NIGERIA
STANDARD TREATMENT GUIDELINES

2ND EDITION, 2016

Standard Treatment Guidelines

NIGERIA | 2016

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AND CLINTON HEALTH ACCESS INITIATIVE

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FOREWORD

I feel honoured to write the foreword to this 2nd edition of the National Standard Treatment Guidelines (NSTG) for the Healthcare delivery system of Nigeria. I am aware that the process for the review of the 1st edition started in 2014 involving support, recommendations and contributions from expert clinicians of various Tertiary Health Institutions and relevant stakeholders in the health care sector. For the NSTG to maintain its relevance in the healthcare delivery system in treating prevalent diseases, there is need for regular review.

The NSTG consists of systematically developed statements designed to assist practitioners and patients in making decisions about appropriate healthcare in clinical practice. The document also identifies, evaluates, summarizes the highest quality of evidence and provides the most current information on the diagnosis, therapy with appropriate dosage of medications including its risk/benefit and cost effectiveness. It also provides information on the prognosis and importantly the prevention of diseases.

The document has additional objectives such as to standardize medical care, to raise quality of care, to reduce several kinds of risks and to achieve the best balance between cost and medical parameters.

It is expected that this document will be well utilized by every Healthcare Provider at the designated levels of our Healthcare system including the private sectors.

I am also aware that the process for the development of this document strictly followed due process where conflict of interest forms were filled by the experts prior to the review exercise. Wider consultations and series of meetings were carried out involving various stakeholders and interest groups where inputs and submissions were considered for inclusion into the current NSTG. Without doubts, this document will be utilized as a quick guide and reference in the management of patients by Doctors and equally serve as a training manual for medical students and other Healthcare Providers towards improving the Health indices of Nigeria.

I feel proud to commend the Committee, Experts and the Secretariat who worked day

and night, driven by real commitment towards the completion of the 2nd edition of NSTG. And appreciation goes to World Health Organization (WHO) and Clinton Health Access Initiative (CHAI) as well as other partners for supporting the review exercise.

Finally, I wish to assure all, of the Ministry's support regarding the wide circulation of this document for use by Healthcare Providers.

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June, 2016.

PREFACE

This is the second edition of the Nigerian Standard Treatment Guidelines, the revision coming up eight years after the first edition due to logistic reasons. The guidelines provide the standard of care for the Nigerian population taking also into consideration in a holistic manner the peculiarities and local circumstances in the health sector, the personnel as well as the resources available for provision of healthcare.

As was the case for the first, enormous effort has been made to improve the standard and appeal of this edition. Expert clinicians have taken time and painstaking effort to update the various sections ensuring evidence based, state of the art and international best practices. In order to accommodate the tertiary level of healthcare, due consideration has been given to a number of medicines so as to cover the scope of medicines seen at this level of care. These medicines will be reflected in the appropriate section of the National Essential Medicines list to prevent irrational use. Furthermore, the choice of medicines have been based on evidence of effectiveness, safety and cost considerations in as much as this does not compromise the health of Nigerians.

In this revision the Chapters have been increased to include geriatric medicine – a specialty of growing importance with the aging population, a separate chapter for mental health, important concerns in

therapeutics and toxicology and relevant information in the appendices. New information following paradigm shifts will be found in chapters covering endocrinology, infectious diseases, respiratory medicine and rheumatology. Due attention have been given to the public health programmes and the dynamics of their therapy
The need for adequate distribution and use of this guideline to foster rational use of medicines is paramount and should be encouraged at all levels of healthcare. Healthcare providers should also note that while the guidelines provide a schema for practice, the need to individualize therapy when the need arises must be borne in mind with a sense of responsibility and without any prejudices. The Committee take due responsibility for any shortcomings and request to be duly informed to ensure correction and improvement of later editions.

I note with due gratitude the support of the Honourable Minister of Health to the Committee. I appreciate with thanks the contribution of the various experts and programme managers, directors and other relevant staff in the Ministry of Health. The support of the World Health Organisation, the Clinton Health Initiative is appreciated with thanks.

Professor Ambrose O. Isah MBBS, FMCP, FWACP, FRCP (Edin.), MD (UK)

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GASTROINTESTINAL AND HEPATOBILIARY DISORDERS

AMOEBIASIS

Introduction

A common parasitic infection of the gastrointestinal system caused by the *protozoan Entamoeba histolytica*

Acquired through faeco-oral transmission.

Clinical features

It may present as:

Amoebic dysentery

Persistent mucoid/bloody diarrhoea

Abdominal pain

Fever/chills

Amoebic abscess

This can occur in any of the following forms as a result of spread via the blood stream:

Liver abscess: swelling, pain in the right sub-costal area

Intracranial space-occupying lesion

Lungs: cough and blood stained sputum

Amoeboma: swelling anywhere in the abdomen

Anal ulceration: may occur by direct extension from the intestinal infection

Chronic Carriers
Symptom-free

Differential diagnoses
Bacillary dysentery
Any other cause of bloody diarrhoea
Cancer of the liver
Other causes of liver enlargement

Complications
Rupture of abscess into the lungs, peritoneum, spacespace-occupying lesion in the brain, right inguinal mass

Investigations
Stool: microscopy for cysts and motile organisms (amoebic dysentery)
Full Blood Count
Serology: amoebic precipitin (if available)
Chest radiograph
Abdominal ultrasound scan (in amoebic liver abscess)

Treatment objectives
Rehydrate adequately
Eradicate the protozoa

Drug treatment

Amoebic dysentery

Correct dehydration (see section on rehydration)

Metronidazole

Adult: 800 mg 8 hourly for 5 days

Child: 30 mg/kg/day in 3 divided doses for 5 days

Or:

Tinidazole

Adult: 2 g daily orally for 3 days (with food)

Child: 50-60mg/kg daily for 3 days

Amoebic liver abscess

Metronidazole

Adult: 800 mg 8 hourly for 10 days

*Child:50*mg/kg/day in 3 divided doses for 7-10 days

Tinidazole

Adult: 2 g daily orally for 3 days (with food)

Child: 50-60mg/kg daily for 5 days

Non-drug treatment

Aspiration is indicated to prevent spontaneous rupture of abscesses.

Consult a surgeon.

Asymptomatic cyst carriers

Treat cyst carrier if patient is a food handler:

Diloxanide furoate

Adult: 500 mg every 8 hours for 10 days

Child: over 25 kg -:20 mg/kg orally every 8 hours for 10 days;under 25kg(I month – 12 years)- 6.6mg/kg every 8 hours for 8 -10 days; 12 years – 18 yeas-500mg every 8 hours for 10 days

Notable adverse drug reactions, caution

Metronidazole is contraindicated in pregnancy.

Avoid alcohol during and at least 48 hours after treatment.

Prevention

Provision of safe drinking water, sanitary disposal of faeces, regular examination of food handlers and appropriate treatment where necessary.

BACILLARY DYSENTRY

Introduction

An important cause of colonic diarrhoea in developing countries.

Caused by pathogenic species of Shigella A-D *(dysenteri, flexneri, boydii and sonnei).*

Transmitted via the faeco-oral route.

Clinical features

Mucoid bloody diarrhoea associated with severe central and lower abdominal pain,

tenesmus
moderate-grade pyrexia
Sometimes only mild, self-limiting diarrhoea lasting for 23 days
Articular features occasionally
Septicaemic spread with multi-system involvement occasionally.

Differential diagnoses
Amoebic dysentery
Idiopathic enterocolits (ulcerative)
Campylobacter jejuni infection
Colorectal cancer
Complications
Septicaemia/bacteraemia
Severe rectal bleeding
Intestinal perforation
Reiter's syndrome

Investigations
Stool microscopy, culture and sensitivity
Full Blood Count
Urea, Electrolytes and Creatinine

Treatment objectives
Adequate rehydration.
Eradicate bacterial pathogens

Drug treatment
Oral Rehydration Therapy (see rehydration under diarrhoea)

Parenteral hydration therapy (see rehydratrion under diarrhoea)

Antibacterial drugs are not usually necessary: even diarrhoeas resulting from bacterial infection are usually self-limiting. Appropriate systemic antibiotics are however required when systemic infections occur.

- Ciprofloxacin 500 mg - 1 g orally 12 hourly for 5 days
- Azithromycin 500 mg daily for 3 days for resistant strains

For Children give

Cotrimoxazole:

6 weeks – 12 years: 24mg/kg every 12 hours

6weeks – 6 months: 120mg every 12 hours

6 months – 6 years: 240mg every 12 hours

6years – 12years: 480mg every 12 hours

12years - 18 years: 960mg every 12 hours

Notable adverse drug reactions

Ciprofloxacin may induce tendinitis especially in children.

Precaution

Ciprofloxacin is not recommended for use in children less than 18 years.

Antidiarrhoeal medicines are not advised.

Prevention
Safe drinking water
Sanitary disposal of human waste material

CHOLERA
Introduction
An acute severe diarrhoeal illness of worldwide importance; endemic in many developing countries.
Caused by *Vibrio cholerae* bacilli (classical and *El Tor* species)
Excessive secretion of fluid is mediated by the release of enterotoxin (released by the bacilli), which acts on the enterocytes of the small intestine via cyclic AMP.
Highly infectious; spread by faeco-oral route.

Clinical features
Mild watery diarrhea, severe life-threatening diarrhea leading to hypovolaemic shock if untreated.
Occasionally, vomiting

Complications
Hypovolaemic shock with multiple end organ failure leading to death
Hypoglycaemia
Paralytic ileus

Investigations
Stool microscopy, culture and sensitivity
Full Blood Count
Urea, Electrolytes and Creatinine

Treatment objectives
Rehydrate adequately and rapidly.
Eradicate the infective organism.
Prevent spread of the infection

Drug treatment
Intravenous Ringer's lactate/Darrow's solutions.
Oral Rehydration Therapy
Antibiotic therapy
Tetracycline:
Adult: 500 mg orally every 6 hours for 5 days

Or:
Doxycycline:
Adult: 200 mg orally once daily for 5 days
Child: 12 - 18 years, 200 mg on first day, then 100 mg daily
- Severe infections, 200 mg orally daily

Erythromycin:
Adult and child over 8 years: 250 - 500 mg orally every 6 hours for 5 days or 500 mg -1 g every 12 hours
Child up to 2 years: 125mg every 6 hours; 2 - 8 years: 250 mg every 6 hours

- Doses doubled in severe infection

Or:

Sulfamethoxazole-trimethroprim (Co-trimoxazole)

Adult: 960 mg orally every 12 hours for 5 days

Child: 6 weeks - 6 months 120 mg 12 hourly; 6 months - 6 years 240 mg; 6 - 12 years 480 mg; 12 - 18 years 960 mg orally every 12 hours for 5 days

Supportive measures

Monitor fluid intake and output (vomitus, urine and stool)

Prevention

Provide access to safe drinking water

Food hygiene

Safe disposal of human waste

Cholera vaccine

CONSTIPATION

Introduction

A clinical condition characterized by infrequent bowel opening and/or passage of hard stools.

Aetiology

Inadequate fibre in diet (simple constipation)

Drugs e.g. antidepressants, narcotic analgesics, etc

Diseases of the anus, rectum and colon e.g.

fissures, haemorrhoids, cancer
Functional: irritable bowel syndrome
Metabolic diseases e.g. hypothyroidism, hypercalcaemia

Clinical features
Stools are often hard
Abdominal bloating
Excessive flatulence. Relevant associated history to determine aetiology should be vigorously pursued
Physical examination should be thorough, and must include a rectal examination

Complications
Megacolon
Anal fissures/tears
Haemorrhoids
Rectal bleeding

Investigations
Stool examination including microscopy.
Proctoscopy/sigmoidoscopy.
Barium enema
Serum hormonal levels e.g. thyroxine, triiodotyronine, thyroid stimulating hormone to exclude hypothyroidism

Treatment objectives
Identify and eliminate cause(s)
Evacuate hard faecal matter

Indications for use of laxatives

Situations where straining will exacerbate pre-existing medical/surgical conditions

- Angina
- Risk of rectal bleeding
- Increased risk of anal tear

Other indications

- Drug-induced constipation
- To clear the alimentary tract before surgery or radiological procedures

Non-drug treatment

Avoid precipitants

High fibre diet (including fruits and vegetables).

Adequate fluid intake.

Megacolon: Saline enema

Surgical: resection of large bowel

Drug treatment

Stimulant laxatives

- Senna 7.5 mg tablet (as sennoside B)

Adult: 2 - 4 tablets at night

Child 6-12years: 1 -2 tablet stat at night (or in the morning if preferred)

12 - 18 years: 2 - 4 tablets at night

Or:

Bisacodyl tablets 10 mg orally at night; suppositories 10 mg per rectum at night

Caution

Laxatives should generally be avoided. Most times these drugs are needed for only a few days

In children, laxatives should be prescribed by a healthcare professional experienced in the management of constipation in children.

DIARRHOEA (Acute)

Introduction

A very common clinical problem the world over, particularly in developing countries.

Accounts for significant morbidity and mortality, especially in children.

Infective agents are recognized in about 70% of cases and are transmitted by the faeco-oral route.

Viruses (particularly Rotavirus) are responsible for over 70% of diarrhoeas in children below 2 years.

Many bacteria and some parasites are also important aetiologic agents, particularly in adolescents and adults.

Endemic and epidemic presentations can occur. Contamination of food and water by bacterial toxins can also lead to acute diarrhoea, sometimes with associated vomiting (i.e. food poisoning). This is usually self-limiting.

Clinical features

Watery diarrhoea of varying volumes, sometimes with vomiting: this is the commonest presentation, and suggests pathology in the small intestine.
Bloody mucoid stools: suggests disease in the colon
Fever, abdominal pain and dehydration
Fast and small volume pulse with low blood pressure: indicates significant fluid loss

Complications
Hypovolaemic shock with multiple organ failure
Septicaemia
Intestinal perforation
Gastro-intestinal bleeding
Paralytic ileus

Differential diagnoses
Non-infectious diarrhoea e.g. drug-induced
Gut allergy (e.g. gluten).
Psychogenic stress.
Metabolic and endocrine causes (e.g. thyrotoxicosis, uraemia, diabetes mellitus)

Investigations
Stool examination including microscopy, culture and sensitivity
Full Blood Count
Urea, Electrolytes and Creatinine
Serology (e.g. Widal test)

Treatment objectives

Achieve adequate hydration.

Eliminate infectious agent (where possible).

Treat complications

Drug treatment

Rehydrate with:

Oral Rehydration Therapy - ORT (low osmolarity) for mild to moderate dehydration

- 500 mL orally over 2 - 3 hours, 3 - 4 times daily

Intravenous sodium chloride 0.9%

- 1 litre 2 - 6 hourly for moderate-to- severe dehydration
- Alternate with Darrow's solution depending on serum potassium

Children: Use of zinc supplementation

- 20 mg per day for 10 - 14 days
- Under 6 months old: 10 mg per day

Specific anti-infective agents for infectious diarrhoeas e.g. metronidazole for amoebiasis, giardiasis

Supportive measures

Monitor fluid intake/output

Notable adverse drug reactions

Heart failure: from overhydration. Initial increase in diarrhoea with ORT: this is self limiting

Hyperkalaemia: from excessive use of

potassium-containing fluids

Prevention

Provide access to safe drinking water

Sanitary disposal of human waste

Personal hygiene: hand-washing, care in food-handling

GASTRITIS

Introduction

Inflammation of the gastric mucosa.Can be acute or chronic. The most important risk factors for acute gastritis include use of drugs (NSAIDs in particular) and alcohol.

H.pylori infection is the most important risk factor for chronic gastritis

All agents of gastritis work through the common path of disrupting the protective mucosal barrier of the stomach.

Acute gastritis may evoke pain that mimics peptic ulcer disease. Chronic gastritis is a precursor of peptic ulcer disease (type B gastritis) and gastric cancer (type A gastritis).

Clinical features

Chronic gastritis is essentially asymptomatic.

Acute gastritis evokes acute abdominal pain that mimics peptic ulcer disease (see peptic ulcer disease)

Occasionally acute gastritis may be haemorrhagic with melaenal stools or rarely

haematemesis

Complications
Acute gastritis: haemorrhage.
Chronic gastritis: peptic ulcer disease; gastric cancer
Differential diagnosis
Peptic ulcer disease (acute gastritis)

Investigations
Endoscopy (macroscopic diagnosis).
Histology of gastric biopsy for definitive diagnosis
Treatment objectives
Eliminate pain (acute gastritis)
Prevent progression to peptic ulcer disease or gastric cancer
Re-establish normal histology

Drug treatment
Acute Gastritis:
Antacids
- Magnesium trisilicate 1 - 2 tablets or suspension 10 mL orally three times daily or as required

Or:
H receptor antagonist 2
- Ranitidine 150 mg orally once daily as required

Or:

Proton pump inhibitors

- Omeprazole 20 mg orally once daily as required

Type A gastritis:

Endoscopic surveillance every 2 - 3 years for early detection of cancer

Type B gastritis: Eradication of H.*pylori* using triple therapy with

- Clarithromycin 500 mg orally twice daily for 7 days

Plus:

- Amoxicillin 1g orally every 12 hours for 7 days

Plus:

- Omeprazole 20 mg orally every 12 hours for 7 days

Prevention

Avoid risk factors (NSAIDs, alcohol, etc)

GIARDIASIS

Introduction

A parasitic infection caused *by Giardia lamblia.* Worldwide in distribution but more common in ***developing countries. Spread by the faeco-oral route.***

Pathogenesis

Invasion of the upper small intestine by the parasite evokes inflammation, leading to

progressive villous atrophy.

Clinical features
Acute disease: watery diarrhoea with abdominal bloating
Chronic disease: diarrhoea, steatorrhoea and weight loss from malabsorption syndrome-with lactose intolerance, xylose malabsorption and vitamin B deficiency

Complications
Diseases related to Vitamin B deficiency

Differential diagnoses
Other causes of upper gastrointestinal malabsorption such as coeliac disease and tropical sprue

Investigations
Full blood count
Stool microscopy and faecal fat assessment
Jejunal biopsy

Treatment objectives
Rehydrate adequately
Eradicate parasite
Replace malabsorbed (deficient) nutrients

Drug treatment
Metronidazole
Adult: 2g orally daily for 3 days or 400mg 8

hourly for 5 daysor 500mg twelve hourly for 7 -10 days

Child: 1 - 3 years 500 mg orally daily; 3 - 7 years 600 -800 mg daily; 7-10 years 1 g daily for 3 days Tinidazole

Adult: 40 mg/kg orally as a single dose;or 2g stat, repeat after 1 week,once if necessary

Child: 50 to 75 mg/kg as a single dose; repeat after 1 week

Supportive

Vitamin B supplementation12 Avoidance of milk

Notable adverse drug reactions

Metallic taste and vomiting from metronidazole

Prevention

Good sanitary habits,proper hand washing and/or use of alcoholic hand sanitizers

Uncontaminated water and food supplies

HAEMORRHOIDS

Introduction

Enlarged or varicose veins of the tissues at the anus or rectal outlet.

Engorgement of the vascular complex or thrombus often leads to the symptoms of disease.

The pathophysiologic mechanisms are

complex and vary with the subject. May be external or internal.

Clinical features
Internal haemorrhoids: typically painless but present with bright red rectal bleeding
May become thrombosed and protrude into the anal canal
External haemorrhoids when thrombosed cause acute perineal pain with or without necrosis and bleeding. Fibrosed external haemorrhoids present as anal tags

Differential diagnoses
Colorectal cancer.
Adenomatous polyps.
Inflammatory bowel disease

Complications
Bleeding, necrosis, perineal sepsis, mucus discharge

Investigations
Anoscopy
Full blood count including blood film

Treatment objectives
Relieve pain and prevent complications

Non-drug treatment
Increase fibre in foods

Increase fluid intake
Avoid foods that cause constipation
Stool softeners
Regular exercise

Drug treatment

Suppositories/ointments of preparations containing hydrocortisone acetate with or without lidocaine hydrochloride plus astringent(s)

Surgery

Elastic band ligation
Sclerosis, photocoagulaton, cryosurgery, excisional haemorrhoidectomy

Caution

Each drug treatment course should not exceed 7 days

PANCREATITIS

Introduction

A state of inflammation of the pancreas, which can be acute or chronic.

Aetiology

Varied, but most important are: Gallstones, alcohol ingestion, abdominal, trauma, Infections
Idiopathic in as many as 20-30% cases
Occurrence is worldwide, but commoner in areas of the world where gallstones and

alcohol ingestion are common.

Pathophysiology
Autolysis of pancreatic tissue by pancreatic enzymes as a result of "secretory block" in the pancreatic bed (often caused by stones).

Clinical features
Acute pancreatitis:
Epigastric pain which may radiate to the back in over 50% of cases
Nausea, vomiting, abdominal distension, severe abdominal tenderness with features of hypovolaemia in severe cases

Differential diagnoses
Peptic ulcer disease.
Cholecystitis

Investigations
Serum amylase: raised in 80% of acute cases.
Serum lipase: if raised is more specific than serum amylase
Alanine aminotransferase: a rise above 3-fold suggests pancreatitis of gallstone origin.
CT scan
Abdominal ultrasound: least useful in acute pancreatitis

Complications
Hypovolaemic shock

Acute renal and respiratory failure
Phlegmos
Gastrointestinal bleeding
Electrolyte imbalance (hypo & hypercalcaemia)
Pancreatic pseudocysts

Treatment objectives
Relieve pain
Prevent complications
Non-drug treatment
Renal failure: haemodialysis.
Respiratory failure: mechanical ventilation.
Gallstones: Endoscopic Retrograde Cholangio Pancreatography (ERCP) with sphincterotomy. Pancreatic pseudocyst: surgery

Drug treatment
Analgesics
Treat specific complications

Supportive measures
Bed rest
Monitor vital signs; fluid intake/output
Nasogastric tube suctioning.
Decrease pancreatic inflammation.
Prevent, identify and treat complications

Caution
Avoid narcotic analgesics, which may cause

spasm of the sphincter of Oddi and worsen pancreatitis

Prevention
Control alcohol ingestion

PEPTIC ULCER DISEASE

Introduction
Caused by peptic ulceration that involves the stomach, duodenum and lower oesophagus. An increasingly common problem in developing countries.
Most ulcers are duodenal

Aetiology/Predisposing factors
H pylori gut infection
Use of NSAIDs
Smoking

Clinical features
Recurrent epigastric pain
- Often radiating to the back
- Worse at night
- Improved by antacids
- May be made worse by some food types (general better with bland diet)

Complications
Upper gastrointestinal bleeding
Perforation
Penetration

Gastric outlet obstruction
Gastric cancer

Investigations
Full Blood Count
Liver Function Tests, Urea, Electrolytes and Creatinine
Occult blood test. Stool microscopy
Endoscopy, Double contrast barium meal
Direct/indirect detection of H. *pylori (by* CLO test or by CO_2 breath test)

Differential diagnoses
Gastritis
Duodenitis
Non-Ulcer Dyspepsia
Gastro-duodenal malignancy
Oesophagitis
Gall bladder diseases

Treatment objectives
Relieve pain
Promote healing of ulcers
Eradicate *H. pylori*
Prevent/reduce recurrence

Drug treatment
Symptomatic treatment with antacids may be used prior to confirming the diagnosis of peptic ulcer disease
H. pylori eradication

Triple therapy with:

- Clarithromycin 500 mg orally every 12 hours for 7 days

Plus:

- Amoxicillin 500 mg orally every 8 hours for 7 days

Plus:

- Omeprazole 20 mg orally every 12 hours for 7 days

Adjunct therapy

Magnesium trisilicate suspension 15 mL orally three times daily as required

Supportive therapy

Regular meals

Avoidance of provocative factors (NSAIDs, alcohol, spicy foods etc.)

Notable adverse drug reactions

Gastric irritation, diarrhoea from triple therapy

Diarrhoea/constipation from adjunct therapy

Treatment of complications

Mild upper gastrointestinal bleeding

Intravenous omeprazole 20 mg 12 hourly for 5 days then standard triple therapy

Severe upper gastrointestinal bleeding

Interventional endoscopic treatment

Blood transfusion
Surgery Perforation
Surgery Gastric outlet obstruction
Rest the gut
Surgery

HEPATIC AND BILIARY DISORDERS

HEPATITIS

Introduction

Inflammation of the liver that can be caused by infective agents, drugs and other toxins
The most predominant and important presentation of liver disease worldwide
The suffixes acute, chronic, viral, autoimmune, alcoholic etc. define the agents causing hepatic injury or their duration as the case may be

Aetiology

Varies, depending on geographical region.Hepatitis A, B and C Viruses, alcohol and drugs are the commonest aetiologic agents

Important risk factors

Family history
Alcohol ingestion
Previous blood transfusion
Contamination of food and water by sewage
Drug ingestion

Sexual contact
Exposure to contaminated sharp objects/instruments

Clinical features
Acute hepatitis:
Mild-to-moderate jaundice
Vague right upper quadrant discomfort
With or without mild fever
There may be enlargement of the liver below the costal margin with varying consistency (depending on the stage of the liver disease)
Chronic hepatitis:
Re-occurence of jaundice may suggest a chronic illness

Differential diagnoses
Liver abscess
Metabolic liver disease/disorder
Primary or secondary liver cancer
Cholecystitis

Complications
Fulminant hepatic failure
Bleeding tendencies
Investigations
Liver Function Tests
Serologic markers of Hepatitis A, B, C, D and E
Abdominal ultrasonography

Treatment objectives
Provide supportive measures
Prevent progression to chronic phase (applicable to Hepatitis C virus only)

Non-drug treatment
High carbohydrate and low protein diet
Discontinuation of hepatotoxic medication
Bed rest
Drug treatment
Hepatitis A
Self-limiting disease.
No specific drug treatment

Hepatitis B
Acute:
Self-limiting to fulminant
Treatment is supportive
Chronic:
Treat only those with viral load exceeding 2000 International Units/ml for e antigen negative patients or 20000 International Units/ml for e antigen positive patients.
Monitor those not meeting treatment criteria with bi annual viral load.
Pegylated interferon alfa -2b:180 microgram subcutaneously weekly for 48 weeks.
Tenofovir 300 mg daily for 2-5 years (used when patient has liver failure)
Liver transplant

Chronic Hepatitis C:
Pegylated interferon alfa -2b:180 microgram subcutaneously weekly for 48 weeks.
Plus:
Ribavirin

- 400 mg orally twice daily for adults with body weight less than 65 kg; 400 mg in the morning and 600 mg in the evening for adults weighing 65-85 kg; 600 mg twice daily for adults weighing over 85 kg

Hepatitis D
Interferon alfa -2b: 3 million units subcutaneously 3 times weekly for 4 months
Plus:
Lamivudine: 100 mg orally once daily for 4 months
Hepatitis E
Largely supportive

Notable adverse drug reactions
Interferon alpha 2b and Ribavirin reduce haematopoiesis
Flu-like illness
Leucopenia
Psychiatric-like symptoms
Development of early resistance if therapy

exceeds one year

Prevention

Prevention of faecal contamination of food and water

Screen blood and blood products for hepatotrophic viruses

Immunization against hepatitis A, B

Reduction of drug misuse/abuse

Pre-exposure prophylaxis (as for NPI/EPI)

Post-exposure prophylaxis.

Avoid exposure to unsterilized sharps

JAUNDICE

Introduction

A common clinical state of varying aetiologies.Classified as haemolytic, hepatic or obstructive Clinical jaundice occurs when the level of serum bilirubin exceeds 2.5 mg/dL. The bilirubin may be conjugated, unconjugated or mixed

Important causes

Diseases of the liver and the biliary tract

Conditions that cause excessive red cells haemolysis: infections, haemoglobinopathies

Clinical features

Discolouration of the sclerae and other mucus membranes

Associated general pruritus (especially with cholestatic jaundice)

Associated features of the underlying disease

Investigations

LFTs: determine levels and nature of bilirubin, liver enzymes (AST, ALT, Alkaline phosphotase)

Abdominal ultrasound scan: look out for canalicular dilatations, biliary stones

Treatment objectives

Treat underlying cause

Prevent complications

Drug treatment

Specific treatment depends on the identified cause

Colestyramine:

- 3-6g orally 6 hourly in severe obstructive jaundice

Phenobarbital in neonatal jaundice:

- 5-8mg/kg orally daily

Notable adverse drug reactions

Colestyramine: diarrhoea

Phenobarbital may cause dose-dependent respiratory depression

Surgical treatment

Obstructive jaundice

ERCP sphincterotomy with stone removal

Stent insertion

Pancreatic head/duodenal head realignment

Supportive measures
Reassurance and monitoring
Phototherapy in neonatal jaundice

LIVER CIRRHOSIS

Introduction
An advanced stage of chronic liver disease associated with permanent distortion of the liver architecture and replacement of some destroyed hepatocytes with fibrous tissue
Accompanied by some loss of liver function leading to certain recognized symptoms and signs

Aetiology
Similar to some causes of acute liver diseases
No known aetiology in up to 30% of cases

Clinical features
Varies with the extent of liver damage:
Fatigue
Ascites
Pedal oedema
Haematemesis
Liver may be shrunken or enlarged below the costal margin; it is typically firm

Differential diagnoses
Granulomatous lesion of the liver
Primary or secondary neoplasms of the liver

Complications
Intractable oedema
Upper gastrointestinal tract bleeding
Coagulopathy
Hepatic encephalopathy
Hepato-renal syndrome

Investigations
LFTs and Serum proteins
PT, PTTK,
Liver biopsy
Ultrasound examination of the liver
Screening for aetiologic factors in chronic liver disease e.g. viral markers for hepatotrophic viruses (e.g. Hepatitis B & C)

Treatment objectives
Prevent further liver damage
Prevent deterioration of liver function
Symptomatic relief from anaemia, fatigue and oedema

Non-drug treatment
Encourage high fibre and low salt diet
Enhance opening of bowel
Correction of anaemia
Reduce oedema and ascites

Drug treatment
Ascites and pedal oedema:

Spironolactone tablets 25 - 100 mg orally 12 hourly
Furosemide 20 - 80 mg orally 12 hourly
Salt-poor albumin for intractable ascites
Prevention of variceal bleeding:
Propranolol 40 - 80 mg orally daily
Isosorbide mononitrate 10mg orally thrice daily
Replacement of damaged liver:
Liver transplant
Prevention of encephalopathy
Institute measures as outlined in section on hepatic encephalopathy

Prevention
Immunization against hepatitis B, C
Abstinence from alcohol

NUTRITIONAL DISORDERS

KWASHIORKOR AND MARASMUS
Introduction
Adequate nutrition is the intake and utilization of energy-giving and body building foods and nutrients in the right proportions to maintain well-being, and productivity.
Malnutrition manifests as stunting, underweight, wasting (kwashiorkor and marasmus), obesity as well as deficiencies of

micronutrients.

Epidemiology
High prevalence in under-developed countries, especially sub-Saharan Africa

Clinical features
Kwashiorkor: Growth retardation, muscle wasting, anaemia, apathy, moon face, lack-luster skin easily plucked hair, pedal oedema, hypo-pigmented skin patches, exfoliation, diarrhoea Marasmus: Thin; protruding bones, hungry-looking 'old-looking face,' whimpering cry

Investigations
Full Blood Count, ESR
Stool microscopy, Urinalysis, Serum proteins, Chest radiograph, Mantoux test

Non-drug treatment
Nutritional counselling
Adequate nutrient intake: may require assistance and special preparations e.g. nasogastric feeding, Periodic growth monitoring

Drug treatment
May be indicated where there are specific infections/infestations

MICRONUTRIENT DEFICIENCIES

Definition

Deficiencies of minerals (iron, iodine, zinc, calcium, phosphorus, magnesium, copper, potassium, sodium, chloride, fluoride etc); folic acid and vitamins

Aetiology

Inadequate dietary intake
Increased requirements
Increased loss (e.g. worm infestation)

Epidemiology

Global; high prevalence in under-developed countries, especially sub-Saharan Africa

Clinical features

Iron: anaemia
Iodine: goitre
Zinc, copper: manifestations of enzyme and insulin deficiencies
Calcium: rickets, osteomalacia
Phosphorus and fluoride: teeth and bone abnormalities
Vitamins:

- A: keratomalacia, corneal xerosis, night blindness
- B (thiamine): beri-beri
- B (riboflavin): scrotal and vulval dermatoses, angular stomatitis, scars, magenta tongue, cheilosis

- B (niacin): scarlet and dry tongue, pellagra 6
- Ascorbic acid: scurvy, petechiae and musculo-skeletal haemorrhages
- D: rickets, epiphyseal enlargement, muscle wasting, bossing of skull bone, 'thoracic rosary', persistently open anterior fontanelle, genu valgum or varum

Investigations
Blood, urine and stool tests. Other investigations as appropriate

Treatment objectives
Correct nutrient deficiencies
Ensure adequate intake
Prevent complications

Treatment
Administration of specific nutrients (as concentrates in foods)
Food supplementation
Treat underlying diseases

Prevention
Nutritional counselling
Optimal breastfeeding and appropriate weaning practices
Adequate intake of locally available, nutritious foods

Personal/food/water hygiene
Prophylactic therapies for malaria

OBESITY

Introduction

A major component of the metabolic syndrome.

Being overweight or obese significantly increases the risk of morbidity and mortality from Type 2 diabetes and its co-morbidities.

Successful weight reduction has a positive impact on morbidity and mortality outcomes.

Constitutional obesity is a result largely of diet and lifestyle.

Measurements for evaluation

Body mass index (BMI): calculation for overall obesity

BMI = weight in kg divided by height in m^2, expressed as kg/m^2

Waist circumference: determination of central fat distribution

Classification of BMI

Underweight: <18.5 kg/m^2

Normal weight: 18.5 - 24.9 kg/m^2

Overweight: 25 - 29.9 kg/m^2

Obesity (Class1): 30 - 34.9 kg/m^2

Obesity (Class 2): 35 - 39.9 kg/m^2

Extreme obesity (Class 3): > 40 kg/m^2

The pattern of distribution of fat in the body

(whether mostly peripheral or central) is assessed by the use of the waist/hip ratio (WHR)

Waist/Hip ratio=Waist circumference (in cm) divided by Hip circumference (in cm)

Waist circumference: measured midway between the lower rib margin and the iliac crests

Hip circumference: the largest circumference of the hip

Waist circumference better depicts central or upper body obesity than waist/hip ratio

Upper limits: 102 cm and 88 cm in men and women, respectively

Investigations

Non-specific

- Always bear in mind the possibility of an underlying cause: although these may not be common, specific therapy may be available
- Clinical presentation may therefore require specific investigations to exclude conditions such as hypothyroidism, hypercortisolism, male hypogonadism, insulinoma and CNS diseases that affect hypothalamic function

Complications

Cardiovascular: Coronary artery disease

Stroke

Congestive heart failure
Pulmonary:
Obstructive sleep apnoea, 'Obesity hypoventilation syndrome'
Endocrine: Insulin resistance and type 2 diabetes mellitus
Hepatobiliary: Gall stones
Reproductive: Male hypogonadism; Menstrual abnormalities; Infertility
Cancers:
In males, higher mortality from cancer of the colon, rectum and prostate
In females, higher mortality from cancer of the gall bladder, bile ducts, breasts, endometrium, cervix and ovaries
Bone, joint and cutaneous disease: Osteoarthritis
Gout
Acanthosis nigricans
Increased risk of fungal and yeast infections
Venous stasis

Treatment objectives
To educate patient and care givers
Achieve an ideal body weight
Prevent complications

Management
Assess dietary intake, level of physical activity, BMI (total body fat) and waist circumference (abdominal fat) on

presentation and at regular monitoring
Assess efficacy of weight loss measures
Integrate weight control measures into the overall management of diabetes mellitus and co-morbidities if BMI is >25, and Waist circumference is more than 102 cm and 88 cm in men and women respectively
Educate patients and other family members
Set realistic goals
Use a multi-disciplinary approach to weight control
Dietary changes and increased level of physical activity are the most economical means to loose weight
Maintain records of goals, instructions and weight progress charts
Surgical intervention may be required in extreme cases

CHAPTER 2:

HAEMATOPOIETIC SYSTEM DISORDERS

ANAEMIAS

Introduction

Anaemia is a reduction in the haemoglobin concentration in the peripheral blood below the normal range expected for the age and sex of an individual

The determination of haemoglobin concentration should always take the state of hydration and altitude of residence of the individual into consideration

Anaemia can be classified on the basis of red cell morphology and aetiology/pathogenesis

Morphological classification

Macrocytic Megaloblastic:

- Folic acid deficiency
- Vitamin B deficiency 12
- Inherited disorders of DNA synthesis
- Non-megaloblastic:
- Accelerated erythropoiesis
- Increased membrane surface area
- Obscure

Hypochromic-microcytic:

Iron deficiency
Disorders of globin synthesis
Other disorders of iron metabolism
Normochromic-normocytic:
Recent blood loss
Haemolytic anaemias
Hypoplastic bone marrow
Infiltrated bone marrow
Endocrine abnormality
Chronic disorders
- Renal disease
- Liver disease

Classification based on aetiology and pathogenesis

Blood Loss: Acute

Chronic (leads to iron deficiency) Increased red cell destruction (haemolytic anaemias):

Corpuscular defects (intracorpuscular or intrinsic abnormality)

Disorders of the membrane e.g. elliptocytosis, spherocytosis

Disorders of metabolism e.g. Glucose-6-Phosphate dehydrogenase deficiency, Haemoglobinopathy e.g. sickle cell disease

Paroxysmal nocturnal haemoglobinuria.

Abnormal haemolytic mechanisms (extra-corpuscular or intrinsic abnormality)

Autoimmune

Rhesus-incompatibility, mismatched transfusion, hypersplenism

Infections e.g. malaria, *Clostridium welchii*, drugs and toxins

Others e.g. burns decreased red cell production

Nutritional (due to deficiencies of substances essential for erythropoiesis)

- Iron
- Folate
- Vitamin B12
- Various deficiencies e.g. protein, ascorbic acid

Bone marrow stem cell failure:

Primary (idiopathic):

- Aplastic anaemia
- Pure red cell aplasia Secondary:
- Drugs (phenylbutazone, cytotoxic agents, etc)
- Chemicals
- Irradiation

Anaemias associated with systemic disorders:

Infection
Liver disease
Renal disease
Connective tissue disease
Cancer (including leukaemia)
Marrow infiltration
Thyroid or pituitary disease

Clinical features

Depend on the degree of anaemia, severity

of the causative disorder and age of the patient

The clinical effects of anaemia are due to anaemia itself and the disorder(s) causing it.

Common:

Tiredness, lassitude, weakness, dyspnoea on exertion, palpitations, pallor

Less common:

Angina of effort, faintness, giddiness, headache, ringing in the ears, high output state congestive cardiac failure.

Differential diagnoses

Cardiac failure

Respiratory failure

Complications

Cardiac failure

Investigations

Haematologic: Haematocrit, haemoglobin concentration, red cell indices, reticulocyte count, total leukocyte and differential counts, platelet count

Erythrocyte sedimentation rate, Blood film examination for morphology of cells, thick and thin films for malaria parasites.

Urine analysis: Colour, pH, clarity, and specific gravity, microscopic examination of fresh urine specimen, protein, glucose, occult blood

Stool: Colour, consistency, examination for ova and parasites, occult blood
Plasma: Blood Urea Nitrogen (BUN), Total protein and albumin bilirubin, creatinine (if BUN is abnormal)
Others:
Coombs test for the presence of antibodies to red cells
Ham's test (acidified serum test)
Bone marrow aspiration and trephine biopsy
Haemoglobin electrophoresis
Sickling test (metabisulphite and solubility)
Family studies

Treatment objectives
Restore haemoglobin concentration to normal levels.
Prevent/ treat complications
Supportive measures
Bed rest in severe cases: initially necessary, especially when cardiovascular symptoms are prominent.
Treat cardiac failure by standard measures.
Balanced diet with adequate protein and vitamins.
Correct dietary deficiencies (e.g. iron, folic acid)
Blood transfusion: a very important measure in the treatment of anaemia, but should not be used as a substitute for investigation, or specific treatment of the cause

Arrest blood loss
Treat any underlying systemic disorder.
Remove any toxic chemical agent or drug.
Correct anatomical gastro-intestinal abnormalities

Drug treatment
Haematinics e.g. iron, vitamin B_{12}, folic acid.
The specific haematinic indicated should be given alone
Response to adequate treatment is important in confirming diagnosis
Iron deficiency:
Oral iron therapy:
- Ferrous sulfate 200 mg (containing 65 mg of iron) tablet 2-3 times daily

Treat for 3- 6 months to correct deficits in haemoglobin
Parenteral therapy:
Not necessary unless there is intolerance to oral iron
Indications for parenteral iron:
Anaemia diagnosed in late pregnancy.
Correction of anaemia just before an operative procedure
Haemorrhage expected to continue unabated
Iron preparations:
Iron dextran given as "total dose" infusion
By deep intramuscular injection into the gluteal muscle or by slow intravenous injection or by intravenous infusion,

calculated according to body weight and iron deficit dose in mL (of 50 mg/mL preparations) = [Patient's wt. in kg X (14 Hb in g/dL)]-10

Periodic hematologic determination of haemoglobin and hematocrit is a simple and accurate technique for monitoring haematological response and should be used as a guide in therapy. Evidence of therapeutic response can be seen in a few days as an increase in reticulocyte count

Notable adverse drug reactions, caution

Oral iron preparations:

Nausea, epigastric pain, diarrhoea, constipation, skin eruptions

Reduce dosage and frequency of administration to reduce these effects

Parenteral iron:

Local reactions: phlebitis and lymphadenopathy

Systemic reactions: may be early or late-headache, fever, vomiting; general aches and pains, backache, chest pain, dyspnoea, syncope; death from anaphylaxis

Test doses are no longer recommended but caution is needed with every dose of intravenous iron Patients should be monitored for signs of hypersensitivity during and for at least 30 minutes after every administration

Total-dose infusion should be avoided in patients with history of allergy

Not recommended for children under 14 years

Avoid in first trimester and use in the second and third trimesters only when the benefit outweighs the potential risks for both mother and fetus

Anaphylaxis and other Hypersensitivity can occur with parenteral iron and facilities for cardiopulmonary resuscitation must be available; Discontinue oral iron prior to administration of iron dextran injection.

Oral iron should not be given until 5 days after last injection

Megaloblastic anaemia

Response to therapy is satisfactory if administered dose is limited to the minimal daily requirement

Treatment with vitamin B_{12}(cobalamin) to replace body stores

- Six-1000 micrograms intramuscular injections of hydroxocobalamin given at 3 -7 day intervals

Maintenance therapy: patients will need to take vitamin B_{12} for life

- 1000 micrograms hydroxocobalamin intramuscularly once every 3 months

Notable adverse drug reactions, caution

Toxic reactions are very rare and are usually not due to cobalamin itself

Pharmacologic doses of folic acid produce haematological response in vitamin B_{12} –deficient

patients but worsen the neurological complications

Large doses of vitamin B_{12} also give haematological response in folate-deficient patients

Prevention

Balanced diet

Prompt treatment of all illnesses

BLOOD TRANSFUSION

Introduction

Blood transfusion is the administration of blood for therapy

It is potentially hazardous: blood should be given only if the dangers of not transfusing outweigh those of transfusion.

Indication(s) must be clearly established. Transfusion of whole blood or red cell concentrates is important in the treatment of acute blood loss and of anaemia.

Red cells can be stored at 4°C for 5 weeks in media that are specially designed to maintain the physical and biochemical integrity of the erythrocytes and which maintain their viability after transfusion.

Citrate Phosphate Dextrose with Adenine (CPDA) is commonly used for collections of whole blood.

The use of whole blood as a therapeutic agent has been almost completely replaced by the use of blood fractions.

Types of blood transfusion

Autologous blood transfusion: Transfusion of the patient's own blood to him/her

- Safest blood for patients

The three main types are:

- Pre- deposit autologous transfusion
- Immediate pre-operative phlebotomy with haemodilution
- Intra-operative blood salvage

Exchange transfusion:

To remove deleterious material from the blood, for example, in severe jaundice resulting from haemolytic disease of the newborn

Alternatives to red cell transfusion:

Perfluorochemicals such as Fluosol-DA

Polymerised haemoglobin solutions with good intravascular recovery

Indications for blood transfusion

Symptomatic anaemias:

- Recurrent haemorrhage
- Haemolysis
- Bone stem cell failure

- Pure red cell aplasia
- Severe anaemia of chronic disorders
- Haematological malignancies (e.g. leukaemia, lymphoma)
- Chemotherapy complicated by anaemia

In neonates:

- Severe acute haemorrhage
- Haemolytic disease of the new born
- Septicaemia
- Prematurity bleeding disorders:
- Congenital e.g. haemophilia
- Acquired e.g. disseminated intravascular coagulopathy

Prevention or treatment of shock:

- Clinical situations in which there is need to restore and/or maintain circulatory volume e.g. trauma, haemorrhage

To maintain the circulation (as in extracorporeal or cardiac by-pass shunts)

Whole blood preparations: Should be limited to correction or prevention of hypovolaemia in patients with severe acute blood loss

Fresh Blood: Justified by the recognition that there is a relatively rapid loss of platelets, leucocytes and some coagulation factors with liquid storage.

There is also progressive increase in the levels of undesirable products such as potassium, ammonia, and hydrogen ions

Erythrocyte preparations

Four types are in common use:

- Packed red blood cells
- Washed red blood cells
- Leucocyte-reduced red blood cells
- Frozen red blood cells

Washed red blood cells:

Obtained from liquid-stored blood by saline washing using a continuous-flow cell separator or from frozen erythrocytes extensively washed to remove the cytoprotective agents

Leucocyte-reduced red blood cells:

Best prepared by passing whole blood or packed cells through specifically designed filters.

Three main reasons for the use of leucocyte-reduced red blood cells:

- To prevent non-haemolytic febrile reactions to white cell and platelet antibodies in recipients exposed to previous transfusions or pregnancies
- To prevent sensitization of patients with aplastic anaemia who may be candidates for bone marrow transplantation
- To minimize risk of transmission of viruses such as HIV or cytomegalovirus

Transfusion therapy

Informed consent should be obtained from patients except in life-threatening emergencies

The risks and benefits of the proposed

transfusion therapy should be discussed with the patient and documented in the patient's medical records

Blood for emergencies:

There may be no time available to type, select and cross-match compatible blood

A rare occurrence, except for

- Trauma
- Unexpected intra-operative haemorrhage
- Massive gastro-intestinal bleeding
- Ruptured aneurysm

Uncross-matched or partially cross-matched blood is administered; routine cross-match should be carried out retrospectively to identify any incompatibility

Complications of blood transfusion

Immunological:

Sensitization to red cell antigens

Haemolytic transfusion reactions

- Immediate
- Delayed

Reactions due to white cell and platelet antibodies

- Febrile transfusion reactions
- Post-transfusion purpura

Reactions due to white cell and plasma protein antibodies

- Urticaria
- Anaphylaxis

Non-immunological:

Transmission of disease
Reactions due to bacteria and bacterial pyrogens
Circulatory overload
Thrombophlebitis
Air embolism
Transfusion haemosiderosis
Complications of massive transfusion

Tests of Compatibility

A minimum of three major procedures must be carried out:

- Determine the recipient's ABO and Rhesus groups
- Select compatible donor blood
- Cross-match donor cells against recipient's serum

Donor blood should be screened for infective agents: HIV, hepatitis B, and C viruses

Other investigations

Haemoglobin concentration, haematocrit
Red cell indices: MCH, MCV, MCHC
Total leucocyte and differential counts
Reticulocyte count
Erythrocyte sedimentation rate
Platelet count

Treatment objectives

To raise haemoglobin concentration and other blood parameters to normal levels

To prevent blood transfusion complications

Non-drug treatment

Transfusion of red blood cells, platelet concentrates or platelet rich plasma as required

Provision of fresh frozen plasma or other blood products as necessary

Drug treatment

Furosemide 40 mg on administration of one unit of blood

In the event of transfusion reactions, stop the transfusion immediately and administer the following:

Promethazine 25 mg intramuscularly or intravenously

Epinephrine 0.5 mL of 1:1000 solutions to be administered subcutaneously

Hydrocortisone sodium succinate 100 mg injection intravenously

Supportive measures

Appropriate nutrition

Adequate hydration

Notable adverse drug reactions, caution

Furosemide: dehydration and hypersensitivity

Promethazine: drowsiness, hypersensitivity

Prevention

Avoid/prevent accidents

Prompt treatment of illnesses that could be complicated by anaemia

Regular medical check-ups

HAEMOSTASIS AND BLEEDING DISORDERS – refer for specialist care

LEUKAEMIAS

Introduction

A heterogeneous group of diseases characterized by infiltration of the blood, bone marrow and other tissues by neoplastic cells of the haematopoietic system

Two main types

- Myeloid leukaemia
- Lymphoid leukaemia

Each is further divided into acute and chronic

Acute leukaemias are defined pathologically as blast cell leukaemias or malignancies of immature haematopoietic cells. The bone marrow shows > 30% blast cells

Two main groups of acute leukaemias

- Acute myeloid leukaemia (AML)
- Acute lymphoblastic leukaemia (ALL)

Childhood leukaemias: patients aged <15 years

Adult leukaemias: patients aged >15 years
Leukaemias in adults aged > 60 years: an important group because their responses to current treatment protocols both for ALL and AML are inferior. These patients are not usually considered for more radical treatment approaches such as autologous or allogeneic bone marrow transplantation
80% of adult cases: AML

Epidemiology/predisposing conditions

Acute lymphoblastic leukaemia (ALL) and Acute myeloid leukaemia (AML)
More common in industrialized than rural areas.
Environmental agents implicated in the induction of certain types of leukaemia:
Ionising radiation: X-rays and other ionizing rays
Chemical carcinogens
- Benzene and other petroleum derivatives
- Alkylating agents

Host susceptibility e.g. genetic disorders:
Bloom's syndrome
Fanconi's anaemia (AML)
Ataxia telangiectasia (ALL)
Down's syndrome
Blast transformation in pre-existing myeloproliferative disorders:
Aplastic anaemia (ALL)

Oncogenic viruses:
HTLV-1 (Human T-cell Lymphotropic virus 1): implicated in adult T cell leukaemia /lymphoma

Clinical features
General symptoms of anaemia
Bleeding
Infections
Anorexia
Weight loss
Lymphadenopathy (not common in AML except in the monocytic variant)
Skin: Macules, papules, vesicles, pyoderma gangrenosum, neutrophilic dermatitis, leukaemic cutis, granulocytic sarcoma

Differential diagnoses
Septicaemia
Miliary tuberculosis
Malignant histiocytosis

Complications
Worsening ill-health

Investigations
Full blood count with ESR, reticulocyte count, Coomb's test
Bone marrow examination
Biochemical tests: serum electrolytes, urea, creatinine, uric acid, liver function tests

Prothrombin time, partial thromboplasnime human leucocyte antigen typing
HIV I and II
Cytochemical tests:
- Peroxidase
- Sudan Black B-Non-specific esterase reaction e.g. alpha napthyl acetate esterase
- Bone marrow cultures
- Cytogenetic studies

Electron microscopy
Cell markers e.g. using a panel of antibodies combined with flow cytometric analysis or the alkaline phosphase-antialkaline phosphate (APAAP) technique to classify the blast cells into lymphoid or myeloid lineages
Abdominal ultrasound/CT scans
Immunological classification
Terminal deoxynucleotidyl transferase demonstration in nuclei of B and T lymphocytes

Treatment objectives
Induce remission to achieve complete remission
Maintain disease-free state

Non-drug treatment
Appropriate nutrition
Adequate hydration (at least 3 litres/24 hours)

Erythrocyte transfusion as required
Platelet concentrate transfusion as required
Maintain electrolyte balance

Drug treatment

Acute lymphoblastic leukaemia

Allopurinol 300 mg daily orally DVP Regime
Daunorubicin 30 mg/m^2intravenously on days 8, 15, 22 and 29
Vincristine 1.4mg/m^2 to a maximum of 2 mg intravenously on days 8, 15, 22 and 29
Prednisolone 60 mg orally once daily from day 1 - 28
L-asparaginase 1000 IU/rm intravenously on days 12, 15, 18, 21, 24, 27, 30 and 33

Or:

COAP Regime
Cyclophosphamide 650 mg/m^2intravenously on days 1 and 8; 14 and 22
Vincristine 1.4mg/m^2intravenously to a maximum of 2mg: days 1 and 8; 14 and 22
Cytosine Arabinoside 50 mg/m^2subcutaneously 12 hourly for 12 days or bolus intravenous injection 100 mg/m^2daily for 7 days
Prednisolone 40 mg/m^2oral for 14 days

- Drugs are given every 28 days for 3 courses

Nervous system prophylaxis

- Methotrexate 12.5 mg/m^2 intrathecally twice weekly to a maximum of 15 mg i.e. 5

doses over 3 weeks.

Consolidation:

To be given on day 29

COAP regime to be given once provided WBC count is = 1x10s/L and platelet count is = 100 x10VL

Maintenance:

6-Mercaptopurine 75 mg/m^2orally daily

Methotrexate 20 mg/m^3orally weekly

- For 3 years if remission is maintained, otherwise reassessment

Pulse therapy (Intensification). To be given every 3 months with

- Vincristine 1.4 mg/mmta a maximum of 2 mg weekly on days1 and 8

Acute myeloblastic leukaemia

Either TAD or COAP as shown below:

TAD:

Cytarabine 100 mg/m^2(continuous infusion) on days 12 and 2, and 100 mg/m every 12 hours by intravenous infusion over 30 minutes on days 3 - 8

Thioguanine 100 mg/m^2 every 12 hours orally on days 3-9

Daunorubicin 60 mg/$m^2$by intravenous infusion over one hour on days 3 - 5

Or:

COAP:

Cyclophosphamide 650 mg/m^2intravenously

on days 1 and 8
Vincristine 1.4 mg/m^2 intravenously to a maximum of 2 mg on days 1 and 8
Cytarabine 50 mg/m^2 subcutaneously every 12 hours for 7 days
Prednisolone 40 mg/m^2 orally for 14 days
- Nervous system prophylaxis is not required
- Assess for remission after 3 courses

Maintenance
COAP every 6 weeks for 2 years
Intrathecal treatment as for ALL if there is CNS disease of the monocytic type

Chronic Myeloid Leukaemia (CML)
Also Chronic Myelogenous Leukaemia; Chronic Granulocytic Leukaemia (CGL)
A clonal disease that results from acquired genetic change in a pluri-potential haematopoietic stem cell
Altered stem cell proliferation generates a population of differentiated cells, and a greatly expanded total myeloid mass

Classification
Majority of patients have relatively homogenous disease characterized by:
- Splenomegaly
- Leucocytosis
- Presence of Philadelphia (Ph) chromosone in all leukaemia cells

Minority of patients has less typical disease (atypical CML)

- These variants lack Ph chromosome.

Examples:

- Chronic myelomonocytic leukaemia
- Chronic neutrophilic leukeamia
- Juvenile chronic myeloid leukaemia

Epidemiology, aetiology and natural history

Rare below the age of 20 years but occurs in all age groups

Increased risk of developing CML with exposure to high doses of irradiation

A biphasic or triphasic disease, usually diagnosed in the initial "chronic" or stable phase Distinguishing features between phases of CGL

Chronic phase.

Untreated patient: <12% blast cells in blood or marrow

Treated patient: Normal or near-normal blood count without immature granulocytes in peripheral blood

Accelerated phase

Rising leucocyte count despite treatment

Rapid leucocyte doubling time

Immature granulocytes in blood

Blast cells >5% but <30°/o in marrow

Anaemia (Hb <10 g/dL) not attributable to treatment

Thrombocytosis (>1000 x 109/L)

Acquisition of specific new cytogenetic abnormalities
Increasing marrow fibrosis
Blastic transformation
More than 30% blasts

Or:
Blasts plus promyelocytes in blood or bone marrow

Clinical features
Asymptomatic Abdominal swelling/pain
Lethargy
Shortness of breath on exertion
Weight loss
Unexplained haemorrhage at various sites e.g. gums, intestinal/urinary tracts
Increased sweating
Visual disturbances
Gout
Priapism
Splenomegaly
Anaemia
Haemorrhage
Fever
Lymphadenopathy (rare in chronic phase)

Complications
Blastic transformation
Death

Investigations
As above for acute leukaemia
plus:
Determination of Philadelphia chromosome
Lactic dehydrogenase
Serum calcium

Treatment objectives
Induce remission to achieve complete remission
Maintain disease-free state
Achieve absence of Philadelphia chromosome

Non-drug treatment
Appropriate nutrition
Adequate hydration
Electrolyte balance

Drug treatment
Hydroxycarbamide (hydroxyurea)
Adult: 20-30 mg/kg orally daily or 80 mg/kg every third day
Child: Not recommended
Interferon alpha
Adult: 9 million units subcutaneously or intravenously thrice weekly for 6 - 12 months

Or:
Imatinib mesylate
- 400 mg orally daily

- *To be used strictly under specialist supervision*

Notable adverse drug reactions, caution
The above drugs (except the steroids) all cause profound myelosuppression
Profound nausea, vomiting, diarrhoea and abdominal discomfort
Secondary malignancies
Steroids: Cushing's syndrome, hypertension, diabetes mellitus, immunosuppression, infections Vincristine: neurotoxicity
Cylophosphamide: alopecia, haemorrhagic cystitis
Daunorubicin: myelosuppression, alopecia, cardiotoxicity
All are contraindicated in patients with history of hypersensitivity reactions to the respective medicines

Prevention
Avoid exposure to ionizing radiation
Early detection and treatment

Chronic Lymphocytic Leukaemia
Neoplastic proliferations of mature lymphocytes
The diseases involve the blood bone marrow and other tissues
Characterized by accumulation of small mature-looking CD5+ B lymphocytes in the

blood, marrow and lymphoid tissues
B-cell disorders are more common
B-cell CLL is more common in males than females

- Accounts for 60% of cases
- Rarely diagnosed below the age of 40 years

Clinical features
Asymptomatic (30% of cases)
Symptoms of anaemia
Lymph node enlargement (painless)
Rare: pyrexia, sweating or weight loss
Severe chest infection/ pneumonia
Splenomegaly (50% of cases)
Hepatomegaly (not frequent)

Differential diagnoses
Lowgrade non-Hodgkin's lymphomas with frequent blood and bone marrow involvement (leukaemia / lymphoma syndromes)
Tuberculosis
Viral infections
Toxoplasmosis

Complications
Richter transformation
Progression of disease

Investigations

Cell morphology:
Size
Nuclear: cytoplasmic (N: C) ratio
Regularity or irregularity of the nuclear outline
Characteristics of the cytoplasm (presence and length or absence of azurophil granules)
Degree of nuclear chromatin condensation and its pattern
Prominence, frequency and localization of the nucleolus

Investigations
As for anaemia and other leukaemias

Treatment objectives
Induce remission to achieve complete remission
Maintain disease-free state

Non-drug treatment
Appropriate nutrition
Adequate hydration
Maintenance of electrolyte balance
Bone marrow transplant
Red cell and platelet concentrate transfusion as required

Drug treatment
Chronic Lymphocytic Leukaemia
Allopurinol 100 mg orally every 8 hours

Chlorambucil 5 mg/ml orally on days 1 to 3
Prednisolone 75 mg orally on day 1; 50 mg orally on day 2 and 25 mg orally on day 3
- Repeat every 2 weeks

Or:

Fludarabine 25 - 30 mg intravenously over 30 minutes on days 1-5
- Repeat every 4 weeks

Or:

Combination chemotherapy
- Cyclophosphamide 400 mg/m^2
- Vincristine 1.4 mg/m^2
- Prednisolone 100 mg orally days 1 - 5
  Repeat every 3 weeks

Or:

Fludarabine 30 mg/m^2 intravenously over 30 minutes on days 1 - 3
Cyclophosphamide 250 - 300 mg/m^2 intravenously over 30 minutes on days 1 - 3
- Repeat every 4 weeks

Supportive measures

Appropriate nutrition.
Adequate hydration

Notable adverse drug reactions, caution

Same as for other leukaemias

Prevention

Avoid chemicals on body (e.g. benzene)
Avoid ionizing radiation (X rays)
Early detection and treatment

LYMPHOMAS

Introduction

Solid neoplasms that originate in lymph nodes or other lymphatic tissues of the body.

A heterogeneous group of disorders

- Can arise at virtually any site
- More often occurs in regions with large concentrations of lymphoid tissues, e.g. lymph nodes, tonsils, spleen and bone marrow

Two main groups:

- Hodgkin's disease
- Non-Hodgkin's lymphomas

Hodgkin's disease

characterized by Reed-Sternberg cells (large binucleate cells with vesicular nuclei and prominent eosinophilic nucleoli)

- Reed-Sternberg cells are occasionally found in other clinical conditions e.g. hyperplastic or inflammatory lesions of lymph nodes

Non-Hodgkin's lymphomas: a heterogeneous collection of lymph proliferative malignancies

- Vary widely according to histological subtype, stage and bulk of disease

Investigations

Mandatory

Full Blood Count (i.e. haemoglobin, haematocrit leucocyte and differential counts; red cell indices, reticulocyte count)
Erythrocyte sedimentation rate
Coombs test
Bone marrow aspiration and needle biopsy
Serum Urea, Electrolytes, Serum Uric acid
Liver Function Tests: transaminases-ALT, AST, ALP; bilirubin; serum proteins HIV screening Immunoglobulins, Chest X-ray

Optional

Examination of post-nasal space
Serum copper level
Neutrophil alkaline phosphatase
Tomograms of lung or mediastinum
Skeletal X-ray
Abdominal ultrasound scans
Intravenous pyelography
CT scans of chest and abdomen
Supplementary node biopsy

Treatment objectives

Induce remission
Restore patient to disease-free state
Maintain state of well-being

Non-drug treatment

Appropriate nutrition

Adequate hydration
Red cell and platelet concentrate transfusions as required

Drug treatment
Malaria prophylaxis: proguanil 200 mg orally daily
Antibiotics as indicated
Allopurinol 300 mg orally daily (when uric acid is high)

Non-Hodgkin's lymphomas
CHOP (3 weekly):
Cyclophosphamide 750 mg/m2 intravenously on day 1
Doxorubicin 50 mg/m^2 intravenously on day1, 2
Vincristine 1.4 mg/m^2 (maximum of 2 mg) intravenously on day 1
Prednisolone 100 mg orally on days 1 - 5
CHOP (4 weekly):
Cyclophosphamide 750 mg/m^2intravenously on days 1 and 8
Doxorubicin 25 mg/m^2intravenously on days 1 and 8
Vincristine 1.4 mg/m^2(maximum 2 mg) on days 1 and 8
Prednisolone 100 mg orally on days 1 - 8

Hodgkin's lymphoma
MOPP

Mechlorethamine 6 mg/m^{2}intravenously on days 1 and 8
Vincristine 1.4 mg/m^2 (maximum 2 mg) intravenously on days 1 and 8
Procarbazine 100 mg/m^{2}orally on days 1 and 4
Prednisolone 40 mg orally on days 1 - 14

ChlVPP
Chlorambucil 6 mg/m^{2}orally on days 1 and 14
Vinblastine 6 mg/m^2 (maximum 10 mg) intravenously on days 1 and 18
Procarbazine 100 mg/m^{2}orally on days 1 and 14
Prednisolone 40 mg orally on days 1 - 14

Supportive measures
Appropriate nutrition
Adequate hydration

Notable adverse drug reactions, caution
All the drugs are contraindicated in patients with hypersensitivity reactions to the respective medicines
Profound nausea, vomiting, diarrhoea and abdominal discomfort
Secondary malignancies
Myelosuppression (except the steroids)
Steroids (prednisolone) may cause Cushing's syndrome, hypertension, diabetes mellitus, suppression of immunity, infections

Vincristine: neurotoxic
Cyclophosphamide: alopecia and haemorrhagic cystitis
Doxorubicin: cardiotoxic

Prevention
Avoid unnecessary exposure to irradiation and chemicals

SICKLE CELL DISEASE
Introduction
A group of conditions with pathological processes resulting from the presence of Haemoglobin S Usually inherited from the parents who have themselves inherited haemoglobin S

The principal genotypes include:
- Homozygous sickle cell disease (SS)
- Sickle cell-haemoglobin C disease (SC)
- Sickle cell-B thalassaemia (SB thal)

Sickle cell-B+ thalassaemia Type I (SB+thal. Type I)
Sickle cell-B+thalassaemia. Type II. (SB+thal Type II)
Sickle cell-B+thalassaemia. Type III. (SB+thal. Type III)

Sickle cell trait
Inheritance of one normal gene controlling formation of B Haemoglobin (HbA), and a sickle gene (HbS)

Total haemoglobin A is more than haemoglobin S Normal haemoglobin F

Sickle cell disease

Inheritance of two abnormal allelemorphic genes controlling formation of B chains of haemoglobin, at least one of which is the sickle gene

Polymerization of the sickle haemoglobin may lead to vaso-occlusion

Pathophysiology

Red cells have reduced deformability and easily adhere to vascular endothelium, increasing the potential for decreased blood flow and vascular obstruction.

Abnormalities in coagulation, leucocytes, vascular endothelium, and damage to the membranes of red cells contribute to sickling

Haemolytic anaemia and vasculopathy are the result of the various pathophysiologic processes

Organ damage is on-going and is often silent until far advanced

The course of the disease is punctuated by episodes of pain

Clinical features

Vary widely from one patient to another:

Persistent anaemia/pallor, growth retardation (variable); Jaundice (variable);

Bone pains (recurrent)
Prominent facial bones due to increased bone marrow activity
Leaner body build and less weight (on average)
Some fingers are shortened as a result of infarction (destruction due to blockage of blood supply)
Hand-foot syndrome (painful and swollen hands and feet) in childhood
Life span on average shorter than normal
Sexual development is delayed in both sexes: menarche occurs at a mean age of 15.5 years (range 12 - 20 years) compared to non-sicklers (mean 13.2years)
Impotence can occur from prolonged priapism
High foetal loss in pregnancy

Sickle cell crises

Patient has acute symptoms/signs attributable directly to sickle cell disease
Two main types:
Pain (vaso-occlusive) crisis
Anaemia crisis

Vaso-occlusive crises

Painful
Tender, swollen bones
Acute hepatopathy
Acute chest syndrome

Priapism
Painless
Haematuria
Cerebrovascular disease (accident) - in descending order of prevalence
- Thrombotic stroke
- Seizures
- Haemorrhage

Retinopathy (commonest in SC patients)

Anaemic crises

Acute splenic (or hepatic) sequestration
Hyper-haemolytic (e.g. precipitated by malaria)
Megaloblastic (folic acid deficiency)
Hypoplastic (due to infection or renal failure)
Aplastic (e.g. due to epidemic parvo virus B19)

Differential diagnoses

Connective tissue disorders e.g. rheumatoid arthritis
Liver disease
Other causes of failure to thrive

Complications

Kidneys:
- Hyposthenuria (reduced ability to concentrate urine/conserve body fluids)
- Haematuria
- Albuminuria

- Reduced kidney function

Leg ulcers:

- Occur around ankles
- Heal slowly and tend to recur

Bones and Joints

- Osteomyelitis
- Avascular necrosis

These may cause:

- Hip pain
- Limping gait
- Kyphoscoliosis when necrosis affects spinal vertebral bones

Infections:

- Salmonella osteomyelitis
- Pneumococcal pneumonia
- Pneumoccoccal meningitis (rare in adolescents and adults)
- Tonsillitis and pharyngitis

Brain and nerves:

- Strokes, seizures (not common in adults)
- Meningitis (not common in adults)
- Cerebral haemorrhage
- Mental neuropathy (rare)

Cardiovascular/respiratory:

- Heart failure
- Pulmonary hypertension
- Acute chest syndrome

Investigations

Full Blood Count (haemoglobin, haematocrit, total leucocyte count and differential counts,

platelet counts)
Erythrocyte sedimentation rate
Red cell indices (MCH, MCHC, MCV)
Reticulocyte count
Sickling tests: solubility test; metabisulphite test
Haemoglobin electrophoresis
- Using cellulose acetate paper at pH 8.4 (alkaline) or citrate agar gel at pH 5.6 (acidic)

Serum Electrolytes, Urea and Creatinine
Liver function tests (transaminases, bilirubin, serum albumin, alkaline phosphatase and prothrombin time)
Others as may be indicated:
Urinalysis; microscopy, culture and sensitivity:
Sputum
- Acid Fast Bacilli
- Microscopy, culture and sensitivity

Stool:
- Ova and parasites
- Occult blood

Ultra sound scan:
- Abdominal ultrasound scan
- Transcranial Doppler ultrasonography

Chest radiograph

Treatment objectives
Maintain (or restore) a steady state of health
Prevent and treat complications

Provide accurate diagnosis, relevant health education and genetic counselling to patients, relatives and heterozygotes
Improve quality of life
Provide a positive self-image in affected persons

Treatment strategies

Counselling and health education
Encouraging membership of support groups
Providing infection prophylaxis (antimalarial; anti-pneumococcal, hepatitis B virus vaccines)
Providing folate supplementation
Avoiding pain-inducing conditions
Providing prompt treatment of symptoms
Advising on contraception
Supervising pregnancy/Labour
Providing regular health checks
Limiting family size

Non-drug treatment

Balanced diet
Adequate fluid intake (at least 3 litres/24 hours)
Avoidance of pain-inducing conditions

- Strenous physical exertion or stress
- Dehydration
- Sudden exposure to extremes of temperature
- Infections e.g. malaria
- Emotional stress

Adjunct treatment
Blood transfusion (especially red cell transfusion)
Anti-pneumococcal vaccine

Drug treatment
Steady state(when patient is well with no complaints):
Proguanil
Adult:200 mg orally daily
Child: under 1 year 25 mg daily; 1 - 4 years 50 mg; 5-8 years 100 mg; 9 - 14 years 150 mg orally daily
Plus:
Folic acid 5 mg orally daily

Pain crises
Mild pain
Paracetamol
Adult: 1 g, every 4 - 6 hours to a maximum of 4g daily
Child: 1-5 years120-250 mg; 6-12 years 250-500 mg; 12 -18 years 500 mg every 4 - 6 hours (maximum 4 doses in 24 hours)

Or:
Aspirin (acetylsalicylic acid) 600 mg orally every 8 hours daily
- Not recommended for children under 16 years

Or:

Ibuprofen 200 mg every 8 hours daily (or other non-steroidal anti-inflammatory drugs)

- Not recommended for children under 16 years

Moderate-to-severe painful crises

Parenteral therapy:

Diclofenac sodium

Adult: 75 mg or 100 mg intramuscularly (as necessary)

- Not recommended for children

Oral therapy:

Paracetamol

Child: 1 -5 years 20mg/kg every 6 hours (maximum 90 mg/kg daily in divided doses) for 48 hours or longer if necessary and if adverse effects are ruled out

Then:

15 mg/kg every 6 hours (maintenance)

6 - 12 years: 20 mg/kg (maximum 1 g) 6 hourly (maximum 90 mg/kg daily in divided doses, not to exceed 4 g for 48 hours or longer if necessary and if adverse effects are ruled out

Then:

15 mg/kg every 6 hours (maximum 4 g daily)

12 - 18 years: 500 mg - 1g every 4 - 6 hours (maximum 4 doses in 24 hours)

Diclofenac potassium 50 mg every 12 hours daily

Or:
Diclofenac sodium 100 mg once daily

Or:
Morphine 15 mg every 8 - 12 hours daily

Antimalarials
Artemisinin-based combination therapy (see section on malaria)

Supportive measures
Counselling and health education
Membership of support group
Regular health checks

Notable adverse drug reactions, caution and contraindications
Paracetamol should be used with caution in patients with hepatic impairment
Opioid analgesics cause varying degrees of respiratory depression and hypotension. They should be avoided when intracranial pressure is suspected to be raised

Prevention
Advice on the risks involved in marriages between carriers, and between sicklers
Anti-pneumococcal vaccine (once every 5 years)

MULTIPLE MYELOMA

Definition:

A malignant proliferation of plasma cells derived from a single clone. The terms multiple myeloma and myeloma may be used interchangeably. The tumour, its product, and the host response to it result in a number of organ dysfunctions and symptoms of bone pain or fracture, renal failure, susceptibility to infection, anaemia, hypercalcemia, and occasionally clotting abnormalities, neurologic symptoms, and vascular manifestations of hyperviscosity.

Clinical features

Classified as asymptomatic or symptomatic, depending on the absence or presence of myeloma related organ or tissue dysfunction, including hypercalcemia, renal insufficiency, anaemia, and bone disease

Bone pain is the most common symptom in myeloma, affecting about 70% of patients.

Usually involves the back and ribs,

The pain of myeloma is precipitated by movement (Unlike the pain of metastatic carcinoma, which often is worse at night)

Persistent localized pain in a patient with myeloma usually signifies a pathologic fracture.

The bone lesions of myeloma are caused by the proliferation of tumour cells and the

activation of the osteoclasts that destroy the bone.

The next most common clinical problem in patients with myeloma is susceptibility to bacterial infections.

Anaemia occurs in about 80% of myeloma patients:

- usually normocytic and normochromic
- related both to the replacement of normal marrow by expanding tumour cells and to the inhibition of haematopoiesis by factors made by the tumour.

Bony lesions develop in almost 80% of patients with newly diagnosed disease; in one study, 58% of patient reported bone pain.

Renal impairment occurs in 20 – 40% of patients with newly diagnosed disease, mainly as a result of direct tubular damage from excess protein load, dehydration, hypercalcemia and the use of nephrotoxic medications.

The risk of infection is increased with active disease but decreases with response to therapy.

Diagnosis

Based on the presence of at least 10% clonal bone marrow plasma cells and monoclonal protein in serum or urine.

In patients with true nonsecretory myeloma, the diagnosis is based on 30% monoclonal

bone marrow plasma cells or a biopsy proven plasmcytoma.

The recommended test for diagnosis of myelomia include:

- the taking of a detailed medical history and physical examination,
- routine laboratory testing :
  - Complete blood count,
  - Chemical analysis,
  - Serum and urine protein electrophoresis with immunofixation, and qualification of monoclonal protein
  - Bone ,marrow examination (trephine biopsy plus aspirate of cytogenetic analysis or fluorescence In situ hybridization[FISH]).
  - Conventional radiography of the spine, skull, chest, pelvic, humeri, and femoral remains the standard to identify myeloma related bone lesions.
  - Magnetic resonance imaging (MRI) is recommended to evaluate symptoms in patients with normal results in conventional radiography and in all patients with radiographs suggesting the presence or solitary plasmacytoma of the bone.
  - Computed tomography and MRI are the procedures of choice to asses suspected cord compression and should be performed on an urgent

basis.

Treatment

About 10% patients with myeloma will have an indolent course demonstrating only very slow progression of disease over many years. The vast majority of patients with myeloma require therapeutic intervention. In general such therapy is of two sorts: systemic chemotherapy to control progression of myeloma, and symptomatic supportive care to prevent serious morbidity from the complications of the disease.

Treatment objectives

Reduce total mass of tumour

Maintain this by continuation therapy

Induction therapy(3-4 weeks)

Chemotherapy of cancer is complex and should be confined to specialists in oncology and in line with National Guidelines on Cancer Chemotherapy.

Due to the complexity of dosage regimens in the treatment of malignant diseases, dose statements have been omitted from majority of the entries in this guideline

Single agent combination

Melphalan/prednisolone.

Combination chemotherapy

VMCP (vincristine, melphalan,

cyclophosphamide, prednisolone)
VAD regimen (Vincristine, adriamycin, high dose dexametasone)
VAMP-use of methyl prednisolone in place of dexametasone
Alpha interferon (3mu)
Novel Therapies
Thalidomide (FOLLOW THE GUIDELINES STRICTLY) and dexamethazone
Adverse effects: sedation, constipation, fatigue, Perpheral Neuropathy, DVT
Treatment
Transplantation
Allogeneous and autologous stem cell
Radiotherapy
Caution
Cytotoxic medicines have both anticancer activity and the potential to damage normal tissue; most are teratogenic
All cytotoxic medicines cause side effects and a balance has to be struck between likely benefit and acceptable toxicity
Trained personnel should reconstitute cytotoxics in designated pharmacy areas
Pregnant staff should avoid exposure to cytotoxics
Prescriptions should not be repeated except on the instructions of a specialist

CHAPTER 3:

CARDIOVASCULAR SYSTEM DISORDERS

CORONARY ARTERY DISEASE (CAD)

Introduction

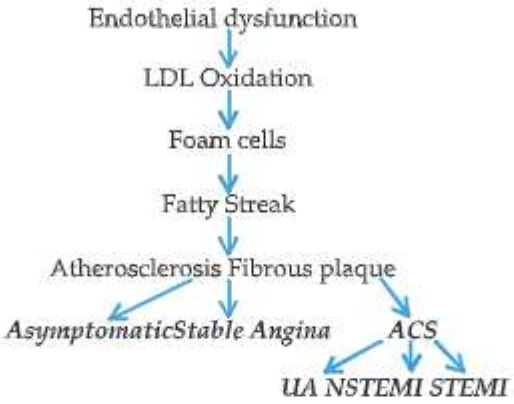
A condition due to imbalance in oxygen demand and supply resulting predominantly from atherosclerotic disease of the coronary artery. Its incidence is on the rise in Nigeria as a result of epidemiologic transition from communicable to non-communicable diseases.

Traditional risk factors

- Diabetes mellitus
- Hypertension
- Cigarette smoking
- Obesity
- Dyslipidaemia
- Advanced aging
- Male gender

Pathogenesis

Atherosclerosis is the major underlying pathogenic factor (See diagram below)



Pathogenesis of Coronary artery disease

Epicardial coronary artery stenosis:

- ⩽ 60% occlusion is compensated for at rest
- 60%-90% occlusion causes ischaemia when there is increased oxygen demand
- 90% occlusion results in ischaemia even at rest

CHRONIC STABLE ANGINA (ANGINA PECTORIS)

Clinical features

Symptoms

- Typically retrosternal chest pain or heaviness worsened by exertion that radiates to the left upper arm

Angina equivalents include:

- Dyspnoea

- Palpitation
- Giddiness
- Fatigue
- Silent ischemia may occur in the diabetics & elderly

Signs

There may be no specific abnormal finding. Nicotine stain of the nails and cardiomegaly may be observed

Differential diagnoses

- Pulmonary embolism
- Gastro-esophageal reflux disease (GERD)
- Pericarditis
- Aortic dissection
- Mitral valve prolapse syndrome
- Esophageal spasm
- Costochondritis

Investigations

Resting ECG:

- Normal in 50% of patients
- ST depression
- May show Q wave of previous MI
- Exercise stress ECG: Positive test evidenced by ST depression ≥ 1 mm (planar or down sloping)
- Arrhythmias or fall in BP

Sensitivity (68%); Specificity (77%)

Echocardiography

Laboratory evaluation:
- Fasting Blood Glucose
- Lipid profile
- Electrolytes, urea & creatinine

Coronary angiography: Gold standard invasive, expensive & done only when coronary artery bypass graft CABG or angioplasty is planned.

Treatment

Antipatelet therapy:
- Acetyl salicylic acid: 75 mg orally daily

Or:
- Clopidogrel 75 mg orally daily

Lipid lowering drugs:
- HMG-CoA reductase inhibitors
- Atovarstatin 10-20 mg orally daily

Nitrates:
Short acting Glycerin Trinitrate tablets 0.3-1mg sublingually, repeated as required; 400 microgramspray/metered dose 1-2 doses under tongue

OR

Intravenous infusion, 10-200 micrograms/minute, adjusted according to response; max. 400 micrograms/minute

Long acting (Isosorbide dinitrate 30-120 mg three times daily, and up to 240 mg if required
Or

Intravenous infusion, 2-10 mg/hour, higher doses up to 20mg/hour may be required

Beta blockers:
- Cardioselective (atenolol 50-100 mg daily; metoprolol 50-200 mg daily).
- Target pulse rate of 55-60 bpm. Taper over 3-10 days

Calcium channel blockers
Dihydropyridine-
- Amlodipine 5 - 10 mg daily

Or:
- Nifedipine 20 - 60 mg daily

Or:
Non-dihydropyridine:
- Verapamil 80 - 120 mg 8 hourly

Angiotensin converting enzyme inhibitor:
- Captopril 6.25 - 12.5 mg daily. Other ACEIs may be used in patients with hypertension

Angiotensin receptor blockers:
- Valsartan 80 - 160 mg daily

Revascularization:
Percutaneous coronary intervention
Coronary artery bypass graft

ACUTE CORONARY SYNDROME

Introduction

Spectrum of CAD comprising myocardial infarction with ST segment elevation (STEMI), or without ST segment elevation (NSTEMI) and unstable angina (UA) depending on clinical, ECG and enzyme changes.Guidelines for management of ACS are issued by ACC/AHA and ESC.

- STEMI: Angina, ST ≥1 mm (in ≥2 adjacent Limb leads) or ≥ 2mm in ≥ 2 contiguous praecordial leads or New onset LBBB and Elevated Troponin
- NSTEMI: Angina ST depression ≥ 1mm or T wave abnormalities, No ST segment elevation, Elevated Troponin level
- Unstable angina: Rest Angina, ST or T wave abnormalities, No ST segment elevation, No rise in Troponin level

Clinical features

- Focused history
- Chest pain similar in character to angina pectoris but greater in severity, longer in duration (>30 mins), not relieved by nitrates, not specifically provoked by exercise or relieved by rest
- Autonomic disturbance: diaphoresis, vomiting, giddiness & anxiety
- History of risk factors, previous MI/intervention, stroke/asthma/bleeding tendencies.

- Pulse: Tachycardia/bradycardia/arrhythmia
- BP: Normal/transient elevation/hypotension
- Pulmonary oedema

Differential diagnosis
- Acute pulmonary embolism
- Acute Pericarditis
- Acute aortic dissection
- Esophageal spasm
- Peptic ulcer disease

Complications
- Arrhythmia
- Pulmonary oedema
- Septal/chordae/myocardial wall rupture
- Stroke
- Ventricular aneurysm
- Pericarditis

Investigations
- 12 lead ECG within 10 minutes of presentation with cheat pain.
- Serial ECG if there is diagnostic uncertainty or change in clinical status.
- In inferior wall MI: do a right sided ECG (4VR) to exclude RV infarct (ST elevation >1 mm)
- ECG diagnosis of STEMI in a setting of preceding LBBB is difficult but the followings are useful:
  - ST changes in the same direction

- ST segment elevation > 7-8 mm
- Coving of ST segment
- Pathological Q wave in 2 consecutive leads and
- Reciprocal changes

- Cardiac enzymes:
  - Cardiac troponin I & T are released within 4 hrs of MI and remain elevated up to 2 weeks & is the most sensitive marker of myocardial damage. Useful in late presentation
  - MB-CK rises within 4-6 hour, peak in a day and disappears by the *second day*
- Blood chemistry: Blood sugar, electrolytes (Na, K, Cl, Ca, Mg), urea, Cr, lipid levels, arterial blood gas
- C-X ray: Cardiomegaly & Pulmonary oedema
- Echocardiography: Regional wall motion abnormality, Pericardial effusion, septal/chordae/ papillary muscle rupture, MR, LV function, RV function, PAP
- Coronary angiography: Delineate site(s) of lesion & number of vessels involved.

- **Management of ACS**
- General
- Oxygen (100% 2-4 L/min) via nasal prongs if SaO2 < 90%
- Set up an IV line
- Dual antiplatelet therapy: ASA 300 mg

stat; 75 mg daily + Clopidrogel 300 mg stat; 75 mg daily.

- Short acting nitrates: Route: Sublingual (0.4 mg); Buccal (1-5 mg 6 hrly); Aerosol (400 ug per spray)
- Long acting nitrates: Iso-sorbide dinitrate 10-20 mg tds. IV route: IV nitroglycerin (5-10 g/min)
- Morphine: (2-4 mg) given slowly through IV canula
- Lipid lowering drugs: statin 10-40 mg daily
- ACEI: Captopril (6.25-12.5 mg daily), Lisinopril (2.5-10 mg daily depending on blood pressure status)
- ARB: Valsartan (80 mg daily), lorsatan (12.5-25 mg daily) There is increased risk of hyperkalaemia, hypotension, and impaired renal function when angiotensin-II receptor antagonists are taken with ACE Inhibitors
- Beta blocker: Carvedilol (6.25-12,5 mg daily) Metoprolol (25-50 mg daily).
- CCB: Rate limiting such as verapamil (40-80 mg tds) & diltiazem (30-60 mg tds). Non rate limiting such as amlodipine (2.5-10 mg daily)
- Anxiolytics: Diazepam 5mg daily
- Stool softener: Liquid paraffin 15-30 mls nocte
- Anticoagulant therapy
- Heparin: Bolus of 60 units/Kg body weight followed by 12 units/Kg/hr

- Heparin should not be given with streptokinase
- LMWH: 1 mg/Kg given twice daily
- Bed rest within the first 24 hours
- **Management of STEMI**
- Reperfusion therapy (pharmacological)
- Indicated only in STEMI
- Patient aged < 75 years
- Best benefit if given within 1-3 hours of AMI ("GOLDEN HOUR"). May be tried if patient report within 6-12 hrs though benefits after 6 hrs is uncertain
- Chances of successful reperfusion is about 50%
- Streptokinase
  - Activates fibrinolytic system
  - Antigenic & dosage cannot be repeated until after at least 1 year
  - Dose 1.5 million units in 100 mls of normal saline given over 30-60 mins
- Recombinant tissue plasminogen activator (tPA)
  - Superior to and more expensive than streptokinase
  - Not antigenic
  - Preferred to SK if there is hypotension
  - Higher risk of intracerebral haemorrhage
- Alteplase
  - Dose: 15 mg stat;
  - then 0.75 mg/Kg (max: 50 mg) over 30 mins; then 0.5 mg/Kg (max: 35

mg) over 60 mins

Contraindications

- Absolute contraindications: Active bleeding, bleeding diathesis, stroke within 3 months, intracranial tumour.
- Relative contraindications: Severe hypertension (> 180/110mm Hg), recent trauma/CPR/c surgery, Active peptic ulcer, Oral anticoagulant therapy, Advanced liver diseases, Active cavitation PTB and Pregnancy and within 1 week post-partum
- Medical intervention
- Refer to centres for percutaneous coronary intervention where available and affordable: Ballon angioplasty, Stenting (Bare metal Thrombectomy
- Surgical intervention:
- Refer for Coronary artery bypass graft (CABG) where available and affordable
- Indicated in: Triple vessel disease, Proximal Left main coronary artery disease, Double vessel disease with proximal LAD lesion & Calcification

Adverse drug reactions

- Nitrates may cause headache and tolerance. Contraindicated in bradycardia and when SBP < 90 mmHg
- Beta blockers may precipitate bradycardia, Heart Failure, asthma and hypotension

- Thrombolytic agents may cause bleeding
- Heparin may induce thrombocytopenia

Prevention

- Life style modifications including regular exercise, optimum weight, high fibre and low saturated fat diet; cessation of cigarette smoking and moderate alcohol intake
- Treatment of hypertension, diabetes and hyperlipidaemia
- Others
  - Educate on benefits, outcome and complications of patient's condition and treatment modalities
  - Emphasize primary and secondary preventive measures as essential irrespective of treatment modality offered

CARDIAC ARRHYTHMIAS

Introduction

Abnormalities of cardiac rhythm

Usually complicate acquired and congenital heart diseases

Classification:

Supraventricular such as atrial fibrillation which is the commonest cardiac arrhythmia in Nigeria

Ventricular such as ventricular tachycardia

Clinical features
Mild arrhythmias might go unnoticed
May present with:
Palpitations
Sudden collapse
Dizziness
Syncope
Near-syncope
Features of complications/underlying causes(s): cardiac failure, stroke, structural heart diseases, etc
Differential diagnoses
Anxiety

Complications
Cardiac failure
Stroke
Peripheral embolic phenomena
Sudden death

Investigations
Electrocardiograph (resting, 24 hour Holter, 1 month Holter monitoring)
Atrial fibrillation is characterized by absence of p waves, presence of fibrillary waves and varying RR interval.
Electrolytes including K and Mg; Urea and Creatinine
Echocardiography
Electrophysiology

Treatment objectives
Abolish the arrhythmias
Treat underlying cause
Treat complications
Prevent further arrhythmias

Non-drug treatment
Pacemaker insertion
Ablation (electrophysiology)
Cardioversion: acute arrhythmias

Drug treatment
Depends on the type of arrythmia
Atrial fibrillation
Rate Control:
- Digoxin (0.125-0.375 mg daily) is the drug of choice in chronic atrial fibrillation.
- Beta blockers (atenolol 50-100 mg)
- Calcium channel blocker (Verapamil 40-120 mg daily)

Rhythm control: External cardioversion
Prevention of atrial fibrillation: Amiodarone 100-400 mg daily
Anticoagulation (Warfarin 5 mg daily; Maintain Target INR 2-2.5)

Refer cases to a specialist for appropriate management

Supportive measures
Patient education

Efficient systems to facilitate patient recovery

Notable adverse drug reactions

Anti-arrhythmias are pro-arrhythmics themselves
Cardiac failure (all anti-arrhythmics)
Blindness (amiodarone)

Prevention

Prevention and prompt management of predisposing conditions such as hypertension, rheumatic heart disease, diabetes mellitus, ischaemic heart disease, congenital heart diseases etc

DEEP VENOUS THROMBOSIS

Introduction

Formation of blood clot(s) in the deep veins of the calf muscles or pelvis
It has the potential of being dislodged to the lungs, causing pulmonary embolism
Predisposoing factors:
Old age
Obesity
Hyper-coagulable states
Long periods of immobilization e.g. cardiac failure, following surgery, long-distance travel, etc
Varicose veins
Pregnancy and pueperium
Malignancies

Clinical features
Could be asymptomatic
Pain and swelling of the leg (calf muscles)

Differential diagnoses
Cellulitis
Infarctive crisis in sicklers
Abscess (pyomyositis)

Complications
Pulmonary embolism

Investigations
Full Blood Count and differentials
Prothrombine time
KCCT
Doppler of the leg/ pelvic vessels (veins)
Echocardiography
Electrocardiography
Venography (pelvic or calf veins)
Treatment objectives
Lyse the clot
Prevent clot from being dislodged
Relieve inflammation

Non-drug treatment
Avoid stasis

Drug treatment
Achieve APTT of 1.5 to 2.5 of control:

Heparin 5000 - 10,000 units by intravenous injection followed by subcutaneous injection of 15,000 units every 12 hours or intravenous infusion at 15 - 25 units/kg/hour, with close laboratory monitoring.
Warfarin 1 - 5 mg orally daily for 6 - 12 weeks.
Maintain Target INR at 2 – 2.5

Notable adverse drug reactions
Bleeding from heparin, warfarin
Osteoporosis (heparin)

Prevention
Low molecular weight heparin 5000 units subcutaneously every 12 hours
Early mobilization

HEART FAILURE
Introduction
A clinical state (syndrome) in which the heart is unable to generate enough cardiac output to meet the metabolic demands of the body or does so at an increased filling pressure
The common causes in Nigeria include hypertension, dilated cardiomyopathy and rheumatic heart disease.
Cardiac failure may be acute or chronic with the latter being the most frequently encountered in the Nigerian setting because of late presentation

Clinical features

Major Diagnostic criteria:
Paroxysmal nocturnal dyspnoea
Orthopnoea
Raised jugular venous pressure
S3
Pulmonary edema
Minor diagnostic criteria
Signs include:
Cough productive of frothy sputum
Leg swelling
Abdominal swelling
The prominence of particular symptoms will depend on which side is affected
Oedema
Tachycardia (about 100 beats per minute)
Displaced apex
Abdomen: hepatomegaly, Ascites
A diagnosis of heart failure requires 2 major or I major and 2 or more minor criteria

Differential diagnoses

Bronchial asthma
Chronic obstructive airways disease (COAD)
Chronic kidney disease
Chronic liver disease

Complications

Thrombo-embolic phenomena: stroke, pulmonary embolism
Pre-renal azotaemia

Arrhythmias

Investigations
Chest radiograph
Electrocardiography
Echocardiography Urea, Electrolytes and Creatinine
Fasting blood glucose
Urine micro-analysis
Full Blood Count with differentials

Treatment objectives
Relieve symptoms
Treat cause where feasible
Treat precipitating factors
Enhance quality of life
Prevent complications
Prolong life

Non-drug treatment
Bed rest
Low salt diet
Exercise (within limits of tolerance)
Stop cigarette smoking
Avoid excessive alcohol

Drug treatment
Diuretics
- Furosemide 40 - 160 mg intravenously or orally
- Spironolactone 25 daily

Potassium supplements

- Potassium chloride 600 mg orally once, every 8 – 12 hours daily depending on the serum levels of potassium

Vasodilators

- Angiotensin converting enzyme inhibitors (ACEIs)

Captopril 6.25 - 25 mg every 12 hours preferably at bedtime

Or:

Lisinopril 2.5 - 20 mg daily especially if there is hypertension

Cardioselective blockers (moderate to severe cardiac failure)

- Carvedilol 3.125 – 25 mg daily. Initially 3.125 mg once to twice daily(with food); dose increased at intervals of at least 2 weeks to 6.25 mg twice daily, then to 12.5 mg twice daily, then to 25mg twice daily; increase to highest dose tolerated, max. 25 mg twice daily in patients with severe heart failure or body weight less than 85 kg and 50 mg twice daily in patients over 85 kg

Or

- Metoprolol 25 -150mg daily

Venodilators

- Nitrates

Glyceryl trinitrate 0.3 - 1 mg sublingually and repeated as required

Ionotropes

Digoxin

- 125 - 250 micrograms daily (the elderly may require 62.5 - 125 micrograms daily)
- Dopamine 2 - 5 microgram/kg/minute by intravenous infusion

Anticoagulants

- Warfarin: monitor INR 2 - 2.5
- Important in atrial fibrillation

Supportive measures

Pacemakers for arrythmias

Ventricular assist devices

Notable adverse drug reactions

Digoxin: arrhythmias

Potassium-sparing drugs: hyperkalaemia

ACEIs: hypotension, hyperkalaemia

Do not combine potassium supplements with potassium-sparing drugs

Precautions

The dose and infusion rate for dopamine are critical

- Low dose infusion rates will cause excessive hypotension
- Higher infusion rates will elevate the blood pressure

The use of P blockers, atrial natriuretic peptide analogues and endothelin receptor antagonists should be reserved for specialist

care

Prevention

Adequate treatment of hypertension and diabetes mellitus

Good sanitation and personal hygiene (to prevent rheumatic fever)

HYPERLIPIDAEMIA

Introduction

An increase in plasma lipid levels:

Cholesterol, or its fractions, or triglyceridaemia can be primary (hereditary) or secondary with underlying diseases especially diabetes mellitus and hypertension

A major risk factor for ischemia heart disease

Clinical features

Patients present with ischaemic heart disease or the underlying cause hyperlipideaemia

Signs include xanthomata, xanthelasmata, and corneal arcus

Differential diagnoses

Primary hyperlipidaemia

Secondary hyperlipidaemia: diabetes mellitus,

Nephrotic syndrome

Complications

Ischaemic heart disease

Peripheral vascular disease
Stroke,
Hypertension

Investigations
Lipid profile
Urea, Electrolytes and Creatinine
Fasting blood glucose

Urine proteins
Serum proteins (total and differential)

Treatment objectives
Lower lipid levels
Prevent or treat complications
Treat underlying causes

Non-drug treatment
Stop smoking
Reduce weight
Exercise moderately and regularly
Water soluble fibre: oat, bran

Drug treatment
Atovastatin

Notable adverse drug reactions, caution and contraindications
Caution in patients with history of liver disease, high alcohol intake
Hypothyroidism should be adequately

managed before starting treatment with a statin

Liver function tests mandatory before and within 1 - months of starting treatment; thereafter at intervals of 6 months for 1 year

Statins may cause reversible myositis, headache, diarrhoea, nausea, vomiting, constipation, flatulence, abdominal pain; insomnia

Prevention

Dietary manipulation

Early identification of individuals at risk

HYPERTENSION

Introduction

A persistent elevation of the blood pressure above normal values (≥ 140/90 mmHg) taken 2-3 times on at least two different occasions

The commonest non-communicable disease in Nigeria

Clinical features

Largely asymptomatic until complications arise ("silent killer")

Symptoms and signs of target organ diseases e.g. cardiac failure, stroke and chronic kidney disease

Complications

Heart: Heart failure, ischaemic heart disease

Brain: Stroke (ischaemic, hemorrhagic)
Eye: Hypertensive retinopathy
Kidney: Renal failure
Peripheral artery disease

Investigations
Urinalysis; urine microscopy
Electrolytes, Urea and Creatinine. Uric acid
Fasting blood glucose
Lipid profile
Chest radiograph
Electrocardiography
Others as may be indicated:
Echocardiography
Abdominal ultrasound
Renal angiography

Treatment objectives
Educate patient about disease and need for treatment adherence
Reduce blood pressure to acceptable levels
Prevent complications (primary, secondary, tertiary)

Non-drug treatment (lifestyle modification)
Low salt diet: Not more than 1 level teaspoon of salt per day; No added salt; Avoid food preserved with salt
Achieve/maintain ideal body weight (BMI 18.5 - 24.9 Kg/m^2)
Stop smoking years

Reduce alcohol intake
Regular exercise
Reduce polysaturated fatty acid intake

Drug treatment

Principles of drug treatment

- Treatment should be individualized
- Most patients will require combination chemotherapy using drug from different classes
- Fixed dose combination is desirable when 2 or more drugs are required
- Drugs with at least 24 hours duration of action to ensure once daily dosing
- Diuretics should be included unless contraindicated
- ACEI and beta blockers are ineffective when used as monotherapy in blacks
- Treat coexisting cardiovascular risk factors
- All patients require lifestyle modifications

Choice of drugs

Diuretics: years:

Thiazides

- Bendroflumethiazide 2.5 - 10 mg orally daily

Or:

- Hydrochlorothiazide 12.5- 50 mg orally daily

Or:

- Hydrochlorothiazide/amiloride 25/2.5 mg daily

Beta Blockers:
Atenolol 25 - 100 mg orally daily
Calcium channel antagonists:
Nifedipine retard 20 - 40 mg orally once or twice daily

Or:
Amlodipine 2.5 - 10 mg orally once daily
Angiotensin converting enzyme inhibitors:
Captopril 6.25 - 50 mg orally once or every 8 - 12 hours

Or:
Lisinopril 2.5 - 20 mg orally once daily
Angiotensin receptor blockers:
Losartan 50 - 100 mg orally daily
Valsartan 80-160 mg daily
Other vasodilators:
Hydralazine 25 - 100 mg orally once daily or every 12 hours

Or:
Prazosin 0.5 - 1 mg orally daily
Centrally acting drugs:
Alpha methyldopa 250 - 500 mg orally twice, three or four times daily

Hypertensive emergencies

Treatment should be done by the experts:
Involves the administration of antihypertensives by the parenteral route

(usually intravenous hydralazine or sodium nitoprusside)

Supportive measures

Patient/care giver education

Notable adverse drug reactions, caution and contraindications

Angiotensin converting enzyme inhibitors, angiotensin receptor blockers: angioedema; dry cough with ACEIs.

Alpha methyldopa, thiazides and potentially other anti-hypertensive drugs may cause erectile dysfunction

Alpha methyl dopa may cause postural hypotension

SLE-like syndrome: hydralazine

Do not use beta blockers in asthmatics and heart failure

Prevention

Weight reduction

Exercise moderately and regularly

Public education

Individual and Population based approaches

Advocacy for the positive lifestyle change

Target blood pressure: **BP< 140/90 mmHg for general population. BP < 130/80 mmHg for patients with diabetes or end stage renal disease**

Hypertension in pregnancy

ACEI and ARB are terratogenic, contraindicated in pregnancy & to be used with caution in women in reproductive age group. Alpha methyl dopa, hydralazine, calcium channel blockers are safe in pregnancy. Diuretics are relatively safe.

INFECTIVE ENDOCARDITIS

Introduction

A microbial infection of the endocardium and diseased heart valves (rheumatic heart disease, congenital heart disease, shunts, and prosthetic valves).

May be acute or sub-acute

Some acute cases occur in normal valves in intravenous drug users or may be part of systemic illness

Sub-acute form usually occurs on diseased valves

Causative organisms include:

Streptococci, Staphylococci, Enterococci; Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, and *Kingella* species ('Hacek' Organisms)

Clinical features

Acute:

High fever with rigors

Delirium

Shock

Development of new murmurs
Severe cardiac failure
Abscesses may form in many parts of the body (e.g. brain)
Subacute:
Low-grade fever
Signs of carditis
Finger clubbing
Arthralgia
Splenonegaly
Osler's nodules
Janeway lesions
Roth spots

Differential diagnoses
Myocarditis
Rheumatic heart disease

Complications
Cardiac failure
Destruction of heart valves
Systemic embolism (could be infective)

Investigations
Full Blood Count and differentials; ESR
Urinalysis; urine microscopy
Blood cultures X 3 (the yield is higher at the time of pyrexia)
Chest X-ray
ECG
Echocardiography

Treatment objectives
Stop the infection
Treat cardiac failure
Prevent coagulation disorders

Non-drug treatment
Bed rest
Low salt diet

Drug treatment
Initiate therapy (culture results awaited) with:
Benzylpenicillin 7.2 g daily by slow intravenous injection or intravenous infusion in 6 divided doses for 4 -6 weeks
- May be increased up to 14.4 g daily if necessary (e.g. in endocarditis)

Plus:
Gentamicin 60 - 80 mg intravenously 12 hourly for 2 weeks
Following bacteriological confirmation institute appropriate antimicrobial therapy
Staphylococci:
- Flucloxacillin: 250 mg - 2 g intravenously every 6 hours for 4 - 6 weeks
- Vancomycin: 1 gm intravenously 12 hourly; Gentamicin 60-80 mg intravenously 12 hourly for 2 weeks

Enterococci:
Amoxicillin 2 gm intravenously 4 hourly;

intravenously Gentamicin 60-80 mg 12 hourly
Candida: Systemic antifungals

Notable adverse drug reactions
Penicillin: rashes, anaphylaxis Gentamicin: nephropathy
Monitor patients on gentamicin and vancomycin carefully

Prevention
Prophylactic antibiotics for patients at risk who are undergoing:

1. Dental procedures
   Under local or no anaesthesia, for those who have NOT had endocarditis, and have NOT received more than a single dose of penicillin in the last one month:
   Amoxicillin Adult: 3g orally 1 hour before procedure
   Child under 5 years: 750 mg orally 1 hour before procedure; 5-10 years: 1.5 g
   For penicillin-allergic patients or patients who have received more than a single dose of a penicillin in the previous one month:
   Azithromycin
   Adult: 500 mg orally one hour before procedure
   Child under 5 years: 200 mg orally; 5 - 10 years: 300 mg Patients who have had endocarditis:

- Amoxicillin plus gentamicin intravenously as for procedures under general anaesthesia (see below)

Dental procedures under general anaesthesia, and no special risk:

Amoxicillin

Adult: 1 g intravenously at induction of anaesthesia; mg orally 6 hours later

Child under 5 years: a quarter of adult dose; 5 - 10 half adult dose

Or:

Adult: 3g orally 4 hours before induction, then 3g as soon as possible after the procedure

Child under 5 years: a quarter of adult dose; 5 – 10 half adult dose

Special risk, e.g. previous infective endocarditis, or patients with prosthetic valves:

Amoxicillin plus gentamicin intravenously

Adult: 1 g amoxicillin plus 120 mg gentamicin at induction

- Then oral amoxicillin 500 mg 6 hours after procedure

  Child under 5 years: a quarter of adult dose of plus 2 mg/kg gentamicin intravenously at induction 5 - 10 years: half adult dose for amoxicillin; 2 mg/kg gentamicin

  Patients who are penicillin-allergic or have received more than a single dose of a penicillin in the last one month:

Vancomycin

Adult: 1g intravenously over at least 100minutes

Plus

Gentamicin

Adult: 120 mg intravenously - Given at induction or 15 minutes before procedure

Child under 10 years: Vancomycin 20 mg/kg; gentamicin 2 mg/kg

2. Genito-urinary tract manipulation

   As for special risk patients undergoing dental procedures under general anaesthesia

3. Obstetrics, gynaecological and gastrointestinal procedures

   As for genitourinary tract manipulation

MYOCARDITIS

Introduction

Acute inflammatory process affecting the myocardium that may occur in association with endocarditis and pericarditis.

Possible causes:

Infections: viral including HIV, bacterial, protozoa

Toxins e.g. scorpion sting

Poisons e.g. alcohol

Drugs/ Allergy e.g. penicillin

Deficiencies e.g. thiamine

Physical agents e.g. radiation

Clinical features
Largely asymptomatic
A few may present with palpitations; symptoms of cardiac failure
Physical examination:
Arrhythmias
Tachycardia
Raised JVP
Cardiomegaly
S3 or S4 (with or without murmurs of regurgitation in the mitral/tricuspid areas)

Differential diagnoses
Other forms of cardiac failure, e.g. peripartum cardiomyopathy

Complications
Cardiac failure
Arrhythmias
Thrombus formation

Investigations
Electrocardiography
Echocardiography
Full Blood Count and differentials, ESR
Urea, Electrolytes and Creatinine
Cardiac enzymes
Endomyocardial biopsy

Treatment objectives
Eliminate/withdraw the offending agent(s)

Treat the effect on the heart
Treat complications

Non-drug treatment
Bed rest

Drug treatment
Treat underlying cause(s)
Anti arrhythmics (depends on the type of arrhythmias)
Anticoagulant: warfarin
Anti-cardiac failure: digoxin, diuretics, potassium supplements
Steroids: prednisolone (not in all cases)
Multivitamins
Anti-oxidants: ascorbic acid (vitamin C), vitamin E

Notable adverse drug reactions
Antiarrhythmics may be pro-arrhythmic
Anticoagulants: bleeding
Steroids: fluid retention, dyspepsia
Diuretics: dehydration, electrolyte imbalance

Prevention
Prevent infection (viral, bacterial, etc)
Prevent exposure to toxins
Nutrition education

PAEDIATRIC CARDIAC DISORDERS
(Refer for Specialist Care)

CONGENITAL HEART DISEASE

Introduction

A heart defect that occurs during the formation of the heart in utero could be fatal (i.e. causes intrauterine death, or death at anytime afterwards)

- An important cause of perinatal morbidity/mortality

Classified as
Cyanotic
Acyanotic

Clinical features

Will depend on the type of the defect:
Mild defects go unnoticed
Stunted growth
Cyanosis
Failure to thrive
Heart murmurs

Differential diagnoses

Rheumatic heart disease
Endomyocardial fibrosis

Complications

Embolic phenomena
Cardiac failure

Investigations

Full Blood Count and differentials
Urea, Electrolytes and Creatinine

Chest radiograph
Electro cardiograhy
Foetal echocardiography
Angiography

Treatment objectives
Relieve symptoms
Treat the definitive defect(s)

Non-drug treatment
Low salt diet

Drug treatment
Treatment of cardiac failure if present
- Digoxin, diuretics and potassium supplements

Supportive measures
Oxygen
Counselling

Prevention
Pre-conception nutrition education
Antenatal care
Genetic counselling

HEART FAILURE IN CHILDREN

Introduction:

Heart failure is a clinical syndrome in which the heart is unable to pump enough blood to meet the metabolic demands of the body

despite adequate atrial filling.

Causes:

- Unlike adults, the most common causes of heart failure in children are:
- Congenital heart diseases (CHDs),
- Non-cardiac causes including pneumonia, severe anaemia
- In neonates, metabolic factors such as hypoglycemia, hypoxia and acidosis
- Acquired heart diseases especially rheumatic heart diseases and cardiomyopathies.

Clinical features:

- Breathlessness, cough
- Sweating on feeding (diaphoresis especially on feeding)
- Feeding difficulty: 'suck-rest-suck cycle' as a result of effort dyspnoea
- Poor weight gain/Failure to thrive
- Pedal or facial swelling
- Features of underlying disease
- Note! Cardinal signs for the diagnosis of heart failure in children are
- tachypnoea, tachycardia, tender hepatomegaly, ± cardiomegaly

Investigations:

- Chest X-ray,
- ECG,
- Echocardiography,
- Cardiac catheterization.
- Electrolyte, urea and creatinine

- Packed cell volume
- Others-depending on the cause

Treatment Objectives

- Decrease preload
- Relieve symptoms of pulmonary and systemic venous congestion.
- Increases myocardial contractility

Drug treatment:

Diuretics:. Frusemide (1-2mg/Kg/day), Spironolactone (1-2mg/Kg/day)

Inotropes:

Digoxin

Dosage regimen of digoxin)

Age	*Digitalization (mg/kg/day)	Maintenance (mg/kg/day)
<1 month	0.04-0.06	0.01
1 month - 2 years	0.04-0.08	0.01-0.02
>2 years	0.04-0.06	0.01
Adult	0.05-1.0 (mg/day)	0.25-0.5 (mg/day)

**How to Digitalise: give half the total digitalizing dose (TDD) immediately, followed by ¼ and then the final ¼ of the TDD at 6-8hour intervals.*

Maintenance dose: Start maintenance dose 12hours after the final TDD. Maintenance dose is 25% of TDD

Other inotropes: Dopamine and Dobutamine in ICU or high dependent unit

ACE inhibitors:

Captopril, Enalapril (in patients with Left to right shunts: (VSD, PDA, left sided regurgitatant lesions (mitral or aortic regurgitations) or poor systolic function (dilated cardiomyopathy, myocarditis)

Beta-blockers: Carvidilol in chronic heart failure

Dilated cardiomyopathy

Non-drug treatment:

- Bed rest- reduces demand on the heart
- Oxygen therapy
- Nurse in prop up (Cardiac) position
- Correct acidosis
- Nutritional rehabilitation- ensure adequate caloric intake, give frequent small feeds
- *Salt restriction in form of low salt formula- not recommended in infants*

Treatment of underlying cause-

- Blood transfusion- for anaemia,
- Antibiotics for pneumonia
- Surgical repair of congenital heart defects.

Notable Adverse Reactions

As in adults

PERICARDITIS

Introduction

An inflammation of the pericardium, which may arise from viral, bacterial, fungal or protozoal infections

Other causes: metabolic, malignancy,

connective tissue disease, radiation, trauma etc
May be acute or chronic

Clinical features
Acute pericarditis:
Chest pain
- Retrosternal
- Sharp
- Radiating to the left shoulder
- Made worse by breathing or coughing
- Relieved by the upright position

Low-grade fever
Pericardial friction rub
Chronic pericarditis:
- Insidious onset

There may be:
- Dyspnoea on exertion
- Leg and abdominal swelling

Differential diagnoses
Endomyocardial fibrosis
Sarcodosis
Amyloidosis

Complications
Pericardial tamponade
Constrictive pericarditis

Investigations
Electrocardiography

Full Blood Count and differentials
Chest radiograph
Echocardiography

Treatment objectives
Relieve distress from pain and tamponade
Relieve constriction
Treat the effect on the heart
Treat complications
Eradicate the organism (if cause is infection)

Non-drug treatment
Bed rest

Drug treatment
NSAIDs
- Indomthaem 50 mg orally every 8 hours

Or:
- Ibuprofen 400 - 800 mg orally every 12 hours Steroids
- Prednisolone 30 mg orally every 8 hours and tapered
- Anti-tuberculous drugs or other antimicrobial agents (if mycobacterium or other microbes are causative)

Supportive measures
Pericardiocentesis
Pericardiectomy
Notable adverse drug reactions
NSAIDs/steroids: dyspepsia and upper GI

bleeding

Prevention
Avoid radiation
Prevent infection

PULMONARY OEDEMA
Introduction
- Occurs when there is congestion of the lungs with fluid, usually in a scenario of left-sided cardiac failure
- Results in stiffness of the lungs and flooding of the alveoli, with difficulty in breathing
- May also follow inflammatory processes
- May be acute or chronic

Clinical features
- Difficulty in breathing, with a sensation of drowning
- Cough productive of frothy (sometimes pink) sputum
- Central cyanosis
- Sweating, agitation etc
- Other symptoms of left-sided cardiac failure
- Examination:
- Wide-spread crepitations
- Rhonchi (in severe cases)
- Other signs of left-sided cardiac failure

Differential diagnoses

Pulmonary embolism
Pneumonia

Complications
Hypoxaemia
Coma

Investigations
- Chest radiograph
- Electrocardiography
- Echocardiography
- Blood gases
- Urea, Electrolytes and Creatinine
- **D-Dimer**

Treatment objectives
Relieve oedema
Relieve discomfort
Treat underlying cause

Non-drug treatment
Propped up position
Bed rest
Sit on bed with legs hanging down

Drug treatment
Oxygen 3 - 5L/min
Morphine 10 mg stat
Loop diuretics - Furosemide 40 - 120 mg intravenously stat; maintenance with 40 - 500 mg daily in single or divided doses
Venodilator: 0.3-1 mg by mouth or 10-200

microgram/min intravenously

Vasodilator: Hydralazine 25-50 mg 12 hourly; ACEI (Captopril: 6.25-25 mg by mouth)

Aminophylline 250-500 mg or 5mg/Kg intravenously over 10 minutes

Supportive measures

Nursing care (e.g. nurse in cardiac position)

Notable adverse drug reactions

Diuretics: hpokalaemia

ACEIs: First dose hypotension, dry cough, hyperkalaemia

Nitrates: Hypotension

Aminophylline: Arrhythmias

Prevention

Treat cause(s) of cardiac failure or fluid overload (e.g. renal failure)

Judicious administration of blood and intravenous fluids

RHEUMATIC FEVER

Introduction

A result of abnormal reaction of antibodies developed against antigens of group A B-haemolytic streptococcus

Infection is usually of the throat; occasionally the skin in a sensitized individual

Antigen-Antibody complex damages the

heart (endocardium, myocardium and pericardium)
Commonest streptococcal strains in Africa are C and G

Clinical features
Duckett-Jones' diagnostic criteria
Major:
Carditis
Sydenham's chorea
Erythema marginatum
Subcuoeous nodules
Arthritis (migratory polyarthritis)
Minor:
Fever
Leucocytosis
Arthralgia
Raised ESR
Raised ASO titre (> 200 IU)
Prolonged PR interval
Supporting evidence of antecedent group A streptococcal infection:
Positive throat culture or rapid streptococcal antigen
Diagnosis
2 major criteria

Or:
1 major **plus** 2 (or more) minor criteria

Differential diagnoses
Malaria
Viral infection
Pyrexia of undetermined origin
Connective tissue disease

Complications
Rheumatic heart disease
Arrhythmias
Cardiac failure

Investigations
Full Blood Count and differentials
ASO titre
ESR
Electrocardiograph
Echocardiography
Chest radiograph
Throat swab for microscopy, culture and sensitivity

Treatment objectives
Relieve symptoms
Treat the bacterial throat infection
Reduce or abolish inflammatory process
Treat cardiac failure if present

Non-drug treatment
Bed rest

Drug treatment

Antibiotics

- Penicillin V

Adult: 500 mg orally every 6 hours, increased up to 1g 6hourly in severe infections

Child: 1 month - 1 year 62.5 mg orally every 6 hours increased in severe infection to ensure at least 12.5mg/kg/dose

1 - 6 years: 125 mg every 6 hours increased in severe infection to ensure at least 12.5 mg/kg/dose

6 - 12 years 250 mg every 6 hours, increased in severe infection to ensure at least 12.5 mg/kg/dose

12 - 18 years 500 mg every 6 hours, increased in severe infection up to 1 g/dose

Or

- Erythromycin

Adult and child over 8 years: 250 - 500 mg orally every 6 hours **or** 500 mg - 1 g every 12 hours; up to 4 g daily in severe infections

Child: up to 2 years, 125 orally mg every 6 hours; 2 – 8 years 250 mg every 6 hours; doses doubled for severe infections

Salicylates - Aspirin (acetylsalicylic acid)

Adult: 300 mg - 1 g orally every 4 hours after food; maximum dose in acute conditions 8 g daily

Child: not recommended for use

Steroids (if salicylates are ineffective)

- Prednisolone

- Initially, up to 10 - 20 mg orally daily; up to 60 mg daily in severe disease (preferably taken in the morning after breakfast); dose can often be reduced within a few days, but may need to be continued for several weeks or months
- Maintenance 2.5 -15 mg orally daily

Prophylaxis against rheumatic fever

Benzathine penicillin 720 mg (1.2 million units) intramuscularly 3 - 4 weekly until the age of 25 years or for life

Notable adverse drug reactions

Penicillin: anaphylactic reaction

Salicylates; steroids: peptic ulceration

Cushingnoid effects are increasingly likely with doses of prednisolone above 7.5 mg daily

Prevention

Good sanitation.

School surveys - identify carriers of streptococcus and treat

Secondary prevention and prophylaxis against endocarditis

RHEUMATIC HEART DISEASE

Introduction

A complication of rheumatic fever

A common cause of cardiac failure in Nigeria

In Africa manifests later compared to

Caucasians
The mitral valve is most affected, followed by the aortic, then the tricuspid
The lesions can occur in various combinations of regurgitation and stenosis

Clinical features
Exertional dyspnoea
Paroxysmal nocturnal dyspnoea
Orthopnoea
Leg and abdominal swelling
Cough with production of frothy sputum
Pedal and sacral oedema
Small volume pulse, which may be irregular
With or without tachycardia
With or without hypotension
Raised JVP
Displaced apex
Left ventricular hypertrophy
Right ventricular hypertrophy
Thrills
Palpable P2
Soft S1; loud P2
S3 or S4
Systolic/diastolic murmurs

Differential diagnoses
Constrictive pericarditis
Endomyocardial fibrosis
Dilated cardiomyopathy

Complications
Arrhythmias e.g. atrial fibrillation, heart block
Cardiac failure
Embolic phenomena
Endocarditis

Investigations
Chest radiograph
Electrocardiography
Echocardiography

Coronary angiography
Electrolytes, Urea and Creatinine

Treatment objectives
Relieve symptoms
Prevent recurrence of rheumatic attack
Repair and replace affected valves

Non-drug treatment
Bed rest
Low salt diet

Drug treatment
Treat for heart failure if present
Use anticoagulants if necessary
Prophylaxis against endocarditis (see Infective Endocarditis)
- Benzathine penicillin 720 mg (1.2 million units) intra musculary monthly for life

Other measures:

- Valve replacement
- Valve repair
- Treat endocarditis

Notable adverse drug reactions, caution

Penicillin may cause hypersensitivity reaction / anaphylaxis

- Caution in patients with a history of penicillin allergy

Prevention

Personal hygiene and good sanitation to prevent recurrence of rheumatic fever

CHAPTER 4:

CENTRAL NERVOUS SYSTEM

DIZZINESS

Introduction

Simply means 'light-headedness'

Usually due to impaired supply of blood, oxygen and glucose to the brain

May suggest some form of unsteadiness, or could precede a fainting spell

Causes:

Side effects of medications, notably anti-hypertensives and sedatives

Anaemia

Arrhythmias

Fever

Hypoglycaemia

Brain stem lesions

Alcohol overdose

Excessive blood loss

Prolonged standing

Autonomic neuropathy (especially in diabetic
patients)

May be accompanied by vertigo (giddiness) in some individuals

May culminate in loss of consciousness

Clinical features
Light-headedness
Feeling faint especially on attempting to stand or after squatting
Weakness

Differential diagnoses
Benign positional vertigo
Labyrinthine disorders
Hysteria
Premonitory symptoms of epilepsy
Migraine aura
Warning symptom of posterior circulation stroke (posterior inferior cerebellar artery)
Cervical spondylosis with compression of vertebral artery
Brain tumour (acoustic neuroma)

Complications
Falls with injury
Stroke
If due to intracranial tumour: raised intracranial pressure with coning
If due to other intracranial pathology: cranial nerve palsies

Investigations
Full Blood Count and differentials
Electrocardiography

Echocardiography
Random blood glucose
X-ray sinuses
Neuro-imaging: CT scan, MRI, carotid Doppler etc

Management
Depends on the aetiological factor identified

Treatment objectives
Eliminate symptom
Prevent recurrence
Drug treatment will depend on underlying cause(s)

Non-drug treatment
Stop all medicines suspected to be responsible
Physiotherapy: pressure stockings

Drug treatment:
Prochlorperazine/Cinnarazine for severe attacks
Aspirin tablets as anti-platelet agent

Notable adverse drug reactions, cautions, contraindications etc.
Aspirin and other NSAIDs to be used with caution in patients with history of dyspepsia, asthmatics (especially aspirin
Prevention:

Avoid precipitants
These must be identified early for effective prevention

HEADACHES

Introduction

The commonest neurological disease in Nigeria
Defined as pain or discomfort in the head and the surrounding structures
They may be:
Primary (idiopathic)
Secondary

Primary headache types

Tension type
Migraine with or without aura
Cluster headache

Secondary causes

Intracranial space-occupying lesions like brain tumours, subdural haematoma
Vascular lesions: strokes
Infections
Following generalized convulsions
Metabolic derangements
Alcohol hangover
Drugs
Irritation of sensory cranial nerves
Inflammation or diseases of structures/organs in the head region: eyes,

nose, sinuses, ears, cervical vertebrae

Atypical headache
Sleep disorders (hypoxia)
Brain stem malformations
HIV infection
Clinical features
Depend on the underlying type/cause(s):

Tension type
Heaviness in the head
Crawling sensation
"Peppery sensation"
Tight-band sensation
Poor sleep
Disturbed concentration

Cluster type
Recurrent, frequent, brief attacks of disturbing pain in the head
Pain around the eyes and forehead
Redness of the eyes
Nasal stuffiness
Drooping of the eyelids

Migraine headache
- See below

Secondary headaches: presence of additional symptoms:
Fever

Vomiting
Neck stiffness
Alteration in level of consciousness
Convulsions
Cranial nerve deficits
Limb weakness (hemiparesis, quadriparesis)
Papilloedema as evidence of raised intracranial pressure
Evidence of disease in other organs
Evidence of drug or alcohol abuse

Persistent daily Headache
- daily attacks of pain on the head
- mimics tension-type pain and could have pulsating quality
- associated with chronic use of analgesic drugs which then sets up a vicious cycle
- may point to underlying stress or emotional disturbance

Differential diagnoses
Meningitis
Hysteria
Refractive error
Cervical spondylosis
Brain tumour
Haemorrhagic stroke

Complications
Depend on the cause and type

Some are benign with no sequelae apart from reduced economic productivity/wastage of medications
Coning (depending on cause)
Blindness (following temporal arteritis, unrelieved raised intracranial pressure)

Investigations
Neuro-imaging: skull X-ray, computerized tomographic scan, MRI
Electroencephalography
Cerebrospinal fluid examination for pressure, cells and chemistry
Erythrocyte sedimentation rate

Treatment objectives
Eliminate pain
Treat the precipitating factor or disease
Prevent recurrent attacks

Non-drug treatment
Psychotherapy
Physiotherapy/biofeedback

Drug treatment
Primary headaches
Simple analgesics and non-steroidal anti-inflammatory agents
Tricyclic antidepressants
- Amitriptyline 10 - 25 mg daily at night

Anxiolytics

- Lorazepam 1 - 2.5 mg at night. Use lower doses for the elderly patient

Stop analgesic use in individuals with persistent daily headaches

Secondary headaches

Medical or surgical management of identified causes

Antibiotics for infections like meningitis, sinusitis Steroids for vasculitis

Notable adverse drug reactions, caution

Aspirin and other NSAIDs: use with caution in patients with history of dyspepsia, and asthma

Tricyclic antidepressants: use with caution in respiratory diseases, cardiac symptoms, muscle weakness and myasthenia gravis, organic brain damages, history of drug or alcoholic dependence, personality disorder, may increase risk of dependence, avoid prolonged use or abrupt withdrawal thereafter. Drowsiness may affect performance of skilled tasks(e.g. driving), effects of alcohol enhanced anticholinergic effects e.g. urinary retention in the elderly

Prevention

Reduce stress levels

Prophylactic medications if attacks last more than 15 days a month, or are severely

incapacitating (in the absence of other causes)
Early detection and correction of refractive errors, sinusitis, oto-rhino-laryngologic and dental problems.

MENINGITIS

Introduction

An infection of the meninges with the presence of pus and inflammatory cells in the cerebrospinal fluid
A medical emergency, and associated with considerable morbidity and mortality
May be bacterial (pneumococcus, meningococcus, tubercle bacilli, *Haemophilus),* viral, fungal, protozoal, neoplastic or chemical
Organism may vary with age of the patient
May occur in epidemics in the Savannah region
Epidemic meningitis is usually due to *Neisseria meningitidis*

Clinical features

Fever
Headache
Vomiting
Photophobia
Alteration in level of consciousness
Neck stiffness and positive Kernig's sign
May present in epidemics

Systemic features of infection by offending organisms include:

Pneumococcal and Haemophilus infection
- Jaundice, pneumonia, heart failure

Meningococcal infection
- Joint pain, joint swelling, red eyes, skin rash

Tuberculous infection
- Weight loss, cough with blood in sputum

HIV infection
- Severe weight loss, diarrhea, mouth lesions, skin rash

Other presentations

Fever of unknown origin: chronic meningitis

Mass lesion with focal neurological deficits: tuberculoma, empyema

Stroke-like syndrome: resulting from inflammation of blood vessels

Seizures which may be uncontrolled and prolonged (status epilepticus)

Acute psychosis (Organic Brain Syndrome)

Dementia

Differential diagnoses

Subarachnoid haemorrhage

Tetanus

Brain abscess

Cerebral malaria

Septicaemia with meningism

Complications

Cranial nerve palsies notably blindness and deafness
Subdural pus collection (empyema)
Stroke
Epilepsy
Heat stroke
Syndrome of Inappropriate Anti-Diuretic Hormone secretion (SIADH)

Investigations

Lumbar puncture for CSF analysis
- To demonstrate presence of inflammatory cells (after exclusion of raised intracranial pressure by fundoscopy or CT scan)

Full Blood Count and differentials
Blood culture
Erythrocyte sedimentation rate
Random blood glucose
Electrolytes, Urea and Creatinine
Chest radiograph
Mantoux test (if tuberculosis is suspected)
HIV screening

Management

Treatment objectives

Eliminate the organism
Reduce raised intracranial pressure
Correct metabolic derangements

Treat complications (if any)

Non-drug treatment

Tepid-sponging

Attention to calories and fluid/electrolyte balance

Physiotherapy (for passive muscle exercises)

Nursing care (e.g. frequent turning and bladder care) to prevent decubitus ulcers and urinary tract infection

Drug treatment

Initial therapy will depend on the age of the patient (and causative agent)

Bacterial infections

Third generation cephalosporins:

Ceftriaxone is the drug of first choice

1 g daily by deep intramuscular injection, or by intravenous injection over at least 2-4 minutes, or by intravenous injection; 2-4 g daily in severe infections; intramuscular doses over 1 g should be divided between more than one site; single intravenous doses above 1 g by intravenous infusion only

Neonate

By intravenous infusion over 60 minutes, 20-50 mg/kg daily(max. 50mg/kg daily)

Children under 50kg, by deep intramuscular injection or by intravenous infusion, 20-50 mg/kg daily; up to 80mg/kg daily in severe

infections; doses of 50 mg/kg and over by intravenous infusion only

Or:
Penicillin V 2 - 4 g by slow intravenous injection every 4 hours

Or:
Chloramphenicol 100 mg/kg intravenously every 6 hours

- May be useful for. *influenza*infection

Tuberculosis:
Standard anti-tuberculous drugs (including pyrazinamide and isoniazid for their good penetration of the blood-brain barrier)

Anti-pyretics: Aspirin (acetylsalicylic acid)
Adult: 300 mg - 1 g orally every 4 hours after food; maximum dose in acute conditions 8 g daily *Child:* not recommended for use

Diazepam (for seizures)
Adult: 10 - 20 mg at a rate of 0.5 ml per 30 seconds, repeated if necessary after 30 - 60 minutes; may be followed by intravenous infusion to a maximum of 3 mg/kg over 24 hours
Child: 300 - 400 micrograms/kg (maximum 20 mg) by slow intravenous injection into a large vein for protracted or frequent recurrent convulsions

- Not required in single, short-lived convulsions Acute cerebral decompression:

Furosemide

Adult: 40 - 80 mg every 8 hours by slow intravenous injection (for a maximum of 6 doses)

Child: neonate 0.5 - 1 mg/kg every 12 - 24 hours (every 24 hours in neonates born before 31 weeks gestation)

1 month - 12 years: 0.5 - 1 mg/kg (maximum 4 mg/kg), repeated every 8 hours as necessary

12 - 18 years: 20 - 40 mg, repeated every 8 hours as necessary; higher doses may be required in resistant cases

Or:

Mannitol 20% solution

Adult: 50 - 200 g by intravenous infusion over 24 hours, preceded by a test dose of 200 mg/kg by slow intravenous injection

Child: neonate 0.5 - 1 g/kg (2.5 - 5 ml/kg of 20% solution) repeated if necessary 1 - 2 times after 4 - 8 hours

1 month - 18 years: 0.5 - 1.5 g/kg (2.5 - 7.5 ml/kg of 20% solution); repeat if necessary 1 - 2 times after 48 hours

Chemoprophylaxis

Treat contacts during meningococcal epidemics with either ciprofloxacin or rifampicin

- Rifampicin

Adult: 600 mg orally every 12 hours for 5 days

Child: 10 mg/kg orallyevery12 hoursfor5days
Under 1 year: 5 mg/kg orallyevery12 hours for 5 days

- Ciprofloxacin

Adult: 500 mg orally as a single dose
Child: 5 - 12 years 250 mg orally as a single dose

Notable adverse drug reactions, caution and contraindications

Diazepam

- Must be administered slowly intravenously to avoid respiratory depression

Chloramphenicol

- May cause aplastic anaemia
- Mannitol
- May cause chills and fever
- Extravasation causes inflammation and thrombophlebitis
- Contraindicated in congestive cardiac failure and pulmonary oedema

Prevention

Immunize against communicable diseases

- Meningococcus, heamophilus, streptococcus (especially for sicklers).

Chemoprophylaxis (Rifampicin or ciprofloxacin)

- As determined by national policy
- For close contacts of clinical cases

MIGRAINE

Introduction

Headache resulting from changes in the calibre of certain blood vessels in the brain with resulting physical, autonomic and emotional disturbance

It can be very incapacitating

Each headache attack lasts for a few hours to a maximum of 3 days but can be aborted with appropriate intervention

Affects more females than males, usually between the ages of 15 and 50 years

Clinical features

Vascular Headaches

Common migraine (or migraine without aura)

- Throbbing pain usually affecting one side of the head around the temples, associated nausea and vomiting
- Dislike of light and noise

Classical migraine (or migraine with aura):

- Attacks of pain preceded by seeing flashes of light
- Disturbances in the field of vision (scotomas)
- Visual hallucinations

Childhood periodic syndromes:

- Abdominal pain and vomiting
- Alternating hemiplegia
- Benign positional vertigo

Basilary artery migraine - predominantly brain stem symptoms
- Dysarthria
- Vertigo
- Tinnitus
- Decreased hearing
- Diplopia
- Ataxia

May coexist with tension-type headache

May present without headache (migraine equivalent) usually seen in psychiatry

May present with complications:
- stroke-like manifestations
- ophthalmoplegia
- status attacks: unrelieved, persistent headaches

Differential diagnoses

Epilepsy, Hysteria, Glaucoma, Multiple sclerosis, Brain tumours

Complications

Stroke
Epilepsy
Blindness

Investigations

Neuro-imaging

Computerized tomographic scan
MRI
Electroencephalography

Treatment objectives
Eliminate pain
Prevent recurrence
Non-drug treatment
Manage in a quiet (and dark) room
Psychotherapy
Physiotherapy/biofeedback

Drug treatment
Acute attack
Aspirin (acetylsalicylic acid) tablets 300 - 900 mg every 4 - 6 hours when necessary maximum 4g daily. Child and adolescent - not recommend (risk of Reye's syndrome)

- With an anti-emetic agent (e.g. metoclopramide), or other non-steroidal anti-inflammatory agents plus metoclopramide

Ergotamine preparations (useful only during the aura phase)
Adult: 1 - 2 mg orally at first sign of attack; maximum 4 mg in 24 hours

- Do not repeat at intervals of less than 4 days; maximum 8 mg in any one week
- Not to be used more than twice in any one month

Child: not recommended

Narcotic analgesics – Pethidine, Codeine
Triptans – Sumatriptan.
Sumatriptan

Oral:
Initial dose: 25mg, 50mg, or 100mg orally, once. Dose may be repeated after at least two hours if migraine recurs, (max. 300mg in 24 hours)

Prophylaxis
Consider for patients who:
Suffer at least 2 attacks a month
Suffer an increasing frequency of headaches
Suffer significant disability in spite of suitable treatment for acute attacks
Cannot take suitable treatment for acute attacks
Available options are: Propranolol
- 40 mg orally every 8 - 12 hours

Tricyclic antidepressants, notably amitriptyline
- 10 mg orally at night, increased to a maintenance dose of 50 - 75 mg at night

Sodium valproate
- Initially 300 mg orally every 12 hours, increased if necessary to 1.2 g daily in 2 divided doses

In refractory cases: Cyproheptadine
- An antihistamine with serotonin-antagonist and calcium channel-blocking

properties

4 mg orally; a further 4 mg if necessary; maintenance 4 mg every 4 - 6 hours

Notable adverse drug reactions, caution and contraindications

Aspirin and other NSAIDs: use with caution in patients with history of dyspepsia and in asthmatics

Tricyclic antidepressants used with caution in patients with cardiac symptoms

Ergotamine: use should not exceed 4 - 6 mg per attack

- Caution in patients with vascular and renal disorders
- Not recommended for children

Opiates: risk of addiction

B-blockers: slow down cardiovascular function; reduce sensitivity to hypoglycaemia in diabetics

Prevention

Avoid precipitants

These must be identified for effective prevention

Reduce stress levels as much as possible

Give prophylactic medicines if attacks last more than 15 days a month, or are severely incapacitating (in the absence of other causes)

PARKINSONISM

Introduction

Synonyms: 'shaking palsy'; 'paralysis agitans'; 'akinetic-rigid syndrome'

A common neuro degenerative disease that results from deficiency of the neurotransmitter dopamine, in the striato-nigral pathway.The idiopathic variety is called Parkinson's disease while parkinsonism is used when an aetiologic agent is found

Causes: -

Drugs:

- Antipsychotics e.g. phenothiazines
- Antihypertensives: alpha methyl dopa, reserpine
- Infections:
  - Encephalitis
  - Typhoid fever
  - Vascular diseases:
  - Arteriosclerosis
  - Neurotoxins
  - Carbon monoxide
  - Manganese
  - Cyanide
  - Heroin analogues e.g. MPTP
  - Head trauma as in boxing Tumours

Metabolic diseases (Wilson's disease)

Idiopathic:- Parkinson's disease

Clinical features

Classical disease:

Rest tremors: coarse, distal tremors described as pill-rolling type

Rigidity

Slowness of movement; loss of arm swinging when walking

Retropulsion, propulsion, turning en bloc

Postural instability with frequent falls

Gait changes: shuffling gait with flexed posturing

Parkinsonism in association with other neurodegenerative diseases

- Dementia with Parkinsonism
- Parkinsonism with Amyotrophic Lateral Sclerosis
- Parkinsonism with Spinocerebellar degeneration
- Parkinsonism with Dementia

Atypical Parkinsonism

- Multi-system atrophy - Parkinsonism with postural hypotension – Shy Dragger Syndrome
- Progressive supranuclear palsy
- Corticobasal Ganglionic Degeneration
- With brain stem and cerebellar degeneration (Olivo-ponto-cerebellar syndrome)

Dementia

- Diffuse Lewy Body Disease with fluctuating confusion and Phenothiazine sensitivity.

Differential diagnoses

Multi-infarct dementia
Alzheimer's disease
Normal pressure hydrocephalus
Brain tumour
Benign essential tremor
Depression
Creutfeldt-Jakob disease

Complications

Recurrent falls with attendant complications e.g. fractures, subdural haematoma
Dementia
Depression

Investigations

Diagnosis is essentially clinical
Neuro-imaging: CT scan/MRI for exclusion of possible differentials

Management

Treatment objectives

Replace dopamine
Ensure mobility and avoidance of falls

Non drug treatment

- Physiotherapy for postural adjustment

Drug treatment

L-dopa/carbidopa (dose expressed as levodopa)

- 50 mg orally every 6 - 8 hours increased by 100 mg once or twice weekly depending on response

Anti-cholinergic drugs for tremors

- Trihexyphenidyl (benzhexol) 1 mg orally daily, increased gradually (usually 5 - 15 mg in 3 - 4 divided doses up to a maximum of 20 mg)

Dopamine receptor agonists

- Bromocriptine 1 - 1.25 mg orally nocte in the first week; 2 - 2.25 mg nocte in the 2 ndweek- 2 5 mg twice daily in the 3 week, 2.5 mg three times daily in the 4 week, increasing by 2.5 mg every 1 - 2 weeks according to response (usual range is 10 - 40 mg daily)
- Ropinirole 1 - 3 mg orally once daily (in resistant cases)

Supportive measures

Physiotherapy for postural adjustments

Antidepressants

- Amitryptiline for pain (which could be quite incapacitating) especially with dopamine-replacement drugs

Notable adverse drug reactions, caution and contraindications

Dopamine replacement drugs: dyskinesia, pain

- Advisable to start with small doses and gradually increase
- There is need for dosage and timing adjustments when side effects manifest

Dopa-agonists: postural hypotension; may cause vomiting

- Caution is advised to avoid falls

Anticholinergic drugs: constipation; memory problems

- Contraindicated in the presence of glaucoma

Prevention

Avoid identified causative agents where feasible

Timely and appropriate treatment to prevent/reduce complications

SEIZURES/EPILEPSIES

Introduction

A seizure results from abnormal excessive electrical discharge of brain cells

Epilepsy is a condition characterized by recurrent seizures unprovoked by any immediate identifiable cause

May be idiopathic or could follow:

- Cerebral infections
- Metabolic derangements (glucose, electrolytes, fluids)

- Stroke
- Tumours
- Head trauma
- Birth injury/asphyxia
- Drug abuse/overdosage/withdrawal
- Alcoholism
- Neuro-degeneration

Clinical features

Classical attack with sudden loss of consciousness, convulsions (tonic and/or clonic)
Tongue biting
Abnormal sensation or perception
Autonomic disturbances: epigastric discomfort, sphincteric incontinence
Semi-purposive actions (automatisms)
Aura
Loss of postural tone (sudden falls without convulsions)
Limb paralysis (Todd's paralysis) usually after attacks

Differential diagnoses

Migraine headache
Syncope
Narcolepsy
Panic attacks
Catatonic schizophrenia
Transient ischaemic attacks
Hysteria

Meniere's disease

Complications

Status epilepticus
Cardiac arrhythmias
Renal failure from myoglobinuria
Cerebral hypoxia/anoxia resulting in brain damage
Sudden death

Investigations

- Electroencephalography
- Neuro-imaging: CT scan,
- MRI
- Random blood glucose
- Urea,
- Electrolytes and Creatinine

Treatment objectives

- Stop convulsions/attacks
- Treat underlying cause if identified
- Improve quality of life

Drug treatment

Parenteral drugs are recommended for acute attacks/status epilepticus

Diazepam

Intravenous injection, 10 mg at a rate of 1 mL(5 mg) per minute, repeated once after 10 minutes, if necessary200-300 micrograms/kg **or** 1 mg per year of age or Under 12 years , 300

– 400 micrgrams/kg (max. 10 mg) (off label), repeated once after 10 minutes if necessary

Could be given per rectum as rectal solution in restless patients

- 500 micrograms/kg (up to a maximum of 30 mg) in adults and children over 10 kg

Phenytoin

Adult: initially 15 mg/kg by slow intravenous injection or infusion (with blood pressure and

Electrocardiograph monitoring) at a rate not more than 50 mg/minute; then 100 mg every 6-8 hours

Child: neonate- initial loading dose 20 mg/kg by slow intravenous injection, then 2 - 4 mg/kg orally every 12 hours, adjusted according to response (usual maximum dose 7.5 mg/kg every 12 hours)

1 month - 12 years: initially 1.5 - 2.5 mg/kg every 12 hours, adjusted according to response to 2.5 - 5mg/kg every 12 hours (usual maximum dose 7.5mg/kg every 12 hours or 300 mg daily)

12 - 18 years: initially 75 - 150 mg every 12 hours, adjusted according to response to 150 - 200 mg 12 hourly (usual maximum 300 mg every 12 hours) Paraldehyde (see important precaution below):

- Useful where facilities for rescucitation are poor

- Causes little respiratory depression when given rectally
- Administer 10 - 20 mL per rectum as an enema Child neonate- 0.4 mL/kg (maximum 0.5 mL) as a single dose; up to 3 months: 0.5 mL; 3 - 6 months: 1 mL; 6 - 12 months: 1.5 mL; 1 - 2 years 2 mL; 3 - 5 years 3 - 4 mL; 6 -12 years 5 - 6 mL (administered as a single dose per rectum) per kg body weight
- Not recommended in pregnancy

Cerebral decompression with marnitol 20% infusion or furosemide if indicated (see meningitis)

Give 50 mls of 50% Glucose to supply brain cells with calories

Give Thiamine in case of alcohol withdrawal seizures

Maintenance therapy in day-to-day care

Generalized epilepsies

Phenobarbital

Adult: 60 - 180 mg orally daily

Child: 5-8 mg orally daily

Phenytoin

Adult: 150 - 300 mg orally daily

Child: neonate- initial loading dose by slow intravenous injection then 2 - 4 mg/kg by mouth every 12 hours adjusted according to response (usual maximum 7.5 mg/kg every

12 hours)
1 month - 12 years: 1.5 - 2.5 mg/kg orally every 12 hours (usual maximum 7.5 mg/kg every 12 hours or 300 mg daily)
12 - 18 years: initially 75 - 150 mg every 12 hours, adjusted according to response up to 150 - 200 mg every 12 hours (usual maximum 300 mg every 12 hours)

Sodium valproate

Adult: 600mg daily in 2 divided doses

Child: neonate, initially 20 mg/kg orally or per rectum once daily; usual maintenance dose 10 mg/kg twice daily
1 month - 12 years: initially 5-7.5 mg/kg every 12 hours; maintenance 12.5 - 15 mg/kg every 12 hours 12 - 18 years: usually 300 mg every 12 hours, increased in steps of 200 mg at 3-day intervals; usual maintenance 500 mg - 1 g twice daily (maximum 1.25 g twice daily)

Partial seizures

Carbamazepine

Adult: 100 - 200 mg orally 1-2 times daily

- Not recommended in pregnancy

Child 1 month - 12 years: initially 5 mg/kg orally at night or 2.5 mg/kg twice daily, increased as necessary by 2.5 - 5 mg/kg every 3 - 7 days; usual maintenance 5 mg/kg every 8 - 12 hours 12 - 18 years: initially 100 - 200 mg 1 - 2 times daily, increased slowly to usual maintenance of 400-600 mg every 8 - 12 hours

Absence attacks

Ethosuximide

Adult: 500 mg daily initially,in two divided doses; increase by 250 mg at intervals of 5-7 days to doses of 1 - 1.5 g dailyin two divided doses (maximum dose 2 g daily)

Child: over 6 years: same as adult dose1 month to 6 years:initially 10 mg/kg(max 250 mg daily); in 2 divided doses increased every 5 – 7 days to usual dose of 20 -40 mg/kg (max. 1 g daily in 2 - 3 divided doses

Sodium Valproate

Adult: Initially 600 mg daily in 1-2 divided doses, increased gradually (in steps of 150-300 mg) every 3 days; usual maintenance dose 1-2 g daily(20-30 mg /kg daily), max. 2.5 g daily

Child: 1 month -12 years, initially 10 -15 mg/kg (max. 600mg)daily in 1-2 divided doses, usual maintenance dose 25-30 mg/kg daily in 2 divided doses

Initiation of valproate treatment by intravenous administration, Adult and child over 12 years, initially 10 mg/kg (usually 400-800 mg) by intravenous injection (over 3-5 minutes) followed by intravenous infusion or intravenous injection (over 3-5 minutes) in 2-4 divided doses or by continuous intravenous

infusion up to max. 2.5 g daily; usual range 1 - 2 g daily (20-30 mg/kg daily);
Child 1 month -12 years, 10mg /kg by intravenous injection(over 3-5 minutes) in 2-4 divided doses or by continuous intravenous infusion up to usual range 20-40 mg/kg daily(doses above 40 mg/kg daily monitor clinical chemistry and haematological parameters)
Continuation of valproate treatment by intravenous injection (over 3-5 minutes) or intravenous infusion in 2-4 divided doses, or by continuous intravenous infusion, same as established oral daily dose.
Other drugs:
Clonazepam for Myoclonic seizures
Primidone which is metabolized to Phenobarbitone

Non-drug treatment
Psychotherapy
Health education to patients, relations and public
Discourage harmful cultural practices e.g. burning, mutilation

Notable adverse drug reactions, caution and contraindications
Antiepileptics: foetal damage if used in pregnancy
- Serial measurements of alpha-fetoprotein and ultrasound studies are

necessary with close monitoring by an obstetrician

Phenytoin: gingival hypertrophy; may not be the first choice in young children

Phenobarbital: sedation and mental dullness and may affect school performance in children

Most antiepileptics: skin rashes, especially Stevens-Johnson syndrome; exfoliative dermatitis

Introduce drugs singly because of possible interaction between drugs

Doses must be gradually increased to avoid toxicity and other side effects

Do not use paraldehyde if it has a brownish colour or the odour of acetic acid

All antiepileptics must be withdrawn slowly so as not to precipitate status epilepticus

Prevention

Prompt treatment of fever in children to avoid febrile convulsions

Prevention of head injuries mainly from automobile accidents

Treat diseases of the brain early to avoid poor healing and death of brain cells

Immunization of children against communicable diseases

Address causative factors (see above)

Avoid driving and swimming unattended, and operation of machinery

STROKE

Introduction

Synonyms: Brain attack, Cerebrovascular disease, Apoplexy

It is a condition resulting from disruption of blood supply to brain cells (either occlusion (infarction) or blood vessel rupture (haemorrhage).

There should be pathological and/or radiological demonstration of the lesion.

The disability may result in death.

The duration of disability is no longer critical and acute stroke is now referred to as "Brain Attack" because the longer brain cells are deprived of blood, the bigger the size of brain cells affected and the more severe the damage or disability.

Clinical features

Classical stroke:

- Sudden motor weakness of one side of the body but could be bilateral, with/without speech, visual and sensory impairment

Subarachnoid haemorrhage:

- Severe headache, neck stiffness and positive Kernig's sign

Stroke-in-evolution:

- Gradual onset of deficit with progression

Mass lesion:

- Sudden rise in intracranial pressure
- Loss of consciousness, respiratory changes, pupillary changes
- Sudden death

Lacunar syndrome:

- Incomplete deficits: speech defects with clumsy hand involvement
- Pure motor and/or pure sensory deficits

Dementia:

- Arises from small, recurrent strokes resulting in cognitive impairment and functional dependence

Differential diagnoses

- Brain tumour

Subdural haematoma

- Brain abscess
- Meningitis/encephalitis
- Cerebral malaria
- Migraine headache
- Multiple sclerosis
- Metabolic derangements e.g. hypoglycaemia, hyperosmolar non-ketotic coma

Complications

Short and Long term

- Tentorial herniation with coning and death
- Cardiac arrhythmias
- Depression
- Epilepsy

- Dementia
- Parkinsonism
- Hyperglycaemia
- Sepsis
- Pneumonia
- UTI
- Decubitus ulcers
- Deep vein thrombosis
- Contractures

Investigations

Neuro-imaging with CT scan/MRI to determine stroke type and choice of management

Lumbar puncture for CSF analysis in suspected subarachnoid haemorrhage

Electrocardiography

Echocardiography

Carotid Doppler ultrasound study

Cerebral angiography

Full Blood Count with differentials

Random blood glucose

Urea, Electrolytes and Creatinine

Chest radiograph

HIV screening

Management

Treatment objectives

Restore cerebral circulation

Limit disability

Treat identified risk/predisposing factors

Reduce raised intracranial pressure
Treat complications (if any)

Non-drug treatment
Attention to calories, fluid balance
Physiotherapy for passive muscle exercises
Nursing care (frequent turning and bladder care) to prevent decubitus ulcers and urinary tract infection
Rehabilitation

Drug treatment
Cerebral decompression if there is evidence of raised intracranial pressure
- -20% mannitol 250 mL repeated every 12 hours for 4-6 doses

And/OR
- Furosemide 40 mg every 8 hours by slow intravenous injection for 6 doses

And/Or:
Thrombolysis with tissue plasminogen activator (Atelpase) - if patient is brought to medical attention within 4 1/2 hours and CT scan did not show haemorrhage or big infarct, no previous bleeding and BP not severely elevated

Treat underlying conditions such as diabetes mellitus, hypertension, and thrombosis
- Treat hyperglycaemia with Insulin

- Treat seizures with Intravenous Diazepam
- Treat fever with antipyretics and antibiotics if infection is suspected
- For subarachnoid haemorrhage: Nimodipime is recommended for the
- control of BP and to prevent vasospasm

Use of antacids to prevent and treat stress ulcers

Use of anti-epileptic drugs to prevent seizures in the acute phase

Notable adverse drug reactions, caution

Rebound cerebral oedema when mannitol is discontinued

Thrombolytic agents: bleeding tendencies

Diazepam by the intravenous route must be administered slowly to avoid respiratory depression and laryngeal spasm

Prevention

Treat/control known risk factors

-Hypertension
-Diabetes mellitus
-Cardiac diseases
-Hyperlipidaemia
-Obesity
-Smoking
-Excessive alcohol consumption

Give low dose aspirin (acetylsalicylic acid) to patients at risk if tolerated

SYNCOPE

Synonym: Fainting

Introduction

Loss of consciousness and postural tone as a result of diminished cerebral blood flow

May be due to:

Vaso-vagal attack or Cardiac causes.

It may result from

Prolonged standing

Severe emotional disturbance

Site of blood (e.g. witnessed by medical student for the first time in theatre)

The more severe form is associated with various heart diseases:

Arrhythmias (especially complete heart block)

Hypertrophic cardiomyopathy

'Heart attack' (mycardial infarction)

Atrial myxoma

Aortic stenosis

Dissecting aneurysm other causes:

Pulmonary embolism

Vertebro-basilar insufficiency

Subclavian steal syndrome

Carotid sinus pressure

Migraine headache

Clinical features

Sudden fall to the ground with loss of consciousness

Cold extremities
Bluish discolouration of extremities (cyanosis)
Pulse irregularities (or pulselessness)
Hypotension (or unrecordable blood pressure)
Fainting induced by pressure on the neck
Fainting induced by coughing, micturition

Differential diagnoses
Epilepsy
Myocardial infarction
Stroke
Aortic dissection
Hysteria

Complications
Cerebral hypoxia/anoxia resulting in brain damage
Stroke
Sudden death

Investigations
Electrocardiography
Echocardiography
Neuro-imaging: CT scan, MRI, carotid Doppler
Random blood sugar

Management
Depends on the cause(s)

Treatment objectives

Restore circulation and ensure brain perfusion by elevating the legs to increase venous return Identify cause and treat accordingly

Prevent recurrence

Non-drug treatment

Physiotherapy: pressure stockings

Drug treatment

Specific treatment for cardiac arrhythmias: refer to cardiologist

If hypotensive, give pressor agents

Notable adverse drug reactions, caution

Aspirin and other NSAIDs: use with caution in patients with history of dyspepsia, and in asthmatics

Prevention

Avoid prolonged standing

Treat underlying cardiac disease

Avoid dehydration or excessive fluid loss

Ensure adequate calorie and avoid prolonged fasting

Use medication as prescribed by physicians and avoid overdosage to compensate for missed doses

THE UNCONSCIOUS PATIENT

Introduction

An unresponsive patient who may also have breathing and circulatory problems

May be neurological or may result from other systemic diseases

An easy way of finding the cause is to think in terms of the vowels:

A Apoplexy (stroke)

E Epilepsy

I Infections e.g. meningo-encephalitis

O: Overdosing with drugs, alcohol intoxication, toxins

U Uraemia and other metabolic disorders

Other causes include: Head injury, Brain tumours (with complications)

Clinical features

Varying levels of impaired consciousness: Comatose: no response to stimulus, however painful Semi-comatose: some response to pain

Stuporose: a state deeper than sleep; vigorous stimulation required to stimulate response

Other features:

Cessation of respiration or abnormal ventilatory patterns: Cheyne-Stokes, ataxic, apneustic, gasping etc

Unresponsiveness or variable response to

painful stimuli

Features of the underlying cause(s)

- Stroke: may present with hemiparesis, facial asymmetry, crossed-eye defects, speech defects etc
- Epilepsy: frothing or tongue biting; abrasions of the extremities; positive past history
- Infections: may present with fever, neck stiffness
- Drug overdosage/toxins: pin-point pupils; respiratory problems; suggestive history
- Uraemia: characteristic fetor; skin rashes; oedema; severe dehydration
- Head trauma: haematomas; subconjuctival haemorrhages
- Bleeding from orifices (if coma is due to trauma or bleeding diathesis)

Features of raised intracranial pressure:

- Slow pulse (Cushing's reflex)
- Rising blood pressure
- Papilloedema

Differential diagnoses

Stroke
Post-epilepsy state
Syncope
Mycardial infarction
Hysteria
Substance abuse

Complication

Cerebral hypoxia/anoxia resulting in brain damage

Investigations

Neuro-imaging: CT scan, MRI
Random blood glucose
Urea, Electrolytes and Creatinine
Electroencephalography
Cerebrospinal fluid analysis
Drug levels/toxicology screen
Full Blood Count
Blood culture

Treatment objectives

Clear airway and restore breathing
Nurse in left lateral position and clear secretions by suction
Maintain circulation
Eliminate the cause
Prevent complications:

- decubitus ulcers
- atelectasis
- contractures etc.

Correct metabolic derangements
Admit in intensive care unit if facility available and connect to a ventilator with monitor

Non-drug treatment

Physiotherapy to prevent contractures/deep

vein thrombosis, and for passive muscle exercises Nursing care (frequent turning and bladder care) to prevent decubitus ulcers and infections

Drug treatment

Infections: appropriate antibacterial agent

Epilepsy: use effective parenteral anticonvulsant drugs; diazepam (see Epilepsy)

Renal failure: dialysis

Appropriate treatment of other metabolic causes

Supportive measures

Subcutaneous Low Molecular Weight heparin (LMWH) to prevent deep vein thrombosis (see Pulmonary Embolism)

Notable adverse drug reactions

Diazepam, if required, should be administered slowly intravenously to avoid respiratory depression

Prevention

Accessible, efficient and effective health care service delivery

Early reporting/detection of ill health

Adherence to medications and non-drug measures in managing disease states

Public Health Education

Promote awareness on avoidance of risk factors

CHAPTER 5:

PSYCHIATRIC DISORDERS

ALCOHOLISM (Alcohol dependence)

Introduction

A disorder characterized by a wide spectrum of problems. Central feature is the use of alcohol, which takes an increasingly dominant place in the user's life in spite of experience of harm related to drinking

Social and genetic factors are thought to be important in pathogenesis

A life time prevalence of about 0.2 - 0.5% in Nigerian adult males

Clinical features

Tolerance

Withdrawal episodes

Compulsive desire to use alcohol

Associated physical, social, or occupational impairments

Differential diagnoses

Dependence on (and withdrawal from) other substances

Complications

Liver cirrhosis

Damage to other organs (including the brain)

Accidents

Delirium tremens

Increased mortality (reduce life expectancy)

Family, social and occupational disability

Investigations

Full Blood Count and differentials Liver function tests

Other investigations as indicated for medical/physical complications

Treatment objectives

Reduction in alcohol consumption as an interim measure

Abstinence as the desired goal

Rehabilitation

Prevention of relapse

Non-drug treatment

Psychosocial interventions.

Cognitive behavioural therapy

Marital and family therapy Group therapy

Drug treatment

Only occasionally required, and following careful assessment

Note

- Detoxification is required for severe

withdrawal syndrome or delirium tremens
- This will involve the administration of a long-acting benzodiazepine and thiamine supplements over 7 - 10 days

Supportive measures

Rehabilitation to
- Sustain abstinence
- Acquire an alcohol-free life style
- Prevent relapse

Prevention

Health education (including school health education, peer group education and self help group e.g. alcoholic anonymous)
Government regulation of alcohol use

ANXIETY DISORDER

Introduction

Generalized anxiety disorder (GAD) is characterized by exaggerated worry and tension, even when there is little or no cause for anxiety
A chronic disorder affecting about 2 - 3% of the population

Clinical features

Pre-occupations: often of diverse nature
Poor concentration
Muscle aches and headaches
Irritability

Sweating
Fatigue
Insomnia
Shortness of breath

Differential diagnoses
Medical causes of suggestive symptoms and signs (e.g. hyperthyroidsm)

Complications
Chronicity
Co-morbid depression
Medical morbidity (e.g. hypertension)

Investigations
To exclude medical/ physical cause(s)

Treatment objectives
Achieve remission of symptoms Prevent relapse

Non-drug treatment
Cognitive-behavioural therapy

Drug treatment
Diazepam 10 - 20 mg orally daily

Or:
Fluoxetine 20 - 60 mg orally daily

Supportive measures
Relaxation techniques

Exercise
Psychotherapy

Notable adverse drug reaction, caution
The risk of dependence (and withdrawal syndromes) limits the utility of benzodiazepines for treatments of long duration

Prevention
Avoid of undue and extreme stress
Avoid psycho-active substances

BIPOLAR DISORDERS

Introduction

A type of mood disorder in which there is (typically) alternation of a depressive phase and a manic or hypomanic phase

Experienced by about 1% of the adult population at some point in their lifetime

About equal incidence between males and females

May be precipitated by psychosocial stress; strong genetic vulnerability often present

Clinical features

Depressive phase:

- Low mood
- Impaired appetite and sleep
- Ideas of worthlessness or hopelessness
- Suicidal ideation

- Other depressive symptoms and signs

Manic or hypomanic phase:

- Elation
- Euphoria
- Irritability
- Expansive mood
- Disturbed sleep
- Grandiosity
- Disinhibition

Differential diagnoses

Schizo-affective disorder Schizophrenia

Organic mood/affective disorder (including effects of drug abuse)

Complications

Social and personal consequences of inappropriate behaviour (e.g. unplanned pregnancy, sexually-transmitted infections, etc)

Suicide

Increased risk of morbidity (reduce life expectancy) (e.g. trauma and accidents)

Increased mortality

Investigations

Investigations as indicated to rule out organic/medical causes

Full Blood Count and renal function tests (to determine suitability of mood stabilizers)

Treatment objectives

Reduce risk to self and others
Normalize mood
Return to full functional status
Prevent recurrence

Non-drug treatment

Cognitive-behavioural therapy as sole treatment in mild cases, and adjunct in all others Electroconvulsive therapy (ECT)

- An effective and essentially safe treatment for severe and acute presentations
- A course of 8-12 treatments are usually needed

Drug treatment

Treat underlying causes

Lithium

- 1stline drug following established diagnosis

Adult :initially 1 - 1.5 g daily Prophylaxis: initially 300 - 400 mg daily

Child: not recommended

- Measure serum lithium concentration regularly (every three months on established regimens)
- Adjust dosage to achieve serum levels of 0.6 - 1.2 mEq/L

Sodium valproate

Adult: 750 mg - 2 g mg orally/day

Child: neonate, initially 20 mg/kg orally once

daily; usual maintenance dose 10 mg/kg every 12 hours daily 1 month - 12 years: initially 5 - 7.5 mg/kg every 12 hours, usual maintenance dose 12.5 -15 mg/kg every 12 hours (up to 30 mg/kg twice daily)
12 - 18 years: initially 300 mg every 12 hours, increased in steps of 200 mg daily at 3-day intervals; usual maintenance dose 0.5 - 1 g twice daily (maximum 1.5 g daily)

Carbamazepine

Adult: 600 - 1,800 mg orally daily

Child: 1 month - 12 years: initially 5 mg/kg orally at night or 2.5 mg/kg twice daily, increased as necessary by 2.5 - 5 mg/kg every 3 - 7 days

- Maintenance dose 5 mg/kg 2 - 3 times daily, increased slowly to usual maintenance of 400 - 600mg 2 - 3 times daily

Antidepressants

- TCAs or SSRIs may be indicated in depressive phase

Antipsychotics

- Haloperidol 1.5 to 3 mg orally 2 - 3 times daily (may be indicated in acute manic phase)

Child 2 - 12 years: initially 12.5 - 25 micrograms/kg orally twice daily, adjusted according to response to maximum 10 mg daily

12 - 18 years: initially 0.5 - 3 mg daily, adjusted according to response to lowest effective maintenance dose (as low as 5 -10 mg daily)

Supportive measures

Psychotherapy and social intervention for patient and relatives/caregivers

Notable adverse drug reactions

- More likely with doses above recommended upper limits

Lithium

- Gastrointestinal disturbances
- Tremors
- Confusion
- Myoclonic twitches

Carbamazepine:
hypersensitivity reactions
Transient memory impairment is common following ECT

Prevention

No primary preventive measures are clearly delineated

Adherence to therapy with mood stabilizers until discontinuation is considered prudent (this is individually determined)

DELIRIUM

Introduction

A transient disorder of brain function

Manifests as a global cognitive impairment and behavioural disturbance

More common at the extremes of life though it can occur at any age

Incidence up to 15% has been reported among general medical inpatients; up to 40% among acutely ill geriatric patients

Poor detection and mis-diagnosis are common

The most common causes are:

Trauma

Infections

Metabolic derangements

Side effects of drugs

Clinical features

Disturbance of consciousness

Disorientation

Memory deficits

Language disturbances

Perceptual disturbances

Rapid fluctuations

Disruption of sleep-wake cycle

Psychomotor hyperactivity

Mood alterations

Differential diagnoses

Dementia

Acute (idiopathic) psychotic disorders

Complications

Usually transient but may be associated with increased morbidity (e.g. from falls) and mortality

Investigations
Determined by any causal or contributing medical conditions

Treatment objectives
Identify and ameliorate any causal or contributing medical conditions
Improve cognition
Normalize behaviour

Non-drug treatment
Nurse in a quiet, well-lit environment
Support physical care, including food and fluid intake
Provide orienting cues
Physical restraint judiciously used when indicated

Drug treatment
High-potency antipsychotics in low dosages for sedation

- Haloperidol

Adult: 0.5 - 1 mg orally or parenterally *every 6 – 8 hours*
Child 2 - 12 years: initially 12.5 - 25 micrograms/kg orally twice daily, adjusted according to response to maximum 10 mg

daily; 12 - 18 years: initially 0.5 - 3 mg daily, adjusted according to response to lowest effective maintenance dose (as low as 5 -10 mg daily)

Benzodiazepines

- For severe agitation (i.e. life-threatening features) or patient seriously disrupting management

Supportive measures

Give reassurance to patient and relatives/caregivers

- The transient nature of condition
- No risk of "madness"

Caution

Close nursing care is required to prevent injuries and falls

Avoid over-medication, especially as antipsychotics and sedatives used may worsen delirium

Prevention

Early treatment of infective and metabolic conditions

Care with the use of drugs (especially anticholinergic medications) in the elderly

DEPRESSION

Introduction

A disorder of mood and affect in which the predominant emotion is sadness/

unhappiness
Can occur alone (unipolar depression) or as part of an alternation disorder in which elevation of mood also occurs (bipolar disorder)
Varies in severity from mild to severe
Life events, especially those involving loss, are often (but not always) the triggers
Strong genetic is vulnerability sometimes present
Occurs in about 2 - 5% of the population at any given time and in about 10 - 25% in their lifetime
Women are generally at an elevated risk

Clinical features
Sadness, unhappiness, feeling low
Loss of interest in usual activities
Reduced energy
Disturbance of sleep and appetite
Impaired concentration
Ideas of worthlessness, guilt, or failure
Morbid or suicidal rumination or ideation
Somatic complaints of various types

Differential diagnoses
Normal grief reaction
Medical conditions causing lowering of mental and physical activities (e.g. anaemia, hypothyroidism)
Infections (e.g. viral)

Complications
Worsening of co-morbid physical illness
Suicide
Recurrence (in 50% or more)

Investigations
Full Blood Count and differentials
Thyroid function test
Indicative infection screen

Treatment objectives
Normalize mood
Prevent suicide attempts
Return to active life
Prevent recurrence

Non-drug treatment
Cognitive-behavioural treatment
Inter-personal psychotherapy
Drug treatment
Tricyclic antidepressants (TCAs)
- Amitriptyline in increasing doses up to 150 mg orally / day
- Fluoxetine 20 - 80 mg orally / day

Supportive measures
Supportive psychotherapy for patients and family / caregivers

Notable adverse drug reactions, caution

Tricyclic antidepressants:

- Dryness of the mouth
- Urinary retention
- Constipation
- Blurring of vision

Selective Serotonin Reuptake Inhibitors (SSRIs)

- Sleep disturbance
- Sexual dysfunction
- Serotonin syndrome

Cardiac toxicity, especially in overdose with TCAs and SSRIs

Increased suicidal ideation in adolescents

- Should be used with caution in patients with epilepsy, history of mania, cardiac disease, diabetes mellitus, and bleeding disorders
- Caution is also required in patients receiving concurrent electroconvulsive therapy (reports of prolonged seizures with fluoxetine)

Prevention

Recurrence is reduced by continuing medication for at least 6 months after acute symptoms resolve

INSOMNIA

Introduction

Difficulty in falling asleep or staying asleep

May be primary and unrelated to any

physical or mental disorder
May relate to a mental disorder, medical or physical conditions
May be an adverse effect of medication (or psychoactive substances)
A common, often chronic problem; tends to increase with age

Clinical features
Early insomnia: difficulty in initiating sleep
Middle insomnia: difficulty in going back to sleep after waking up at night
Terminal insomnia: early awakening, commonly 2 hours or more before desiring to do so

Differential diagnoses
Useful to consider possible aetiological factors: medical, mental, situational, environmental
Pain is a common factor

Complications
Deteriorating physical and/or mental health
Decline in overall well being and quality of life

Investigations
Mainly of the presumed underlying cause(s)

Treatment objectives

To improve sleep, especially sleep satisfaction
To remove underlying/associated factors

Non-drug treatment
Sleep hygiene
Behavioural modifications to enhance relaxation
Avoid habits and lifestyles that promote insomnia
Improve environmental/sleeping conditions

Drug treatment
General principles
Treat underlying cause(s)
Avoid sedatives: use for only short periods when indicated
Short-acting benzodiazepines
- For early insomnia

Or:
Longer-acting benzodiazepines e.g.
- Diazepam at low doses: 2.5 - 10 mg for no more than 2-3 weeks
- For middle insomnia

Supportive measures
Relaxation therapy: a useful adjunct for the most common forms of insomnia

Notable adverse drug reactions

Benzodiazepines: dependence and rebound insomnia

Prevention

Reduced stress exposure

Caution with alcohol and psychoactive substances, such as coffee, kolanut

Discourage misuse of "sleeping pills" e.g. Bromazepam, diazepam

PANIC DISORDER

Introduction

A disorder characterized by episodic attacks of extreme fear, mostly unrelated to specific objects or situations

Associated with multiple somatic and cognitive symptoms

Each attack lasts for about 5 - 30 minutes

Often begins abruptly

Affects about 0.5 - 1.0% of the population

Clinical features

A feeling of choking

Pounding heart

Chest pressure or pain

Dizziness

Shortness of breadth

Trembling

Sweating

Tingling or numbness in the hands or feet Hot flushes

Differential diagnoses

Other causes of intense fear (phobias, obsessive-compulsive disorders, etc.)

Medical causes (e.g. hyperthyroid states, episodic hypoglycemia, etc)

Seizure disorders

Complications

Phobia

Depression

Suicide

Investigations

As indicated to exclude medical aetiologies

Treatment objectives

To reduce intensity and frequency of attacks

To reduce anticipatory anxiety

Non-drug treatment

Cognitive-behavioural treatment

Drug treatment

Fluoxetine

Adult: initially 20 mg orally once daily, increased after two weeks (if necessary) to 20 - 60 mg once daily (maximum 80 mg)

Elderly: 20 - 40 mg (maximum 60 mg for elderly) once daily

- Discontinue if no improvement within 10

weeks

Child and adolescent under 18 years: not recommended

Or:

Amitryptiline 50 - 150 mg orally / day

Supportive measures

Psychotherapy Relaxation techniques

Notable adverse drug reactions

Tricyclic antidepressants are cardiotoxic in overdose

Increased risk of suicidal attempts by patients with panic disorder

Prevention

No specific primary prevention measures

SCHIZOPHRENIA

Introduction

A serious psychotic disorder characterized by multiple impairments in emotional, behavioural, cognitive, social, and occupational domains (among others)

Affects about 1% of the population

Onset usually in late adolescence or early adulthood Strong genetic component to its etiology; environmental factors, including pre-natal and obstetric factors, also implicated

Clinical features

Disorders of:
Thought
Perception
Speech
Cognition
Behaviour
Motor function

Differential diagnoses
Psychosis of other origin (including those due to organic factors)
Affective psychosis
Epilepsy, especially of temporal lobe origin
Drug effect, e.g. amphetamine intoxication

Complications
Chronicity
Suicide
Increased physical morbidity
Increased mortality

Investigations
To exclude organic causes of acute psychotic presentations

Treatment objectives
Relieve acute symptoms
Return to full functional status
Rehabilitate
Prevent relapse

Non-drug treatment

Psycho-social interventions as indicated (including social and occupational therapy)

Psycho-education for patient and relatives / caregivers

Supportive psychotherapy

ECT (especially for catatonic forms)

Drug treatment

Chlorpromazine

Adult: initially 25 mg orally every 8 hours (or 75 mg at night), adjusted according to response to usual maintenance dose of 75 - 300 mg daily

- Elderly: a third to half adult doses

By deep intramuscular injection: 25 - 50 mg every 6 - 8 hours

Child: 1 - 5 years: 500 micrograms/kg orally every 6 - 8 hourly (maximum 40 mg daily); 6 - 12 years: a third to half adult dose (maximum 75 mg daily)

Haloperidol

Adult: initially 1.5 - 3 mg every 8 - 12 hours daily or 3 -5mg every 8 - 12 hours in severely affected or resistant patients

- In resistant schizophrenia, up to 30 mg daily may be needed, adjusted according to response to the lowest effective maintenance dose (as low as 5 - 10 mg daily)

Elderly, initially half adult dose

Child: initially 25 - 50 mg micrograms/kg daily in 2 divided doses (maximum 10 mg)

Fluphenazine

Adult: initially 2 - 10 mg every 8 - 12 hours, adjusted according to response to 20 mg daily

- Doses above 20 mg daily (10 mg in elderly) only with special precaution

Or:

25 - 100 mg intramuscularly fortnightly to monthly

Child: not recommended

Supportive measures

Supportive psychotherapy

Social and occupational therapy

Cognitive therapy (as adjunct in the treatment of persisting psychotic experience)

Rehabilitation

Notable adverse drug reactions

Extrapyramidal and Parkinsonian symptoms (may require anticholinergic medication)

Tardive dyskinesia

Weight gain

Agranulocytosis (monitor blood counts in patients on clozapine)

Prevention

No clear/specific scope for primary prevention at present

Secondary and tertiary:

- Early and effective treatment

Rehabilitation to reduce disability

CHAPTER 6:

DENTAL AND ORAL DISORDERS

ACUTE NECROTIZING ULCERATIVE GINGIVITIS

Definition

A polymicrobial, endogenous infection

Aetiology

Fusiform and spirochaete bacteria

Epidemiology

In developing countries, seen almost exclusively in children

Related to poverty and malnutrition (predisposing factors)

In industrialized countries, most common in young adults with neglected mouths; smoking and stress have been associated

Clinical features

Crater ulcers striating at the tips of the interdental papillae

Ulcers spread along gingival margins

Gingival soreness and bleeding

Foul breath

Metallic taste
Increased salivation
Cervical lymphadenopathy and fever in advanced cases

Differential diagnoses
Primary herpetic gingivo-stomatitis
HIV-associated acute ulcerative gingivitis
Gingival ulceration in acute leukaemia or aplastic anaemia

Investigations
Smears from ulcers show predominantly spirochaetes and gram-negative fusiform bacteria

Treatment objectives
Treat infection
Restore oral health

Non-drug treatment
Oral hygiene (debridement) is essential

Drug treatment
Metronidazole
Adult: 200 mg orally 8 hourly for 3 days
Child: 1-3years: 50mg orally every 8 hours for 3 days; 3-7years: 100mg every12 hours; 7 - 10 years: half adult dose

Supportive therapy

Ascorbic acid

Adult: not less than 250 mg orally daily (in divided doses)

Child: 1 month - 4 years: 125 - 250 mg in 1 - 2 divided doses

4-12 years: 250-500 mg daily in 1-divided doses; 12 - 18 years 500 mg - 1 g daily in 1 - 2 divided doses

Ferrous sulfate

Adult: 200 mg orally three times daily taken before food *Child* 6-12years: half adult dose

Follow-up treatment

Rehabilitation of the mouth

Once the acute phase has subsided, oral hygiene should be brought to as high a standard as possible to lessen the risk of recurrence

Sequestrectomy

Notable adverse drug reactions, caution

Metronidazole: nausea, vomiting, unpleasant taste; disulfiram-like effect with alcohol.

ACUTE PERIAPICAL ABSCESS

Definition

A localized collection of pus in the periapical region of a tooth contained within the alveolar bone

Aetiology

May develop either directly from acute periapical periodontitis or more usually from a chronic periapical granuloma

Generally the result of a mixed bacterial infection

Culture of the pus yields a wide range of different organisms

- Strict anaerobes (e.g. *prevotella, porphyromonas)* usually predominante, but facultative anaerobes may be found

Clinical features

Painful swelling at the root of tooth

Sinus (may be present)

Tooth is tender to biting or percussion

Tooth mobility

Differential diagnoses

Inflammatory radicular cyst

Osteomyelitis

Periodontal abscess

Investigations

Radiographs (periapical)

Treatment objectives

Relieve symptoms

Eliminate the infection

Non-drug treatment

Drain abscess using local anaesthesia

by root canal extirpation if tooth is to be retained
Otherwise extract the involved tooth
Treat residual infection

Drug treatment
Amoxicillin
Adult: 250 mg orally every 8 hours for 5 to 7 days *Child:* up to 10 years 125 mg every 8 hours, doubled in severe infections
Metronidazole
Adult: 200 mg orally every 8 hours for 3-7 days
Child: 1 - 3 years: 50 mg orally 8 hourly for 3-7 days; 3 - 7 years: 100 mg 8 hourly; 7 - 10 years: 100mg 8 hourly for 3-7 days
In Dentoalveolar abscess that has not responded to penicillins or metronidazole
Clindamycin
Adult: 150-300 mg every 6 hours; up to 450 mg every 6 hours in severe infections
Child: 1 month – 18 years 3-6 mg/kg(max. 450 mg) every 6 hours
Neonate
Under 14 days 3-6 mg/kg 3 times daily
14 -28 days 3-6 mg/kg(max.450mg) 4 times daily
By deep intramuscular injection or by intravenous infusion, 0.6-2.7 g daily (in 2-4 divided doses) life threatening infection, up to 4.8 g daily; single doses above 600 mg by intravenous infusion only; single doses by

intravenous infusion not to exceed 1.2 g;
Caution
Discontinue immediately if diarrhoea or colitis develops; monitor liver and renal function if treatment exceeds 10 days, and in neonates and infants; avoid rapid intravenous administration; avoid in acute porphyria

ACUTE PERIODONTAL DISEASES

Gingival abscess

Risk factors

Contributing systemic risk factors may affect progress, treatment and therapeutic outcomes for chronic periodontitis. These may include diabetes, smoking, certain periodontal bacteria, aging, gender, genetic predisposition, systemic diseases and conditions (immunosuppression), stress, nutrition, pregnancy, HIV infection, substance abuse, and medications. Elimination, alteration, or control of risk factors which may contribute to chronic periodontitis should be attempted. Consultation with the patient's physician may be indicated.

Treatment considerations include drainage to relieve the acute symptoms and mitigation of the etiology.

Periodontal abscess- Treatment considerations include establishing drainage

by debriding the pocket and removing plaque, calculus, and other irritants and/or incising the abscess. Other treatments may include irrigation of the pocket, limited occlusal adjustment, and administration of antimicrobials and management of patient comfort.

A surgical procedure for access for debridement may be considered. In some circumstances extraction of the tooth may be necessary. A comprehensive periodontal evaluation should follow resolution of the acute condition

Clinical features

These abscesses follow an inflammation of the gingiva and periodontium. As it progresses and following attachment loss gums become red, slightly swollen, with obvious pus accumulation and bleeds on slight provocation. The associated teeth show different degrees of mobility and mostly vital.

Non-drug treatment

Instruction, reinforcement, and evaluation of the patient's plaque control should be performed.

Supra- and subgingival scaling and root planning should be performed to remove microbial plaque and calcul

Antimicrobial agents or devices may be used

as adjuncts
Antiseptic mouthwashes e.g. chlorhexidinegluconate 2% three times daily for 1 - 2 weeks
Hexetidine mouthwashes to alternate with warm saline mouthwashes

Drug treatment
Analgesics
- Paracetamol

Adult: 1 g orally every 8 hours for 3 - 5 days
Child: 1 -5 years: 125-250 mg, 6- 12 years 250-500 mg orally every 8 hours
Antibiotics
- Amoxicillin

Adult: 250 mg orally every 8 hours for 5 days
Child: 1 month - 1 year 62.5 mg orally every 8 hours:
dose doubled ill severe infections
1 -5 years: 125 mg every 8 hours; 5- 12 years: 250 mg 8 hourly; 12-18 years 500 mg 8 hourly; all doses doubled in severe infections
- Metronidazole

Adult: 200 mg orally every 8 hours for 5 days
Child: 1 - 3 years 50 mg orally every 8 hours: 3-7 years:
100 mg every 12 hours; 7 - 10 years: 100 mg every 8 hours

ALVEOLAR/DRY SOCKET

Introduction

The most frequent painful complication of extractions

Caused by destruction of the clot that normally fills the socket

Predisposing factors

Excessive extraction trauma
Limited local blood supply
Local anaesthesia
Oral contraceptives
Osteosclerotic disease
Radiotherapy
Female gender

Clinical features

Pain delayed for few days up to a week after extraction
Deep seated, throbbing pain
Mucosa around socket is red and tender
No clot in socket - bare whitish alveolar bone exposed

Differential diagnosis

Osteomyelitis

Complication

Osteomyelitis

Treatment objective

Alleviate pain and suffering of the patient
Optimize condition for epitheliasation of the extraction socket

Keep open socket clean and protect exposed bone

Non-drug treatment

Irrigate with mild warm saline and antiseptic
Fill with an obtudant dressing containing some non-irritant antiseptic
Warm saline mouth rinse

Drug treatment

Local anaesthesia

- Lidocaine 2% (1in 80,000)
- Co-amoxiclav
- Severe dental infection with spreading cellulitis
- 250/125 mg orally every 8 hours for 5 days (dose/doubled in severe infections)

Chlorhexidene gluconate 2%

- 10 mL for mouth washes three times daily

Prevention

Minimal trauma during extractions
Immediately after extraction, squeeze socket edges firmly together and hold for a few minutes till clot has formed

Antibiotics if patients have had irradiation, or have Paget's disease

CELLULITIS

Definition

A rapidly spreading, poorly localized / diffuse erythematous inflammation of the soft tissues particularly associated with streptococcal infection to break down fibrin and ground substance, lyse cellular debris and enhance spread of infection.

Pathogenesis

Rapid spread is most likely related to release of large amounts of streptokinase and hyalurondinase which are produced by most strains of streptococci

The fascial space infections may involve sublingual, submandibular and/or parapharyngeal spaces

Ludwig's angina is bilateralbrawny cellulitis of the sublingual and submandibular spaces

Clinical features

Diffuse, tense, painful swelling of the involved soft tissues

Malaise

Elevated temperature

Ludwig's angina

This is a surgical emergency!

It causes airway obstruction, which can quickly result in asphyxia
Suppuration and abscess formation may occur later if treatment is neglected or delayed

Complications
Extension towards the eyes, and risk of cavernous sinus thrombosis: cellulitis affecting maxillary teeth Respiratory difficulty: cellulitis affecting mandibular teeth

Investigations
Culture (blood and swab) and sensitivity testing

Non-drug treatment
Drainage of the swelling to reduce pressure (oral drain may also be placed)
Secure the airway by tracheostomy if necessary

Drug treatment
Aggressive antibiotic treatment - Intravenous co-amoxiclav (given over 3 to 4 minutes) in combination with intramuscular gentamicin for 5 days
Injection co-amoxiclav
Adult: 1,000/200 mg intravenously every 8 hours

Child: neonate and premature infants, 25 mg/kg every 12 hours; infants up to 3 months, 25 mg/kg every 8 hours, 3 months to 12 years, 25 mg/kg every 8 hours increased to 25 mg/kg every 6 hours in more severe infections

Injection gentamicin:

Adult: 3-5mg/kg daily in divided doses every 8 hours

Child: up to 2 weeks: 3 mg/kg every 12 hours; 2 weeks -12 years: 2 mg/kg every 8 hours

Precaution

Gentamicin may cause significant ototoxic and nephrotoxic effects

Prevention

Early treatment of carious teeth

DENTAL CARIES

Definition

A progressive bacterial breakdown of teeth exposed to the carbohydrate and bacterial plaque

Classification

Enamel caries

Dentine caries

Root surface caries

Aetiology

Develops over time in the presence of certain interacting variables

- Carbohydrate diet
- Viridans streptococci bacteria
- Susceptible tooth surface

Pathogenesis

Enamel caries progress in the following stages:

- Incipient caries
- Early (sub-microscopic) lesion
- Phase of non-bacterial enamel crystal destruction
- Cavity formation
- Bacterial invasion of enamel

- *Clinical features*

Cavity formation in affected tooth

- Starts as a white spot Pain
- On exposure of the cavity to thermal changes or food particles

Complications

Pulpitis

- If not treated can cause apical periodontitis and dentoalveolar abscess

Investigations

Periapical radiographs
Bitewing radiographs
Electric pulp testers
Thermal test

Non-drug treatment

Depending on the stage of the lesion:

Excision of lesion followed by:

Amalgam filling, Glass Ionomer Cement (GIC) composite and Atraumatic Restorative Technique (ART) for enamel caries

Amalgam filling, GIC for dentine caries

Root Canal Therapy, pulp capping pulpotomy, pulpectomy for pulpal involvement

Drug treatment

Analgesics pre-operatively

- Paracetamol 1 g 4 - 6 hourly orally to a maximum of 4 g daily

Prevention

Oral health education

Regular scaling and polishing

Systemic and topical fluoride application

Fissure sealants

Routine dental check-ups

PLAQUE-INDUCEDGINGIVITIS

Introduction

An inflammatory response of the gingivae to plaque bacteria

The most common type is chronic marginal gingivitis

Clinical features

Chronic gingivitis is asymptomatic, low-grade inflammation of the gingivae
Gums become red slightly swollen and bleed on slight touch

Non-drug treatment

Oral hygiene instructions
Scaling and polishing
Antiseptic mouthwashes e.g. chlorhexidine gluconate 2% three times daily for 1 - 2 weeks
- Hexetidine mouthwashes to alternate with warm saline mouthwashes

Drug treatment

Analgesics
- Paracetamol

Adult: 1gorally every 8hours for 3-5days
Child: 1-5years: 125-250mg, 6-12years 250-500mg orally every 8 hours Antibiotics
- Amoxicillin

Adult: 250 mg orally every 8 hours for 5 days
Child: 1 month - 1 year 62.5 mg orally every 8 hours; dose doubled in severe infections 1-5 years: 125mgevery 8 hours; 5-12 years: 250mg 8 hourly; 12 - 18 years 500 mg 8 hourly; all doses doubled in severe infections
- Metronidazole

Adult: 200 mg orally every 8 hours for 5 days
Child: 1-3years 50mgorally every 8 hours for 3 days; 3-7years: 100 mg every 12 hours; 7 - 10

years: 100 mg every 8 hours

Notable adverse drug reactions, caution

Metronidazole: nausea, vomiting and metallic taste

Metronidazole is contraindicated in pregnancy

Avoid alcohol during treatment with metronidazole, and for at least 48 hours after treatment

Prevention

Oral health education

Scaling and polishing every six months

NEOPLASMS OF THE ORAL CAVITY refer to specialist care

ORAL THRUSH (Candidiasis)

Introduction

A clinical infection of mucous membranes due to the fungus species *Candida*

Candida albicans is the most frequently isolated strain

Classification

Acute oral candidosis

Chronic oral candidosis

Denture association candidosis/denture stomatitis

Pathogenesis/aetiology

Immunosupression results in the *Candida albicans* (a normal oral commensal) becoming virulent

- It invades and proliferates in superficial epithelium
- Results in a thick plaque which is oedematous and not easily rubbed off

Clinical features

A creamy/whitish, soft and friable slough located on the soft tissues of the oral cavity: tongue, palate, cheek, pharynx

May be asymptomatic, or painful, with difficulty in swallowing

Predisposing factors

Denture wearing
Reduced salivation (e.g. drug induced)
Antibiotic therapy (especially broad spectrum)
Poorly controlled diabetes mellitus
Steroid therapy (chronic)
Salivary gland damage (e.g. post radiation)
Malnutrition
HIV infection
Leukaemia
Iron, vitamin B, folic acid deficiency and Agranulocytosis

Investigations

Smear of the affected region and Gram

staining or PAS with or without potassium hydroxide to demonstrate hyphae
Swab sample for microscopy, culture and sensitivity
Biopsy and histopathologic examination
Identify predisposing factors (including immunosuppresion)
Define extent of involvement

Non-drug treatment
Manage any underlying predisposing factors
Replace worn dentures
Proper counselling of patients as to use of dentures
Diet modification and improvement
Chlorhexidine mouthwash three times daily for 1 – 2 weeks

Drug treatment
Topical anti-fungal medication e.g.
- Nystatin suspension

Adult: 100,000 units/mL 4 times daily, after food (usually for 7 days)
- Continue for 48 hours after lesions have resolved

Child 1 month - 18 years, prophylaxis and treatment: 100,000 units 6 hourly after food for 7 days
- Continue for 48 hours after lesions have healed Immunocompromised children:
- 500,000 units 6 hourly for 7 days

Or:

- Miconazole oral gel 2%

Adult: place 5 - 10 mL in the mouth after food and retain near lesions 4 times daily

Child under 2 years: 2.5 mL twice daily; 2 - 6 years: 5 mL twice daily; 6 - 12 years: 5 mL 4 times daily; 12 - 18 years: 5-10mL 4 times daily

- Leave in the mouth after food and retain near lesions

Some patients may require systemic antimicrobial medicines

- Fluconazole

Adult: 50 mg orally daily for 7 - 14 days

Child: 3-6mg/kg on the first day, then 3mg/kg daily For neonates up to 2 weeks old: administer every 72 hours; 2 - 4 weeks old: administer every 48 hours

PERICORONITIS

Introduction

An inflammatory condition of the operculum or gumflap around a partially erupted/impacted tooth

Common around the lower last molars or wisdom teeth Upper canine may also be affected

Classification

Subacute

Acute Chronic

Acute-on-chronic

Aetiology
Mixed microbial infection

Food impaction and plaque accumulation under gum flap
Trauma to gum flap from opposing tooth
Ulcerative gingivitis
Reduced resistance
Anaerobes in plaque

Clinical features
Soreness and tenderness around partially-erupted tooth
Pain
Swelling
Enlargement of regional lymph nodes
Fever
Abscess formation

Investigations
Radiographs
- To establish the position of the affected tooth and its relationship to the second molar
- May show impacted third molar

Non-drug treatment
When mouth opening is possible: careful irrigation under the gum flap to clear debris, using warm saline mouthwash

- To be done frequently until stagnation area is removed

Operculectomy

Disimpaction of the third molar by surgical extraction

Occlusal reduction of opposing tooth

Extraction of opposing tooth to forestall supraeruption and sequelae

Drug treatment

Appropriate antibiotics

Analgesics

Supportive therapy

Possible complications

Cellulitis

Ludwig's angina

Osteomyelitis

Submasseteric abscess

Temporomandibular joint ankylosis

PERIODONTITIS

Introduction

An inflammatory condition of the periodontium: periodontal ligament, cementum, alveolar bone, gingivae

Classification

Acute periodontitis

Chronic periodontitis

Juvenile periodontitis

Other sub-classifications

Acute periodontitis

Relatively uncommon Of short duration; may be due to trauma, abscess or ulceration

Characterized by pain - May be associated with bleeding, fever, swelling and redness of the mucosa, unpleasant taste in the mouth

Chronic periodontitis

This is inflammation within the supporting tissues of the teeth with bone loss

The most frequently occurring form of periodontitis

Characterized by pocket formation and/or recession of the gingiva

Prevalent in adults, but can occur at any age

Progression of attachment loss usually occurs slowly, but periods of rapid progression can occur

A sequala to untreated gingivitis

Clinical features

May be asymptomatic initially, with a low grade inflammation of the periodontium and gingiva

As it progresses, and following attachment, lost gums become red, slightly swollen and bleed on slight touch

Associated teeth show different degrees of mobility

Risk factors
Diabetes mellitus
Smoking
Certain periodontal bacteria
Aging
Gender
Genetic predisposition
Immunosuppression
Stress
Nutrition
Pregnancy
HIV infection
Substance abuse
Medications
Eliminate, alter, or control above risk factors which may contribute to chronic
Consultationwith the patient's physician may be indicated

Non-drug treatment
Instruction, reinforcement, and evaluation of the patient's plaque control should be performed
Supra- and sub-gingival scaling and root planning to remove microbial plaque and calculi

Drug treatment
Analgesics

- Paracetamol

Adult: 1 g orally every 8 hours for 3 - 5 days
Child: 1 -5 years: 125 - 250 mg; 6 - 12 years: 250 - 500 mg orally every 8 hours

Antibiotics

- Amoxicillin

Adult: 250 mg orally every 8 hours for 5 days
Child: 1 month - 1 year: 62.5 mg orally every 8 hours;
dose doubled in severe infections
1 -5 years: 125 mg every 8 hours; 5 - 12 years: 250 mg 8 hourly; 12 - 18 years 500 mg 8 hourly; all doses doubled in severe infections

- Metronidazole

Adult: 200 mg orally every 8 hours for 5 days
Child: 1 - 3 years 50 mg orally every 8 hours; 3 - 7 years:
100 mg every 12 hours; 7 - 10 years: 100 mg every 8 hours

Antiseptic mouthwashes

- 2% Chlorhexidine gluconate (alcohol free) Rinse mouth with 10 ml for about 1 minute twice daily for 1 - 2 weeks
- Hexetidine mouthwashes to alternate with warm saline mouthwashes

Notable adverse drug reactions, caution

Metronidazole: nausea, vomiting and metallic taste
Metronidazole is contraindicated in

pregnancy
Avoid alcohol during treatment with metronidazole, and for at least 48 hours after treatment

Prevention
Oral health education
Scaling and polishing every six months

Juvenile periodontitis

An uncommon disease characterized by periodontal destruction, often in the absence of overt gingival inflammation

Epidemiology

Prevalence 1:1000; male = female
Onset at puberty or earlier

Clinical features

Affects the first permanent molar and incisors

Actinobacillus, *Actinomycetes comitans* has been isolated from the affected sites

Results in drifting and loss of the first permanent molar and incisors

Investigation

Radiology may reveal marked bone loss interdentally, inter-radicularly and apically

Complications

Tooth loss

Malocclusion
Temporo-Mandibular Joint (TMJ) dysfunction syndrome

Non-drug treatment
Control of plaque bacteria by use of antiseptic solution
Establishing a healthy gingival and periodontal attachment
Oral hygiene instruction and motivation
Regular scaling and polishing
Root planning
Splinting of mobile tooth
Periodontal surgery
Bone regenerative techniques e.g. using Polytetrafluoroethylene (PTFE) membranes, Bio-Oss, Bio-membrane

Drug treatment
Metronidazole
Adult: 200 mg orally every 8 hours for 5 days
*Child 1 - 3 years:50*mg orally every 8 hours; 3 - 7 years: 100 mg every 12 hours; 7 - 10 years: 100 mg every 8 hours; 10 -18 years: 200 mg every 8 hours
Plus:
Tetracycline 250 mg orally daily for up to 21 days
Child under 12 years: metronidazole and amoxicillin (or erythromycin for those sensitive to penicillin)

Precaution

Tetracyclines should not be given to children under 12 years

PULPITIS

Introduction

Inflammation of the dental pulp

The single most important disease process affecting the dental pulp

Accounts for virtually all pulpal disease of any clinical significance

Clinical features

Pain which is difficult to localize

- May radiate to the adjacent jaw and occasionally to the face, ear or neck

May be triggered by:

- Cold or hot stimulants
- Arecumbentposition
- Occasionally by mastication when food particles get into a carious cavity

Important to determine whether pulpitis is reversible or irreversible

Reversible pulpitis:

The pulp can recover with removal of stimulus

Pain lasts for only a few moments after removal of the initiating stimulus

Irreversible pulpitis:

The pulp cannot recover even after removal of

stimulus
Characterized by pain which lingers for at least one minute after removal of stimulus
May be spontaneous

Complications
The sequelae of untreated pulpitis (in the order in which they occur) are:
Reversible pulpitis
Irreversible pulpitis
Pulpal necrosis
Apical periodontitis
Periapical abscess
Cellulitis

Investigations
Of primary importance is the use of a pulp tester to test the vitality of the pulp
The following can be used:
- Electric pulp tester
- Cold or hot water bath
- Ethyl chloride spray
- Hot gutta percha sticks
- Ice sticks

Treatment objectives
To exclude the pulp from the stimulus (or stimuli) in reversible pulpitis
To remove the pulp in irreversible pulpitis

Non-drug treatment

Reversible:
- Indirect pulp capping
- Direct pulp capping
- Conventional filling using amalgam, composite or GIC
- Desensitization with strontium chloride

Irreversible:
- Root canal therapy
- Extraction

Drug treatment

Paracetamol

Adult: 500 mg - 1 g orally every 4 - 6 hours (to a maximum of 4 g) for 5 - 7 days

Child: same as adult dosing 6 - 12 years: 250 - 500 mg; 1 - 5 years: 125 - 250 mg; 3 months - 1 year: 125 - 250 mg for 5 - 7days

NSAIDs may be required in some patients

Notable adverse drug reactions

Aspirin and other NSAIDs
- Gastrointestinal haemorrhage, allergic reactions
- Do not prescribe for patients with peptic ulcer disease
- May exacerbate symptoms in asthmatics

Aspirin is contraindicated in children less than 16 years as it may precipitate Reye's syndrome

Prevention

Prevent dental caries (the most important cause of pulpitis)

Seek prompt dental attention

SALIVARY GLAND DISEASES

Introduction

A wide spectrum of disorders

Diseases due to obstruction

Salivary calculi
Parotid papilla and duct strictures
Salivary fistulae
Mucoceles and cysts
Ranula

Sialadenitis

Acute infection and inflammation of the salivary glands

Types:

Parotitis (mumps – acute, non-suppurative)

The most common sialadenitis
Mainly affects children
Usually not related to sialolithiasis

Suppurative parotitis

Chronic sialadenitis

Sub-mandibular sialadenitis

Less common in children
Abundant salivary flow with rich mucus component
Rapid excretion

Sublingual sialadenitis

This is rare

- Mumps (acute non-suppurative)

Introduction

- Painful non-erythematous swelling of one or both parotid glands, occurring 1-2 weeks following exposure to the aetiologic virus

Epidemiology

- Most common in children aged 6 - 8 years with epidemics occurring during winter and spring, before the onset of routine vaccination against measles, mumps and rubella

Clinical features

- Preauricular swelling
- Preauricular pain
- Fever
- Chills
- Headache

Diagnosis

- Based on history and clinical findings
- Virological evaluations may be employed

Differential Diagnosis

- Bacterial infections
- Obstructive diseases

Complications

- Meningitis
- Pancreatitis
- Otitis
- Nephritis
- Orchitis
- Testicular atrophy
- Sterility

Treatment

- Supportive care for fever, headache and malaise
- Antipyeritics
- Analgesics
- Adequate rehydration

Xerostomia

Dry mouth

It can be caused by the following:

- Sjogren's syndrome
- Irradiation
- Dehydration
- Psychogenic
- Drugs

Sjogren's syndrome

- Presents with dryness of the eyes and mouth (primary type)
- In the secondary type, dryness occurs in association with rheumatoid arthritis or other connective tissue disease

Neoplasms of the salivary gland

The next most common neoplasms of the mouth after squamous cell carcinomas [PSI]

Above 70% develop in the parotid gland

Over three-quarters are benign

Women are slightly more frequently affected

Classification

The modified WHO classification (1972) includes:

Epithelial tumours

Adenomas:

- Pleomorphic adenoma ('mixed tumour')
- Monomorphic adenomas
- Warthin's tumour, oxyphoitic adenoma
- Carcinomas:
- Mucoepidermoid carcinoma
- Acinic cell carcinoma
- Adenocarcinoma
- Epidermoid carcinoma
- Undifferentiated carcinoma
- Malignant mixed tumour

Non-epithelial tumours

- Lymphomas
- Sarcomas

Clinical features

Benign tumours are generally asymptomatic enlargements

Malignant varieties are painful, irregular, ulcerative and metastatic

Investigations

Sialography

- Postero-anterior view of the skull
- Oblique lateral view of the jaws

Management

Benign and malignant lesions: surgical excision

Malignant lesions: radiotherapy and chemotherapy in addition to excision

Secondary bacterial infections: treat with antibiotics e.g. ampicillin/cloxacillin 250/250 mg every 6 hours for 5-7 days

- Adjust doses as appropriate for children

TEMPOROMANDIBULAR JOINT DISORDERS

Introduction

These disorders can be grouped under the following conditions:

Temporo-Mandibular Joint (TMJ) pain-dysfunction syndrome

Osteoarthritis

Rheumatoid arthritis

Trauma

Developmental defects

Ankylosis

Infection

Neoplasia

TMJ pain dysfunction syndrome

The most common problem in or around the TMJ

Clinical features

Equal frequency between genders, but five times as many females seek treatment

Patients are usually between 15 and 40 years

Unilateral or bilateral dull pain within the TMJ and/or surrounding muscles, sometimes on waking or during eating or speech

TMJ may lock in the open or closed positions, occasionally

TMJ sounds such as clicking, crunching or grating are often described

Associated headache is usually located in the temporal region

Pain is cyclical and usually resolves, but may recur

May be associated with psychological stress

Differential diagnoses

Migraine

Psychologic depression

Treatment objectives

Most symptoms are self-limiting and do not require treatment

Treatment should be conservative and reversible

Non-drug treatment

Educate patient about the condition, emphasizing its frequency and self-limiting nature

Soft diet

Apply moist heat to painful muscles

Physiotherapy

Drug treatment

Analgesics as appropriate

Anxiolytics

- Diazepam 5 mg orally 1 hour before sleep, then 2 mg every 12 hours, for up to 10 days (maximum)

Supportive measures

Occlusal splints

Osteoarthritis

Rare

Increasing incidence after 50 years

Joint crepitus denotes degenerative joint disease

May be accompanied by pre-auricular pain, but not involving the masticatory muscles

Radiographs (e.g. panoramic, trans-pharyngeal, trans-cranial, oblique, lateral, open and closed) show degenerative joint disease

Rheumatoid arthritis

A disease of unknown aetiology

Autoimmune mechanisms and immune complex formation have been implicated

Usually begins in early adult life and affects females more frequently

Patients rarely complain of pain from TMJ but clinical examination shows TMJ involvement in 50% of cases

Limitation of mouth opening; softness, crepitus, referred pain, and tenderness on biting

Severe disability is unusual

Trauma

Clinical features include:

Condyle fracture or trauma arthritis

Pain and trismus of traumatic arthritis resolve after one week

Micro-trauma from parafunction may result in chronic symptoms

Dislocation is usually a result of trauma and is rare; very rarely it occurs after yawning

Developmental defects

Aplasia of the condyle is extremely rare and may be unilateral or bilateral

Hypoplasia of the condyle may be congenital or acquired

Cause of congenital hypoplasia is not known; either one or both condyles may be involved

Acquired hypoplasia may be secondary to

trauma, infection or radiation

Hyperplasia of the mandibular condyle is rare and self-limiting. Cause is unknown. It is generally unilateral with resultant facial asymmetry, deviation of mandible to the opposite side and malocclusion

Ankylosis

Follows trauma, infection or other inflammatory condition

A disorder that leads to restriction of mouth opening from partial reduction to complete immobility of the jaw

Clinical features

An extremely disabling affliction that causes problems in mastication, digestion, speech, appearance, and oral hygiene

In growing patients, deformities of the mandible

and maxilla may occur together with malocclusion

Treatment

Temporo-mandibular joint (TMJ) ankylosis in children is challenging

Surgical correction is technically difficult

Incidence of recurrence after treatment is high

7-step protocol consists of:

- Aggressive excision of the fibrous and/or bony ankylotic mass

- Coronoidectomy on the affected side
- Coronoidectomy on the contralateral side, if the above 2 steps do not result in a maximal incisal opening greater than 35 mm or to the point of dislocation of the unaffected TMJ
- Lining of the TMJ with a temporalis myofascial flap or the native disc, if it can be salvaged
- Reconstruction of the ramus condyle unit with either distraction osteogenesis or costochondral graft and rigid fixation
- Early mobilization of the jaw. If distractionosteogenesis is used to reconstruct the ramus condyle unit, mobilization begins the day of the operation. In patients who undergo costochondral graft reconstruction, mobilization begins after 10 days of maxillomandibular fixation.
- Finally, all patients receive aggressive physiotherapy

Adults may be treated with one or several osteotomies or joint replacement

Neoplasia

Primary neoplasms arising from the structures of the TMJ are extremely rare

Benign tumours such as chondromas and osteomas are more frequent than sarcomas arising from bone or synovial tissues

Others are secondary carcinomas

CHAPTER 7:

SKIN, HAIR AND NAILS DISEASES

BACTERIAL INFECTIONS

CELLULITIS

Introduction

An acute suppurative bacterial infection of the skin and soft tissue, often with involvement of underlying structures: fascia, muscles and tendons. Most often due to ß-haemolytic streptococci or*Staphylococcus aureus*.Less common causes include: Anaerobic bacteria, *Mycobacteria, Proteus, Pseudomonas* and rarely *Crytococcus*.Usually (but not always) follows some discernible wound.Often a complication of immunosuppression like diabetes and HIV/AIDS

Epidemiology

The prevalence is unclear. It is commoner in adult malesabove 45 years of ageand young children. Risk factors include: immunosuppression, malnutrition, obesity,

elderly persons, peripheral vascular disease, lymphoedema and recent injuries to the skin.

Clinical features

Areas of oedema; rapidly spreading

Erythema (rapidly becomes intense and spreads)

Tenderness and warmth

- Often accompanied by fever, lymphangitis, regional lymphadenitis

Systemic signs of toxicity

Area becomes infiltrated and pits on pressure

Sometimes the central part becomes nodular and surrounded by a vesicle that ruptures and discharges pus and necrotic material

Differential diagnoses

Erysipelas

Deep vein thrombosis

Complications

- Unusual in immunocompetent adults; children and compromised adults are at higher risk immuno

Septicaemia

Gangrene

Metastatic abscesses

Recurrent cellulitis may predispose to chronic lymphoedema

Investigations

Blood culture
Full Blood Count with differentials
Fasting blood glucose
HIV screening
Wound swab for microscopy, culture and sensitivity
Urinalysis

Treatment objectives

Eradicate infection
Treat underlying immune suppression
Prevent complications

Drug treatment

Ampicillin/cloxacillin
*Adult:500*mg - 1 g orally every 6 hours for 5 - 7 days
Child under 5 years: a quarter adult dose; 5 - 10 years: half adult dose

Or:

Cloxacillin
Adult: 500 mg orally every 6 hours for 5 - 7 days
Child under 5 years: a quarter adult dose; 5 - 10 years: half adult dose
Ciprofloxacin
Adult:250- 750 mg orally every 12 hours for 5 - 7days

Child: see note on caution

Ceftriaxone

Adult: 1 g intravenously or intramuscularly daily for 3 days

Child: neonate, 20 - 50 mg/kg by intravenous infusion over 60 minutes; 1 month - 12 years, body weight less than 50 kg: 50 mg/kg by deep intramuscular injection or intravenous injection over 2 -4 minutes, or by intravenous infusion

- Intramuscular injections over 1 g should be divided over more than 1 site
- Doses of 50 mg/kg and more should be given by intravenous infusion only
- Use only when there is significant resistance to other drugs

Tetanus Prophylaxis

Surgical treatment

May need incision and drainage or debridement

Caution, contraindications

Ciprofloxacin is contraindicated in growing adolescents and children below 12 years; also contraindicated in pregnancy

Prevention

Treat any wound promptly

FURUNCULOSIS (Boils)

Introduction

Infection of a hair follicle by staphylococcal organisms, that leads to an inflammatory nodule, with a pustular centre

A carbuncle is merely two or more confluent furuncles, with separate heads

Recalcitrant cases may occur with a background of immune suppression

Alcoholism:

- Malnutrition
- Blood dyscrasias
- Disorders of neutrophil function
- Diabetes
- AIDS

May occur in patients with atopic dermatitis

May be iatrogenic

Clinical features

Can be found on all body sites where hairs are present Starts with a small, yellow creamy pustule that rapidly evolves into a red nodule, often with a central yellowplug

As the lesion expands, it becomes: Painful and tense

Associated with local oedema, lymphangitis, regional lymphadenopathy and fever

- Eventually, the central part of the nodule becomes soft and drains spontaneously

Healing occurs after about 1 - 2 weeks with scar formation

Differential diagnoses
Folliculitis
Cutaneous myiasis
Acne inversa in the axilla or groin

Complications
Cellulitis
Septicaemia
Carvenous sinus thrombosis when the lesions are on the head and neck

Investigations
Wound swab for bacteriology and sensitivity
Full Blood Count with differentials
Fasting blood glucose
HIV screening
Urinalysis

Treatment objectives
Treat infection
Correct predisposing factors
Prevent complications

Drug treatment
Topical antibiotics
- Gentamicin 0.3% cream
- Resistance may set in with prolonged use of Systemic antibiotics

Usually unnecessary except for head and neck lesions, or when the boil is accompanied by fever, chills, regional lymphadenopathy,

or a feeling of being unwell

- Co-trimoxazole

Adult: 960 mg orally every 12 hours for 5 - 10 days

Child: 6 weeks - 5 months: 120 mg; 6 months - 5 years: 240 mg; 6 - 12 years: 480 mg taken orally every 12 hours for 5 - 10 days

- Erythromycin

Adult and child over 8years 250 - 500 mg orally every 6 hoursor-1g12hourlyfor5-10days

Child: up to 2 years: 125 mg orally every 6 hours; 2 – 8 years: 250 mg every 6 hours for 5 - 10days

Surgical treatment

A small puncture wound often gives less of a scar than allowing spontaneous rupture; it also reduces the pain

Should be under antibiotic cover to prevent septicaemia

IMPETIGO CONTAGIOSA

Introduction

A superficial, highly contagious, bullous skin disorder caused by coagulase positive staphylococci and occasionally -haemolytic streptococci

Clinical features

Children are more commonly affected

Initial lesions are superficial vesicles, or

bullae found around orifices: eyes, nose and ears

Begins with a 2 mm erythematous macules which quickly develop into vesicles or bullae

- Blisters are superficial and rupture easily, releasing a thin straw-coloured seropurulent discharge
- The exudate dries to form loosely stratified golden yellow crusts

Auto-inoculation from fluid (from ruptured blister) leads to multiple lesions

As the lesions spread peripherally and the skin clears centrally, large circles are formed by fusion of the spreading lesions to produce gyrate patterns

Lesions heal without scarring, but may leave behind erythema and hyperpigmentation

Other pruritic dermatoses may become impetiginized (i.e. infected with the above organisms):

- Scabies
- Pediculosis
- Papular urticaria
- Atopic eczema

Differential diagnoses

Ringworm Ecthyma

Herpes simplex

Complications

Regional lymphadenopathy

Cellulitis
Rarely: septicaemia
Rarely: acute glomerulonephritis, if nephritogenic strain of streptococcoci is involved

Investigations
Wound swab for bacteriology and sensitivity

Treatment objectives
Treat infection
Treat underlying pruritic dermatoses
Prevent complications

Non-drug treatment
Debride crusted lesions with soap and water **or** desloughing antibacterial agents
Dry weepy lesions with astringent such as potassium permanganate, sodium chloride 0.9% solution, hydrogen perioxide

Drug treatment
Erythromycin
Adult and child over 8 years:250- 500 mg orally every 6 hours**or**500 mg - 1 g every 12 hours for 5 - 10 days *Child:* up to 2 years: 125 mg orally every 6 hours; 2 - 8 years: 250 mg every 6 hours

Or:
Co-trimoxazole

Adult: 960 mg orally every 12 hours for 5 - 10 days
Child: 6 weeks - 5 months: 120 mg; 6 months - 5 years: 240 mg; 6 - 12 years: 480 mg taken orally every 12 hours for 5 - 10 days

Supportive measures
Debride crusted lesions: Dislodging antibacterial agen Avoid auto-inoculation e.g. with fingers, shaving brushes, handkerchiefs, or pillow cases
- Strict personal hygiene Treat underlying skin disease(s)

Notable adverse drug reactions
Sulphonamide and co-trimoxazole: fixed drug eruption

DERMATITIS AND ECZEMA

ATOPIC DERMATITIS (Atopic eczema)
Introduction
Inflammation of the superficial dermis and epidermis, leading to disruption of the skin
Dermatitis and eczema are used interchangeably, although eczema was initially used to refer to blistering dermatitis, being derived from a Greek term meaning 'to boil over'
Atopic dermatits is a hereditary disorder characterised by dry skin, the presence of

eczema, and onset less than 2 years

Epidemiology

A common condition in children; the overall prevalence is rising with a slight male preponderance

Clinical features

Atopic dermatitis looks different at different ages and in people of different races Essential features are:

Pruritic, exudative, or lichenified eruptions on face, neck, upper trunk, wrists and hands, and in the antecubital and popliteal folds

Personal or family history (in about 70% of cases) of

- Allergic manifestations e.g. asthma, hay fever, allergic rhino-conjunctivitis, or eczema

Chronic or chronically relapsing dermatitis

Dry skin

The age at which eczema ceases to be a problem varies

- Many children show a significant improvement by the age of 5 years
- Most will have only occasional flare-ups by the time they are teenagers
- A few continue to have troublesome eczema in adult life, especially those children that suffer from hay fever

There is no "cure" for atopic eczema

Differential diagnoses

Seborrhoeic dermatitis (especially in the infant)
Irritant or allergic contact dermatitis
Nummular dermatitis
Scabies
Psoriasis (especially palmo-plantar)
In infants certain immunodeficiency syndromes

Complications

Bacterial infections of the skin Eczema herpeticum
Complications of over treatment with steroids

Investigations

RAST or skin tests may suggest dust mite allergy
Eosinophilia and increased serum IgE levels may be present but are nonspecific
Blinded food challenges: for diagnosing food allergy

Treatment objectives

Suppress inflammation
Reduce itching
Prevent complications

Drug treatment

Topical:

Hydrocortisone 1% or betamethasone valerate 0.1%

- Apply twice a day until the skin improves then decrease to once a day or less frequently as needed

Tacrolimus (0.03% or 1%) apply twice daily

Systemic therapy: Steroids (only to control acute exacerbations)

- Prednisolone

Adult initially up to 10 - 20 mg orally daily

- Preferably taken as a single dose in the morning after breakfast
- In severe disease: up to 60 mg orally daily, as a short course for 5-10 days

Or:

- Triamcinolone acetonide 40 mg by deep intramuscular injection, into gluteal muscle

Criteria for systemic steroid therapy

Failed maximal therapy; little improvement after environmental changes

Chronic unbearable, unrelenting itch

Erythroderma without infections

Social setting in which other modalities are impossible

Smallpox vaccination is absolutely contraindicated

Guidelines for the use of potent topical

steroids in infants
Do not use on the face, axillae, diaper area or flexures
Do not use under occlusion
Do not use for an area greater than about 25% of total body surface area
Do not use for more than 2 weeks consecutively and do not give refills
Do not dispense more than 50 g per week
Always use sparingly

Adjunctive measures
Exclusive breastfeeding; milk substitute if need be
Attention to cleanliness especially in the diaper region
Avoid excessive bathing, vigorous rubbing, or chafing
Avoid unduly heavy, tight, or soiled clothing
Treat local infections
Pat (rather than rub) skin dry after bath and immediately lubricate skin with petroleum jelly or emulsifying ointment
Showers should be warm to cool, not hot
Tub soaking is good, if followed by adequate lubrication
Avoid wool; its fibers are irritating
Emotional stress leads to increased scratching
In patients and parents of affected children, other psychologic techniques may be useful
Secondary skin infection with bacteria such as

Staphylococcus aureus may worsen the dermatitis and itching

Patients must consciously be shielded from anyone with varicella or herpes simplex

Keep finger nails trimmed short

Some kinds of soap may irritate and dehydrate the skin; use synthetic soap powders

Reassure patients and/or anxious parents

Use patient education handouts

Allergy tests, restriction diets and environmental hypoallergenic changes will not cure eczema

Notable adverse drug reactions

Steroids

- Increased susceptibility to and severity of infection
- Activation or exacerbation of tuberculosis, amoebiasis, strongyloidiasis
- Risk of severe chickenpox in non-immune patients
- Nausea, dyspepsia, hiccups
- Hypersensitivity reactions
- Atrophy of the skin; striae, telangiectasia, petechiae
- Glaucoma, cataracts
- Cushingoid syndrome, adrenal/pituitary suppression, hyperglycaemia and diabetes mellitus
- Suppression of growth in children
- Menstrual irregularities

- Oedema
- Electrolyte imbalance
- Hypertension
- Pseudotumour cerebri

CONTACT DERMATITIS

Introduction

An acute or chronic dermatitis that results from direct skin contact with chemicals or allergens These agents could be Chemicals

Animal or plant products

Physical agents like heat, cold, ultraviolet rays or ionizing radiation

Contact dermatitis is classified as:

- Irritant dermatitis
- Acute irritant dermatitis
- Cumulative insult dermatitis
- Allergic contact dermatitis
- Phototoxic dermatitis
- Photo-allergic dermatitis

Clinical features

Acute phase

- Tiny vesicles, weepy and crusted lesions

Resolving or chronic contact dermatitis

- Scaling, erythema, and possibly thickened (lichenified) skin
- Itching, burning, and stinging may be severe

Contact dermatitis is recognized by the distribution and configuration of the lesion

which usually corresponds to the contactant e.g.

- Face: cosmetics
- Photodermatitis: airborne allergens e.g. dust, fumes, sprays
- Neck: nickel necklace, perfume, and collars of garments
- Hands: various chemicals handled at home, at work and at leisure hours
- Feet: shoes, socks, remedies for athletes' foot, etc

Differential diagnoses
Atopic dermatitis
Seborrhoeic dermatitis
Psoriasis
Dermatophyte infection
Lichen planus
Face: lupus erythematosus, pellagra, rosacea

Complications
Impetiginization
Secondary dissemination

Investigations
Patch test
Occupational site assessment

Treatment objectives
Cure the dermatitis
Identify cause(s) and avoid further contact

Drug treatment
As for atopic dermatitis

Supportive measures
Counselling (after identifying the cause)
Allergen replacement

EXFOLIATIVE DERMATITIS (Erythroderma)
Introduction
Refers to the involvement of all or most of the skin surface by a scaly erythematous dermatitis
Usually a secondary or reactive process to an underlying cutaneous or systemic disease
Some causes:
Contact dermatitis
Atopic eczema
Seborrhoeic dermatitis
Drug eruptions
Lichen planus and lichenoid eruptions
Crusted scabies
Pediculosis corporis
Dermatophytosis
Psoriasis
Pemphigus foliaceus
Lymphomas and leukaemia
Ichthyosiform erythroderma
Pityriasis rubra pilaris

Clinical features

May be acute or chronic
The irritating process is followed by a patchy erythema which spreads rapidly within 24 hours Pyrexia, malaise and shivering
Scaling
Irritation and tightness
Skin feels cold
The periorbital skin is inflamed and oedematous, resulting in ectropion, with consequent epiphora
Moderate-to-gross generalized enlargement of lymph nodes in the absence of an underlying malignant lymphoma (dermatopathic lymphadenopathy)
The nodes are rubbery in consistency
The general picture is modified by the initial cause
Pruritus is often intense if due to atopic eczema or lymphoma

Differential diagnoses
All the causes of exfoliative dermatitis listed above

Complications
Hypothermia
Hypoalbuminaemia
Dehydration
High output cardiac failure
Septicaemia
Enteropathy

Steatorrhoea
Anaemia

Investigations
Full Blood count and differentials; ESR Urea and Electrolytes
Histopathology
Blood culture

Treatment objectives
Restore the skin to normal
Treat underlying disease
Prevent or treat complications

Drug treatment
Systemic steroids in high doses - Prednisolone 40 - 60 mg orally per day
Treat impetiginization and septicaemia as appropriate (depending on results of culture and sensitivity)
Further treatment depends on the cause of exfoliative dermatitis

Adjuvant therapy
Adequate hydration
Emolients for skin (see Atopic eczema)
Keep warm
Adequate nursing care
Appropriate nutrition and haematinics

Prevention

Avoid over-treatment of skin diseases and polypharmacy, generally

Do not abuse the skin with "medicated" soaps and herbal concoctions

Get appropriate management of skin disease(s) from qualified personnel

PARASITIC DERMATOSES

CUTANEOUS LARVA MIGRANS (Creeping eruption)

Introduction

An infection of the skin by various nematode larvae which migrate, but never reach internal organs or complete their life cycles

Migration leads to twisting, winding linear skin lesions produced by the burrowing of larvae Victims are usually:

People who go barefoot at the beaches

Children playing in sandboxes and crawling on the bare ground

Carpenters and plumbers working under homes

Gardeners

The most common causes are cat and dog hookworm

- *Ancylostoma braziliense*
- *Ancylostoma caninum*
- *Necator americanus*
- *Gnathostoma spinigerum*
- *Strongyloides stercoralis*

Clinical features

Shortly after entering the skin:

The larvae elicit intense pruritus

Tiny papules and even papulovesicles develop

As the larvae begin to migrate:

Intermittent stinging pain occurs

Thin red, tortuous and minimally elevated lines are formed in the skin

- Rate of migration varies with the species
- Pruritus and excoriation promote secondary bacterial infections

Intestinal infections with *Strongyloides stercoralis* may be associated with perianal larva migrans syndrome called 'larva currens' because of the rapidity of larval migration (up to 10 cm/hr)

- Larva currens is an autoinfection caused by penetration of the perianal skin by *Strongyloides stercoralis*

Differential diagnosis

Ring worm

Complications

Secondary bacterial infection

Fatal *Strongyloides stercoralis* hyperinfection in immunocompromised patients

Investigation

None useful to management

Treatment objectives
Eradicate the larvae
Eradicate gut Strongyloides
Treat impetiginization
Prevent re-infection

Drug treatment
Ivermectin
Adult: 150 microgram/kg orally as a single dose
Child over 5 years old: 200 micrograms/kg orally daily for 2 days

Or:
Albendazole
Adult: 400 mg orally twice daily for 2 days, repeated after 3 weeks if necessary
Child over 2 years: 400 mg once or twice daily for 3 days, repeated after 3 weeks if necessary
Antihistamines for pruritus
Antibiotics for secondary bacterial infections

Prevention
Avoid direct contact of skin with sand

GUINEA WORM DISEASE (Dracunculiasis)
Introduction
An infection by a very long nematode, *Dracunculus medinensis*

Contracted through drinking water contaminated with water fleas (cyclops) infected with Dracunculus

Except for remote villages in Rajastan desert of India and Yemen the disease is now only seen in Africa, between the Sahara and Equator

Nigeria is one of the few countries with reports of >1,000 new cases a year

Efforts are currently going on to eradicate the disease in Nigeria

Pathophysiology

In the stomach, the larvae penetrate into the mesentery, where they mature sexually in 10 weeks

The female worm burrows to the cutaneous surface to deposit her larvae, causing specific skin manifestations

When the parasite comes in contact with water, the worm rapidly discharges its larvae, which are ingested by the cyclops

Clinical features

As the worm approaches the surface it may be felt as a cordlike thickening

It forms an indurated cutaneous papule

Several hours before the head appears at the skin surface there is (at the point of emergence)

- Local erythema
- Burning sensation

- Pruritus
- Tenderness

Soon after, the papule blisters and a painful ulcer develops, usually on the leg

- Ulcer may occur on other parts of the body e.g. the genitalia, buttocks, or arms

Differential diagnoses

Sickle cell ulcer
Stasis ulcer

Complications

Secondary infection
Cellulitis
Erysipelas
Progressive lymphoedema
Oesteomyelitis
Arthritis
Tetanus

Investigations

Radiograph of the affected area

- If osteomyelitis and arthritis (or calcified worms) are suspected

Treatment objectives

Resolve local inflammation to permit easier removal of the worm
Extract the worm Prevent and treat complications

Drug treatment

Metronidazole

Adult: 500 mg orally every 8 hours for 7 days

Child: 7.5mg/kg orally every 8 hours

Or:

Mebendazole

Adult: 400 - 800 mg orally daily for 6 days

Child over 1 year: usually 100 mg orally twice daily for 3 days

Or:

Ivermectin

Adult: 200 micrograms/kg orally as a single dose

Child. Consult specialist

Treat or prevent complications with antibiotics

Worm extraction

Traditionally:

Extract the worm slowly by winding it about a match stick or twig, removing 3 - 5 cm daily, with care not to rupture it

- In the event of such an accident, the larvae escape into the tissues and produce fulminating inflammation
- The process appears to be facilitated by placing the affected part in water several times a day

Notable adverse drug reactions, caution and contraindications

Metronidazole

- Avoid high dose regimens in pregnancy
- Avoid drinking alcohol during treatment and at least 48 hours after

Ivermectin

- Oedema (face and limbs)
- Fever, pruritus, lymphadenitis, malaise, hypotension
- Should not be used in the presence of concurrents, *loa* infection: risk of encephalopatic reactions to dying *loaloa* microfilariae
- Should not be used in patients with central nervous system diseases (e.g. meningitis): increased penetration of ivermectin into the CNS

Caution in early pregnancy

Prevention

Provide universal access to safe and portable water In hyperendemic areas, treat the whole population twice
yearly with ivermectin

MYIASIS

Introduction

Invasion of mammalian tissue by fly larvae
Furuncular myiasis may be caused by *Dermatobia hominisor* the Tumbu *fly*
Cordylobia anthropophaga
Larvae of *D. hominis* are often transferred by

mosquitoes
Usual host is cattle. People living near cattle-rearing areas are particularly vulnerable
Eggs, living larvae, or both are deposited on the skin or mucous membranes or on clothing
- Eggs hatch and produce larvae that then burrow into the skin and cause mild or severe inflammatory changes

Clinical features
Furuncular myiasis looks like a furuncle (boil)
Key feature is the presence of a tiny hole in the inflammed erythematous papule
There may be a sensation of motion within the furuncle
There may be intermittent stinging sensation
- In accidental myiasis, there is a pre-existing lesion, usually a leg ulcer, wound or ulcerated basal cell carcinoma

Differential diagnoses
Furuncles and carbuncles

Complications
Secondary bacterial infection

Investigation
Nil

Treatment objectives
Extract the maggot

Treat or prevent bacterial infection

Non-drug treatment
Apply petrolatum: the maggot crawls out to avoid asphyxiation

Or:
Extract the maggot by compressing simultaneously from beneath on both sides with a pair of spatulae

Drug treatment
Prevent bacterial infection with oral antibiotics if lesions are multiple
Wound myiasis is flushed out surgically with antiseptics: surgical debridement

Prevention
Iron clothes that are dried in the open air

ONCHOCERCIASIS (River blindness)
Introduction
A common chronic filarial disease in tropical regions which frequently cause pruritus and blindness
Causative organism *is Onchocerca volvulus*
The microfilariae are transmitted by female *Simulium,* tiny black flies which breed along small, rapidly moving streams
Female worms release motile microfilariae into the skin, subcutaneous issues,

lymphatics, and eyes

Clinical features

Interval from exposure to onset of symptoms can be as long as 1 - 3 years Skin lesions

- May be localized or cover large areas Intense pruritus
- A cardinal symptom; may occur in the absence of the skin lesions

Dermatitis

- Skin eventually becomes lichenified from chronic scratching
- Post inflammatory confetti-like depigmentation on the skin ("leopard skins") may occur in late onchodermatitis

Onchocercomata

- Subcutaneous nodules which develop on various sites of the body and contain myriad adult worms which can live for up to 14 years.

Firm, non-tender lymphadenopathy is a common finding in patients with chronically infected onchocerciasis

- "Hanging groin" describes the pendulous loose, atrophic skin sac that contains these large nodes

Microfilariae in the eye may lead to visual impairment and blindness

Differential diagnoses

Scabies

Pediculosis
Papular urticaria
Papulonecrotic tuberculids
Pruritic papular eruption of HIV
Other causes of generalized pruritus without a rash
Other causes of subcutaneous nodules e.g.

- *Sparganosis*
- *Paragonimiasis*
- *Gnathostomiasis*
- *Cysticercosis*
- *Echinococcosis*

Complication
Blindness

Investigations
Skin snips or punch biopsy for microfilariae
Excise nodule for adult worms
Mazzotti test reaction
Slit lamp eye examination

Treatment objectives
Kill the microfilariae
Eliminate source of microfilarial release
Prevent blindness

Drug treatment
Ivermectin
- As a single oral dose of 150 microgram/kg in adults and children over 5 years
- Repeat every 6 months for 2 years and yearly

for 12-15 years or longer

Eye involvement

- Prednisolone 1 mg/kg orally should be started several days before treatment with ivermectin

Surgical

Excise individual nodules (nodulectomy)

Notable adverse drug reactions, caution and contraindications

No food or alcohol should be taken for at least 2 hours before or after dosage

Pregnant women should not receive ivermectin until after delivery

Breastfeeding mothers should not be treated until the infant is at least 1 week old

Prevention

Use biodegradable insecticides to kill flies

Netting and repellents remain crucial.

Provide access to safe and portable water

In hyperendemic areas, treat the whole population twice yearly with ivermectin

PEDICULOSIS (Lice)

Introduction

Diseases due to blood sucking lice

Can be divided into three conditions:

Pediculosis capitis (head lice):

- Caused by *Pediculus humanus var. capitis*

Pediculosis corporis (body lice):

- Caused by *P. humanus var. corporis*

Phthiriasis pubis (pubic lice):

- Caused by Phthirus pubis

The arthropods are transmitted from human to human via:

Direct contact

Sharing of combs, brushes, towels *(P. capitis)*

Sharing clothing *(P. corporis)*

Shearing underwear

Sexual intercourse or any intimate personal contact *(P. pubis)*

Clinical features

Pediculosis capitis:

Generally the only complaint is pruritus:

Nits can easily be seen at the base of the hairs; careful inspection may reveal the adult louse

Secondary impetiginization is common because of the itching

- Cervical nodes may become enlarged

Children and individuals with long hair are more likely to be affected

Homeless people and refugees are also vulnerable

No age or economic stratum is immune

- School children who share school caps, hair brushes and combs, pillow cases are particularly vulnerable

Pediculosiscorporis :

Pruritus may be the only symptom in some patients

Chronic scratching may result in characteristic

hemorrhagic puncta and linear excoriations

Patient eventually develops intensely pruritic papules and nodules, numerous excoriations, secondary infections and even lymphadenopathy

The combination of excoriations, hyperpigmentation, healed scars and secondary impetiginization is quite typical and known as "vagabond's skin"

Overcrowding and poor personal hygiene promote infestation

Refugees, destitutes and vagrants are particularly vulnerable

Pediculosis pubis:

Most often found in the pubic and axillary hairs

Occasionally may be found on abdominal or trunk hairs

On rare occasions may be seen on the scalp, eyebrows and even eyelashes

Pruritus is also a symptom

Classic clinical finding is the maculae cerulae

- Indistinct blue-grey or slate-coloured macules ranging in size from several millimetres to several centimeters

- They result from the bite of the louse causing small intracutaneous haemorrhages
- The colour is due to blood whose haemoglobin has been altered by the saliva

Differential diagnoses

P. capitis:
- Seborrhoeic dermatitis
- Pityriasis amiantacea
- Peripilar keratin
- Hair casts
- Piedra

P. corporis :
- Scabies
- Atopic dermatitis
- All pruritic dermatoses

P. pubis:
- Scabies
- Candidiasis
- In the axillae trichomycosis axillaris

Complications

Secondary bacterial infections

The body louse serves as a vector for diseases:

Epidemic typhus *(Rickettsia prowazekii)*

Trench fever *(Bartonella quintana)*

Relapsing fever *(Borrelia recurrentis)*

Investigations

P. capitis and pubis:

- Examine louse or the nits on epilated hair strands (especially from behind the ears) under the microscope

P. corporis:

- Examine the seams of clothing for nits and lice

Treatment objectives

Eradicate the lice

Prevent re-infection

Treat complications

Drug treatment

P. capitis:

1% permethrin cream rinse

- The cream is lathered through the hair, left on for 10 minutes and thoroughly rinsed out. A fine-tooth comb should be used to remove adherent nits
- Repeat treatment after a week

P. corporis:

Treat dermatitis with antipruritics or corticosteroids

Treat secondary infection with oral antibiotics

Supportive measures

P. capitis:

All contact individuals should be examined

and treated as necessary

Pillow cases should be disinfested as for clothing.

P. corporis:

Eradicate lice from clothing by laundering in hot water or machine-drying at a high temperature, followed by ironing the seams

P. pubis:

Treatment is the same as for pediculosis capitis, with the exception that pediculosis of the eyelashes should be treated with an occlusive ophthalmic ointment applied to the eyelid margins for 10 days

- Affected persons' sexual contact(s) should be treated simultaneously

Notable adverse drug reactions, caution

As stated under scabies

Prevention

Improve personal hygiene

Do not share hair combs, brushes, clothing, pants and pillows

SCABIES

Introduction

An intensely pruritic infestation caused by human mite *Sarcoptes scabiei*

Contracted by close contact and rarely via fomites

Occurs commonly in children and inmates of

overcrowded institutions such as prisons and boarding

Houses Infection of households is common

Sexual intercourse is also another possible method of spread among adults

Sharing a bed or using the same underwear will also suffice to contact the disease

Clinical features

Severe pruritus worse at night is characteristic

The typical lesion is the burrow - It is hardly seen because of the marked excoriation and secondary infection on the skin

Papulo-pustular eruptions with excoriation and impetiginized.

Characteristic sites of predilection:

Interdigital spaces of the fingers

Flexural surfaces of the wrist

Extensor surfaces of the elbows and knees

Anterior axilliary area

Nipples

The phallus (especially in adults)

General immune status and experience with *S. scabiei* play a role

In a normal host, the initial infection is asymptomatic for about 3 - 6 weeks during which time the individual is capable of transmitting the disease

- All family or living unit members must therefore be treated, not just the itching

ones

After a reinfestation, symptoms appear within 24 hours

Crusted scabies (Norwegian scabies)

An uncommon variant of scabies

Patient fails to mount a resistance and the mites proliferate dramatically

May be found among HIV/AIDS patients, institutionalized inmates like prisoners, refugees, and psychiatric patients

Differential diagnoses

Infantile acropustulosis
Atopic dermatitis
Papular acral dermatitis of childhood
Dermatitis herpetiformis

Complications

Secondary bacterial infection leading to acute glomerulonephritis

Investigations

Burrow scraping on to a glass slide for microscopy Video dermatoscopy

Treatment objectives

Treat the infestation
Treat secondary bacterial infection
Relieve pruritus

Drug treatment

Scabicides:

Permethrin 5% cream

Adult: apply over the whole body and wash off after 8-12 hours

Child: supervision required with application and rinsing

Or:

Benzyl benzoate 25% in emulsion

Adult: apply over the whole body; repeat without bathing next day and wash off 24 hours later

- If necessary apply a third time

Child: Benzyl benzoate is an irritant and should be avoided in children

Or:

Precipitated sulfur 5 - 10% in petroleum jelly

Adult and child: apply over all the body daily for 7 - 10 days

Antihelminthic: Ivermectin

Adult: Single 200 microgram/kg oral dose for crusted scabies

Child: over 5 years: 200 micrograms/kg daily for 2 days

Antihistamine:

Chlorphenamine

Adult: 4 mg orally every 4 - 6 hours; maximum 24 mg a day

Child: 1 month - 2 years 1mg orally every 12 hours; 2 - 5 years: 1 mg every 4 - 6 hours; 6 - 12

years: 2 mg every 4-6 hours
Topical antipruritic:
Crotamiton cream (for residual itching)
Adult: apply every 8 - 12 hours
Child: less than 3 years: apply once daily only

PAPULOSQUAMOUS DISORDERS

LICHEN PLANUS

Introduction

A chronic, pruritic, papular skin disease
The three cardinal features are:
Skin lesions
Mucosal lesions
Histopathologic features of band-like infiltration of lymphocytes and melanophages in the upper dermis
Some of the drugs known to cause lichen planus (LP):
Chloroquine
Quinacrine
Quinidine
Gold
Streptomycin Tetracycline
NSAIDs
Phenothiazines
Hydrochlorothiazide

Clinical features

LP has been found in children, young and middle-aged adults

The skin lesions are flat-topped polygonal papules with a characteristic colour

- Violaceous in fair skinned people but slate-grey on black skin

Itching is mild-to-severe

Like psoriasis, lesions often occur on sites of trauma and scratch marks (Koebner's or isomorphic phenomenon)

Wickham's striae are fine white streaks present on the tops of papules

The lesions are distributed mainly on:

- Flexor surfaces of the wrist
- Lumbar area
- The penis, tongue, buccal and vaginal mucous membranes

On the buccal mucous membrane it may present as white reticulate pattern or plaque which may after several years transgress into squamous cell carcinoma

The nails are also affected with:

- Pitting, roughening and splitting (trachyonychia)
- Thickening (pachyonychia)
- Encroachment of the nail fold on the nail plate (pterygium ungium)

Total destruction of all 20 nails may precede, accompany, or follow the onset of skin lesions

The hair follicles in the scalp may also be affected (lichen planopilaris) with post-inflammatory scarring alopecia

Hepatitis C infection is found with greater

frequency in lichen planus than in controls

Healing of the skin lesions leave post-inflammatory hyperpigmentation

Differential diagnoses

Consider other papulosquamous disorders:

Psoriasis

Pityriasis rosea

Lupus erythematosus

Secondary syphilis

Lichen striatus

Parap soriasis

Pityriasis rubra pilaris

Nummular eczema Oral lesions:- Erosive lesions may mimic

Aphthous stomatitis and herpes simplex

- White plaques may be confused with Pre-malignant leukoplakia

White sponge naevus

Complications

20-nail dystrophy

Rarely, squamous cell carcinoma of oral and hypertrophic lichen planus

Investigations

Histopathology

Hepatitis C antigen

Treatment objectives

Relieve itching

Clear lesions
Suppress inflammation

Drug treatment

Topical corticosteroids: Beclomethasone dipropionate 0.1% cream

- Apply 1 - 2 times daily
- Not licensed for use in children under one year

Bethamethasone valarate 0.1% cream and ointment

- Apply 1 - 2 times daily

For isolated or hyperkeratotic lesions apply corticosteroids under occlusion or use intralesional triamcinolone (see Psoriasis)

Scalp lesions:

Topical corticosteroids

Clobetasol propionate 0.05% lotion

- Apply thinly 1 - 2 times daily for up to 4 weeks

Mouth lesions:

Triamcinolone acetonide 0.1% in adhesive base

- Apply a thin layer 2 - 4 times daily for a maximum of 5 days; do not rub in

Or:

Tretinoin 0.025% cream

Adult and child: apply thinly 1 - 2 times daily

Systemic corticosteroids

Prednisolone

Adult: 20 - 40 mg orally daily for several weeks with reduction of dosage or switch to alternate-day therapy as soon as improvement is seen
Child: not recommended for children for this indication

Or:
Triamcinolone acetonide 40 mg intramuscularly once or twice (at a 6-week interval)

Or:
Ciclosporin
Adult and child over 16 years: 2.5 mg/kg daily in two divided doses
- If good results not achieved within two weeks increase rapidly to maximum 5 mg/kg daily

Notable adverse drug reactions
See Psoriasis

Prevention
Avoid precipitating drugs

PITYRIASIS ROSEA
Introduction
A common, mild, inflammatory exanthem
Tends to be seasonal
- More common during the fall, winter and spring in temperate countries

- In Nigeria more common during the early part of the rainy season (though cases are seen throughout the year)

Common among siblings or other family/household members

The seasonal clustering and household concurrence are suggestive of an infective origin

- Increasingly regarded as a delayed reaction to a viral infection (most likely Human Herpes Virus 7)

Clinical features

Largely a disease of adolescents and in young adults, but it has been described all age groups

Rarely, there is an observable prodrome of pharyngitis, malaise and mild headache

The initial lesion in 20 - 80% of cases ("herald patch") is often larger than the later lesions and precedes the general eruption by 1 - 30 days

- Often found on the trunk, but may appear on the face or extremities
- Oval with a collarette of scales
- May be diagnosed as "ringworm" before the other lesions appear

Other lesions consist of multiple erythematous macules progressing to small, red papules on the trunk Sun-exposed areas are spared

Papules enlarge and become oval with long axes parallel to each other, and following lines of cleavage: the so-called "Christmas tree" pattern

Pruritus is mild or absent

Some lesions may be atypical: vesicular, crusted, purpuric, follicular, lichenoid, and psoriasiform A variant, inverse pityriasis rosea also occurs

- Believed to be commoner in blacks
- Affects the face, neck, distal extremities and the flexures

Use of ampicillin early in the course of the eruption causes an explosive exacerbation of eruptions which become more inflammatory and urticarial

- Lesions may become impetiginized

The disease persists for about 6 weeks but may last for 3-4months

Healing may occur with postinflammatory hyper/hypopigmentation

Recurrences are uncommon (about 1%) but the lesions are usually mild and localized

Differential diagnoses

Secondary syphilis

Exanthematic or pityriasis rosea-like drug eruptions

Lichen planus

Guttate psoriasis

Tinea corporis

Tinea versicolor
Seborrhoeic dermatitis
Viral exanthems
Pityriasis lichenoides chronica

Complications
None

Investigations
Non-specific
VDRL
- If secondary syphilis is suspected (e.g. lesions on palms and soles with/without lymphadenopathy)

Treatment objectives
To relieve symptoms (if any)
Reassure patients about the harmless, self-limiting nature of the eruption

Drug treatment
Topical: Urea cream
- Useful as a hydrating agent: apply twice daily Systemic:

Oral antihistamine
- If pruritus is bothersome (see Urticaria)

Systemic corticosteroids:
- If complicated by ampicillin exanthematic eruption

Triamcinolone acetonide 40 mg intramuscularly as a single dose Antibiotics:

If lesions are impetiginized
Erythromycin 500 mg orally every 6 hours for 14 days

Notable adverse drug reactions, caution
Antihistamine; Triamcinolone: see Urticaria

Prevention
Unknown

PSORIASIS

Introduction

A chronic inflammatory skin disease which is characterized by

- Increased epidermal proliferation
- Epidermal thickening
- Erythematous lesions with silvery white scales
- Affects people of all ages in all countries

Cause remains largely unknown but it has been to variously attributed to genetic, climatic, nutritional, ecological and immunological factors

Triggers include:
Streptococcal or viral infections
Emotional crises
Pregnancy and delivery
Trauma (Koebner phenomenon)
Diet Alcohol
Cigarette smoking
Hypocalcemia

Stress

Infections e.g. streptococcal pharyngitis

May occasionally be provoked or exacerbated by drugs:

ACE inhibitors

Calcium channel blockers

P- adrenoceptor antagonists

Chloroquine

Lithium

Non-Steroidal Anti-inflammatory Drugs (NSAIDs)

Terbinafine

Lipid lowering drugs

Clinical features

Lesions are characterized by:

Sharp borders

Erythema

Increased scales

When scratched, scales fall off as tiny flakes that resemble scrapings from a candle (Candle sign)

If the scales are removed (exposing the dermal papillae) punctate bleeding from the enlarged capillaries occur (Auspitz sign)

Eruptive lesions may be intensely or mildly pruritic, or may be asymptomatic

All lesions begin as small scaly macules but may take divergent paths as they spread centrifugally Patterns seen may be:

- Guttate

- Follicular
- Numular
- Geographic
- Erythrodermic
- Annular
- Gyrate or serpenginous

Favoured sites are:

- Knees and elbows
- Scalp
- Palms and soles
- Nails

Intertriginous regions such as the gluteal cleft, groin, penis, labia, axillae, beneath the breasts and between the toes are involved (inverse psoriasis or psoriasis inversa)

There could also be other organ involvement e.g. psoriatic arthritis

The disease runs a chronic and highly variable course (waxes and wanes)

- New lesions may replace older, regressing ones
- Unstable lesions may evolve into psoriatic erythroderma or generalized pustular psoriasis

HIV/AIDS can lead to the onset or worsening of psoriasis

Differential diagnoses

Guttate psoriasis:

Pityriasis lichenoides et varioliformis

Acuta Pityriasis rosea

Secondary syphilis (psoriasiform syphilis)

Scalp, face, chest lesions:
Seborrhoeic dermatitis
Lupus erythematosus
Chronic truncal psoriasis:
Nummular dermatitis
Lichen planus
Small plaque parapsoriasis
Tinea corporis
Pityriasis rubra pilaris
Intertriginous areas:
Candidiasis
Intertrigo
Hailey-Hailey disease
Nail:
Tinea unguium
Lichen planus
Trachyonychia

Complications
Erythroderma
Arthritis mutilans

Investigations
Histopathology

Treatment objectives
To retard epidermal proliferation
Reduce inflammation
Prevent complications

Drug treatment

Choice of treatment depends on the site, severity and duration of the disease, previous treatment, and the age of the patient

Topical treatment: Corticosteroid ointment

- Hydrocortisone for the face and flexures
- Betamethasone or clobetasol for the scalp, hands and feet
- Application is followed by an occlusive dressing of a polyethylene film, which may remain in place for 12 - 24 hours to augment effectiveness

Dithranol ointment 0.1% - 2% (for moderately severe psoriasis)

- Initiate under medical supervision
- Start with 0.1%; carefully apply to lesions only, leave in contact for 30 minutes, then wash off thoroughly
- Repeat application daily, gradually increasing strength to 2% and contact time to 60 minutes at weekly intervals
- Wash hands thoroughly after use
- Avoid contact with eyes and healthy skin

Coal tar solution (for chronic psoriasis)

- Use either alone or in combination with exposure to ultraviolet light
- Apply 1 - 4 times daily, preferably starting with a lower strength preparation

Coal tar bath

- Use 100 mL in bath of tepid water and soak for 10 - 20 minutes
- Use once daily, to once every 3 days for at

least 10 - 20 minutes, and for at least 10 baths

- Often alternated with ultraviolet (UVB) rays, allowing at least 24 hours between exposure and treatment with coal tar
- Urea 10% cream or ointment (for dry scaling and itching skin)
- Apply twice daily, preferably to damp skin

Salicylic acid 3 - 5% in cold cream or hydrophilic ointment (for thick scaling)

Tazarotene 0.05% and 0.1% gels

- May be combined with topical steroids for mild-to-moderate plaque psoriasis

Tacrolimus ointment 0.1% or 0.03%

- For psoriasis in the flexures, face and penis, when potent steroids cannot be used and other agents are poorly tolerated

Small lesions and nail psoriasis

Intra-lesional corticosteroid injections of triamcinolone are frequently used

- Triamcinolone acetonide suspension 10 mg/mL may be diluted with sterile saline to make a concentration of 2.5 - 5 mg/mL
- For nail lesions inject triamcinolone in the region of the matrix and the lateral nail fold

Scalp

Soften scales with salicylic acid 3% in mineral/olive oil, massage in and leave on

overnight

- Then shampoo with a tar shampoo, and remove scales mechanically with a comb and brush
- Repeat daily until the scales are gone
- If 3% is not very effective, use 6% salicylic acid

Or:

Fluocinolone acetonide 0.01% in oil

- Apply and leave under a shower cap at night and shampoo in the morning
- After shampooing and while the hair is still wet, massage thoroughly into the scalp skin
- Attempting to remove scales by excessive brushing, scrubbing, or combing may result in sufficient trauma worsen psoriasis (Koebner's effect)

Ultraviolet light (UVL)

- For psoriasis involving more than 30% of the body surface 290 - 320 nm ultraviolet B (UVB) three times weekly for 18 - 24 treatments
- Lubricating the skin surface with mineral oil or petroleum jelly before UVL produces uniform penetration by reducing the reflection of light from the disrupted skin surface

PUVA (psoralen plus ultraviolet A)

- For patients who have not responded to standard UVB treatment

Severe psoriasis unresponsive to outpatient UVL, may be treated in a day care centre with the Goeckerman

- Use of crude coal tar for many hours and exposure to UVB light

Systemic therapy: Antibiotics to eliminate streptococcal pharyngitis

Methotrexate

Adult: 20 mg orally once weekly *Child: not* licensed for this indication Indicated for:

- Psoriatic erythroderma
- Moderate-to-severe psoriatic arthritis
- Acute pustular psoriasis (von Zumbusch type)
- Involvement of more than 20% total body surface
- Localized pustular psoriasis that causes functional impairment (e.g. hands)
- Lack of response to phototherapy, PUVA, or retinoids

Cyclosporine

- Induction therapy is 2.5 - 3.0 mg/kg given in a divided dose twice daily
- Can be increased to 5.0 mg/kg/day until a clinical response is noted. The dose is then tapered
- On discontinuation a severe flare-up may occur, suggesting that an alternative treatment (e.g. phototherapy or acitretin) should be instituted as the cyclosporine dose is reduced

Adjuvant therapy

Diet: fish oils rich in Q-3 polyunsaturated fatty acids

Patient education

Emotional support

Notable adverse drug reactions, caution and contraindications

Coal tar:

Contraindicated in inflammed, broken or infected skin

- May cause irritation, photosensitivity reactions, Hypersensitivity

Skin, hair, fabrics and bathtubs discoloured brown and smelly

Dithranol:

Irritant: avoid contact with eyes and healthy skin

Contraindicated in hypersensitivity; avoid use on face, acute eruptions, and excessively inflamed areas

- Discontinue use if excessive erythema occurs or lesions spread

Conjunctivitis following contact with eyes

Staining of skin, hair, and fabrics brown

Urea:

Avoid application to face or broken skin; avoid contact with eyes

May cause transient stinging and local irritation

Steroids:

When extensive areas are treated or when there is erythrodermic psoriasis, sufficient may be absorbed to cause adrenal suppression

May induce tachyphylaxis

Rebound often occurs after stopping treatment, resulting in a more unstable form of psoriasis

Intralesional injection may cause reversible atrophy at the injection site

Salicylic acid:

Widespread application may lead to salicylate toxicity

Ultraviolet light:

Burning of skin may cause Koebner's phenomenon and an exacerbation

Increased risk of skin cancer particularly in persons with fair complexions and albinos.

Examine periodically

Use protective glasses to prevent cataracts

Causes premature ageing of the skin

Should be administered only by experienced dermatologists

Methotrexate:

May cause blood disorders (bone marrow suppression), liver damage, pulmonary toxicity, GIT disturbances

- If stomatitis and diarrhoea occur, stop treatment

- Renal failure, skin reactions, alopecia, osteoporosis, arthalgia, myalgia, ocular irritation, may also occur
- May precipitate diabetes
- Monitor before and throughout treatment: blood counts and hepatic and renal function tests
- Contraception during and for at least 6 months after treatment for both males and females
- Contraindicated in pregnancy and breast feeding. Folic acid may be given to reduce toxicity Cyclosporin:

Nephrotoxic: monitor kidney function

Other side effects- hypertrichosis, hyperuricaemia, thrombocytopenia, malignancies and lymphoproliferative disorders (similar to other immunosuppressive therapies)

Tacrolimus:

See Atopic eczema

Prevention

Avoid exacerbating factors e.g. abrasions, scratches, harsh fibre bathing sponges, and the drugs listed above

Prevent streptococcal sore throat and treat promptly when it occurs

DERMATOPHYTE INFECTIONS (Tinea)

Introduction

Superficial fungal infection that affects keratinized tissues

Fungi that usually cause only superficial infections on the skin are called dermatophyte- classified in three genera: *Microsporum, Trichophyton and Epidermophyton*

Can be acquired from humans, animals, soil or vegetable matter

Common in tropical climate (which is hot and humid)

Infection could be spread by fomites

The mycoses caused by dermatophytes are called dermatophytosis, tinea, or ringworm

On certain parts of the body they have distinctive features characteristic of that particular site; therefore the tineas are divided into:

Tinea capitis (scalp)
Tinea barbae (beard)
Tinea faciei (face)
Tinea corporis (trunk)
Tinea cruris (groin)
Tinea manuum (hand)
Tinea pedis (feet)

Tinea unguium or onychomycosis (nail)

Clinical features

Varied: depending on the site of the body involved

Pruritis is a notable symptom

Tinea capitis:

Scalp involvement is seen predominantly in children

Lesions are varied in appearance: usually scaly, dry and annular, with or without alopecia

Some appear diffuse and scaly and may involve the whole of the scalp

Inflamed, pustular lesions (kerion) may develop when infection is from animal to man

Pruritus usually leads to excoriation of lesions and secondary bacterial infection

Hypersensitivity to the presence of the fungal elements may occur at distant sites ("Id" reaction) ***Tinea barbae***:

Ringworm of the beard is not a common disease

Occurs chiefly among those in agricultural pursuits, especially those in contact with farm animals

Lesions present as severe, deep folliculitis with erythema, nodular infiltrates, scales and pustules Marked regional lymphadenopathy is the rule Tinea faciei:

Fungal infection of the face (apart from the

beard) - Frequently misdiagnosed, since the typical ringworm not commonly seen on the face

Erythematous, slightly scaling, indistinct borders are usually seen

People who use corticosteroids such as cosmetic bleaching creams are prone to *T. faciei*

The steroid effect makes the lesions atypical hence, *T. incognito*

Tinea corporis:

One or more circular, sharply circumscribed, slightly erythematous, dry, scaly patches

Lesions may be slightly elevated, particularly at the borders, where they are more inflammed and scaly than at the central parts

Progressive central clearing produces annular outlines that give them the name "ringworm" In the presence of immune suppression from underlying illness, or chronic use of topical steroid creams lesions may be very extensive and atypical in appearance (Tinea incognito)

Tinea cruris:

Occurs more commonly in adult men

Leads to severe itching in the groins (crotch)

Presents as slowly spreading erythematous patches with scaly borders on the upper inner

aspects of the thighs

Treatment objectives

To clear lesions and prevent recurrence

Drug treatment

TopicalKetoconazole

- 2% cream apply twice daily

Miconzole

- 2% cream apply twice daily

Systemic

Fluconazole

Adult: 50 mg orally daily for 2 - 4 weeks; up to 6 weeks in tinea pedis

Child: 1 month - 18 years 3 mg/kg (maximum 50 mg) daily for 2 - 4 weeks; up to 6 weeks in tinia pedis

Notable adverse drug reactions

Fluconazole: numerous drug interactions

Hepatotoxicity during long-term daily therapy

Prevention

Do not share combs, hair brushes, school caps, shoes, socks or underwears

Keep the feet dry; avoid tight-fitting covered shoes

Aerate the feet as often as possible

Use good antiseptic powder on the feet after bathing e.g. Tolnaftate 1% powder

Reduce perspiration and enhance evaporation from the crural areas by wearing loose pants (e.g. boxer pants) made of absorbent cotton fabric

Apply plain talcum powder or antifungal powders in the flexures e.g. armpits, under the breasts, in the groins

Avoid exposure to animals with ringworm *(M. canis)* especially cats, dogs and (less commonly), horses and cattle

Excessive perspiration is the most common predisposing factor in adult T. corporis

- Avoid excessively hot, humid environments, or take a cold shower after sweating

PITYRIASIS VERSICOLOR (Tinea versicolor)

Introduction

Superficial yeast infection of the skin caused by *Malassezia furfur* species (normal commensals on the skin)

Common in warm humid climates

Predisposing factors:

Occlusion of the skin with pomades and greases

Immune suppression

Hyperhidrosis

Heat

Clinical features

Usually asymptomatic (or just mild itching)

May be generalized in the immunocompromised

Fine scaly, guttate or nummular patches, particularly on young adults who perspire freely

Individual patches are dirty, yellowish/brownish/hypopigmented macules (hence the term versicolor)

Larger irregular patches may evolve

Sometimes follicular tendency is marked; more noticeable at the advancing edges of the irregular patches

Sites of predilection:

- Sternal region
- Sides of the chest
- Shoulders
- Upper back
- Face

Differential diagnoses

Seborrhoeic dermatitis

Pityriasis alba

Pityriasis rosea

Leprosy

Complications

None usually; only of cosmetic significance

M. furfur sepsis

- From contamination of the lipid-containing medium in immunocompromised patients

receivinghyperalimentation through tubes

Investigations
Skin scraping for KOH microscopy

Treatment objectives
Improve appearance of skin

Drug treatment
Topical:
Selenuim sulphide shampoo
- Apply on affected areas daily, leave on for 10 – 30 minutes and wash off
- Continue for 3 weeks

Ketoconazole shampoo
- Use as above

Miconazole cream
- For limited areas
- Apply twice daily for 3 weeks

Supportive measures
Deal with underlying predisposing factor(s)

Prevention
Avoid hot, humid environments or clothings that promote perspiration
Take a cold shower after perspiration
Use any of the above shampoo washes once a month if predisposed

HERPES ZOSTER

Introduction

A second infection with varicella-zoster virus (VZV), usually in adults and limited to a dermatome Synonyms:
Zoster, from the Greek "zostrix", meaning belt
Shingles, from the Latin "cingulus", also meaning belt

Clinical features

Vesicles arranged in one or more dermatomes unilaterally
Initial pruritus, pain and paraesthesia
Multidermatomal and disseminated forms may occur in immuno-compromised states especially HIV infection
The early rash is vesicular, later becomes pustular and then ulcerates
The whole episode may last 2 weeks

Differential diagnosis

Chicken pox

Complications

Pain may persist long after rash has healed (post-herpetic neuralgia)
Dissemination of infection in the immunocompromised
Hemorrhagic and necrotic lesions

Ramsay-Hunt syndrome (Herpes zoster of the ear resulting in severe ear pain, hearing loss and vertigo)
Visual impairment due to corneal ulcers (Zoster ophthalmicus-V1)

Investigations
HIV screening for all patients
Full Blood Count with differentials
ESR
Exclude Hodgkin's disease and leukaemia
Treatment objectives
Provide symptomatic relief
Treat secondary infection
Treat any identified predisposing factor

Drug treatment
Drying agents e.g. zinc oxide 5% (calamine) lotion
- Apply twice daily

Aciclovir
Adult: 800 mg orally five times daily for 5 - 7 days
- Continue for at least 3 days after complete healing

Child: 12 - 18 years: 5 mg/kg orally every 8 hours usually for 5 days

Or:
Aciclovir cream 5%
Adult apply five times daily for 5 - 10 days

Child: not listed for this indication in children
Oral antibiotics to treat or prevent secondary bacterial infection

Herpetic neuralgia
Amitriptyline
10 - 25 mg orally initially, gradually increased to 75 mg daily

Or
Pregabalin
Start with 75mg bid or tid, gradually increase dose depending on efficacy and tolerabilty to a maximum of 600mg per day in divided doses

Notable adverse drug reactions, caution
Aciclovir
- Ensure adequate hydration
- Caution in pregnancy and breastfeeding
- May cause nausea, vomiting, dizziness
- Fatigue pruritus and photosensitivity

Pregabalin
- Hypersensitivity reactions
- Ataxia, dizziness
- Suicidal tendencies
- Blurred vision
- Muscle spasms
- Peripheral edema

MOLLUSCUM CONTAGIOSUM

Introduction

A common infection caused by a large epidermotropic pox virus

Common in children

Spread by direct human to human contact

In adults it is often transmitted during sexual intercourse

Clinical features

Individual lesions are smooth-surfaced, firm, dome-shaped, pearly papules; average diameter 3 - 5 mm

Some "giant" lesions may be up to 1.5 cm in diameter

Characteristic central umbilication

Spontaneous resolution is expected

Host response plays an important role

Children with widespread molluscum contagiosum usually have atopic dermatitis

Consider HIV in adults

Differential diagnoses

Viral warts

Giant molluscum contagiosum may mimic basal cell epithelioma

Complications

Secondary bacterial infection

Investigations

Histopathology of the expressed pasty core

Treatment objectives
Eradicate the skin lesions

Non-drug treatment
Light electrosurgery with a fine needle
Cryotherapy with trichloroacetic acid 35% - 100%
Curettage and paint with iodine

Drug treatment
Cimetidine
Adult: 40 mg/kg/day orally for 2 months
Child: not licensed for use in children less than 1 year. 1 month - 12 years: 5 - 10 mg/kg (maximum 400 mg) 4 times daily 12 - 18 years: 400 mg orally 4 times daily
Antibiotics - To prevent or treat secondary infection

Prevention
Avoid direct skin contact with an infected person

VARICELLA (Chickenpox)
Introduction
Varicella Zoster virus is Human Herpes Virus 3
Transmission is by direct contact with the lesions and by the respiratory route

Initial replication occurs in the nasopharynx and conjunctivae

After the primary infection, the virus remains dormant in nervous tissue

- Reactivation later in life is typically manifested as Herpes zoster

Clinical features

Incubation period is 10 - 21 days

Vesicular eruptions consist of delicate "teardrop" vesicles on an erythematous base

The eruption starts with faint macules that develop rapidly into vesicles within 24 hours

Successive fresh crops of vesicles appear for a few days, mainly on the trunk, face, and oral mucosa

New lesions usually stop appearing by the fifth day; the majority is crusted by the sixth day

- Most disappear in less than 20 days without a scar, except larger and secondarily infected lesions

Low grade fever

Malaise

Headaches

The severity of the disease is age-dependent

- Adults have more severe disease and a greater risk of visceral disease

Differential diagnoses

Variola minor

Disseminated zoster in immunosuppressed patients
Widespread papular urticaria
Coxsackie and ECHO viruses eruption

Complications
Secondary bacterial infection
Pneumonia
Cerebellar ataxia and encephalitis
Reye's syndrome

Investigations
Tzanck smear
Direct fluorescent antibody (DFA) staining
Polymerase Chain Reaction (PCR)

Treatment objectives
Relieve itching and treat secondary bacterial infection
Reduce severity and scarring

Drug treatment
Aciclovir
Adult: 10 mg/kg intravenously three times daily for 7 days in immunocompromised patients
Child: see Herpes zoster
Antihistamine for pruritus
Co-trimoxazole or erythromycin for secondary infection

Notable adverse drug reactions, caution

Aciclovir

- Ensure adequate hydration
- Caution in pregnancy and breastfeeding
- May cause nausea, vomiting, dizziness, fatigue pruritus and photosensitivity

Prevention

Isolate patients from non-immune persons

VIRAL WARTS (Verrucae)

Introduction

Infections caused by human papilloma viruses (HPV); include more than 80 types

Transferred between humans, or from animals to humans

Cause cutaneous tumours which tend to regress spontaneously but may rarely progress into cutaneous malignancies

Clinical features

Infection may be clinical, subclinical, or latent Clinical lesions are visible by gross inspection Subclinical lesions may be seen only by aided examination (e.g. the use of acetic acid soaking) Latent infection:

- HPV virus or viral genome is present in apparently normal skin
- Thought to be common, especially in genital warts, and explains in part the failure of destructive methods to eradicate warts

Incubation period is highly variable; from weeks to years

Auto-inoculation is the rule

Lesions may also occur on scratches (Koebner phenomenon)

Lesions are classified according to their positions and shape:

Common warts

Firm growths with rough surface; round or irregular, greyish or brown

Generally appear on areas that are frequently injured, such as the fingers, around the nails (periungual warts); knees, face and scalp

Plantar warts

Develop on the soles of the feet, where they are usually flattened by the pressure of walking

- A reactive callus forms around lesions

Multiple warts may coalesce, resembling a tile or mosaic floor (mosaic warts)

May be extremely tender

Unlike corns and calluses, plantar warts tend to bleed from many tiny spots, like pinpoints when pared down with a blade

Filiform warts

Long, thin, small growths that usually crop up on the eyelids, face, neck, or lips

People who chronically use corticosteroids

as cosmetic bleaching creams are prone to multiple filiform warts

Plane warts

More common in children and young adult.

Usually appear in groups as smooth, yellow-brown, small, flat papules; most frequently on the face

Genital warts

Occur most often on warm, moist surfaces of the body In men, usual sites are the end and shaft of the penis, and below the foreskin (if uncircumcised)

In women, lesions occur on the vulva, vaginal wall, cervix, and skin surrounding the vaginal area May develop in the perianal region or rectum

- Especially in homosexual men, and in women who engage in anal sex

Usually appear 1 - 6 months after infection as soft

erythematous papules, which may be greyish if hyperkeratotic

New lesions develop rapidly and all coalesce, producing a cauliflower-like picture

May grow rapidly in pregnant women, and immunocompromised patients

Differential diagnoses

Common warts

Keratoacanthoma
Squamous cell carcinoma
Seborrhoeic keratosis
Hypertrophic lichen planus
Tuberculosis
verrucosa cutis
Palmoplantar keratoderma
Arsenical keratoses
Plane warts
Epidermodysplasia verruciformis
Syringomas
Dermatosis papulosa nigra
Lichen planus
Lichen nitidus
Genital warts
Condyloma lata
Pemphigus vegetans

Complications
Squamous cell carcinoma of the perianal skin
Cervical carcinoma from anogenital warts
Obstructive laryngeal papillomatosis in babies infected through maternal birth canal

Investigations
Histopathology if in doubt

Management
Treatment depends on their location, type, and severity, as well as duration of lesions

Treatment objectives

Eradicate the skin lesions

Prevent complications

Non-drug treatment

Liquid nitrogen freeze

Electro-desiccation

Laser surgery

Drug treatment

Salicylic acid with lactic acid plaster

- Apply carefully to wart; rub wart surface gently with file or pumice stone once weekly
- May need to treat for as long as 3 months

Podophyllum resin

- Apply weekly under supervision e.g. in genitourinary clinic

Imiquimod 5% cream

- Apply thinly once daily on 3 alternate days per week until lesions resolve (maximum 16 weeks)

Notable adverse drug reactions, caution and contraindications

Salicylic acid plaster

- Avoid broken skin
- Not suitable for anogenital region or large areas
- Podophyllum
- Avoid normal skin and open wounds

- Keep away from face
- Should not stay on treated skin for more than 6 hours before washing

Prevention

Women with genital HPV infection should have routine cervical cytologic screening

- Pappanicolaou (PAP) smear to detect cervical dysplasia

MISCELLANEOUS DISORDERS

ACNE VULGARIS (Pimples)

Introduction

One of the most common skin diseases

A disorder of the pilosebaceous follicles

Typically first appears during puberty when androgenic stimulation triggers excessive production of sebum

Many factors interact to produce acne in a given patient

- Genetics
- Sebum production
- Hormones
- Bacteria
- Properties of the sebaceous follicle
- Immunologic

Over-production of stratum corneum cells (hyperkeratosis) obstructs the hair follicles at the follicular mouth producing open comedones, or blackheads

Just beneath the follicular opening in the neck of the sebaceous follicle it causes microcomedones (closed comedones, or whiteheads)

There is an overgrowth of gram-positive bacteria in the obstructed follicle: *Propionibacterium acnes*or *Staphylococcus epidermidis;* distally *Pityrosporum ovale*

Rupture of the comedonal contents into the dermis induces a foreign body reaction and inflammation

Clinical features

Almost every individual has some degree of acne during puberty, with spontaneous resolution occurring in early adult life

Occasionally, the disease persists into the fourth decade, or even remains a life-long problem

Favoured sites are the face, upper back and upper chest and shoulders

There may be mild soreness, pain, or itching

May present differently in different age groups

- Pre-teens often present with comedones as their first lesions
- Teenage acne is invariably inflammatory and the lesions include firm red papules, pustules, abscesses, indurated nodules, cysts and rarely interconnecting draining sinus tracts

Inflammatory acne can be classified as mild, moderate, or severe

Mild acne:

- Few-to-several inflammatory papules and pustules, but no nodules

Moderate acne:

- Several-to-many papules, pustules, and a few to several nodules

Severe acne (acne conglobata):

- Numerous fistulated comedones; extensive inflammatory papules; pustules; many cysts, abscesses, nodules, and draining sinuses
- The lesions may be generalized, involving even the buttocks
- Excoriation of acne papules and microcomedones are common, and scarring may result
- Usually, multiple shallow erosions or crusts are found

Differential diagnoses

Acne rosacea
Dermatosis papulosa nigra
Steatocystoma multiplex
Syringoma
Trichoepithelioma
Warts
Angiofibromas of tuberous sclerosis
Molluscum contagiosum
Steroid acne from the use of systemic

steroids or topical fluorinated steroids on the face (often as cosmetic skin lightening creams)

Some drugs may produce acneiform eruptions

- Androgens
- Adrenocorticotropic hormone (ACTH)
- Glucocorticoids
- Hydantoins
- Isoniazid
- Halogens

Complications

Psychosocial problems from cosmetic disfigurement

Post-inflammatory pigmentary changes

Pitted scars Keloids

Acne fulminans (acute febrile ulcerative acne conglobata with polyarthritis and leukemoid reaction)

Investigations

Usually, none required

In the presence of unusual acne, hirsutism, premature pubarche, or androgenic alopecia (especially when associated with obesity and/or menstrual irregularities):

Screen for hyperandrogenism

Blood levels of free testosterone, dehydroepiandrosterone, and androstenedione

- If raised, test response of the hormones and cortisol to dexamethasone suppression

Treatment objectives
Reduce severity of acne
Prevent complications

Drug treatment
Comedonal acne
Topical treatment only:
Tretinoin cream
Adult: 0.025% **or** 0.05% **or** 0.1% cream or gel applied nightly
Child: apply thinly 1 - 2 times daily

Or:
Benzoyl peroxide
Adult: 2.5% **or** 5% water-based or alcohol-based gels, applied twice daily
Child 12-18 years: apply 1 - 2 times daily preferably after washing with soap and water
- Start with lower strength preparations
- Infantile acne:

Child 1 month to 2 years; neonate: apply 1 - 2 times daily
- Start with lower strength preparations

Or:
Salicylic acid solution 2%
Adult and child: apply up to 3 times daily
- Tretinoin may be used at night and

benzoyl peroxide or topical antibiotics in the morning because they have different modes of action and are complementary

- It may take 8 - 12 weeks before observable improvement occurs

Mild inflammatory acne

Treat as above

Moderate inflammatory acne

Topical and systemic drugs:

Tetracycline

Adult and child over 12 years: 500 mg orally every 12 hours

Or:

Doxycycline

Adult and child over 12 years: 100 mg orally every 12 hours

Or:

Erythromycin

*Adult and child over 12 years:*500mg - 1 g every 12 hours

Infants requiring oral therapy: 250 mg once daily or 125 mg every 12 hours

Or:

Clarithromycin 250 - 500 mg orally every 12 hours

- In patients who do not tolerate any of the tetracyclines or who fail to improve

Review patient in 6 weeks and 3 - 4 months later

- If there is marked improvement, taper the

dose by 250 mg for tetracycline every 6 - 8 weeks while treating with topicals to arrive at the lowest systemic dose needed to maintain clearing

Antibiotic-resistant acne

Spironolactone may be added as an antiandrogen

Adult: 50 - 200 mg orally daily

Severe acne

Start with systemic antibiotics as above

Oral isotretinoin (13-cis retinoic acid)

Adult: 0.5 - 1 mg/kg/day for 20 weeks for a cumulative dose of at least 120 mg/kg

Child 12 - 18 years: 500 micrograms/kg once daily, increased if necessary to 1 mg/kg in 1 - 2 divided doses

- Occasionally, acne does not respond or promptly recurs after therapy, but may clear after a second course
- At least a 4-month rest period from the drug is recommended before a second treatment course is considered

Acne fulminans

Prednisolone 1.0 mg/kg daily for 7 - 10 days then taper off rapidly as isotretinoin is started

Success has been reported with dapsone but only in toxic doses (100 mg three or four times daily)

Adjuvant measures

Use non-irritating cleansing agents to reduce

facial sheen and bacterial flora
Emotional support
Comedone extraction
Intralesional injection for deeper papules and occasional cysts
Dilute suspensions of triamcinolone acetonide
- 2.5 mg/mL or 0.05 mL per lesion
Laser, dermabrasion for cosmetic improvement of scars

Notable adverse drug reactions, caution and contraindications

Topical preparations:
Creams and water-based gels are less irritating than alcohol/acetone-based gels
- Always initiate treatment with lower strength and increase as tolerance develops to initial irritant reaction
- Occasionally contact sensitivity may occur

Benzoyl peroxide
- May bleach fabrics, hair and skin
- Avoid contact with eyes, mouth, and mucous membranes

Antibiotic resistance may occur
- Avoid the use of different oral and topical antibiotics at the same time
- Vaginitis and perianal itching due to Candida may occur
- Tetracyclines, and doxycycline are

contraindicated in pregnancy and in children less than 12 years

- May reduce the effectiveness of oral contraceptives
- Often cause GIT symptoms
- doxycycline may cause photodermatitis
- Erythromycin cannot be used in conjunction with astemizole or terfenadine, as serious cardiovascular complications may occur

Salicylic acid

- Significant absorption may occur from the skin in children

Isotretinoin:

Dry skin, lips and eyes
Decreased night vision
Epistaxis
Hypercholesterolaemia
Hypertriglyceridaemia
Pseudotumour cerebri and headaches
Depression
Musculoskeletal or bowel symptoms
Thinning of hair
Bony hyperosteoses

Premature epiphyseal closure in children

- Absolutely contraindicated during pregnancy (teratogenicity)
- Obtain informed consent before use; start oral contraceptives one month before commencing therapy and continue for another month after conclusion of

therapy
- Women of childbearing age are strongly advised to avoid pregnancy for up to 3 years following cessation of therapy
- Check cholesterol and triglyceride levels every 2 - 4 weeks while on therapy
- Dapsone at such high doses is likely to cause methhaemoglobinemia
- Where leprosy is still endemic (e.g. Nigeria), reserve for treatment of leprosy

Prevention

Avoid
- Oil-based cosmetics, hair styling mousse, face creams and hair sprays
- Medicines that may induce acne

PRURITUS

Introduction

Commonly known as itching

The most common unpleasant experience involving the skin; provokes a desire to scratch

May be elicited by many normally occurring stimuli e.g.
- Light touch
- Temperature change
- Emotional stress
- Chemical, mechanical, thermal and electrical stimuli

Mediated by the release of chemical substances e.g. histamine, kinins, and

proteases

- Prostaglandin E lowers the threshold for histamine-induced pruritus, while enkephalins, pentapeptides which bind to opiate receptors in the brain modulate pain and itching centrally

Clinical features

At a low level, may merely be annoying

May actually torture the patient, interfere with sleep and lead to less than optimal performance

There are great variations from person to person

- In the same person there may be variation in reactions to the same stimuli

In the elderly, senile pruritus due to dry skin may be particularly bothersome

Psychologic trauma, stress, absence of distractions, anxiety, and fear may all enhance itching

Tends to be most severe at the time of undressing for bed

There are also regional variations

- The ear canals, eyelids, nostrils, and perianal and genital areas are especially susceptble to pruritus

May be localized or generalized

May or may not be associated with skin lesions

Excoriations are typically linear and occur

where the patient can reach with his hands

- The middle of the back is typically spared except when the patient has used a back scratcher
- The scratch is usually erythematous, with many tiny erosions scattered along it

Fresh marks are usually weepy or bloody; older ones crusted

- Lesions may become impetiginized

In addition to excoriations, some patients may have smooth, shiny fingernails (the polished nails of chronic pruritus)

Pruritus without skin lesions suggests

- Biliary obstruction
- Diabetes mellitus
- Uraemia
- Lymphoma
- Hyperthyroidism
- Adverse reaction to medicines e.g. Histamine liberators, opioids
- Occult scabies
- Pediculosis
- Onchodermatitis
- Dermatitis herpetiformis
- Atopic eczema in remission
- HIV/AIDS
- Systemic mastocytosis

Polycythaemia vera is a notable cause of pruritus; usually induced by temperature changes

Some patients complain of pruritus provoked

by bath or immediately post-bath
Factors include:

- Aquagenic pruritus
- Temperature-dependent pruritus due to cold/heat
- Cholinergic pruritus (when the core temperature is increased and there is sweating)
- Allergy to bath sponge or soap
- Mechanical scrubbing of the skin with coarse sponge causing degranulation of mast cells
- A forceful jet of water from the shower may trigger pruritus in some cases.

Differential diagnoses
All the above causes of pruritus

Complications
Sleep disturbance
Less than optimal performance at home, work or school
Emotional disturbance
Suicidal ideation

Investigations
As suggested by meticulous history and physical examination
Treatment objectives
Suppress itch

Identify and treat cause(s)
Improve quality of life
Prevent complications

Drug treatment
Ketotifen
Adult: 2 mg orally taken before bath (with food)
Child 3 years and over: 1 mg orally twice daily
Depressed, itchy individuals
Doxepin
*Adult*initially 75 mg orally daily in divided doses or as a single dose at bedtime
- Increased if necessary to a maximum of 300 mg daily in 3 divided doses

Up to 100 mg may be given as a single dose
Elderly: initially 10 - 50 mg daily; range of 30 - 50 mg daily may be adequate
Not recommended for children
Pruritus associated with partial biliary obstruction and primary biliary cirrhosis
Colestyramine
Adult: 4 - 8 g orally daily in water (or other suitable liquid)
Child 1 month - 1 year: 1 g orally once daily mixed with water; 1 - 6 years: 2 g once daily; 6 - 12 years: 4 g once daily; 12 - 18 years: 4 - 8 g daily, adjusted according to response in all age groups
Pruritus of renal failure
Activated charcoal

Adult: 50 g orally initially then 50 g every 4 hours.

- Treat vomiting with an anti-emetic because it may reduce the efficacy of charcoal treatment

In cases of intolerance reduce the dose and increase frequency of administration (e.g. 25 g every 2 hours or12.5 g every hour). This may however compromise efficacy

Or:

Ultra Violet B therapy

Localized pruritus

Corticosteroid creams for inflammatory skin disease

Or:

Crotamiton cream 10%

Adult: apply topically 2 - 3 times daily

Child: apply once daily for child below 3 years; over 3 years: apply 2 - 3 times daily

Or:

Urea 10% hydrocortisone cream 1 %, *Adult and* child: dilute with aqueous cream in first 1 week of use if stinging occurs

Or:

Emulsifying ointment BP

Adult and child: can be used as soap substitute; rub on skin before rinsing off completely

Or:

- Doxepin hydrochloride

Adult: apply thirly 3 - 4 times daily (coverage should be less than 10% body surface area)

Adverse drug reactions, caution and contraindications

Colestyramine: Counsel patients

Other drugs should be taken at least 1 hour before, or 4 -6 hours after colestyramine to reduce possible interference with absorption

May cause constipation and gastrointestinal discomfort

Interferes with the absorption of fat-soluble vitamins

- Supplements of vitamins A, D and K may be required

Activated charcoal:

Risk of aspiration in drowsy or comatose patients

Risk of intestinal obstruction in patients with reduced gastro-intestinal motility

Black stools

Doxepin:

Caution in patients with glaucoma, urinary retention, and severe liver impairment

May cause drowsiness, local burning, stinging, irritation and dry mouth

Prevention

Use a cleansing bar (instead of soap) for baths
Pat rather than rub skin dry after bath and immediately lubricate skin with petroleum jelly or emulsifying ointment

URTICARIA AND ANGIOEDEMA

Introduction

An eruption of evanescent wheals or hives which can result from many different stimuli on an immunologic or non-immunologic basis

The most common immunologic mechanism is hypersensitivity mediated by IgE - Another mechanism involves activation of the complement cascade.

The activation of cutaneous mast cells and their release of mediators is the unifying feature of most urticaria Mast cells are found in the immediate vicinity of blood vessels

- They release preformed mediators (histamine, heparin and various enzymes) as well newly manufactured ones (prostaglandins, leukotrienes)

A hive or urticarial lesion is the result of localized oedema in the dermis

Causes:

Medications

Food

Aero-allergens

Latex; seminal fluid (contact urticaria)

Insect antigens (bees, wasps or hornet toxins)

Infections and infestations (parasitic, fungal, Bacterial and viral)

Foreign proteins (antisera, vaccinations)

Physical stimuli (pressure, heat, cold, cholinergic stimuli, water, light and irradiations)

Auto-immune disorders, enzyme defects (C1 esterase inihibitor deficiency)

Psychosocial conflicts (stress, depression)

Excessive mast cells (mastocytoma, urticaria pigmentosa)

Pseudoallergy (mast cell degranulators e.g. NSAIDS; dyes, preservatives, contact urticaria)

Serum sickness

Malignancies

Idiopathic

Clinical features

May be acute or chronic:

Acute urticaria is of sudden onset and lasts less than 6 weeks

Chronic urticaria persists for more than 6 weeks with either:

- Daily emergence of new wheals (chronic continuous) or
- Occasional hive-free periods (chronic recurrent)

The typical urticarial reaction is similar to the triple response of Lewis

- Initial erythema
- Next oedema (the hive)
- Finally an erythematous ring surrounding the hive

Urticarial lesions may:

- Vary in size and shape over minutes to hours
- Present an orange-skin appearance
- Become bullous

The pruritus associated with urticaria is usually extreme

Excoriations are extremely unusual because the lesions are almost invariably rubbed, not scratched

Dermographism is characterized by wheal and erythema after minor stroking of, or pressure on the skin

- Commonly found under pressure areas e.g. the belt line

May persist for years, but spontaneous regression usually occurs within 2 years

Angioedema is the involvement of deeper vessels

- Characterized by painless, deep, subcutaneous swelling
- Often involves periorbital, circumoral and facial regions; palms, soles and the genitalia
- May target the gastrointestinal and respiratory tracts, causing abdominal pain, coryza, asthma and respiratory problems
- Respiratory tract involvement may cause

airway obstruction
- Anaphylaxis and hypotension may also occur

Differential diagnoses

Gyrate erythemas
Urticarial vasculitis
Mastocytosis
Pityriasis rosea (early lesions)
Bullous lesions:
Pemphygus
Pemphygoid
Erythema multiforme
Fixed drug eruption
Angioedema:
"Calabar swelling"
Cellulitis
Idiopathic scrotal oedema of children
Melkerson-Rosenthal syndrome
Cold uriticaria:
Cryoglobulinemia Immune complex diseases
Systemic lupus erythematosus and other collagen vascular diseases
Macroglobulinemia
Mycoplasma infections (cold hemagglutinins)
Syphilis
Familial cold urticaria
Acquired cold urticaria

Complications

Emotional distress in chronic cases
Fatality

Investigations
Suggested by meticulous history and physical examination

Treatment objectives
To alleviate symptoms
Eliminate and treat cause

Drug treatment
Chlorphenamine maleate
Adult: 4 mg orally every 4 - 6 hours (maximum 24 mg daily)
Child: under 1 year, not recommended 1-2years:1mg every 12 hours; 2-5years :1mg every4-6 hours (maximum 6 mg daily); 6 - 12 years: 2 mg every 4 -6hours (maximum12mgdaily)
- If less sedation is required (e.g. day time)

Or:
Loratadine
Adult and Child over 6 years: 10 mg orally daily
*Child*2-5years5mgdaily
If persistent and chronic urticaria
Add Doxepin (oral form discontinued)
Adult: apply thinly 3 - 4 times daily; usual maximum 3g per application (total daily maximum 12 g)

Child: not recommended for children under 12 years

Or:

(For symptomatic dermographism and chronic urticaria)

Add:

Ranitidine hydrochloride *Adult:* 150 mg orally every 12 hours or 300 mg at night

- Not to be used alone for the treatment of urticaria

Refractory cases

Systemic corticosteroids

- Prednisolone 0.5 to 1.0 mg/kg orally daily

Adjuvant measures

To relieve itching: Tepid or cold tub baths or showers

Add starch, or sodium bicarbonate, menthol, or magnesium sulfate to bath water

Do not scrub the body with sponge (it promotes degranulation of cutaneous mast cells)

Avoid medicines likely to cause urticaria/angioedema

Eliminate any suspected food

Counselling

Notable adverse drug reactions, caution and

contraindications

Chlorphenamine maleate:

Patients not to drive or operate machinery

Ranitidine:

Tachycardia, agitation, visual disturbances, alopecia, gynaecomastia and impotence

Caution in hepatic impairment, pregnancy and in breast feeding

loratadine:

Headache, dry mouth, drowsiness, dizziness and nausea

Caution in the elderly especially if renal function is compromised

Doxepin:

Caution in cardiac disease

Contraindicated in recent myocardial infarction, arrhythmias, glaucoma and severe liver disease

May cause dry mouth, sedation, blurred vision, constipation, nausea, difficulty with micturition

Prevention

Eliminate/avoid any identified/possible causal factor(s)

VITILIGO

Introduction

A disease characterized by acquired loss of melanocytes, leading to areas of depigmentation

Sometimes associated with uveitis and other autoimmune phenomena

- Many autoantibodies can be demonstrated in vitiligo patients; those against melanocytes may rarely be demonstrable

There is also a neural hypothesis

- Vitiliginous patches often follow a dermatome
- A neurochemical mediator responsible for destroying the melanocytes has therefore been suggested

There is also an occupational vitiligo

- Due to chemically induced depigmentation
- Seen among workers who are in contact with para-phenolic compounds or hydroquinones (but this is considered a different disorder)

Clinical features

All ages are affected

The dermatomal type is more common in the paediatric age

The completely depigmented patches have distinct borders

- A few patients may have inflammatory vitiligo with raised erythematous borders
- Some may have hypopigmented skin between the depigmented and normal skin (trichrome vitiligo)

The distribution may be:

Generalized (autoimmune type)
Segmental (dermatomal type)

The hairs on the patches eventually turn white (acquired poliosis)

The generalized type may be symmetrically distributed in the extremities

- Generalized vitiligo continues to spread while new lesions develop for years

Spontaneous repigmentation may occur
Favoured sites are

- Extensor surfaces of the extremities
- Face and peri-orificial surfaces (around the mouth, eyes, nipples, umbilicus, penis, vulva, and anus)

Focal vitiligo may affect one non-dermatomal site e.g. lips, vulva or penis

Universal vitiligo applies to cases where the entire body surface is depigmented

Generalized vitiligo may be associated with:

- Hyperthyroidism
- Hypothyroidism
- Pernicious anaemia
- Diabetes mellitus
- Addison's disease

Local loss of pigment may occur around a naevus and melanomas, the so-called halo phenomenon

Vitiligo-like leucoderma occurs in about 1% of melanoma patients

- Usually a good prognostic sign since it suggests an effective immune reaction

against the tumour cells

Segmental vitiligo affects only one part of the body

- It spreads rapidly in that area and then stabilizes
- It is not associated ith autoimmune diseases
- Favoured sites are the trigeminal area or an intercostal nerve distribution (zosteriform pattern)

Just as with albinism, the interplay between the melanocytes of the eyes, ears, and skin is apparent The prototype is Vogt-Koyanagi-Harada syndrome:

Vitiligo of the face, eyelashes, and scalp hair in association with

- Uveitis
- Dysacoussis
- Alopecia areata

Chemical vitiligo affects sites of contact with the chemicals

- When the chemicals are inhaled or a substantial quantity is absorbed through the skin, the distribution of the white patches may simulate the generalized autoimmune type

Differential diagnoses

Post-burns depigmentation

Tertiary stage of pinta Morphoea

Lichen sclerosis

Pityriasis alba
Tinea versicolor
Piebaldism Hypomelanosis of Ito

Complications
Emotional problems due to cosmetic disfigurement

Investigations
Exclude other autoimmune diseases if clinically suggestive
See also notes on caution below

Treatment objectives
Re-pigmentation
Improve cosmetic appearance
Emotional support

Topical
Corticosteroids
- Hydrocortisone 1% or betamethasone valerate

Adult: 0.1% apply once or every 12 hours (for focal or limited lesions)
Child: apply 1 - 2 times daily Psoralens
- 8-methoxypsoralen (MOP)

0.05% - 0.1% in combination with ultraviolet-A radiation (PUVA) for focal or limited lesions
Adult and child: apply twice weekly
Tacrolimus 0.1% ointment twice daily for 24

weeks

Systemic

Systemic 8-methoxypsoralen

Adult: 0.5 mg/kg orally

The initial UVA dose is 1 or 2 J/cm2, gradually increased

Two or three treatments are done per week for 3 – 6 months

Systemic corticosteroids

- May occasionally be used to arrest the autoimmune process
- Prednisolone tablets 0.5 - 1.0 mg/kg orally day

Surgical

Pigmented skin grafted onto vitiliginous patches

- Often the transferred melanocytes repigment the depigmented areas

The various techniques include:

- Suction blister grafts
- Mini-punch grafts
- Transfer of either pure melanocyte cultures or mixed epidermal cultures to a prepared site

Adjuvant measures

Camouflage (cover-up cosmetics)

Patient education and emotional support

Notable adverse drug reactions, caution and contraindications

Corticosteroids:

See Dermatitis and Eczema

8-MOP:

Inadvertent sunburns with blistering

Systemic psoralen is contraindicated in:

- Known photosensitivity
- Porphyria
- Liver disease
- Systemic lupus erythematosus

If systemic therapy is to be used the following should be done before therapy

- Ophthalmological examination
- Full Blood Count
- Liver function tests
- Antinuclear Antibody Test

Acquired ochronosis

PUVA therapy should be supervised by an experienced dermatologist

Prevention

Unknown

CHAPTER 8:

EAR, NOSE AND THROAT

ACUTE OTITIS MEDIA

Introduction

Acute inflammation of the middle ear due to pyogenic organisms

Usually secondary to upper respiratory infection spreading from nasopharynx

Common in infants and young children; more frequent during winter and rainy periods

Usual organisms are streptococcus pneumococcus and staphylococcus

Clinical features

Main symptoms:

Earache

Fever

Deafness

Ear discharge

Malaise

In babies, irritability

Clinically increasing inflammation and redness of the eardrum

Later, perforation and pulsating mucopurulent discharge

Differential diagnoses
Acute otitis externa
Referred otalgia

Complications
Acute mastoiditis
Facial nerve paralysis
Labyrinthitis
Intracranial
- Meningitis
- Brain abscesses
- Lateral sinus thrombosis

Investigations
Ear swab for culture and sensitivity- swab taken properly without contamination
Full Blood Count

Treatment objectives
Control infection
Restore normal hearing

Non-drug treatment
Ear toilet and antiseptic dressings
Myringotomy for persistent mucopurulent collection in middle ear with bulging eardrum

Drug treatment
Antibiotics

- Amoxicillin

Adult: 500 mg -1 g orally every 8 hours for 5 - 7 days

Child: 40 mg/kg orally every 8 hours

Analgesics

- Paracetamol

Adult: 500 mg - 1 g orally every 4 - 6 hours (to a maximum of 4 g) for 5 - 7 days

Child over 50 kg: same as adult dosing

6 - 12 years: 250 -500 mg; 1 - 5 years: 125 - 250 mg; 3 months - 1 year: 125 - 250 mg for 5 - 7days

Systemic decongestant

- Psuedoephedrine

Adult: 60 mg orally every 4 - 6 hours (up to 4 times daily)

Child:6-12years: 30mg (5mL of syrup) 3 times daily; 2-5years:15mg,(2.5mL of syrup)

Supportive measures

Bed rest and adequate fluids

Notable adverse drug reactions, caution

Many preparations of pseudoephedrine contain antihistamines and may cause drowsiness

Avoid ear drops

Prevention

Good general health and clean airy environment to reduce incidence of upper

respiratory infections (colds)

ADENOID DISEASE

Introduction

A manifestation of hyperplasia/hypertrophy of the adenoid tissue in the nasopharynx

Usually occurs in children aged 2 - 6 years

Excessively large adenoids may cause obstruction of the nasopharyngeal airway with symptoms of nasal obstruction

Large adenoids may encroach on the Eustachian tube openings causing secretory otitis media with deafness in the child

Chronic infection of adenoid tissue is also often present

Symptoms usually subside spontaneously as adenoids regress physiologically and become atrophic with age

Clinical features

Nasal obstruction and mouth-breathing

Snoring at night

Obstructive sleep apnoea

Progressive deafness due to secretory otitis media

Differential diagnoses

Allergic rhinitis

Sinusitis

Otitis media

Complications
Sinusitis
Recurrent otitis media
Pneumonitis

Investigations
X-ray of nasopharynx
Xray sinuses and chest

Treatment objectives
To significantly improve nasopharyngeal airway and thereby improve nasal breathing
Treat concurrent infection
Non-drug treatment
Adenoidectomy in severe cases

Drug treatment
Decongestants
- Psuedoephedrine syrup

6-12 years: 30mg (5mL of syrup) orally every 8 hours; 2-5years2.5mL

Or:
- Ephedrine nasal drops (0.5%)

Instil into nostrils twice daily and at night time
Antibiotic
- Amoxicillin syrup 125 - 250 mg orally every 8 hours for 5-7days.

CHRONIC OTITIS MEDIA

Introduction

A chronic inflammatory condition of the middle ear mucosa with recurrent ear discharge

- Often over a period of years

Occurs in two clinical varieties

- The more common simple type with a central eardrum perforation
- The much less common, serious type often associated with the presence of cholesteatoma

Bacteriology is usually mixed, mostly gram negative organisms (Proteus, Pseudomonas)

Clinical features

Main complaints: recurrent ear discharge and increasing deafness

Pain is uncommon

Discharge is mucoid in the simple type but thick and foul-smelling in the serious variety

Usually central eardrum perforation is of varying size

- Cholesteatoma and marginal or attic perforation is seen in the serious type

Complications

Generally more with the serious type:

Intracranial suppuration

- Extradural abscess

- Meningitis
- Brain abscess

Lateral sinus thrombosis
Facial nerve paralysis
Labyrinthitis

Investigations
Ear swab taken properly for microscopy, culture and sensitivity
Audiogram: conductive deafness
X-ray of the mastoids: shows sclerosis, hypopneumatization

Treatment objectives
To give the patient a safe and dry ear
To preserve or restore hearing as much as possible

Non-drug treatment
Careful ear toilet and regular ear dressing with antiseptic pack
With dry ear, persistent perforation may be closed surgically (myringoplasty) to protect middle ear and improve hearing
In the serious type with cholesteatoma not responding to treatment, mastoid operation is done to clear out disease and prevent complications

Drug treatment
Antibiotic - Co-amoxiclav

Adult: 500/125 mg orally every 8 hours for acute exacerbations up to 14 days
Child: 6 - 12 years: 250 mg orally every 12 hours; under 6 years: 125 mg every12 hours
If infection does not settle with systemic antibiotics refer to specialist

Supportive measures

Protect ears from water with Vaseline/cotton wool while bathing

Caution

Topical treatment with ototoxic antibiotics is contraindicated in the presence of a perforation

EPISTAXIS

Introduction

A condition of bleeding from the nose
A clinical presentation rather than a disease entity on its own

Bleeding is most often from ruptured vessels in the anterior nasal septum, sometimes from the posterior nose especially in the elderly

Can arise from a wide variety of causes
Local (in the nose)
Trauma
Inflammation of nose or sinuses
- Acute e.g. acute rhinitis/sinusitis
- Chronic e.g. tuberculosis, leprosy,

Neoplasms

Manifestation of systemic diseases

Bleeding diatheses

Blood dyscrasias

Hypertension

Clinical features

Bleeding from nose; often spontaneous but may follow obvious trauma or injury

Varying amounts of blood, from few drops to torrential life-threatening haemorrhage

Often intermittent; most bleeds stop spontaneously

Differential diagnoses

Various pathological conditions, both local and systemic present with nasal bleeding

Complications

Haemorrhagic shock

Fatality

Investigations

Full Blood Count, including platelet count

Bleeding and clotting time; partial thromboplastin time

Urea and Electrolytes and Creatinine

X-ray sinuses

CT scan

Treatment objectives

To arrest bleeding in actively bleeding cases
Replace significant blood losses and treat shock
Identify and treat aetiological factors

Non-drug treatment

Pressure and compression of the nose between fingers to arrest bleeding

Cotton wool pack soaked in epinephrine 1:1000 may be placed on bleeding area before compression to induce vasoconstriction

Nasal packing with lubricated ribbon gauze

Arrest of posterior bleed with rubber tampon or improvised Foley's catheter balloon

Cauterization of bleeding point or dilated vessels in anterior nasal septum

- Diathermy cautery (electrical) or chemical cautery with silver nitrate stick

Drug treatment

Treat underlying aetiologies
Sedation if necessary

- Diazepam 5 mg orally twice daily for 1 - 2 days
- Antibiotics if infection is present
- Amoxicillin

Adult: 500 mg orally every 8 hours for 5 - 7 days
Child: 250 - 500 mg orally for 5 - 7 days
Other drugs depending on identified

causative factors

Supportive measures

Intravenous infusion, crystalloids and blood as necessary

Bed rest

Prevention

Avoid/treat predisposing conditions

FOREIGN BODIES IN THE AIRWAYS

Introduction

Children (most commonly) may aspirate pieces of play objects or food items accidentally into the airway

May present as serious emergencies with imminent asphyxia

The object if arrested at laryngeal level causes acute upper respiratory obstruction

Sharp objects such as fish bone or pins may be impacted on the vocal cord and the resulting oedema causes progressive obstruction

Small objects such as seeds may traverse the larynx and become arrested in the trachea or bronchus lower down

Vegetables such as peanuts often cause severe reaction in the lungs with pneumonitis

Clinical features

Difficulty in breathing with stridor occurs

immediately or progressively

Initial dyspnoea and cough may subside if the object passes down.

Symptoms gradually return later

Severe cases: stridor and severe cyanosis with imminent asphyxia requiring immediate intervention to prevent a fatal outcome

Two-way stridor often occurs with tracheal foreign bodies

In the lower airways objects may remain for long periods, with unexplained chest symptoms

Differential diagnoses

Acute laryngitis
Acute laryngeal oedema
Bronchopneumonia
Pulmonary tuberculosis

Complications

Life-threatening asphyxia
Lung collapse and atelectasis

Investigations

Radiograph of neck and chest

Treatment objectives

To maintain the airway and adequate respiratory function

Remove the foreign object as expeditiously

as possible

Non-drug treatment

Immediate removal under anaesthesia by direct laryngoscopy or bronchoscopy as appropriate Tracheostomy where necessary to maintain airway

Drug treatment

Antibiotic prophylaxis if necessary (for 3 days)

- Amoxicillin

Child: 6-12 years: 250mg orally every 12 hours; under 6 years: 125 mg orally every 12 hours

Steroid

- Hydrocortisone (for pneumonitis)

Child 1 month - 1 year: initially 25 mg by intravenous or intramuscular injection every 8 hours; 1 - 6 years: initially 50 mg every 8 hours; 6 - 12 years: initially 100 mg every 8 hours; 12 - 18 years: initially 100 - 500 mg 3 times daily, adjusted in all age groups according to response

Supportive measures

Oxygen

Steam inhalation/nebulizer

Prevention

Vigilant supervision of young children

FOREIGN BODY IMPACTION

Introduction

Various objects ingested and stuck in pharynx or upper oesophagus , mostly accidentally. In children play objects, toy pieces, coins, safety pins ; fish bones, poorly chewed meat, dentures mainly in adults. Sharp objects may be arrested in the oropharynx, most others at the cricopharyngeal sphincter.

Clinical features

Dysphagia, drooling of saliva, cough at times.

Complication:

Perforation of oesophagus, mediastinitis, aspiration pneumonitis

Investigation

Radiogragh of neck and chest

Treatment objectives:

To remove foreign body expeditiously

Non-drug treatment

Removal under local anaesthetic if object visualised in oropharynx

Removal under general anaesthetic with oesophagoscopy if object is in hypopharynx or cervical oesophagus

Drug treatment

Antibiotic prophylaxis if indicated
Amoxicillin.

FOREIGN BODIES IN THE EAR

Introduction

A common presentation in ENT emergency practice

Children usually involved as they insert various objects into ears while playing: beads, plastic toys, seeds, etc

Live insects may also crawl into the ear in adults/children

Clinical features

Symptoms are often absent

Little pain (sometimes)

Sensation of blockage may be reported by older children

Object usually seen with good light in the ear canal

Differential diagnoses

Impacted wax

Otitis externa

Complications

Otitis externa

Perforation of tympanic membrane from attempts at removal

Treatment objectives

Remove object expeditiously without damage to ear structures or causing undue pain to patient

Non-drug treatment

Removal by ear syringing

Removal with appropriate hook, or alligator forceps

Examination and removal under anaesthesia if difficult in the clinic

Prevention

Vigilant supervision of young children

FOREIGN BODIES IN THE NOSE AND RHINOLITHS

Introduction

Children often insert various objects into the nostrils while playing: pieces of plastic toys, rolled paper, foam, seeds, some metal objects, etc

The objects may remain undetected for long periods, particularly organic items, until they become infected

Typically result in foul smelling unilateral nasal discharge

Some inorganic objects may (after long periods) become coated by hard calcific deposits and become known as rhinoliths

Clinical features

Often no indication or symptom

May be accidentally noticed by parent

Later, complaints of foul purulent unilateral nasal discharge of unknown origin

Differential diagnoses

Acute or chronic rhinitis Sinusitis

Nasal growth/polyp

Complication

Secondary infection: rhinosinusitis

Investigation

Radiograph of nose: for metallic or radio-opaque objects

Treatment objectives

Remove object safely with little discomfort to patient

Non-drug treatment

Careful removal with appropriate hook or forceps

Removal under anaesthesia as necessary

Prevention

Vigilant supervision of young children

MASTOIDITIS

Introduction

Develops as a complication of acute suppurative otitis media, mostly in children

Follows acute otitis media (untreated or inadequately treated), or due to particularly virulent organisms

Infection spreads from the tympanum posteriorly into the mastoid antrum and aircells

Colliquative necrosis of the air cells and suppuration in the mastoid bone follows

A subperiosteal abscess forms behind the ear in a child with a discharging ear

Clinical features

Fever

Pain behind the ear

Mucopurulent ear discharge

Progressive inflammatory swelling over the mastoid region

Swelling is tender and fluctuant

Differential diagnosis

Suppurating post-aural lymphadenitis from otitis externa

Complications

Spread of infection into cranial cavity with:

Extradural abscess

Meningitis

Brain abscess
Lateral sinus thrombophlebitis

Investigations

Ear swab for microscopy, culture, culture and sensitivity
Radiographs of the mastoid

Treatment objectives

Control and eradicate infection
Prevent more serious complications

Non-drug treatment

Cortical mastoidectomy to open the mastoid
- Exenterate the infected air cells and drain the mastoid

Drug treatment

Large doses of parenteral antibiotics
- Amoxicillin

Adult: 500 mg -1 g intravenously every 6 - 8 hours for 7 days
Child: 50 - 100 mg/kg intravenously every 6 - 8 hours in divided doses daily for 7 days
- Ceftriaxone

Adult: 1 g every 12 hours intravenously for 7 days
Child: by intravenous infusion over 60 minutes Neonates: 20 -50 mg/kg once daily, by deep intramuscular injection, intravenous injection over 2 - 4 minutes, or by intravenous infusion 1 month - 12 years (body weight

under 50 kg) 50 mg/kg once daily, up to 80 mg/kg in severe infections

Analgesics

- Paracetamol

Adult: 500 mg -1 g orally every 4 - 6 hours (to a maximum of 4 g) for 5 - 7 days

Child over 50 kg: same as adult dosing 6-12years:250-500mg;3months-5years:125-250 mg taken orally every 4 - 6 hours for 5 - 7 days

Supportive measures

Bed rest: in-patient care

Intravenous infusion as appropriate

Prevention

Adequate and timely treatment of acute otitis media

NASAL ALLERGY

Introduction

Hypersensitivity of the nasal mucosa to various foreign substances, of the atopic type

Manifests as recurrent episodes of sneezing, rhinorrhoea and nasal obstruction whenever patient comes in contact with the offending allergen

Symptoms are attributed to the effect of histamine and other chemical substances released from ruptured mast cells in the nasal mucosa

Common allergens are pollens of various

plants, flowers and trees; house-dust; hairs; some foods; fungi and cosmetics
A common condition and affects all age groups
May be familial, often associated with allergic asthma or dermatitis

Clinical features
Repeated episodes of sneezing
Watery nasal discharge
Nasal obstruction with itching and conjunctival irritation whenever patient is in contact with allergen
Nasal mucosa may be congested or sometimes normal at the time of clinical examination
Presentation may be seasonal as with pollen allergy, or perennial with allergy to house dust, etc
Nasal polyps may develop
Differential diagnoses
Chronic rhinitis from other causes
Vasomotor rhinitis
Chronic sinusitis

Complications
Chronic sinusitis
Pharyngitis

Investigations
Skin tests for allergens: intradermal or prick

tests

Smear of nasal secretions for eosinophilia

Serological tests: radio-immunoassay for IgE antibodies

Sinus X-ray

Treatment objectives

Control or suppress the allergic symptoms

Prevent allergic reactions

Non-drug treatment

Elimination of allergens

Hyposensitisation by vaccination

Drug treatment

Antihistamines

- Chlorphenamine

Adult: 4 mg orally every 4 - 6 hours; maximum 24 mg daily

Child: not recommended under 1 year 6 - 12 years: 2 mg orally every 4 - 6 hours; maximum 12 mg daily; 2 - 5 years: 1 mg every 4 - 6 hours; maximum 6 mg daily

Or:

- Promethazine

Adult: 25 mg orally at night, increased to 25 mg twice daily if necessary or, 10 - 20 mg every 8 - 12 hours

*Child: not*recommended under 2 years 5-10years: 10-25mg orally daily in 1-2 divided

doses; 2-5years:5-15mg daily in 1-2 divided doses

Topical steroid

Non-sedative antihistamines - Loratidine

Adult and child over 12 years 10mg once daily; Child 2-12 years, body weight under 30kg, 5mg/5ml once daily; body weight over 30 kg, 10 mg once daily

- Beclomethasone nasal spray

Adult and child over 6 years: 100 micrograms (i.e. 2 sprays) into each nostril twice daily

- Or 50 micrograms into each nostril every 8 hours
- Reduce dose to 50 micrograms into each nostril twice daily when symptoms are controlled

Decongestant

- Psuedoephedrine

Adult: 60 mg orally 4 - 6 hourly (up to 4 times daily)

Child: 6 - 12 years: 30 mg (5 mL of syrup) orally every 8 hours; 2 - 5 years: 2.5 mL

Notable adverse drug reactions, caution

Drowsiness with antihistamine drugs

Avoid prolonged use of medications

Prevention

Avoid known allergenic substances, inhalants, foods, etc

OTITIS EXTERNA

Introduction

Inflammation of the external ear

May be:

Infective: bacteria or fungi

Reaction of the canal skin to chemical irritant(s)

Part of a generalized dermatitis

Localised otitis externa or furuncle (boil) is a Staphylococcal infection of a hair follicle in the canal

Diffuse otitis externa may be bacterial or fungal or reactive

- May be acute or chronic

Bacterial infection often follows trauma from scratching the canal skin

Fungal otitis (otomycosis) commonly follows swimming in the tropics, usually infection by *Aspergillus niger*

Clinical features

Pain and itching

Ear discharge

Sensation of blockage due to accumulated debris in canal

Deafness is variable

Canal is red and swollen, full of inflammatory debris

- In otomycosis whitish mass of debris with black spots

Differential diagnoses
Otitis media
Acute mastoiditis

Complications
Acute perichondritis

Investigations
Ear swab, taken properly for microscopy, culture and sensitivity
Urinalysis for glycosuria
Blood glucose estimation in cases of recurrent furunculosis to exclude diabetes mellitus

Treatment objectives
Control infection/inflammation
Relieve discomfort

Non-drug treatment
Careful ear toilet to clear out debris
Daily dressing with antiseptic gauze packed with Acriflavin in spirit
Furunculosis: dressing with magnesium sulfate wick or steroid and antibiotic ointment dressing

Drug treatment
Antibiotics
- Amoxicillin

Adult: 500 mg -1 g orally every 8 hours for 5 - 7 days
Child: 40 mg/kg orally in every 8 hours for 5 - 7 days

- Neomycin/hydrocortisone ear drops

Adult and child: instil 2 - 3 drops 3 - 4 times daily

Analgesics

- Paracetamol

*Adult:500*mg -1 g orally every 4 - 6 hours (to a maximum of 4 g) for 5 - 7 days

Child over 50 kg: same as adult dosing

6 - 12 years: 250 - 500 mg; 3 months - 5 years: 125 – 250 mg taken orally every 4 - 6 hours

Supportive measures

Prevent water from entering ear for one month

Prevention

Avoid trauma to ear canal (especially scratching)

Keep ears dry

PERITONSILLAR ABSCESS (Quinsy)

Introduction

The main common local complication of acute tonsillitis

A virulent streptococcal infection; may spread beyond the tonsillar capsule into the peri-tonsillar space, causing, first cellulitis,

and later suppuration in the space
More common in adults with tonsillitis

Clinical features
Follows an attack of acute tonsillitis
Increasing pain, fever and dysphagia
Trismus- spread of oedema and infection to pterygoid muscles
Often referred pain to ipsilateral ear
Difficulty in opening mouth for examination; mouth full of saliva
Affected tonsil displaced downwards and medially, with swelling above and lateral to it, all inflamed and oedematous
Uvula pushed to opposite side

Differential diagnoses
Parapharyngeal abscess
Retropharyngeal abscess
Tonsillar tumours

Complications
Septicaemia
Parapharyngeal suppuration/abscess

Investigations
Throat swab
Full Blood Count with differentials

Treatment objectives
Rapid control of infection

Relief of pain and discomfort

Non-drug treatment
Improves
Incision and drainage, preferably under local anaesthetic when suppuration is definite

Drug treatment
Antibiotics
- Amoxicillin

Adult: 500 mg -1 g intravenously every 6 hours for 7 days
Child: 50 - 100 mg/kg orally every 8 hours
Analgesics
250
- Paracetamol

Adult: 500 mg - 1 g orally every 4 - 6 hours (to a maximum of 4 g) for 5 - 7 days
Child over 50 kg: same as adult dosing
6 - 12 years: 250 - 500 mg; 3 months - 5 years: 125 – 250 mg taken orally 4 - 6 hourly for 5 - 7 days

Or:
- Aspirin (Acetysalicylic acid)

Adult: 300 - 900 mg orally every 4 - 6 hours when necessary; maximum 4 g
Not recommended in children (risk of Reye's syndrome)

Supportive measures

Intravenous infusion
Bed rest

Notable adverse drug reactions
Aspirin may cause gastrointestinal irritation

Prevention
Elective tonsillectomy is advised after an episode of quinsy to prevent further (more severe) attacks

PHARYNGITIS (Sore Throat)
Introduction
A common cause of persistent sore throat in young and most middle-aged adults, usually unaccompanied by other symptoms

Often secondary to chronic nasal conditions with nasal obstruction e.g.
- Vasomotor rhinitis
- Nasal polyps
- Septal deviation

Obstruction causes mouth breathing with dryness of the throat
Other causes:
Secondary inflammation from postnasal discharge of sinusitis

Chronic exposure to irritants such as tobacco smoke and alcohol
Secondary infection from carious teeth

Clinical features

Persistent sore throat with no systemic upset or dysphagia
Sore throat is often worse in the mornings

Differential diagnoses
Chronic tonsillitis
Pharyngeal or laryngeal tumour

Complications
More often related to the primary sources of irritation or infection

Investigations
Throat swab: microscopy, culture and sensitivity
X-ray of paranasal sinuses

Treatment objectives
Control symptoms by identifying and treating primary causes

Non-drug treatment
Treat sinusitis
Surgery for obstructive nasal conditions
Treat dental caries

Drug treatment
Appropriate antibacterial agent if indicated

Supportive measures
Reduction or avoidance of exposure to

known irritants-tobacco, alcohol, etc

SINUSITIS

Introduction

Inflammation of the mucosal lining of the paranasal sinuses

May be acute or chronic and affect one or more of the sinuses

- Most commonly the maxillary sinus or antrum (in very young children the ethmoidal sinuses)

Acute sinusitis is often sequel to acute rhinitis

- Common organisms are streptococcus, pneumococcus, and haemophilus Chronic sinusitis is more insidious
- May be associated with chronic rhinitis and allergy but other factors such as air pollution, smoking, dental sepsis and poor general health may be contributory

Bacteriology is mixed: sometimes Gram negative and fungal organisms

Clinical features

Rhinorrhoea

Nasal obstruction

Fever with pain over affected sinus in acute cases

Less dramatic symptoms in chronic sinusitis

- Intermittent nasal obstruction and discharge over a long period
- Little pain

- Mucopurulent postnasal discharge ("drip")

Differential diagnoses
Acute rhinitis (coryza)
Allergic rhinitis
Vasomotor rhinitis

Complications
Orbital cellulitis (complicating ethmoidal sinusitis)
Cavernous sinus thrombosis (sphenoidal sinusitis)
Intracranial infection
- Subdural abscess
- Meningitis
- Cerebral abscess
- Dural vein thrombophlebitis

Osteomyelitis of frontal or maxillary bones
Chronic pharyngotonsillitis
Chronic laryngitis and bronchitis

Investigations
Nasal swab for microscopy, culture and sensitivity
X-ray of sinuses: four-view
Antrum roof puncture/lavage: specimen for culture
CT scan in complicated cases

Treatment objectives

Control and eradicate infection
Restore adequate drainage of sinuses

Non-drug treatment
Antrum wash-out/lavage
Trephining of frontal sinus
Radical surgery for non-responsive cases
- Intranasal antrostomy
- Caldwell-Luc operation
- Fronto-ethmoidectomy

Functional Endoscopic Sinus Surgery (FESS)

Drug treatment
Antibiotics
- Amoxicillin

*Adult:500*mg - 1 g orally every 8 hours for 5 - 7 days
*Child:40*mg/kg orally every 8 hours for 5 - 7 days

Or:
Amoxicillin/clavulanic acid
Adult:500/125 mg orally every 12 hours
Child:0.25 mL/kg of 125/31 mg suspension orally every
8 hours; dose doubled in severe infections
1-6years: 5mL of 250/62 mg suspension every 8 hours; dose doubled in severe infections
6 - 12 years: 5 mL of 250/62 mg suspension every 8 hours; dose doubled in severe infections 12 - 18 years: one 250/125 mg

strength tablet every 8 hours, daily increased in severe infection to one 500/125 strength tablet every 8 hours daily

Or:

Cotrimoxazole

*Adult:960*mg orally every 12 hours

Child 6 weeks to 5 months: 120 mg orally every 12 hours; 6 months - 5 years: 240 mg every 12 hours; 6 - 12 years: 480 mg every 12 hours

- Ceftriaxone

Adult: 1 g intravenously or intramuscularly every 12 hours for 7 days for patients with severe or nosocomial disease

Child: by intravenous infusion over 60 minutes Neonates: 20 - 50 mg/kg once daily, by deep intramuscular injection, intravenous injection over 2 - 4 minutes, or by intravenous infusion 1 month - 12 years (body weight under 50 kg) 50 once daily, up to 80 mg/kg in severe infections Decongestant

- Psuedoephedrine tablets

Adult: 60 mg orally twice daily until congestion

Child 2-6 years: 15 mg orally 3 - 4 times daily; 6 - 12 years: 30 mg 3 - 4 times daily; 12 - 18 years: 60 mg 3 - 4 times daily Analgesic

- Paracetamol

Adult: 500 mg -1 g orally every 4 - 6 hours (to a maximum

of 4 g) for 5 - 7 days

Child over 50 kg: same as adult dosing
6 - 12 years: 250 - 500 mg; 3 months - 5 years: 125 - mg taken orally every 4 - 6 hours for 5 - 7 days

Supportive measures

Steam inhalations with menthol

Treat contributory nasal pathology as appropriate

- Allergy, nasal polyps, septal deviations, dental pathology, etc

Notable adverse drug reactions

Amoxicillin

- Minor gastrointestinal disturbance

Cotrimoxazole

- Fixed drug eruption
- Nausea and vomiting
- Erythema multiforme
- Steven-Johnson syndrome

Prevention

Avoid airway irritants, smoking, and alcohol

Avoid air pollution

Maintain good general health and nutrition

TONSILLITIS

Introduction

An inflammatory condition of the palatine tonsils, common in children

In half or more cases infection is by beta-haemolytic streptococcus, in others viral

Typically an acute infection

Chronic tonsillitis presents usually as recurrent acute infection

Essentially a disease of children but also occurs in young adults

Clinical features

Fever
Sore throat
Dysphagia
Systemic upset and malaise
Tonsils are swollen, inflamed and covered with purulent exudates
Jugulo-digastric lymph nodes are enlarged and tender

Differential diagnoses

Infectious mononucleosis
Vincent's angina
Agranulocytosis

Complications

Quinsy: main common complication
Parapharyngeal infection/abscess
Rheumatic fever and nephritis following streptococcal tonsillitis

Investigations

Throat swab for microscopy, culture and sensitivity
Full Blood Count

Treatment objectives
Control the infection
Control pain
Prevent further episodes

Non-drug treatment
Oral hydration
Salt/warm water gargle
Tonsillectomy in chronic cases with frequent recurrent tonsillitis
Drug treatment
Antibiotics
- Amoxicillin
Adult: 250 - 500 mg orally every 8 hours for 5 - 7 days
Child: 40 mg/kg orally every 8 hours for 5 - 7 days
The parenteral route may be required when there is vomiting or severe dysphagia

Or:
- Cotrimoxazole
Adult: 960 mg orally every 12 hours 5 - 7 days
Child 6 weeks to 5 months: 120 mg orally every 12 hours; 6 months - 5 years: 240 mg every 12 hours; 6 – 12 years: 480 mg every 12 hours
Analgesic
- Paracetamol
Adult: 500 mg -1 g orally every 4 - 6 hours (to a maximum of 4 g) for 5 - 7 days

Child over 50 kg: same as adult dosing
6-12years:250-500mg; 3months-5years:125-250
mg taken orally every 4 - 6 hours for 5 - 7 days

Supportive measures
Bed rest
Intravenous infusion as necessary

Notable adverse drug reactions
Cotrimoxazole
- Fixed drug eruption
- Nausea and vomiting
- Erythema multiforme
- Steven-Johnson syndrome

TRACHEOSTOMY

Introduction
A surgical procedure in which an opening is created into the trachea from the outside, commonly to bypass an upper respiratory obstruction

May also be done to provide easier access for care of the chest in some seriously ill patients
- Also for respiratory support and artificial ventilation in patients with respiratory insufficiency or paralysis

Most cases are done to by-pass upper airway obstruction:
- Acute infections of the larynx
- Trauma

- Foreign body aspiration
- Acute laryngeal oedema
- Vocal cord paralysis
- Tumours

Some cases are done as part of, or to facilitate major head and neck surgery

An appropriate-sized tracheostomy tube, portex or metal, is inserted to maintain the opening

Clinical features

Acute presentation with clinical features of airway obstruction, stridor and incipient asphyxia following trauma

Acute inflammatory conditions of the larynx, which would require the operation as an emergency

Progressive lesions: may require less urgent intervention in anticipation of likely obstruction

Cases with medical indications requiring respiratory support are usually done on a more elective basis

Complications

Haemorrhage

Infection: wound and chest

Damage to nerves and large vessels in the neck

Treatment objectives

To secure the airway

Non-drug treatment

Postoperative care of tracheostomy preferably in an intensive care unit, with suction, humidification, stoma care as appropriate

Drug treatment

Broad spectrum antibiotic cover

WAX IN THE EAR

Introduction

Wax (or cerumen) is a normal product of the human external ear

- A dark brownish mixture of the secretions of the ceruminous and sebaceous glands in the outer third of the external auditory canal

Small quantities are produced continuously and function to lubricate the canal

Quantities produced and the consistencies vary

- May be excessive in some people, causing deafness, ear ache, secondary infection and even vertigo

Clinical features

Sensation of blockage and some degree of deafness are the most common complaints

Sometimes, pain and irritation

Ear discharge in some cases
Quantity seen varies
- May be soft or hard
- May be impacted in the deep meatus

Differential diagnoses
Foreign bodies
Otitis externa

Complications
Superimposed infection: otitis externa
Hearing impairment

Treatment objectives
Evacuate the wax and clear the ear
Non-drug treatment
Removal with probe and cotton wool: for soft wax
Ear syringing: for hard wax, often after preliminary softening with oily drops
Occasionally, removal under anaesthesia if syringing is unsuccessful

Drug treatment
Ear drops to soften and loosen wax
- Warm olive oil

CHAPTER 9:

ENDOCRINE SYSTEM DISORDERS

DIABETES MELLITUS

Introduction

Diabetes mellitus is a common metabolic disorder characterised by persistent hyperglycaemia, a consequence of defects in the action or secretion of insulin resulting in disturbances of metabolism of carbohydrates, fat and protein. It is associated with acute as well as long-term complications affecting the eyes, kidneys, feet, nerves, brain, heart and blood vessels

Its classification has been revised by the WHO and is based on aetiology:

Type 1:

- Results from destruction (usually autoimmune) of the pancreatic P cells
- Insulin is required for survival

Type 2:

- Characterized by insulin resistance and/or abnormal insulin secretion (either may predominate); both are usually present
- It is the most common type of diabetes

Other specific types of diabetes- less common, and include:

- Genetic disorders
- Infections
- Diseases of the exocrine pancreas
- Endocrinopathies
- Drugs

Gestational diabetes: appears for the first time in pregnancy

Clinical features

Type 1 diabetes:

Patients present at a young age (usually teens or twenties); earlier presentation may also occur

Rapid onset of severe symptoms: weight loss, thirst and polyuria

Blood glucose levels are high and ketones are often present in the urine

If treatment is delayed, ketoacidosis (DKA) and death may follow

The response to insulin therapy is dramatic and gratifying

Misclassification of patients as "Type 1" is relatively common

- Insulin-treatment is not the same as insulin-dependence

Type 2 diabetes:

Most patients present with the classical symptoms

including polyuria, polydipsia and polyphagia
Some patients present with sepsis, diabetic coma (hyperosmolar non-ketotic states)
A minority is asymptomatic and therefore identified at screening
The patients usually do not seek medical attention early because of the insidious nature of the disease
Many present at diagnosis with features of diabetic complications

- Visual difficulties from retinopathy
- Pain and/or tingling in the feet from neuropathy
- Foot ulcerations
- Stroke

Gestational diabetes (GDM):
Diabetes which arises in pregnancy
Must be distinguished from existing diabetes in women who become pregnant
Of particular importance because it is associated with poor pregnancy outcomes, especially if not recognised and not treated
Particular problems associated with GDM:
Foetal macrosomia Eclampsia
Intra-uterine growth retardation Birth difficulties Neonatal hypoglycaemia
Neonatal respiratory distress

Diagnosis

Straightforward in the majority of cases

May pose a problem for those with a minor degree of hyperglycaemia, and in asymptomatic subjects

- In these circumstances, two abnormal blood glucose results on separate occasions are needed to make the diagnosis
- If the results of point blood glucose testing are equivocal, an oral glucose tolerance test should be performed
- If diagnosis remains in doubt maintain surveillance with periodic re-testing until the diagnostic situation becomes clear

Take into consideration additional risk factors for diabetes before deciding on a diagnostic or therapeutic course of action

The diagnosis of diabetes must be confirmed biochemically prior to initiation of any therapy

Symptoms of hyperglycaemia

Plus:

Random venous plasma glucose 11.1 mmol/L

Or

Fasting venous plasma glucose ≥7.0 mmol/L

Or

HbA1c≧7%

Confirms the diagnosis of diabetes

In asymptomatic subjects, a single abnormal blood glucose result is inadequate to make a diagnosis of diabetes

- Abnormal values must be confirmed at the earliest possible date using any of the following
- Two separate fasting or random blood samples

Or

- A 75g oral glucose tolerance test

Glucose Tolerance State	Venous plasma (mmol/L)	Venous plasma (mg/dL)
Diabetes mellitus Fasting 2 hour post-75 g glucose load	> 7 ≥ 11.1	> 126 ≥ 200
Impaired glucose tolerance Fasting AND 2 hour post-75 g glucose load	< 7.0 - 7.8 and < 11.1	< 110 - 140and < 200
Impaired fasting glycaemia Fasting	6.1 - < 7.0	5.6 - < 6.1

Unless there is unequivocal hyperglycaemia with acute metabolic decompensation or obvious symptoms, the diagnosis of diabetes should always be confirmed by repeating the test on another day

Management

Goals:

Early diagnosis

Prevent and/or reduce short and long term morbidities

Prevent premature mortality

Improve quality of life and productivity of affected persons

Promote self care practices and empowerment of people with diabetes

Reduce the personal, family and societal burden of diabetes

Achievement of these goals is dependent on:

Successful establishment of diabetes health care team, and infrastructure to support it, including provision of education for health care professionals and for people living with diabetes

Core components of diabetes care

Treatment of hyperglycaemia

Treatment of co-morbidities

Prevention and treatment of macrovascular and microvascular complications

***Non-drug treatment* Education**

The provision of knowledge and skills to people with diabetes mellitus

- To empower them to render self-care in their management

Priciples of Diabetes EducationShould be locally applicable, simple and effective

All members of the diabetes care team should be trained to provide the education

It must empower people with diabetes as well as their families

- Provide them with adequate knowledge

of diabetes and its sequelae
- Create the right attitudes and provide resources to provide appropriate self care

The effectiveness of the programme must be evaluated and modified as necessary

What people with diabetes need to know
Diabetes is serious but can be controlled
Complications can be prevented
That the cornerstones of therapy are education, diet and exercise
Their metabolic and blood pressure targets
How to look after their feet and thus prevent ulcers and amputations
How to avoid other long term complications
That regular medical check ups are essential
When to seek medical help

Diet

- One of the cornerstones of diabetes management
- Based on the principle of healthy eating in the context of social, cultural and psychological influences on food choices
- Dietary modification (and increasing level of physical activity) should be the first step in the management of newly diagnosed persons with Type 2 diabetes
- Should be maintained throughout the course of diabetes management

Goals of dietary management of Type 2

diabetes mellitus

To achieve an ideal body weight:

- An appropriate diet should be prescribed along with an exercise regimen
- Caloric restrictions should be moderate and yet provide a balanced nutrition
- Eat at least three meals a day. Binge eating should be avoided
- As nack between meals can be healthy for certain groups of people
- The diet should be individualized, based on traditional eating patterns, be palatable and affordable
- Animal fat, salt, and so-called diabetic foods should be avoided
- Pure (simple sugars) in foods and drinks should be avoided
- Eating plans should be high in carbohydrates and fibre, vegetables and fruits should be encouraged
- Dietary instructions should be written out, *even if the person is illiterate:* someone at home should be available to interpret to him/her
- Food quantities should be measured in volumes using available household items (e.g. cups), or be countable (e.g. number of fruits or slices of yam or bread)
- Weighing scales are generally unaffordable and/or difficult to understand
- Appetite suppressants generally yield poor and/or unsustainable weight

reductions and are expensive

Physical activity

- One of the essentials in the prevention and management of Type 2 diabetes mellitus

Regular physical activity:
Improves metabolic control
Increases insulin sensitivity
Improves cardiovascular health
Helps weight loss
Gives a sense of well-being
Two main types of physical activity:

- Aerobic or endurance exercise e.g. walking, running
- Anaerobic or resistance exercise (e.g. lifting weights)

Both types of activity may be prescribed to persons with type 2 diabetes mellitus; the aerobic form is usually preferred

General principles and recommendations
Detailed evaluation

- Cardiovascular, renal, neurological and foot assessments
- Evaluation should be done before a formal exercise programme is commenced
- The presence of chronic complications excludes certain forms of exercises

Prescribed physical activity programmes should be appropriate for:

- The age
- Socio-economic status
- State of physical fitness
- Lifestyle
- Level of control

Exercise generally improves metabolic control, but can precipitate acute complications like hypoglycaemia and hyperglycaemia

Physical activity should :

- Be regular (about 3 days/week)
- Last at least 20 - 30 minutes per session
- Be at least of moderate activity

Activities like walking, climbing steps (instead of taking lifts) should be encouraged

For sedentary persons with diabetes, a gradual introduction using a low intensity activity like walking is mandatory

Avoid exercising if :

- Ambient glycaemia is > 250 mg/dL blood glucose
- Patient has ketonuria
- Blood glucose is less than 80 mg/dL

To avoid exercise-induced hypoglycaemia in patients on insulin

- Increase peri-exercise carbohydrate intake
- Reduce insulin dose
- Adjust injection site (avoid exercising muscles site) For persons with type 2 diabetes mellitus on long acting insulin secretagogues

- Extra carbohydrate should be taken before and after the exercise

In those on short acting secretagogues (e.g. glipizide, repaglinide) the post exercise dose should be omitted

Glycaemia should be monitored (using strips and meters) before and after planned physical activity

- Delayed hypoglycaemia may occur

Proper footwear must always be worn during exercise

For a prescribed formal activity, the exercise session should consist of:

- A warm-up period of 5-10minutes
- The activity proper: 20 - 60 minutes
- A cool-down period of 5-10minutes

In most parts of Africa, prescribing formal exercise in gyms or requiring special equipment is a recipe for non-adherence to the exercise regimen

Patients should be encouraged to integrate increased physical activity into their daily routine

- The programme should impose minimum (if any) extra financial outlay in new equipment and materials

Drug treatment

Oral antidiabetic agents:

- For Type 2 diabetes mellitus indicated :
- When individualized targets are not

met by the combination of dietary modifications and physical activity/exercise

- (In some cases) at the first presentation of diabetes (i.e. fasting blood glucose more than 11 mmol/L or random blood glucose more than 15 mmol/L)

May be used as monotherapy or in combination therapy, targeting different aspects in the pathogenesis of hyperglycaemia in Type 2 diabetes mellitus

- i.e. increasing insulin production and release, decreasing insulin resistance and/or decreasing hepatic glucose production

Category	Example	Mechanism of action	Daily dose	Side effects
Biguanide	Metformin	Improves insulin sensitivity in the liver, muscle and adipocytes	500-2500mg	Nausea, vomiting, abdominal pains
Sulphonylureas	Glibenclamide Climepiride	Stimulates insulin release from the beta cells	2.5-15mg 1 - 6mg	Hypoglycaemia Weight gain
Dipeptidyl peptidase IV inhibitors	Vildagliptin	Stimulate insulin release in a glucose dependent manner	50 - 100mg	Gastrointestinal upset Abnormal liver function tests
Insulin	Various formulations	Increase glucose absorption	Appropriate doses	Hypoglycaemia Lipodystrophy

Sulphonylureas

Initial monotherapy in non-obese patients

Add-on as combination therapy

- Indicated for Type 2 diabetes, maturity-onset diabetes of the young, under specialist care

Contraindications

Allergy to sulpha drugs
Liver impairment
Severe renal failure
Pregnancy
Age > 80 years

Biguanides

Indicated in:

Monotherapy in obese Type 2 diabetes mellitus
Combination therapy
Metabolic syndrome
Allergy to sulphonylureas

- Under specialist supervision ONLY
- Not licensed for use in children less than 10 years old

Important notes on Oral Glucose Lowering Agents (OGLAs)

Sulphonylureas and biguanides are the agents most widely available

- Stocking these agents would meet the diabetes care needs of most diabetes facilities

The choice of OGLAs should be informed by:

- Lifestyle
- Degree of control
- Access to medicines
- Economic status
- Mutual agreement between the doctor and the person with diabetes

Monotherapy with any of the drugs should be the initial choice

- Use of stepped-care approach is recommended

If overweight (BMI > 25 kg/m^2) or if insulin resistance is the major abnormality

- Metformin should be the first choice
- If metformin is contraindicated thiazolidinediones may be used

Avoid metformin and long acting sulphonylureas in elderly patients

- Instead, use short acting sulphonylureas

Combination therapy using OGLAs with different mechanisms of action is indicated if monotherapy with one of the agents has failed

When oral combination therapy fails, insulin should be added to the treatment regimen or should replace the OGLAs

Secondary failure of OGLAs is said to be common (5 -10% of patients annually) although no reports from Africa are available

Insulin Therapy in Type 2 Diabetes

Insulin is increasingly being used

- In combination with OGLAs or as monotherapy in the management of Type 2 diabetes to achieve optimum targets
- Hyperglycaemic emergencies
- Peri-operatively, especially major or emergency surgeries
- Organ failure: renal, liver, heart etc

- Pregnancy
- Latent Autoimmune Diabetes of Adults (LADA)
- Sensitivity to OGLAs

Regimen and dose of insulin therapy will vary from patient to patient

Two forms of insulin therapy are often used in combination with OGLA therapy

- Intermediate/long acting insulin plus OGLA **or** pre-mixed insulin

Referral to an endocrinologist should be considered if more than 30 units of insulin are required per day

Time Course of Action of Insulin Preparations

Insulin Preparation	Onset of Action	Peak Action	Duration of Action	Injections per day
Very rapid acting (insulin analogues)	10 min	1h	3h	Immediately before meals
Short-acting	30 min	2-5h	5-8h	30 min before meals
Intermediate-acting (NPH or lente)	2-3h	6-12h	14 - 24 h	Once or twice daily
Biphasic mixtures (30/70; premixed)	30 min	2-12h	16 - 24 h	Once or twice daily

Monitoring glycaemic control

Clinical and laboratory methods are employed

HbA1c tests are desirable standard tests but are unavailable in most of the primary and secondary healthfacilities in Africa

Fasting plasma glucose performed in the laboratory in place of HbA1c is the best alternative

- Its average for repeated measurements gives a reliable indication of the control

Glycosuria is a poor means of assessment of control

Self Blood Glucose Monitoring (SBGM) should be encouraged

Results of self urine testing or blood glucose tests should be recorded in a logbook

Clinic protocols should set out in some detail, the parameters to be monitored at the initial visits, at regular follow-up visits, and at annual reviews

At the initiation of insulin therapy, appropriate advice on SBGM and diet should be given

Treatment of co-morbidities

Examples are obesity, hypertension and dyslipidaemias

- See relevant chapters

Diabetic foot problems

Introduction

People with diabetes are at increased risk of foot ulcers and amputations which are major causes of morbidity and disability

Both foot ulcers and amputations can be prevented by education, anticipation, early recognition and prompt management

The most common predisposing factors for ulcers and amputations are:

Peripheral neuropathy with loss of sensation

Poor foot hygiene

Peripheral vascular disease
Deformities and abnormal biomechanics
Unsuitable or no footwear
Cornerstones of management
Regular inspection and examination of the foot at risk
Identify the at-risk foot
Education of healthworkers, people with diabetes and their families
Appropriate footwear
Early treatment of non-ulcerative and ulcerative foot problems

Diabetes in pregnancy

Introduction

Gestational diabetes mellitus (GDM) is any degree of glucose intolerance *first recognised* in pregnancy

If inadequately managed, GDM is associated with increased risk of perinatal morbidity and mortality

Diagnosis and prompt institution of therapy reduce the risks of poor outcomes

Screening for GDM

When:

- Between 24 and 28 weeks of gestation

Who: Women with

- High risk for GDM
- BMI 25 kg/m^2
- Previous history of GDM
- Glycosuria
- Previous large baby (> 4 kg)

- Poor obstetric history
- Family history of diabetes
- Known IGT / IFG

Management

Combined health care team- obstetrician, diabetologist, diabetes educator, and paediatrician/ neonatologist Initial therapy is dietary modification

- Spread carbohydrate over 3 small to moderate sized meals and 2 - 3 snacks/day
- Consider an evening snack to prevent starvation ketosis
- Energy intake should provide for desirable weight gain during pregnancy
- For obese women a 30 - 33% calorie restriction is advised

Daily SBGM (urine glucose monitoring) is not useful in pregnancy

Initiate insulin therapy if:

- Fasting plasma glucose is > 5.8 mmol/L
- 1 hour post-prandial glucose is > 8.6 mmol/L
- 2 hour post-prandial plasma glucose is >7.5 mmol/L

Modify insulin regimen to achieve above targets

Regular assessment of maternal wellbeing should include blood pressure and urine protein Regular surveillance for foetal well-being

Delivery at 38 weeks gestation recommended
Withdraw therapy for diabetes after birth
Re-assess classification of maternal status at 6 weeks post partum

Acute metabolic complications of diabetes mellitus(see emergency section)

Differential diagnoses
Stroke
Seizures
Trauma
Drug overdose
Ethanol intoxication

Prevention of diabetes

Generalised obesity, central obesity and physical inactivity are the major modifiable risk factors, and should be avoided/corrected

Onset of diabetes can be delayed in people at high risk by active lifestyle modification

- Lifestyle modification should be the cornerstone of preventative strategies in the following categories of people:

Age > 45years
Overweight and obesity (BMI > 25 kg/mm)
Physical inactivity
First degree relatives with diabetes
Previous gestational diabetes
Previously identified IGT or IFG
Dyslipidaemia

Hypertension

The components of lifestyle modification should include (but not be limited to) the following:

- Lose 5 - 10% weight
- Reduce fat intake (< 30% of total daily calories)
- Reduce saturated fat intake (< 10% of total daily calories)
- Increase fibre intake to >15 g/1000 kcal
- Traditional African diets are high in fibre content
- Increase levels of physical activity e.g. brisk walking producing a heart rate >150/min
- Exercise should last for at least 30 minutes and should be undertaken at least three times a week
- Reduce high alcohol intake

THYROID DISORDERS

THYROTOXICOSIS

Introduction

This refers to the clinical, physiological and biochemical manifestation of the effect of excess thyroid hormone on tissues as opposed to hyperthyroidism which simply means hyperfunctioning of the thyroid gland. There is a female preponderance due to the role of autoimmunity in the aetiology.

Actiology

Grave's disease (commonest cause)
Toxic nodule
Toxic multi-nodular goitre
Pituitary thyrotroph adenoma
Trophoblastic tumours producing HCG with thyrotrophic activity
Pituitary thyroid hormone resistance syndrome producing excess TSH and resultant excess T_3 and T_4
Thyroid cancers (rarely).

Thryroiditis (sub-acute or postpartum)
Iodine induced- drugs such as :
- Amiodarone
- Radiographic contrast media
- Iodine prophylaxis programmes
- Extra-thyroidal sources of thyroid hormone excess
- Factitious hyperthyroidism
- Struma ovarii TSH-induced:

Clinical features

A goitre may or may not be present
- May be diffuse or nodular

Heat intolerance
Dermatological:
Increased sweating and pruritus
Warm and moist skin
Pretibial myxoedema
Pigmentation, vitiligo

Palmar erythema
Onycholysis.
Hair loss
Cardiorespiratory:
Palpitation
Dyspnoea on exertion
Angina and cardiac failure
Increased pulse pressure (tachycardia)
Atrial fibrillation
Exacerbation of asthma
Gastrointestinal:
Weight loss despite increased appetite (hyperphagia)
Weight gain in 10% of patients
Increased stool frequency
Neuromuscular:
Tremors, nervousness, irritability, emotional liability and psychosis
Muscle weakness and proximal myopathy
Hyperkinesia
Hyperreflexia
Insomnia
Reproductive:
Loss of libido, impotence
Amenorrhoea/ oligomenorrhoea
Infertility and spontaneous abortions
Ocular:
Lid lag lid retraction
Grittiness, excessive lacrimation
Exophthalmos diplopia

Papilloedema
Others:
Increased thirst
Polyuria
Fatigue and apathy
Triad of Proptosis, Nail changes (onycholyis) and Skin changes) strongly suggest Graves disease

Differential diagnosis:
Active tuberculosis – weight loss, cough if in heart failure
Advanced retroviral disease –weight loss, hyperdefeacation, skin changes
Malnutrition-weight loss, proptosis
Cancer cachexia- weight loss
Malabsorption syndrome- weight loss
Neuropsychiatric disorders- tremors, pressure of speech and talkativeness even frank psychosis.
Poorly controlled Diabetes mellitus-weight loss, loss of libido, hyperphagia.

Complications
Hyperthyroid crisis (thyroid storm)
Compression of the trachea
Cardiac failure
Loss of visual acuity
Infertility
Periodic paralysis

Investigations:

Diagnostic Investigations:

Thyroid Function Test with free T_4 and T_3 which is both expected to be high and TSH suppressed.

Thyroid antibodies-

Thyroid Peroxidase Antibodies which is elevated in most cases of autoimmune thyroid disease,

Thyroglobulin antibody elevated in most cases of thyroid diseases,

TSH Receptor antibodies especially Thyroid Stimulating Immunoglobulin specifically in Grave's disease.

Radiolabelled thyroid scan with iodine 123 especially if diagnosis is not certain

ANCILLARY INVESTIGATIONS:

Full Blood Count with ESR- which may show anaemia; if megaloblastic –pernicious anaemia may coexist as an autoimmune disease. Elevated ESR.

Serum Electrolyte, urea and creatinine –electrolytes derangement

Fasting blood sugar which could be deranged

Electrocardiogram which may show sinus tachycardia, atrial fibrillation

Echocardiography may reveal evidence of congestive heart failure with poor ejection fraction, dilated heart chambers.

Treatment objectives

Achieve normal metabolic rates

Obtain normal serum T_3, T_4 and TSH Levels

Prevent complications

Drug treatment

Antithyroid drugs

- Carbimazole

Adult: starting dose 30 - 60 mg orally in divided doses daily

Maintenance: 10 - 15 mg oral daily

Child: neonate, initially 250 micrograms/kg orally every 8 hours until euthyroid then adjust as necessary 1 month -12 years: initially 250 micrograms/kg (maximum 10 mg every 8 hours) until euthyroid then adjusted as necessary

12 - 18 years: initially 10 mg every 8 hours until euthyroid then adjusted as necessary

- Higher initial doses occasionally required, particularly in thyrotoxic crisis

Child and carers to inform doctor immediately if sore throat, mouth ulcers, bruising, fever, malaise or nonspecific illness develops

Propylthiouracil (preferred in pregnancy)

Adult: starting dose 300 - 450 mg orally in divided doses daily

Maintenance: 100 - 150 mg orally in 2 or 3 divided doses daily

Child: neonate, initially 2.5 - 5 mg/kg orally every 12 hours until euthyroid, then adjusted

as necessary 1 month - 1 year: initially 2.5 mg/kg every 8 hours until euthyroid; 1 - 5 years: 20 mg/kg 8 hourly until euthyroid;
5- 12years: initially 50mg every 8hours until euthyroid;
12 - 18 years: initially 100 mg every 8 hours until euthyroid

- Higher doses occasionally required particularly in thyrotoxic crisis
- Duration of treatment usually is 18 - 24 months

B- adrenergic blocking drugs

- Propranolol 80 - 160 mg orally daily in divided doses
- Symptoms and signs of hyperthyroidism due to adrenergic stimulation may respond to these agents

Iodine used in:

- The emergency management of thyroid storm
- Thyrotoxic patients undergoing emergency surgery
- For the preoperative preparation of thyrotoxic patients selected for subtotal thyroidectomy

Aqueous iodide oral solution (Lugol's solution):

- Iodine 5%, potassium iodide 10% in purified water; total iodine 130 mg/mL

Adult: 2 - 3 drops of saturated potassium

iodide solution
orally 3 or 4 times daily (300 - 600 mg/ day)
Child: neonate 0.1 - 0.3 mL orally every 8 hours; 1 month
- 18 years: 0.1 - 0.3 mL every 8 hours
  Thyrotoxic crisis:

Child 1 month -1 year: 0.2 - 0.3 mL 8 hourly
- Dilute with milk or water

Use of steroids in autoimmune cases of thyrotoxicosis.

Radioactive sodium iodine
- Used in patients who are past child bearing age
- Dosage difficult to gauge; the response of the gland is unpredictable
- Up to 25% of patients given enough radioactive iodine to achieve euthyroidism, may develop hypothyroidism within one year
- High incidence of recurrence of hyperthyroidism if smaller doses are used

Surgery
Indications include:
Patients < 21 years who should not receive radio iodine
Persons who cannot tolerate other agents because of hypersensitivity, or for other reasons

Patients with very large goitres, having compressive symptoms or signs

Some patients with toxic adenoma and multinodular goitres

Supportive measures

Appropriate care of any system affected e.g. eye care, treatment of heart failure

Thyroid storm would require judicious intravenous fluid use, corticosteroids and treatment of the precipitating cause

Notable adverse drug reactions, caution and contraindications

Carbimazole and propylthiouracil

- May cause severe bone marrow suppression (including pancytopenia and agranulocytosis)
- They are contraindicated in breastfeeding mothers

HYPOTHYROIDISM (Myxoedema)

Introduction

Refers to subnormal amounts of thyroid hormones in the circulation, and the clinical features associated with this

Aetiology

May be primary or secondary

Primary hypothyroidism more common

- Probably an autoimmune disease; may

occur as a sequel to Hashimoto's thyroiditis

- Post therapeutic hypothyroidism (medical or surgical)

Secondary hypothyroidism:

Occurs when there is failure of the hypothalamic-pituitary axis due to

- Deficient secretion of TRH from the hypothalamus

Or:

- Lack of secretion of TSH from the pituitary

Clinical features

Generally in striking contrast to those of hyperthyroidism; may be quite subtle, with an insidious onset

In adults:

Dull facial expression, slow speech and poor memory

Puffiness of the hands, feet and face

Lethargy and fatigue

Thinning, dryness and loss of hair

Hypothermia

Bradycardia

Reduced systolic and increased diastolic blood pressure

Weight gain

Decreased reflexes

Constipation

Menstrual abnormalities

In infants:
Mental and physical retardation
- If not corrected, cretinism

Differential diagnoses
Endogenous depression
Reactive depression

Complications
Myxoedema coma Cretinism in the young

Investigations
Total serum T and T levels
TSH stimulation test
TRH test

Treatment objectives
Establish cause
Establish the severity of hypothyroidism
Restore normal body functions
Prevent complications

Drug treatment
Replacement therapy
- Levothyroxine sodium (thyroxine sodium)

Adult: initially 20 - 100 micrograms (50 micrograms for those over 50 years) orally daily, preferably before breakfast
- Adjusted in steps of 50 micrograms every 3 - 4 weeks until metabolism normalizes

(usually 100 - 200 micrograms daily)

Child 1 month - 2 years: initially 15 micrograms/kg orally once daily, adjusted in steps of 25 micrograms daily every 2 - 4 weeks until metabolism normalizes 2 - 12 years: initially 5 - 10 micrograms/kg once daily adjusted in steps of 25 micrograms daily every 2 - 4 weeks until metabolism normalizes 12 - 18 years: initially 50 - 100 micrograms once daily, adjusted in steps of 50 micrograms daily every 3 - 4 weeks until metabolism normalizes (usual dose 100 - 200 micrograms daily

Or:

Liothyronine sodium (1-tri-iodothyronine sodium)

*Adult*initially 10 - 20 micrograms orally daily, gradually increased to 60 micrograms daily in 2 - 3 divided doses

- Small initial doses in the elderly In hypothyroid coma:
- 5 - 20 micrograms by slow intravenous injection, repeated every 12 hours (as often as every 4 hours if necessary)

Alternatively:

- 50 micrograms by slow intravenous injection initially then 25 micrograms every 8 hours, reducing to 25 micrograms daily

Child 12 - 18 years: 10 - 20 micrograms orally

daily, gradually increased to 60 micrograms daily in 2 - 3 divided doses

In hypothyroid coma:

1 month - 12 years: 2 - 10 micrograms by slow intravenous injection every 8 hours (up to every 4 hours if necessary);

- Reduce to 1 - 5 micrograms in patients with cardiovascular disease

12 - 18 years: 5 - 20 micrograms, repeated every 12 hours] (up to every 4 hours if necessary)

- Reduce to 10 - 20 micrograms in patients with cardiovascular disease

Supportive measures

Treat anaemia, constipation and other complications as appropriate

Immediate mechanical ventilation in myxoedema coma

Notable adverse drug reactions, caution

- Thyroxine should not be used alone for long term replacement therapy
- Monitor serum levels of hormones to ensure that patients are not exposed to cardiac risks

Prevention

Iodinated salt to prevent iodine deficiency*CUSHING'S SYNDROME*

Introduction

Cushing's syndrome is a constellation of features associated with prolonged exposure to inappropriately high levels of plasma glucocorticoids.

The most common cause is from exogenous glucocorticoid use

Endogenous causes of Cushing's syndrome are rare.

Classification/ Aetiology:

ACTH- dependent

- Cushing's disease
- ectopic ACTH Syndrome
- macronodular Adrenal Hyperplasia

ACTH-independent causes:

- cortisol-secreting adrenal tumour or hyperplasia
- Iatrogenic Cushing's Syndrome

Clinical features

Weight gain, Moon facies, fat pads, truncal obesity;

Facial plethora, red- purple striae ecchymoses, facial hair, hirsutism and male pattern balding in women, acne, Acanthosis nigricans, opportunistic fungal infections, poor wound healing.

Ocular: Raised intraocular pressure, exophthalmos, Visual field defects-

Muscle weakness especially proximal myopathy

Menstrual irregularities- decreased libido, men- decreased libido and impotence in men

Lethargy, depression, emotional liability, psychosis, diabetes mellitus, , loss of height, pathologic fractures, hypertension, discriminatory features -reddish purple striae, plethora, proximal muscle weakness, bruising with no obvious trauma, and unexplained osteoporosis.

Investigations

- Serum electrolytes and glucose and FBC
- Urine free cortisol (UFC; at least two measurements)
- Late-night salivary cortisol (two measurements)
- 1-mg overnight dexamethasone suppression test (DST)
- Longer low-dose DST (2 mg/d for 48 h)

Determination of the cause of cushings

- Morning plasma ACTH-
- High-Dose Dexamethasone Suppression Test-
- Inferior Petrosal Sinus Sampling and Selective Venous Catheterization-;
- Imaging- CT/MRI Scanning of Pituitary and Adrenals, scintigraphy

Differential diagnosis

Constitutional obesity

Other causes of weight gain

Pseudo cushings

Pregnancy
Depression
Bulimia
Alcoholism

Management

Exclude causes of weight gain through history and examination

Confirm diagnosis and differential diagnoses before initiating treatment

Remove any underlying cause e.g. iatrogenic CS

General principal reduce level of plasma cortisol and treat any symptoms and complications

Surgical resection of the culprit tumour if patient qualifies.

Start with medical treatment and then surgery or continue medical treatment as indicated..

Medical Treatment

1) Mifepristone,
2) Adrenal steroid inhibitors

Metyrapone

Aminoglutethimide- commonly prescribed in combination with metyrapone.

Surgery: adrenalectomy or pituitary surgery

Radiotherapy: For failed surgery and/or patients not fit for surgery

Complications of CS and its treatment
Diabetes mellitus
Osteoprosis and fracturesv
Hypopituitarism (compression of other gonadotrophs)
Steroid psychosis
Hormone replacement
Lifelong glucocorticoid replacement with pituitary destruction or bilateral adrenalectomy. Lifelong mineralocorticoid replacement is also necessary in those patients who undergo bilateral adrenalectomy.

Prognosis
Favourable for adenomas if surgery is curative.
CS due to carcinoma and adrenal hyperplasia have worse prognosis

OBESITY
Introduction
Overweight and obesity increase the risk for several diseases such as hypertension, ischaemic heart disease, and diabetes mellitus.
Obesity results from an imbalance between energy intake and energy expenditure. However, more recent researches, have suggested that genetics, physiological and

behavioural factors also play significant roles in the aetiology of obesity.
Management of weight disorders is a two step process: assessment and management.

Clinical Features
The main symptom of obesity is a complaint of being too fat by the patient or concerned relatives.

Measurements for evaluation
Body mass index (BMI) is a surrogate measure for global adiposity for ease of clinical use.
Waist circumference: is a surrogate marker for truncal obesity.
BMI is calculated as follows
BMI = weight in kg divided by height in m, expressed as kg/m^2
Underweight: $<18.5\ kg/m^2$
Normal weight: $18.5 - 24.9\ kg/m^2$
Overweight: $25 - 29.9\ kg/m^2$
Obesity (Class1): $30 - 34.9 kg/m^2$
Obesity (Class 2): $35 - 39.9\ kg/m^2$
Morbid obesity (Class 3): $> 40\ kg/m^2$
Super morbid obesity: $> 50\ kg/m^2$
Truncal obesity : waist circumference and/or waist/hip ratio (WHR).

Upper limits: 102 cm and 88 cm in men and women respectively.

Investigations

Non-specific assessment:, fasting blood glucose, oral glucose tolerance test, serum lipid profile. Assessment of other complications as indicated.

Specific assessment should be directed towards identifying underlying specific causes when suspected. These include conditions such as

Endocrinopathies (hypothyroidism, Cushing's syndrome, male hypogonadism, Insulinoma CNS disease that affects hypothalamic function, PCOS),

genetic syndromes associated with obesity,

mental disorders like bulimia nervosa and binge eating disorder,

medications such as steroids, atypical antipsychotics

Pathological consequences of obesity

System	Pathology
Gastrointestinal:	Gallstones, pancreatitis, abdominal hernia, Non Alcoholic Fatty Liver Disease,GERD
Endocrinology/ Metabolic:	Metabolic syndrome, insulin resistance, impaired glucose tolerance, type 2 DM, dyslipidaemia, polycystic ovarian syndrome.

Cardiovascular:	Hypertension, coronary artery disease, congestive heart failure, dysarrhythmia, pulmonary hypertension, stroke, venous stasis, deep venous thrombosis (DVT), pulmonary embolism, chronic kidney disease.
Respiratory:	Abnormal pulmonary function, obstructive sleep apnoea, obesity hypoventilation syndrome.
Musculoskeletal:	Osteoarthritis, gout, low back pain
Gynaecologic:	Menstrual irregularities, infertility
Genitourinary:	Urinary stress incontinence
Ophthalmologic:	Cataracts
Neurologic:	Idiopathic intracranial hypertension
Cancers:	Oesophagus, colon, gall bladder, cervix, breast, uterus, kidney, prostate
Postoperative events:	Atelectasis, pneumonia, DVT, pulmonary embolism

Treatment objectives

Conventional approach is to reduce energy intake and increase physical activity.

Initial goal of therapy should be to reduce body weight by 10% from the individual baseline weight over a period of six months.

This can be achieved through the following:

- Lifestyle modification (diet therapy and physical activity)
- Behavioural and psychological interventions
- Pharmacological intervention
- Bariatric surgery

Lifestyle modifications:

Low caloric diets (with reduction of both carbohydrate and fats components) are to ensure deficit of 500 to 1000kcalories per day from the individual daily nutritional intake to ensure weight loss of 0.5 to 1kg per week.

Physical activity:

Moderate levels of exercise (brisk walking, swimming, cycling) for 30 to 45minutes, five days per week is encouraged. Other forms of exercise must be based on physician prescription.

Behavioural and psychological interventions:

Behavioural therapy should be aimed at

motivating patients in adopting and practising all recommended treatment strategies of obesity management.

Pharmacological intervention:
When lifestyle modification and behavioural therapy has failed
Useful and approved drugs include orlistat,
Bariatric surgery:
BMI >35kg/m2 with co-morbidities or BMI ≥40kg/m2 following failure of lifestyle and pharmacologic therapy.

CHAPTER 10:

EYE DISORDERS

ACUTE ANTERIOR UVEITIS (Iritis)

Introduction

Inflammation of the iris (with or without the cilliary body)

Usually occurs without any associated systemic inflammation

Tends to recur

Clinical features

Blurring of vision
Eyeball is tender
Phoptophobia due to cilliary spasms.
Exudation into anterior chamber
Flare and cells
Keratic precipitates
Hypopion
Posterior synechiae
Miosis due to spasm of sphincter pupillae

Differential diagnoses

Infective conjunctivitis
Acute iritis
Acute glaucoma

Complications
Secondary glaucoma
Cataracts

Investigations
Chest radiograph to exclude sarcoidosis and tuberculosis
Spinal radiograph (especially lumbrosacral segment) to exclude ankylosing spondilytis
Serology for rheumatoid factor, antinuclear antibodyetc

Treatment
Corticosteroid drops for treatment of inflammation:
Betamethasone sodium phosphate 0.1%
- Apply eye drops every 1 - 2 hours until inflammation is controlled then reduce frequency
- Subconjunctival: injection of steroid if severe

Atropine sulfate 0.5% or 1%
- 1 drop up to 4 times daily

Caution and Contraindications to treatment
Avoid atropine drops if there is risk of acute glaucoma
Prevention
No real preventive measures

ACUTE KERATITIS

Introduction

Infection or inflammation of the cornea. Could be secondary to trauma to the cornea
Sometimes associated with infective conjunctivitis but could occur de novo

Clinical features

Irritation, pain
Red eye (conjunctival congestion)
Eye discharge: watery; purulent (if bacterial)
Photophobia
Visual impairment, depending on the site and size of ulcer and if interstitial
Hypopion, if associated with uveitis (no hypopion if viral)
Ulceration of cornea, which stains with fluorescene; no ulcer in interstitial keratitis

Aetiology

Exogenous

- Marginal ulcers secondary to bacterial conjunctivitis *(S. aureus)*
- Central ulcers (Pneumococcus, *Herpes simplex*, fungi)

Keratomalacia (Vitamin A deficiency)
Exposure (7th cranial nerve palsy or dysthyroid eye disease)
Endogenous

- Interstitial keratitis of congenital syphilis

- Interstitial keratitis of Herpes zoster

Differential diagnoses
Infective conjunctivitis
Acute iritis
Acute glaucoma

Complications
Corneal perforation

Investigations
Corneal scraping for microscopy, culture and sensitivity

Drug treatment
Antibiotic drops (if bacterial)
- Chloramphenicol eye drops 0.5%
- Apply 1 drop at least every 2 hours, and then reduce thefrequency as infection is controlled and continue for 48 hours after healing

Atropine drops
- 1 drop 2 times daily

Antivirals (if dendritic ulcer)
conjunctiva,
- Acyclovir

Apply 1 cm ointment 5 times daily(continue for at least 3 days after complete healing
- Idoxuridine 5% in dimethylsulfoxide

Adult and child over 12 years: apply to lesions 4 times daily for 4 days, starting at first sign of

attack
Child under 12 years: not recommended
Topical steroids
- Only for interstitial keratitis where there is no active ulcer

Non-drug measures
Lateral tarsorrhaphy for exposure keratopathy

Caution and contraindications to treatment
Never use topical steroids in the presence of an active ulcer

Prevention
Treat initial infection or trauma promptly to avoid progression to keratitis

ALLERGIC CONJUNCTIVITIS
Introduction
Could occur on its own or in association with generalized atopy (asthma, eczema, spring catarrh)

Clinical features
Itching of the eyes with grittiness
- May be associated with itchy ears and throat, or sinusitis

Brownish discolouration of the conjunctiva
Eyelid oedema
Red eyes occasionally, with watering when

acute
Follicles on the bulbar conjunctiva especially at the limbus
Papillae on the tarsal conjunctiva (seen on eversion of the eyelid)
Phlycten in tuberculosis- appears as a yellow nodule with surrounding leash of engorged vessels

Aetiology
Exogenous allergens
- Topical drugs: atropine, penicillin
- Cosmetics
- Pollen from plants and flowers (hay fever or spring catarrh)
- House dust mite and animals

Endogenous allergens
Phlyctenular conjunctivitis caused by tuberculo-protein

Differential diagnoses
Trachoma
Other forms of conjunctivitis

Complications
Pannus formation
Keratoconus
Corneal plaques

Investigation
Skin sensitivity test to detect allergen

Drug treatment

Antiinflammatory preparations

- Antazoline sulfate 0.5%, xylometazoline hydrochloride

0.05%

Adult and child over 5 years: apply 2 - 3 times daily

- Ketotifen eye dropsChild 3 – 18 years apply twice daily

Or

- Olopatadine eye drops

Adult and Child 3 – 18 years apply twice daily; max. duration of treatment 4 months

Corticosteroids/NSAIDS – low dose, topical, if severe

Diclofenac sodium 0.1% eye drops-

Adult and child: apply once daily

Phlyctenular conjuntivitits:

Treat for tuberculosis using standard regimen

Caution and Contraindication to treatment

Avoid overuse/misuse of steroids. Use only in severe cases

Xylometazoline is a sympathomimetic; use with caution in patients susceptible to angle closure glaucoma

Systemic absorption of antazoline and

xylometazoline may result in interactions with other drugs

Prevention
Avoid allergen(s) as much as possible in cases where it/ they have been identified

CATARACT
Introduction
Cataract is the opacification or clouding of the crystalline lens.
It is the commonest cause of blindness and low vision in the world.

Aetiology – Congenital e.g. Rubella, Toxoplasmosis
- Developmental
- Senile – occurs with ageing
- Secondary e.g. Traumatic, Diabetic, Drug induced, post uveitic etc.

Clinical features
Gradual, painless loss of vision
In the early stages, feels like looking through frosted glass.
White pupillary reflex
If secondary, symptoms and signs of causative disease are also present.

Differential diagnosis
Corneal opacity

Other causes of leucocoria e.g.
Retinoblastoma (in children), retinal detachment, severe ocular toxoplasmosis etc.

Complications
In congenital cases, can lead to amblyopia (lazy eye) if not operated early
Intumescent cataract with secondary glaucoma
Phacolytic uveitis and glaucoma

Investigations
Projection of light
Pupillary light reflex
Macular function test
Ocular ultrasounds scan: especially in cases of trauma to exclude pathology of the posterior segment of the eye

Management
(a) Treatment objective –
To remove the cataractous lens
Replace with a clear plastic lens inserted into the eye (except in infants).
(b) Main treatment is surgical.
An extracapsular cataract extraction with posterior chamber intraocular lens implant (ECCE + IOL) is the most appropriate for older children and adults.

Small incision cataract surgery and phacoemulsification are recent advances of the ECCE technique.
In new born babies and infants, a lensectomy and anterior vitrectomy is the preferred surgical procedure.
Aphakic glasses or contact lenses are worn to correct the resulting refractive error.
Posterior capsular opacity following ECCE could be treated with surgical or Yag laser capsulotomy.

Caution and contraindications to treatment
Intraocular lens implants is not advisable for new born babies and infants.

Prevention
Aspirin and other aspirin like analgesics have been found to delay cataracts in patients with diabetes and rheumatoid arthritis.
Antioxidant vitamins - Vitamin C has been found to have a protective effect against nuclear and posterior subcapsular cataracts. Vitamin E has also been found to protect against nuclear and cortical cataract.

EYE INJURIES

Introduction
Injuries to the eye could be caused by blunt or

sharp objects or chemicals

Aetiology

Blunt injuries e.g. a fist or a ball hitting the eye
Sharp injuries e.g. glass, metal, broom stick etc
Chemicals e.g., alkali or acid

Clinical features

Blunt injury

Eyelids: peri-orbital haematoma and oedema
Conjunctivae: subconjunctival haemorrhage and chemosis
Cornea: abrasion or oedema
Anterior chamber: hyphaema from tears of the iris or cilliary body
Iris: traumatic mydriasis
Traumatic uveitis
Angle recession
Lens: dislocation into anterior or posterior chambers; cataract
Vitreous haemorrhage
Retina: peripheral tear leading to retinal detachment; oedema with haemorrhage (Commotio Retinae)
Choroid: tear with haemorrhage
Rupture of the eyeball, usually posteriorly (rare)
Optic nerve: avulsion
Blow out fracture of the orbital wall

Sharp Injury

Lacerations of eyelids, conjunctivae, cornea, sclerae, or corneo-sclera
Uveal prolapse with or without lens extrusion
Intraocular foreign body
Endophthalmitis

Chemical burns

Acid or alkali e.g. cement or lime are alkaline
Acids coagulate surface proteins
Alkalis penetrate into the anterior chamber causing uveitis
- Symblepharon: adhesions between bulbar and tarsal conjunctivae

Differential diagnoses

Conjuctivitis
Endophthalmitis
Orbital cellulitis

Complications

Ruptured globe
Endophthalmitis
Reversible blindness (compression of optic nerve by orbital haematoma)
Irreversible blindness (optic nerve avulsion)
Corneal opacity/scarring

Investigations

Orbital radiographs
Orbital ultrasound

Management

Blunt injuries - Treat individual injury

Sharp injuries

- Suture lacerations
- Remove intraocular foreign bodies with magnet if possible, or by vitrectomy
- Parenteral antibiotics, if infected
- Evisceration (removal of the contents of the eyeball) if ruptured globe, or if infection not settling on antibiotics

Chemical burns

- Copious rinsing of eyeball and fornices with sodium chloride 0.9% or clean water at site
- In hospital, copious rinsing again, to dilute offending agent
- Remove particles from eye e.g. lime or cement
- Antibiotic ointment
- Rodding of fornices with ointment to prevent symblepharon
- Topical steroids for uveitis once cornea is re-epithelized
- Vitamin C (ascorbic acid)

Caution and contraindications

Avoid the use of topical steroids in active corneal ulceration

Avoid the use of harmful traditional eye medications; may cause more complications

Prevention

Wearing of appropriate protective eye goggles for sports, welding and when working with chemicals

FOREIGN BODIES IN THE EYE

Introduction

Foreign bodies are usually in the form of small particles of metal, vegetable matter or insects which embed on surface of the eye

Occasionally a high velocity material, usually a metal could be propelled into the eye

Clinical features

Foreign body may be embedded on the tarsal conjunctiva, bulbar conjunctiva, and cornea or inside the eye

- Intraocular foreign body (IOFB)

IOFBs may be in the anterior chamber, iris, lens or vitreous; on the retina or even behind the eyeball after doubly perforating the eye

Differential diagnoses

Corneal abrasion

Endophthalmitis

Complications

Perforation of the eye

Endophthalmitis

Retinal toxicity from a metallic IOFB

Investigation
Radiograph of the orbit with a localizing ring

Management
Removal of subtarsal, conjunctival or corneal foreign body under magnification e.g. slit lamp microscope

Caution
Ultrasound should be avoided in an eye with a perforating wound

Prevention
Appropriate protective goggles for sports, welding, game hunting etc

10

GLAUCOMA

Introduction

The glaucomas are a group of diseases in which there is gradual loss of vision (actually visual field) due to a form of optic neuropathy in which raised intraocular pressure is a risk factor.
It is a common cause of blindness worldwide and once a patient is blind from glaucoma, vision cannot be restored.
Classification - Congenital (Buphthalmous)
Developmental
Acquired - Primary - Acute - closed

angle glaucoma

Chronic – open angle glaucoma
Closed angle glaucoma
Secondary glaucomas e.g.
Traumatic, lens
induced uveitis, drug induced etc.

Clinical features

- The commonest type of glaucoma, chronic open angle glaucoma is generally symptomless except in the late stages when symptoms of visual loss present.
- In Congenital glaucoma, there is enlarged eyeball(s) in the newborn, corneal oedema/opacity, photophobia and watering.
- In Acute closed angle glaucoma, there is marked elevation of intraocular pressure with severe pain, reduction in vision, red eye, fixed mid-dilated pupil, shallow anterior chamber.

Investigations

- Intraocular pressure measurement
- Central and peripheral visual field test.
- Fundoscopy to determine cup/disc ratio
- Gonioscopy to view the angle of the eye

Management

Treatment objective:
Stop further loss of retinal ganglion cells

and nerve fibre layer by prompt treatment.
Drug treatment –
Topical and Systemic drugs can be utilized in all forms of glaucoma but surgery may be the only treatment option in some glaucomas (drugs are used before surgery in these cases). These drugs include:

- Betablockers e.g. Timoptol
- Parasympathomimetics e.g. Pilocarpine
- Sympathomimetics e.g. Adrenalin, Dipivefrin
- Carbonic Anhydrase Inhibitors e.g. Acetazolamide, Dorzolamide
- Prostaglandin analogues e.g. Latanoprost
- Hyperosmotic agents e.g. Mannitol, Glycerol

Surgical treatment –
Available surgical procedures include:

- Goniotomy (for Congenital glaucoma)

Trabeculectomy (for all forms of glaucoma)
Trabeculotomy
Laser Trabeculoplasty
Peripheral Iridectomy or Yag Laser Iridotomy for acute closed angle glaucoma (to the affected eye as well as the normal fellow eye).
Laser photo-ablation of the ciliary body

Differential diagnosis
Ocular hypertension

Primary optic atrophy (pallor but no cupping of the disc)

Complications
Central Retinal Vein Occlusion
Rubeosis Irides with resultant secondary glaucoma
Total blindness

Adverse drug reactions
Timoptol – can precipitate heart block, bradycardia and bronchospasms
Pilocarpine – Mioses, spasms of accommodation, retinal detachment
Acetazolamide – Paraesthesia, renal stone formation, Steven – Johnson syndrome, gastrointestinal disturbances, decreased libido, fatigue, weight loss.
Latanoprost –foreign body sensation, mild conjunctival hyperaemia and increased pigmentation of iris.

Contraindications
Timoptol is contraindicated in Asthmatics and patients with heart block.

Acetazolamide is contraindicated in patients with pre-existing systemic acidosis, patients with sicke cell disease and allergy to sulphonamides.

Pilocarpine is contraindicated in eyes with previous retinal detachment, and pathological myopia.

Prevention

Early detection and prompt treatment. Screening for glaucoma should be incorporated into the health plan of any nation

INFECTIVE CONJUNCTIVITIS

Introduction

The commonest cause of a red eye is infective conjunctivitis, which could be caused by bacteria or viruses

Clinical features

Red eye (generalized)

Eye discharge: purulent or catarrhal, worse on waking from sleep

Eye discomfort: grittiness

Photophobia: mild

Swollen eyelids in ophthalmia neonatorum

Aetiology

Staphylococcus aureus,

Pneumococcus

Haemophillus influenza,

Gonococcus: Ophthalmia neonatorum

Older children or adults after use of infected urine to treat a red eye

TRIC agent (chlamydia)
Adenovirus: Epidemic keratoconjunctivitis ('Apollo')

Differential diagnoses
Allergic conjunctivitis
Acute keratitis
Acute iritis/uveitis
Acute glaucoma

Complications
Corneal affectation, which could lead to perforation
Endophthalmitis

Investigations
Conjunctival swab for microscopy, culture and sensitivity

Management
Non-drug measures
Dark glasses for photophobia

Drug treatment
Antibiotic eyedrops or ointments
- Chloramphenicol 0.5%
- Apply one drop at least every 2 hours until infection is controlled then reduce frequency and continue for 48 hours after healing
- Inclusion Conjunctivitis: Sulphonamide drops or tetracycline drops or ointment

- Epidemic keratoconjunctivitis: Antibiotic drops to prevent secondary bacterial infection

(Chloramphenicol 0.5% drops
Adult and child over 2 years: apply every 4 hours for no more than 5 days)

- Ophthalmia Neonatorum
- Gentamicin sulfate 0.3% applied as stated above

Or:

- Ofloxacin 0.3% solution applied as stated above

Plus:

- systemic cephalosporin e.g. ceftriaxone *Adult:* 1 g every 12 hours intravenously for 7 days *Child:* by intravenous infusion over 60 minutes Neonates: 20 - 50 mg/kg once daily, by deep intramuscular injection, intravenous injection over 2 – 4 minutes, or by intravenous infusion 1 month - 12 years (body weight under 50 kg) 50 mg/kg once daily, up to 80 mg/kg in severe infections

Chlamydia

- Systemic erythromycin

*Adult and child over 8 years:*250 - 500 mg orally every 6 hours (or 500 mg - 1 g every 12 hours)
1 month - 2 years: 125 mg orally every 6 hours; dose doubled in severe infections
2 - 8 years: 250 mg 6 hourly; 8 - 18 years: 250 -

500 mg 6 hourly; dose doubled in severe infections

Caution and contraindications

Steroid drops are absolutely contraindicated

Prevention

Wash hands thoroughly after any unhygienic procedure

Avoid sharing towels used for cleaning face

OPHTHALMIA NEONATORUM

Introduction

Infection in both eyes of a newborn baby in the first one month of life, without obstruction of the nasolacrimal ducts

Clinical features

Swollen eyelids:

- It may be impossible to see the baby's eye because of the swelling

Red eyes:

- The conjunctivae are less inflamed in chlamydial infection

Pus:

- Oozes out when the eyelids are opened
- Fever:
- May or may not be present

Aetiology

Bacterial:

- Especially *Neisseria gonorrhoea:* starts within 3 days after birth
- Chlamydia (usually starts 1 week after birth)

Chemicals:

Others

Differential diagnosis

Lid oedema following prolonged difficult labour

Complications

Corneal perforation

Endophthalmitis

Investigation

Conjunctival swab for microscopy, culture and sensitivity

Management

Non-drug measures

Copious irrigation to wash pus from the eyes with cooled boiled water or sodium chloride 0.9%

Drug treatment

Topical antibiotics

- Gentamicin 0.3% eye drops

Apply 1 drop at least every 2 hours, and then reduce frequency as infection is controlled

Or:

Ofloxacin 0.3% eye drops
Apply twice daily. (not to be used for more than 10 days)

Or:
Tetracycline 1% eye ointment
Apply 3 times daily for one week or more, depending on the severity of the condition

Plus

Ciprofloxacin 10 mg/kg per dose intramuscularly 12 hourly for 2 days

Or:

Ceftriaxone 100 mg/kg by deep intramuscular injection or intravenous injection over 2 - 4 minutes every 24 hours

- By intravenous infusion: 1 g daily, 2 - 4 g in severe infections

Child: neonate, infuse over 60 minutes, 20 - 50 mg/kg daily (maximum 50 mg/kg daily)
Child under 50 kg: 20 - 50 mg/kg daily by deep intramuscular injection or by intravenous injection over 2 - -4 minutes, or by intravenous infusion; up to 80 mg/kg daily in severe infections

Caution
Do not use steroids eyedrops

Penicillin drops are not effective in the treatment of opthalmia neonatorum

Prevention

Apply tetracycline eye ointment or silver nitrate drops in both eyes of neonates immediately after delivery

Proper antenatal care for early detection of infection in mothers

SCLERITIS/EPISCELITIS

Introduction

Inflammation of the sclera and episclera

Usually self-limiting but relapses may occur

Usually unilateral and associated with collagen disorders

Clinical features

Dull, deep-seated pain in the eye

Localized conjunctival congestion

Differential diagnoses

Pterygium

Phlyctenular conjunctivitis

Trauma to the eye

Complications

Thinning of the sclera

Anterior staphyloma

Scleral perforation

Investigations

Investigate for collagen diseases

Management
Topical steroids or NSAIDs for the duration of symptoms
Treat arthritis if active

Caution
Avoid prolonged use of steroids

Prevention
No real preventive measures available

STYE (HORDEOLUM)
Introduction
External stye
- Infection of the lash follicle and its associated gland of Zeis or Moll

Internal stye (chalazion)
- Infection of the meibomian gland

Clinical features
Painful lump growing on the eyelid
Red swollen area on the eyelid (like a boil)
Pain in the affected area of the eyelid
Chalazion: firm, painless lump on the eyelid, usually upper lid

Differential diagnoses
Various eyelid cysts and tumours

Complications
Pre-septal cellulitis

Orbital cellulitis
Cavernous sinus thrombosis

Investigations
If recurrent, screen for diabetes

Management
Non-drug measures
Apply warm wet pads for 15 minutes 4 times daily until the stye drains
Incision and curettage (if there is still a chalazion lump) as soon as the infection settles

Drug treatment
Antibiotic eye ointment to stop infection
- Chloramphenicol ointment apply 4 times daily for 2 weeks

Systemic antibiotics
- Amoxicillin 250 - 500 mg orally every 8 hours for 5 - 7 days

Caution
Discourage the use of traditional eye medication

Prevention
Clean eyelids regularly and thoroughly
For recurrent styes, use baby shampoo to clean the eyelashes regularly

THE RED EYE

Causes

Infective conjunctivitis including ophthalmia neonatorum
Allergic conjunctivitis
Keratitis
Scleritis/episcleritis
Trauma to the eye
See relevant sections

TRACHOMA

Introduction

Caused by *Chlamydia trachomatis*, an organism midway between a bacterium and virus
The organism is found in the conjunctival as well as corneal epithelium and is responsible for two different conditions:

- Trachoma (a severe disease)
- Inclusion conjunctivitis (milder)

Trachoma is commonly associated with poverty and unhygienic living conditions

Clinical features

Acute phase:
Irritable red eye
Mucopurulent eye discharge
Eyelid oedema, pain, photophobia in severe cases
Chronic phase:
Follicles on tarsal conjunctivae

Papillae
Superficial punctate keratitis
Pannus formation on superior cornea
End stage:
Eyelid scarring with trichiasis, entropion
Conjunctival scarring
Limbal scarring with Herbert's pits
Corneal scarring

Differential diagnoses
Other forms of infective conjunctivitis (especially viral)
Allergic/vernal conjunctivitis
Corneal scarring from other diseases

Complications
Trichiasis
Entropion
Corneal scarring

Investigations
Conjunctival scraping for microscopy
- Immunofluorescence or ELISA test

Giemsa staining for trachoma inclusion bodies

Drug treatment
Topical:
Tetracycline ointment applied 4 times a day for 6 weeks
Systemic:

Erythromycin, tetracycline (not recommended for young children) or the newer antibiotics e.g. azithromycin as appropriate

- Azithromycin

Adult: 500 mg orally once daily for 3 days

Child over 6 months: 10 mg/kg (maximum 500 mg) orally once daily for 3 days; over 6 months (body weight 15 - 25 kg) 200 mg once daily for 3 days; body weight 26- 35 kg: 300 mg once daily for 3 days; body weight 36 - 45kg: 400 mg once daily for 3 days

Surgical treatment

Indicated for the treatment of trichiasis, entropion, corneal scarring

Corneal graft, but entropion must be corrected first

Caution and contraindications

Systemic tetracycline is contraindicated in young children

Prevention

Improve personal and public hygiene

Treat the whole community with topical or systemic antibiotics

Prompt surgery for trichiasis and entropion to prevent blindness from corneal scarring

XEROPHTHALMIA

Introduction

The term Xerophthalmia refers to the spectrum of eye diseases under Vitamin A deficiency

Ranges from night blindness to conjunctival xerosis, to Bitot's spots, corneal xerosis and finally keratomalacia

Clinical features

Night blindness

Dryness of the conjunctiva and cornea (xerosis)

Tearing

Bitot's spots

Corneal degeneration (keratomalacia)

Differential diagnosis

Measles keratoconjunctivitis

Complications

Corneal perforation

Corneal scarring

Blindness

Investigations Conjunctival impression cytology (where available)

Serum Vitamin A levels

Management

Non-drug treatment

Nutrition education

Drug treatment

Vitamin A capsules 200,000 units orally daily for two days, then one capsule after one week

Topical antibiotics and antivirals where applicable

Padding the eye (for active corneal ulceration)

Caution

Avoid the use of harmful traditional eye medication

Prevention

Distribution of massive dose capsules of vitamin A to affected communities

Nutrition and health education

Fortification of foods with vitamin A

CHAPTER 11:

GENITOURINARY SYSTEM NEPHROLOGY

ACUTE KIDNEY INJURY (AKI)

Introduction

Acute kidney injury is defined as a sudden clinical and/or laboratory manifestation of abnormal kidney function occurring within 48 hours or 7 days of kidney injury.

A reduction in urine output documented as less than 0.5 ml/kg/hour for more than 6 hours.

Absolute increase in serum creatinine of more than or equal to 0.3 mg/dl (26.5 umol/L) occurring within 48 hours of an insult to the kidneys.

A percentage increase in serum creatinine of more than or equal to 50% (1.5 fold from baseline) which is known or presumed to have occurred within 7 days.

Seen in about 5% of patients admitted to medical wards

Up to 30% of intensive care admissions may manifest and develop AKI.

AKI could be classified variably depending on the defining criteria.

Hospital acquired AKI typically develops as a complication of another clinical condition Community acquired AKI typically evolve in these patients while in their homes.

Classification:

Classified according to: the

- Medical specialty in which it evolves ie Surgical AKI if it complicates surgery, Obstetric or Gynaecologic AKI
- Volume of urine being excreted at the time the AKI is developing:
- non-oliguric AKI when urine volume is more than 400mls/day
- oliguric AKI when urine volume is less than 400mls/day and anuric AKI when urine volume is less than 150mls/day.
- Site of physiologic or anatomic derangements or toxic insult into pre-renal, intrinsic renal and post renal AKI:

Pre-renal AKI:

Systemic derangement leading to hypoperfusion of the kidneys leading to AKI e.g. diarrhoea and vomiting, haemorrhage, cardiac failure etc.

Intrinsic renal AKI:

Direct nephrotoxic injury to renal tubules or there could be inflammation of interstitial, glomerular or vascular structures.

Post renal AKI:

Obstruction to the outflow of urine, which could be at different levels e.g. renal pelvis, ureter, urinary bladder or urethra.

Examples of causes of post-renal AKI include:

- Nephrolithiasis
- Netroperitoneal fibrosis and tumours
- Bladder cancer
- Benign prostatic hypertrophy
- Prostate cancer or even urethritis

Peculiar causes of AKI in Nigerians include:

- Nephrotoxins (native herbs, drugs, $CuSO_4$ (green water)
- Ethylene glycol poisoning etc)
- Cholera
- Malaria
- Septicaemia (particularly typhoid)
- Obstetric causes (ante partum haemorrhage, post partum haemorrhage, septic abortion, eclampsia).

AKI could now be staged according to severity (See table below)

Table 1: Staging of Acute Kidney Injury

STAGE	CHANGE IN SERUM CREATININE (Scr)	% INCREASE IN SERUM CREATININE (Scr)	URINE VOLUME
1	Rise in Scr by > 0.3 mg/dL	Increase of >150-190 % (1.5 to 1.9-fold increase) from baseline	Urine output less than 0.5ml/kg/hr for more than 6 Hours

2		Rise in serum creatinine by >200-290 % (2 to 2.9-fold increase) from baseline	Urine output less than 0.5ml/kg/hr for more than 12 Hours.
3		rise in serum creatinine to >300 % (≥ 3 fold) from baseline. OR eGFR <35ml/min/1.73mzin those <18 years.	Urine output less than 0.3ml/kg/hr for ≥12 for 24Hours or anuria for ≥ 12 hours.

Clinical Features:

- Asymptomatic
- Reduction in urine output (oliguria),
- features of uraemia,
- fluid retention leading to oedema.
- Uraemia present with different features in different systems: I

Gastrointestinal system :

- Nausea and vomiting,
- Hiccups,
- Diarrhoea,
- Epigastric pain and rarely haematemesis.

Chest:

- Central chest pain of pericarditis
- breathlessness
- Pulmonary oedema.

Central nervous system:

Uraemia manifest with tremors

Derangement of sleep rhytmn

Drowsiness

Seizures

Stupor or coma
Skin:
dryness and pruritus
Haemopoietic system:
Anaemia (pallor)
Bleeding
Diathesis.

Diagnostic criteria: As in the definition and table above.

Differential Diagnoses:
- Acute exacerbation of Chronic Kidney Disease.
- End Stage Renal Failure

Complications
- Pulmonary oedema
- Infections
- Electrolyte abnormalities:
- Hyperkalemia in acute cases and
- hypokalaemia and
- hyponatreamia in polyuric phases.

Relevant investigations and management:
Urine Examination (Volume / microscopy /Urinalysis/Electrolytes).
- Allow quantification of total daily urine output, which could be used in staging. Microscopy would reveal the types of cells, casts, crystals or other substances in the urine.

- Urinal athy.

Renal biopsy: Mandatory in the following:

- Any evidence of glomerular disease :
  - nephrotic range proteinuria
  - sub-nephrotic range proteinuria with haematuria
  - RBC cast
- AKI not resolving in 6 weeks
- AKI in connective tissue disease
- AKI in renal allograft
- Determine the prognosis and chance of recovery of renal function in dialysis dependent AKI.

Principles of management in patients with AKI include:

- Maintenance of fluid homeostasis
- Control of biochemical abnormalities
- Maintenance of nutrition
- Treat the underlying cause
- Dialysis where indicated
- Maintenance of fluid homeostasis:

Entails strict regulation of fluid intake to insensible loss (500-1200mls/day) Replacement of fluids totalling volume of urine and other documented losses in the previous 24 hours.

Avoid potassium containing fluids

- Control of biochemical abnormalities:

Hyperkalaemia in these patients should be treated in one of 3 ways:

Forcing the potassium (K) into cells using

- Glucose-Insulin Infusion or Glucose Infusion
- Antagonising the effects of K on the heart using 10% Calcium gluconate
- Dialysis or use of ion exchange resin like kayexelate
- Maintenance of nutrition:

AKI patients are usually hypercatabolic hence the following

- High calorie low protein diet is recommended in acute or oliguric phase while
- High calorie normal protein is recommended in recovery phase.
- Parenteral hyperalimentation is seldom necessary in prolonged cases.
- Dialysis where indicated:

Manifestation of clinical and biochemical features of uraemia or development of electrolyte and acid-base complications of AKI:

Clinical features include:

- Encephalopathy,
- Pulmonary oedema,
- Persistent nausea and vomiting,
- Pericarditis,
- Refractory oedema,
- Uncontrolled HT
- Bleeding diathesis

Biochemical indications include:

- Hyperkalemia > 6.5mmol/l,
- Serum bicarbonate < 12mmol/l,

- Urea >25 mmol/l,
- Creatinine >600micromol/l

Manifestations of features of hypercatabolism:

- K+ rate of rise >1mmol/day,
- urea rate of rise >10 mmol/day a
- creatinine rate of rise >100micromol/day.

Dialysis:

The patients could benefit from

- Daily Haemodialysis,
- Extended Daily Dialysis,
- Slow low efficient Dialysis,
- Acute Peritoneal Dialysis,
- intermittent Peritoneal Dialysis,
- Haemofiltration or Haemodiafiltration.

Prevention of AKI:

- Avoidance of nephrotoxins in all forms.
- Provision of pipe borne water.
- Prompt treatment of accident victims.
- Prompt treatment of infections.
- Good maternal, child and reproductive health care
- Adequate hydration during contrast investigations

CHRONIC KIDNEY DISEASE (CKD):

Introduction:

CKD is defined as a structural and functional

abnormality of the kidneys persisting for 3 months or more and manifesting as markers of kidney damage or reduction in glomerular filtration rate (GFR). Markers of kidney damage include persistent microalbuminuria or overt albuminuria or haematuria. Structural abnormalities include abnormalities on imaging or on histology.

CKD is also defined as GFR less than 60ml/min or presence of markers of kidney disease for 3 months or more.

CKD is quite common in clinical practice as 12-26% of Nigerians have covert or overt CKD as revealed in community studies

Markers of kidney disease include:

- Persistent proteinuria - Dipstick positive proteinuria or microalbuminuria
- Persistent Haematuria - by dipstick and/or urine microscopy
- Abnormal renal imaging - by various techniques such as ultrasonography, Computerised Tomography Scan, intravenous urography or plain radiograph, renal scintigraphy.

Abnormal renal histology

Stages of CKD:

Table 1: Definition of the five stages of Chronic Kidney Disease

Stage	Description	GFR (ml/min/1.73m²)
1.	Kidney damage with normal or increased GFR	>90 (with kidney damage)
2.	Kidney damage with mild decrease in GFR	60 - 89
3a.	Mild to Moderate decrease in GFR	45 - 59
3b.	Moderate to severe decrease in GFR	30 - 44
4.	Severe decrease in GFR	15 - 29
5.	Kidney failure	<15 (or dialysis)

Clinical Features:

Asymptomatic

Younger patients may present with peri-orbital swelling early in the disease process

Swelling which may then progressively worsen over time and lead to ascites and anasarca.

Hypetension develops in majority of patients with CKD Congestive cardiac failure or cerebrovascular disease.

Reduction in urine output (oliguria)
Features of uraemia: Gastrointestinal system - nausea, vomiting, hiccups, diarrhoea, epigastric pain and rarely haematemesis.
Chest- central chest pain of pericarditis or breathlessness with development of pulmonary oedema.
Central nervous system-, tremors, derangement of sleep rhytmn, drowsiness, seizures and stupor or coma.
Skin- dryness and pruritus
Haemopoietic system- anaemia (pallor) and bleeding diathesis.

Diagnostic criteria: As in the definition and table above.

Differential Diagnoses
Severe acute kidney injury with cortical necrosis
Poisoning with multiple organ involvement.
Chronic Congestive cardiac failure (Cardio-renal syndrome).

Complications:
Anaemia
CKD Mineral and Bone Disease
Congestive cardiac failure
Cerebrovascular disease
Peripheral vascular disease
Malnutrition

Relevant investigations and management.

Urine Examination (Volume / microscopy /Urinalysis / Electrolytes).Serum chemistry (e.g. creatinine, urea, K): This would

- Allow estimation of GFR using various formulae and staging of the CKD
- For planning or initiation of renal replacement therapy
- To monitor treatment outcomes or improvement.

Haematology including serology:

- To assess the Full Blood Counts, clotting profile and monitor response to treatment with ral or parenteral iron and Erythropoietin stimulating agents
- Serological tests for HBV, HCV and HIV are necessary to be able to isolate infectious patients while applying general precautions in the management of all patients.

Imaging (e.g. USS, IVU, RUCG etc.):

Renal ultrasonography remains the simplest non-invasive diagnostic tool for patients with CKD.

Provides information on size, shape, preservation of cortical thickness and cortico-medullary differentiation as well as echogenicity of the kidneys, which are usually deranged in patients with advanced CKD.

Other structural abnormalities like polycystic kidney disease or hydronephrosis may be

discovered and may suggest need for other imaging modalities particularly in patients with obstructive uropathy.

Renal biopsy: Renal biopsy is mandatory in patients that present with some conditions. These include

Any evidence of glomerular disease

- nephrotic range proteinuria
- sub-nephrotic range proteinuria with or without haematuria
- RBC cast
- Presence of persistent microscopic haematuria
  - Suspicion of connective tissue disease
  - Presence of familial type of nephrotic syndrome
  - Determine the prognosis and response to therapy.

Principles of management of patients with CKD include:

- Counselling to discourage use of nephrotoxins in whatever form and ensure good compliance.
- Good Blood Pressure control.
- Reduction of proteinuria and ensuring maximum renoprotection
- Maintenance of fluid homeostasis
- Control of biochemical abnormalities
- Maintenance of nutrition
- Maintenance of haemoglobin between

11-12g/L
- Maintenance of normal calcium-phosphate homeostasis
- Treatment of any complicating infection
- Treat the underlying cause (more useful if discovered early)
- Dialysis where indicated

What to do when CKD is detected:
- Stage the disease
- Take appropriate measures depending on the stage:

  Stage 1 – 2 : where the cause of CKD is known, treat underlying cause and institute measures to retard progression.

  Stage 1-2 : where cause is unknown, refer to Nephrologist.

  Stage 3-5: refer to Nephrologist.

Treatment of hypertension:

Combination of different groups of antihypertensive drugs is the rule but should include maximal doses of either ACEI or ARBs to achieve target BP.

Blood Pressure Targets:

Proteinuria < 1g/24hrs - - - - - - - - - 130/80mmHg

Proteinuria >1g/24hrs - - - - - - - - - 120/75mmHg

CKD with diabetes - - - - - - - - 120/75mmHg

In children ——— Less than 90th percentile for the age, sex and height.

Anaemia is the commonest haematological complication of CKD and worsens with deterioration of renal function. ***All CKD patients should be screened for anaemia at time of diagnosis and thereafter, at least every 3 months.***

Targets for anaemia treatment:

Predialysis: Hb - 11 – 12g/dl, serum ferritin 100-500ng/ml, TSAT ≥20%

Dialysis: Hb - 11 – 12g/dl, serum ferritin 200 – 500ng/ml, TSAT ≥20%

In all patients, avoid Hb level > 13g/dl because of risk of haemoconcentration and its effect on morbidity and increased cardiovascular mortality[4].

Treatment of anaemia:

1) In predialysis patients and patients receiving peritoneal dialysis or home haemodialysis,optimize iron balance before Epoetin therapy using oral iron. If poor response (TSAT is < 20% and serum ferritin < 100ng/ml for 4 weeks), switch to parenteral iron.

   Dose of oral iron: Ferrous sulphate 200mg or Ferrous gluconate 600mg three times daily (approx. 65mg elemental iron) or 2-6 mg /kg /day of elemental iron for

paediatric patients

Dose of parenteral iron: Iron sucrose intravenously 200mg weekly for 5 weeks (total of 1000mg) or Iron dextran intravenously 250mg weekly for 4 weeks. For patients in whom adherence may be difficult, total dose infusion should be considered as its been found to be effective and safe[5,6].

2) For patients on in-center haemodialysis, start with parenteral iron at 100mg in the last 30-60 minutes of the dialysis session to a total of 1000mg.In cases of poor or inadequate dialysis , higher doses may be given to ensure achieving 1000mg within 4 weeks.

- ***Test dose of iron dextran should be administered before the full dose.***
- ***For patients that have received blood transfusions, check iron stores (serum ferritin) before giving supplemental iron because of risk of iron overload.***

3) Erythropoiesis Stimulating Agents (ESAs)

These should be administered preferably after iron deficiency has been corrected and BP controlled.

Table 2: Types of ESAs and their characteristics

ESA	CLASS	HALF - LIFE	STORAGE CONDITIONS	RISK OF PRCA
Erythropoietin beta	Short acting	8.8 hours	2-8°C normally, but can be kept at 25°C for 3-5 days	minimal

- Short acting ESAs – Epoetin alfa and Epoetin beta.
- Intermediate acting ESAs – Darbepoetin alfa-
- Long acting ESAs – CERA

Dialysis:

Based on manifestation of clinical and biochemical features of uraemia or development of electrolyte and acid-base complications of CKD.

The clinical features include:

Encephalopathy,

Pulmonary oedema,

Persistent nausea and vomiting,

Pericarditis, refractory oedema,

Uncontrolled HT

Bleeding diathesis.

The biochemical indications include:

- hyperkalemia >6.5mmol/l,
- serum bicarbonate <12mmol/l,
- urea >25 mmol/l,
- creatinine >600micromol/l

Manifestations of features of hypercatabolism:

- K+ rate of rise >1mmol/day
- urea rate of rise >10 mmol/day
- creatinine rate of rise >100micromol/day

To maintain good calcium phosphate homeostasis:

- Dietary phosphate intake should be

limited in patients with hyperphosphatemia. Phosphate-binding agents are required in the treatment of hyperphosphatemia.

For choice of phosphate binder, it is reasonable to take into account the following:

- Serum calcium,
- CKD stage,
- Presence of other components of CKD-MBD,
- Concomitant therapies
- Side-effect profile of the drug.

The dose of calcium- based phosphate binders should be restricted in the presence of arterial calcification and/or adynamic bone disease. In such instances the use of non-calcium based phosphate binders (e.g. Sevelamer HCl, lanthanum carbonate etc) could be used.

Hypocalcaemia, is treated with calcium salts and active vitamin D analogues. Calcium intake should however, be restricted in patients with hypercalcemia, soft tissue calcification, low PTH and in patients with adynamic bone disease.

Calcitriol or vitamin D analogs, or calcimimetics, or a combination of calcimimetics and calcitriol or vitamin D

analogs may be used to lower PTH to two to nine times the upper normal limit for the assay in treated patients.
Parathyroidectomy is indicated when medical therapy fails.

Dialysis: The patients could benefit from:

Maintenance Haemodialysis(HD) (Twice or Thrice weekly HD),
Continuous Ambulatory Peritoneal Dialysis (CAPD)
Kidney Transplantation.

NEPHROTIC SYNDROME

Introduction

A clinical complex characterized by
- Proteinuria of 3.5 g per 24 hours
- Hypoalbuminaemia
- Generalized oedema
- Hyperlipidaemia;
- lipiduria
- Hypercoagulability

Aetiology

Idiopathic in a significant proportion of cases
Known causes include:
- Inflammatory diseases of the glomeruli (glomerulopathies)
- Viral infections e.g. Hepatitis B, HIV
- Immunologic disorders e.g. SLE

Allergies: insect bites, poisonous plants

Intravenous drugs e.g. heroin Others:
- Diabetes mellitus
- Carcinomas
- Amyloid deposition

Histologic types
Minimal change disease
Focal segmental glomerulosclerosis
Membranous glomerulopathy
Mebrano- proliferative glomerulonephritis
Mesangio-proliferative glomerulonephritis

Clinical features
Generalized body swelling
Passage of frothy urine

Complications
Peripheral arterial or venous thrombosis
Acceleration of atherosclerosis
Protein malnutrition
Vitamin D deficiency
Increased susceptibility to infections
Iron-resistant microcytic hypochromic anaemia

Differential diagnoses
Other causes of body swelling
- Congestive heart failure
- Decompensated chronic liver disease
- Protein losing enteropathy

Investigations

Blood:
- Serum proteins
- Serum lipids Urine:
- Urinalysis
- 24 hour urine collection for protein estimation
- Abdominal ultrasound scan
- Renal biopsy

Treatment objectives
Reduce proteinuria
Eradicate peripheral oedema

Drug treatment
Diuretics e.g. loop diuretics like furosemide
Glucocorticoids (e.g. prednisolone)
- If renal biopsy and histology reveal a steroid-responsive cause of the nephrotic syndrome

Cytotoxic drugs (e.g. cyclophosphamide) in some steroid-resistant cases

Prevention
Avoid nephrotoxins

Treat bites and stings to prevent p haemolytic streptococcal infection

SEXUALLY TRANSMITTED INFECTIONS

BACTERIAL VAGINOSIS

Introduction

A clinical syndrome resulting from

replacement of the normal hydrogen peroxide-producing *Lactobacillus* sp. in the vagina by high concentrations of anaerobic bacteria, such as

Gardnerella vaginalis Mycoplasma hominis Mobiluncus curtisii

The cause of the microbial alteration is not fully understood

The associated malodour is due to the release of amines produced by anaerobic bacteria that decarboxylate lysine to caverdine, and arginine to putrescine

Predisposing factors are the use of antiseptic/antibiotic vaginal preparations or vaginal douching

Clinical features

Malodorous and increased white vaginal discharge that is homogenous, low in viscosity, and uniformly coats vaginal walls

The fishy-smelling discharge is particularly noticeable after sexual intercourse; usually no pruritus or inflamed vulvae

Differential diagnoses

Other causes of vaginal discharge: see Gonorrhoea

Complications

Acute salpingitis

Premature rupture of membranes

Preterm delivery and low birth weight

Investigations

Homogeneous milky discharge with pH > 4.5 (pH > 6.0 highly suggestive)

Fishy odour from the biogenic amines; altered by addition of 10% KOH (Sniff test)

Clue cells on a wet mount

- Clue cells are normal vaginal epithelial cells studded with bacteria, giving the cells a granular appearance

Treatment objective

To eliminate the organisms

Drug therapy

Recommended regimen:

- Metronidazole 400 mg orally, every12 hous for 7 days

Alternative regimen:

- Metronidazole 2 g orally, as a single dose

Or:

- Metronidazole 0.75% gel 5 g intravaginally, twice for 7 days

Notable adverse drug reactions, caution

Metronidazole: see Trichomoniasis

Advise to return if symptoms persist as re-treatment may be needed

Recommended regimen for pregnant women

Metronidazole 200 orally, every 8 hours for 7

days, after the first trimester
Or: 2gorally, asasingledose
If treatment is imperative in the first trimester of pregnancy
- Give metronidazole 2 g orally as a single dose

Notable adverse reactions, caution and contraindications
Metronidazole: Causes a disulfiram-like reaction with alcohol
Avoid high doses in pregnancy and breast feeding
May cause nausea, vomiting, unpleasant taste, furred
tongue, and gastro-intestinal disturbances
Generally not recommended for use in the first trimester of pregnancy

Prevention
Reduce or eliminate predisposing factors such as antiseptic/antibiotic vaginal preparations or vaginal douching
Treat symptomatic pregnant women
Screen pregnant women with a history of previous pre-term delivery to detect asymptomatic infections
Retreat pregnant women with recurrence of symptoms
Counselling, Compliance, Condom use and Contact treatment

CHANCROID (Ulcus Molle, Soft Chancre)

Introduction

An infectious disease caused by *Haemophilus ducreyi*, a small gram-negative bacillus

Common in the tropics, especially in Africa, the Far East, and the Caribbean

Persons may present with chancroid outside endemic regions; sporadic outbreaks of infection occur in Europe and North America

Clinical features

Incubation period is about 3 - 7 days

Begins as a small, tender papule, changing into a pustule which rapidly progresses to a painful ulcer with a bright red areola

Neither the edge nor base of the ulcer is indurated (unlike syphilis)

- The ulcer feels soft, hence the name 'soft sore' (ulcus molle)

With superimposed bacterial infection it often feels indurated

The ulcers may be multiple due to auto-inoculation

Sites of predilection in men are the prepuce, frenulum, glans or shaft of the penis

In women the labia, fourchette, vestibule, clitoris, cervix, or perineum are favored sites

Lesions may cause dyspareunia, pain on voiding or defaecation and vaginal discharge

Women may be asymptomatic carriers

About 7 - 14 days after the appearance of the ulcer, a bubo appears

- A mass of gland smatted together, of tenad herent to the overlying skin

The glands above the inguinal ligament are usually affected, and often there is a unilateral enlargement

Central softening is often found and if untreated the bubo may rupture and discharge through a fistula

The combination of a painful genital ulcer and suppurative inguinal adenopathy is almost pathognomonic of chancroid

Patient may present with bubo, the initial ulcer having healed

Atypical lesions have been reported in HIV-infected individuals

- More extensive, or multiple lesions sometimes accompanied by systemic manifestations such as fever and chills

Complications

Progressive ulceration and amputation of the phallus, particularly in HIV patients

Differential diagnoses

Other causes of genital ulcers:
Syphilis
Herpes
Granuloma inguinale
Lymphogranuloma venereum

Fixed drug eruption
Erythema multiforme
Behcet's disease
Trauma
Tuberculous ulcer
Cancers

Investigations

Microscopy, culture and sensitivity of discharge from ulcer
Serological tests e.g. complement fixation (CF); microimmuno-fluorescence (MIF) test; PCR

Treatment objectives

Same as for Gonorrhoea

Drug therapy

Recommended regimen: Ciprofloxacin 500 mg orally every 12 hours for 3 days

Or:
Erythromycin 500 mg orally every 6 hours for 7 days

Or:
Azithromycin 1 g orally as a single dose
Alternative regimen:
Ceftriaxone, 250 mg by intramuscular injection, as a single dose

Adjuvant therapy

Keep ulcerative lesions clean Aspirate fluctuant lymph nodes through the surrounding healthy skin, preferably from a superior approach to prevent persistent dripping and sinus formation

Incision and drainage, or excision of nodes may delay healing and is not recommended

Follow-up

All patients should be followed up until there is clear evidence of improvement or cure

In patients infected with HIV, treatment may appear to be less effective, but this may be a result of co-infection with genital herpes or syphilis

Chancroid and HIV infection are closely associated and therapeutic failure is likely to be seen with increasing frequency

- Patients should therefore be followed up weekly until there is clear evidence of improvement

Notable adverse drug reactions, caution and contraindications

Ciprofloxacin and ceftriaxone (see gonorrhoea)

Erythromycin and azithromycin (see chlamydia)

Prevention

Counselling, Compliance, Condom use and

Contact treatment

CHLAMYDIAL INFECTION (Other than Lymphogranuloma venereum)

Introduction

The chlamydiae occupy a special place between bacteria and viruses

- They are a large group of obligate intracellular organisms

Chlamydia trachomatis has a number of serovars and causes many different human infections

- Eye: trachoma; inclusion conjunctivitis
- Genital tract: lymphogranuloma venereum, non-gonococcal urethritis, cervicitis, salpingitis
- Respiratory tract: pneumonia

C. trachomatis immunotypes D - K are isolated in about 50% of cases of non-gonococcal urethritis and cervicitis by appropriate techniques

Clinical features

Infections are asymptomatic, but when an incubation period can be determined, it is usually about 10 - 20 days

Co-infection with gonococci and chlamydiae is common

C. trachomatis is an important cause of non-gonococcal urethritis in males, and in females cervicitis, salpingitis, or pelvic inflammatory

disease

Urethral or cervical discharge tends to be less painful, less purulent, and watery in chlamydial compared with gonococcal infection

On physical examination, the cervix may show contact bleeding in addition to the discharge

A patient with urethritis or cervicitis and absence of gram-negative diplococci on Gram stain and of *N. gonorrhoeae* on culture is assumed to have chlamydial infection

Complications

Epididymo-orchitis and sterility in males.

Pelvic inflammatory disease (PID) and infertility in females.

Adverse pregnancy outcomes.

Conjunctivitis and pneumonia in the newborn

Differential diagnoses

Other causes of urethral and vaginal discharge (see Gonorrhoea)

Investigations

Microscopy, culture and sensitivity (of discharge)

Direct immunofluorescence assay

Enzyme-linked immunoassay

DNA probe test

Ligase chain reaction (LCR)

Treatment objectives

Same as for gonococcal infection

Drug therapy

Recommended regimen: Doxycycline 100 mg orally, every 12 hours for 7 days

Or:

Azithromycin 1 g orally, in a single dose

Chlamydial infection during pregnancy

Recommended regimen:

Erythromycin 500 mg orally every 6 hours for 7 days

Or:

Amoxycillin 500 mg orally every 8 hours for 7 days

Neonatal chlamydial conjunctivitis

Typically has an incubation period of 10 - 14 days compared to 2 - 3 days for gonococcal opthalmia Recommended regimen:

Erythromycin syrup 50 mg/kg per day orally, every 6 hours for 14 days

Alternative regimen:

Trimethoprim 40 mg with sulfamethoxazole 200 mg orally, every 12 hours for 14 days

Note

There is no evidence that additional therapy with a topical agent provides further

benefit

If inclusion conjunctivitis recurs after therapy has been completed, erythromycin treatment should be reinstituted for 2 weeks

It is important to treat the mother and her sexual partner

Notable adverse drug reactions, caution and contraindications

Doxycycline and tetracycline

- Caution in patients with hepatic impairment, systemic lupus erythematosus and myasthenia gravis
- Antacids, aluminium, calcium, iron, magnesium and zinc salts, and milk decrease the absorption of tetracyclines
- Deposition of tetracyclines in growing bones and teeth (by binding to calcium) causes staining and occasionally dental hypoplasia
- Should not be given to children under 12 years, or to pregnant or breast-feeding women
- With the exception of doxycycline, tetracyclines may exacerbate renal failure and should not be given to patients with kidney disease
- May cause nausea, vomiting and diarrhoea; hypersensitivity reactions. Headache and visual disturbances may indicate benign intracranial hypertension
- Candidal superinfection with prolonged

therapy

Azithromycin and Erythromycin

- Erythromycin estolate is contraindicated during pregnancy because of drug-related hepato-toxicity; only erythromycin base or erythromycin ethylsuccinate should be used
- Erythromycin should not be taken on an empty stomach
- Caution in persons with arrhythmias
- Infants should be followed up for symptoms and signs of infantile hypertrophic pyloric stenosis (has been reported in infants less than 6 weeks exposed to this drug)

Ofloxacin See ciprofloxacin- Gonorrhoea

Amoxicillin

- Caution where there is a history of allergy
- Erythematous rashes common in glandular fever, cytomegalovirus infection, acute or chroni lymphocytic leukaemia with pityriasis rosea, and allopurinol use

Prevention

Counselling, Compliance, Condom use and Contact treatment

GONORRHOEA

Introduction

Caused by *Neisseria gonorrhoeae,* a gram-negative aerobic diplococcus

It prefers the columnar epithelium of the urethra, the cervical canal, the rectum and the conjunctivae.

The keratinizing epithelium of the adult vagina is quite resistant to *N. gonorrhoeae*, but that of the pre-pubertal girls, pregnant women and the elderly is more easily colonized

Occasionally *N. gonorrhoeae*reaches the blood stream causing sepsis

Gonorrhoea in males

Clinical features

Presents as foul-smelling urethral discharge of pus with dysuria 2 - 6 days after exposure

Some patients have a scanty discharge that cannot be distinguished from non-gonococcal urethritis

Often asymptomatic during the day but there may be a drop of discharge in the morning

Urethral orifice is usually inflamed; there may be balanitis because of the irritation from the discharge and secondary infection

About half of infected males are asymptomatic

Ascending infection is common and may lead to inflammation of the epididymis (epididymitis)

Epididymitis usually manifests by acute onset of unilateral testicular pain and

swelling, often with tenderness of the epididymis and vas deferens

- Occasionally there is erythema and oedema of the overlying skin
- The adjacent testis is often also inflamed (orchitis), giving rise to epididymo-orchitis

Complications

Local complications (now uncommon):

Littre abscess involving periurethral glands

Paraurethral abscesses

Proximal urethral involvement with frequency and terminal haematuria

Cowper's gland abscess involving the bulbourethral glands, producing a swelling behind the base of the scrotum that can produce a proximal or Cowper's stricture

Prostatitis Proctitis

Urethral stricture leading to hydroureters and hydronephrosis

Chronic epididymo-orchitis leading to sterility

Contaminated fingers or other fomites can also lead to infection of the eyes- gonococcal conjunctivitis

- Haematogenous spread leading to meningitis, arthritis etc

Differential diagnoses

Urethral discharge:

Spermatorrhoea/prostatorrhoea (sexual arousal)

- *Trichomonas vaginalis and Candida albicanscan* also give rise to urethral discharge and balanitis Ascending infections:

Escherichia coli, a common cause in the insertive male homosexuals

- Other organisms may be transmitted non-sexually following genitourinary infections, surgery and instrumentation (including catheterization)

Scrotal swelling (epididymo-orchitis): In older men, where there may have been no risk of STIs, other general infections may be responsible, e.g. *Escherichia coli, Klebsiella* spp. or *Pseudomonas aeruginosa*

Tuberculous epididymo-orchitis, secondary to lesions elsewhere, especially in the lungs or bones

Brucellosis, caused by *Brucella melitensis or Brucella abortups*

- Orchitis is usually clinically more evident than an epididymitis

In pre-pubertal children the usual aetiology is coliform, pseudomonas infection or mumps virus Non-infectious causes of scrotal swelling:

Trauma (haematocoele)

Testicular torsion

Tumour
Hydrocoele of the tunica vaginalis
Cyst of epididymis
Varicocoele
Inguinoscrotal hernia

Investigations
Urethral swab for microscopy and culture and sensitivity

Gonorrhoea in women

Clinical features
Inflammation of the cervix and cervical canal (cervicitis) is the commonest presentation in women
Urethritis: the urethra becomes the most common site in women who have had hysterectomy
The most frequent complaint is discharge, often accompanied with burning on urination
Over 50% of infected women are asymptomatic
Oropharyngeal gonorrhoea from orogenital sex (fellatio) may present as sore throat

Complications
Local:
Infections of Skene's periurethral glands and Bartholin's labial glands; a Bartholin's gland abscess may cause pain on sitting or walking

Vulvitis
Ascending infection to the endometrium, fallopian tubes, ovaries and peritoneum (pelvic inflammatory disease)
Ectopic pregnancy
Infertility
Perihepatic abscess (Fitz-Hugh-Curtis syndrome)
Risk of disseminated gonococcal infection during pregnancy and menstruation
Risk to the newborn infant:

- Premature rupture of membranes
- Premature labour
- Chorioamnionitis
- Septic abortion
- Ophthalmia neonatorum
- Oropharyngeal gonorrhoea

Differential diagnoses
Other causes of vaginal discharge:
Accentuation of physiological discharge

- Premenstrually
- At the time of ovulation
- In pregnancy
- Use of contraceptive pills or an intrauterine device

Infective causes:

- Candidiasis
- Trichomoniasis
- Bacterial vaginosis

- Chlamydia
- Cervical herpes genitalis
- Cervical warts
- Syphilitic chancre
- Toxic shock syndrome *(Staphylococcus aureus)*
- P-haemolytic streptococcal infection, *Mycoplasma* infection

Non-infective causes:

- Cervical ectropion
- Cervical polyp(s)
- Neoplasia e.g. cancer of the cervix
- Retained products (tampon, post-abortion, post-natal)
- Trauma
- Semen (post-coital)
- Contact irritants and sensitizers e.g. from douches or feminine hygiene sprays
- Bullous diseases of the mucous membranes

Investigations

Endocervical swab (through a vaginal speculum) for microscopy, culture and sensitivity

Gonorrhoea in children

Clinical features

Sexual abuse is a common cause of gonorrhoea in young girls

Usually symptomatic in young girls

Pruritus and dysuria are common complaints
Discharge may cause irritant dermatitis of the upper thighs

Differential diagnoses
Other causes of vaginal discharge in young girls:
A vaginal foreign body such as a small toy, bead, or even a piece of food
Other infections caused by *T. vaginalis*, and *C. albicans*
Intestinal bacteria or pin worms due to inadequate cleaning after defeacation

Ophthalmia neonatorum
Gonococcal conjunctivitis in the neonate can be acquired perinatally
Purulent conjunctivitis; the lids swell; eyes are red and tender
If not treated promptly, the cornea may be eroded and perforated, leading to secondary glaucoma, conophthalmus and blindness
About 30% of babies infected will also have oropharyngeal gonorrhoea

Differential diagnoses
The silver nitrate prophylaxis can produce a chemical conjunctivitis, usually appearing 6 - 8 hours after treatment and resolving over 24 hours
The most common cause of neonatal

conjunctivitis in most countries is *C. trachomatis, E. coli, Staphylococci, Streptococci* and *Pseudomonas* sp. can also cause conjunctivitis in the neonate.

Treatment objectives

Eliminate the organism in the patient and sexual partner(s)

Prevent re-infection

Prevent complications

Counsel and screen for possible co-infection with HIV so that appropriate management can be instituted

Drug therapy

Recommended regimen:

Ciprofloxacin 500 mg orally, as a single dose

Or:

Ceftriaxone 125 mg by intramuscular injection, as a single dose

Neonatal gonococcal conjunctivitis

Recommended regimen:

Ceftriaxone 50 mg/kg by intramuscular injection, as a single dose, to a maximum of 125 mg

mg/kg

Note

Single-dose ceftriaxone and kanamycin are of proven efficacy

The addition of tetracycline eye ointment to these regimens is of no documented benefit

Adjunctive therapy for gonococcal ophthalmia

- Systemic therapy, as well as local irrigation with saline or other appropriate solution
- Irrigation is particularly important when the recommended therapeutic regimens are not available
- Careful hand washing by personnel caring for infected patients is essential

Follow-up

Review patients after 48 hours

Notable adverse drug reactions, caution and contraindications

Ciprofloxacin

- Avoid in pregnancy and breast feeding; children below 12 years
- Reduce dose in renal impairment

Ceftriaxone

- Caution in persons with known sensitivity to beta-lactam antibiotics
- May cause diarrhoea (and rarely antibiotic-associated colitis); nausea, vomiting and abdominal discomfort

Prevention

Counselling, Compliance, Condom use and Contact treatment

Ocular prophylaxis provides poor

protection against *C. trachomatis* conjunctivitis

Prevention of ophthalmia neonatorum

Clean the eyes carefully immediately after birth. The application of 1% silver nitrate solution or 1%

tetracycline ointment to the eyes **of all infants** at the time of delivery is strongly recommended as a prophylactic measure

Infants born to mothers with gonococcal infection should receive additional antibiotic treatment (as those with clinical neonatal conjunctivitis)

GRANULOMA INGUINALE (Donovanosis Granuloma venereum)

Introduction

A mildly contagious disease caused by *Klebsiella granulomatis*

Currently rare in several parts of Africa Endemic in Southeast Asia, Southern India, the Caribbean and South America

Clinical features

A chronic mildly contagious disease with a potentially progressive and destructive character

Incubation period ranges from 10 - 40 days. The early lesion is a papule or nodule which soon becomes ulcerated and has an offensive discharge

The floor of the ulcer may be covered with a dirty grey material; its walls may be overhanging, or a papillomatous fungating mass may arise from the growth of vegetations

Progressive indolent, serpiginous ulceration of the groins, pubis, genitals and anus may form.

Pain on walking may be excrutiating

Persisting sinuses and hypertrophic depigmented scars are fairly characteristic

Regional lymph nodes are not enlarged but with cicatrisation, the lymph channels may be blocked causing pseudoelephantiasis of the genitalia. Both the fibrotic scarring and elephantiasis-like lesion could cause obstructed labour

Subcutaneous extension and abscesses may occur and form a pseudo-bubo in the inguinal region

Healing is unlikely without treatment; the locally destructive lesion may eventually involve the groins, pubis and anus

A squamous cell carcinoma may arise from chronic lesions.

Differential diagnoses

Syphilis Chancroid

Lymphogranuloma venereum

Lupus vulgaris

Deep mycosis

Amoebic ulcer
Pyoderma gangrenosum
Squamous cell and basal cell carcinoma

Complications
Obstructed labour
Squamous cell carcinoma

Investigations
Direct microscopy

Treatment objectives
Same as for gonococcal infection

Drug therapy
Recommended regimen: Azithromycin-1 gorallyonfirstday,then500mgorally,once a day

Or:
Doxycycline
- 100 mg orally every 12 hours

Therapy should be continued until the lesions have completely epithelialized
Alternative regimen:
Erythromycin
- 500 mg orally every 6 hours

Or:
Tetracycline 500 mg orally every 6 hours

Or:

Trimethoprim 80 mg/sulfamethoxazole 400 mg, 2 tablets orally, 12 hourly
All treatment should be for a minimum of 14 days

Note

The addition of a parenteral aminoglycoside such as gentamicin should be carefully considered for treating HIV-infected patients

Follow-up

Patients should be followed up clinically until signs and symptoms have resolved

Notable adverse drug reactions, caution and contraindications

Sulfamethoxazole/trimethoprim

- Contraindicated in persons with hypersensitivity to sulfonamides or trimethoprim; porphyria
- Caution required in renal impairment (avoid if severe); hepatic impairment (avoid if severe); maintain adequate fluid intake (to avoid crystalluria)
- May cause nausea, vomiting, diarrhoea, headache, hypersensitivity reactions, including fixed drug eruption, pruritus, photo-sensitivity reactions, exfoliative dermatitis, and erythema nodosum

Others

- See Chlamydia

Prevention

Counselling, Compliance, Condom use and Contact treatment

LYMPHOGRANULOMA VENEREUM

(Climatic bubo; lymphogranuloma inguinale;! lymphopathia venereal; Durand-Nicolas-Favrq Disease)

Introduction

A chronic disease caused by *Chlamydia trachomatis* (serotypes L1, L2, L3), an obligate intracellular microorganism

Most common in Asia, Africa, and South America In Europe and North America, it is most prevalent among homosexuals, immigrants from endemic areas and people returning from endemic areas, such as soldiers, seamen, and vacationers

Clinical features

A chronic granulomatous, locally destructive disease that is characterized by progressive, indolent, serpiginous ulceration of the groins, pubes, genitals and anus

May be classified into primary, secondary, and late stages

Primary stage

After an incubation period of 7 - 15 days, a papule or small non-indurated painless ulcer appears - Usually goes unnoticed

Extra-genital lesions (rectal, oral) have also been described

Women probably act as asymptomatic carriers

Patients are very rarely seen at the primary stage

Secondary stage

About 3 - 6 weeks post-contact a uni-or bilateral massive inguinal lymphadenopathy (bubo) appears

The glands elongate along the Poupart's ligament to become sausage shaped

Buboes progress to involve the glands above and below the ligament, so that the depression formed by the ligament which separates these two groups of glands gives the "sign of the groove"

Pain in the gland is usual, and as the glands are matted together, the overlying skin develops an erythematous or violaceous hue

The glands eventually become fluctuant, break down and discharge

Inguinal lymphadenopathy occurs in only 20 - 30% of women with LGV

There is primary involvement of the rectum, vagina, cervix, or posterior urethra, which drain to the deep iliac or perirectal nodes

- This may produce symptoms of lower abdominal or back pain

Systemic symptoms usually present with:

- Fever
- Malaise
- Arthritis
- Loss of weight

Skin manifestations (erythema nodosum, papulo-pustular lesions and photodermatosis)

- Raised ESR

Late stage

Spontaneous remission is common, though some patients enter the late stage

Characterized by disfiguring and destructive sequelae Impairment of the lymphatic drainage from fibrotic scarring leads to distant oedema and gross elephantiasis of the genitalia

- There could be associated anorectal and vaginal strictures

Complications

Systemic spread of *C. trachomatis* in the secondary stage resulting in arthritis, pneumonia, hepatitis or rarely perihepatitis

Other rare systemic complications include pulmonary infection, cardiac involvement, aseptic meningitis, and ocular inflammatory disease

The late stage may be complicated by the genito-anorectal syndrome

- Reported more in homosexual men, and

women who engage in receptive anal intercourse

Patients may also complain of fever, pain, and tenesmus. Obstructed labour from elephantiasis of the vulva

Differential diagnoses

Buboes:

- Chancroid
- Infections of the lower limbs
- Hodgkins disease and other lymphomas
- Plague
- Tularemia Late stage:
- Tuberculosis
- Deep mycosis of the genitalia
- Squamous cell or basal cell carcinoma

Investigations

Culture and cell typing of the isolate from an aspirate of involved lymph node

Serological tests e.g. CFT and MIF; PCR

Treatment objectives

Same as for gonorrhoea

Drug treatment

Recommended regimen: Doxycycline

- 100 mg orally every 12 hours for 14 days

Or:

Erythromycin

- 500 mg orally every 6 hours for 14 days

Alternative regimen:

Tetracycline

- 500 mg orally every 6 hours for 14 days

Adjuvant measures

Aspirate fluctuant lymph nodes through healthy skin Incision and drainage or excision of nodes may delay healing and is not recommended

Some patients with advanced disease may require treatment for longer than 14 days, and sequelae such as strictures and/or fistulae may require surgery

Notable adverse drug reactions, caution and contraindications

See Chlamydia

Prevention

Counselling, Compliance, Condom use and Contact treatment

SYPHILIS

Introduction

Infection caused by the spirochaete *Treponema pallidum*

Occurs worldwide Can be classified as:

Congenital (transmitted from mother to child*in utero)*

Acquired (through sex or blood transfusion)

Acquired syphilis may be early or late

Primary syphilis is characterized by an ulcer or chancre at the site of infection or inoculation

Manifestations of secondary syphilis include a skin rash, condyloma lata, mucocutaneous lesions and generalized lymphadenopathy

Early syphilis: primary, secondary and early latent stages

Primary syphilis: an ulcer or chancre at the site of infection or inoculation

Secondary syphilis: skin rash, condyloma lata, **mucocutaneous lesions and generalized**lymphadenopathy

Late syphilis: late latent syphilis, gummatous, neurological and cardiovascular syphilis

This section is only on primary syphilis

Clinical features

After an incubation period of 2 - 4 weeks (full range 90 days) the first lesion of syphilis may appear at the site of exposure, most commonly, the genitals

Chancres may also be located on the lips or tongue; ano-rectal chancres frequently seen in male homosexuals - Begins as a small, dusky-red macule which soon develops into a papule

The surface of the papule erodes to form an ulcer which is typically round and painless with a clean surface and exudes a scanty yellow serous discharge teeming with spirochaetes

Lesion is indurated and feels firm or hard on palpation; surrounding skin is oedematous

Regional inguinal (or generalized) lymphadenopathy follows

The glands are painless, moderately enlarged (not buboes), discrete and never suppurate

Atypical lesions may be seen for various reasons e.g. bacterial superinfection, trauma or co-infection with chancroid.

Even without treatment, the primary lesion(s) gradually heals up and will disappear after approximately 3 - 8 weeks, sometimes leaving a thin atrophic scar which is easily overlooked

Differential diagnoses

Other causes of genital ulcers:

Chancroid Herpes

Lymphogranuloma venerum

Granuloma inguinale

Trauma

Fixed drug eruption

Behcet's disease

Erythema multiforme

Tuberculous ulcer

Amoebic ulcer
Cancer

Complications

Phimosis and paraphimosis Late syphilis: gummatous, neurological and cardiovascular syphilis

Investigations

Dark field examination
Direct fluorescent antibody tests of lesion exudates or tissue
VDRL
RPR

Treatment objectives

Eliminate the organism in the patient and sexual partner(s)
Prevent re-infection
Prevent complications
Counsel and screen for possible co-infection with HIV so that appropriate management can be instituted

Drug therapy

Recommended regimen: Benzathine benzylpenicillin - 4g (2.4 million units) by intramuscular injection, ata single session

- Because of the volume involved, this dose is usually given as two injections at separate sites Alternative regimen for

penicillin-allergic (non -pregnant) patients

Doxycycline

- 100 mg orally, every 12 hours for 14 days

Or:

- Tetracycline 500 mg orally, every 6 hours for 14 days

Alternative regimen for penicillin-allergic pregnant patients

Erythromycin

- 500 mg orally, every 6 hours for 14 days

Notable adverse drug reactions, caution and contraindications

Benzylpenicillin (Penicillin G)

- Caution in patients with history of allergy; atopic patients; in severe renal impairment, neurotoxicity; high doses may cause convulsions
- Contraindicated in penicillin hypersensitivity
- May cause hypersensitivity reactions including! urticaria, fever, joint pains, rashes, angioedema, anaphylaxis, serum sickness-like reaction, rarely intestitial nephritis, haemolytic anaemia, leucopaenia, thrombocytopaenia and coagulation disorders

Other antibiotics

- See Chlamydia

Prevention

Counselling, Compliance, Condom use and Contact treatment

All infants born to seropositive mothers should be treated with a single intramuscular dose of benzathine penicillin

- 50,000 units/kg, whether or not the mothers were treated during pregnancy (with or without penicillin)

Prevention of congenital syphilis is feasible

- Programmes should implement effective screening strategies for syphilis in pregnant women

Screening for syphilis should be conducted at the first prenatal visit

Some programmes have found it beneficial to repeat the tests at 28 weeks of pregnancy and at delivery in populations with a high incidence of congenital syphilis

TRICHOMONIASIS

Introduction

Caused by the flagellated protozoan, *Trichomonas vaginalis*

An extremely common infection, almost always transmitted via sexual contact

Women are far more frequently affected and more likely to have symptoms

Men are more likely to be asymptomatic and serve as carriers

Clinical features

Vaginal discharge: a white-yellow frothy discharge is characteristic

Burning sensation

Dysuria

Dyspareunia

The liabia are often swollen

The cervix may have punctuated haemorrhages producing a strawberry-like surface when viewed with a colposcope

Some men may have dysuria or a minimal urethral discharge and balanoposthitis

Co-infection with *N. gonorrhoeae* is common

Differential diagnoses

Other causes of vaginal discharge or urethral discharge: see Gonorrhoea

Complications

Acute salpingitis

Adverse pregnancy outcomes, particularly premature rupture of membranes, pre-term delivery and low birth weight

Investigations

Microscopy and culture of vaginal discharge

Treatment objectives

Eliminate the organism in the patient and sexual partner(s) Prevent re-infection Prevent complications

Counsel and screen for possible co-infection with HIV so that appropriate management can be instituted

Drug treatment

Recommended regimen:

Metronidazole

- 2g orally in a single dose

Or:

Tinidazole

- 2 gorally in a single dose

Alternative regimen:

Metronidazole

- 400 mg or 500 mg orally every 12 hours for 7 days

Or:

Tinidazole

- 500 mg orally every 12 hours for 5 days

Note

Other 5-nitroimidazoles are also effective, both in single and in multiple dose regimens

Asymptomatic women with trichomoniasis should be treated with the same regimen as symptomatic women

Recommended regimens for male urethral infections: same as for women

Patients not cured with the repeated course of metronidazole may be treated with a regimen consisting of metronidazole 2 g orally daily, together with 500 mg applied intravaginally

each night for 3 - 7 days

Vaginal preparations of metronidazole are available in many parts of the world, but are only recommended for the treatment of refractory infections, *not for the primary therapy of trichomoniasis* Recommended regimen for neonatal infections

Metronidazole

- 5 mg/kg orally, every 8 hours for 5 days

Infants with asymptomatic trichomoniasis, or urogenital colonization persisting past the fourth month of life should be treated with metronidazole

Notable adverse drug reactions, caution and contraindications

Metronidazole

Causes a disulfiram-like reaction with alcohol

- Avoid high doses in pregnancy and breast feeding
- May cause nausea, vomiting, unpleasant taste, furred tongue, and gastro-intestinal disturbances
- Generally not recommended for use in the first trimester of pregnancy

Prevention

Counselling, Compliance, Condom use and Contact treatment

VULVO-VAGINAL CANDIDIASIS

Introduction

Inflammation of the vagina and vulva, usually evolving from vaginal discharge and secondary external irritation

Candida albicans is the commonest cause of candidal vulvo-vaginitis; *Candida glabrata* has also been identified

Candidal vaginitis is most common in :

- Pregnancy
- Patients with diabetes mellitus
- Those on long-term antibiotic therapy or oral contraceptives
- Conditions associated with immune suppression
- Corticosteroid use

Usually not acquired through sexual intercourse

Because of the close proximity between the anus and female genitalia, re-infections may occur from the gastrointestinal tract

Clinical features

Up to 20% of women with the infection may be asymptomatic

If symptoms occur, they usually consist of vulval itching, soreness and a non-offensive vaginal discharge which may be curdy

Clinical examination:

Vulval erythema (redness) or excoriations from scratching

Vulval oedema
Erosions and crusting on the adjacent intertriginous skin
Although treatment of sexual partners is not recommended, it may be considered for women who have recurrent infections
A minority of male partners may have balanitis, which is characterized by erythema of the glans penis or inflammation of the glans penis and foreskin (balanoposthitis)

Differential diagnoses
Other causes of vaginal discharge: see Gonorrhoea in women

Complications
Emotional problems because of the recurrent nature of the infection, and dyspareunia
Very serious emotional problems in a non-sexually active person wrongly "accused" by parents, spouse or health care providers

Investigations
Positive KOH examination
Culture of vaginal discharges

Treatment objectives
Cure the infection
Prevent recurrence

Drug therapy

Recommended regimen: Clotrimazole 1% vaginal cream

- Insert 5 g at night as a single dose; may be repeated once if necessary

Or:

Miconazole 2% intravaginal cream

- Insert 5 g applicator once daily for 10 - 14 days or twice daily for 7 days

Or:

- Clotrimazole 500 mg intravaginally, as a single dose

Or:

- Fluconazole 150 mg orally, as a single dose
- Recommended topical regimen for balanoposthitis
- Clotrimazole 1% cream apply twice daily for 7 days

Or:

- Miconazole 2% cream twice daily for 7 days

Notable adverse drug reactions, caution and contraindications

Fluconazole:

- Caution in patients with renal impairment
- Avoid in pregnancy and breastfeeding
- Monitor liver function

- Discontinue if signs or symptoms of hepatic disease develop (risk of hepatic necrosis)
- May cause nausea, abdominal discomfort, diarrhoea, flatulence, headache, skin rash and Steven-Johnson syndrome
- Discontinue treatment or monitor closely if infection is invasive or systemic)

Prevention

Reduce or eliminate predisposing factors

After defecation cleaning should be done backwards to prevent faecal contamination of the vulva and vagina

UROLOGY

BENIGN PROSTATIC HYPERPLASIA

Introduction:

Non-cancerous enlargement of the prostate causing clinical symptoms

Increase in size impacts on the urethra and partially or completely obstructs urine outflow

A common cause of lower urinary tract obstruction among elderly males.

Pathological enlargement occurs usually after the age of 40.

Symptoms are due to mechanical obstruction (static cause) or spasm of the smooth muscles

in the prostate and around the bladder neck.

Clinical Features

Lower Urinary Tract Symptoms (LUTS);

a. Filling/Storage Symptoms
   - Daytime frequency
   - Urgency
   - Urgency incontinence
   - Nocturia

b. Voiding/Emptying Symptoms
   - Poor stream
   - Excessive straining
   - Hesitancy
   - Intermittency
   - Terminal dribbling
   - Acute retention of urine

c. Postmicturition Symptoms
   - Feeling of incomplete emptying of the bladder
   - Post-micturition dribbling of urine

d. Other Symptoms
   - Haematuria
   - Swelling of the lower abdomen

Signs

Mass in the lower abdomen

Digital Rectal Examination (DRE) - Anatomic enlargement of the prostate, firm, smooth-surfaced, with median groove and lateral sulci present.

Signs of progressive renal failure in severe longstanding obstruction

Investigations

Full blood count and ESR
Urinalysis, urine microscopy, culture and sensitivity
Serum electrolytes, urea, and creatinine
Prostate Specific Antigen (PSA)
Transrectal ultrasound (TRUS)
Abdominal ultrasound
X-rays – Chest, Abdomen (KUB)
Uroflowmetry
Cystoscopy

Differential Diagnosis

Prostate Cancer
Bladder Cancer
Bladder neck stenosis/ contracture
Bladder neck dysnerrgia
Urinary Tract Infection
Urethral stricture
External sphincter dysnerrgia
Bladder calculus
Prostatitis
Neurological Diseases
Bladder wall diseases, especially chronic schistosomiasis and tuberculosis of the bladder.

Complications

Urinary retention
Recurrent Urinary Tract Infection
Bladder calculus

chronic renal insufficiency & Acute renal failure
Haematuria
Obstructive uropathy

Treatment

Objectives –To relieve obstruction and treat complication

a. Non-drug: In severe symptoms (IPSS≥19) and associated complications

Surgical Operation

- Open prostatectomy
- Transurethral resection of the prostate
- Transurethral incision of the bladder neck
- Transurethral Vaporization of the Prostate
- Transurethral laser surgery

Non-medical minimally invasive alternatives

Urethral catheterization (Size 16 or 14 FG)

Prostatic urethral stents

Balloon dilatation of the prostatic urethra

Transurethral or transrectal hypothermia

Transurethral thermotherapy

Interstitial therapies

b. Drug: In mild to moderate symptoms (IPSS<19)

α-1 adrenergic blockers

Alfuzosin- 10mg nocte (preferable in

reproductive age)

5- α -reductase inhibitors

Finasteride – 5mg daily

c. Adverse Reactions

α -1 adrenergic blockers – retrograde ejaculation, dizziness, hypotension and syncopal attacks. So should be taken at bedtime

5- α -reductase inhibitors – loss of libido, erectile dysfunction, gynaecomastia.

CARCINOMA OF THE PROSTATE

Introduction

The most commonly diagnosed malignancy affecting men above middle age

The commonest malignancy of the male genitourinary tract.

About 90% are adencarcinomas

Exact cause not known

Risk factors

Increasing age

Presence of testicles

Heredity – Cancer of the prostate in first degree relatives, inheritance of faulty genes e.g. cancer of the breast in mother

Ethnicity – commoner and more aggressive in the black race

Environment –Industrial workers (rubber, textile, fertilizer)

Obesity

Clinical Features

a. Lower urinary tract symptoms (LUTS)
   Daytime frequency
   Poor stream
   Excessive straining
   Nocturia
   Terminal dribbling

b. Features of advanced disease
   Low back pain
   Paraplegia/Paraparesis
   Pedal oedema
   Weight loss
   Pathological fractures
   Azotaemia

c. Digital Rectal Examination (DRE) - Enlarged, assymetrical, hard, nodular prostate with obliteration of the median groove and lateral sulci

Differential diagnosis

Benign Prostatic Hyperplasia
Chronic Prostatitis
Bladder cancer
Prostatic calculi
Urethral Stricture

Complications

Urinary retention
Recurrent Urinary tract infection
Obstructive uropathy

Progressive renal failure
Paraplegia/Paraparesis
Pathological fractures
Lymphoedema

Investigation
Full blood count and ESR
Serum, electrolytes, urea and creatinine
Transrectal ultrasound
Abdominal ultrasound
Prostate biopsy
X-ray- Lumbosacral spine, chest
MRI/CT scan
Radionuclide bone scan

Treatment
Objective – Cure for early disease
- Palliation and improvement of quality of life for advanced disease

Treatment should be in a specialist centre.

a. Non-drug:
Active surveillance (Watchful waiting)
Radical Prostatectomy
Radical radiotherapy
Bilateral orchiectomy
Cryoablation therapy
Laser therapy

b. Drug:
Used usually in advanced diseases
Leutenising hormone releasing hormone agonist:-

Goserelin – 3.6mg subcutaneously every month or 10.8mg 3-monthly

Leuprolide – 3.75mg-7.5mg subcutaneously monthly or 11.5mg-22mg 3-monthly

Leutenising hormone releasing hormone antagonist:-p

Degarelix– 280mg subcutaneously stat, then 80mg monthly

Antiandrogens:- Used with LHRH agonist, antagonist, or orchiectomy. Monotherapy is not advised

Bicalutamide – 50mg daily orally

Flutamide–250mg three times daily orally

For castration resistant prostate cancer (CRPC): This must be treated in tertiary centres with appropriate facility and personnel.

Ketoconazole- 400mg orally three times daily with prednisolone 20mg mane, 10mg evening

Dietylstilboestrol -3mg daily

Docetaxel 75mg daily to be used with Prednisolone

For bone metastasis:

Zoledronic acid 400mg over 1 hr once per month

Radiotherapy – Radium-223

c. Notable ADR, caution, and contraindications

Antiandrogens
Loss of libido
Gynaecomastia
Erectile dysfunction
Fluid retention
Hypertension
Thromboembolic disease
Loss of libido
Gynaecomastia
Ketoconazole
Liver toxicity-monitor liver function
Other drugs for CRPC fraught with ADRs including blood dyscrasias, and reduced quality of life

ERECTILE DYSFUNCTION

Introduction

Erectile dysfunction (ED) is the persistent inability to achieve and maintain an erection sufficient for satisfactory sexual intercourse.

It results from disorder in any of the factors involved in the complex processes that lead to erection which involves the brain, hormones, blood vessels, emotions, and muscles.

It can occur at any age, but is uncommon in boys, common in the elderly.

By the age of 45 years, most men have experienced ED at some time. Generally, 40% of men experience some degree of ED at 40 years, and 70% at 70 years. Complete ED

occurs in approximately 5% of men at 40 years and 15% at 70 years.

Types of ED
Organic – Physical
Psychogenic- Emotion and mental health
Mixed – occasionally

Risk factors
Advanced age

Diabetes mellitus
Cardiovascular diseases e.g. hypertension, coronary heart disease
High cholesterol (dyslipidaemia)
Tobacco intake – smoking, snuffing
Recreational drug use –Indian hemp, cocaine etc
Depression and other psychiatric illnesses

Clinical features
Difficulty getting erection
Difficulty keeping an erection
Reduced sexual drive
History suggestive of possible causes- e.g. drugs, systemic diseases
Focused history to confirm it is ED and not any other sexual dysfunctions (see differential diagnosis)
Focused physical examination to identify significant abnormalities, e.g. gynaecomastia,

penile deformity or plaques, impaired sensations etc

Differential diagnosis

Ejaculatory Dysfunctions (premature ejaculation, delayed ejaculation, anejaculation, anorgasmia)
Long refractory period
Decreased libido
Peyronie's Disease

Complications

Psychological disturbance –stress and anxiety
Unsatisfactory sex life
Embarrassment and low esteem
Relationship problems
Infertility

Investigations

FBC + ESR
Urinalysis
Blood glucose screening
Blood lipid profile (Fasting)
Hormone assay – Total and free testosterone, prolactin, FSH, LH
Specialist Investigations:

- Nocturnal penile tumescence testing (Rigidscan)
- Colour Duplex Ultrasound – for suspected vascular cause.
- Cavernosogram – suspected venous

leak

Treatment

Objective – to achieve and sustain erection

a. Non active treatment:

Life-style and home remedies

- Quitting smoking,
- Losing excess weight
- Exercising regularly
- Reducing or stopping alcohol and hard drugs
- Couples or marriage counselling, if relation problem

Adjusting medications:

Important part of ED treatment

- alpha methyl dopa, Beta blockers & Hydrochlorothiazide cause ED
- Ca channel blockers & ACEs have no effect
- ARBs may increase libido, improve ED and sexual, performance.

b. First line active treatment – Drugs

Oral phosphodiesterase-5 inhibitors (PDES-5)

- Sildenafil- 25-100mg daily

Adequate medical evaluation must be done before PDES-5 (or any OTC drug for ED) is given.

MALE INFERTILITY

Introduction

Failure to achieve conception after one year of regular, unprotected sexual intercourse in a couple trying to achieve pregnancy

Primary:

- When the man has never impregnated a woman Secondary:
- When the man had impregnated a woman in the past

Male factor is responsible for about 50% of infertile unions

Clinical features

Vital points in the history: Duration of infertility

Ability to have erection, penetration and ejaculation Family history of infertility

History of systemic disease e.g. diabetes mellitus, hypertension, chronic liver disease and tuberculosis

History of sexually transmitted infections and urinary tract infections

History of genital trauma

History of surgery: herniorraphy, orchidopexy, urethral surgeries, etc

Examination:

Gynaecomastia

Penis: epispadias, hypospadias, penile deformities

Scrotum: absence of testis, small sized testis, varicocoeles, hard and irregular epididymis

Investigations

Semen analysis x 3
Hormone profile (LH, FSH, testosterone, and prolactin)
Scrotal ultrasound
Trans-rectal ultrasound
Testicular biopsy
Vasography

Treatment objectives

To improve semen quality and restore reproductive capability

Non-drug treatment

Surgical options:
Varicocoelectomy
Vasovasotomy
Epididymo-vasotomy
Transurethral resection of obstructed ejaculatory duct
Assisted reproductive techniques:
Intra-uterine insemination
In vitro fertilization
Gamete intra-fallopian tube transfer
Intra-cytoplasmic sperm injection

POSTERIOR URETHRAL VALVES

Introduction

Congenital mucosal folds situated in the prostatic/membranous urethra, causing urine outflow obstruction

Occurs in males - The most common mechanical cause of renal deterioration in children

Clinical features
Obstructive urinary symptoms
Urinary retention
Failure to thrive
Distended bladder with palpable kidneys

Differential diagnoses
Anterior urethral valves
Congenital bladder neck hypertrophy
Congenital urethral stricture
Meatal stenosis
Posterior urethral polyp

Complications
Recurrent urinary tract infections
Septicaemia
Bladder dysfunction
Bladder stones
Hydroureter/hydronephrosis
Progressive renal impairment
Failure to thrive

Investigations
Urinalysis
Urine microscopy, culture and sensitivity
Full Blood Count
Serum Urea, Electrolytes and Creatinine

Abdominal ultrasound
Micturating cysto-urethrogram
Urethrocystoscopy

Treatment objectives
To relieve obstruction
Treat any complications

Non-drug treatment
Valve resection with endoscopes
Valve avulsion with valvotomes

Drug treatment
None

Supportive measures
Correct dehydration and electrolyte imbalance
Treat infection with appropriate antibiotics
Urinary diversion: vesicostomy

Prevention
Not applicable

PRIAPISM
Introduction
Persistent penile erection that continues beyond, or is not related to sexual stimulation
Predisposing factors:
Thromboembolic disorders e.g. sickle cell disease, leukaemia

Spinal injuries
Perineal and genital trauma
Drugs e.g. chlorpromazine, prazosin and prostaglandins

Clinical features
Persistent painful erection lasting several hours
Penis is rigid and tender but the glans penis and corpus spongiosum are soft

Complication
Erectile dysfunction

Investigations
Full Blood Count
Haemoglobin electrophoresis
Colour Doppler/duplex ultrasound

Treatment objectives
To increase venous drainage from the corpora cavernosa
Decrease arterial inflow in high flow priapism
Treat the primary cause(s)

Non-drug treatment
Shunting procedures
- Caverno-glandular shunt
- Caverno-spongiosum shunt intensity
- Caverno-saphenous shunt

Spinal or epidural anaesthesia

Drug treatment

Intracavernosal injection of alpha adrenergic agonist:

Phenylephrine testicle

- 250 - 500 microgram

Or:

Ephedrine

- 50 - 100 mg

Supportive measures

Adequate hydration

Pain relief

Prevention

Avoid causative drugs

PROSTATITIS

Introduction

An inflammation of the prostate or pain in the prostate, similar to that caused by an inflammation

Accounts for 2% of prostatic pathology

Classified into:

Acute bacterial prostatitis

Chronic bacterial prostatitis

Chronic non-bacterial prostatitis

Prostatodynia

Risk factors:

Ductile reflux
Urinary tract infection
Indwelling urethral catheterization
Penetrating anal sex
Sexually transmitted infections

Acute bacterial prostatitis

Results from direct spread of ascending urethral infection or reflux of infected urine into the prostatic ducts

- *E. coli* is the main causative organism.

Others are *Klebsiella, Pseudomonas, Streptococcus faecalis and Staph aureus*

Chronic bacterial prostatitis

Caused by *E. coli,* Klebsiella, Mycoplasma and
Chlamydia

Non-bacterial prostatitis

An inflammation of indeterminate cause

Clinical features

Acute prostatitis

Systemic features

- Fever
- Chills
- Malaise
- Nausea

Local features

- Dysuria
- Frequency
- Haematuria
- Urethral discharge

Rectal examination:

- Hot boggy, swollen and very tender prostate

Chronic prostatitis

Voiding symptoms: dysuria, frequency, urgency, haematuria
Poor stream
Urethral discharge
Low back pain
Perineal pain
Haemospermia
Painful ejaculation
Rectal examination: enlarged, tender, firm prostate

Differential diagnoses

Benign prostatic hypertrophy
Cystitis
Urethral stricture
Prostate cancer

Complications

Prostatic abscess
Prostatic calculi
Infertility
Septicaemia

Investigations

Urinalysis

Urine microscopy, culture and sensitivity

Prostatic massage: microscopy, culture and sensitivity (chronic prostatitis only)

Trans-rectal ultrasound

Biopsy: culture and histology

Urethrocystoscopy (chronic prostatitis only)

Full Blood Count; ESR

Treatment objectives

To eradicate causative organisms

Control pain

Drug treatment

Antibiotics (based on local sensitivity

- Ciprofloxacin 500 mg orally every 12 hours for 28 days

Or:

- Cotrimoxazole 960 mg orally every 12 hours for 28 days

Anti-inflammatory drugs

- Non-steroidal e.g. diclofenac, ibuprofen etc
- Steroids e.g. prednisolone, dexamethasone

Alpha blockers e.g. prazocin,

Hormonal therapy e.g. finasteride,

Non-drug treatment

Prostatic massage (chronic prostatitis only)

Physiotherapy
Sitz baths

SCROTAL MASSES

The empty scrotum

Introduction

A clinical situation in which the testis is absent from the scrotum
May be bilateral or unilateral
Causes include:
Undescended testis
Ectopic testis
Retractile testis
Absent (vanishing) testis
Atrophic testis
Surgical removal (for treatment of other conditions)

Undescended testis
The testis is arrested in its normal path of descent
Unilateral arrest is more common than bilateral arrest
Incidence at birth is about 3% in full term infants, 30% in preterm infants and 1% in adulthood

Clinical features

Absence of one or both testes from the scrotum
Pain from trauma to the testis

Infertility (in adulthood)
Atrophic testis
The testis, if palpable cannot be manipulated into the scrotum
Inguinal hernia may be present on the affected side

Complications
Torsion of the spermatic cord
Trauma to the testis
Malignancy
Infertility

Investigations
Urinary 17-ketosteroids, gonadotropins
Serum testosterone
Ultrasonography
Computed tomography
Laparoscopy
Magnetic Resonance Imaging

Management
Hormone therapy:
Human chorionic gonadotropin
- 1,500 units/week intramuscularly, for a total of 9 injections
- Applicable only to special cases

Surgical treatment:
In those with undescended testes
- Bring testis down and fix it in the scrotum

TORSION OF THE TESTIS

Introduction

Twisting of the spermatic cord with compromise of the blood supply to the testis
An uncommon affliction that is most commonly seen in adolescent males.
A few cases occur in infancy

Clinical features

Pain in one testicle: of sudden onset, severe in and radiates to the lower abdomen
Nausea and vomiting
Swollen, high lying testis with reddening of the scrotal skin
Tenderness. Pain can be increased by lifting
Absence of the cremasteric reflex
Abnormal lie of the testis on the opposite side

Differential diagnoses

Acute epididymo-orchitis
Mumps orchitis
Trauma to the testis
Strangulated inguinal hernia
Insect bites
Inflammatory vasculitis (Henoch-Schonlein purpura)
Idiopathic scrotal oedema
Testicular tumour
Fournier's gangrene

Complications

Testicular atrophy
Sympathetic orchidopathy
Abnormal sperm count
Infertility

Investigations

Colour Doppler sonography

- An absence of arterial flow is typical

Radionuclide scan using Tc-99m pertechnetate

- The twisted testis is avascular

Treatment objectives

Detorsion
Fixation of the testis to prevent recurrence

Treatment

Fixation on the affected side and prophylactic fixation on the opposite side

URETHRAL STRICTURE

Introduction

An abnormal narrowing or loss of distensibility of any part of the urethra, as a result of fibrosis

One of the commonest causes of urine retention in tropical Africa

Very rare in females.

May result from trauma or inflammation; may be iatrogenic

Traumatic causes:

Penetrating or blunt injury to the urethra

- From pelvic fractures or falling astride an object

Infective causes:

Gonococcal urethritis or non-gonococcal urethritis from chlamydia, tuberculosis or schistosomiasis

Iatrogenic causes:

Urethral instrumentations e.g. catheterization and urethroscopy

May be congenital

May be complete or partial, single or multiple

Can affect any part of the urethra, anterior or posterior

Clinical features

Dysuria

Frequency

Urgency

Poor stream

Straining

Hesitancy

Dribbling

Examination of the external genitalia may reveal:

Urethral indurations

Periurethral or perineal abscess

Urinary fistula

Differential diagnoses

Benign prostatic hypertrophy

Prostate cancer

Bladder calculi
Bladder neck stenosis

Complications
Urinary tract infections
Urethral/bladder calculi
Urinary retention
Fournier's gangrene
Perineal urinary fistulae
Progressive renal failure

Investigations
Urinalysis
Urine microscopy, culture and sensitivity
Urethroscopy
Urethrogram
Uroflowmetry
Abdominal ultrasound
Serum Urea, Electrolytes and Creatinine
Full Blood Count

Treatment objective
To restore urethral patency

Drug treatment
None

Non-drug treatment
Serial dilatation/bouginage
Endoscopic direct visual urethrotomy
Urethroplasty: excision and end-to-end

anastomosis
Substitution urethroplasty

Prevention
Ensure prevention of sexually transmitted infections
Prompt and appropriate treatment of sexually transmitted infections
Care and attention to asepsis during instrumentation procedures involving the urethra

URINARY SCHISTOSOMIASIS
Introduction
A common parasitic infection of the urinary tract caused
by a body fluke, *Schistosoma haematobium*
Acquired while bathing/wading in infected water
Endemic in many parts of Africa
Gets to the urinary tract through the blood vessels after penetrating the skin

Clinical features
Soon after penetration of the skin:
Pricking sensation and itching (cercarial dermatitis)
Four weeks later:
Intermittent fever, malaise, urticaria and cough
Six - 24 months later:

Intermittent, painless terminal haematuria (may be total)
Symptoms of bladder irritability: dysuria, frequency, urgency, strangury

Differential diagnoses
Tuberculous cystitis
Abacterial cystitis
Bladder carcinoma

Complications
Bladder fibrosis and contracture
Ureteral stricture
Urethral stricture
Bladder calculi
Bladder cancer

Investigations
Urine examination for schistosomal ova
Cystoscopy: tubercles, sandy patches, nodules, ulcers
Plain abdominal radiograph (KUB)
Intravenous urogram
Serological tests
Full Blood Count

Treatment objectives
To eradicate the fluke and ova
Prevent complications

Drug treatment

Praziquantel

- The schistosomicide with the most attractive

combination of effectiveness, broad-spectrum activity and low toxicity

Adult: Single oral dose of 50 mg/kg

Child over 4 years: 20 mg/kg orally, repeated after 4 – 6 hours

- In *S. japonicum* infection, 20 mg/kg 3 times daily for one day after initial dose

Notable adverse drug reactions, caution

Nausea, epigastric pain, pruritus, headache, dizziness

Prevention

Provision of and access to pipe-borne water

Improvement in socio-economic conditions

Mass chemotherapy in endemic areas

Eradicating the intermediate hosts (water snails)

URINARY TRACT CALCULI

Introduction

Occurrence of stone(s) in the kidney, ureter, bladder or urethra

Incidence in Nigeria is 7 - 34 per 100,000

Stones are different with respect to their composition

- Oxalate stones, phosphate stones, uric acid stones and cystine stones

Factors promoting stone formation:
Obstruction to urine outflow
Infection in the urinary tract
Crystallization on foreign bodies
Dehydration
Change in pH
In-born errors of metabolism

Clinical features
Renal and ureteric stones:
Sudden onset loin pain radiating to the groin
Haematuria
Nausea and vomiting
Stones in the bladder:
Frequency
Urgency
Difficulty in passing urine
Stones in the urethra:
Urinary retention

Differential diagnoses
Acute pyelonephritis
Renal tumour
Acute appendicitis
Other causes of urinary obstruction e.g. enlarged prostate, urethral strictures

Complications
Recurrent and intractable urinary tract infection
Secondary hydronephrosis

Progressive renal failure
Periurethral abscess/urethral fistula

Investigations
Urinalysis
Urine culture
Serum calcium, phosphate and albumin
Intravenous urography (IVU)
Ultrasonography
Computerized tomography (non-contrast enhanced)

Treatment objectives
Relieve symptoms
Remove stones
Prevent recurrence

Non-drug treatment
Increased fluid intake
Endoscopic Short Wave Lithotripsy (ESWL)
Endoscopic removal of stones
Open surgical removal

Drug treatment
Analgesics
Antibiotics to treat infections
Drugs used to prevent recurrence:
Thiazide diuretics
- Hydrochorothiazide 5 mg orally daily

Or:
Potassium citrate

- 60 mEq orally daily

Or:
Allopurinol 100 mg orally daily

CHAPTER 12:

GERIATRICS

DECUBITUS (PRESSURE) ULCERS

Introduction

- Localized physical manifestation of changes in blood supply to the skin, usually manifesting over bony promises
- Result from pressure or pressure in combination with friction/shear
- May occur in institutional or home settings
- Can be a useful indicator of quality of care

Risk factors

- Age (higher risk the older the patient)
- Decreased serum albumin (3-fold increase in risk)
- Emergency admission (up to 36-fold increase in risk in patients admitted for surgical indications)
- Faecal incontinence (up to 3-fold increase in risk)
- Fractures (up to 5-fold increase in risk)
- Number of days in bed
- Number of days without nutrition

In older persons with functional limitations (bed- or chair-bound):

- Decreased body weight (2-fold increase in risk)
- Dry skin
- Immobility or reduced mobility
- Lymphopaenia (5-fold increase in risk)

Facility dependent factors

- Ambulation difficulties
- Diabetes mellitus
- Faecal incontinence
- Feeding difficulties
- History of cerebrovascular accident (5-fold increase in risk)
- Male gender

Tissue loss is described as follow:

Stage I

- Intact skin
- Non-blanchable erythema
- May be painful, warm or cool, firm or soft, compared to adjacent skin
- May be missed in darkly pigmented skin

Stage II

- Ulcer extends through epidermis
- No slough
- May present as blister with intact or ruptured blister
- Does not include other skin breaches e.g. excoriation, maceration, tape burns or dermatitis associated with incontinence

Stage III

- Full thickness loss of tissue
- Subcutaneous fat may be visible
- Bone, tendon, muscle not exposed
- Tunneling, undermining may be present
- Depth varies by anatomical location

Stage IV

- Full thickness tissue loss
- Slough present
- Depth varies by anatomical location
- May extend to muscle, fascia, joint capsule
- Bone/or muscle may be exposed or palpable
- Tunneling, undermining frequent
- May be associated with osteitis or osteomyelitis

Unstageable/Unclassified

- Full thickness tissue loss
- Depth obscured by slough and/or eschar in the wound bed
- ***Adherent, dry, intact eschar on the heel is protective and should not be removed***

Suspected deep tissue injury

- Localized area of discoloured skin
- Initial boggy swelling
- May become blood-filled blister
- May be cooler or warmer than surrounding skin

Clinical features

- Skin lesions at different stages (see above)

Signs of wound infection:
- Erythema around edges of wound
- Enlarging wound
- Foul odour
- Friable granulation tissue
- Increased necrotic tissue
- Increasing pain
- Marked oedema
- Purulent exudate
- Tunneling
- Warmth
- Wound breakdown

Differential diagnoses
- Arterial ulcers
- Diabetic ulcers
- Venous ulcers

Complications
- Secondary wound infection
- Systemic Inflammatory Response Syndrome
- Osteomyelitis

Investigations
- Levine's technique for obtaining wound swabs:
  Cleanse wound with 0.9% saline
  Rotate a swab over a 1-cm square area of viable wound tissue (not necrotic tissue

or wound exudate)

Apply enough pressure to get fluid from beneath the wound surface

- Tissue biopsy

** Wound contamination is not synonymous with wound infection*

** Culture of wound surface exudates is not useful to diagnose wound infection*

** Cultures obtained using Levine's technique give results comparable to those from tissue specimens*

Treatment objectives

- Achieve wound healing
- Prevent secondary wound infection

Non-drug treatment

Remove debris from wound

- Clean wound using 0.9% saline or lactated Ringer's solution at each dressing change

** Antiseptic solutions may be cytotoxic*

** Contraindicated in patients with ulcers in lower extremities who have arterial disease*

Remove necrotic tissue

- Autolytic methods (hydrogels or moisture retaining dressings)
- Chemical debridement
- Sharp debridement

Close dead spaces in wound

- Pack tunnels and undermining loosely with moist gauze dressings

Adjunct treatment to facilitate wound healing

- Electrical therapy
- Electromagnetic therapy
- Laser therapy
- Ultrasound therapy
- Vacuum-assisted closure

Prevent further injury

- Avoid pressure on wound
- Chair cushions for sitting
- Keep heels off pressure
- Pressure-reducing mattresses
- Reposition every 2 hours (or more frequently, depending on host factors)

Support wound healing process

- Adequate protein intake, unless contraindicated by renal disease: 1.25-1.5g/kg/day)
- Adequate calorie intake (30-35kcal/kg/day)
- Avoid exposure to cold (causes vasoconstriction and impaired tissue perfusion)
- Correct deficiencies e.g. of vitamin C and zinc

Drug treatment

- Analgesia: give appropriate pain medicines 30 minutes before wound procedures
- Antibiotics:

Topical:

Indicated for ulcers:

- With ≥1 million CFU/g of tissue
- That grow any level of β-haemolytic streptococci
- Which are clean but fail to heal after 2-4 weeks of optimal care

Spectrum should cover Gram-negative, Gram-positive and anaerobic organisms

Limit use to avoid tissue toxicity and development of resistance

Systemic:

Indicated if there is evidence of:

- Cellulitis
- Localized infection
- Systemic Inflammatory Response Syndrome (SIRS)
- Osteomyelitis

Superficial wound with localized signs of infection; no signs of SIRS or osteomyelitis

- Amoxicillin/clavulanate
- Clindamycin
- Clindamycin plus ciprofloxacin.

Superficial to deep wound with SIRS

- Clindamycin plus ciprofloxacin
- Clindamycin plus ceftriaxone
- Vancomycin (for MRSA)
- *Given for 2-4 weeks*

Deep tissue involvement with SIRS, osteomyelitis and/or threat to limb or life

- Clindamycin plus ceftriaxone
- Clindamycin plus gentamicin
- Vancomycin (for MRSA)
- *Given for 2-12 weeks; prolonged oral*

treatment required for bone and joint infections after initial intravenous therapy

Surgical Therapy

- Involves direct wound closure
- Suitable for a select group of patients e.g. those with spinal cord injury, deep Stage III/IV decubiti

  High risk of recurrence if factors contributing to ulcers are not corrected

  Notable adverse drug reactions

  Vancomycin may be oto- and nephrotoxic; may also cause blood dyscrasias, rashes, Steven-Johnson's syndrome, toxic epidermal necrolysis; muscle spasm, pain, phlebitis, vasculitis, severe hypotension and flushing of the upper body ('red man' syndrome) are other possible complications. It's use should be restricted to cases of MRSA).

  Prevention

- Address potentially modifiable risk factors e.g. dry skin, immobility, nutritional factors, etc.
- Incidence can be reduced up to 30% with aggressive prevention/intervention strategies

  A multi-disciplinary wound team is an asset, where available

Pressure relief

- Improve mobility ('bed is bad')

- Frequent turning (interval may be reduced from traditional 2 hours depending on host risk factors)
- Pressure-relieving devices

Static:
- To redistribute local pressure over a wider area
- Foam mattresses
- Devices containing air, gel or water

Dynamic:
- Redistribute pressure over wider body area e.g.:
- Alternating pressure pads
- Air suspension devices
- Air-fluid surfaces
- Use power sources

Nutritional Interventions

Increase daily caloric intake to 30 kcal/kg/day for malnourished patients

Achieve daily protein intake between 1.2 – 1.5 g/kg/day

* *Current evidence does not support routine supplementation with vitamin C or zinc unless in those with demonstrated deficiency*

* *High serum zinc levels may interfere with healing and with metabolism of copper*

DELIRIUM (Please see Appendix for screening tool - Confusion Assessment Method)

Introduction

- Acute onset disorder of attention and cognitive function; fluctuating course
- Common in the elderly
- Potentially reversible
- May occur in home settings but more common during hospitalization
- More prevalent postoperatively and in Intensive Care Units
- Up to 80% of terminally ill patients may be delirious before death

Aetiology

There is/are usually an underlying cause(s)

Cardiovascular diseases

- Arrhythmias
- Heart failure
- Shock
- Myocardial infarction

Environmental

- Emotional stress
- ICU admission
- Lack of sensory stimulation
- Physical restraints
- Sleep deprivation

Infections

- Respiratory system
- Skin
- Urinary tract infections
- Etc.

Medications

- Alcohol (toxicity or withdrawal)
- Anticholinergics
- Anti-inflammatory agents including corticosteroids
- Benzodiazepines
- Cardiovascular medicines (e.g. antihypertensives, digoxin, diuretics)
- H_2-receptor antagonists
- Lithium
- Opioid analgesics
- Polypharmacy
- Tricyclic antidepressants

Metabolic disturbances

- Acid-base imbalance
- Acute blood loss
- Adrenal dysfunction
- Dehydration
- Electrolyte imbalance
- End-organ failure
- Hyperglycaemia/hypoglycaemia
- Hyper-/hypocalcaemia
- Hypoalbuminaemia
- Hypoglycaemia
- Hypoxia
- Thyroid dysfunctions

Neurologic

- CNS infections
- Head trauma
- Seizures
- Stroke

- Subdural haematoma
- Transient Ischaemic Attacks
- Tumours

Miscellaneous
- Bladder catheterization
- Constipation
- Fever
- Hypothermia
- Multiple procedures e.g. bladder catheterization, nasogastric tube insertion, etc.
- Pain
- Post-operative state
- Physical restraints
- Urinary retention

Surgery
- Cardiac
- Orthopaedic
- Prolonged cardiopulmonary bypass

Risk factors

Cognitive status
- Cognitive impairment/dementia
- Depression
- Previous delirium

Co-morbidities
- Anaemia
- HIV infection
- Hypoalbuminaemia
- Metabolic derangements (see above)
- Multiple co-morbidities

- Neurologic disease (encephalitis, meningitis, stroke, Parkinson's disease, etc.)
- Severe illness
- Terminal illness
- Trauma

Decreased oral intake
- Dehydration
- Malnutrition

Demographic
- Age ≥60 years
- Low educational attainment
- Male sex

Functional status
- Functional dependence
- Gait instability
- History of falls
- Immobility
- Reduced physical activity
- Sensory impairment

Clinical features

Diagnosing delirium (Confusion Assessment Method)

Delirium is present if the following 2 are present:

(i) Acute deterioration in cognition, fluctuating over time

(ii) Inattention

Plus *one or the other* of:

(iii) Disorganized thinking

(iv) Altered level of consciousness (agitation,

coma, drowsiness, hyper-alertness, irritability, stupor)

Three forms present clinically:

- Hyperactive, hyperalert
- Hypoactive, hypoalert, lethargic
- Mixed – combining features of the above 2 forms

Differential diagnoses

- Dementia
- Depression
- Mania
- Organic psychosis e.g. schizophrenia

Complications

Adverse outcomes of hospitalization outcomes:

- Increased length of hospital stay: functional decline, immobility, pressure ulcers, deep vein thrombosis, pulmonary embolism, etc.
- Complications associated with underlying causes
- Increased risk of dementia
- Worsening of pre-existing cognitive impairment
- Mortality

Investigations

Depending on the history and clinical features:

- Full blood count

- Electrolytes and urea
- Serum calcium and albumin
- Liver function tests
- Blood glucose
- Oxygen saturation
- Blood culture
- Urinalysis
- Urine culture
- Chest X-ray
- Thyroid function tests
- Arterial blood gases
- Vitamin B_{12} levels
- Toxicology screens
- Cortisol levels
- Cerebrospinal fluid examination

Treatment objectives

- Return patient to baseline cognitive functioning

Non-drug treatment

Should be directed at underlying cause(s)/risk factors:

- *Agitation*: ensure safety; use physical restraints only as a last resort to prevent self-harm; allow family participation – even if only to sit by
- *Cognitive impairment*: sensory orientation, family presence, increase nursing attention, calendars, clocks, newspapers, familiar music, games, etc.
- *Dehydration*: rehydrate
- *Hearing loss*: hearing aids
- *Immobility*: ambulate out of bed as soon

and as frequently as possible, involve in self-care as much as possible, minimize tethers (IV lines, nasogastric tubes, urinary catheters, etc.), physiotherapy
- ***Medications***: taper and discontinue unnecessary medicines (see Beer's list of potentially inappropriate medicines), consider renal and liver function in relation to medications, use alternatives, avoid medicines with long half lives
- ***Sleep deprivation***: avoid sedatives, sleep protocol (back massage, soothing music, minimize vital sign checks/procedures, reschedule medication regimen to exclude night time if possible, caffeine-free drinks; maintain sleep-wake cycle
- ***Visual impairment***: provide glasses or other appropriate visual aids

Drug treatment
- *Haloperidol: 0.25-1 mg every 20-30 minutes as loading dose until patient is calmer; divide loading dose by 2 and give every 12 hours; taper over 2-3 days. Maximum daily dose 3-5 mg; doses above 5 mg per day not beneficial; increase harm.*
- *Lorazepam: 0.25 - 0.5 mg starting dose*

Notable adverse drug reactions

Haloperidol should not be given by the intravenous route because of the risk of QTc prolongation. Obtain ECG prior to the first dose if possible.

Caution with haloperidol if patient is taking other medicines that prolong QTc

Prevention

- Identify potential risk factors and avoid or mitigate them

DEPRESSION (Please see Appendix for screening tool - Geriatric Depression Scale)

ELDER MISTREATMENT

Introduction

- Acts that cause harm to vulnerable elder persons
- Occur deliberately or through neglect
- Meted out by caregivers or persons held in trust
- May occur in home or institutional settings
- May affect older persons across all social classes
- May affect males as well as females
- Diagnosis may be missed if not carefully sought

Risk factors

For suffering abuse

- Advanced age
- Disability (physical or mental)
- Lack of financial skills

- Recent bereavement or other personal loss
- *Conditions that increase dependence*

For committing abuse

- Elder dependence
- Family caregiving
- Lone caregiver and stress
- Mental illness
- Physical abuse in the past
- Substance abuse

Types

- Financial or material neglect:
- Exploitation, theft of financial documents, forcible transfer of property, etc.
- Neglect or abandonment neglect:
- Caregiver fails to meet the older person's needs e.g. clothing, hygiene, healthcare, shelter, social stimulation, etc.
- Physical neglect:
- Injury inflicted through striking, force feeding, use of restraints, etc.
- Psychological neglect:
- Mental anguish inflicted through insulting statements, threats, social isolation, etc.
- Self-neglect neglect:
- Acts by older adult that threaten their own wellbeing or safety
- Sexual neglect:
- Forced sexual activity, touching or

fondling without consent

Clinical features

- Bruising in unusual locations
- Burns in patterns suggesting intentional injury
- Delay in seeking medical care
- Decubiti
- Dehydration
- Inattention to personal hygiene
- Injury patterns suggesting use of restraints
- Malnutrition
- Traumatic alopecia
- Unexplained fractures
- Unexplained weight loss

Differential diagnoses

- Allergic reactions
- Adverse reactions to medications
- Contact dermatitis
- Mental illness
- Senile purpura
- Systemic diseases presenting with features of neglect e.g. diabetes mellitus, malabsorption, urinary tract infection, etc.

Complications

- Physical harm
- Psychological harm e.g. living in fear, depression, anxiety, etc.
- Morbidity worsening

- Mortality

Investigations
- As indicated by history and clinical features

Treatment objectives
- Improve safety
- Improve health status
- Improve overall wellbeing

Non-drug treatment
Individualized, multi-disciplinary approaches:
- Assess decision making capacity
- Social work referral
- Comprehensive geriatric assessment
- Other referrals as indicated

Drug treatment
- As indicated by the clinical scenario e.g. analgesia for pain

Prevention
- Recognize warning signs (in the older adult, caregiver, home or institution) and intervene

FALLS

Introduction
- A fall is to inadvertently come to rest on the ground or lower level with or without loss of consciousness or injury

- The diagnosis excludes falls associated with intrinsic events e.g. seizures, stroke or syncope, or falls from overwhelming environmental hazards
- May be an atypical presentation of acute illness in an older adult
- A marker of frailty and impaired mobility in older adults
- Older adults are more likely to fall with increasing age
- Risk of falling increases with previous falls
- Falls account for significant morbidity and mortality in older adults
- Incidence of falls is approximately equal in older men and women but women are more likely to be injured

Aetiology

- Multifactorial

Risk factors

- Modifiable, difficult to modify, non-modifiable

Intrinsic

- Age
- Alcohol use
- Fear of falling
- Hearing impairment
- Medical conditions e.g. arthritis, dementia, stroke, Parkinson's disease, etc.

- Muscle weakness
- Orthostatic hypotension
- Pain
- Poor balance
- Urinary incontinence
- Vertigo
- Visual impairment

Extrinsic
- Medications
- Anticonvulsants
- Antidepressants
- Antihypertensives
- Antipsychotics
- Benzodiazepines
- Diuretics
- Improper or improper use of assistive devices
- Inappropriate footwear

Environmental/Situational
- Clutter
- Haste
- Inadequate lighting
- Lack of non-skid surfaces in bath tubs
- Loose mats/rugs
- Poor housing design/disrepair e.g. uneven steps
- Slippery floors

Clinical features
- Patient is found on the ground or at a lower level than s/he was

- Complications arising from fall

Evaluation

Get Up and Go test

Patient sitting on an arm chair is timed when asked to:

- Rise from the chair without using the arms
- Walk a distance of 3 m across the room
- Turn, walk back to the chair and sit down

Increased risk of falling if the above activities take more than 13.5 seconds

Complications

Economic

Costs of:

- Disability
- Dependence
- Hospitalization and interventions to treat complications

Physical:

- Dehydration
- Dependency
- Disability
- Fractures
- Haemorrhage e.g. subdural haematoma
- Hypothermia
- Immobility
- Increased likelihood of falling again
- Self-imposed reduction in physical activity
- Severe injuries requiring hospitalization

- Skin lacerations
- Mortality
- Pneumonia
- Pressure ulcers

Psychological

- Anxiety
- Depression
- Fear of falling; falls phobia syndrome
- Loss of confidence
- Social withdrawal

Investigations

- Should be tailored identify underlying causes e.g. anaemia, arrhythmias, electrolyte imbalance, infections, etc.
- Additional investigations to confirm or rule out complications as indicated by clinical presentation

Treatment objectives

Eliminate or reduce modifiable predisposing factors, for example:

- Reduce total number of medications and/or dose of individual medicines if feasible, e.g. in patients with postural hypotension

Non-drug treatment

- Please see below (Prevention)

Drug treatment

- Specific treatments for underlying conditions and complications arising

from falling

Prevention

Vitamin D 800 IU daily plus Calcium 1,200 mg for all older adults at risk of falling

Environmental modification

- Adequate lighting, preferably with switches located at entrances to rooms or hallways
- Arrange furniture to avoid obstruction
- Clean up spills without delay
- Light source (e.g. flashlight) to be kept handy in the event of power failure
- Non-skid mats in bath tubs
- Provide elevated toilet seats
- Provide handrails on both sides of bathroom

Address predisposing factors

- Educate patients on:
- Proper use of assistive devices
- Arising and changing position slowly
- Signs and symptoms of hypoglycaemia
- Examine eyes regularly
- Maintain adequate hydration
- Provide frequent, small meals
- Encourage rest after meals

Rehabilitation

Important to address risk of falling again

- Physical
- Psychological
- Social

FRAILTY

Introduction

- A physiological state characterized by increased vulnerability to stressors
- Caused by decreased physiologic reserves
- An endpoint of multiple disease processes

Aetiology

- Multifactorial
- Lack of physical exercise and undernutrition play major roles

Clinical features

Symptoms

- Anorexia
- Fatigue
- Inactivity
- Weakness
- Weight loss
- Inactivity

Signs

- Abnormalities of gait and balance
- Deconditioning (loss of physical functioning)
- Osteopenia
- Sarcopenia (decreased skeletal muscle mass, strength and exercise tolerance, thermoregulation, energy expenditure, etc.)

- Slow gait speed (Timed Up and Go test ≥ 15 seconds)
- Undernutrition

Fried's phenotypic criteria for diagnosis of frailty

- Reduced physical activity
- Self-reported exhaustion/poor endurance
- Slow gait
- Weakness
- Weight loss

Each of the above is scored 1 if present

Total scores are interpreted as follow:

- Robust = 0
- Pre-frail = 1-2
- Frail = 3-5

Differential diagnoses

Complications

- Acute illnesses
- Dependency
- Disability
- Falls
- Hospitalizations
- Injuries
- Institutionalization
- Mortality

Investigations

- Full blood count
- Electrolytes, urea and creatinine

- Liver function tests
- Thyroid function tests
- Serum calcium and albumin
- Urinalysis

Treatment objectives
- Return patient to baseline status

Non-drug treatment
- Should be tailored to address clinical presentation

Drug treatment
- Should be tailored to address clinical presentation

Prevention

Multi-dimensional approaches:
- Identify patients who are pre-frail and intervene to return to robustness
- Exercise (endurance and muscle strengthening; stretching and balance exercises)
- Nutrition (healthy food; supplements as indicated)
- Reduce chronic disease prevalence
- Treat sarcopenia
- Promote healthy aging

ATYPICAL PRESENTATION OF ILLNESS IN OLDER ADULTS (See Appendix VI)

CHAPTER 13:

INFECTIOUS DISEASES/ INFESTATIONS

FEVERS: MANAGEMENT APPROACH

Introduction

A leading cause for seeking medical care

In health, temperature is controlled within limits (in adults at a mean of 36.8C) with diurnal variations of about 0.5C

'Fever' is elevation of body temperature that exceeds the normal daily variation and occurs in conjunction with an increase in hypothalamic set point

In children younger than 5 years of age:

A rectal temperature greater than 38C

Oral temperature above 37.8C

Axillary temperature above 37.2C

Important points in the history are:

Chronology of symptoms

Occupational history

Travel history

Geographic region

Family history

Physical examination:

Vital signs (axillary temperatures are unreliable)

Skin, lymph nodes, eyes, nail beds, CNS, chest, abdomen, cardiovascular, musculo-skeletal and nervous systems
Rectal examination is imperative
The penis, prostate, scrotum and testes (for men)
Pelvic examination (for women)

Investigations

The number of investigations will depend on the clinical circumstances. On occasions, patients may need to be extensively investigated
General:
Full Blood Count
Differential white blood cell count
Urinalysis with examination of the urinary sediment
Examination of any abnormal fluid collection
Microbiology:
Smears and culture of specimens from the throat, urethra, anus, cervix, and vagina (as indicated)
Sputum smears; culture
Blood culture
Urine microscopy, culture and sensitivity
Cerebrospinal fluid examination
Abnormal fluid collection: specimens for microscopy, culture and sensitivity testing
Chemistry:
Urine examination

Serum urea, electrolytes and creatinine
Blood glucose
Liver function tests
Cerebrospinal fluid examination
Radiology:
Chest radiograph
Other investigations as may be indicated in the clinical circumstances

Complications

Heat stroke in adults
Febrile convulsions in children
Complications associated with underlying cause(s) of fever

Treatment objectives

To lower the temperature
To treat underlying causes

Non-drug treatment

Tepid sponging
Liberal oral sips of water (if clinical state is not a contraindication)

Drug treatment

Paracetamol
*Adult:500*mg - 1 g orally every 4 - 6 hours; maximum 4 g daily
Child: 3 months - 1 year: 60 - 125 mg; 1 - 5 years: 120 -
250 mg; 6 - 12 years: 250 - 500 mg; repeated every 4 – 6 hours if necessary to a maximum of

4 doses in 24 hours
- Infants under 3 months should not be given paracetamol unless advised by a doctor
Aspirin: (acetylsalicylic acid)
Adult: 300 - 900 mg orally (with or without food) very 4 - 6 hours if necessary; maximum 4g daily
Treat the identified (or suspected) cause of fever
Child: under 16 years, not recommended because of the risk of Reye's syndrome

Notable adverse drug reactions, caution
Paracetamol:
Liver damage (and less frequently, renal damage) following over dosage
Aspirin
Gastrointestinal discomfort, nausea
Ulceration with occult bleeding
Hearing disturbances such as tinnitus (rarely deafness)
Use with caution in the following clinical conditions:
Asthma
Allergic disease
Impaired renal or hepatic function
Pregnancy
Breastfeeding
Elderly
Dehydration

FOOD POISONING

Introduction

A spectrum of disorders arising from:

Infections acquired by eating contaminated food

Clinical problems that result from eating food contaminated with toxins

Clinical sequelae from inherently poisonous animals, plants or mushrooms

Clinical forms:

Staphylococcal food poisoning:

- Food is contaminated by *S. aureus* when prepared unhygienically by individuals who are carriers
- Subsequent growth of *S. aureus* in the food and enterotoxin production occurs if the food is not cooked at temperatures sufficient to kill the bacteria, or is not refrigerated

Food-borne botulism

Non-typhoidal Salmonellosis

Shigellosis

E. coli food poisoning

Campylobacter food poisoning

Listeria monocytogenes food poisoning

Yersinia enterocolitica food poisoning

Norwalk virus food poisoning

Hepatitis A virus food poisoning

Giardiasis

Helminthic parasitic food poisoning

Clinical features

Staphylococcal food poisoning:

Nausea

Diarrhoea 2 - 6 hours after eating food contaminated by enterotoxin

Food-borne botulism:

Incubation period is 18 - 36 hours, but depending on toxin dose, can extend from a few hours to several days

Symmetric descending paralysis

Diplopia

Dysarthria/dysphagia

Nausea, vomiting and abdominal pain may precede or follow the onset of paralysis

Non-typhoidal Salmonellosis:

Diarrhoea

Nausea

Vomiting

Abdominal cramps

Fever

Headache

Myalgia

Shigellosis:

Fever

Self-limiting watery diarrhoea

Bloody diarrhoea

Dysentry

- Frequent passage, 10 - 30 times/day of small volume stools containing blood, mucus and pus

Abdominal cramps
Tenesmus
Campylobacter food poisoning:
A prodrome with fever, headache, myalgia and/or malaise
12 - 48 hours later:
Diarrhoea and abdominal pain
E. coli food poisoning:
Watery diarrhoea accompanied by cramps
L. monocytogenes food poisoning:
Common source of outbreaks of acute gastritis
Not a major cause of sporadic diarrhoea
Norwalk virus food poisoning:
Abrupt onset of nausea and abdominal cramps followed by vomiting and/or diarrhoea
Hepatitis A virus food poisoning:
May cause large outbreaks of diarrhoea and vomiting from contaminated food, water, milk and shellfish
- Intrafamily and intrainstitutional spread common

Diagnosis
Essentially clinical
Laboratory confirmation of the specific microbe(s) involved

Differential diagnoses
Other causes of acute onset diarrhoea,

nausea,
abdominal cramps and vomiting with or without systemic manifestations

Complications
Fluid and electrolyte derangements
Others
- By no means limited to the stated organisms

Shigellosis:
Dehydration
Rectal prolapse
Protein-losing enteropathy
Malnutrition
Haemolytic-uraemic syndrome
Toxic megacolon
Perforation
Campylobacter food poisoning:
Bacteraemia
Cholecystitis
Pancreatitis
Cystitis
Meningitis
Endocarditis
Arthritis
Peritonitis
Cellulitis
Septic abortion

Treatment objectives
Restore fluid and electrolyte balance

Neutralize toxin
Eradicate microbe

Non-drug measures
Gastric lavage in food-borne botulism

Drug treatment
Appropriate fluid and electrolyte replacement
Trivalent (types A, B, and E) equine anti-toxin should be administered as soon as possible after specimens are obtained for laboratory analysis for food-borne botulism
Emetics in food-borne botulism
Administer appropriate medicines
Shigellosis
Oral Rehydration Therapy
Plus:
Adult: Amoxicillin 50 - 100 mg/kg/day orally every 8 hours; up to 2 g/day
Child up to 10 years: 125 mg every 8 hours, doubled in severe infections

Or:
Trimethoprim/sulfamethoxazole (co-trimoxazole)
Adult: 960 mg orally every 12 hours for 5 days
Child weeks to 5 months: 120 mg orally; 6 months - 5 years: 240 mg; 6 - 12 years: 480 mg given every 12 hours for 5 days

Or:

Ceftriaxone:
Adult: 1 g intravenously slowly
Child: 50 mg/kg/day intravenously for 5 days

Campylobacter food poisoning

Fluid and electrolyte replacement
Plus:
Erythromycin
Adult: 250 mg orally every 6 hours for 5 - 7 days
Child: 30-50 mg/kg orally every 6 hours for 5 - 7 days

E. coli food poisoning

Ciprofloxacin
Adult: 500 - 750 mg orally every 12 hours

Or:
200 - 400 mg 12 hourly by intravenous infection over 30 -60 minutes
Child and adolescent: not recommended

L. monocyogenes food poisoning

Amoxicillin
Plus:
Gentamicin
Treat specific complications as appropriate e.g.

- Antibiotic-unresponsive toxic megacolon: colectomy
- Haemolytic-uraemic syndrome: dialysis
- Malnutrition from protein-losing enteropathy: nutritional support; optimal

nutritional management

Prevention

Appropriate environmental and personal hygiene

- Hand washing with soap and water
- Decontamination of water supplies by
- Use of sanitary latrines or toilets

Identify and treat chronic carriers among food handlers

Hygienic preparation and storage of food

Ensure that food is cooked at temperatures sufficient to kill bacteria

Refrigerate food whenever possible

Encourage exclusive breastfeeding

Encourage measures to reduce the burden of malnutrition (with its attendant predisposition to severe infections)

Administer a pentavalent vaccine (A, B, C, D, and E) for persons at high of botulism

Report new cases to public health authorities

HELMINTHIASIS

Introduction

Parasitic worm infestations can arise from different groups:

Nematodes (round worms)

Ascaris

Ancylostoma (hookworm)

Enterobius (pinworm)

Trichiuris (whipworm)

Cestodes (flat worms/tapeworms)

- *Taenia solium* and *T. saginata*
- Trematodes (flukes)
- *Schistosoma haematobium* and *S. mansoni*

Round worm infestations are associated with rural living and poor hygiene

- Prevalent among school children and young adults
- Acquired through soil and faeco-oral contamination
- Flat worms and tape worms are acquired by eating under-cooked contaminated meat or fish

Bladder worms *(S. haematobium)* are acquired by wading through streams and ponds contaminated with the vector snails

Clinical features

Depend on the infecting helminth:

Ascariasis

Lung phase:

Irritating, non-productive cough

Burning substernal discomfort, aggravated by coughing or deep inspiration

Dyspnoea

Blood-tinged sputum

Intestinal phase: Usually no symptoms Pain

Features of small bowel obstruction

Features of perforation, Intussusception. Volvulus

Biliary tree occlusion: biliary colic,

cholecystitis, cholangitis, pancreatitis, intrahepatic abscess
Effects of migration of an adult worm up the oesophagus:
Coughing
Oral expulsion of the worm
Hookworm
Most are asymptomatic
Maculo-papular dermatitis
Mild transient pneumonitis
Epigastric pain, often with post-prandial accentuation
Diarrhoea
Weakness
Shortness of breath
Skin depigmentation
Enterobiasis
Perianal pruritus, worse at night owing to the nocturnal migration of the female worms
Skin excoriation and bacterial superinfection
Abdominal pain
Weight loss
Vulvo-vaginitis
Pelvic/perineal granulomas
Trichuriasis
Abdominal pain Anorexia
Bloody or mucoid diarrhoea
Rectal prolapse
Growth retardation
Strongyloidiasis

Distinguished by its ability to replicate in the human host

- Can thus persist for decades without further exposure of the host to exogenous infective larvae

Recurrent urticaria: buttocks and wrists

Pruritic raised erythematous skin lesions: advance as rapidly as 10 cm/hour along the course of larval migration

- The pathognomonic serpiginous eruption

Mid-epigastric abdominal pain
Nausea
Diarrhoea
Gastrointestinal bleeding
Mild chronic colitis
Weight loss
Small bowel obstruction

Disseminated strongyloidiasis in patients with unsuspected infection who are given glucocorticoids can be fatal

Trichinellosis

In the first week after infection (gut invasion):
Diarrhoea Abdominal Pain
Constipation
Nausea
Vomiting

In the second week after infection (muscle invasion): Fever
Periorbital and facial oedema
Haemorrhages (subconjunctival, retinal and

nail bed)
Maculopapular rash
Headache
Cough
Dyspnoea
Dysphagia
Tachyarrhythmias
Heart failure
Encephalitis
Pneumonitis
Schistosomiasis
- See Urology

Differential diagnoses

Other causes of acute-onset diarrhoea and/or vomiting -Other conditions depending on the predominant clinical presentation

Investigations

Stool examination for ova and parasites
Urine examination: microscopy
Haematology: eosinophilia and anaemia may be present Serology and CT scan may be required in some instances

Drug Treatment

Hookworm
Mebendazole
Adult and child: 100 mg orally every 12 hours for 3 days Iron supplementation may be given

if anaemia is present

Ascaris

Mebendazole

Adult and child: 100 mg orally every 12 hours for 3 days

Trichiuris

Mebandazole

Adult and child: 100 mg orally every 12 hours for 3 days

Enterobius

Pyrantel embonate

Adult and child: 10 mg/kg orally once

- Repeat dose 2 weeks later; several treatments may be necessary

Trematodes

Praziquantel

Adult: 40 mg/kg given orally at once

- Provides up to 80% cure rates

Child over 4 years: 20 mg/kg followed after 4 - 6 hours a further dose of 20 mg/kg

Praziquantel is effective in all human cases caused by schistosomes

Cestodes

Praziquantel

Adult: 40 mg/kg given orally at once

Or:

- 20 mg/kg followed by another 20 mg/kg after 4 - 6 hours

Child over 4 years: 20 mg/kg followed after 4 - 6

hours a further dose of 20 mg/kg (20 mg/kg 3 times daily for one day for S. japonicum infections)

Notable adverse drug reactions, caution and contraindications

Avoid mebendazole in pregnant women

Side effects of praziquantel include abdominal pain, headache, dizziness and skin rashes

Prevention

Good personal and food hygiene

Access to safe and potable water

Regular deworming

Adequate cooking of food and meats

HUMAN IMMUNODEFICIENCY VIRUSINFECTION (HIV)

Introduction

Human Immunodeficiency Virus (HIV) is a retrovirus, which infects primarily CD4 T cells (T helper cells)

Infection leads to a progressive destruction of the immune system with a consequent myriad of opportunistic infections and the development of certain malignancies

Acquired Immuno Deficiency Syndrome (AIDS) is defined as the presence of an AIDS-defining illness (see table 1) with a positive antibody test for HIV

HIV transmission

Sexual transmission through vaginal and anal sex is the commonest route globally and in Nigeria, accounting for about 80%

Transfusion of infected blood and blood products Use of contaminated instruments; sharing needles, tattooing and occupational exposures Mother-to-child transmission of HIV: from an infected mother to her baby during pregnancy, at delivery and, after birth through breast-feeding

Clinical Course of HIV Disease:

- *Acute (Primary) HIV infection: This occurs 1-4 weeks after infection during which infected people experience transient flu-like symptoms, which may include:*
  - Mild fever
  - Muscle aches and pains
  - Fatigue
  - Enlargement of lymph nodes
  - Sore throat
  - Fever
  - Skin rash

  This stage is difficult it is difficult to diagnose by standard laboratory assays.
  - *Seroconversion: Usually occurs within 4 weeks. Patients develop antibody response, which is detectable by a positive HIV Ab test.*
  - *Asymptmatic Infection: The*

individual feels well despite on-going viral replication. Usually last avariable amount of time and is marked by a gradual decline in CD4 cell counts.

- ***Early Symptomatic Infection:***
- Generalized lymphadenopathy
- Weight loss
- Night sweats
- Pruritic skin rash
- Unexplained fever
- Chronic diarrhea
- Oral candidiasis
- Oral hairy leukoplakia
- Herpes zoster
- Pneumococcal infections
- Pulmonary Tb

Late Disease/AIDS defining Illness:

This period is marked by the appearance of opportunistic infections and neoplasms

Opportunistic infections:

- Pulmonary/extrapulmonary tuberculosis and Disseminated TB
- Pneumocytis *jiroveci* (carinii) pneumonia.
- Cryptococcal meningitis
- Recurrent bacterial pneumonia
- Candida oesophagitis
- CNS toxoplasmosis
- Kaposi sarcoma
- Non-Hodkin's lymphoma
- Disseminated/extrapulmonary coccidiomycosis, crytococcosis or histoplasmosis

- Chronic (> 1month) intestinal cryptosporidiosis or isosporiasis
- Disseminated extrapulmonary mycobacteria (non-tuberculous)
- Progressive multifocal leukoencephalopathy (PML)
- Recurrent salmonella septicaemia
- HIV wasting syndrome

Staging of HIV/AIDS

WHO Staging System for HIV Infection and Disease in Adults and Adolescents

Clinical Stage I:

Asymptomatic

- Generalised lymphadenopathy
- Performance scale 1: asymptomatic, normal activity

Clinical Stage II:

- Weight loss < 10% of body weight
- Minor mucocutaneous manifestations (seborrhoeic dermatitis, prurigo, fungal nail infections, recurrent oral ulcerations, angular cheilitis)
- Herpes zoster within the last five years
- Recurrent upper respiratory tract infections (i.e. bacterial sinusitis)
- And/or performance scale 2: symptomatic, normal activity

Clinical Stage III:

- Weight loss > 10% of body weight Unexplained chronic diarrhoea, > 1 month Unexplained prolonged fever (intermittent or constant)

- >1month Oral candidiasis (thrush) Oral hairy leucoplakia Pulmonary tuberculosis within the past year Severe bacterial infections (i.e. pneumonia, pyomyositis)
- And/or performance scale 3: bedridden < 50% of the day during last month

Clinical Stage IV:

- HIV wasting syndrome
- Pneumocystic carinii pneumonia
- Toxoplasmosis of the brain
- Cryptosporidiosis with diarrhoea > 1 month
- Cryptococcosis, extrapulmonary Cytomegalovirus disease of an organ other than liver, spleen or lymph node (e.g. retinitis)
- Herpes simplex virus infection, mucocutaneous (>1month) or visceral
- Progressive multifocal leucoencephalopathy
- Any disseminated endemic mycosis
- Candidiasis of oesophagus, trachea, bronchi
- Atypical mycobacteriosis, disseminated or lungs
- Non-typhoid salmonella septicaemia
- Extrapulmonary tuberculosis
- Lymphoma
- Kaposi sarcoma 2
- HIV encephalopathy
- And/or performance scale 4: bedridden > 50% of the day during last month

- 1: Weight loss of > 10% plus either unexpl ained chronic diarrhoea > 1 month, or chronic weakness and unexplained prolonged fever > 1 month.

2: Clinical findings of disabling cognitive and/or motor dysfunction interfering with activities of daily living, progression over weeks or months in absence of concurrent illness or condition other than HIV infection that could explain the finding

WHO Improved Clinical Staging

Laboratory indices		Clinical stage			
Lymphocytes	CD4	Stage 1 Asym. PGL	Stage 2 Early HIV	Stage 3 Intermed. (ARC)	Stage 4 Late AIDS
A > 2000	> 500	1A	2A	3A	4A
B 1000 - 2000	200 - 500	1B	2B	3B	4B
C < 1000	<200	1C	2C	3C	4C

CDC classification

CD4	Stage A Asym. PGL	Stage B Symp. not A or C	Stage C AIDS indicator condition
> 500	A1	B1	C1
200 - 500	A2	B2	C2
< 200	A3	B3	C3

Differential diagnoses:

-Tuberculosis
-Malignancies
-Diabetes mellitus
-Other wasting syndromes

Complications:

Table 1: Complications of HIV disease at different CD4 cells cut-offs.

CD4 count (cells/ mms)	Infectious complications	Non-infectious complications
> 500	Acute HIV, candidal vaginitis	PGL, Guillain -Barre syndrome, myopathy, aseptic meningitis
200 - 500	Pneumococcal and other bacterial pneumonias, pulmonary TB, Herpes zoster, oropharyngeal candidiasis, oral hairy leukoplakia, Kaposi sarcoma	Cervical cancer, anaemia, lymphomas
< 200	**Milliary/extrapulmonary TB,** pneumocystis carinii pn eumonia (PCP), disseminated histoplasmosis anc coccidiomycosis, progressive multifoca leukoencephalopathy (PML)	Wasting, peripheral neuropathy, progressive polyradiculopathy, HIV - associated dementia, cardiomyopathy
< 100	Disseminated herpes simplex, **toxoplasmosis, crytococcosis, cryptosporidium, chronic** microsporidiosis, and oesophageal candidiasis	
< 50	Disseminated cytomegalovirus (CMV), disseminated Mycobacterium avium complex (MAC)	Central nervous system lymphomas

Investigations

Full Blood Count and differentials
VDRL (or RPR)
Tuberculin test (PPD)
Sputum smears for TB Electrolytes,
Urea and Creatinine
Blood glucose
Liver function tests
Lipid studies (fasting trigycerides, LDL, HDL)
HBV, HCV serology
Cervical (PAP) smears
CD4 T cell counts
HIV RNA level (viral load)
HIV DNA (paediatric diagnosis <18 months of age)
Genotype and phenotype assays for resistance testing

Treatment objectives

Clinical: prevent disease progression
Immunological: restore immunity
Virological: control or suppress viral replication
Public health: reduce infectivity

Criteria for initiating ART based on Nigerian ART guidelines

Criteria for initiating ART in adults and adolescents (including pregnant women)

When to start ART in adults and adolescents (including pregnant women)

Target population	Recommendation
Severe/advanced HIV disease (WHO stage 3 or 4)	Initiate ART in all individuals regardless of the CD4 cell counts
HIV infection (WHO stage 1 or 2)	Initiate ART if CD4 is <500 cells/mm³
TB disease	Initiate ART in all individuals with active TB disease regardless of CD4 cell count*
Hepatitis B co-infection	Initiate ART in all individuals regardless of CD4 cell count in the presence of severe chronic liver disease**
HIV-associated nephropathy	Initiate ART if CD4 ≤500 3. Consider ART in persons with CD4+ >500 cell/mm3
HIV-sero-discordant couples	Consider ART in sero-discordant couples

** In patients with dual TB/HIV disease TB treatment should be initiated first, followed by ART as soon as possible thereafter (and within the first eight weeks of initiating TB treatment). For those with a CD4 count less than 500 cells/ mm3, ART should be provided within two weeks of starting TB treatment.*

***Severe chronic liver disease includes cirrhosis and end-stage liver disease and is categorized into compensated and decompensated stages.*

Decompensated cirrhosis is defined by the development of clinically evident complications of portal hypertension (ascites, variceal haemorrhage and hepatic encephalopathy) or liver insufficiency (jaundice).

Children

Criteria for initiating ART in Children and non-pregnant adolescents up to 15 years old:[2]

Target population	**Recommendation**
Adolescents (10-15 years)	Initiate ART if CD4 cell count =<500 cells/mm 3 or WHO clinical stage 3 or 4
Children (5-10 years)	Initiate ART regardless of WHO clinical stage and CD4 cell count for those with: – Active TB – HBV co -infection + severe chronic liver disease – WHO clinical stage 3 or 4
Children (<5 years old)	Initiate ART in all regardless of WHO clinical stage and/or CD4 cell count

Drug treatment

Recommended first-line ART regimens for adults, adolescents, pregnant and breastfeeding women and children.
Recommended first-line ARV regimen for ART naïve adults and adolescents

First-line ART	Preferred first-line regimens	Alternative first - line regimen
Adults (Including pregnant and breastfeeding women and adults with TB and HBV coinfection)	TDF + 3TC + EFV	AZT + 3TC + NVP AZT + 3TC +EFV ABC + 3TC + EFV
Adolescents (10 -19 years) ≥ 35 kg		AZT + 3TC + NVP AZT + 3TC + EFV ABC + AZT + 3TC

Recommended first-line ARV regimen for children

Age group	Consideration	Preferred 1st line regimen	Alternative 1 st line regimens (in order of preference)
0 to 36 months (0-3 years)	With no prior exposure to NNRTIs	AZT + 3TC + NVP	ABC + 3TC + NVP AZT+ 3TC+ ABC**
	With prior exposure to NNRTIs (through PMTCT)	AZT + 3TC + LPV/r* *	ABC+ 3TC + LPV/r
	With unknown exposure to NNRTIs	AZT + 3TC + NVP NB: Closely monitor	AZT + 3TC + EFV ABC + AZT + 3TC D4T + 3TC + EFV

		for treatment failure	
3 to 10 years	Regardless of NNRTI exposure	AZT+3TC+EFV***	AZT + 3TC + NVP ABC + AZT + 3TC D4T + 3TC + EFV
Special Circumstances	Severe Anaemia/ Neutropenia	AZT should not be used in persons with Hb <8g/dl	
	HBV in children >3 years	TDF+ 3TC + EFV*** (or NVP)	
	TB in children	See TB treatment	

*** *EFV is only indicated for use in children >3 years of age and >10kg.*

++ *In case of ABC hypersensitivity reactions, do not ever re-use in-patient.*

Triple NRTI therapy (i.e. ABC + AZT + 3TC) is suboptimal and should only be used if there is absolutely no alternative

Recommendations for ART in HIV + children with active TB

Considerations	Time of initiation of ART*	Preferred ART regimen
Active TB diagnosed, not yet on HAART	Start anti -TB treatment* Start ART 2 to 8 weeks after commencing anti-TB treatment	Children <3 years and PMTCT exposure to NNRTI Use Triple NRTI (AZT + 3TC + ABC) Children < 3 years and no PMTCT exposure to NNRTI Initiate NVP -based regimen and increase NVP dose to 200 mg/m²per day, OR Triple NRTI (AZT + 3TC + ABC) Children >3 years: Standard 1st line AZT + 3TC + EFV^is preferred. Consider 1stline alternatives if prefe rred regimen not applicable.
Active TB diagnosed, already on HAART	If <3 years and on NVP - based regimen	Continue regimen but increase NVP to maximum dose (200mg/m²/day)
	If >3 years and on NVP - based regimen	Substitute: Replace NVP with EFV
	If on LPV/r based regimen	Increase dose of Ritonavir to make 1:1 ratio with LPV

'Administration of CPT is important in children with TB/HIV co-infection.

** Regimen assumed to contain Rifampin*

^Careful clinical monitoring with lab support, is recommended where NVP is used with rifampicin. This combination should only be used if there are no other options

'EFV is not currently recommended for children <3 years of age.

Monitoring Response to ART and diagnosis of Treatment Failure

Clinical and laboratory monitoring of patient on ART is critical to achieving the treatment objectives. Through this, treatment response and possible toxicity of ARVs are monitored and patients that are eligible for drug switch are easily detected.

Recommended and desirable laboratory tests during monitoring of ART

Laboratory Tests after the initial baseline investigations and during follow-up on ART

Phase of HIV management	Recommended	Desirable (if feasible)
Follow-up before ART	CD4 cell count (Every 6 -12 months)	
ART initiation	CD4 cell count	Haemoglobin test for AZT* Pregnancy test Blood pressure measurement Urine

		dipsticks for glycosuria and eGFR and serum creatinine for TDF[b] Alanine aminotransferase[c] for NVP
Receiving ART	CD4 cell count (every 6 months) HIV viral load (at 6 months after initiating ART and every 12 months thereafter)	Urine dipstick for glycosuria and serum creatinine for TDF[d]
Treatment failure	CD4 cell count HIV viral load	HBV (HBsAg) serology[f] (before switching ART regimen if this testing was not done or if the result was negative at baseline)

WHO definitions of clinical, immunological and virological failure for the decision to switch ART regimens

Definition of clinical, immunological and virological failure

Failure	*Definition*	*Comments*
Clinical Failure	**Adults and adolescents** New or recurrent clinical event indicating severe immunodeficiency (WHO clinical stage 4 condition) after 6 months of effective treatment. **Children** New or recurrent clinical event indicating advanced or severe immunodeficiency (WHO clinical stage 3 and 4 clinical condition with exception of TB) after 6 months of effective treatment	The condition must be differentiated from Immune Reconstitution Inflammatory Syndrome occurring after initiating ART For adults, certain WHO clinical stage 3 conditions (pulmonary TB and severe bacterial infections) may also indicate treatment failure

Immuno logical failure	**Adults and adolescents** CD4 count falls to the baseline (or below) Or Persistent CD4 levels below 100 cells/ mm3 50% decline from on-therapy CD4 cell peak level	Without concomitant or recent infection to cause a transient decline in the CD4 cell count A systematic review found that current WHO clinical and immunologi cal criteria have low sensitivity
	Children Younger than 5 years Persistent CD4 levels below 200 cells/mm3 or <10% **Older than 5 years** Persistent CD4	

Notable adverse drug reactions, caution and Contraindications

Nevirapine (NVP)

- Life-threatening skin rash (Stevens-Johnson syndrome); occurs in < 5% of patients, usually within 8 weeks of treatment
- DRESS syndrome (drug rash, eosinophilia and systemic symptoms): manifests as fever, athralgia, etc
- Hepatitis and jaundice reported

Efavirenz (EFV)

- Morbilliform rash may appear; usually not life-threatening

CNS side effects in about 50% of patients (usually self-limiting)

- Hallucinations
- Insomnia
- Abnormal dreams
- Somnolence
- Amnesia
- Abnormal thinking
- Confusion
- Euphoria

For these reasons, EFV is contraindicated in patients who already have psychiatric manifestations

- Foetal abnormalities observed in animal models; efavirenz should not be used in pregnant women or women who might become pregnant while on therapy

Zidovudine (ZDV)

- Bone marrow suppression resulting in:
- Anaemia with macrocytosis
- Thrombocytopaenia
- Leucocytopaenia
- Gastro-intestinal intolerance is fairly common: hypersalivation, nausea, abdominal discomfort

Stavudine (d4T)

- Peripheral neuropathy presenting with painful sensations in the lower limbs more than the upper limbs
- Lactic acidosis with hepatic steatosis
- Stop treatment or switch to a drug less

toxic to mitochondria (worse when d4T is used in combination with ddI)
- Peripheral fat atrophy
- Ascending motor weakness resembling Guillain-Barre syndrome

Lamivudine (3TC)
- No major side effect but class side effects may occur

Didanosine (ddI)
- Dose-related pancreatitis; worse when combined with hydroxycarbamide (hydroxyurea)
- Peripheral neuropathy; worse if combined with d4T
- Lactic acidosis (a class adverse effect)

Tenofovir (TDF)
- Infrequent; not more than what is observed in placebos in controlled trials
- Renal insufficiency and bone demineralization

Abacavir (ABC)
- Life-threatening hypersensitivity in 3 - 9% of patients
- Lactic acidosis with or without hepatic steatosis

Indinavir (IDV)
- Class-specific events
- Nephrolithiasis with or without haematuria in 10 - 28% of patients; (fluid intake should be increased)
- Alopecia

Nelfinavir (NFV)

- Diarrhoea: 10 - 30% of patients; (should be managed with agents such as loperamide)
- Fat accumulation
- Hyperlipidaemia

Lopinavir/ritonavir (LPV/r)

Well tolerated except for occasional class adverse reactions:

- Gastrointestinal
- Hepatic transaminitis especially in patients with hepatitis B or C
- Hyperlipidaemia
- Fat accumulation

Saquinavir (SQV)

GIT intolerance in 5 - 30% leading to:

- Nausea
- Abdominal pain
- Diarrhoea

Amprenavir (AMP)

- Class adverse effects
- GIT intolerance; oral paraesthesia in 28% of patients

Oral solution contains propylene glycol which may precipitate:

- Seizures
- Stupor
- Tachycardia
- Hyperosmolality
- Lactic acidosis
- Renal failure
- Haemolysis

Oral solution is contraindicated in children below

4 years; should be changed to capsules as soon as possible

Ritonavir (RTV)

- Class side effects
- Perversion of taste
- Circumoral and peripheral paraesthesia
- Hepatotoxicity
- Aesthenia
- Atazanavir (ATV)
- Unconjugated hyperbilirubinaemia
- Gastrointestinal effects
- No effect on lipids

Note

Refer to standard texts for possible drug-drug interactions in all cases

Prevention

Prevention Of Mother-To-Child Transmission (PMTCT)

- Pregnancy or breastfeeding is an indication for triple prophylaxis in HIV
- Positive womenirrespective of the baseline CD4 cell count or viral load and ARV should be provided as soon as possible irrespective of the gestational age.
  - For positive pregnant women with a baseline CD4 < 500 cells/mm^3, ARV should be provided for life
  - Those with CD> 500 cells/mm^3, triple prophylaxis should be provided and

this should continue up to one week after cessation of breastfeeding. Thereafter, the mother and child should be evaluated at a competent ART center for comprehensive care.

Drug of choice

- Preferred first-line regimen: TDF+3TC + EFV
- Alternative first-line Regimens
- AZT + 3TC + EFV*(Recommended alternative to TDF for pregnant and breatfeeding women)*
- AZT + 3TC + NVP*(For pregnant women who cannot tolerate EFV)*
- TDF + 3TC + NVP*(For pregnant women who cannot tolerate AZT and EFV)*

Prophylaxis for HIV exposed infants

All infants born to HIV mothers are exposed and should receive post-exposure prophylaxis

NVP dosing for Infant HIV prophylaxis*

Infant age	Daily dosing
Birth to 6 weeks:	
Birth weight <2,500 grammes	10 mg (1 ml) once daily
Birth weight > 2,500 grammes	15 mg (1.5 ml) once daily
>6 weeks to 6 months*	20 mg (2 ml) once daily
>6 months to 9 months*	30 mg (3 ml) once daily
>9 months to 12 months*	40 mg (4 ml) once daily

**Dosing beyond 6 weeks of age should be considered in special situations.*

** For the duration of therapy, refer to the Integrated National Guideline on HIV

Post-exposure prophylaxi

Evaluate the risk of exposure and potential eligibility of post-exposure prophylaxis

- *Occupational exposure: The chosen regimen must be continued for at lenst 28*
- *days or until the result of the HIV status of the source person is known to be*
- *negative*

(a) 2-drug combination

- TDF/3TC (300/300mg) o.d. or
- AZT/3TC (300mg/150mg) b.d.

(b) 3-drug combination

- TDF/3TC/EFV (300/300/600mg) o.d. or
- AZT/3TC (300/150mg) b.d. + EFV (600mg) o.d.

NB:

1. *Nevirapine should never be used for PEP as the risk of fatal hepatotoxicity outweighs the risk of HIV infection.*
2. *Where Efavirenz is contraindicated, either of the 2drug combinations may be combined with ATV/r or LPV/r*

Sexual Assault

- *This should be initiated as soon as possible (and up to 72 hours post-assault) to optimize the potential benefit.*

Mechanisms with established merit:

A: Abstinence

B: Be faithful (mutual fidelity to infected partner)

C:Consistent and correct use of male and female condoms

D: Delay onset of sexual activity

E: Examine yourself

F: Find out your status

Screening and treatment of sexually transmitted infections

Encourage Partner Disclosure and Voluntary

Confidential Couple Counselling (VCCCT)

Promote the rights and protection of children and infusion women

MALARIA

Introduction

An infectious protozoan disease transmitted by the female Anopheles mosquito

A major public and private health problem and indeed a cause and consequence of national underdevelopment Five species of the parasite cause the disease in humans:

Plasmodium falciparum, P. malariae, P. vivax, P. ovale and *P.knowlesi*

P. *falciparum* accounts for 98% of all cases of malaria in Nigeria and is responsible for the severe form of the disease

Principal mode of spread: bites from infected female Anopheles mosquito

Peak feeding times are usually dusk and

dawn, but also throughout the night Other uncommon modes are:
Blood transfusion
Mother-to-child transmission

Classification*based on clinical course of P. *falciparum

- Asymptomatic parasitaemia
- Acute Uncomplicated malaria
- Severe malaria
- Asymptomatic parasitaemia:
  older children and adults living inhigh malaria endemicity
  Have acquired natural immunity to clinical disease
  Have malaria parasites in the peripheral blood but no symptoms
- Acute Uncomplicated malaria:

Present with clinical features , usaually non-specific:
Fever
Chills
Headache Malaise
Aches and body pain
Weakness
Tiredness
Pallor
Anorexia
Vomiting
Bitterness in the mouth
Excessive sweating

Pallor
Hepatosplenomegaly
Jaundice
- Severe (Complicated) malaria:
- Malaria is severe when there is:

Repeated vomiting
Prostration
Impaired consciousness
Severe anaemia (Haemoglobin< 5g/dl)
Circulatory collapse (Algid malaria)
Hypoglycaemia(whole blood glucose <2.2mol/L)
Pulmonary oedema
Abnormal bleeding/DIC
Jaundice(Serum bilirubin >3g/dl)
Haemoglobinuria(Black water fever)
Febrile seizures
Acute renal failure
Hyperparasitaemia(>5% of RBCs are parasitized)
Hyperpyrexia(Temp > 40°C)
Lactic acidosis

Other early complications include:
- Pneumonia
- Septicaemia
- Preterm contractions/preterm labour
- Abortions
- Low birth weight
- Intrauterine deaths
- Congenital malaria

Late complications:

- Hyperreactive malaria splenomegaly
- Quartan malaria nephropathy
- Possibly, Burkitt's lymphoma

Cerebral malaria

A severe form of malaria

Occurs usually in children and in non-immune adults Manifests with diffuse and symmetric encephalopathy; focal neurologic signs are unusual Requires prompt and effective therapy to avoid fatality

Diagnosis of malaria

Clinical diagnosis alone is presumptive, gives room for over-diagnosis

Confirmatory diagnosis is based on the detection of parasites in the blood

Parasitological confirmation is recommended in all suspected cases of malaria

Light microscopy remains the gold standard

Microscopic diagnosis should not delay appropriate treatment if there is a clinical suspicion of severe malaria

Rapid Diagnostic Test is used in Primary Health Care levels

Differential diagnoses

Typhoid fever

Meningitis

Encephalitis

Septicaemia
Other causes of fever

Investigations
Blood smear for malaria parasites
Packed cell volume; haemoglobin concentration
White cell count with differentials
Blood sugar
Urinalysis
Electrolytes and Urea; Creatinine Stool microscopy for ova; occult blood Chest radiograph
Cerebrospinal fluid biochemistry; microscopy, culture and sensitivity

Treatment objectives
Eradicate parasitaemia
Preventprogression to severe malaria
Attend to the immediate threats of life
Prevent transmission of gametocytes
Provide personal protection against malaria
Provide chemoprophylaxis in susceptible groups

Drug treatment
Uncomplicated malaria
Artemisinin-based Combination Therapy(ACTs) are the current recommended treatments for uncomplicated malaria globally

Artemether-Lumefantrine(AL) is the medicine of choice while Artesunate-Amodiaquine(AA) is the alternative

Dosage Regimen for AL:

Weight	No of tablets/dose (20/120)mg tab	No of tablets/dose (40/240)mg tab	No of tablets/dose (80/480)mg tab
5 – <15kg	1tab twice dlyx3days	NA	NA
15 – <25kg	2tabs twice dlyx3days	1tab twice dlyx3days	NA
25 – <35kg	3tabs twice dlyx3days	NA	NA
≥ 35kg	4tabs twice dlyx3days	2tabs twice dlyx3ady	1tab twice dly x3ays

Dosage Regimen for AA:

Weight/Age	Tablet strength	Dosage Regimen
4.5kg– <9kg (2mnths– 11months)	25mg/67.5mg	1 tablet once dly x 3days
9kg– <18kg (1yr–5yrs)	50mg/135mg	1 tablet once dly x3days
18kg– <36kg (6yrs– 13yrs)	100mg/270mg	1 tablet once dlyx3days
36kg and above 14 years and above	100mg/270mg	2 tablets once dlyx3days

Other ACTs available for the treatment of uncomplicated malaria:

- Artesunate-Mefloquine
- Dihydroartemisinin-Piperaquine
- Artemisinin-Piperaquine

Children < 5kg with uncomplicated malaria with an ACT at the same mg/kg between target dose as for children weighing 5kg.

It is vital to prevent severe disease, therefore as soon as a presumptive diagnosis of malaria is made:

Insert artesunate suppository per rectum as a single dose

Re-insert if expelled; in young children the buttocks may need to be held or taped together for 10 minutes to ensure retention of the rectal dose, in adults one or more artesunate suppositories should be inserted however dose should be given once and followed as soon as possible by definitive therapy

Severe malaria

Parenteral Artesunate is the drug of choice, and treatment should be started without delay

If not available other effective parenteral antimalarial should be commenced

Adults and Children >20kg:

Artesunate 2.4mg/kg (BW) IV or IM given

on admission (time 0), then 12hrs and 24hrs, then once a day. There is no upper limit to the total dose of artesunate

Children ≤20kg:

Artesunate 3mg/kg (BW) IV or IM given on admission(time 0),then 12hrs and 24hrs,then once a day.

Parenteral artemether or quinine is an alternative if parenteral artesunate is not available

Artemether 3.2mg/kg (BW) IM given on admission then 1.6 mg/kg (BW) per day; or Quinine 20mg salt/kg (BW) on admission (IV infusion or divided IM injections), then 10mg/kg (BW) every 8 hrs; infusion rate should not exceed 5mg/kg (BW) per hr.

Note: Give parenteral antimalarials in the treatment of severe malaria for a minimum or 24hrs once started (irrespective of the patients ability to tolerate oral medications early) and thereafter complete treatment by giving a complete course of the recommended ACT

In all cases, patient's progress should be monitored and management changed as deemed necessary

Supportive measures

Paracetamol (oral/rectal) for symptomatic relief

If temperature is >38.5°C, wipe with wet towel, and fan to lower the temperature

Pulmonary oedema

- Nurse in cardiac position
- Give oxygen
- Furosemide 2 - 4 mg/kg intravenously
- Exclude anaemia as the cause of heart of the heart failure

Renal failure

- Give fluids if patient is dehydrated: 20 ml/kg of chloride injection 0.9%, and challenge with furosemide 1-2mg/kg
- Catheterize to monitor urinary output

If no urine within the next 24 hours, refer for peritoneal or haemodialysis

Profuse bleeding

- Transfuse with screened fresh whole blood
- Give pre-referral treatment and refer urgently

If meningitis is suspected, and cannot be excluded immediately by lumbar puncture, give appropriate antibiotics

Other severe diseases should be treated accordingly

Treatments not recommended

Corticosteroids and other anti-inflammatory agents; agents used for cerebral oedema e.g. urea, adrenaline, heparin

- Have no role in the treatment of severe malaria

Notable adverse drug reactions, caution and contraindication

Mefloquine should be avoided if the patient

had cerebral malaria because of the increased risk of seizure, encephalopathy and psychosis

Prevention

Personal protection

- Reduce the frequency of mosquito bites by avoiding exposure to mosquitoes at their peak feeding times
- Use insect repellants
- Put on suitable clothing
- Use insecticide-impregnated bed nets (ITN)

Chemoprohylaxis

- Indicated for:
- Children born to non-immune mothers in endemic areas
- Pregnant women (see section on antenatal care)
- Travellers to endemic areas
- *People with sickle cell anaemia should have regularchemoprophylaxis*(see Sickle Cell Diseases)

Recommended antimalarial prophylaxis in Nigeria:

Mefloquine

Atovaquone-Proguanil

Mefloquine: 5 mg base/kg weekly, giving an adult dose of 250 mg base weekly

- If tablets are available, an appropriate fraction can be given to child aged 8 - 13

years

- Contraindicated in children <8 years and in pregnant women
- Commence 2-3 weeks prior to arrival, weekly in country and thereafter for 2-3 weeks after departure

Atovaquone-Proguanil

Fixed dose combination, administered daily

Commence 1-2 days prior to arrival, then continue throughout stay, and for 7days after departure

Atovaquone-Proguanil Dosage Regimen

Weight (kg)	Total daily dose	Dosage Regimen
11-20kg	62.5mg/25mg	1 Paediatric Tablet dly
21-30kg	12.5mg/50mg	2 Paediatric Tablet dly as a single dose
31-40kg	187.5mg/75mg	3 Paediatric Tablet dly as a single dose
>40kg	250mg/100mg	1 Tablet(adult strength) as a single dose

Chemoprophylaxis is not recommended for individuals living with areas of intense transmission

RABIES

Introduction

An acute disease of the CNS caused by a bullet-shaped rhabdovirus that affects all mammals

The virus is a single-stranded RNA virus found in animals, in all regions as urban rabies or sylvatic rabies

Transmitted by infected secretions, usually saliva

Most exposures are through bites of an infected animal; occasionally contact with a virus-containing aerosol or the ingestion or transplant of infected tissues may initiate the disease process

Human infection is through contact with un-immunized domestic animals

Dogs are the most important vectors worldwide

Clinical features

There are four stages:

A non-specific prodrome of 1 - 4 days consisting of

- Fever
- Headache

- Malaise
- Myalgia
- Anorexia
- Nausea
- Vomiting
- Sore throat
- Cough
- Paraesthesia

An acute encephalitic stage

- Excitement
- Agitation
- Confusion
- Hallucinations
- Combativeness
- Bizarre aberrations of thought
- Muscle spasms
- Meningismus
- Seizures
- Focal paralysis
- Hydrophobia

Brainstem dysfunction

- Diplopia
- Facial paralysis
- Optic neuritis
- Difficulty with deglutition
- Priapism
- Spontaneous ejaculation
- Coma

Death or recovery

Differential diagnoses
Gullain-Barre syndrome
Other causes of viral encephalitis
Poliomyelitis
Allergic encephalomyelitis
Complications
Inappropriate secretion of ADH
Diabetes insipidus
Cardiac arrythmias
Adult Respiratory Distress Syndrome (ARDS)
Gastro Intestinal (GI) bleeding
Thrombocytopenia
Paralytic ileus

Investigations
Full Blood Count and differentials
Urea and Electrolytes
Culture of secretions
Cerebro Spinal Fluid (CSF) analysis
Serology
Pulmonary Chain Reaction (PCR)

Treatment objectives
Disinfect wound; avoid early suturing
Provide passive immunization with antirabies
antiserum
Provide active immunization with the vaccine

Non-drug treatment

Wound care

The wound or site of exposure should be:

Cleansed under running water

Washed for several minutes with soapy water

Disinfected and dressed simply

It should not be sutured immediately

Drug treatment

Unimmunized persons or those whose prophylaxis is probably incomplete

- Rabies (cell mediated) vaccine

Adult: 1 ml by deep subcutaneous or intramuscular injection in the deltoid region on days 0, 3,7,14 and 30

Plus:

Rabies immunoglobulin given on day 0 *Child: same* as for adult For fully immunized persons:

- Rabies (cell mediated) vaccine

Adult: 1 ml by deep subcutaneous or intramuscular injection in the deltoid region on days 1 and 3 *Child: same* as for adult

Post-exposure prophylaxis (PEP)

Should be initiated as soon as possible after exposure The decision to initiate PEP should include:

Whether the individual came into physical contact with saliva or another substance likely to contain rabies virus

Whether rabies is known or suspected in the

species and area associated with the exposure
The circumstances surrounding the exposure e.g. whether the bite was provoked or unprovoked

- Consider the use of rabies vaccine whenever a patient has been attacked by an animal in an environment where rabies is enzootic, even if there is no direct evidence of rabies in the attacking animal
- Pregnancy not a contraindication

Supportive measures
Allay anxiety: reassure
Other measures as appropriate for clinical situation

Notable adverse drug reactions, caution
Concomitant chloroquine administration interferes with antibody response to rabies vaccine there are no specific contraindications

Prevention
Pre-exposure prophylaxis
Should be offered to persons at high risk of exposure and/or contact with rabies virus:
Veterinnarians
Cave explorers
Laboratory workers who handle the rabies virus
Animal handlers

Workers in quarantine stations

Field workers who are likely to be bitten by infected wild animals

Certain port officials Bat handlers

Persons living in (or travelling to) areas where rabies is enzootic and/or where there is limited access to prompt medical care

Those caring for patients caring for patients with rabies

- Although there is no proven evidence of human-transmission

Pregnancy is not a contraindication: if there is substantial risk of exposure, and rapid access to post-exposure prophylaxis is limited, give pre-exposure prophylaxis Rabies vaccine:

- 1 ml by deep subcutaneous or intramuscular injection in the deltoid region on days 0, 7 and 28

Booster doses every 2 - 3 years for those at continued risk

TETANUS

Introduction

A common, infectious disease affecting individuals of all ages and sexes, particularly the socio-economically deprived

A neurologic disorder characterized by increased muscle tone and spasm that is caused by tetanospasmin, a powerful protein toxin elaborated by *Clostridium tetani* The

bacteria are found in the soil, inanimate environment, animal faeces and occasionally in human faeces Portals of entry: Umbilical stump Female genital mutilation (FGM) Male circumcision Abortion sites

Penetrative wounds (e.g. nail puncture or intramuscular injection) Head injury; scalp wounds Traditional scarification (e.g. for tribal identity) Trado-medical incisions Post-operative surgical sites Chronic otitis media Clinical forms: Generalized tetanus Neonatal tetanus Localized tetanus Cephalic tetanus

Clinical features

Generalized tetanus

Lock jaw Dysphagia

Stiffness or pain in the neck, shoulder and back muscles

Rigid abdomen and stiff proximal limb muscles

The hands and feet are relatively spared

Neonatal tetanus

Poor feeding

Rigidity

Spasms

Localized tetanus

Increased tone; spasms are restricted to the muscles near the wound

Prognosis is excellent

Cephalic tetanus

Follows head injury or ear infection
Trismus
Dysfunction of one or more cranial nerves, often the 7* nerve
Mortality is high

Diagnosis
Entirely clinical

Differential diagnoses
Alveolar abscess
Strychnine poisoning
Dystonic drug reactions
Hypocalcaemic tetani
Meningitis/encephalitis
Acute abdomen

Complications
Autonomic dysfunction
- Labile or sustained hypertension
- Tachycardia
- Dysarrhythmias
- Hyperpyrexia
- Profuse sweating
- Peripheral vasoconstriction
- Cardiac arrest Aspiration pneumonia Fractures

Muscle rupture
Deep vein thrombophlebitis
Pulmonary emboli
Decubitus ulcers

Rhabdomyolysis

Investigations

Wound swab for microscopy, culture and sensitivity

Cerebrospinal fluid for biochemistry; microscopy, culture and sensitivity most

Full Blood Count; ESR

Urinalysis; urine microscopy, culture and sensitivity

Blood glucose

Electrocardiography

Serum Electrolytes, Urea and Creatinine

Electromyography

Treatment objectives

Eliminate the source of toxin airborne

Neutralize unbound toxin

Prevent muscle spasms

Monitor the patient's condition and provide support (especially respiratory support) until recovery

Non-drug treatment

Admit patient to a quiet room

Protect airway

Explore wounds

Cleanse and thoroughly debride the wound

Provide intubation or tracheostomy for hypoventilation

Physiotherapy

Monitor bowel, bladder and renal function
Prevent decubitus ulcers

Drug treatment
Antibiotics
- Benzylpenicillin (Penicillin G)

Adult: 0.6 - 2.4 g daily by slow intravenous injection or infusion in 2 - 4 divided doses; higher doses in severe infections
Child: 1 month - 18 years, 100 mg/kg in 4 divided doses, every 6 hours; dose doubled in severe infections (maximum 2.4 g ,every 4 hours)
1 - 4 weeks: 75 mg/kg daily in 3 divided doses, every 86 hours; dose doubled in severe infection Preterm neonate and neonate under 7 days: 25 mg/kg ,every 12 hours; dose doubled in severe infection

Or:
- Metronidazole

Adult: 500 mg intravenously ,every 6 hours for 10 days
Child: neonate, initially 15 mg/kg by intravenous infusion then 7.5 mg/kg twice daily; 1 month - 12 years: 7.5 mg/kg (maximum 400 mg) every 8 hours; 12 - 18 years: 400 mg every 8 hours Antitoxin
- Human tetanus immune globulin (TIG)

Adult: 250 units by intramuscular injection, increased to

500 units if:

- The wound is older than 12 hours
- There is risk of heavy contamination
- Patient weighs more than 90 kg

A second dose of 250 units should be given after 3 - 4 weeks if patient immunosuppressed or if active immunization with tetanus vaccine is contraindicated

- Administer antitoxin before manipulating the wound
- Control of muscle spasm
- Diazepam

Adult: 20 mg intravenously slowly stat and titrate up to 250 mg/day in infusion

Child: 1 month - 18 years: 100 - 300 micrograms/kg repeated every 1 - 4 hours by slow intravenous injection

- Could also be administered by intravenous infusion or by nasoduodenal tube as follows

3 - 10 mg/kg over 24 hours, adjusted according to response

Or:

Phenobarbital (dilute injection, 1 in 10 with water for injection)

Adult: 10 mg/kg intravenously at a rate of not more than 100 mg/minute, up to maximum total dose of 1g

Child: 5-10mg/kg at a rate not more than 30mg/minute

Treat autonomic dysfunction with

- Vasopressors, chronotropic agents if necessary Hydration
- To control insensitive and other fluid losses
- Enteral or parenteral nutrition
- As determined by clinical situation

Treat intercurrent infections

Notable adverse drug reactions, caution and contraindications

Diazepam is adsorbed from plastics of infusion bags and giving sets; causes drowsiness and light headedness; hypotension

Benzyl penicillin: hypersensitivity reactions

Metronidazole: taste disturbances

Phenobarbital: caution in renal and hepatic impairment

May cause paradoxical excitement, restlessness and confusion in the elderly; hyperkinesia in children

Prevention

Active immunization of all partially or un-immunized adults, those recovering from tetanus, all pregnant women, infants and un-immunized (missed) children

Health education

Improvement in socio-economic status

TRYPANOSOMIASIS (Sleeping sickness)

Introduction

African trypanosomiasis is an acute or chronic disease caused by *Trypanosoma brucei*namely *T. brucei*rhodesiense (East Africa) *T. brucei*gambiense (West Africa)

Clinical features

(Gambian Sleeping Sickness)

Two clinical stages:

- Early stage
- CNS stage

Early stage:

A nodule or chancre following a bite

Fever

Headache

Dizziness

Weakness

Significant posterior cervical (Winterbottom sign) and supraclavicular lymphadenopathy

Splenomegaly

CNS stage:

Occurs six months to several years later

Characterized by behavioural changes with hallucinations, delusions, and disturbances of sleep with drowsiness during the day and terminating with stupor

Investigations

Peripheral blood film for the detection of trypanosomes

Rapid Card Agglutination Trypanosomiasis Test (CATT) for antibody detection

Diagnosis

Presumptive

- Based on the clinical suspicion and history of exposure to the tsetse fly

A finding of the trypanosome in peripheral blood, lymph node aspirate or CSF is confirmatory

Differential diagnoses

Malaria fever
Meningitis
Viral infections involving the CNS

Treatment

Early stage

Suramin

Adult and child: 5 mg/kg on day 1, 10 mg/kg on day 3, and 20 mg/kg on days 5, 11, 17, 23 and 30

Late stage

Melarsoprol

Adult: 2.0 - 3.6 mg/kg intravenously in 3 divided doses for 3 days, followed 1 week later with 3.6 mg/kg intravenously in 3 divided doses for 3 days 10 - 21 days later: 3.6 mg/kg intravenously in 3 divided doses for 3

days

Caution

Urine should be examined for casts and protein before and after treatment with suramin

Lumbar puncture follow-up for at least 1 year after treatment with melasoprol is required

Prevention

Surveillance and treatment

Chemoprophylaxis

Vector control by selective clearing of vegetation and use of insecticides

TYPHOID FEVER

Introduction

A systemic disease characterized by fever and abdominal pain, caused by dissemination of *Salmonella typhi* or *S. paratyphi*.

Transmitted only through close contact with acutely infected individuals or chronic carriers (from ingestion of contaminated food or water)

Incidence of chronic carriage is higher among women and persons with biliary abnormalities: gall stones, carcinoma of the gall bladder; also higher in persons with gastrointestinal malignancies

Clinical features

Incubation period ranges from 3 - 21 days
Prolonged fever (38.8C to 40.5-C)
A prodrome of non-specific symptoms:

- Chills
- Headache
- Anorexia
- Cough
- Weakness
- Sore throat
- Dizziness
- Muscle pains
- Gastro-intestinal:

Diarrhoea or constipation
Abdominal pain Rash (rose spots)
Hepato-splenomegaly Epistaxis
Relative bradycardia

Complications

Neuropsychiatric symptoms
Intestinal perforation
Gastro-intestinal haemorrhage
Pancreatitis
Hepatitis
Splenic abscesses
Meningitis
Nephritis
Pneumonia
Osteomyelitis
Chronic carrier state

Investigations

A positive culture is the 'gold standard' for the diagnosis of typhoid fever

Specimens for culture may be obtained from the blood, stool, urine, bone marrow; gastric and intestinal secretions

There are no diagnostic tests other than positive cultures

Non-specific

Full Blood Count

- Leucopenia, neutropenia, leucocytosis can develop early, especially in children; late if complicated by intestinal perforation or secondary infection

Liver function tests

- Values may be elevated
- Electrocardiography:

ST and T wave abnormalities may be present

- Serological tests
- Widal test gives high rates of false positives and negatives

Treatment objectives

Eliminate *S. typhi* and *S. paratyphi*

Prevent complications

Prevent chronic carrier status

Drug treatment

Ceftriaxone

Adult: 1 g daily by deep intramuscular

injection or by intravenous injection over at least 2 - 4 minutes; 2 - 4 g daily in severe infection

- May also be given by intravenous infusion

Child: neonate, 20 - 50 mg/kg daily by intravenous injection over 60 minutes; infant and child under 50 kg: 20 - 50 mg/kg daily; up to 80 mg/kg in severe infection; over 50 kg: adult dose

Doses of 50 mg/kg and above should be given by intravenous infusion only

Intramuscular doses over 1 g should be divided between more than one site; single intravenous doses above 1 g should be given by intravenous infusion only

Or:

Ciprofloxacin *Adult:* 500 - 750 mg orally every 12 hours

Or:

200 - 400 mg every 12 hours by intravenous infection over 30 - 60 minutes

Child and adolescent: not recommended

Parenteral fluid administration

Treat complications

Notable adverse drug reactions, caution

Ciprofloxacin:

Diarrhoea, nausea, vomiting, abdominal discomfort, headache (which are themselves

features of the disease)

Should be given with caution in pregnancy and during breastfeeding

- Not recommended for children or adolescents

Non-drug treatment

Nursing care

Enteral or parenteral nutrition

Prevention

Eliminate Salmonella by effective treatment of cases, improved sewage management, improved water treatment and improved food hygiene (production, transit, storage and utilization) Typhoid immunization is recommended for those at risk

- Not a substitute for scrupulous personal and environmental hygiene

Identify, and treat chronic carriers with amoxicillin or ciprofloxacin daily for 4 - 6 weeks

- In patients with urolithiasis and schistosomiasis appropriate treatment should be instituted

Correct anatomic abnormalities associated with the disease surgically

- Cholecystectomy may be required in some cases

CHAPTER 14:

RHEUMATIC & MUSCULOSKELETAL DISORDERS

BACK PAIN

Introduction

Back pain is defined as pain along the spine stretching from the neck downward and including the buttocks area.

It affects the cervical, thoracic, lumbar, sacral and coccyx spines.

Probably the commonest affliction of mankind.

Affects all ages groups and sex

Low Back pain is the commonest form

Most cases are due to mechanical causes –poor posture, overuse, unaccustomed exercises

Most back pain are from the soft tissues of the back viz, muscles, ligaments, tendons, and not from the bony or joint structure.

Most back pain will resolve within six weeks with or without treatment, hence there is no need to investigate most cases of back pain.

It could however be recurrent.

There are 'Red flag' clinical features that necessitate further investigations (X-ray,

MRI, CT).

These include:

- Back pain that disturb sleep
- Back pain that persists with recumbent position
- Back pain associated with constitutional disturbances such as fever, loss of weight, nausea, and general feeling of being unwell.
- Back pain associated with bowel disorders.
- Back pain associated with urinary symptoms.
- Back pain associated with muscle weakness
- Back pain associated with deformities of the back.

Other causes of back pain apart from mechanical include:

Degenerative Arthritis- spondylosis
Spinal canal stenosis
Spondylolisthesis
Osteomyelitis
Primary or secondary malignancies
Osteoporotic fracture
Multiple myeloma
Tuberculosis of the spine
Spinal abscess

Clinical features

Back pain

Radicular pain in the arms or legs
Back Stiffness
Saddle anaesthesia
Paraesthesia in the feet
Back pain worsened by coughing or sneezing is suggestive of disc lesions
Back deformity

Differential Diagnosis

Referred pain from gastro-intestinal structures, liver, gall bladder, pancreatic disease.
Aortic aneurysms
Tumours of the pleura, pericardium
Pelvic inflammatory diseases
Psychosomatic disorders

Investigations

None if the pain is mechanical
Laboratory investigations to exclude other causes
Imaging- Plain X-rays, CT Scan, MRI, Bone Densitometry, Radioisotope studies

Treatment Objectives

Relief of pain
Treatment of underlying disease
Treatment of complications

Management

I. Non Pharmacologic:

Weight loss
Avoidance of precipitating factors
Physical therapy
Acupuncture
Bio feed back
Back exercises e.g. Mackenzie Extension Exercises

II. Pharmacologic

- Simple analgesics- Paracetamol up to 1gm tds, with or without -
- Non steroid Anti-inflammatory drugs e.g. Ibuprofen up to 2,400mg dly, Naproxen – 500mg BD, Diclofenac – 75mg BD, Cox 2 inhibitors – Celecoxib-200mg daily.
- Muscles relaxants –Tizanidine – up to 4mg tds , Baclofen 5-10mg daily
- Narcotic analgesics- Tramadol- 50mg tds, Codeine based compounds
- Anti depressants – Amitriptyline- 25-50mg nocte
- Selective Serotonin Re–uptake inhibitors e.g. Fluoxetine
- Anti–convulsants – **Pregabalin** – up to 600mg daily Gabapentin- up to 1,500g daily.

GOUT

Introduction

Crystalline inflammatory disease, due to monosodium urate monohydrate crystals

deposition joint and tissues

Due either to excessive uric acid production from intrinsic purine metabolism or extrinsic (dietary)

Also due to decrease renal excretion of uric acid and resultant accumulation in the blood.

Commonly seen in males (40 years and above) and post menopausal females

4 major types:

- Asymptomatic
- Acute
- Inter critical
- Chronic Tophaceous
- It could be either primary (no identifiable cause) or secondary.

Secondary causes-

Dehydration

Fasting

Renal impairment

Hypertension

Malignancy such as lymphoma, leukemia

Psoriasis

Sickle Cell Disease

Use of cytotoxic agents

Excessive alcohol intake especially beer

Low dose Aspirin

Drugs such as Pyrazinamide, Diuretics

Clinical Presentation

- Usually mono-articular, occasionally

Polyarticular (in elderly persons and in renal failure)
- Sudden onset of pain and swelling
- Pain and swelling maximal within 24–48 hours
- Tendency to start at night or early hours of the morning
- Usually follows binge of alcohol or consumption of offal of animals
- Recurrent painful episodes
- Affects big toe (*Podagra*), but also large joints – forefoot, ankles, knees, wrists
- There may be associated fever, vomiting, diaphoresis, rigors

Chronic Tophaceous Gout
- Follows long standing attacks of acute gout, usually up to 7 years
- Tophi deposit in the skin, ear lobes, over joints
- Tophi may deposit in the kidneys and brain

Differential Diagnosis
- Septic Arthritis
- Osteoarthritis
- Rheumatoid arthritis
- Gonococcal arthritis
- Traumatic synovitis
- Pseudogout
- Osteomyelitis
- Reactive Arthritis

Investigations

- Serum Uric Acid- may be normal in acute Gout
- Haematocrit, white blood cell count, ESR
- Creatinine, Urea- to exclude associated renal impairment
- Cholesterol, Triglycerides- usually co-exist with Gout
- Plasma glucose (high serum uric acid may be a component of metabolic syndrome)

Treatment objectives

Treatment of pain

Lower Serum Uric Acid to below 6mg/100ml

Non – drug treatment

Dietary control – avoid offal, salmon, sea food, red meat

Avoid alcohol especially beer, wine

Reduce weight

Control cholesterol level

Avoid tight shoes

Rest affected joints

Avoid surgical operation of tophi

Drug Treatment

Acute Gout

- NSAIDs at higher doses above normal e.g.
  - Naproxen – 500mg tds
  - Diclofenac – 75mg BD
- Prednisolone – 40mg dly for 1 week

then taper quickly and stop
- Intra articular steroids

Chronic Tophaceous Gout
- Allopurinol – Xanthine Oxidase
  - Inhibitor – gradual escalation from 100mg dly up to 600mg daily
- Sulfinpyrazone – uricosuric agent

Adverse Drug Reactions

NSAIDs – Dyspepsia, Peptic Ulcer Disease, Gastro – intestinal haemorrhage, renal insufficiency, hepatotoxicity, heart failure (especially elderly persons)

Allopurinol – elevated liver enzymes, Allopurinol Hypersensitivity Syndrome– liver failure, renal failure, eosinophilia, hypersensitivity skin.

OSTEOARTHRITIS – DEGENERATIVE ARTHRITIS

Introduction
- Heterogeneous group of diseases
- All forms of assault on the joint or any other arthritis will result in OA
- Degenerative disease of synovial joint – osteoarthritis, degenerative disease of intervertebral disc spondylosis
- Commonest type of arthritis affects mostly middle aged to elderly

- Females more frequent affected than males
- Mostly primary, but can be secondary
- Secondary causes include – previous trauma to the joint, meniscal injury, any previous arthritis e.g. Gout, RA; Congenital hip dysplasia; Epiphyseal: dysplasia; hypermobility syndromes, previous poliomyelitis in the limb, glycogen storage disorder.
- Joints affected – mostly knee, also hip, ankle, Distal interphalangeal joints, cervical, Lumbosacral. Rarely thoracic spine.

Clinical features:

- Joint pain – developing over several weeks or even years
- Initially pain on movement but later at rest
- Joint warm to touch
- Minimal joint morning stiffness
- Creakiness (crepitus) on walking
- Swelling of the joint may be bony – osteophytes, or soft due to effusion
- Presence of Heberden's nodes (Distal Interphalangeal joint); Bouchards nodes (proximal interphalangeal joint)
- Joint deformities – knee – Genu Varus , Genu Valgum

Complications

- Joint deformity

- Immobility
- Joint subluxation

Differential Diagnosis
- Rheumatoid Arthritis
- Gout
- Psoriatic arthritis
- Bursitis
- Ankylosing spondylitis

Investigations
- To exclude other diagnosis
- No blood tests diagnostic
- Imaging – X-ray; CT, MRI (hardly done because of cost)

Treatment objectives

Reduce pain

Enhance mobility

Prevent deformities

Treatment

A. Non-pharmacologic
- Patient education
- Weight loss
- Avoidance of excessive flexion of joint such as the knee
- Knee brace, feet insoles, walking sticks
- Regular exercise – walking, bicycling, swimming. Avoid jogging, if knee is affected
- Physical therapy – Quadriceps strengthening exercise, range of motion

exercises
- Occupational therapy
- Acupuncture
- TENS

B. Pharmacologic
- Paracetamol – up to 4gm daily in divided dose
- Mainstay of treatment is NSAIDs (none is superior to the other); depends on patients response
- Ibuprofen - 200mg daily up to 2,400mg daily in divided doses
- Diclofenac – 75mg – 150mg daily
- Naproxen 500mg BD
- NSAIDs with misoprostol or PPI
- COX – 2 inhibitors- Celebrex – 200mg daily;
  Local application – Diclofenac gel,
  Intra – articular steroid – not to be given more than four times in the year
- Intra – articular platelet rich plasma
- Narcotic analgesics:
  - Codeine based compound,
  - Tramadol

Adverse Drug reactions

NSAIDS – Dyspepsia, Peptic Ulcer Disease, Gastro–intestinal haemorrhage, perforation, hepatotoxicity, impairment of renal blood flow, pyloric stenosis, fixed drug eruptions, constipation, skin rashes

Indications for surgery

- Intractable pain
- Disability
- Deformity

Prevention

- Reduce weight
- Regular exercise

SEPTIC ARTHRITIS

Introduction

- Septic arthritis is accompanies by articular manifestation due to presence of pathogen within a joint.
- Mostly due to bacteria, but can also be due to fungal, viral, or protozoan agent, rickettsia
- Two major entities – gonococcal and Non – gonococcal
- Staphylococcus aureus is the commonest organism
- Other organisms are Klebsiella pneumonia (alcoholics); pseudomonas aeruginosa (intravenous drug abuse) Gram negative bacteria (malignancy use of immunosuppressive drugs); salmonellae, Neisseria meningitides; streptococcus pneumonia (SLE)
- Joint infection mostly as a result of haematogenous seeding during a bacteriaenic episode
- May also occur secondary to penetrating

Cutaneous trauma
- Rarely iatrogenic from local corticosteroid joint injection
- Any joint may be involved but usually large joint such as knee or ankle

Risk Factors
- Extremes of life – very young and persons above 80yrs
- Immunosuppressive/ cytotoxic agents
- Immunosuppression – HIV, chronic renal, failure, hypogammaglobulinaemia
- Diabetes mellitus
- Previous intra – articular steroid
- Osteoarthritis
- Alcoholism
- Haemoglobinopathies
- Cutaneous ulcers
- Prosthetic joint

Clinical Features
- Usually monoarticular
- Acutely swollen joint
- Joint hot to touch, extremely painful
- Tenderness on palpation and movement
- High fever
- Rigors, diaphoresis

Differential Diagnosis
- Gout
- Pseudogout
- Reactive arthritis
- Haemarthrosis – possibly from aspirin

- Osteoarthritis
- Intra articular injury
- Osteonecrosis
- Metastic, carcinoma

Investigations

- Haematocrit, white blood cell count and differentials
- Joint aspirate – microscopy, culture and sensitivity
- Blood culture
- ESR, CRP
- X-ray of affected joints
- CT, MRI

Treatment

- Antibiotic treatment depending on bacteria isolated
- Cloxacillin or Flucloxacillin – 2mg IV qds
- Vancomycin – for MRSA resistant staphylococcus
- Ceftriaxone – 1 – 2gm once daily (IM or IV) for suspected gonococcus or meningococcus
- NSAIDs
- Surgical

  Daily needle aspiration

  Open drainage

  Arthroscopic debridement with lavage

  Intravenous therapy for 10 -14 days and then oral antibiotics for up to 6 weeks

Complications:

- Septicaemia
- Joint ankylosis

RHEUMATOID ARTHRITIS

- Rheumatoid arthritis is a chronic auto-immune inflammatory arthritis.
- Typically affects females more than males and constitutes 10 – 15% of all patients seen in rheumatology clinics in Nigeria.
- Affects all age groups but especially 40yrs and above
- Untreated badly managed, it could result in deformities, cardiovascular morbidities and decreased life expectancy
- It is characterized by affectation of small and medium sized joints in a symmetric fashion
- Primary lesion is a synovitis whereby immune competent all infiltrate the acellular synovium, leading to formation of 'pannus'. Auto antibodies such as rheumatoid factor.
- Pannus produces a lot of digestive enzymes, proteases, collagenases, metalloproteinase, cathepsin D
- Ultimately, destruction of cartilage, bone, tendons, ligaments
- Resultant vasculitis and atherosclerosis may affect internal organs such as lungs, heart, eyes

- Risk factors – genetic such as Shared Epitope of HLA DRBI; environmental such as infective agent – parvovirus, Epstein Barr virus, mycoplasma, smoking

Clinical features

- Diagnosis based on the American College of Rheumatology criteria (ACR)
- Lately the ACR and European League Against Rheumatism (EULAR) criteria
- Symptoms include constitutional features such as fever, loss of weight, loss of appetite, fatigue, nausea, anaemia
- Polyarthritis involving especially joints of the hands, elbows, shoulders, feet, knees, hip and temporo-mandibular
- Hardly ever affects joints of the spine except Atlanto – axial joint
- Dryness of the mouth and eyes
- Subcutaneous nodules especially the elbow
- Internal organ involvement may include pleural effusion, interstitial pneumonitis, pericarditis, pericardial effusion, ischemic heart disease, conjunctivitis, hepatoslenomegaly

Differential Diagnosis

- Polyarticular osteoarthritis
- Polyarticular gout
- Systemic Lupus Erythematosus
- Spondyloarthropathy

- Reactive Arthritis

Investigations
- Haematocrit, white blood cell count, ESR, CRP
- Liver function tests (as most of drugs used could be hepatotoxic)
- Cholesterol, triglycerides
- Rheumatoid factors
- Anti Cyclic Citrullinated Peptide

Imaging
- Plain radiographs especially of the hands and feet to demonstrate erosions
- Ultrasound of the joints to demonstrate synovitis and erosions
- CT scan
- MRI
- Radioisotope

Treatment of Objectives
To reduce pain and swelling
To prevent joint destruction and deformities
To prevent cardiovascular morbidities

Management
Non-pharmacologic treatment as in osteoarthritis
Disease modifying Antirheumatic drugs (DMARDS) are the mainstay and must be started immediately
- NSAIDs are supportive and should

be stopped if no pain or if any adverse effects

- Synthetic DMARDs:
  - Methotrexate – gold standard – 7.5mg – 25mg once weekly orally or subcutaneously
  - Sulfasalazine tablets – 1gm – 3gm daily in divided doses
  - Hydroxychloroquine – 200mg daily
  - Leflunomide – 20mg – 30mg daily after loading dose
  - Azathioprine – 2-3mg/kg body weight daily
  - Cyclophosphamide
- In some cases, combination therapy with Methotrexate + Sulfasalazine + Hydroxychloroquine or Methotrexate + Leflunomide

Biologic DMARD

- Used accordingly to guidelines if disease activity high after synthetic DMARDs
- Numerous groups

Anti TNF –Etanercept,

B Lymphocyte Depletor – Rituximab,

Corticosteroids – Prednisolone at low doses, less than 15mg eventually phased out within 6 months; intra – articular steroid

NSAIDs - used only in exacerbation of pain

Adverse Effects

NSAIDs – Dyspepsia, Peptic ulceration,

Gastro intestinal bleeding, hepatotoxicity, fixed drug eruptions.

DMARDs – susceptibility to infections, hepatotoxicity, elevated blood lipids, pancytopenia, bone marrow suppression, malignancy, teratogenicity

SYSTEMIC LUPUS ERYTHEMATOSUS (LUPUS)

- Systemic lupus erythematosus is a multisystemic auto immune disorder with a broad spectrum of manifestations
- Affects all organs and systems
- Deposition of immune complexes
- It presents as a chronic disease with a waxing and waning course
- Significant morbidity and possible mortality
- Affects mainly African Americans and Hispanic
- Affects mostly females of child bearing age [16 – 55yrs]
- Early damage due to the disease; late disease due to infections and atherosclerosis

Aetiology and pathogenesis

No definite aetiology, but genetic and environment factors implicated

- Female sex
- Genetic factors – commoner in monozygotic twins

- Epigenetic factors
- Environmental – ultraviolet rays, drugs; Epstein-Barr virus (EBV); viral(e.g. procainamide, hydralazine, methyldopa)
- Oral drugs such as oral contraceptives

Clinical features

- Affects all organs and systems – skin, joints, heart, lungs, kidneys, brain, pregnancy
- Diagnosis mostly by American College of Rheumatology (ACR) Criteria and lately, Systemic Lupus international collaborating clinics [SLICC] criteria
- Usually presents with fever, polyarthralgia, fatigue, loss of weight. Hair loss, skin rashes, mouth or pharyngeal ulcers.
- Specific organ involvement:

  Lung – pleurisy, pleural effusion, pulmonary fibrosis

  Heart – Pericarditis, pericardial effusion, ischemic heart disease

  Kidneys – Proteinuria, acute and chronic renal failure, nephrotic syndrome

  CNS – meningitis, encephalitis, seizure, psychosis

  Blood – Hemolytic anaemia, leucopenia, thrombocytopenia

  Pregnancy – Antiphospholipid syndrome – recurrent pregnancy losses, intra-uterine growth retardation

Differential Diagnosis

- Rheumatoid Arthritis
- Scleroderma, inflammatory myopathies
- Fibromyalgia
- Benign Hypermobility Syndrome

Investigations

- Haematocrit, white blood cell count, platelet count
- ESR, CRP
- Urine Analysis and microscopy – for casts, RBCs, protein
- Electrolytes and urea
- Chest X-ray
- ECG
- Serology – Anti-nuclear Antibody (ANA); Extractable Nuclear Antigen (ENA); Double stranded DNA
- Kidney Biopsy – as indicated

Management

- Corticosteroids- Prednisolone – 1–2mg/kg
- Anti-malarial – Hydroxychloroquine – 200mg–400mg daily
- Azathioprine – 2mg-3mg/kg body weight
- Mycophenolate Mofetil – 1gm–3gm daily
- Cyclosporine
- Cyclophosphamide
- Biologic DMARDs – Rituximab,

Non-pharmacologic

- Avoid sunlight
- Avoid physical and emotional stress
- Physical Exercise
- Sunscreen

CHAPTER 15:

OBSTETRICS AND GYNAECOLOGY

ABORTION

Introduction

Expulsion from the mother's uterus of a growing and developing embryo or fetus prior to the stage of viability (about 20 weeks), with fetal weight less than 50 g

Abortion is one of the leading causes of maternal morbidity and mortality in Nigeria

It may be:

Spontaneous- Occurring from natural causes or

Induced – Brought about purposefully by drugs or mechanical means

The different types are:

Complete-- With complete expulsion or extraction from the mother of a fetus or embryo, and of any other products of conception

Incomplete:- Parts of the products of conception have been expelled but some (usually the placenta) remain in the uterus

Unsafe

- Termination of a pregnancy in

circumstances where the law is restrictive

Safe: When it is carried out in accordance with legal prescriptions.

In Nigeria, abortion is legal when it is necessary to save the life of a woman. When therefore a doctor determines that the life of a woman is at stake and therefore cannot continue with a pregnancy and therefore carries out an abortion, the abortion is a safe abortion.

Abortions done in other circumstances is unsafe.

Abortion can also be:

Habitual:- A single experience of an abortion, or

Habitual:- When a woman has had three or more consecutive, spontaneous abortions

Clinical features

Threatened abortion:

Slight vaginal bleeding is often the only symptom

The fetus is often alive and viable

Imminent/incipient/impending abortion:

Copious vaginal bleeding

Uterine contractions

Inevitable abortion:

The bleeding is more severe

The cervix has started to dilate with

separation of the conceptus from the uterine bed

Rupture of the membranes in the presence of cervical dilatation in a pre-viable pregnancy

Ampulla/tubal abortion:

- Abortion of pregnancy in the ampulla of the fallopian tube or the tube itself
- Rupture of an oviduct, the seat of ectopic pregnancy
- Extrusion of the products of pregnancy through the fimbriated end of the oviduct
- Aborted ectopic pregnancy, the pregnancy having originated in the fallopian tube

Septic abortion: Complicated by fever, endometritis and parametritis

Differential diagnoses

Ectopic pregnancy, and Hydatidiform mole

Investigations

Pelvic ultrasound scan

Pregnancy test will be necessary in cases of threatened abortion

Microscopy, culture and sensitivity test of vaginal discharge

Urinalysis; urine microscopy, culture and sensitivity

Full Blood Count

Blood Group

HIV test, Hepatitis B and C antibodies

Complications

Endometritis, Parametritis, Peritonitis Haemorrhage, HIV infection, secondary infertility,

Perforation of the uterus and/or intestines rupture of the bladder

Treatment objectives

Restore hemostasis, prevent/treat complications, provide health education, and provide post-abortion family planning

Non-drug treatment

Nursing care

Psychological support

Personal hygiene

Drug treatment

Treat infection(s)

Replace fluid, electrolyte, and blood losses.

Complete/incomplete abortion: Surgical correction evacuation of retained products using manual vacuum aspiration (MVA) or misoprostol

Prevention

Promote personal and family understanding of basic reproductive health

- Universal basic education

- Girl child education
- Moral instruction

Protect vulnerable groups (young females) from undue exposure to their male folks

- At home
- In school
- Within peer groups

Legislation against street hawking for vulnerable groups Provide access to Primary Health Care and referral to efficient and effective higher levels of care Enforce existing laws on the criminality of abortion

Review existing laws on abortion with a view to promoting and protecting the overall wellbeing of mother and unborn child

ANTENATAL CARE (ANC)

Introduction

Antenatal Care (ANC) is the clinical assessment of mother and fetus, with the overall goal to obtain the best possible outcomes for both

ANC is an excellent example of preventive health care, as it deals mainly with normal individuals with an emphasis on the practice of health promotion

Availability, accessibility and utilization of ANC remain poor in Nigeria as in many other developing nations

Aims of antenatal care

- Assessment and management of maternal risk and symptoms
- Assessment and management of fetal risks
- Prenatal diagnosis and management of fetal abnormality
- Diagnosis and management of perinatal complications.
- Decisions regarding timing and mode of delivery.
- Parental education regarding pregnancy and childbirth
- Parental education regarding child-rearing

Providers of antenatal care

Community care, supervised predominantly by the midwife

- Shared care between the woman's general practitioner, midwife and obstetrician, with visits interspersed between all health professionals concerned-

Basic care component-

75% of pregnant women usually qualify for this

Hospital-only care:

- In cases where there is increased risk to the mother, fetus, or both- specialized care component
- A critical 25% of women will usually fall under this category

Schedule of visits during pregnancy

Previously, antenatal visits were:

- Monthly until 28 weeks gestation, then fortnightly until 36 weeks, and weekly thereafter until delivery, resulting in up to 14 hospital visits during pregnancy

Best available evidence indicates that there is no difference in outcome between a four-visit schedule and a twelve-visit schedule

- Current trends favour fewer visits, while establishing clearly defined objectives to be achieved at each visit.

This pattern of ANC is called Focused Antenatal Care

Pre-conception visit

1stANC visit

- Best before, and not later than the 12 week

2ndANC visit

- Scheduled around the 26, weeks

3rdANC visit

- Scheduled around the 32 week

4thANC visit

- Between the 36a, and 38a, week

Postnatal visit- scheduled within 1 week postnatally

This model is suited for the basic care component; the specialized care component is better managed with the 12-visit schedule

Intermittent preventive treatment in pregnancy using Sulphadoxine-pyrimethamine (IPTp-SP).

Administered as directly observed therapy (DOT) of three tablets of Sulphadoxine-pyrimethamine (each tablet containing 500mg/25mgSP).

Three doses of IPTp-SP is recommended

Starting as early as possible in 2nd Trimester (after quickening or 16-18 weeks)

IPTp-SP is recommended at each scheduled ANC visit until time of delivery provided that the doses are given one month apart.

IPTp-SP should be avoided in first trimester.

SP should not be administered to women receiving co-trimoxazole.

SP can be safely used in a woman on daily dose of 0.4mg of Folic acid.

Folic acid at a daily dose equal or above 5mg should not be given together with SP as this counteracts its efficacy as antimalaria.

Activities during each visit

Pre-conception visit

Assess the general health and well-being of the patient

Take appropriate action based on the outcome assessment

General advice regarding nutrition and life style

1stANC visit

Should be in the 1st trimester, preferably before the 12«,week

Should last between 30 - 40 minutes. Key objective is to obtain the patient's medical and obstetric history:

- Assess the woman's eligibility to follow the basic component of the new WHO model using the classifying form which contains 18 sets of questions.

Activities during the visit should include:

Physical examination

- General examination including height and weight
- Blood pressure
- Chest and heart auscultation
- Symphyio-fundal height (SFH) measurement and abdominal palpation
- Vaginal examination - specifically for Pap smear if the woman has not done one in the past 2 years; also for women with past history suggestive of cervical incompetence - assessment and referral
- Any medical or obstetric conditions that require specialized care

Investigations -Urinalysis for bacteriuria, proteinuria and glycosuria

- Haemoglobin, genotype
- Blood group
- HIV screening
- VDRL
- Hepatitis B and C screening

- Haemoglobin concentration/packed cell volume

Interventions

Iron and folate

Tetanus toxoid- 1st injection.

Treat for syphilis if VDRL is positive.

Refer if other investigation results so require.

Allow time for advice, questions and answers, and scheduling of next appointment.

Maintain complete clinic records of all transactions of the visit

2nd ANC visit

Should be close to, or at 26thweek

Expected to take about 20 minutes.

Activities during the visit should include:

Review of history for any changes.

Assessment of adherence to routine ANC medicines.

Assess for referral

- Update the risk status and refer if the need arises.

Physical examination

- General examination: pallor, edema
- Blood pressure
- SFH measurement

Investigations

Urinalysis for bacteriuria,

proteinuria -for nulliparous women and those with a history of hypertension or pre-eclampsia/ eclampsia

Haemoglobin concentration/packed cell volume only if there is evidence of anaemia

Interventions

Iron

Folic acid

Malaria prophylaxis

- Intermittent treatment with sulfadoxine/pyrimethamine
- One full treatment dose in the 2 and 3rdtrimesters
- Last dose not later than 1 month before the Expected Date of Delivery

Or:

- Proguanil 100 - 200 mg orally daily

Maintain complete clinic records as well as ANC card records

3rd ANC:

Should be around the 32nd week

Expected to take about 20 minutes.

Activities during the visit:

Review history for any changes

Assess adherence to routine ANC medicines.

Extra attention to advice on

- What to do if labour occurs
- What to do if membranes rupture
- Birth spacing and counselling on contraception.
- Assess for referral

Physical examination

- General examination: pallor, edema, dyspnea
- Breast examination
- Blood pressure
- Abdomen: SFH palpation to exclude twin gestation, and fetal growth retardation

Investigations

- Haemoglobin concentration or packed cell volume
- compulsory for all in this visit
- Urinalysis: bacteriuria, proteinuria; for nullipara and those with hypertension, pre-eclampsia/eclampsia

Interventions

Iron

Folic acid

Tetanus toxoid (2 injections)

Antimalarial drugs

Maintain complete records: clinic as well as ANC card records

4thANC visit

The final visit before labour or delivery

Should take place about or between the 36 - 38weeks

Activities during the visit include:
Review history for any changes
Assessment of adherence to routine ANC medicines
Physical examination
- General examination
- Blood pressure
- Abdomen: SFH, fetal lie and presentation; presence of multiple gestations
- Advise on the concept of prolonged pregnancy and the need to present if still not in labour by the 41 week

Investigations
Urine: proteinuria; only in nullipara, hypertension, pre-eclampsia/eclampsia
Assess for referral

Interventions
Iron
Folic acid
Malaria prophylaxis
Advice, questions and answers; scheduling next appointment
Maintain complete records: clinic as well as ANC card records

Malaria treatment for breakthrough episodes
Quinine is safe and can be used in all trimesters

Artemisinin-based combinations are safe in the 2 and 3rdtrimesters

- Artemether-lumefantrine is considered safe

Postnatal visit

Should hold within 1 week postpartum

Offer contraception

Complete tetanus prophylaxis with tetanus toxoid

Continue interventions: iron, folic acid and malaria prophylaxis

ANAEMIA IN PREGNANCY

Anaemia is the most common complication of pregnancy in sub-Saharan Africa

It is a diminution below normal of the total circulating haemoglobin mass

World Health Organization definition of anaemia

- Haemoglobin concentration less than 11 g/dL or a hematocrit less than 33% in peripheral blood

For practical purposes in developing and tropical countries a haemoglobin concentration of 10 g/dL or hematocrit of 30% is taken as cut off

- Below these levels there may be adverse fetal and maternal outcomes

Classification

Mild - PCV 25 - 29%

Moderate - PCV 20 - 24%
Severe - PCV < 20%

Clinical presentation
Varies; depends on the severity
- May be asymptomatic or symptomatic

Symptoms
Generalised weakness
Lassitude
Easy fatigability
Headaches
Dyspnea on mild exertion
Ankle swelling

Signs
Pallor
Jaundice may or may not be present
Pedal edema
Tachypnea
Tachycardia
Haemic murmurs
Pseudo-toxaemia
- Systolic hypertension, edema and albuminuria

There may, or may not be clinical evidence of causative pathology
- Sickle cell facies, urinary tract symptoms, etc.

Hepatomegaly: not invariable
Splenomegaly: not invariable
Anaemic heart failure in extreme cases

Differential diagnoses

Nutritional deficiencies

- Iron, folic acid, protein, vitamin C; trace elements, and rarely vitamin B 12

Physiological demands of pregnancy

Excessive red cell haemolysis as in malaria, haemoglobinopathies

Infections: urinary tract infection, HIV/AIDS

Hookworm infestation

Antepartum haemorrhage

Bone marrow pathologies

Miscellaneous: e.g. bleeding duodenal ulcer

Complications

Maternal

Abortion

Cardiac failure

Reduced ability to tolerate blood loss at delivery

Reduced ability to tolerate anaesthesia

Diminished resistance to infection

Preterm labour. Precipitate labour may occur

Death

Spontaneous abortion

Intrauterine growth restriction. Intrauterine fetal death

Still birth

Prematurity

Risk of developing anaemia within 2 - 3 months of birth if mother suffered iron

deficiency anaemia

Investigations

Haematocrit
Haemoglobin concentration
White blood cell count and differentials
Blood picture
Reticulocyte count
Blood smear
Midstream urine: microscopy, culture and sensitivity
Stool analysis: ova, cysts, parasites, occult blood
Group and cross-match blood
Haemoglobin genotype Blood Group
VDRL
HIV screening. Urinalysis and culture.
Ultrasound scan (e.g. of abdomen, pelvis).
Bone marrow biopsy if bone marrow involvement is suspected

Treatment objectives

Correct hematocrit.
Treat underlying cause(s)
- See differential diagnoses.

Fetal surveillance:
- Of growth and wellbeing to exclude IUGR and intrauterine asphyxia

Correction of hematocrit:

Oral haematinics
- For mild and moderate anaemia -

Ferrous sulfate

- 200 mg daily and folic acid 5 mg daily Vitamin C (ascorbic acid)
- 100 mg three times daily.
- Parenteral iron: indicated in
- Mild to moderate anaemia, near term
- Malabsorption of oral iron, or when it causes serious gastroenteritis

Administration:

Calculate haemoglobin deficit

For each 1 g/dL deficit, 250 mg of iron dextran injection is required

Additionally, 50% of the total calculated is added onto the deficit value to take care of the iron stores

Administer by deep intramuscular injection into the gluteal muscle, by slow intravenous injection or by intravenous Infusion (after a negative test dose)

Intramuscular injection

- 250 mg daily; after a negative test dose of 25 mg Intravenous
- If the total calculated dose of iron dextran is less than 1,500 mg it can be given over 8 hours in one litre of sodium chloride 0.9%
- If greater than 1,500 mg, it should be given in divided doses daily, not exceeding 1,500 mg/day *Antihistamine (chlorphenamine injection), epinephrine and hydrocortisone injection must be available: iron dextran could cause severe anaphylaxis*

Blood transfusion

- Consider as from the 37 week for mild anaemia and from the 32 week for moderate anaemia
- Usually, packed cells under furosemide cover

Indications:

Severe anaemia irrespective of gestational age

Cardiac failure

Moderate anaemia detected in labour or during an abortion, or co-existing with other conditions such as sepsis, renal failure, haemorrhage or eclampsia

Prevention

Counselling on contraception; adequate spacing of pregnancies.

Malaria prophylaxis in pregnancy.

Chemoprophylaxis against helminthiasis.

Prompt and appropriate treatment of febrile illnesses in pregnancy

Improvement of the socioeconomic status of the people

Provision of accessible and affordable maternity care facilities

CANCER OF THE CERVIX

Introduction

The second most common malignancy and the leading cause of death among women in developing countries

- 75% of the patients present in advanced stages; lack of organized screening programmes for detection of the preclinical stages in many countries

Aetiology/risk factors

Aetiology not known but several risk factors have been implicated:

Early sexual exposure

Multiple sexual partners

A promiscuous male partner

History of sexually transmitted infections particularly Human Papilloma Virus infection; Herpes simplex type 2; chlamydia trachomatis

Early first child birth

High parity

Low socio-economic status. Smoking

Micronutrient deficiency. Poor sexual hygiene

Clinical features

Two age groups with highest incidence: 35 - 40 years; 45 - 55 years.

May be asymptomatic

- Picked up in the early stage by routine PAP smear screening

Abnormal vaginal bleeding:

- Post-coital
- Contact
- Spontaneous

- Inter-menstrual

Post-menopausal Vaginal discharge.Becomes offensive in advanced disease. Pyometria with uterine enlargement

Haemorrhagic, ulcerative or fungating lesion on the cervix, with extension to the vagina wall in advanced stages. Vesico-vaginal fistula in advanced stages. Also, there may be recto-vaginal fistula in advanced stages Cachexia

- The presence of a lesion on the cervix

Presumptive Diagnosis

Based on :

- Typical history of risk factors
- Histological confirmation of malignancy

Differential diagnoses

Endometrial cancer.Endometrial hyperplasia.Endometrial polyps. Cervicitis, Cervical polyps, Cervical erosion

Vaginal lesions: vaginitis, vaginal malignancy Functioning tumours of the ovary leading to endometrial hyperplasia and vaginal bleeding Iatrogenic: hormonal drugs and IUCD in-situ, Blood disorders: bleeding dyscrasias, leukaemia

Investigations

Packed cell volume; haemoglobin concentration

Urinalysis
Blood Group
White cell count, and differentials. Electrolytes and Urea, Liver function tests
Midstream urine specimen for microscopy, culture and sensitivity; Chest radiograph; HIV screening. Intravenous urography

Principles of management
Examination Under Anaesthesia
- Staging and Biopsy

Treatment of invasive carcinoma of the cervix
- Surgery
- Radiotherapy
- Surgery plus radiotherapy
- Chemo-radiation

Treatment options will depend on
the skill of the surgeon, availability of facilities, the stage of the disease, Age of the patient, and the
Ability of available personnel to manage untoward effects of the modality of treatment chosen

Stages I to IIA
Surgery or radiotherapy (as primary modes of treatment respectively)
- Radiotherapy can be used as primary mode of treatment in all stages of the disease

Follow up

This is for life. Regular cytology of vault smears for early detection and prompt treatment of recurrence.

Prevention

Adequate screening programmes: Papanicolaou smear Visual inspection of the cervix after acetic acid lavage (VIA). Testing for the human papilloma virus. DNA Specific programmes targeted at eliminating or mitigating the effects of recognized risk factors

CARDIAC DISEASE IN PREGNANCY

Introduction

A rare but potentially serious clinical entity.Occurs in about 1% of all pregnancies. Incidence and prevalence of all heart disease varies from place to place.

- Rheumatic heart disease is more commonly found in less affluent societies while congenital heart disease now accounts for approximately 50% of cardiac diseases in pregnancy in the UK.

Types of cardiac diseases in pregnancy

Acquired

Rheumatic heart diseases

- Mitral > Aortic > Tricuspid > Pulmonary

Cardiomyopathies

- Particularly peripartum cardiomyopathy which could be either congestive or obstructive

Pre-existing hypertensive heart disease
Ischaemic heart disease

Congenital

Acyanotic heart disease

- Atrial septal defect, ventricular septal defect, patent ductus arteriosus, etc.

Cyanotic heart disease

- Tetralogy of Fallot, Eisenmenger's syndrome, Acquired forms of cardiac disease appear to be more lethal in association with pregnancy, in women aged 25 years or more, and in third or later pregnancies

Congenital malformations are more prevalent in younger women and in those of lower parity

Clinical features

Severity of heart disease in pregnancy

The New York Heart Association Guidelines (1965) is used.

- Relies on the cardiac response to physical activity; may not bear any relationship to the extent of the lesion present

Class 1

- No limitation of physical activity Class 2
- Slight to moderate limitation of physical

activity: ordinary day-to-day activities cause dyspnoea

Class 3

- Marked limitation of activity. Minimal exertion causes dyspnoea

Class 4

- Symptoms at rest; unable to carry out any physical activity without dyspnoea; orthopnoea may be present

Other symptoms

Palpitations Nasal stuffiness

Dizziness; light headedness; syncope

Epigastric or sub-xiphoid pain; bloating, heartburn

Heat intolerance, sweating and flushing

Signs

Plethoric facies

Odema (legs; occasionally hands and face) Varicoseveins

Bounding pulses and capillary pulsations. Capillary telangiectasia.Prominent jugular venous pulsations. Lateral displacement of cardiac apex. Sinus tachycardia; ectopic beats Third heart sound

Widely split S and S heart sounds1

Murmurs. Crepitations

Investigations

Full Blood Count
Serum Electrolytes, Urea and Creatinine
Urinalysis
Blood Glucose
Bedside crude clotting time
Echocardiography (Doppler)
Electrocardiography
Serial blood cultures (if infective endocarditis is suspected)
Chest radiograph is better avoided in pregnancy

Management

Pre-pregnancy

Fully evaluate patient in conjunction with a cardiologist
Surgically correct any defect that is amenable
Counsel on the following points:

- Risk of maternal death
- Possible reduction of maternal life expectancy
- Risk of fetus developing congenital heart disease; fetal growth restriction
- Possibility of pre-term labour
- Need for frequent hospital attendance; possible admission
- Need for intensive maternal and fetal monitoring in labour

Antenatal Care

Joint management with the cardiologist
Extreme vigilance: most features of cardiac

failure are present in pregnancy

Watch out for respiratory tract infection or urinary tract infection and treat aggressively

Watch out for anaemia, obesity and multiple gestations for intensive care. Intensive care also required when other medical or psychological conditions co-exist.

Examination:

- Ankle and sacral edema
- Pulse rate and rhythm
- Blood pressure
- Jugular venous pressure
- Basal crepitations
- Symphysio-fundal height (SFH) measurement.
- Competent dental care:
- Full inspection
- Advise on oral hygiene
- Dental treatment e.g. tooth extraction should be done under antibiotic cover to prevent infective endocarditis

Admission

- Individualised; usually when complications or intercurrent illnesses occur

Supportive measures

Elastic stockings or tights to prevent pooling of blood in the veins of the lower limb

- Indicated for example in patients with congenital heart disease, with pulmonary hypertension; artificial valve replacements; those with atrial

fibrillation

- Heparin safer in pregnancy; warfarin is teratogenic Termination of pregnancy and sterilization
- Best option in severe debilitating cases

Congestive Cardiac Failure

Manage as if non-pregnant (in conjunction with a cardiologist)

Fetal surveillance:

- Ultrasound scan particularly for cardiac anomaly at 22 weeks

Delivery:

- Either for maternal or fetal indications. Cardiac surgery in pregnancy if indicated

Management of labour in women with cardiac disease

Avoid induction of labour if possible

Prophylactic antibiotics to prevent bacterial endocarditis

Careful fluid balance

Avoid the supine position

Epidural anaesthesia by a senior anaesthetist

Shorten 2 stage with low cavity forceps delivery Oxytocin for third stage; ergometrine is contraindicated Oxygen should be available and used if needed

Complications

Maternal

Mortality:

- 25 - 50% in Eisenmenger's syndrome; 5% in tetralogy of Fallot; 1% in rheumatic heart disease

Congestive cardiac failure:

- Greatest risk in the immediate post-partum period

Fetal

Rheumatic heart disease:
Intrauterine growth restriction;
pre-term delivery
Cyanotic congenital heart disease:
Poor outcomes; up 40% fetal loss
Uncorrected coarctation of aorta:
Fetal growth restriction in > 10% of cases
Pre-maturity
Small for gestation age
Intrauterine growth restriction
Intrauterine fetal death
10 - 15% chance of baby having congenital heart disease

ECLAMPSIA

Introduction

The occurrence of generalized convulsions, associated with signs of pre-eclampsia during pregnancy, labour, or within 7 days of delivery; not anticoagulation

Referred to as atypical eclampsia if it occurs

- In the absence of high blood pressure
- After 7 days post-partum

Incidence is widely variable. Worldwide range reported to be 1 in 100 - 1 in 3,448 pregnancies In Nigeria, it is commoner among un-booked patients.

Aetiology

Not exactly known. Its precursor is pre-eclampsia. A disease of primigravidae, or multigravidae with pregnancy for a new consort.

Clinical features

Generalized tonic-clonic seizures, usually heralded by:

- Headaches
- Dizziness and blurring of vision
- Nausea and vomiting
- Epigastric pain
- Rapidly progressive edema.
- Exaggerated tendon reflexes.
- Oliguria

Hypertension.

Worsening proteinuria

Complications

Maternal

Cerebral haemorrhage

Disseminated intravascular coagulopathy

Renal failure

Cardiopulmonary failure

Liver dysfunction (as in HELLP syndrome)

Fatality

Fetal

Prematurity

Intrauterine growth restriction.Intrauterine fetal death. Fetal brain damage

Differential diagnoses

Idiopathic epilepsy: sometimes accompanied by transient proteinuria

Cerebral malaria: sometimes accompanied by hypertension and albuminuria

Pneumococcal meningitis

Hyper and/or hypo-glycaemia, particularly among diabetics.

Terminal phase of severe anaemia

Terminal phase of hepatic failure.

Severe infections and septicaemia.

Others:

- Uremia
- Brain tumours or abscesses
- Cerebral haemorrhage
- Poisoning (accidental or intentional)
- Hysteria

Investigations

Haemoglobin concentration/hematocrit

Haemoglobin genotype, Platelet count, Blood Group

Serum Urea and Electrolytes; Creatinine

Liver function tests

Urinalysis

Management

Manage in conjunction with the physician

Treatment objectives

Stabilise the patient

Deliver fetus by the safest and most expeditious route

Prevent complications

Stabilization

Control (and prevent further) fits

Control blood pressure

Maintain the airway

Ensure adequate urinary output

Monitor

Controlling fits

Intravenous diazepam -10mg stat to abort seizures or prevent fits during examination; then

- Intravenous infusion of glucose 5% in water with 40 mg of diazepam added, and titrated against the patient's level of consciousness

Magnesium sulfate (see details below)

Treatment packs are contained in cardboard boxes containing magnesium sulfate for the loading dose, 24hour maintenance therapy and treatment of one (recurrent) convulsion. Syringes, swabs, drip sets and fluids also contained in treatment packs.;

- Calcium gluconate should always be available to manage toxicity

Intravenous infusion of magnesium sulfate

- Loading dose: 4 g by slow intravenous injection over a period not less than 5 minutes (preferably over 10 - 15 minutes)
- Maintenance: 10 g in 1 litre of sodium chloride 0.9%, given by intravenous infusion at a rate of 1g per hour

The intramuscular magnesium sulfate (Pritchard) regimen

- Loading dose: 4 g by slow intravenous injection over a period not less than 5 minutes, then 10 g intramuscularly, 5 g by deep intramuscular injection into each buttock
- Maintenance therapy: 5 g by deep intramuscular injection, 2.5 g in each buttock every 4 hours
- Continue for 24 hours after last convulsion, or delivery.

Recurrent convulsions

Magnesium sulfate

- 2-4g intravenously over 5 minutes
- Give lower dose (2 g) if the patient is small and/or weight is less than 70 kg

Monitoring during magnesium sulfate therapy

Continue with intravenous infusion or give the next intramuscular dose **only** if

- Patellar reflexes are normal

- Respiratory rate is > 16 cycles/minute
- Urine output is > 25 mL/hour (or > 100 mL in 4 hours)

Consider reducing the dose if
- Renal function is impaired
- Respiratory depression occurs
- Urine output is < 100 mL in 4 hours

More frequent monitoring is required in the first two hours on intravenous therapy

Magnesium toxicity

Absent patellar reflexes:
Stop magnesium sulfate treatment
Administer oxygen by face mask
Administer 1g calcium gluconate by slow intravenous injection.

If respiratory rate is abnormal:
Stop further magnesium sulfate

If there are no respiratory abnormalities or abnormal patellar reflexes:
Reduce the dose by half.

Respiratory arrest:
Stop magnesium sulfate treatment
Intubate and ensure ventilation (manage with the anaesthetist)
Calcium gluconate 1 g by slow intravenous injection.

Control of blood pressure

Intravenous hydralazine

- 5 mg bolus slowly over 15 minutes, stat.

Further boluses can be given every 20 - 30 minutes as long as diastolic blood pressure is 110 mg and above

Or:

Labetalol

- 20 mg intravenously as a bolus
- Repeat after 15 - 20 minutes (if need be, increasing the doses)

The airway:

- Intermittent suction of the nostrils and oropharynx
- Insert an airway

Urinary output:

- In-dwelling Foley's catheter for strict fluid input and output monitoring.

Monitoring:

- Quarter-hourly vital signs
- Record any further fits

Delivery

Induction of labour

- Is the first option if the cervix is favourable, particularly if the patient is not yet in established labour
- Can be done by the use of escalating doses of oxytocin infusion or with misoprostol tablets

Elective forceps delivery

- Should be done if patient is in the second stage to reduce the stress and cardiovascular changes, especially peaks of elevated blood pressure that accompany expulsive efforts at this stage in labour

Emergency Caesarean section is indicated when:

- Cervix is unfavourable for induction
- There is fetal distress
- Patient is unconscious (unless delivery is imminent)
- Vaginal delivery is unlikely within 6 - 8 hours from the onset of the first eclamptic fit and there is an obstetric indication for a Caesarean section

Post-partum

Continue parenteral anticonvulsant for another 24 hours after delivery (or after last seizure), whichever comes first

Prevention

Adequate antenatal, intrapartum and postpartum care.

Early detection of pregnancy-induced hypertension.

Aggressive management.

This is the 'gold standard' towards achieving good fetal and maternal outcomes

Re-occurrence

- Occurs in 15.6% of cases
- Adequate counselling on the need for early booking, regular antenatal clinic attendance and hospital delivery in subsequent deliveries required.

ECTOPIC PREGNANCY

Introduction

Pregnancy in which the conceptus implants either outside the uterus (fallopian tube, ovary or abdominal cavity) or in an abnormal position within the uterus (cornua, cervix, angular and rudimentary horn)

The most common surgical emergency in women in many developing countries.

A substantial cause of maternal mortality:

- Rapidity with which haemorrhage and shock occur
- Pre-rupture diagnosis is elusive, with consequent delay in surgical management

Clinical features:

The clinical subsets include:

Acute ectopic gestation

- 25% or less of cases

Sub-acute ectopic gestation

- 75% of cases

"Silent" ectopic/chronic ectopic gestation

Acute Ectopic Gestation

Amenorrhoea

Features of acute abdomen particularly lower abdominal pain, vaginal bleeding or brownish discharge, severe pallor, shoulder tip pain.

Difficulty with sitting on hard surfaces.

Features of shock with cardiovascular collapse: hypotension and tachycardia

The uterus is slightly enlarged with tenderness on one side

- Some advice that examination should be avoided if there is a strong suspicion of an ectopic pregnancy

Positive cervical excitation tenderness

Sub-acute Ectopic Gestation

Slow-leaking ectopic prior to rupture, with most of the signs and symptoms of acute ectopic gestation present but in the mildest form.

"Silent" vs. "Chronic" Ectopic Gestation

Asymptomatic

- May just be picked up during a pelvic examination in the course of booking or antenatal clinic, or found on ultrasound for another pelvic pathology

Complications

Shock

Sterility (with the loss of both tubes). Often requires blood transfusion (with its attendant cost and risk of blood-borne infections) 5-20% risk of having another ectopic gestation

Fatality

Diagnosis

Requires a high index of suspicion particularly in the case of atypical, slow-leaking or chronic ectopic gestation where diagnosis could be difficult

Differential diagnoses

For unruptured ectopic pregnancy: Acute pelvic inflammatory disease, Adnexal torsion, incomplete abortion, Endometriosis Degenerating uterine fibroid, Acute appendicitis, Accidented ovarian cysts

Investigations

Haemoglobin concentration/packed cell volume

Blood grouping and cross matching

Urinalysis

Ultrasound scan of the pelvis (preferably trans-vaginal) ; Serum B-hCG (where available) especially in silent cases

Laparoscopy may be necessary when still in doubt, but the availability of trans-vaginal

ultrasound scan may obviate this

- Final arbiter when the diagnosis is in doubt is exploratory laparotomy

Treatment objectives

Depend on the clinical subset. The objective is to preserve maternal life

Acute ectopic

Immediate resuscitation (fluids/blood). Stop haemorrhage by emergency surgery. Replace lost blood.

General principles and treatment modalities

Surgery

- Salpingectomy (total or partial) for ruptured ectopic pregnancy
- Partial salpingectomy if the remaining segment of the tube is about 4 cm long; this could be used for reconstructive surgery subsequently
- Salpingostomy for unruptured cases.

Non-surgical options

- Used in unruptured cases: expectant management and medical agents

Expectant management

- Monitor pregnancy by -hCG levels
- Vaginal scans: spontaneous resorption can occur provided gestation sac is < 4 cm and hCG is <1,500 IU

Medical treatment

- Methotrexate

Administered systemically or locally to induce dissolution of trophoblastic tissue (Ru 486)

- Hyperosmolar glucose solution, potassium chloride and prostaglandins can also been used

Auto transfusion

- During surgery for ectopic gestation; very important in developing countries with inadequate blood banking services
- The risks of transfusion with donated blood are avoided
- Use only fresh blood.

On discharge:

- Counsel for contraception and advise to report immediately to the hospital if a pregnancy is suspected so that its site can be confirmed

HYPEREMESIS GRAVIDARUM

Introduction

A clinical situation in which vomiting in early pregnancy considered to be physiological becomes persistent or severe enough to disturb the patient's health and/or require hospitalization. Occurs in approximately a third to 50% of women

- Often the first sign of pregnancy, beginning at about the 6 week and stops spontaneously before the 14thweek

Generally limited to the early morning, but may occur at other times of the day. Cause is essentially unknown, but hypotheses include
Hormonal:

- Increased sensitivity to placental hormones such as hCG, estrogen or progesterone.
- May be psychogenic:
- The woman thinks she should have early morning sickness because generations before her have had it.

Clinical features

Persistent and severe vomiting that leads to electrolyte and nutritional derangements

Differential diagnoses

It is a diagnosis of exclusion. Concerted effort must be made to exclude the under listed causes of pathological vomiting:
Multiple gestations
Hydatidiform mole
Malaria in pregnancy
Gastrointestinal disorders:
Heartburn due to hiatus hernia: a common cause of vomiting in late pregnancy
Enteritis
Appendicitis
Peptic ulcer disease
Hepatitis
Acute fatty liver of pregnancy

Pancreatitis
Cholecystitis
Urinary tract disorders: pyelonephritis
Acute polyhydramnios
- Commonly associated with monozygotic twinning and diabetic pregnancies

Pre-eclampsia Accidents to ovarian cysts
- Torsion, haemorrhage, infection and rupture Red degeneration in a fibroid

Complications

Biochemical abnormalities
- Usually sequel to vomiting, starvation and dehydration
- Ketosis, electrolyte imbalance (alkalosis and hypokalemia); vitamin deficiencies

In neglected or poorly managed cases: severe weight loss, Tachycardia, Hypotension, Oliguria

Neurologic disorders from vitamin B deficiency

Retinal haemorrhages, Jaundice (from hepatic necrosis), Oesophageal tears and spontaneous rupture of the oesophagus, Mendelson's syndrome, Fetal loss, Maternal mortality

Investigations

Full Blood Count with differentials; Urea, Electrolytes and Creatinine; Liver function tests;

Midstream urine for microscopy, culture and sensitivity; Urinalysis for ketones;
Blood film for malaria parasites; Ultrasound scan of the pelvis/abdomen to rule out multiple or abnormal pregnancies

Management
Admit
Strict intake-output monitoring Intravenous fluid therapy to:
- Correct electrolyte disturbances
- Provide calories
- Rehydrate the patient
- Anti-emetics
- Those that have been proven not to be teratogenic:
- Cyclizine 50 mg orally

Or:
- Promethazine 25 mg orally

All of these are taken three times daily.
Total parenteral nutrition
- In severe cases

In persistent and intractable cases with significant maternal complications, termination of pregnancy may be considered

JAUNDICE IN PREGNANCY
Introduction
Usually indicates a liver/biliary disorder and becomes clinically apparent when the serum bilirubin exceeds 2 - 2.5 mg/dL
Many indicators of liver disease in the non-

pregnant State are normal findings in pregnancy. These include:

- Spider naevi
- Decreased plasma albumin
- Increased serum lipids

Prothrombin time, transaminases and bilirubin are unaltered in normal pregnancy

Jaundice occurs in about 1 in 1,500 - 2,000 pregnancies

Aetiology

Aetiology peculiar to pregnancy

Hyperemesis gravidarum

Pre-eclampsia and eclampsia as seen with HELLP syndrome

Acute yellow atrophy (acute fatty liver in pregnancy; acute hepatic failure)

Intra-hepatic cholestasis of pregnancy

Cholestasis in pregnancy Gallstones

Aetiology not peculiar to pregnancy

Viral hepatitis

Haemolytic jaundice

Adverse reactions to drugs e.g. chlorpromazine, tetracycline

Congenital hyperbilirubinaemias such as Dubin-Johnson syndrome

Liver cirrhosis

Clinical features

Acute yellow atrophy

A rare and serious disorder associated with

high mortality
Occurs in the order of 1: 10,000 pregnancies
Unknown aetiology
Typically noted in primigravidae, occurring after the
30th week of pregnancy or few days after birth
The jaundice is classically obstructive
Onset usually sudden with

- Abdominal pain (right upper quadrant)
- Headaches
- Nausea and vomiting
- Progressive jaundice
- Encephalopathy
- Hypertension is not uncommon

Histology
Perilobular fatty infiltration of the liver cells
There is no place for liver biopsy because of risk of severe bleeding complications

Management
Early diagnosis is mandatory

- Clinical features with evidence of deranged LFTs and of renal failure

The management requires a combined team of obstetrician, physician and anaesthetist

Definitive treatment
Deliver the baby as soon as possible (frequently by
Caesarean section)

Supportive measures
Transfusion with blood, fresh frozen plasma, platelets as indicated
Renal dialysis

Complications
Disseminated intravascular coagulopathy
Hypotension
Significant risk of maternal and fetal death due to:
- Maternal liver failure
- Metabolic disturbance
- Encephalopathy
- Overwhelming haemorrhage associated with clotting defects

Prognosis
Good
Post-natally, liver function returns to normal over a few weeks and there is no evidence of long-term liver dysfunction

Cholestasis of pregnancy
Uncommon, in the order of 1: 2,000 pregnancies
Common in certain southern American countries particularly Chile
Presents commonly in late third trimester, after 36 weeks
Clinically significant because of its

association with intra-uterine fetal growth retardation (IUGR) and intra-uterine fetal death (IUFD) (mechanism unclear)
It is not as a rule associated with maternal complications

Clinical features
Generalized pruritus
Decreased fetal movements
Upper abdominal pain
Dark urine
Steatorrhea
Occasionally there is jaundice (particularly in the later stages of the disease)

Investigations
Liver function tests:
- Mildly deranged
- Serum bilirubin and bile salts may be elevated

Differential diagnoses
Viral hepatitis
Early HELLP syndrome
Acute fatty liver

Management
Careful maternal follow-up with LFTs
Fetal surveillance: by growth (serial ultrasound biometry) and wellbeing (cardio-tocographic fetal) monitoring

If all is well induce at 38 weeks
Management of associated pruritus:
(Difficult to manage)
Topical agents offer little help
Colestyramine
- To bind bile salts

Vitamin K
- To decrease bleeding tendencies
- (Colestyramine binds fat soluble vitamins)

Antihistamines
- May offer brief respite

Ursodeoxycholic acid and colestyramine (orally) decrease itching and normalize liver function
Adult: 10 - 15 mg/kg daily in 2 - 4 divided doses
Child 1 month -18 years: 10 - 15 mg/kg twice daily; total dose may be given in 3 divided doses
Recurrence
Quite high

Prognosis
Good
- Complete recovery in days to weeks

Dubin-Johnson syndrome
Intermittent bilirubinaemia (conjugated)
Often chronic and familial
No itching, usually asymptomatic

Cause is unknown

Treatment
None is required

Intra-hepatic cholestasis of pregnancy
Also termed 'recurrent obstructive jaundice' or 'idiopathic cholestasis'
Thought to be due to the effect of high estrogen levels on the liver, which results in decreased conjugation of bilirubin
A rare condition
- Incidence of 1:500 pregnancies

More commonly seen in among Scandinavians
Its exact etiology is unknown

Clinical features
Intense pruritus due to retention of bile salts
The most common presenting symptom and may occur in the absence of other symptoms
Onset of symptoms usually in the third trimester
Jaundice is not often seen

Investigations
Bilirubinuria
Elevated bile acids
Elevated alkaline phosphatase
Elevated liver transferase enzymes
Prothrombin time

Always exclude viral disease, gallstones and treatment with chlorpromazine

Complications

Maternal

Haemorrhage
Preterm labour
Steatorrhea

Fetal

Fetal distress
Still-birth
Perinatal death
Prematurity and its problems
Meconium staining of the liquor

Management

Careful maternal follow-up with LFTs
Fetal surveillance: by growth (serial USS biometry) and well-being (CTG) monitoring
If all is well, induce at 38 weeks
Management of pruritus
-See Cholestasis of pregnancy

Recurrence

Risk of recurrence is 50%
Can be precipitated by estrogen-containing oral contraceptive pills

Viral hepatitis

The most common cause of jaundice in

pregnancy, accounting for about 40% of cases
Incidence during pregnancy is probably no more than in the normal population
Pregnancy does not alter the course of the disease
Hepatitis A virus does not affect the fetus, but hepatitis B and C can cross over the placenta to affect the fetus

- Unlike other hepatotrophic viral infections, which carry a significant risk of vertical transmission (particularly in the third trimester)

A severe attack may influence fetal outcome

- Slight increase in premature labour and stillbirths (as seen in any severe medical illness)

Treatment
Avoid any further damage to the liver by drugs
Bed rest
Adequate nutrition
If hepatitis B is present then the infant requires protection with immunoglobulins against HBsAg

- Hepatitis B immunoglobulin by intramuscular

Injection *Neonate: 200* units as soon as possible after birth
Child 1 month - 5 years: 200 units; 5 - 10 years: 300 units; 10 - 18 years: 500 units

Avoid breastfeeding

Delivery room personnel must exercise great care in dealing with these patients, as all their body fluids are highly infectious

Immediate delivery if hepatitis becomes fulminant

Ideally, all women should be tested for hepatitis B and C antibodies at the first antenatal visit. And women should be immunized against these viruses before pregnancy.

PELVIC INFLAMMATORY DISEASE

Introduction

Ascending pelvic infection involving the upper genital tract

Usually involves sexually transmitted organisms e.g. *Neisseria gonorrhoeae* and *Chlamydia trachomatis*

- It may also be caused by organisms endogenous to the lower genital tract.

In severe cases, organisms may migrate via the peritoneum to the upper abdomen causing perihepatic adhesions: the so- called "violin strings" (Fitz-Hugh-Curtis syndrome).

Responsible for significant morbidity in women, accounting for about 30% of all gynaecological admissions in sub-Saharan Africa. It is thought that 3% of women have

Pelvic
Inflammatory Disease (PID) during their lifetime

Risk factors
Age:
- Peak incidence between 15 - 25 years

Sexual activity:
- Multiplicity of sexual partners

Use of intrauterine contraceptive devices:
- Usually within the first 4 months of use

Previous episode(s) of PID

Clinical features
Major criteria (the Westrom triad):
Lower abdominal pain and tenderness
Cervical excitation tenderness
Adnexal tenderness

Minor criteria
Fever (38 degrees C)
Leucocytosis
Purulent vaginal discharge
Adnexal mass

Diagnosis
Based on the presence of the Westrom triad of symptomatology **plus** one of the minor criteria Confirmation by demonstration of causative organism(s) on microscopy, culture and sensitivity testing

Differential diagnoses
Acute appendicitis
Ovarian cyst accident
Endometriosis
Urinary tract infections
Renal disorders (e.g. nephrolithiasis)
Pelvic adhesions
Lower lobe pneumonia
Ectopic gestation

Complications
Pelvic abscess
Septicaemia
Chronic pelvic pain
Ectopic gestation
Infertility
Fitz-Hugh-Curtis syndrome
Recurrence (about 25% rates)

Investigations
Packed cell volume
Haemoglobin genotype
Blood Group
White Blood Cell count
Electrolytes and Urea
Midstream urine microscopy, culture and sensitivity
Endocervical swab
High vaginal swab culture: to exclude trichomoniasis, bacterial vaginosis
Urethral swab

Ultrasound scan: to exclude cyesis, ectopic gestation, adnexal mass (e.g. ovarian mass)
Indications for admission
Uncertain diagnosis
Intolerance of oral medication or non-response to outpatient therapy
Presence of a pelvic mass
Presence of an intrauterine device
Upper abdominal pain
Non-adherence to therapy
Pregnancy
Nulliparity

Treatment objectives
Rehydrate adequately
Eradicate the infecting organism(s)
Prevent complications

Drug treatment
Appropriate antibiotics for an adequate period
- The antibiotic chosen should cover all possible causative organisms while awaiting culture/sensitivity results

Outpatient therapy while awaiting culture results:
Ceftriaxone (or equivalent cephalosporin)
- 1g intramuscularly stat

Plus:
Doxycycline
- 100 mg orally every 12 hours for 14 days

Plus or minus:
Metronidazole
- 400 mg orally every 12 hours for 14 days

If no response in 48 - 72 hours
- Admit, re-evaluate and give appropriate intravenous therapy

Inpatient triple therapy-
Ceftriaxone/doxycycline/metronidazole

Or:
Clindamycin/gentamicin/metronidazole
Triple antibiotic regimen to be continued for 48 hours after the patient improves clinically
Subsequently, the patient should continue therapy with
Doxycycline
- 100 mg orally every 12 hours

Plus:
Metronidazole
- 400 mg orally every 8 hours for 10-14 days

Prevention
Encourage the use of barrier contraceptive with spermicides
Modify risky sexual behaviour: avoid multiplicity of sexual partners
Contact tracing: to break the existing chain of infection and prevent recurrence
Prompt diagnosis and treatment to prevent long-term complications

RAPE

Introduction

Performance of the act of sexual intercourse by force, duress, intimidation or without legal consent (as with a minor)

A growing social disorder afflicting the poor and rich, alike, with devastating and longstanding emotional consequences for the afflicted, family and society at large.

An enormous societal problem that appears to be poorly recognized and grossly under-reported.

An average of one in five adult women may have experienced sexual assault during her lifetime.

Adult women are much more likely to be raped by a spouse, ex-spouse, or acquaintance than by a stranger.

The girl-child is much more likely to be raped by her close male associates (non-strangers), not excluding her father, uncle, brother, cousin, neighbour, school teacher, family driver, security personnel, and even faith-based instructor

Mental illness, alcohol and drug abuse appear to be predisposing factors; neglect and inattentiveness to the needs of the girl-child also contribute

Clinical features

Indirect presentation
Vague symptoms
Physical features:
- Perineal pain
- Bleeding per vaginam
- Bruised face/body
- Arthritis
- Disordered gait

Psychological symptoms/disorders
- Sadness
- Depression
- Refusal to respond to simple questions
- Avoidance of eye contact
- School/work absenteeism

Differential diagnoses
Vaginitis
Threatened abortion
Domestic violence
Alcoholism
Drug abuse
Depression

Investigations
Early
Vaginal/perineal swab for microscopy, culture and sensitivity
Semen: DNA analysis
Late
Urinalysis; urine microscopy, culture and sensitivity

Pregnancy test (blood)
HIV screening

Treatment objectives
Evaluate safety of the patient
Assess and treat physical injuries
Provide emotional support
Assess and deal with the risk of sexually transmitted infections and pregnancy
It is important to document clinical findings

Non-drug measures
Reassure patient
Provide information about legal services

Drug treatment
Treat physical injury (as appropriate)
Treat STIs, UTI (as appropriate)
Treat HIV infection (if detected); Post-exposure prophylaxis if clinical situation so requires
- See section on HIV infection

Manage pregnancy (as appropriate). If seen within 120 hours, give emergency contraceptives (e.g. prostinor2) to prevent unwanted pregnancy.
Rape is an indication for safe termination of pregnancy in some countries. If indeed, it is adjudged to threaten the life of the woman, this could also be a justification for termination of pregnancy in Nigeria

Treat depression (if present)

Prevention

Promote Basic Education for All
Reduce adult illiteracy
Promote family/community moral values
Promote Basic Health Education
Promote safe shelter and neighbourhoods
Enforce existing laws on rape
Legislate for new laws to deter potential rapists and protect females
Promote socio-economic well-being for all

IMMUNIZATION SCHEDULES(See Appendix VII)

CHAPTER 16:

RESPIRATORY DISEASES

ACUTE EPIGLOTTITIS

Introduction

Epiglottitis is inflammation of the epiglottis and adjacent supraglottic structures.

Epiglottitis can progress rapidly to life-threatening airway obstruction if not treated.

The condition is commonest in children.

Pathogens in children include *H. influenza* type B, types A, F, *Streptococci* and *Staph. aureus*

The commonest is *H. influenza*type B.

In adults a wide range of pathogens, including viruses, bacteria, fungi are involved but H. influenza type B appears to be the most common.

In immunocompromised hosts, epiglottitis may be caused by Pseudomonas aeruginosa and Candida.

Non-infectious causes include, thermal injury, corrosive ingestion, foreign body ingestion

Rarely may occur as a result of graft-versus-host disease in transplantation.

Clinical features

Common presentation in children

- Difficulty with breathing
- Stridor
- Hoarse voice
- Pharyngitis
- Fever
- Sore throat
- Tenderness of anterior neck
- Cough
- Difficulty swallowing
- Change in voice

Adult presentation usually less fulminant

- Sore throat
- Fever
- Muffled voice
- Drooling
- Stridor
- Hoarseness
- Difficulty swallowing
- Difficulty breathing

1. Differential Diagnosis

- Laryngotracheitis or spasmodic croup
- Uvilitis
- Bacterial tracheitis
- Peritonsillar or retropharyngeal abscesses
- Foreign body lodged in the larynx
- Angioedema
- Upper airway congenital anomalies

- Diphtheria

Complications
- Airway obstruction
- Epiglottic abscess
- Secondary infection
- Necrotizing epiglottitis (rare, in immunodeficiency)
- Death **Investigations**
- Radiograph (lateral neck x-ray)
- "Thumb sign" appearance of the enlarged epiglottitis
- Ultrasound
- Microbiology

Treatment objectives
- Safeguard airway
- Control infection

Drug treatment
- Amoxicillin/Clavulinic acid 625mg 12hourly for 7 – 10days

OR
- Cefuroxime
- *Adult* : 250mg orally every 12hours for 5 – 10days
- *Child* : 125mg orally every 12hours for 5 – 10days

OR
- Ceftriaxone

Adult : 250mg – 500mg IM/IV for 5 – 10days

Child : neonate, infuse over 60mins, 20 – 50mg/kg daily

Child under 50kg : 20 -50 mg/kg daily by deep im injection or by IV injection over 2 – 4 minutes or by IV infusion ; up to 80mg/kg daily in severe infections.

Supportive measures

- Oxygen
- Steam inhalation
- Nasotracheal intubation may be necessary
- Maintain adequate caloric intake and hydration

Notable adverse drug reactions , caution

Cefuroxime : avoid in pregnancy and in patients with renal impairment.

Ceftriaxone : rashes, fever, GIT disturbances

Dose reduction in the elderly.

Prevention

- Haemophilus influenza vaccine
- Child 2months – 18years : 0.5mls
- Should be part of childhood immunization.

ACUTE BRONCHITIS

Introduction

An inflammation of the bronchial tubes.

Commonly caused by a variety of viruses, same as those that are responsible for common cold. Primary bacterial aetiology may also occur.

Acute bronchitis can last from a few days to 10days but the associated cough may last for several weeks after the infection has cleared up.
Bronchitis lasting up to 90days is still usually classified as acute bronchitis.

Clinical features
- Cough
- Sputum production
- Sputum may be clear, yellow or greenish
- Wheezing
- Muscle and backache
- Low grade fever
- Shortness of breath in severe cases
- Chest pain especially while coughing

Differential diagnosis
- Cough-variant asthma
- Mycoplasma pneumonia
- Chlamydia pneumonia
- Bordetella pertussis

Complications
- Pneumonia
- Acute respiratory failure
- Repeated bouts of acute bronchitis over time may lead to COPD

Relevant investigations and management
- Chest x-ray
- Sputum tests

- (Quality sputum for culture and tests for evidence of allergy)
- Pulmonary function tests

Management

- Antibiotics usually NOT required unless there is clear evidence of primary bacterial aetiology or secondary bacterial infection
- **Drug treatment**

  Empirical antimicrobials e.g.

  Amoxicillin 500mg PO 8 hourly for 5 – 7 days

  Macrolide e.g. erythromycin 500mg 8hourly 5 – 7 days

  Co-trimoxazole 960mg 12hourly 5 – 7 days

- **Adverse events related to drugs**

  Nausea, Skin rashes, rarely Stevens-Johnson syndrome with co-trimoxazole.

ACUTE LARYNGO-TRACHEO-BRONCHITIS (Croup)

Introduction

An infection of the upper and lower respiratory tract affecting children 2 - 3 years of age

Causes significant sub-glottic oedema

Most common aetiology is parainfluenza virus infection preceded by an upper respiratory tract infection

Clinical features
Fever
Hoarseness
'Bovine cough'
Inspiratory stridor

Differential diagnosis
Acute epiglottitis

Complication
Respiratory obstruction

Investigations
Radiograph of the neck (postero-anterior view)

Treatment objectives
Prevent asphyxiation
Treat inflammatory oedema

Supportive measures
Humidification
Hospitalization may be necessary

Drug treatment
Nebulized epinephrine
Child: 400 micrograms/kg (maximum 5 mg)
- Repeat after 30 minutes if necessary

Glucocorticoids
- Dexamethasone

Child 1 month -18 years:10 - 100 micrograms/kg orally daily in 1 - 2 divided doses, adjusted according to response up to 300 micrograms/kg daily especially in emergencies

- Give parenterally in more severe cases
- May repeat dose after 12 hours if necessary

Caution

Effects of nebulized epinephrine last 2 - 3 hours; the child should be monitored carefully for recurrence of the obstruction

ACUTE RHINITIS (common cold, coryza)

Introduction

An acute inflammation of the nasal mucosa with variable degrees of pharyngitis.

Rhinoviruses are the commonest aetiologically important agents followed by the coronaviruses, the parainfluenza, RSV, influenza and adenoviruses in that order. Others include enteroviruses, rubella, varcella and possibly a sizeable group of undiscovered viruses.

Clinical Features

Systemic complaints are often absent or modest if present.

Fever is usual.

Features may include

- Tickling sensation in the nose associated with itching of the nose and palate
- Occasional vertigo due to associated viral labyrinthitis.
- Watery nasal discharge (rhinorrhoea), which may later become purulent
- Sneezing
- Headaches
- Nasal obstruction (usually alternating)

Differential Diagnosis

- Allergic rhinitis
- Bacterial rhinitis (often supervenes after the viral onset)

Complication

- Superimposed bacterial rhinitis
- Suspect this if symptoms last longer than 7-10days
- Sinusitis
- Lower respiratory infection
- Otitis media
- Obstruction of internal auditory meatus: may cause deafness.

Management

Treatment objectives

- Relieve nasal mucosal oedema and obstruction
- Relieve pain/discomfort
- Treat complications

Drug treatment

- Symptoms of rhinorrhea and sneezing can be relieved with non-selective sedating antihistamines such as chlorpheniramine,.
- Selective H_1 receptor inhibitors are not effective.
- Eg. Adult chlorpheniramine 4mg orally every 4-6hrs up to a maximum of 24mg/day, maximum of 12mg/day in elderly.
- For children

  1-2years 1mg b.d

  2-6years 1mg every 4-6hours (max 6mg/day)

  6-12years 2mg every 4-6hours (max 12mg/day)

  - Symptoms of headache, myalgias and occasional fever

Analgesics

- Paracetamol

Adults: 1gm three times daily

Children: 1-5years 120-250mg

6-12years 250-500mg

12-18years 500mg 6hourly (max 4 doses/day)

- Non-steroidal anti-inflammatory drugs (IBUPROFEN) may improve symptoms in adults with rhinovirus infection.
- Symptoms of nasal decongestion

Nasal sprays containing decongestants should not be used for more than 5-7days to avoid rebound rhinitis medicamentosa on

withdrawal
e.g. Ephedrine hydrochloride nasal drops 1-2drops into each nostril up to 3-4 times daily for a maximum duration of 7days. Applicable to adults and children over 12years.

Notable adverse drug reactions
Paracetamol: raised liver enzymes, renal papillary necrosis

Non-steroidal
Anti-inflammatory: Upper G.I bleeding drugs.

BRONCHIAL ASTHMA

Introduction

A chronic inflammatory disease of the airways that is characterized by hyper-responsiveness of the tracheo-bronchial tree to a multiplicity of stimuli
Manifests physiologically by wide-spread airway narrowing and clinically by paroxysmal attacks of dyspnoea, cough and wheezing
Acute episodes are interspersed with symptom-free periods

Clinical features

Episodic dyspnoea
Cough: unproductive, or productive of scanty sputum

Wheezing
Tachypnoea
Tachycardia
Pulsus paradoxus in severe attacks
Mildly raised blood pressure
Rhonchi: inspiratory and expiratory
Prolonged expiration
Silent chest (an ominous sign)

Differential diagnoses
Chronic bronchitis
Left ventricular failure
Glottic dysfunction with respiratory obstruction
Recurrent pulmonary emboli
Eosinophilic pneumonia
Carcinoid tumour

Complications
Spontaneous pneumothorax
Pneumo-mediastinum
Atelectasis

Investigations
Diagnosis is based on:
Airway reversibility to inhaled β - adrenergic agonist
Isocapnoeic response to hyperventilation of cold air
Sputum eosinophilia
Chest radiograph: hyperinflation

Treatment objectives

Arrest and reverse acute episodes

Prevent (or at least reduce) frequencies of asthmatic attacks

Achieve a stable asymptomatic state

Maintain the best pulmonary function possible

Drug treatment

Acute asthma episodes:

Nebulised salbutamol

Adult and child over 18 months: 2.5 mg repeated up to 4 times daily; may be increased to 5 mg if necessary

Child under 18 months: 1.25 - 2.5 mg up to 4 times daily

- More frequent administration may be needed in severe cases

Intravenous aminophylline in patients not previously treated with theophyilline and without contraindications.

Adult: 250 - 500 mg slowly (with close monitoring) over 20 minutes

Child 1 month - 18 years: by intravenous injection 5mg/kg (maximum 500 mg), and then by intravenous infusion

Intravenous steroids

Adequate hydration

Oxygen

Chronic management is based on severity:

Intermittent symptoms

Inhaled salbutamol on as-needed basis

Mild persistent asthma

Inhaled salbutamol

Adult: 100 - 200 micrograms for persistent symptoms up to 4 times daily

Child 1 month - 18 years: 100 - 200 micrograms (1 - 2 puffs) up to 4 times daily (for occasional use only)

Plus:

Inhaled corticosteroid

- Beclomethasone dipropionate 100 microgram 3 - 4 times daily

Moderate persistent asthma

Inhaled salbutamol

Adult: 100 - 200 micrograms for persistent symptoms up to 4 times daily

Child 1 month - 18 years: 100 - 200 micrograms (1 - 2 puffs) up to 4 times daily (for occasional use only)

Plus:

Inhaled corticosteroid

- Beclomethasone dipropionate ()

Adult: 100 microgram 3 - 4 times daily

Child under 2 years: 50 micrograms every 12 hours; 2 -5 years: 100 - 200 micrograms every 12 hours; 5 - 12 years: 100 -200 micrograms every 12 hours; 12 - 18 years: 100 - 400 micrograms every 12 hours

Plus:

Long- acting B agonist

2 - Salmeterol

Adult: 50 micrograms twice daily, up to 100 micrograms

Child 2 - 4 years: 25 micrograms (1 puff) every 12 hours; 4 - 12 years: 50 micrograms (2 puffs) every 12 hours; 12 - 18 years 50 - 100 micrograms (2 - 4 puffs) every 12 hours

Severe persistent asthma

Inhaled salbutamol

Adult and child up over 18 months: nebulizer 2.5 mg repeated up to 4 times daily; may be increased to 5 mg if necessary

Child under 18 months: 1.25 - 2.5 mg up to 4 times daily

- Repeated administration may be required in severe cases

Long- acting B_2 agonist

Adult: 50 micrograms twice daily up to 100 micrograms

Child 2 - 4 years: 25 micrograms (1 puff) every 12 hours; 4 - 12 years: 50 micrograms (2 puffs) every 12 hours; 12 - 18 years 50 - 100 micrograms (2 - 4 puffs) every 12 hours Oral corticosteroid

- Prednisolone

Adult: 40 - 50 mg orally daily for a few days, and then reduce gradually

Child: 1-2mg/kgorallyoncedailyfor3-5days

Supportive measures

Supplemental oxygen Hydration
Education on care and precipitating factors

Notable adverse reactions, caution

In all cases, prescribers/dispensers should consult product literature to confirm the strengths of various aerosol preparations

Aminophylline

- Do not exceed 500 mg in 24 hours because of the risk of cardiac arrhythmias
- Avoid in elderly or in patients with arrhythmias and hyperthyroidism
- Exercise caution in hypertensive patients
- May cause CNS stimulation with insomnia and convulsions

Steroids

- Immuno suppression, metabolic derangements, etc
- Care should be taken in withdrawing steroids

Prevention

Avoid precipitating factors

Appropriate use of medicines

Training of patients in the techniques of the proper use of aerosols/spacer devices is important

CHRONIC OBSTRUCTIVE AIRWAYS DISEASE

Introduction

A pulmonary disorder of adults

characterized by chronic airflow limitation in the small airways Complicates chronic bronchitis and emphysema Obstruction to air flow is only partially reversible with bronchodilator therapy

Two extreme types of COAD are recognized although there is a lot of overlap

Clinical features

Depending on the predominant syndromes, could be described as follows:

Pink puffers

Slowly progressive dyspnoea
Cough with scanty sputum
Aesthenic features
Barrel-shaped chest
Wheeze

These patients mainly have emphysema

Blue bloaters

Prolonged periods of cough and copious sputum production
Dyspnoea
Frequent respiratory infections
Central cyanosis

These patients mainly have chronic bronchitis

Differential diagnoses

Chronic persistent asthma Cystic fibrosis

Complications

Respiratory failure

Recurrent bronchial infections with *Haemophilus influenza* and *Streptococcus pneumoniae*

Cor pulmonale

Left ventricular failure

Pulmonary thromboembolism

Investigations

Chest radiograph: hyperinflation, pulmonary hypertension

Ventricular function tests: FEV_1/FVC ratio

Blood gas analysis

Blood pH

Haematocrit

Sputum microscopy and culture (during symptom exacerbation)

Electrocardiogram

Airways reversibility test

Treatment objectives

Maintain optimal level of oxygenation and ventilation

- Supplemental oxygen, at 24 - 28% or 1 - 2 litres/minute

Treat infections

Reverse airways obstruction

Clear airways secretions

Drug treatment

Long acting β_2 - agonist

See bronchial asthma

Theophylline

- Aminophylline (see bronchial asthma)

Antibiotics (when necessary to control infection)

- Erythromycin

Adult: 250 - 500 mg orally every 6 hours, or 500 mg - 1 g every 12 hours (up to 4 g daily in severe infections)

Co-amoxiclavulanate

Adult: 500/125 mg orally every 12 hours

Supportive measures

Assisted ventilation

Hydration

Pulmonary physiotherapy

Prevention

Avoidance of cigarette smoking

Avoid/remove atmospheric pollutants

BRONCHIECTASIS

Introduction

An abnormal and permanent dilatation of medium sized airway due to damage of their walls. Usually arises from repeated bacterial or viral infections which result in inflammation and destruction of the

structural components of the bronchial tree.
May be focal or diffuse
Bronchiectasis has both congenital or acquired causes.
The most important cause is severe or repeated respiratory infections.
Other causes include:

- Cystic fibrosis
- Other hereditary disorders e.g. ciliary dyskinesia
- Immunodeficiency disorders
- Autoimmune disorders e.g. rheumatoid disease, ulcerative colitis, Sjogren syndrome
- Mechanical factors e.g. chronically enlarged lymph nodes with pressure effect, lung tumour
- Inhaled toxic substances e.g. silica, cola dust, tobacco smoke.

Clinical features

- Persistent or recurrent cough
- Purulent fetid sputum
- Haemoptysis
- Pleuritic chest pain

With or without a history of preceding pneumonic illness.

- Digital clubbing
- Crepitations, rhonchi and wheezes
- Cor pulmunale and right ventricular failure in chronically hypoxic patients

Differential diagnoses
- Pulmonary tuberculosis
- Lung abscess
- Chronic bronchitis
- Bullous emphysema

Complications
- Massive haemoptysis
- Lung abscess
- Mycotic abscess
- Pulmonary amyloidosis
- Ventilatory failure
- Cor pulmunale and right ventricular failure

Investigations
- Chest radiograph: cystic spaces with air-fluid levels
- Bronchography: saccular, cylindrical or varicose bronchial dilatations
- CT scan (of the chest)
- Bronchoscopy: biopsys of endobronchial lesion
- Sputum microscopy, culture, Ziehl Nielson microscopy
- Ventilatory function test: obstructive pattern

Treatment objectives
- Eliminate underlying pathology
- Improve mucus clearance
- Control infection
- Reverse airflow obstruction

Drug treatment

- Empirical antibiotics in acute exacerbations
  - Amoxicillin

Adult: 500mg – 1g orally every 8 hours for 5 – 7 days

Child: 40mg/kg orally in 3 divided doses daily

  - Cotrimoxazole

Adult: 960mg orally every 12 hours for 5 – 7 days

Child: 6 weeks to 5months: 120mg orally; 6months – 5years: 240mg; 6-12years: 480mg

Appropriate antibiotics as soon as culture results are available.

- Bronchodilators
  - Salmeterol

Adult: 2 puffs (50 micrograms) twice daily

  - Can be doubled in severe airway obstruction

Child: same as adult dose (for children > 4years)

  - Salbutamol

Adult: 1 – 2 puffs (100 – 200micrograms) 3 – 4 times daily

Child: usually 100 microgram (1puff) may be increased to 200 microgram with more severe symptoms.

Supportive measures

- Supplemental oxygen
- Postural drainage or suction
- Cessation of cigarette smoking

Notable adverse drug reactions, caution

- Prescribers / dispensers should consult product literature to confirm the strength of various aerosol preparations.
- Salbutamol: palpitations, tremors, nervous tension, muscle cramps, sleep disturbances, tachycardia, peripheral vasodilation, hypotension.

Prevention

- Avoidance of smoking
- Timely and effective treatment of bacterial infections
- Respiratory care during childhood measles

LUNG ABSCESS

Introduction

Defined as necrosis of the lung parenchyma, usually caused by microbial infection, often with an air-fluid level.

May be classified as acute (symptoms ≤ 1month) or chronic (symptoms ≥ 1month).

May also be classified as primary if it occurs in a previously healthy person or in a person prone to aspiration.

Secondary lung abscess commonly occurs in association with bronchogenic carcinoma or

immunodeficiency states e.g. HIV infection.

Lung abscess may be associated with the following

- Pyogenic bacteria
- Tuberculosis
- Fungi
- Parasites
- Pulmonary infarction
- Primary or metastatic malignancies
- Silicosis
- Coal miner's pneumoconiosis

Clinical features

Symptoms are indolent lasting several weeks:

- Cough, with purulent offensive sputum
- Fever, chills
- Night sweats
- Weight loss
- Pleurtic chest pain

Signs:

- Digital clubbing
- Crepitations
- Pleural friction rub

Differential diagnoses

- Localized bronchiectasis
- Pneumonia
- Tuberculosis

Complications

- Cerebral abscess
- Empyema
- Amyloidosis

Investigations
- Sputum: Gram stain and culture
- Bronchoscopy
- Transthoracic aspiration
- Blood culture
- Chest radiograph

Treatment objectives
- Eradicate bacterial cause
- Drain abscess
- Preserve normal lung function

Non-drug treatment
- Hydration
- Pain relief
- Physiotherapy

Drug treatment
- Antibiotics
  - Metronidazole

Adult: 500mg orally every 8hours
Child: neonate, initially 15mg/kg orally then 7.5mg/kg every 12 hours; 1 month – 12 years: 7.5 mg/kg (maximum 400mg) every 8 hours; 12 – 18years: 400mg every 8hours
Plus:
- Amoxicillin

Adult: 500mg orally every 8 hours for 7 –

10days
Child less than 5 years: a quarter adult dose; 5 – 10years: half adult dose
Or
Amoxicillin/clavulanic acid
Adult: 1g/200mg orally every 8hours for 7 – 10days (Definitive antibiotic therapy should be based on culture and sensitivity results)

Prevention

- Good dental care
- Adequate treatment of acute pneumonia
- Preventive with vaccination in person at risk
  - HIV infected patients who are still capable of responding to a vaccine challenge.
  - Patients with recurrent sinopulmonary infection
  - Patients with or acquired hypogammaglobulinaemia

CHEST PAIN

Introduction

A common clinical symptom that may or may not have significant clinical implications

Clinical features (with differential diagnoses)

Sharp, lancinating lateral chest pain, worse with breathing and coughing: pleurisy
Dull aching lateral chest pain: chest wall pain,

pleural effusion

Central chest pain precipitated by a dry harking cough: suggestive of tracheitis or tracheobronchitis

Central chest discomfort/pain with sensation of heaviness or chest compression: suggestive of myocardial ischaemia

Lateral burning chest pain associated with tenderness on physical contact: Bornholm's disease

Investigations

Chest radiography
Electrocardiography
Echocardiography

Treatment objectives

Treat primary cause Relieve pain

Drug treatment

Non narcotic analgesics

- Paracetamol

Adult: 1 gorallyevery8hours

Child 1 - 3 months: 30 - 60 mg every 8 hours; 3 - 12 months: up to 120 mg every 4 - 6 hours; 1 - 5 years: 120 -250 mg every 4 - 6 hours; 6 - 12 years: 250 - 500 mg every 4 - 6 hours; 12 - 18 years: 500 mg every 4 - 6 hours Non-steroidal analgesics

- Diclofenac sodium

Adult: 25 - 50 mg orally three times (daily

depending on severity)

Child 6 months -18years: 0.3 1 mg/kg by mouth or by rectum 3 times daily (maximum total dose 150 mg daily) Pain of more serious aetiology e.g. pain of lower or upper respiratory tract infection, or pain of myocardial ischaemia

- Refer to an appropriate specialist

COUGH

Introduction

The explosive expiration that clears the tracheo-bronchial tree of secretions and foreign particles or noxious gaseous materials A defensive reflex reaction

Comes to medical attention only when it becomes troublesome, affects life style and/or when there is concern about its cause

Clinical features

Cough may be:

Acute or chronic

Seasonal

Associated with breathlessness and or wheezing Productive of sputum: note colour, smell; haemoptysis Associated with fever

Associated with chest pain: note location and character of pain

Associated with risk factors, e.g. cigarette smoking Associated with the use of drugs for

other illnesses Associated with other constitutional symptoms

Differential diagnoses

Triggers of cough may rise from the upper or lower airways, or lung parenchyma Upper airways:

- Inhaled irritants: dust, fumes, smoke
- Upper airways secretion
- Gastric reflux Lower airways:
- Inflammation
- Viral bronchitis
- Bronchiectaesis
- Bacterial infection
- Bronchial asthma
- Endobronchial tuberculosis
- Bronchial infiltration/compression Parenchymal lung disease
- Pneumonia
- Lung abscess
- Interstitial or endobronchial oedema due to heart disease

Drugs:

- ACE inhibitors

Investigations

Macroscopic and microscopic examination of sputum Sputum culture

Exclude tuberculosis if cough is chronic

Sputum cytology for malignant cells

Chest radiograph where indicated

HIV screen if history and clinical features are

suggestive

Treatment objectives

Identify and treat the underlying cause(s)

Abolish cough

Non-drug measures

Adequate rehydration to prevent inspissation

Encourage expectoration for productive cough

Do not use antitussives unless cough is dry, unproductive and distressing

Drug treatment

Cough suppressants: for dry, unproductive cough

- Codeine cough linctus *Adult:* 5-10mL3-4timesdaily
- Not recommended in children Appropriate antibiotics for bacterial infections

Notable adverse drug reactions, caution

Codeine cough linctus: sedation, constipation

DYSPNOEA

Introduction

An abnormal and uncomfortable awareness of breathing

Effort of breathing is out of proportion with exertion needs

Patients often have difficulties in describing

the discomfort of dyspnoea

Clinical features

Will depend on the underlying cause(s) of dyspnoea

Differential diagnoses

Pulmonary:

- Obstructive airways disease: asthma, chronic bronchitis, emphysema
- Parenchymal lung disease: pneumonia, pneumoconiosis, pulmonary fibrosis
- Pulmonary vascular obstruction: pulmonary emboli
- Chest wall disorders: respiratory muscle paralysis, kyphoscoliosis

Cardiogenic:

- Congestive cardiac failure
- Left ventricular failure Metabolic:
- Diabetic ketoacidosis

Neurogenic:

- Anxiety neurosis

Treatment objectives

Treat cause(s) of dyspnoea

Restore normal respiration

Non-drug treatment

Oxygen in appropriate concentration

Other treatment will depend on theunderlying/ precipitating cause

NON-OBSTRUCTIVE (SIMPLE) CHRONIC BRONCHITIS (NCB)

Introduction

- Chronic bronchitis denotes chronic or recurrent bronchial mucus hyper secretion resulting in Chronic expectoration of sputum.
- For clinical or epidemiological purposes, the term is applied to patients who have coughed up sputum on most days during at least three consecutive months in two successive years.

Non-obstructive Chronic bronchitis:

- In this condition there is Chronic or recurrent mucoid hyper secretion sufficient to cause expectoration but there is no air flow obstruction.
- Between 10 -25% of adult population are affected by NCB.
- It tends to be common in men.
- It is not well understood why some of these persons progress to chronic obstructive airway disease and some do not.
- NCB has a generally good prognosis.
- With smoking cessation and vigorous treatment early in the disease process the disease may be reversed.

The exact cause of the illness is not known

- More common in urban or industrial areas.

- Some inhaled irritants play a role in persistence and aggravation of symptoms and pathology. These include, inhaled tobacco smoke, air pollutant, dusts, powder and noxious fumes.
- Viral or bacterial infection many precipitate or aggravate disease.
- Although history of heavy smoking is common, disease may be observed in non smokers.
- Pathologically, there is hypertrophy and hyperplasia of mucus secreting glands relative to wall thickness.
- There are diffuse inflammatory changes of bronchial epithelium with ulceration, neutrophil infiltration, loss of cilia, bacterial invasion and area of squamous metaplasia. These changes interfere with muco-ciliary function.

Clinical features:

History

- Most striking features are impressive history of cough with sputum production for many years.
- Initially cough present during cold seasons, especially in the morning.
- Over the years cough increases in frequency, severity and duration until cough is present all year round.
- Sputum is usually scanty, mucoid and more in the mornings and occasionally blood stained.

Physical signs

- Patient may be overweight
- Patient may not be in respiratory distress and respiratory rate may be normal.
- Palpation of chest may reveal local tenderness over recently fractured rib.
- Percussion note resonant over the lungs.
- Liver dullness and cardiac dullness normally preserved.
- Breath sound is vesicular
- Positive signs are almost all referable to bronchial secretions.
- Transient basal rales may be noted on inspiration. May clear completely with cough.
- Finger clubbing is not commonly observed in pure chronic bronchitis.

Differential diagnosis

- Asthma
- Bronchiectasis
- Pulmonary TB
- Bronchogenic Ca

Complications

- Muco-purulent relapses due to secondary bacterial infection.
- Progression to chronic obstructive airway disease.

Relevant investigations in non-obstructive(simple) chronic bronchitis

- Spirometry may reveal no abnormality in lung function, since there is no airflow obstruction.
- Chest Xray does not show any characteristic abnormality in simple chronic bronchitis.
- Bronchography may reveal irregular narrowed or distorted bronchi. There is however, no need for routine bronchography in chronic bronchitis.
- Sputum examination; In early stages, sputum may be mucoid. Sputum M/C/S may be necessary to detect bacterial infection.
- Arterial blood gas studies may be unnecessary in straightforward uncomplicated NCB.

Management

a. **Reduction of bronchial irritation**
   - Smoking cessation
   - Avoidance of dusty and smoke laden environment.

b. **Treatment of respiratory infections**
   - Purulent sputum should be treated with amoxicillin 500mg 8hourly for seven days.
   - In the absence of response a sputum culture and sensitivity is done and antibiotics changed to sensitive antibiotics.

c. **Mucolytics**- Mucolytic expectorant appear to improve quality of life and

decreases cough. Iodinated glyceryl at a dose of 60mg four times daily for 1 to 8 weeks can be used.

d. **Bronchodilators and steriods** may not be necessary in simple chronic bronchitis since there is no airway obstruction.

e. **Physiotherapy-** postural drainage may be of value in patients with increased sputum production.

Adverse drug reaction

Maculopapular reactions may occur in patients taking amoxicillin.

PNEUMONIA

Introduction

An inflammation of the lung parenchyma

Various bacterial species, fungi and viruses may cause pneumonia

The setting in which infection is acquired could be a predictor of the infecting pathogen

Bacterial Pneumonia: is defined as bacterial infection of the lung parenchyma associated with recently developed radiological shadowing which may be segmental, lobar or multi lobar.

Types

- Community Acquired Pneumonia (CAP)
- Hospital Acquired pneumonia (HAP)

- Ventilator Associated pneumonia (VAP)
- Health care associated Pneumonia(HCAP)
- Pneumonia in the immunocompromised
- Aspiration pneumonia

Streptococcus pneumoniae is the most common pathogen in community-acquired pneumonia

Common bacteria causing CAP:

- Streptococcus pneumonia
- Mycoplasma pneumonia
- Legionella pneumophilia
- Chlamydia pneumonia
- Haemophilus influenza
- Staphylococcus aureus
- Chlamydia psittaci
- Coxiella burnetti
- Klebsiella pneumonia
- Actinomyces israelli

Other causative organisms:

Haemophilus influenzae

Mycoplasma pneumoniae

Pseudomonas aeruginosa (usually implicated in nosocomial pneumonia)

Clinical features

Typical pneumonia:

Sudden onset fever, chills and rigors

Cough with purulent sputum production

Pleuritic chest pain

Breathlessness with short inspiratory efforts

Signs:
Fever
Herpes labialis
Tachypnoea
Signs of lung consolidation
Pleural friction rubs
Chest signs are very helpful depending on the phase of the inflammatory response
When consolidated

- Dull percusision
- Increased Tactile and vocal fremitus
- Bronchial breath sounds
- Whispering pectoriloquy
- Crepitations

Signs of severity

- Confusion
- Urea > 7mmol/L
- Respiratory rate > 30/min
- Systolic BP < 90
- Age 65years

Score 1 point for any of the above features present
0 or 1- home treatment
2 - Hospital-supervised treatment
3 or more- manage in Hospital as severe pneumonia
4 or 5 – ICU Admission
Atypical pneumonia:
Gradual onset

Dry cough
Prominent extra-pulmonary symptoms
Headache
Sore throat
Fatigue
Myalgia
Chest crackles or rales

Differential diagnosis
- Acute bronchitis
- COPD Exacerbation
- Pulmonary embolism/infarction
- TB
- Pulmonary eosinophilia

Complications
- Empyema Thoracis
- Pleural effusion
- Lung abscess
- Lobar collapse
- Deep vein thrombosis and pulmonary embolism
- Pneumothorax
- ARDS
- Multi organ failure
- Hepatitis, pericarditis, myocarditis, meningoencephalitis
- Pyrexia from drug hypersensitivity

Relevant Investigations
- FBC +ESR+ CRP
- Serum E/U/Cr

- LFT
- Blood Culture
- Serology
- Cold agglutinins
- Arterial blood gases/ SPO_2
- Sputum gram stain, M/C/S
- Urine pneumococcal and legionella antigen
- Chest X-ray
- Pleural fluid M/C/S

Treatment objectives

Eliminate the infection

Return to normal lung function

Treatment

GENERAL

- Oxygen to maintain $Pa0_2$ at or above 8kPa
- IV fluids especially in severe cases
- Anti pyretics
- Antibiotics
  - Uncomplicated CAP + No modifying factor, no antibiotics use in the last 3months:

Co-amoxiclavulanate

Adult : 1g 12hourly for 5 – 7 days

Child : Neonate and premature infants, 25mg/kg 12 hourly; Infants up to 3 months, 25mg/kg 8 hourly; 3months – 12years, 25mg/kg 8hourly increased to 6hourly in more severe infections.

OR

Benzyl penicillin

Adult : initially 2million units 6 hourly.

Child : preterm and neonate under 7 days, 25mg/kg by IM injection

Or by slow IV injection or infusion every 12 hours; double dose in severe infections.

Neonate 7 – 28 days: 25mg/kg 8hourly; double dose in severe infections.

1 month – 18years : 25mg/kg 4 – 6hourly. Double dose in severe infections.

Commence oral therapy as soon as possible.

OR

Macrolide (azithromycin 500mg stat ,then 250mg daily, or Clarithromycin 500mg twice daily for up to 14days)

OR

Cefuroxime axetil

Adult: 500mg orally 8 hourly for 5 – 7 days

Child: 3 months – 2 years 10mg/kg(maximum 125mg) orally 12 hourly

2 – 12 years 15mg/kg orally 12 hourly

12 – 18 years 12 hourly. May double doses in severe infections.

Patients with history of recent use of Antibiotics

- Respiratory quinolone (levofloxacin).
  - Quinolones are generally better avoided in TB endemic areas because of

their potential use as part of 2nd line regimen in the treatment of MDR-TB.

- Advanced macrolide+ amoxycillin
- Advanced macrolide+amoxycillin+clavulanic acid
- Complicated CAP
- IV β lactam + advanced macrolide
- Iv respiratory quinolones+advanced macrolide
- Penicillin G+advanced macrolide

Adverse reactions

Co-amoxiclav: nausea, diarrhoea, skin rashes, contraindicated in penicillin hypersensitive individuals.

Cefuroxime: nausea, vomiting, abdominal discomfort, headaches, rarely antibiotic associated colitis.

Macrolides: similar to those mentioned above but usually milder. Hepatoxicity and antibiotic associated colitis are quite rare.

Prevention

Pneumococcal vaccine

Haemophilus influenzae vaccine

PULMONARY TUBERCULOSIS

Introduction

Tuberculosis (TB) is one of the oldest diseases known to affect humans.

It is caused by bacteria of mycotuberculosis complex which includes *M. tuberculosis, M.*

bovis, and *M. africanum*.
M. tuberculosis is the most common cause of tuberclosis worldwide.

Transmission is by droplet infection.
Nigeria ranks 11th among the high burden countries of TB in the world.
Federal government of Nigeria established the National Tuberculosis and Leprosy Control Program (NTBLCS) in 1993 with the objective to reduce the prevalence of TB and Leprosy to a level at which they no longer constitute public health problem in the country.

Almost every organ can be affected. The lung parenchyma are affected in more than 80% of the cases. This can be either as primary pulmonary disease (occurs mainly in childhood) or post primary pulmonary disease.
Those at risk of acquiring tuberculosis are:
- Contacts of patients with smear positive pulmonary disease
- Immunocompromised individuals, health works and people living in overcrowded conditions.

Clinical Features
Common symptoms of pulmonary tuberculosis:-

- Persistent cough
- Weight loss
- Drenching night sweats
- Chest pain (dull or pleuritic)
- Haemoptysis
- Anorexia

Signs:-

Physical examination may be normal

- Crepitations usually in the upper zone (earliest physical sign)
- Physical signs of consolidation, cavitation and fibrosis develop later

Other signs:- palor and finger clubbing, Erythema nodusum and phlynctenular conjuctivitis (Primary PTB)

Differential Diagnosis

- Pneumonia
- Carcinoma of the bronchus
- Lung abscess (especially due to *Klebsiella pneumoniae*)
- Pulmonary infarction

Complications

- Lung collapse
- Bronchiectasis
- Chronic Obstructive Pulmonary Disease
- Pleural effusion
- Corpulmonale
- Destructive lung syndrome
- Lung abscess

- Acute Respiratory Distress Syndrome
- Spontaneous Pneumothorax
- Haemorrhage/Mycetoma

Extrapulmonary TB:

Lymph node TB:

- Painless swelling of lymph nodes (usually cervical and supracervical sites
- Usually discrete in early disease; may become inflamed and have a fistulous tract draining caseous material)

Pleural TB:

- Fever
- Pleuritic chest pain
- Dyspnoea
- Dullness to percussion
- Absence of breath sounds

TB of the upper airways:

Nearly always a complication of advanced cavitatory pulmonary TB

May involve the laynx, pharynx and epiglottis:

- Hoarseness
- Dysphagia
- Dysphonia

Chronic productive cough

Genitourinary TB:

Urinary frequency Dysuria Haematuria Flank pain

Skeletal TB:

Weight bearing joints are affected: spine, hips

and knees

Spinal TB (Pott's disease):

Paraparesis Paraplegia

TB meningitis:

- Headache
- Mental changes
- Confusion
- Lethargy
- Altered sensorium
- Neck rigidity
- Ocular nerve paresis
- Hydrocephalus

Gastrointestinal TB:

- Commonly affects the terminal ileum and caecum
- Abdominal pain (may be similar to that of appendicitis)
- Diarrhoea
- Intestinal obstruction
- Haematochezia
- Palpable mass Fever
- Weight loss
- Night sweats
- TB peritonitis

Pericardial TB:

- Fever
- Dull retrosternal pain
- Friction rub
- Cardiac tamponade

Military TB:

- Fever
- Night sweats

- Anorexia
- Weakness
- Weight loss
- Cough
- Hepatomegaly
- Splenomegaly
- Lymphadenopathy
- Choroidal tubercles (pathognomonic)
- Meningitis

There are no clinical findings specific for a diagnosis of pulmonary TB; a history of contact with a smear positive pulmonary TB case, respiratory symptoms for more than 2-3 weeks not responding to broad spectrum antibiotics, and weight loss, failure to thrive may suggest TB

Relevant Investigations

Bacteriologic examination:-

- Sputum AFB X 3 (spot, morning, spot)
- Sputum mycobacterial culture and drug susceptibility testing/Gene xpert-Xpert MTB-RIF Assay

Radiologic Examination

- Chest X-ray

Other tests:-

- Tuberculin skin test (low sensitivity and low specificity)
- Hematologic - Full blood count and ESR

In cases of diagnostic difficulties one may need High Resolution CT and fibre-optic

brouchoscopy with bronchoalveolar levage and trans-bronchial biopsy

Treatment

Treatment objectives

Cure the disease

Prevent death from active TB or its late effects

Prevent relapse of TB

Decrease transmission of TB

Prevent the development of acquired drug resistance

NTBLCP recommends that all TB cases and TB suspects should be managed in DOTs centres. This programme provides good quality drugs free of charge to all patients. It also implements international standards for tuberculosis care.

Chemotherapy

Standard drug regimens for Adults

A. 6 months regimen

- **Initial Intensive Phase:-** This is for 2 months using Rifampicin, Isoniazid, Pyrazinamide and Ethambutol.

Continuation Phase:- 4 months using Isoniazid and Rifampicin. In this regimen, there is fully supervised administration of drugs in both intensive and continuation phases.

Daily doses and adverse reactions of commonly used anti-tuberculosis drugs (Adults)

Drugs	Daily dose	Adverse drug reactions
Rifampizin taken 30 mins before breakfast	10 mg/kg (maximum dose) 600mg	Drug interactions, hypersensitivity hepatitis fever
Isoniazid	5-10mg/kg (maximum dose) 300mg	Hypersensitivity, polyneuropathy.
Ethambutol	15-20mg/kg (maximum dose) 1.6g	Optic neuritis hypersensitivity
Pyrazinamide	20-35mg/kg (maximum dose) 2.5g	Hepatitis, gout, hypersensitivity

- Pyridoxine 10mg is usually added to prevent peripheral neuropathy
- These drugs can be provided as fixed dose combinations to enhance adherence to therapy or loose tablets.

Other aspects of management include:-

- Contact tracing
- Health education of the patient

Standard drug regimens for Children

Two standardized treatment Regimen

adopted for the treatment of all children diagnosed with susceptible TB in Nigeria:

1. Standard six month treatment Regimen for all children with newly diagnosed or previously treated PTB disease.
   a. Regimen 1 for Children: **2(RHZ+E)/4(RH)**
2. Standard six month treatment Regimen for all children with newly diagnosed or previously treated EPTB disease.
   a. Regimen 2 for children with EPTB: **2(RHZ+E)/10(RH)**

Tb Paediatric drugs and dosages

Drug	Dosage(mg/kg)	Range(mg/kg)	Maximum dose (mg/day)
Isoniazide (H)	10	7-15	300
Pifampicin (R)	15	10-20	600
Pyrazinamide (Z)	35	30-40	2000
Ethambutol (E)	20	15-25	1200

Use of Pyridoxine (Vitamin B6) in children

Pyridoxine (vitamin B6) protects against isoniazid-induced peripheral neuropathy

Not routinely given but is recommended for severely malnourished and HIV-infected children

The recommended dose is 25 mg/ day until treatment is completed

Monitoring of PTB Treatment

Monitoring for response to therapy (clinical improvement and bacteriologic clearance in sputum) and adverse drug reactions.

Routine laboratory monitoring for drug toxicity may not be necessary when there are no symptoms, signs or co-morbid factors like hepatic and renal disease.

MULTIDRUG RESISTANCE TUBERCULOSIS (MDR-TB)

Drug resistance in mycobacteria comes about through random spontaneous mutation.

Emergence of this is creating additional barriers to effective tuberculosis control.

MDR-TB is caused by an organism resistance to at least isoniazid and rifampicin.

XDR-TB is MDR-TB plus resistance to any of the floroquinolones and one of the second line injectables.

MDR-TB SUSPECTS

- Failure of treatment with first line anti-tuberculosis drugs
- Symptomatic contacts to a known MDR-

TB case

Diagnosis

Send sputum samples to specialized facility for culture, molecular line probe and drug susceptibility

Treatment – refer to designated treatment centres

HIV ASSOCIATED PULMONARY TUBERCULOSIS

Tuberculosis is an important opportunistic among HIV infected persons and commonest cause of death in such patients.

It directly attacks the critical immune mechanism involved in protection against Tuberculosis.

Its presentation depends on the stage of HIV infection.

Diagnosis of tuberculosis in HIV patients may be difficult when the immunity is highly compromised (low CD4 count) because of atypical presentation, increase frequency of sputum smear negativity and atypical radiographic features.

All TB patients should be offered HIV counselling and testing; also all HIV patients should be screened for TB.

Treatment:

Commence anti-tuberculosis treatment.

Offer Co-trimoxazole preventive therapy
Commence anti-retroviral therapy
First line Anti-tuberculosis drugs though very effective but the problems in the treatment of such patients are:

- Pill burden
- Drug interaction

Potentials for drug interactions with rifampicin and antiretroviral agents such as protease inhibitors (PIs) and non nucleoside reverse transcriptase inhibitor (NNRTIs)
Rifabutin may be used in place of rifampicin but not easily available and expensive.
Efavirenz in place of nevirapine

- Immune reconstitution syndrome
- Increased incidence of drug resistance cases

CHAPTER 17:

INJURIES AND ACUTE TRAUMA

Dog bites

Responsible for 80% of bite wounds

Bacteriology usually mixed

- Alpha haemolytic streptococci, pasteurella species, staphylococci, Eikenella *chorrodeus, actinomyces,* fusobacterium, prevotella, pophyomonas species,

Capnocytophaga canimorsus

15 - 20 % of wounds become infected

Lower limbs are most commonly affected

Infections occur 8 - 24 hours after bite and may manifest as:

- Pain
- Fever
- Lymphadenopathy
- Cellulitis

If the canine tooth penetrates synovium or bone:

- Septic arthritis
- Osteomyelitis

Cat bites

Less common
More than 50% result in infection
Females are more affected than males
The hands and arms are more commonly affected
Usual organisms include *P. mutocida* and those ones following dog bites

Rats, mice, gerbils and animals that prey on them
May transmit *Streptobacillus moniliformis* or *Spirillus minor*
Usually affect hunters or laboratory handlers of rats
Manifests as:
Fever
Chills
Myalgias
Headaches
Severe migratory arthralgia
A maculopapular rash involving the palms and soles

Human bites
May be:
Self-inflicted
Sustained by medical personnel caring for patients
Sustained during fights, rapes or during sexual activity
May become infected more than bites from

other animals
The oral microflora include multiple species of aerobic and anaerobic bacteria
Those of hospitalized and debilitated patients often include *Enterobacteriacae*
HIV, HBV have been reported due to human bites

Sharks and crocodiles
Cause death by:
Tissue destruction
Crush syndrome
Haemorrhage
Infection

Marine invertebrates
Have specialized organelles called nematocysts for poisoning and capturing prey
May cause serious ill health and death

Initial assessment
Careful history
Contact local authorities to determine if the specie is rabid; if possible locate animal for observation
Antibiotic allergy, immunization of patient and other morbid condition(s) should be documented
Inspect wound for evidence of infection.
Conduct general physical examination,

including vital signs

Investigations

Depend on the type of injury, the clinical presentation and the onset/type of complications:

Full Blood Count
Electrolytes and Urea
Blood clotting profile
Arterial blood gas estimations
Chest radiographs
Wound and blood cultures

Treatment objectives

Neutralize envenomation
Limit systemic effects
Local wound care
Prevent onset of complications
Prevent specific infections such as rabies in high risk cases

Non-drug measures

Limb splinting (and rest the limb)
Use of venom detection kit (if available)
Application of pressure bandage
Control/care of the airway *Incision is discouraged; the mouth should not be used to suction*
Wound debridement and fasciotomy for compartment syndrome may become necessary

Drug treatment

Administration of high flow oxygen

Intravenous fluid administration to maintain circulation: use colloids or cystalloids as clinically appropriate

Treatment of anaphylaxis with antihistamines (H blockers), epinephrine (adrenaline) and corticosteroids

Analgesia

Prophylactic antibiotics as appropriate

Tetanus prophylaxis

For animal bites in which rabies is considered a significant risk it is imperative that anti-rabies prophylaxis be instituted

- If the patient is not previously vaccinated local wound cleansing should be done, rabies immune globulin administered and the vaccine given

Antirabies prophylaxis

Rabies immune globulin

Adult and child: 20 units/kg body weight by infiltration in and around the cleansed wound; if whole volume not exhausted, give remainder by intramuscular injection into anterior-lateral thigh (distant from vaccine site)

- Half of the dose is infiltrated around the wound and the rest given intramuscularly into the gluteal muscles

Human Diploid Cell Vaccine (HDCV) or

Rabies Vaccine Adsorbed (RVA)

- 1 mL is given into the deltoid on days 0, 3, 7, 14, and 28
- Should not be administered in the gluteal area

If the patient has previously been vaccinated clean the wound and give the vaccine given on days 0 and 3 only ***Adrenaline (epinephrine), hydrocortisone must be immediately on hand for the treatment of anaphylaxis if it occurs Prevention***

Appropriate clothing and footwear while outdoors Attention and care to observe general safety measures

BURNS

Introduction

A common form of trauma in our environment

Involves coagulative necrosis of tissue cells following varied insults

- Flames
- Chemicals
- Electricity
- Friction
- Cold or hot fluids

The various types occur with varying frequencies in various segments of the population

- For example scalds occur with great frequency in children while flame burns

occur commonly in young adults

Clinical features(and complications)

Extensive skin loss with dehydration

Airway burns leading to dyspnoea, tachypnoea, stridor, hypoxia, hypercarbia, airway obstruction and death

Breathing difficulties from circumferential chest burns

Acute respiratory distress syndrome, acute lung injury and pulmonary oedema

Massive fluid losses from evaporation and interstitial fluid shifts leading to hypovolaemic shock

Acute renal failure from pre renal failure, acute tubular necrosis, and the crush syndrome

Electrolyte abnormalities: hyper or hypokalaemia with cardiac dysrhythmias and/or arrest

Anaemia from destruction of red cells.

Also nutritional anaemia

Hypothermia

Immune dysfunction

Burns wound sepsis and septicaemia

Tetanus

Acute gastric dilatation

Stress ulcerations in the gastrointestinal system

Limb compartment syndrome

Crush syndrome

Deep vein thrombosis
Systemic Inflammatory Response Syndrome (SIRS)
Multiple Organ Dysfunction Syndrome (MODS)

Investigations

Full Blood Count
Electrolytes and Urea Grouping and cross-matching
Arterial blood gases
Chest radiograph
Electrocardiogram
Wound swab for microscopy, culture and sensitivity
Blood culture
Intracompartmental pressure monitoring

Treatment objectives

At the scene: to stop the burning process or remove victim from the burn situation
Transfer the patient to hospital as soon as possible In the hospital identify life threatening injuries and treat
Perform a detailed survey
Restore patient's physiology as much as possible
Promote wound healing
Prevent complications
Rehabilitation

Treatment

Copiously irrigate the wound with cold water (not ice cold) for 10 - 15 minutes

Avoid hypothermia and the use of agents such as raw eggs and palm oil

- They are not useful and may promote wound sepsis In hospital perform a quick primary survey

Check:

- Airway
- Breathing
- Circulation
- Disability
- Exposure

Correct problems identified Give patient 100% oxygen

Pass an endotracheal tube if there is risk of airway obstruction

Obtain specimens for investigations as detailed above

Determine percentage total body surface area (TBSA) burned

- Wallace rule of nines is recommended in adults
- In children there are several charts e. g Lund and Browder charts

Calculate the total fluid requirement in the first 24 hours using appropriate formulae

- We recommend the Parkland's formula

Determine burn depth

Apply burns dressing

Pass all relevant tubes and gadgets

- Nasogastric tube, urethral catheter, etc

Perform a detailed secondary survey (especially if combined with other trauma)

- Obtain the **AMPLE** history

Allergies, **M**edications,

Past medical history, pregnancy, **L**ast meal

Environment (including details of the incident)

Administer tetanus prophylaxis depending on immune status

Apply relevant splintage

Commence prophylaxis against deep venous thrombosis

Physiotherapy

Decide whether patient should go to a burns unit or burns centre following standard criteria

Drug treatment

Oxygen

Tetanus toxoid

Anti tetanus serum, antitetanus globulin as appropriate

Narcotic analgesics e. g. morphine, pethidine, tramadol

Nonsteroidal anti inflammatory analgesics e. g. diclofenac

H_2 receptor antagonists e.g. ranitidine

Prophylactic antibiotics e.g. cephalosporins

Topical wound dressing agents e. g with zinc

oxide based creams, antibiotic-containing dressings

Prevention

Health education to promote healthy life style and avoidance of risky behaviour

Installation of fire warning systems such as smoke detectors in buildings

Control of petroleum products

An efficient fire service

Fire protocols in all establishments

DISASTER PLAN

Introduction

A disaster is an event which causes serious disruption to community life, threatens or causes death or injury in that community, and/or damage to property

It is beyond the day-to-day capacity of the prescribed statutory authorities and requires special resources other than those normally available to those authorities

Could arise from natural causes cyclones, earthquakes and tsunamis or from man-made situations such as plane crashes and wars

Occur with little or no warning - Only well-prepared systems will be able to limit the damages and losses that follow disasters

The effectiveness and quality of response to a disaster is highly dependent on the level of preparation

An ill-prepared system will lead to an ineffective and uncoordinated response

Apart from an effective response, other advantages of preparation include cost savings and an improved and alert system

There are four phases of disaster management:

- Prevention
- Preparation
- Response
- Recovery

Prevention

Essentially the evolution and implementation of strategies to prevent or mitigate the impact of disasters if/when they arise e.g. designing tsunami warning systems or fire alarm systems

Preparation

Involves system upgrade, overhaul, protocol design, implementation and quality assessment for disaster management

Response

Involves the interaction of the various emergency response agencies to the disaster to save as many casualties as possible; quick transfer to hospitals, coordination of the hospitals and creation of temporary shelters

Recovery

A phase that involves:

- Rebuilding,
- Reconstruction
- Rehabilitation,

with a goal to restoring the community to its pre-event state or as close to it as possible

For a disaster plan to be effective it needs to involve all the stake holders in its design

Disaster plan is necessary at various levels of health care and political terrain:

- National,
- Regional,
- State and local government levels

There should be disaster plans within organizations such as the hospitals, fire service, Army, Air force and Navy; the Ministries of health, the police and the Emergency Medical Service (EMS)

There is need for a coordinating agency such as the National Emergency Management Agency (NEMA) to supervise, monitor and coordinate inter-agency procedures, protocols, joint training sessions and drills

Personnel in all the relevant response agencies must be familiar with the policies, protocols and procedures to be implemented following a disaster

Training and retraining is essential

The hospital disaster plan

There should be a Disaster Committee in the hospital which should:

Design a disaster plan for the hospital

Put in place procedures and protocols to be implemented in a disaster situation

Supervise staff training for disaster management

Be engaged in capacity building

Promote staff awareness regarding disaster prevention and preparation

Promote inter-departmental interaction regarding disaster management

Determine staff competency levels in disaster management

Allocate staff roles in disaster management

Ensure regular drills, seminars, tabletop exercises, computer simulations and interactions on disasters

Ensure stockpile of drugs and equipment to be mobilized in disaster situation

Ensure quality assurance and audit

Promote inter-hospital and inter-agency interaction within the municipality with regard to disaster management

Ensure management commitment to disaster management

Committee composition

The committee should be composed of the following:

The Hospital Trauma Director

The Emergency Department Chief
The Head of Surgery
The Head of Anaesthesia
The Chief of Nursing services
The Head of Security
The Head of Stores
The Head of Pharmacy
A representative of the Hospital Manager

The disaster protocol in the hospital should address the following principal issues:

Who activates the disaster protocol?
What are the criteria for activation?
Information relay to critical departments: laboratories, blood bank, theatres, ICU, radiology, anaesthesia, Emergency Department (ED) Management, Hospital Management, Portage and Security
Pattern of staff call up to the Emergency Department in a disaster situation
Method of staff call
Pre-determined plan for Emergency Department evacuation
Information centre constitution for distressed relatives
Departmental disaster procedures
Logistic issues in a disaster situation
"Standing down" criteria and procedure

HEAD INJURY

Introduction

The term refers to any injury to the head

- Includes bruises and lacerations to the scalp

For practical purposes it is preferable to talk of:

Traumatic brain injury (TBI)

Craniocerebral injury

Craniofaciocerebral injury

This section will focus on TBI

- TBI is common in trauma patients
- Present in up to 50% of multiply injured patients
- Isolated TBI is uncommon

In up to 50% of cases of severe TBI there is multisystem trauma

Classification

Can be considered from the point of view of:

Mechanism of injury

Severity of injury

Morphology Mechanism:

Blunt or penetrating Severity:

- Depends on the patient's position on the Glasgow

Coma Scale (GCS).

- 13 - 15: mild
- 9-12: moderate
- 8 or less: severe

Morphology:

Skull fractures

Intracranial lesions

- Skull fractures could involve the vault or

base of the skull

- Vault fractures may be linear, stellate, depressed or non-depressed; open or closed
- Basilar fractures may be with or without CSF leaks and also with or without facial nerve palsy
- Intracranial lesions may be focal or diffuse.
- Focal lesions include epidural, subdural and intracerebral haematomas
- Diffuse lesions include concussions and diffuse axonal injury (DAI)

Pathophysiology

The brain is covered by the meninges: dura, arachnoid and pia mater with the subdural and the subarachnoid spaces

CSF is produced in the lateral ventricles

- The normal circulating volume of CSF is 140 mL The brain normally regulates its blood flow by a process of autoregulation, which is for the most time undisturbed in TBI

Normal CBF is 800 mL/min or 20% of total cardiac output

- CBF = CPP/CVR = 50 mL/100 g of brain tissue/min
- CPP is the Cerebral Perfusion Pressure
- CVR is Cerebral Vascular Resistance
- CPP = MAP - ICP
- MAP is Mean Arterial Pressure

- ICP is Intracranial Pressure

The normal ICP is 10 mmHg (136 mm H_2O)

- Changes in intracranial volume result in compensation, with alterations in CSF volume and blood volume within the cranium but with minimal change in intracranial pressure

At some point minimal changes in volume result in geometric increases in ICP (The Monro-Kellie doctrine), and decompensation occurs

An expanding intracranial mass (such as a subdural haematoma) leads to:

- Uncal herniation through the incisura in the tentorium with compression of the oculomotor nerve and the motor tracts in the mid brain
- This leads to ipsilateral pupllary dilatation and contralateral hemiparesis or hemiplegia

In the Kernohan's notch syndrome which occasionally occurs there isipsilateral papillary dilatation and hemiparesis.

With progressive expansion of an intracranial mass the cerebellar tonsils eventually herniate through the foramen magnum (coning)

- This is associated with hypertension and bradycardia (Cushing's reflex)
- Sequentially apnoea, arrythmias, hypotension and death ensue

Clinical features

These patients may present with:

Features of multisystem trauma

Altered level of consciousness

Skull fractures and mass effect from intracranial lesions

Features of raised intracranial pressure

- Headaches
- Nausea
- Projectile vomiting
- Drowsiness
- Papilloedema

Complications of TBI:

- A lucid interval (often occurs in extradural haematoma)
- Post injury, the patients maintain a satisfactory level of consciousness until suddenly consciousness is lost

Extradural haematoma:

Rare; overall, occurs in less than 1% of head injuries

More common in young patients

Often results from torn middle meningeal vessels

CT shows a biconvex or lenticular opacity

Subdural haematoma:

More common

Occurs in 20 - 30% of severe head injuries, more commonly in the elderly (due to brain atrophy)

Results from torn bridging veins

The opacity on CT follows the contour of the brain

Basal skull fracture:

May be suggested by:

- Periorbital ecchymosis (racoon eyes)
- Retroauricular ecchymosis (Battle sign)
- CSF leaks
- Facial nerve palsy

Complications of TBI

Early:

Coma

Post concussion headaches

Post traumatic amnesia

Retrograde amnesia

Abnormalities of salt and water metabolism such as diabetes insipidus and syndrome of inappropriate ADH

Anterior pituitary dysfunction such as ACTH abnormalities and poor cortisol stress response

Late:

Chronic subdural haematoma

Infections such as meningitis and brain abscess

Hydrocephalus

Epilepsy

CSF leaks

Carotico-cavernous fistulae

Traumatic aneurysms

Chronic headaches
Personality changes

Treatment objectives
Identify life threatening injuries and treat
Limit primary injury
Prevent secondary brain injury
Provide critical care
Rehabilitate

Primary survey:
Assess airway and maintain patency
- Suctioning and manoeuvers to elevate the tongue (jaw thrust and chin lift) may be useful
- Apatent airway is important in optimizing outcome in TBI

Ventilation is next addressed:
- Administer 100% oxygen
- Hypoxia is one of the causes of secondary head injury and must be avoided
- Conduct a quick chest examination to identify tension pneumothorax, pneumothorax, haemothorax, flail chest etc
- Institute urgent treatment as may be indicated

Maintenance of the circulation:
- Equally important in optimizing outcomes
- Hypotension is a cause of secondary brain

injury and must be avoided
- Intravenous lines should be set up; administer crystalloids

Asses the GCS and the state of the pupils
Expose the patient to perform a quick general examination but avoid hypothermia.
Secondary Survey:
(See section on multiple injuries)
Secondary brain injury
Neuronal injury that is not present at the time of the primary insult but develops in response to subsequent intracranial or extracranial events
Extracranial causes:
Hypoxia
Hypotension
Seizures
Hyperthermia
Hyponatraemia
Hypernatraemia
Hypoglycaemia
Hyperglycaemia
Intracranial causes:
Extradural haematoma
Subdural haematoma
Intracerebral haematoma
Cerebral oedema
Cerebral contusion
Hydrocephalus
Meningitis

Brain abscess

CT scan in TBI:

- Has revolutionalized the management of traumatic brain injury as it can readily diagnose intracranial haematomas and skull fractures
- In trauma it is advisable to do a non-contrast CT scan
- Indications for CT scan:
- GCS of 14 or less
- GCS of 15 with:
- Loss of consciousness > 5minutes
- Amnesia for injury
- Focal neurological deficit
- Signs of calvarial or basal skull fracture

Intracranial pressure monitoring:

Best done through a ventriculostomy catheter, with or without concomitant intraparenchymal transducer

Indications for ICP monitoring in TBI:

- Patients with post resuscitation GCS of 8 or less
- Intubated patients in ICU
- Patients with intracranial haematomas but are adjudged not to need surgery

Emergency management of raised intracranial pressure:

- Endotracheal intubation
- Controlled ventilation to a pCO of 35 mmHg2
- Volume resuscitation
- Maintain normal blood pressure

- Narcotic sedation
- Neuromuscular blockade
- Bolus mannitol (1 g/kg)
- (See Meningitis)
- Head up tilt at 30 degrees
- Controlled hypothermia

Surgery in TBI:

Often indicated in head injury for the evacuation of intracranial haematomas or elevation of depressed skull fractures

Indications may depend on the centre and the neurosurgeon, but all agree that an intracranial haematoma causing significant mass effect should be removed

A midline shift of more than 5 mm is considered significant

Indications for surgery will depend on:

- The neurological status of the patient
- Findings on CT
- Extent of intracranial injury
- Intracranial pressure.

The procedures include:

- Burr holes
- Craniotomy
- Craniectomy
- Elevation of depressed skull fractures

Drugs in TBI:

- Diuretics to reduce intracranial pressure e.g. mannitol (see Meningitis)
- Sedatives e.g. diazepam (see Tetanus)
- Muscle relaxants e.g. diazepam, suxamethonium

- Anticonvulsants e.g. phenytoin, phenobarbital (see Epilepsy)
- Antibiotics as appropriate
- Vasopressors e.g. noradrenaline, dobutamine if there is hypotension, and in collaboration with a physician

Prevention

Measures aimed at reducing accidents in transportation (especially road traffic accidents), in homes and in factories:

- Motorbike crash helmet laws and enforcement
- Alcohol laws
- Speed limits
- Better motor licensing rules
- Health education
- Better motor engineering
- Good road designs
- Safety procedures at work and a good EMS and trauma system

MULTIPLE INJURIES

Introduction

The multiply injured patient is that patient with injury to more than one organ system

Often victims of motor vehicle crashes, motor bike accidents, pedestrians hit by cars, or falls from heights

Present a challenge to the managing team in terms of priority of medical intervention

- If the priorities are not well ordered the results can be catastrophic

Difficult to outline clinical features for these patients as virtually any injury is possible

Treatment objectives

Identify life threatening injuries and treat

Identify all injuries, institute primary management and limit progress of injuries and further tissue damage

Restore patient's physiology paying special attention to the triad of:

- hypothermia,
- acidosis
- coagulopathy

Format a prioritized plan of definitive treatment and rehabilitation

Management

Advanced trauma life support (ATLS) principles should apply

Patient should be received by a trauma team consisting of at least :

- A trauma team leader
- An airway and a procedure doctor
- Two nurses in similar capacity
- Aradiographer
- A scrub nurse
- A social worker

It is important that hospitals which regularly manage trauma patients should maintain a

standing trauma team on a 24-hour basis

- This helps to optimize outcomes in patient management

Prehospital information

The trauma team needs this information from the pre-hospital team

- Relayed in the **MIST** format, preferably before the patient's arrival to enable adequate preparation to be made before hand

M: Mechanism of injury

I: Injuries sustained

S: Prehospital vital signs: pulse, blood pressure, respiratory rate, oxygen saturation, temperature

T: Treatment given e. g cervical collar, intravenous fluids etc

Primary Survey

- Quick survey to identify life threatening injuries and treat

Airway

- Talking? Assume airway is alright. If not suction, Guedel's airways
- Careful with airway manoeuvers such as the jaw thrust and chin lift
- Always protect the cervical spine
- Apply rigid cervical collar
- May need endotracheal intubation.

Breathing

- Check the breathing, respiratory rate,

oxygen saturation

Examine the chest:

- Tension pneumothorax? Haemothorax? Flail chest?

Chest tube decompression?

- Always obtain a chest radiograph before decompression if possible
- Perform arterial blood gas estimations

Circulation:

- Check the pulse, blood pressure, capillary refill
- Listen to the heart sounds
- Apply electrocardiograph leads
- Set up an intravenous line with a large bore cannula size 14 or 16 FG
- Collect blood for investigations: ABGs, FBC, electrolytes and urea, grouping and cross matching; pregnancy tests
- Focused Assessment using Sonography in Trauma

(FAST)

Disability and Neurology

- Assess patient's level of consciousness using the

Glasgow coma scale

- Check the state of the pupils and their reaction to light
- Expose the patient to perform a quick general examination but prevent hypothermia

- Cover with warm blanket or put on artificial warmer if available
- Record core temperature

The trauma series of radiographs is part of the primary survey. These are

- A-P chest view
- A-P pelvic view
- Lateral cervical view
- (In the above order)

Secondary survey

This is a total body examination to detect injuries sustained

Involves obtaining the **AMPLE** history (allergies, medications, past medical history, pregnancy, last meal, environment including details of the accident)

Head:

- Check for scalp haematomas, lacerations, skull fractures, CSF leaks (rhinorrhoea, otorhoea); facial fractures, raccoon eyes
- Remove contact lenses; examine pupils, oral examination; Battle sign

Neck:

- Perform a careful neck examination
- Leave in collar if there is a high index of suspicion for cervical injury

Chest:

- Inspect for dyspnoea, tachypnoea, chest movements, flail chest, open

pneumothorax or obvious penetration
- Palpate for chest expansion, crepitus (subcutaneous emphysema) and rib fractures
- Assess position of the trachea and determine any tracheal shift
- Determine percussion notes in both lung fields (dull in haemothorax and hyperresonant in pneumothorax)
- Auscultate for breath sounds and air entry

Abdomen:
- Examination findings often unreliable in the multiply injured patient
- This may be as a result of altered sensorium due to head injury, inebriation or drugs, neurological injury, or distracting injury
- There is need to augment examination with bedside investigations like FAST and DPL (Diagnostic Peritoneal Lavage) if indicated
- In the haemodynamically stable patient the best imaging modality is the CT scan with contrast
- Inspect for seat belt marks, lacerations, abdominal contour and movements with respiration
- Palpate for tenderness, rebound tenderness and rigidity
- Percuss if indicated
- Auscultate for bowel sounds
- Pass a nasogastric tube Pelvis:

- Perform anteroposterior and lateral compression tests to check for pelvic fractures
- If fracture is suspected, apply a pelvic girdle or pelvic sheet to decrease pelvic volume, improve tamponade and decrease pelvic haemorrhage

Examine the perineum:

- Check for perineal bruising, bogginess, scrotal haematomas, and blood at the tip of the penis
- If there is blood at the tip of the penis it is inadvisable to pass a urethral catheter: a partial urethral rupture may be converted to a complete rupture. Do an urethrocystogram to confirm urethral rupture
- If not contraindicated pass an indwelling urethral catheter to monitor urinary output and tissue perfusion
- Haematuria is suggestive of bladder or kidney injury Perform a vaginal examination, checking for bleeding and lacerations Lower limb examination:
- Check for obvious lacerations, deformity, fractures and dislocations
- Undertake an appropriate neurovascular assessment
- Assess muscle power in each limb Upper limb examination:
- Same as for lower limb

'LOG ROLL'

- The patient is now log rolled by four persons so as to examine the back
- The spine is examined from the occiput to the coccyx checking for deformity, swellings, steppings, and tenderness
- While still in this position perform a digital rectal examination to assess anal tone, presence of blood in the rectum and the position of the prostate
- A high riding prostate is suggestive of urethral rupture
- Return patient to the supine position Neurological examination:
- Perform a detailed neurological examination as indicated

The trauma team should now note all the observed injuries and format a plan for:

- The further management of the patient
- Removal from the emergency department and

Definitive management of the patient under the appropriate surgical units and consultants

CHAPTER 18:

SURGICAL CARE

PERI-OPERATIVE CARE

Consists of:

Pre-operative care

Intra-operative care

Post-operative care

Targeted at ensuring that the patient is fit for anaesthesia and intended surgical procedure, as well as prevent complications after surgery

The focus on peri-operative care is based on the evidence that majority of complications and mortality following elective surgery are avoidable and can be prevented.

It involves what happens to the patient before going into the operating room, what happens in the operating room and what happens after the patient leaves the operating room.

The *goals* of peri-operative care include:

- Prevent avoidable complications
- Ensure early identification of complications and prompt treatment

- Prevent avoidable mortality
- Ensure quick recovery and return to activities
- Ensure safe and smooth surgery
- Ensure optimal patient outcomes

Pre-operative care

The goal is to prevent and/or minimize the risk of adverse cardio-pulmonary events during and after surgery.

Clinical evaluation

Efforts should be made to identify the following by history and physical examination: Cardiopulmonary disorders:
Cough
Chest infection
Bronchial asthma
Chronic obstructive airways disease
Hypertension
Cardiac failure Metabolic disorders:
Diabetes mellitus Haematologic disorders:
Sickle cell disease Allergy:
Drug allergies (e.g. penicillins, talc, elastoplast, antiseptics etc.)
Drug history:
Propranolol, diuretics, steroids and other hormonal agents; prednisolone, oral contraceptives; tricyclic antidepressants
Social habits:
Cigarette smoking, alcohol use

Previous anaesthetic experience:
How long ago, type of anaesthesia

Investigations
Cardiopulmonary:
Chest radiograph: especially for patients 60 years and above, and those with chest infection
- Look for evidence of chest infection and cardiomegaly

Electrocardiogram: especially for patients over 60 years and those with heart disease or hypertension
Pulmonary function tests may be necessary in patients with obstructive airways disease
Metabolic:
Urine sugar to exclude diabetes mellitus
- All adults and patients with history suggestive of diabetes mellitus

Serum Electrolytes and Urea
Haematologic: Haemogram/packed cell volume
Haemoglobin genotype
Clotting profile (prothrombin time and kaolin cephalin clotting time) where there is suspicion of bleeding diathesis e.g. in jaundiced patients
Others:
Other investigations as may be indicated by individual clinical circumstances

Correction of abnormalities and preparation for surgery

Cardiopulmonary: Rehydrate patient adequately, using appropriate fluids

Control blood pressure

Treat/control chest infections with appropriate antibiotics

Control obstructive airways disease

Metabolic conditions and derangements:

Correct electrolyte deficits, especially hypokalaemia

Acidosis is usually corrected by adequate rehydration (provided the patient has no renal disease)

Diabetes should be controlled

- Patients already controlled will need their therapy to be converted to soluble insulin for long surgical procedures (this should be done in conjunction with the physician and anaesthetist)

Haematological: Correct anaemia

- Cause(s) of anaemia should be identified and treated
- The minimum haemogram for a patient undergoing elective surgery should be 10 g/dL
- Haemogram 6 - 9 g/dL: correction may be achieved by haematinics; reschedule surgery
- Haemogram <6 g/dL: correction may require blood transfusion

- Emergency surgery: correct anaemia by blood transfusion

Blood transfusion should be avoided as much as practicable.

- Patient with sickle cell anaemia: haemogram should be brought up to 8 g/dL
- These patients must be adequately hydrated to avoid sickling and sludging within the bloodstream
- Short day case procedure: imperative to admit the patient with sickle cell anaemia at least a day before surgery to achieve adequate hydration

Suspected bleeding diathesis

- Intramuscular vitamin K (10 mg daily), at least 48 - 72 hours before surgery
- For major surgery, blood should be grouped, cross-matched and stored

Other disorders:

Any associated medical condition should be treated / controlled before embarking on surgery

- This should be done in conjunction with the physician as much as possible

Patients who require nutritional rehabilitation

- If surgery is elective reschedule it, and give adequate time to achieve improved nutritional status, otherwise morbidity and mortality may be increased

High-risk patients:

- At high risk of developing postoperative complications
- Deliberate and meticulous efforts should always be made to adequately evaluate them and ensure optimal fitness for surgery
- Elderly patients (age >60 years): - risk of deep vein thrombosis, atelectasis
- Obesity- risk of deep vein thrombosis, atelectasis
- Cancer-risk of deep vein thrombosis, atelectasis, haemorrhage
- Women on oral contraceptive pills-risk of deep vein thrombosis
- Co-existing chronic medical conditions-risk of wide ranging complications
- Sickle cell anaemia-risk of sickling crises, deep vein thrombosis

Consent for surgery

Details of the surgery should always be explained to the patient (or relatives) in very simple language before surgery

Should include a mention of the possible/common complications

A signed consent should be obtained, in the presence of a witness (usually a nurse)

Obtaining consent should be done by the surgeon himself

There are a number of evidence-based risk stratification to guide in predicting the risk of adverse events and help in instituting

appropriate measures to prevent these events (Tables 1 and 2).

Table 1: Cardiac risk stratification in patients undergoing non-cardiac surgery

Risk	Examples
High (≥5% cardiac risk)	• Emergent major operations, particularly elderly • Aortic or major vascular surgery • Peripheral vascular surgery • Upper abdominal
Intermediate (1%–5% cardiac risk)	• Intraperitoneal and intrathoracic surgery • Carotid endarterectomy • Head and neck surgery • Gynaecologic surgery • Neurosurgery • Orthopedic surgery • Urologic surgery
Low (<1% cardiac risk)	• Endoscopic procedures • Superficial procedures • Cataract surgery • Breast surgery • Ambulatory surgery

Cardiac events include fatal and nonfatal cardiac events
Incorporates perioperative cardiovascular events within 30 days after surgery

Table 2:American Society of Anaesthesiologist physical status classification system

ASA PS Classification	Definition	Examples, including, but not limited to:
ASA I	A normal healthy patient	Healthy, non-smoking, no or minimal alcohol use
ASA II	A patient with mild systemic disease	Mild diseases only without substantive functional limitations. Examples include (but not limited to): current smoker, social alcohol drinker, pregnancy, obesity (30 < BM < 40), well-controlled DM/HTN, mild lung disease
ASA III	A patient with severe systemic disease	Substantive functional limitations; One or more moderate to severe diseases. Examples include (but not limited to): poorly controlled DM or HTN, COPD, morbid obesity (BMI ≥ 40), active hepatitis, alcohol dependence or abuse, implanted pacemaker, moderate reduction of ejection fraction, ESRD undergoing

The addition of "E" denotes Emergency surgery: (An emergency is defined as existing when delay in treatment of the patient would lead to a significant increase in the threat to life or body part)

Intra-operative care

The focus of intra-operative care is to ensure a safe and smooth surgical procedure. The entire team in the operating room, including surgical team, anaesthesia team and peri-operative nursing team, should work together as a team and take responsibility for intra-operative care to ensure a smooth and safe operation. The World Health Organisation's 'Surgical Safety Checklist' is a helpful guide for safe and effective intra-operative care This checklist can be modified to suit each hospital based on local realities.

Post-operative care

An excellently performed operation can be marred by poor post-operative care and inadequate attention to patient's post-operative needs.

Meticulous and efficient care in the postoperative period is paramount for adequate patient recovery and success of surgery

A well-planned and supervised postoperative care ensures a smooth recovery, and helps to prevent or limit postoperative morbidity and mortality

Preoperative, intraoperative and postoperative care is a continuum and interlinked

- Many of the instructions and therapy started in the preoperative period may need to be continued into the postoperative period

The surgeon himself must be involved in the postoperative care and not leave it to others, who may not have much ideas or information about the surgery

Initial recovery

Close monitoring and observation:

The first 4 - 6 hours after a major surgery and general anaesthesia are critical

- The patient is still drowsy and recovering from the effects of anaesthesia

The cardiopulmonary status (pulse rate, blood pressure, respiration) needs to be monitored very closely (every 15 minutes) in order to promptly detect any abnormality

Where available, electronic monitors with an alarm system should be used

Airways management

The patient may still be under some effect of

anaesthesia

- Airways need to be kept patent

Prevent the tongue from falling backwards by positioning patient in the left lateral position
The neck should be prevented from falling on itself as this can occlude the airway
Secretions should also be cleared using a low-pressure suction

Nursing position

Different operations require specific positioning in the postoperative period to reduce venous pressures, keep airways patent, enhance drainage etc
The surgeon should be conversant with the specific positions and give appropriate instructions

Analgesia

Pain is a most undesirable effect of surgery
Patients should not be allowed to suffer from pain unduly
The appropriate analgesic technique should be chosen for the nature of surgical procedure performed
Adequate analgesia will ensure early ambulation and help to limit atelectasis
The following principle should guide the use of analgesia for control of postoperative pain:

- Multimodal approach: should be pre-emptive and preventative

- Use of ocal anaesthetics:
  - Improve analgesia
  - Decrease opioid requirements
  - Decrease opioid-related side effects
  - Can be given via:
    - Wound infiltration
    - Epidural
    - Peripheral nerve blocks
    - Opioids remain the mainstay of surgical pain control
    - Pain should be continuously evaluated using appropriate pain assessment tools to help in ensuring adequate and appropriate management

The following analgesic guide is helpful in the control of postoperative pain (Figure 1)

SEVERITY OF PAIN	TREATMENT APPROACH
SEVERE PAIN/MAJOR SURGERY (e.g.) Laparotomy Thoracotomy Major Limb surgery Cesarean section	Strong Opioids + NSAIDs or Paracetamol + LA/RA
MODERATE PAIN/MODERATE SURGERY (e.g.) Appendectomy Herniorrhaphy Excision of lumps	Week Opioids + NSAIDs or Paracetamol + LA/RA
MILD PAIN/MINOR PROCEDURE (e.g) Minor lacerations Venipuncture	NSAIDs or Paracetamol + LA

Strong Opioid example: Morphine
Weak Opioid examples: Tramadol, Pentazocine, Pethidine
Non-steroidal anti-inflammatory agents (NSAIDs) examples: ketorolac, Ibuprofen, Diclofenac, Piroxicam
LA: local anaesthesia using Bupivacaine or Lignocaine
RA: regional anaesthesia Bupivacaine or Lignocaine
LA can be done by local wound infiltration or topical application
RA can be done by nerve blocks, epidoral block, caudal block

Figure 1:Guide for postoperative analgesia

Nasogastric decompression

The stomach may need to be kept decompressed for 24 - 48 hours, particularly following gastrointestinal surgery

Decompression:

- Prevents abdominal distension and
- Prevents tension on abdominal fascial closure I
- Prevents splinting of the diaphragm and atelectasis

The widest possible bore of nasogastric tube for patient's age should be chosen

The nasogastric tube should be removed as soon as it is no longer needed, evidenced by:

- Progressively diminishing effluent (<500 mL/24 hours in an adult)
- Change from bilious colour to clear colour of gastric juice

Fluid and electrolyte balance

Ensure that the patient receives adequate amounts of intravenous fluids if oral intake is prohibited

Choose an appropriate fluid to provide enough calories and electrolytes

Glucose 5% in sodium chloride 0.9% or lactated Ringer's solution is appropriate for most adults

After the 48 hours, the daily requirement of potassium should be provided if oral intake is

still prohibited, especially if nasogastric drainage is ongoing

- This should be in form of potassium chloride added to intravenous fluids

Assess fluid and electrolyte balance on a daily basis and correct deficits

All intake (intravenous fluids, drugs, blood etc.) and output (urine, nasogastric drainage, other tubes, etc.) as well as insensible losses should be carefully recorded

Nutrition

Following major surgery, adequate nutrition should be provided for the patient, particularly if oral intake is going to be prohibited for more than 48 - 72 hours

- This can be done in the form of parenteral nutrition

Chest physiotherapy:

Bed-ridden patients and patients who have had chest or upper abdominal surgery are prone to basal atelectasis and hypostatic pneumonia.

- These should be prevented by appropriate chest physiotherapy
- Ensure adequate analgesia to enhance chest excursion
- Encourage coughing and expectoration, with a hand supporting any abdominal wound

- Periodic chest percussion to loosen bronchial secretions
- Ambulate as early as possible

Mobilization and ambulation

Mobilize and ambulate patients as early as is practicable to avoid the complications of prolonged recumbency

Ambulation should be gradual: prop up in bed, sit out of bed, short walks etc.)

Early ambulation should help prevent hypostatic pneumonia and deep vein thrombosis (very important in obese and elderly patients)

Antibiotics

Appropriate antibiotics as indicated

Irrational or indiscriminate use is not to be encouraged

Wound care

Specific surgical wounds are cared for in different ways

Clean surgeries: do not open wound (unless indicated) until day 5 - 7

Inspect wounds immediately if there are features suggestive of surgical site (wound) infection

- Undue pain
- Undue swelling
- Discharge of serosanguinous fluid or pus

Infected wounds:
Wound swab for microbiological culture and sensitivity tests
Adequate local wound care
Appropriate antibiotics
If there are systemic features (e.g. fever, anorexia) systemic treatment with antibiotics may be necessary

Care of indwelling tubes, catheters and drains

All indwelling catheters, tubes and drains should be monitored and appropriately managed to avoid infection, dislodgement/displacement
They should be removed as soon as they have served their purpose(s)

General complications in the post-operative period

Look out for general complications and treat accordingly
Postoperative pyrexia may be due to:

- Malaria
- Atelectasis and hypostatic pneumonia
- Wound infection
- Urinary tract infection
- Deep vein thrombosis
- Wound infection

ACUTE ABDOMEN

These are abdominal conditions causing

sudden or severe pain, that require immediate or urgent attention.

Cause may be surgical in nature or medical diseases.

Medical conditions should always be borne in mind as they would usually not require surgical intervention.

Common surgical and medical causes are detailed in Table 3. In newborns, intestinal obstruction (Table 4) is the commonest cause of acute abdomen and their care is different from that of adults and older children.

Table 3: Causes of acute abdomen

Surgical pathology	Medical disease
Inflammatory/Infecti ve Appendicitis, cholecystitis, pancreatitis, salpingitis, diverticulitis, primary peritonitis, intra-abdominal abscess	Metabolic disease Diabetes mellitus, porphyria
Gastrointestinal perforation Perforated typhoid enteritis, perforated peptic ulcer, GI perforation from	Haematologic disease Haemoglobinopat hy (e.g. sickle cell disease, thalasaemia),

trauma, perforated GI neoplasms,	leukaemia
Intestinal obstruction/strangulation Inguinal & other external hernias, internal hernias, intussusception, peritoneal adhesions, volvulus, obstructing GI neoplasms	Gastrointestinal infections/infestations Gastroenteritis, typhoid enteritis, parasitic infestation
Intra-abdominal haemorrhage Trauma (spleen, liver, other solid viscera), ectopic pregnancy, ruptured aortic aneurysm, ruptured neoplasm (e.g. primary liver cell carcinoma)	Extra-abdominal Infection/infestations Lower lobar pneumonia, malaria
Biliary tract obstruction	
Urinary tract obstruction	
Gynaecologic disease Twisted ovarian cyst, salpingitis, ectopic pregnancy, bleeding	

Table 4: Causes of neonatal intestinal obstruction

Common causes
Anorectal malformation
Intestinal atresia
Hirschsprung's disease
Intestinal malrotation and midgut volvulus
Incarcerated and strangulated inguinal/other hernia
Less common causes
Meconium obstruction (plug and ileus)
Extra-luminal compression from masses
Congenital peritoneal bands
Others

Clinical Evaluation

A detailed history should be taken and meticulous and thorough physical examination done. However, prompt resuscitation should not be sacrificed for taking too much time for history and

examination. Clinical evaluation and resuscitation should as much as feasible be done simultaneously to save time.

In Acute abdominal pain note the following:

- Location
- Onset and progression
- Nature and character
- Aggravating and relieving factors
- Abdominal distension
- A pasthistory of similar pain suggests complication of an underlying condition

In typhoid perforation, fever precedes abdominal pain, while the reverse is true for acute appendicitis

Nausea and vomiting:

- A frequent finding
- Common in intestinal obstruction

Altered bowel habits:

- Diarrhoea may suggest an infective/inflammatory condition
- Constipation occurs in intestinal obstruction and late in peritonitis
- The presence or absence of blood, mucus in stool should be ascertained

Fever:

- An early feature in inflammatory/infective conditions
- A late feature in most other causes of acute abdomen

Gynaecologic history:

- In every female, the following should be ascertained
- Last menstrual period: this will help in the suspicion of ectopic gestation and bleeding Graffian follicle
- Vaginal discharge: salpingitis

Urinary symptoms:
- Ascertain the presence or absence of the following
- Pain on micturition
- Pus in urine or cloudy urine
- Urethral discharge
- Loin pain

Past medical history:
- Diabetes mellitus
- Sickle cell disease

Physical examination:

General examination
- Dehydration
- Temperature (the exact temperature should be taken with a thermometer: oral, axillary or rectal temperature)
- Pallor
- Jaundice
- Foetor (as in diabetic ketoacidosis etc.)

Haemodynamic status:
- Pulse rate: >100/minute is abnormal
- Blood pressure: <100 mmHg systolic and <60 mmHg diastolic pressures indicate hypotension in an adult

Chest:

- Examine carefully for evidence of chest infection

Abdomen:

- Distension
- Presence of scars of previous surgery or bruising in trauma
- Visible peristalsis (suggests intestinal obstruction)
- General peritonitis:

There may be no movement with respiration

Ascertain the site of tenderness

Localized:

- Right iliac fossa (appendicitis, gynaecologic conditions etc.)
- Right hypochondrium (cholecystitis)

Generalised: varied causes

As much as possible any palpable mass should be characterized

If tenderness is not too marked, ascertain the presence of free fluid in the peritoneal cavity by shifting dullness or fluid thrill (ascites)

Listen for bowel sounds

- Diminished or absent in peritonitis; exaggerated in early stages of intestinal obstruction

Rectal examination:

- Look for perianal soilage
- Presence or absence of faeces in rectum
- Palpate rectovesical pouch or rectouterine pouch (of Douglas) for

bogginess and tenderness indicating a pelvic collection of pus or blood

Examine the faeces on the examining finger for blood, mucus Vaginal examination:

- May be necessary to exclude gynaecological conditions

Differential Diagnosis

Give very careful thought to findings at clinical evaluation and list of possible causes in table 1, and then make a list 3 – 5 possible differential diagnosis before proceeding to carry out relevant investigations.

Investigations

Plain radiography

Abdomen:

- Supine and upright films to identify features of intestinal obstruction (dilated bowel loops and multiple fluid levels)
- Aradio-opaque shadow may be seen in the region of the urinary tract in ureteric colic

Chest:

- An upright film may identify gas under the diaphragm in gastrointestinal perforation
- Chest infection should also be looked for

Abdomino-pelvic ultrasonography:

Should help to ascertain the cause of pain in a proportion of the patients (e.g. cholecystitis,

gynaecologic conditions, urinary calculi, and degenerating masses)

- May identify injured solid organ in trauma

Diagnostic peritoneal lavage:

- Useful in abdominal trauma to identify haemoperitoneum and leakage of gastrointestinal contents and secretions of other organs into the peritoneal cavity

Biochemical tests:

- Urinalysis: test the urine for sugar, protein, ketones, etc
- Random blood sugar to exclude diabetes mellitus
- Serum electrolytes and urea; correction may be needed
- Serum amylase to exclude acute pancreatitis

Haematological tests:

- Haemogram to exclude anaemia
- Packed cell volume may not be reliable because of haemoconcentration from dehydration
- If there is suspicion of sickle cell disease, the haemoglobin genotype should be obtained
- A complete blood count may show evidence of acute infection (leucocytosis, neutrophilia)
- Blood should be grouped, and compatible blood cross-matched and made ready

Other investigations:

- Computed tomography may be needed when there is diagnostic confusion
- Cultures: any suspicious fluid and materials should be obtained and sent for microbiology and culture (e.g. vaginal discharge, peritoneal fluid)

Management

Resuscitation and General Measures

In most patients, resuscitation and institution of some general measures are necessary before proceeding to definitive treatment of the condition

Time taken to adequately resuscitate the patient is critical to achieving a good outcome and preventing/minimizing morbidity and mortality. ,

Monitored very closely by repeated examinations and electronic monitoring to identify when patient is adequately resuscitated

Avoid time wasting as well as identify patients who are not responding to resuscitation and require additional measures

Surgery may become part of resuscitation as a damage control measure but such surgery is usually limited in extent.

General measures

Resuscitation

Rehydration and correction of electrolyte derangements

Correct shock by giving crystalloids (sodium chloride 0.9%, Ringer's lactate) or colloid (e.g. dextran)

Maintenance fluids are calculated based on degree of dehydration

Correct electrolyte deficits (especially potassium)

Nasogastric decompression: the largest possible size of tube for patient

Aspirate intermittently using low pressure suction or large syringe

Urethral catheterization (to monitor urine output)

Correct anaemia (by blood transfusion)

Commence broad spectrum, intravenous antibiotics effective against likely microorganisms

- Do not give aminoglycosides until urine output is adequate

Monitor the following parameters to ensure adequate rehydration:

- Cardio-respiratory stability
- Pulse rate
- Blood pressure
- Central venous pressure
- Pulmonary capillary wedge pressure
- Urine output, volume, colour
- Hydration status
- Skin turgor

- Sensorium

Ascertain level of consciousness

Evidence of adequate resuscitation

- Pulse rate begins to fall towards, or below 100 beats/minute
- Blood pressure: begins to towards normal
- Urine output: 50 - 100mL/hr (1 - 2 mL/kg/hr); clear or amber

Definitive Treatment

Once the patient is adequately resuscitated, definitive treatment can proceed. The treatment of newborns with intestinal obstruction is summarized in Table 5.

Surgical conditions:

Most of the surgical conditions will require urgent laparotomy after adequate resuscitation

- Evacuation of pus, blood and all infected material
- Meticulous examination of all organs and recesses
- Identify primary pathology
- Identify other associated/coexisting pathology
- Treat identified pathologies on their merits
- Cleanse peritoneal cavity with large volumes of warm sodium chloride 0.9%

Medical conditions:

Consult a physician as appropriate, to treat

the condition accordingly
It's important that repeated examination and monitoring continues during and after definitive treatment to identify any problems and promptly attend to them.

Table 5: Neonatal Intestinal Obstruction: recognition and management

Recognition	Diagnostic Evaluation	Management
Key Symptoms	Plain abdominal X-ray (upright/supine): number/distribution of gas shadows, free peritoneal gas	Ensure patent airways: secretions/vomitus Ensure adequate breathing and oxygenation Decompress stomach with N/G tube to improve respiration
Bilous vomiting	Serum electrolytes/urea	Rehydration Insert urinary catheter to monitor urine output
Abdominal distension	Complete blood count	Parenteral broad spectrum antibiotics
Non/delayed passage of	Specialised imaging as	Keep warm

meconium, constipation	necessary	
Others		Refer to appropriate hospital if no capacity to handle (only when stable after resuscitation)
Key Physical Findings		Definitive treatment at appropriate hospital with capacity for newborn surgery
Dehydration, anaemia, jaundice		

Prognosis

Outcome and survival depends on:

Early presentation and diagnosis

Prompt and adequate resuscitation before surgery

Appropriate and meticulous surgery and other treatments as indicated

PREVENTION OF BLOOD LOSS AND BLOOD TRANSFUSION IN SURGERY

Blood transfusion is the introduction of whole blood or blood components into the blood stream of an individual

Should be used appropriately because its use is not without complications and untoward effects

Several techniques and manouvres (Table 6) are available to help minimize bloss loss at surgery, and hence minimize blood transfusion.

Table 6: Use of simple techniques to minimize blood loss at surgery

Techniques
Elevation of site of surgery
Pressure (digital and sponge)
Electrocautery
Clipping and ligature
Surgical glues (e.g. fibrin glue)
Tourniquets (elastic bandage or pneumatic, useful in limbs, remember tourniquet time, remember to remove before closure of skin wound)

Blood and its commonly used components:

Whole blood

Packed red cells

Fresh frozen plasma
Clotting factor concentrates
Platelet concentrate

Basic principles of blood transfusion:
Appropriate use
Adequate evaluation before transfusion to ascertain the indication, amount and component required
Screening for communicable diseases (HIV, hepatitis, etc.) before transfusion
Adequate grouping and cross-matching before transfusion
Store under at appropriate temperature
Use blood fractions whenever possible to avoid wastage
Use autologous blood whenever possible to minimize risk of transfusing communicable diseases *Transfusion is not a substitute for meticulous and appropriate surgical techniques*

Indications for blood transfusion

- To replace lost blood volume
- Haemorrhage from trauma and other forms of blood loss
- Operative haemorrhage
- To improve oxygen carrying capacity
- Various types of anaemias To replace clotting factors
- Some liver diseases
- Deficiency states

Complications

Early complications:

- Immune reactions
- ABO incompatibility
- Rhesus incompatibility
- Febrile reactions
- Allergic reactions
- Reactions to plasma proteins

Biochemical complications:

- Hyperkalaemia
- Citrate toxicity (hypocalcaemia) Haemoglobinaemia Infective complications: Bacteraemia
- Transfusion of parasites (e.g. malaria)
- Transfusion of viruses (HIV, Hepatitis B, C, D)

Physical complications:

- Volume overload
- Air embolism
- Hypothermia

Complications of massive blood transfusion:

Massive transfusion refers to the single transfusion of 50 - 100% of the equivalent of an individual's blood volume in less than 24 hours

- 2.5 - 5 litres in adults and 40 - 80 mL/kg body weight in children

The complications are related to:

Volume overload

Transfusion of old blood

Electrolyte derangements (especially potassium and calcium)

Transmission of infections
Delayed complications:
- Haemosiderosis
- Post transfusion purpura

Autologous transfusion
Transfusion of the patients' own blood

Advantages
Reduced risk of transmitting communicable diseases
Overcomes the problem of shortage of blood
Types and methods
Pre-deposit blood
- Usually best done in conjunction with haematology staff
- The patient donates one unit of blood at a time (e.g. weekly) several weeks before the elective surgery
- Following donation, the patient is given haematinics, and sometimes erythropoietin to enhance bone marrow function; the blood is stored for later use

Pre-operative isovolaemic haemodilution
- Just before elective surgery, 1 - 2 units of blood are taken from the patient and replaced by volume expanders such as Ringer's lactate, sodium chloride 0.9%, or colloid
- The blood taken is transfused intraoperatively after all haemostasis has been secured

Intraoperative blood salvage

- Appropriate for patients undergoing laparotomy or thoracotomy for haemorrhage into these cavities (e.g. traumatic haemothorax, splenic injury, ectopic gestation)
- The blood is collected in an appropriate blood bag and then transfused using a blood giving set with filter
- Special salvage equipment may be available sometimes
- Contaminated blood must not be transfused

Contraindications to autologous transfusion

Pregnancy

Chronic medical conditions

Cancer

Situations where the blood may have become contaminated (this is for intraoperative blood salvage)

Children:

Other sources of blood

Umbilical cord blood

Alternatives to blood transfusion

Since blood transfusion is attended by several untoward effects and complications, efforts are continuously being made to identify alternatives to transfusion

- Most of these are experimental at the

moment and are not practicable in the clinical setting

PREVENTION AND CONTROL OF SURGICAL SITE INFECTION

Postoperative surgical site infection (SSI) is a rather common, but undesirable complication. Increase postoperative morbidity and may sometimes lead to mortality. Efforts therefore need to be made to prevent it.

Prevention requires a good appreciation of the risk stratification according to type of surgical wound as well as the indications for the utilization of antibiotic prophylaxis (Tables 7 and 8).

Table 7: Class of surgical wounds and risk of SSI

Class of surgical wound	Characteristics	Risk of SSI (%)
I. Clean	Uninfected operative wound No inflammation Respiratory/alimentary, urinary tract not	<2

	entered Primarily closed or drained with closed drain	
II. Clean-Contaminated	Respiratory/alimentary/urinary tract entered under controlled conditions No unusual contamination No major break in technique	4 – 10
III. Contaminated	Open, fresh accidental wounds Major break in sterile technique Gross spillage from respiratory/alimentary/urinary tract Acute non-purulent inflammation	>20
IV. Dirty/Infected	Old traumatic wounds with retained devitalised	>40

	tissue Existing clinical infection or perforated viscera (organism	

www.cdc.gov/hicpac/SSI/table7-8-9-10-SSI.html

Table 8: Evidence-based indications for antibiotic prophylaxis

Class of surgical wound	Use of antibiotics
Clean	None
Clean *(implant, prosthesis, valvular heart disease, immunosuppression, on steroids etc.)*	Prophylactic
Clean-contaminated	Prophylactic
Contaminated	Prophylactic
Dirty/Infected	Therapeutic

Prophylactic antibiotics for prevention of SSI

Objective of antibiotic prophylaxis

To prevent postoperative infection in

susceptible patients

Principles of antibiotic prophylaxis

Should be used only where there is a high risk of bacterial contamination

Intravenous route is preferred to achieve optimum effect

Should be given not >2 hours before surgical incision

- Many surgeons prefer to give at the time of induction of anaesthesia

Should be repeated intraoperatively if the surgery lasts for >3 hours

Not more than 2 - 3 doses (not longer than 24 hours) should be given after surgery

Antibiotics should be reinstituted if infection occurs

Choice of antibiotics

Should depend on the known prevalent bacteria in the part of the body

Broad spectrum antibiotics are preferred

Combination of antibiotics (with synergistic actions) is preferred to a single antibiotic

Should be used only when scientific evidence shows benefit

Indications for antibiotic prophylaxis

Clean-contaminated and contaminated surgical wounds

Patients with increased risk of infection: e.g. Immunosuppresion, diabetes mellitus, severe malnutrition, patients on steroids, patients on cytotoxic chemotherapy
When prosthesis or implants are used
To prevent infective endocarditis in patients with valvular heart disease or prostethic heart valves
Patients with peripheral vascular disease undergoing surgery on that limb

Complications

- Antibiotic misuse
- Antibiotic resistance
- Side effects of antibiotics (e.g. pseudomembranous colitis)
- False sense of surgical security

Antibiotic prophylaxis should be effective and efficient

CHAPTER 19:

PAEDIATRIC PERSPECTIVES

MEASLES (Rubeola)

Introduction

An acute viral infection caused by an RNA virus of the genus Morbillivirus in the family Paramyxoviridae

- Only one serotype is known

Endemic throughout the world

A major contributor to childhood mortality, 79% reduction in deaths from measles due to the global push to improve vaccine coverage

Approximately 114900 deaths in 2014

Also a major cause of preventable blindness

Transmission is by droplet infection during the prodromal stage

Incubation period: 9 - 11 days

Time of exposure to appearance of rash: about 14 days

Clinical features

The essential lesion is found on the skin, mucous membranes of the nasopharynx, bronchi, intestinal tract and conjunctivae

Three stages:

Incubation period
Prodromal stage with an enanthem
Final stage
Incubation period:
Mild fever; 10 - 11 days
Prodromal stage:
3-5days
Low grade to moderate fever
Dry cough
Coryza
Conjunctivitis
Koplik spots
Photophobia
Final stage:
Temperature rises abruptly as the rash appears
Rash begins from the upper lateral part of the neck, behind the ears, along the hairline and posterior parts of the cheek then spreads to the rest of the body
Rash fades in the same pattern in 3 - 4 days
Associated lymphadenopathy

Differential diagnoses
Rubella
Roseola infantum
Infections from Echovirus, Coxsackie Virus and
Adenovirus
Infectious mononucleosis
Toxoplasmosis

Meningococcaemia
Scarlet fever
Rickettsial diseases
Kawasaki disease
Serum sickness
Drug rashes

Complications

Diarrhoea
Otitis media
Pneumonia
Laryngo-tracheobronchitis
Malnutrition
Encephalitis
flaccid
Seizures
or
Blindness
Subacute sclerosing panencephalitis

Investigations

Isolation of the virus by tissue culture
ELISA: first IgM and later IgG response
Demonstration of Warthin Finkeldy giant cells in smears of the nasal mucosa
Full Blood Count: low white blood cell count with relative lymphocytosis
Lumbar puncture: increase in CSF protein; and small increase in lymphocytes, normal glucose level

Treatment objectives

Relieve symptoms
Hydrate adequately
Treat secondary bacterial infection
Prevent complications

Non-drug treatment
Humidification of the room for those with croup
Protection from strong light for those with photophobia
Nutrition
Fluids

Drug treatment
No specific drugs
Some children require supplemental vitamin A
- 100,000 IU stat for age 6 months - 1 year
- 200,000 IU stat for age above 1year
- Repeat on days 2 and 14 for those with ophthalmologic evidence of vitamin A deficiency

Specific treatment of complications:
- use of dexamethazone for Croup

Notable adverse drug reactions
Vitamin A may cause features of pseudotumour cerebri
- Nausea, vomiting, drowsiness, bulging fontanelle, diplopia, papilloedema and cranial nerve palsies

Prevention

Isolation precaution from the 5th day of exposure until

5days after appearance of the rash

Measles vaccine at 9 months

- Vaccine may be given at 6 months for measles post-exposure, and in outbreak prophylaxis

Post-exposure prophylaxis

- Passive immunization with immune globulin within 6 days of exposure

POLIOMYELITIS

Introduction

An acute infectious disease of humans (particularly children) caused by any of three serotypes of poliovirus

P1, P2, and P3

Immunity to one serotype does not confer immunity to others

Occurs in many regions of the developing world

The global polio eradication initiative was launched in 1988

- In 15 years, the number of cases had fallen by 99% and the number of infected countries reduced from 125 to 7
- There was an increase in global cases as a result of an epidemic in India

In 2015, Nigeria was declared a polio free

country.

Pathogenesis

Entry into mouth (via faecally-contaminated food/water)
Replication in pharynx, gastrointestinal tract, local lymphatics
Haematologic spread to lymphatics and central nervous system
Viral spread along nerve fibres
Destruction of motor neurons

Clinical features

Incubation period: 6 - 20 days, with a range of 3 – 35 days
Asymptomatic infection: 95%
Minor non-specific symptoms: 4 - 8%
Symptoms occur in less than 2 %

- Slight fever
- Headache
- Malaise
- Sore throat
- Vomiting

Non-paralytic polio (1-2%)

- Symptoms last 1 - 2 weeks

Moderate fever
Headache
Vomiting
Diarrhoea
Fatigue

Irritability
Pain or stiffness of the back, arms, legs, abdomen
Muscle tenderness and spasms in any part of the body
Neck pain and stiffness
Skin rash

Paralytic polio

3 types depending on the level of involvement

- Spinal polio: 79%
- Bulbar polio: 2%
- Bulbospinal: polio 19%

Fever 5 - 7 days before other symptoms
Headache
Stiff neck and back
Assymmetric muscle weakness
Rapid onset
Progresses to paralysis

- Location of paralysis depends on region affected

Abnormal sensation
Hyperaesthesia
Difficulty in initiating micturition
Constipation
Bloated abdomen
Dysphagia
Muscle spasms
Drooling
Dyspnoea

Irritability
Positive Babinski's sign

Complications
Multiple intestinal erosions
Acute gastric dilatation
Hypertension
Hypercalcaemia
Nephrocalcinosis
Vascular lesions
Myocarditis
Pulmonary oedema
Pulmonary embolism
Paralysis of limbs, muscles of respiration and swallowing which can be fatal

Differential diagnoses
Guillain- Barre syndrome
Lead toxicity
Cranial nerve Herpes zoster
Post-diphtheric neuropathy
Arthropod borne viral encephalitis
Rabies
Tetanus
Botulism
Encephalomyelitis: demyelinating type
Neoplasms in and around the spinal cord
Familial periodic paralysis
Myasthenia gravis
Acute porphyrias
Hysteria and malingering

Conditions causing pseudoparalysis
Unrecognized trauma
Transient toxic synovitis
Acute osteomyelitis
Acute rheumatic fever
Scurvy
Congenital syphilis: pseudoparalysis of Parrot

Complications
Multiple intestinal erosions
Acute gastric dilatation
Hypertension
Hypercalcaemia
Nephrocalcinosis
Vascular lesions
Myocarditis
Pulmonary oedema
Pulmonary embolism
Paralysis of limbs, muscles of respiration and swallowing which can be fatal

Investigations
Viral isolation from stool, pharynx or cerebrospinal fluid
If the virus is isolated from a person with acute paralysis, it must be tested further, using fingerprinting genomic sequencing to determine if it is the wild type or vaccine type
Serology: a fourfold rise in antibody may be demonstrated

Cerebrospinal fluid examination:

- Raised white cell count, 10 - 200 cells/mm3(primarily lymphocytes)
- Mild increase in protein: 40 - 50 mg/mL

Treatment objectives

Allay fear
Minimize ensuing skeletal deformities
Anticipate and treat complications
Prepare the child and family for a prolonged management of permanent disability if it seems likely

Non-drug treatment

Bed rest
Avoidance of exertion
Application of hot packs
Lying on a firm bed
Hospitalization for those with paralytic disease
Suitable body alignment to avoid excessive skeletal deformity
Active and passive motions as soon as pain disappears
Manual compression of the bladder
Adequate dietary and fluid intake
Review by orthopaedist and psychiatrist
Gravity drainage of accumulated secretions
Tracheostomy in case of vocal cord paralysis

Drug treatment

Bethanicol 5 - 10 mg orally or2.5 - 5 mg subcutaneously for bladder paralysis

Analgesics

- Avoid opiates if there is impairment of ventilation

Treat urinary tract infection with appropriate antibiotics

Prevention

Hygienic practices

- To prevent / limit contamination of food and water by the virus

Vaccination

- The only effective method of prevention

Oral Polio Vaccine

Given at:

Birth

6 weeks

10 weeks

14 weeks

- Highly effective
- 50% immune after 1 dose
- >95% immune after 3 doses
- Confers herd immunity
- Immunity probably life long
- Limits spread of wild polio virus

Inactivated Polio Vaccine:

Given at: 2 months 4 months 12 months

- Highly effective
- >90% immune after 2 doses

- >99% immune after 3 doses
- Duration of immunity not known with certainty ***Notable adverse drug reactions, caution and contraindications***

Oral polio vaccine:

- Paralytic poliomyelitis
- Should not be administered to persons who are immunocompromised (it is a live vaccine)

Contra indicated in :

- Persons with history of severe allergic reaction to a vaccine component or following prior dose
- Moderate or severe acute illness

Inactivated vaccine may be used in immune compromised persons

- It may (rarely) cause local reactions

VITAMIN A DEFICIENCY

Introduction

Vitamin A was the first fat-soluble vitamin to be discovered

It comprises a family of compounds called the retinoids

In nature, the active retinoids occur in 3 forms - Alcohol (retinol), aldehyde (retinal or retinaldehyde) and acid (retinoic acid)

In the human body, retinol is the predominant form, and 11-cis-retinol is the active form

Retinol-binding protein (RBP) binds vitamin

A and regulates its absorption and metabolism Vitamin A is essential for:
Vision (especially dark adaptation)
Immune response
Epithelial cell growth and repair
Bone growth
Reproduction
Maintenance of the surface linings of the eyes
Epithelial integrity of respiratory, urinary, and intestinal tracts
Embryonic development
Regulation of adult genes
It functions as an activator of gene expression by retinoid alpha-receptor transcription factor and ligand-dependent transcription factor

Deficiency of vitamin A is found among malnourished children, the elderly, and chronically ill populations in the United States, but it is more prevalent in developing countries.

Among the first signs of vitamin A deficiency (VAD) are:

- Abnormal dark adaptation
- Dry skin and dry hair
- Broken fingernails
- Decreased resistance to infections

Epidemiology

An estimated 250 million children in developing countries are at risk for vitamin

deficiency syndromes

The most widely affected group includes up to 10 million malnourished children who develop xerophthalmia and have an increased risk of complications and death from measles

Each year 250,000 - 500,000 children become blind because of VAD

Improving the vitamin A status of children (aged 6 - 59 months) with deficiencies can reduce rates of death from measles by 50%; from diarrhoea by 33%, and from of all causes of mortality by 23%

Pathophysiology

Vitamin A deficiency may be secondary to:
Decreased ingestion
Defective absorption and altered metabolism
Increased requirements

An adult liver can store up to a year's reserve of vitamin A, whereas a child's liver may have enough stores to last only several weeks

Serum retinol concentration reflects an individual's vitamin A status

Because serum retinol is homeostatically controlled, its levels do not drop until the body's stores are significantly limited

The serum concentration of retinol is affected by several factors:

- Synthesis of Retinol Binding Protein in the liver

- Infection
- Nutritional status
- Adequate levels of other nutrients such as zinc and iron

Recommended Daily Allowance

Infant (1 year or younger)

- 375 micrograms Child 1 - 3 years
- 400 micrograms Child 4 - 6 years
- 500 micrograms

Child 7 - 10 years

- 700 micrograms

All males older than 10 years

- 1000 micrograms

All females older than 10 years

- 800 micrograms

Aetiology

Malnutrition

- The commonest cause of VAD in this part of the world

Inadequate intake

Measles infection

Increased risk of deficiency in:

Fat malabsorption

Cystic fibrosis

Tropical sprue

Pancreatic insufficiency

Inflammatory bowel disease

Cholestasis

Small bowel bypass surgery

Vegans

Refugees
Recent immigrants
Alcoholism
Toddlers and pre-school children living below the poverty line

Clinical features

VAD may be asymptomatic
Increased risk of respiratory and diarrhoeal infections
Decreased growth rate
Retarded bone development
Increased fatigue as a manifestation of VAD anaemia
Bitot spots
Poor dark adaptation (nyctalopia)
Dry skin
Dry hair
Pruritus
Broken fingernails
Keratomalacia
Xerophthalmia
Follicular hyperkeratosis (phrynoderma) from blockage of hair follicles with plugs of keratin
Excessive deposition of periosteal bone secondary to reduced osteoclastic activity
Anaemia
Keratinization of mucous membranes

Differential diagnoses

Cataract
Refractive errors
Zinc deficiency

Complications
Blindness
Corneal ulceration

Investigations
Serum retinol
- Costly but is a direct measure
- A value of less than 0.7mg/L in children younger than 12 years is considered low

Serum RBP
- Easier and less expensive to perform than retinol
- Less accurate because levels are affected by serum protein concentrations; types of RBP cannot be differentiated

Serum zinc
- Useful because zinc deficiency interferes with RBP production

Iron panel
- Useful because iron deficiency can affect the

metabolism of vitamin A
Serum albumin
- Levels are indirect measures of levels of vitamin A

Full Blood Count with differentials
- If anaemia, infection, or sepsis is a

possibility

Serum electrolytes

Liver function tests

- To evaluate nutritional status

Radiographs of the long bones

- To evaluate bone growth and excessive deposition of periosteal bone

Clinical testing for dark-adaptation threshold

Treatment objectives

Reduce morbidity

Prevent complications

Treat complications

Non-drug treatment

Eat foods rich in vitamin A

- Liver
- Beef
- Chicken
- Eggs
- Whole milk; fortified milk
- Carrots
- Mangoes
- Orange fruits
- Sweet potatoes
- Spinach
- Green vegetables

At least 5 servings of fruits and vegetables per day is recommended to provide a comprehensive distribution of carotenoids

Drug treatment

Daily oral supplements of vitamin A

Child:

Less than, or 3 years

- 600 microgram (2,000 IU) orally once daily

4-8years

- 900 microgram (3,000 IU) orally once daily

9-13years

- 1,700 microgram (5,665 IU) orally once daily

14 - 18 years

- 2,800 microgram (9,335 IU) orally once daily

Adult: all ages 3,000 microgram (10,000 IU) orally once daily

Severe disease

- 60,000 microgram (200,000 IU) orally for a minimum of 2 days
- Has been shown to reduce child mortality rates by 35 - 70%

Notable adverse drug reactions, caution

Risk of teratogenicity increases in pregnant women at doses >800 micrograms/day (not recommended at these doses)

Contraindicated in

- Documented hypersensitivity
- Hypervitaminosis A:

Parenteral vitamin A in infants of low birth

weight may be associated with:
Thrombocytopenia
Renal dysfunction
Hepatomegaly
Cholestasis
Ascites
Hypotension
Metabolic acidosis (E-Ferol syndrome)

Prevention
Eat foods rich in vitamin A, in adequate amounts
Family and community health education

SICKLE CELL DISEASE

Introduction
A group of inherited red blood cell disorders with one abnormal haemoglobin called Haemoglobin S and another that is also abnormal in the rbc.
Haemoblobin molecule is unusually sensitivity to low oxygen tension making it to denature affecting its shape to sickle and its oxygen carrying capacity
If the other abnormal haemoglobin is another S, the condition is called sickle cell anaemia, the commonest variant of sickle cell disorders.
Manifestations which vary with age and may have intermittent episodic events called crises tend to occur.

The two broad forms are :

- vaso-occlusive (painful/thrombotic crisis)
- Anaemic

Can also occur together in the same patient.

CLINICAL PRESENTATION

< 6 months

Usually do not show any feature of the disease due to HbF protection

Late infancy (> 6mths – 2yrs)

Persistent pallor/anaemia, jaundice, hepatosplenomegaly, and bone pains – extremities commonly.

Pre-school (2 – 5 years)

Persistent pallor/anaemia, jaundice, hepatosplenomegaly, bone pains, *dactilytis – painful swelling of the digits, and *hand-foot syndrome – bilateral, painful, warm, non-pitting swelling of the dorsa of hands and feet, sickle cell habitus – bossing and gnathopathy.

**Due to ischaemic necrosis of the bones of extremities. They are earliest specific manifestation*

School age (6 – 12 years)

Persistent pallor/anaemia, jaundice, hepatomegaly ± splenomegaly (due to autosplenectomy), sickle cell habitus –

bossing (prominent facial and skull bones due to increased bone marrow activity); gnathopathy (overridding of maxillary bone over the mandible); asthenia (leaner and less weight) - lower weight, height and BMI (though some have catch-up during adolescence) and bone pain – usually back bones.

Adolescents
Persistent pallor/anaemia, jaundice, hepatomegaly ± splenomegaly, sickle cell habitus, delayed sexual development in both sexes (mean menarcheal age 15.5 years as against 13.4 years in AA girls) and bone pain.

CRISES IN SCD
Vaso Occlusive Crises
Bone pain crisis, abdominal pain crisis, acute hepatopathy, acute chest syndrome, priapism and cerebrovascular accident (CVA).
Precipitating factors to VOC – fever, infection, physical exertion, extremes of weather and emotional disturbance
Anaemic Crises
- Aplastic crisis – due to parvovirus B19
- Hyperhaemolytic crisis – precipitated by malaria and bacterial infection
- Sequestration crisis – trapping of

significant proportion of rbc in the spleen
- Megaloblastic changes – folic acid deficiency

COMPLICATIONS OF SCD

Occur in almost all the body organs.

- Musculoskeletal system complications
  - Osteomyelitis – commonly staph aureus
  - Avascular necrosis of hip or shoulder joints
  - Pathological fracture
  - Digital clubbing
  - Chronic leg ulcer – common in adolescents, affects the skin around the malleoli, heals slowly and recurs.
  - Kyphosis
  - Scoliosis
  - Kyphoscoliosis
- Hepatobiliary system complications
  - Hepatomegaly
  - Hepatic coma – rare
  - Haemosiderosis – transfusion associated
  - Cholelithiasis – uncommon in Africa
- Genito-urinary system complications
  - Hyposthenuria, polyuria, nocturia, enuresis – due to impaired urinary concentrating ability. Begins by 4-

5years
- Haematuria – usually from the left kidney
- Proteinuria – early manifestation of sickle cell nephropathy
- Bacteriuria – affects 22% of febrile SCD children
- Pyelonephritis
- Renal tubular acidosis
- Cardiovascular system complications
  - Cardiomegaly – due to functional adaptation to chronic anaemia
  - CCF
  - Cor pulmonare
- Respiratory system complications
  - Pulmonary hypertension
  - Hypoxaemia – hallmark of pulmonary function abnormality in SCD
  - Reduced lung volume – Due to frequent pneumonia and reduced thoracic size
- Central nervous system complications
  - CVA
  - Seizures
  - Sensorineural hearing loss
  - Ocular lesions – anterior and posterior chamber lesions
- Susceptibility to infection
  - Sepsis and other bacterial infections especially pneumonia, H. influenza,

salmonella due to
  - Altered splenic function
  - Functional asplenia
  - Defective alternate pathway of complement activation
  - Elevated serum iron
  - Deranged pulmonary alveolar macrophage function from chronic hypoxia
- Impaired psychosocial function
  - Physical, skeletal and sexual maturation problems
  - Societal attitude / discrimination
  - Frequent ill-health and hospitalization
  - School absenteeism
  - Role limitations
  - Loss of job
  - Persistent jaundice
  - Neurocognitive impairment
  - Academic performance
  - Parents and siblings psychological problems

INVESTIGATIONS

- Full blood count (FBC)-
- Haemoglobin, PCV, RBC count
- Total leukocyte count, differential leukocyte count (Neutrophils including bands, lymphocytes, monocytes, basophils, eosinophils)
- Platelet count
- Erythrocyte sedimentation rate (ESR)

- Red blood cell indices (MCV, MCHC, MCH)
- Blood film may show sickled and other abnormally shaped red cells, malaria parasites
- Reticulocyte count
- Sickling test using 2% sodium methabisulphite as a deoxygenating agent
- Solubility test using sodium dithionate as buffer
- Haemoglobin electrophoresis using cellulose acetate paper at pH 8.4 (alkaline) or citrate agar gel at pH 5.6 (acidic). This detects the variant haemoglobin bands
- High performance liquid chromatography (HPLC) for Haemoglobin quantitation
- Serum electrolytes, urea and creatinine
- Liver function test:
- serum bilirubin-total and conjugated
- transaminases
- alkaline phosphatase
- serum albumin
- prothrombin time
- Urinalysis, microscopy, culture and sensitivity
- Stool microscopy for ova and parasites, occult blood
- Sputum M/C/S, acid fast bacilli
- Chest X-Ray
- Ultrasound scan

- Abdominal ultrasound
- Trans cranial Doppler ultrasound (TCD)- detects those at increased risk of cerebrovascular accident (CVA)
- Magnetic Resonance Imaging (MRI)- detects brain micro infarcts
- Organ dysfunction screen:
- kidney: urinalysis, 24-hour urine protein quantitation, renal ultrasound, measurement of GFR, screen for microalbuminuria
- echocardiography detects pulmonary hypertension
- Pulse oximetry

TREATMENT

- Objectives:
  - Maintain (or restore) a steady state of health
  - Prevent and treat complications
  - Provide accurate diagnosis, relevant health education and genetic counselling to patients, relatives and heterozygotes
  - Improve quality of live
  - Provide a positive self-image in affected persons
- Strategies:
  - Counseling and health education
  - Encouraging membership of support groups
  - Providing infection prophylaxis
    - Anti-malarial

- Anti-pneumococcal
- Hepatitis B, pneumococcal, H influenza and other childhood vaccines
- Providing folate supplementation
- Avoiding pain-inducing conditions
- Providing prompt treatment of symptoms
- Advising on contraception
- Supervising pregnancy/Labour
- Providing regular health checks
- Limiting family size
- Non-drug treatment:
- Balanced diet- encourage adequate intake of vegetables and fruits
- Adequate fluid intake (at least 3 litres per/24 hours or 1.5L/m^2/24 hours)
- Avoidance of
- Pain-inducing conditions
- Strenuous exertion or stress
- Dehydration
- Sudden exposure to extremes of temperature
- Infections e.g. malaria (encourage use of insecticide treated nets)
- Emotional stress
- Adjunct treatment:
- Blood transfusion (especially red cell transfusion)
- Drug treatment:
- Steady state (when patient is well with no complaints)
- Proguanil

- Infants 25mg orally daily
- 1-4 years 50mg orally daily
- 5-8 years 100mg orally daily
- 9-14 years 150mg orally daily

Plus

- Folic acid 5mg orally daily

Plus

- Other vitamins as may be required
- Omega 3 fatty acids
- K-thiocyanate
- Penicillin for children
- >2 months-3 years, 125mg orally 12 hourly
- Over 3 years, 250mg orally 12 hourly

Pain crisis:

- Mild pain

Paracetamol:

Child 10mg/kg/dose orally 4-6 hourly (max. 4 doses/24 hours)

OR

- Ibuprofen:
- child 5-10mg/kg/dose orally 6-8 hourly (max. 40-60mg/kg/24 hours)
- Moderate to severe pain

Diclofenac sodium:

child 2mg/kg/24 hours intramuscularly in 2-4 divided doses

OR

- Pentazocin:
- adult and child> 14 years 30mg/dose 4-6 hourly intramuscularly

OR

- Dihydrocodeine (DF118) orally

OR

- Morphine,
- infants and children, 0.1-0.2mg/kg/dose 2-4 hourly IM, IV, SC or 0.2-0.5mg/kg/dose orally 4-6 hourly
- Adolescents, 3-4mg/dose (as necessary)
  - Anti-malarial:
    Artemisinin-based combination therapy (ACT): see section on malaria
- Detection and treatment of precipitating factors e.g. malaria, sepsis, dehydration etc.
- Supportive measures:
  - Counseling and health education
    - Membership of support groups
    - Regular health checks
- ***Notable adverse drug reactions, cautions and contra-indications:***
  - Paracetamol should be used with caution in patients with hepatic impairment
  - Opioid analgesics cause varying degrees of respiratory depression and hypotension. They should be avoided when raised intra-cranial pressure is suspected
  - NSAIDS can cause abdominal pain, gastrointestinal bleeding, ulceration and perforation. It should not be taken in an empty stomach

NEPHROLOGY

URINARY TRACT INFECTION

Introduction

UTI is a potential cause of renal scarring, calculi, ht, crf.

Can be associated with various anomalies and voiding dysfunction.

Urinary tract obstruction is a risk factor

Refers to the invasion of urinary tract by pathogenic organisms.

UTI include cystitis, pyelonephritis and asymptomatic bacteriuria

Breast feeding has been associated with reduced risk of UTI in child< 6months

Incidence varies according to the age and sex: 1-3% of girls. At <1 year, males > female with a ratio of 2.8-5.4:1 and at 1—2 years females > males at a ratio of 10:1

Neonates: 1-4%; M>F (preterms 2.9%,full term 0.7%)

Infants(1 month-1 year):1.1-1.2&(2%) M=F

>7yrs: 2.5% F>M

Clinical features:

Neonates: Non-specific - Vomiting, irritability, diarrhea, poor feeding, failure to thrive, dehydration

Infants and toddlers: fever may or may not

be present
Older children:
Frequency, urgency, dysuria, abdominal pain, enuresis, flank pain, hematuria

Investigations

Urinalysis: Best specimen for this and MCS is the supra-pubic aspirate.
Mid Stream Urine can also be used
Clean voided bag specimen in 2months – 2yrs

- WBC > 5HPF
- pH- proteus produces alkaline pH
- microscopic hematuria

Urine MCS: any colony count following a Supra Pubic Aspirate is diagnostic
100,000 cfil/ml – female
10,000 cfil/ml - male

Others :
Renal ultrasound
DMSA(dimecaptosuccinic acid)- parenchymal filling defect in acute pyelonephritis
It is superior to RUSS and IVU
MCUG: may be indicated in suspected anatomic anomaly e.g. reflux, PUV.
IVU: - produces information regarding precise anatomic image
- estimate renal function not reliable for detecting renal scarring or

pyelonephritis

- large dose radiation is required

Renal cortical scintigraphy

Complications

Immediate – bacteremia, dehydration

Late – chronic urinary tract infection, Renal scarring, Hypertension

Treatment

Goal:

To eradicate the causative organism and correct associated symptoms

specific – antibiotics for 10-14days

-Cotrimoxazole

Ceftriaxone

Cefixime

Amoxyl/clavulanic

Supportive - hydration, feeding, perineal education

NEPHROTIC SYNDROME

Definition

It is a clinical syndrome characterized by heavy protenuria , hypo albuminemia, oedema , hyper cholesteronemia.

Clinical features

Facial oedema, pedal oedema, anasarca, anuria/oliguria, frothy urine, hypertension in10% of cases

Complications

Congestive cardiac failure, pulmonary oedema, spontaneous bacteria peritonitis, renal vein thrombosis, acute kidney injury, myocardial infarction

Investigations

Urinalysis, 24hour urinary protein, serum protein (total and albumin), serum lipids, serum electrolytes, urea, creatinine, renal biopsy (when indicated)

Treatment

Objectives:

To reduce oedema, to reduce proteinuria, to correct lipedemia

Steroid at a dose of 60mg/m2/day single dose for 6-8weeks

Reduce to 40mg/m2 alternate days for 4 weeks and thereafter gradually taper off

Note that steroid is not indicated in ALL categories of nephrotic syndrome

Diuretics slow acting like thiazides at a dose of 1-2mg/day divided doses

Slow k at 1-2mg/kg/day, transfusion with 25% salt poor albumin/plasma at a dose of 10ml/kg

Low salt diet, high protein

Immunization with Pneumococcal conjugate vaccine

ACUTE KIDNEY INJURY

Introduction

A significant cause of morbidity (electrolyte derangements, disordered coagulation and endocrine dysfunction) and mortality in children.

Defined as a sudden, rapid and progressive deterioration in renal function resulting in the inability of the kidneys to perform its homeostatic function.

Manifested as a rise in plasma urea, creatinine and accompanied by oliguria and occasionally polyuria .

Recently referred to as acute kidney injury with RIFLE staging (RISK INJURY FAILURE LOSS END STAGE). Serum creatinine and urinary output are parameters used, the worst of the two being used to stage.

Key words

Oliguria: reduction in U.O <= 300ml/m^2 or <1ml/kg/hr

Anuria: reduction in U.O to < 1ml kg/day

Polyuria: urine output > 4ml kg/hr

Azotemia: high nitrogeneous waste indicated by high urea

Uraemia: symptom complex reflecting organ dysfunction occurring when the kidney fails to regulate body composition

Cause of renal injury:

Pre-renal,
Intrinsic renal
Post renal.
Commonest causes in pediatrics are often pre-renal and are due largely to preventable causes
least common cause is post renal.
50% AKI in children are non oliguric
Incidence is difficult because of non standardized definition .
Seen in 5% of children admitted into ICU.
Reported rates have been between 3.13% to 57.9%
Pre renal: Mainly due to volume loss – diarrhoea, vomiting, haemorrhage
Intrinsic : Native disease of the kidney – acute glomerulonephritis, nephrotic syndrome,
complicated malaria, septicaemia
Post renal: usually due to obstruction – posterior urethral valve, urethral stricture, pelvi- ureteric junction obstruction, vesico –ureteric junction obstruction, neurogenic bladder

Clinical features
Fluid retention, oliguria, anuria, oedema, dyspnea, hypertension →convulsion, congestive cardiac failure, pallor, acidotic breathing
Features of underlying disease

Investigations

Complete blood count, malaria parasite, serum electrolyte urea, creatinine

Others as indicated may include MCUG

Treatment

Objectives:

To prevent progression of failure, to rapidly restore volume in pre-renal cases, to treat the underlying cause

Fluid management, blood transfusion in case of acute loss, relieve obstruction, dialysis

Anti malarial

Antibiotics

CHRONIC RENAL FAILURE

Introduction:

A situation of an irreversible reduction in GFR with a prevalence at 18 per million and may be congenital or acquired

KDQI defines chronic kidney disease (CKD) as a kidney damage with GFR of <60ml/m2/1.73m^2 for >= 3 months.

Whatever is the kidney cause, there is irreversible sclerosis and loss of nephrons which leads to reduction in GFR

Clinical staging

Stage 1- kidney damage with normal GFR > 90ml/min

2- mild reduction in GFR 60-69

ml/mm/1.73m^2

3- mod reduction in GFR 30-59 ml/mm/1.73m^2

4- severe " " " 15-29 " " " "

5- kidney failure GFR < 15ml/mm

Different formulae for GFR but Schwatz is the most commonly used :

Ht x k(constant)Kis age and sex dependent SCR

The cause is age dependent: <5yrs commonly from anatomic anomalies and for > 5yrs, acquired glomerular dx Glomerulonephritis, Urinary Tract Obstruction, REFLUX, Congenital anomalies and urinary tract infection are leading causes

Clinical features

Non-specific: headache, fatigue, lethargy, anorexia, vomiting, growth failure

Specific: anaemia, oliguria, anuria, puffiness

Investigation

RUSS, renal radionucleid scan, CT scan, MRI, VCUG, urinalysis, spot urine for total protein : cr, 24 HUP -total protein, creatinine clearance, serum complements C3, anti GBM antibodies, HBV, HCV, Retroviral screening, serum electrolyte, urea

and creatinine, serum Ca, phosphate, uric acid, complete blood count

Treatment

Goal is to delay progression if possible, treat pathologic manifestation, renal transplantation

Treatment of underlying conditions like UTI, Glomerulonephritides; control of BP with ACE inhibitors which also delay progression through the reduction of protein excretion; control of lipidemia; avoid nephrotoxins

For Anaemia-

- Avoid blood Transfusion – do not initiate transfusion until PCV is <30% and aim to maintain PCV at 30%;
- Treat Fe deficiency either orally or parenterally;
- Erythropoetin either SC or IV ;
- Electrolyte; diet – protein restriction

ACUTE GLOMERULONEPHRITIS

Introduction

A disorder of structure and or a functional anomaly.

Of an abrupt onset with a tendency to spontaneous recovery

May be acute or chronic.

Chronic type is a common cause of ESKD in

children.
Could be caused by post streptococcal glomerulonephritis, diffuse proliferative glomewrulonephritis, mesangial proliferative glomerulonephritis, focal segmental glomerulosclerosis, membranous glomerulopathy, rapidly progressive glomerulonephritis, systemic illness, SLE, Henoch Schonlein Purpura, HBV, poly arteritis nodosa
The commonest cause in this environment is post streptococcal

Clinical features
History of passage of dark smoky urine, preceding sore throat, skin infection, facial or pedal oedema, seizure, oliguria, anorexia
Physical examination - Hypertension, oedema, respiratory distress in presence of severe fluid accumulation, seizures, hypertensive encaphalopathy

Diagnostic Criteria
Gross hematuria, hypertension, oliguria are the hall mark of diagnosis

Complication
Hypertensive encephalopathy, acute kidney injury, hyperkalemia, -fluid overload, congestive cardiac failure

Investigation:

Urinalysis:

- pH-acid
- colour-dark smoky urine
- Red cell casts commonly may be absent and repeated urinalysis of fresh urine may be needed

leucocyte (pyuria)

- waxy –suggest pre existing nephritis
- pr- +,2+ not massive<500mg/dl
- non selective pr (>0.2 IgG/alb)
- Na^+, $Ca2^+$ reduced
- FeNa <0.5% Electrolyte, urea and creatinine.

Throat Swab-:

grp A strept organism

ASO >200 todds unit

Streptozyme:-

Complement: -C3

FBC - anaemia-

Treatment

Goal:

Treatment of hypertension, eradication of organism

Hypertension: Tabs aldomet at a dose of 10mg/kg /dose in divided doses

ACE inhibitors like Lisinopril

Diuretics e.g. thiazides at 1-2 mg/kg /day in 2 divided doses

Salt restriction

Diet: normal protein with 60-70% high biologic value but reduced to 0.6-1gm/kg in renal failure

Organism: Oral penicillin at a dose of 100mg/kg/day divided doses

Cephalosporins

Erythromycin a dose of 40-50mg/kg/day divided doses when there is penicillin allergy

PNEUMONIA

Introduction

Pneumonia accounts for 15% of all under-five deaths (including neonatal death due to pneumonia) in Nigeria, the highest in Africa.

Burden of disease mainly in the younger age groups;

81% of deaths from pneumonia in children less than 2 years.

Male to female ratio is 1.5:1.

Classified clinically as lobar pneumonia or bronchopneumonia

Clinical Features

Cough

Fast breathing.

Fever is very common in childhood pneumonia.

Vomiting

Poor feeding
Diarrhea
Convulsion
Chest pain (due to pleuritis) in older children.
Tachypnoea is a sensitive marker of pneumonia and is commonly present and/or difficult breathing
Dull or resonant percussion note on percussion
Bronchial breath sounds and or crepitations on auscultation
For neonates:

- Fever or hypothermia
- Poor feeding
- Vomiting
- Lethargy or irritability
- Abdominal distension
- Convulsion
- Jaundice
- Tachypnoea (≥ 60 breaths/min)
- Tachycardia etc

Complications

Complications can be acute or chronic.

Acute:

- Heart failure
- Pleural effusion
- Empyema
- 3 Ps: Pneumatocoele, pneumothorax, pyopneumothorax, pyopneumothorax

- Atelectasis
- Septicaemia
- Acute respiratory failure

Chronic:

- Lung abscess
- Bronchiectasis

Diagnostic Criteria

Gold standard' for diagnosis is chest radiography,(although does not reveal the aetiology.)

Investigations

Chest radiography

Blood culture to determine the bacterial aetiology

Full blood count will determine anaemia and *suggest* a bacterial (polymorphonuclear leukocytosis) or viral aetiology (lymphocytosis).

Electrolye and urea, may show hyponatraemia and azotaemia especially in those children with accompanying diarrhea and vomiting, and poor feeding.

Treatment

Clear the airway using gentle suction

Supplemental oxygen if oxygen saturation is less than 90% in room air or signs of severe respiratory distress are present. If pulse oximetry is not available give oxygen if

signs of respiratory distress and or cyanosis are present.

- Give oxygen via nasal prongs or nasal catheters: 0.5-1L/min for children 0-2months, 2-3L/min for children 3 months to 5 years; maximum of 4L/min for older children)

Allow small frequent feeds if tolerated; feeding may also be done using nasogastric tube

If feeds are not tolerated give intravenous isotonicfluid. Ensure it contains at least 5% glucose (e.g. 5% dextrose in 0.9% saline or Ringer's lactate with added glucose)

Nursing care should be provided at least every 3 hours: check vital signs including oxygen saturation

The doctor should review the child at least twice each day

For high grade fever (temperature ≥39°C), give paracetamol 10-15mg/kg 4-6 hourly or ibuprofen 6mg/kg. If widespread wheeze is present (high-pitch musical sound during expiration only or during both phases of respiration) give first dose of short acting bronchodilator such as salbutamol or albuterol and re-assess.

The National Guideline on the antibiotictreatment of community-acquired pneumonia in under-5s is summarised below

Category of children	Outpatients		Inpatients	
	First line	Alternatives*	First line	Alternatives*
<2 months	Admit and treat as neonatal sepsis			
months	High dose Oral amoxicillin (45mg/kg per dose 12 hrly) for at least 5 days	Oral amoxicillin-clavulanic acid (amoxicillin component 45mg/kg per dose 12 hrly) OR oral cefpodoxime (5mg/kg per dose 12 hrly) OR oral cefuroxime (10-15mg/kg per dose 12 hrly) for at least 5 days	IV amoxicillin (50mg/kg every 8 hrs) AND IV/IM genticin (5-7.5mg/kg once daily)	IV cefuroxime (50mg/kg 8 hrly) AND IV/IMgenticin (5-7.5mg/kg once daily) for at least 5 days OR IV ceftriaxone (50-100mg/kg/day every 12-24hours). OR IV cefotaxime (25-50mg/kg 6 hrly). OR IV/IM genticin (5 -7.5mg/kg once daily) AND IV cloxacillin (25-50mg/kg 6 hrly) for at least 5 days.
HIV-infected children	High dose Oral amoxicillin (45mg/kg per dose 12 hrly) for 10 days	Oral amoxicillin-clavulanic acid (amoxicillin component 45mg/kg per dose 12 hrly) OR oral cefpodoxime (5mg/kg per dose 12 hrly)	IV amoxicillin (50mg/kg every 8 hrs) AND IV/IM genticin (5-7.5mg/kg once daily) PLUS high dose co-trimoxazole (5mg/kg e	IV cefuroxime (50mg/kg 8 hrly) AND IV/IM genticin (5-7.5mg/kg once daily) for at least 5 days OR IV ceftriaxone (50-100mg/kg/da

Notes:

1. Step down to appropriate oral antibiotics when improvement is sustained. For instance, cefpodoxime after ceftriaxone.
2. Target pathogens in outpatients' treatment are *Streptococcus pneumoniae* and *Haemophilus influenzae* type B; whereas in cases on admission, these as well as *Staphylococcus aureus* and other bacilli are included.
3. Maximum dose of gentamicin should not exceed 120mg.

*Alternatives: Consider alternatives when first line drugs are not available or applicable or child has not responded to the first line drugs.

NEONATOLOGY

POST-RESUSCITATION CARE OF ASPHYXIATED BABIES

Clinical manifestations (This depends on the severity and degree of hypoxic -Ischaemic encephalopathy)

- Inability to cry or suck, global hypotonia and poor activity or global hypertonia with brisk deep tendon reflexes, shock, poor temperature regulation, paralytic ileus, respiratory distress, bleeding tendencies, seizures,

oliguria

Management

Largely anticipatory.

Serial monitoring of parameters such as: oxygen saturation, via pulse oximetry, random blood glucose, serum electrolytes, urea and creatinine, serum calcium, serum troponin, serum aspartate aminotransferase, serum alanine aminotransferase should be done where facilities are available.

Derangements should be appropriately corrected according to standard protocols:

- Prophylactic phenobarbitone by slow intravenous administration (even in the absence of seizures) 10-15mg/kg loading dose and maintained with 2.5mg/kg 12-hourly till neurologic functions are restored.
- Seizures should be managed with slow intravenous phenobarbitone (loading dose of 15mg/kg over 15-20 minutes to be maintained with 2.5mg/kg 12 hourly. The dose should be tailed off over many days after serial neurologic physical examination and electroencephalography have consistently shown normal parameters.
- Fluid restriction in the management of perinatal asphyxia is no longer favored because of the existing risks of renal insufficiency coupled with the uncertain

risk of the Syndrome of Inappropriate ADH Secretion. Efforts should be to prevent over-hydration rather than fluid restriction. Administer the exact daily maintenance fluid requirement.

- Apnea should be managed with frequent airway clearing and ventilation by bag and mask.
- *Nil per os* should be maintained until peristalsis is present and respiratory rate is normal. Feeding should be instituted even when the sucking reflex is still depressed with the use of expressed breast milk administered via a nasogastric tube. The volume of milk to be administered will depend on the daily maintenance fluid requirement.

Outcome.

The persistence of abnormal cry, abnormal motor functions and primitive reflexes beyond the first week of life highly suggests severe cerebral damage.

The risk of long term neurologic deficits has been shown to be minimized with the use of interventions such as magnesium sulphate and selective cerebral cooling.

APNEA

Introduction.

Cessation of breath for more than 15 seconds with bradycardia and cyanosis.

There may also be hypotonia or pallor in severe cases.

Particularly common among very preterm infants with estimated gestational age of < 32 weeks. It may also occur among sick term babies.

Prematurity and immaturity of the central respiratory centre causes a separate entity known as "Apnea of Prematurity".

Other causes include septicaemia, pneumonia, necrotizing enterocolitis, gastro-oesophageal reflux, congestive cardiac failure, meningitis, intra-cranial haemorrhage, seizures, hypoglycaemia, severe anaemia and urinary tract infection.

Management

The first step in the management of an apneic infant is to provide tactile stimulation such as flicking the sole of the feet. If there is no response to tactile stimulation, the following steps are recommended:

- Resuscitation: airway clearance, assisted breathing and circulation according to standard protocols.
- 10% Dextrose-in-Water (4ml/kg) for the correction of blood glucose.
- Start any of the following depending on availability:

methyl-xanthine.

Caffeine citrate in a loading dose of 20mg/kg

oral or slow intravenous and maintenance of 5mg/kg per 24 hours is the drug of choice.
Alternative drugs include aminophylline in a loading dose of 6mg/kg slow intravenous and maintenance of 2.5mg/kg 12-hourly.

- Apnea monitor should be used if available or a pulse oximeter with an alarm system turned for hypoxaemia may be a good substitute.
- If apnea persists, nasal Continuous Positive Airway Pressure (CPAP) is recommended at 4-5cm H_2O. If apnea still persists with persisting cyanosis, intubation and subsequent commencement of Intermittent Positive Pressure Ventilation is recommended.
- For preterm infants, the medications may be continued until the infant attains the post-conceptional age of 33-34 weeks. For other infants, medications may be continued till the infant no longer requires oxygen therapy or has been free of apnea for at least 5 days.
- Nursing in the prone position with cessation of oral feeds may be helpful if apnea becomes recurrent and gastro-oesophageal reflux is highly suspected.

HYPOGLYCAEMIA

Introduction.

Whole blood glucose level of < 2.6 mmol/l irrespective of age and gestational age.
Most infants with mild to moderate

hypoglycaemia are asymptomatic.
Normal glucose utilization rate is 4-6mg/kg/minute but high-risk infants require 6-10mg/kg/minute. In the extreme cases of hyperinsulinism, the infants utilize glucose at a rate greater than 10mg/kg/minute.
The following predispose to it: prematurity, intra-uterine growth restriction, hypothermia, perinatal asphyxia, polycythaemia, septicaemia, maternal diabetes, in-born errors of metabolism – galactosaemia, glyogen storage diseases – liver diseases, haemolytic disease of the newborn, Beckwith-Wiedemann syndrome, nesidioblastosis and other pancreatic tumours.

Clinical features
Mild cases of hypoglycaemia may be asymptomatic.
Symptoms include diaphoresis, irritability, hypotonia, lethargy, apnea and seizures.

Investigation.
Screening for hypoglycaemia is mandatory for all high-risk infants (EGA <37weeks, Weight <2.5kg, SGA, infants of diabetic mothers, infants of mothers on β-blockers, infants with cold-stress).
The diagnosis of hypoglycaemia should be confirmed using test strips which are not

affected by haematocrit, oxygen saturation or bilirubin level.

These modern strips usually provide blood glucose values very close to values obtained by spectrophotometery.

Management

- Intravenous 10% Dextrose-in-Water should be administered at the rate of 60ml/kg/day on the first day. RBG should be checked at 1hour after commencement of infusion, 6 hourly for the first 24 hours and thereafter, 12 hourly.
- If RBG is < 2.6 mmol/l and the infant is asymptomatic, it is important to either increase the 10% Dextrose-in-Water drip rate by 50% or increase the concentration of dextrose to 15%. A central vein should be used to administer dextrose concentration of >12.5%. Urine should be monitored for glycosuria when high concentration of glucose is infused. As RBG normalizes, the concentration of dextrose infusion should be gradually reduced back to maintenance concentration of either 4.3% or 8%.
- Enteral feeding should be continued if the infant can tolerate oral feeding.
- If RBG is < 2.6 mmol/l and drowsiness, seizures or coma are present, bolus of 4ml/kg of 10% dextrose should be

administered and maintenance infusion should be done at the rate of 8 mg/kg/minute. RBG should be checked after 1 hour. If RBG is >2.6 mmol/l, infusion rate should be reduced to 5mg/kg/minute and RBG should be checked after another 1 hour. If RBG is < 2.6 mmol/l and the infant is asymptomatic, the infusion should be maintained and RBG checked in 3 hours. However, if RBG is < 2.6 mmol/l and symptoms persist, the infusion rate should be increased to 12mg/kg/minute and RBG should be repeated in 1 hour. If RBG increases, glucose infusion rate should be reduced slowly over 4-6 hours.

- If hypoglycaemia persists despite 12mg/kg/minute
  serum insulin should be measured and intravenous hydrocortisone 2.5-5mg/kg should be given 12 hourly.
  If this step is ineffective, oral diazoxide 1.7-5 mg/kg 8 hourly with oral chlorthiazide 10mg/kg 12 hourly should be administered with close monitoring for hypotension and dehydration.
- In the extreme cases requiring more than 20mg/kg/minute of glucose, subcutaneous somatostatin octreotide 1µg/kg 4 hourly or intramuscular glucagon 20µg/kg 6 hourly may be effective.

- If hypoglycaemia persists with hyperinsulinism, partial pancreatectomy is indicated.

HYPERGLYCAEMIA

Introduction

Random blood glucose of > 8.0 mmol/l. Preterm infants particularly when treated with 10% dextrose infusion or total parenteral nutrition are prone to it. Hypothermia, sepsis, post-operative cases, neonatal diabetes (transient or persistent) are also associated.

Clinical features

Persistent dehydration with glycosuria. Transient diabetes is suspected among infants with wasting or intra-uterine growth restriction who develop hyperglycaemia whilst on enteral feeds.

Management

- The concentration of dextrose in infusion should be reduced.
- Insulin 0.05-0.1 iu/kg/hour should be commenced if RBG remains above 10 mmol/l with glycosuria despite reduction of glucose concentration in infusion.
- Treat the identified cause if possible.

VITAMIN K DEFICIENCY BLEEDING

Introduction

Formerly known as the Haemorrhagic Disease of the Newborn.

It is due to the deficiency of the clotting factors II, VII, IX and X.

In the newborn, the levels of the clotting factors are naturally low and coupled with low Vitamin K production, the newborn infant has a natural tendency for haemorrhage.

Both term and preterm infants are affected but the risk is higher among preterm infants. Infants of mothers on anti-convulsant or anti-coagulant therapies and infants on prolonged exclusive breastfeeding are also at higher risk of the disease.

Clinical Presentation

This disease in the classical type presents between the second and fourth days of life. The early type presents within the first 24 hours of life while the late type occurs as late as three to six weeks of life.

Classical features include oozing from puncture sites or circumcision sites, umbilical stump haemorrhage, haematemesis and or melaena, ecchymoses, epistaxis or scalp haemorrhage, intra-viscera haemorrhage (kidney, brain) may also occur in severe cases.

Investigations

- Prothrombin time – very prolonged

- Activated partial thromboplastin time – prolonged
- Thrombin time – normal
- Fibrin degradation products – normal
- Platelet count - normal

Management

Once haemorrhage occurs, intramuscular injections should be avoided to prevent haematoma formation. Intravenous Vitamin K_1 (1-3mg) should be administered daily for up to 3 days.

- Fresh Frozen Plasma should be administered along with therapeutic doses of Vitamin K_1 because the corrective action of Vitamin K_1 is usually delayed.
- If bleeding is severe and baby is shocked, immediate replacement transfusion with O Rhesus-negative whole blood (20ml/kg) is indicated. This is preferably done through a central vein (umbilical vein) over 20-30 minutes. Important supportive care includes oxygen therapy and frequent monitoring of oxygen saturation and central venous pressure.
- In the absence of shock, gradual correction using single-volume exchange blood transfusion (80ml/kg) is important. This may be followed up with top-up transfusion (10ml/kg of packed cells or 15ml/kg of partially packed cells)

in case the haematocrit remains low.

NECROTIZING ENTEROCOLITIS

Introduction

This refers to extensive necrosis of the intestine of multi-factorial origin. It may ultimately result in intestinal perforation. It commonly affects the terminal ileum and proximal colon but it may involve the entire length of the gut.

Prematurity and very low birth weight, early infant formula feeding, intra-uterine growth restriction, polycythaemia , septicaemia, umbilical catheterization, congenital heart diseases can predispose to it.

Clinical features

Grading

This is different from the Bell's classification but it involves clinical, laboratory and radiologic features. This grading is useful for prognostication and management planning.

Grade 1 (Better Prognosis)

Feed intolerance, abdominal distension, bilious vomiting or gastric aspirate, haematochezia or malaena, systemic illness – lethargy, hypotonia, apnea – plain abdominal X-Ray shows gaseous bowel distension or presence of gas within the bowel wall (pneumatosis intestinalis).

Grade 2 (Worse Prognosis)

In addition to features of Grade 1, cases also have:
Abdominal tenderness and rigidity (evidence of perforation), abnormal or spontaneous bleeding, shock, leucopenia, thrombocytopaenia, pneumomediastinum or portal vein gas.

Investigations.
- Full blood count
- Plain abdominal X-Ray
- Random Blood Glucose
- Serum electrolytes, urea and creatinine
- Blood culture

Management
- The baby should be put on *Nil per Os* (NPO) and while Total Parenteral Nutrition (TPN) is instituted. In the alternative, intravenous fluid therapy can be used to meet the maintenance fluid, caloric and electrolyte requirements.
- Abdominal girth should be monitored as progressive increment may indicate intestinal perforation.
- Insert a nasogastric tube to decompress the stomach and for regular aspiration.
- Antibiotics are administered intravenously: a triple regimen of cephalosporin (ceftriaxone, cefotaxime or ceftazidime – 100mg/kg/day), gentamicin – 5mg/kg/day (or

kanamycin) and
metronidazole 7mg/kg 8-hourly.

- Serial plain abdominal X-Ray (supine and right lateral) is useful in monitoring the progress of the disease
- Thrombocytopaenia and deranged coagulation profile should be corrected with the appropriate blood product available.
- Shock is managed using crystalloids or colloids and inotropes. Urinary output must be monitored to confirm the success of anti-shock therapy.
- If diagnosis is confirmed (with X-Ray findings), antibiotics and *nil per os* are continued for 7 – 10 days but if diagnosis remains unconfirmed and baby recovers quickly, gradual oral feeds may be re-introduced after 48 hours while antibiotic therapy continues for 5 days.
- Surgery is indicated by (i) clinical deterioration (ii) intestinal perforation
- Follow-up Care should anticipate strictures presenting with intestinal obstruction.

JAUNDICE

Introduction

This refers to the yellowish discolouration of the sclera, skin and mucous membranes as a result of excessive accumulation of bilirubin in the blood. It is clinically visible at total

serum bilirubin (TSB) level of 5mg/dl (85μmol/L). Close to 60% of term and 80% of preterm infants develop jaundice within the first week of life. Jaundice can be physiologic or pathological.

Physiologic jaundice is characterized by the following:

Usually a diagnosis of exclusion, appears after 36 hours of life, peaks at the 5th day of life with total serum bilirubin (TSB) of 12mg/dl (205 μmol/L) for term babies or 15mg/dl (255 μmol/L) for preterm babies, conjugated bilirubin of > 2mg/dl (34 μmol/L), jaundice clears spontaneously by the 7th day of life for the term infants and between the 10th and 14th days for the preterm infants.

Pathological jaundice is characterized by:

Jaundice observed within the first 24hours of life, jaundice lasting more than 14 days in term infants and 21 days in the preterm infants (this is known as Prolonged Jaundice), jaundice with TSB >12mg/dl (205 μmol/L), jaundice with fever and other signs of sickness, conjugated bilirubin more than 2mg/dl (34 μmol/L) or deep yellow urine.

Clinical assessment of infants with jaundice:

- Note should be taken of risk factors: preterm birth, history of jaundice in the siblings, history of exclusive

breastfeeding, small size <2.5kg at birth, evidence of haemolysis, sepsis.

- Examine the baby close to the window or under very bright light.
- Examine the sclera for yellowish discoloration.
- Other examination sites include the gum, the tip of the nose, the upper chest wall, the lower abdominal wall, the palms and soles. Using the tip of the finger, slight pressure is applied to these sites for up to 10 seconds and when the finger is lifted, the yellowish discolouration underneath the blanched skin becomes obvious.

Investigations

- Serum bilirubin (only TSB is essential in the first week of life; split bilirubin – total, unconjugated and conjugated- is only required in cases of prolonged jaundice)
- Trans-cutaneous Bilirubin (TcB) estimation is reliable as it correlates well with serum bilirubin except in severe cases of hyperbilirubinaemia. Therefore, this can be reliably used when serum TSB is not available.
- Blood typing (for mother and baby)
- Full Blood Count
- Peripheral blood film examination
- Coomb's test
- G6PD assay or screening
- Thyroid Function Tests

- Hepatobiliary scan

Management.

The goal of treatment is to rapidly reduce serum bilirubin levels and prevent bilirubin encephalopathy.

- Blue light phototherapy delivering irradiance from a distance of 30cm from the baby in a cot or incubator. Important steps during phototherapy: (a) infant should be nursed naked except for diaper (b) infant must be blind-folded (c) body temperature to be monitored 4-hourly (d) turning of infant every 2-4 hours (e) TSB must be monitored 12-hourly and at worst, on a daily basis.
- Double-volume Exchange Blood Transfusion (EBT) using 160-170ml/kg of compatible fresh whole blood fresh. This procedure should be carried out over at least 2 hours using a three-way valve. Infants undergoing EBT should be maintained on phototherapy to minimise rebound hyperbilirubinaemia.
- All babies with visible jaundice within the first 24 hours of life must have phototherapy until a diagnosis is made.
- Intensive phototherapy is achieved with further reduction of the distance between baby and light source and increased irradiance from multiple directions. The use of Bili-blanket serves this purpose

well especially when it is combined with the conventional phototherapy.

- Phototherapy should be continued until Total Serum Bilirubin is 3-5mg/dl lower than the threshold range for the age or until the jaundice has cleared significantly.
- Without obvious sepsis, antibiotics are **not** indicated in the treatment of neonatal jaundice. If fever is present, results of relevant tests should guide treatment. Note: Ceftriaxone and sulphur-containing antibiotics or antimalarial drugs should be avoided because of the risk of displacement of bilirubin from albumin-binding sites.
- Breastfeeding should be increased during care for jaundice. If this is difficult, intravenous fluid should be administered with extra 10ml/kg added to the daily maintenance fluid requirement. Both caloric intake and hydration enhance the excretion of bilirubin.
- Phenobarbitone is not recommended because it is slow-acting and not effective for jaundice cases characterized by rapidly rising TSB.

SEPSIS

Introduction

This is a clinical syndrome resulting from bacterial blood stream infection with active proliferation of the organisms.

Early-Onset sepsis manifests within the first 48 hours of life or at most 7 days of life while Late-Onset sepsis manifests after 7 days of life.

Terminologies.

- Early-Onset sepsis is presumed when risk factors for sepsis are present but the infant has no clinical feature suggestive of sepsis.
- In probable sepsis, an infant has clinical and/or laboratory features suggestive of sepsis without bacteriological confirmation.
- Sepsis is latent when clinical features are present with laboratory features such as deranged full blood count or elevated serum C-Reactive Proteins and serum Interleukins but without positive blood culture.
- Sepsis is confirmed when blood culture is positive in addition to clinical manifestations.

Clinical Features

Non-specific features include fever, vomiting, poor feeding, poor activity

More specific features include hypothermia, poor skin color, jaundice, abdominal distension, respiratory distress, apnea and bleeding tendencies.

There may be local manifestations of serious illnesses such as omphalitis, otitis media, pneumonia, diarrhoea or urinary tract infestation.

Investigations

Anaemia, leucopaenia, thrombocytopaenia, elevated C-Reactive Protein, elevated procalcitonin, elevated interleukins 1,6,8 and positive blood culture. Others will include specific ancillary tests for localized diseases such as chest X-Ray, mid-stream urine and other swabs for bacteriological studies.

Management

- For presumed sepsis in the developing world, it is recommended that blood culture should be requested and the infant is immediately commenced on empirical antibiotic therapy based on the local pattern of organisms and their sensitivity pattern. Intravenous cefuroxime 50mg/kg 12-hourly and gentamicin 2.5mg/kg 12-hourly. The infant should be closely monitored for any evidence of clinical deterioration. If the infant remains asymptomatic and the blood culture yields no growth, the antibiotics can be safely discontinued after 48 hours.
- For probable sepsis, blood culture should be requested while the infant is

commenced on antibiotics: intravenous second or third generation cephalosporins; Cefuroxime 50mg/kg 12-hourly or Ceftriaxone 75mg/kg daily or Cefotaxime 50mg/kg 8-hourly and gentamicin 2.5mg/kg 12-hourly for 10 to 14 days depending on the clinical response. It may be necessary to change the antibiotics if the sensitivity report from the laboratory suggests resistance to the drugs in use.

- Support care: Fluid and caloric balance, dextrose for hypoglycaemia, blood transfusion for severe anaemia, oxygen therapy and ventilators supports for hypoxaemia.

BACTERIAL MENINGITIS

Introduction

This refers to inflammation of the lepto-meninges as a result of bacterial infection. This condition occurs more frequently in Late-Onset Sepsis compared to Early-Onset Sepsis. It is associated with high mortality because early diagnosis is usually difficult since the early features are non-specific.

Clinical Features

The early, non-specific features include: fever, vomiting, poor feeding and poor activity.

The late features are more specific and these

include: hypothermia, tone abnormalities, particularly, hypertonia, high-pitched cry, setting-sun eye appearance, opisthotonus, bulging and tense anterior fontanelle, seizures, apnea and altered sensorium.

Investigations

- A lumbar tap for cerebrospinal fluid analysis is mandatory. Skip it if the infant has cardio-respiratory instability. Inability to carry out this important investigation must not be a reason to delay empirical antibiotic therapy. Meningitis is diagnosed with cell count greater than 30/mm^3, pleocytosis with polymorphonuclear cells, protein greater than 150mg/dl and glucose less than 50% of simultaneously assayed blood glucose.
- Neuro-imaging studies are required if: (a) fever persists or recurs (b) seizures recur after initial control (c) occipitofrontal circumference is increasing. It will be necessary to exclude subdural and intra-cerebral collections.

Treatment.

- The empirical antibiotic therapy should be tailored to the local pattern of bacterial aetiology of neonatal meningitis.
  Staphylococcus aureus, Streptococcus pneumoniae and the Gram-negative bacilli are frequently encountered in Nigeria.

Therefore, the recommended antibiotics include a combination of intravenous third generation cephalosporin: ceftriaxone 50mg/kg 12-hourly or cefotaxime 50mg/kg 6-hourly and gentamicin 2.5mg/kg 12-hourly.

Intravenous ampicillin 50mg/kg 6-hourly may be useful in places where resistance of organisms to the drug is not remarkable and in known cases of *Listeria monocytogenes* infection.

The duration of antibiotic therapy is 14 days for Gram-positive organisms and 21 days for Gram-negative organisms.

- Phenobarbitone is recommended through the intravenous route for seizures; 15mg/kg loading dose followed up with 2.5mg/kg 12-hourly maintenance doses. The loading dose could be repeated at 10mg/kg when seizures are difficult to control.
- Fluid therapy: The current body of evidence is not in favour of fluid restriction for all infants. Critically-ill infants tend to be dehydrated from poor feeding or vomiting and fluid restriction is likely to be harmful – risk of shock and renal shut-down – In such babies. More important is the need to avoid over-hydration (in lieu of the risk of cerebral edema) and that could be achieved by stringent administration of not more than

the recommended maintenance fluid requirement.

- Corticosteroids: Unlike in older infants and children, corticosteroids are presently not recommended for use in neonatal meningitis for lack of evidence of usefulness.
- Support care: Fluid and caloric balance, blood transfusion for severe anaemia, oxygen therapy and ventilators supports for hypoxaemia.

MALARIA

Malaria in the newborn period masquerades as sepsis in most instances as the clinical features are very similar.

Investigations

- Rapid Diagnostic Tests for rapid detection of *Plasmodium falciparum* in situations of poor laboratory services
- Peripheral blood film microscopy with Giemsa staining which most frequently reveals *Plasmodium falciparum.*
- Full blood count: anaemia and leucocytosis.
- Blood culture is expected to be negative.

Management

- Every symptomatic infant and asymptomatic infants in whom parasitaemia persists beyond 72 hours of life should be treated. Due to the extreme

similarities in the clinical manifestations of malaria and sepsis in the newborn, it may be safer to start acutely ill babies with probable malaria on anti-malarial drugs as well as antibiotics until sepsis could be confidently excluded.

- Oral quinine 20mg/kg stat, followed with 10mg/kg 8 hourly for 5 to 7 days is recommended.
- Oral amodiaquine as 25mg/kg total dose administered as 10mg/kg/day on the first two days and 5mg/kg on the third day may be used when quinine is contraindicated.

In areas of high resistance to the 4-aminoquinolines, amodiaquine may not be useful.

Treat infants weighing <5 kg with uncomplicated *P. falciparum* malaria with ACT at the same mg/kg bw target dose as for children weighing 5kg with close monitoring of response

BLOOD TRANSFUSION IN THE NEWBORN

Introduction

Indicated by:

- Acute haemorrhage
- Anaemia

For haemorrhage, the decision to transfuse should be based on the infant's haemodynamics – pulse, blood pressure –

and not on haematocrit which is usually normal immediately after haemorrhage. Cross-match is desirable only if the condition is not life-threatening, otherwise fresh Group O Rhesus negative must be obtained for urgent transfusion.

For anaemia, transfusion is recommended when: PCV is < 36% and infant is critically ill; PCV is < 30% and infant is stable or surgery is urgently needed; cardiac failure is present and can be attributed only to anaemia.

When severe anaemia occurs within the first week of life, single volume exchange blood transfusion with 80ml/kg of fresh cross-matched blood is recommended.

Complications

- Transfusion reactions are extremely rare in the newborn.
- Risk of transmission of infections (HIV, Hepatitis B Virus, Hepatitis C Virus, CMV) is germane
- Hyperkalaemia, Hypocalcaemia, acidosis, hypothermia.

Circulatory overload is common. The recommended measures include slowing of the drip rate and intravenous frusemide 1mg/kg.

CHAPTER 20:

EMERGENCIES

ACUTE LEFT VENTRICULAR FAILURE

Introduction

Sudden diminution in the function of the left ventricle. Pulmonary capillary and venous pressure increase beyond plasma oncotic pressure. There is resultant accumulation of oedema fluid in the pulmonary interstitial spaces and alveoli

Aetiology

Hypertension
Arhythmias
Myocardial infarction

Clinical features

Dyspnoea
Orthoponea
Paroxysmal nocturnal dyspnoea
Cough
Heamoptysis
Restlessness
Wheezes
Hypoxia

Differential diagnoses
Pulmonary thromboembolism
Bronchial asthma
Cardiac tamponade

Complications
Acute renal failure
Right-sided heart failure

Investigations
Electrocardiography
Plain chest radiograph
Echocardiography
Cardiac catheterization
Pulmonary function tests
Arterial blood gasses
Electrolyte, Urea and Creatinine

Treatment objectives
To improve pump performance of the failing ventricle
To reduce the cardiac workload
To control salt and water retention

Non-drug treatment
As in hypertension

Drug treatment
Diuretics
- Furosemide

Adult: 40 - 80 mg by slow intravenous injection stat

- Then 40 - 160 mg orally or intravenously daily in 1or 2 divided doses for maintenance

Child: neonate, 0.5 - 1 mg/kg by slow intravenous injection every 12 - 24 hours (every 24 hours if post-menstrual age is under 31 weeks) 1 month - 12 years: 0.5 - 1 mg/kg (maximum 4 mg/kg), repeated every 8 hours as necessary 12 - 18 years: 20 - 40 mg every 8 hours; higher doses may be necessary in resistant cases Angiotensin converting enzyme inhibitors

- Captopril

Adult: 6.25 - 12.5 mg daily orally, then 25 mg in divided doses daily (maximum 150 mg daily) for maintenance

Child: not licensed for use in children

Or:

- Lisinopril

Adult: 2.5 mg orally daily; 5 - 20 mg daily for maintenance

Child: neonate, initially 10 micrograms/kg orally once daily; monitor blood pressure carefully for 1 - 2 hours, increased as necessary up to 500 micrograms/kg daily in 1-3 divided doses

1 month - 12 years: initially 100 micrograms/kg orally once daily, monitor

blood pressure carefully for 1 - 2 hours, increased as necessary up to a maximum of 1 mg/kg daily in 1-2 divided doses
12 - 18 years: initially 2.5 mg daily, monitor blood pressure carefully for 1 - 2 hours; usual maintenance dose 10 - 20 mg daily in 1 - 2 divided doses (maximum 40 mg daily if body weight is >50 kg) May require morphine
Adult: 5 - 10 mg orally, subcutaneously or intramuscularly (usually a single initial dose)
Child: not listed for this indication Digoxin
Adult: 125 - 250 micrograms orally daily may be required
Aminophylline *Adult:* up to 250 mg by slow intravenous injection stat

Supportive measures
Oxygen
Nurse in cardiac position

Notable adverse drug reactions, caution and contraindications
Use ACE inhibitors, and aminophylline and digoxin with caution
- Monitor potassium levels closely
- Monitor fluid input and output

Prevention
Adequate control of hypertension

ACUTE SEVERE ASTHMA

Introduction

An exacerbation of asthma that has not been controlled by the use of standard medication Feared by patients and may be life-threatening. Unfortunately, the severity of attack is easily underestimated.

Clinical Features

Patients with acute severe asthma typically have:

- The inability to complete a sentence in one breath
- A respiratory rate of ≥25breaths/min
- Tachycardia ≥ 110beats/min (pulsus paradoxus not particularly useful as it is only present in 45% of cases)
- PEFR <50% of predicted normal or best

Features of life-threatening attack are:

- A silent chest ,cyanosis or feeble respiratory effort
- Exhaustion, confusion or coma
- Bradycardia or hypotension
- PEFR <30% of predicted normal or best(approximately 150L/min in adults)
- Arterial blood gases: -Normal or high $PaCO_2$>6kPa(45mmHg)
  - Severe hypoxaemia PaO_2 <8kPa(60mmHg) despite oxygen therapy
  - A low and falling arterial Ph e.g. <7.35

- $SPO_2 < 92\%$

Differential Diagnosis
- Acute infective exacerbation of COPD
- Acute pulmonary oedema
- Tension pneumothorax
- Pulmonary embolism
- Anaphylaxis

Complications
- Respiratory failure (type 1)
- Pneumothorax or pneumomediastinum
- Cardiac arrest
- Hypoxaemia with hypoxic ischaemic CNS injury
- Toxicity from medications

Investigations
- Pulmonary function tests
- Arterial blood gases
- CXR
- Sputum culture if yellowish, offensive or copious.
- Blood culture if pyrexial
- FBC and ESR
- EUCr

Management

A medical emergency thus intervention is started immediately along with history taking and physical examination.
- Quickly assess severity of attack. Alert

ICU if life-threatening

- Sit patient up and give high dose Oxygen 40-60% via a non-rebreathing bag
- Nebulised salbutamol 5mg() is given. This can be repeated 4hourly for 4 doses
- IV Hydrocortisone sodium succinate 200mg 4hourly for 24hours. Prednisolone is continued at 40-60mg orally daily for 2 weeks.
- Nebulised ipratropium bromide 0.5mg may be added.

If life-threatening features are present:

- Inform ICU and senior colleagues.
- Add $MgSO_4$ 1.2-2g IV over 20mins
- Give salbutamol nebulizer every 15mins or 10mg continuously per hour. Monitor ECG; watch for arrhythmias.

Further management

If patient is improving:

- 40-60% oxygen
- Tab Prednisolone 40-50mg per day for at least 5days.
- Nebulised salbutamol 4hourly
- Monitor peak flow and oxygen saturations

If patient is not improving after 15-30mins:

- Continue 100% oxygen and steroids
- IV Hydrocortisone 100mg or Tab prednisolone 30mg if not already given.

Give salbutamol nebulizer every 15mins or

10mg continuously per hour.

- Continue ipratropium 0.5mg every 4-6hrs

If patient still not improving:

- Discuss with ICU and seniors
- Continue 100% oxygen
- Repeat nebulised salbutamol every 15mins or give IV infusion 3-20ug/min - consider Aminophylline: load with 5mg/kg IVI over 20mins, then 500ug/kg/hr.

  If still no improvement or life-threatening features are present; consider transfer to ICU. Do arterial blood gases and if $PaCO_2$ >7kPa, ventilation may be required.

Monitoring Treatment

- Repeat PEF 15-30mins after initiating treatment
- Pulse oximeter monitoring: maintain $SaPO_2$>92%
- Check ABG within 2hrs if: initial $PaCO_2$ was normal or raised or initial PaO_2 <8kPa(60mmHg) or patient is deteriorating.
- Record PEF pre- and post-β-agonist in hospital at least 4 times.
- Once patient is improving,
- Wean down and stop Aminophylline over 12-24hours.
- Reduce nebulised salbutamol and switch to inhaled β-agonist

- Initiate inhaled steroids and stop oral steroids if possible.
- Continue to monitor PEF. Look for deterioration on reduced treatment and beware early morning dips in PEF
- Look for the cause of the acute exacerbation and admission and take care of it.

Discharge

Patients before discharge, must have:

- Been stable on discharge medications
- Had inhaler technique checked
- PEF >75% Predicted or best with diurnal variability <25%
- Steroids(inhaled and oral) and bronchodilator therapy
- Own a PEF meter and have management plan
- Respiratory clinic appointment within 4 weeks and GP appt within a week.

Common Adverse Drug Effects

- Oxygen-seizure, retinal detachment, ARDS etc
- Steroids-DM, osteoporosis, proximal myopathy, PUD, Cushing's syndrome, growth retardation.
- Beta-agonists-fine tremors, nervous tremors, palpitation, headache ,muscle cramps, tachycardia, arrhythmias, peripheral vasodilatation, insomnia, hypokalemia

- Ipratropium–taste disorders, GERD, Pharyngitis, dysuria, insomnia
- Aminophylline–palpitations, tachycardia, arrhythmia, nausea, vomiting, gastric irritation, headache, convulsion, HTN(better avoided in the elderly, patients with arrhythmias, high BP)
- $MgSO_2$ -nausea, vomiting, flushing of skin, hypotension, arrhythmias, respiratory depression and muscle weakness.

CARDIAC ARREST

Introduction

Sudden cessation of cardiac pump function

If there is no spontaneous reversal or resuscitatory measure, death results

Commonest cause of cardiovascular deaths among Caucasians

Peaks between ages 0 - 6 months and 45 - 75 years

Aetiology

Myocardial infarction

Arrhythmias

Drugs and other substances of abuse

Sudden infant death syndrome

Miscellaneous

Clinical features

Usually sudden collapse Unrecordable blood pressure Loss of peripheral pulses

Cessation of respiration May be asymptomatic Complaints may be non-specific Presentation may be that of underlying cause

Differential diagnoses
Syncope Seizures

Complications
- Acute renal failure
- Cerebrovascular accident

Investigations (after the initial rapid assessment and resuscitation)
Electrocardiography
Echocardiography
Urea, Electrolytes and Creatinine

Blood gases
Chest radiograph
Lipid profile

Treatment objectives
Prompt restoration of cardiac and respiratory function
Monitoring of impact of cardiac arrest on the various associated organs
Intervention to restore normal functions
Formulation of a broader and more comprehensive diagnostic and treatment plan

Eliminate/control aetiological factor(s) in order to reduce morbidity/prevent mortality

Non-drug treatment

Ensure clear airway by tilting the head backwards, lifting the chin and exploring to remove foreign bodies/dentures

Remove wears/ornaments which may negate the above

Basic life support (CPR)

Ensure that patient is lying on a firm/hard surface

Cardiac massage (80-100 per minute)

Assisted ventilation using a masked ambu bag

Twice in succession for every 15 cardiac massages (once every 5th massage when 2 people are in attendance)

- Watch out for spontaneous respiration during this exercise

Advanced life support

Intubation with an endotracheal tube

Defibrillation/cardioversion for patients with ventricular fibrillation/ventricular tachycardia

- Defibrillate with 200 J shock. Additional shock up to 360 J may be required

Epinephrine (adrenaline) 1mg intravenously after failed defibrillation Repeat defibrillation Insert intravenous line

Monitor arterial blood gases

Drug treatment

Sodium bicarbonate

- 1 milliequivalent/kg
- Additional 50% of this dose every 10 to 15 minutes as deemed clinically appropriate

Lidocaine 1 mg/kg intravenously if there is unstable cardiac electrical activity. Repeat as required

Other antiarhythmic drugs if necessary For cardiac arrest secondary to bradyarrhythmias or asystole:

Continue CPR

Insert intravenous line

Prevention

Family and community basic support education

DROWNING AND NEAR-DROWNING

Introduction

Refers to death by suffocation due to immersion in water

May be classified as "wet"- where the victim has inhaled water or "dry"- a less common condition, but one that involves the closing of the airway due to spasms induced by water

Wet drowning could occur by either fresh or salt water

Drowning typically accounts for a small but significant percentage of accidental deaths

Near-drowning episodes refer to instances where rescue was successful and death prevented

Near-drowning can be associated with considerable disability e.g. head injury, paralysis, and respiratory complications

Contributory factors

Swimming in deep waters Falling unexpectedly into water Not being able to swim Breath-holding swimming and diving Alcohol consumption High water temperatures Easy, illicit access to pools Inadequate pool and spa covers Muscle cramps or epileptic attacks developing during swimming

Pathophysiology

Inhalation of water results in ventilation-perfusion imbalance with hypoxaemia and pulmonary oedema

Absorption of hypotonic fresh water results in collapse of the alveoli, resulting in right-to-left shunting of un-oxygenated blood

Absorption of hypertonic salt water results in alveolar oedema, but the overall effects are the same for both inhalation of fresh and salt water

Infection may develop subsequently and is

more likely when contaminated water is inhaled

Clinical features

If alive, patient is unconscious and not breathing

Hypoxemia and tissue hypoxia

Acidosis

Hypothermia

Pneumonia

Acute renal failure

Haemolysis

Complications of near-drowning

Hypoxic brain injury with cerebral oedema (which may occur within 24 hours)

Cardiac arrhythmias

Dehydration

Acute Respiratory Distress Syndrome (ARDS) Acute renal failure

Disseminated Intravascular Coagulopathy

Investigations

Full Blood Count; ESR

Chest radiograph

Electrolytes, Urea and Creatinine

Liver function tests

Acid base status evaluation

Arterial blood gases

Skull and spine radiographs

CT Scan (if available)

Treatment objectives

Immediate resuscitation and stabilization to prevent or minimize complications

Non-drug measures

Airway management Immobilize the cervical spine, as trauma may be present

Treat hypothermia vigorously

Endotracheal intubation with mechanical ventilation and Positive End-Expiratory Pressure if patient is apneic or in severe respiratory distress or has oxygen-resistant hypoxemia

Admission for observation for at least 24 hours if any of the complications are observed even if briefly

Drug treatment

Ventilate with 100% oxygen

Establish an intravenous infusion with 0.9% saline or lactated Ringer's solution

Manage pulmonary complications with the administration of 100% oxygen initially, titrated thereafter reviewing arterial blood gases Bronchodilators if bronchospasm is present Manage metabolic acidosis: give NaHCO if pH is 3 persistently less than 7.2

Treat cerebral oedema

- Hyperventilation
- Intravenous mannitol (1 - 2 g/kg every 4

hours) Appropriate management of pulmonary oedema

Prevention

Teach the unskilled to stay away from water
Teach persons not to swim beyond skill level
Parental/caregiver supervision of children
Diving only under suitable conditions
Education/public awareness

Isolation fences around outdoor pools, and locked doors for indoor pools Locked safety covers for spas and hot tubs

ELECTROLYTE ABNORMALITIES

Introduction

Detection of deranged electrolytes and fluid balance does not constitute a diagnosis

Efforts should be made to determine the underlying causes in every case

Hyperkalaemia

Plasma K concentration > 5 mmoles/L

Aetiology

Usually occurs as a result of potassium release from cells

Decreased renal excretion of K as in renal failure Decreased potassium secretion: Impaired sodium reabsorption in

- Primary hypoaldosteronism
- Adrenal insufficiency

- Secondary hypoaldosteronism
- Medications such as ACE inhibitors, NSAIDs and heparin

Enhanced chloride reabsorption (chloride shunt) as seen in Gordon's syndrome

Clinical features

Weakness, flaccid paralysis, metabolic acidosis

ECG changes

- Increased T wave amplitude
- Peaked T waves
- Prolonged PR intervals, QRS duration
- Atrioventricular conduction delays
- Loss of P waves
- Ventricular fibrillation or asystole

Investigations

Serum Urea,
Electrolytes and Creatinine
Other renal function tests
Acid base balance

Treatment objectives

Correction of hyperkalaemia
Preservation of cardiac function
Treatment of underlying cause(s)

Management

Depends on the degree of hyperkalaemia, associated physical features and ECG

changes
The measures are aimed at:
Promoting potassium loss
Limiting exogenous potassium intake
Discontinuation of anti-kaliuretic drugs
Shifting potassium into cells

Drug treatment

Calcium gluconate

- 10 ml of 10% solution intravenously over 2-3 minutes Insulin plus glucose infusion
- 10 - 20 units of regular insulin plus 25 - 50 g of glucose given as 10 units in 100 ml of 50% glucose

Other alternatives to cause influx of potassium: Sodium bicarbonate (134mmoles/L) if there is metabolic acidosis

- See Cardiac Arrest

Or:

Parenteral/nebulised salbutamol (see Bronchial asthma) Removal of potassium with diuretics (loop plus thiazide diuretics in combination)

Sodium polysterene sulphonate (a cation exchange resin)

- Administered as a retention enema of 50 g of resin and 50 ml of 70% sorbitol mixed in 150 ml of tap water

Haemodialysis

- The most rapid and effective way of

lowering plasma potassium concentration

- Reserved for patients in renal failure and those with severe hyperkalaemia unresponsive to more conservative measures

HYPERNATRAEMIA

Introduction

Defined as plasma sodium > 145 mmoles/Litre

Majority of cases result from water loss in the absence of sodium loss, when the thirst mechanism is impaired, or (infrequently) due to primary sodium gain

Clinical features

Mainly neurologic:

Altered mental status

Weakness

Neuromuscular irritability Focal neurological deficits Occasionally coma and seizures

As in hyponatraemia severity of the clinical features are related to the rapidity of onset and the magnitude of the rise in plasma sodium concentration

Treatment objectives

Correct water deficit

Stop on-going water loss

Calculation of water deficit

Deficit = (Plasma Na -140)/140 X 0.5(males) **or** 0.4 (females)Xbody weight in kg

Water replacement in glomerulo nephropathy

Mineralocorticoid excess (primary deficit should be corrected slowly over 48 - 72 hours to prevent cerebral oedema

Water replacement can be given by mouth or nasogastric tube pressure

- Glucose 5% injection is also suitable for water replacement, being a hypotonic fluid mmHg

HYPOKALAEMIA

Introduction

Plasma potassium less than 3.5 mmol/Litre

Mostly associated with increase in potassium loss

Increased renal loss: Diuretics and salt-waste and secondary hyperaldosteronism 6

Increased distal delivery of non-reabsorbable anions (vomiting, DKA, renal tubular acidosis) Amphotericin B

Cushing's syndrome, Bartter's syndrome

Increased non-renal loss:

GIT loss (diarrhoea, integumentary sweat)

Redistribution into cells:

Metabolic alkalosis

Drugs

Insulin

P adrenergic agonists aadrenergic antagonists Decreased intake: Starvation

Clinical features

- Vary between patients and depend on the level of potassium loss

Serum K <3mmoles/ Litre: Patigue Myalgia Weakness of the lower extremities More severe hypokalaemia results in

- Progressive weakness
- Hypoventilation
- Complete paralysis

ECG changes are due to ventricular depolarisation and sulphonamide do not correlate with the plasma potassium levels

- Flattening/inversion of the T wave
- Aprominent Uwave
- ST segment depression
- Prolonged QT interval
- Severe depletion results in prolonged PR interval
- Decreased voltage and widening of the QRS complex

- *Investigations*

Electrocardiography
Electrolytes, Urea and Creatinine
Acid-base status
Identifying the underlying disease

Treatment objectives

Correction of potassium deficit

Minimize/stop on-going loss

Drug treatment (oral route preferred)

Potassium chloride

- Doses depend on deficits, on-going losses and renal status

Intravenous potassium (given in an infusion)

- Do not exceed 20 mmoles/L

Calculation of potassium requirement

Deficit body weight (kg) 0.3

- Add daily requirement of potassium and correct over 3 days

Caution

Oral potassium supplements should be taken in an erect position or sitting upright and with plenty of water to avoid oesophageal erosions

Hyponatraemia

Plasma Na+ < 135mmol/L

Different types with varied aetiologies

Pseudo-hyponatraemia:

With normal plasma osmolality as seen in hyperlipidaemia or hyper-proteinaemia

With increased plasma osmolality as seen in hyperglycaemia, infusion of mannitol

Hypo-osmolar hyponatraemia:

Due to a primary water gain and secondary sodium loss, or a primary sodium loss and secondary water gain

Integumentary loss: sweating, burns
Loss from the GIT: vomiting, tube drainage, fistula
Renal loss: diuretics, hypoaldosteronism, salt wasting neuropathy, obstructive diuresis
Primary polydypsia
Cardiac failure
Hepatic cirrhosis
Nephritic syndrome
Decreased solute intake:
SIADH
Glucocorticoid deficiency
Hypothyroidism
Chronic renal insufficiency

Clinical features
Cerebral oedema
May be asymptomatic
Otherwise nausea, malaise, headache, lethargy, confusion, and altered consciousness
Coma when plasma sodium is less than 120 millimoles per litre

Differential diagnoses
Congestive cardiac failure
Hepatic cirrhosis
Nephritic syndrome

Investigations
Directed at establishing the cause and

severity of hyponatraemia

Treatment objectives

To correct plasma sodium concentration by restricting water intake and promoting water loss

To correct the underlying disorder

Management

Mild asymptomatic hyponatraemia requires no treatment

Mild hyponatraemia with ECF volume contraction: Sodium releption with isotonic saline infusion

Hyponatraemia associated oedematous states: Restriction of both sodium and water intake

Promotion of water loss in excess of sodium by use of a loop diuretic

For severe cases which are symptomatic (plasma sodium concentration <115 mmoles/L):

Hypertonic saline to raise sodium concentration by 1 - 2 mmol/L/hour for the first 3 hours, but not more than 12 mmoles/L during the first 24 hours

Calculation of the total amount of sodium to administer

Amount of sodium = (desired concentration – actual concentration) X body weight X 0.6

HYPERTENSIVE EMERGENCIES

Introduction

Severely elevated blood pressure (>200/120 mmHg) with evidence of target organ damage such as: Neurologic (e.g. altered consciousness) Cardiovascular (myocardial ischeamia, left ventricular failure)
Renal deterioration
Fundoscopic abnormalities
Presentations include:
Aortic dissection
Hypertensive encephalopathy
Eclampsia
Malignant hypertension

Aetiology

Improperly managed hypertension
Renal vascular disease
Pheochromocytoma
Accelerated essential hypertension

Clinical features

Severely elevated blood pressure (>200/120mmHg)
Headaches, malaise, vomiting, dizziness, blurred vision, chest pain, palpitations, dyspnoea, oliguria
Fundoscopic changes
Evidence of left ventricular failure
Changes in level of consciousness

Complications
Target organ damage
Cerebrovascular accident
Myocardial infarction
Cardiac failure
Renal failure
Death

Investigations
Plain chest radiograph
Echocardiography
Full Blood Count
Urea, Electrolytes and Creatinine
Urinalysis
Echocardiography

Treatment objectives
Prompt but gradual reduction in mean arterial by not more than 25% within the first 2 hours Further reduction of BP to (not less than) 160/100 within 2 to 6 hours
- Lower pressures may be indicated for patients with aortic dissection

Initiate/re-initiate long term therapy to normotensive levels

Drug treatment
labetalol
Notable adverse drug reactions, caution
Stop infusion if response is unsatisfactory after 10 minutes at maximum dose

Lower doses in patients already on anti-hypertensives
Hypotension may occur
Monitor blood cyanide and thiocyanate concentrations
Discontinue if adverse drug reaction to metabolites develop: tachycardia, sweating, hyperventilation, arrhythmias, acidosis)
Reduce infusion over 15 - 30 minutes to avoid rebound effect when stopping therapy

HYPOGLYCEMIA

Introduction

Blood glucose level less than 2.5 mmol/L (45 mg/dL)
May occur in a fasting state or may be post-prandial

Aetiology

Most commonly iatrogenic
Antidiabetic drugs
Associated with quinine, salicylates and use
After overnight fast
Missed meal(s)
During exercise
Can be due to intensive insulin therapy
May follow weight loss
May follow alcohol ingestion
Reduced insulin clearance Sepsis
Secondary to non-B cell

tumours/insulinoma

Clinical features

The two types are neuroglycopenic and neurogenic

Neurogenic manifestations:

Palpitations
Tremors
Anxiety
Sweating
Hunger
Paresthesia

Neuroglycopenic manifestations:

Confusion
Fatigue
Seizures
Loss of consciousness
Death

Diagnosis

The Whipples's triad provides a framework for diagnosis of hypoglycaemia:

Symptoms of hypoglycaemia

Low plasma glucose concentration (<2.5mmole/L)

Alleviation of hypoglycemic symptoms after glucose administration

Differential Diagnoses

Other causes of acute confusional state

Investigations

Random blood sugar on presentation

Other tests to confirm the cause of hypoglycaemia

Treatment objectives

Prompt restoration of normal blood glucose level

Prevention of rebound or recurrent hypoglycaemia

Prevention of occurrence of neural damage or death

Treatment

Urgent treatment must be given if irreversible complications are to be avoided

Oral glucose tablets or glucose drinks if tolerated (and if patient is conscious)

If there is neuroglycopaenia preventing the use of oral glucose:

- Give 50% glucose (dextrose) - 50 ml/25 g in double dilution intravenously
- Followed by 5 - 10% glucose (dextrose) for at least 48 hours in hypoglycaemia secondary to sulphonylurea therapy
- Intravenous glucagon 1mg stat (give subcutaneously or intramuscularly if intravenous route is impractical)

Supportive measures

Discontinue or reduce the dosage of causative drugs
Treat identified underlying cause(s)

Precaution

Glucagon is not effective in glycogen-depleted individuals e.g. those with alcohol induced-hypoglycaemia

MYXOEDEMA COMA

Introduction

A life-threatening complication of hypothyroidism
Follows a background of long-standing hypothyroidism

Clinical features

May be precipitated by exposure to cold, infection, trauma and CNS suppressants
Coma with extreme hypothermia, temperatures 24 - 32oC
Seizures Areflexia
CO retention and respiratory depression due to2 decreased cerebral blood flow

Differential diagnoses

Corna due to CNS depressants
Adrenal insufficiency
Morbid depression

Complications
Cardiac failure
Respiratory failure
Death

Investigations
T_3, T_4, TSH assay

Treatment objectives
To restore normal body metabolism
To prevent death

Drug treatment
Triiodothyronine - 20 micrograms intravenously stat, then 20 micrograms every 8 hours until there is sustained clinical improvement
May also require hydrocortisone 100 mg intravenously every 8 hours
Maintain therapy with oral thyroxine in a dose of 50 micrograms per day
Treat precipitating factor(s)

Precaution
Patients should not be re-warmed rapidly because of risk of cardiac arrhythmias

PNEUMOTHORAX

Introduction
The presence of air or gas in the pleural cavity which can impair oxygenation and /or

ventilation Occurs in apparently normal lung or in the presence of an underlying lung disease.

Clinical results are dependent on the degree of collapse of the lung on the affected side.

Classified as:

- Spontaneous pneumothorax which develop without preceding trauma (e.g. Primary and Secondary)
- Traumatic pneumothorax which develop as a result of direct or indirect trauma to the chest, including diagnostic or therapeutic maneuvers (Iatrogenic Pneumothorax).

Primary Spontaneous Pneumothorax (PSP):

Presents in an otherwise healthy individual with no underlying lung disease.

Occurs in people aged 20-30 years, with a peak incidence in the early twenties.

Rarely observed in people older than 40 years.

Secondary Spontaneous Pneumothorax (SSP):

Complicates an underlying lung disease like pulmonary infections e.g. Tuberculosis, pneumocystis pneumonia, bacterial pneumonia or pulmonary airway disease e.g. COPD especially Emphysema, acute severe asthma, and cystic fibrosis

Occurs more frequently in patients aged 60-65

yrs

Catamenia Pneumothorax:
Recurrent pneumothorax associated with menstruation
Respiratory Symptoms occur within 24-48hrs of menstruation in women aged 30-40 years
Mostly right-side and associated with parity
Pelvic endometriosis may be demonstrated in up to one-third of patients.

Tension Pneumothorax:
life threatening condition develops when air is trapped in the pleural cavity under positive pressure
Displaces mediastinal structures and Complicates approximately 1-2% of the cases of spontaneous pneumothorax.

Clinical features
Varies widely from being asymptomatic to life threatening respiratory distress.

Symptoms
- Acute onset of chest pain (sharp and stabbing) worse on inspiration
- Shortness of breath
- Sweating
- Fainting
- Anxiety
- Cough

- History of previous Pneumothorax
- Acute epigastric pain

Signs

Respiratory system

- Dyspnea
- Tachypnoea (or bradypnoea as a pre-terminal event)
- Tracheal deviation to contra-lateral side
- Decreased chest expansion
- Decreased tactile fremitus
- Normal or hyper-resonant chest wall percussion notes
- Decreased breath sounds on the affected side

Central nervous system

- Altered consciousness
- Anxiety
- Syncope

Cardiovascular system

- Tachycardia
- Pulsus paradoxus
- Hypotension
- Raised jugular venous pressure

Differential diagnosis

- Acute coronary syndrome
- Acute respiratory distress syndrome
- Aortic dissection
- Congestive cardiac failure and pulmonary edema
- Esophageal rupture and tears

- Myocarditis and cardiac tamponade
- Pulmonary Embolism
- Rib fracture

Complications
- Failure to re-expand
- Recurrence
- Haemopneumothorax
- Pyopneumothorax
- Re-expansion pulmonary edema
- Respiratory failure

Investigations
- Chest Computer Tomography (CT)
- Arterial Blood Gas Analysis
- Chest Radiography
- Chest ultrasonography

Goals of Treatment:
Relieve the Pneumothorax
Prevent recurrence.
Modality of treatment depends on clinical presentation and actiology.
Patients with PSP or SSP and significant breathlessness associated with any size of Pneumothorax should undergo active intervention on admission(Tension pneumothorax)

Management of PSP
- Observation is the treatment of choice for small PSP without significant

breathlessness.

- Selected asymptomatic patients with a large PSP may be managed by observation alone for the natural tendency of the gases in pleural space is to be reabsorbed.
- Patients with a small PSP without breathlessness should be considered for discharge with early outpatient review. These patients should also receive clear written advice to return in the event of worsening breathlessness.

Drainage of Pneumothorax

- Needle aspiration (NA) using size 14-16G needle drains.
- NA should not be repeated unless there were technical difficulties.
- Following failure of NA, small core (< 14F) chest drain insertion is recommended.
- Large-bore chest drains are not needed for pneumothorax.

Suction

- Suction should not be routinely used
- Caution is required because of the risk of re-expansion pulmonary edema
- High – volume low-pressure suction systems are recommended

Specialist referral

- Referral to a respiratory physician

should be made within 24hours of admission.

- Complex drain management is best effected in areas where specialist medical and nursing expertise are available.

Management of SSP

- All patients with SSP should be admitted to hospital for at least 24hours and receive supplemental oxygen.
- Most patient will require the insertion of a small-bore chest drain.
- All patient will require early referral to a chest physician to treat underlying cause.
- Those with a persistent air leak should be discussed with a thoracic surgeon at 48hours on admission.

SSP patient who are unfit for surgery

- Medical pleurodesis may be appropriate for inoperable patients
- Patients with SSP can be considered for ambulatory management with a Heimlich valve

Discharge and follow-up

- Patients should be advised to return to hospital if increasing breathlessness develops.
- All patients should be followed up by a respiratory physicians until full resolution.
- Air travel should be avoided until full

resolution.

- Diving should be permanently avoided unless the patient has undergone bilateral pleurotomy and has normal lung function and chest CT scan post-operatively.

Medical chemical pleurodesis

- Chemical pleurodesis can control difficult or recurrent Pneumothoraces but, since surgical options are more effective, it should only be used if a patient is either unwilling or unable to undergo surgery.
- Chemical pleurodesis for pneunothorax should only be performed by a respiratory specialist using 5g sterile talc in Normal saline.

Referral to thoracic surgeons

In cases of persistent air leak or failure of the lung to re-expand, an early (3-5 days) thoracic surgical option should be sought.

Surgical strategies:

- Open thoracostomy
- video – Assisted Thoracoscopic surgery (VATS)

Tension Pneumothorax treatment

- Tension Pneumothorax is a medical emergency

- Treatment is with oxygen and emergency needle decompression at the second intercostal space.
- Pleurodesis with 5% talc in Normal saline is done when all the air is out.

PULMONARY THROMBOEMBOLISM

Introduction

Clinically significant obstruction of a part or the whole of the pulmonary arterial tree
Usually by thrombus that becomes detached from its sites of formation outside the lung
The emboli is swept downstream until it is arrested at points of intrapulmonary vascular narrowing

Predisposing factors: the virchow's triad:
- Relative venous statis
- Injury to the wall of a vein
- Increased coagulability of blood itself.

Pulmonary thromboembolism most commonly results from deep vein thrombosis (a blood clot in the deep veins of the legs or pelvis) that breaks off and migrate to the lung ie. Venous thromboembolism (VTE).
Sources of non-thrombotic emboli: Air, fat, amniotic fluid, bone marrow
Factors that contribute to venous thromboembolism:
Immobilization,
Trauma,

Heart disease
Malignancy
Pregnancy
Puerperium
Oestrogen therapy
Other risk factors include: obesity, chronic bronchitis emphysema, diabetes mellitus, homocyteinuria and polycythaemia.

Massive pulmonary thromboembolism is a medical emergency.

Clinical features

- Sudden onset of breathlessness
- Chest pain
- Cough
- Haemoptysis
- Anxiety
- Cyanosis
- Syncope
- Sudden death

Signs

- Varying degrees of dyspnea
- Pleuritic pain on inspiration
- Pleural rub
- Tachycardia
- Gallop rhythm
- Accentuated pulmonary component of the second heart sound.
- Jugular venous engorgement
- Wheeze

- Pyrexia
- Clinical signs of deep venous thrombosis (DVT)

Differential diagnosis

- Acute coronary syndrome
- Acute respiratory distress syndrome
- Anxiety disorder
- Atrial fibrillation
- Cardiogenic shock
- Pulmonary hypertension

Investigations

- Electrocardiogram
- CT pulmonary angiography
- Ventilation / perfusion scan
- Chest x-ray.
- Arterial blood gases.
- Echocardiagrphy
- D – dimer level

Wells score

Pulmonary embolism can be predicted using the wells score.

- Clinically suspected DVT – 3.0 points
- Alternative diagnosis is less likely than P.E – 3 points
- Tachycardia (H.R > 100) – 1.5 points
- Immobilization ≥ 3days or surgery in previous four weeks – 1.5 points
- History of DVT – 1.5 points
- Hemoptysis – 1.0 points
- Malignancy (with treatment within 6

months) or palliative – 1.0 points

Traditional interpretation

- Score > 6 – High (probability 59% based on pooled data)
- Score 2.0 to 6.0 – moderate (probability 29% based on pooled data).
- Score < 2.0 – Low (probability 15% based on pooled data).

Alternative interpretation

- Score > 4 – PE likely
- Score 4 or less – PE unlikely

Complications

- Sudden cardiac death
- Obstructive shock
- Pulseless electrical activity
- Atrial or ventricular arrhythmias
- Cor – pulmonale
- Severe hypoxemia
- Paradoxical embolism
- Thrombophlebits

Management

1. Anticoagulation

   Enoxaparin (LMWH e.g. lovenox)- 1mg/kg SC q12hr or 1.5mg/kg SC qDay

   Warfarin (e.g. Coumadin, Jantoven) – initial dose: 2-5mg PO qDay (overlap warfarin and parenteral anticoagulant for at least 5days then discontinue parenteral

therapy)
The INR is maintained 2.0 – 3.0
Side effect is spontaneous bleeding

2. Thrombolysis – with fibrinolytic therapy
   Streptokinase – rarely used because of ananphylaxis
   Alteplase (e.g. Activase, TPA,) 100mg iv infused over 2 hours; 10mg bolus followed by 90mg over 2hr .followed immediately with heparin therapy when PTT returns to < 2times normal. Side effects is spontaneous bleeding.
3. Inferior vena cava filters – when anticoagulant therapy is contraindicated
4. Surgery
   Pulmonary embolectomy
5. Prophylaxis for pulmonary embolism
   Low – dose heparin e.g. levonox 40mg SC qDay
   Antiplatelets e.g. Asprin 75- 81mg PO qDay

Notable adverse drug reactions, caution and contraindications

Heparin:

- Thrombocytopaenia and haemorrhage
- Osteoporosis
- Pathologic fractures
- May cause hyperkalaemia (inhibition of aldosterone secretion)
- Contraindicated after recent surgery or

trauma, in haemophilia and other bleeding disorders, peptic ulcer, severe liver disease, acute bacterial endocarditis

Enoxaparin:

- Haemorrhage
- May cause hyperkalaemia (inhibition of aldosterone secretion)

Warfarin:

- Haemorrhage
- Skin necrosis
- Avoid during pregnancy

Recombinant tissue plasminogen activator

- Intracranial haemorrhage

Prevention

- Prophylactic warfarin or heparin in patients at risk
- Inferior vena cava filters, when anticoagulation cannot be undertaken because of active bleeding.

THYROID STORM (THYROTOXIC CRISIS)

Rare but life-threatening

Mortality rate is up to 30% even with treatment

Causes of death include cardiac failure, arrythmias and hyperthermia

Precipitants include the following:

Infections

Trauma & Surgery

Stroke

Diabetic ketoacidosis

Radio iodine treatment of patients with partially treated or untreated hyperthyroidism

Clinical features

Fever

Diarrhoea

Vomiting

Jaundice

Seizures

Coma

Complications

Cardiac failure

Arrythmias

Hyperthermias

Investigations

Diagnosis is mainly clinical and laboratory investigations are done to identify possible precipitants-infection.

Level of thyroid hormones may not be markedly elevated.

Correlation does not exist between levels of thyroid hormones and thyroid storm.

FBC may show leucocytosis even in the absence of infection

Raised bilirubin,

Alkaline phosphatase

Management

Requires intensive monitoring Supportive care

Identification and treatment of precipitating cause(s)

Treatment objectives

Reduction in T synthesis/action and restoration to3 normal values

Treatment of identified precipitating factors

Prevention of complications

Drug treatment

Propylthiouracil

Adult: 600 mg loading dose; 200 - 300 mg orally every 6 hours by nasogastric tube or per rectum *Child 5 - 12 years:* Initially 50 mg orally 3 times daily until euthyroid then adjusted as necessary *12 - 18* years. Initially 100 mg 3 times daily administered until euthyroid then adjusted as necessary; higher doses sometimes required

Saturated Solution of Potassium Iodide (SSKI)

Adult: 5 drops every 6 hours; to be commenced 1 hour after the first dose of propylthiouracil

Child 1 month -1 year: 0.2 - 0.3 mL orally 3 times daily

- Dilute well with milk and water

Propranolol

Adult: 40 - 60 mg orally every 4 hours or 2 mg intravenously every 4 hours
Child: neonate, initially 250 - 500 micrograms /kg every 6 -8 hours, adjusted according to response 1 month - 18 years: initially 250 - 500 micrograms/kg every 6 - 8 hours, adjusted according to response; doses up to 1 mg/kg may be required; maximum 40 mg every 8 hours
Dexamethasone
- 2 mg intravenously every 6 hours
Intravenous Chlorpromazine 50-100mg. Intramuscular route to treat agitation
Intravenous anti-arrhythmic if arrhythmia is present- choice of drug depends on arrhythmia type.
Antibiotics (if infection is present)

Supportive measures
Adequate hydration with intravenous fluids and cooling

UPPER GASTROINTESTINAL BLEEDING

Introduction

Bleeding from the lower oesophagus, stomach or duodenum up to the level of ligament of Treitz.

Occurs worldwide and is responsible for significant mortality and morbidity. Major causes include bleeding from:
- Peptic ulcer disease

- Oesophageal and gastric varices
- Mallory-Weiss tear
- NSAID-related mucosal bleeding
- Neoplasia

Bleeding is either from rupture of engorged varices or from disruption of the oesophageal or gastro-duodenal mucosa with ulceration or erosion into an underlying vessel.

Clinical features

Depends on whether the bleeding is acute or chronic, mild or severe

Various presentations:

- Haemetemesis
- Melaena
- Haematochezia
- Hypovolaemia
- Iron deficiency anaemia (with its associated symptoms)

Differential diagnoses

Black stools from ingestion of iron tablets

Haematemesis/melaena from previously swallowed

blood (from the upper respiratory tract and oral cavity)

Complications

Hypovalaemic shock

Congestive heart failure from chronic severe anaemia

Investigations

Upper gastrointestinal endoscopy: picks up lesions in 90% of cases

Upper gastrointestinal barium radiography: 80% detection rate

Selective mesenteric arteriography

Radio isotope scanning

Stool- occult blood test

Full Blood Count

Treatment objectives

Restore and maintain haemodynamic status

Control bleeding

Prevent recurrence of bleeding

Non-drug treatment

Carefully monitor vital signs (pulse, blood pressure, respiration and temperature) as frequently as necessitated by the patient's condition Insert a nasogastric tube to aspirate gastric contents and/or to introduce agents to constrict the blood vessels

Blood transfusion: whole blood (acute bleeding) or packed cells (chronic) bleeding. Up to 5 - 6 pints of blood may be needed in severe cases

- Plasma expanders in the absence of blood

Continuous Central Venous Pressure (CVP) monitoring

Drug treatment

Bleeding peptic ulcers/erosions

Proton Pump Inhibitors

- Omeprazole 20 mg orally once daily for 4 weeks

Or:

- Omeprazole 40 mg by slow intravenous injection over 5 minutes once daily until patient can take orally

Anti-Helicobacter pylori therapy set above.

Endoscopic treatment for actively bleeding ulcer or visible non-bleeding vessel

- Injection therapy with 98% alcohol (total volume less than 1mL)

Or:

- Injection therapy with epinephrine (1:10,000) up to 1mL

Or:

- Thermal coagulation with heat probe

Or:

- Laser therapy

Bleeding varices

Intravenous Octreotide 50 microgram stat, then infusion or 25-50 microgram hourly for 24-48 hours, until endoscopy

Intravenous vasopressin 20 units over 20 minutes bolus then infusion of 0.1 - 0.5

units/min
Plus:
Intravenous nitroglycerin 40 microgram/min (titrated upward to maintain systolic blood pressure above 90 mmHg)
Endoscopic treatment
Variceal band ligation
Injection sclerotherapy: equal volume mixture of 3% sodium tetradecyl sulfate, 98% ethanol, sodium chloride 0.9% injection (2-5 mL/site; maximum 50 mL)
Radiologic therapy
Venous embolization
Transjugular Intrahepatic Portosystemic Shunt (TIPS)
Oesophageal transection and devascularization
Liver transplant
Peptic ulcers/erosions/tumours
Surgical repair or resection as appropriate

Supportive
Monitor vital signs and urine output to detect early features of hypovolaemic shock
Look out for features of hepatic encephalopathy

Notable adverse drug reactions
Vasopressin can cause abdominal cramps. It lowers blood pressure drastically and could worsen ischaemic heart disease

Prevention

Peptic ulcers/erosions related upper gastrointestinal bleeding

Avoid NSAIDs.

Treat *H. pylori* infection

Oesophageal varices

pblockers (propranolol 40 mg orally 12 hourly and titrate up to 160 mg depending on the heart rate)

Maintenance sclerotherapy

CHAPTER 21:

CLINICAL PHARMACOLOGY AND THERAPEUTICS

ADVERSE DRUG REACTIONS

Introduction

The use of medicines is inextricably linked to unintended responses

The safe use of medicines is therefore an important consideration in therapy

Adverse drug reaction

Response to a medicine which is noxious and unintended and occurs at doses normally used in man for the prophylaxis, diagnosis or therapy of disease, or for the modification of physiologic function

Adverse drug event

Any untoward medical occurrence that may present during treatment with a pharmaceutical product, but which does not necessarily have a causal relationship with the treatment

A serious adverse event (experience, or reaction)

Any untoward medical occurrence that at any dose

- Results in death
- Is life-threatening
- Requires patient hospitalization or prolongs existing hospitalization
- Results in persistent or significant disability/incapacity
- Causes a congenital anomaly or birth defect
- Requires an intervention to prevent permanent impairment or damage

Side effect

Any unintended effect of a pharmaceutical product occurring at doses normally used in humans and is related to the pharmacological properties of the drug

There is need to have a high index of suspicion during therapy so as to recognize and adequately manage adverse effects

Report any suspected adverse response to a drug to the hospitals' Adverse Reaction Registry or directly to the National Agency for Food and Drug Administration and Control (NAFDAC), Abuja A sample of the Yellow Form is shown in Appendix VIII

Analysis of such reports enables appropriate decisions to ensure safe and judicious use of medicines

Pharmacovigilance:

The science and activities relating to the detection, assessment, understanding and preventing adverse effects of drug or any other possible drug related problems (WHO 2002). The scope also includes herbals, traditional and complementary medicines, blood products, biologics, medical devices and vaccines.

Other issues relevant to pharmacovigilance are:

- Substandard medicines
- Medication errors
- Lack of efficacy reports
- Use of medicines for indications that are not approved and for which there is
  - inadequate scientific basis
- Case reports of acute and chronic poisoning
- Assessment of drug-related mortality
- Abuse and misuse of medicines
- Adverse interactions of medicines with chemicals, other medicines and food.

Objectives:

- Improve patient care and safety in relation to the use of medicines and all medical and paramedical interventions
- Improve public health and safety in relation to the use of medicines
- Contribute to the assessment of benefit,

harm, effectiveness and risk of medicines, encouraging their safe, rational and more effective (including cost-effective) use
- Promote understanding, education and clinical training in pharmacovigilance and its effective communication to the public.

Working of pharmacovigilance

Pharmacovigilance relies on the reporting of adverse events to a medical products to the National Pharmacovigilance Center housed in NAFDAC in order to facilitate early detection of hitherto unknown adverse reaction and interactions; and detect increase frequency of known adverse reactions. (see appendix VIII for pharmacovigilance reporting form)

Who reports adverse reactions?

- Patients
- Physicians
- Nurses
- Pharmacist
- Other health care personnel
- Patient - Public

ENVENOMATION

Snake bites

- In Africa, often occur among farmers who walk unshod

- Occasionally occur around homes when snakes are accidentally stepped upon

Poisonous snakes belong to the families of:

- Viperidae (responsible for most snake bites in Africa)
- Subfamilies: viperinae (old world vipers), Crotalinae (New world vipers, Asian pit vipers)
- Elapidae (e. g. cobras)
- Colubridae (e. g. boomslang)
- A large group; only a few species are dangerously toxic to humans
- Hydrophidae (sea snakes)

Clinical features

- Depend on the type of snake, location of bite and promptness of intervention

Local effects:

- Pain, Swelling, Bruising, Tender enlargement of regional lymph nodes

Systemic effects:

- Early anaphylactoid symptoms, Transient hypotension with syncope, Angioedema, Urticaria, Abdominal colic, Diarrhoea, Vomiting, Late persistent or recurrent hypotension, Electrocardiograph abnormalities, Spontaneous systemic bleeding, Coagulopathy, Adult respiratory distress syndrome, Acute renal failure

Viperidae and crotalidae

- Local and systemic bleeding,

Impairment of organ function, Reduction of cardiac output, Inhibition of peripheral nerve impulses
- Multisystem effects: Rhabdomyolysis, Haemolysis, Blood vessel damage

Elapidae
- Neurotoxic effects

Snake bite wounds may become secondarily infected with:
- *Clostridium tetani*, causing tetanus
- *Clostridium welchi*, causing gas gangrene

Initial assessment
- Brief history: time and circumstances of bite, progression of local and systemic symptoms; antibiotic allergy, immunization of patient and other morbid condition(s).
- Examination: to look for tooth marks, local swelling, bleeding, shock, cardiovascular and respiratory systems, evidence of paralysis, level of consciousness,

Investigations
- **FBC and differentials**
- **Test for haemostasis (20-Minute Whole Blood Clotting Test)**
- Electrolytes and Urea
- Blood clotting profile
- Arterial blood gas estimations
- Chest radiographs
- Wound and blood cultures

Treatment objectives

Neutralize envenomation

Limit systemic effects

Local wound care

Prevent onset of complications

First-Aid and Transport to the Hospital

- Move the victim to safety from area where they may have been bitten
- Reassure the patient (only 50% of the bites by venous snakes cause envenoming)
- Remove constricting clothings, rings, shoes, bracelets, bands from the bitten limb
- Immobilize the whole patient, especially the bitten limb, using a splint or sling
- Transport the patient to the hospital as quickly and passive as possible
- Avoid harmful practices: never attempt to suck out or aspirate the poison, do not make incisions, never attempt to catch the snake, a tight arterial tourniquet should never be used)

Hospital management

Non-drug measures

- Assess and observe the patient for at least 24 hours
- Cardiopulmonary resuscitation may be necessary: clearing of the airway,

oxygen administration by face mask or nasal cannula, intravenous access to allow treatment of shock with intravenous fluids

- Identification of the snake would help in the choice of antivenom (where specific antivenoms are available)
- Wound debridement and fasciotomy for compartment syndrome may become necessary

Drug treatment

- Intravenous fluid administration to maintain circulation: use colloids or cystalloids as clinically appropriate
- Antivenom:
  - Indications for antivenom treatment:
  - **Systemic envenomation (neurotoxicity, spontaneous systemic bleeding, incoagulable blood (20MWBCT), cardiovascular abnormality (hypotension, shock, arrhythmia, abnormal electrocardiogram)**
  - **Local envenomation: extensive swelling (involving more than half of the bitten limb), rapidly progressive swelling, bites on fingers and toes**
  - **Usually polyvalent antivenoms are used, but where specific species are identified, a monospecific antivenom may be used**
  - *Adult and child: No dose difference*

between adult and child in terms of dosage. Contents of the antivenom vial diluted in sodium chloride 0.9% intravenous infusion, and infused intravenously over 30 minutes

- **Intradermal, subcutaneous testing with diluted venom before administration are not predictive of venom reaction and should not be done**
- Prophylactic antibiotics as appropriate Tetanus prophylaxis

Antivenom reactions:

- **Early reactions (3-60 minutes): cough, tachycardia, itching, urticarial, fever, nausea, vomiting, headache.**
- Treatment of anaphylaxis with antihistamines (H blockers), epinephrine (adrenaline) and corticosteroids
- Pyrogenic reactions: due to pyrogen contamination of antivenom during manufacture.
- begins 1-2 hours after treatment and characterized by fever and chills
- Tepid sponging and administration of paracetamol are useful
- Late reactions: occurs 5-24 days (average 7 days) following antivenom. There
- Characterized by itching, urticaria, fever, arthralgia, periarticular swellings, proteinuria and sometimes

- neurological symptoms.
- Antihistamines are used for milder attacks, but in severe cases, including those with neurological symptoms, a short course of prednisolone should be used.

Scorpion Sting

Scorpion stings occur worldwide. Most cases are minor but significant envenomation resulting to death has been reported. Children are particularly at risk of scorpion stings and envenomation

Clinical Features:

Local symptom:

- **Localized pains, edema, erythema, paraesthesia, muscle fasciculation and numbness may occur at the site of sting**

Systemic symptom: usually due to autonomic discharge

- hypersalivation, profuse diaphoresis, lacrimation, miosis, diarrhea, vomiting, bradycardia, hypotension, increased respiratory secretions, priapism, tachycardia, hypertension, mydriasis, hyperthermia, hyperglycemia, agitation, and restlessness may occur.

Investigations

- **There is no specific diagnostic investigations, and in most cases, no**

investigation is required
- Investigations, when required, should be tailored towards the complications that may occur

Treatment

Non-drug treatment
- Allay anxiety
- Cold presses may be applied

Drug Treatment
- Analgesic: paracetamol, ibuprofen may be used
- Local anaesthetic agent like lidocaine may be infilterated at the site of bite
- Midazolam, diazepam: in addition to allaying anxiety, they also act as muscle relaxant
- Prazosin for hypertension
- Dobutamine for hypotension
- Nitroglycerin for pulmonary edema
- Antivenom: are available and increase the rate of toxin elimination

POISONS AND POISONING

Introduction

Poisons: are chemical or physical agents that produce adverse responses in biological systems.

Paracelsus (1493-1541) famously said *" All things are poison and nothing without poison. Solely, the dose determines that a thing is not a*

poison"

Poisoning: The ingestion by, or exposure of a patient to excessive doses of a medicine or other substances that may cause harm.

The pattern of poisoning may be influenced by age and gender.

Poison may occur in the following ways:

- Self poisoning (may be suicidal or parasuicide)
- Accidental
- Homicidal
- Occupational
- Environmental

Common substances causing poisoning in the Nigeria include (but are not limited to):

- Pharmaceuticals-Analgesics, hypnosedatives, antidepressants, alcohol
- Petroleum distillates
- Industrial chemicals
- Agrochemicals
- Household products
- Natural toxins
- Toiletries

Duration of exposure:

- Acute: if exposure occurs for less than 24 hours. To be described as acute toxicity, the adverse effect must occur within 14 days of exposure.

- Subacute: if exposure occurs for up to 1 month
- Subchronic: if exposure occur for between 1-3 months
- Chronic: if exposure occur for > 3 months

Clinical presentation

Determined (amongst others) by:

- Type of Agent
- Inherent toxicity
- Dose and duration following exposure
- Concurrent therapy
- Co-existing disease states etc

This guideline provides only a brief overview.

NB: Practitioners are advised to seek advice from experts, standard texts in medicine and toxicology, in the absence of a Poison Information Centre

Principles of management of poisoning

Aims:

1. **Stabilize the patient**
2. **Decrease the absorption of the substance**
3. **Increase elimination of the substance**
4. **Prevent and treat complications**
5. **Reduce the risk of future occurrence**

Management:

1) Emergency stabilization
   - Remove the patient from the

environment if there is immediate sign of danger (important in environmental poisoning).
- Provide fresh air and oxygen (respiratory decontamination)
- Life-saving measures take priority over all other decontamination techniques.

The following ABC approach is recommended:

A. Establish a clear **A**irway (patient may have to be intubated if they are unconscious; all patient should be placed in left lateral position to prevent aspiration of poison and gastric content)
B. Ensure adequate **B**reathing and ventilation (Give high flow oxygen, except in paraquat Poisoning where this may worsen mild-moderate hypoxia)
C. Ensure adequate **C**irculation
D. Address **D**rug-induced depression of the central nervous and respiratory systems
E. Correct any **E**lectrolyte and metabolic abnormalities (hypoglycemia and altered potassium handling are common in

severe poisoning, cardiac monitoring for arrhythmias may be required especially in poisoning with TCA)

2) **Clinical Evaluation**

- Detailed history, physical examinations and appropriate investigations (Remember that absolutely diagnostic features are rare, however, some recognition of some toxidromes may give a clue e.g. pinpoint pupil in opioid poisoning).
- Information from relatives, friends, and emergency services personnel may be very useful.
- The patient may have no symptoms and signs when seen early in the course of the poisoning
- Amount, route and extent of exposure should be ascertained.
- Check for comorbidities
- Calm the patient down
- Appropriate laboratory investigations (blood screen, urine screen, blood biochemistry, liver enzymes etc).
  - *NB: Blood or other specimen assay may be very important to determine if the condition is actually a case of drug poisoning for legal purposes)*
- Watch out for and control convulsions, hypothermia etc

3) **Decrease the absorption of the substance**

Principle:it is based on thepresumption that both the dose and duration of exposure are determinants of toxicity, and limiting continued exposure is beneficial.

Skin and mucuous areas:

- Flushing the areas (e.g. skin and eyes) with large volumes of water/normal saline to remove the toxic substance

Gut decontamination:

- Induced emesis: where there are no contraindications.
- Gastric lavage: Most effective when used within the 1st hour of poisoning.
- Activated charcoal: Adsorb drugs and other substances in a non-specific manner and reduce absorption into the body. Repeated doses may be used in cases of poisons with drugs known to undergo entero-hepatic or entero-enteric re-circulation (e.g. carbamazepine, cardiac glycoside, dapsone, phenobarbitone, quinine and theophylline).
  - *NB: Activated charcoal is very unpalatable and conscious patients must be made to understand this.*
  - *Substances like boric acid,*

cyanide, ethanol, ethylene glycol, iron, lithium, malathion, methanol, petroleum products and strong acids and alkalis are not adsorbed by activated charcoal.

- Gastric aspiration: should be considered in a patient that has ingested potentially life-threatening doses of drugs that are not adsorbed by oral activated charcoal (e.g. Lithium and Iron)
- Cathartics or whole bowel irrigation: osmotic cathartics are no longer recommended because of the risk of electrolyte abnormality. Whole bowel irrigation may be performed to enhance rectal elimination of unabsorbed drugs.

A combination of the above methods may be used.

4) **Increase Elimination of poisons**

Clearance of the toxic substances may be enhanced by:

- Manipulation of urine pH
- Haemodialysis/Haemoperfusion

5) **Use specific Antidotes**

An antidote is a drug that antagonizes the toxicity of another substance in a specific manner. Examples of common antidotes are:

a) Naloxone for opioids

b) N-acetylcysteine for paracetamol
c) Flumazenil for benzodiazepines
d) Penicillamine for copper and lead
e) Digoxin-specific antibody for digoxin
f) 100% oxygen for carbon monoxide
etc

6) **Treat complications**
Treat dysrhythmia, hypoxia, hypotension, convulsions and hypothermia

7) **Reduce the risk of future occurrence**
This may involve counselling and psychiatrist support

SPECIFIC POISONS

Paracetamol

Toxicity often occurs following an acute ingestion (within 24 hours) of 7.5 - 10 g (15-20 tablets)
in adults or 150 mg/kg in children. In patients with certain risk factors, dose of up to 5g can cause liver damage.

Risk factors:

1) Concurrent treatment with drug that induces hepatic CYP450 activity (rifampicin, carbamazepines, St. John's wort etc).

2) Decrease glutathione reserve
   a. Acute starvation
   b. Alcoholism
   c. Chronic malnutrition
   d. HIV

Clinical features

Acute poisoning:
Often divided into 4 overlapping phases with varying physical signs:

Phase 1 (0.5-24 hours after ingestion)
- *May be asymptomatic*
- *May present with anorexia, nausea, vomiting and malaise*
- *Examination may show palor ± diaphoresis*

Phase 2 (18-72 hours after ingestion)
- Anorexia, nausea and vomiting ± right upper quadrant abdominal pain
- ± Right upper quadrant tenderness
- ± Tachycardia and hypotension (ongoing volume loss)
- ± Decrease urinary volume

Phase 3 (Hepatic phase: 72-92 hours after ingestion)
- Anorexia, nausea, vomiting, abdominal pain + tender hepatic edge
- Jaundice, coagulopathy, hypoglycemia and hepatic

encephalopathy may develop
- Acute renal failure may develop
- Multi-organ failure and death

Phase 4 (recovery phase: 4 days-3 weeks after ingestion)
- Patient who survives phase 3 have complete resolution of symptoms and organ failure

Poor prognostic factors:
- Encephalopathy or hepatic failure
- Greater than two fold prolongation of Prothrombin time
- Serum bilirubin > 68 micromol/L (4 mg/dL)
- Serum creatinine > 3.3 mg/L

Chronic poisoning: is usually similar but alcoholics may present with a syndrome of severe combined hepatic and renal insufficiency.

Investigations:
- LFTs including prothrombin time and serum proteins
- Urea, Electrolytes and Creatinine
- Blood sugar estimation
- Blood levels of paracetamol at least 4 hours post-ingestion (where facility is available). Paracetamol levels done before earlier than this may be

misleading.

Treatment objectives

- To prevent or reduce damage to organs
- To restore normal metabolic functions

Drug treatment

Activated charcoal, especially within 4 hours of ingestion

- *Adult:* 50 g orally, repeated if necessary
- Child: under 12 years, 25 g (50g in severe poisoning)

Acetylcysteine

- *Adult and child:* initially 50 mg/kg by intravenous infusion over 15 minutes, then 50 mg/kg over 4 hours and then 100 mg/kg over 16 hours
- Diluted 3: 1 with a non-alcoholic, non-dairy beverage
- Loading dose is 140 mg/kg; maintenance dose 70 mg/kg every 4 hours for 17 doses
- Treatment is effective if started within 8 - 10 hours Alternatively:

Methionine

- *Adult and child over 6years:* 2.5 g orally followed by a further dose of 2.5 g every 4 hours Child under 6 years: initially 1 g followed by 3 further doses of 1g every 4 hours

Supportive measures

- As for all cases of acute poisonings

Notable adverse drug reactions, caution and contraindications

Acetylcysteine may cause:

- Nausea,
- Vomiting and,
- Epigastric discomfort.

Antiemetics (metoclopramide) may be required.

Methionine may cause:

- Nausea,
- Vomiting,
- Drowsiness,
- Irritability

Aspirin:

Toxic doses are associated with increased sensitivity of the respiratory center, incomplete oxidative phosphorylation and increased rate of metabolism

Clinical features

Initial manifestations (occur 3 - 6 hours after an overdose of>150 mg/kg):

- Vomiting
- Sweating
- Tachycardia
- Hyperventilation
- Tinnitus
- Fever
- Lethargy

- Confusion
- Respiratory alkalosis
- Impaired renal function
- Increased anion gap
- Metabolic acidosis may result

Severe poisoning:
- Coma
- Respiratory depression
- Seizures
- Cardiovascular collapse
- Cerebral and pulmonary oedema

Investigations
- FBC, ESR
- Electrolytes, Urea and serum Creatinine
- Random Blood Glucose
- LFTs including prothrombin time
- Blood aspirin levels

Treatment objectives
- As for paracetamol poisoning

Non-drug treatment
- Gastric lavage and whole bowel irrigation

Drug treatment
- Activated charcoal can be used up to 12 - 24 hours after ingestion (see Paracetamol poisoning)
- Intravenous infusion of sodium chloride 0.9% (preferably with glucose)
- Correct dehydration and ensure good

urine output (saline diuresis)
- Supplemental oxygen
- Supplemental glucose
- Intravenous vitamin K 10 mg daily for coagulopathy
- Intravenous $NaHCO_3$ to alkalinize urine
- Correct other electrolyte derangements
- Haemodialysis for severe salicylate poisoning

Indications for haemodialysis
- Severe clinical toxicity
- Aspirin (acetylsalicylic acid) levels > 7 mmol/L (100 mg/dL)
- *a rapidly rising plasma salicylate level even if not up to 7 mmol/L is an indication for dialysis*
- Failure of other treatment modalities
- Renal failure
- Heart failure
- Seizures
- Severe acidosis

Benzodiazepines
- Most commonly involves diazepam and bromazepam
- These drugs potentiate the inhibitory effect of GABA on CNS neurons

Clinical features
- CNS depression may occur within 30 minutes of acute overdose
- Respiratory depression

- Coma, especially when benzodiazepines are combined with other CNS depressants
- Paradoxical excitement may occur early in the course of poisoning

Treatment objectives

- As for paracetamol poisoning

Non-drug treatment

- Respiratory support

Drug treatment

- Activated charcoal: method of choice for gastrointestinal decontamination
- Flumazenil, a competitive benzodiazepine receptor antagonist, can reverse CNS and respiratory depression *(Given as 200 micrograms Flumazenil over 15 seconds; then 100 microgram at 1 minute intervals if need. Usual dose range: 300-600 microgram IV over 3-6 minutes; up to 1mg maximum or 2mg if in intensive care unit)*

Notable adverse drug reactions

- Flumazenil may trigger seizures
- Activated charcoal colours stools black

Carbon monoxide poisoning

- Results from inhalation of smoke, car or generator fumes caused by incomplete

combustion in a confined space
- Carbon monoxide reversibly binds to haemoglobin, myoglobin and mitochondria, inhibiting cellular respiration

Clinical features
- Respiratory system: Dyspnoea
- Tachypnoea
- Headache
- Emotional liability
- Confusion
- Impaired judgement
- Clumsiness
- Syncope
- Nausea, vomiting and diarrhoea may occur
- Ischaemic chest pain,
- Arrhythmias,
- Heart failure and hypotension may occur

In severe poisoning:
- Coma and cerebral oedema may occur
- Pulmonary oedema
- Respiratory depression
- Cherry-red colour of skin and mucus membranes (rarely cyanosis)

Investigations
Full Blood Count and ESR
Serum Urea, Electrolytes and Creatinine
Liver function tests

Acid-base status
Blood gases

Non-drug treatment

- Remove from carbon monoxide exposure; move to fresh air

Drug treatment

- Oxygen administration - face mask in conscious patients and endotracheal intubation in comatose patients after clearing the airways
- Treat hypotension and arrhythmia
- Mannitol (10 - 20%; 250 mL intravenously over 30 minutes. May repeat every 8 hours)

Methanol poisoning

- Methanol is commonly in many industrial solvents and in adulterated alcoholic beverages. Methylated spirit contains 10% methanol
- Other sources are: automotive windshield washer fluids, paint thinners and removers, varnishes, copy machine fluids, and fuel additives (octane boosters)
- Toxicity of methanol is attributed to its metabolism to formaldehyde and formic acids which are toxic and reactive metabolites

Clinical features

- Initial lag period of 12-24 hours before toxic manifestation occur
- Visual disturbance: dimming or blurring of vision, scintillations, photophobia, visual field defects and blindness may occur. Fundoscopy may show enlarged blind spot
- GIT: Nausea, vomiting, epigastric pain, and pancreatitis
- Others: dizziness, headache, malaise, agitation, generalized weakness, and sensorineural depression

Laboratory findings:

- Elevated anion gap
- Metabolic acidosis
- Elevated osmol gap
- Positive serum methanol assay
- CT scan may show characteristic finding of bilateral cerebral infarction selectively involving the putamenand adjacent areas

Treatment:

- Ethanol is the antidote (see standard text for guidelines)
- In severe poisoning, hemodialysis removes methanol

Kerosene poisoning

- Similar to poisoning by other petroleum distillates; commoner in children
- Petroleum distillate hydrocarbons are

poorly absorbed following ingestion
(Aspiration may occur resulting in aspiration pneumonitis)

Clinical features
- CNS excitation (low doses); depression (high doses)
- Rarely coma and seizures
- Others: nausea, vomiting, abdominal pain and diarrhoea

Investigations
- Electrolytes, Urea and serum Creatinine
- Liver function tests
- Chest radiograph
- Electrocardiography

Non-drug treatment
- *Gastric lavage and decongestion are contraindicated because of the risk of aspiration*

Supportive measures
- Oxygen administration
- Respiratory support
- Monitoring liver, renal and myocardial function
- Correct metabolic abnormalities

Drug treatment
- Antibiotics for aspiration pneumonitis
  NB: Glucocorticoids are ineffective

ORGANOPHOSPHATE/INSECTICIDE POISONING

Introduction

- Cause irreversibly inhibition of acetylcholinesterase leading to accumulation of acetylcholine at muscarinic and nicotinic synapses and in the CNS.
- Organophosphates are absorbed through the skin, lungs, and gastrointestinal tract and are distributed widely in tissues
- Elimination is slow- by hepatic metabolism

Clinical features

- Onset is usually between 30 minutes -2 hours after exposure

Muscarinic effects:

- Nausea
- Vomiting
- Abdominal cramps
- Increased urinary frequency;
- Urinary and fecal incontinence
- Increased bronchial secretions
- Cough
- Dyspnoea
- Sweating
- Salivation
- Miosis
- Blurred vision
- Lacrimation
- Bradycardia

- Hypotension, and
- Pulmonary oedema may occur

Nicotinic effects:
- Twitching
- Weakness
- Hypertension
- Tachycardia
- Paralysis in severe cases

CNS effects: Anxiety, Restlessness, Tremor
- Confusion
- Weakness
- Seizure
- Coma

Non-drug treatment
- Remove contaminated clothing
- Wash skin with soap and water
- Ventilatory support

Drug treatment
- Oxygen administration
- Atropine (Effective for muscarinic symptoms)
- *Adult:* 0.5 - 2 mg intravenously every 5 - 15 minutes until bronchial and other secretions have dried
- *Child:* 20 micrograms/kg (maximum 2 mg) intramuscularly or intravenously depending on the severity of poisoning, every 5 - 10 minutes until the skin becomes flushed and dry, pupils

dilate and tachycardia develops

Treat seizures with intravenous diazepam 10 mg stat

Lead poisoning

- Lead is a component of paints, gasolines, storage batteries, glass, ceramics and as part of plumbing pipes
- Inorganic lead is absorbed slowly via the respiratory and gastrointestinal tracts, but poorly from the skin; organic lead compounds are volatile and are absorbed from through the skin and respiratory tract
- Absorption via the gastrointestinal tract is more in children, and is increased in the presence of low dietary calcium, iron deficiency and ingestion on an empty stomach.
- In the blood stream, most of the lead is bound to RBC and remainder is distributed to soft tissues of the brain and kidney.

Clinical features:

Acute:

- manifest with signs and symptoms of encephalopathy and colic
- hemolytic anaemia and basophilic stippling may be present
- Subacute:
- headache, fatigue, intermittent

abdominal cramps, myalgia and arthralgia

Chronic:

Presentation is mutisystemic:

- Fatigue, myalgia
- CNS: headache, difficulty in concentrating, irritability, peripheral neuropathy, and seizures
- GI: colics, anorexia, epigastric tenderness, constipation and metallic taste may occur

Diagnosis:

- Blood level of lead

Management:

- Remove from exposure
- Supportive care
- Chelation therapy: Dinercaprol, penicillamine or Calcium disodium EDTA

RATIONAL PRESCRIPTION WRITING

Introduction

The writing of a prescription is the culmination of a clinical encounter with a patient

The decision to issue a prescription follows a complex process of professional analysis and must be based on following considerations: Knowledge of the patient's clinical state Factors likely to influence the drug's

pharmacokinetics and pharmacodynamics; the efficacy, safety and cost of the drug
Rational prescribing entails the following process with various steps:

Step 1:
- Define the patient's problem

Step 2:
- Specify the therapeutic objectives

Step 3:
- Verify whether your proposed treatment is suitable for this patient

Step 4:
- Start the treatment

Issuing a prescription is not conclusive treatment. Two further steps must be considered:

Step 5:
- Give information, instructions and warnings

Step 6:
- Monitor (and/or stop) the treatment

Details of this process will be found in the WHO's "Guide to Good Prescribing" A prescription order should specify: What is to be administered To whom

By whom prescribed It should clearly indicate:

How much should be taken (the amount e.g. in milligrams, grams) How often (frequency) The route of administration

And:

Duration of therapy *Apart from its use in therapy, a prescription order is important as a medico-legal document* Essential elements of a prescription order Identity of prescriber:

- Name
- Address/institution of prescriber
- Telephone number Date of prescription:
- Near top/beginning of left margin of a chart order Identity of patient:
- Name
- Age (especially in children)
- Gender
- Address of patient
- Hospital number

Elements specifying medication:

- Name of medication *(generic name)*
- Strength *(metric units)and* quantity
- Dosage
- Frequency
- Duration
- Directions for use (drug- and patient-specific)
- Refill instructions
- Waiver of requirements for child-proof containers
- Additional labelling instructions

Prescriber's signature and other identification data e.g. code. Prescriptions may be hand written or computer-issued:

- Hand written prescriptions should be written in indelible ink and the hand

writing should be legible (important, to avoid medication errors)

- Any alteration(s) made in a computer-issued prescription should be duly endorsed

Abbreviations

Only standard, official abbreviations should be used. The following are some notable abbreviations

a.c	ante cibum (before food)
b.d	bis die (twice daily)
o.d	omni die (every day)
o.m	omni mane (every morning)
p.c	post cibum (after food)
p.r.n	pro re nata (when required)
q.d.s	quarter die sumendum (to be taken four times daily)
q.q.h	quarter quaque hora (every four hours) stat immediately
t.d.s	ter die sumendum (to be taken three times daily)
t.i.d	ter in die (three times daily)

NOTE

Avoid abbreviations of drug names

Doses should be written in the metric system or in international units (IU) when metric doses are not practicable

If a drug is to be administered 'as required', specify the minimum dose interval and the total amount of drug to be administered

Avoid unnecessary use of decimal points

1 mg not 1.0 mg

If >1 g state as g

If < 1 g state as milligram e.g. 500 mg not 0.5 g

If < 1 mg state as microgram: 100 microgram not 0.1 mg If the decimal point is unavoidable, insert zero (0) in front of the point e.g. 0.5 mL, not .5 mL Microgram and nanogram should not be abbreviated Millilitre (mL) should be used for volume and not cubic centimetre, c.c or cm^3

Prescription for special cases:

Special precaution should be taken in children (especially neonates and infants), and the elderly when considering drug therapy

- There are differences in drug handling (pharmacokinetics) and sensitivity in drug response (pharmacodynamics) in the different age groups

Particular care should also be taken when prescribing for pregnant women

Precaution should also be taken in clinical states associated with organ system failure (renal, hepatic) where dosage adjustment may be required

Children (including neonates and infants)

There are notable differences in the proportions and constituents of body fluids between adults and children

The immature enzyme systems result in poor oxidation and conjugation and may cause adverse effects

- Grey Baby syndrome with chloramphenicol is an example

Drugs predominantly excreted by the kidneys e.g. aminoglycosides, penicillins may require dose reduction

Use appropriate formulations for various routes e.g. rectal route (for diazepam, theophylline) in the uncooperative child

(See appendix IV for calculation of dose requirements for children)

The Elderly

Persons 65 years or over: a growing segment of the Nigerian population

A number of factors interplay to increase the incidence of adverse drug reactions in this group of patients

- Bodily changes affecting drug handling and tissue response
- The increasing number of medicines prescribed to treat multiple diseases, each with a potential to cause an adverse drug reaction as well as a drug-drug interaction
- Poor adherence to therapy due to factors inherent in the elderly

Dosage reduction may be required for some drugs because of

- Changes in volume of distribution
- Reduced metabolism
- Reduced renal elimination

Particular care is necessary in administration of drugs where sensitivity in the elderly is increased e.g.:

- Hypno-sedatives
- Neuroleptics
- Diuretics

Where no drug is needed avoid unnecessary prescriptions.

Relevant drugs should be prescribed in the appropriate dose and monitored closely

Consideration should be given to the formulation that is most appropriate in the clinical circumstances

The possibility of drug-drug interactions should always be borne in mind

Pregnancy and Lactation

Changes in fluid and tissue composition occur during pregnancy

Reduced gastrointestinal motility delays gastric emptying and may delay drug absorption after oral administration

Vasodilation may result in enhanced absorption following drug administration by the intramuscular route

There is increased volume of distribution, increased hepatic metabolism and increased elimination of drugs

Extreme care must be taken when administering drugs with teratogenic potential to women in the reproductive age group (See appendix IV)

Some drugs may cause harm to infants when administered to nursing mothers (see appendix V)

Other drugs e.g. bromocripine inhibit lactation Drugs excreted significantly in milk and likely to cause toxicity are shown in appendix V

CHAPTER 22:

NOTIFIABLE DISEASES

List of Notifiable diseases

1. AIDS
2. Anthrax (human)
3. Brucellosis (human)
4. Cerebro-spinal meningitis
5. Chicken pox
6. Cholera
7. Diarrhoea (simple without blood)
8. Diarrhoea with blood (dysentery)
9. Diphtheria
10. Dracuncolasis
11. Filariasis
12. Food poisoning
13. Gonorrhoea
14. Hepatitis
15. Lassa Fever
16. Leprosy
17. Louse-borne typhus fever
18. Malaria
19. Measles
20. Onchocerciasis (River blindness)
21. Ophthalmia neonatorum
22. Pertussis (Whooping cough)

23. Plague
24. Pneumonia
25. Poliomyelitis
26. Rabies (human)
27. Schistosomiasis
28. Smallpox
29. Syphilis
30. Other sexually transmitted diseases (STD)
31. Tetanus (other)
33. Tetanus (neonatal)
33. Trachoma
34. Trypanosomiasis (sleeping sickness)
35. Tuberculosis
36. Typhoid and paratyphoid fevers
37. Viral influenza
38. Yaws
39. Yellow fever

List of emergency and immediate notifiable disease

1. AIDS (Acquired Immune Deficiency syndrome)
2. Acute Flaccid Paralysis
3. Anthrax
4. Cerebro-spinal Meningitis (CSM)
5. Cholera
6. Lassa fever
7. Plague
8. Rabies (human)
9. Small pox

10. Typhoid and paratyphoid fevers
11. Yellow fever

APPENDICES

APPENDIX I

WHO CLINICAL STAGING OF HIV FOR INFANTS AND CHILDREN WITH ESTABLISHED HIV INFECTION

Clinical Stage 1

Asymptomatic

Persistent generalized lymphadenopathy

Clinical Stage 2 (I)

Unexplained persistent hepatosplenomegaly

Papular pruritic eruptions

Fungal nail infections

Angular cheilitis

Lineal gingival erythema

Extensive wart virus infection

Extensive molluscum contagiosum

Recurrent oral ulceration

Unexplained persistent parotid enlargement

Herpes zoster

Recurrent or chronic upper respiratory tract infections (otitis media, otorrhoea, sinusitis, tonsillitis)

Clinical Stage 3 (I)

Unexplained moderate malnutrition or wasting not adequately responding to standard therapy Unexplained persistent diarrhoea (14 days or more)
Unexplained persistent fever (above 37.6 °C, intermittent or constant, for longer than one month)
Oral hairy leukoplakia
Acute necrotizing ulcerative gingivitis or periodontitis
Lymph node tuberculosis
Pulmonary tuberculosis
Severe recurrent bacterial pneumonia
Symptomatic lymphoid interstitial pneumonitis
Chronic HIV-associated lung disease including bronchiectasis
Unexplained anaemia (<8.0 g/dl), neutropaenia (<0.5 x 109/L3) and or chronic thrombocytopenia

Clinical stage 4 (i) (ii)
Unexplained severe wasting, stunting or severe malnutrition not responding to standard therapy Pneumocystis pneumonia Recurrent severe bacterial infections (e.g. empyema, pyomyositis, bone or joint infection, meningitis, but excluding pneumonia) Chronic herpes simplex infection; (orolabial or
cutaneous of more than one month's

duration, or visceral at any site Extrapulmonary tuberculosis Kaposi sarcoma

Oesophageal candidiasis (or Candida of trachea, bronchi or lungs)

Cytomegalovirus infection; retinitis or cytomegalovirus infection affecting another organ, with onset at age over 1 month Central nervous system toxoplasmosis (after the neonatal period)

Extrapulmonary cryptococcossis (including meningitis)

HIV encephalopathy

Disseminated endemic mycosis (extrapulmonary histoplamosis, coccidiomycosis) Chronic cryptosporidiosis (with diarrhoea) Chronic isosporiasis

Disseminated non-tuberculous mycobacteria infection

Cerebral or B cell non-Hodgkin lymphoma Progressive multifocal leukoencephalopathy HIV-associated cardiomyopathy or nephropathy

(i) Unexplained refers to where the condition is not explained by other causes

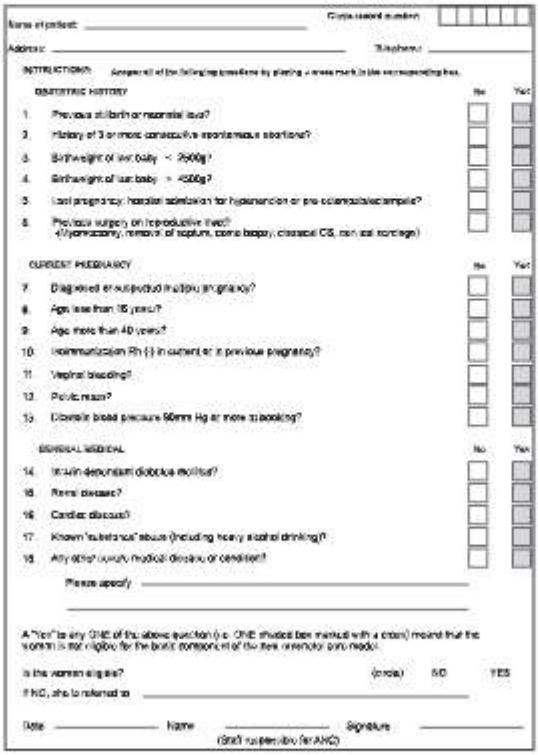
(ii) Some additional specific conditions can be included in regional classifications (e.g. Disseminated Penicilliosis in Asia, HIV-associated rectovaginal fistula in Africa), and reactivation of American trypanosomiasi

APPENDIX II

WHO NEW ANTENATAL CARE MODEL CLASSIFYING FORM 2001

Figure 2: CLASSIFYING FORM

Criteria for classifying women for the basic component of the new antenatal care model



Name of patient: _____ Clinic record number: _____

Address: _____ Telephone: _____

INSTRUCTIONS: Assessment of the following questions by placing a cross mark in the corresponding box.

OBSTETRIC HISTORY	No	Yes
1. Previous stillbirth or neonatal loss?		
2. History of 3 or more consecutive spontaneous abortions?		
3. Birthweight of last baby < 2500g?		
4. Birthweight of last baby > 4500g?		
5. Last pregnancy: hospital admission for hypertension or pre-eclampsia/eclampsia?		
6. Previous surgery on reproductive tract? (Hysterectomy, removal of septum, cone biopsy, classical CS, non-tubal surgery)		

CURRENT PREGNANCY	No	Yes
7. Diagnosed or suspected multiple pregnancy?		
8. Age less than 16 years?		
9. Age more than 40 years?		
10. Haemmunization Rh (-) in current or a previous pregnancy?		
11. Vaginal bleeding?		
12. Pelvic mass?		
13. Diastolic blood pressure 90mm Hg or more at booking?		

GENERAL MEDICAL	No	Yes
14. Insulin-dependent diabetes mellitus?		
15. Renal disease?		
16. Cardiac disease?		
17. Known "substance" abuse (including heavy alcohol drinking)?		
18. Any other severe medical disease or condition?		

Please specify: _____

A "Yes" to any ONE of the above question(s) (i.e. ONE shaded box marked with a cross) means that the woman is not eligible for the basic component of the new antenatal care model.

Is the woman eligible? (cross) NO YES

If NO, she is referred to _____

Date _____ Name _____ Signature _____
(Staff supervisor for ANC)

APPENDIX III

CALCULATION OF DOSAGE REQUIREMENTS IN CHILDREN

Introduction

Medicine doses are generally based on body weight (in kilogram) or the following age ranges:

First one month (neonate)

Up to 1 year (infant)

1 - 5 years

6 - 12 years

Unless the age is specified, the term child includes persons aged 12 years and below

Dose Calculation

Calculated based on body weight (in kilogram) or the body surface area (in m2). Use this rather than attempting to calculate doses on the basis of doses used in adults

Body Surface Area (BSA) estimates are more accurate for calculation of paediatric doses- Many physiological phenomena correlate better to BSA

For most medicines the adult maximum dose should not exceeded

For example if the dose is stated as 4 mg/kg (max. 180 mg), a child weighing 10 kg should receive 40 mg but a child weighing 50 kg

should receive 180 mg and not
200mg

Young children may require higher doses per kilogram than adults because of their higher metabolic rate Calculation by body weight in an overweight child may result in much higher doses being administered than necessary. Such doses should be calculated based on idea body weight in relation to height and age. See table below.

Age	Ideal body weight		Height		Body Surface
	Kg	lb	cm	Inch	
Newborn*	3.5	7.7	50	20	0.23
1 Month*	4.2	9	55	22	0.26
3 Month*	5.6	12	59	23	0.32
6 Month	7.7	17	67	26	0.40
1 year	10	22	76	30	0.47
3 years	15	33	94	37	0.62
5 years	18	40	108	42	0.73
7 years	23	51	120	47	0.88
12 years	39	86	148	58	1.25

The figures relate to full term and not preterm infants who may need reduced dosage according to their clinical condition

APPENDIX IV

MEDICINES WITH TERATOGENIC POTENTIAL

Medicine	Comment
Antiepileptics	Risk of teratogenicity greater if more than one medicine used
Bleomycin	Avoid (teratogenic and carcinogenic in animal studies)
Busulfan	Avoid (teratogenic in animals)
Carboplatin	Avoid (teratogenic and embryotoxic in animal studies)
Cisplatin	Avoid (teratogenic and embryotoxic in animal studies)
Co-trimoxazole	Teratogenic risk (trimethoprim -a folate antagonist)
Cytarabine	Avoid (teratogenic in animal studies)
Dactinomycin	Avoid (teratogenic in animal studies)
Daunorubicin	Avoid (teratogenic and carcinogenic in animal studies)
Doxorubicin	Avoid (teratogenic and toxic in animal studies)
Sulfadoxine/ pyrimethamine	Possible teratogenic risk (pyrimethamine is a folate antagonist)
Griseofulvin	Avoid (fetotoxicity and teratogenicity in animal studies)
Hydroxocarbamide (hydroxyurea)	Avoid (teratogenic in animal studies)
Idoxuridine	Avoid (teratogenic in animal studies)
Isotretinoiin	Avoid (teratogenic in animal studies)
Lithium salts	Avoid (teratogenic in animal studies)
Phenytoin	Avoid (teratogenic in animal studies)
Trenitoin	Avoid (teratogenic in animal studies)

Trimethoprim	Teratogenic in animal studies
Vinblastine	Teratogenic
Vincristine	Avoid if possible (risk of teratogenicity) Congenital malformation (screening advised) Teratogenic Teratogenic risk (folate antagonist) Avoid (limited experience suggest fetal harm; teratogenic in animal studies) Avoid (teratogenicity and fetal loss in animal studies)

APPENDIX V

MEDICINES THAT COULD CAUSE HARM WHEN ADMINISTERED TO BREASTFEEDING MOTHERS

Medicine	Comment
Abacavir	Breastfeeding not advised in HIV infection Amount too small to be
Acetazolamide	harmful
Alcohol	Large amounts may affect infant and reduce milk consumption
Amantadine	Avoid; present in milk; toxicity in
Amiloride	infants reported
Aminophylline	Manufacturer advises avoid
Amitryptilline	Present in milk- irritability in infants
Amlodipine	reported
Amoxicillin	Manufacturers advise avoid
Amphetamines	Manufacturers advise avoid
Amprenavir	Trace amounts in milk
	Significant amount in milk
	Breast feeding not advised in HIV infection
Aspirin	Avoid- possible risk of Reye's syndrome; regular use of high doses could impair platelet function and
Androgens	produce hypoprothrombinaemia in infants if neonatal vitamin K stores low
Antihistamines	Avoid. May cause masculinization in the female infant or precocious
Antipsychotics	development in the male infant; high doses suppress lactation
Apomorphine	Anticoagulants, oral Risk of haemorrhage; increased by Vitamin

Atenolol	K deficiency; warfarin appears safe but phenindione should be avoided
Atropine	Significant amounts of some
Azithromycin	antihistamines present in milk,
Barbiturates	although not known to be harmful
Benzodiazepines	Avoid unless absolutely necessary
Beta-blockers	Manufacturer advises avoid
Caffeine	Toxicity due to bet -blockage. Avoid
Captopril	or use with caution (monitor infant)
Carbimazole	Manufacturer advises caution
Ceftriaxone	Present in milk; manufacturer
Cefuroxime	advises use only if no suitable alternative
Chloramphenicol	Avoid if possible.
Chlorpromazine	Large doses may produce
Ciprofloxacin	drowsiness
	Avoid if possible
Contraceptives, oral	Monitor infant; possible toxicity due to beta-blokade
Corticosteroids Co-trimoxazole	Regular intake of large amounts can affect infant
	Manufacturers advise avoid
Cyclophosphamide	Use lowest effective dose

APPENDIX VI

ATYPICAL PRESENTATION OF ILLNESS IN OLDER ADULTS

Introduction

Diseases in older persons may not present with 'typical' features

Major disease may manifest with only behaviour change or loss of Activities of Daily Living (ADLs)

Core features of disease usually seen in younger adults may be missing

Presentation may be:

- Vague
- Altered
- None at all (illness not reported)

Aetiology

Typical features of disease are confounded by decline in physiological reserve, multi-morbidities and/or coexistence of geriatric syndromes

Factors underlying atypical presentation of illness in the elderly

- Cognitive impairment
- Functional impairment
- Increasing age
- Multi-morbidity
- Polypharmacy

Examples of atypical disease presentations

- Acute onset confusion (delirium)
- Anorexia
- Atypical laboratory results
- Generalized weakness
- Falls
- Fatigue
- New onset urinary incontinence
- New onset decline in ADLs (e.g. difficulty with walking)
- No fever
- No pain
- Pain at site different from expected

Illness presenting atypically	Presentation
Acute abdomen	Absent symptoms
	Constipation
	Mild discomfort
	Tachypnoea
	Vague respiratory symptoms
Cognitive impairment	Changes outside language and memory domains may be missed
	Behavioural changes may be misdiagnosed as primary psychiatric illness
Depression	Hyperactivity
	No sadness
	Sadness misunderstood as 'normal ageing'
	Sleep disturbances
	Vague symptoms

Heart failure	Anorexia, fatigue may overshadow breathlessness
Infections	Anorexia
	Confusion
	Decreased fluid intake
	Falls
	Functional decline
	No fever
	No leukocytosis
Malignancy	Abdominal masses may not be detected
	Back pain from metastases
Myocardial infarction	Fatigue
	Functional decline
	Nausea
	No chest pain
	No ECG changes
	No fever
	No leukocytosis
Non-dyspnoeic pulmonary oedema	Shortness of breath more common than chest pain
	Anorexia
	Classical PND, cough may be absent
	Confusion
	Functional decline
	Onset may be insidious
Thyroid disease	Agitation, confusion may spell hypothyroidism
	Fatigue, overall 'slowing

Consequences of atypical presentation of disease in the elderly

-Delayed diagnoses
-Missed diagnoses
-Prolonged hospitalization
-Mortality

** These can be mitigated by clinicians being aware that atypical presentation of illness is common in older adults, and being alert to think more broadly when dealing with older adults*

APPENDIX VII

CONFUSION ASSESSMENT METHOD FOR DELIRIUM

The Modified Barthel Index (20-point version)

	Score			
Functioning	0	1	2	3
Mobility	Immobile	Wheelchair independent	Needs help	Independent
Stairs	Unable	Needs help	Independent	
Transfers (e.g. from bed to chair, vice versa)	Unable	Needs major help	Needs minor help	Independent
Bladder	Incontinent	Occasional incontinence	Continent	
Bowels	Incontinent	Occasional incontinence	Continent	
Grooming	Needs help	Independent		
Toilet use	Unable	Needs help	Independent	
Feeding	Unable	Needs help	Independent	
Dressing	Completely dependent	Needs some help	Independent	
Bathing	Needs help	Independent		
Scores below 10: patient is heavily dependent for care				

APPENDIX VIII

ADVERSE DRUG REACTION REPORTING FORM

NATIONAL PHARMACOVIGILANCE CENTRE (NPC) NIGERIA

National Agency for Food and Drug Administration & Control (NAFDAC), Headquarters, Office, Plot 1632 Olusegun Obasanjo Way, Wuse Zone 7, Abuja

FORM FOR REPORTING OF SUSPECTED ADVERSE DRUG REACTIONS

IN STRICT CONFIDENCE

1. * PATIENT'S DETAILS

Full Name or Initials: Patient Record No:

AGE/DATE OF BIRTH: SEX: M F AD/GHT (kg):

HOSPITAL/Treatment Centre:

2. * ADVERSE DRUG REACTION (ADR)

A. DESCRIPTION

C. OUTCOME OF REACTION (TICK AS APPROPRIATE)

Recovered fully | Recovered with disability (specify)

Congenital Abnormality (Specify) | Life Threatening (specify)

Death | Others (specify)

DATE Reaction Started | DATE Reaction Stopped

B. Was Patient Admitted Due to ADR: Yes No

If Already Hospitalized, Was it Prolonged Due to ADR: Yes No

Duration of Admission (days):

Treatment of Reaction:

3. * SUSPECTED DRUG (Including Biologicals Traditional/Herbal Medicines & Cosmetics)

A. DRUG DETAILS (Give name and other details if available / Attach product label / Sample if possible)

Brand Name: Generic Name: Batch No:

NAFDAC No: Expiry Date:

Name & Address of Manufacturer:

B. Indications for Use	Dosage	Route of Administration	Date Started	Date Stopped

4. * CONCOMITANT MEDICINES (All medicines taken within the last 3months including herbal and self medication)

Brand or Generic Name	Dosage	Route	Date Started	Date Stopped	Reason for Use

5. * SOURCE OF REPORT:

Name of Reporter:

Address:

Profession:

Signature: Tel No/E-mail:

* = MANDATORY FIELDS

*Source: NAFDAC

APPENDIX IX

IMMUNIZATION SCHEDULE

Revised EPI Schedule for infants less than 1 year

Contact	Minimum target age of child	Type of vaccine	Dose	Route of administration	Site
1st	At birth	BCG*	0.05ml	Intradermal	Right upper arm
		OPV0**	2 drops	Oral	Mouth
		HepB0***	0.5ml	Intramuscular	Antero-lateral aspect of thigh
2nd	6 weeks	Pentavalent1 (DPT+HepB+Hib)	0.5ml	Intramuscular	Antero-lateral aspect of thigh
		OPV1	2 drops	Oral	Mouth
		PCV1	0.5ml	Intramuscular	Antero-lateral aspect of thigh
		Rota1	1ml	Oral	Mouth
3rd	10 weeks	Pentavalent2 (DPT+HepB+Hib)	0.5ml	Intramuscular	Antero-lateral aspect of thigh
		OPV2	2 drops	Oral	Mouth
		PCV2	0.5ml	Intramuscular	Antero-lateral aspect of thigh
		Rota2	1ml	Oral	Mouth
4th	14 weeks	Pentavalent3 (DPT+HepB+Hib)	0.5ml	Intramuscular	Antero-lateral aspect of thigh
		OPV3	2 drops	Oral	Mouth
		PCV3	0.5ml	Intramuscular	Antero-lateral aspect of thigh
		IPV	0.5ml	Intramuscular	Antero-lateral aspect of thigh
5th	9 months	Measles	0.5ml	Subcutaneous	Left upper arm
		Yellow fever	0.5ml	Subcutaneous	Left upper arm

*BCG dose for children ≥ 12 months is 0.1ml
**OPV0 must be given within first weeks after birth
***HepB0 should be preferably given within 24 hours of birth. If this is not possible, it may be given up to 2 weeks after birth

***Recommended schedule for children with HIV/AIDS**

Vaccine	Asymptomatic HIV infection	Symptomatic HIV infection
BCG	Yes	No
OPV	Yes	Yes
Pentavalent	Yes	Yes
Rota	Yes	Yes
PCV	Yes	Yes
IPV	Yes	Yes
Measles	Yes	Yes
Yellow fever	Yes	No
Tetanus	Yes	Yes
Vitamin A	Yes	Yes

*Note: BCG, Yellow fever, Mumps and Rubella are contraindicated in symptomatic HIV infection

TT immunization schedule for women of childbearing age (15-49 years)

Dose	When to give	Expected duration of protection
TT1	At first contact or as early as possible in pregnancy	None
TT2	At least 4 weeks after TT1	1-3 years
TT3	At least 6 months after TT2 or during subsequent pregnancy within 3 years	5 years
TT4	At least 6 months after TT3 or during subsequent pregnancy	10 years
TT5	At least 6 months after TT4 or during subsequent pregnancy	All the child bearing years

Vitamin A supplementation

Age	Dose	Capsule
6 months	100,000 IU*(30 mg RE**) for 6-11 month old	1 blue capsule or half red capsule (4 drops)
12 months	200,000 IU (60 mg RE) for ≥ 12 month old	1 red capsule or 2 blue capsule

*IU – international units **RE – retinol equivalent

Note: After first dose (6-11 month), repeat Vit A every 6 months until 5 years of age.

General Notes:

1. The manufacturer's instructions should be followed strictly.
2. *The immunization status of every child should be checked at each contact with a health service(including hospital outpatient departments and wards), verified by sighting the immunization card and immunization should be offered as appropriate to eliminate missed opportunities.
3. *Therefore mothers must be encouraged to always bring their children's immunization cards whenever visiting a health facility for any reason (curative or preventive).
4. * Health workers to always request to sight the immunization card while attending to any child.
5. When multiple injectable vaccines are required during the same visit, they should be given at different sites.
6. When a dose is delayed, resume without repeating the previous dose.
7. BCG - for children ≥ 6 months, mantoux test should first be carried out to exclude active infection or previous immunity

before BCG is given (children less than 6 months do not mount sufficient reaction to tuberculin test).

8. Pertussis vaccine – give acellular vaccine in place of whole cell if child is ≥ 3 years.
9. Beside above exceptions, children less than 5 years who are not previously immunized should follow the normal recommended schedule maintaining the minimum intervals between doses.

***Awareness on numbers 2, 3 & 4 above to be created nationwide.**