standard treatment guidelines

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FOREWORD

I am indeed very pleased to write the foreword to this maiden edition of the Standard Treatment Guidelines (STG) for the Nigerian health care system. I am aware that the process of its production began in 2005 involving contributions and recommendations of various experts and stakeholders in the health care sector.

The STG is an important tool for the attainment of comprehensive and effective health care delivery services thereby achieving the goals of the National Drug Policy, which inter alia are: the availability of safe, efficacious and affordable medicines to satisfy the healthcare needs of the majority of the population and ensure the rational use of drugs. The fulfillment of the above mentioned goals is part of the strategic thrust of the Health Sector Reform Programme aimed at the reduction of disease burden and the improvement of access to quality health services. It is expected that the STG will become a major reference document for all health workers both in the public and private sectors.

It is instructive to note that the development of the STG followed due process with wide consultations and meetings involving various stakeholders and interest groups. The document that has come out of this process is a reflection of the quality of the inputs that went into its development. In my opinion, this maiden edition of the STG has been produced and serialized in such a way as to assist health care providers especially doctors in the effective discharge of their duties as prescribers. It will also ensure discipline as only those medicines recommended will be prescribed for patients within a given health facility.

I commend all those who worked tirelessly towards the completion of this maiden edition STG. Special mention and gratitude must go to the World Health Organization (WHO) for sponsoring and providing sustained technical support to the committee. Without this support, this STG would not have seen the light of the day.

Finally, let me quickly add that this STG must be widely circulated and disseminated. Everything possible must be done to ensure that practitioners maximize the benefit of such a useful document. If it has worked in other parts of the world, it should also work in Nigeria. It must also be subjected to regular reviews in view of the dynamic nature of health care management.

Dr. Hassan Muhammed Lawal, CON
Supervising Minister of Health

PREFACE

This first edition of Standard Treatment Guidelines (STG) for the Nigerian health practitioner is coming relatively later than those of many other countries. It is indeed a welcome development.

The standard of medical practice and the wage bill of health services are usually remarkably improved by health personnel putting to use STG. This among other benefits can only lead to improved health of the community.

In Nigeria our health indices are among the worst in the world. Our country Nigeria does not lack the manpower or the necessary infrastructure to turn things around. What appears to be lacking is the organization of health services required to put both to optimal use. Efforts such as the actualization of our own national STG and the various health reforms currently in progress will definitely improve our situation.

It is therefore my pleasure and privilege to write the preface to this maiden edition of the STG. This is the outcome of a long journey that started several years ago. The previous chairmen of the National Formulary and Essential Drugs Review Committees made efforts to start the project but were unsuccessful due to lack of funds.

The current committee had the luck of being assisted by the country office of the World Health Organization (WHO) in not only this endeavor but in the preparation and printing of the last edition of the Nigerian Essential Medicines List. The desk officer, Dr Ogori Taylor showed great commitment to the project and the country owes a debt of gratitude to WHO.

In preparing this document every effort was made to ensure that the stakeholders own the project so that it is not seen as an imposition. Accordingly, the major contributions came from various practitioners and their associations as well as from many practitioners whose input were judged crucial to the success of the project. We also adopted the acceptable practices in the field that were in use by special health projects such as HIV/AIDS, Malaria, TB/Leprosy programmes etc. The academia was also involved. There were several fora where the contributions were discussed openly with the stakeholders and consensus arrived at.

It is my hope therefore that this document will be widely used by Nigerian health practitioners. I salute the contributors and those that helped in one way or the other. The committee of course accepts responsibility for any lapses but also hopes that these would be brought to our attention for correction in subsequent editions.

Professor Ibrahim Abdu-Aguye,MBBS; FMCP;SFIAM; FIICA;D. Sc (Hon)
Chairman, National Formulary and Essential Drugs Review Committee.

TABLE OF CONTENTS

SECTION A

Section B

Section C

APPENDICES

SECTION A

AMOEBIASIS

Introduction

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

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

Space-occupying lesion in the brain

Right inguinal mass

Investigations

Stool: microscopy for cysts and motile organisms (amoebic dysentery)

Full Blood Count

Chest radiograph (in amoebic liver abscess)

Abdominal Ultrasound Scan

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

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

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

Notable adverse drug reactions, caution

Metronidazole is contraindicated in pregnancy.

Avoid alcohol during treatment 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 a mild, self-limiting diarrhoea lasting 2-3 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.

- Amoxicillin 500 mg 8 hourly for 5 days

Or:

- Cotrimoxazole 960 mg 12 hourly for 3-5 days

Or:

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

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 diarrhoea

Severe life-threatening diarrhoea 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: 125 mg 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 - 12 years*:* 1 - 2 tablets 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

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_2 receptor antagonist
- 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

Or:
- Metronidazole 400 mg orally every 8 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

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_{12} deficiency

Complications

Diseases related to Vitamin B_{12} 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: 2 g orally daily for 3 days or 400 mg 8 hourly for 5 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; repeat after 1 week
Child: 50 to 75 mg/kg as a single dose; repeat after 1 week

Supportive

Vitamin B_{12} supplementation
Avoidance of milk

Notable adverse drug reactions
Metallic taste and vomiting from metronidazole
Prevention
Good sanitary habits
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
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: 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 intaket/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 (generally 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
<u>*H. pylori* eradication</u>
Triple therapy with:
- Metronidazole 400 mg orally every 8 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
Or:
- Clarithromycin 500 mg orally every 12 hours for 7 days
Plus:
- Amoxicillin 1g orally every 12 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
<u>Mild upper gastrointestinal bleeding</u>
Intravenous omeprazole 20 mg 12 hourly for 5 days then standard triple therapy
<u>Severe upper gastrointestinal bleeding</u>
Interventional endoscopic treatment
Blood transfusion
Surgery
<u>Perforation</u>
Surgery
<u>Gastric outlet obstruction</u>
Rest the gut
Surgery

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

<u>Bleeding peptic ulcers/erosions</u>

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

<u>Bleeding varices</u>

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

Injection sclerotherapy: equal volume mixture of 3% sodium tetradecyl sulfate, 98% ethanol, sodium chloride 0.9% injection (2-5 mL/site; maximum 50 mL)

Variceal band ligation

Radiologic therapy

Venous embolization

Transjugular Intrahepatic Portosystemic Shunt (TIPS)

Oesophageal transection and devascularization

Liver transplant

<u>Peptic ulcers/erosions/tumours</u>

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

<u>Peptic ulcers/erosions related upper gastrointestinal bleeding</u>

Avoid NSAIDs.

Treat *H. pylori* infection

<u>Oesophageal varices</u>

β blockers (propranolol 40 mg orally 12 hourly and titrate up to 160 mg depending on the heart rate)

Maintenance sclerotherapy

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:

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

Clinical features

Acute hepatitis:

Mild-to-moderate jaundice

Vague 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

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

Non-drug treatment

High carbohydrate and low protein diet

Discontinuation of hepatotoxic medication

Bed rest

Drug treatment

<u>Hepatitis A</u>

Self-limiting disease. No specific drug treatment

<u>Hepatitis B</u>

Acute:

Self-limiting to fulminant

Treatment is supportive

Chronic:

Interferon alfa -2b: 10 million units subcutaneously three times weekly for 4 months

Lamivudine: 100 mg orally daily for 1year

Liver transplant

Chronic Hepatitis C:

Interferon alfa -2b

- 3 million units subcutaneously 3 times weekly for 4 months

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

<u>Hepatitis D</u>

Interferon alfa -2b: 3 million units subcutaneously 3 times weekly for 4 months

Plus:

Lamivudine: 100 mg orally once daily for 4 months

<u>Hepatitis E</u>

Largely supportive

Notable adverse drug reactions

Interferon alpha 2b and Ribavirin haematopoietic toxicity

Flu-like illness

Leucopenia

Psychiatric-like symptoms

Development of early resistance if therepy exceeds 1 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

HEPATIC ENCEPHALOPATHY

Introduction

A state of disturbed central nervous system function as a result of hepatic insufficiency

Characterized by changes in personality, cognition, motor function, level of consciousness

One-year survival rate is 40%

Nitrogenous substances, particularly ammonia, reach the brain via portosystemic shunts leading to alteration of neurotransmission

Predisposing factors

Reduced blood supply to the liver

Infection of the liver

Bleeding into the gut

Electrolyte imbalance (hypokalaemia and hypomagnesaemia)

Poor bowel evacuation

Clinical features

Cognitive abnormalities: may be mild and recognizable only with psychometric testing but may be severe with frank confusion, altered level of consciousness and coma

Hyper-reflexia

Fetor hepaticus

Insomnia

Flapping tremor (asterixis)

Differential diagnoses

Intracranial lesions (haemorrhage, tumour, abscess etc.)

CNS infections (encephalitis, meningitis)
Other metabolic encephalopathies (uraemia, hyper/hypoglycaemia etc.)
Hypertensive encephalopathy
Alcohol intoxication
Drug toxicity e.g. sedatives, heavy metals

Investigations
As appropriate to identify possible precipitating factors
Full Blood Count
Urea, Electrolytes and Creatinine,
Blood sugar
Microscopy and culture of the stool and blood

Treatment objectives
Reverse neuropsychiatric symptoms
Minimize nitrogenous substances
Treat precipitating factors

Drug treatment
Lactulose syrup (10 g/15 mL)
- Initially 30 - 45 mL orally three times daily titrated to either the resolution of symptoms or production of three soft stools per day

Or:
- As rectal retention enema 300 mL in 1 litre water retained for 1 hour; duration usually for days or weeks

Or:
Metronidazole 800 mg orally 12 hourly
Treat underlying cause(s) e.g hypokalaemia, anaemia, infection

Supportive measures
High carbohydrate, low protein diet
Adequate hydration
Rectal wash out

Notable adverse drug reactions
Lactulose: excess gas, diarrheoa
Metronidazole: peripheral neuropathy, dysgeusia

Prevention
Avoid precipitating factors

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
With or without 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 - 6 g orally 6 hourly in severe obstructive jaundice

Phenobarbital in neonatal jaundice
- 5 - 8 mg/kg orally daily

Notable adverse drug reactions
Colestyramine: diarrhoea
Phenobarbital may cause dose-dependent respiratory depression

Surgical treatment
<u>Obstructive jaundice</u>
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
PT, PTTK, Serum albumin
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
<u>Ascites and pedal oedema</u>
Spironolactone tablets 25 - 100 mg orally 12 hourly
Furosemide 20 - 80 mg orally 12 hourly
Salt-poor albumin for intractable ascites

<u>Prevention of variceal bleeding</u>
Propranolol 40 - 80 mg orally daily

<u>Replacement of damaged liver</u>
Liver transplant

<u>Prevention of encephalopathy</u>
Lactulose 30 mL orally twice daily
- Doses to be titrated upward until at least 3 bowel motions daily are achieved

Saline rectal enema

Prevention
Immunization against hepatitis B, C
Abstinence from alcohol

NUTRITIONAL DISORDERS

KWASHIOKOR AND MARASMUS

Introduction
Adequate nutrition is the intake and utilization of energy-giving and body building foods and nutrients, to maintain well-being, and productivity.
"Malnutrition" includes generalized malnutrition that manifests as stunting, underweight, wasting (kwashiorkor and marasmus), obesity as well as deficiencies of micronutrients.
Kwashiorkor is protein-energy malnutrition .
Marasmus is malnutrition resulting from inadequate calorie intake.

Obesity is a commonly nutritional disorder (results from excessive intake of calories).

Epidemiology
High percentages 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, etc.
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 percentages 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_1 (thiamine): beri-beri
- B_2 (riboflavin): scrotal and vulval dermatoses, angular stomatitis, scars, magenta tongue, cheilosis
- B_6 (niacin): scarlet and dry tongue, pellagra
- 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
Waist circumference: determination of central fat distribution
BMI is calculated as follows

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

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
BMI represents overall adiposity

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: measure 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
CNS disease that affects hypothalamic function

Complications

Cardiovascular:
Coronary 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
- 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: BLOOD AND BLOOD-FORMING ORGANS

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

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

Morphological classification

Macrocytic
Megaloblastic
- Folic acid deficiency
- Vitamin B_{12} deficiency
- 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 B_{12}
- 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
Death

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, 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

<u>Iron deficiency</u>

Oral iron therapy:
- Ferrous sulfate 200 mg (containing 65 mg of iron) 1 tablet 2-3 times daily

Treat for 3- 6 months to correct deficits in haemoglobin and in stores
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
Dose in mL (of 50 mg/mL preparations) = [Patient's wt. in kg X (14 Hb in g/dL)] ÷ 10

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
A test dose should be administered: 25 mg intramuscularly or by intravenous infusion over 5 to 10 minutes
Total-dose infusion should be avoided in patients with history of allergy

<u>Megaloblastic anaemia</u>

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 4ºC 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)

<u>Whole blood preparations</u>

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

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 complictated 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 disoders:

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^2 intravenously on days 8, 15, 22 and 29

Vincristine 1.4 mg/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/m^2 intravenously on days 12, 15, 18, 21, 24, 27, 30 and 33

Or:

COAP Regime

Cyclophosphamide 650 mg/m^2 intravenously on days 1 and 8; 14 and 22

Vincristine 1.4 mg/m^2 intravenously to a maximum of 2 mg: days 1 and 8; 14 and 22

Cytosine Arabinoside 50 mg/ m^2 subcutaneously 12 hourly for 12 days or bolus intravenous injection 100 mg/m^2 daily for 7 days

Prednisolone 40 mg/m^2 oral 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 = 1x10^9/L and platelet count is = 100 x10^9/L

Maintenance

6-Mercaptopurine 75 mg/m^2 orally daily

Methotrexate 20 mg/m^2 orally weekly

- For 3 years if remission is maintained, otherwise re-assessment

Pulse therapy (Intensification)

To be given every 3 months with

- Vincristine 1.4 mg/ m^2 to 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 1 and 2, and 100 mg/m^2 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^2 intravenously 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 differented 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 have 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% in marrow

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

Thrombocytosis (>1000 x 10^9/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

Low grade 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/m^2 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 is 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 lymphoproliferative 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/m^2 intravenously on day 1

Doxorubicin 50 mg/m^2 intravenously on day 1

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^2 intravenously on days 1 and 8

Doxorubicin 25 mg/m^2 intravenously 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 4

Prednisolone 40 mg orally on days 1 - 14

ChIVPP

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-ß thalassaemia (Sß thal)

Sickle cell-ß+ thalassaemia Type I (Sß+thal.Type I)

Sickle cell-ß+thalassaemia. Type II. (Sß+thal. Type II)

Sickle cell-ß+thalassaemia. Type III. (Sß+thal. Type III)

Sickle cell trait

Inheritance of one normal gene controlling formation of ß 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 ß 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)
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 4 g daily
Child: 1 - 5 years 120 - 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 20 mg/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

CHAPTER 3: CARDIOVASCULAR SYSTEM

ANGINA PECTORIS

Introduction

A symptom complex characterised by chest pain or discomfort caused by transient myocardial ischaemia usually due to coronary heart disease

Less common in this environment though current studies show increasing prevalence

In 90% (or more) of cases there is a hereditary factor

Major risk factors:

- Hypertension
- Diabetes mellitus
- Hypercholesterolemia
- Smoking
- Obesity
- Male sex
- Age

Clinical features

Stable angina (chest discomfort on exertion and relieved by rest)

Unstable angina (discomfort on exertion and at rest)

Myocardial infarction (chest pain or discomfort that lasts more than 30 minutes; may be associated with symptoms of cardiac failure, shock, arrhythmias)

Differential diagnoses

- Myalgia
- Pericarditis
- Aortic dissection
- Pleurisy

Complications

- Cardiac failure
- Myocardial infarction
- Arrhythmias
- Sudden death

Investigations

- Full Blood Count and differentials
- Urea, Electrolytes and Creatinine
- Fasting blood glucose
- Urinalysis; urine microscopy
- Electrocardiograph: resting, treadmill exercise
- Echocardiography (resting/exercise)
- Radio nuclide studies
- Cardiac enzymes (CK-MB)
- Coronary angiography

Treatment objectives

- Relieve discomfort
- Improve quality of life
- Prevent complications
- Relieve the obstruction
- Address the risk factors present

Non-drug treatment

- Dietary manipulation (low salt, low cholesterol diet)
- Exercise
- Stop smoking
- Reduce alcohol consumption

Drug treatment

ß blockers

- Atenolol 50 - 100 mg daily

Nitrates

- Glyceryl trinitrate 0.3 - 1 mg sublingually, repeated as required

Or:

- Isosorbide dinitrate 30 - 120 mg orally daily (up to 240 mg)

Calcium channel antagonists

- Verapamil 80 - 120 mg orally 8 hourly

Anti-platelets

- Aspirin (acetylsalicylic acid) 75 mg orally daily

Unstable angina

Treat as for acute myocardial infarction

Other measures

- Angioplasty (PTCA)
- Coronary artery bypass graft (CABG)
- Treat/reduce risk factors
- Patient education (very important)

Notable adverse drug reactions, caution and contraindications

ß blockers

- Bradycardia
- Caution in asthmatics and patients with chronic obstructive airways disease because of bronchoconstriction.

Nitrates: hypotension

Calcium channel antagonists: hypotension

Aspirin, thrombolytics: bleeding

- Avoid in recent stroke and in upper gastrointestinal bleeding

Avoid concurrent use of ß-blockers with verapamil

Prevention

- Nutrition education
- Address risk factors
- Healthy living

CARDIAC ARRHYTHMIAS

Introduction

Conditions in which cardiac rhythms become abnormal

Usually complicate acquired and congenital heart diseases

- Abnormal arrangements of the cardiac impulse fibres or fibrosis affect the conduction fibres

Clinical features

Mild arrhythmias might go unnoticed

May present with:

- Palpitations
- Sudden collapse
- Dizziness
- Syncope
- Near-syncope

- May be complicated by cardiac failure, stroke, etc

Differential diagnoses

- Sinus arrhythmias
- Anxiety

Complications

- Cardiac failure
- Stroke
- Peripheral embolic phenomena
- Sudden death

Investigations

Electrocardiograph (resting, 24 hour Holter, 1 month Holter monitoring)

- Urea, Electrolytes and Creatinine
- Echocardiography
- Electrophysiology

Treatment objectives

- Abolish the arrhythmias
- Treat complications
- Prevent further arrhythmias

Non-drug treatment

- Pacemaker insertion
- Ablation (electrophysiology)
- Cardioversion: acute arrhythmias

Drug treatment

- Depends on the type of arrythmia
- Refer to a specialist for appropriate management

Supportive measures

- Patient education
- Efficient systems to facilitate patient recovery

Notable adverse drug reactions

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

Prevention

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

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

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

Brought about by:

- Hyper-coagulable states

Long periods of immobilization e.g. cardiac failure, following surgery, long-distance travel, etc

- Malignancies

Clinical features

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

Differential diagnoses

- Cellulitis
- Infarctive crisis in sicklers
- Abscess (myositis)

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

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 up with the metabolic demands of the body

The commonest cause in Nigeria is hypertension

Other causes include dilated cardiomyopathy and rheumatic heart disease

Cardiac failure can be classified as:

Left or right-sided
Congestive
Acute
Chronic

- Chronic cardiac failure is the commonest syndrome encountered in our setting

Clinical features

Difficulty with breathing on exertion
Paroxysmal nocturnal dyspnoea
Orthopnoea
Cough productive of frothy sputum
Leg swelling
Abdominal swelling

The prominence of particular symptoms will depend on which side is affected

Signs include:

Oedema
Tachycardia (about 100 beats per minute)
Raised jugular venous pressure
Displaced apex
S3 or S4 or both (With or without murmurs)
Chest: with or without crepitations
Abdomen: hepatomegaly

Differential diagnoses

Bronchial asthma
Chronic obstructive airways disease (COAD)
Renal failure
Liver failure

Complications

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

Investigations

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

Treatment objectives

Relieve symptoms
Enhance quality of life
Prevent complications
Prolong life

Non-drug treatment

Bed rest
Low salt diet
Exercise (within limits of tolerance)

Drug treatment

Digoxin

- 125 - 250 micrograms daily (the elderly may require 62.5 - 125 micrograms daily)

Diuretics

- Furosemide 40 - 80 mg intravenously or orally

Or:

- Bendroflumethiazide 5 mg orally daily

Or:

- Spironolactone 25 - 100 mg once, every 8 - 12 hours 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

Or:

Lisinopril 2.5 - 20 mg daily

Venodilators

- Nitrates

Glyceryl trinitrate 0.3 - 1 mg sublingually and repeated as required

Ionotropes

- 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 β 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

A clinical syndrome in which there are high lipid levels: cholesterol, or its fractions, or triglyceridaemia

Can be primary (hereditary) or secondary - as a result of other diseases

Incidence in Nigeria is thought to be low but recent studies show increasing incidence in association with diabetes mellitus and hypertension

A major risk factor for ischemia heart disease

Clinical features

Patients present with complications of hypertension, ischaemic heart disease or the cause of secondary 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

Urea, Electrolytes and Creatinine
Fasting blood glucose
Lipid profile
Urine proteins
Serum proteins (total and differential)

Treatment objectives

Lower lipid levels
Prevent complications
Treat complications

Non-drug treatment

Stop smoking
Reduce weight
Exercise moderately and regularly
Water soluble fibre: oat, bran

Drug treatment

Fluvastatin

- Initially 20 mg orally once daily at bedtime
- Adjust dose at 4-week intervals as needed and tolerated
- Maintenance 20 - 40 mg orally once daily in the evening
- A 40 mg daily dose may be split and taken every 12 hours

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 - 3 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 (taken three times on at least two different occasions with intervals of at least 24 hours)

Blood pressure ≥ 140/90 mmHg irrespective of age is regarded as hypertension

The commonest non-communicable disease in Nigeria

The commonest cause of cardiac failure and stroke

Hypertension may be:

Diastolic and systolic
Diastolic alone
Isolated systolic

Clinical features

Largely is asymptomatic until complicated ("silent killer")

Non-specific symptoms: headache, dizziness, palpitations etc

Other symptoms and signs depending on the target organs affected e.g. cardiac or renal failure, stroke etc

Differential diagnoses
White coat hypertension
Anxiety/fright/stress
Complications
Heart:
- Heart failure, ischaemic heart disease
Brain:
- Stroke (ischaemic, hemorrhagic)
Eye:
- Hypertensive retinopathy
Kidney:
- Renal failure
Large arteries:
- Aortic aneurysm
Investigations
Full Blood Count
Urinalysis; urine microscopy
Urea, Electrolytes and Creatinine
Uric acid
Fasting blood glucose
Lipid profile
Chest radiograph
Electrocardiography
Echocardiography (not in all cases)
Abdominal ultrasound
Renal angiography (not in all cases)
Treatment objectives
Educate patient about disease and need for treatment adherence
Reduce blood pressure to acceptable levels
Prevent complications (primary, secondary, tertiary)
Rehabilitate
Non-drug treatment (lifestyle modification)
Low salt diet
Achieve/maintain ideal body weight (BMI 18.5 - 24.9 kg/m^2)
Stop smoking
Reduce alcohol intake
Regular moderate exercise
Reduce polysaturated fatty acid intake
Drug treatment
Diuretics:
Thiazides
- Bendroflumethiazide 2.5 - 10 mg orally daily
Or:
- Hydrochlorothiazide 12.5 - 50 mg orally daily
Or:
- Hydrochlorothiazide/amiloride 25/2.5 mg daily
Loop diuretics
- Furosemide 40 - 80 mg orally daily
ß-blockers:
Propranolol 40 - 80 mg orally every 8 - 12 hours
Or:
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
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
Fixed combinations:
Reserpine plus dihydroergocristine plus clopamide 0.25/0.5/5 mg one-two tablets orally daily
Or:
Lisinopril plus hydrochlorothiazide 20/12.5 mg 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
All antihypertensive drugs may themselves cause hypotension
Angiotensin converting enzyme inhibitors, angiotensin receptor blockers: angioedema; cough with ACEIs
Alpha methyldopa, thiazides (and potentially other anti-hypertensive drugs): erectile dysfunction
SLE-like syndrome: hydralazine
Do not use β blockers in asthmatics
Prevention
Weight reduction
Exercise moderately and regularly
Public education
Individual approach
Population approach
Advocacy for the positive lifestyle change

INFECTIVE ENDOCARDITIS

Introduction
A microbial infection of the endocardium and the valves of the heart
May be acute or sub-acute
Some acute cases occur in normal valves or may be part of systemic illness

The sub-acute form usually occurs on damaged valves (e.g. rheumatic heart disease, congenital heart disease), shunts, and atherosclerotic lesions
Causative organisms include staphylococci, streptococci 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)
Echocardiography
Treatment objectives
Stop the infection
Treat cardiac failure
Prevent coagulation disorders
Non-drug treatment
Bed rest
Low salt diet
Drug treatment
Initiate therapy 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 or intramuscularly every 8 hours for 2 weeks

Following bacteriological confirmation institute appropriate antimicrobial therapy
Staphylococci:
Flucloxacillin
- 250 mg - 2 g intravenously every 6 hours for 4 - 6 weeks
Candida:
Systemic antifungals
Notable adverse drug reactions
Penicillin: rashes, anaphylaxis
Gentamicin: nephropathy
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 a penicillin in the last one month:
Amoxicillin
Adult: 3 g 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; 500 mg orally 6 hours later
Child under 5 years: a quarter of adult dose; 5 - 10 years: half adult dose
Or:
Adult: 3 g orally 4 hours before induction, then 3 g orally as soon as possible after the procedure
Child under 5 years: a quarter of adult dose; 5 - 10 years: 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 amoxicillin 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: 1 g intravenously over at least 100 minutes
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

MYOCARDIAL INFARCTION

Introduction

Occurs when an area of heart muscle is necrosed or permanently damaged because of an inadequate supply of oxygen (heart attack)

Reported to be uncommon in Nigeria, although recent reports suggest a rising incidence

Clinical features

Precordial pain: discomfort, heaviness, tightening lasting 30 minutes or more
Shortness of breath
Palpitations
Cough productive of frothy sputum
Signs of right or left-sided cardiac failure and shock

Differential diagnoses

Pulmonary embolism
Aortic dissection
Pericarditis

Complications

Cardiac failure
Ventricular aneurysm
Arrhythmias: heart block, ventricular tachycardia, ventricular fibrillation, atrial fibrillation
Sudden death

Investigations

Full Blood Count; ESR
Urea, Electrolytes and Creatinine
Uric acid
Fasting blood glucose
Lipid profile
Enzyme assays: AST, CK-MB, and LDH
Electrocardiograph monitoring throughout admission
Coronary angiography (in case of secondary angioplasty)

Treatment objectives

Relieve pain (discomfort)
Relieve obstruction
Treat complications
Prevent future episodes

Non-drug treatment

Bed rest
Dietary control (low cholesterol)
Exercise (later)
Weight reduction (later)
Stop smoking

Drug treatment

Aspirin (acetylsalicylic acid) 150 - 300 mg orally stat, then 75 - 150mg daily

Morphine 10 mg by slow intravenous injection over 5 minutes (i.e. 2 mg/minute)

Unfractionated heparin
Adult: 5,000 - 10,000 units (75 units/kg) by intravenous injection as loading dose followed by continuous infusion of 15 - 25 units/kg/hour
- 15,000 units 12 hours by subcutaneous injection
Small adult or child: lower loading dose, then 15 - 25/kg/hour by intravenous infusion, or 250 units/kg every 12 hours by subcutaneous injection
Or:
Lowmolecular weight heparin
- Enoxaparin: 30 mg intravenous bolus (optional) then 1 mg/kg subcutaneously every 12 hours for 7 - 8 days

Thrombolytics
- Streptokinase
Adult: 1,500,000 units by intravenous infusion over 60 minutes, then 250 units over 30 minutes according to condition (with monitoring)
Child: 1 month - 12 years, initially 2,500 - 4,000 units/kg over 30 minutes followed by continuous infusion of 500-1,000 units/kg/hour for up to 3 days until reperfusion occurs
- 12 - 18 years: initially 250,000 units intravenously over 30 minutes, followed by intravenous infusion of 100,000 units/hour for up to 3 days until reperfusion occurs

Recombinant plasminogen activator (*use by specialist physician*)
- Alteplase 15 mg intravenously over 1 - 2 minutes, followed by intravenous infusion of 50 mg over 30 minutes then 35 mg over 60 minutes
(Total dose, 100 mg over 90 minutes; lower doses in patients less than 65 kg

ß blockers
- Atenolol 50 - 100 mg orally daily
- Propranolol 180 - 240 mg orally in 2 - 4 divided doses daily

Angiotensin converting enzyme inhibitors
- Captopril 6.25 - 50 mg orally once, twice or three times daily
- Lisinopril 2.5 - 10 mg daily

Maintenance anti-anginal therapy

Non-drug therapy

Coronary artery bypass graft (CABG)
Secondary or rescue PTCA

Supportive measures

Treat arrhythmias
Oxygen: 100% at 5L/minute

Notable adverse drug reactions, caution

Heparin or streptokinase: bleeding (risk of bleeding in recent stroke, diabetic retinopathy, brain tumours, peptic ulcer disease or surgery)
- Laboratory monitoring is essential: preferably daily, and dose adjusted accordingly

Aspirin: dyspepsia
ß-blockers: bradycardia
- Should be avoided in patients presenting with this symptom

Prevention

Treat hypertension, diabetes mellitus, and hyperlipidaemia
Stop smoking
Nutrition education

MYOCARDITIS

Introduction

Inflammatory process affecting the myocardium

A common disorder; usually occurs in association with endocarditis and pericarditis

Possible causes:
Infections: viral, bacterial, protozoal
Toxins e.g.scorpion sting
Poisons e.g. alcohol
Drugs e.g. chloroquine
Allergy e.g. to 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 cardiac failure

Complications

Cardiac failure
Arrhythmias
Thrombus formation

Investigations

Full Blood Count and differentials
Urea, Electrolytes and Creatinine
Electrocardiography
Echocardiography
Myocardial 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)*

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 EMBOLISM

(Also see in Respiratory system)

Introduction

Blockage of the pulmonary artery or one of its branches by a blood clot, fat, air, or clumped tumour cells

The most common form is thrombo-embolism; occurs when
- A blood clot (generally a venous thrombus) becomes dislodged from its site of formation and embolizes to the arterial blood supply of one of the lungs

The calf veins (deep vein thrombosis) and right ventricle are sources of embolism

Some predisposing factors:

Congestive cardiac failure
Trauma
Surgery
Prolonged immobilization
Malignancies
Stroke

Clinical features

Depend on how massive the embolism is:

No symptoms

Moderate-to-severe cases:

Difficulty in breathing
Chest pain
Sweating
Collapse (shock)
Haemoptysis

Signs:

Small volume pulse
Low blood pressure
Cyanosis
Raised JVP
Cool, clammy skin
Pallor
Tachycardia
Fever
Pleural friction rubs
Loud P2

Differential diagnoses

Lobar pneumonia
Myalgia
Pleuritis (pleurisy)

Complications

Right-sided cardiac failure
Haemorrhagic pleural effusion

Investigations

Full Blood Count and differentials
Electrocardiograph
- Sinus tachycardia
- New onset atrial fibrillation/flutter
- S wave in lead 1, Q wave in lead 3 and an inverted T wave in lead 3
- QRS axis >90º, quite often

Chest radiograph
Blood gasses (arterial)
Ventilation/perfusion lung scanning
Pulmonary artery angiogram

Treatment objectives

Relieve discomfort
Relieve the obstruction(s)
Prevent complications
Prevent further episodes

Non-drug treatment

Bed rest
Mobilization

Drug treatment

Heparin
- 5000 - 10,000 units intravenously stat, followed by 1000 - 2000 units per hour (APTT or INR 1.5 - 2.5 greater than normal)

Or:

Enoxaparin
- 1.5 mg/kg (150 units/kg) subcutaneously every 24 hours, usually for at least 5 days (and until adequate oral anticoagulation is established)

Or:

Warfarin 1 - 5 mg (INR 1.5 - 2) for 6 - 12 weeks (as maintainance after initial parenteral anticoagulation)

Or:

Streptokinase
- 250,000 units over 30 minutes, then 100,000 units every hour for 24 - 72 hours

Or:

Recombinant plasminogen activator (alteplase)
- 10 mg intravenously over 1 - 2 minutes, followed by intravenous infusion of 90 mg over 2 hours

To be used by a specialist physician

Notable adverse drug reactions

Heparin, warfarin or streptokinase: bleeding

Risk of bleeding in:
- Recent stroke
- Diabetic retinopathy
- Brain tumours
- Peptic ulcer disease
- Surgery

Prevention

Low molecular weight heparin for immobilized patients
Early mobilization of patients
Appropriate, moderate and frequent exercises

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

Blood gases
Urea, Electrolytes and Creatinine
Echocardiography
Chest radiograph
Electrocardiography

Treatment objectives

Relieve oedema
Relieve discomfort
Treat underlying cause

Non-drug treatment

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

Aminophylline 250 mg intravenously slowly over 10 - 15 minutes

Treat underlying cause(s)

Supportive measures

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

Notable adverse drug reactions

Aminophylline, digoxin: arrhythmias
Diuretics, ACEIs: hypotension

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 ß- haemolytic streptococcus

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

Antibodies damage the heart(endocardium, myocardium and pericardium)

Commonest streptococcal strains in Africa are C and G

Clinical features

Fever
Arthralgia
Abnormal movements of the hands (upper hands)
Diagnosis: 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)
Previous history of rheumatic fever

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 6 hourly in severe infections
Child: 1 month - 1 year 62.5 mg orally every 6 hours increased in severe infection to ensure at least 12.5 mg/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 infective endocarditis
Benzathine penicillin 720 mg (1.2 million units) intramuscularly 3 - 4 weekly until the age of 25 years (or 10 years after the attack-whichever is longer)
Notable adverse drug reactions
Penicillin: anaphylactic reaction
Salicylates; steroids: peptic ulceration
Cushingoid 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 stenosis and regurgitation
Clinical features
Shortness of breath on exertion
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
Electrocardiography (resting/exercise)
Lipid profile
Echocardiography
Chest radiograph
Coronary angiography
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

NON-PSYCHIATRIC DISORDERS

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
Investigtions
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

Prevention

Avoid precipitants
- These must be identified early for effective prevention

HEADACHES

Introduction

The commonest neurological disease in Nigerian communities
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

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
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 recurrence

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

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 patients with cardiac symptoms
Tricyclic antidepressants: 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 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
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

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
Subdural pus collection (empyema)
Stroke
Epilepsy
Heat stroke
SyndroVme 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

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
- 2 - 4 g daily by intravenous injection or by intravenous infusion over 2 - 4 minutes
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 *H. influenzae* 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 every12 hours for 5 days
Child: 10 mg/kg orally every12 hours for 5 days
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

Can be very incapacitating

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

Prophylaxis

Consider for patients who:
Suffer at least 2 attacks a month
Suffer an increasing frequency of headaches
Suffer significant disability inspite of suitable treatment for acute attacks
Cannot take suitable treatment for acute attacks

Available options are:

Propanolol
- 40 mg orally every 8 - 12 hours

Tricyclic antidepressants, notably amitryptiline
- 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; maintainance 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

ß-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 dopamine in the striato-nigral pathway

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

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 may occur in association with other neurodegenerative diseases

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. subdural haematoma
Dementia
Depression

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

Treatment objectives
Replace dopamine
Ensure mobility and avoidance of falls

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 2nd week; 2.5 mg twice daily in the 3rd week, 2.5 mg three times daily in the 4th 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 (≥ 2) 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)
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
Ménière'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
Arrest convulsions/attacks
Treat underlying cause if identified
Improve quality of life

Drug treatment
Parenteral drugs are recommended for acute attacks/status epilepticus

Diazepam
Adult: 10 - 20 mg by slow intravenous injection; repeat if necessary in 30 - 60 minutes
Child: 200 - 300 micrograms/kg **or** 1 mg per year of age

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 7.5 mg/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 mannitol 20% infusion or furosemide if indicated (see meningitis)

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: 600 mg 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; increase by 250 mg at intervals of 4 - 7 days to doses of 1 - 1.5 g daily (maximum dose 2 g daily)
Child over 6 years: same as adult dose
Up to 6 years: 250 mg daily; increase gradually to 20 mg/kg daily (maximum 1 g daily)

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

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

A condition resulting from disruption of blood supply to brain cells with disability lasting more than 24 hours or resulting in death

Could result from:

Occlusion (ischaemic)

Rupture of blood vessels with bleeding into the brain substance or into the subarachnoid space (haemorrhagic)

Clinical features

Classical stroke:

- Sudden motor weakness, 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

Tentorial herniation with coning and death

Cardiac arrhythmias

Depression

Epilepsy

Dementia

Parkinsonism

Hyperglycaemia

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

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

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

And/Or:

- 20% mannitol 250 mL repeated every12 hours for 4 - 6 doses

Treat underlying conditions such as diabetes mellitus, hypertension, and thrombosis

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

Introduction

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

May be due to:

Vaso-vagal attack

Cardiac causes

Prolonged standing

Severe emotional disturbance

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 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

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

Give aspirin tablets as anti-platelet agent

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: **A**poplexy (stroke)

E: **E**pilepsy

I: **I**nfections e.g. meningo-encephalitis

O: **O**verdosing with drugs, alcohol intoxication, toxins

U: **U**raemia 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
Maintain circulation
Eliminate the cause
Prevent complications: decubitus ulcers, atelectasis, contractures etc
Correct metabolic derangements

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 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

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:
Imipramine 50 - 150 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

- 1st line 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 e.g.

- Nitrazepam 5 -10 mg at night for short term use
- For the elderly, 2.5 - 5 mg
- 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 of 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 5: 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
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 - 3 years: 50 mg orally *every* 8 hour*s* for 3 days; 3 - 7 years: 100 mg *every* 12 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 - 2 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 - 12 years: 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
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
Remove source of infection e.g. fish-bone, other foreign objects
Drain abscess using local anaesthesia
Treat residual infection
Non-drug treatment
Extraction (or endodontic treatment) i.e. root canal therapy
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 days
Child: 1 - 3 years: 50 mg orally 8 hourly for 3 days; 3 - 7 years: 100 mg every 12 hours; 7 - 10 years: half adult dose

ALVEOLAR OSTEITIS

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 anesthesia
Oral contraceptives
Osteosclerotic disease
Radiotherapy
Clinical features
More common in women
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 lamina dura exposed
Differential diagnosis
Osteomyelitis
Complication
Osteomyelitis
Treatment objective
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 inflammation of the soft tissues particularly associated with streptococcal 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 bilateral cellulitis of the sublingual and submandibular spaces
Clinical features
Diffuse, tense, painful swelling of the involved soft tissues
Malaise
Elevated temperature
Ludwig's angina 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-amoxiclavulanate
Adult: 1,000/200 mg intravenoulsly 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 - 5 mg/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 damage to teeth exposed to the saliva
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:

- 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:

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

GINGIVITIS

Introduction

An inflammatory response of the gingivae to plaque bacteria

The most common type is chronic gingivitis

Clinical features

Chronic gingivitis is asymptomatic, low grade inflammation of the gingivae

Gums become red and slightly swollen

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: 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

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

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_2, folic acid deficiency
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 - 10 mL 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 - 6 mg/kg on the first day, then 3 mg/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 gum/flap around a partially erupted tooth

Common around the lower last molars or wisdom teeth
Upper canine may also be affected

Classification

Acute
Chronic
Acute-on-chronic

Aetiology

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

Drug treatment

Appropriate antibiotics
Analgesics
Supportive therapy

Possible complications

Cellulitis
Ludwig's angina
Osteomyelitis

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

A sequela of chronic gingivitis

Symptoms are the same as in the acute type, but with less pain and longer history

Clinical features

Inflammation

Destruction of the periodontal membrane fibres

Resorption of the alveolar bone

Migration of the epithelial attachment along root towards the apex

Pocket formation around the tooth

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 planing

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
- A recumbent position
- 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 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

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)s

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

Diseases which result from inflammation of the salivary glands

- Mumps
- Suppurative parotitis
- Chronic sialadenitis

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

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

TEMPORO-MANDIBULAR 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

Infection
Follows penetrating trauma to joint or spread from middle ear

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 6: DERMATOLOGY

BACTERIAL INFECTIONS

CELLULITIS
Introduction
A 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*
Usually (but not always) follows some discernible wound
Often a complication of immunosuppression like diabetes and HIV/AIDS
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 immunosuppression
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
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 yellow plug
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

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 8 years 250 - 500 mg orally every 6 hours **or** - 1 g 12 hourly for 5-10 days
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 β-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

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

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

<u>Criteria for systemic steroid therapy</u>
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

<u>Guidelines for the use of potent topical steroids in infants</u>
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: 7.5 mg/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 companies
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 concurrent *L. loa* infection: risk of encephalopatic reactions to dying *L. loa* 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 hominis* or 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 blackflies 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

Pediculosis corporis :

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 millimeters 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 - 4 months
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 variously attributed to genetic, climatic, nutritional, ecological and immunological factors

Triggers include:
Streptococcal or viral infections
Emotional crises
Pregnancy and delivery
Trauma (Köebner 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
β- 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

Vitamin D analogue calcipotriol
- Suitable for childhood psoriasis

Combination therapy with calcipotriol and high-potency (Class I) steroids may provide:
- Greater response rates, fewer side effects, and steroid sparing, allowing a shift to a less potent topical steroid or less frequent use of a Class I steroid

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

<u>Small lesions and nail psoriasis</u>

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

<u>Scalp</u>

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 to 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

Aciteritin

Adult: Initially 25 - 30 mg orally daily for 2 - 4 weeks; adjusted according to response. Usual range 25 - 50 mg daily (maximum 75 mg)
- For pustular, erythrodermic and plaque types, and psoriatic arthritis

Child: severe extensive psoriasis resistant to other forms of therapy, palmo-plantar pustular psoriasis

1 month - 12 years: 500 micrograms/kg orally once daily with food or milk; occasionally up to 1 mg/kg/day

To be administered under expert supervision in both adults and children

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
TNF inhibitors (Efaluzimab)
- Indicated for moderate-to-severe chronic plaque psoriasis unresponsive to, or intolerant of other systemic therapy or photochemotherapy
- Initially 700 micrograms/kg by subcutaneous injection then 1 mg/kg weekly
- Discontinue if inadequate response after 12 weeks
- Not recommended for children and adolescents

Adjuvant therapy
Diet: fish oils rich in Ω-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
Vitamin D_3 (calcipotriol):
May irritate the skin (stinging)
Very expensive
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)
Aciteritin:
See Acne- isotretinoin
Tacrolimus:
See Atopic eczema
Efalizumab:
Thrombocytopenia, hepatic and renal impairment. Monitor platelet count during initial herapy, then every 3 months
Contraindicated in immunodeficiency, severe infection, active tuberculosis; history of malignancy; pregnancy and breastfeeding
May cause influenza-like symptoms, leucocytosis, arthralgia, paradoxical exacerbation of psoriasis or development of variant forms including psoriatic arthritis (discontinue treatment)
Expensive

Prevention
Avoid exacerbating factors e.g. abrasions, scatches, harsh fibre bathing sponges, and the drugs listed above
Prevent streptococcal sore throat and treat promptly when it occurs

SUPERFICIAL FUNGAL INFECTIONS

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
Topical
Ketoconazole
- 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 immuno-compromised
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 receiving hyperalimentation 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 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

VIRAL INFECTIONS

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:
Capsaicin 0.075% cream

- For use after lesions have healed

Adult: apply 3 - 4 times daily
Child: may not be suitable for children because of its irritant properties
Topical local anaesthetics

- Helpful in some patients

Notable adverse drug reactions, caution

Aciclovir

- Ensure adequate hydration
- Caution in pregnancy and breastfeeding
- May cause nausea, vomiting, dizziness
- Fatigue pruritus and photosensitivity

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 Cham. 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 caues 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:

Clindamycin or erythromycin gel or solution twice daily

Adult and child: apply twice daily

Or:

Azelaic acid 20% cream

Adult and child: apply twice daily; initially once daily for sensitive skin

- Suitable for acne patients with atopic dermatitis

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:

Minocycline

Adult and child over 12 years: 50 - 100 mg orally every 12 hours

Or:

Erythromycin

Adult and child over 12 years: 500 mg - 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

Oral contraceptives containing a non-androgenic progestin

Co-cyprinidiol:

- A mixture of cyproterone acetate and ethinylestradiol 2000 parts to 35 parts

- 1 tablet orally daily for 21 days starting on day 1 of menstrual cycle and repeated after a 7-day interval, usually for several months

- For *women* with severe acne refractory to prolonged antibiotic therapy

Or:

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, minocycline and doxycycline are contraindicated in pregnancy and in children less than 12 years

- May reduce the effectiveness of oral contraceptives

- Often cause GIT symptoms

- Minocycline and doxycycline may cause photodermatitis

- Erythromycin cannot be used in conjunction with astemizole or terfenadine, as serious cardiovascular complications may occur

Salicylic acid

- Significant absorbtion 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

Hydroxyzine hydrochloride

Adult: initially 25 mg at night, increased if necessary to 25 mg 3 - 4 times daily

Child: 6 months - 6 years: initially 5 - 15 mg daily, increased if necessary to 50 mg daily in divided doses

Over 6 years: initially 15 - 25 mg daily, increased if necessary to 50 - 100 mg daily in divided doses

Aquagenic pruritus, mastocytosis, and pruritus of neurofibromatosis

Sodium cromoglycate

Adult: 200 mg orally taken before bath and immediately after

Child 2 - 14 years: 100 mg orally 4 times daily before meals
- Dose may be increased after 2 - 3 weeks to a maximum of 40 mg/kg daily, reduced according to response

Or:

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 or 12.5 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:

Capsaicin cream 0.75%

Adult: apply topically 3 - 4 times daily

Child: not recommended because of associated burning

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 thinly 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

Soduim cromoglycate:

Occasional nausea, rashes, and joint pain

Ketotifen:

Drowsiness; dry mouth; slight dizziness; CNS stimulation; weight gain

Driving, swimming and operating machines should be avoided

Enhances the effects of alcohol

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 - 2 years: 1 mg every 12 hours; 2 - 5 years: 1mg every 4 - 6 hours (maximum 6 mg daily); 6 - 12 years: 2 mg every 4 - 6 hours (maximum12 mg daily)
- If less sedation is required (e.g. day time)

Cetirizine
Adult and Child over 6 years: 10 mg orally daily or 5 mg every 12hours
Child 2 - 6 years: 5 mg orally daily or 2.5 mg every 12 hours
Or:
Acrivastine
Adult: 8 mg orally every 8 hours
Child under 12 years and *elderly*: not recommended
Or:
Loratadine
Adult and Child over 6 years: 10 mg orally daily
Child 2 - 5 years 5 mg daily
If persistent and chronic urticaria
Add Doxepin (oral form discontinued)
Adult: apply thinly 3 - 4 times daily; usual maximum 3 g 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
Cetirizine, loratadine, and acrivastine:
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
Topical depigmentation
- Monobenzyl ether of hydroquinone

20%, apply twice daily for 3 - 6 months (if more then

50% of the body is affected)

Systemic

Systemic 8-methoxypsoralen (or 5-methoxypsoralen)

Adult: 0.5 mg/kg orally

The initial UVA dose is 1 or 2 J/cm^2, 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

Monobenzylether of hydroquinone:

Depigmentation at distant sites

Acquired ochronosis

PUVA therapy should be supervised by an experienced dermatologist

Prevention

Unknown

CHAPTER 7: 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 - 12 years: 30 mg (5 mL of syrup) 3 times daily; 2 - 5 years 15 mg, (2.5 mL)

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: 30 mg (5 mL of syrup) orally every 8 hours; 2 - 5 years 2.5 mL

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 - 7 days

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: 250 mg 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 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 inexpert 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 - 12 years: 250 - 500 mg; 3 months - 5 years: 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 - 10 years: 10 - 25 mg orally daily in 1 - 2 divided doses; 2 - 5 years: 5 - 15 mg daily in 1 - 2 divided doses
Topical steroid
- 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

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

- 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 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

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 - 6 years: 5 mL 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 mg/kg once daily, up to 80 mg/kg in severe infections

Decongestant

- Psuedoephedrine tablets

Adult: 60 mg orally twice daily until congestion improves

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 - 250 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, most 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 - 12 years: 250 - 500 mg; 3 months - 5 years: 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

Or:
Chlorobutanol 5% paradichlorobenzene 2%, arachis (peanut) oil 57.3%

CHAPTER 8: ENDOCRINE SYSTE

DIABETES MELLITUS

Introduction
A group of metabolic diseases characterized by chronic hyperglycaemia
Results from defects in insulin secretion, insulin action or both
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 β 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
- 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 75 g oral glucose tolerance test

Values for the Diagnosis of Categories of Hyperglycaemia

Glucose Tolerance State	Venous plasma (mmol/L)	Venous plasma (mg/dL)
Diabetes mellitus		
Fasting	≥ 7	≥ 126
2 hour post-75 g glucose load	≥ 11.1	≥ 200
Impaired glucose tolerance		
Fasting	< 7.0	< 110
AND		
2 hour post-75 g glucose load	≥ 7.8 and < 11.1	≥ 140 and < 200
Impaired fasting glycaemia		
Fasting	≥ 6.1 and < 7.0	≥ 5.6 and < 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 Education
Should 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
- A snack 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 foot wear must always be worn during exercise

For a prescribed formal activity, the exercise session should consist of:
- A warm-up period of 5 - 10 minutes
- The activity proper: 20 - 60 minutes
- A cool-down period of 5 - 10 minutes

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 hypoglycaemic 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

Sulphonylureas

Initial monotherapy in non-obese patients

Add-on as combination therapy

Adult: Glibenclamide 1.25 - 10 mg orally twice daily

Child 12 - 18 years: initially 2.5 mg orally daily with, or immediately after breakfast, adjusted according to response; maximum 15 mg daily
- Indicated for Type 2 diabetes, maturity-onset diabetes of the young, under specialist care

Notable adverse drug reactions

Weight gain
Hypoglycaemia
Syndrome of inappropriate ADH secretion
Blood dyscrasias
Heart burn
Abdominal pain

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

Adult: Metformin 500 mg - 1 g orally twice or three times daily

Child 10 - 18 years: initially 500 mg orally once daily, adjusted according to response at intervals of not less than 1 week; maximum 2 g daily in 2-3 divided doses
- Under specialist supervision ONLY
- Not licensed for use in children less than 10 years old

Notable adverse drug reactions

Gastrointestinal upset/nausea/loose bowel motions
Metallic taste
Lactic acidosis

Contraindications

Impaired hepatic and renal function
Congestive cardiac failure
Contrast studies
Chronic obstructive airways disease
Alcoholism

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 and/or glinides or glitazones

Combination therapy using OGLAs with different mechanisms of action is indicated if monotherapy with one of the agents has failed

The rapid acting secretagogues (glinides) and the alpha glucosidase inhibitors make for flexibility in the glycaemic management of Type 2 diabetes mellitus but are relatively very expensive

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	1 h	3 h	Immediately before meals
Short-acting	30 min	2 - 5 h	5 - 8 h	30 min before meals
Intermediate-acting (NPH or lente)	1 - 3 h	6 - 12 h	16 - 24 h	Once or twice daily
Biphasic mixtures (30/70; premixed)	30 min	2 - 12 h	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 health facilities 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 $\geq 25\ kg/m^2$
- 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

These are:

Diabetic ketoacidosis
Non-ketotic hyperosmolar states
Hypoglycaemia
Lactic acidosis

- Acute hyperglycaemic complications may present with coma or altered levels of consciousness in people with diabetes

Differential diagnoses

Stroke
Seizures
Trauma
Drug overdose
Ethanol intoxication

Diabetic ketoacidosis

Introduction

Severe uncontrolled diabetes requiring emergency

treatment with insulin and intravenous fluids

Blood ketones (acetoacetate and 3-hydroxbutyrate) concentration > 5 mmol/L

Carries a high mortality in Africa

- Through late presentation, delayed diagnosis and inadequate treatment

Presents at any age although there is a well defined peak at puberty

Causes include:

Infection
Management errors
New cases of diabetes (treatment not commenced)
No obvious cause in about 40% of cases

Indications for immediate hospital admission

Repeated vomiting or inability to take adequate oral fluids

Hyperventilation
Any disturbance of consciousness
Persistent ketonuria
Presence of infections

Initial treatment plan for Diabetic Ketoacidosis in adults

Fluids and electrolytes

- One litre per hour for 3 hours; thereafter according to need
- Sodium chloride 0.9% injection
- Hypotonic (half-normal) saline: 75 mmol/L if plasma sodium exceeds 150 mmol/L
- Glucose 5% when blood glucose level falls below14 mmol/L

Plus:

Potassium (K^+) replacement

- To be added into each litre of fluid

Plasma K^+ less than 3.5 mmol/L:

Add 40 mmol KCl

Plasma K^+ 3.5 - 5.5 mmol/L:

Add 20 mmol KCl

Plasma K^+ greater than 5.5mmol/L:

Do not add KCl

Plus:

Insulin

- To be added into intravenous fluid for rehydration
- Initially, 5 - 10 units/hour; by continuous intravenous infusion
- Maintenance 2 - 4 units/hour, titrated against blood glucose levels (until able to eat)

Intramuscular injections:

- 20 units immediately, then 5 - 10 units/hour, titrated against blood glucose levels

Other measures:

Treat precipitating cause (e.g. infection, myocardial infarction)

Correct hypotension (should respond to adequate fluid replacement)

Pass nasogastric tube if consciousness is impaired

Ventilate if adult respiratory distress syndrome develops

- 100% oxygen by intermittent positive pressure ventilation

Intravenous dexamethasone, mannitol for cerebral oedema (see cerebral oedema)

Treat specific thromboembolic complications if they occur

Diabetic non-ketotic hyperosmolar state

Introduction

Characterized by the insidious development of:

Marked hyperglycaemia (usually > 50 mmol/L)
Dehydration
Pre-renal uraemia

- Significant hyperketonaemia does not develop

Two-thirds of cases occur in previously undiagnosed cases of diabetes

Usually affects middle- aged or elderly patients and carries a mortality of over 30%

Precipitating factors include:

Infections
Diuretic treatment
Drinking glucose-rich beverages

Treatment

Rehydration
Insulin therapy
Electrolyte replacement

- In a manner similar to that used for diabetic ketoacidosis

Hypoglycaemia

Introduction

Affects over 70% of patients on insulin therapy

Common causes of hypoglycaemia in persons with diabetes mellitus

Engaging in more exercise than usual
Delay or omission of a snack or main meal
Administration of too much insulin
Eating insufficient carbohydrate
Overindulgence in alcohol
Overdosing with sulphonylureas

In the presence of low blood glucose (< 2 mmol/L) characteristic symptoms and signs include:

Light headedness
Headaches
Tremulousness
Palpitations
Sweating
Feeling of hunger
Tachycardia
Hypertension (usually systolic)
Stroke-like presentations
Coma

Acute management

Oral glucose if patient is conscious

If patient is unconsious:

Some important features of the main types of diabetic emergencies are shown below:

Diabetic Ketoacidosis	Hyperosmolar non-ketotic state	Hypoglycaemic coma	Lactic acidosis
Hyperventilation **Dehydration Tachypnoea;** **Kaussmaul breathing** **Acetone breath** **More common in insulin-dependent persons; may occur in Type 2 diabetes Warm skin** **Normal or low blood pressure** **Hyperglycaemia and glycosuria** **Hyperketonaemia and ketonuria** **Fall in blood pH** **Increased free fatty acid** **Levels in blood**	No hyperventilation Dehydration more severe Marked polydipsia and polyuria Absence of acetone breath Usually seen in Type 2 diabetes Normal, low or elevated blood pressure Hyperglycaemia more marked Absence of ketones in blood and urine No change in blood pH Normal fatty acid levels	Normal breathing No dehydration Absence of acetone breath May occur in all categories of persons with diabetes Cold, clammy skin; profuse sweating Systolic hypertension may precede coma Low blood glucose Absence of ketones in blood and urine No change in pH	Hyperventilation Absence of acetone odour Common in those taking biguanides Diagnosis made only when other causes of metabolic acidosis have been excluded Blood lactate levels not commonly measured

Intravenous glucose
- 50% glucose given as a bolus of 40 - 50 mL

Or:
- 20% glucose 100 - 150 mL followed by 8 - 10% glucose infusion if necessary

Or:

Injectable glucagon
- 1 mg intramuscularly stat

If hypoglycaemia is due to long acting sulphonylureas, or long and intermediate acting insulin or alcohol

Prolonged intravenous glucose infusion (5 - 10% for 12 - 24 hours; even longer) may be necessary

If intravenous access is impossible:

Consider nasogastric or rectal glucose

Or:

Give glucagon 1 mg intramuscularly

As a last resort:

Administer epinephrine (adrenaline)
- 1 mL of 1 in 1,000 strength, subcutaneously stat

On recovery:

Give a long acting carbohydrate snack

Attempt to identify the cause of hypoglycaemia and correct it

Assess the type of insulin used, injection sites and injection techniques
- Lipohypertophy can alter the rate of absorption

Enquire into, and correct inappropriate habits of eating, exercise and alcohol consumption

Review other drug therapy and renal function

Adjust insulin or OGLA dosages as appropriate

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/m^2)

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

HYPERTHYROIDISM (Thyrotoxicosis)

Introduction

A clinical syndrome which results from exposure of the body to excess levels of the thyroid hormones, Thyroxine (T_4) and Tri-iodothyronine (T_3)

More females are affected than males (usually in the ratio of 5:1)

Aetiology

Grave's disease (80% of patients)

Multinodular goitre

Autoimmune functioning solitary thyroid nodule

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:
- Inappropriate TSH secretion by the pituitary
- Choriocarcinoma
- Hydatiform mole

Follicular carcinoma of the thyroid with metastasis

Clinical features

A goit may or may not be present
- May be diffuse or nodular

Dermatological:

Increased sweating and pruritus

Pretibial myxoedema

Pigmentation, vitiligo

Palmar erythema.

Cardiorespiratory:

Dyspnoea on exertion

Angina and cardiac failure

Increased pulse pressure

Exacerbation of asthma

Gastrointestinal:

Weight loss despite increased appetite

Diarrhoea

Steatorrhoea

Neuromuscular:

Tremors, nervousness, irritability, emotional lability and psychosis

Muscle weakness and proximal myopathy

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

Fatigue and apathy

Differential diagnosis

Simple goitre

Malignant tumours of the thyroid

Complications

Hyperthyroid crisis (thyroid storm)

Compression of the trachea

Cardiac failure

Loss of visual acuity

Infertility

Periodic paralysis

Investigations

Specific:

Serum T_3, T_4 and TSH levels

Measurement of I^{131} intake by the thyroid gland

Non-specific:

Liver function tests
- Slightly raised concentrations of bilirubin, alanine aminotransferase

Serum calcium
- Mild hypercalcaemia

Fasting blood glucose
- Glycosuria may be present

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 non-specific illness develops

Propylthiouracil

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 - 12 years: initially 50 mg every 8 hours 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

β- 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
Radioactive sodium iodine (I^{131})
- 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 goiters, 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 pancytopemia 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_3 and T_4 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

- T_3 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

CHAPTER 9: 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

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 X-ray (especially lumbrosacral segment) to exclude ankylosing spondilytis

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

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
Sometimes associated with infective conjunctivitis
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 frequency as infection is controlled and continue for 48 hours after healing

Atropine drops
- 1 drop up to 4 times daily

Antivirals (if dendritic ulcer)
- 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 it 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
- Sodium cromoglycate eye drops

Adult and child: apply four times daily
- Diclofenac sodium 0.1% eye drops

Adult and child: apply once daily

Phlyctenular conjuntivitits:
Treat for tuberculosis using standard regimen

Caution

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

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

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 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 the surface of the eye
Occasionally a high velocity material, usually a metal could be propelled into the eye

Clinical features

May be embedded on the tarsal or bulbar conjunctiva, the 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

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 influenzae
Gonococcus: ophthalmia neonatorum
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

Investigation

Conjunctival swab for microscopy, culture and sensitivity

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

Epidermic 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:

- A 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

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 the upper lid

Differential diagnoses

Various eyelid cysts and tumours

Complications

Pre-septal cellulitis
Orbital cellulitis
Cavernous sinus thrombosis

Investigations

If recurrent, screen for diabetes

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 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 Eliza 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 - 45 kg: 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 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
Non-drug treatment
Nutrition education
Drug treatment
Vitamin A capsules 200,000 IU 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 10: GENITO-URINARY SYSTEM

NEPHROLOGY

ACUTE RENAL FAILURE
Introduction
A syndrome characterized by rapid decline in glomerular filtration rate with retention of nitrogenous waste products, disturbance of extracellular fluid volume, electrolytes and acid-base homeostasis
Classification/aetiology
Pre-renal Acute Renal Failure
Hypovolaemia (e.g. from haemorrhage, severe diarrhoea and vomiting etc)
Low cardiac output (e.g myocarditis)
Renal hypoperfusion (e.g. from use of angiotensin converting enzyme inhibitors)
Systemic vasodilatation (e.g. sepsis)
Hyperviscosity syndromes (e.g polycythaemia)
Intrinsic renal failure
Renovascular obstruction (e.g. renal vein thrombosis)
Glomerular disease e.g. glomerulonephritis
Acute tubular necrosis (e.g. from ischemia)
Interstitial nephritis (e.g. infections, allergic, from antimicrobials like rifampicin)
Intratubular deposition and obstruction (e.g. uric acid, oxalate stones)
Renal allograft rejection
Post renal Acute Renal Failure
Ureteric obstruction (from calculi, blood clots etc)
Bladder neck obstruction from prostate hypertrophy
Urethral obstruction (e.g. from strictures, congenital urethral valves)
Clinical features
Thirst, dizziness, hypotension, tachycardia in pre-renal ARF
Oliguria (not invariable)
Oedema, hypertension
Flank pain, hesitancy, nocturia, in post-renal ARF
Complications
Volume overload
Hyperkalaemia
Metabolic acidosis
Uraemic encephalopathy
Hypertension
Differential diagnoses
Acute-on-chronic renal failure
Chronic renal failure
Investigations
Urine microscopy: casts (granular, hyaline)
Urinalysis: proteinuria, haemauria
Serum Electrolytes, Urea and Creatinine
Full Blood Count with differentials
Abdominal ultrasound scan
Treatment objectives
Correct primary haemodynamic abnormality
Correct biochemical abnormalities
Prevent further renal damage
Non-drug treatment
Fluid challenge (where indicated)
Low potassium, low salt, low protein diet
Avoid or discontinue nephrotoxic drugs
Drug treatment
Antihypertensive drugs (see treatment of hypertension)
Loop diuretics
Furosemide:
- Initially 250 mg by intravenous infusion over 1 hour at a rate not exceeding 4 mg/minute
- Give another 500 mg by intravenous infusion over 2 hours if urine output is satisfactory
- Effective dose can be repeated every 24 hours
- If no response, dialysis is probably required

Supportive therapy
Regular intermittent haemodialysis
Peritoneal dialysis
Prevention
Close attention to cardiovascular function and intravascular volume in high risk patients, especially those with pre-existing renal insufficiency
Avoid hypovolaemia (especially in patients on nephrotoxic drugs)
Adequate hydration and sodium loading in patients to be exposed to radiocontrast dye investigations (for example)

CHRONIC KIDNEY DISEASE
Also chronic renal failure

Introduction
A progressive and persistent deterioration in kidney structure and function ultimately resulting in accumulation of nitrogenous waste products and disruption of acid-base homeostasis.
- Also associated with derangement in the kidney's osmoregulatory, metabolic and endocrine function

Aetiology
Hypertension
Diabetes mellitus
Chronic glomerulonephritis
Systemic lupus erythematosus
Chronic pyelonephritis
Genetic e.g. adult polycystic kidney disease, Alport's syndrome
Clinical features
Nocturia
Oliguria
Bleeding tendencies
Anaemia
Hypertension (not invariable)

Body swelling
Pruritus
Bone pains

Complications

Hyperkalaemia
Severe anaemia
Hypertensive heart disease
Atherosclerosis
Uraemic pericarditis
Renal osteodystrophy
Metabolic acidosis

Investigations

Urine
- Urinalysis
- Urine microscopy, culture and sensitivity

Blood
- Serum Electrolytes, Urea and Creatinine
- Creatinine clearance
- Full Blood Count; ESR
- Serum lipids
- Serum proteins
- Serum calcium and phosphate

Abdominal ultrasound scan

Treatment objectives

Slow down rate of decline of GFR
Manage hypertension
Control hypertension
Provide renal replacement therapy (if in end stage)

Non-drug treatment

Diet: low salt, low protein, low potassium
Avoid nephrotoxic agents

Drug Treatment

Anthypertensive agents (see treatment of hypertension)
Diuretics (furosemide at doses appropriate for clinical condition)
Vitamin D and calcium supplements
Erythropoietin
- Initially 50 units/kg 3 times weekly; adjusted according to response in steps of 25 units/kg 3 times weekly at intervals of at least 4 weeks
- Maintenance dose (when Hb concentration 10 -12 g/100 mL is achieved)
- Total 75 - 300 units/kg weekly, as a single dose or in divided doses

Iron supplements
- Ferrous sulphate

Adult: 200 mg orally 3 times daily
Child: 6 - 18 years: prophylactic 1 tablet (200 mg) daily; therapeutic 200 mg 2 - 3 times daily

Treat hyperkalaemia (see chapter on hyperkalaemia)
Phosphate binding agents
Calcium carbonate:
Adult: 500 mg - 1.25 g orally
- Starting dose usually 500 mg - 1 g orally 2 times daily after meals

Child: 1 month - 1 year: 120 mg 3 - 4 times daily with feeds; 1 - 6 years: 300 mg; 6 - 12 years: 600 mg; 12 - 18 years: 1.25 g; all 3 - 4 times daily prior to, or with meals and adjusted as necessary
Aluminium hydroxide:
Adult: 300 - 600 mg orally 3 times a day with meals
Child 5 - 12 years: 1 - 2 capsules orally 3 - 4 times daily; 12 - 18 years: 1 - 5 capsules 3 - 4 times daily; adjusted as necessary

Supportive measures

Haemodialysis
Peritoneal dialysis

Definitive treatment is renal transplantation

Notable adverse drug reactions, caution and contraindications

See furosemide
Potential for adverse drug reactions with drugs eliminated primarily by the kidneys e.g. aminoglycoside antibiotics, NSAIDs, metformin, etc
Calcium-containing phosphate-binding agents are preferred in children but are contraindicated in hypercalcaemia or hypercalciuria

Prevention

Appropriate management of known causes of chronic renal failure e.g. hypertension and diabetes mellitus
Cautious use of nephrotoxic agents: avoid their use in patients with low renal reserves
Early detection and treatment of renal disease when renal function is still adequate

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 β 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 the 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 daily 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:
2 g orally, as a single dose
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, **C**ompliance, **C**ondom use and **C**ontact 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 glands matted together, often adherent 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 chancre
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, **C**ompliance, **C**ondom use and **C**ontact 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 and minocycline, 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, **C**ompliance, **C**ondom use and **C**ontact 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 bloodstream 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):

Littré 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 albicans* can 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 abortus*
- 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)*
- β-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

<u>Neonatal gonococcal conjunctivitis</u>

Recommended regimen:

Ceftriaxone 50 mg/kg by intramuscular injection, as a single dose, to a maximum of 125 mg

Or:

Spectinomycin 25 mg/kg by intramuscular injection as

a single dose, to a maximum of 75 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

Spectinomycin

- Nausea, dizziness, fever and urticaria

Prevention

Counselling, **C**ompliance, **C**ondom use and **C**ontact 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 g orally on first day, then 500 mg orally, 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, **C**ompliance, **C**ondom use and **C**ontact treatment

LYMPHOGRANULOMA VENEREUM

(Climatic bubo; lymphogranuloma inguinale; lymphopathia venereal; Durand-Nicolas-Favre 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, **C**ompliance, **C**ondom use and **C**ontact 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 and 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
- 4 g (2.4 million units) by intramuscular injection, at a single session
- Because of the volume involved, this dose is usually given as two injections at separate sites

Alternative regimen:

Procaine benzylpenicillin
- 2 g (1.2 million units) by intramuscular injection, daily for 10 consecutive days

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, **C**ompliance, **C**ondom use and **C**ontact 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
- 2 g orally in a single dose

Or:

Tinidazole
- 2 g orally 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, **C**ompliance, **C**ondom use and **C**ontact 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 immunosuppression - 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

A common cause of lower urinary tract obstruction among elderly males

Non-cancerous increase in size of the prostate gland

Increase in size impacts on the urethra and partially or totally obstructs urine outflow

Occurs after the age of 40 years; cause is uncertain

Symptoms are due to mechanical obstruction or spasms of the smooth muscles around the bladder neck and prostate

Clinical features

Lower urinary tract symptoms

Irritative symptoms:

Frequency

Urgency

Nocturia

Urge incontinence

Obstructive symptoms:

Poor stream

Hesitancy

Straining

Intermittency

Retention of urine

Haematuria

Recurrent urinary tract infections

Progressive renal failure

Digital rectal examination:

Enlarged prostate; firm and symmetrical

Differential diagnoses

Prostate cancer

Bladder cancer

Bladder calculi

Urethral stricture

Prostatitis

Neurogenic bladder

Complications

Acute or chronic urine retention

Recurrent urinary tract infections

Bladder calculi

Haematuria

Hydroureter/hydronephrosis

Progressive renal failure

Investigations

Urinalysis

Urine microscopy, culture and sensitivity

Serum Urea, Electrolytes and Creatinine

Prostate Specific Antigen (PSA)

Trans-rectal ultrasound

Abdominal ultrasound scan

Full Blood Count

Treatment objectives

Relieve obstruction

Treat or prevent complications

Non-drug treatment

Surgery: open prostatectomy or transurethral resection

Minimally invasive procedures

High intensity focused ultrasound

Transurethral balloon dilatation

Intraurethral stent

Transurethral vaporization of the prostate

Intermittent self-catheterization

Drug treatment

Alpha adrenergic blockers

- Prazosin, doxazosin, tamsulosin

Doses are titrated from 1 -10 mg depending on individual response

- 400 microgram orally daily as single dose for tamsulosin

5- Alpha reductase inhibitors

- Finasteride 5 mg orally daily

Notable adverse drug reactions, caution

Alpha-adrenergic blockers: dizziness, syncopal attacks, tachycardia

- Should therefore to be taken at night before going to bed

5- Alpha reductase inhibitors: loss of libido, erectile dysfunction, gynaecomastia

CARCINOMA OF THE PROSTATE

Introduction

The most commonly diagnosed malignancy affecting men beyond the middle age

The commonest malignancy of the genitourinary tract

Exact cause is not known

About 90% are adenocarcinomas

Risks factors

Increasing age

Familial and genetic factors

High levels of testosterone and dihydrotestosterone

Clinical features

Lower urinary tract symptoms

Frequency

Urgency

Nocturia

Poor stream

Straining

Terminal dribbling

Haematuria
Features of metastasis
Low back pain
Paraplegia
Pathological fractures
Pedal oedema
Azotaemia
Weight loss
Rectal Examination: hard, nodular, asymmetrical prostate

Differential diagnoses
Benign prostatic hyperplasia
Chronic prostatitis
Bladder cancer/calculi
Prostatic calculi
Urethral stricture

Complications
Urinary retention
Urinary tract infection
Hydroureter/hydronephrosis
Progressive renal failure
Paraplegia
Pathological fractures
Lymphoedema

Investigations
Prostate Specific Antigen
Prostate biopsy
Trans-rectal ultrasound
Abdominal ultrasound
CT scan
Liver function tests
Chest radiograph
Serum Urea, Electrolytes and Creatinine
Full Blood Count

Treatment objectives
Aim at cure for early disease
Palliation for advanced disease

Non-drug treatment
Watchful waiting
Radical prostatectomy
Radiotherapy (brachytherapy or external beam radiation)
Bilateral orchidectomy
Cryoablation therapy
Laser therapy

Drug treatment
LHRH agonist:
Goserelin acetate
- 3.6 mg by subcutaneous injection into the anterior abdominal wall every 28 weeks

Anti-androgens:
Cyproterone acetate
- 100 mg orally twice daily for long term palliative therapy

Or:
Bicalutamide 50 mg orally daily in advanced cases, with orchidectomy

Or:
Flutamide 250 mg orally three times daily

Or:
Diethyl stilbestrol 3 mg orally daily
Cytotoxic chemotherapy:
Docetaxel 75 mg/m^2 every 3 weeks

Notable adverse drug reactions, caution and contraindications
Anti-androgens:
- Loss of libido
- Gynaecomastia
- Impotence

Diethyl stilbestrol:
- Fluid retention
- Hypertension
- Thrombo-embolic disease
- Loss of libido
- Gynaecomastia
- *Contraindicated in patients with cardiovascular diseases*

ERECTILE DYSFUNCTION (Impotence)

Introduction
Persistent inability to obtain and sustain an erection sufficient for sexual intercourse

May be non-organic (psychogenic) or organic, resulting from physical causes
- Vascular, neurologic or endocrine dysfunction

Other causes include drugs and trauma

Clinical features
Inability to obtain or sustain erection
History suggestive of possible causes e.g. drugs, systemic disease like hypertension, diabetes mellitus
With or without gynaecomastia
With or without penile deformity, plaques or impaired sensation

Complications
Psychological disturbances
Infertility

Investigations
Full Blood Count
Hormonal assay (LH, FSH, testosterone, prolactin)
Serum Urea, Electrolytes and Creatinine
Blood glucose
Nocturnal penile tumescence test

Treatment objective
To obtain and sustain erection

Non-drug treatment
Psychotherapy
Use of vacuum suction devices
Placement of intracorporal prosthesis
Microsurgical vascular anastomosis

Drug treatment

Androgen replacement in those with androgen deficiency:
Testosterone enanthate
- 250 mg intramuscularly every 2-4 weeks

Or:
Oral methyl testosterone or fluoxymesterone
120 - 160 mg daily for 2 - 3 weeks; maintenance 40 - 120 mg daily

Intra-corporal administration of:
Prostaglandin E_1
- 5 - 15 microgram

5-Phosphodiesterase inhibitors:
Sildenafil citrate
- 25 - 100 mg one hour before intercourse

Notable adverse drug reactions, caution and contraindications
Androgens
- Not to be given to patients with prostate carcinoma

Phosphodiesterase inhibitors
- Altered vision, headache, dizziness and nasal congestion
- Contraindicated in patients taking nitrates
- Should be used with caution in patients with ischaemic heart disease

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
- Caverno-saphenous shunt

Spinal or epidural anaesthesia

Drug treatment
Intracavernosal injection of alpha adrenergic agonist:
Phenylephrine
- 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, S*treptococcus 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 sensivity
- 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, doxazocin
Hormonal therapy e.g. finasteride, cyproterone

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 intensity 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 the testicle up
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-Schönlein 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 instrumention 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
Treament 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
Or:
Metrifonate
Adult: 10 mg/kg orally, fortnightly for three doses
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 wih 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 11: 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 36.8°C) with diurnal variations of about 0.5°C

'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 38°C

Oral temperature above 37.8°C

Axillary temperature above 37.2°C

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 cirmcumstances

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
- Use of sanitay 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 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
Or:
Piperazine phosphate
Adult: 4 g (i.e. the contents of one satchet) stirred into water or milk and taken at bedtime
- Reapeat after 14 days

Child: 1 - 6 years: 750 mg (i.e. 5 mL) orally in the morning, repeated after 14 days
Infants 3 months - 1 year: 2.5 mL orally in the morning, repeated after 14 days
- Repeated treatments may be necessary

Trichiuris
Mebandazole
Adult and child: 100 mg orallyevery 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 by a further dose of 20 mg/kg
Praziquantel is effective in all human cases caused by all 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 by 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 VIRUS INFECTION

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 features

Transient early acute symptoms: commonly "flu" -like illness, often not recognized in the first 2 - 3 weeks of HIV infection:

Generalized lymphadenopathy
Sore throat
Fever
Skin rash

Asymptmatic period:

The individual feels well despite on-going viral replication

Initial symptoms:

Generalized lymphadenopathy
Wasting syndrome/fever/night sweats
Neurologic disease
Early immune failure
Oral thrush
Herpes zoster
Hairy leukoplakia
AIDS (opportunistic infections)
Recurrent bacterial pneumonias
Pulmonary and extrapulmonary tuberculosis
Pneumocytis carinii infection
Kaposi sarcoma
Viral infections including cytomegalo virus
Other protozoan infections including cryptosporidium, cryptocooccocus.
Systemic fungal infections
Other cancers (lymphomas, cervical cancer, etc.)

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) > 1 month
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[1]
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
HIV encephalopathy[2]
And/or performance scale 4: bedridden > 50% of the day during last month

1: Weight loss of > 10% plus either unexplained 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

CD4 count (cells/mm^3)	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 pneumonia (PCP), disseminated histoplasmosis and coccidiomycosis, progressive multifocal 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

Complications

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

Adults and Adolescents

Initiation of therapy depends on availability of CD4 cell count testing

If CD4 testing is available:

WHO Stage IV disease irrespective of CD4 cell count
WHO Stage III disease with CD4 cell counts < 350/mm^3
WHO Stage I or II disease with CD4 cell counts ≥ 200/mm^3

If CD4 testing is unavailable:

WHO Stage IV disease irrespective of total lymphocyte count (TLC)
WHO Stage III disease irrespective of TLC
WHO Stage II disease with a TLC ≥ 1200/mm^3

A TLC of ≥ 1200/mm^3 does not predict a CD4 cell count of ≥ 200/mm^3 in asymptomatic patients

TLC of ≥ 1200/mm^3 may not be used as criterion for the initiation of therapy in asymptomatic patients (WHO Stage 1 disease)

Children

Children are monitored using CD4 percentage (CD4 %) i.e. percentage of lymphocytes that are CD4 cells

CD4% of an HIV-negative child is around 40%

Diagnosis depends on the age of the child and availability of virological testing

Children < 18 months

Serological diagnosis is unreliable as maternally-derived antibodies may persist for up to 15 - 18 months

Diagnosis of HIV has to be made by identifying HIV DNA using PCR

HIV-seropositive children aged <18 months

If HIV status is virologically-proven ART is recommended when the child has:

WHO Paediatric Stage III disease irrespective of CD4 %

WHO Paediatric Stage II disease, with consideration of using CD4 <20% to assist in decision making

Or:

WHO Paediatric Stage I (asymptomatic) and CD4 <20%

- If HIV-seropositive status is not virologically proven but CD4 cell assays are available, ART can be initiated when the child has :

WHO Stage II or III disease and CD4 <20%

- In such cases, HIV antibody testing must be repeated at age 18 months to definitively confirm that the child is HIV infected
- Only children with confirmed infection should have ARV therapy continued

HIV-seropositive children aged >18 months

ART can be initiated when child has:

WHO Paediatric Stage III disease (e.g. clinical AIDS) irrespective of CD4 count

WHO Paediatric Stage II disease with CD4 <15%

Or:

WHO Paediatric Stage I disease (e.g. asymptomatic-appendix I) and CD4 <15% (Appendix I)

For children > 8 years adult criteria for initiation of therapy are applicable

Drug treatment

Preferred first line regimen (adults and adolescents)

- d4T / 3TC / NVP or EFZ

Alternative first line regimens

- TDF/3TC/NVP or EFZ

Or:

- ABC/3TC/NVP or EFZ

Alternative first line drugs for special category of adults

Pregnant women with CD4 count <250 cells/mm^3 or women who are likely to become pregnant

- ZDV / 3TC / NVP

Adult dosages

Nevirapine (NVP)
- 200 mg orally once daily for 2 weeks; then 200 mg twice daily

Efavirenz (EFV)
- 600 mg orally once daily; 800 mg once daily when using anti-tuberculosis drug

Zidovudine (ZDV)
- 250 - 300 mg orally twice daily

Stavudine (d4T)
- 40 mg orally twice daily
- If weight <60 kg: 30 mg twice daily

Lamivudine (3TC)
- 150 mg orally twice daily

Didanosine (ddI)
- 400 mg orally once daily
- If weight < 60 kg or combined with TDF: 250 mg once daily

Tenofovir (TDF)
- 300 mg once daily

Abacavir (ABC)
- 300 mg orally twice daily

Indinavir (IDV)
- 800 mg orally three times daily

Nelfinavir (NFV)
- 1.25 g orally twice daily

Or:
- 750 mg three times daily

Lopinavir/Ritonavir (LPV/r)
- 3 capsules (498 mg) orally twice daily

Saquinavir (SQV)
- 1.2 g orally three times daily

Amprenavir (AMP)
- 1.2 g twice daily

Ritonavir (RTV)
- 100 mg orally twice daily

Atazanavir (ATV)
- 400 mg orally once daily

Children

Preferred first line regimen

- d4T or ZDV/ 3TC / NVP or EFV
  - EFV for age 3 years and above; avoid liquid formulations

Alternative first line regimens

- ddI/3TC/NVP or EFV
  - EFV for age 3 years and above, avoid liquid formulations

Alternative first line drugs for special category of children

Children with tuberculosis require rifampicin-containing regimen for TB treatment

- D4T **or** ZDV/3TC/EFV (3 years and above)

Age less than 3 years: please refer to paediatric HIV consultant

First line recommendations for HIV/TB patients

Adults/Adolescents and Pregnant Women:

- (ZDV or dT4) + 3TC + NVP during non-rifampicin-containing continuation phase

Or:

- (ZDV or dT4) +3TC + EFV during rifampicin-containing intensive or continuation phase

Management of virological treatment failure

Treatment failure due to resistance

The three drugs reserved for the first line regimens are replaced with three totally new drugs-second line regimens

- If resistance testing cannot be done (see second line treatment regimens)

Where resistance testing is available, the failing drug may be identified and replaced

Recommended second line regimens

Adults and adolescents

First line	Second line
d4T or ZDV/3TC/NVP or EFV	TDF/FTC/IDV/r or SQV/r or LPV/r Or: ABC/ddI/IDV/r or SQV/r or LPV/r Or:
TDF/FTC/NVP or EFV	ZDV/3TC or ddI/IDV/r or SQV/r or LPV/r Or:
ABC/3TC/NVP or EFV	TDF/FTC/IDV/r or SQV/r or LPV/r

Note

The dose of ddI should be reduced from 400 mg to 250 mg when co-administering with TDF in an adult > 60 kg
Reduce dose to 125 mg in adult < 60 kg
IDV/r, LPV/r and SQV/r require secure cold chain for storage
Co-formulations of the medications above may be used to reduce the pill burden

Children

First line	Second line
d4T or ZDV/3TC/NVP or EFV	d4T or ZDV/3TC/ABC/LPV/r (preferred) or NFV
Or:	Or:
ddI/3TC/NVP or EFV	ZDV/3TC/LPV/r (preferred) or NFV

LPV/r requires secure cold chain
All treatment failures at first and second level health facilities should be referred to a paediatric consultant

Child dosages

Didanosine (ddI)
- 2 weeks - 8 months: 100 mg/m^2 orally twice daily
- > 8 months: 120 mg/m^2 twice daily

Lamivudine (3TC)
- <1 month: 2 mg/kg orally twice daily
- >1 month: 4 mg/kg orally twice daily
- Adolescents < 50 kg: 2 mg/kg orally twice daily

Stavudine (d4T)
- 1mg/kg orally twice daily up to a maximum of 40 mg per dose

Zalcitabine (ddC)
- Not available

Zidovudine (ZDV)
- 160 mg/m^2 orally every 8hours

Efavirenz (EFZ)
- Taken orally once daily
- 10 to <15 kg: 200 mg; 15 to < 20 kg 250 mg; 20 to <25 kg 300 mg; 25 to <32.5 kg 350 mg; 32.5 to <40 kg 400 mg; >40 kg 600 mg

Nevirapine (NVP)
- 15 - 30 days: 5 mg/kg orally once daily for 14 days, then 120 mg/m^2 twice daily for 14 days, and 200 mg/m^2 twice daily
- 1 month - 13 years: 120 mg/m^2 twice daily for 14 days, then 200 mg/m^2 twice daily

Indinavir (IDV)
- <4 years: not used
- 4 - 17 years: 500 mg/m^2 orally twice daily; (maximum 800 mg) three times daily

Nelfinavir (NFV)
- <1 year: 40 - 50 mg/kg orally three times daily; or 65 - 75 mg/kg twice daily
- 1 - 13 years: 55 - 65 mg/kg twice daily

Lopinavir/rotinavir (Lop/r)
- 7 kg to <15 kg: lopinavir 12 mg/kg, rotinavir 3 mg/kg orally twice daily with food
- 15 - 40 kg: lopinavir 10 mg/kg, rotinavir 2.5 mg/kg orally twice daily with food
- >40 kg lopinavir 400 mg, rotinavir 100 mg orally twice daily with food

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 (**d**rug **r**ash, **e**osinophilia and **s**ystemic **s**ymptoms): 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 chronic 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

Mechanisms with established merit:

Prevention of mother-to-child transmission (PMTCT)
Prophylactic AZT/NVP or HAART
Caesarian section
Infant feeding choices (Exclusive Formula)
Safer sex (condom use)
Post exposure prophylaxis among healthworkers
Treatment of STIs
Voluntary counselling and testing (VCT)
Needle exchange programmes for IVUs

Mechanisms with anticipated (potential) merit:

Reduction of viral load with HAART
Post exposure prophylaxis following sexual exposure (rape)
Sexual risk reduction
Promotion of safer sex and low-risk behaviour

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 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

Four species of the parasite cause the disease in humans: *Plasmodium falciparum*, *vivax*, *ovale* and *malariae*

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

Uncomplicated

There are no life-threatening manifestations

Complicated

P. *falciparum* asexual parasitaemia, with the presence of clinical and/or laboratory life-threatening features

Clinical features

These are non-specific:

Fever
Chills
Headache
Malaise
Aches and body pain
Weakness
Tiredness
Pallor
Anorexia
Vomiting
Bitterness in the mouth
Excessive sweating
Pallor
Hepatosplenomegaly
Jaundice

Malaria is severe when there is:

Repeated vomiting
Prostration
Impaired consciousness
Severe anaemia
Circulatory collapse
Hypoglycaemia
Pulmonary oedema
Abnormal bleeding
Jaundice
Haemoglobinuria
Febrile seizures
Renal failure
Hyperparasitaemia

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

Absence of fever does not exclude a diagnosis of malaria

Microscopic diagnosis should not delay appropriate treatment if there is a clinical suspicion of severe malaria

Differential diagnoses

Typhoid fever
Meningitis
Encephalitis
Septicaemia
Other causes of fever

Complications

Early

Hypoglycaemia
Lactic acidosis
Haematological abnormalities
Liver dysfunction
Pneumonia
Septicaemia
Non-cardiogenic pulmonary oedema
Cerebral malaria
'Blackwater' fever
Acute tubular necrosis

In pregnancy

Anaemia
Preterm contractions/preterm labour
Abortions
Low birth weight
Intrauterine deaths
Congenital malaria

Late

Hyperreactive malaria splenomegaly
Quartan malaria nephropathy
Possibly, Burkitt's lymphoma

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
Prevent severe malaria
Attend to the immediate threats of life
Prevent complications
Provide personal protection against malaria
Provide chemoprophylaxis in susceptible groups

Drug treatment

Uncomplicated malaria

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

Artemisin-based combination therapy is the treatment of choice

Adult and child over 16 years < 40 kg: 10 mg/kg; 40 - 59 kg: 400 mg (one 400 mg suppository); 60 - 80 kg: 800 mg (two 400 mg suppositories); >80 kg: 1,200 mg (three 400 mg suppositories)

Child: 30 - 39 kg: 300 mg (three 100 mg suppositories); 20 - 29 kg: 200 mg (two 100 mg suppositories); 9 - 19 kg: 100 mg (one 100 mg suppository); 5 - 8.9 kg: 50 mg (one 50 mg suppository)

- Dose should be given ONCE and followed as soon as possible by definitive therapy for malaria

Definitive treatment

Artemisin-based combination therapy is recommended

Monotherapy with dihydroartemisin or other artemisinin derivatives is not recommended

Artemether-lumefantrine (20 mg/120 mg)

Adult and child over 14 years: 4 standard tablets orally every 12 hours

Child: 9 - 14 years: 3 tablets twice daily for 3 days; 4 - 8 years 2 tablets every 12 hours for 3 days

6 months - 3 years: 1 tablet every 12 hours for 3 days

- Not recommended for children under 3 months or <5 kg

Or:

Artesunate-amodiaquine (4 mg/10 mg base)

Adult: 4 standard tablets every 12 hours

Child: 1 - 2 standard tablets orally every 12 hours, adjusted according to age or body weight

Severe malaria

Quinine or artemisinin derivatives given parenterally are the drugs of choice

- Quinine:

Adult: 20 mg/kg of salt to a maximum of 1.2 g loading dose intravenously, diluted in 10 ml/kg isotonic fluid over 4 hours

- 8 hours after start of the loading dose: 10 mg/kg salt to a maximum of 600 mg over 4 hours, every 8 hours until the patient is able to take orally
- Then change to tablets 10 mg/kg 8 hourly for 7 days **or** give full dose of artemether-lumefantrine

Child: 20 mg/kg of salt as loading dose diluted in 10 mL/kg of 4.3% glucose in 0.18% saline or in 5% glucose over 4 hours 12 hours later, give 10 mg salt/kg as infusion over 4 hours, and every 8 hours until patient is able to take orally

Change to tablets 10 mg/kg every 8 hours to complete a total of 7 days

Or:

- Where intravenous access is not possible, give quinine dihydrochloride 20 mg/kg salt as loading dose, diluted to 60 -100 mg/ml intramuscularly in different sites
- 8 hours after loading dose, give 10 mg/kg 8 hourly until patient is able to take orally
- Thereafter, change to tablets 10 mg/kg 8 hourly for 7 days or give a full dose of artemether-lumefantrine

Or:

- Artesunate

Adult: 2.4 mg/kg intravenous bolus; repeat 1.2 mg/kg after 12 hours then 1.2 mg/kg daily for 7 days

Child: intravenous use reserved for specialists

- Once patient can tolerate oral medication give a full dose of artemether-lumefantrine

Or:

- Artemether
- 3.2 mg/kg intramuscular loading dose followed by 1.6 mg/kg daily for 6 days

Alternatively:

- Once patient can tolerate oral medication, give full dose of artemether-lumefantrine

In all cases, patient's progress should be monitored and management changed as deemed necessary

Supportive measures

Paracetamol (oral/rectal) for symptomatic relief of fever

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 sodium chloride injection 0.9%, and challenge with furosemide 1 - 2 mg/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 can not 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

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

Mefloquine 5 mg base/kg weekly, giving an adult dose of 250 mg base/week

Or:

1.5 mg of salt/kg administered daily (100 mg of salt daily)
- 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 one week before departure and continue until 4 weeks after leaving the region

Chemoprophylaxis is not recommended for individuals living with areas of intense transmission

People with sickle cell anaemia should have regular chemoprophylaxis (see Sickle Cell Diseases)

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; ocassionally 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-Barré 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 (C SF) 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-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 7th 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
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
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 30 mg/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 contrainndications
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 diagnsoses
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 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

TUBERCULOSIS

Introduction
One of the oldest diseases known to affect humans, globally
Nearly one third of the global population (i.e. 2 billion) people are infected with *Mycobacterium tuberculosis* and at risk of developing the disease
More than 8 million people develop active tuberculosis (TB) every year; about 2 million die
More than 90% of global TB cases and deaths occur in the developing world where 75% of cases are in the most economically productive age group (15 - 54 years)
M. tuberculosis usually affects the lungs although in up to one third of cases other organs are involved
If properly treated, TB caused by drug-susceptible strains is curable in virtually all cases; however if untreated it may be fatal within 5 years in more than half of cases
Transmission usually takes place through the airborne spread of droplet nuclei produced by patients with infectious pulmonary TB and aerosolized by coughing
- As many as 3,000 infectious nuclei per cough can be produced
- Droplet nuclei could also by spread by sneezing and speaking

Poverty and widening gap between rich and poor, hunger, neglect of the disease, the collapse of health infrastructure plus the impact of HIV pandemic

contribute to the worsening global burden of TB

Determinants of transmission: from exposure to infection (exogenous factors)

The probability of contact with a case of TB

The intimacy and duration of that contact

Degree of infectiousness of the case

The shared environment of the contact (crowding in poorly ventilated rooms)

Determinants of developing TB: from infection to disease (endogenous factors)

Innate susceptibility to disease

Level of function of the individual's cell mediated immunity

Age

- Incidence highest during late adolescence and early childhood, women aged 25 - 34 years and the elderly

Other diseases

The outcome of infection by M.tuberculosis is affected by the presence of:

HIV co- infection
Silicosis
Lymphoma
Leukaemia
Chronic renal failure and haemodialysis
Insulin dependent diabetes mellitus
Immunosuppressive treatment
Malnutrition
Old, self-healed fibrotic TB lesions

Clinical features

Generally non-specific:

Fever (low grade and intermittent)
Night sweats
Wasting
Anorexia
General malaise
Weakness
Cough (initially non-productive, subsequently productive of purulent and/or blood streaked sputum)
Haemoptysis
Chest pain
Dyspnoea
Adult respiratory distress syndrome (ARDS)
Pallor
Finger clubbing

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

Differential diagnoses

Will vary depending on the system affected:

Asthma
Bronchiectasis
Whooping cough
Inhaled foreign body
Cardiac disease
Carcinomas
Intracranial space-occupying lesions
Osteoarthritis, etc

Investigations

Sputum for AAFB, microscopy, culture and sensitivity
Tuberculin skin test
Chest radiograph
Full Blood Count; ESR
HIV screening
Urinalysis; microscopy, culture and sensitivity
CSF microscopy, culture, sensitivity; chemistry
Nucleic acid amplication
Drug susceptibility testing
Others: IVP, bone biopsy, etc as indicated

Complications

Lung abscess
Destroyed lung syndrome
Pressure effects from enlarged lymph nodes
Obstructive uropathy
Chronic kidney disease
Infertility
Skeletal deformities (varum and valgus; kyphosis, scoliosis)

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

Treatment

Regimen should include at least 4 drugs in the initiation phase

Standardized regimens are the choice in settings where susceptibility testing of reserve drugs is not available

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 (38.8°C 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 12: MUSCULOSKELETAL SYSTEM

BACK PAIN

Introduction

A common complaint which most adults will have had at one time or the other

Defined as any pain of the back, at any site between the neck and the buttocks

Low back pain is the commonest; involves essentially the lumbosacral/coccygeal spine

Most cases result from mechanical causes and usually last less than six weeks

Causes include:

Spondylosis
Intra-spinal abscess
Tumours (primary or secondary)
Osteoporosis
Osteomyelitis
Trauma
Pregnancy

Clinical features

Patients will complain of aches, pains, or sometimes peppery sensation

Pain is usually worsened on bending forward if due to a disc pathology

-Worsened when the intra-abdominal pressure is increased as in sneezing and coughing

Worsened on extension of the back if it is due to apophyseal lesion

- Most back pains are from mechanical causes and are self-limiting

There are danger or 'red flag' features that indicate more serious causes as infections, or malignanacy

- Starting for the first time in persons aged 50 years and above
- Worsened at night
- Worse on lying supine
- Associated with constitutional disturbances such as fever, loss of weight, anorexia, anaemia
- Associated with radicular pain
- Associated with structural abnormalities such as kyphosis or scoliosis

Differential diagnoses

Pancreatic or gall bladder, stomach, or intestinal disorders with referred pain
Retro-peritoneal tumours
Alcoholic gastritis
Aortic aneurysms
Tumours or inflammation of the pleura, pericardium
Metastatic bone disease
Psychosomatic disorders
Pelvic inflammatory disease

Complications

Complications of underlying cause(s) or pressure effects on the spinal cord and nerve roots

Investigations

Full Blood Counts; ESR
C-Reactive Protein
Calcium, phosphate, alkaline phosphatase levels
Radiograph of the lumbosacral spine, myelogram
CT Scan
MR1
Bone densitometry

Treatment objectives

Treat underlying cause
Relieve pain
Treat complications

Drug treatment

Paracetamol

- 1 g orally every 8 hours

NSAIDs

- Ibuprofen 1.2 - 1.8 g orally in 3 - 4 divided doses daily

Narcotic analgesics

- Morphine 10 mg orally every 4 hours (if necessary)

Antidepressants

- Amitriptyline initially 25 mg orally daily

Non-drug treatment

Physical therapy
Acupuncture
Surgery

Notable adverse effects, caution and contraindications

NSAIDs

- Individuals vary in their responses
- Should not be taken on empty stomach because of increased risk of gastric erosions and bleeding
- Particular caution in the elderly; paracetamol is very useful in treating pain of mild to moderate severity
- Combinations of different NSAIDs increases gastro-toxicity without conferring any advantage
- Interaction with antihypertensive medicines may lead to poor blood pressure control
- Interaction with warfarin: increased risk of bleeding

Morphine

- Nausea and vomiting; constipation; drowsiness; difficulty with micturition; biliary spasm; hypotension
- Dependence

GOUT

Introduction

Arises from a disorder of uric acid metabolism

Deposition of uric acid crystals in joints results in recurrent episodes of arthritis, usually in one joint

Deposition of uric acid crystals in tissues and joint destruction may occur if untreated

Clinical features

Acute presentation: acute gout

Chronic tophaceous gout: there is deposition of uric acid in tissues such as skin and kidneys

Most common in men aged 30 years and over

It has also been seen in post-menopausal women, especially those on diuretic therapy

Sudden onset of pain in a joint: usually the ankles, foot, or knee

May also present as arthritis in the big toe: podagra

Arthritis may be recurrent before attention is sought

Affected joint is exquisitely warm to touch, painful and swollen

There may be a skin reaction over the affected joint

The attack may be accompanied by fever and other constitutional symptoms

If untreated, subsequent attacks may be polyarticular or more painful

Complications

Joint-destruction if untreated
Nephrolithiasis and renal failure
Septic arthritis

Differential diagnoses

Septic arthritis
Osteoarhritis
Cellulitis
Gonococcal arthritis
Traumatic synovitis

Investigations

Serum uric acid

- Normal: 2 - 6 mg/100 mL in females; 2 - 7 mg/100 mL in males
- Normal during acute attacks in 20% of patients
- Always elevated in chronic tophaceous gout

Synovial fluid analysis and examination under polarized light microscopy for intracellular crystals of uric acid

24 hour-urine for uric acid
Radiographs of affected joints

Treatment objectives

Lower serum uric acid if above 9 mg/100mL in acute attacks

Lower the serum uric acid level in chronic tophaceous gout

Prevent joint deformity

Non-drug treatment

Dietary control: restrict purine intake by avoiding red meat, alcohol, offals of animals, salmon and sardines

Weight reduction
Physical exercise
Avoid using inflammed joint(s) during acute attacks
Avoid operating on tophi deposits

Drug treatment

Non Steroidal Anti-inflammatory Drugs (NSAIDs):

Indomethacin

- 50 mg orally three or four times daily

Or:

Ibuprofen

- 1.2 - 1.8 g orally daily in 3 - 4 divided doses

Or:

Naproxen

- 500 mg orally three times daily for 3 days then 500 mg twice daily thereafter

Or:

Diclofenac sodium
- 75 mg orally twice daily

Oral corticosteroids:
Prednisolone
- 40 mg in divided doses for 3 days, tapered over 2 weeks

Intra-articular steroids:
Triamcinolone
- 5 - 40 mg by intra-articular/intradermal injection according to patient's size (maximum 80 mg); may be repeated when relapse occurs

Or:
Methylprednisolone
- 4 - 80 mg (depending on patient's size) intra-articularly; may be repeated at intervals of 7 - 35 days

Uricosuric agents:
Allopurinol
- Initially 100 mg orally once daily then maintenance 300 - 400 mg/day

Or:
Probenecid
- 250 mg orally twice daily for 1 week, then 500 mg twice daily
- Increase up to 3 g/day

Notable adverse drug reactions, caution and contraindications
Allopurinol
- Hypersensitivity rashes
- Reduce dose in renal insufficiency

Probenecid
- Blood dyscrasias

NSAIDs
- Risk of peptic ulceration, bleeding, perforation, renal insufficiency, cardiac decompensaton

Uricosuric agents
- Not to be used during acute gout: arthritis may worsen or evolve into polyarticular disease

Prevention
Avoid alcohol
Prevent/treat obesity
Avoid drugs that elevate serum uric acid

OSTEOARTHRITIS

Introduction

A heterogenous group of diseases manifesting with symptoms and signs in the synovial joints, attributable to dysfunction of the articular cartilage and subchondral bone

It is the end result of all forms of diseases in the joints
- When such changes occur in the intervertebral disc, it is called spondylosis

Clinical features

Affects mostly females 40 years and above. If less than 40 years, underlying causes e.g. trauma or repetitive injuries should be looked for

Affects mostly weight-bearing joints such as knees, ankles. Other joints such as hips (especially in sickle cell disease), hands and spine may be affected

Presenting features are:
- Pain
- Morning stiffness of short duration
- Swelling
- Creakiness while walking
- Loss of function and deformity

Complications
Joint deformity
Septic arthritis

Differential diagnoses
Rheumatoid arthritis
Gouty arthritis
Benign Hypermobility Syndrome
Bursitis
Psoriatic arthritis

Investigations
None diagnostic:
Radiographs of affected joints
Investigations to exclude other differentials

Treatment objectives
Reduce pain
Enhance mobility
Prevent deformity

Non-drug treatment
Patient education
Exercise
Physiotherapy
Hydrotherapy
Occupational therapy
Intra-articular lavage

Drug treatment
Paracetamol
- 500 mg -1 g orally every 8 hours

NSAIDs
- Orally or local application
- Ibuprofen

Adult: 400 - 800 mg orally every 8 hours
- Naproxen

Adult: 500 mg orally every 12 hours
- Diclofenac sodium

Adult: 75 - 150 mg orally in 2-3 divided doses daily

Narcotic analgesics
- Morphine

Adult: 5 - 20 mg orally every 4 hours

Anti-depressants for night pain
- Amitriptyline

Adult: 25 - 75 mg orally daily in divided doses or as a single dose at bedtime

Capsaicin cream
0.075% cream, apply small amounts up to 3 - 4 times daily

Intra-articular steroids

- See Gout

Hyaluronate
- Injected into the joint (usually the knee), results in pain relief in 1 - 6 months, but increases inflammation in the short term

Glucosamine/chondroitin (triple strength i.e. 750/600 mg) one tablet orally every 12 hours

Indications for surgery
Intractable pain
Deformity
Disability

Alternative therapies
Acupuncture
Osteopathy
Transcutaneous Electrical Nerve Stimulation (TENS)

Notable adverse drug reactions, caution and contraindications
NSAIDs
- Gastro-intestinal side effects which may be mild (e.g. dyspepsia, nausea, constipation and diarrhoea) or serious (e.g. perforation, ulceration, bleeding and stenosis)
- May also cause pruritus, rashes, fixed drug eruptions; dizziness and drowsiness; renal insufficiency/renal failure, especially in the elderly

Prevention
Reduce weight
Regular exercise
Treat early

RHEUMATOID ARTHRITIS

Introduction

A chronic inflammatory disease of unknown cause

Possibly occurs as a result of auto-immunity

Affects primarily the peripheral joints in a symmetric pattern; may affect other organs

Clinical features

Clinical manifestations are usually preceeded by constitutional symptoms such as fatigue, malaise, fever, weight loss, loss of appetite

Joint involvements are characterized, serially or simultaneously, by the following
Significant joint morning stiftness
Polyarthritis
Arthritis of joints of the hands
Bilaterally symmetrical arthritis
- Any joint could be affected but mostly the knees, ankles, hips, shoulders, elbows; not joints of the back

Other clinical features
Rheumatoid nodules
Lymph glands enlargement
Anaemia
Hepatosplenomegaly

Differential diagnoses
Systemic Lupus Erythematosus
Polyarticular gout
Fibromyalgia syndrome
Sjogren's syndrome
Osteoarthritis
Hepatitis B

Complications
Chronic pain
Joint instability and deformity
Pulmonary fibrosis
Ischaemic heart disease
Eye involvement
Malignancies: lymphoma

Investigations
Full Blood Count; ESR
Rheumatoid factor
Synovial fluid analysis
Radiographs of affected joints

Treatment objectives
Reduce pain and disability
Limit joint damage
Improve quality of life

There is no cure

Non-drug treatment
Education
Physiotherapy
- Improve mobility
- Increase muscle power
- Reduce pain and disability

Drug treatment
- Paracetamol

Adult: 500 mg orally three times daily

Child 1 - 5 years: 120 - 250 mg; 6 -12 years: 250 - 500 mg; 12 - 18 years: 500 mg every 4 - 6 hours (maximum 4 doses in 24 hours)

Non-steroidal anti-inflammatory drugs
- Ibuprofen

Adult: 400 - 800 mg orally every 8 hours

Child 1-3 months: (and body weight >5 kg), 5 mg/kg orally3 - 4 times daily preferably after food; in severe conditions and weight >5 kg, maximum 30 mg/kg in 3 - 4 divided doses

3 months - 1 year and body weight >5 kg: 50 mg 3 - 4 times daily; in severe conditions up to 30 mg/kg in 3 - 4 divided doses

1 - 4 years: 100 mg every 6 - 8 hours daily; in severe conditions up to 30 mg/kg in 3 - 4 divided doses

4 - 7 years: 150 mg every 8 hours; in severe conditions up to 30 mg/kg in 3 - 4 divided doses, maximum 2.4 g daily

7 - 10 years: 200 mg every 8 hours; in severe conditions up to 30 mg/kg in 3 - 4 divided doses, maximum 2.4 g daily

10 - 12 years: 300 mg every 8 hours; in severe conditions up to 30 mg/kg in 3 - 4 divided doses, maximum 2.4 g daily

12 - 18 years: 300 - 400 mg very 6 - 8 hours daily, preferably after food, increased if necessary to maximum 2.4 g daily

- Diclofenac potassium
Adult: 25 - 50 mg orally every 8 hours
Child 14-18 years: 75-100 mg daily in 2-3 divided doses
Corticosteroids
- Prednisolone: low dose, up to 15 mg orally daily
- Triamcinolone and methylprednisolone into joints
(See Gout)
Disease Modifying Anti-Rheumatic Drugs (DMARDs)
- Methotrexate
Adult: 10 - 25 mg orally once weekly
Child 1 month - 18 years: 10 - 15 mg/m^2 once weekly, increased if necessary to a maximum of 25 mg/m^2 once weekly: by oral, subcutaneous or intramuscular route
- Azathioprine
Adult: 50 - 150 mg orally daily
Child 1 month - 18 years: initially 1 mg/kg daily, adjusted according to response to a maximum of 3 mg/kg daily
(Consider withdrawal if no improvement within 3 months)
- Hydroxychloroquine sulphate
Adult: initially 400 mg orally daily in divided doses; maintenance 200 - 400 mg (but not exceeding 400 mg) daily **or**
Child 1 month - 18 years: 5 - 6.5 mg/kg orally (maximum 400 mg) once daily
Or:
- Chloroquine base
Adult: 150 mg orally daily (maximum 2.5 mg/kg daily)
Child: up to 3 mg/kg orally daily
- To be administered on expert advice
In unresponsive cases, refer for specialist care
Notable adverse drug reactions, caution and contraindications
NSAIDs
- May cause severe gastrointestinal side effects e.g. peptic ulceration, bleeding, perforation
- Renal and cardiac failure especially in elderly persons (should be used with caution)
DMARDs
- Bone marrow suppression
- May also cause lymphoma
Methotrexate
- Pulmonary fibrosis, hepatotoxicity
- Regular Full Blood Count including differentials, renal and liver function tests are required
- Concomitant administration of folic acid may reduce mucosal and gastrointestinal side effects

SEPTIC ARTHRITIS

Introduction

An inflammation of synovial tissues by bacteria, with production of pus into the joint space

Also variously called suppurative, purulent or infective arthritis

Rare, but may cause a lot of illness and early joint destruction or deformity

Septic arthritis is broadly categorized as:
Gonococcal
Non-gonococcal

S. aureus, streptococci, candida species, *M.tuberculosis*, HIV, hepatitis B virus

Clinical features

Frequency in most studies is about 2 - 10 cases per 100,000

May occur on its own, or in association with other forms of arthritis such as gout, rheumatoid arthritis and osteoarthritis

Causative organisms are mostly *S.aureus*, and streptococci. Other organisms include *H.influenzae, Neisseria gonorrhoeae*

Typical presentations:
Fever
Hot, painful and distended joint with pus
Markedly decreased range of motion

Occasionally, septic arthritis may present with a migratory polyarthralgia and dermatitis, especially with gonococcal infection

Constitutional symptoms such as nausea, vomiting, headaches, loss of weight, loss of appetite may also be seen

Differential diagnoses
Malaria fever
Acute gouty arthritis
Osteoarthritis
Rheumatoid arthritis

Complications
Irreversible joint destruction
Degenerative joint disease
Osteomyelitis
Soft tissue injury

Investigations
Full Blood Count and differentials
ESR
Blood cultures
Urethral, cervical and rectal cultures
Synovial fluid analysis
Main radiographs of affected regions
Ultrasonography

Treatment objectives

Initiate appropriate antibiotics therapy early to prevent joint damage

Prevent septicaemia arising from the joint

Drug treatment

Antibiotic choice (based on culture report)
- Cefriaxone 1 g intravenously every 24 hours
Treatment may be continued for 4 weeks
- There can be a change to oral antibiotics after the first week

Joints infected with *N. gonorrhoeae* respond to 1 week of intravenous ceftriaxone followed by ciprofloxacillin

500 mg orally twice a day for another 1 week

Surgical measures
Needle aspiration
Arthroscopic drainage and lavage
Open drainage and lavage

Prevention

Effective treatment of the primary infective agents and other predisposing disease states e.g. sickle cell disease, complicated fractures

Attention to asepsis in joint manipulation procedures and during intra-articular diagnostic/therapeutic interventions

SYSTEMIC LUPUS ERYTHEMATOSUS

Introduction

A chronic, multisystemic, auto-immune inflammatory disease that affects virtually any organ in the body

Typically runs a relapsing and remitting course

Affects mainly women of child-bearing age

Particularly common among Blacks and Asians, in whom it runs a more devastating course

Clinical features

Affects one or more organs simultaneously

Skin and joints most affected but may also affect the central nervous system and kidneys

Onset usually preceded by constitutional symptoms:
Fever
Marked weight loss
Loss of appetite
Aches and pains all over the body

Typical characteristics are seen serially or simultaneously:
Joint pains
Malar rash
Discoid skin rash
Photosensitivity
Mouth or pharyngeal ulcers
Pleurisy
Pericarditis
Renal failure
Nephritis
Nephritic syndrome
Seizures
Psychosis
Peripheral neuropathy
Transverse myelitis
Eye involvement
Recurrent abortions

Complications
Opportunistic infections
Avascular necrosis
Premature atherosclerotic disease
Myocardial infarction

Differential diagnoses
Malaria
Rheumatoid arthritis
Typhoid fever
Hepatitis
Fibromyalgia syndrome
Scleroderma
Mixed connective tissue disease
Benign hypermobility syndrome
Drug-induced SLE

Complications
Opportunistic infections
Avascular necrosis
Premature atherosclerotic disease
Myocardial infarction

Investigations

Full Blood Count: leucopaenia, thrombocytopaenia, anaemia
ESR, CRP
Urine analysis and microscopy: albuminuria, casts, haematuria
Urea, Electrolytes and Creatinine
LE cell test
Serology: ANA, Anti-ds DNA, Anti-SM
Ro/Ssa; La/SSB, Anti-Cardiolipin antibody
Radiographs of affected joints
Echocardiogram
MRI

Treatment objectives
Reduce pain
Improve mobility
Prevent such organ involvement as kidney and brain
There is no cure for the disease

Non-drug treatment
Patient education
Physiotherapy
Occupational therapy
Adequate nutrition
Exercise to prevent contractures

Drug treatment
Non-Steroidal Anti-inflammatory drugs (NSAIDs)
- See above
Anti-malarials
- Hydroxychloroquine
Adult: 200 mg orally daily
Child 1 month - 18 years: 5 - 6.5 mg/kg orally (maximum 400 mg) once daily
Or:
Chloroquine
Adult: 150 - 300 mg base daily
Child: up to 3 mg/kg orally daily
Corticosteroids
- Pulse methylprednisolone
Adult: 1 g/day intravenously for 3 days
- Used for organs or life-threatening exarcerbations
Or:
- Prednisolone
Adult: 0.5 mg - 1 mg/kg orally daily

Immunosuppressives
- Methotrexate
Adult: 7.5 mg - 15 mg orally weekly
Child 1 month - 18 years: 10 - 15 mg/m^2 once weekly, increased if necessary to a maximum of 25 mg/m^2 once weekly: by oral, subcutaneous or intramuscular route
Or:
- Azathioprine
Adult: 2.3 mg/kg orally daily
Child 1 month - 18 years: initially 1 mg/kg daily, adjusted according to response to a maximum of 3 mg/kg daily
Or:
- Cyclophosphamide
Adult: 500 - 750 mg/m^2 intramuscularly or intravenously monthly
Child: not listed for this indication

Notable adverse drug reactions

NSAIDs:
- Gastrointestinal side effects: perforation, bleeding, ulceration
- Renal failure
- Cardiac failure
- Hepatotoxicity
- CNS involvement

Methotrexate
- Pulmonary fibrosis
- Hepatocellular damage
- Immune suppression

Azathioprine
- Risk of neoplasia
- Hepatocellular damage
- Bone marrow suppression

Cyclophosphamide
- Haemorrhagic cystitis
- Ovarian failure
- Bone marrow suppression
- Bladder malignancy

Prevention

No primary prevention
Relapses can be prevented by:
Avoiding ultraviolet light exposure to sun
Anti-malarial therapy
Treating hypertension adequately
Correcting dyslipidaemia
ACE inhibitors (to limit renal damage)

Chapter 13: OBSTETRICS AND GYNAECOLOGY

ABORTION

Introduction

Expulsion from the mother's uterus of a growing and developing embryo or foetus prior to the stage of viability (about 20 weeks), with foetal weight less than 50 g

One of the leading causes of maternal mortality and morbidity in Nigeria

May be:
Spontaneous
- Occurring from natural causes

Induced
- Brought about purposefully by drugs or mechanical means

Accidental
- Due to a fall, blow or other injury

Complete
- With complete expulsion or extraction from the mother of a foetus 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

Illegal (criminal)
- Termination of a pregnancy without legal justification

Legal
- With or without medical justification but done in a manner that is legal

Solitary
- A single experience of an abortion

Habitual
- When a woman has had three or more consecutive, spontaneous abortions

Clinical features

Threatened abortion:
Cramp like pains
Slight show of blood
May or may not be followed by the expulsion of the foetus
Occurs during the first 20 weeks of intrauterine life ('pre viability' period)

Imminent /incipient/impending abortion:
Copious vaginal bleeding
Uterine contractions
Cervical dilatation

Inevitable abortion:
Rupture of the membranes in the presence of cervical dilatation in a pre-viable pregnancy

Ampular/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

Antepartum haemorrhage
Ectopic pregnancy
Hydatidiform mole
Carcinoma of the cervix
Rape

Investigations

Pelvic ultrasound scan
Abdominal radiograph
Chest radiograph
Microscopy, culture and sensitivity test of vaginal discharge
Urinalysis; urine microscopy, culture and sensitivity
Full Blood Count
Blood Group

Complications

Endometritis
Parametritis
Peritomitis
Haemorhage
HIV infection
Secondary infertility
Perforation of the uterus and/or intestines
Rupture of the bladder

Treatment objectives

Restore haemostasis
Prevent/treat complications
Provide health education

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 of complication(s)

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

ANC is clinical assessment of mother and foetus, with an overall goal of obtaining the best possible outcomes for both

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 foetal risk
Prenatal diagnosis and management of foetal 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, foetus, 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

Pre-conception visit
1st ANC visit

- Best before, and not later than the 12th week
2nd ANC visit
- Scheduled around the 26th week
3rd ANC visit
- Scheduled around the 32nd week
4th ANC visit
- Between the 36th and 38th 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

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

1st ANC visit

Should be in the 1st trimester, preferably before the 12th 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
- SFH 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 for referral
- Any medical or obstetric conditions that require specialized care
Investigations
-Urinalysis for bacteriuria, proteinuria and glycosuria
- Haemoglobin genotype
- Blood group
- HIV screening
- VDRL
- Haemoglobin concentration/packed cell volume

Interventions

Iron
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 26th week
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, oedema
- Blood pressure
- SFH

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 sufadoxine / pyrimethamine
- One full treatment dose in the 2nd and 3rd trimesters
- 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 visit

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, oedema, dyspnoea
- Breast examination
- Blood pressure
- Abdomen: SFH palpation for twin gestation

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 (2nd injection)
Antimalarials
Maintain complete records: clinic as well as ANC card records

4th ANC visit

The final visit before labour or delivery
Should take place about or between the 36th - 38th weeks
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, foetal 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 41st 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 2nd and 3rd trimesters
- 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

Introduction

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 haematocrit less than 33% in peripheral blood
For practical purposes in developing and tropical countries a haemoglobin concentration of 10 g/dL or haematocrit of 30% is taken as cut off
- Below these levels there may be adverse foetal 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
Dyspnoea on mild exertion
Ankle swelling

Signs

Pallor
Jaundice may or may not be present
Pedal oedema
Tachypnoea
Tachycardia
Haemic murmurs
Pseudo-toxaemia
- Systolic hypertension, oedema 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
Excessive sweating in the tropics
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
Death

Foetal
Abortion
Intrauterine growth restriction
Intrauterine foetal 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
Ultrasound scan (e.g. of abdomen, pelvis)
Bone marrow biopsy if bone marrow involvement is suspected

Treatment objectives
Correct haematocrit
Treat underlying cause(s)
- See differential diagnoses

Foetal surveillance
- Of growth and wellbeing for IUGR and intrauterine asphyxia

Correction of haematocrit
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 37th week for mild anaemia and from the 32nd 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 in 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 pre-clinical 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; chlamydiae
Early first child birth
High parity

Low socio-economic status
Smoking
Micronutrient deficiency
Oral contraceptive usage
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
- Postcoital
- 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 on to the vagina wall in advanced stages
Vesico-vaginal fistula in advanced stages
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
Endometritis: particularly atrophic
Choriocarcinoma
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, 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
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

Classs 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 subxiphoid pain; bloating, heartburn
Heat intolerance, sweating and flushing

Signs

Plethoric facies
Odema (legs; occasionally hands and face)
Varicose veins
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_1 and S_2 heart sounds
Murmurs
Crepitations

Investigations

Full Blood Count
Serum Electrolytes, Urea and Creatinine
Urinalysis
Blood Glucose
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 foetus developing congenital heart disease; foetal growth restriction
- Possibility of pre-term labour
- Need for frequent hospital attendance; possibly admission
- Need for intensive maternal and foetal 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 oedema
- 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
Anticoagulation
- 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)

Foetal surveillance:
- Ultrasound scan particularly for cardiac anomaly at 22 weeks

Delivery:
- Either for maternal or foetal 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 anesthesia by a senior anesthetist
Shorten 2nd 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

Foetal

Rheumatic heart disease:
Intrauterine growth restriction; pre-term delivery
Cyanotic congenital heart disease:
Poor outcomes; up 40% foetal loss
Uncorrected coarctation of aorta:
Foetal growth restriction in > 10% of cases
Pre-maturity
Small for gestation age
Intrauterine growth restriction
Intrauterine foetal 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 caused by epilepsy or other convulsive disorders

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 unbooked 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 oedema

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

Foetal

Prematurity
Intrauterine growth restriction
Intrauterine foetal death
Brain damage
Death

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:
- Uraemia
- Brain tumours or abscesses
- Cerebral haemorrhage
- Poisoning (accidental or intentional)
- Hysteria

Investigations

Haemoglobin concentration/haematocrit
Bedside crude clotting time
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 foetus 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, 24-hour 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 1litre 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 - 4 g 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
1 g 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
Indwelling 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 foetal 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 foetal and maternal outcomes

Re-occurence
- 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 advise 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 but in the mildest form

"Silent"/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
Adnexial 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/abdomen
Serum ß-hCG (where available) especially in silent cases
Paracentesis abdominis (should be considered)
Laparoscopy
- Final arbiter when the diagnosis is in doubt

Treatment objectives
Depend on the clinical subset
Preserve maternal life

Acute ectopic
Immediate resuscitation (fluids/blood)
Stop haemorrhage: by 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
- 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 6th week and stops spontaneously before the 14th week

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

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
Cholescystitis
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 hypokalaemia); vitamin deficiencies

In neglected or poorly managed cases:

Severe weight loss
Tachycardia
Hypotension
Oliguria
Neurologic disorders from vitamin B_1 deficiency
Retinal haemorrhages
Jaundice (from hepatic necrosis)
Oesophageal tears and spontaneous rupture of the oesophagus
Mendelson's syndrome
Foetal 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

Management

Admit
Strict intake-output monitoring
Intravenous fluid therapy to:
- Correct electrolyte disturbances
- Provide calories
- Rehydrate the patient

Anti-emetics

Those which have been proven not to be teratogenic:
- Meclozine 25 mg orally

Or:
- 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

IMMUNIZATION SCHEDULES

Introduction

Tetanus immunization for the pregnant woman is geared towards protecting the mother (and baby) against tetanus

Tetanus Immunization Schedule in Pregnancy

TIMING OF IMMUNIZATION	PROTECTION OFFERED
1st dose at booking or on 1st contact	Confers no protection
2nd dose at 4 weeks after 1st dose	Confers protection for 3years
3rd dose at 6 months after 2nd dose	Confers protection for 5years
4th dose at 1 year after 3rd dose or in next pregnancy	Confers protection for 10 years
5th dose at 1 year after 4th dose or in next pregnancy	Confers protection for life

Immunization and Vitamin A Schedule

At Delivery	Vitamin A to Mother
At Birth	BCG; $POLIO_0$; HBV_1
6 Weeks	DPT_1; $POLIO_1$; HBV_2
10 Weeks	DPT_2; $POLIO_2$
14 Weeks	DPT_3; $POLIO_3$; HBV_3
9 Months	MEASLES; YELLOW FEVER; 1st Dose Vitamin A_1
15 Months	Vitamin A_2

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 alkaline phosphatase

- 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

Cogenital 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 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 bleeding complications

Management

Early diagnosis is mandatory

- Clinical features with evidence of deranged LFTs and of renal failure

The management it requires a combined team of obstetrician, physician and anesthetist

Definitive treatment

Deliver the baby as soon as possible (frequently by Caesarean section)

Supportive measures

Transfusion with blood, fresh frozen plasma, platelets as indicated

Dialysis

Complications

Disseminated intravascular coagulopathy

Hypotension

Significant risk of maternal and foetal 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 36weeks

Clinically significant because of its association with IUGR and IUFD (mechanism unclear)

It is not as a rule associated with maternal complications

Clinical features

Generalized pruritus

Decreased foetal 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

Foetal surveillance: by growth (serial USS biometry) and wellbeing (CTG) 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 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

Foetal

Foetal distress

Still-birth

Perinatal death

Prematurity and its problems

Meconium staining of the liquor

Management

Careful maternal follow-up with LFTs

Foetal 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 oestrogen-containing oral contraceptive pills

Viral hepatitis

The most common cause of jaundice in pregnancy, accounting for about 40% of the causes

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 foetus

- Unlike other hepatotrophic viral infections, which carry a significant risk of vertical transmission (particularly in the third trimester)

A severe attack may influence foetal 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

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

Adnexial tenderness

Minor criteria

Fever (38^0C)

Leucocytosis

Purulent vaginal discharge

Adnexial 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, adnexial mass (e.g. ovarian mass)

<u>Indications for admission</u>

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

Out patient therapy while awaiting culture results:

Ceftriaxone (or equivalent cephalosporin)

- 1 g 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

<u>Inpatient triple therapy</u>

- 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

<u>Early</u>

Vaginal/perineal swab for microscopy, culture and sensitivity

Semen: DNA analysis

<u>Late</u>

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)

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

CHAPTER 14: RESPIRATORY SYSTEM

ACUTE EPIGLOTTITIS

Introduction

A life threatening, rapidly progressive cellulitis of the epiglottis that may cause complete airway obstruction

Most common in children, in whom *Haemophilus influenzae* is the most common pathogen

In adults, is often caused by *Strept. pneumoniae* and group A streptococcus

Clinical features

Fulminant presentation in children with:

Fever
Irritability
Cough
Dysphonia
Airway occlusion
Dysphagia
Dyspnoea
Drooling
Stridor

Adults' symptoms are less fulminant, presenting with:

Sore throat
Dysphagia
Dyspnoea

Absence of hoarseness distinguishes acute epiglottitis from acute laryngitis

Differential diagnoses

Acute laryngitis
Laryngo-tracheo-bronchitis (Croup)

Complications

Complete airways obstruction and asphyxiation

Investigations

Lateral X-ray of the neck

"Thumb sign" appearance of the enlarged epiglottis

Blood culture

Do not view the epiglottis using a tongue depressor: this may cause laryngospasm, with complete respiratory obstruction

Treatment objectives

Safeguard the airway
Control infection

Drug treatment

Cefuroxime

Adult: 250 mg orally every 12 hours for 5 - 10 days

Child: 125 mg orally every 12 hours for 5 - 10 days

Or:

Ceftriaxone

Adult: 250 - 500 mg intramuscularly or intravenously for 5 - 10 days

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

Supportive measures

Oxygen
Steam inhalation
Nasotracheal intubation may be required
Maintain adequate caloric intake and hydration

Notable adverse drug reactions, caution

Cefuroxime: avoid in pregnancy and in patients with renal impairment

Ceftriaxone: rashes, fever, gastrointestinal disturbances

- Dose reduction in the elderly

Prevention

Haemophilus influenzae vaccine

Child 2 months - 18 years: 0.5 mL

- Should be available as part of childhood immunization

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, adju sted 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)

Introduction

Inflammation of the mucosal surface of the nose, most commonly due to infection with respiratory viruses

Clinical features

Tickling sensation in the nose associated with itching of the nose and palate

Watery nasal discharge (rhinorrhoea), which may later become purulent

Sneezing
Headaches
Nasal obstruction (usually alternating)

Differential diagnoses

Allergic rhinitis
Vasomotor rhinitis
Bacterial rhinitis (often supervenes after the viral onset)

Complications

Superimposed bacterial rhinitis

- Suspect this if symptoms last longer than 7 - 10 days

Sinusitis
Lower respiratory infection
Otitis media

Obstruction of internal auditory meatus: may cause deafness

Treatment objectives

Relieve nasal mucosal oedema and obstruction
Relieve pain/discomfort
Treat complications

Drug treatment

Analgesics

- Paracetamol

Adult: 1 g orally three times daily to relieve headaches or fever

Child 1 - 5 years: 120 - 250 mg; 6 -12 years: 250 - 500 mg; 12 - 18 years: 500 mg 4 - 6 hourly (maximum 4 doses in 24 hours)

Antibiotics

- Only if secondary bacterial infection occurs

Supportive measures

Steam inhalation with a drop of eucalyptus oil

Notable adverse drug reactions

Paracetamol: raised liver enzymes, renal papillary necrosis

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

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 $ß_2$ agonist
- 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 $ß_2$ 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 - 2 mg/kg orally once daily for 3 - 5 days
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 prepartations
Aminophylline
- Do not exceed 500 mg in 24 hours because of the risk of cardiac arrhythmias
- May cause CNS stimulation with insomnia and convulsions
Steroids
- Immunosuppression, 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

BRONCHIECTASIS

Introduction
Abnormal and permanent dilatation of medium sized bronchi
A consequence of inflammation and destruction of the structural components of the bronchial wall, caused by bacterial or viral infections
May be focal or diffuse
Clinical features
Persistent or recurrent cough
Purrulent 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 brain 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: biopsy 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: 500 mg -1 g orally every 8 hours for 5 - 7 days
Child: 40 mg/kg orally in 3 divided doses daily
- Cotrimoxazole
Adult: 960 mg orally every 12 hours for 5 - 7 days
Child: 6 weeks to 5 months: 120 mg orally; 6 months - 5 years: 240 mg; 6 - 12 years: 480 mg
Appropriate antibiotics as soon as culture results are available
Bronchodilators
- Salmeterol xinafoate
Adult: 2 puffs (50 micrograms) twice daily
- Can be doubled in severe airway obstruction
Child: same as adult dose (for children > 4 years)
- Salbutamol
Adult: 1 - 2 puffs (100 - 200 micrograms) 3 - 4 times daily
Child: usually 100 microgram (1 puff) 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 prepartations
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

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 g orally every 8 hours
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 - 18 years: 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

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 influenzae* and *Streptococcus pneumooniae*
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 $ß_2$ - agonist
- See bronchial asthma
Theophylline
- Aminophylline (see bronchial asthma)
Antibiotics (when necessary to control infection)
- 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: 2 - 8 years: 250 mg orally every 6 hours
Up to 2 years: 125 mg every 6 hours
- Co-amoxiclavulanate

Adult: 500/125 mg orally every 12 hours
Child 1 month -1 year: 0.25 mL/kg of 125/31 mg suspension orally every 8 hours; dose doubled in severe infections
1 - 6 years: 5 mL 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

Supportive measures
Assisted ventilation
Hydration
Pulmonary physiotherapy

Prevention
Avoidance of cigarette smoking
Avoid / remove atmospheric pollutants

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 - 10 mL 3 - 4 times daily
- Not recommended in children

Appropriate antibiotics for bacterial infections

Notable adverse drug reactons, 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 the underlying/precipitating cause

LUNG ABSCESS

Introduction
Suppuration of the lung parenchyma

May be due to:
Infection by aspirated oro-pharyngeal anaerobes
Inadequately treated pneumonia caused by *Staphylococcus aureus*, *Mycobacterium tuberculosis*
Bronchial obstruction.

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
Pulmonary amyloid

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: 500 mg orally every 8 hours
Child: neonate, initially 15 mg/kg orally then 7.5 mg/kg every 12 hours; 1 month - 12 years: 7.5 mg/kg (maximum 400 mg) every 8 hours; 12 - 18 years: 400 mg every 8 hours

Plus:
Amoxicillin
Adult: 500 mg orally every 8 hours for 7 - 10 days
Child less than 5 years: a quarter adult dose; 5 - 10

years: half adult dose
Or:
Amoxicillin/clavulanic acid
Adult: 1 g/200 mg orally every 8 hours for 7 - 10 days
(Definitive antibiotic therapy should be based on culture and sensitivity results)

Prevention

Good dental care
Adequate treatment of acute pneumonia
Prevent pneumonia with vaccination in persons 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

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
Streptococcus pneumoniae is the most common pathogen in community-acquired pneumonia
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
Atypical pneumonia:
Gradual onset
Dry cough
Prominent extra-pulmonary symptoms
Headache
Sore throat
Fatigue
Myalgia
Chest crackles or rales

Differential diagnoses

Pulmonary embolism
Septicaemia

Complications

Lung abscess
Pleural effusion
Empyema thoracis
Septicaemia
Endocarditis
Meningitis

Investigations

Sputum examination
Haematological evaluation
Sputum culture
Chest radiograph
Blood cultures
Serologic studies

Treatment objectives

Eliminate the infection
Return to normal lung function

Drug treatment

Antibiotics
- Co-amoxiclavulanate

Adult: 1 g/200 mg orally every 12 hours for 5 -7 days
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
Or:
- Benzyl penicillin

Adult: initially 1.2 g (2 million units) intravenously every 6 hours
Child: preterm and neonate under 7 days, 25 mg/kg by intramuscular injection or by slow intravenous injection or infusion every 12 hours; dose doubled in severe infection
Neonate 7 - 28 days: 25 mg/kg every 8 hours; dose doubled in severe infection
1 month - 18 years: 25 mg/kg every 4 - 6 hours, increased to 50 mg/kg every 4 - 6 hours (maximum 2.4 g every 4 hours) in severe infection
- Commence oral therapy as soon as practicable

Or:
- Cefuroxime axetil

Adult: 500 mg orally every 8 hours for 5 - 7 days
Child 3 months - 2 years: 10 mg/kg (maximum 125 mg) orally every 12 hours; 2 - 12 years: 15 mg/kg (maximum 250 mg) every 12 hours daily; 12 - 18 years: 250 mg every 12 hours; dose doubled in severe infection

Supportive measures

Analgesics
Hospitalization may be necessary in severe infection
Adequate hydration.
Supplemental oxygen if cyanosis is present

Notable adverse drug reactions, caution and contraindications

Co-amoxiclavulanate: nausea, diarrhoea, skin rashes
- Contra indicated in penicillin-hypersensitive individuals

Cefuroxime: nausea, vomiting, abdominal discomfort,

headaches
- Rarely, antibiotic-associated colitis

Prevention

Pneumococcal vaccine
Haemophilus influenzae vaccine

PULMONARY EMBOLISM

Introduction

Occurs when a venous thrombus is dislodged from its site of formation (thrombotic embolus) or a fat globule from a long bone fracture or crush tissue injury or even a tumour fragment (non-thrombotic embolism), is carried in the blood stream to the pulmonary arterial circulation causing obstruction to alveolar perfusion

Clinical features

Massive embolus in main pulmonary artery:
Sudden death
Sudden onset dyspnoea
Tachypnoea
Tachycardia
Small volume pulse
Hypotension
Circulatory collapse
Raised jugular venous pressure
Small-to-moderate embolus:
Cough
Pleurtic chest pain
Haemoptysis
Tachycardia
Left parasternal heave
Loud pulmonary component of second heart sound
Fever
Signs of lung consolidation
Pleural friction rubs

Differential diagnoses

Myocardial infarction
Unstable angina
Pericarditis
Exacerbation of chronic bronchitis
Congestive cardiac failure
Pneumothorax

Complications

Sudden death
Pulmonary infarction
Lung abscess

Investigations

Electrocardiography
- Sinus tachycardia
- Atrial fibrillation
- Right bundle branch block
- Right axis deviation <90°
- T wave inversion
- Q waves in leads III, AVF, V3

Chest radiograph
May be normal or show:
- Focal oligaemia
- Pleural effusion
- Wedge-shaped opacity (Hampton's hump)

Ventilation/perfusion scan
Arterial blood gas analysis: hypoxaemia, respiratory alkalosis
Full Blood Count: leucocytosis
Raised ESR
Raised LDH levels

Treatment objectives

Prevent fatality
Restore normal lung perfusion

Non-drug treatment

Primary measures:
Embolectomy
Supplemental oxygen
Psychological support

Drug treatment

Anticoagulants
- Heparin

Adult: 5,000 units (10,000 in severe pulmonary embolism) loading dose then continuous infusion at a rate of 15 - 25 units/kg/hour
Child: neonate, initially 75 units/kg (50 units/kg if under 35 weeks post-menstrual age), then 25 units/kg/hour by intravenous injection, adjusted according to APTT
1 month - 1 year: same as for neonate
1 year - 18 years: initially 75 units/kg by intravenous injection, then 20 units/kg/hour by continuous intravenous infusion, adjusted according to APTT
Or:
- Enoxaparin

Adult: 1.5 mg/kg (or 150 units/kg) by subcutaneous injection every 24 hours, for at least 5 days (until adequate oral anticoagulation is established)
Child: neonate, 1.5 - 2 mg/kg by subcutaneous injection twice daily; 1 - 2 months: 1.5 mg/kg twice daily; 2 months - 18 years: 1 mg/kg twice daily
- Warfarin

Adult: initially 10 mg orally daily for 2 days
Child: neonate (under specialist advice), 200 micrograms/kg once daily as a single dose on first day, then on the following 2 days
1 month - 18 years: 200 micrograms/kg (maximum 10 mg) as a single dose on first day, reduced to 100 micrograms/kg (maximum 5 mg) once daily for following 2 days
- Usual maintenance dose: 100 - 300 micrograms/kg once daily
- Subsequent doses depend on prothrombin time (INR)

Thrombolytic agents
- Recombinant tissue plasminogen activator

Adult: 10 mg by intravenous injection given over 1 - 2 minutes; then intravenous infusion of 90 mg given over 2 hours
- Not exceeding 1.5 mg/kg in persons less than 65 kg

Notable adverse drug reactions, caution and contraindications

Heparin:
- Thrombocytopaenia and haemorrhage
- Osteopaenia
- 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

CHAPTER 15: INJURIES AND ACUTE TRAUMA

BITES AND STINGS

Introduction

Bites occur from:
Humans
Domestic animals such as cats and dogs
Wild animals e.g. snakes, sharks and crocodiles

Stings often occur from:
Bees, wasps and other insects
Marine invertebrates such as the jellyfish, corals, scorpions and anemones

The microbiology of bite wound infections reflects the oro-pharyngeal flora of the biting animal
- Organisms from the soil, skin of the animal and victims, animal feaces may also be present

Clinical features

Depend on the type of injury, and the delay before presentation in hospital

Bites from common domestic animals usually result in bruises, lacerations and haemorrhage;

Rabies may complicate dog bites

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

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 :
- Subfamily viperinae (the Old World vipers)
- Crotalinae (the 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)

In Africa the vipers are responsible for most snake bites.

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

<u>Viperidae and crotalidae</u>

Local and systemic bleeding
Impairment of organ function
Reduction of cardiac output
Inhibition of peripheral nerve impulses
Multisystem effects
Rhabdomyolysis
Haemolysis
Blood vessel damage

<u>Elapidae</u>

Neurotoxic effects

Snake bite wounds may become secondarily infected with:
- *Clostridium tetani,* causing tetanus
- *Clostridium welchi*, causing gas gangrene

<u>Indications for antivenom treatment</u>

Hypotension
Vomiting
Hand or foot bite swellings extending beyond the wrist or ankle within 4 hours of the bite
Electrocardiograph abnormalities

Sharks and crocodiles

Cause death by:
Tissue destruction
Crush syndrome
Haemorrhage
Infection

Bees and wasps

Are the most common causes of stings

They leave their stinging apparatus behind in the skin

The symptoms that follow bee stings are those due to anaphylaxis to their venom

Marine invertebrates

Have specialized organelles called nematocysts for poisoning and capturing prey

May cause serious ill health and death

Initial assessement

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

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

Administration of high flow oxygen

Intravenous fluid administration to maintain circulation: use colloids or cystalloids as clinically appropriate

Treatment of anaphylaxis with antihistamines (H_1 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

Indications for anti-snake venom treatment

Symptoms or signs of systemic envenoming: hypotension, angioedema, urticaria, diarrhoea and vomiting, spontaneous bleeding, adult respiratory distress syndrome, acute renal failure, etc

Electrocardiograph abnormalities

Marked local envenoming e.g. swelling extending beyond wrist within 4 hours of bite on hand, or beyond ankle after bite on foot

Adult and child: contents of the antivenom vial diluted in sodium chloride 0.9% intravenous infusion, and infused intravenously over 30 minutes

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:

- **A**irway
- **B**reathing
- **C**irculation
- **D**isability
- **E**xposure

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

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,
Medications,
Past medical history, pregnancy,
Last 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 and 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
- A patent 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)

<u>Secondary brain injury</u>

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

<u>CT scan in TBI</u>

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

<u>Indications for CT scan</u>

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

<u>Intracranial pressure monitoring</u>

Best done through a ventriculostomy catheter, with or without concomitant intraparenchymal transducer

<u>Indications for ICP monitoring in TBI</u>

Patients with post resuscitation GCS of 8 or less
Intubated patients in ICU

Patients with intracranial haematomas but are adjudged not to need surgery

<u>Emergency management of raised intracranial pressure</u>

Endotracheal intubation
Controlled ventilation to a pCO_2 of 35 mmHg
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

<u>Surgery in TBI</u>

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

<u>Drugs in TBI</u>

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*** and ***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
- A radiographer
- 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 prehospital 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 16: SURGICAL CARE AND ASSOCIATED DISORDERS

ACUTE ABDOMEN

Introduction

An abdominal condition of sudden onset requiring immediate (urgent) attention

A common surgical emergency

Aetiology

Surgical:

Inflammatory/infective conditions:

- Acute appendicitis: the commonest cause of acute abdomen
- Acute salpingitis: a common cause in sexually active young females
- Acute cholecystitis
- Acute pancreatitis
- Acute diverticulitis: not very common in this environment

These conditions usually begin with a localized peritonitis which progresses to generalized peritonitis if left untreated.

Perforation of hollow viscera:

- Perforated chronic duodenal ulcer
- Perforated typhoid ileitis: a common cause in this environment
- Traumatic gastrointestinal perforation
- Perforated gastrointestinal malignancies

Intestinal obstruction:

- Strangulated external and internal hernias
- Intussusception
- Peritoneal adhesions and bands (congenital or acquired)
- Gastrointestinal tumours

Intra-abdominal haemorrhage

- Trauma (injury to solid viscera e.g. spleen and liver)
- Ruptured abdominal aortic aneurysm
- Haemorrhage from tumours (e.g. primary liver cell carcinoma)

Obstruction to urinary/biliary tract:

These usually present as colics due to stones

- Ureteric colic
- Biliary colic

Gynaecologic (outside those listed above)

- Bleeding Graffian follicle
- Twisted ovarian cyst
- Ectopic pregnancy
- Salpingitis
- Degenerating fibroids

Non-specific abdominal pain:

- Includes a variety of conditions that do not come under the above causes

Medical:

These should always be borne in mind so as to avoid unnecessary surgery

Metabolic disorders:

- Diabetes mellitus
- Porphyria

Haematologic conditions:

- Sickle cell disease
- Leukaemia

Infections and infestations:

- Lower lobe pneumonia
- Gastroenteritis
- Malaria
- Parasitic infestations

Clinical features

Acute abdominal pain

Note the following:

- Location
- Onset and progression
- Nature and character
- Aggravating and relieving factors
- Abdominal distension
- A past history 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

Investigations

Plain radiography

Abdomen:
- Supine and upright films to identify features of intestinal obstruction (dilated bowel loops and multiple fluid levels)
- A radio-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)

Differential diagnoses

Follow a detailed evaluation (as above) and make a reasonable (probable) list of not more than 3 - 5 differential diagnoses

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

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

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

ANTIMICROBIAL PROPHYLAXIS IN SURGERY

Introduction

Postoperative surgical site infection (wound infection) is a rather common, but undesirable occurence in this environment

Surgical site infection tends to increase postoperative morbidity and may lead to mortality

Efforts therefore need to be made to prevent surgical site infection

Antibiotic prophylaxis is not a substitute for adherence to basic principles of surgical asepsis and meticulous attention to technical details

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

Where endogenous contamination is expected (breaching of hollow organs):

Oesophageal surgery

Hepatobiliary surgery

Colorectal surgery

Urinary tract surgery and procedures

Vaginal and uterine surgery

Patients with valvular heart disease

Use of prostheses and implants

Orthopaedic implants

Neurosurgical implants

Patients with cardiac prostheses

Other prostheses

Immunocompromised patients:

HIV/AIDS

Diabetes mellitus

Cancer; patients on cytotoxic chemotherapy

Patients on steroids

Severely malnourished patients

Others:

Patients with peripheral vascular disease undergoing surgery on that limb

Complications

Antibiotic misuse

Antibiotic resistance

Complications of antibiotics (e.g. pseudomembranous colitis)

False sense of surgical security

Antibiotic prophylaxis should be effective and efficient

INTESTINAL OBSTRUCTION

Introduction

A condition in which there is failure of onward propulsion of intestinal contents

A common surgical emergency

Aetiology

Mechanical (dynamic):

Extra-luminal (compression from outside the intestinal wall)

- Strangulated external hernias (e.g. inguinal hernia), internal hernias
- Volvulus
- Peritoneal adhesions and bands
- Intra-abdominal masses (e.g. lymph nodes, tumours)

Intramural (due to causes within the wall of the intestine):
- Intussusception
- Intestinal atresia and stenosis
- Strictures
- Hirschsprung's disease
- Intestinal tumours

Intraluminal (due to causes within the lumen of the intestine):
- Impacted faeces
- Impacted worms (e.g. ascaris lumbricoides)
- Foreign bodies
- Pedunculated polyps

Non-mechanical (adynamic, paralytic ileus):
Electrolyte derangements
- Hypokalaemia

Septicaemia (especially in neonates and infants)
Diabetes mellitus
Other metabolic conditions e.g. uraemia

Pathophysiology

Simple obstruction

Only the intestinal lumen is affected; there is no evidence of strangulation

Strangulated obstruction

Vascular compromise has occurred and may progress to gangrene and/or perforation

Closed loop obstruction

A segment of intestine is blocked at 2 ends (e.g. colonic obstruction with competent ileocaecal valve, intestinal volvulus)
- Dangerous because the risk of perforation is high

Irrespective of the cause or type of obstruction, the symptoms, signs and physiologic consequences are the result of the following
- Stasis proximal to the level of obstruction (gases, fluid)
- Dilatation above level of obstruction
- Increased secretion from the involved segment(s)
- Compression of the veins and later arteries leading to ischaemia, gangrene, necrosis and perforation

The end results are:
- Dehydration
- Electrolyte derangements
- Anaemia
- Peritonitis
- Septicaemia

Clinical features

Symptoms:

Colicky abdominal pain: not a prominent symptom in adynamic obstruction

Abdominal distension

Vomiting: usually bilious and occurs early in small intestinal obstruction
- A late symptom in large intestinal obstruction
- May be faeculent in advanced obstruction

Constipation: occurs early in large intestinal obstruction and late in small intestinal obstruction

Obstipation (non-passage of faeces or flatus) signifies complete obstruction

Stools may be blood-stained (intussusception, volvulus, strangulation)

Diarrhoea: may be present in the face of obstruction (spurious diarrhoea)

Fever: signifies strangulation or perforation

Signs:

General:

Dehydration
Pyrexia
Pallor

Cardiorespiratory: assess the following
- Lung fields
- Pulse rate
- Blood pressure

Abdomen:
- Distension: usually marked in large intestinal obstruction
- Visible peristalsis
- Tenderness
- Tympanitic percussion notes
- Bowel sounds: increased, diminished or absent

Rectal examination
- Perianal soilage
- Empty or full rectum
- Any palpable mass
- Examine finger for faeces, blood, mucus

Complications

Fluid and electrolyte derangements (especially hypokalaemia)
Intestinal gangrene
Intestinal perforation
Peritonitis
Septicaemia and septic shock

Investigations

Plain radiographs
- Abdomen

Supine:

Dilated bowel loops

Should identify affected bowel (jejunum, ileum, large intestine)

Upright (erect):

Multiple fluid levels
- Chest

To identify gas under diaphragm (suggests perforation) and chest infection

Biochemical tests;
- Electrolytes and urea
- Blood Glucose

Haematological:
- Haemogram
- Complete blood count (leucocytosis and neutrophilia suggest strangulation)
- Group and cross match blood and store appropriately

Ultrasonography
- Useful in intussusception, suspected intra-abdominal tumours

Laparoscopy:
- May be helpful in some instances to identify the cause of obstruction

In difficult cases, other investigations may be necessary depending on the presentation and clinical suspicion
- Avoid contrast studies (as much as possible) in acute intestinal obstruction

General measures

Resuscitate:
- Rehydrate and correct electrolyte deficits (especially potassium)
- Nasogastric decompression using a wide bore nasogastric tube

Urethral catheterization to monitor urine output

Broad-spectrum intravenous antibiotics (anaerobes, gram negatives, gram positives)

Correct anaemia by blood transfusion

Definitive treatment

Should only be embarked upon after adequate resuscitation

Mechanical obstruction

Most of the causes will require laparotomy

Treat identified cause on its merits:

Gangrenous or perforated bowel: resect

Small intestine:

Re-anastomose if patient is fit
Bring ends out as stomas if patient is too ill

Large intestine:

Re-anastomose if on right side
Bring ends out as stomas if on left side
Evacuate any peritoneal collection
Suspicious lesions: take specimens for histopathology

Non-mechanical (adynamic) obstruction

Treat accordingly
Surgery is not required

PREOPERATIVE EVALUATION and POSTOPERATIVE CARE

Preoperative Evaluation

Introduction

The assessment of a patient before surgery to ensure that the patient is in optimal physiologic state and fitness for the surgical procedure

A most important aspect of the care of a surgical patient

No elective operation should be carried out without an adequate preoperative assessment

In the emergency situation, all efforts must be made to ensure that the patient can withstand anaesthesia and the surgical procedure

Occasionally (e.g. with severe on-going haemorrhage, airway obstruction) resuscitation, anaesthesia and surgery may commence simultaneously

Objectives of preoperative evaluation

To detect any fluid and electrolyte derangements

To detect any haematological derangements (e.g. anaemia, bleeding diathesis, sickle cell disease)

To detect any coexisting medical conditions that may adversely affect the outcome of anaesthesia and surgery
- All patients scheduled to have surgery should be in a haemodynamically stable condition before surgery

The above may not always be possible, but efforts must be made to improve cardiopulmonary and renal function

Correct any detected abnormality

Patient evaluation and correction of abnormalities may need to be done in conjunction with others: the anaesthetist, physician, paediatrician etc

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 conjuction 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

Postoperative Care

Introduction

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
Minor/moderate surgery
Patient taking orally:
Paracetamol
Non steroidal antiinflammatory drugs
Patient not taking orally:
Injectable nonsteroidal antiinflammatory drugs (e.g. diclofenac sodium)
Major surgery:
Parenteral analgesics
- Narcotic analgesics (e.g. morphine)
- NSAIDs (e.g. diclofenac sodium)

Nasogastric decompression

The stomach may need to be kept decompressed for 24 - 48 hours, particularly following gastrointestinal surgery
Decompression prevents abdominal distension and tension on abdominal fascial closure
It also 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

USE OF BLOOD TRANSFUSION IN SURGERY

Introduction

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

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

CHAPTER 17: 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 through out the world

30 - 40 million cases and 745,000 deaths for the year 2001

- 50 - 60% of estimated deaths due to vaccine-preventable diseases

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 - 5 days
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
Encephalitis
Seizures
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

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 has 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, and increase in cases in Nigeria

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- Barré 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 flaccid paralysis, it must be tested further, using fingerprinting or 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/mm^3 (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 **or** 2.5 - 5 mg subcutaneously for bladder paralysis
Analgesics
- Avoid opiates if there is impairment of ventilation
Treat urinary tract infecton with appropriate antibiotics

Prevention
Hygienic practices
- To prevent / limit contamination of food and water by the virus
Vaccination
- The only effective method of prevention

<u>Oral Polio Vaccine</u>
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 immunocompromised 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 0.7 mg/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 - 8 years
- 900 microgram (3,000 IU) orally once daily

9 - 13 years
- 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

CHAPTER 18: 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
Insipient left ventricular failure secondary to hypertension
Arhythmias
Myocardial infarction

Clinical features
Dyspnoea
Orthoponea
Paroxysmal nocturnal dyspnoea
Cough
Heamoptysis
Restlessness
Wheezes
Hypoxia

Differential diagnoses
Pulmonary thromboembolism
Bronchial asthma
Pulmonary tuberculosis
Cardiac tamponade

Complications
Right-sided heart failure
Acute renal failure
Myocardial infarction

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

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 caucasions
Peaks between ages 0 - 6 months and 45 - 75 years

Aetiology
Congenital and acquired structural defects of the heart
Abnormal electrical activities of the heart
Inflammatory, infiltrative, neoplastic and degenerative processes
Fluids and electrolyte imbalances
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
Death
Sequelae involving the vital organs
- Acute renal failure
- Myocardial infarction
- Cerebrovascular accident

Investigations (after the initial rapid assessment and resuscitation)
Electrocardiography
Echocardiography
Urea, Electrolytes and Creatinine
Lipid profile
Blood gases
Chest radiograph

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

Hemolysis

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 $NaHCO_3$ if pH is 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 Aarrest

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) **X** body 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
- Glucose 5% injection is also suitable for water replacement, being a hypotonic fluid

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
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
β adrenergic agonists
α adrenergic antagonists
Decreased intake:
Starvation

Clinical features

- Vary between patients and depend on the level of potassium loss

Serum K <3mmoles/Litre:
Fatigue
Myalgia
Weakness of the lower extremities
More severe hypokalaemia results in
- Progressive weakness
- Hypoventilation
- Complete paralysis

ECG changes are due to ventricular depolarisation and do not correlate with the plasma potassium levels
- Flattening/inversion of the T wave
- A prominent U wave
- 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 pressure by not more than 25% within the first 2 hours
Further reduction of BP to (not less than) 160/100 mmHg 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

Sodium niprusside
- 0.3 micrograms/kg/min intravenously initially, 0.5 - 6 micrograms/kg/min maintenance (maximum of 6 micrograms/kg/min)

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
Use sodium nitroprusside with caution in ischaemic heart disease, renal impairment, raised intracranial pressure and impaired pulmonary function

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 sulphonamide 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-ß 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 (<2.5 mmole/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 - 32°C
Seizures
Areflexia
CO_2 retention and respiratory depression due to decreased cerebral blood flow

Differential diagnoses

Coma 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

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

Thyroid function tests

Other tests to identify precipitating factors

Management

Requires intensive monitoring
Supportive care
Identification and treatment of precipitating cause(s)

Treatment objectives

Reduction in T_3 synthesis/action and restoration to 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
Antibiotics (if infection is present)

Supportive measures

Adequate hydration with intravenous fluids and cooling

POISONING

Introduction

The ingestion by, or exposure of a patient to excessive doses of a medicine or other substances may cause harm
This may be:
Self poisoning (may be suicidal)
Accidental
Homicidal

Clinical presentation

Determined (amongst others) by:
Type of drug
Inherent toxicity
Dose and duration following exposure
Concurrent therapy
Co-existing disease states etc
This guideline provides only a brief overview.
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

Verify, validate or confirm all of the events related to the poisoning
Take good clinical history
- Information from relatives, friends, emergency services personnel may be very useful especially where the patient is unwilling or unable to provide useful information
Emergency stabilization
Quick clinical evaluation
Elimination of the poison or decontamination
Enhancing systemic clearance
Administration of antidotes
Supportive measures
Observation
Disposition

Emergency stabilization

Life-saving measures take priority over all other decontamination techniques
The following ABC approach is recommended:

A Establish a clear **A**irway
B Ensure adequate **B**reathing and ventilation
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

Clinical evaluation

A quick clinical evaluation should be carried to:
Obtain a good history of the drug ingestion/exposure
- Amount, time, etc
- Circumstances surrounding the event (from the patient, relations and other eyewitnesses)
The patient may have no symptoms when seen early in the course of the poisoning
A thorough physical examination may further provide clues on the drug class causing toxicity e.g pinpoint pupils with opioid overdose
- The absence of a significant sign does not negate the diagnosis
Clinical laboratory patient data e.g. urine drug screens
- Useful in patients with coma of unknown aetiology

Elimination of poisons (or Decontamination)

The removal of the offending substance from the patient
The presumption is that both the dose and duration of exposure are determinants of toxicity, and limiting continued exposure is beneficial
Remove the patient from the toxic environment
Provide fresh air and oxygen (respiratory decontamination)
Flushing the areas (e.g. skin and eyes) with large volumes of fluid to remove the toxic substance
Gastrointestinal decontamination:
Emesis or lavage to evacuate the gastric contents

Administer activated charcoal as an absorbent to bind the toxic substance in the gastrointestinal tract

Use cathartics or whole bowel irrigation to increase the rectal elimination of unabsorbed drugs

A combination of the above methods may be used.

Enhancing systemic clearance

Clearance of the toxic substances may be enhanced by:

Manipulation of urine pH

Haemodialysis

Haemo perfusion

Antidotes

An antidote is a drug that antagonizes the toxicity of another substance in a specific manner

Examples:

- Naloxone for opioids
- N-acetylcysteine for paracetamol

Looked out for, and address the peculiarities related to specific poisonings

- Important where multiple drugs are involved

The pattern of poisoning is influenced by age and gender

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

SPECIFIC POISONS

Paracetamol

Toxicity often occurs following an acute ingestion (within 24 hours) of =10 - 15 g (20 - 30 tablets) or 150 mg/kg

It could also in conditions with enhanced P_{450} enzyme activity (e.g. on-going use of anticonvulsants, rifampicin)

Less often hepatotoxicity occurs following chronic ingestion of therapeutic or slightly greater amounts in conditions with decreased gluthatione reserve

- Acute starvation
- Alcoholism
- Childhood
- Chronic malnutrition

Clinical features

Early manifestations are non-nspecific and also non-predictive of subsequent hepatotoxicity. They include:

Nausea and vomiting

Excessive sweating

Onset of hepatotoxicity is heralded by right upper quandrant tenderness and hepatomegaly

Features of liver damage include:

Encephalopathy

Haemorrhage

Hypoglycaemia

Cerebral oedema

Death

These symptoms are maximal in 3 - 4 days

Poor prognostic indices:

Encephalopathy or hepatic failure

Greater than two fold prolongation of Prothrombin time

Serum bilirubin > 68 micromol/L (4 mg/dL)

Serum creatinine > 3.3

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 (where facility is available)

Laboratory evidence of hepatotoxicity includes:

Prolongation of prothrombin time

Elevation of serum bilirubin and transaminase activity

Renal function may also be impaired

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 6 years: 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 centre, 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)

- To correct dehydration and produce brisk urine flow (saline diuresis)

Supplemental oxygen

Supplemental glucose

Intravenous vitamin K 10 mg daily for coagulopathy

Intravenous $NaHCO_3$ to alkalinize urine (see Cardiac Arrest for administration)

Correction of other electrolyte derangements

Haemodialysis for severe salicylate poisoning

Indications for haemodialysis

Severe clinical toxicity

Aspirin (acetylsalicylic acid) levels = 7 mmol/L (100 mg/dL)

Contraindications, failure of other treatment modalities

Benzodiazepines

Most commonly involves diaazepam and bromazepam

These drugs potentiate the inhibitory effect of GABA on CNS neurons

Clinical features

Mainly CNS depression occuring 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

- See Paracetamol poisoning

Flumazenil, a competitive benzodiazepine receptor antagonist, can reverse CNS and respiratory depression

- Give 0.1 mg intravenously at 1 minute intervals until desired effect is achieved

Notable adverse drug reactions

Flumazenil with tricyclic antidepressants can cause seizures

Activated charcoal colours stools black

Prevention of Drug Poisoning

- Keep all medicine out of reach when not needed
- Label all medicines appropriately
- Kerosene poisoning prevention

Keep kerosene and other hydrocarbons away from children

Use dedicate on tenants kerosene and other hydrocarbon

Co-poison prevention

(1) Keep working generator safely away from explosions

(2) Do not run mobile engine/vehicles within explosions

(3) Enact and enforce laws for safe engine/generator purchasing and use

Carbon monoxide poisoning

Usually due to inhalation of smoke, car or generator exhaust fumes caused by incomplete combustion in a confined space

Carbon monoxide binds to haemoglobin, myoglobin and to mitochondria, inhibiting cellular respiration

Toxic effects of carbon monoxide are related to hypoxia

Clinical features

Dyspnoea

Tachypnoea

Headache

Emotional lability

Confusion

Impaired judgement

Clumsiness

Syncope

Nausea, vomiting and diarrhoea may occur

Cardiovascular manifestations:

Ischaemic chest pain, arrhythmias, heart failure and hypotension
In severe poisoning:
Cerebral oedema
Pulmonary oedema
Respiratory depression
Coma may be seen in severe poisoning
Cherry-red colour of skin and mucus
Rarely cyanosis

Investigations

To identify complications and establish a diagnosis
- 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. Repeat every 8 hours

Kerosene poisoning

Similar to poisoning by other petroleum distillates
Petroleum distillate hydrocarbons are poorly absorbed following ingestion but can be aspirated, causing significant toxicity to the airways
More common in children

Clinical features

CNS excitation in low doses; depression in high doses
Rarely coma and seizures
Other effects: nausea, vomiting, abdominal pain and diarrhoea
Aspiration may occur and cause aspiration pneumonia

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

Glucocorticoids are ineffective

Organophosphate/insecticide poisoning

Introduction

These substances irreversibly inhibit acetylcholinesterase and cause 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 from exposure to toxicity is between 30 minutes - 2 hours
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
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
- Effective for muscarinic symptoms

Plus:
Pralidoxine
- Diluted to 10 - 15 mL with water for injection and administered by slow intravenous injection over 5 - 10 minutes

Adult: 1 - 2 g; can be repeated in 30 minutes
Child: initially 30 mg/kg, then either 30 mg/kg every 4 hours or by intravenous infusion, 8 - 10 mg/kg/hour (usual maximum 12 g in 24 hours)
Treat seizures with intravenous diazepam 10 mg stat

CHAPTER 19: THERAPEUTICS

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 the 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
<u>Essential elements of a prescription order</u>
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

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

In this text the following WHO definitions will apply

Adverse drug reaction

A response to a medicine which is noxious and unintended

- 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

- 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 VI

Analysis of such reports enables appropriate decisions to ensure safe and judicious use of medicines

In the text a number of known adverse reactions are listed for medicines used for the treatment of the stated diseases

- This list is by no means complete or comprehensive
- There may be unknown adverse reactions peculiar to our population

CHAPTER 20: 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

APPENDIX 1

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 trypanosomiasis

APPENDIX II:

WHO NEW ANTENATAL CARE MODEL CLASSIFYING FORM 2001

Criteria for classifying women for the basic component of the new antenatal care model

Name of patient: _____ **Clinic record number:** | | | | | | |

Address: _____ **Telephone:** _____

INSTRUCTIONS: Answer all 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? (Myomectomy, removal of septum, cone biopsy, classical CS, cervical cerclage)	☐	■
	CURRENT PREGNANCY	**No**	**Yes**
7.	Diagnosed or suspected multiple pregnancy?	☐	■
8.	Age less than 16 years?	☐	■
9.	Age more than 40 years?	☐	■
10.	Isoimmunization Rh (-) in current or in 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 questions (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? (circle) **NO** **YES**

If NO, she is referred to _____

Date _____ Name _____ Signature _____
(staff responsible 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 fo 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 th 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	/m2
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	Teratogenic in animal studies
Isotretinoiin	Teratogenic
Lithium salts	Avoid if possible (risk of teratogenicity)
Phenytoin	Congenital malformation (screening advised)
Trenitoin	Teratogenic
Trimethoprim	Teratogenic risk (folate antagonist)
Vinblastine	Avoid (limited experience suggest fetal harm; teratogenic in animal studies)
Vincristine	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
Acetazolamide	Amount too small to be harmful
Alcohol	Large amounts may affect infant and reduce milk consumption
Amantadine	Avoid; present in milk; toxicity in infants reported
Amiloride	Manufacturer advises avoid
Aminophylline	Present in milk- irritability in infants reported
Amitryptilline	Manufacturers advise avoid
Amlodipine	Manufacturers advise avoid
Amoxicillin	Trace amounts in milk
Amphetamines	Significant amount in milk
Amprenavir	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 produce hypoprothrombinaemia in infants if neonatal vitamin K stores low
Androgens	Avoid. May cause masculinization in the female infant or precocious development in the male infant; high doses suppress lactation
	Anticoagulants, oral Risk of haemorrhage; increased by Vitamin K deficiency; warfarin appears safe but phenindione should be avoided
Antihistamines	Significant amounts of some antihistamines present in milk, although not known to be harmful

Medicines that could cause harm when administered to breastfeeding mothers contd.

Medicine	Comment
Antipsychotics	Avoid unless absolutely necessary
Apomorphine	Manufacturer advises avoid
Atenolol	Toxicity due to bet-blockage. Avoid or use with caution (monitor infant)
Atropine	Manufacturer advises caution
Azithromycin	Present in milk; manufacturer advises use only if no suitable alternative
Barbiturates	Avoid if possible. Large doses may produce drowsiness
Benzodiazepines	Avoid if possible
Beta-blockers	Monitor infant; possible toxicity due to beta-blokade
Caffeine	Regular intake of large amounts can affect infant
Captopril	Manufacturers advise avoid
Carbimazole	Use lowest effective dose
Ceftriaxone	Present in milk in low concentrations
Cefuroxime	Present in milk in low concentrations
Chloramphenicol	Use another antibiotic; may cause bone marrow toxicity in infant
Chlorpromazine	Drowsiness in infant reported
Ciprofloxacin	Manufacturer advises avoid
Contraceptives, oral	Avoid until weaning or for 6 months after birth (adverse effects on lactation)
Corticosteroids	Avoid maternal dose of prednisolone beyond 40 mg daily
Co-trimoxazole	Risk of kernicterus in jaundiced infants and of haemolysis in G6PD-deficient infants
Cyclophosphamide	Discontinue breastfeeding during and for 36 hours after stopping treatment
Dapsone	Haemolytic anaemia; although significant amount in milk, risk to infant very small unless infant is G6PD deficient
Desferroxamine	Use only if potential benefit outweighs risk
Enoxaparin	Irritability and disturbed sleep reported
Ergotamine	Avoid
Furosemide	May inhibit lactation
Ibuprofen	Manufacturer advises avoid
Indometacin	Manufacturers advise avoid
Iodine and iodides	Stop breastfeeding; danger of neonatal hypothyroidism and goitre
Lisinopril	Manufacturers advise avoid
Morphine	Withdrawal symptoms in infants of dependent mothers; breastfeeding not best method of treating dependence in offspring
Nicotinic acid	Avoid
Nifedipine	Manufacturers advise avoid
Oestrogens	Avoid
Phenobarbital	Avoid when possible
Phenytoin	Manufacturer advises avoid
Progesterone	Manufacturers advise avoid
Statins	Manufacturers advise avoid
Tetracycline	Avoid
Theophylline	Irritability in infants reported
Thiamine	Severely thiamine-deficient mothers should avoid breastfeeding as toxic methyl-glyoxal present in milk
Tinidazole	Avoid breastfeeding during and for 3 days after stopping treatment
Vitamin A	Theoretical risk of toxicity in infants of mothers taking large doses
Vitamin D	Caution with systemic doses; may cause hypercalcaemia in infant. Manufacturers advise avoid

NATIONAL PHARMACOVIGILANCE CENTRE (NPC) NIGERIA

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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 ☐ WEIGHT (kg): _____

HOSPITAL/Treatment Centre: _____

2. * ADVERSE DRUG REACTION (ADR)

A. DESCRIPTION

DATE Reaction Started	DATE Reaction Stopped

C. OUTCOME OF REACTION

TICK AS APPROPRIATE

☐ Recovered fully ☐ Recovered with disability (Specify) _____

☐ Congenital Abnormality (Specify) _____ ☐ Life Threatening (Specify) _____

☐ Death ☐ Others (specify) _____

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 (State name and other details if available / Attach product label / Sample (if available)

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**