



standard treatment guidelines

NIGERIA | 2008

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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

Chapter 1: Alimentary Tract

Gastrointestinal Disorders	1
Amoebiasis	1
Bacillary Dysentery	1
Cholera	2
Constipation	2
Diarrhoea (acute)	3
Gastritis	4
Giardiasis	4
Haemorrhoids	5
Pancreatitis	5
Peptic Ulcer Disease	6
Upper Gastrointestinal Bleedin	6
Hepatic And Biliary Disorders	7
Hepatitis	7
Hepatic Encephalopathy	8
Jaundice	9
Liver Cirrhosis	9
Nutritional Disorders	10
Kwashiokor And Marasmus	10
Micronutrient Deficiencies	10
Obesity.....	11

Chapter 2: Blood And Blood-forming Organs

Anaemias	13
Blood Transfusion	14
Haemostasis And Bleeding Disorders	16
Leukaemias	16
Lymphomas	19
Sickle Cell Disease.....	20

Chapter 3: Cardiovascular System

Angina Pectoris.....	23
Cardiac Arrhythmias	23
Congenital Heart Disease	24
Deep Venous Thrombosis	24
Heart Failure	25
Hyperlipidaemia	26
Hypertension	26
Infective Endocarditis	27
Myocardial Infarction.....	29
Myocarditis	30
Paediatric Cardiac Disorders.....	30
Pericarditis	30
Pulmonary Embolism	31
Pulmonary Oedema	32
Rheumatic Fever	32
Rheumatic Heart Disease	33

Chapter 4: Central Nervous System

Non-psychiatric Disorders	34
Dizziness	34
Headaches.....	35
Meningitis	36
Migraine	37
Parkinsonism.....	38
Seizures/epilepsies	39
Stroke	41
Syncope	42
The Unconscious Patient	42
Psychiatric Disorders	43
Alcoholism (alcohol Dependence)	43
Anxiety Disorder	44
Bipolar Disorders	44
Delirium.....	45
Depression	46
Insomnia.....	47
Panic Disorder.....	47
Schizophrenia.....	48

Chapter 5: Dental And Oral Disorders

Acute Necrotizing Ulcerative Gingivitis.....	49
Acute Periapical Abscess	49
Alveolar Osteitis	50
Cellulitis.....	50
Dental Caries	50
Gingivitis	51
Neoplasms Of The Oral Cavity	51
Oral Thrush (candidiasis)	51
Pericoronitis	52
Periodontitis.....	52
Pulpitis	53
Salivary Gland Diseases	54
Temporo-mandibular Joint Disorders.....	54

Chapter 6: Dermatology

Bacterial Infections	56
Cellulitis	56
Furunculosis (boils)	56
Impetigo Contagiosa	57
Dermatitis And Eczema	58
Atopic Dermatitis (atopic Eczema).....	58
Contact Dermatitis	59
Exfoliative Dermatitis (erythroderma).....	59
Parasitic Dermatoses	60
Cutaneous Larva Migrans (creeping Eruption).....	61

Guinea Worm Disease (dracunculiasis).....	61	The Red Eye	104
Myiasis	62	Trachoma.....	105
Onchocerciasis (river Blindness)	62	Xerophthalmia	105
Pediculosis (lice)	63		
Scabies	64		
Papulosquamous Disorders	65	Chapter 10: Genito-urinary System	
Lichen Planus	65	Nephrology	106
Pityriasis Rosea	66	Acute Renal Failure.....	106
Psoriasis	67	Chronic Kidney Disease	106
Superficial Fungal Infections	69	Nephrotic Syndrome	107
Dermatophyte Infections (tinea)	69	Sexually Transmitted Infections	108
Pityriasis Versicolor (tinea Versicolor)	70	Bacterial Vaginosis	108
Viral Infections	71	Chancroid (ulcus Molle, Soft Chancre)	109
Herpes Zoster	71	Chlamydial Infection	110
Molluscum Contagiosum	72	Gonorrhoea	111
Varicella (chickenpox)	72	Granuloma Inguinale (donovanosis; Granuloma Venereum).....	113
Viral Warts (verrucae)	73	Lymphogranuloma Venereum	114
Miscellaneous Disorders	74	Syphilis	115
Acne Vulgaris (pimples)	74	Trichomoniasis	116
Pruritus	76	Vulvo-vaginal Candidiasis	117
Urticaria And Angioedema	78	Urology	118
Vitiligo	80	Benign Prostatic Hyperplasia	118
		Carcinoma Of The Prostate	118
Chapter .7: Ear, Nose And Throat		Erectile Dysfunction (impotence)	119
Acute Otitis Media.....	81	Male Infertility	120
Adenoid Disease	82	Posterior Urethral Valves	120
Chronic Otitis Media	82	Priapism	121
Epistaxis	83	Prostatitis	121
Foreign Bodies In The Airways	83	Scrotal Masses	122
Foreign Bodies In The Ear	84	Torsion Of The Testis	122
Foreign Bodies In The Nose And Rhinoliths.....	84	Urethral Stricture	123
Mastoiditis.....	84	Urinary Schistosomiasis	123
Nasal Allergy	85	Urinary Tract Calculi	124
Otitis Externa	86		
Peritonsillar Abscess (quinsy)	86	Chapter 11: Infectious Diseases / infestations	
Pharyngitis (sore Throat)	86	Fevers: Management Approach	125
Sinusitis	87	Food Poisoning	125
Tonsillitis	88	Helminthiasis	127
Tracheostomy	89	Human Immunodeficiency Virus Infection	129
Wax In The Ear	89	Malaria	135
		Rabies.....	137
Chapter 8: Endocrine System		Tetanus	138
Diabetes Mellitus	90	Trypanosomiasis (sleeping Sickness)	140
Hyperthyroidism (thyrotoxicosis)	97	Tuberculosis	140
Hypothyroidism (myxoedema)	99	Typhoid Fever	142
Chapter 9: Eye Disorders		Chapter 12: Musculoskeletal System	
Acute Anterior Uveitis (iritis)	100	Back Pain	143
Acute Keratitis	100	Gout	144
Allergic Conjunctivitis	101	Osteoarthritis	145
Eye Injuries	101	Rheumatoid Arthritis	146
Foreign Bodies In The Eye	102	Septic Arthritis	147
Infective Conjunctivitis	103	Systemic Lupus Erythematosus	148
Ophthalmia Neonatorum	103		
Scleritis / Episcleritis	104		
Stye (hordeolum)	104		

Chapter 13: Obstetrics And Gynaecology	
Abortion	149
Antenatal Care	150
Anaemia In Pregnancy	152
Cancer Of The Cervix	153
Cardiac Disease In Pregnancy	154
Eclampsia	156
Ectopic Pregnancy	158
Hyperemesis Gravidarum	159
Immunization Schedules	160
Jaundice In Pregnancy	160
Pelvic Inflammatory Disease	162
Rape	163
Chapter 14: Respiratory System	
Acute Epiglottitis	165
Acute Laryngo-tracheo-bronchitis (croup)	165
Acute Rhinitis (common Cold)	166
Bronchial Asthma	166
Bronchiectasis	167
Chest Pain	168
Chronic Obstructive Airways Disease(coad)	168
Cough	169
Dyspnoea	170
Lung Abscess	170
Pneumonia	171
Pulmonary Embolism	172
Section B	
Chapter 15: Injuries And Acute Trauma	
Bites And Stings	173
Burns	175
Disaster Plan	176
Head Injury	177
Multiple Injuries	180
Chapter 16: Surgical Care And Associated Disorders	
Acute Abdomen	182
Antimicrobial Prophylaxis In Surgery	184
Intestinal Obstruction	184
Preoperative Evaluation and Postoperative Care	186
Use Of Blood Transfusion In Surgery	189
Chapter 17: Paediatric Perspectives	
Measles (rubeola)	190
Poliomyelitis	191
Vitamin A Deficiency	193

Section C**Chapter 18: Emergencies**

Acute Left Ventricular Failure	195
Cardiac Arrest	196
Drowning And Near-drowning	197
Electrolyte Abnormalities	198
Hypertensive Emergencies	200
Hypoglycemia	200
Myxoedema Coma	201
Thyroid Storm (thyrotoxic Crisis)	201
Poisoning	202

Chapter 19: Therapeutics

Prescription Writing	206
Adverse Drug Reactions	208

Chapter 20: Notifiable Diseases209**APPENDICES****Appendix I**

WHO clinical staging of HIV for infants and children with established HIV infection.....	211
---	-----

Appendix II:

WHO new antenatal care model classifying form 2001.....	212
--	-----

Appendix III

Calculation of dosage requirements in children.....	214
--	-----

Appendix IV:

Medicines with teratogenic potential.....	215
---	-----

Appendix V:

Medicines that could cause harm when administered to breastfeeding mothers.....	215
--	-----

Appendix VI:

NAFDAC Adverse Drug Reaction Reporting form	217
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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 furate

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 DYSENTERY**Introduction**

An important cause of colonic diarrhoea in developing countries.

Caused by pathogenic species of *Shigella* A-D (*dysenteriae*, *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 enterocolitis (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 rehydration 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-trimethoprim (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, triiodothyronine, 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 aetiological 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 melaena 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:

H₂ 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 B₁₂ deficiency

Complications

Diseases related to Vitamin B₁₂ 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 B₁₂ 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, photocoagulation, 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 intake/output

Nasogastric tube suctioning

Decrease pancreatic inflammation
Prevent, identify and treat complications

Caution

Avoid narcotic analgesics which may cause spasm of the sphincter of Oddi and worsen pancreatitis

Prevention

Control alcohol ingestion

PEPTIC ULCER DISEASE**Introduction**

Caused by peptic ulceration that involves the stomach, duodenum and lower oesophagus.

An increasingly common problem in developing countries.

Most ulcers are duodenal

Aetiology/Predisposing factors

H. pylori gut infection
Use of NSAIDs
Smoking

Clinical features

Recurrent epigastric pain
- Often radiating to the back
- Worse at night
- Improved by antacids
- May be made worse by some food types (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 CO₂ breath test)

Differential diagnoses

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

Treatment objectives

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

Drug treatment

Symptomatic treatment with antacids may be used prior to confirming the diagnosis of peptic ulcer disease

H. pylori eradication

Triple therapy with:

- 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**Mild upper gastrointestinal bleeding**

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

Severe upper gastrointestinal bleeding

Interventional endoscopic treatment
Blood transfusion
Surgery
Perforation
Surgery
Gastric outlet obstruction
Rest the gut
Surgery

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

- Hypovolaemic shock
- Congestive heart failure from chronic severe anaemia

Investigations

Upper gastrointestinal endoscopy: picks up lesions in 90% of cases

Upper gastrointestinal barium radiography: 80% detection rate

- Selective mesenteric arteriography
- Radio isotope scanning
- Stool- occult blood test
- Full Blood Count

Treatment objectives

- Restore and maintain haemodynamic status
- Control bleeding
- Prevent recurrence of bleeding

Non-drug treatment

Carefully monitor vital signs (pulse, blood pressure, respiration and temperature) as frequently as necessitated by the patient's condition

Insert a nasogastric tube to aspirate gastric contents and/or to introduce agents to constrict the blood vessels

Blood transfusion: whole blood (acute bleeding) or packed cells (chronic) bleeding. Up to 5 - 6 pints of blood may be needed in severe cases

- Plasma expanders in the absence of blood
- Continuous Central Venous Pressure (CVP) monitoring

Drug treatment

Bleeding peptic ulcers/erosions

- Proton Pump Inhibitors
- Omeprazole 20 mg orally once daily for 4 weeks

Or:

- Omeprazole 40 mg by slow intravenous injection over 5 minutes once daily until patient can take orally

Anti *Helicobacter pylori* therapy set above.

Endoscopic treatment for actively bleeding ulcer or visible non-bleeding vessel

- Injection therapy with 98% alcohol (total volume less than 1mL)

Or:

- Injection therapy with epinephrine (1:10,000) up to 1mL

Or:

- Thermal coagulation with heat probe

Or:

- Laser therapy

Bleeding varices

Intravenous 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

Peptic ulcers/erosions/tumours

Surgical repair or resection as appropriate

Supportive

Monitor vital signs and urine output to detect early features of hypovolaemic shock

Look out for features of hepatic encephalopathy

Notable adverse drug reactions

Vasopressin can cause abdominal cramps. It lowers blood pressure drastically and could worsen ischaemic heart disease

Prevention

Peptic ulcers/erosions related upper gastrointestinal bleeding

Avoid NSAIDs.

Treat *H. pylori* infection

Oesophageal varices

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 aetiological 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-occurrence 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

Hepatitis A

Self-limiting disease. No specific drug treatment

Hepatitis B

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 1 year

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

Hepatitis D

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

Plus:

Lamivudine: 100 mg orally once daily for 4 months

Hepatitis E

Largely supportive

Notable adverse drug reactions

Interferon alpha 2b and Ribavirin haematopoietic toxicity

Flu-like illness

Leucopenia

Psychiatric-like symptoms

Development of early resistance if therapy exceeds 1 year

Prevention

Prevention of faecal contamination of food and water

Screen blood and blood products for hepatotropic 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, diarrhoea
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 mucous 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 phosphatase)
Abdominal ultrasound scan: look out for canalicular dilations, 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**Obstructive jaundice**

ERCP sphincterotomy with stone removal
Stent insertion
Pancreatic head/duodenal head realignment

Supportive measures

Reassurance and monitoring
Phototherapy in neonatal jaundice

LIVER CIRRHOSIS**Introduction**

An advanced stage of chronic liver disease associated with permanent distortion of the liver architecture and replacement of some destroyed hepatocytes with fibrous tissue

Accompanied by some loss of liver function leading to certain recognized symptoms and signs

Aetiology

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

Clinical features

Varies with the extent of liver damage:

Fatigue
Ascites
Pedal oedema
Haematemesis

Liver may be shrunken or enlarged below the costal margin; it is typically firm

Differential diagnoses

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

Complications

Intractable oedema
Upper gastrointestinal tract bleeding
Coagulopathy
Hepatic encephalopathy

Hepato-renal syndrome

Investigations

LFTs
PT, PTTK, Serum albumin
Liver biopsy
Ultrasound examination of the liver
Screening for aetiological factors in chronic liver disease e.g. viral markers for hepatotropic viruses (e.g. Hepatitis B & C)

Treatment objectives

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

Non-drug treatment

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

Drug treatment**Ascites and pedal oedema**

Spirolactone tablets 25 - 100 mg orally 12 hourly
Furosemide 20 - 80 mg orally 12 hourly
Salt-poor albumin for intractable ascites

Prevention of variceal bleeding

Propranolol 40 - 80 mg orally daily

Replacement of damaged liver

Liver transplant

Prevention of encephalopathy

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**KWASHIORKOR 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
- B₁ (thiamine): beri-beri
- B₂ (riboflavin): scrotal and vulval dermatoses, angular stomatitis, scars, magenta tongue, cheilosis
- B₃ (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 m², expressed as kg/m²

Classification of BMI

Underweight: <18.5 kg/m²
Normal weight: 18.5 - 24.9 kg/m²
Overweight: 25 - 29.9 kg/m²
Obesity (Class 1): 30 - 34.9 kg/m²
Obesity (Class 2): 35 - 39.9 kg/m²
Extreme obesity (Class 3): > 40 kg/m²
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
 - Hypocortisolism
 - 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 B₁₂ 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 (intracorpuseular 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-corpuseular or intrinsic abnormality):
 - Autoimmune
 - Rhesus-incompatibility, mismatched transfusion
 - Hypersplenism
 - Infections e.g malaria, *Clostridium welchii*
 - Drugs and toxins

Others e.g. burns
 Decreased red cell production:
 Nutritional (due to deficiencies of substances essential for erythropoiesis)
 - Iron
 - Folate
 - Vitamin B₁₂
 - Various deficiencies e.g. protein, ascorbic acid
 Bone marrow stem cell failure:
 Primary (idiopathic):
 - Aplastic anaemia
 - Pure red cell aplasia
 Secondary:
 - Drugs (phenylbutazone, cytotoxic agents, etc)
 - Chemicals
 - Irradiation
 Anaemias associated with systemic disorders:
 Infection
 Liver disease
 Renal disease
 Connective tissue disease
 Cancer (including leukaemia)
 Marrow infiltration
 Thyroid or pituitary disease
Clinical features
 Depend on the degree of anaemia, severity of the causative disorder and age of the patient
 The clinical effects of anaemia are due to anaemia itself and the disorder(s) causing it
 Common:
 Tiredness
 Lassitude
 Weakness
 Dyspnoea on exertion
 Palpitations
 Pallor
 Less common:
 Angina of effort
 Faintness
 Giddiness
 Headache
 Ringing in the ears
 High output state
 Congestive cardiac failure
Differential diagnoses
 Cardiac failure
 Respiratory failure
Complications
 Cardiac failure
 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 B₁₂, folic acid
 The specific haematinic indicated should be given alone
 Response to adequate treatment is important in confirming diagnosis
Iron deficiency
 Oral iron therapy:
 - Ferrous sulfate 200 mg (containing 65 mg of iron) 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
Megaloblastic anaemia
 Response to therapy is satisfactory if administered dose is limited to the minimal daily requirement
 Treatment with vitamin B₁₂ (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 B₁₂ 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 B₁₂ -deficient patients but worsen the neurological complications
 Large doses of vitamin B₁₂ also give haematological response in folate-deficient patients
Prevention
 Balanced diet
 Prompt treatment of all illnesses
BLOOD TRANSFUSION
Introduction
 Blood transfusion is the administration of blood for therapy.
 It is potentially hazardous: blood should be given only if the dangers of not transfusing outweigh those of

transfusion.
 Indication(s) must be clearly established.
 Transfusion of whole blood or red cell concentrates is important in the treatment of acute blood loss and of anaemia.
 Red cells can be stored at 4°C for 5 weeks in media that are specially designed to maintain the physical and biochemical integrity of the erythrocytes and which maintain their viability after transfusion.
 Citrate Phosphate Dextrose with Adenine (CPDA) is commonly used for collections of whole blood.
 The use of whole blood as a therapeutic agent has been almost completely replaced by the use of blood fractions.
Types of blood transfusion
 Autologous blood transfusion:
 Transfusion of the patient's own blood to him/her
 - Safest blood for patients
 The three main types are:
 - Pre-deposit autologous transfusion
 - Immediate pre-operative phlebotomy with haemodilution
 - Intra-operative blood salvage
 Exchange transfusion:
 To remove deleterious material from the blood, for example, in severe jaundice resulting from haemolytic disease of the newborn
 Alternatives to red cell transfusion:
 Perfluorochemicals such as Fluosol-DA
 Polymerised haemoglobin solutions with good intravascular recovery
Indications for blood transfusion
 Symptomatic anaemias:
 - Recurrent haemorrhage
 - Haemolysis
 - Bone stem cell failure
 - Pure red cell aplasia
 - Severe anaemia of chronic disorders
 - Haematological malignancies (e.g. leukaemia, lymphoma)
 - Chemotherapy complicated by anaemia
 In neonates:
 - Severe acute haemorrhage
 - Haemolytic disease of the newborn
 - Septicaemia
 - Prematurity
 Bleeding disorders:
 - Congenital e.g. haemophilia
 - Acquired e.g. disseminated intravascular coagulopathy
 Prevention or treatment of shock:
 - Clinical situations in which there is need to restore and/or maintain circulatory volume e.g. trauma, haemorrhage
 To maintain the circulation (as in extracorporeal or cardiac by-pass shunts)
Whole blood preparations

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

Fresh Blood

Justified by the recognition that there is a relatively rapid loss of platelets, leucocytes and some coagulation factors with liquid storage. There is also progressive increase in the levels of undesirable products such as potassium, ammonia, and hydrogen ions

Erythrocyte preparations

Four types are in common use:

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

Washed red blood cells

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

Leucocyte-reduced red blood cells

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

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

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

Transfusion therapy

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

The risks and benefits of the proposed transfusion therapy should be discussed with the patient and documented in the patient's medical records

Blood for emergencies

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

A rare occurrence, except for

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

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

Complications of blood transfusion

Immunological:

Sensitization to red cell antigens
Haemolytic transfusion reactions

- Immediate
 - Delayed
- Reactions due to white cell and platelet antibodies

- Febrile transfusion reactions
 - Post-transfusion purpura
- Reactions due to white cell and plasma protein antibodies

- Urticaria
- Anaphylaxis

Non-immunological:

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

Tests of Compatibility

A minimum of three major procedures must be carried out:

- Determine the recipient's ABO and Rhesus groups
 - Select compatible donor blood
 - Cross-match donor cells against recipient's serum
- Donor blood should be screened for infective agents: HIV, hepatitis B, and C viruses

Other investigations

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

Treatment objectives

To raise haemoglobin concentration and other blood parameters to normal levels

To prevent blood transfusion complications

Non-drug treatment

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

Provision of fresh frozen plasma or other blood products as necessary

Drug treatment

Furosemide 40 mg on administration of one unit of blood

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

- Promethazine 25 mg intramuscularly or intravenously
- Epinephrine 0.5 mL of 1:1000 solutions to be administered subcutaneously
- Hydrocortisone sodium succinate 100 mg injection

Supportive measures

- Appropriate nutrition
- Adequate hydration

Notable adverse drug reactions, caution

- Furosemide: dehydration and hypersensitivity
- Promethazine: drowsiness, hypersensitivity

Prevention

- Avoid/prevent accidents

Prompt treatment of illnesses that could be complicated by anaemia

Regular medical check-ups

HAEMOSTASIS AND BLEEDING DISORDERS - refer for specialist care

LEUKAEMIAS

Introduction

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

Two main types

- Myeloid leukaemia
 - Lymphoid leukaemia
- Each is further divided into acute and chronic
- Acute leukaemias are defined pathologically as blast cell leukaemias or malignancies of immature haematopoietic cells. The bone marrow shows > 30% blast cells

Two main groups of acute leukaemias

- Acute myeloid leukaemia (AML)
 - Acute lymphoblastic leukaemia (ALL)
- Childhood leukaemias: patients aged <15 years
- Adult leukaemias: patients aged >15 years
- Leukaemias in adults aged > 60 years: an important group because
- Their responses to current treatment protocols both for ALL and AML are inferior
 - These patients are not usually considered for more radical treatment approaches such as autologous or allogeneic bone marrow transplantation

80% of adult cases: AML

Epidemiology/predisposing conditions

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

More common in industrialized than rural areas

Environmental agents implicated in the induction of certain types of leukaemia:

- Ionising radiation: X-rays and other ionizing rays
- Chemical carcinogens

- Benzene and other petroleum derivatives
- Alkylating agents

Host susceptibility e.g. genetic disorders:

- Bloom's syndrome
- Fanconi's anaemia (AML)
- Ataxia telangiectasia (ALL)
- Down's syndrome

Blast transformation in pre-existing myeloproliferative disorders:

- Aplastic anaemia (ALL)

Oncogenic viruses:

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

Clinical features

General symptoms of anaemia

Bleeding

Infections

Anorexia

Weight loss

Lymphadenopathy (not common in AML except in the monocytic variant)

Skin:

- Macules, papules, vesicles
- Pyoderma gangrenosum
- Neutrophilic dermatitis
- Leukaemic cutis
- Granulocytic sarcoma

Differential diagnoses

- Septicaemia
- Miliary tuberculosis
- Malignant histiocytosis

Complications

Worsening ill-health

Investigations

Full blood count with ESR, reticulocyte count

Coomb's test

Bone marrow examination

Biochemical tests: serum electrolytes, urea, creatinine, uric acid

Liver function tests

Prothrombin time, partial thromboplastin time

Human Leucocyte Antigen typing

HIV I and II

Cytochemical tests

- Peroxidase

- Sudan Black B

- Non-specific esterase reaction e.g. alpha naphthyl 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 phosphatase-antialkaline phosphatase (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/m² intravenously on days 8, 15, 22 and 29
 Vincristine 1.4 mg/m² 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/m² intravenously on days 12, 15, 18, 21, 24, 27, 30 and 33
 Or:
COAP Regime
 Cyclophosphamide 650 mg/m² intravenously on days 1 and 8; 14 and 22
 Vincristine 1.4 mg/m² intravenously to a maximum of 2 mg; days 1 and 8; 14 and 22
 Cytosine Arabinoside 50 mg/m² subcutaneously 12 hourly for 12 days or bolus intravenous injection 100 mg/m² daily for 7 days
 Prednisolone 40 mg/m² oral for 14 days
 - Drugs are given every 28 days for 3 courses
 Nervous system prophylaxis
 - Methotrexate 12.5 mg/m² 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 = $1 \times 10^9/L$ and platelet count is = $100 \times 10^9/L$
Maintenance
 6-Mercaptopurine 75 mg/m² orally daily
 Methotrexate 20 mg/m² 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/m² to a maximum of 2 mg weekly on days 1 and 8
Acute myeloblastic leukaemia
 Either TAD or COAP as shown below:
TAD
 Cytarabine 100 mg/m² (continuous infusion) on days 1 and 2, and 100 mg/m² every 12 hours by intravenous infusion over 30 minutes on days 3 - 8
 Thioguanine 100 mg/m² every 12 hours orally on days 3 - 9
 Daunorubicin 60 mg/m² by intravenous infusion over one hour on days 3 - 5
 Or:
COAP
 Cyclophosphamide 650 mg/m² intravenously on days 1 and 8
 Vincristine 1.4 mg/m² intravenously to a maximum of 2 mg on days 1 and 8
 Cytarabine 50 mg/m² subcutaneously every 12 hours

for 7 days
 Prednisolone 40 mg/m² orally for 14 days
 - Nervous system prophylaxis is not required
 - Assess for remission after 3 courses
Maintenance
 COAP every 6 weeks for 2 years
 Intrathecal treatment as for ALL if there is CNS disease of the monocytic type
Chronic Myeloid Leukaemia (CML)
 Also Chronic Myelogenous Leukaemia; Chronic Granulocytic Leukaemia (CGL)
 A clonal disease that results from acquired genetic change in a pluri-potential haematopoietic stem cell
 Altered stem cell proliferation generates a population of differentiated cells, and a greatly expanded total myeloid mass
Classification
 Majority of patients have relatively homogenous disease characterized by:
 - Splenomegaly
 - Leucocytosis
 - Presence of Philadelphia (Ph) chromosome in all leukaemia cells
 - Minority of patients have less typical disease (atypical CML)
 - These variants lack Ph chromosome. Examples:
 - Chronic myelomonocytic leukaemia
 - Chronic neutrophilic leukaemia
 - 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 10⁹/L)
 Acquisition of specific new cytogenetic abnormalities
 Increasing marrow fibrosis
Blastic transformation
 More than 30% blasts
 Or:
 Blasts plus promyelocytes in blood or bone marrow

Clinical features
 Asymptomatic
 Abdominal swelling/pain
 Lethargy
 Shortness of breath on exertion
 Weight loss
 Unexplained haemorrhage at various sites e.g. gums, intestinal/urinary tracts
 Increased sweating
 Visual disturbances
 Gout
 Priapism
 Splenomegaly
 Anaemia
 Haemorrhage
 Fever
 Lymphadenopathy (rare in chronic phase)
Complications
 Blastic transformation
 Death
Investigations
 As above for acute leukaemia plus:
 Determination of Philadelphia chromosome
 Lactic dehydrogenase
 Serum calcium
Treatment objectives
 Induce remission to achieve complete remission
 Maintain disease-free state
 Achieve absence of Philadelphia chromosome
Non-drug treatment
 Appropriate nutrition
 Adequate hydration
 Electrolyte balance
Drug treatment
 Hydroxycarbamide (hydroxyurea)
Adult: 20-30 mg/kg orally daily or 80 mg/kg every third day
Child: Not recommended
 Interferon alpha
Adult: 9 million units subcutaneously or intravenously thrice weekly for 6 - 12 months
 Or:
 Imatinib mesylate
 - 400 mg orally daily
 - To be used strictly under specialist supervision
Notable adverse drug reactions, caution
 The above drugs (except the steroids) all cause profound myelosuppression
 Profound nausea, vomiting, diarrhoea and abdominal discomfort
 Secondary malignancies
 Steroids: Cushing's syndrome, hypertension, diabetes mellitus, immunosuppression, infections
 Vincristine: neurotoxicity
 Cyclophosphamide: 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 azurophilic 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/m² 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/m²
- Vincristine 1.4 mg/m²
- Prednisolone 100 mg orally days 1 - 5
- Repeat every 3 weeks

Or:

- Fludarabine 30 mg/m² intravenously over 30 minutes on days 1 - 3
- Cyclophosphamide 250 - 300 mg/m² 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/m² intravenously on day 1
- Doxorubicin 50 mg/m² intravenously on day 1
- Vincristine 1.4 mg/m² (maximum of 2 mg) intravenously on day 1
- Prednisolone 100 mg orally on days 1 - 5

CHOP (4 weekly):

- Cyclophosphamide 750 mg/m² intravenously on days 1 and 8
- Doxorubicin 25 mg/m² intravenously on days 1 and 8

- Vincristine 1.4 mg/m² (maximum 2 mg) on days 1 and 8
- Prednisolone 100 mg orally on days 1 - 8

Hodgkin's lymphoma

MOPP

- Mechlorethamine 6 mg/m² intravenously on days 1 and 8
- Vincristine 1.4 mg/m² (maximum 2 mg) intravenously on days 1 and 8
- Procarbazine 100 mg/m² orally on days 1 4
- Prednisolone 40 mg orally on days 1 - 14

ChIVPP

- Chlorambucil 6 mg/m² orally on days 1 and 14
- Vinblastine 6 mg/m² (maximum 10 mg) intravenously on days 1 and 18
- Procarbazine 100 mg/m² 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-β thalassaemia (Sβ thal)
- Sickle cell-β+ thalassaemia Type I (Sβ+thal.Type I)
- Sickle cell-β+thalassaemia. Type II. (Sβ+thal. Type II)

Sickle cell-β+thalassaemia. Type III. (Sβ+thal. Type III)

Sickle cell trait

Inheritance of one normal gene controlling formation of β 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 β 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 pathophysiological 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 13.2 years)
- 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/conserves 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
 - Pneumococcal 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
- Strenuous 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

- β 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

- β 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 β-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 arrhythmia
- 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 cardiography
- 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 VEIN 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
- Prothrombin 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
Inotropes
- Dopamine 2 - 5 microgram/kg/minute by intravenous infusion
Anticoagulants

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

Supportive measures
Pacemakers for arrhythmias
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 hyperlipidaemia

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 \geq 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/m²)
 - 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
- β-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 methyl dopa 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 nitroprusside)

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 methyl dopa, 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
- Splenomegaly
- 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:
 - Benzyloxy penicillin 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*)

β 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

β-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

Sarcoidosis

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
- Indomethacin 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
- Electrocardiography
- Sinus tachycardia
- New onset atrial fibrillation/flutter
- S wave in lead I, Q wave in lead III and an inverted T wave in lead III
- QRS axis >90°, quite often
- Chest radiograph
- Blood gases (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 maintenance 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 β-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

Subcutaneous 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 1 g 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) intramuscularly 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

Investigations

- Full Blood Count and differentials
- Electrocardiography
- Echocardiography
- Random blood glucose
- X-ray sinuses

Neuro-imaging: CT scan, MRI, carotid Doppler etc

Management

Depends on the aetiological factor identified

Treatment objectives

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

Non-drug treatment

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

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, quadriplegia)
- 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
- Syndromes 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 funduscopy 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 every 12 hours for 5 days

Child: 10 mg/kg orally every 12 hours for 5 days

Under 1 year: 5 mg/kg orally every 12 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, hemophilus, 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

Basilar 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: unrelied, 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:

Propranolol

- 40 mg orally every 8 - 12 hours

Tricyclic antidepressants, notably amitriptyline

- 10 mg orally at night, increased to a maintenance dose of 50 - 75 mg at night

Sodium valproate

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

In refractory cases:

Cyproheptadine

- An antihistamine with serotonin-antagonist and calcium channel-blocking properties

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

Notable adverse drug reactions, caution and contraindications

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

Tricyclic antidepressants used with caution in patients with cardiac symptoms

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

- Caution in patients with vascular and renal disorders

- Not recommended for children

Opiates: risk of addiction

β-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
Creutzfeldt-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

- Amitriptyline 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/overdose/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 resuscitation 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 every 12 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' (myocardial 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: Apoplexy (stroke)

E: Epilepsy

I: Infections e.g. meningo-encephalitis

O: Overdosing with drugs, alcohol intoxication, toxins

U: Uraemia and other metabolic disorders

Other causes include:

Head injury

Brain tumours (with complications)

Clinical features

Varying levels of impaired consciousness:

Comatose: no response to stimulus, however painful

Semi-comatose: some response to pain

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

Other features:

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

Unresponsiveness or variable response to painful stimuli

- Features of the underlying cause(s)
- Stroke: may present with hemiparesis, facial asymmetry, crossed-eye defects, speech defects etc
- Epilepsy: frothing or tongue biting; abrasions of the extremities; positive past history
- Infections: may present with fever, neck stiffness
- Drug overdose/toxins: pin-point pupils; respiratory problems; suggestive history
- Uraemia: characteristic fetor; skin rashes; oedema; severe dehydration
- Head trauma: haematomas; subconjunctival 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
- Myocardial 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. hyperthyroidism)

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 breath

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:

Amityriptiline 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 hours 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 predominant, 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 anaesthesia

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 obtundant dressing containing some non-irritant antiseptic

Warm saline mouth rinse

Drug treatment

Local anaesthesia

- Lidocaine 2% (1 in 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 hyaluronidase 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 intravenously every 8 hours

Child: neonate and premature infants, 25 mg/kg every 12 hours; infants up to 3 months, 25 mg/kg every 8 hours, 3 months to 12 years, 25 mg/kg every 8 hours increased to 25 mg/kg every 6 hours in more severe infections

Injection gentamicin:

Adult: 3 - 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 pulp 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

Immunosuppression 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 B₁₂, 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 immunosuppression)

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, *Actinomyces 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 - 7 days

NSAIDs may be required in some patients

Notable adverse drug reactions

- Aspirin and other NSAIDs
- Gastrointestinal haemorrhage, allergic reactions
- Do not prescribe for patients with peptic ulcer disease
- May exacerbate symptoms in asthmatics

Aspirin is contraindicated in children less than 16 years as it may precipitate Reye's syndrome

Prevention

Prevent dental caries (the most important cause of pulpitis)
Seek prompt dental attention

SALIVARY GLAND DISEASES**Introduction**

A wide spectrum of disorders

Diseases due to obstruction

- Salivary calculi
- Parotid papilla and duct strictures
- Salivary fistulae
- Mucocele 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 β -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 - 7 days

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 - 10 days

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 streptococci 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 peroxide

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 agent
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 dermatitis 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

Criteria for systemic steroid therapy

Failed maximal therapy; little improvement after environmental changes

- Chronic unbearable, unrelenting itch
- Erythroderma without infections

Social setting in which other modalities are impossible

Smallpox vaccination is absolutely contraindicated**Guidelines for the use of potent topical steroids in infants**

- Do not use on the face, axillae, diaper area or flexures
- Do not use under occlusion
- Do not use for an area greater than about 25% of total body surface area
- Do not use for more than 2 weeks consecutively and do not give refills

Do not dispense more than 50 g per week

Always use sparingly

Adjunctive measures

- Exclusive breastfeeding; milk substitute if need be
- Attention to cleanliness especially in the diaper region
- Avoid excessive bathing, vigorous rubbing, or chafing
- Avoid unduly heavy, tight, or soiled clothing
- Treat local infections

Pat (rather than rub) skin dry after bath and immediately lubricate skin with petroleum jelly or emulsifying ointment

Showers should be warm to cool, not hot
Tub soaking is good, if followed by adequate lubrication

Avoid wool; its fibers are irritating
Emotional stress leads to increased scratching

In patients and parents of affected children, other psychologic techniques may be useful

Secondary skin infection with bacteria such as *Staphylococcus aureus* may worsen the dermatitis and itching

Patients must consciously be shielded from anyone with varicella or herpes simplex

Keep finger nails trimmed short

- Some kinds of soap may irritate and dehydrate the skin; use synthetic soap powders
- Reassure patients and/or anxious parents
- Use patient education handouts
- Allergy tests, restriction diets and environmental hypoallergenic changes will not cure eczema*
- Notable adverse drug reactions**
- Steroids
 - Increased susceptibility to and severity of infection
 - Activation or exacerbation of tuberculosis, amoebiasis, strongyloidiasis
 - Risk of severe chickenpox in non-immune patients
 - Nausea, dyspepsia, hiccups
 - Hypersensitivity reactions
 - Atrophy of the skin; striae, telangiectasia, petechiae
 - Glaucoma, cataracts
 - Cushingoid syndrome, adrenal/pituitary suppression, hyperglycaemia and diabetes mellitus
 - Suppression of growth in children
 - Menstrual irregularities
 - Oedema
 - Electrolyte imbalance
 - Hypertension
 - Pseudotumour cerebri

CONTACT DERMATITIS

Introduction

- An acute or chronic dermatitis that results from direct skin contact with chemicals or allergens
- These agents could be
 - Chemicals
 - Animal or plant products
 - Physical agents like heat, cold, ultraviolet rays or ionizing radiation
- Contact dermatitis is classified as :
 - Irritant dermatitis
 - Acute irritant dermatitis
 - Cumulative insult dermatitis
 - Allergic contact dermatitis
 - Phototoxic dermatitis
 - Photo-allergic dermatitis

Clinical features

- Acute phase
- Tiny vesicles, weepy and crusted lesions
- Resolving or chronic contact dermatitis
- Scaling, erythema, and possibly thickened (lichenified) skin
- Itching, burning, and stinging may be severe
- Contact dermatitis is recognized by the distribution and configuration of the lesion which usually corresponds to the contactant e.g
 - Face: cosmetics
 - Photodermatitis: airborne allergens e.g. dust, fumes, sprays
 - Neck: nickel necklace, perfume, and collars of garments

- Hands: various chemicals handled at home, at work and at leisure hours
- Feet: shoes, socks, remedies for athletes' foot, etc

Differential diagnoses

- Atopic dermatitis
- Seborrheic 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
- Seborrheic 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
- Stearorrhoea
- 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
- Emollients 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 Rajasthan 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

Osteomyelitis

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 encephalopathic 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 inflamed 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 tissues, 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 confluent-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

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 unguium)
 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
 Parapsoriasis
 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 valerate 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 serpinginous
- 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

Small lesions and nail psoriasis

Intra-lesional corticosteroid injections of triamcinolone are frequently used

- Triamcinolone acetamide suspension 10 mg/mL may be diluted with sterile saline to make a concentration of 2.5 - 5 mg/mL
- For nail lesions inject triamcinolone in the region of the matrix and the lateral nail fold

Scalp

- Soften scales with salicylic acid 3% in mineral/olive oil, massage in and leave on overnight
- Then shampoo with a tar shampoo, and remove scales mechanically with a comb and brush
- Repeat daily until the scales are gone
- If 3% is not very effective, use 6% salicylic acid

Or:

Fluocinolone acetonide 0.01% in oil

- Apply and leave under a shower cap at night and shampoo in the morning
- After shampooing and while the hair is still wet, massage thoroughly into the scalp skin
- Attempting to remove scales by excessive brushing, scrubbing, or combing may result in sufficient trauma 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
- Acitretin

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 inflamed, 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 D₃ (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, arthralgia, 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)

Acitretin:

- See Acne- isotretinoin

Tacrolimus:

- See Atopic eczema

Efaluzimab:

- Thrombocytopenia, hepatic and renal impairment.
- Monitor platelet count during initial therapy, 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, scratches, 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:

Microsporium, *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 tineaes 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 inflamed 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
- Miconazole
- 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 tinea 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:
 Selenium sulphide shampoo
 - Apply on affected areas daily, leave on for 10 - 30 minutes and wash off
 - Continue for 3 weeks
 Ketoconazole shampoo
 - Use as above
 Miconazole cream
 - For limited areas
 - Apply twice daily for 3 weeks

Supportive measures
 Deal with underlying predisposing factor(s)

Prevention
 Avoid hot, humid environments or clothings that promote perspiration
 Take a cold shower after perspiration
 Use any of the above shampoo washes once a month if predisposed

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 pearly 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
 Cocksackie 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 pinpoint 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
- Seborrheic 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

- Papanicolaou (PAP) smear to detect cervical dysplasia

MISCELLANEOUS DISORDERS**ACNE VULGARIS (Pimples)****Introduction**

One of the most common skin diseases

A disorder of the pilosebaceous follicles

Typically first appears during puberty when androgenic stimulation triggers excessive production of sebum

Many factors interact to produce acne in a given patient

- Genetics
- Sebum production
- Hormones
- Bacteria
- Properties of the sebaceous follicle
- Immunologic

Over-production of stratum corneum cells (hyperkeratosis) obstructs the hair follicles at the follicular mouth producing open comedones, or blackheads

Just beneath the follicular opening in the neck of the sebaceous follicle it causes microcomedones (closed comedones, or whiteheads)

There is an overgrowth of gram-positive bacteria in the obstructed follicle: *Propionibacterium acnes* or *Staphylococcus epidermidis*; distally *Pityrosporum ovale*

Rupture of the comedonal contents into the dermis induces a foreign body reaction and inflammation

Clinical features

Almost every individual has some degree of acne during puberty, with spontaneous resolution occurring in early adult life

Occasionally, the disease persists into the fourth decade, or even remains a life-long problem

Favoured sites are the face, upper back and upper chest and shoulders

There may be mild soreness, pain, or itching

May present differently in different age groups

- Pre-teens often present with comedones as their first lesions

- Teenage acne is invariably inflammatory and the lesions include firm red papules, pustules, abscesses, indurated nodules, cysts and rarely interconnecting draining sinus tracts

Inflammatory acne can be classified as mild, moderate, or severe

Mild acne:

- Few-to-several inflammatory papules and pustules, but no nodules

Moderate acne:

- Several-to-many papules, pustules, and a few to several nodules

Severe acne (acne conglobata):

- Numerous fistulated comedones; extensive inflammatory papules; pustules; many cysts, abscesses, nodules, and draining sinuses

- The lesions may be generalized, involving even the buttocks

- Excoriation of acne papules and microcomedones are common, and scarring may result

- Usually, multiple shallow erosions or crusts are found

Differential diagnoses

Acne rosacea

Dermatosis papulosa nigra

Steatocystoma multiplex

Syringoma

Trichoepithelioma

Warts

Angiofibromas of tuberous sclerosis

Molluscum contagiosum

Steroid acne from the use of systemic steroids or topical fluorinated steroids on the face (often as cosmetic skin lightening creams)

Some drugs may produce acneiform eruptions

- Androgens
- Adrenocorticotropic hormone (ACTH)
- Glucocorticoids
- Hydantoins
- Isoniazid
- Halogens

Complications

Psychosocial problems from cosmetic disfigurement

Post-inflammatory pigmentary changes

Pitted scars

Keloids

Acne fulminans (acute febrile ulcerative acne conglobata with polyarthritis and leukemoid reaction)

Investigations

- Usually, none required
 In the presence of unusual acne, hirsutism, premature pubarche, or androgenic alopecia (especially when associated with obesity and/or menstrual irregularities):
 Screen for hyperandrogenism
 Blood levels of free testosterone, dehydroepiandrosterone, and androstenedione
 - If raised, test response of the hormones and cortisol to dexamethasone suppression

Treatment objectives

- Reduce severity of acne
- Prevent complications

Drug treatment

Comedonal acne

Topical treatment only:

- Tretinoin cream
Adult: 0.025% **or** 0.05% **or** 0.1% cream or gel applied nightly
Child: apply thinly 1 - 2 times daily

Or:

- Benzoyl peroxide
Adult: 2.5% **or** 5% water-based or alcohol-based gels, applied twice daily
Child 12-18 years: apply 1 - 2 times daily preferably after washing with soap and water
 - Start with lower strength preparations
 Infantile acne:
Child 1 month to 2 years; neonate: apply 1 - 2 times daily
 - Start with lower strength preparations

Or:

- 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-cyprindiol:

- 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 sebum 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 absorption may occur from the skin in children

Isotretinoin:

Dry skin, lips and eyes

Decreased night vision

Epistaxis

Hypercholesterolaemia

Hypertriglyceridaemia

Pseudotumour cerebri and headaches

Depression

Musculoskeletal or bowel symptoms

Thinning of hair

Bony hyperostoses

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 methaemoglobinemia

- 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 susceptible 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

Sodium 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 inhibitor 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:

- Pemphigus
- Pemphigoid
- Erythema multiforme
- Fixed drug eruption

Angioedema:

- “Calabar swelling”
- Cellulitis
- Idiopathic scrotal oedema of children
- Melkerson-Rosenthal syndrome

Cold urticaria:

- 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: 1 mg every 4 - 6 hours (maximum 6 mg daily); 6 - 12 years: 2 mg every 4 - 6 hours (maximum 12 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 12 hours

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 dermatographism 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 with 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
- Dysacusis
- 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 sclerosus
- 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
- 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
- Monobenzy ether of hydroquinone
- 20%, apply twice daily for 3 - 6 months (if more than

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/cm², gradually increased

Two or three treatments are done per week for 3 - 6 months

- Systemic corticosteroids
- May occasionally be used to arrest the autoimmune process
- Prednisolone tablets 0.5 - 1.0 mg/kg orally day

Surgical

- Pigmented skin grafted onto vitiliginous patches
- Often the transferred melanocytes repigment the depigmented areas

The various techniques include:

- Suction blister grafts
- Mini-punch grafts
- Transfer of either pure melanocyte cultures or mixed epidermal cultures to a prepared site

Adjuvant measures

- Camouflage (cover-up cosmetics)
- Patient education and emotional support

Notable adverse drug reactions, caution and contraindications

Corticosteroids:

- See Dermatitis and Eczema

8-MOP:

- Inadvertent sunburns with blistering

Systemic psoralen is contraindicated in:

- Known photosensitivity
- Porphyria
- Liver disease
- Systemic lupus erythematosus

If systemic therapy is to be used the following should be done before therapy

- Ophthalmological examination
- Full Blood Count
- Liver function tests
- Antinuclear Antibody Test

Monobenzylether of hydroquinone:

- Depigmentation at distant sites
- Acquired ochronosis

PVA 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*
- 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

- Pseudoephedrine

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
- Pseudoephedrine 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 every 12 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 air cells
 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

- Pseudoephedrine

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 (Acetylsalicylic 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
- Pseudoephedrine 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 SYSTEM

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

Principles 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/m²) 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 premixed 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

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 $\geq 25 \text{ kg/m}^2$
- Previous history of GDM
- Glycosuria
- Previous large baby ($> 4 \text{ kg}$)
- 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 $> 5.8 \text{ mmol/L}$
 - 1 hour post-prandial glucose is $> 8.6 \text{ mmol/L}$
 - 2 hour post-prandial plasma glucose is $> 7.5 \text{ mmol/L}$
- 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-hydroxybutyrate) concentration $> 5 \text{ mmol/L}$

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 below 14 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^+ $3.5 - 5.5 \text{ mmol/L}$:

Add 20 mmol KCl

Plasma K^+ greater than 5.5 mmol/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 $> 50 \text{ mmol/L}$)
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 ($< 2 \text{ mmol/L}$) 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 unconscious:

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
 - Lipohypertrophy 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 > 45 years
 - Overweight and obesity (BMI > 25 kg/m²)
 - 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 (T₄) and Tri-iodothyronine (T₃)
 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
 Thyroiditis (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 T₃, T₄ and TSH levels
 - Measurement of I¹³¹ 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 T₃, T₄ 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 pancytopenia and agranulocytosis)
- They are contraindicated in breastfeeding mothers

HYPOTHYROIDISM (Myxoedema)

Introduction

Refers to subnormal amounts of thyroid hormones in the circulation, and the clinical features associated with this

Aetiology

May be primary or secondary

Primary hypothyroidism more common

- Probably an autoimmune disease; may occur as a sequel to Hashimoto's thyroiditis

- Post therapeutic hypothyroidism (medical or surgical)

Secondary hypothyroidism:

- Occurs when there is failure of the hypothalamic-pituitary axis due to

- Deficient secretion of TRH from the hypothalamus

Or:

- Lack of secretion of TSH from the pituitary

Clinical features

Generally in striking contrast to those of hyperthyroidism; may be quite subtle, with an insidious onset

In adults:

- Dull facial expression, slow speech and poor memory
- Puffiness of the hands, feet and face
- Lethargy and fatigue
- Thinning, dryness and loss of hair
- Hypothermia
- Bradycardia
- Reduced systolic and increased diastolic blood pressure

Weight gain

Decreased reflexes

Constipation

Menstrual abnormalities

In infants:

- Mental and physical retardation

- If not corrected, cretinism

Differential diagnoses

- Endogenous depression

- Reactive depression

Complications

- Myxoedema coma

- Cretinism in the young

Investigations

- Total serum T_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 ciliary body)

Usually occurs without any associated systemic inflammation

Tends to recur

Clinical features

- Eyeball is tender
- Photophobia due to ciliary 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 lumbosacral segment) to exclude ankylosing spondylitis

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 fluorescein; 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 its own or in association with generalized atopy (asthma, eczema, spring catarrh)

Clinical features

Itching of the eyes with grittiness

- May be associated with itchy ears and throat, or sinusitis

Brownish discolouration of the conjunctiva

Eyelid oedema

Red eyes occasionally, with watering when acute

Follicles on the bulbar conjunctiva especially at the limbus

Papillae on the tarsal conjunctiva (seen on eversion of the eyelid)

Phlycten in tuberculosis- appears as a yellow nodule with surrounding leash of engorged vessels

Aetiology

- Exogenous allergens
- Topical drugs - atropine, penicillin
- Cosmetics
- Pollen from plants and flowers (hay fever or spring catarrh)
- House dust mite and animals
- Endogenous allergens
- Phlyctenular conjunctivitis caused by tuberculo-protein

Differential diagnoses

Trachoma
Other forms of conjunctivitis

Complications

Pannus formation
Keratoconus
Corneal plaques

Investigation

Skin sensitivity test to detect allergen

Drug treatment

- Antiinflammatory preparations
- Antazoline sulfate 0.5%, xylometazoline hydrochloride 0.05%

Adult and child over 5 years: apply 2-3 times daily

- Sodium cromoglycate eye drops
Adult and child: apply four times daily

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

Phlyctenular conjunctivitis:

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 ciliary 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 (Comotio 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

Conjunctivitis
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
- Haemophilus 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 ophthalmia 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 REDEYE**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
Antihypertensive 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
- Ferrus 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
Membrano-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 curtisi

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 cadaverine, 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, every 12 hours 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, Compliance, Condom use and Contact treatment

CHANCROID (Ulcus Molle, Soft Chancre)**Introduction**

An infectious disease caused by *Haemophilus ducreyi*, a small gram-negative bacillus

Common in the tropics, especially in Africa, the Far East, and the Caribbean

Persons may present with chancroid outside endemic regions; sporadic outbreaks of infection occur in Europe and North America

Clinical features

Incubation period is about 3 - 7 days

Begins as a small, tender papule, changing into a pustule which rapidly progresses to a painful ulcer with a bright red areola

Neither the edge nor base of the ulcer is indurated (unlike syphilis)

- The ulcer feels soft, hence the name 'soft sore' (ulcus molle)

With superimposed bacterial infection it often feels indurated

The ulcers may be multiple due to auto-inoculation
Sites of predilection in men are the prepuce, frenulum, glans or shaft of the penis

In women the labia, fourchette, vestibule, clitoris, cervix, or perineum are favored sites

Lesions may cause dyspareunia, pain on voiding or defaecation and vaginal discharge

Women may be asymptomatic carriers

About 7 - 14 days after the appearance of the ulcer, a bubo appears

- A mass of 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, Compliance, Condom use and Contact treatment

CHLAMYDIAL INFECTION

(Other than Lymphogranuloma venereum)

Introduction

The chlamydiae occupy a special place between bacteria and viruses

- They are a large group of obligate intracellular organisms

Chlamydia trachomatis has a number of serovars and causes many different human infections

- Eye: trachoma; inclusion conjunctivitis

- Genital tract: lymphogranuloma venereum, non-gonococcal urethritis, cervicitis, salpingitis

- Respiratory tract: pneumonia

C. trachomatis immunotypes D - K are isolated in about 50% of cases of non-gonococcal urethritis and cervicitis by appropriate techniques

Clinical features

Infections are asymptomatic, but when an incubation period can be determined, it is usually about 10 - 20 days

Co-infection with gonococci and chlamydiae is common

C. trachomatis is an important cause of non-gonococcal urethritis in males, and in females cervicitis, salpingitis, or pelvic inflammatory disease

Urethral or cervical discharge tends to be less painful, less purulent, and watery in chlamydial compared with gonococcal infection

On physical examination, the cervix may show contact bleeding in addition to the discharge

A patient with urethritis or cervicitis and absence of gram-negative diplococci on Gram stain and of *N. gonorrhoeae* on culture is assumed to have chlamydial infection

Complications

Epididymo-orchitis and sterility in males

Pelvic inflammatory disease (PID) and infertility in females

Adverse pregnancy outcomes

Conjunctivitis and pneumonia in the newborn

Differential diagnoses

Other causes of urethral and vaginal discharge (see Gonorrhoea)

Investigations

Microscopy, culture and sensitivity (of discharge)

Direct immunofluorescence assay

Enzyme-linked immunoassay

DNA probe test

Ligase chain reaction (LCR)

Treatment objectives

Same as for gonococcal infection

Drug therapy

Recommended regimen:

Doxycycline 100 mg orally, every 12 hours for 7 days

Or:

Azithromycin 1 g orally, in a single dose

Chlamydial infection during pregnancy

Recommended regimen:

Erythromycin 500 mg orally every 6 hours for 7 days

Or:

Amoxicillin 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 ophthalmia

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 reinstated 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 chronic lymphocytic leukaemia with pityriasis rosea, and allopurinol use

Prevention

Counselling, Compliance, Condom use and Contact treatment

GONORRHOEA

Introduction

Caused by *Neisseria gonorrhoeae*, a gram-negative aerobic diplococcus

It prefers the columnar epithelium of the urethra, the cervical canal, the rectum and the conjunctivae.

The keratinizing epithelium of the adult vagina is quite resistant to *N. gonorrhoeae*, but that of the pre-pubertal girls, pregnant women and the elderly is more easily colonized

Occasionally *N. Gonorrhoeae* reaches the 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):

Littre abscess involving periurethral glands

Paraurethral abscesses

Proximal urethral involvement with frequency and terminal haematuria

Cowper's gland abscess involving the bulbourethral glands, producing a swelling behind the base of the scrotum that can produce a proximal or Cowper's stricture

Prostatitis

Proctitis

Urethral stricture leading to hydronephrosis and hydroureteritis

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

Varicocele

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 defaecation

Ophthalmia neonatorum

Gonococcal conjunctivitis in the neonate can be acquired perinatally

Purulent conjunctivitis; the lids swell; eyes are red and tender

If not treated promptly, the cornea may be eroded and perforated, leading to secondary glaucoma, conophthalmus and blindness

About 30% of babies infected will also have oropharyngeal gonorrhoea

Differential diagnoses

The silver nitrate prophylaxis can produce a chemical conjunctivitis, usually appearing 6 - 8 hours after treatment and resolving over 24 hours

The most common cause of neonatal conjunctivitis in most countries is *C. trachomatis*.

- *E. coli*, staphylococci, streptococci and *Pseudomonas* sp. can also cause conjunctivitis in the neonate

Treatment objectives

Eliminate the organism in the patient and sexual partner(s)

Prevent re-infection

Prevent complications

Counsel and screen for possible co-infection with HIV so that appropriate management can be instituted

Drug therapy

Recommended regimen:

Ciprofloxacin 500 mg orally, as a single dose

Or:

Ceftriaxone 125 mg by intramuscular injection, as a single dose

Neonatal gonococcal conjunctivitis

Recommended regimen:

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

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, Compliance, Condom use and Contact treatment

Ocular prophylaxis provides poor protection against *C. trachomatis* conjunctivitis

Prevention of ophthalmia neonatorum

Clean the eyes carefully immediately after birth

The application of 1% silver nitrate solution or 1% tetracycline ointment to the eyes of **all infants** at the time of delivery is strongly recommended as a prophylactic measure

Infants born to mothers with gonococcal infection should receive additional antibiotic treatment (as those with clinical neonatal conjunctivitis)

GRANULOMA INGUINALE (Donovanosis; Granuloma venereum)

Introduction

A mildly contagious disease caused by *Klebsiella granulomatis*

Currently rare in several parts of Africa
Endemic in Southeast Asia, Southern India, the Caribbean and South America

Clinical features

A chronic mildly contagious disease with a potentially progressive and destructive character

Incubation period ranges from 10 - 40 days

The early lesion is a papule or nodule which soon becomes ulcerated and has an offensive discharge

The floor of the ulcer may be covered with a dirty grey material; its walls may be overhanging, or a papillomatous fungating mass may arise from the growth of vegetations

Progressive indolent, serpiginous ulceration of the groins, pubis, genitals and anus may form. Pain on walking may be excruciating

Persisting sinuses and hypertrophic depigmented scars are fairly characteristic

Regional lymph nodes are not enlarged but with cicatrization, 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, Compliance, Condom use and Contact treatment

LYMPHOGRANULOMAVENEREUM

(Climatic bubo; lymphogranuloma inguinale; lymphopathia venerea; 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, papulopustular lesions and photodermatitis)

- Raised ESR

Late stage

Spontaneous remission is common, though some patients enter the late stage

Characterized by disfiguring and destructive sequelae
Impairment of the lymphatic drainage from fibrotic scarring leads to distant oedema and gross elephantiasis of the genitalia

- There could be associated anorectal and vaginal strictures

Complications

Systemic spread of *C. trachomatis* in the secondary stage resulting in arthritis, pneumonia, hepatitis or rarely perihepatitis

Other rare systemic complications include pulmonary infection, cardiac involvement, aseptic meningitis, and ocular inflammatory disease

The late stage may be complicated by the genito-anorectal syndrome

- Reported more in homosexual men, and women who engage in receptive anal intercourse

Patients may also complain of fever, pain, and tenesmus.

Obstructed labour from elephantiasis of the vulva

Differential diagnoses

Buboes:

- Chancroid

- Infections of the lower limbs

- Hodgkins disease and other lymphomas

- Plague
 - Tularemia
- Late stage:
- Tuberculosis
 - Deep mycosis of the genitalia
 - Squamous cell or basal cell carcinoma

Investigations

- Culture and cell typing of the isolate from an aspirate of involved lymph node
- Serological tests e.g. CFT and MIF; PCR

Treatment objectives

- Same as for gonorrhoea

Drug treatment**Recommended regimen:**

- Doxycycline
- 100 mg orally every 12 hours for 14 days

Or:

- Erythromycin
- 500 mg orally every 6 hours for 14 days

Alternative regimen:

- Tetracycline
- 500 mg orally every 6 hours for 14 days

Adjuvant measures

- Aspirate fluctuant lymph nodes through healthy skin
- Incision and drainage or excision of nodes may delay healing and is not recommended
- Some patients with advanced disease may require treatment for longer than 14 days, and sequelae such as strictures and/or fistulae may require surgery

Notable adverse drug reactions, caution and contraindications

- See Chlamydia

Prevention

- Counselling, Compliance, Condom use and Contact treatment

SYPHILIS**Introduction**

Infection caused by the spirochaete *Treponema pallidum*

- Occurs worldwide

Can be classified as:

- Congenital (transmitted from mother to child *in utero*)
- Acquired (through sex or blood transfusion)
- Acquired syphilis may be early or late
- Primary syphilis is characterized by an ulcer or chancre at the site of infection or inoculation

Manifestations of secondary syphilis include a skin rash, condyloma lata, mucocutaneous lesions and generalized lymphadenopathy

Early syphilis: primary, secondary and early latent stages

Primary syphilis: an ulcer or chancre at the site of infection or inoculation

Secondary syphilis: skin rash, condyloma lata, mucocutaneous lesions and generalized lymphadenopathy

Late syphilis: late latent syphilis, gummatous, neurological and cardiovascular syphilis

This section is only on primary syphilis

Clinical features

After an incubation period of 2 - 4 weeks (full range 90 days) the first lesion of syphilis may appear at the site of exposure, most commonly, the genitals

Chancres may also be located on the lips or tongue; ano-rectal chancres frequently seen in male homosexuals

- Begins as a small, dusky-red macule which soon develops into a papule

The surface of the papule erodes to form an ulcer which is typically round and painless with a clean surface and exudes a scanty yellow serous discharge teeming with spirochaetes

Lesion is indurated and feels firm or hard on palpation; surrounding skin is oedematous

Regional inguinal (or generalized) lymphadenopathy follows

The glands are painless, moderately enlarged (not buboes), discrete and never suppurate

Atypical lesions may be seen for various reasons e.g. bacterial superinfection, trauma or co-infection with chancroid.

Even without treatment, the primary lesion(s) gradually heals up and will disappear after approximately 3 - 8 weeks, sometimes leaving a thin atrophic scar which is easily overlooked

Differential diagnoses

Other causes of genital ulcers:

- Chancroid
- Herpes
- Lymphogranuloma venereum
- Granuloma inguinale
- Trauma
- Fixed drug eruption
- Behcet's disease
- Erythema multiforme
- Tuberculous ulcer
- Amoebic ulcer
- Cancer

Complications

- Phimosi and paraphimosi
- 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 interstitial nephritis, haemolytic anaemia, leucopaenia, thrombocytopenia and coagulation disorders
- Other antibiotics
- See Chlamydia

Prevention

Counselling, Compliance, Condom use and Contact treatment

All infants born to seropositive mothers should be treated with a single intramuscular dose of benzathine penicillin

- 50,000 units/kg, whether or not the mothers were treated during pregnancy (with or without penicillin)

Prevention of congenital syphilis is feasible

- Programmes should implement effective screening strategies for syphilis in pregnant women

Screening for syphilis should be conducted at the first prenatal visit

Some programmes have found it beneficial to repeat the tests at 28 weeks of pregnancy and at delivery in populations with a high incidence of congenital syphilis

TRICHOMONIASIS**Introduction**

Caused by the flagellated protozoan, *Trichomonas vaginalis*

An extremely common infection, almost always, transmitted via sexual contact

Women are far more frequently affected and more likely to have symptoms

Men are more likely to be asymptomatic and serve as carriers

Clinical features

Vaginal discharge: a white-yellow frothy discharge is characteristic

Burning sensation

Dysuria

Dyspareunia

The labia 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, Compliance, Condom use and Contact treatment

VULVO-VAGINAL CANDIDIASIS

Introduction

Inflammation of the vagina and vulva, usually evolving from vaginal discharge and secondary external irritation

Candida albicans is the commonest cause of candidal vulvo-vaginitis; *Candida glabrata* has also been identified

Candidal vaginitis is most common in:

- Pregnancy
- Patients with diabetes mellitus
- Those on long-term antibiotic therapy or oral contraceptives
- Conditions associated with 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
- Hydrourerter/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/m² 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 E₁
 - 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: herniorrhaphy, orchidopexy, urethral surgeries, etc

Examination:

Gynaecomastia
 Penis: epispadias, hypospadias, penile deformities
 Scrotum: absence of testis, small sized testis, varicoceles, 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:

Varicocelectomy
 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, *Streptococcus faecalis* and *Staph aureus*

Chronic bacterial prostatitis
Caused by *E. coli*, Klebsiella, Mycoplasma and Chlamydia

Non-bacterial prostatitis
An inflammation of indeterminate cause

Clinical features

Acute prostatitis
Systemic features
- Fever
- Chills
- Malaise
- Nausea
Local features
- Dysuria
- Frequency
- Haematuria
- Urethral discharge

Rectal examination:
- Hot boggy, swollen and very tender prostate

Chronic prostatitis
Voiding symptoms: dysuria, frequency, urgency, haematuria
Poor stream
Urethral discharge
Low back pain

Perineal pain
Haemospermia
Painful ejaculation
Rectal examination: enlarged, tender, firm prostate

Differential diagnoses
Benign prostatic hypertrophy
Cystitis
Urethral stricture
Prostate cancer

Complications
Prostatic abscess
Prostatic calculi
Infertility
Septicaemia

Investigations
Urinalysis
Urine microscopy, culture and sensitivity
Prostatic massage: microscopy, culture and sensitivity (chronic prostatitis only)
Trans-rectal ultrasound
Biopsy: culture and histology
Urethrocystoscopy (chronic prostatitis only)
Full Blood Count; ESR

Treatment objectives
To eradicate causative organisms
Control pain

Drug treatment
Antibiotics (based on local sensitivity)
- Ciprofloxacin 500 mg orally every 12 hours for 28 days
Or:
- Cotrimoxazole 960 mg orally every 12 hours for 28 days
Anti-inflammatory drugs
- Non-steroidal e.g. diclofenac, ibuprofen etc
- Steroids e.g. prednisolone, dexamethasone
Alpha blockers e.g. prazosin, doxazosin
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/bougination
Endoscopic direct visual urethrotomy
Urethroplasty: excision and end-to-end anastomosis
Substitution urethroplasty

Prevention

Ensure prevention of sexually transmitted infections
Prompt and appropriate treatment of sexually transmitted infections

Care and attention to asepsis during instrumentation procedures involving the urethra

URINARYSCHISTOSOMIASIS**Introduction**

A common parasitic infection of the urinary tract caused by a body fluke, *Schistosoma haematobium*

Acquired while bathing/wading in infected water

Endemic in many parts of Africa

Gets to the urinary tract through the blood vessels after penetrating the skin

Clinical features

Soon after penetration of the skin:

Pricking sensation and itching (cercarial dermatitis)

Four weeks later:

Intermittent fever, malaise, urticaria and cough

Six - 24 months later:

Intermittent, painless terminal haematuria (may be total)

Symptoms of bladder irritability: dysuria, frequency, urgency, strangury

Differential diagnoses

Tuberculous cystitis
Abacterial cystitis
Bladder carcinoma

Complications

Bladder fibrosis and contracture
Ureteral stricture
Urethral stricture
Bladder calculi
Bladder cancer

Investigations

Urine examination for schistosomal ova
Cystoscopy: tubercles, sandy patches, nodules, ulcers
Plain abdominal radiograph (KUB)
Intravenous urogram
Serological tests
Full Blood Count

Treatment objectives

To eradicate the fluke and ova
Prevent complications

Drug treatment

Praziquantel

- The schistosomicide with the most attractive combination of effectiveness, broad-spectrum activity and low toxicity

Adult: Single oral dose of 50 mg/kg

Child over 4 years: 20 mg/kg orally, repeated after 4 - 6 hours

- In *S.japonicum* infection, 20 mg/kg 3 times daily for one day after initial dose

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)

URINARYTRACT CALCULI**Introduction**

Occurrence of stone(s) in the kidney, ureter, bladder or urethra

Incidence in Nigeria is 7 - 34 per 100,000

Stones are different with respect to their composition

- Oxalate stones, phosphate stones, uric acid stones and cystine stones

Factors promoting stone formation:

Obstruction to urine outflow
Infection in the urinary tract
Crystallization on foreign bodies
Dehydration
Change in pH
In-born errors of metabolism

Clinical features

Renal and ureteric stones:

Sudden onset loin pain radiating to the groin
Haematuria
Nausea and vomiting

Stones in the bladder:

Frequency
Urgency
Difficulty in passing urine

Stones in the urethra:

Urinary retention

Differential diagnoses

Acute pyelonephritis
Renal tumour
Acute appendicitis
Other causes of urinary obstruction e.g. enlarged prostate, urethral strictures

Complications

Recurrent and intractable urinary tract infection
Secondary hydronephrosis
Progressive renal failure
Periurethral abscess/urethral fistula

Investigations

Urinalysis
Urine culture
Serum calcium, phosphate and albumin
Intravenous urography (IVU)
Ultrasonography
Computerized tomography (non-contrast enhanced)

Treatment objectives

Relieve symptoms
Remove stones
Prevent recurrence

Non-drug treatment

Increased fluid intake
Endoscopic Short Wave Lithotripsy (ESWL)
Endoscopic removal of stones
Open surgical removal

Drug treatment

Analgesics
Antibiotics to treat infections
Drugs used to prevent recurrence:
Thiazide diuretics
- Hydrochlorothiazide 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 circumstances

Complications

Heat stroke in adults

Febrile convulsions in children

Complications associated with underlying cause(s) of fever

Treatment objectives

To lower the temperature

To treat underlying causes

Non-drug treatment

Tepid sponging

Liberal oral sips of water (if clinical state is not a contraindication)

Drug treatment

Paracetamol

Adult: 500 mg - 1 g orally every 4 - 6 hours; maximum 4 g daily

Child: 3 months - 1 year: 60 - 125 mg; 1 - 5 years: 120 - 250 mg; 6 - 12 years: 250 - 500 mg; repeated every 4 - 6 hours if necessary to a maximum of 4 doses in 24 hours

- Infants under 3 months should not be given paracetamol unless advised by a doctor

Aspirin: (acetylsalicylic acid)

Adult: 300 - 900 mg orally (with or without food) every 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**

Aspectrum 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

Dysentery

- 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.monocytogenes 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 sanitary latrines or toilets
 Identify and treat chronic carriers among food handlers
 Hygienic preparation and storage of food
 Ensure that food is cooked at temperatures sufficient to kill bacteria
 Refrigerate food whenever possible
 Encourage exclusive breastfeeding
 Encourage measures to reduce the burden of malnutrition (with its attendant predisposition to severe infections)
 Administer a pentavalent vaccine (A, B, C, D, and E) for persons at high of botulism
 Report new cases to public health authorities

HELMINTHIASIS**Introduction**

Parasitic worm infestations can arise from different groups:

Nematodes (round worms)

Ascaris
 Ancylostoma (hookworm)
 Enterobius (pinworm)
 Trichiuris (whipworm)
Cestodes (flat worms/tapeworms)

- *Taenia solium* and *T. saginata*
Trematodes (flukes)
 - *Schistosoma haematobium* and *S. mansoni*
 Round worm infestations are associated with rural living and poor hygiene

- Prevalent among school children and young adults
 - Acquired through soil and faeco-oral contamination
 Flat worms and tape worms are acquired by eating under-cooked contaminated meat or fish

Bladder worms (*S. haematobium*) are acquired by wading through streams and ponds contaminated with the vector snails

Clinical features

Depend on the infecting helminth:

Ascariasis

Lung phase:

Irritating, non-productive cough
 Burning substernal discomfort, aggravated by coughing or deep inspiration
 Dyspnoea
 Blood-tinged sputum

Intestinal phase:

Usually no symptoms
 Pain
 Features of small bowel obstruction
 Features of perforation
 Intussusception
 Volvulus

Biliary tree occlusion: biliary colic, cholecystitis, cholangitis, pancreatitis, intrahepatic abscess

Effects of migration of an adult worm up the oesophagus:

Coughing
 Oral expulsion of the worm

Hookworm

Most are asymptomatic
 Maculo-papular dermatitis
 Mild transient pneumonitis
 Epigastric pain, often with post-prandial accentuation
 Diarrhoea
 Weakness
 Shortness of breath
 Skin depigmentation

Enterobiasis

Perianal pruritus, worse at night owing to the nocturnal migration of the female worms
 Skin excoriation and bacterial superinfection

Abdominal pain
 Weight loss
 Vulvo-vaginitis
 Pelvic/perineal granulomas

Trichuriasis

Abdominal pain
 Anorexia
 Bloody or mucoid diarrhoea
 Rectal prolapse
 Growth retardation

Strongyloidiasis

Distinguished by its ability to replicate in the human host

- Can thus persist for decades without further exposure of the host to exogenous infective larvae

Recurrent urticaria: buttocks and wrists
 Pruritic raised erythematous skin lesions: advance as rapidly as 10 cm/hour along the course of larval migration

- The pathognomonic serpiginous eruption

Mid-epigastric abdominal pain
 Nausea
 Diarrhoea
 Gastrointestinal bleeding
 Mild chronic colitis
 Weight loss
 Small bowel obstruction

Disseminated strongyloidiasis in patients with unsuspected infection who are given glucocorticoids can be fatal

Trichinellosis

In the first week after infection (gut invasion):

Diarrhoea
 Abdominal Pain
 Constipation
 Nausea
 Vomiting

In the second week after infection (muscle invasion):

Fever
 Periorbital and facial oedema
 Haemorrhages (subconjunctival, retinal and nail bed)
 Maculopapular rash
 Headache
 Cough
 Dyspnoea
 Dysphagia
 Tachyarrhythmias
 Heart failure
 Encephalitis
 Pneumonitis

Schistosomiasis

- See Urology

Differential diagnoses

Other causes of acute-onset diarrhoea and/or vomiting
 -Other conditions depending on the predominant clinical

presentation

Investigations

Stool examination for ova and parasites

Urine examination: microscopy

Haematology: eosinophilia and anaemia may be present

Serology and CT scan may be required in some instances

Drug TreatmentHookworm

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

- Repeat 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

Mebendazole
Adult and child: 100 mg orally every 12 hours for 3 days

Enterobius

Pyrantel embonate
Adult and child: 10 mg/kg orally once

- Repeat dose 2 weeks later; several treatments may be necessary

Trematodes

Praziquantel
Adult: 40 mg/kg given orally at once

- Provides up to 80% cure rates

Child over 4 years: 20 mg/kg followed after 4 - 6 hours 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

Asymptomatic 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
- Pneumocystis carinii infection
- Kaposi sarcoma

Viral infections including cytomegalo virus

Other protozoan infections including

cryptosporidium, cryptocococcus.

- 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 leukoplakia
- 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 syndrome¹
- Pneumocystis 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 (> 1 month) 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 encephalopathy²
- 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			
	CD4	Stage 1	Stage 2	Stage 3	Stage 4
Lymphocytes		Asym.PGL	Early HIV	Intermed. (ARC)	Late AIDS
A	> 2000	1A	2A	3A	4A
B	1000 - 2000	1B	2B	3B	4B
C	< 1000	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, cryptococcosis, 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 triglycerides, 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/mm³
WHO Stage I or II disease with CD4 cell counts ≥ 200/mm³

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/mm³
A TLC of ≥ 1200/mm³ does not predict a CD4 cell count of ≥ 200/mm³ in asymptomatic patients
TLC of ≥ 1200/mm³ may not be used as criterion for the initiation of therapy in asymptomatic patients (WHO Stage I 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/mm³ 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**

d4T or ZDV/3TC/NVP or EFV

TDF/FTC/NVP or EFV

ABC/3TC/NVP or EFV

Second line

TDF/FTC/IDV/r or SQV/r or LPV/r

Or:

ABC/ddI/IDV/r or SQV/r or LPV/r

Or:

ZDV/3TC or ddI/IDV/r or SQV/r or LPV/r

Or:

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**

d4T or ZDV/3TC/NVP or EFV

Or:

ddI/3TC/NVP or EFV

LPV/r requires secure cold chain

All treatment failures at first and second level health facilities should be referred to a paediatric consultant

Second line

d4T or ZDV/3TC/ABC/LPV/r (preferred) or NFV

Or:

ZDV/3TC/LPV/r (preferred) or NFV

Child dosages

- Didanosine (ddI)
- 2 weeks - 8 months: 100 mg/m² orally twice daily
- > 8 months: 120 mg/m² 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)
- 1 mg/kg orally twice daily up to a maximum of 40 mg per dose
- Zalcitabine (ddC)
- Not available
- Zidovudine (ZDV)
- 160 mg/m² orally every 8 hours
- 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/m² twice daily for 14 days, and 200 mg/m² twice daily
- 1 month - 13 years: 120 mg/m² twice daily for 14 days, then 200 mg/m² twice daily
- Indinavir (IDV)

- <4 years: not used
 - 4 - 17 years: 500 mg/m² 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/ritonavir (Lop/r)
 - 7 kg to <15 kg: lopinavir 12 mg/kg, ritonavir 3 mg/kg orally twice daily with food
 - 15 - 40 kg: lopinavir 10 mg/kg, ritonavir 2.5 mg/kg orally twice daily with food
 - >40 kg lopinavir 400 mg, ritonavir 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 (drug rash, eosinophilia and systemic symptoms); manifests as fever, arthralgia, 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
 - Thrombocytopenia
 - Leucocytopenia
- 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
- Aesthesia
- 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)

Chemoprophylaxis-

- 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; occasionally contact with a virus-containing aerosol or the ingestion or transplant of infected tissues may initiate the disease process

Human infection is through contact with unimmunized 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 arrhythmias

Adult Respiratory Distress Syndrome (ARDS)

Gastro Intestinal (GI) bleeding

Thrombocytopenia

Paralytic ileus

Investigations

Full Blood Count and differentials

Urea and Electrolytes

Culture of secretions

Cerebro Spinal Fluid (CSF) analysis

Serology

Pulmonary Chain Reaction (PCR)

Treatment objectives

Disinfect wound; avoid early suturing

Provide passive immunization with antirabies

antisera

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:

- Veterinarians
- 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 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 insensitve and other fluid losses
Enteral or parenteral nutrition
- As determined by clinical situation

Treat intercurrent infections
Notable adverse drug reactions, caution and contraindications
Diazepam is adsorbed from plastics of infusion bags and giving sets; causes drowsiness and light headedness; hypotension
Benzyl penicillin: hypersensitivity reactions
Metronidazole: taste disturbances
Phenobarbital: caution in renal and hepatic impairment
- May cause paradoxical excitement, restlessness and confusion in the elderly; hyperkinesia in children

Prevention
Active immunization of all partially or un-immunized adults, those recovering from tetanus, all pregnant women, infants and un-immunized (missed) children
Health education
Improvement in socio-economic status

TRYPANOSOMIASIS (Sleeping sickness)**Introduction**

African trypanosomiasis is an acute or chronic disease caused by *Trypanosoma brucei* namely
T. brucei rhodesiense (East Africa)
T. brucei gambiense (West Africa)

Clinical features

(Gambian Sleeping Sickness)

Two clinical stages:

Early stage

CNS stage

Early stage:

A nodule or chancre following a bite

Fever

Headache

Dizziness

Weakness

Significant posterior cervical (Winterbottom sign) and supraclavicular lymphadenopathy

Splenomegaly

CNS stage:

Occurs six months to several years later

Characterized by behavioural changes with hallucinations, delusions, and disturbances of sleep with drowsiness during the day and terminating with stupor

Investigations

Peripheral blood film for the detection of trypanosomes

Rapid Card Agglutination Trypanosomiasis Test (CATT) for antibody detection

Diagnosis

Presumptive

- Based on the clinical suspicion and history of exposure to the tsetse fly

A finding of the trypanosome in peripheral blood, lymph node aspirate or CSF is confirmatory

Differential diagnoses

Malaria fever

Meningitis

Viral infections involving the CNS

Treatment**Early stage**

Suramin

Adult and child: 5 mg/kg on day 1, 10 mg/kg on day 3, and 20 mg/kg on days 5, 11, 17, 23 and 30

Late stage

Melarsoprol

Adult: 2.0 - 3.6 mg/kg intravenously in 3 divided doses for 3 days, followed 1 week later with 3.6 mg/kg intravenously in 3 divided doses for 3 days

10 - 21 days later: 3.6 mg/kg intravenously in 3 divided doses for 3 days

Caution

Urine should be examined for casts and protein before and after treatment treatment with suramin

Lumbar puncture follow-up for at least 1 year after treatment with melarsoprol 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 be 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 supra-cervical 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 cavitary pulmonary TB
- May involve the larynx, 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 amplification
- 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*

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 malignancy

- 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

MRI

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

Osteoarthritis

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 inflamed 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 decompensation

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 heterogeneous 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 preceded 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 stiffness

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 orally 3 - 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 every 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/m² once weekly, increased if necessary to a maximum of 25 mg/m² 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)
- Ceftriaxone 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 ciprofloxacin

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, thrombocytopenia, 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-Cardioliipin 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 exacerbations

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/m² once weekly, increased if necessary to a maximum of 25 mg/m² 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/m² 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
- Peritonitis
- Haemorrhage
- 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 sulfadoxine / 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
- Interventions**
- 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 B₁₂
 - 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

Class 1

- No limitation of physical activity

Class 2

- Slight to moderate limitation of physical activity; ordinary day-to-day activities cause dyspnoea

Class 3

- Marked limitation of activity. Minimal exertion causes dyspnoea

Class 4

- Symptoms at rest; unable to carry out any physical activity without dyspnoea; orthopnoea may be present

Other symptoms

Palpitations

Nasal stuffiness

Dizziness; light headedness; syncope

Epigastric or subxiphoid pain; bloating, heartburn

Heat intolerance, sweating and flushing

Signs

Plethoric facies

Oedema (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 S₁ and S₂ 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 septicemia

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-occurrence

- Occurs in 15.6% of cases
- Adequate counselling on the need for early booking, regular antenatal clinic attendance and hospital delivery in subsequent deliveries required

ECTOPIC PREGNANCY

Introduction

Pregnancy in which the conceptus implants either outside the uterus (fallopian tube, ovary or abdominal cavity) or in an abnormal position within the uterus (cornua, cervix, angular and rudimentary horn)

The most common surgical emergency in women in many developing countries

A substantial cause of maternal mortality

- Rapidity with which haemorrhage and shock occur
- Pre-rupture diagnosis is elusive, with consequent delay in surgical management

Clinical features

The clinical subsets include:

- Acute ectopic gestation
- 25% or less of cases
- Sub-acute ectopic gestation
- 75% of cases
- "Silent"/ectopic/chronic ectopic gestation

Acute Ectopic Gestation

Amenorrhoea

Features of acute abdomen particularly lower abdominal pain

- Vaginal bleeding or brownish discharge
- Severe pallor
- Shoulder tip pain
- Difficulty with sitting on hard surfaces

Features of shock with cardiovascular collapse: hypotension and tachycardia

The uterus is slightly enlarged with tenderness on one side

Some 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
- Accidental ovarian cysts

Investigations

- Haemoglobin concentration/packed cell volume
- Blood grouping and cross matching
- Urinalysis
- Ultrasound scan of the pelvis/abdomen
- Serum β -hCG (where available) especially in silent cases
- Paracentesis abdominis (should be considered)
- Laparoscopy
- Final arbiter when the diagnosis is in doubt

Serum β -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 be 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

Cholecystitis

Urinary tract disorders: pyelonephritis

- Acute polyhydramnios
- Commonly associated with monozygotic twinning and diabetic pregnancies
- Pre-eclampsia
- Accidents to ovarian cysts
- Torsion, haemorrhage, infection and rupture
- Red degeneration in a fibroid

Complications

- Biochemical abnormalities
- Usually sequel to vomiting, starvation and dehydration

- Ketosis, electrolyte imbalance (alkalosis and hypokalaemia); vitamin deficiencies

In neglected or poorly managed cases:

- Severe weight loss
- Tachycardia
- Hypotension
- Oliguria
- Neurologic disorders from vitamin B₁ 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
1 st dose at booking or on 1 st contact	Confers no protection
2 nd dose at 4 weeks after 1 st dose	Confers protection for 3years
3 rd dose at 6 months after 2 nd dose	Confers protection for 5years
4 th dose at 1 year after 3 rd dose or in next pregnancy	Confers protection for 10 years
5 th dose at 1 year after 4 th 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; 1 st 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
- Congenital hyperbilirubinaemias such as Dubin-Johnson syndrome
- Liver cirrhosis

Clinical features**Acute yellow atrophy**

- A rare and serious disorder associated with high mortality
- Occurs in the order of 1: 10,000 pregnancies
- Unknown aetiology
- Typically noted in primigravidae, occurring after the 30th week 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 anaesthetist

Definitive treatment

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

Supportive measures

- Transfusion with blood, fresh frozen plasma, platelets as indicated
- 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 36 weeks
- 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 hepatotropic 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°C)
- 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)

Indications for admission

- Uncertain diagnosis
- Intolerance of oral medication or non-response to outpatient therapy
- Presence of a pelvic mass
- Presence of an intrauterine device
- Upper abdominal pain
- Non-adherence to therapy
- Pregnancy
- Nulliparity

Treatment objectives

- Rehydrate adequately
- Eradicate the infecting organism(s)
- Prevent complications

Drug treatment

- Appropriate antibiotics for an adequate period
- The antibiotic chosen should cover all possible causative organisms while awaiting culture/sensitivity results

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

Inpatient triple therapy

- Ceftriaxone/doxycycline/metronidazole

Or:

- Clindamycin/gentamicin/metronidazole

Triple antibiotic regimen to be continued for 48 hours after the patient improves clinically

Subsequently, the patient should continue therapy with

- Doxycycline
- 100 mg orally every 12 hours

Plus:

- Metronidazole
- 400 mg orally every 8 hours for 10-14 days

Prevention

- Encourage the use of barrier contraceptive with spermicides
- Modify risky sexual behaviour: avoid multiplicity of sexual partners
- Contact tracing: to break the existing chain of infection and prevent recurrence
- Prompt diagnosis and treatment to prevent long term complications

RAPE

Introduction

Performance of the act of sexual intercourse by force,

duress, intimidation or without legal consent (as with a minor)

A growing social disorder afflicting the poor and rich, alike, with devastating and longstanding emotional consequences for the afflicted, family and society at large

An enormous societal problem that appears to be poorly recognized and grossly under-reported

An average of one in five adult women may have experienced sexual assault during her lifetime

Adult women are much more likely to be raped by a spouse, ex-spouse, or acquaintance than by a stranger

The girl-child is much more likely to be raped by her close male associates (non-strangers), not excluding her father, uncle, brother, cousin, neighbour, school teacher, family driver, security personnel, and even faith-based instructor

Mental illness, alcohol and drug abuse appear to be predisposing factors; neglect and inattentiveness to the needs of the girl-child also contribute

Clinical features

Indirect presentation

Vague symptoms

Physical features:

- Perineal pain
- Bleeding per vaginam
- Bruised face/body
- Arthritis
- Disordered gait
- Psychological symptoms/disorders

- Sadness

- Depression

- Refusal to respond to simple questions

- Avoidance of eye contact

- School/work absenteeism

Differential diagnoses

Vaginitis

Threatened abortion

Domestic violence

Alcoholism

Drug abuse

Depression

Investigations

Early

Vaginal/perineal swab for microscopy, culture and sensitivity

Semen: DNA analysis

Late

Urinalysis; urine microscopy, culture and sensitivity

Pregnancy test (blood)

HIV screening

Treatment objectives

Evaluate safety of the patient

Assess and treat physical injuries

Provide emotional support

Assess and deal with the risk of sexually transmitted

infections and pregnancy

It is important to document clinical findings

Non-drug measures

Reassure patient

Provide information about legal services

Drug treatment

Treat physical injury (as appropriate)

Treat STIs, UTI (as appropriate)

Treat HIV infection (if detected); Post-exposure prophylaxis if clinical situation so requires

- See section on HIV infection

Manage pregnancy (as appropriate)

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
- Droling
- 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, adjusted according to response up to 300 micrograms/kg daily especially in emergencies
- Give parenterally in more severe cases
- May repeat dose after 12 hours if necessary

Caution

Effects of nebulized epinephrine last 2 - 3 hours; the child should be monitored carefully for recurrence of the obstruction

ACUTE RHINITIS (Common cold)**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 β_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 β_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 preparations

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

Purulent fetid sputum

Haemoptysis

Pleuritic chest pain

With or without a history of preceding pneumonic illness

Digital clubbing.

Crepitations, rhonchi and wheezes

Cor pulmonale 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 pulmonale 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 preparations

Salbutamol: palpitations, tremors, nervous tension, muscle cramps, sleep disturbances, tachycardia, peripheral vasodilation, hypotension

Prevention

Avoidance of smoking

Timely and effective treatment of bacterial infections

Respiratory care during childhood measles

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

Aesthetic 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 pneumoniae*
 Cor pulmonale
 Left ventricular failure
 Pulmonary thromboembolism

Investigations
 Chest radiograph: hyperinflation, pulmonary hypertension
 Ventricular function tests: FEV₁/FVC ratio
 Blood gas analysis
 Blood pH
 Haematocrit
 Sputum microscopy and culture (during symptom exacerbation)
 Electrocardiogram
 Airways reversibility test

Treatment objectives
 Maintain optimal level of oxygenation and ventilation
 - Supplemental oxygen, at 24 - 28% or 1 - 2 litres/minute
 Treat infections
 Reverse airways obstruction
 Clear airways secretions

Drug treatment
 Long acting β_2 - agonist
 - See bronchial asthma
 Theophylline
 - Aminophylline (see bronchial asthma)
 Antibiotics (when necessary to control infection)
 - Erythromycin
Adult 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
 - Bronchiectasis
 - 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 reactions, caution

Codeine cough linctus: sedation, constipation

DYSPNOEA**Introduction**

An abnormal and uncomfortable awareness of breathing

Effort of breathing is out of proportion with exertion needs

Patients often have difficulties in describing the discomfort of dyspnoea

Clinical features

Will depend on the underlying cause(s) of dyspnoea

Differential diagnoses

Pulmonary:

- Obstructive airways disease: asthma, chronic bronchitis, emphysema

- Parenchymal lung disease: pneumonia, pneumoconiosis, pulmonary fibrosis

- Pulmonary vascular obstruction: pulmonary emboli
 - Chest wall disorders: respiratory muscle paralysis, kyphoscoliosis

Cardiogenic:

- Congestive cardiac failure

- Left ventricular failure

Metabolic:

- Diabetic ketoacidosis

Neurogenic:

- Anxiety neurosis

Treatment objectives

Treat cause(s) of dyspnoea

Restore normal respiration

Non-drug treatment

Oxygen in appropriate concentration

Other treatment will depend on 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

Pleuritic 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

Pleuritic 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

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 faeces 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 *Enterobacteriaceae*

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

Viperidae and crotalidae

- Local and systemic bleeding
- Impairment of organ function
- Reduction of cardiac output

- Inhibition of peripheral nerve impulses
- Multisystem effects
- Rhabdomyolysis
- Haemolysis
- Blood vessel damage

Elapidae

Neurotoxic effects

Snake bite wounds may become secondarily infected with:

- *Clostridium tetani*, causing tetanus
- *Clostridium welchi*, causing gas gangrene

Indications for antivenom treatment

- 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 assessment

Careful history

Contact local authorities to determine if the specie is rabid; if possible locate animal for observation

Antibiotic allergy, immunization of patient and other morbid condition(s) should be documented

Inspect wound for evidence of infection.

Conduct general physical examination, including vital signs

Investigations

Depend on the type of injury, the clinical presentation and the onset/type of complications:

- Full Blood Count
- Electrolytes and Urea
- Blood clotting profile
- Arterial blood gas estimations
- Chest radiographs
- Wound and blood cultures

Treatment objectives

- Neutralize envenomation
- Limit systemic effects
- Local wound care
- Prevent onset of complications

Prevent specific infections such as rabies in high risk cases

Non-drug measures

- Limb splinting (and rest the limb)
- Use of venom detection kit (if available)
- Application of pressure bandage
- Control/care of the airway

Incision is discouraged; the mouth should not be used to suction

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 crystalloids as clinically appropriate

Treatment of anaphylaxis with antihistamines (H₁ 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:

- Airway
- Breathing
- Circulation
- Disability
- Exposure

Correct problems identified

Give patient 100% oxygen

Pass an endotracheal tube if there is risk of airway obstruction

Obtain specimens for investigations as detailed above

Determine percentage total body surface area (TBSA) burned

- Wallace rule of nines is recommended in adults

- In children there are several charts e. g Lund and Browder charts

Calculate the total fluid requirement in the first 24 hours using appropriate formulae

- We recommend the Parkland's

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

H₂ 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 H₂O)

- 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 pupillary dilatation and contralateral hemiparesis or hemiplegia

In the Kernohan's notch syndrome which occasionally occurs there is ipsilateral pupillary 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, arrhythmias, 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 (raccoon 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 manoeuvres 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

Assess the GCS and the state of the pupils

Expose the patient to perform a quick general examination but avoid hypothermia.

Secondary Survey:

(See section on multiple injuries)

Secondary brain injury

Neuronal injury that is not present at the time of the primary insult but develops in response to subsequent intracranial or extracranial events

Extracranial causes:

- Hypoxia
- Hypotension
- Seizures
- Hyperthermia
- Hyponatraemia
- Hypernatraemia
- Hypoglycaemia
- Hyperglycaemia

Intracranial causes:

- Extradural haematoma
- Subdural haematoma
- Intracerebral haematoma
- Cerebral oedema
- Cerebral contusion
- Hydrocephalus
- Meningitis
- Brain abscess

CT scan in TBI

Has revolutionized the management of traumatic brain injury as it can readily diagnose intracranial

haematomas and skull fractures

In trauma it is advisable to do a non-contrast CT scan

Indications for CT scan

- GCS of 14 or less
- GCS of 15 with:
 - Loss of consciousness > 5 minutes
 - Amnesia for injury
 - Focal neurological deficit
 - Signs of calvarial or basal skull fracture

Intracranial pressure monitoring

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

Indications for ICP monitoring in TBI

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

Patients with intracranial haematomas but are adjudged not to need surgery

Emergency management of raised intracranial pressure

- Endotracheal intubation
- Controlled ventilation to a pCO₂ 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

Surgery in TBI

Often indicated in head injury for the evacuation of intracranial haematomas or elevation of depressed skull fractures

Indications may depend on the centre and the neurosurgeon, but all agree that an intracranial haematoma causing significant mass effect should be removed

A midline shift of more than 5 mm is considered significant

Indications for surgery will depend on:

- The neurological status of the patient
- Findings on CT
- Extent of intracranial injury
- Intracranial pressure.

The procedures include:

- Burr holes
- Craniotomy
- Craniectomy
- Elevation of depressed skull fractures

Drugs in TBI

Diuretics to reduce intracranial pressure e.g. mannitol (see Meningitis)

Sedatives e.g. diazepam (see Tetanus)

Muscle relaxants e.g. diazepam, suxamethonium

Anticonvulsants e.g. phenytoin, phenobarbital (see Epilepsy)

Antibiotics as appropriate

Vasopressors e.g. noradrenaline, dobutamine if there is hypotension, and in collaboration with a physician

Prevention

Measures aimed at reducing accidents in transportation (especially road traffic accidents), in homes and in factories:

- Motorbike crash helmet laws and enforcement
- Alcohol laws
- Speed limits
- Better motor licensing rules
- Health education
- Better motor engineering
- Good road designs
- Safety procedures at work and a good EMS and trauma system

MULTIPLE INJURIES**Introduction**

The multiply injured patient is that patient with injury to more than one organ system

Often victims of motor vehicle crashes, motor bike accidents, pedestrians hit by cars, or falls from heights

Present a challenge to the managing team in terms of priority of medical intervention

- If the priorities are not well ordered the results can be catastrophic

Difficult to outline clinical features for these patients as virtually any injury is possible

Treatment objectives

Identify life threatening injuries and treat

Identify all injuries, institute primary management and limit progress of injuries and further tissue damage

Restore patient's physiology paying special attention to the triad of **hypothermia, acidosis** 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 manoeuvres 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, otorrhoea); 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 boggy 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 occurrence 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 reinstated 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 conjunction with the physician and anaesthetist)

Haematological:

- Correct anaemia
- Cause(s) of anaemia should be identified and treated
- The minimum haemogram for a patient undergoing elective surgery should be 10 g/dL
- Haemogram 6 - 9 g/dL: correction may be achieved by haematinics; reschedule surgery
- Haemogram <6 g/dL: correction may require blood transfusion
- Emergency surgery: correct anaemia by blood transfusion

Blood transfusion should be avoided as much as practicable.

- Patient with sickle cell anaemia: haemogram should be brought up to 8 g/dL
- These patients must be adequately hydrated to avoid sickling and sludging within the bloodstream
- Short day case procedure: imperative to admit the patient with sickle cell anaemia at least a day before surgery to achieve adequate hydration
- Suspected bleeding diathesis
- Intramuscular vitamin K (10 mg daily), at least 48 - 72 hours before surgery
- For major surgery, blood should be grouped, cross-matched and stored

Other disorders:

Any associated medical condition should be treated / controlled before embarking on surgery

- This should be done in conjunction with the physician as much as possible

Patients who require nutritional rehabilitation

- If surgery is elective reschedule it, and give adequate time to achieve improved nutritional status, otherwise morbidity and mortality may be increased

High- risk patients:

- At high risk of developing postoperative complications
- Deliberate and meticulous efforts should always be made to adequately evaluate them and ensure optimal fitness for surgery
- Elderly patients (age >60 years): - risk of deep vein thrombosis, atelectasis
- Obesity- risk of deep vein thrombosis, atelectasis
- Cancer-risk of deep vein thrombosis, atelectasis, haemorrhage
- Women on oral contraceptive pills-risk of deep vein thrombosis
- Co-existing chronic medical conditions-risk of wide ranging complications
- Sickle cell anaemia-risk of sickling crises, deep vein thrombosis

Consent for surgery

Details of the surgery should always be explained to the patient (or relatives) in very simple language before surgery

Should include a mention of the possible/common complications

A signed consent should be obtained, in the presence of a witness (usually a nurse)

Obtaining consent should be done by the surgeon himself

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 1 year
- 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 5 days 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/mm³ (primarily lymphocytes)

- Mild increase in protein: 40 - 50 mg/mL

Treatment objectives

Allay fear

Minimize ensuing skeletal deformities

Anticipate and treat complications

Prepare the child and family for a prolonged management of permanent disability if it seems likely

Non-drug treatment

Bed rest

Avoidance of exertion

Application of hot packs

Lying on a firm bed

Hospitalization for those with paralytic disease

Suitable body alignment to avoid excessive skeletal deformity

Active and passive motions as soon as pain disappears

Manual compression of the bladder

Adequate dietary and fluid intake

Review by orthopaedist and psychiatrist

Gravity drainage of accumulated secretions

Tracheostomy in case of vocal cord paralysis

Drug treatment

Bethanicol 5 - 10 mg orally or 2.5 - 5 mg subcutaneously for bladder paralysis

Analgesics

- Avoid opiates if there is impairment of ventilation

Treat urinary tract infection with appropriate antibiotics

Prevention

Hygienic practices

- To prevent / limit contamination of food and water by the virus

Vaccination

- The only effective method of prevention

Oral Polio Vaccine

Given at:

Birth

6 weeks

10 weeks

14 weeks

- Highly effective

- 50% immune after 1 dose

- >95% immune after 3 doses

- Confers herd immunity
 - Immunity probably life long
 - Limits spread of wild polio virus
- Inactivated Polio Vaccine**
- Given at:
- 2 months
 - 4 months
 - 12 months
- Highly effective
 - >90% immune after 2 doses
 - >99% immune after 3 doses
 - Duration of immunity not known with certainty
- Notable adverse drug reactions, caution and contraindications**
- Oral polio vaccine:
 - Paralytic poliomyelitis
 - Should not be administered to persons who are immunocompromised (it is a live vaccine)
 - Contra indicated in:
 - Persons with history of severe allergic reaction to a vaccine component or following prior dose
 - Moderate or severe acute illness
- Inactivated vaccine may be used in 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)

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
- Orthopnoea
- Paroxysmal nocturnal dyspnoea
- Cough
- Haemoptysis
- 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 1 or 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 caucasians

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 antiarrhythmic 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 NaHCO₃ 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 Arrest

Or:

- Parenteral/nebulised salbutamol (see Bronchial asthma)
- Removal of potassium with diuretics (loop plus thiazide diuretics in combination)

- Sodium polystyrene 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) \times 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 polydipsia

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 repletion 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 ischaemia, left ventricular failure)

Renal deterioration

Fundoscopy 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

Fundoscopy 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 nitroprusside

- 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- β 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 neuroglycopenia 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

CO₂ 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

T₃, T₄ 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, arrhythmias 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
Arrhythmias
Hyperthermia

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 T₃ 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 Airway

B Ensure adequate Breathing and ventilation

C Ensure adequate Circulation

D Address Drug-induced depression of the central nervous and respiratory systems

E Correct any Electrolyte 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, hypnotosedatives, 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 P₄₅₀ 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 glutathione reserve

- Acute starvation
- Alcoholism
- Childhood
- Chronic malnutrition

Clinical features

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

- Nausea and vomiting
- Excessive sweating
- Onset of hepatotoxicity is heralded by right upper quadrant 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 1 g 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 NaHCO₃ 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 diazepam and bromazepam
These drugs potentiate the inhibitory effect of GABA on

CNS neurons

Clinical features

Mainly CNS depression occurring 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:

Pralidoxime

- 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

Essential elements of a prescription order

Identity of prescriber:

- Name

- Address/institution of prescriber

- Telephone number

Date of prescription:

- Near top/beginning of left margin of a chart order

Identity of patient:

- Name

- Age (especially in children)
- Gender
- Address of patient
- Hospital number
- Elements specifying medication:
- Name of medication (*generic name*)
- Strength (*metric units*) and quantity
- Dosage
- Frequency
- Duration
- Directions for use (drug- and patient- specific)
- Refill instructions
- Waiver of requirements for child-proof containers
- Additional labelling instructions
- Prescriber's signature and other identification data e.g code. Prescriptions may be hand written or computer-issued:
- Hand written prescriptions should be written in indelible ink and the hand writing should be legible (important, to avoid medication errors)
- Any alteration(s) made in a computer-issued prescription should be duly endorsed

Abbreviations

- Only standard, official abbreviations should be used. The following are some notable abbreviations
- a.c ante cibum (before food)
 b.d bis die (twice daily)
 o.d omni die (every day)
 o.m omni mane (every morning)
 p.c post cibum (after food)
 p.r.n pro re nata (when required)
 q.d.s quarter die sumendum (to be taken four times daily)
 q.q.h quarter quaque hora (every four hours)
 stat immediately
 t.d.s ter die sumendum (to be taken three times daily)
 t.i.d ter in die (three times daily)

NOTE

- Avoid abbreviations of drug names
- Doses should be written in the metric system or in international units (IU) when metric doses are not practicable
- If a drug is to be administered 'as required', specify the minimum dose interval and the total amount of drug to be administered
- Avoid unnecessary use of decimal points
 1 mg not 1.0 mg
 If >1 g state as g
 If <1 g state as milligram e.g. 500 mg not 0.5 g
 If <1 mg state as microgram: 100 microgram not 0.1 mg
 If the decimal point is unavoidable, insert zero (0) in front of the point e.g 0.5 mL not .5 mL
 Microgram and nanogram should not be abbreviated
 Millilitre (mL) should be used for volume and not cubic centimetre, c.c or cm³

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 bromocriptine 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 physiological 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. Dracunculosis
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 cryptococcosis (including meningitis)
HIV encephalopathy
Disseminated endemic mycosis (extrapulmonary histoplasmosis, 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: <input type="text"/>
Address: _____	Telephone: _____
INSTRUCTIONS: Answer all of the following questions by placing a cross mark in the corresponding box.	
OBSTETRIC HISTORY	
1. Previous stillbirth or neonatal loss?	No <input type="checkbox"/> Yes <input checked="" type="checkbox"/>
2. History of 3 or more consecutive spontaneous abortions?	No <input type="checkbox"/> Yes <input checked="" type="checkbox"/>
3. Birthweight of last baby < 2500g?	No <input type="checkbox"/> Yes <input checked="" type="checkbox"/>
4. Birthweight of last baby > 4500g?	No <input type="checkbox"/> Yes <input checked="" type="checkbox"/>
5. Last pregnancy: hospital admission for hypertension or pre-eclampsia/eclampsia?	No <input type="checkbox"/> Yes <input checked="" type="checkbox"/>
6. Previous surgery on reproductive tract? (Myomectomy, removal of septum, cone biopsy, classical CS, cervical cerclage)	No <input type="checkbox"/> Yes <input checked="" type="checkbox"/>
CURRENT PREGNANCY	
7. Diagnosed or suspected multiple pregnancy?	No <input type="checkbox"/> Yes <input checked="" type="checkbox"/>
8. Age less than 16 years?	No <input type="checkbox"/> Yes <input checked="" type="checkbox"/>
9. Age more than 40 years?	No <input type="checkbox"/> Yes <input checked="" type="checkbox"/>
10. Isoimmunization Rh (-) in current or in previous pregnancy?	No <input type="checkbox"/> Yes <input checked="" type="checkbox"/>
11. Vaginal bleeding?	No <input type="checkbox"/> Yes <input checked="" type="checkbox"/>
12. Pelvic mass?	No <input type="checkbox"/> Yes <input checked="" type="checkbox"/>
13. Diastolic blood pressure 90mm Hg or more at booking?	No <input type="checkbox"/> Yes <input checked="" type="checkbox"/>
GENERAL MEDICAL	
14. Insulin-dependent diabetes mellitus?	No <input type="checkbox"/> Yes <input checked="" type="checkbox"/>
15. Renal disease?	No <input type="checkbox"/> Yes <input checked="" type="checkbox"/>
16. Cardiac disease?	No <input type="checkbox"/> Yes <input checked="" type="checkbox"/>
17. Known 'substance' abuse (including heavy alcohol drinking)?	No <input type="checkbox"/> Yes <input checked="" type="checkbox"/>
18. Any other severe medical disease or condition?	No <input type="checkbox"/> Yes <input checked="" type="checkbox"/>
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 for calculation of paediatric doses- Many physiological phenomena correlate better to BSA

For most medicines the adult maximum dose should not exceeded

For example if the dose is stated as 4 mg/kg (max. 180 mg), a child weighing 10 kg should receive 40 mg but a child weighing 50 kg should receive 180 mg and not 200mg

Young children may require higher doses per kilogram than adults because of their higher metabolic rate

Calculation by body weight in an overweight child may result in much higher doses being administered than necessary. Such doses should be calculated based on ideal body weight in relation to height and age.

See table below.

Age	Ideal body-weight		Height		Body Surface /m ²
	Kg	lb	cm	Inch	
Newborn*	3.5	7.7	50	20	0.23
1 Month*	4.2	9	55	22	0.26
3 Month*	5.6	12	59	23	0.32
6 Month	7.7	17	67	26	0.40
1 year	10	22	76	30	0.47
3 years	15	33	94	37	0.62
5 years	18	40	108	42	0.73
7 years	23	51	120	47	0.88
12 years	39	86	148	58	1.25

* The figures relate to full term and not preterm infants who may need reduced dosage according to their clinical condition

APPENDIX IV:

MEDICINES WITH TERATOGENIC POTENTIAL

Medicine	Comment
Antiepileptics	Risk of teratogenicity greater if more than one medicine used
Bleomycin	Avoid (teratogenic and carcinogenic in animal studies)
Busulfan	Avoid (teratogenic in animals)
Carboplatin	Avoid (teratogenic and embryotoxic in animal studies)
Cisplatin	Avoid (teratogenic and embryotoxic in animal studies)
Co-trimoxazole	Teratogenic risk (trimethoprim -a folate antagonist)
Cytarabine	Avoid (teratogenic in animal studies)
Dactinomycin	Avoid (teratogenic in animal studies)
Daunorubicin	Avoid (teratogenic and carcinogenic in animal studies)
Doxorubicin	Avoid (teratogenic and toxic in animal studies)
Sulfadoxine/pyrimethamine	Possible teratogenic risk (pyrimethamine is a folate antagonist)
Griseofulvin	Avoid (fetotoxicity and teratogenicity in animal studies)
Hydroxocarbamide(hydroxyurea)	Avoid (teratogenic in animal studies)
Idoxuridine	Teratogenic in animal studies
Isotretinoin	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
Amityptilline	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

National Agency for Food and
Drug Administration & Control
(NAFDAC), Headquarters Office
Plot 2032 Olusegun Obasanjo Way
Wuse Zone 7 Abuja



FORM FOR REPORTING OF
SUSPECTED ADVERSE DRUG
REACTIONS

IN STRICT CONFIDENCE

Tel: 08086899571 or Fax: 09-5241108

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	C. OUTCOME OF REACTION TICK AS APPROPRIATE
DATE Reaction Started _____ DATE Reaction Stopped _____	<input type="checkbox"/> Recovered fully <input type="checkbox"/> Recovered with disability (Specify) _____ <input type="checkbox"/> Congenital Abnormality (Specify) _____ <input type="checkbox"/> Life Threatening (Specify) _____ <input type="checkbox"/> Death _____ <input type="checkbox"/> Others (specify) _____

B. Was Patient Admitted Due to ADR

Yes No

If Already Hospitalized, Was it Prolonged Due to ADR Yes <input type="checkbox"/> No <input type="checkbox"/>			
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

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