

standard treatment guidelines

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FOREWORD

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

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

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

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

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

Dr. Hassan Muhammed Lawal, CON
Supervising Minister of Health

PREFACE

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

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

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

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

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

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

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

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

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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

- T₃ 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

Hypopyon

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

Anti-inflammatory 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 affectionation 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

Epidemic keratoconjunctivitis

Antibiotic drops to prevent secondary bacterial infection

- Chloramphenicol 0.5% drops

Adult and child over 2 years: apply every 4 hours for no more than 5 days

Ophthalmia Neonatorum

- Gentamicin sulfate 0.3% applied as stated above

Or:

- Ofloxacin 0.3% solution applied as stated above

Plus:

- 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 staphylooma

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, haematuria

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)