



CHAPTER 18

EYE DISORDERS

18.4 HERPES ZOSTER OPHTHALMICUS

B02.3+

DESCRIPTION

Herpes zoster ophthalmicus occurs when the varicella-zoster virus reactivates in the trigeminal ganglion and passes down the ophthalmic division of the trigeminal nerve. Patients present with a vesicular rash on the forehead, upper lid and side of the nose. A minority of patients may develop conjunctivitis, keratitis, uveitis, retinitis and cranial-nerve palsies. Permanent sequelae of ophthalmic zoster infection may include chronic ocular inflammation, loss of vision, and debilitating pain. Patients < 50 years old should be offered HIV testing.

DRUG TREATMENT

- aciclovir, oral, 800 mg five times daily for 7–10 days
- chloramphenicol 1%, ophthalmic ointment, apply four times daily to affected eye

For neuralgic pain:

- amitriptyline, oral, 25 mg at night for 3 months

For skin lesions:

- potassium permanganate. 1:10 000 aqueous solution, topical, cleanse twice daily
- PLUS**
- silver sulphadiazine, topical, apply twice daily after cleansing

Follow patient weekly until skin lesions healed.

Best results are obtained if treatment is initiated within the first three days of onset of symptoms.

REFERRAL

- fluorescein uptake by the cornea (keratitis)
- decreased vision, i.e. a 2 line fall off in snellen acuity in affected eye compared to healthy eye
- afferent pupil defect
- signs of uveitis

18.5 KERATITIS

H16

18.5.1 KERATITIS, HERPES SIMPLEX

H13.1*

DESCRIPTION

Associated features: previous history often, decreased corneal sensation.
Morphology: dendritic ulcer seen on staining with fluorescein.



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DRUG TREATMENT

- aciclovir ophthalmic ointment inserted in the lower cul-de-sac five times per day at four hour intervals
Continue for 3 days after ulcer has healed.

Note:

Topical corticosteroids are contraindicated in the treatment of dendritic ulcers. In other settings topical corticosteroids may be used only by personnel with experience in ophthalmology and with access to both a tonometer and a slit lamp.

18.5.2 KERATITIS, SUPPURATIVE

H16.0

DESCRIPTION

Painful red eye with corneal lesion that stains with fluorescein and has creamy white appearance. If RVD + or history of injury to eye with plant matter, need high index of suspicion for fungal infection.

DRUG TREATMENT

Treat only if access to slit lamp, otherwise refer.

Scrape ulcer for microscopy, culture and sensitivity and modify treatment accordingly.

- ciprofloxacin, ophthalmic drops, instill 1 drop hourly for 3 days then reduce frequency to 1 drop 3–4 hourly
OR
ofloxacin, ophthalmic drops, instill 1 drop hourly for 3 days then reduce frequency to 1 drop 3–4 hourly

If gram positive cocci:

ADD

- vancomycin 25 mg/mL, topical

If fungal infection, change to:

- natamycin 5%, ophthalmic drops, instill 1 drop 1–2 hourly, initially
After 3–4 days reduce frequency to 1 drop 3–4 hourly.
Continue for 14–21 days until resolution of infection.

REFERRAL

- no access to slit lamp
- no facilities for microscopy, culture and sensitivity



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18.6 RETINITIS, HIV CMV

h30.9

DESCRIPTION

CMV retinitis is seen in advanced HIV, with CD 4 count < 100. The characteristic appearance is necrosis, i.e. white exudates, and hemorrhages at the edges of the exudates. Irreversible blindness occurs once the optic disk is involved.

DRUG TREATMENT

- ganciclovir, intravitreal, 200 mcg once a week
Once immune function has been restored with antiretroviral therapy, i.e. CD4 >100, maintenance ganciclovir can be stopped but monitor for recurrence.

18.7 UVEITIS

H20.0

DESCRIPTION

An inflammation of the uveal tract and adjacent structures. The commonest form is acute anterior uveitis, which presents with pain and photophobia, brow ache, loss of vision, circumciliary injection and a miotic pupil. Chronic uveitis may lead to cystoid macular oedema with decreased central acuity, cataract formation and secondary glaucoma. Numerous systemic diseases can cause uveitis.

This condition should be managed at an ophthalmology unit.

DRUG TREATMENT

Cycloplegic agents, e.g.:

- homatropine 5%, ophthalmic drops, instill 1–2 drops 3–4 hourly

AND

Corticosteroids, e.g.:

- prednisolone 1%, ophthalmic drops, instill 1–2 drops 4 times daily



CHAPTER 19 POISONING

19.1 ENVENOMATION

19.1.1 INSECT BITES AND STINGS

T63

DESCRIPTION

Insect bites and stings usually have local effects. Systemic effects are rare. Local inflammatory or systemic/immunological forms of toxicity are encountered, which may vary between minor local reactions and acute anaphylaxis.

Multiple bee stings may require ICU care.

NON-DRUG TREATMENT

Observe patient for sufficient period of time, as a rapid worsening in the condition may occur and the effects of treatment may be transient.

DRUG TREATMENT

ANAPHYLAXIS

See Section 20.1: Anaphylaxis/Anaphylactic Shock.

19.1.2 SNAKEBITES

T63.0

NON-DRUG TREATMENT

Minimise movement of affected limb.

Emergency treatment by bandaging affected limb with a crepe bandage without compromising blood supply.

Observe patient closely for a period of 24 hours after contact.

Do not apply a tourniquet.

Sucking or cutting the wound has not been found to be of any benefit.

COBRA AND MAMBA

Application of a tight crepe bandage proximal to the bite-site may be life saving.

The venom of cobras and mambas bites is predominantly neurotoxic. Ventilatory and cardiovascular support may be needed in an ICU.

If venom occurs in the eyes, irrigate extensively with copious amounts of water for 15–20 minutes.

BOOMSLANG OR BERGADDER

The venom of boomslang is haemotoxic.

Observe for 36 hours.

VIPERS AND ADDERS

The venom of vipers and adders is predominantly cytotoxic.

Crepe bandaging is inappropriate in all cases of cytotoxic snakebites.

Surgical intervention, i.e. decompression surgery for established compartment syndrome and debridement of necrotic tissue should be done only when absolutely necessary and as conservatively as possible.

DRUG TREATMENT

Cleanse wound:

- chlorhexidine 0.05% in water

Secondary infection:

- amoxicillin/clavulanic acid, oral, 375 mg 8 hourly for 5 days

Immunisation, primary or booster:

- tetanus toxoid vaccine, IM, 0.5 mL immediately

In unimmunised or partially immunised patients:

- tetanus immunoglobulin, human, IM, 250 units

Intravenous Fluids

Reverse circulatory shock, if present.

Analgesia

For mild pain:

- paracetamol, oral, 1 g 4–6 hourly as needed

OR

For severe pain:

Opioids, e.g.:

- morphine, slow IV, 3–10 mg in increments of 2 mg
Dilute morphine to 10 mL with water for injection or sodium chloride 0.9%.
Beware of respiratory arrest and hypotension when administering high dose morphine intravenously.
Opioids should be used cautiously in neurotoxic snakebite.

The use of an NSAID is not recommended due to the potential danger of renal failure in a hypotensive patient.

Polyvalent Antivenom

Obtainable from SA Vaccine Producers - See full details in package insert.

Effective against the venom of:

- puff adder
- gaboon adder
- rinkhals
- Cape cobra
- Egyptian cobra
- black necked spitting cobra
- forest cobra
- black and green mamba

It is ineffective against the venom of:

- night and berg adder and other minor adders
- boomslang
- vine and twig snakes

Never administer antivenom without being fully prepared to manage acute anaphylaxis.

Note:

- in most cases patients do not need and should not be given antivenom
- the dose of antivenom is the same for adults and children
- serum sickness is relatively common, especially after administration of large doses of antivenom

Criteria for antivenom administration:

- all patients with confirmed mamba bites should receive antivenom, even before onset of symptoms
- patients with confirmed gaboon adder or puff adder bites should receive antivenom at the onset of any symptoms

Signs of systemic poisoning:

- muscle weakness and/or difficulty in breathing
- difficulty in swallowing
- weakness
- double vision
- drooping eyelids
- spreading of local tissue damage
- swelling of a hand or foot within 1 hour of a bite (the majority of bites occur on the hands or feet)
- swelling extends to the elbows or knees within 4 hours of a bite
- swelling of the groin or chest at any time or if actively advancing
- significant swelling of head or neck

- polyvalent snake antivenom, slow IV infusion
Dilute 100 mL in 300 mL sodium chloride 0.9%.
Administer slowly for the first 15 minutes, as most allergic reactions will occur within this period.
Increase the flow rate gradually until the infusion is completed within one hour.
Repeat if there is no clinical improvement after the infusion.

Black mamba bites to reverse respiratory paralysis:

- polyvalent snake antivenom, slow IV infusion, 200 mL or more may be required

To prevent airway obstruction in swelling of head or neck (cytotoxic bites):

- polyvalent snake antivenom, slow IV infusion, 500 mL

Difficulty in breathing with muscle weakness:

- polyvalent snake antivenom, slow IV infusion, 100 mL. May be repeated - See package insert.

BOOMSLANG POISONING

Envenomation rarely occurs, causing disseminated intravascular coagulation usually a few days later but has occurred within hours in isolated cases.

In suspected boomslang bite a whole blood clotting time is a useful bedside test, especially in rural areas. Place 5 mL of blood in a dry test tube and leave at room temperature for 20 minutes. Normal clotting time varies from 5–20 minutes.

The initial blood clotting parameters may be normal.

It is important to follow these over a few days.

Special investigations include FBC, activated PTT, Prothrombin time (INR), fibrinogen, D-dimer and monomers.

Correct haematological abnormalities.

Boomslang antivenom

Obtainable from SA Vaccine Producers - See full details in package insert.

Do not administer polyvalent antivenom.

Never administer antivenom without being fully prepared to manage acute anaphylaxis.

- boomslang antivenom, slow IV infusion, 20 mL diluted in 50–100 mL sodium chloride 0.9% or dextrose 5%, administered over 5–10 minutes

19.1.3 SCORPION ENVENOMATION

T63.2

DESCRIPTION

Poisonous scorpions in Southern Africa are of the Genus *Parabuthus*. Features useful in its identification are a relatively large tail and small pincers.

The venom typically causes immediate pain, followed by systemic symptoms within 1–4 hours. These include:

- general paraesthesias
- muscle cramps
- pain
- excessive sympathetic stimulation

Dysphagia, dysarthria and loss of pharyngeal reflexes with possibly respiratory impairment may be encountered.

NON-DRUG TREATMENT

Observe all cases for 12–24 hours.

Ventilatory support may be required in severe cases.

DRUG TREATMENT

Routine antivenom therapy is recommended only in severe cases with systemic signs.

- scorpion antivenom, slow IV, 10 mL administered over 3–5 minutes

OR

scorpion antivenom, IV infusion, 10 mL diluted in 50–100 mL sodium chloride 0.9% or dextrose 5%, administered over 5–10 minutes

Never administer antivenom without being fully prepared to manage acute anaphylaxis.

Immunisation, primary or booster:

- tetanus toxoid vaccine, IM, 0.5 mL immediately

In unimmunised or partially immunised patients:

- tetanus immunoglobulin, human, IM, 250 units

Intravenous Fluids

Reverse circulatory shock, if present.

Monitor response.

Analgesia

Analgesics such as paracetamol and opiates have not been shown to be effective.

“Ring block”

- lidocaine (lignocaine) 1–2%, 2 mL injected around the bite as local anaesthetic

Severe muscle pain and cramps:

- calcium gluconate 10%, bolus IV infusion, 10 mL over 10 minutes
Followed by 60–120 mL diluted in 1 L sodium chloride 0.9%, administered over 12–24 hours.
May need to be repeated frequently, i.e. every 20–30 minutes.
Only moderately effective.

19.1.4 SPIDER ENVENOMATION

T63.3

BLACK WIDOW SPIDER

Features useful in the identification of black widow spiders are black or dark brown colour with variable red markings on the dorsal aspect of the abdomen, which diminish with age and no ventral markings. Is to be differentiated from *Latrodectus geometricus* with a geometric hourglass shaped marking on the ventral abdomen and much less potent venom.

The venom typically causes:

- severe general muscle pain and cramps especially of the large girdle muscles,
- muscle rigidity
- feeling of tightness of the chest
- board-like rigidity of a non tender abdomen

Profuse sweating may be prominent, as well as neurological hyperreactivity.

Pain increases with time and may last for days to a week if antivenom is not given

Secondary infection with cellulitis is a common complication.

Other spider species, may cause cytotoxic damage.

NON-DRUG TREATMENT

Observe all cases for 12–24 hours.

DRUG TREATMENT

- spider antivenom, IV infusion, 5–10 mL diluted in 50–100 mL sodium chloride 0.9% or dextrose 5%, administered over 5–10 minutes

Never administer antivenom without being fully prepared to manage acute anaphylaxis.

Severe muscle pain and cramps:

- calcium gluconate 10%, bolus IV infusion, 10 mL over 10 minutes
Followed by 60–120 mL diluted in 1 L sodium chloride 0.9%, administered over 12–24 hours.
May need to be repeated frequently, i.e. every 20–30 minutes.

Immunisation, primary or booster:

- tetanus toxoid vaccine, IM, 0.5 mL immediately

In unimmunised or partially immunised patients

- tetanus immunoglobulin, human, IM, 250 units

Intravenous Fluids

Reverse circulatory shock, if present.

Monitor response.

Analgesia

For mild pain:

- paracetamol, oral, 1 g 4–6 hourly as needed
- Opioids and NSAIDS have not been shown to be effective.

Antihistamines, e.g.:

- chlorpheniramine, oral, 4–8 mg as a single dose
OR
promethazine, IM, 25–50 mg as a single dose

For secondary infection: See Section 4.2: Cellulitis and Erysipelas.

South African Vaccine Producers (Pty) Ltd
1 Modderfontein Road Sandringham, Johannesburg Tel: (011) 386 6000 Fax: (011) 386 6016 After hours: 082 905 3329 http://www.savp.co.za/
Products Polyvalent snake venom (10 mL) Boomslang antivenom (10 mL) Spider antivenom (<i>Latrodectus indistinctus</i>) (5 mL) Scorpion antivenom (<i>Parabuthus spp.</i>) (5 mL)

19.2 EXPOSURE TO POISONOUS SUBSTANCES**GENERAL MEASURES**

- Limit further exposure to toxin.
In case of skin exposure, wash body and remove clothes. Showering may be useful.
Eye contaminants, especially alkalis, acids and other irritants, should be removed by continuous irrigation of the eye for 15–20 minutes.
- Take a complete and accurate history, ascertain all relevant facts and do a complete clinical examination. A high index of suspicion is important.
Obtain a collateral history, especially for patients with impaired consciousness.
A special effort should be made to obtain tablets, packets, containers, etc. to identify agents involved.
- Limit the use of toxicology investigations to those that may influence/alter management.
Avoid non-specific toxicology screens.
- Establish baseline laboratory values.
Quantify toxin level in blood, to monitor progress where relevant and follow urine and electrolytes, pH and blood gases, glucose and others, as indicated.
- Maintain and follow basic clinical parameters, i.e.:
 - pulse rate
 - blood pressure
 - hydration
 - ventilation
 - patent airway and oxygenation
 - control seizures and prevent physical injury in the restless. Avoid excessive sedation.

INITIATION OF TREATMENT**Neutralise poison**

Administer only if patient has ingested a potentially toxic amount of a poison which is known to be adsorbed by charcoal.

Administer within 1–2 hours after ingestion of poison.

- charcoal, activated, oral, 50–100 g diluted in 300–600 mL water
In poisoning with large amounts and/or slow release formulations, repeat at least 12.5 mg hourly.
When mixing, add charcoal to water and not vice versa.

Alkalinisation

Possible benefit in salicylate, lithium and less clearly, tricyclic antidepressant poisoning.

Note:

Salicylate poisoning may cause a respiratory alkalosis, which may aggravate the metabolic acidotic state. The infusion of large volumes sodium and water may precipitate hypernatraemia and fluid overload.

The increase in pH may also be associated with hypokalaemia, which may cause dysrhythmias in a patient with a tricyclic antidepressant overdose.



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In this setting consider only in the presence of cardiac involvement, i.e. prolonged QRS duration or QRS axis abnormality on ECG.

- sodium bicarbonate, IV, 50–100 mEq in 1 L sodium chloride 0.45%
Administer 250–500 mL over 1–2 hours.
Attempt to achieve urine pH of 7.5 or higher.

Haemodialysis

PATIENT SHOULD BE REFERRED TO A TERTIARY (DIALYSIS) CENTRE.

Limited to a number of agents, i.e. salicylates, lithium (serum levels over 4.0 mmol/L), ethylene glycol, methanol, ethanol and theophylline that does not respond to initial/alternative management.

Charcoal haemoperfusion may be of benefit in cases of extremely high theophylline levels, benzodiazepine, carbamazepine and methaqualone poisoning.

REFERRAL

- severely ill patient for ventilatory/circulatory support
- relevant diagnostic testing not available, e.g. paracetamol levels
- relevant medication/antidotes is not available
- where dialysis/haemoperfusion is required
- for psychiatric evaluation

19.3 SPECIFIC POISONINGS

19.3.1 BENZODIAZEPINE POISONING

T42.4

DESCRIPTION

Patients present with depressed mental and respiratory function.

Benzodiazepines are unlikely to cause significant respiratory suppression on their own.

Management is supportive.

Ventilatory support may be required.

19.3.2 CARBON MONOXIDE POISONING

T58

DESCRIPTION

Poisoning caused by accidental or suicidal exposure to fires in poorly ventilated areas, combustion engines, faulty stoves and faulty heating systems.

Patients present with:

- dizziness
- seizures and other CNS symptoms
- nausea
- vomiting
- retinal haemorrhages
- impaired level of consciousness
- cherry red skin and lips
- tachycardia
- ECG changes
- normal arterial PO₂ but low oxygen saturation
- high arterial carboxyhaemoglobin - test not commonly available

NON-DRUG TREATMENT

Remove patient from toxic environment.

Oxygen via facemask.

Ventilation may be needed in deeply comatose patients.

In a Cochrane review, hyperbaric oxygen therapy has not been shown to be of benefit.

DRUG TREATMENT

For seizures:

- diazepam, slow IV, 10 mg

19.3.3 INGESTION OF CAUSTIC SUBSTANCES

T54.3/T54.2

DESCRIPTION

Alkaline: Detergent, toilet bowl cleaners and liquid drain cleaners.

Acids: Various.

Causes tissue necrosis of gut resulting in strictures later.

NON-DRUG TREATMENT

No emesis or gastric lavage.

Rinse mouth with copious amounts of cold water.

Patients require urgent endoscopic evaluation and possible surgical intervention.

19.3.4 COCAINE POISONING

T40.5

DESCRIPTION

Cocaine may be absorbed through any mucous membrane, smoked or injected intravenously. Persons who smuggle cocaine, may ingest packets of this agent.

Patients may present with one or more of the following:

- acute myocardial infarction
- cardiac dysrhythmias
- tachycardia and hypertension
- stroke
- seizures
- alterations in mood and confusion
- pulmonary oedema
- rhabdomyolysis with acute renal failure and intestinal ischaemia

NON-DRUG TREATMENT

Supportive management aimed at preventing and managing complications.

Cool patients with hyperthermia.

Abdominal X-rays may show packages of cocaine. In these patients, conservative management is recommended. Surgery is reserved for those who develop obstruction or perforation.

Raised serum creatine kinase may indicate rhabdomyolysis or myocardial infarction.

The ECG may be normal in some cases of acute myocardial infarction.

For severe dysrhythmias, DC cardioversion.

Note:

Lidocaine (lignocaine) may precipitate seizures.

β-blockers may aggravate hypertension and myocardial ischaemia.

DRUG TREATMENT

For sedation or seizures:

- diazepam, IV, 10 mg

STATUS EPILEPTICUS

See Section 14.4.1: Status Epilepticus.

Psychosis or delirium with severe agitation:

- haloperidol, IM, 2–5 mg

OR

lorazepam, IM, 2 mg

Severe hypertension:

- labetalol, IV, 2 mg/minute to a maximum of 1–2 mg/kg

As relatively unopposed stimulation of alpha-receptors may result, this should be preceded by:

- chlorpromazine, IV, 25 mg

19.3.5 ETHANOL POISONING

T51.0

DESCRIPTION

Acute poisoning usually presents with:

- central nervous system depression
- hypoglycaemia
- hypothermia
- changes in fluid and electrolyte status such as hypokalaemia and hyponatraemia

High levels of ethanol may influence the osmolar gap and cause a pseudohyponatraemia.

See Section 19.3.6: Ethylene Glycol Poisoning.

Consider other causes for the patient's condition, including hypoglycaemia and head trauma.

NON-DRUG TREATMENT

Supportive management aimed maintaining stable cardiorespiratory function.
Manage hypothermia.

DRUG TREATMENT

- thiamine, IV, 100 mg in 1 L dextrose 5%

19.3.6 ETHYLENE GLYCOL POISONING

T52.3

DESCRIPTION

Ethylene glycol is a component of motor vehicle radiator coolant/antifreeze and brake fluid. It is also found in homemade toilet and drain cleaners.

Clinical signs:

- resembles alcohol intoxication
- vomiting
- later hypotension
- cardiac failure
- oliguric renal failure
- significant metabolic acidosis with a large anion gap, i.e.:
($[\text{Na}^+] - [\text{Cl}^-] - [\text{HCO}_3^-] > 12$)
- hypocalcaemia
- a higher measured serum osmolality when compared to the calculated equivalent
- oxalate crystals in urine

DRUG TREATMENT

In acidotic patients, haemodialysis is the treatment of choice.

Ethanol

In other patients and where access to dialysis facilities is not readily available:

- ethanol 95% BP, IV, diluted to 10% in dextrose 5%
Administer 10 mL/kg of dilute solution over 30–45 minutes (0.6–0.7 g/kg ethanol).
Follow with the dilute solution at:
 - 1 mL/kg/hour for non-drinkers.
 - 2 mL/kg/hour for patients with hepatic enzyme induction, such as chronic alcohol users.

If intravenous ethanol cannot be given:

- ethanol 95% BP, oral, diluted to 20% in any suitable liquid
Administer 1 mL/kg of dilute solution.
Follow with the dilute solution at:
 - 0.1 mL/kg/hour for non-drinkers.
 - 0.2 mL/kg/hour for patients with hepatic enzyme induction, such as chronic alcohol users.

If ethanol 95% BP is not available, orally administer any commercially available alcoholic beverage with an alcohol content of \pm 40% (80 proof), at the above oral dose.

Note:

The patient needs to be co-operative or administration via nasogastric tube may be required.

The aim is to maintain plasma ethanol levels of 1–1.3 g/L (0.1–0.13 g/dL).

Several days of ethanol therapy may be required. Continue treatment until clinical condition improves.

- thiamine, oral, 100 mg daily

METABOLIC ACIDOSIS

The ultimate goal is a pH of not more than 7.2.

- sodium bicarbonate, IV, 50–100 mmol/L administered over 30–45 minutes

Note:

The rapid infusion of large volumes in an already oliguric patient may precipitate pulmonary oedema and cardiac dysrhythmias.

Correct hypoglycaemia.

Correct severe or clinical evident hypocalcaemia.

19.3.7 HEAVY METALS: MERCURY, ARSENIC, GOLD, COPPER AND LEAD POISONING

T56.1/T57.0/T56.8/T56.4/T56.0

DESCRIPTION

Acute toxicity of organ-systems may be summarised as follows:

	Pneu monitis	GIT	Blood cells	CVS collapse	Kidneys	Hepato toxicity	CNS
Mercury	X	X		X	X		X
Arsenic	X	X	X	X	X		X
Gold		X		?		X	
Thallium		X	X				X
Copper		X	X		X	X	X
Lead		X			X		X
Cadmium	X	X					

Dimercaprol

Dimercaprol chelates metals, enabling excretion of the less toxic complexes.

It is the agent of choice for mercury, gold and arsenic poisoning.

Its role is less established in cases of lead, copper and thallium poisoning.

Do not use for iron poisoning.

- dimercaprol 10%, deep IM, 400–800 mg in divided doses on the first day, then 200–400 mg/day in divided doses for 2 days and then 100–200 mg/day in divided doses for 4–7 days

These injections are painful and sterile abscesses may form.

In severe cases, consider a loading dose of 5 mg/kg.

Penicillamine

Penicillamine also chelates metals.

Indicated in copper, mercury, arsenic, zinc and lead poisoning.

- penicillamine, oral, 0.5–1.5 g/day in 4 divided doses

19.3.8 HYDROCARBON POISONING

T52.0

DESCRIPTION

Poisoning due to petroleum products, including paraffin, turpentine, petrol, mineral spirits and halogenated hydrocarbons.

Clinical signs:

- aspiration pneumonia
- GIT effects
- arrhythmias
- CNS effects

NON-DRUG TREATMENT

If contaminated, remove clothing and wash skin.

Do not attempt gastric emptying/lavage.

DRUG TREATMENT

The usefulness of activated charcoal is limited.

Observe and examine for aspiration pneumonia. Prophylactic antibiotics are not indicated.

19.3.9 IRON TOXICITY

T45.4

DESCRIPTION

Iron is a commonly prescribed drug, especially in pregnancy, and causes initial gastrointestinal toxicity.

More significant exposure may be associated with:

- metabolic acidosis
- hypotension
- CNS side effects
- renal failure
- hepatitis

DRUG TREATMENT**Chelation therapy**

Patients with serum iron levels < 54 micromol/L and absence of symptoms more than 6 hours after overdose do not require chelation therapy.

- desferrioxamine, IV, 1–2 g every 3–12 hours to a maximum of 6 g every 24 hours

For levels > 180 micromol/L, consider exchange transfusion.

If serum iron levels are not available and the probability of this poisoning is high administer a single dose of desferrioxamine 1 g and observe for “vin rosé” discoloration of urine, which indicates high blood iron levels. If present, continue with chelation therapy, as above.

Give intravenous fluids for hypotension.

19.3.10 POISONING WITH AMPHETAMINE DERIVATIVES

T43.6

DESCRIPTION

These include:

- “Ecstasy”: 3,4-methylenedioxymethamphetamine (MDMA)
- “Ice” and “Eve”: 3,4-methylenedioxy-N-ethylamphetamine (MDEA)
- “Tic tic”: Methamphetamine

Drug effects are due to effects on dopaminergic and serotonergic neurons in the CNS and include:

- hyperthermia, especially with Ecstasy
- tachycardia
- hypertension
- angina pectoris and myocardial infarction
- stroke
- hyperactivity
- delirium
- tremors
- seizures and coma.

Further complications include:

- rhabdomyolysis, which presents with elevated serum creatine kinase
- hyperkalaemia
- later acute tubular necrosis
- potentially fatal hyponatraemia
- dehydration

NON-DRUG TREATMENT

Supportive management aimed at maintaining stable cardiorespiratory function. Manage hypothermia, hypoglycaemia and fluid and electrolyte status.

DRUG TREATMENT

Haemodialysis may be required for acute renal failure.

For seizures:

- diazepam, IV, 10 mg

Severe hypertension:

- labetalol, IV, 2 mg/minute to a maximum of 1–2 mg/kg

As relatively unopposed stimulation of alpha-receptors may result, this should be preceded by:

- chlorpromazine, IV, 25 mg

19.3.11 METHANOL POISONING

T51.1

DESCRIPTION

Previously found in methylated spirits but has recently been replaced with less toxic agents.

Presents with:

- initially, CNS and GIT effects
- later, large anion gap (> 12), metabolic acidosis, retinal toxicity and renal failure due to formic acid production.

DRUG TREATMENT

If acidotic and there is an osmolar gap, [measured osmolarity minus calculated ($2 \{ \text{sodium} + \text{potassium} \} + \text{urea} + \text{glucose}$)], start dialysis urgently, if available.

If dialysis not available, use ethanol.

See Section 19.3.6: Ethylene Glycol Poisoning.

19.3.12 POISONING WITH NITRITES, NITROPRUSSIDE, NITROGLYCERINE CHLORATES, SULPHONAMIDES AND OTHERS

D74.8/D74.9

DESCRIPTION

Nitrites are used to cure meat in the informal butchery sector.

Patients present with:

- normal oxygen levels and deep central cyanosis, due to methaemoglobinaemia
- CNS depression
- arrhythmias.

Note:

The measured PO_2 is normal and that only the pH and a directly measured oxygen saturation give an indication of severity.

NON-DRUG TREATMENT

Oxygen via facemask.

DRUG TREATMENT

In symptomatic cases or patients with high methaemoglobin values $> 30\%$:

- methylene blue (tetramethyl thionine chloride) 1% dilute solution, slow IV infusion, 1–2 mg/kg administered over 5 minutes

Repeat in 1 hour and if necessary every 4 hours up to total of 7 mg/kg.

Side effects include precordial pain, restlessness and dyspnoea.

In severe cases, not responding to methylene blue or if methylene blue is not available, consider exchange transfusion.

Administration of ascorbic acid (vitamin C) has not been shown to be effective, mainly due to slow onset of effect.

19.3.13 OPIOID POISONING

T40

DESCRIPTION

Poisoning due to morphine, pethidine, methadone (long-acting) and heroin ingestion.

Patients present with:

- especially respiratory depression, and/or CVS-, and/or CNS depression
- miosis.

Non-cardiogenic pulmonary oedema may be present.

NON-DRUG TREATMENT

Supportive management aimed at maintaining cardiorespiratory function.

DRUG TREATMENT

- naloxone, IV, 0.4–2 mg immediately
Repeat 0.4 mg every 5 minutes until reversal or pupils dilate.
Total effective dose is 10 mg.
May be administered endotracheally.
Duration of action is short, i.e. 45 minutes. Repeat doses over 24 hours may be required.

19.3.14 ORGANOPHOSPHATE POISONING

T60.0

* Notifiable condition.

DESCRIPTION

Poisoning due to parathion, malathion and other insecticides, possibly in hydrocarbon solvent.

Absorption occurs through the skin or agent is taken orally.

Patients present with muscarinic and/or nicotinic manifestations of intoxication.

Muscarinic overstimulation causes salivation, lacrimation, vomiting, diarrhoea and increased bronchial secretions. Nicotinic overstimulation causes muscle fasciculations and paresis or paralysis. Patients may present with either bradycardia or tachycardia. May be confirmed by measuring red cell pseudocholinesterase levels - serum levels are unreliable.

NON-DRUG TREATMENT

Decontamination of skin and clothes, where applicable.

Maintain adequate ventilation and circulation.

Ventilatory support in ICU may be required due to excess of nicotinic effects.

DRUG TREATMENT

- atropine, IV, 1 mg, initial dose
Follow with 0.05 mg/kg every 15 minutes.
Assess degree of atropinisation by increase in pupil size (do not monitor continuous atropinisation on pupil size), pulse rate, bronchial secretions and salivation.
A continuous IV infusion of 0.05 mg/kg/hour may be required.
Atropine therapy should not be stopped abruptly and should be weaned at a rate of no more than 1–2 mL/hour. During this phase it is important to follow the patient as a worsening in condition commonly occurs a few days following ingestion.

19.3.15 PARACETAMOL POISONING

T39.1

DESCRIPTION

The liver is the main organ acutely damaged in paracetamol poisoning. Acute ingestion of doses of 7.5–15 g in a healthy adult may cause severe centrilobular hepatic necrosis. In patients on enzyme inducers, particularly alcohol, lower doses of paracetamol cause damage. Renal tubular necrosis may also develop. Hepatic and renal failure typically manifest after 2–5 days.

Clinical featuresWithin 0.5–24 hours after overdose

The patient may experience symptoms of gastrointestinal irritability with anorexia, nausea, vomiting, abdominal pain, as well as pallor, malaise and sweating. During this phase the patient may, however, appear normal or asymptomatic. Activated charcoal should be administered within 1–2 hours after overdose.

24–48 hours after overdose

Signs and symptoms may be less pronounced, but the blood chemistry may become abnormal. In severe intoxication, the clinical picture of progressive liver failure develops within 2–5 days.

Liver damage is likely to occur in patients with paracetamol levels > 300 mcg/mL at 4 hours or > 45 mcg/mL at 15 hours post ingestion.

Levels < 120 mcg/mL at 4 hours are unlikely to be associated with hepatotoxicity. For reliable hepatotoxicity risk assessment, blood for plasma paracetamol levels should be drawn 4 hours post ingestion or as possible thereafter as this represents peak levels.

If paracetamol levels are in the toxic range, defined as above the predictive graph line joining 150 mcg/mL at 4 hours and 20 mcg/mL at 15 hours after ingestion on the Rumack-Matthew nomogram, liver function tests (ALT) should be performed daily.

With significant hepatitis, investigate degree of liver failure with INR and refer patient for further management, which may involve liver transplant. Patients with paracetamol levels in the toxic range, who have been managed appropriately and are asymptomatic with normal liver functions after 48–72 hours, may be discharged.

Monitor renal function and serum potassium in patients with significant toxicity.

DRUG TREATMENT

Acetylcysteine

Acetylcysteine is the antidote of choice and should be given intravenously. Although it is more effective when given within 8 hours of ingestion of paracetamol, there may be benefit even if liver failure has developed.

It is never too late to administer acetylcysteine.

Indications for acetylcysteine

Administer without waiting for plasma paracetamol levels in substantial overdose, defined as 7.5 g or 125 mg/kg, whichever is smaller. Discontinue if plasma levels are in the non-toxic range.

Administer immediately if there is doubt about the time interval since ingestion. Do not use the Rumack-Matthew nomogram.

Give to patients presenting 24 hours or later after an overdose, who have detectable plasma paracetamol levels or biochemical evidence of hepatotoxicity.

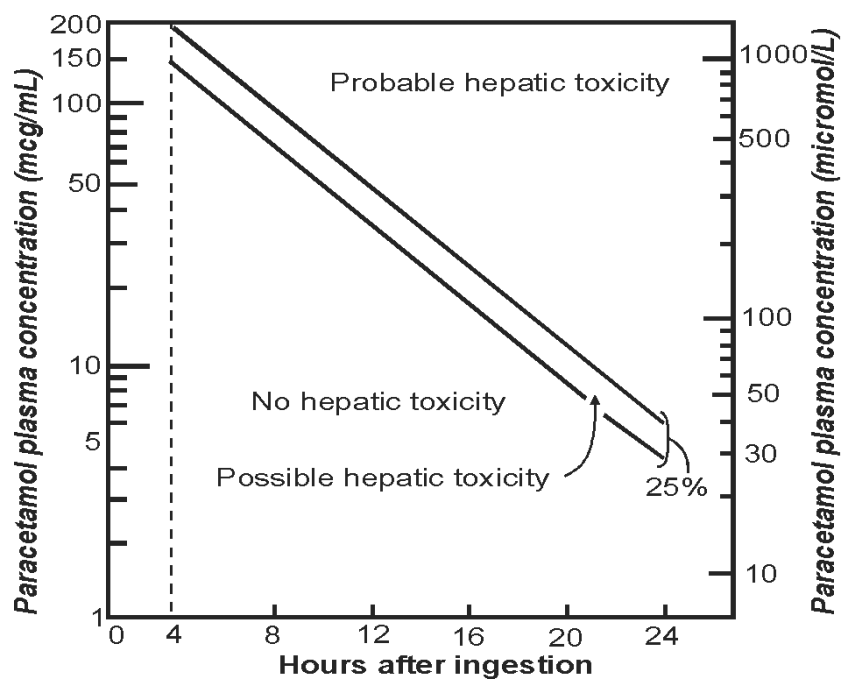
Acetylcysteine therapy is necessary if the initial paracetamol level is in the toxic range.

Paracetamol toxicity develops at lower plasma concentrations in:

- patients taking drugs that induce hepatic enzymes, e.g. barbiturates, phenytoin, carbamazepine, rifampicin and meprobamate
- alcohol abusers
- patients with conditions causing glutathione depletion, e.g. malnutrition and HIV infection.

In these patients a lower threshold for instituting antidote therapy should be used, i.e. 25% lower than the line joining 150 mcg/mL at 4 hours and 20 mcg/mL at 15 hours.

- acetylcysteine, IV, 150 mg/kg in 200 mL dextrose 5% over 15 minutes as a loading dose
Follow with 50 mg/kg in 500 mL dextrose 5% over next 4 hours by continuous infusion.
Then 100 mg/kg in 1 L dextrose 5% over 16 hours.
Beware of allergic reactions. In less severe cases of allergy administer the loading dose over 1–2 hours under antihistamine cover and continue at a lower infusion rate.



Modified and reproduced from Rumack BH, Matthew H: Acetaminophen poisoning and toxicity. Paediatrics 1975; 55:871

19.3.16 SALICYLATES OR NSAID POISONING

T39/T39.3

DESCRIPTION

Patients present with:

- nausea
- vomiting
- CNS depression
- respiratory alkalosis followed by metabolic acidosis or one or both disorders
- tinnitus
- convulsions
- non cardiogenic pulmonary oedema

NON-DRUG TREATMENT

Consider ICU admission for pulmonary and/or cerebral oedema.

DRUG TREATMENT

Alkalinisation often with potassium replacement. Exercise caution in patients with respiratory alkalosis.

Monitor closely with laboratory data, where available.

Where acidosis does not respond rapidly to sodium bicarbonate, consider haemodialysis.

19.3.17 THEOPHYLLINE POISONING

T44.9

DESCRIPTION

Patients present with:

- tachycardia and tachydysrhythmias
- vomiting
- agitation
- seizures
- nausea
- abdominal pain
- restlessness
- profound hypokalaemia

NON-DRUG TREATMENT

Monitor cardiac function and treat dysrhythmias.

Monitor and correct fluid status and other electrolyte abnormalities.

DRUG TREATMENT

Correct hypokalaemia actively:

- potassium chloride, IV, not more than 40 mmol/L and rate not more than 20 mmol/hour

For seizures:

- diazepam IV, 10 mg

In severe cases, consider charcoal haemoperfusion.

19.3.18 TRICYCLIC ANTIDEPRESSANT POISONING

T43.0

DESCRIPTION

Patients present with:

- signs of cholinergic blockade
- hypotension
- agitation progressing to coma
- both tachy- and bradyarrhythmias
- pulmonary oedema
- seizures

The antimuscarinic effects of these agents may cause transient gastrointestinal ileus and urinary retention.

NON-DRUG TREATMENT

Monitor with ECG and blood gases.

ICU admission for ventilatory/circulatory support, when indicated.

Manage gastrointestinal ileus and urinary retention appropriately by keeping patients nil per mouth and inserting a urinary catheter.

DRUG TREATMENT

Repeated or prolonged administration of activated charcoal.

Although the evidence for this indication is limited, consider alkalinisation in severe cases with evidence of cardiac involvement, i.e. prolongation of QRS duration or QRS axis deviation on ECG and /or severe hypotension.

Note:

Alkalosis and resultant hypokalaemia may aggravate cardiac dysrhythmias.

For torsade des pointes:

- magnesium sulphate, IV 2 g administered over 30 minutes, then 1 g hourly

Manage broad complexes with:

- sodium bicarbonate to a pH of 7.55

For seizures or if sedation is required for restlessness

- diazepam, IV, 10 mg

Intravenous Fluids

Reverse circulatory shock, if present.

In severe cases, provide inotropic support

Monitor response.

See Section 20.1.2.2: Tension Pneumothorax, Suspected

19.3.19 ANTICOAGULANT POISONING

T45.5

DESCRIPTION

Poisoning due to warfarin ingestion and ingestion of supercoumarins, e.g. rat poison and other vermin poisons.

DRUG TREATMENT

- vitamin K₁, IV/IM, 10 mg
May be repeated depending on the INR response.
Note the delay in effect.

Patients bleeding require additional fresh frozen plasma and the first dose of intravenous vitamin K.

Follow up doses by any route may be required if INR continues to rise, as vitamin K has a shorter half-life than warfarin.

Administration of vitamin K may induce resistance to warfarin.

Patients on chronic warfarin therapy and at high risk for thromboembolic complications, but who are not actively bleeding with INR between 5–9:

- vitamin K₁, IV, 0.5–1 mg low dose

Resume warfarin therapy, once the INR is less than 4.0.

For INR > 9:

- vitamin K₁, IV, 2.5 mg

OR

- vitamin K₁, oral, 5 mg

For oral administration of low doses, the parenteral form may be used.

In all cases repeat INR in 24 hours.

SUPER COUMARINS

Treatment may be prolonged.

19.4 POISON CENTRES

Western Cape	Tygerberg	021 931 6129
	Red Cross	021 689 5227
Gauteng	Johannesburg	0800 333 444 082 911
	Garden City	011 495 5000
	MEDUNSA	012 521 4145/4359 (office hours)
Free State	Bloemfontein	082 491 0160
KwaZulu-Natal	St. Augustine's	031 268 5559
Telephone numbers tested on 31 May 2006.		



CHAPTER 20 EMERGENCIES AND INJURIES

20.1 EMERGENCIES

20.1.1 ANAPHYLAXIS/ANAPHYLACTIC SHOCK

R57.9

DESCRIPTION

An acute, potentially life-threatening hypersensitivity reaction starting within seconds to minutes after administration of or exposure to a substance to which the individual has been sensitised. Clinical manifestations range from mild urticaria and angioedema to upper airway obstruction, bronchospasm, hypotension, shock and death. The reaction can be short-lived, protracted or biphasic, i.e. acute with recurrence several hours later. Immediate reactions are usually the most severe and/or life threatening.

NON-DRUG TREATMENT

Cardiopulmonary resuscitation.
Maintain an open airway. Intubate if necessary.
Monitor all vital parameters closely.
Check pulse and blood pressure.
Reassure and comfort patient.
Patient counselling to prevent recurrence.
Medical alert bracelet should be worn at all times.

DRUG TREATMENT

Administer adrenaline and hydrocortisone early to prevent circulatory collapse and severe bronchospasm.

Intravenous fluids

Establish a large bore intravenous line and keep open with:

- sodium chloride 0.9%, IV

If IV access not possible, administer the first dose of adrenaline IM.

- adrenaline 1:1 000, **IM**, 0.3–0.5 mL by deep intramuscular injection. **Not subcutaneously.**

OR

adrenaline, IV, 3–5 mL of 1:10 000 solution

Give very slowly. Start with 1 mL then repeat after every minute.

Maximum dose: 1 mg/dose or 5 mg/day.

To make a 1:10 000 solution: dilute 1 mL in 9 mL sodium chloride 0.9%.

PLUS

- hydrocortisone, IV, 200 mg, immediately

PLUS