Chapter 16:

POISONINGS

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This chapter covers the following topics:

- Epidemiology of poisonings and toxidromes
- Approach to the child with suspected poisoning
- Iron poisoning/overdose
- Organophosphate poisoning
- Paraffin poisoning (hydrocarbons)
- Tricyclic antidepressants (TCAs) overdose
- Caustic and corrosive substance ingestion
- Neuroleptic overdose
- Salicylate poisoning
- Snake bites
- Oral contraceptive overdose

EPIDEMIOLOGY OF POISONING AND TOXIDROMES

Children may be exposed to a large spectrum of potentially harmful substances. Most poisoning cases are in children <6 years old. Internationally, poisoning is responsible for 3.9% of injury-related deaths, with children in low- and middle-income countries making up the greatest proportion of these cases. A study of poisoning cases encountered at Red Cross War Memorial Children's Hospital revealed that pharmaceuticals are responsible for most poisoning cases, followed by paraffin, pesticides, household products and cosmetics.

Below are important toxidromes which can help one identify the class of the implicated toxin:

Table 16.1: Clinical Features of Important Toxidromes

Toxidrome	Clinical Features	Examples
Anticholinergic	Agitation, hallucinations, dilated pupils, dry mouth, dry skin, fever, urinary retention	Antihistamines, atropine, *Amanita pantherina*
Cholinergic	Pinpoint pupils, bradycardia, respiratory secretions (bronchorrhoea), fasciculations, salivation, wheezing, diarrhoea	Organophosphates
Sympathomimetic	Agitation, hallucinations, dilated pupils, sweating, tremor, seizures, tachycardia, hypertension	Cocaine, amphetamines, nasal decongestants
Sedative/hypnotic	Coma, hypothermia, hypotension	Alcohol, benzodiazepines
Opioid	Coma, pinpoint pupils, hypothermia, hypotension, respiratory depression	Codeine

APPROACH TO THE CHILD WITH SUSPECTED POISONING

One must:

- Resuscitate
- Identify the poison
- Prevent further absorption of the poison
- Enhance and support elimination of the poison

Resuscitate

Assess airway, breathing, circulation and disability, noting the child's mental status, heart rate, respiratory rate, temperature, pupils, skin colour and temperature, and the presence of fasciculations.

Identify the Poison

One must take a history from the child (if possible) and caregiver, and read the drug name and concentration of the suspected poison from its bottle or container. In South Africa, one may call the Poisons Information Helpline (0861 555 777; a 24-hour emergency line) to find out if the symptoms correspond with any toxidromes.

Prevent Absorption

Decontaminate the skin or mucous membranes by washing or irrigating them with water for 15-20 mins (use saline to irrigate the eye, if possible). Gut decontamination is performed if a potentially life-threatening poison is ingested and the patient presents within 1 hour of ingestion. One can consider using multiple doses of activated charcoal to adsorb the poison if the child has an intact gag reflex or the airway is protected. Activated charcoal binds certain toxins (e.g. carbamazepine, theophylline) in the GIT to prevent absorption. Haemodialysis may also be of benefit in some poisonings e.g. ethylene glycol poisoning.

Support and Enhance Elimination of the Poison

One may support elimination preventing and reversing complications:

- Monitor the child
- Support respiration
- Treat hypotension or arrhythmias and give fluids
- Control convulsions
- Manage hypoglycaemia and hypothermia

If possible, one should measure the drug levels.

The effect of the drug may be limited by administering an antidote, such as:

- Desferrioxamine for iron poisoning
- Atropine for organophosphate poisoning
- Naloxone for opioid overdose

- Flumazenil for benzodiazepine overdose
- N-acetylcysteine for paracetamol overdose

Urine alkalinisation (administer IV sodium bicarbonate to increase the pH of the urine) is done to enhance the elimination of acids, such as salicylates.

IRON POISONING/OVERDOSE

Children are usually exposed to iron because their parents have iron supplements in the medicine cabinet. Risk of serious poisoning is dependent on the child's exposure to elemental iron. Doses of more than 20 mg/kg may result in symptoms and doses of 40-60 mg/kg may result in symptoms of serious toxicity. See related image here.

Clinical Features

The clinical picture progresses through five phases which often overlap. They are:

- Gastrointestinal phase (30 mins to 6 hours)
- Latent phase (6-24 hours)
- Shock (6-72 hours)
- Hepatotoxicity (12-96 hours)
- Bowel obstruction (2-8 weeks)

The presentation varies and may include:

- Mild abdominal symptoms e.g. abdominal tenderness, vomiting and diarrhoea
- Tachycardia
- Altered mental state
- Hypotension suggestive of shock
- Liver failure after about 48 hours (the loss of synthetic function results in coagulopathy, INR >1.5 and encephalopathy)

Investigations

Special investigations should include serum iron levels (at least 4 hours post-ingestion and may need to be repeated) and abdominal X-ray (may demonstrate ingested pills).

Management

Iron poisoning is a medical emergency and must be treated with urgency. Delay in starting therapy may prove fatal. Treatment is based on three principles which must be simultaneously applied in all patients with iron poisoning:

- Maintain an effective circulating blood volume
- Eliminate as much iron from the gut as possible, before it can be absorbed
- Chelate absorbed iron with desferrioxamine before it can cause mitochondrial damage

General and Supportive Measures

Resuscitate the patient as needed. Should the child have a reduced level of consciousness, secure the airway and consider ventilation.

Specific Management

If the child presented early and mainly has gastrointestinal manifestations, gastrointestinal decontamination should be considered. However, activated charcoal is not recommended (has poor iron binding capacity) and gastric emptying and lavage are not indicated (the child will most likely be vomiting and adult iron preparations cannot be removed with an orogastric tube). Whole bowel irrigation therapy can be performed.

Desferrioxamine is an IV antidote which is given in serious overdose at a rate of 15 mg/kg/hour (maximum total dose of 80 mg/kg). It chelates iron and, thus, limits exposure. Desferrioxamine may be used in conjunction with sodium bicarbonate (if the child is acidotic).

ORGANOPHOSPHATE POISONING

Children are mainly exposed to organophosphates in the form of insecticides (used in rural or farming communities) or rat poison (in urban communities). Toxicity is the result of inhibition of the acetylcholinesterase enzyme, which causes the accumulation of acetylcholine.

Clinical Features and Diagnosis

Acute toxicity presents with a cholinergic toxidrome:

- Sweating
- Salivation
- Lacrimation
- Urination
- Diarrhoea
- Constriction of pupils
- Bradycardia
- Respiratory symptoms e.g. bronchospasm, bronchorrhoea, respiratory depression
- Muscle weakness

The diagnosis is made based on clinical features and suggestive history. If there is doubt surrounding the diagnosis, an atropine challenge may be done (give 0.01-0.02 mg/kg of atropine) and the patient watched for the development of anticholinergic symptoms. The plasma acetylcholinesterase level may be measured to confirm the diagnosis.

Management

General and Supportive Measures

One must first secure the child's airway. Decontamination is guided by history. The person who is performing the decontamination must wear the appropriate protective clothing. If the child’s skin is contaminated, discard the child’s clothes and thoroughly wash the child.

If the organophosphate was ingested, activated charcoal may be given (within 2 hours of ingestion). A gastric lavage should not be performed.

Intravenous benzodiazepines (e.g. diazepam 0.3 mg/kg; to a maximum of 10 mg) should be used in all but the mildest cases to relieve anxiety and treat or prevent breakthrough seizures. Doses should be repeated as necessary.

Organophosphate poisoning is a notifiable condition (as are all symptomatic poisonings), thus a form should be filled out and sent to the relevant local health authority.

Specific Treatment

One may administer:

- Atropine antidote:
    - One should give an initial bolus dose of atropine (0.05 mg/kg) and then double the atropine (see related image here) bolus dose every 5 minutes until some signs of atropinisation occur i.e. clearing of secretions (the most important sign of adequate atropinisation; improving heart rate and BP, drying of skin and dilating of pupils are late signs).
    - Once there is clinical improvement and signs of atropinisation, one should start an atropine infusion (10-20% of the total bolus dose of atropine given, administered hourly). Regularly monitor the patient and give additional bolus doses of atropine if any deterioration occurs until the patient is re-atropinised.
- Inhaled ipratropium:
    - 0.5 mg inhaled ipratropium can be given to help resolve bronchospasms.

PARAFFIN POISONING (HYDROCARBONS)

Hydrocarbons are found in a wide range of industrial and domestic products. Paraffin is the most ubiquitous of the hydrocarbons and is often stored in unmarked cooldrink bottles in unsecure locations, giving children easy access to it (see related image here). Toxicity depends on the route of exposure:

- Direct contact (local toxicity) – causes local irritation of the skin and mucous membranes due to leaching of oils and fat from skin
- Inhalation/aspiration – even small amounts can cause necrotising and haemorrhagic pneumonitis i.e. chemical pneumonitis
- Ingestion – causes systemic toxicity, CNS depression, cardiac sensitisation to catecholamines and even liver and kidney damage

Clinical Features

Clinical features suggestive of inhalation or aspiration include coughing, tachypnoea or dyspnoea with nasal flaring, wheezing and cyanosis. Air in the alveoli is displaced by vaporised hydrocarbons causing oedema and worsening hypoxia.
Systemic toxicity is typified by CNS effects – headache, dizziness, blurred vision, lethargy progressing to seizure and coma. Myocardial sensitisation results in arrhythmia.

Investigations

Important special investigations include arterial blood gas, FBC, RFTs and serum glucose. If respiratory symptoms develop, take a chest X-ray 6-8 hours after ingestion (or sooner if clinically indicated). The X-ray may demonstrate small patchy opacification.

Management

There is no role for gastrointestinal decontamination in patients with paraffin poisoning, as activated charcoal (see related image here) does not adsorb hydrocarbons and induced emesis/gastric lavage are contraindicated.
There is no specific treatment for paraffin poisoning, therefore the mainstay of treatment is supportive (careful monitoring of respiratory symptoms and pulse oximetry). If the child is severely distressed or has a decreased level of consciousness, one should intubate. Give oxygen and bronchodilators (β_2-antagonists) for distress. Closely monitor the child's oxygen to detect late-onset respiratory failure. If seizures develop, treat with benzodiazepines. Keep the child hydrated; monitor input and output.

TRICYCLIC ANTIDEPRESSANTS (TCAs) OVERDOSE

There is a high potential for toxicity with TCAs because of their narrow therapeutic indices (>5 mg/kg in children).

Clinical Features and Investigations

The symptoms are predominantly neurological (altered mental state, seizures) but also include cardiotoxicity, acidosis and respiratory depression. The child will also have anticholinergic features, such as decreased GI motility and urinary retention.

Management

Monitoring

It is important to monitor input and output, arterial blood gas results (for acid-base levels) and RFTs (especially potassium) because of the anticholinergic effects of TCAs. One must also monitor drug levels. Baseline and continuous 12-lead ECGs should be done because cardiac toxicity may occur. BP should also be monitored.

Treatment

Gastrointestinal decontamination with activated charcoal is helpful up to several hours after exposure because of delayed gastric emptying. However, one must always ensure that the airway is protected before using activated charcoal due to the risks of rapid CNS depression, loss of airway control and seizure.

One should consider ventilation if the patient becomes hypoxic (pCO_2 >6.7 kPa or pO_2 <8 kPa). Sodium bicarbonate is the mainstay of treatment and 0.5-2 mmol/kg sodium bicarbonate by slow IV infusion with 5% dextrose should be administered. This should be followed by an infusion to keep the pH around 7.50. Wide QRS arrhythmias may develop and must be treated with sodium bicarbonate as above. Hypotension should be treated and normotension maintained through volume expansion (give Ringer's lactate or normal saline). Torsades de Pointes should be managed with magnesium sulphate (25-100 mg repeated every 10 mins to a maximum of 2 g). Convulsions are managed with benzodiazepines. Phenytoin is contraindicated.

CAUSTIC AND CORROSIVE SUBSTANCE INGESTION

Corrosive or caustic injury occurs as the result of the ingestion of acid or alkaline substances. Acids that have a corrosive effect include hydrochloric acid and battery acid. Alkalis which have corrosive effects include sodium hydroxide, calcium

hydroxide and potassium permanganate. Potassium permanganate is a commonly ingested or corrosive substance. See related image here.

Clinical Features

The main clinical feature is pain. Younger children who are unable to give a history of pain will present with crying, refusal to swallow, drooling or vomiting. Other symptoms include stridor or hoarseness. On examination, oral burns may be seen.

Management

If the child has been asymptomatic for 12 hours and is able to tolerate oral fluids, no intervention is necessary. All symptomatic patients will be investigated by endoscopy, which will influence further management.
Symptomatic patients should be kept nil per mouth and thoroughly examined to exclude an oesophageal perforation. If airway involvement is suspected, endotracheal intubation is indicated. A gastrointestinal bleed or oesophageal perforation should be surgically managed.

NEUROLEPTIC OVERDOSE

Neuroleptics are a group of antipsychotic and sedating agents. Commonly used neuroleptic drugs include chlorpromazine, haloperidol and phenothiazine antiemetics e.g. promethazine.

Clinical Features

The main clinical symptoms of neuroleptic overdose are:

- Decreased level of consciousness
- Respiratory difficulty/depression e.g. dyspnoea, cyanosis, apnoea
- Hypotension
- Extrapyramidal side effects

These agents also have antimuscarinic effects, namely dry mouth, urinary retention, dilated pupils and blurred vision.

Management

Monitoring

The child must be attached to an ECG monitor to detect arrhythmias. Other routine monitoring which should be performed are temperature, blood pressure and heart rate monitoring (tachycardias are common). Arterial blood gases should be done to monitor urea, electrolyte and glucose levels.

The child's level of consciousness must be regularly evaluated to ensure the child can maintain his/her own airway. If not, intubation is indicated.

General and Supportive Measures

If the child presents early (within two hours) and a significant overdose is suspected, give activated charcoal.

The associated hypotension should be managed with fluids and placing the child in Trendelenburg position to increase venous return. Occasionally, neuroleptic malignant syndrome (NMS) can develop. It causes:

- Hyperthermia
- Altered level of consciousness
- Muscle rigidity
- Abnormal U&E
- Features of autonomic disturbance (fluctuating BP, sweating incontinence)

NMS is an emergency and must be differentiated from dystonia for appropriate clinical management.

Specific Treatment

Children are prone to extrapyramidal side effects e.g. dystonia (prolonged, painful muscle contraction with abnormal postures and movements). This is managed with anticholinergics e.g. biperiden (Akineton®) given IM/IV at an aged-dependent dose which is repeated 6-8 hourly over 48-72 hours:

- Age <1 year – 1 mg
- 2-6 years – 2 mg
- 7-10 years – 3 mg
- >10 years – 5 mg

If there is no response, one should consider giving diazepam (0.1-0.4 mg/kg).

SALICYLATE POISONING

Salicylates are found in many over-the-counter medications, such as aspirin, Grand-Pa headache powders®, Tylenol®, Compral® and wintergreen ointment.

Clinical Features

Patients will commonly present with tachypnoea, hyperventilation and metabolic acidosis. They may also have nausea, vomiting, diarrhoea or tinnitus. With more severe intoxication they may have altered mental states, fever or pulmonary oedema, and may die.

Investigations

The plasma salicylate concentration should ideally be measured in anyone suspected of salicylate poisoning, and should be measured in 2-4-hour intervals. The results should be interpreted in the context of the patient's clinical condition. Doctors should continue careful monitoring of patients, even if salicylate concentrations are on the decline.

Management

As with any emergency patient, the Advanced Life Support (ALS) algorithm should be followed. One must pay special attention to and correct electrolyte imbalances, particularly glucose and potassium imbalances.

GIT decontamination with activated charcoal is recommended in acute overdose (1 g/kg; maximum single dose of 50 g). This should be reconsidered or omitted in patients with altered mental states who cannot maintain their airways (unless intubated). Urinary alkalinisation is recommended in addition to gastrointestinal decontamination with activated charcoal. This is done by administering an IV bolus of 1-2 mL/kg 8.4% sodium bicarbonate, followed by an infusion of sodium bicarbonate (add 100 mL of 8.4% sodium bicarbonate to 900 mL of 5% dextrose) at a rate approximately double the maintenance requirement. Potassium levels should be monitored and replaced as needed.

SNAKE BITES

There are about 35 venomous snake species in South Africa, but only ~10 of these are potentially fatal. These include cobras, mambas, adders, vipers, boomslangs and vine snakes.

Classification of Venom and Associated Syndromes

There are three types of venom – neurotoxic, cytotoxic and haemotoxic.

Table 16.2: Snake Venom Types

Neurotoxic Venom e.g. black mambas, small adders and non-spitting cobras	**Cytotoxic Venom** e.g. puff adders and some vipers	**Haemotoxic Venom** e.g. boomslangs and vine snakes
• Mainly acts in the synaptic clefts • Patients may present with dizziness, slurred speech, impaired coordination, hypersalivation and ptosis • Can cause hyponatremia, respiratory failure and paralysis of skeletal muscles • Associated syndrome – progressive weakness (PW) syndrome	• Contains proteolytic enzymes, therefore directly injures tissues (causes local tissue damage with necrosis) • Patients may present with tender lymphadenopathy, rash, low-grade fever and headaches • Associated syndrome – painful progressive swelling (PPS) syndrome	• Activate certain clotting factors (II and X), causing a consumptive coagulopathy • Patient may present with profuse bleeding, swelling and necrosis • May lead to DIC and multi-organ failure • Associated syndrome – bleeding (B) syndrome

Management

Identifying the type of snake is not always accurate and is not recommended. Instead, treatment should be given based on clinical presentation i.e. syndromic management. Management should always start with resuscitation (ABCDE). Tourniquets should not be used. Instead the patient must be kept still and the

affected limb may be gently splinted. One must check that the child's tetanus immunisation is up-to-date.

90% of envenomations are treated with supportive measures alone, unless severe envenomation is suspected. This includes:

- For PW syndrome – airway and ventilator support
- For PPS syndrome (90% of envenomation in SA) – elevate the bitten limb, remove tight clothing, give adequate analgesia and IV fluids, and mark the area of swelling and monitor hourly to assess progression and whether antivenom will be required.
- For B syndrome – perform coagulation studies; give blood and other blood products

Specific Treatment

Antivenom is only indicated in ~10% of snakebite patients. The antivenom may be polyvalent or monovalent. Polyvalent antivenom is used most commonly, as it covers ten types of snake bites e.g. for patients with PW. Monovalent antivenom is given to patients with a bleeding syndrome (as it may be due to a boomslang bite).

ORAL CONTRACEPTIVE OVERDOSE

Depending on the specific product, ingested oral contraceptives (see example here) are generally comprised of a mix of synthetic progesterones (progestins) and oestrogens, which have different ways of causing toxicity.

Clinical Features

Progestins do not cause acute toxicity; however, chronic exposure has been known to cause thromboembolic phenomena, jaundice and altered liver function. It also potentially precipitates porphyria attacks.

Oestrogens do have the potential to cause acute toxicity, but hospitalisation is rarely required. Symptoms of oestrogen toxicity include nausea, vomiting, diarrhoea and lethargy. Theoretically, a withdrawal vaginal bleed can occur in girls 1-3 days post-exposure but this is not supported by evidence. One must identify chronic exposure in children as it may lead to premature closure of epiphyseal plates and stunting growth.