Poisoning

General approach to the poisoned patient

Taking a history in poisoning

• What toxin(s) have been taken and how much?

• What time were they taken and by what route?

• Has alcohol or any other substance (or substances, including drugs of misuse) been taken as well?

• Obtain details from witnesses (e.g. family, friends, ambulance personnel) of the circumstances of the overdose

• Assess immediate suicide risk in those with apparent self-harm (full psychiatric evaluation when patient has recovered physically)

• Assess capacity to make decisions about accepting or refusing treatment

• Establish past medical history, drug history and allergies, social and family history

• Record all information carefully

Triage and resuscitation

• immediately assessing vital signs

• identifying the poison(s) involved and obtaining adequate information about them

• identifying patients at risk of further attempts at self-harm and removing any remaining hazards

Clinical assessment and investigations

- Glasgow Coma Scale (GCS) and AVPU (alert/verbal/painful/ unresponsive) scale are used to assess conscious level
- Vital signs
- Systemic examination (neurological, cardiac, respiratory)
- ECG and cardiac monitoring instituted in all patients with cardiovascular features or where exposure to potentially cardiotoxic substances is suspected.
- Patients who may need antidotes should be weighed if possible
- RBS, urea, electrolytes and creatinine
- Arterial blood gases and calculation of anion and osmolar gaps
- measurement of the amount of toxin in the blood
- Psychiatric assessment

General management

- Resuscitation (ABC measures)
- Remove patient from poising area
- Patients presenting with eye/skin contamination should undergo local decontamination measures.
- Supportive care
- Treatment of complications
- antidots

Gastrointestinal decontamination

Patients who have ingested potentially life-threatening quantities of toxins may be considered for gastrointestinal decontamination if poisoning has been recent

1. **Activated charcoal**
   Given orally as a slurry, activated charcoal absorbs toxins in the bowel as a result of its large surface area. It can prevent absorption of an important proportion of the ingested dose of toxin, but efficacy decreases with time and current guidelines do not encourage use more than 1 hour after overdose, unless a sustained-release preparation has been taken or when gastric emptying may be delayed. Use is ineffective for some toxins like iron, lithium, acids, alkalis, ethanol, Ethylene glycol, Mercury, Methanol, Petroleum distillates . In patients with impaired swallowing or a reduced level of consciousness, activated charcoal, even via a nasogastric tube, carries a risk of aspiration pneumonitis, which can be reduced (but not eliminated) by protecting the airway with a cuffed endotracheal tube. A laxative is generally given with the charcoal to reduce the risk of constipation or intestinal obstruction .

2. **Gastric aspiration and lavage**
   Gastric aspiration and/or lavage is very infrequently indicated in acute poisoning, as it is no more effective than activated charcoal

for most substances and complications are common, especially pulmonary aspiration. It is contraindicated if strong acids, alkalis or petroleum distillates have been ingested. Use may be justified for life-threatening overdoses of those substances that are not absorbed by activated charcoal

3. Whole bowel irrigation

This involves the administration of large quantities of osmotically balanced polyethylene glycol and electrolyte solution (1–2 L/ hr for an adult), usually by a nasogastric tube, until the rectal effluent is clear. It is occasionally indicated to enhance the elimination of ingested packets of illicit drugs or slow-release tablets such as iron and lithium that are not absorbed by activated charcoal. Contraindications include inadequate airway protection, haemodynamic instability, gastrointestinal haemorrhage, obstruction or ileus. Whole bowel irrigation may precipitate nausea and vomiting, abdominal pain and electrolyte disturbances.

Urinary alkalinisation

If the urine is alkalinised (pH > 7.5) by the administration of sodium bicarbonate (e.g. 1.5 L of 1.26% sodium bicarbonate over 2 hrs) resulting in enhanced urinary excretion of salicylates and methotrexate. Urinary alkalinisation is currently recommended for patients with clinically significant salicylate poisoning when the criteria for haemodialysis are not met . It is also sometimes used for poisoning with methotrexate. Complications include alkalaemia, hypokalaemia, occasionally alkalotic tetany and hypocalcaemia may occur but is rare.

Haemodialysis and haemoperfusion

These techniques can enhance the elimination of poisons that have a small volume of distribution and a long half-life after overdose; use is appropriate when poisoning is sufficiently severe. The toxin must be small enough to cross the dialysis membrane (haemodialysis) or must bind to activated charcoal (haemoperfusion) . Haemodialysis can also correct acid–base and metabolic disturbances associated with poisoning .

Lipid emulsion therapy

Lipid emulsion therapy is increasingly used for poisoning with lipid-soluble agents, such as local anaesthetics, tricyclic antidepressants, calcium channel blockers and lipid-soluble β-adrenoceptor antagonists (β-blockers) such as propranolol. It involves intravenous infusion of 20% lipid emulsion (e.g. Intralipid).

Antidotes

7.9 Specific antidotes used to treat poisoning

Mechanism of action	Examples of antidote	Poisoning treated
Glutathione repleters	Acetylcysteine Methionine	Paracetamol
Receptor antagonists	Naloxone	Opioids
	Flumazenil	Benzodiazepines
	Atropine	Organophosphorus compounds Carbamates
Alcohol dehydrogenase inhibitors	Fomepizole Ethanol	Ethylene glycol Methanol
Chelating agents	Desferrioxamine	Iron
	Hydroxocobalamin Dicobalt edetate	Cyanide
	DMSA Sodium calcium edetate	Lead
Reducing agents	Methylthioninium chloride	Organic nitrites
Cholinesterase reactivators	Pralidoxime	Organophosphorus compounds
Antibody fragments	Digoxin Fab fragments	Digoxin

(DMSA = dimercaptosuccinic acid)