

## COMMON POISONS

### Principles in approach to poisoning

- there is no role for the use of emetics in the modern treatment of poisoning.
- the use of activated charcoal for reducing drug absorption should be considered if patient presents within 1 hour of ingestion. A single dose of 1g/kg body wt can be given by mouth or nasogastric tube within 1 hour of ingestion of a well charcoal absorbed poison and > 1 hour in the case of a slow release drug preparation.
- gastric lavage not recommended unless patient has ingested a potentially life threatening amount of poison and procedure can be done within 1 hour of ingestion
- when in doubt about the nature of poison, contact the poison centre for help.

### Laboratory investigations

- a careful history may obviate the need for blood tests
- blood glucose should be taken in all cases
- blood gas analysis in any patient with respiratory insufficiency, hyperventilation or metabolic acid base disturbance is suspected
- electrolyte estimation may be useful as hypokalaemia may occur in acute poisoning
- routine measurement of paracetamol level should be performed in deliberate poisoning in the older child
- radiology may be used to confirm ingestion of metallic objects, iron salts
- ECG is of limited diagnostic value although prolonged PR and QRS in an unconscious patient should prompt a diagnosis of Tricyclic Antidepressant poisoning

### PARACETAMOL

Paracetamol is also called acetaminophen. Poisoning occurs when > 150mg/kg ingested. Fatality is unlikely if < 225mg/kg is ingested.

### Clinical Manifestations

- Clinical staging:

*Table 1. Clinical staging in paracetamol poisoning*

Stage 1 - nausea vomiting within 12-24 hours, some asymptomatic

Stage 2 - liver enzymes ↑ by 24 hours after ingestion; symptoms often abate

Stage 3 - liver enzymes abnormalities peak at 48-72 hours

- symptoms of nausea and vomiting, anorexia return

- clinical course results in recovery or hepatic failure

- there may be renal impairment

Stage 4 - recovery phase lasts 7-8 days

- most serious effect is liver damage which may not be apparent for the first 2 days

### Management

- communicating the diagnosis is preferably handled in private by a senior medical officer or specialist who is familiar with the natural history, genetic aspect and management of Down syndrome.
- careful examination to look for associated complications.
- investigations: 1. echocardiogram by 2 weeks (if clinical examination or ECG were abnormal) or 6 weeks. 2. Chromosomal analysis. 3. T4 /TSH at birth or by 1-2 weeks of life.

## Management

- measure plasma paracetamol level at 4 hours after ingestion and then 4 hourly.  
Other investigations: RBS/LFT/PT/PTT/RFT daily for 3 days
- initiation of N-Acetylcysteine (NAC) within 10 hours of ingestion; it is still beneficial up to 24 hours of ingestion
  - IV NAC if 4 hour plasma paracetamol level > 150µg/ml.  
Give 150mg in 200mls D5 over 15 min,  
followed by 50mg/kg in 500mls D5 over 4 hours,  
then 100mg/kg in 500mls D5 over 16 hours.
  - NAC is less effective if given > 15 hours after ingestion. Decision is based on the Rumack- Matthew nomogram (Figure 1). Plot the serum level of paracetamol drawn at least 4 hours following ingestion.
  - for patients who are already taking enzyme-inducing drugs, they should be given NAC if the levels are 50% or more of the standard reference line.
- If no blood levels are available, start treatment based on clinical history. Therapy can be stopped once level obtained is confirmed in the non toxic range.
- ensure NAC is appropriately diluted and patient does not become fluid overloaded
- if PT ratio > 3.0, give IM Vitamin K 1- 10mg. Fresh frozen plasma or clotting factor concentrate may be necessary.
- treat complications of acute hepatorenal failure.

## Prognosis

- Without treatment,  $\frac{2}{3}$  will develop severe liver and/or renal damage and 5 % will die
- If treatment given within 15 hours of ingestion, prognosis is excellent

## SALICYLATE

Ingestion of > 0.15 mg/kg will cause symptoms; fatal dose is 0.2 - 0.5g/kg.

Its main effects are as a metabolic poison causing metabolic acidosis and hyperglycaemia.

- clinical features
  - general: hyperpyrexia, profuse sweating and dehydration
  - CNS: delirium, seizures, cerebral oedema, coma, Reye's syndrome
  - respiratory: hyperventilation
  - gastrointestinal: epigastric pain, nausea, vomiting, upper gastrointestinal bleeding, acute hepatitis
  - renal: acute renal failure
  - metabolic: hyper/hypoglycaemia, anion gap metabolic acidosis, hypokalaemia
  - cardiovascular: non-cardiogenic pulmonary oedema
- investigations
  - FBC, PCV; BUSE/Serum creatinine; LFT/PT/PTT, RBS; ABG
  - serum salicylate level at least 6 hours after ingestion

## Management

- use activated charcoal and alkalinisation to enhance elimination.  
Dose of activated charcoal is 1-2g/kg/dose 4-8 hourly.
- Correct dehydration, hypoglycaemia, hypokalaemia, hypothermia, metabolic acidosis
- Give vitamin K if there is hypoprothrombinaemia.
- Plot the salicylate level on the normogram (Figure 2)
- Forced alkaline diuresis - see Table 2.
- haemodialysis if :
  - severe cases, blood level > 100mg/dl
  - refractory acidosis
  - renal failure
  - non-cardiogenic pulmonary oedema
  - severe CNS symptoms e.g. seizures

## Prognosis

The presence of coma, severe metabolic acidosis together with plasma salicylate concentrate > 900mg/L indicate a poor prognosis even with energetic treatment.

**Table 2. Forced alkaline diuresis**

<p><b>Indication:</b> for moderate to severe cases (salicylate level &gt; 35 mg/dl 6 hrs after ingestion) <i>Needs close monitoring as it is potentially dangerous</i></p> <ol style="list-style-type: none"><li>1. give 30mls/kg in 1st hour (<math>\frac{1}{2}</math> saline/D5% + 1ml/kg 8.4% NaHCO<sub>3</sub>).</li><li>2. continue at 10mls/kg/hr till the salicylate level is at the therapeutic range.</li><li>3. give IV frusemide (1mg/kg/dose) after 1st hr and then 8hrly.</li><li>4. add 1g KCl to each 500mls drip solution, to the above regime (discontinue KCl if serum K<sup>+</sup> &gt; 5mmol/L).</li><li>5. aim for plasma pH of &gt;7.5 and urine output of &gt; 3-6ml/kg/h.</li><li>6. monitor BUSE/RBS/ABG every 6 hrs.</li><li>7. treat hypoglycaemia (5ml/kg of 10% dextrose)</li></ol>
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## IRON

Dangerous dose of iron can be as small as 30mg/kg. The toxic effect of iron is due to unbound iron in the serum.

- Clinical features

Table 3. Clinical staging in iron poisoning

Stage 1 (6-12 hrs)	gastrointestinal bleeding, vomiting, abdominal pain, diarrhoea, hypotension, dehydration, acidosis and coma
Stage 2 (8-16 hrs)	symptom free period but has nonspecific malaise
Stage 3 (16-24 hrs)	profound hemodynamic instability and shock
Stage 4 (2-5 wks)	liver failure and gastrointestinal scarring with pyloric obstruction

## Management

- ingestion < 30mg/kg - patients are unlikely to require treatment
- ingestion > 30mg/kg - perform an abdominal XRay:
  - if pellets seen then use gastric lavage with wide bore tube
  - if pellets seen in small bowel then whole bowel irrigation with polyethylene glycol  
volume to use: 500ml/h in children < 6 yrs, or  
1000ml/h in children 6-12 yrs. or  
1500 – 2000ml/h in children >12 yrs old  
(contraindications: paralytic ileus, significant hematemesis and hypotension)
- blood should be taken at 4 hrs after ingestion
  - if level < 55µmol/l : unlikely to develop toxicity
  - if level 55-90µmol/l, observe for 24 - 48 hrs.  
Chelate if symptomatic (haematemesis or malaena)
  - if level > 90µmol/l or significant symptoms, chelate with  
IV Desferrioxamine 15 mg/kg till max of 80 mg/kg in 24 hours

### Notes:

1. If serum iron is not available, severe poisoning is indicated by nausea, vomiting, leucocytosis  $>15 \times 10^9$  and hyperglycaemia  $> 8.3 \mu\text{mol/l}$
2. Administer Desferrioxamine with caution because of hypotension and pulmonary fibrosis. Continue chelation therapy till serum iron normal, metabolic acidosis resolved and urine colour returns to normal.
3. Critical care management: Focus on cardiopulmonary failure, hypotension, severe metabolic acidosis, hypoglycaemia or hyperglycaemia, anaemia, gastrointestinal bleeding, liver and renal failure.

## Prognosis

Gastrointestinal bleeding, hypotension, metabolic acidosis, coma and shock are poor prognostic features.

## KEROSENE AND HYDROCARBON INGESTION

- **strict contraindication** to doing gastric lavage and emesis: it increases risk of chemical pneumonitis.
- admit the child for observation for respiratory distress and treat symptomatically.
- cerebral effects may occur from hypoxia secondary to massive inhalation.
- antibiotics and steroids may be useful in lipoid pneumonia (esp. liquid paraffin).
- chest X-ray

## TRICYCLIC ANTIDEPRESSANTS

- clinical features
  - anticholinergic effects: fever, dry mouth, mydriasis, urinary retention, ileus
  - central nervous system: agitation, confusion, convulsion, drowsiness, coma
  - respiratory system: respiratory depression
  - cardiovascular system: sinus tachycardia, hypotension, complex arrhythmias

### Management

- there is no specific antidote. Give activated charcoal 1-2 g/kg/dose 4-8 hourly.
- continuous ECG monitoring. Meticulous monitoring required: If no QRS widening, cardiac conduction abnormality, hypotension, altered sensorium or seizures within the 6 hours; it is unlikely the patient will deteriorate.
- treatment should be instituted for prolonged QRS and wide complex arrhythmias. QRS > 100ms (seizures) and QRS >160ms (arrhythmia).
- correct metabolic acidosis. Give bicarbonate (1-2mmol/kg) to keep pH 7.45 - 7.55 when QRS is widened or in the face of ventricular arrhythmias. Administration of NaHCO<sub>3</sub> is targeted at narrowing the QRS and is titrated accordingly by bolus or by infusion. Watch out for hypokalemia and treat accordingly.
- convulsions should be treated with diazepam.
- use propranolol to treat life-threatening arrhythmias.
- if *torsades de pointes* occurs treat with MgSO<sub>4</sub>
- treat hypotension with Norepinephrine. Dopamine is not effective.
- haemodialysis/PD is not effective as tricyclics are protein bound
- important to avoid the use of flumazenil for reversal of co-ingestion of benzodiazepines as this can precipitate tricyclic induced seizure activity

## ORGANOPHOSPHATES

- clinical features:
  - cholinergic effects: miosis, sweating, lacrimation, muscle twitching, urination, excessive salivation, vomiting, diarrhoea
  - central nervous system: convulsions, coma
  - respiratory system: bronchospasm, pulmonary oedema, respiratory arrest
  - cardiovascular system: bradycardia, hypotension

### Management

- Remove contaminated clothing and wash exposed areas with soap and water.
- gastric lavage and give activated charcoal.
- resuscitate the patient. Protect the airway by early intubation. Use only non depolarising neuromuscular agents
- IV atropine 0.05mg/kg every 15 minutes till fully atropinized. Atropine administration is guided by drying of secretions rather than heart rate or pupil size.
- keep patient well atropinized for the next 2-3 days.
- a continuous infusion of atropine can be started at 0.05mg/kg/hr and titrated.
- give IV pralidoxime 25-50mg/kg over 30 min, repeated in 1-2 hrs and at 10-12 hr intervals as needed for symptom control (max 12g/day) till nicotinic signs resolves.
- treat convulsions with diazepam.
- IV furosemide for pulmonary congestion after full atropinisation.
- a rapid sequence intubation involves the potential for prolonged paralysis.

## PARAQUAT

- clinical features:
  - ulcers in the mouth and oesophagus
  - diarrhoea and vomiting
  - jaundice and liver failure
  - renal failure

## Management

- remove contaminated cloth and wash with soap and water.
- gastric lavage till clear.
- to give Fuller's earth in large amount.
- general supportive care.

## CHRONIC LEAD POISONING

This is an important diagnosis to be considered in any child who has raised intracranial pressure or encephalopathy.

- clinical features:
  - usually no history of ingestion
  - colicky abdominal pain, constipation, lethargy, anaemia, drowsiness, vomiting, headache, fits, coma due to encephalopathy.
  - behavioural change
- investigations
  - increase blood lead levels  $> 80\mu\text{g}/100\text{ml}$ .
  - lead lines (lines of increased density) at growing ends of long bones.
  - basophilic stippling of red cells.
  - increased coproporphyrin urinary excretion.

## Management

- identify source and prevent further ingestion.
- decrease cerebral oedema. Use dexamethasone  $0.2\text{--}0.4\text{mg}/\text{kg} \pm \text{IV}$  mannitol.
- chelating agents:

The 2 agents used are IV EDTA ( $50\text{mg}/\text{kg}/\text{day}$  in divided 4 hourly doses) and oral 2,3 dimercaptosuccinic acid (DMSA ).

(Older agents (BAL and penicillamine) are rarely used now)
- when to treat:
  - if blood level  $> 750\mu\text{g}/\text{l}$  admit to PICU for urgent chelation
  - if blood level  $> 450\mu\text{g}/\text{l}$  treat with oral DMSA
  - if blood level  $< 450\mu\text{g}/\text{l}$  treatment has no effect on outcome