Cyanide (CN)

Significant cyanide exposure causes precipitous cardiovascular collapse and seizures. Antidotes are most effective when administered shortly after exposure.

Toxicity / Risk Assessment

Sources: Hydrogen cyanide gas (HCN) / salts

Industry – mining, manufacture of plastics

House fires – burning of plastics / polyurethane

(note: coexisting CO exposure is common)

Plants - amygdalin (apricot kernels, almonds)

- large quantities required to cause toxicity

Others - warfare agents, fumigants, prolonged sodium nitroprusside infusions (> 24 hours)

Acetonitrile - metabolised to cyanide (delayed onset of toxicity), absorbed dermally

Potentially lethal dose: 1 mg/kg of cyanide salt

Clinical features:

- Onset of clinical toxicity is within 30 minutes

Mild toxicity: headache, vomiting, anxiety, tachycardia, dizziness, mild dyspnea

Moderate to severe: collapse, seizures, respiratory distress, hypotension, confusion, severe metabolic acidosis, CVS collapse, arrhythmias, death

Management

Inhalation of HCN gas commonly leads to death at the scene of exposure. Supportive care and early administration of an antidote in critically unwell patients are Rx priorities. Survival to hospital portends a good prognosis.

Decontamination: There is no role for decontamination once patient has been removed from source of HCN gas

Administer 50 g AC to patients who have ingested cyanide salts within the previous 2 hours

Airway: Early intubation and administration of 100% O_2

Hypotension: Treat initially using ANTIDOTES (see below) and a bolus of intravenous crystalloid (20-30 mL/kg)

Investigations: Blood [cyanide] can be measured, but is not available rapidly enough to aid management

- Cyanide poisoning produces a raised anion-gap metabolic acidosis with a raised lactate
- A lactate of >10 mmol/L following a house fire in the absence of significant burns is suggestive of cyanide toxicity
- A difference between arterial O_2 saturation % and venous O_2 saturation % < 10 is suggestive of cyanide toxicity (venous O_2 saturation % > 90% is consistent with an inability to utilise oxygen at a tissue level)

ANTIDOTES: ***(discuss with a clinical toxicologist and see separate Hydroxocobalamin / Sodium thiosulfate guideline)***

[Mild toxicity: Hydroxocobalamin **OR** Na thiosulphate] [*Mod-severe toxicity:* Hydroxocobalamin **AND** Na thiosulphate]

Hydroxocobalamin (Cyanokit®): 5 g in 200 mL N/saline over 15 mins (slow IV push if critically unwell).

Paediatric dose: 70mg/kg up to 5 g diluted in 0.9% sodium chloride 2.8ml/kg up to 200ml over 15 minutes.

Sodium thiosulfate: 50 mL sodium thiosulfate 25% (12.5 g) IV over 10 mins. Can be repeated after 30-60 mins.

Paediatric dose: 1.6 mL/kg (max 50 mL) sodium thiosulfate 25% (400 mg/kg up to 12.5g) IV over 10 minutes

Disposition: asymptomatic 4-hours post exposure can be discharged pending mental health assessment

- Patients with severe toxicity, or those requiring treatment with an antidote should be admitted to a critical care bed