

## Overview

Rare but potentially rapidly lethal poisoning - coma, shock, seizures & profound lactic acidosis.

## Toxic mechanism

Inhibits oxidative phosphorylation by binding to  $\text{Fe}^{3+}$  of cytochrome oxidase → lactic acidosis. Also pulmonary & coronary vasoconstriction, and in the CNS, triggers NMDA release & seizures.

## Toxicokinetics

Rapidly abs, small Vd & protein bound. Probably met in liver to thiocyanate by rhodanese and elim in urine with  $T_{\frac{1}{2}}$  2-3h.

## Clinical features

If exposure is to hydrogen cyanide gas (HCN) then rapid loss of consciousness.

May smell of bitter almonds (not all people can detect this)

Ingestion of cyanide salts → N&V, headache, dyspnoea, ↑BP, ↑HR, agitation, collapse, seizures.

May progression to: ↓BP, ↓HR, confusion, tetany, ↓RR, coma & death

Delayed neurotoxicity (Parkinsonism) after weeks/months may occur.

## Investigations

*Screening:* ECG (early sinus tachy most common, later bradycardia), paracetamol, BSL

*Specific bloods:* EUC, ABG (AG met acidosis, ↑lac [corresponds with sev], venous  $\text{PO}_2$  &  $\text{SvO}_2$  approach arterial values as  $\text{O}_2$  cannot be utilised), CN level (not avail for acute Mx)

Cyanide Level	Clinical effect
>20 $\mu\text{mol/L}$ (0.05mg/dL)	Symptomatic
>40 $\mu\text{mol/L}$ (0.1mg/dL) or lactate >10mmol/L	Potentially toxic
>100 $\mu\text{mol/L}$ (0.26mg/dL)	Lethal

*Other:* FBC

## Risk assessment

Time critical response req. Immediate threat to life. If patient reaches hospital before arrest → good chance of survival with supportive care. Chronic exposure → non-specific symptoms.

## Management

*Resus:* ABCs. **Immediate** intubation and ventilation with 100%  $\text{O}_2$  if severe poisoning.

*Supportive Care*

*Decontamination:* Remove from source if HCN exposure. Bag clothing. Wash skin with soap & water. Charcoal may be given after intubation.

*Antidotes:* Needed only if significant CN ingestion likely (↓LOC, ↓BP, seizures, metabolic acidosis) then give cyanide antidote - **hydroxocobalamin, sodium thiosulphate, dicobalt EDTA, amyl/sodium nitrite**. (See Antidotes)

## Disposition

If clinically well at 4h → d/c. Otherwise admit and if any significant exposure will be intubated so → ICU.

## Notes

HCN used industrially as fumigant, also nitriles used in manufacture of plastics. Cyanide compounds, incl HCN, produced in fires. Na nitroprusside therapy can lead to CN toxicity.