

Overview

Common lethal poisoning due to tissue hypoxia. Symptoms non-specific

Toxic mechanism

Hb binding affinity 210x that of O₂ → hypoxia. Also binds to cellular cytochromes and initiates endothelial oxidative injury and inflammatory cascade.

Toxicokinetics

T_½ depends on pO₂. In room air: 4hr, 100% O₂: 90min, hyperbaric O₂ at 3atm: 23min.

Clinical features

CNS: headache, N&V, dizziness, confusion, poor conc, cerebellar signs, seizures, syncope, coma

CVS: ↑HR, ↑BP, ischaemic ECG or MI, dysrhythmias, ↓BP

Resp: Non-cardiogenic APO

Metabolic: Lactic acidosis, rhabdo, ↑BSL

Other: myalgia, DIC, bullae, alopecia, sweat gland necrosis, neurological & neuropsychiatric SE.

Investigations

Screening: ECG, paracetamol, BSL

Specific bloods: ABG, CO-Hb, lactate, β-hCG, FBC, UEC

COHb Level	Effect
<10%	Background level in smokers
10%	Asymptomatic, slight headache
20%	Dizziness, nausea, dyspnoea, throbbing headache
30%	Vertigo, ataxia, visual disturbance
40%	Confusion, coma, seizures, syncope
50%	CVS & resp failure, arrhythmia, seizures, death

Other as indicated: Trop/CK, CT/MRI brain, neuropsychiatric testing at 3-12mo.

Risk assessment

Deaths occur prehospital, however morbidity may be affected by hospital Rx.

Higher risk for sequelae when low conc prolonged exposure (accidental) than high conc short duration (car exhaust parasuicide).

Other high risk factors:

- Significant LOC/coma
- Persistent neurological dysfunction or confusion
- Cerebellar signs
- Metabolic acidosis
- Myocardial ischaemia
- Age >55y
- Fetus

Loose correlation with COHb level.

Management

Resus & Supportive Care: ABCs.

Decontamination: Remove source.

Enhanced Elimination:

- Normobaric O₂ until asymptomatic (24h if pregnant) - no evidence ↓sequelae.
- Consider hyperbaric O₂ if high risk. Controversial (proven benefit, end point, & avail)

Disposition

D/C when asymptomatic. If end organ ischaemia → HDU/ICU. Neuropsych follow up at 1-2mo.