Carbon Monoxide (CO)

CO exposure causes tissue hypoxia and a multi-faceted inflammatory response, producing acute organ injury and chronic neurological dysfunction.

Toxicity / Risk Assessment

CO toxicity is more likely to occur in a poorly ventilated, enclosed space in which there is a prolonged period of CO generation

Sources: *vehicle exhaust fumes, domestic/industrial fires, cigarette (up to 10-15%) or hookah smoking (up to 30%), machinery emitting CO in an enclosed space, incomplete combustion of carbonaceous materials in an enclosed space (e.g., charcoal briquettes, faulty heating devices), exposure to methylene chloride*

Clinical features:

Vary from mild and non-specific through to life-threatening:

General - weakness, nausea, vomiting, headache

CNS - Dizziness, ataxia, confusion, coma

CVS - sinus tachycardia, atrial fibrillation, PVCs, ventricular arrhythmias, myocardial infarction, pulmonary oedema

Severe complications: - rhabdomyolysis, renal failure, hepatic injury

Delayed neurological sequelae

- Appear after days-months following exposure. (impaired judgment / memory / concentration, dementia)
- More likely with LOC, existing cerebrovascular disease, prolonged exposures, focal neuro. deficits post exposure

Investigations

COHb concentration (co-oximetry measurement via venous blood gas sample. ABG not necessary)

- COHb concentration correlates poorly with clinical features in the ED, severity of poisoning + outcome
- A normal COHb concentration (0.5-5.0%) measured in ED does not exclude CO poisoning

ECG / troponin: measure if there has been LOC, CVS symptoms (e.g., chest pain) or CVS instability

CT / MRI brain: may detect changes in caudate nuclei, globus pallidus, basal ganglia and putamen

- Should not be ordered routinely, but may aid in cases of poor Rx response or diagnostic dilemma

Management

Patients with altered conscious state or CVS instability should be managed in a resuscitation area

Provision of high concentration oxygen (O_2) ASAP is the mainstay of management:

- Intubated patients: FiO_2 100% for at least 6 hours and COHb concentration < 5%
- Non-intubated patients: O_2 at 15L/minute via tight fitting non-rebreather mask with reservoir bag

Endpoint of O_2 Rx: at least 6 hours duration, COHb concentration < 5% + asymptomatic

Hyperbaric oxygen (HBO) therapy is controversial, and clear evidence of benefit is lacking

Discuss patients with abnormal ECG, troponin or neuroimaging and those with an altered conscious state, focal neurological deficits, CVS instability or pregnancy with a clinical toxicologist

Disposition: altered conscious state or, CVS instability: manage in an HDU / ICU setting

Patients with normal conscious state AND COHb concentration < 5% after 6 hours of O_2 Rx can be discharged pending mental health assessment as required. *Neuropsychiatric assessment post recovery from acute exposure may be beneficial in selected cases (e.g., occupational exposures / severe toxicity)*