POISINDEX(R) Toxicologic Management

Topic: CARBON MONOXIDE

0.0 OVERVIEW

0.1 LIFE SUPPORT

This overview assumes that basic life support measures have been instituted.

0.2 CLINICAL EFFECTS

0.2.1 SUMMARY OF EXPOSURE

A. Carbon monoxide poisoning causes a multitude of effects due to tissue hypoxia and possibly cellular poisoning.

0.2.3 HEENT

A. Retinal findings, visual field deficits, or retrobulbar neuritis may occur.

0.2.4 CARDIOVASCULAR

A. Atrial and ventricular arrhythmias, heart block, and EKG changes suggestive of cardiac ischemia may occur.

0.2.6 NEUROLOGIC

A. Headache, dizziness, seizures, coma, and death.

B. Delayed effects may occur 48 to 72 hours following exposure.

0.2.7 GASTROINTESTINAL

A. Nausea and vomiting are common. May mimic acute gastroenteritis or food poisoning.

0.2.14 HEMATOLOGIC

A. Carboxyhemoglobin level may be elevated initially. Diminishes rapidly following carbon monoxide source removal and institution of oxygen therapy, which greatly reduces the half life of COHb.

B. COHb level correlates poorly with symptoms, especially if measured after oxygen initiated.

0.2.18 PSYCHIATRIC

A. Neuropsychiatric disorders, usually personality changes and impairment of memory may occur after a delay of 1 to 3 weeks.

0.2.19 PREGNANCY/BREAST MILK

A. CO exposure during pregnancy is teratogenic.

0.2.22 OTHER

A. High-risk groups include infants, pregnant women, the elderly, and patients with a history of ischemic heart disease or chronic obstructive lung disease.

0.3 LABORATORY

A. Determine COHb level when the patient is first seen and repeat every 2 to 4 hours until less than 15% in symptomatic patients.

B. Monitor EKG, electrolytes, CPK, urinalysis, arterial blood gases if symptomatic or if the COHb level is greater than 20%.

C. Monitor cardiac function.

D. CT scan or MRI should be considered if neurologic symptoms develop or persist.

0.4 TREATMENT OVERVIEW

0.4.3 INHALATION EXPOSURE

A. DECONTAMINATION: Move patient to fresh air. Monitor for respiratory distress. If cough or difficulty in breathing develops, evaluate for respiratory tract irritation, bronchitis, or pneumonitis. Administer 100 percent humidified supplemental oxygen with assisted ventilation as required.

B. ADMINISTER 100% OXYGEN by tight-fitting face mask to reduce the biological half-life of CO.

C. CARBOXYHEMOGLOBIN (COHb) LEVEL:

1. Determine when the patient is first seen and repeat at 2 to 4 hourly intervals until less than 15% (0.15).
2. A COHb level should be obtained prior to oxygen therapy as long as this does not delay the initiation of this treatment.
3. COHb concentrations frequently do not correlate well with the severity of the poisoning. In an appropriate setting, any patient found unconscious, seizing, or with EKG changes or metabolic acidosis should be treated as a severe carbon monoxide poisoning, regardless of the COHb concentration.

D. CONSIDER HYPERBARIC OXYGEN therapy for severely poisoned patients.

E. SEIZURES: Administer diazepam IV bolus (DOSE: ADULT: 5 to 10 mg initially which may be repeated every 15 minutes PRN up to 30 mg. CHILD: 0.25 to 0.4 mg/kg dose up to 10 mg/dose) or lorazepam IV bolus (DOSE: ADULT: 4 to 8 mg; CHILD: 0.05 to 0.1 mg/kg).

1. If seizures are uncontrollable or recur, give phenobarbital and/or phenytoin unless these agents are involved in the poisoning.

F. EKG: Indicated in adult patients found unconscious, with chest pain or other significant symptoms, or with COHb level greater than 20%, to detect signs of myocardial damage.

G. CEREBRAL EDEMA

1. Patients with signs of increased intracranial pressure should be hyperventilated with 100% oxygen via an endotracheal tube to keep the arterial pCO2 level at 25 to 30 mmHg.
2. Parenteral fluids should be limited to 2/3 to 3/4 of normal maintenance. Osmotic diuretics (eg, mannitol) or other methods to reduce intracranial pressure may be used but are unlikely to affect outcome.

H. ADMISSION CRITERIA: Neurological symptoms or signs, abnormal EKG, metabolic acidosis, COHb level above 30% (0.3).

0.5 RANGE OF TOXICITY

A. COHb levels above 30% (0.3) are usually considered toxic,

although COHb levels do not correlate well with clinical severity; patients may have significant toxicity with mildly elevated or even normal COHb levels, particularly if they were obtained after oxygen therapy.

B. Toxicity is best determined by the patient's cardiovascular and mental status, arterial pH and bicarbonate levels.