InBrief

Carbon Monoxide Poisoning

Exposure to carbon monoxide (CO), a colorless, odorless gas, can cause significant toxicity and is the most common cause of fatal poisoning in the United States. Exposure can be accidental or intentional as part of a suicide attempt.

Signs and Symptoms

As CO poisoning can present subtly, a high level of suspicion and good history-taking may help make the diagnosis. This is particularly true in mild cases where presentation is nonspecific and the cause is not obvious. A history of other people or pets having similar symptoms or a history of using devices that produce CO can be helpful.

Mild CO poisoning can present with headache, flu-like symptoms without fever, or nausea/vomiting.

Chronic CO poisoning may cause more insidious symptoms, such as trouble concentrating, personality changes, or memory loss. More severe intoxication can cause chest pain, ataxia, seizures, syncope, focal neurologic deficits, confusion, visual disturbances, retinal hemorrhages, bullous skin lesions, dyspnea, coma, and respiratory or cardiac arrest.

CO poisoning should be considered in comatose patients with an unexplained elevated anion gap metabolic acidosis or lactic acidosis. If the patient has been in a fire and has lactate levels > 10 mmol/L, coexisting cyanide poisoning should be considered.

The cherry-red color change of skin and oral mucosa classically described for CO poisoning is rarely seen in living patients.

Causes and Risk Factors

CO is produced during house or building fires; use of wood/charcoal/propane/gas heaters or stoves, natural gas-powered motors, generators and furnaces, gasoline powered generators and motors, and industrial equipment; and from car and boat exhaust.

CO poisoning typically occurs indoors in a poorly ventilated space. However, a leaky or clogged exhaust system on a vehicle or boat can produce symptoms outdoors.

Methylene chloride is a substance used in Christmas bubble lights, varnishes, and paint strippers. It is converted by the liver to CO and can cause prolonged toxicity when inhaled or ingested.

Adverse effects

The affinity of hemoglobin for CO is 200 times that of oxygen. CO displaces oxygen from the hemoglobin molecule and binds to hemoglobin to form carboxyhemoglobin (COHb), resulting in hypoxemia. The half-life of COHb in room air averages 240-320 minutes and can vary depending on the amount of respiration. On 100% oxygen, the half-life is about 80 minutes. In methylene-chloride exposure, the half-life can be up to 13 hours due to continued CO production.

COHb shifts the oxygen dissociation curve to the left; hemoglobin will hold on to oxygen molecules more tightly than normal rather than delivering it to the tissues, which exacerbates the tissue hypoxemia already caused by the CO.

CO results in a relative uncoupling of oxidative phosphorylation and causes lactic acidosis. It also causes release of guanylate cyclase and nitric oxide, which can cause hypotension. A cellular inflammatory process involving white blood cells and release of free radicals can also occur. The hypoxia, hypotension, and inflammation can lead to cell injury or death. The basal ganglia and globus pallidus are extremely sensitive to the effects of CO toxicity.

In pregnant women, even mild CO poisoning can affect the fetus, potentially causing fetal demise or congenital malformations, since fetal hemoglobin has a stronger affinity for CO than does adult hemoglobin.

Adverse outcomes

Survivors of CO poisoning can suffer from long-term neurocognitive sequelae, including impaired memory, cognitive dysfunction, depression, anxiety, or vestibular and motor deficits.

About one-third of patients may have subtle memory deficits or show personality changes after CO poisoning.

Pearl to Know

CO is produced in the body during the normal breakdown of heme. Normal physiologic CO levels are about 1% in nonsmokers, whereas smokers can have levels up to 10%.