Carbon Monoxide Poisoning and Pregnancy: Critical Nursing Interventions

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In 2011, 13,862 people were reportedly exposed to carbon monoxide (CO).[1] The Centers for Disease Control and Prevention estimates that CO poisoning is responsible for more than 20,000 ED visits, 4000 hospitalizations, and 400 deaths annually.[2] Because of the vague symptoms caused by CO poisoning, underreporting of exposures and misdiagnosing of patients makes calculation of the frequency of pregnant women exposed to CO especially difficult. The incidence of CO poisoning in pregnancy is believed to be between 4.6% and 8.5%.[3]

Understanding the pathophysiology, signs and symptoms, and treatment options for CO poisoning is important for optimal management and improving outcomes for pregnant women exposed to CO. Failing to perform a thorough assessment can lead to a missed diagnosis with ramifications that can affect an entire family system. It can necessitate hospitalization, increase stress levels for the patient and his or her loved ones, cause financial stress from expensive hospital bills and missed days of work, and cause long-term neurologic damage that can impair memory and cognitive functioning. Pregnant women exposed to CO have additional stressors. The mother may feel anxious for the duration of the pregnancy because of concerns about the well-being of the fetus. Emergency nurses play a critical role in identifying and treating pregnant women to prevent these outcomes.

Pathophysiology

CO is a colorless and odorless gas produced from the incomplete combustion or burning of fossil fuels such as oil, gas, or coal.[2] Common sources of CO poisoning include oil or gas furnaces and wood-burning stoves, oil or gas water heaters, gas stoves, gas or charcoal grills, kerosene heaters, gasoline powered tools, lawn equipment, automobile exhaust, generators, and house fires.

Once inhaled, CO easily diffuses across the alveolar membrane into the blood, where it binds with hemoglobin and creates a complex called carboxyhemoglobin (COHb). CO has an affinity to hemoglobin that is 200 to 250 times that of oxygen, but fetal hemoglobin (HbF) has an even higher affinity to CO than adult hemoglobin (HbA).[4]

A COHb complex that also has oxygen bound to it has a decreased capacity to release the oxygen to tissues and causes a left shift of the oxyhemoglobin dissociation curve.[5] The partial pressure of maternal arterial oxygen (Pao_2) is 100 mm Hg compared with the fetal Pao_2, which is about 20 mm Hg.[6] Because fetal Pao_2 levels are already so low, even a small shift of the oxyhemoglobin dissociation curve can result in a great insult to the fetus.[3] The combination of decreased oxygen bound to hemoglobin and reduced ability to release oxygen that is bound can lead to permanent organ and brain damage.

Intracellularly, CO binds to the cytochrome c oxidase enzyme of the electron transport chain and stops adenosine triphosphate production. The disturbance of the electron transport chain causes a cascade effect, producing free radicals and oxidative stress that damages blood vessel walls. White blood cells adhere to the damaged vessel walls and release more free radicals, causing lipid peroxidation and inflammation and further disrupting capillary integrity.[4] Nitric oxide is released, causing vasodilation that reduces venous return and myocardial perfusion.[7] Decreased myocardial perfusion increases the risk for cardiac damage and a life-threatening cardiac event.

Studies of pregnant sheep with CO poisoning demonstrated that fetal oxygen tensions began to decrease when maternal COHb levels began to increase, but the uptake of CO to the fetus lagged behind the mother for the first 4 to 5 hours of an exposure. However, because HbF has a much higher affinity to CO, after 5 hours of exposure, fetal COHb (COHbF) levels exceeded that of the mother.[8] Depending on the duration of the exposure, COHbF levels exceeded that of the mother by as much as 58%, and the fetal half-life of CO was nearly 4 times longer.[8] However, these figures remain unknown in humans.

Mortality rates from acute CO poisoning in pregnant adults is associated with COHb levels between 19% and

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24%. In fetuses, mortality rates have been estimated to be between 36% and 67%.[5] Unfortunately, no method is available to determine COHb levels of a fetus in utero, and maternal well-being can be a misleading prediction of fetal well-being.[3]

Signs and Symptoms

Signs and symptoms of CO poisoning can be vague and difficult to diagnose. Mild symptoms may be flu-like, including dizziness, disorientation, weakness, nausea, and vomiting. Ninety percent of patients report having a headache.[6] In severe CO exposures, hypertension may be a sign of renal failure caused by red blood cell destruction. Additional symptoms of severe exposure include hypotension; tachycardia, tachypnea, dysrhythmias, and other electrocardiogram changes; coma; and death.[9]

A single episode of hypoxia from CO can be teratogenic to the fetus, with permanent consequences.[3] In the first trimester, anatomic malformations such as skeletal abnormalities and limb malformations are more frequently seen.[3] Exposures in the second and third trimesters have greater effects on fetal brain development and can cause anoxic brain damage, Down syndrome, cerebral palsy, and psychomotor and mental development functional changes.[3] When assessing the status of the fetus, the fetal heart rate tracing can provide clues of fetal distress. An increased baseline fetal heart rate can indicate fetal hypoxemia. Fetal heart rate variability is also an indication of fetal oxygenation status, and moderate variability is reassuring. Fetal heart rate accelerations and decelerations measure uteroplacental sufficiency; heart rate accelerations are a reassuring sign of a healthy pregnancy.[10] After CO exposure, an elevated baseline fetal heart rate, decreased heart rate variability, absence of accelerations, and an absence of decelerations are signs of fetal distress.[11]

Treatment

Hyperbaric oxygen therapy (HBOT) is recommended by the Undersea and Hyperbaric Medical Society for the treatment of acute CO poisoning.[12] The half-life of CO when breathing room air is 320 minutes but reduces to 23 minutes when breathing 100% oxygen at 3 atmospheres absolute (ATA) in the hyperbaric chamber.[6] Also at 3 ATA, CO dissociates from hemoglobin at a much faster rate, cytochrome c oxidase function is restored within minutes, lipid peroxidation is inhibited, and cardiovascular function improves.[6] Patients who receive HBOT for CO poisoning are reported to have a 25% lower incidence of long-term neurologic sequelae compared with patients who do not receive HBOT.[13]

When evaluating the benefits and risks of using a hyperbaric chamber to treat a pregnant woman who has been exposed to CO, several animal studies have concluded that theoretical risks to the fetus are associated with HBOT. These theoretical risks include retinopathy, prematurity, premature closure of the ductus arteriosus, retinal detachment, and skeletal malformations.[14] Extrapolation to human fetuses is difficult because these studies were conducted on small animals using treatment protocols that should not be used with humans. The conclusions from these studies were based on protocols that exceeded 3 ATA. Hyperbaric therapy with 100% oxygen at pressures greater than 3 ATA increases risk for oxygen-induced seizures.[15] Thus the conclusions from the animal studies do not reflect actual or safe clinical practice.

Several published case reports (Table) describe fetal outcomes after HBOT for pregnant women with CO poisoning and demonstrate that HBOT appears to be safe for the fetus. There may even be a greater potential risk of treating pregnant mothers with 15 L/min high-flow surface oxygen because the treatment duration required to drive CO off of HbF is significantly longer than when providing treatment with hyperbaric oxygen. In one of the cases, the patient required 16 hours of breathing 100% high-flow surface oxygen.[11] High fetal arterial partial pressures of oxygen for a long duration may impose greater risks to retinal development or premature closure of the ductus arteriosis than a short hyperbaric treatment. However, this issue requires further investigation.

Nursing Assessment and Interventions

Nurses have an important role in both identifying CO exposure and ensuring that patients receive the proper care. Symptoms of mild CO poisoning can be very vague and can mimic other illnesses such as the flu. Without an obvious history of present illness, it can easily be missed. Weaver[16] describes a case of a 39-year-old woman who went to multiple specialists after several months of experiencing headaches, fatigue, and memory lapses; she never received a diagnosis until a friend found her unconscious and brought her to the emergency department. It was then determined that she had CO poisoning from her furnace. Laing[9] describes another case of a 29-year-old patient who was found unconscious at home after failing to report to work. The patient was brought to the emergency department where a series of tests were completed before the patient was