ACCIDENTAL CARBON MONOXIDE POISONING WITH SEVERE CARDIORESPIRATORY COMPROMISE IN 2 CHILDREN

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Carbon monoxide is a colorless, odorless gas produced as a by-product of incomplete combustion. Carbon monoxide poisoning accounts for approximately 3500 deaths in the United States annually.1,2 Carbon monoxide in exhaust fumes from motor vehicles accounts for most deaths due to carbon monoxide poisoning; however, poorly functioning heating systems, inhaled smoke, and propane-operated forklifts also can cause carbon monoxide poisoning.3,4 We report 2 cases of severe cardiorespiratory compromise in 3-year-old identical twin girls that occurred after they were exposed for an estimated 12 to 18 hours to a faulty furnace producing high levels of carbon monoxide.

Case Reports

Case 1

S.G. was intubated at the scene because of poor respiratory effort and extensor posturing. The arterial carboxyhemoglobin level within 1 hour after discovery was 10.3%; 2 hours later, it was reduced to 1.3%. Pertinent laboratory values included a serum base deficit of 13.9, a prothrombin time of 17 seconds (normal, 10.6-11.4 seconds), a partial thromboplastin time of 47 seconds (normal, 24-36 seconds), and urine positive for myoglobin.

The patient was initially treated with volume resuscitation, fresh frozen plasma, cryoprecipitate, and alkalization to a urinary pH greater than 7.0. One hour after admission, hypotension necessitated the initiation of infusions of epinephrine and dopamine. The infusions were titrated to effect, with a maximum of 0.6 µg/kg per minute of epinephrine and 10 µg/kg per minute of dopamine. Echocardiography showed an ejection fraction of 0.22. Nine hours after admission, the serum level of cardiac troponin I was 6.6 µg/L (normal, <2 µg/L).5 Acute pulmonary edema developed and was treated with 100% oxygen, a maximum positive end-expiratory pressure of 16 cm H2O, and pressure-control inverse-ratio ventilation to maintain a PaO2 greater than 50 mm Hg and an oxygen saturation greater than 85%.

During the next several days, S.G.’s cardiovascular and respiratory status improved. Neurological findings remained a concern; S.G. had extensor posturing in response to noxious stimuli and no spontaneous movement. On day 5, she was successfully weaned off all infusing inotropic agents, and she was electively extubated on day 9. On day 11, supraventricular tachycardia developed and was initially managed with an infusion of procainamide hydrochloride and subsequently with oral propranolol. Follow-up echocardiography at this time showed an ejection fraction of 0.69. Results of neurological examination improved; S.G. had spontaneous eye opening and focused on family members.

S.G. was transferred from the pediatric intensive care unit to the pediatric ward on day 15; at the time, she was receiving oxygen via nasal cannula. She was transferred to the rehabilitation service on day 23, and she was discharged to home on day 40. At physical

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examination at the time of discharge, she was alert and able to converse and had limited flexion in the lower extremities. Six months after the poisoning, she continued to receive an antispasmodic agent for spasticity in the lower extremities, was attending preschool, and was reaching age-appropriate developmental milestones.

Case 2

H.G. was found unconscious next to her sister. She was given 15 L/min of oxygen via a nonrebreathing mask by emergency medical services and taken to a local emergency department. Her carboxyhemoglobin level within approximately 1 hour of discovery was 4.8%. On neurological examination, she had a score of 14 (confused) on the Glasgow Coma Scale and hyperreflexia of the lower extremities. Results of a cardiovascular examination were unremarkable. Arterial blood gas analysis revealed a compensated metabolic acidosis with an arterial pH of 7.39, a PaO₂ of 139 mm Hg, an oxygen saturation of 98.2%, and a base deficit of 10.9. Findings on a chest radiograph and a computed tomography scan of the brain were normal. Results of coagulation studies were abnormal, with a prothrombin time of 17 seconds and a partial thromboplastin time of 52 seconds. Her urine tested positive for myoglobin.

She was transported to our facility, and within 2 hours of her arrival, she began to have increasing respiratory distress. A chest radiograph showed bilateral air-space disease consistent with pulmonary edema. She was intubated for tachypnea and hemodynamic collapse. She required infusions of dopamine and dobutamine at 10 µg/kg per minute and an infusion of epinephrine at 0.5 µg/kg per minute to maintain normal blood pressure. An echocardiogram showed an ejection fraction of 0.23, and the serum level of cardiac troponin I was 13 µg/L. Her respiratory status began to deteriorate, and she required 100% oxygen and a positive end-expiratory pressure of 16 cm H₂O to maintain a PaO₂ greater than 50 mm Hg and an oxygen saturation greater than 85%. A chest radiograph showed worsening pulmonary edema with increasing bilateral alveolar consolidation.

H.G. had a few brief episodes of supraventricular tachycardia thought to be due to her initial myocardial injury. These episodes did not require treatment. She was extubated on hospital day 9 and was transferred to the pediatric ward on day 11. By day 20, she was weaned off oxygen, and she was discharged to home on day 24. At the time of discharge, she had no neurological deficits. Follow-up 6 months after discharge showed no detectable cognitive or motor deficits.

Discussion

In the presented cases, cardiopulmonary compromise and tachydysrhythmias occurred after carbon monoxide poisoning in a pair of 3-year-old patients. Carbon monoxide reversibly binds to hemoglobin with an affinity approximately 240 times greater than that of oxygen, thus reducing the total oxygen-carrying capacity of the hemoglobin. This competitive binding shifts the oxygen-hemoglobin dissociation curve to the left, resulting in impaired release of oxygen at the tissue level and cellular hypoxia.6

Myoglobin and mitochondrial cytochrome c oxidase are other heme-containing proteins that bind carbon monoxide, reducing muscular oxygen stores, possibly leading to poor myocardial function.6 The affinity of myoglobin for carbon monoxide is 30 to 50 times greater than its affinity for oxygen, whereas cytochrome c oxidase binds carbon monoxide and oxygen equally. Despite nonpreferential binding, Miro et al7 found 76% inhibition of cytochrome c oxidase activity after acute carbon monoxide poisoning. This inhibition persisted at lower levels (48%) 3 days after poisoning. These results suggest that mitochondrial cytochrome c oxidase may also be important in carbon monoxide poisoning. Furthermore, this phenomenon could explain the persistence of clinical signs and symptoms in patients with carbon monoxide poisoning after carboxyhemoglobin levels have returned to normal.

Cardiorespiratory Effects

Signs and symptoms associated with carbon monoxide poisoning are related to the severity of exposure. Tissue and cellular hypoxia can be mild to severe, and carboxyhemoglobin levels do not correlate well with the severity of signs and symptoms in a substantial number of cases. The duration of exposure appears to be an important factor; exposure to carbon monoxide for 1 hour or more may increase morbidity.8 On the basis of their medical histories and clinical features, these 2 girls most likely were exposed to high levels of carbon monoxide for several hours, although their carboxyhemoglobin levels were lower than the range generally defined as toxic. However, these levels were measured 25 minutes after the start of treatment with 100% oxygen via an endotracheal tube in 1 case and via a nonrebreathing face mask in the other case.

The half-life of carboxyhemoglobin is 4 to 6 hours when a patient is breathing room air and 40 to 80 minutes when the patient is breathing 100% oxygen.9 Hyperbaric oxygen therapy at 2 to 3 atmospheres
absolute can further reduce the half-life of carboxyhemoglobin to less than 30 minutes.\textsuperscript{10} Because of delays in transport to our facility, initiation of treatment with 100% oxygen via endotracheal tube and face mask, carboxyhemoglobin levels less than 5%, and the instability of their conditions, S.G. and H.G. were not candidates for hyperbaric oxygen therapy.

Calculated oxygen saturations do not reflect the presence of carboxyhemoglobin, so direct measurements of both carboxyhemoglobin and oxyhemoglobin levels are necessary. Compared with arterial hemoglobin oxygen saturation measured by cooximetry, measurements of fractional arterial oxygen saturation obtained with pulse oximetry are consistently overestimates. Furthermore, this difference increases with increasing carboxyhemoglobin levels.\textsuperscript{11} The pulse oximeter value generally decreases only 1% for each 10% of carboxyhemoglobin present.

S.G. and H.G. both were in shock with resulting cardiopulmonary collapse. Each had compromised cardiac output, pulmonary edema, and delayed-onset tachydysrhythmias. This clinical scenario is consistent with known cardiac effects of carbon monoxide poisoning. Direct toxic effects on the heart are thought to occur at a threshold level of 100 to 180 ppm of carbon monoxide for a 4-hour exposure.\textsuperscript{12} Cardiovascular effects of carbon monoxide may also include myocardial ischemia, pulmonary edema, arrhythmias, and stunned myocardium syndrome.\textsuperscript{13} These cardiovascular effects may be due to decreased cardiac output caused by cellular hypoxia, binding of carbon monoxide with myoglobin, and diminished oxygen release.\textsuperscript{14}

Pulmonary edema occurs in 10% to 30% of cases of acute carbon monoxide poisoning.\textsuperscript{15} Possible causes of pulmonary edema include tissue hypoxia,\textsuperscript{14} toxic effects of carbon monoxide on alveolar membranes, myocardial damage leading to left ventricular failure, aspiration of gastric contents after loss of consciousness, and neurogenic pulmonary edema.

Both S.G. and H.G. experienced delayed supraventricular tachycardia after left ventricular ejection fractions had returned to normal. H.G. had supraventricular tachycardia on hospital day 4; S.G. had it on hospital day 11. Studies in humans and animals have indicated that the threshold for induced ventricular fibrillation is lowered after exposure to carbon monoxide.\textsuperscript{14} Anderson et al\textsuperscript{16} found conduction abnormalities after carbon monoxide poisoning, including ST-segment and T-wave abnormalities, atrial fibrillation, intraventricular block, and extra systoles, in addition to myocardial ischemia and infarction. The pathological changes associated with cardiac injury found by Anderson et al\textsuperscript{16} included focal myocardial necrosis, leukocyte infiltration, and punctate hemorrhage.

Renal Effects

Rhabdomyolysis and acute renal failure also occur after carbon monoxide poisoning.\textsuperscript{17} The treatment of myoglobin-induced nephrotoxic effects is aggressive hydration, diuresis, and alkalinization of the urine to increase myoglobin solubility. S.G. and H.G. were treated aggressively with volume resuscitation and alkalinization until the urine pH was greater than 7.0 to treat myoglobinuria. Neither patient had acute renal failure or indications of nephrotoxic effects.

Neurological Effects

The most common neurological manifestations of carbon monoxide poisoning include fatigue, headaches, dizziness, difficulty in thinking, nausea, weakness, and confusion. Patients often have nystagmus, ataxia, and in severe poisonings, cerebral edema. Even low-level exposure to carbon monoxide results in long-term impairment of higher cognitive functions such as memory, new learning ability, attention, and concentration.\textsuperscript{18} Acute hydrocephalus has also resulted from carbon monoxide exposure.\textsuperscript{19} The most common neuroimaging findings in acute carbon monoxide poisoning are low-density lesions in the globus pallidus, detected in 7 of 18 patients in 1 study\textsuperscript{20} and in 7 of 19 patients in another study.\textsuperscript{21} Although gray matter has greater metabolic oxygen needs than white matter does, white matter is particularly sensitive to cerebral hypoxia due to carbon monoxide intoxication, and white matter injury correlates directly with clinical status and outcome.\textsuperscript{22} The greater sensitivity of white matter to hypoxia may be explained by its more restricted vascular supply, which limits its ability to tolerate reduced oxygen tensions.\textsuperscript{23}

Recent studies suggest that carbon monoxide-induced tissue hypoxia may be followed by reoxygenation injury of the central nervous system. This hypoxic event facilitates the production of partially reduced oxygen species, resulting in a reperfusion injury.\textsuperscript{24} In addition, exposure to carbon monoxide causes a reversible demyelinization of lipids in the central nervous system.\textsuperscript{25}

Approximately 10% to 30% of patients with carbon monoxide poisoning experience a delayed onset of neuropsychiatric signs and symptoms 3 to 240 days after exposure. The full range of causes of the late sequelae of carbon monoxide exposure is uncertain, but the following factors may play a role: cellular hypoxia, postischemic reperfusion injury, the effects
of carbon monoxide on the cerebral vascular endothelium, and peroxidation of brain lipids mediated by reactive oxygen species. Signs and symptoms include cognitive and personality changes, parkinsonism, dementia, and psychosis. No clinical or laboratory results are predictive of which patients are at risk for this complication, but advanced age appears to be a risk factor. Randomized clinical trials of hyperbaric oxygen therapy in patients with carbon monoxide poisoning showed no difference in persistent and delayed neurological sequelae and indicated that such therapy may lead to worse outcomes. Recovery from delayed neuropsychiatric syndrome occurs in more than half of affected patients within 1 year.

Little is known about the neurological consequence and prognosis in children who survive carbon monoxide poisoning. Although a small number of children in 1 study had a decline in school function, both S.G. and H.G. were functioning at a developmentally appropriate level without long-term neurological sequelae 12 months after their exposure.

Prevention

Prevention of carbon monoxide poisoning begins with education of the public about the dangers of carbon monoxide poisoning and the potential sources of carbon monoxide in residences and vehicles. The public must be advised that properly maintaining all fuel-burning appliances and motor vehicles is necessary to prevent carbon monoxide poisoning. A recent review of 136 deaths due to carbon monoxide poisoning revealed that approximately half of the deaths could have been prevented by having a carbon monoxide detector. Electronic carbon monoxide detectors generally respond at carbon monoxide levels of 35 ppm and generate a visible and audible alarm loud enough to arouse a sleeping person. The Environmental Protection Agency and the Occupational Safety and Health Administration provide regulations and suggestions for prevention of carbon monoxide poisoning.

REFERENCES

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