

Recurrent Acute Life-threatening Events and Lactic Acidosis Caused by Chronic Carbon Monoxide Poisoning in an Infant

Marta Foster, MD*; Salvatore R. Goodwin, MD*‡; Charles Williams, MDS§; and Janice Loeffler, MD, PC||

ABSTRACT. Acute severe carbon monoxide poisoning is usually easy to recognize and diagnose. However, chronic or less severe exposure may produce more subtle symptoms. We report on a 3½-year-old girl who was admitted to the hospital several times with acute, life-threatening events, acidosis, and flu-like symptoms. The diagnosis was elusive, but after careful questioning of family members and a home visit, chronic carbon monoxide poisoning was diagnosed. *Pediatrics* 1999;104(3). URL: <http://www.pediatrics.org/cgi/content/full/104/3/e34>; carbon monoxide poisoning, carboxyhemoglobin, children, diarrhea, acute life-threatening events, lactic acidosis, flu-like symptoms.

ABBREVIATIONS. CO, carbon monoxide; O₂, oxygen; COHb, carboxyhemoglobin; LCHAD, long-chain 3-OH acyl CoA dehydrogenase.

The incidence of carbon monoxide (CO) poisoning is ~3500 to 4000 cases per year, caused by deliberate or accidental exposure. Of this number, 10% are children.¹ For most individuals, CO is nontoxic at low concentrations. However, with the increased use of tobacco products, natural gas, gasoline engines, and home appliances that use gas or kerosene, more cases are being reported.²

CASE REPORT

The patient was a 3½-month-old girl born at 37 weeks' gestation weighing 5 lb, 14 oz. She did well until 3 to 4 weeks of age, when she developed frequent loose stools that were followed 2 weeks later by a dry cough and rapid labored breathing. Over the next 5 to 7 days, her symptoms worsened, and she was taken to her primary physician. The following day she was admitted to the hospital for respiratory distress and wheezing; her oxygen (O₂) saturation was 50%. Supplemental O₂ was started, and she was given nebulized albuterol. A chest radiograph revealed an interstitial infiltrative pattern, hyperinflation, and decreased volume at the left apex. She was transferred to a nearby regional medical center at this time. Laboratory tests demonstrated the following: potassium, 5.3 mEq/L; sodium, 129 mEq/L; and carbon dioxide, 19 mEq/L. Immunofluorescent staining of nasopharyngeal secretions was negative for respiratory syncytial virus. The patient then was transferred to our children's hospital for additional evaluation. Adrenal insufficiency and factitious causes of hyponatremia, such as elevated glucose or triglycerides, were ruled out. Her low sodium was attributed to diarrheal stools. She was discharged to

home after 4 days in good condition with nebulized albuterol treatment as needed.

At 2 months of age, her mother noted breathing difficulty and wheezing for which she administered nebulized albuterol. However, the infant developed apnea with circumoral cyanosis and stiffness of her extremities. Her father administered several mouth-to-mouth breaths after which she began to breathe again, and she was transported to the local hospital. Her O₂ saturation was 80% breathing room air, and she was given supplemental O₂. A chest radiograph showed prominent interstitial markings at the base of both lungs with marked changes in the upper lobes that were relatively unchanged from her previous chest radiograph. An evaluation for the cause of the apnea included an electrocardiogram and an upper gastrointestinal series, which showed normal results. She was transferred once again to our children's hospital for additional management. Initial laboratory work revealed a metabolic acidosis as follows: pH, 7.30; Pco₂, 37 mm Hg; Po₂, 81 mm Hg; Hco₃, 19 mEq/L; base excess, -3.0 mEq/L; and O₂ saturation, 96.2%. Her potassium was 6.3 mEq/L, and lactic acid level was 8.3 mmol/L. She was also hypertensive without apparent etiology. Kayexalate, bicitra, and captopril were administered, and a genetics consultation was obtained to assist in evaluating her lactic acidosis. Serum amino acids were normal, but urinalysis demonstrated elevated levels of free dicarboxylic acids and derivatives, and long-chain 3-OH acyl CoA dehydrogenase (LCHAD) defect was considered. However, a plasma acylcarnitine profile was normal, and an LCHAD defect was considered to be very unlikely. The hyperkalemia and lactic acidosis resolved spontaneously. The etiology of her hypertension was believed to be related to a renal hypoxic/ischemic insult that was consistent with echogenicity seen on a renal ultrasound. She was weaned successfully from supplemental O₂ and was discharged to her home with an apnea monitor and instructions for administering captopril, Bicitra, and Kayexalate.

She did well until 3 months of age, when again she was hospitalized for respiratory distress and suspected bronchiolitis. She was transferred to our intensive care unit after experiencing apnea that required a brief period of cardiopulmonary resuscitation without tracheal intubation. On arrival, physical examination revealed a hyperactive and irritable infant with a 40°C temperature, a ≥60 breaths/minute respiration rate, and a ≥200 beats/minute heart rate with an S4 gallop and grade 2/6 midsystolic murmur. Her trachea was intubated, and she was ventilated mechanically because of persistent apnea and bradycardia. Her chest radiograph demonstrated pulmonary edema and an enlarged cardiac silhouette. The electrocardiogram showed evidence of biventricular enlargement, and a two-dimensional echocardiogram demonstrated an ejection fraction of 40% without dilation or hypertrophic cardiomyopathy. The pulmonary edema and cardiomegaly resolved with the administration of Lasix; her blood pressure remained normal and captopril and Bicitra were discontinued. She was extubated on the fifth hospital day.

A review of cooximetry values from her previous hospitalizations revealed slightly elevated carboxyhemoglobin (COHb) levels. This finding prompted an investigation into her home environment. We discovered that the infant's mother had been using a kerosene space heater to heat the house. When asked specifically about headaches, she said that everyone in the household was experiencing them and that visitors would develop headaches that resolved after leaving the house. The local fire department was asked to analyze urgently the air in the house; they documented a CO level of 0.43% near the space heater and of 0.13% in the infant's

From the Departments of *Anesthesiology, †Pediatrics, and §Genetics, University of Florida College of Medicine, Gainesville, Florida.

||Private practice pediatrician in Valdosta, GA.

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Reprint requests to (S.R.G.) Editorial Office, Department of Anesthesiology, PO Box 100254, Gainesville, FL 32610-0254. E-mail: goodwin@anest1.anest.ufl.edu

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room, with 0.15% considered lethal. Members of the household were evacuated emergently.

The infant was transferred subsequently to our pediatric ward, where she remained stable. She was discharged to a new home environment, and 6 months later she had been doing well except for a short hospital admission for a documented respiratory syncytial virus infection.

DISCUSSION

We believe that chronic CO poisoning was likely the cause of all of these hospital admissions and acute life-threatening events. To our knowledge, this is the first case report describing recurrent chronic CO poisoning in an infant. The infant's first symptom, diarrhea, and resultant hyponatremia have been described in CO poisoning in infants.¹ The acidosis and ischemia discovered during the second and third admissions were probably the result of CO poisoning; no other etiology was discovered. Each time, the lactic acidosis resolved with only supportive care and hydration. Other causes of lactic acidosis that were evaluated and considered very unlikely were adrenal insufficiency and congenital lactic acidosis. Because COHb rapidly returns to normal with supplemental oxygen, CO poisoning was not suspected initially. Since her return to a CO-free home, she has remained well and has had no additional symptoms. Family members also no longer experience headaches.

The toxic nature of carbon monoxide is caused primarily by its tremendous affinity for hemoglobin. Hemoglobin affinity is 200 to 250 times greater for CO than for O₂. The enhanced O₂ affinity limits the amount of O₂ released to the tissues and the O₂-carrying capacity of hemoglobin.³ CO also binds to myoglobin and to the electron transport system, thus inhibiting cellular respiration. This creates pronounced tissue hypoxia, anaerobic metabolism, and lactic acidosis.^{2,4} The definite diagnosis is obtained by measuring COHb levels. In normal adults, chronic exposure to moderate levels of CO may produce symptoms that mimic those of the flu (ie, nausea, lethargy, and headaches).^{1,5} Exposure to higher levels of CO may cause shortness of breath, dyspnea, tachypnea, headache, emotional lability, confusion, impaired judgment, clumsiness, syncope, nausea, vomiting, and diarrhea. Cerebral edema, coma, respiratory depression, and pulmonary edema are seen in severe cases. Cardiovascular manifestations are ischemic in nature and include chest pain, arrhythmias, heart failure, and hypotension.⁶ Bullae and blisters also may be seen over pressure points that could appear as burns.^{4,7} Renal failure secondary to ischemia and myoglobinuria from muscle necrosis also can occur. Deafness, visual field defects, blindness (temporary or permanent), venous engorgement with papilledema, and optic nerve atrophy have occurred.^{7,8}

COHb levels >15% usually produce symptoms; levels >20% are considered toxic; levels >40% are associated with more severe neurologic effects; and levels >50% produce irreversible central nervous system damage.⁹ Pregnant women, fetuses, and newborn infants are especially vulnerable to CO toxicity,

because fetal hemoglobin has a higher affinity for O₂, and O₂ tensions are usually lower.^{2,10}

In children, acute intoxication may occur earlier because of higher metabolic rates, respiratory exchange requirements, and smaller blood volumes resulting in a more rapid CO uptake. The clinical presentation differs from adults in that it often mimics gastroenteritis, as it did in our patient.^{1,11} Lethargy and syncope are also more likely to occur in children at lower COHb levels than in adults.¹¹ As with adults, children with COHb levels <15% are asymptomatic.¹¹ The pediatrician should be aware that rare delayed complications can occur as a result of CO toxicity. Delayed neurologic complications caused by post hypoxic demyelination has occurred after CO exposure in adults.¹² Hydrocephalus has been reported in a child 100 hours after CO exposure.¹³

The treatment consists of removing the patient from the site of exposure and administering O₂ therapy. By the laws of mass action, dissociation of CO-hemoglobin complex occurs, and CO is excreted via the lungs. In room air, CO half-life is 5 to 6 hours. The half-life decreases to ~1 to 1½ hours when receiving 100% O₂ and to ~30 minutes with the use of hyperbaric O₂ therapy.^{8,9} In conscious patients, 100% O₂ should be administered via a nonbreathing mask until CO levels have decreased to 10% and symptoms have resolved. Endotracheal intubation with mechanical ventilation may be necessary in patients with central nervous system dysfunction or cardiovascular instability. Hyperbaric O₂ at 2 to 3 atmospheres shortens the duration of symptoms.⁷ It also is believed that patients with normal COHb levels and persistent neurologic deficits may have improved outcome with hyperbaric oxygen treatment.^{4,7,8}

CONCLUSION

In summary, we present a case of ongoing CO poisoning in an infant who had recurrent unexplained constitutional symptoms, acute life-threatening events, and lactic acidosis. Once additional history was obtained regarding method of home heating and symptoms experienced by other family members, an analysis of CO in the home was performed, and the home was evacuated emergently because of the near lethal levels of CO. Once removed from this environment, the child has thrived and has had no additional problems. This case illustrates the importance of including exposure to toxins high on the list of differential diagnoses when the cause of illness is elusive. In such an elusive case, a home visit may prove helpful in making the diagnosis.

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