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

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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/104/3/34; carbon monoxide poisoning, carboxyhemoglobin, children, diarrhea, acute life-threatening events, lactic acidosis, flu-like symptoms.

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 upper lobes that required a brief period of cardiopulmonary resuscitation and intubation. The patient was transferred to our children’s hospital for additional evaluation. A review of cooximetry values from her previous hospitalization revealed an ejection fraction of 40% without dilation or hypertrophy. She was extubated on the fifth hospital day.

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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 8.5 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.

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The patient was transferred to our intensive care unit after experiencing apnea caused by apparent hypoxia/ischemic insult that was consistent with echocardiogram seen on a renal ultrasound. 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 8.5 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 echocardiogram 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.

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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. 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 experienced 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. 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. 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). Exposure to higher levels may produce symptoms that mimic those of the flu (ie, nausea, lethargy, and headaches). Exposure to moderate levels of CO may produce symptoms that mimic those of the flu (ie, nausea, lethargy, and headaches). 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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. 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. 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. Lethargy and syncope are also more likely to occur in children at lower COHb levels than in adults. As with adults, children with COHb levels <15% are asymptomatic. 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. Hydrocephalus has been reported in a child 100 hours after CO exposure.

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. In conscious patients, 100% O2 should be administered via a nonrebreathing 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. It is also believed that patients with normal COHb levels and persistent neurologic deficits may have improved outcome with hyperbaric oxygen treatment.

**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.

**REFERENCES**

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