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/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 circulatory 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 8.5 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 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 3½ 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 respirations, and a >200 beats/minute heart rate with an S4 gallop and grade 2/6 mid systolic 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 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 environment.

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PEDIATRICS Vol. 104 No. 3 September 1999 1 of 3
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CHRONIC CARBON MONOXIDE POISONING IN AN INFANT
tom, diarrhea, and resultant hyponatremia have
time, the lactic acidosis resolved with only support-
care and hydration. Other causes of lactic acidosis
that were evaluated and considered very unlikely
were adrenal insufficiency and congenital lactic aci-
dosis. Because COHb rapidly returns to normal with
supplemental oxygen, CO poisoning was not sus-
pected initially. Since her return to a CO-free home,
she has remained well and has had no additional
symptoms. Family members also no longer experi-
ence 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 pro-
nounced 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
of CO may cause shortness of breath, dyspnea,
tachypnea, headache, emotional liability, confusion,
impaired judgment, clumsiness, syncope, nausea,
vomiting, and diarrhea. Cerebral edema, coma, res-
piratory depression, and pulmonary edema are seen
in severe cases. Cardiovascular manifestations are
ischemic in nature and include chest pain, arrhyth-
mias, heart failure, and hypotension. Bullae and
blesters also may be seen over pressure points that
could appear as burns. Renal failure secondary to
ischemia and myoglobinuria from muscle necrosis
also can occur. Deafness, visual field defects, blind-
ness (temporary or permanent), venous engorge-
ment with papiledema, and optic nerve atrophy
have occurred.

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 new-
born 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 ex-
change requirements, and smaller blood volumes
resulting in a more rapid CO uptake. The clinical
presentation differs from adults in that it often mim-
ics gastroenteritis, as it did in our patient. Leth-
argy 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 expo-
sure.

The treatment consists of removing the patient
from the site of exposure and administering O2 ther-
apy. 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 re-
ceiving 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 pa-
tients with central nervous system dysfunction or
cardiovascular instability. Hyperbaric O2 at 2 to 3
atmospheres shortens the duration of symptoms. It
also is believed that patients with normal COHb
levels and persistent neurologic deficits may have
improved outcome with hyperbaric oxygen treat-
ment.

CONCLUSION
In summary, we present a case of ongoing CO
poisoning in an infant who had recurrent unex-
plained constitutional symptoms, acute life-threaten-
ing events, and lactic acidosis. Once additional his-
tory was obtained regarding method of home
heating and symptoms experienced by other family
members, an analysis of CO in the home was per-
formed, and the home was evacuated emergently
because of the near lethal levels of CO. Once re-
moved from this environment, the child has thrived
and has had no additional problems. This case illus-
rates 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 diagno-
sis.

REFERENCES
2. Vreman HJ, Mahoney JJ, Stevenson DK. Carbon monoxide and carboxy-
3. Goldbaum LR, Ramirez RG, Absalon KB. What is the mechanism of
392–399


