



Perinatal Carbon Monoxide Poisoning: Treatment of a 2-Hour-Old Neonate with Hyperbaric Oxygen

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Abstract

A 41-year-old gravida 4 para 3 (G4P3) and 38^{5/7} weeks pregnant woman presented to labor and delivery with dizziness, headache, and decreased fetal movement after 12 hours of exposure to carbon monoxide (CO) from a grill that was used inside for heat. The mother was hemodynamically stable, and her neurologic examination was intact. Her carboxyhemoglobin level, which was obtained 12 hours after removal from the CO exposure, was 7.4%. The fetus's heart rate was 173 beats per minute with moderate variability and one late appearing deceleration, not associated with contractions. The biophysical profile score was 2 of 8. The obstetrics team performed a routine cesarean section. The 1- and 5-minute Apgar's scores were 7 and 8, respectively. The arterial cord gas result was as follows: pH = 7.05, PCO₂ = 71 mm Hg, pO₂ = 19 mm Hg, bicarbonate = 14 mmol/L, and carboxyhemoglobin = 11.9%. The mother and infant were treated with hyperbaric oxygen therapy consisting of 100% oxygen at 2.4 atmosphere absolutes (ATA) for 90 minutes at 2.5 hours after delivery. Following one hyperbaric oxygen treatment, the infant was transitioned to room air and routine postpartum treatment and was discharged 3 days later in good condition. Hyperbaric oxygen treatment was well tolerated in this neonate.

Keywords

- ▶ perinatal
- ▶ carbon monoxide
- ▶ poisoning
- ▶ hyperbaric oxygen
- ▶ pregnancy

Perinatal carbon monoxide (CO) poisoning is a rarely reported occurrence.^{1–5} CO binds to hemoglobin with more than 200 times the affinity of oxygen for hemoglobin. As a result, in the setting of CO poisoning, oxygen is displaced from hemoglobin, and oxygen-carrying capacity may be

decreased leading to cellular hypoxia.⁶ A fetus is particularly susceptible to the effects of CO in that CO binds with even greater affinity to fetal hemoglobin than to maternal hemoglobin.⁷ CO also shifts the already left-shifted fetal oxygen dissociation curve further to the left, thereby additionally

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decreasing the release of oxygen from hemoglobin to the tissues.^{1,7} Treatment of CO poisoning consists of high-flow oxygen and treatment with hyperbaric oxygen (HBO) when indicated.⁸ We present a case of a term fetus inadvertently poisoned with CO resulting in delivery and followed by emergent HBO treatment 2 hours after birth. We also propose recommendations for perinatal treatment of the CO-exposed fetus.

Case Report

A 41-year-old pregnant gravida 4 para 3 (G4P3) woman at 38^{5/7} weeks of gestational age by ultrasound presented to our hospital's Labor and Delivery Unit with symptoms of dizziness, mild headache, and decreased fetal movement. Prior to arrival, the patient and her spouse had used an indoor charcoal grill to heat their apartment, and she and her spouse were exposed to the burning coals consistently for 12 hours from the afternoon to overnight. When she awoke, she felt lightheaded and noted decreased fetal movement. Her spouse experienced syncope and was transported by ambulance to a local emergency department (ED). For unclear reasons, at the outside hospital, he did not receive HBO treatment and was not transferred for HBO treatment and was discharged from the emergency department. Our patient removed herself from the CO exposure, and approximately 12 hours later, she took a ride share to our hospital's Labor and Delivery Unit. Measurements of CO levels at the residence were not available.

Approximately 12 hours after symptom onset and removal from the CO source, she presented to our hospital's Labor and Delivery Unit. The patient complained of a mild headache and decreased fetal movement for the prior 8 hours. She denied vaginal bleeding, leakage of vaginal fluid, and contractions. The patient had a past medical history of type-2 diabetes for which she was taking insulin. She was a former smoker but no longer used cigarettes, tobacco, or nicotine. There were no smokers in the house. Other than the patient's diabetes, the pregnancy was uncomplicated. The mother had received routine prenatal care. Outside of the finding of cystic kidneys on prenatal ultrasound, the fetus had normal anatomy scans with normal intrauterine growth.

The patient's examination was notable for a blood pressure of 122/79 mm Hg, a heart rate of 64 beats per minute, a temperature of 98.2°F, and a respiratory rate of 18 breaths per minute. The patient was awake, alert, and oriented, strength was intact throughout, her gait was normal, her finger–nose–finger exam was intact, her face was symmetric, and her speech was clear. Her abdomen was gravid and nontender. Her skin was pink, warm, and dry. The electrocardiogram revealed a sinus bradycardia at 52 beats per minute without ischemic changes. The patient's white blood cell count was 8,400 mm³, hemoglobin was 12.6 g/dL, and platelet count was 251,000 mm³. Arterial carboxyhemoglobin was 7.4%; however, this level was obtained approximately 12 hours after symptom onset and removal from the source of CO. The elimination half-life of carboxyhemoglobin while breathing room air is 320 minutes.⁸ Extrapolating

back, the mother's carboxyhemoglobin at the time of symptom onset may have been over 30%. Upon arrival to the hospital, the mother was administered high flow oxygen of 10 L/minute via nonrebreather mask.

The fetus' heart rate was 173 beats per minute with moderate variability and one late appearing deceleration, not associated with contractions. Tocometer readings were absent for contractions. On ultrasound, the amniotic fluid index was 16 mm. The biophysical profile (BPP) was 2/8 (2 for adequate amniotic fluid). Given the decreased fetal movement and BPP score, the obstetrics team decided to perform a routine cesarean section.

The baby was delivered via cesarean section and had a spontaneous cry. The 1-minute Apgar's score was 7. The 5-minute Apgar's score was 8. The infant's weight was 3,105 g, length was 49.5 cm, and head circumference was 34.5 cm. The infant's vital signs were as follows: blood pressure was 60/40 mm Hg, heart rate was 137 beats per minute, respiratory rate was 45 breaths per minute, and temperature was 98.6°F. The infant was noted to have significantly decreased muscle tone throughout and dusky colored extremities, both of which improved with warming and stimulation.

The infant's arterial cord gas result was the following: pH = 7.05, PCO₂ = 71 mm Hg, pO₂ = 19 mm Hg, bicarbonate = 14 mmol/L, and carboxyhemoglobin = 11.9%. The infant's white blood cell count was 18,500 mm³, hemoglobin was 12.9 g/dL, and platelets were 236,000 mm³. The infant was administered high-flow 100% oxygen of 10 L per minute via nonrebreather mask.

The neonatologist consulted our Hyperbarics service and Medical Toxicology Consult service just prior to delivery. The mother's prolonged exposure to CO and persistent symptoms despite removal from the CO exposure, indicated more severe poisoning and potential risk to the fetus. The fetus' BPP confirmed fetal distress necessitating delivery and a decision to treat with HBO. The mother and newborn infant were treated emergently with HBO therapy consisting of 100% oxygen at 2.4 atmosphere absolutes (ATA) for 90 minutes at 2.5 hours after delivery. The hyperbaric treatment table for CO poisoning at our institution is 100% oxygen at 2.8 ATA for 90 minutes. Given the age of the newborn, we decided to treat at 2.4 ATA to limit the potential effects of pressure on the neonate's lungs and to reduce the risk of pulmonary and central nervous system (CNS) oxygen toxicity. A chamber attendant and neonatal intensive care unit (NICU) nurse accompanied the neonate and her mother. Chamber-side was the chamber operator, the Hyperbaric and Toxicology teams, a respiratory therapist, and additional nurses. Oxygen was delivered to the mother via hood and to the infant via enclosed bassinet within the chamber. The newborn was wrapped in a blanket to minimize movement and for warmth, and the environment was kept warmer on ascent. The umbilical line was kept patent and connected to a pressure tested pump, and vitals were monitored through a pressure tested machine. The newborn was given a pacifier to assist with equalization of the Eustachian tubes during the 10-minute descent (to prevent middle ear barotrauma) but was removed during the 10-minute ascent to prevent breath

holding on ascent and pulmonary barotrauma. Prior to HBO treatment, the infant had been on 10 L high-flow 100% oxygen for 2.5 hours. With this normobaric oxygen treatment, her carboxyhemoglobin had decreased to 4.6%. Following HBO treatment, her carboxyhemoglobin level was 1.4% ($\leq 3\%$ is considered normal⁸). The half-life of carboxyhemoglobin with 100% oxygen treatment is up to 80 minutes, whereas the half-life of carboxyhemoglobin with hyperbaric oxygen treatment is 15 to 30 minutes.⁶ No further treatments of the infant were done, and the infant was transferred to the NICU on room air. The mother received additional HBO therapy the following day given on-going symptoms. The infant was discharged in good condition 3 days after delivery. Of note, the toxicology service had contacted the mother of the infant's spouse to recommend evaluation at our health system's ED for evaluation and possible HBO treatment, but he did not present for evaluation.

Discussion

CO poisoning during pregnancy can have serious adverse effects including severe neurological sequelae or death to the mother and fetus. CO poisoning is associated with a maternal mortality rate between 19 and 24% and a fetal mortality rate between 36 and 67%, with variability arising from severity of maternal poisoning and gestational age.^{9,10} Treatment of CO poisoning involves normobaric oxygen therapy with or without HBO treatment. Few cases of neonates or very young children poisoned with CO have been described. Bar et al reported a case of a 39-week pregnant woman poisoned with CO whose fetus was in distress and who underwent an emergent cesarean section delivery.¹ The neonate was then treated with 100% normobaric oxygen for 12 hours and was subsequently discharged in good condition. Turan et al described a 7-day-old male infant with CO poisoning who was treated with hyperbaric oxygen and discharged in good condition. Rudge described 14 patients who were younger than 2 years of age and were treated with HBO; one was a 4-day-old neonate.¹¹ To our knowledge, our case is the youngest CO-poisoned patient to be treated with HBO.

The mechanisms of CO-induced tissue toxicity are complex. CO displaces oxygen from hemoglobin and decreases the oxygen-carrying capacity of hemoglobin, thereby causing tissue hypoxia.^{6,12} At a cellular level, CO acts as an asphyxiant by inhibiting human cytochrome oxidase.¹³ CO also has proinflammatory mechanisms via nitric oxide liberation in a platelet and neutrophil-dependent reaction¹⁴ and mediates brain lipid peroxidation, thereby likely contributing to deleterious neurologic effects.¹⁵ Other mechanisms of CO toxicity involve apoptosis, mitochondrial oxidative stress, and immune-mediated injury. These are proposed mechanisms of CO toxicity which manifests acutely with symptoms of headache, nausea, and vomiting.⁸ Delayed neurological sequelae may follow CO poisoning.¹⁶

Obtaining a carboxyhemoglobin level can confirm exposure and support the diagnosis of CO poisoning, but symptoms may not correlate with the initial carboxyhemoglobin

level as was thought in this case.^{6,17,18} The mother's initial carboxyhemoglobin level was 7.4%, and she was symptomatic with regard to CO poisoning; however, she had been removed from the CO source of poisoning and had been breathing room air for 12 hours prior to obtaining that level. This time allowed for elimination of CO. Carboxyhemoglobin levels of greater than 10% in smokers and greater than 3% in nonsmokers are considered elevated.^{6,19}

Administration of supplemental 100% oxygen is the standard treatment for acute CO poisoning.¹⁶ Oxygen hastens dissociation of CO from hemoglobin and enhances tissue oxygenation.²⁰ Hyperbaric oxygen accelerates CO elimination and is considered to prevent CO-mediated cognitive sequelae of CO poisoning.¹⁶

Maternal CO poisoning in pregnancy jeopardizes the well-being of the fetus. The fetus is more vulnerable to the hypoxic effects induced by CO than the mother. Operating on the steep part of the oxyhemoglobin dissociation curve, a small drop oxygen tension can dangerously lower fetal arterial oxygen concentration. CO competes with oxygen for the same hemoglobin binding sites, blocking oxygen uptake, and placing the fetus at high risk for fetal hypoxia. As fetal carboxyhemoglobin levels increase, the fetal oxyhemoglobin dissociation curve shift further to the left, intensifying the effect and causing critical hypoxia in the fetus.²¹ Maternal CO poisoning has been associated with fetal demise, physical malformations, and developmental cognitive deficits.^{18,20-24} Predicting fetal outcomes is challenging, as the severity of maternal poisoning as measured by maternal carboxyhemoglobin concentrations do not correlate with fetal carboxyhemoglobin, and poor fetal outcomes have been observed with low maternal carboxyhemoglobin concentrations.¹⁸

When a pregnant woman experiences CO poisoning, decisions regarding her medical management and that of her fetus can be difficult. Given the rare nature of this condition, no specific treatment guidelines are available from the American College of Obstetricians and Gynecologists (ACOG) or American Academy of Pediatrics. For infants greater than or equal to 34 weeks' gestational age, signs of fetal distress caused by maternal CO toxicity may raise concerns about the need for urgent delivery versus expectant management.²⁵ Maternal and newborn risks, however, must be balanced with those of continuing of the pregnancy, especially if the fetus is less than 39 weeks of gestation.²⁵⁻²⁷ In the case presented here, significant fetal distress was observed, as documented by a biophysical profile of 2 out of 10, the infant was term gestation, and the woman had experienced previous cesarean births; therefore, the decision was made to deliver the baby via repeat cesarean.

Following exposure to CO, options for treatment of the CO-poisoned pregnant patient include normobaric oxygen treatment, hyperbaric oxygen treatment, delivery and subsequent treatment of the neonate, and mother with normobaric oxygen or hyperbaric oxygen. Following maternal CO poisoning, we recommend the administration of high-flow oxygen via nonrebreather mask to the exposed pregnant mother. If the fetus is not in distress, HBO treatment of the pregnant mother can be considered, as HBO treatment of the fetus following

maternal CO exposure appears to be well tolerated and may be beneficial.^{9,22,24,28} If the decision is made to deliver the fetus due to signs of fetal distress following CO exposure, we recommend HBO treatment of the newborn, if feasible, and if the mother has a measurable concentration of CO greater than 10% or any evidence of ischemia (syncope and cardiac ischemia). If HBO treatment is not available following the delivery, the neonate should be treated with 100% normobaric oxygen until carboxyhemoglobin levels are less than 3% and any symptoms have resolved.⁸ Given the limitations of available data, decisions regarding the timing of delivery should always be individualized to the needs of the patient, weighing competing risks and benefits for the woman and her fetus.²⁵ If delivery is advised before 39^{0/7} weeks of gestation, then indications for delivery should be clearly documented and discussed with the patient.²⁶

Conclusion

HBO treatment was well tolerated in this newborn infant who sustained inadvertent perinatal CO poisoning. HBO is a treatment option for perinatal CO exposures.

Conflict of Interest

None declared.

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