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## CASE REPORT

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# Carbon monoxide poisoning during pregnancy treated with hyperbaric oxygen

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**Abstract**

Acute carbon monoxide (CO) intoxication during pregnancy causes fetal death and teratogenic effects. Hyperbaric oxygen (HBO2) therapy has the potential to improve them. HBO2 therapy should be considered to treat CO intoxication during pregnancy.

**KEYWORDS**

carbon monoxide, case reports, hyperbaric oxygen therapy, pregnancy

## 1 | INTRODUCTION

Although acute carbon monoxide (CO) intoxication during pregnancy is an uncommon event, transplacental passage of CO may cause high maternal and fetal mortality, as well as adverse central nervous system (CNS) consequences and teratogenic effects.<sup>1-3</sup> Oxygen treatment should be swiftly offered for all CO poisoning cases. CO is eliminated because higher oxygen levels in the lungs leads to the dissociation of carboxy-hemoglobin (COHb), allowing free CO to be exhaled at a rate dependent on the partial pressures of the two gases.<sup>4</sup> However, when there is significant maternal exposure, a longer duration of treatment is required than that for nonpregnant patients for fetal CO elimination.

Hyperbaric oxygen (HBO2) therapy is the inhalation of 100% oxygen at greater than atmospheric ambient pressures and a widely accepted therapeutic strategy used to treat CO intoxication, particularly for patients with moderate-to-high-risk

CO poisoning. Here, we report a 30-year-old female CO poisoning patient who was 31 weeks pregnant and transferred to our emergency department for indication for HBO2 therapy. This case report and literature review are quite important to increase emergency physicians' awareness of fetal risk in maternal CO intoxication and understand HBO2 as a therapeutic strategy for CO-intoxicated pregnant women.

## 2 | CASE PRESENTATION

A 30-year-old female who was 31 weeks pregnant was transferred to our emergency department complaining of nausea and headache. The patient and her husband had been living in a camping tent and had dinner in front of a fireplace with three other family members. While asleep, the patient noticed nausea and headache symptoms and visited the local clinic. The patient was a nonsmoker with no significant medical history.

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Upon arrival at the local clinic's emergency department, the patient was well oriented with a Glasgow Coma Scale score of 15 (E4V5M6). The patient had no local neurological deficits. Her vital signs included blood pressure of 110/80 mm Hg, respiration rate of 15 breaths/min, heart rate of 70 beats/min, body temperature of 36.9°C, and oxygen saturation 98%. Arterial blood gas test results on ambient air on arrival were as follows: pH 7.440, PaCO<sub>2</sub> 31.6 mm Hg; PaO<sub>2</sub>, 97.0 mm Hg; HCO<sub>3</sub><sup>-</sup> 22.9 mEq/L, lactate 1.6 mmol/L, and COHb 28.6%. Her laboratory data were unremarkable, as follows: white blood cells, 7100/μL; hemoglobin, 11.5 g/dL; serum creatine kinase, 60 IU/L; sodium 133 mmol/L; potassium, 3.4 mmol/L; blood glucose 84 mg/dL; blood urea nitrogen 9.6 mg/dL; creatinine 0.41 mg/dL; total bilirubin 0.3 mg/dL; aspartate aminotransferase 13 IU/L; alanine aminotransferase 10 IU/L; and lactate dehydrogenase 156 IU/L. Urine drug screening tests (Triage<sup>®</sup>, Biosite) were negative.

As the patient was diagnosed with CO intoxication based on her interview, physical examination, and high blood COHb concentration, high-flow oxygen (15 L/min) was given during transportation by ambulance, followed by a decrease in COHb levels of up to 3.9%. Fetal wellbeing was confirmed with normal heart rate, normal umbilical cord blood flow, and an estimated fetal weight of 2019 g.

After arrival to our hospital, HBO<sub>2</sub> therapy was initiated 5 hours after exposure to CO. The patient underwent additional HBO<sub>2</sub> therapy. In accordance with our protocol, which was decided by referring to the report of Weaver et al<sup>5</sup> and consulting with our team, the patient's HBO<sub>2</sub> therapy (100% oxygen at two atmospheres for 60 minutes) was administered three times for 2 days. After the treatment, the COHb level improved to 0.7%, and no abnormalities were observed in the patient and the fetal, so she was discharged.

At 41 weeks, she delivered a healthy infant weighing 2862 g with 1- and 5-minute Apgar scores of 7 and 9, respectively. Currently, after 3-month follow-up, both the infant and mother are doing well.

### 3 | DISCUSSION

Carbon monoxide poisoning is toxic to both the fetus and mother in terms of delayed CNS sequelae and CNS disorders. Hypoxia appears to be the primary mechanism of the deleterious effects on the fetus associated with CO poisoning during pregnancy. CO dissolves in the maternal blood and crosses the placenta via passive diffusion, thus combining with fetal hemoglobin. COHb levels become 10-15% higher in the fetus than in the mother, since fetal hemoglobin has a higher affinity for CO than adult hemoglobin. Fetal CO washout is slower, as the dissociation between CO and hemoglobin takes longer than adult hemoglobin. Moreover, the fetus is unable to adequately increase ventilation or tissue blood flow

to combat the ischemic insult. Therefore, clinicians should be aware that the fetus is at greater risk than the mother for CO-induced adverse effects. Although the fetal brain has a high threshold to withstand the effects of sustained hypoxia, once a critical level of deoxygenation is surpassed, the result is major brain damage with early death.<sup>6</sup>

Effects of CO intoxication on the fetus surviving a poisoning episode include limb malformation, microcephaly, hypotonia, persistent seizures, areflexia, and mental and motor disabilities. Autopsies of infants who later died of CO intoxication have revealed brain damage, including neuronal loss in the cerebral cortex, abnormalities of the cerebellum, and injury to the basal ganglia.<sup>7,8</sup>

Fetuses exposed to mild or moderate accidental CO poisoning usually exhibit normal physical and neurobehavioral development.<sup>3,9</sup> Of note, at the time of presentation, maternal COHb levels did not correlate with concurrent symptoms. It seemed that symptoms of pregnant women exposed to CO predicted associated fetal morbidity or mortality risk.<sup>2,3</sup>

While pregnant women may present alterations of some physiologic functions, clinical symptoms such as nausea, headache, and fatigue may also be present in non-pregnant patients, so they are non-specific. Cases of CO intoxication may be unsuspected or attributed to other etiologies such as stroke without a clear episode of accidental CO exposure. Therefore, CO intoxication requires a high level of suspicion. The condition is diagnosed based on initial history, physical/laboratorial tests including measurement of blood COHb level, and assessments of environmental CO levels like those for non-pregnant woman.

Regardless of pregnancy, HBO<sub>2</sub> therapy is valuable in treating CO poisoning due to its effects. First, HBO<sub>2</sub> increases oxygen concentration and causes the oxygen dissociation curve to shift from the left to the right. Second, by diminishing CO interaction with cytochrome oxidase, HBO<sub>2</sub> allows mitochondria to more effectively use oxygen. Third, HBO<sub>2</sub> may lessen lipid peroxidation, which may be implicated in delayed CNS sequelae and neurological deterioration. Finally, decreased CO influenced through HBO<sub>2</sub> may decrease changes to the myocardium that result from CO poisoning, if the patient already has cardiovascular disease.<sup>10-12</sup> HBO<sub>2</sub> CO intoxication treatment guidelines suggest that patients with COHb levels of 25% or more should receive treatment regardless of symptoms.<sup>13</sup> Cases with COHb levels under 25% should receive HBO<sub>2</sub> therapy when there are neurological findings.

Previous clinical and experimental studies have shown that HBO<sub>2</sub> should treat CO intoxication to avoid adverse fetal consequences, particularly for pregnant women with either 20% or higher COHb levels or impaired consciousness.<sup>7,10,14-17</sup> On the other hand, treatment during pregnancy may be controversial primarily due to concerns regarding fetal oxygen toxicity due to high partial oxygen pressures. Adverse fetal effects induced by high oxygen partial pressures include

retinopathy of prematurity, teratogenicity, and cardiovascular effects, particularly premature closure of the ductus arteriosus and alterations in placental blood flow. Although several studies have shown HBO<sub>2</sub> to be effective for cases of moderate- to high-risk CO poisoning such as our patient, the risk of HBO<sub>2</sub> therapy needs to be weighed against possible maternal and fetal morbidity and mortality to achieve the safest and most effective treatment.<sup>13,17</sup>

The use of HBO<sub>2</sub> as an adjuvant treatment remains limited because of accessibility in some regions, believed lack of evidence to support its benefits, and relatively high cost. However, HBO<sub>2</sub> should be administered more liberally for pregnant patients because of the fetus' greater susceptibility to CO and hypoxia. Importantly, pregnant patients should be instructed regarding prevention of CO exposure.<sup>18</sup>

In conclusion, the present case upholds previously published guidelines for HBO<sub>2</sub> treatment of CO poisoning during pregnancy. As CO negatively affects both the fetus and mother, it is critical for emergency physicians to promptly recognize and manage such poisoning cases.

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## CONFLICT OF INTEREST

None declared.

## AUTHOR CONTRIBUTIONS

HM, TN, TO, AN, and HN: participated in patient management and data collection, contributed to the interpretation of the case, and critically reviewed the manuscript. YK, and AN: participated in patient management, collected and analyzed the data, wrote the manuscript, and critically reviewed the manuscript. All authors approved the final manuscript as submitted.

## ETHICAL APPROVAL

Appropriate consent has been obtained, prior to submission, for the publication of data.

## DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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