

Preterm labor in mild carbon monoxide poisoning: a case report

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Summary

Introduction: Carbon monoxide (CO) is a common toxic gas, and CO poisoning in pregnant women can be particularly dangerous, as the fetus is more vulnerable to CO exposure. Even if the mother experiences only mild symptoms and has low carboxyhemoglobin (COHb) levels following exposure, fetal COHb levels can be much higher. Therefore, there is no clear consensus for defining mild CO poisoning during pregnancy. Here, we report a case of preterm delivery following mild CO intoxication. **Case report:** An 18-year-old female at 36 weeks of gestation visited the hospital with CO poisoning 1 h after exposure. She had only mild headache and COHb level was checked 3.9%. Hyperbaric oxygen (HBO₂) therapy was not provided because she and her husband refused this treatment. Consequently, normobaric oxygen therapy was initiated. Five hours later, her symptoms improved, and thus, the therapy was discontinued. Eight hours later, the patient experienced labor pain, and the baby was delivered by spontaneous vaginal delivery. Neonatal COHb was 1.3% at birth, and the newborn was healthy. **Conclusion:** If maternal symptoms are mild following low CO exposure, normobaric oxygen therapy or HBO₂ therapy can be considered. However, whatever we choose, we should be prepared for premature birth.

Key words: Carbon monoxide poisoning; Preterm labor; Fetal carboxyhemoglobin; Pregnancy.

Introduction

Carbon monoxide (CO), a byproduct of burning carbon-containing materials, is a colorless, odorless, and tasteless gas. CO poisoning during pregnancy can be dangerous to both the mother and the fetus. In an animal study, when the mother was exposed to CO, maternal carboxyhemoglobin (COHb) rapidly increased and was transferred to the fetus through the placenta [1]. To clearly understand the extent of fetal CO poisoning, direct measurement of fetal COHb may be the best alternative. However, collection of blood from the umbilical cord during pregnancy is dangerous, and maternal COHb is not an accurate indicator of fetal COHb [2].

The fetus can experience greater toxicity even if the mother has mild symptoms as fetal COHb reaches greater levels than those observed in the mother. In addition, COHb clearance is five times slower in the fetus than in the mother [3]. However, there is no clear consensus on whether to actively initiate hyperbaric oxygen (HBO₂) therapy in pregnant women with mild CO poisoning. In this case, we report a case of preterm delivery following mild CO intoxication.

Case Report

An 18-year-old female at 36 weeks of gestation presented to the emergency department of a tertiary university hospital via taxi with CO poisoning. She was exposed to CO for approximately 10 min when she was trapped in a fire, and arrived at the hospital 1 h after exposure. She presented with only a mild headache. She was a non-smoker. Her initial evaluation findings were as follows: Glasgow Coma Scale score, 15; blood pressure, 120/80 mmHg; heart rate, 80 beats per minute; respiratory rate, 20 per minute; body temperature, 36.3 °C; and peripheral capillary oxygen saturation, 99%.

Normobaric oxygen therapy was initiated through a non-rebreather mask ventilator with 15 L/min of O₂. At initial presentation, the amount of COHb was 3.9%. The patient's cervix was soft and closed with a station of -2, and the nitrazine test was interpreted as negative with a pH of 5.0. Non-stress test was normal, and fetal heart rate was 150 beats per minute. A 6-mm-long cervix, vertex presentation of the fetus, and an amniotic fluid index of 8.3 cm were observed on the emergency obstetric ultrasound. HBO₂ therapy was not initiated because the patient and her husband refused this treatment. Consequently, normobaric oxygen therapy was initiated. Five hours later, her headache improved, and thus, the therapy was discontinued. Eight hours later, she experienced labor pain, and spontaneous vaginal delivery was performed by the obstetrician. The patient gave birth to a live male with Apgar scores of 6 and 8 at the first and the fifth minutes, respectively, and a weight of 2420 g. Maternal COHb was 1.1%, and fetal COHb was 1.3% as determined using the umbilical cord blood at birth.

On day 3 after birth, neonatal brain ultrasound and echocardiogram were normal. There were no specific findings on the brain ultrasound, which was performed again on day 10 after birth. The baby was discharged on day 10 after birth.

Discussion

The binding affinity of CO to hemoglobin is 200 times higher than that of O₂. CO causes hypoxia in its hemoglobin-bound form of COHb. In this case, the patient's exposure time was approximately 10 min, which resulted in mild headache, and an initial COHb level of 3.9%. Considering that she was a non-smoker and that an hour had lapsed be-

fore receiving treatment, the actual COHb was expected to be higher than the initial COHb. The half-life of CO is 300 minutes in room air and 37-120 minutes under normobaric oxygenation conditions [4]. Moreover, an elevated COHb level (2% for nonsmokers and 9% for smokers) strongly suggests exposure to exogenous CO and supports a clinical diagnosis of CO poisoning [5]. Therefore, the patient was deemed to have experienced CO exposure.

In the late gestational stage, CO poisoning is associated with neurologic sequelae due to anoxic events [6]. The fetus is more vulnerable to CO poisoning because the fetus experiences higher COHb levels than the mother [3]. There is no consensus regarding when to use HBO₂ therapy during pregnancy. However, HBO₂ therapy should be performed if fetal distress exists, because CO can be fatal to the fetus, even if the mother is asymptomatic [7]. In cases of CO poisoning in pregnant women, measurement of fetal COHb may predict how CO affects the fetus. However, it is difficult to measure fetal COHb directly during pregnancy. Thus, in our case, we could not measure fetal COHb. Therefore, we suggested HBO₂ therapy because the patient complained of headache and fetal COHb would eventually equilibrate at oxygen concentrations 10–15% higher than those in the atmosphere [8]. However, the patient refused HBO₂ treatment. Thus, normobaric oxygen therapy was initiated instead of HBO₂ therapy for 5 hours until the headache disappeared. Three hours after discontinuing oxygen therapy, the patient eventually delivered preterm, and maternal COHb was 1.1%, and neonatal COHb at birth was 1.3%.

Possible fetal consequences of CO poisoning during pregnancy include preterm delivery, hypoxic ischemic encephalopathy, hypotonia, cerebral palsy, areflexia, persistent seizures, microcephaly, cardiomegaly, limb malformations, and death [8]. In our case study, although preterm labor occurred, the neonate appeared healthy. The causes of preterm labor include infection, stress, malnutrition, hypertension, and diabetes [9]. CO poisoning may be one of the causes of preterm labor. One study found that increased atmospheric CO levels due to air pollution could cause premature birth. This is because CO interferes with the metabolism in and transport through the placenta [10]. In our case study, the patient did not exhibit any sign of infection, malnutrition, or medical conditions such as hypertension or diabetes. Unfortunately, the cause of preterm labor couldn't be determined. However, we may assume that premature birth was caused by CO poisoning or stress associated with CO poisoning as the patient had no other known causes that could lead to this outcome.

We should consider HBO₂ treatment when treating pregnant women for CO poisoning, and we may be prepared for the possibility of premature birth.

Conclusions

CO poisoning in pregnant women can be fatal to both the fetus and the mother. However, if maternal symptoms are mild following low CO exposure, normobaric oxygen ther-

apy or HBO₂ therapy can be considered. However, no matter what treatment is selected, the healthcare professionals should be prepared for premature birth.

Ethics Approval and Consent to Participate

All subjects gave their informed consent for inclusion before they participated in the study. The study was conducted in accordance with the Declaration of Helsinki, and the protocol was approved by the Ethics Committee of Soonchunhyang University Bucheon Hospital (approval number: 2019-12-011).

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Conflict of Interest

The authors declare no conflict of interest.

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