CASE REPORT

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Fetal Death Due to Nonlethal Maternal Carbon Monoxide Poisoning

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ABSTRACT: Fetal death due to acute carbon monoxide poisoning is rarely reported in the medical literature. Of the eight cases found in literature review, only one documented the fetal carboxyhemoglobin concentration. This paper reports a fetal death due to accidental nonlethal maternal carbon monoxide intoxication in which both maternal and fetal carboxyhemoglobin concentrations were obtained. The corrected carboxyhemoglobin concentration was 61% at the time of death in utero, while the maternal carboxyhemoglobin was measured at 7% after one hour of supplemental oxygen. The authors review the mechanisms of fetal death and emphasize the different carbon monoxide kinetics in the fetal circulation.

KEYWORDS: toxicology, carbon monoxide, fetal death

Carbon monoxide (CO), the gas produced by the incomplete combustion of carbon-containing materials, is the leading cause of poison-induced deaths in the United States [*1*]. This environmental hazard is ubiquitous and, in homes, is usually produced in toxic concentrations by faulty heating units which are poorly ventilated [*2*]. Approximately 3500 to 4000 deaths each year are attributed to carbon monoxide intoxication in the United States [*1*]. Fetal death due to maternal CO poisoning has only rarely been reported. Of the eight cases published in the English-language medical literature, only one report provides the carboxyhemoglobin (COHb) concentrations of both the mother and the fetus [*3*]. We present the circumstances of another fetal death due to a nonlethal maternal CO poisoning in which COHb levels were obtained for both mother and fetus. Documentation of the COHb levels provides us with an opportunity to emphasize the

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difference in CO kinetics between the mother and the fetus and to call attention to the current recommendations for treating this medical emergency when a pregnant female is involved.

Case Report

A 20-year-old white female arrived by ambulance at the hospital approximately 60 min after being found unconscious at her mobile home. She had been intubated by the Emergency Medical Services and had received 100% supplemental oxygen en route to the hospital. The patient's 21-year-old husband was also found at the scene and brought to the hospital. Although initially disoriented, restless, and combative, he was lucid at the time of arrival in the emergency department. From his history, it was determined that the couple's usual heater was in disrepair, and a portable propane heater was the sole source of heat in their unventilated mobile home. He also disclosed that his wife was 28 weeks into her first pregnancy, that her past medical history was unremarkable, and that she was not currently taking medications or using cigarettes.

During the initial physical examination, the patient was noted to be combative and confused. Her blood pressure was palpable by cuff measurement at 80 torr systolic. She was being ventilated by a volume-cycled ventilator which she triggered 26 times a minute. Carbonaceous material was found in the nares, oropharynx, and adherent to the endotracheal tube. No burns of the skin, nasal hair, face, or eyebrows were present. Abdominal examination results were consistent with a 28-week intrauterine pregnancy.

No fetal movement could be detected by ultrasound, and fetal heart sounds were absent. Peripheral cyanosis was noted in her nail beds. The measured carboxyhemoglobin concentration at the time of admission was 7%. A plasma and urine toxicology screen was negative. The initial chest radiograph was interpreted as showing bilateral alveolar infiltrates consistent with the adult respiratory distress syndrome.

On the second hospital day, the patient went into labor spontaneously and delivered a 1050-g stillborn female fetus of approximately 7 months gestation, with a crown-heel length of 39 cm and crown-rump length of 27 cm. The gross autopsy findings were remarkable only for bright red discoloration of the skin and visceral organs. The corrected fetal COHb saturation at the time of the autopsy was 61% by IL 282-CO-Oximeter[3] [4]. On microscopic examination of the tissues, the expected autolytic changes were seen but no other diagnostic abnormalities were found.

The mother began a slow convalescence after delivery of the fetus and subsequently recovered normal pulmonary function. She was discharged on the ninth hospital day.

Discussion

Approximately 3500 to 4000 deaths each year in the United States are caused from carbon monoxide (CO), the nonirritating, odorless, tasteless, and colorless inert gas that is produced by the incomplete combustion of carbon-containing materials [1].

CO has an affinity for reversibly binding with adult hemoglobin that is 250 times greater than that of oxygen [5]. Measurement of the carboxyhemoglobin (COHb) level provides the clinician with an objective parameter to correlate with clinical symptoms and prognosis. Because CO is endogenously produced in humans during metabolism of protoporphyrin to bilirubin during hemoglobin metabolism, a nonsmoking individual may have a normal resting COHb saturation of 1 to 3% [6]. Cigarette smokers will commonly have

[3]The IL 282 CO-Oximeter is manufactured by Instrumentation Laboratory, Inc., Lexington, MA 02173.

COHb levels of 5 to 6%, and, if they smoke continuously, COHb levels may reach 8 to 10% [7].

CO interferes with cellular metabolism by inhibiting the transport, delivery, and utilization of oxygen. CO successfully competes for oxygen binding sites. The hemoglobin molecule will then bind more avidly to the oxygen molecules on its surface, resulting in a shift of the oxyhemoglobin dissociation curve to the left. Finally, CO poisons cellular respiration by displacing oxygen from receptors of the cytochrome oxidase system, particularly cytochrome a3 and cytochrome P-450 [6].

In adults, COHb concentrations of 30 to 40% are generally associated with weakness, dizziness, nausea and vomiting, and cardiovascular collapse. Syncope, seizures, and death may occur with COHb levels of 50% [8]. Inhalation of ambient gas which has a CO concentration of 1% can be fatal within 10 min, depending on the victim's activity and respiratory rate [9]. Likewise, victims with underlying diseases such as anemia, heart dysfunction, or lung disease may succumb at much lower COHb concentrations [7].

Interestingly, Goldbaum [10] has shown that CO is toxic only when inhaled through the lungs and not when introduced by intraperitoneal injection or transfusion with CO-saturated red cells. He asserts that the toxic effects of CO are not caused by elevated COHb alone, such as occurs in the latter two instances, but also by direct action of dissolved CO on the cytochrome oxidase system. Thus, he cautions that COHb determination may be misleading without knowledge of environmental conditions and the respiratory status of the individuals affected.

Investigations of CO exchange between mother and fetus have shown that CO absorption and elimination occur more slowly in the fetal circulation [11,12]. Following maternal exposure to CO, the fetal COHb rises more slowly than the maternal COHb but will continue to rise for several hours after acute exposure until it eventually reaches an equilibrium at a level which is approximately 10% greater than the mother's COHb saturation [12]. The pattern of elimination of CO is similar, and fetal COHb concentrations diminish more slowly than maternal COHb levels [13]. Although there is some evidence that the placenta has some capacity to transport CO actively, passive diffusion through the placenta along a partial pressure gradient is thought to account for the bulk of fetal CO absorption and elimination [14].

Our case further illustrates the differing CO kinetics between the maternal and fetal circulations. The normal COHb half-life of 4 to 5 h when breathing room air with an oxygen concentration of approximately 21%, can be markedly shortened by inhaling oxygen at higher concentrations. If 100% supplemental oxygen is administered, the COHb half-life is shortened to 60 min [8]. Approximately 40 to 50% of the body's CO can be eliminated in 1 h when high fractions of oxygen are rapidly administered by emergency rescue teams [7]. Unfortunately, it is estimated that it takes four to five times as long for the fetal COHb to decrease to the same maternal COHb level [14]. The implications for treatment of the pregnant victim are clear.

Fetal tissues are at greater risk from hypoxia caused by CO since a higher COHb equilibrium is achieved, COHb elimination is delayed, and the fetal hemoglobin experiences a more accentuated left shift than does adult hemoglobin [15]. This particular susceptibility of fetal tissue to CO toxicity has promoted interest in utilizing hyperbaric oxygen for the treatment of the pregnant patient with CO poisoning. With the use of 100% oxygen at 2 to 3 atmospheres of pressure, the COHb elimination can be significantly enhanced in both mother and fetus [14]. In lieu of this modality, supplemental oxygen at concentrations approaching 100% should be administered for five times the length of time needed to reduce the maternal COHb to acceptable concentrations [14].

The mother in our report is assumed to have reached a minimal COHb concentration of 40 to 50% since she sustained loss of consciousness. Therefore, with the kinetics described, the fetus would be expected to develop a COHb level of at least 65%. To

explain the disparity between maternal and fetal COHb concentrations in our case, it is necessary to postulate that fetal death occurred at the time that the fetal circulation reached its maximal COHb saturation. According to available research information, the fetal COHb concentration is not expected to change after death in utero. CO is not produced by decomposition, nor is it absorbed in significant amounts by a body when exposed to an environment rich in CO. Indeed, COHb persists for weeks in the human body and may be accurately quantified even after embalming and burial [7].

In chronic or nonfatal CO poisoning, degenerative changes can often be seen involving the basal ganglia, kidneys, liver, and heart. These lesions are indicative of severe tissue hypoxia and are not pathognomonic of CO poisoning [9]. In acute asphyxia due to CO, death occurs before these lesions can develop. The fetal autopsy suggested COHb poisoning by the cherry red discoloration of the skin and visceral organs.

The carboxyhemoglobin concentration that causes fetal death in humans has not yet been defined. The clinician's ability to predict fetal survival is poor because the fetal COHb concentration cannot be estimated from a single determination of maternal concentration without knowledge of the maternal exposure pattern. At the present time, the best predictive index of fetal morbidity and mortality appears to be the severity of maternal symptoms at the site of exposure.

The authors hope that this account of fetal death associated with nonfatal maternal exposure to CO, which is only the second report to include measurement of both maternal and fetal COHb saturations, will alert both death investigators and clinicians to the fragile relationship that exists between mother and fetus under circumstances of CO exposure.

Addendum

Since the completion of this report, an additional recent case of nonlethal maternal CO poisoning with fetal death has come to the authors' attention.[4]

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