

## Case Report

# Fetal death due to nonlethal maternal acute carbon monoxide poisoning in a 28 weeks pregnancy

Ozhan Ozdemir<sup>1,\*</sup>, Cemal Resat Atalay<sup>1</sup>, Mustafa Erkan Sari<sup>1</sup>, Bunyamin Ugur Ilgin<sup>1</sup>

<sup>1</sup>Department of Obstetrics and Gynecology, Ankara Numune Education and Research Hospital, Ankara, Turkey

## Abstract

Carbonmonoxide poisoning in pregnancy is a relatively rare occurrence, with potentially serious complications for both mother and fetus. It is known that fetal effects of carbon monoxide intoxication are more severe than maternal effects. Because the fetal hemoglobin shows much more affinity to carbonmonoxide than the maternal hemoglobin and the clearance rate of carboxyhemoglobin from fetal blood is five times longer than the maternal blood clearance. During the pregnancy exposure to carbonmonoxide may result in stillbirth, abortion, fetal abnormalities, intrauterine growth restriction, functional deformities due to brain defects. In this case report, we summarize a case of a pregnant woman who was toxicated with carbon monoxide and had intrauterine exitus in the 28th week of pregnancy, well treated with hyperbaric oxygen and then induced for labor. We aimed to discuss the features of carbonmonoxide toxication, importance of the early and exact diagnosis and, the possible fetal effects of the carbonmonoxide toxication and treatment modalities.

## Key Words:

Carbonmonoxide, poisoning, pregnancy

## Introduction

Acute carbonmonoxide (CO) poisoning in pregnancy is a relatively rare condition with serious adverse effects to the mother and fetus, and the resulting intrauterine hypoxia can cause fetal death or severe neurological sequelae [1]. The pathophysiology of CO toxicity primarily involves cellular hypoxia due to impaired oxygen transport by haemoglobin, as CO binds to haemoglobin more avidly than oxygen (230–270 times). Other mechanisms of CO toxicity include direct toxic effects on respiratory cytochromes, brain lipid peroxidation, leucocyte-mediated inflammatory changes in the brain, and release of nitric oxide free radical from platelet and vascular endothelium [2]. It is known that fetal effects of CO intoxication are more

severe than maternal effects. Because the fetal hemoglobin shows much more affinity to carbonmonoxide than the maternal hemoglobin and the clearance rate of carboxyhemoglobin (COHb) from fetal blood is five times longer than the maternal blood clearance. The fetal COHb levels created are 10–15% higher than maternal levels, as fetal haemoglobin has a higher affinity for CO than adult haemoglobin. Moreover, fetal elimination of CO takes more time, for it dissociates much more slowly from fetal than adult haemoglobin [3]. Acute maternal CO poisoning is associated with a maternal mortality between 19 and 24% and a fetal mortality between 36 and 67% [4]. In this case report we summarize a case of a pregnant woman who was toxicated with CO and had intrauterine exitus in the 28th week of pregnancy, well treated with hyperbaric oxygen and then induced for labor.

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\*Correspondence: Ozhan Ozdemir

Address: Department of Obstetrics and Gynecology, Ankara Numune Training and Education Hospital, 06010, Ankara, TURKEY

Tel: +905052255078

E-mail: seyozi@hotmail.com

## Case Presentation

A 31 year old (gravida 3, para 2) at 28 weeks gestational age was exposed to carbon monoxide CO. CO was produced

by a defective water heater. The patient was transferred to the hospital around 5 hours after the exposure. She did not lose consciousness and her vital signs were regular pulse rate of 132 beats per minute, blood pressure of 120/75 mm Hg and respirations of 45/mn. She complained of dizziness and palpitation. Her medical and family history was unremarkable. Peripheral cyanosis was noted in her nail beds. Abdominal examination results were consistent with a 28-week intrauterine pregnancy. No fetal movement could be detected by ultrasound, and fetal cardiac activity was absent. The measured COHb concentration at the time of admission was 33%. The arterial blood gas analysis was as follows; pH-7.36, pCO<sub>2</sub>-25 mmHg, pO<sub>2</sub>-14,5 mmHg and O<sub>2</sub> saturation-40,5%. High flow oxygen therapy was started immediately. The patient was then treated with 100% O<sub>2</sub> at 2.5 atmospheres absolute for 90 minutes. Treatment with hyperbaric oxygen was started in the 5 hours after the end of exposure. A dramatic improvement in oxygen saturation was observed immediately afterwards. On the second hospital day, the patient went into labor with an i.v. infusion of oxytocin and delivered a 1010-g stillborn female fetus of approximately 28 weeks gestation. The gross findings were remarkable only for bright red discoloration of the skin. The level of carboxyhaemoglobin in the cord blood was 59 %. She was discharged in good health three day after exposure.

## Discussion

CO is an odorless, tasteless, colorless, nonirritating gas formed by hydrocarbon combustion. The non-specific symptoms of CO exposure include headache, nausea, vomiting, palpitations, dizziness, and confusion. As exposure increases, patients develop more pronounced and severe symptoms, and oxygen-dependent organs, such as the brain and heart, show the earliest signs of injury [5]. The clinical presentation of acute CO poisoning is variable, but in general, the severity of the observed symptoms correlates roughly with the observed level of COHb. The measurement of COHb levels is, at present, the most useful laboratory method for ascertaining the severity of exposure to CO. In a nonsmoker, the average carboxyhaemoglobin level is 1%, with levels of up to 15% seen in heavy smokers. Poisoning is considered to have occurred at COHb levels of over 10%, and severe poisoning is associated with levels over 20–25%, plus symptoms of severe cerebral or cardiac ischaemia. However, people living in areas of pollution

may have levels of 5%, and heavy smokers can tolerate levels up to 15%. Severe poisoning can be fatal, and up to a third of survivors have delayed neurological sequelae. CO elimination is slower in fetus than mother, fetal haemoglobin has greater affinity for CO than adult haemoglobin [6]. The fetus is particularly vulnerable to CO poisoning. During pregnancy, a woman's oxygen-carrying capacity is reduced because of an increased endogenous CO production and additional endogenous carbon monoxide from the developing fetus, leading to an increased COHb concentration. A higher ventilation rate during pregnancy will lead to increased uptake of carbon monoxide at any given CO concentration [7]. The fetus is also at risk, and there have been occasional fetal deaths in non-fatal maternal exposures. In the developing fetus, oxygen is released at a lower oxygen partial pressure, and fetal haemoglobin binds with CO more quickly compared with adults. Fetal death due to nonlethal maternal CO poisoning [8]. CO may be a teratogen and the effects of CO on fetal development differ by term. During the embryonic phase, CO can cause a variety of birth defects. During the fetal stage, congenital anomalies are less common, but death or permanent neurological damage may occur. Other toxic effects include fetal growth retardation, premature delivery and sudden infant death [9]. The treatment of acute CO poisoning during pregnancy begins by removing the victim from the environmental source of CO. Then 100% normobaric oxygen should be administered immediately and hyperbaric oxygen should be considered. The only possible nonteratogenic treatment for pregnant women with CO poisoning is hyperbaric oxygen therapy. Hyperbaric oxygen is mandatory for all pregnant women with either impaired consciousness or COHb levels of 20% or higher [10]. In conclusion, acute CO poisoning during pregnancy is comparatively uncommon, yet can result in fetal death and functional alterations or anatomical malformations in survivors. Hyperbaric oxygen seems to be the treatment of choice and especially when the carboxy-haemoglobin concentration is > 20%, hyperbaric oxygen therapy is urgent.

### Acknowledgement

None

### Declaration of Interest

None

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