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CASE REPORT

Carbon monoxide poisoning and myocardial injury in pregnancy: a case report

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ABSTRACT

Background: Carbon monoxide (CO) is a colorless and odorless gas from combustion sources causing accidental or intentional poisoning. Patients present with nonspecific symptoms such as headache and dizziness, but severe cases can lead to altered mental status. Myocardial injury is a life-threatening outcome of CO poisoning, requiring timely evaluation and management.

Case Presentation: A 33-year-old pregnant female with no prior co-morbidities presented to the emergency department with dizziness, vomiting, and syncope. She had been exposed to CO from a petrol generator, resulting in a carboxyhemoglobin level of 9.6%. The patient exhibited global hypokinesia and reduced ejection fraction. After receiving oxygen therapy, she showed improvement and was discharged in stable condition.

Conclusion: This case underscores the importance of early recognition and management of myocardial injury in CO poisoning, particularly in pregnant patients. Prompt oxygen therapy and appropriate follow-up are crucial to mitigate long-term cardiovascular and fetal complications.

Keywords: Case report, carbon monoxide poisoning, pregnancy, myocardial injury, cardiomyopathy.

Introduction

Carbon monoxide (CO) is a toxic gas, also known as a silent killer, produced by sources of combustion such as generators and heaters causing accidental poisoning or used for intentional suicide attempts. Patients often present with nonspecific symptoms, hence it is a vague toxidrome often leading to misdiagnosis. In addition to the well-known neurological sequel, myocardial injury is another major outcome of CO poisoning and may lead to acute as well as possible long-term complications if not carefully evaluated and managed in a timely manner by emergency physicians upon initial presentation [1].

In this study, we present a unique case of a pregnant patient with cardiomyopathy as a consequence of acute CO poisoning. Our aim is to provide insight into the possible cardiac complications caused by CO toxicity in otherwise healthy adults, with emphasis on the effect on pregnant individuals, and how to correctly identify and manage cardiac causes in the emergency department (ED). To the best of our knowledge, a comprehensive Medline search yielded no reports of similar presentations previously.

Case Presentation

A 33-year-old female, with no previous known co-morbidities, presented to the ED via the ambulance with complaints of dizziness, vomiting, and an episode of syncope. On arrival at the ED, the patient was alert and oriented with normal vital signs except for a blood pressure of 83/45 mmHg. Chest examination was normal. Oxygen saturation on room air was 100%. There were no signs of respiratory distress or cyanosis.

The patient worked in a villa with two other occupants. Upon further questioning, there was a petrol generator in the vicinity that was currently being used due to a lack of electricity for the past week. The room she shared with another cohabitant had no additional sources of ventilation or windows. Upon waking up that morning, she realized her roommate and dog had passed away, and she felt unwell and called for help. She denied any recent pesticide, construction, or paint use in the house. There was no recent history of illness, fever, chest pain, abdominal pain, or shortness of breath. The patient also denied alcohol, tobacco, and substance abuse.

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Primary and secondary surveys were otherwise unremarkable other than some mild swelling and bruising over the left eye and a 4 cm chin laceration. Bedside point-of-care ultrasound was conducted, following the Rapid ultrasound for shock and hypotension (RUSH) protocol for rapid ultrasound for hypotension and shock, which was negative apart from a dilated inferior vena cava (IVC) and global hypokinesia of the heart.

Laboratory investigations are depicted in Table 1. The patient was suffering from acute CO poisoning with a carboxyhemoglobin (COHb) level of 9.6%. Levels equal to or >2% in non-smokers and 10% or more in smokers, are considered abnormal. She also had an incidental positive beta-human chorionic gonadotropin (β-hCG) in her blood indicating early pregnancy, as well as increased cardiac biomarkers as well as high inflammatory markers. On repeat of labs prior to discharge a significant decrease of the cardiac biomarkers and COHb, and an increase in β-hCG was seen.

An electrocardiogram (ECG) showed sinus rhythm without any QT interval prolongation. Computed tomography brain, face, spine, and chest X-rays were all unremarkable.

A bedside echocardiogram conducted by the cardiologist showed global hypokinesia with depressed left ventricular systolic function with an ejection fraction of about 25%-30% visually. The right ventricle was mildly dilated whereas the left ventricle was normal. Trace tricuspid regurgitation was seen and right ventricular systolic pressure of approximately 30 mmHg. IVC measured 1.8 cm with no inspiratory collapse. There were no valvular lesions or pericardial effusion.

The patient was resuscitated with 1 l of sodium chloride 0.9%, and oxygen therapy at 10 l per minute via non-rebreather mask which improved the CO level to 2.2% in approximately 2 hours and eventually to 0.5% the next day. She was also given metoclopramide and pantoprazole as part of symptomatic treatment. She was then started on piperacillin/tazobactam in view of high inflammatory markers though blood culture later showed no growth and it was discontinued.

The patient was admitted for 5 days under the medical team while being followed by the toxicology, cardiology, and obstetrics departments. She had an uncomplicated stay and pregnancy in the ward and was discharged in stable condition. She was advised to follow up with the obstetrics clinic as this is her first pregnancy and the cardiology clinic as an outpatient for further evaluation. However, the patient was lost to follow up as she traveled back to her home country.

Table 1. *Pertinent laboratory values.*

Laboratory parameter	Value	After 5 days	Reference range
WBC (×10³/µl)	18.4	7.2	3.6-11.0
Hgb (g/dl)	11.1	10.8	12.0-15.0
pH*	7.322	7.412	7.350-7.450
pCO_2* (mmHg)	48	34	40-50
pO_2* (mmHg)	21.5	181	30-55
HCO_3* (mmol/l)	22	21.3	21-28
COHb* (%)	9.6	0.5	0.5-1.5
Lactate* (mmol/l)	2.8	2.0	0.5-1.6
LDH (U/l)	265	226	105-222
CRP (mg/l)	15.1	6.3	<5
PCT (ng/ml)	2.23	1.16	<0.05
Troponin T (ng/l)	593	417	<14
CKMB (nl/ml)	84.8	7.9	<4.89
NT-proBNP (pg/ml)	1,319	503.8	<125
D-dimer (µg/ml)	1.75	1.35	<0.5
β-hCG (mIU/ml)	133	317	<5

*: VBG. WBC: White blood cells; Hgb: Haemoglobin; pH: Potential of hydrogen; VBG: venous blood gas; pCO2: Partial pressure of carbon dioxide; pO2: Partial pressure of oxygen; HCO3: Bicarbonate; COHb: Carboxyhaemoglobin; LDH: Lactate dehydrogenase; CRP: C-reactive protein; PCT: Procalcitonin; CKMB: creatine kinase-myoglobin binding; NT-proBNP: N-terminal pro–B-type natriuretic peptide; β-hCG: Beta human chorionic gonadotropin.

Discussion

CO has approximately 200 times more affinity toward hemoglobin compared to oxygen, forming COHb. CO also binds to mitochondrial cytochrome C, decreasing protocol Adenosine triphosphate (ATP) production. Due to this competitive inhibition of oxygen release, tissue hypoxia occurs which in turn can lead to reperfusion injury. Cardiac dysfunction can result from oxidative stress due to high CO levels binding to myoglobin in myocytes as well as reduced nitric oxide levels [1]. Additionally, CO increases free radical production which in turn causes endothelial dysfunction leading to increased coronary vasoconstriction. At toxic levels, CO may even increase the risk of thrombosis as it increases platelet aggregation by binding to fibrinogen-bound heme [2].

Patients often present with unspecific symptoms such as headache, dizziness, nausea, weakness seizures, or respiratory arrest which may lead to coma and even death [3]. Diagnosis is made on a clinical triad of consistent symptoms, history of CO exposure, and COHb level. A COHb level as low as 2% is abnormal and can produce symptoms, whereas a level of 10% and above is required for diagnosis in chronic smokers [2]. Concomitant cyanide poisoning should be considered especially after fire incidents and exposure.

Cardiovascular injury may occur as a consequence of CO poisoning and should be considered and carefully evaluated. Some common cardiovascular injuries include acute myocardial infarction, arrythmias, left ventricular dysfunction, and acute heart failure [4]. Henry et al. [3] published a cohort study on the long-term mortality outcome of patients with CO toxicity. In their study, cardiovascular death was more common (44% compared to 18%) in those who initially suffered from myocardial injury inpatient. Of these patients, 38% died at a median follow up of 7.6 years [3]. In another study, they found a strong correlation between COHb level and the risk of developing myocardial infarction as a long-term complication [5]. Additionally,

those with preexisting coronary artery disease and/or co-morbidities such as hypertension have an increased risk of suffering from severe cardiovascular events, so one must maintain a high level of suspicion. Hence, it is crucial to investigate by obtaining cardiac biomarkers and an ECG early on, and to keep the patient on a cardiac monitor [6].

CO poisoning in pregnancy is uncommon but can be detrimental to both the mother and fetus. The pregnancy outcome depends on the severity of the initial symptoms, exposure duration, COHb concentration, and baseline health of the individual. CO readily passes through the placenta causing fetal toxicity. Another mechanism is fetal hypoxia which is the result of decreased maternal oxygen release by hemoglobin and reduced oxygen transport through the fetal umbilical vein. According to multiple case reports published in the literature, anatomical malformations have been found to be more common during early gestational age whereas fetal brain hypoxia has been found during late gestation [7]. Maternal COHb level is an inaccurate indicator of fetal toxicity. Fetal COHb concentration is estimated to be around 10%-15% higher. In severe CO toxicity, fetal mortality can reach up to 67% [8].

Normobaric oxygen therapy is the initial treatment of choice until the CO level decreases or there is a resolvement of symptoms. Hyperbaric oxygen therapy (HBOT) is reserved for adult patients who present with loss of consciousness, cardiac ischemia, neurological deficits, metabolic acidosis, and/or COHb of more than 25% [6]. Traditionally, HBOT in pregnancy was a relative contraindication, however, with more research regarding improved fetal outcomes, it is moving toward becoming a strong indication in most circumstances where benefits outweigh risks particularly in severe cases [9]. Unfortunately, there are no studies evaluating HBOT at different stages of pregnancy nor on the development of the fetus. The reported cases regarding fetal teratogenicity are related to CO poisoning rather than the HBOT [10]. In pregnant women, it is advisable to treat more aggressively, as the fetus has a decreased ability to eliminate CO independently. Thus, oxygen therapy up to 5 times greater than the maternal need is required [11]. At the time, the hyperbaric chamber was unavailable at our facility, thus we initially considered transferring the patient to a nearby hospital. Nevertheless, the patient improved with conservative measures and was stable throughout her hospital course with low CO levels.

According to our Medline literature search results, there have not been similar previously published cases, therefore further research is required to establish an appropriate algorithm and proper guidelines for ED physicians to evaluate patients with CO poisoning for possible life-threatening cardiovascular events. Moreover, spreading awareness regarding CO poisoning and educating the community is crucial as a measure of prevention, especially in the more vulnerable pregnant population.

One limitation of our paper is that our patient was lost to follow up, hence we were unable to study the long-term maternal cardiovascular effects as the result of CO poisoning as well as the development of the fetus.

Conclusion

In this report, we highlight the importance of early recognition and evaluation of myocardial injury in all patients who present with CO poisoning, despite the absence of angina. Patients with CO toxicity who have positive cardiac biomarkers on initial presentation, warrant further work-up including an echocardiogram, serial troponin, and ECG. All patients with suspected CO poisoning should be initiated on 100% oxygen and considered for HBOT if they meet the inclusion criteria. Though, priority is for maternal stability, HBOT may also aid in reducing fetal injuries. Furthermore, adequate follow up with a cardiologist for risk stratification and monitoring of possible long-term complications of those who present with myocardial injury initially is strongly advised.

List of Abbreviations

B-hCG	Beta human chorionic gonadotropin
CO	Carbon monoxide
COHb	Carboxyhaemoglobin
ECG	Electrocardiogram
ED	Emergency department
HBOT	Hyperbaric oxygen therapy
IVC	Inferior vena cava

Conflict of interests

The authors declare that there is no conflict of interest regarding the publication of this article.

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Consent for publication

Waiver of consent was obtained as the patient was lost to follow up despite multiple attempts of contact.

Ethical approval

Ethical approval was granted by the hospital Institutional Review Board via reference number: MBRU IRB-2023-206, dated 18/8/2023 as exempted.

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