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Case Report

Acute Heart Failure Associated with Carbon Monoxide Intoxication during Pregnancy: Two Case Reports

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Abstract

Carbon monoxide poisoning is a leading cause of mortality among accidental intoxications, it is responsible for severe tissular hypoxia leading to life-threatening complications. Pregnant women are more at risk to develop severe forms of intoxication due to their physiological changes and their higher oxygen requirements as well as for possible adverse fetal outcomes. We present two cases of monoxide intoxication in two pregnant women with acute heart failure and variant complications with different fetal outcomes.

Keywords: Heart failure, carbon monoxide poisoning, pregnancy, dysrhythmias.

INTRODUCTION

Carbon monoxide (CO) intoxication is more frequently seen during the winter season due to defective heating systems. It is considered a potentially deadly condition that can cause multiple organ damage due to severe tissular hypoxia.

The alterations lie in the high affinity of CO to hemoglobin which is up to 300 times stronger than oxygen, leading to the formation of carboxyhemoglobin, a more stable form and not easily dissociated [1].

Hence, neurological and myocardial injuries are more frequently encountered due to their high oxygen needs. They are also associated with a substantially higher risk of morbidity and mortality [2]. These complications have been widely described in the literature but only a few of them report pregnancy and obstetrical particularities. We present two cases of CO intoxication in pregnant women with variable features of cardiovascular complications.

CASE PRESENTATION 1

A 23-year-old female patient at 27 weeks of gestation with no past medical history was admitted to the emergency department following a severe CO intoxication while bathing in a closed confined room from a firewood heater.

The patient was conscious and complained of palpitations and shortness of breath. Upon admission, her blood pressure was 110/54 mmHg, pulse rate at 240bpm, respiratory rate at 35cpm, and oxygen saturation level 97% at ambient air.

Clinical examination was unremarkable with normal breath sounds and no signs of right or left cardiac failure nor signs of inhalation injury. Initial blood gas results showed respiratory alkalosis with a pH of 7.50, paCO2 of 25.5mmHg, HCO3- of 20.2mmol/l, and paO2 at 88mmHg on 15l/min of oxygen. Measurement of blood concentrations of CO was not available in our hospital.

An electrocardiogram showed regular narrow QRS tachycardia at 240 bpm in favor of

supraventricular tachycardia (Figure 1). Given the hemodynamic stability, vagal maneuvers were undertaken as first-line treatment but were unsuccessful, followed by an intravenous bolus of Adenosine that was also ineffective. Electric cardioversion was performed and a regular sinus rhythm at 70bpm was reached at the third synchronic shock. Fetal heart rhythm was continuously monitored and revealed no signs of acute fetal distress. Shortly afterward, the patient presented acute pulmonary edema that responded to non-invasive ventilation with Continuous Positive Airway Pressure (CPAP) and diuretics.

Bedside cardiac point of care ultrasound showed a moderate systolic dysfunction with left ventricular ejection fraction at 48%, no dilated or hypertrophic ventricular or underlying valve disease. Left ventricular filling pressures were high as attested by the restrictive mitral flow pattern. Laboratory findings revealed a rise in troponin levels at 12 hours from admission (from 333ng/ml to 444ng/ml). Renal and hepatic functions were not impaired and muscle enzymes were within the normal values.

The patient remained in the intensive care unit for 48 hours on continuous non-invasive monitoring as the fetal heart rhythm remained normal. She received a daily dose of 100mg flecainide orally and was progressively weaned from CPAP and oxygen therapy.

Pregnancy was carried out without particular incident giving birth to a healthy baby at full term. 6 months follow-up showed normal electrocardiograms and echocardiography and no neuropsychiatric symptoms.

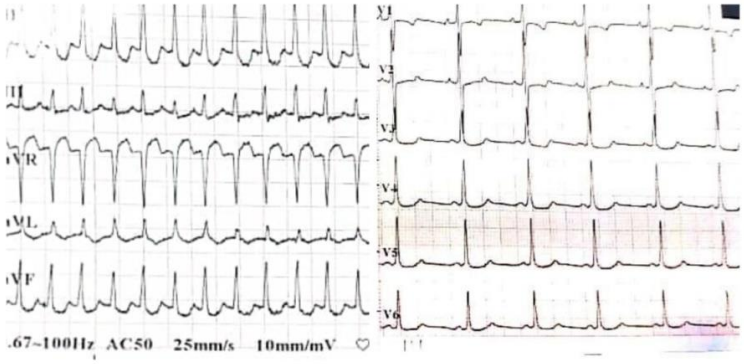

Figure 1: Electrocardiogram showing supraventricular tachycardia (left) and normal sinus rhythm after cardioversion with depression of ST-segment in leads V3 through V6 (right)

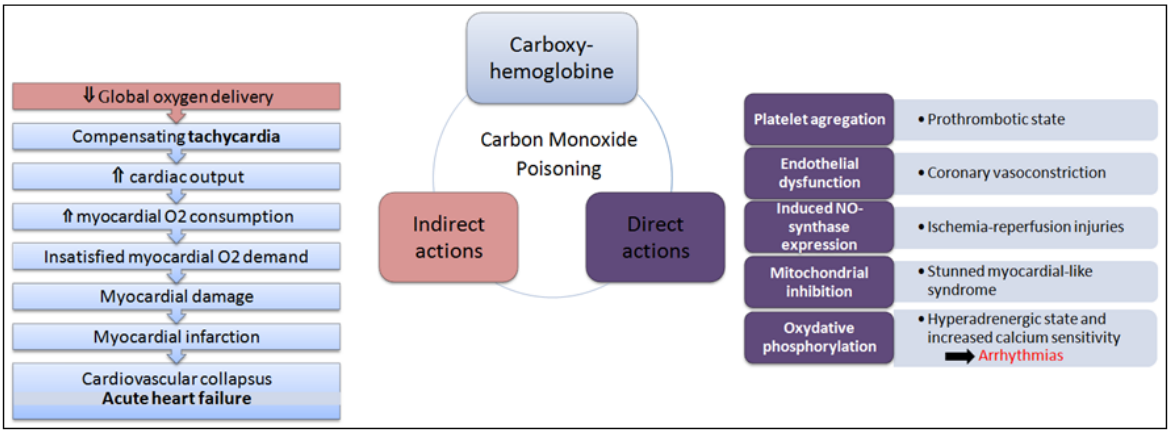

Figure 2: Pathophysiology of cardiovascular complications due to carbon monoxide intoxication

CASE PRESENTATION 2

A 25-year-old patient with no past medical history in her second trimester of pregnancy presented with a cardiogenic shock, rhabdomyolysis, renal and hepatic failures, and intrauterine fetal demise (IUFD) following prolonged exposure to carbon monoxide from a non-conforming water heater in a non-ventilated area.

Initial assessment revealed a confused patient with a Glasgow Coma Scale of 13, apathetic, presenting with hemodynamic and respiratory instability with prolonged capillary refill time and impregnable blood pressure, distended jugular veins, and inspiratory crackles at lung sounds.

Early treatment was quickly initiated consisting of high flow oxygen therapy by a non-rebreathing-face mask at 15 L/min, intravenous fluid

resuscitation, and norepinephrine perfusion restoring adequate tissue reperfusion. Further clinical examination revealed extensive inflammatory edema of the right lower limb, dark brown urine discoloration with oliguria. The obstetrical evaluation revealed intrauterine fetal demise (IUFD).

Blood gas results showed a compensated metabolic acidosis PH: 7, 38, PCO2:14, 9, Po2:173, Hco3-:9, 0, Sao2: 100%; serum lactate levels were at 2.25mmol/l. ECG was significant for sinus tachycardia and inferolateral depression of ST-segment. Bedside echocardiography was in favor of acute heart failure with non-dilated cavities, global hypokinesia, impaired systolic function with LVEF of 42%, and a collapsed inferior vena cava. Faced with persistent hemodynamic instability, dobutamine was used at a dose of 10µ/kg/min along with norepinephrine and cautious fluid challenges determined by invasive arterial and venous pressure measurements and under non-invasive continuous monitoring using ClearSight Edwards ® and repeated transthoracic echography.

Edema of the right lower limb was due to prolonged compression since venous ultrasound and angiography were normal. A biological workup revealed hemoconcentration with hematocrit at 45.5%, elevated serum creatinine levels at 33 mg/l, hyperkaliemia at 6.2mmol/l, severe rhabdomyolysis with creatinine kinase values at 56685UI/l (normal < 200UI/l), and high troponin levels at 2199 pg/ml (169 times normal value).

Fetal extraction was engaged by induced vaginal delivery on the second day of her admission after stabilization. The patient remained in the ICU for a total stay of ten days while being progressively weaned from vasoactive drugs and oxygen therapy by the 4th day, whereas complete recovery of cardiac function was obtained by day 7. Neurological assessment exposed retrograde amnesia without any other neurological deficit and non-oliguric renal failure persisted until discharge with a glomerular filtration rate of 20ml/min/1.73 m². Further follow-up was intended to determine whether it would develop into chronic kidney failure but the patient failed to comply with her consultations deadlines despite persistent efforts to contact her.

DISCUSSION

Pregnancy is a physiological state that places greater demand on both cardiovascular and respiratory systems to satisfy its increased metabolic demand. Several changes justify the vulnerability of pregnant women and their fetuses to mild hypoxia, such as increased cardiac output, decreased respiratory functional residual capacity, a rightward shift of the maternal oxyhemoglobin dissociation curve, and an increase in oxygen consumption.

One of the many clinical features that have been associated with mild and severe CO poisoning are myocardial damage and cardiovascular complications; they count for up to one-third of patients and may be associated with increased long-term mortality [3].

The pathophysiology is multifactorial and caused by reversible direct and indirect lesions due to carboxyhemoglobin (Figure 2). The main consequence is myocardial damage leading to ischemic injury. Other aspects include arrhythmias as observed in the first case, acute heart failure, vasoplegic response, takotsubo cardiomyopathy, and cardiovascular collapse or cardiorespiratory arrest [4-6].

Fetal Repercussions

During CO poisoning in pregnant women, CO dissolved in maternal plasma crosses the placenta by passive diffusion and thus combines with fetal hemoglobin.

In utero fetuses are more vulnerable to carbon monoxide toxicity because of the natural leftward shift of the dissociation curve of fetal hemoglobin that provides it with a higher affinity for CO than adult hemoglobin (10-15% more), a slower dissociation and elimination of CO. Hence, acute nonlethal maternal intoxication may cause severe acute fetal hypoxia and result in fetal demise, abnormal development or permanent neurologic sequelae [7].

Continuous electronic fetal heart monitoring is essential to promptly detect signs of acute fetal distress. First-line in-utero resuscitation should always be carried out before considering emergency delivery since the reestablishment of adequate maternal tissular perfusion and oxygenation is often sufficient to recover normal fetus heart rate.

Treatment

The main treatment of CO poisoning consists of the administration of 100% normobaric oxygen administered via a non-rebreathing face mask for a minimum of 6 hours despite normal oxygen saturation levels and partial arterial oxygen pressure, thus reducing the half-life of maternal carboxyhemoglobin to 40-70 min instead of 4-6 hours [8]. During pregnancy, since fetal clearance of CO is extended, oxygenation therapy should be initiated promptly in all cases and must be sustained at a rate of 15l/min for 16 hours or more [8].

Furthermore, non-invasive ventilation may be useful if associated with acute left ventricular failure; whereas hyperbaric oxygen therapy should be considered in cases of severe and moderate maternal exposure, it was observed to be harmless and beneficial in pregnant women by decreasing fetal hypoxia and improving outcomes [9].

In the presence of dysrhythmias, antiarrhythmic therapy should be wisely considered only when symptomatic or in presence of hemodynamic compromise. The first treatment choice is intravenous adenosine if vagal maneuvers are unsuccessful. If adenosine fails, then IV cardioselective beta-blockers such as propranolol or metoprolol are recommended. Intravenous administration of verapamil may be associated with a greater risk of maternal hypotension and subsequent fetal hypoperfusion. If indicated, electric cardioversion should be performed as it has been proved to be safe during all stages of pregnancy [10].

CONCLUSION

Given the large toxic manifestations of carbon monoxide poisoning on the cardiovascular system, a thorough and prompt cardiac examination is necessary to identify serious acute maternal and fetal complications. Long-term follow-up is essential to detect neuropsychological sequelae and unfavorable cardiovascular and renal outcomes. Although more efforts should be made to control defective water heating systems across the country.

Why should an emergency physician be aware of this?

- Carbon monoxide intoxication is very frequently seen in the emergency department and can cause many life-threatning complications
- Pregnant women are more susceptible to developing myocardial injuries when exposed to carbon monoxide poisoning.
- A thorough clinical examination along with biological markers and a bedside point of care ultrasound should always be performed in high-risk patients.

Financial Disclosures: None

Conflicts of Interest: None

Author contributions

- M.E: Contributed to patients' clinical care and wrote the manuscript
- N.I: Contributed to patients' clinical care and writing the manuscript
- S.E: Contributed to patients' clinical care and data collection
- H.R: Has drafted and revised the work
- A.E: Has made substantial contribution in the conception, design and supervision of the manuscript All authors read and approved the final manuscript

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