Case Report

Cardiogenic Shock Related to Carbon Monoxide Poisoning

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Keywords

Cardiogenic shock · Carbon monoxide poisoning

Abstract

Carbon monoxide (CO) poisoning is one of the leading causes of death by poisoning in occidental countries. We report the presentation and management of a patient who developed a severe cardiac dysfunction, leading to profound cardiogenic shock after CO poisoning despite an initial low CO blood level.

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Introduction

Carbon monoxide (CO) poisoning is one of the leading causes of death by poisoning in occidental countries [1]. It mostly presents with neurological impairment. Despite being less frequent, cardiovascular events can severely alter the patients' prognosis [2]. Here, we report the observation of a patient who developed cardiogenic shock after CO poisoning.

Case Presentation

A previously healthy 25-year-old woman, non-smoker, was taken in charge by the emergency medical services for an acute onset atraumatic coma. Her mother, who had started

suffering from a new-onset headache, found her unconscious in her bedroom. When last seen 12 h earlier, the patient had no complaints.

On site, the patient had normal temperature and glycaemia as well as a Glasgow Coma Scale of 3. She was in a calm, areflexic coma. Endotracheal intubation was performed for airway protection. After 1 h of mechanical ventilation on 100% oxygen, the first arterial carboxyhaemoglobin (COHb) level was 13%. The patient's respiratory rate was at 20 breaths per minute, oxygen saturation at 94% and pulse rate at 90 beats per minute (bpm). Upon arrival at the emergency department (ED), she was under infusions of midazolam for sedation and norepinephrine for haemodynamic instability.

In the ED, transthoracic echocardiography (TTE) revealed global hypokinesia with a severely depressed left ventricular ejection fraction (LVEF) at 10% and a cardiac index (CI) at 1.5 L/min/m². Brain CT scan was normal. Electrocardiogram showed regular sinus rhythm at 89 bpm with no suspicion of myocardial ischaemia. Initial arterial blood gas (ABG) on high flow oxygen showed lactic acidosis: pH: 7.25; PaCO₂: 4.21 kPa; PaO₂: 90 kPa; HCO₃⁻: 13.9 mmol/L; lactate: 4.9 mmol/L and COHb: 8.9%. The first blood tests showed: Hb: 11.6 g/dL; troponin Ic: 4.7 g/L (upper limit <0.09 g/L); β-HCG <0. In presence of neurologic manifestations, hyperbaric oxygen therapy (HBOT) for 90 min at 2.5 ATA was started. In view of the clinical presentation, the absence of cardiovascular risk factor and no segmentary hypokinesia on TTE, no coronarography was performed.

After ICU admission, a Swan-Ganz catheter showed a CI at 1.35 L/min/m², a SvO₂ at 41%, a CVP at 15 mm Hg and a PAOP at 25 mm Hg. Considering the haemodynamic instability, no other HBOT session was performed. Haemodynamic optimisation was obtained with dobutamine and norepinephrine infusions. The haemodynamic parameters improved with a CI at 3.35 L/min/m², a SvO₂ at 71%, a CVP at 9 mm Hg and a PAOP at 12 mm Hg. Her clinical course in ICU was rapidly favourable allowing the weaning of catecholamine support within 48 h. TTE performed at day 3 showed a normalised LVEF.

Neurologically, cognitive impairment persisted and brain MRI revealed bilateral pallidal lesions, compatible with CO poisoning. Follow-up at 3 months showed mild to moderate cognitive impairment with attention and executive disorders.

Discussion and Conclusion

Our patient presented a severe cardiogenic shock secondary to CO poisoning. Despite an initially low COHb level, she required inotropic and vasoactive agents. Nevertheless, they were rapidly weaned.

CO poisoning, also called a “silent killer,” is one of the most crucial health concerns worldwide, primarily because of its severe clinical effects and high toxic morbidity and mortality. CO is an odourless and colourless gas generated because of incomplete combustion of carbon-containing fuels. The mechanism of CO toxicity is tissue hypoxia, mainly because the binding affinity of CO to haemoglobin is 200–240 times that of oxygen, which reduces oxygen-carrying capacity and impairs the oxygen delivery.

CO cardiotoxicity is caused by both tissue hypoxia and direct effects on the myocardium: CO binds to the myoglobin with a 60-times greater affinity than oxygen, thus reducing the oxygen supply to the mitochondria, impairing the oxidative phosphorylation and deteriorating the energy source of the myocardium [3]. ATP production is interrupted, which results in anaerobic metabolism with lactate and free radicals production. Moreover, an

animal study showed increased Ca^{2+} sensitivity of myofilaments and increased diastolic intracellular Ca^{2+} . These abnormalities induce a hyperadrenergic state and a greater risk of developing arrhythmias. Furthermore, the compensatory tachyarrhythmia due to the systemic hypoxia in the early stages of CO poisoning increases the oxygen demand and accelerates CO diffusion, which exacerbates the hypoxic injury to the myocardium [4].

CO-related cardiovascular dysfunction includes angina, myocardial infarction, arrhythmia, left ventricular dysfunction, transient myocardial stunning, cardiogenic shock, and sudden death [5, 6]. In autopsy samples, the pathological features of CO intoxication are variegated and may include scattered and punctiform necrotic areas, subendocardial haemorrhages in the left ventricle, degenerative involvement of papillary and other muscles, as well as focal myocardial necrosis [7]. Other studies have demonstrated that myocardial injury independently predicts the short-term poor outcome in patients with severe CO poisoning and the long-term mortality in those with moderate to severe CO poisoning [8, 9].

HBOT is defined as the breathing of 100% oxygen by patients within hyperbaric chambers compressed to greater than 1.4 atmospheres (atm) of absolute pressure. The half-life of CO in room air is around 4–5 h. These half-life values decrease to approximately 40–80 min with administration of “100% oxygen” and to 23 min with hyperbaric (2 atm) oxygen. This explains why the COHb value was quite low in our patient’s case, after 1 h of mechanical ventilation on 100% oxygen. However, given that the half-life is only around 90 min with high-flow oxygen and that it typically takes at least 2 h to arrange HBOT treatment, the biological rationale that HBOT is more efficient in clearing CO is limited in practice [10].

Intravenous inotropes and vasopressors remain fundamental to the acute management of cardiogenic shock. These agents may increase ventricular contractility and cardiac output, reduce filling pressures, and preserve end-organ perfusion [11]. Limited data support the use of norepinephrine as the preferred first-line agent [12]. However, given their propensity to increase myocardial oxygen demand, these agents should be used in the lowest possible doses for the shortest duration. Until definitive therapy or resolution of the acute precipitating problem, patients with cardiogenic shock should receive temporary intravenous inotropic support to maintain systemic perfusion and preserve end-organ performance [13].

The present case report shows that even if the initial CO blood level measured is low, CO poisoning can be associated with severe cardiac dysfunction, leading to profound cardiogenic shock. Therefore, it appears essential for emergency physicians to identify features of myocardial injury in case of CO poisoning by performing electrocardiogram, cardiac markers and echocardiography in order to quickly implement adequate haemodynamic monitoring and therapeutic support. Cardiac dysfunction is sometimes severe, but often short. In case of failure of maximal medical therapy, a short-term cardiac assistance should be considered [11]. If haemodynamic condition permits, these patients should have access to a faster HBOT, so as to limit cardiac, and more importantly neurological, sequelae.

Statement of Ethics

The authors have no ethical conflicts to disclose. Written informed consent was obtained from the patient for publication of this case report and any accompanying images. There are no patient identifiers in the case report which may link the patient to the report.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

F.B. conceived and designed the paper, collected the data, wrote the paper and performed final edits. M.B. collected the data and assisted with writing the paper and performing final edits.

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