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CASE REPORT

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Non-ST elevation myocardial infarction secondary to carbon monoxide intoxication

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ABSTRACT

Carbon monoxide poisoning has been documented in literature to cause severe neurological and tissue toxicity within the body. However, cardiotoxicity is often overlooked, but not uncommon. Previous research studies and case reports have revealed a significant relationship between carbon monoxide intoxication and myocardial ischemic events. We report a case of a 48-year-old male, who was exposed to severe smoke inhalation due to a house fire and subsequently developed a non-ST elevation myocardial infarction. Ischemic changes were evident on electrocardiogram, which demonstrated T-wave inversion in lead III and ST-segment depression in leads V4-V6. Elevated cardiac enzymes were also present. After standard treatment for an acute cardiac event, the patient fully recovered. This case demonstrates that myocardial ischemic changes due to carbon monoxide poisoning may be reversible if recognized in early stages and treated appropriately, thus reminding physicians that a proper cardiovascular examination and diagnostic testing should be performed on all patients with carbon monoxide poisoning.

Abbreviations: NSTEMI: Non-ST elevation myocardial infarction

1. Introduction

Carbon monoxide (CO) intoxication causes impaired oxygen delivery and utilization at the cellular level, causing tissue hypoxia at different sites within the body, while having profound impact on high-oxygen demand organs, and causing changes at a neurological level. As a result of the high affinity of CO to hemoglobin, even small concentrations of CO can result in significant levels of carboxyhemoglobin (HbCO). As CO binds to cardiac myoglobin with an even greater affinity than to hemoglobin, myocardial depression and hypotension can further exacerbate the tissue hypoxia, leading to myocardial ischemia and damage [1,2]. The following case report demonstrates the significant clinical relationship between acute CO poisoning and myocardial ischemic damage presenting as a non-ST elevation myocardial infarction (NSTEMI) in a patient who was exposed to sever smoke inhalation in a house fire. Post-treatment, the cardiac abnormality fully resolved.

2. Case presentation

A 48-year-old male was found outside his apartment building in acute respiratory distress, leaning against a fence, after escaping from a fire that was taking place in a neighboring apartment. The patient suffered severe smoke inhalation and cuts on his legs from shattered glass but denied any chest pain or numbness of extremities. He was immediately taken by ambulance to a local hospital for first aid management and treatment. Patient has a history of hypertension, no coronary artery disease, and no history of smoking. On arrival (Day 1), patient was placed immediately on a non-rebreather mask for high-concentration oxygen delivery at 100%. Vital signs showed a heart rate (HR) of 98 beats/min, blood pressure (BP) of 129/78 mmHg, and a respiratory rate (RR) of 19 breaths/min. The initial venous blood gas (VBG) revealed COHb of 12.5%, pH 7.32, pCO2 44.7 mmHg, pO2 76.3 mmHg, SaO2 100%, and HCO3- 22 mmol/L. Basic biochemistry data showed glucose 110 mg/dL, BUN 14 mg/dL, Cr 1.2 mg/dL, Na 139 mmol/L, K 4.2 mmol/L, Cl 103 mEq/L, CO2 23 mEq/L, Ca 9.8 mmol/L, and Troponin I of 0.039 ng/mL with normal lipid profile. Electrocardiogram (ECG) revealed sinus rhythm with ST-wave depression in leads V4-V6 which was similar to EKG done by EMS and there was no previous EKG to compare. At the time, despite the patient being stable, conscious, and denying any chest pain, he was kept on continuous high-flow oxygen delivery, and monitored on the medical floor. Later in the evening, a second set of cardiac enzymes was drawn, revealing an elevated Troponin I of 3.06 ng/mL. EKG was repeated and showed T-wave inversion
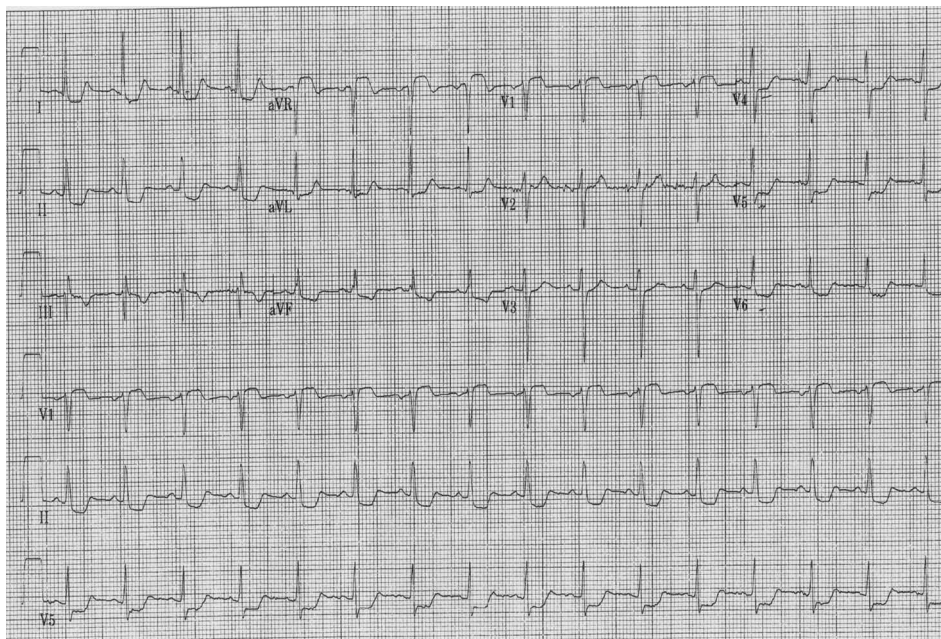
Figure 1. EKG showing T wave inversion in lead III, ST depression in I,II and V4-V6.

in lead III, mild left ventricular hypertrophy (LVH), and no ST-T wave segment changes as compared to earlier EKG. On physical exam patient was stable, alert, awake, oriented, afebrile, but complained of progressive left-sided stabbing type chest pain 9/10 that was non-radiating. He was loaded with oral aspirin 325 mg, Plavix 75 mg, lisinopril, atorvastatin 80 mg and enoxaparin 1 mg/kg. His initially measured serum COHb of 12.5% decreased dramatically within the first 24 hours to 0.8%.

The following day (Day 2), a third set of cardiac enzymes revealed a Troponin I of 1.68 ng/mL. Repeat ECG showed sinus rhythm, with T-wave inversion in lead III, and ST-wave depression in leads V4-V6 (Figure 1). Echocardiography showed a hypercontractile left ventricle, no structural or valvular abnormalities. Patient was taken to the coronary care unit for further care.

After stabilizing the patient (Day 3), a follow-up ABG showed a pO2 of 93.8 mmHg, and a downtrending of cardiac enzymes with a Troponin I of 0.416 ng/mL. Cardiac catheterization revealed normal coronaries, left ventricular ejection fraction (EF) of 59%, and a mildly elevated left ventricular end diastolic pressure (LVEDP). Because the electrocardiographic changes were attributed to CO intoxication, no thrombolytic or percutaneous coronary intervention was needed.

After continuous oxygen therapy and fluid resuscitation, the patient was transferred back to the medical floor on Day 7. A repeat ECG showed T-wave inversion in lead III, with a resolution of the ST-wave depression in leads V4-V6 that was noted on the previous EKG. The abnormal biochemistry and hemogram data returned to normal values. The patient was discharged on Day 8 with low dose aspirin and statins. On follow-up at the pulmonary clinic one month later, the patient's general condition was found to be good, without any cognitive dysfunction or neurological deficits. Since cardiac catheterization revealed normal coronaries, the cardiologist felt that no other interventions are needed and to follow up with his primary care physician.

3. Discussion

CO is an odorless, colorless, non-irritating gas, produced by incomplete combustion of carbonaceous material. It is a leading cause of poison-related deaths in the United States (3–5). Its high affinity for hemoglobin is 210 times that of oxygen, thus interfering with oxygen release and delivery to cells due to a shift in the oxygen-hemoglobin dissociation curve and possibly impairing electron transport, as a result of the reversible inhibition of mitochondrial respiration and oxidative stress which results in tissue inflammation and hypoxia [1,3,6]. A wide array of acute symptoms has been documented, ranging from mild viral-like symptoms to more severe respiratory depression, cerebral edema, and fatal arrhythmias [7].

As CO binds to cardiac myoglobin with an even greater affinity than to hemoglobin, myocardial depression and hypotension can further exacerbate the tissue hypoxia, leading to myocardial ischemia and damage [8]. As a result, moderate to severe CO intoxication can result in myocardial injury and present an increased risk of mortality [9]. Therefore, a decrease in oxygen transport capacity

of the blood leads to a decreased amount of oxygen delivery to the tissues, resulting in myocardial ischemia and damage, even in patients with normal coronary arteries [10,11], which was the case with our patient who had no cardiac risk factors with a normal coronary angiogram. Even though the main mechanism by which CO causes tissue damage is via production of hypoxia, effects of CO are more profound in the myocardium than in peripheral tissues because of very high oxygen extraction by the myocardium at rest [12]. CO may also have direct myocardial effects. Chen demonstrated that in isolated rat hearts, CO caused a greater decrease in heart rate and pulse pressure compared to the same degree of anoxia produced by the inhalation of nitrogen [13]. The role of CO in platelet aggregation is controversial, but some studies have indicated increased platelet activation and aggregation [14] in animals.

With regards to treatment of CO poisoning with hyperbaric oxygen therapy (HBOT), it has been found to be beneficial in reducing the risk of cognitive deficits [15]. Hyperbaric oxygen (HBO2) hastens carboxyhemoglobin (COHb) elimination and favorably modulates inflammatory processes instigated by CO poisoning, an effect not observed with breathing normobaric oxygen. Hyperbaric oxygen improves mitochondrial function, inhibits lipid peroxidation transiently, impairs leukocyte adhesion to injured microvasculature, and reduces brain inflammation caused by the CO-induced adduct formation of myelin basic protein [16]. Animal studies demonstrate HBOT to reduce myocardial infarct size in ischemic rabbit hearts when hyperbaric oxygen is administered at reperfusion [5]. Other investigators have shown no beneficial effect for HBOT on infarct size in dogs [17]. However, the effects of HBOT in human myocardial ischemia are still poorly understood and needs further investigation. Based on limited data, the indications for HBOT include moderate to severe CO poisoning [18,19]. However, in our case report, the patient's presenting symptoms of respiratory distress were significantly improved with high-concentration oxygen under normal pressure. His initially measured serum COHb of 12.5% decreased dramatically within the first 24 hours to 0.8%. Therefore, HBOT was not administered to the patient, and his symptoms and cardiac function fully recovered. Though with his clinical presentation and EKG changes as described above with high troponins he was given aspirin, clopidogrel, statins, lisinopril and enoxaparin.

In conclusion, considering the potential mediating role of CO intoxication in tissue hypoxia, it is of extreme importance to examine the influence of CO poisoning in myocardial ischemia and injury. Research studies by Henry & Satran, et al. (2006) have investigated and linked the role and degree of CO intoxication as being both a predictor of myocardial injury and long-term mortality and as an impediment to successful recovery from tissue damage imposed by the body's hypoxic state. This case further supports and demonstrates the significant relationship between CO poisoning and myocardial ischemic injury mimicking a NSTEMI, which may be fully reversible with a timely diagnosis and proper treatment. It is therefore, fundamental to do a full cardiac work-up on patients exposed to CO poisoning to prevent permanent myocardial injury, and decrease the risk of long-term mortality.

Disclosure statement

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