Non-ST elevation myocardial infarction secondary to carbon monoxide intoxication

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\textbf{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.

\textbf{Keywords:} 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 \cite{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 severe 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....
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 mea-
sured 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 hypercon-
tractile 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 attribu-
ted to CO intoxication, no thrombolytic or percuta-
aneous 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 bio-
chemistry 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 cor-
onaries, the cardiologist felt that no other interven-
tions are needed and to follow up with his primary
care physician.

3. Discussion
CO is an odorless, colorless, non-irritating gas, pro-
duced 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 tis-
sue 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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