Acute Carbon Monoxide Poisoning Resulting in ST Elevation Myocardial Infarction: A Rare Case Report

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Acute carbon monoxide (CO) poisoning with cardiac complications is well documented in the literature. However, ST segment elevation is a rare presentation, and most of these cases with ST elevation have revealed non-occlusive or normal coronary arteries. We report a case of CO poisoning complicated with ST elevation myocardial infarction. Emergency coronary angiography revealed total occlusion of the left anterior descending artery and primary percutaneous coronary intervention was performed. This report of a rare case should remind physicians that cardiovascular investigations, including electrocardiography, must be performed in cases with CO poisoning because mortality might increase if reperfusion therapy or appropriate medical treatments are not performed in patients with acute coronary artery occlusion.

Key Words: acute myocardial infarction, carbon monoxide, carboxyhemoglobin, ST elevation
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Carbon monoxide (CO) is known as a silent killer because it is an odorless, colorless and nonirritating gas. The clinical findings of CO poisoning are highly variable, but neurologic and cardiovascular complications are common [1]. Myocardial infarction related to CO poisoning has been frequently reported in the literature; however, ST elevation myocardial infarction due to coronary occlusion is a very rare presentation. Here, we report a rare case of acute CO poisoning complicated with acute anterior ST elevation myocardial infarction.

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Case Presentation

A 56-year-old female was found collapsed and prone by her family and she had an unclear conscious level. She was immediately sent to our emergency department for first aid. She had a history of hypertension, diabetes and dyslipidemia, but did not smoke. Suicide was suspected because burnt charcoal and some benzodiazepine medications were found at the scene. On arrival, a non-rebreathing mask was used for high-flow oxygen supply. Vital signs showed a heart rate of 98 beats/min, a respiratory rate of 12 breaths/min and her blood pressure was 148/90 mmHg. Initial laboratory data revealed carboxyhemoglobin (COHb) of 19.3%, creatine kinase-MB of 19.9 IU/L, troponin I of 0.97 ng/mL and urine benzodiazepine level of 550.69 ng/mL. Electrocardiography (ECG) showed V1–V4 ST elevation (Figure 1). Although the patient did not complain of chest pain because of impaired consciousness, she

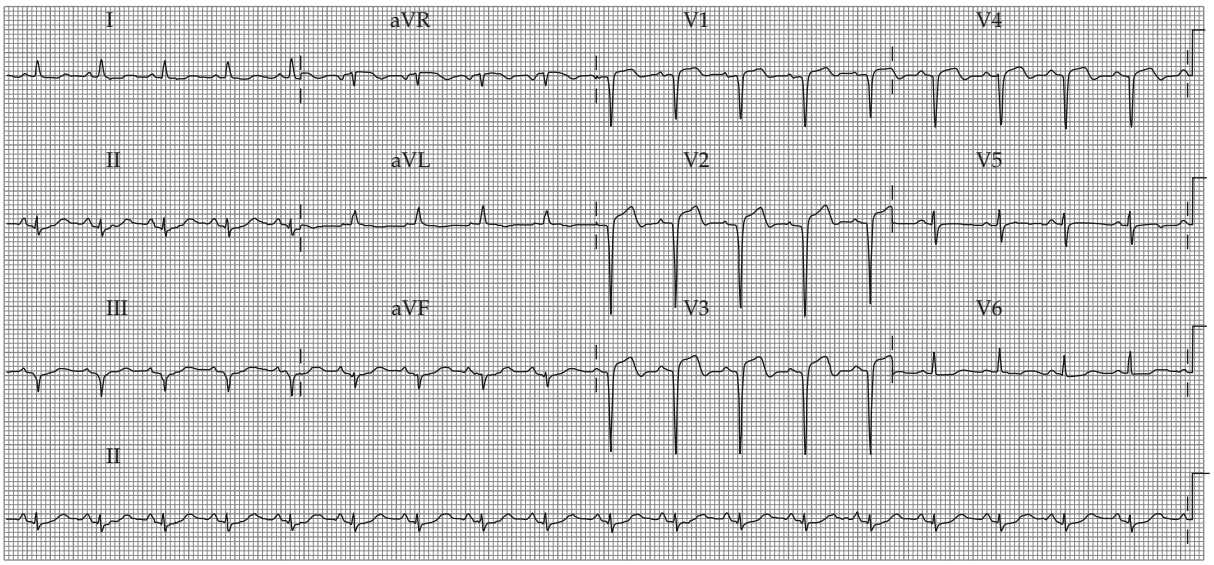

Figure 1. *Electrocardiogram revealed sinus tachycardia and ST elevation over leads V1–V4 with a pathologic Q wave.*

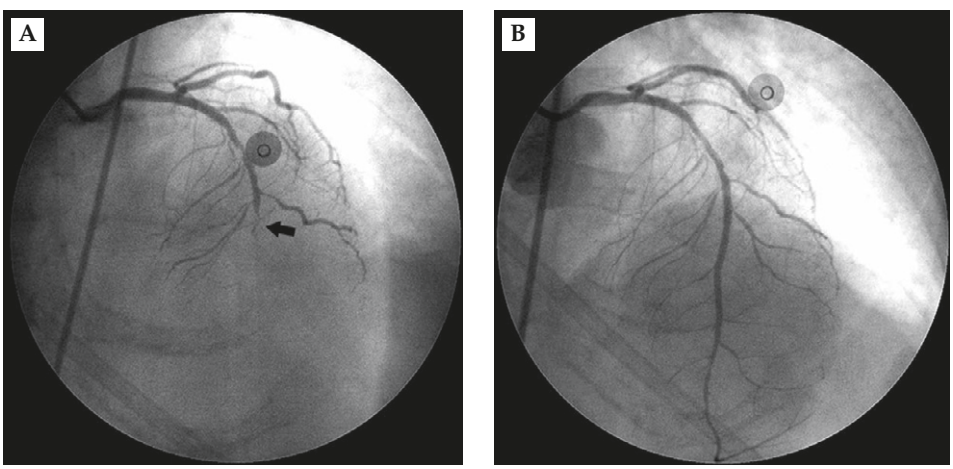

Figure 2. *(A) Left coronary angiography before intervention showing total occlusion over the middle left anterior descending artery (black arrow). (B) Left coronary angiography after intervention showing restored thrombolysis in myocardial infarction-3 flow after angioplasty with stenting.*

was sent for primary percutaneous coronary intervention. Emergency coronary angiography revealed total occlusion of the middle left anterior descending artery (LAD) (Figure 2A), with patent right coronary and left circumflex arteries. Coronary angioplasty with stenting was subsequently performed over the LAD (Figure 2B). After the primary percutaneous coronary intervention, she was transferred to a cardiac care unit for intensive care.

At our cardiac care unit, her consciousness level improved under medication and high-flow oxygen. Further laboratory data revealed her COHb to be 0.9%. Cardiac enzymes also demonstrated an early peak (creatine kinase-MB, 142.1 IU/L; troponin I, 131.4 ng/mL). Follow-up ECG also revealed resolution of the ST elevation. After a 4-day cardiac care unit stay, she was transferred to an ordinary ward and later discharged in a stable condition.

Discussion

CO is one of the leading causes of poisoning-related deaths in the United States. It binds to hemoglobin with greater affinity than oxygen and forms COHb, which leads to impaired oxygen transportation and subsequent tissue hypoxia [1]. In addition, an increased thrombogenic tendency of CO poisoning

has been reported, although this is a cause of some controversy [2].

The half-life of CO while a patient is breathing room air is nearly 300 minutes, but decreases to approximately 90 minutes while breathing high-flow oxygen via a non-rebreathing mask, and to 30 minutes during hyperbaric oxygen treatment. Although some randomized trials have revealed a potential benefit of hyperbaric oxygen [3], a Cochrane review did not favor its use for acute CO poisoning [4]. Therefore, the role of hyperbaric oxygen in the treatment of CO poisoning remains controversial. In addition, it is unclear which group of CO-poisoned patients should receive this therapy, and there are some uncertainties about the optimal chamber pressure, the number of hyperbaric oxygen sessions and the maximal interval between the sessions. Therefore, the Undersea and Hyperbaric Medical Society recommends hyperbaric oxygen therapy for patients with serious CO poisoning. Indeed, a COHb level of ≥25% is considered an indication for the use of hyperbaric oxygen.

Myocardial injury is not uncommon in cases with CO poisoning, and is associated with increased long-term mortality. Satran et al reported two clinical patterns of myocardial injury [5]. One group of patients was younger with few cardiac risk factors but had severe CO poisoning. They were more likely to have left ventricular global hypokinesis as a result of CO poisoning, which improved steadily with treatment. The other group of patients was older with regional wall motion abnormalities and a higher frequency of cardiac risk factors. CO poisoning might reveal underlying coronary artery disease by disturbing the balance between oxygen supply and demand.

Although cardiac manifestations are not rare in cases with CO poisoning patients, ECG findings of myocardial ischemic change often reveal T-wave inversion or ST depression in these patients. In the cases without ECG signs of ST elevation, some patients had coronary atherosclerotic lesions and some had normal coronary arteries [6–9]. However, ST segment elevation is a rare presentation in CO poisoning. Some case reports have described similar finding [10–13], but most of the cases had normal coronary arteries while undergoing coronary angiography [10,11]. Another case showed complete recovery of ST segment elevation after continuous oxygen treatment, so a normal coronary arteries in the case was favored by the author without coronary angiography evidence [12]. In one case with right coronary artery stenting history, thrombolytic therapy was given for inferior ST elevation, and coronary angiography follow-up 1 week later showed 30% in-stent restenosis and 70% stenosis over LAD [13].

The present case, a 56-year-old female with CO poisoning, was found to have precordial leads V1–V4 ST elevation on ECG. Emergency coronary angiography revealed that the LAD was the infarct-related artery and angioplasty with stenting was performed. Even if the initial COHb level is not very high, acute CO poisoning can still lead to severe cardiovascular complications in case with high cardiovascular risk. Our case demonstrates that ST elevation myocardial infarction with coronary occlusion is a possible complication of acute CO poisoning.

This report of a rare case should remind physicians that cardiovascular investigations, including ECG, should be performed in patients with acute CO poisoning. Cardiac enzymes should also be checked if ECG abnormalities are noted. Emergency coronary angiography should also be performed according to the American College of Cardiology/American Heart Association guidelines for ST elevation myocardial infarction in patients with high cardiovascular risks or previous history of coronary artery disease being treated with high-flow oxygen via a non-rebreathing mask or 100% oxygen by mechanical ventilator support.

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急性一氧化碳中毒造成 ST 段上升之心肌梗塞 — 罕見的病歷報告

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急性一氧化碳中毒造成心血管之併發症是有良好的文獻記載的。然而，心電圖上呈現 ST 段上升則是相當罕見的現象，且在這些有 ST 段上升之罕見案例中，大部分都是表現非阻塞性或是正常的冠狀動脈血管。我們在此報告一位一氧化碳中毒之病患併發急性 ST 段上升之心肌梗塞。緊急的冠狀動脈攝影顯示左前降動脈完全阻塞，因此進一步施行了緊急心導管介入性治療。這個罕見的案例報告告訴我們，在一氧化碳中毒的病患，醫師不應該忽略心血管方面的評估，因為在這些合併有急性冠狀動脈阻塞的病人身上，若我們沒有做即時的再灌流或是給予理想的藥物治療，死亡率有可能會增加。

關鍵詞：一氧化碳，碳氧血紅蛋白，ST 段上升，急性心肌梗塞 .

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