Clinical Case Report

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Variant angina induced by carbon monoxide poisoning

A CARE compliant case report

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Abstract

Rationale: Carbon monoxide (CO) poisoning can cause severe damage to the nervous system, and can also cause serious damage to organs, such as the heart, kidneys, and lungs. CO damage to myocardial cells has been previously reported. This can lead to serious complications, such as myocardial infarction.

Patient concerns: A 47-year-old female patient complained of sudden chest pain for 30 minutes. Before admission, the patient had non-radiating burning chest pain after inhalation of soot.

Diagnosis: An electrocardiogram showed that myocardial ischemia was progressively aggravated, manifested by progressive ST-segment elevation, and accompanied by T wave inversion and other changes. No obvious coronary stenosis was observed in a coronary angiographic examination. Therefore, the patient was considered to have developed variant angina resulting from CO poisoning-induced coronary artery spasm.

Interventions: The patient was treated with drugs for improving blood circulation and preventing thrombosis, and underwent hyperbaric oxygen therapy.

Outcomes: Clinical symptoms relieved after the treatment.

Lessons: Findings from this case suggest that CO can cause coronary artery spasm and it is one of the predisposing factors of variant angina. For these patients, hyperbaric oxygen therapy can improve blood circulation and prevent formation of thrombus and encephalopathy.

Abbreviations: BNP = brain natriuretic peptide, CK-MB = creatine kinase-MB, CO = carbon monoxide, COHb = CO hemoglobin, Hb = hemoglobin, MYO = myoglobin, TIMI = myocardial infarction, TNT = Troponin T.

Keywords: carbon monoxide poisoning, myocardial infarction, variant angina

1. Introduction

At present, the pathophysiological mechanism underlying coronary artery spasm in patients with variant angina is not fully understood. This mechanism may be related to endothelial dysfunction and hypersensitivity of vascular smooth muscle. Common causes of variant angina include smoking, use of drugs (e.g., cocaine and amphetamines), emotional stress, excessive mental stress, and cold stimulation. The clinical manifestations of carbon monoxide (CO) poisoning are varied, and CO poisoning can not only cause neurological symptoms, but also cause the incidence of serious cardiovascular complications, such as ST-segment elevation myocardial infarction (STEMI).[1,2] In our case, we found that CO poisoning induced variant angina. The mechanism for this finding may be that CO enters the alveoli and then dissolves in the blood. As the blood circulates throughout the body, CO binds to hemoglobin (Hb) in red blood cells to become carboxyhemoglobin, and then binds to myoglobin to become oxymyoglobin.[3,4] CO can also simultaneously bind to mitochondrial cytochrome oxidase and impede the production of cellular energy, resulting in damage of multiple organs. CO can also promote the expression of inducible nitric oxide synthase, which can mediate NO-induced myocardial damage during ischemia-reperfusion.[5] There have been few case reports on CO poisoning-induced variant angina. This study was approved by the Ethics Committee of First Hospital of Jilin University (approval number 2018–366) and informed consent was obtained from the patient.

2. Case report

A 47-year-old female patient was admitted because of sudden chest pain for 30 minutes. Before admission, the patient experienced burning chest pain after inhalation of soot. The burning chest pain occurred in the upper middle sternum and was not radiating. This symptom was accompanied by dizziness, chest

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tightness, sweating, nausea, and vomiting. The vomit was stomach content and the pain persisted without remission. The patient had a 10-year history of hypertension (blood pressure up to 190/100 mmHg), which was usually controlled at 140–150/80–90 mmHg via oral medication. She had no history of smoking.

At the time of admission, the patient had a clear mind, her blood pressure was 180/125 mmHg, and there were no other abnormalities in other physical examinations. Routine blood test results were normal. Blood gas analysis showed 6.9% CO hemoglobin (COHb). A fibrinolytic assay showed a normal D-dimer level. A cardiac enzyme test showed that the Troponin T (TNT) was 0.011 ng/mL, myoglobin (MYO) level was 90.72 ng/mL, creatine kinase-MB (CK-MB) level was 1.88 ng/mL, and brain natriuretic peptide (BNP) level was 70.45 pg/mL. After admission, an electrocardiogram (Fig. 1) showed ST-segment elevation in leads II, III, and avF. After 15 minutes, ST-segment elevation in leads II, III, and avF was more prominent than before the examination. Additionally, T-wave inversion was observed, and this suggested acute inferior myocardial infarction. Immediate coronary angiography showed no significant left main coronary artery stenosis. Atherosclerotic plaques were visible in the proximal and mid segments of the left anterior descending artery and forward flow was thrombolysis in myocardial infarction (TIMI) grade 3 (Fig. 2). No abnormalities were observed in the left circumflex coronary artery and the forward flow was TIMI grade 3. The proximal and mid segments of the

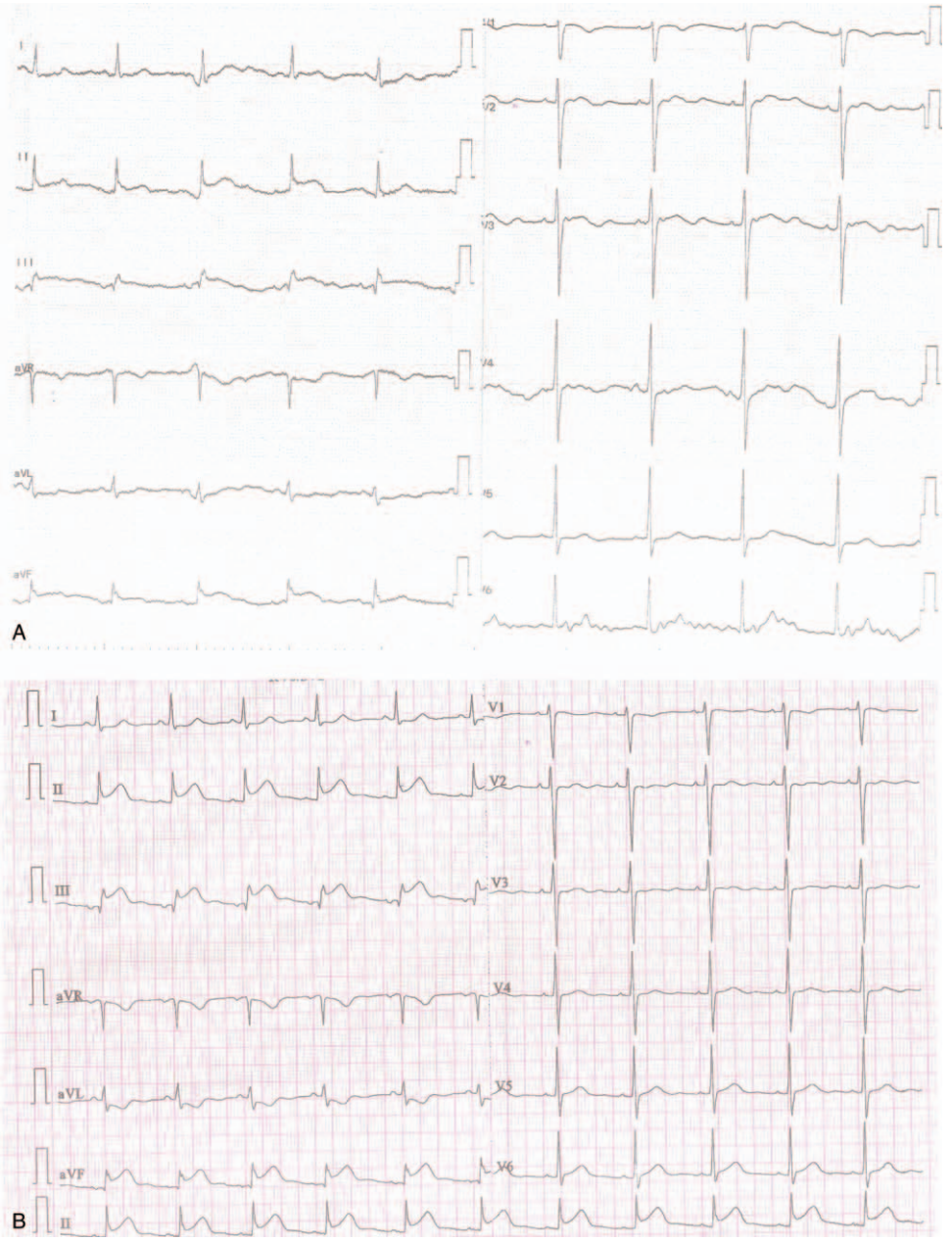

Figure 1. (A) An electrocardiogram shows ST-segment elevation in leads II, III, and avF immediately after admission. (B) An electrocardiogram shows aggravated ST segment elevation, accompanied by T wave inversion 15 minutes after admission.

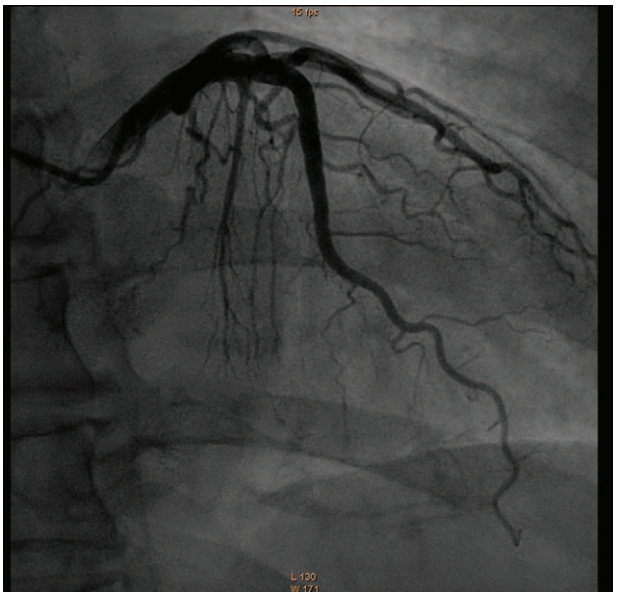

Figure 2. Coronary angiography showing atherosclerotic plaques at the proximal and mid segments of the left anterior descending artery.

right coronary artery had 20% tubular stenosis and its forward flow was TIMI grade 3. No abnormalities were observed in the collateral circulation. The right coronary artery was dominant. No abnormalities were observed on electrocardiograms at 2 and 7 hours after admission. At 7 hours after admission, a cardiac enzyme test showed that the TNT level was 0.768 ng/mL, MYO had returned to a normal level, and the CK-MB level was 22.14 ng/mL. Eleven hours later, the TNT level was 0.552 ng/mL, MYO had decreased to a normal level, and the CK-MB level was 15.32 ng/mL. After treatment with drugs for improving blood circulation and preventing thrombosis, the patient did not have chest tightness or other discomfort. On the next day, she was discharged and underwent hyperbaric oxygen therapy in the outpatient department. The pressure for hyperbaric oxygen therapy was 0.22 MPa, after a 25-minute of compression period, pure oxygen was inhaled through the oxygen mask for 40 minutes with 5-minute intervals in room air, followed by a 25-minute of decompression period. Hyperbaric oxygen therapy was conducted for 90 minutes once daily for 10 days. Hyperbaric oxygen therapy achieved better curative effect, the patient had no chest pain and dizziness after treatment.

3. Discussion

There is evidence to suggest that COHb levels >3% in nonsmokers or >10% in smokers indicate exposure to CO poisoning.[6] In this study, our patient had no history of smoking. After admission, her COHb level was 6.9%, which indicated CO poisoning. Variant angina is also known is a special type of ischemic heart disease characterized by spontaneous episodes of chest pain accompanied by transitory ST-segment elevations during the episodes, clear diagnosis criteria of variant angina includes clinical, electrocardiographic, and angiographic data.[7,8] In the study, the patient had the typical symptoms chest pain, electrocardiogram showed progressive ST-segment elevation, and an increase in cardiac enzyme was also observed, but no abnormalities were found with coronary angiography, which consistent with diagnosis of variant angina. Piantadosi[9] first proposed that the toxic effect of CO poisoning results from formation of carboxyhemoglobin. Carboxyhemoglobin can cause severe damage to organs, such as the heart and brain. Direct toxic effects of CO on mitochondria might produce a "stunned myocardium-like syndrome" with completely normal coronary angiograms and focal hypokinesia.[10] In autopsy samples, the pathological features of CO intoxication are varied and may include punctiform and scattered necrotic areas, subendocardial hemorrhage in the left ventricle, degenerative involvement of papillary muscle and other muscles, and focal myocardial necrosis.[11] CO poisoning can cause non-ST-segment elevation myocardial infarction, accompanied by an increase in cardiac enzymes, depression of the ST segment, and T-wave changes. However, these electrocardiographic indices return to normal levels 7 days later without thrombolysis and coronary stent implantation.[12] Gonullu et al[13] reported a 44-year-old healthy man who had ST segment elevation in leads II, III, and avf due to CO poisoning, but he did not undergo an angiographic examination. These abnormalities on the electrocardiogram gradually returned to normal after drug treatment. Sward et al[14] reported an 80-year-old patient who had a history of diabetes, hypertension, and congestive heart failure. CO poisoning aggravated the patient's heart injury and resulted in myocardial infarction. Sward et al[14] also reported CO poisoning in a 62-year-old female patient who developed severe chest pain after escaping from her environment. Myocardial infarction was diagnosed by coronary angiography. Systemic treatment with coronary stents improved her condition. The 62-year-old female patient was admitted to hospital because of chest tightness. Myocardial damage was considered because of T wave inversion, progressive ST-segment elevation, and an increase in cardiac enzymes. In contrast to the above-mentioned case, coronary angiography was performed immediately after admission in our patient. Coronary angiography showed no severe vascular stenosis. Based on our patient's CO exposure history and COHb levels, CO poisoning was considered to have induced variant angina.

CO poisoning complicated by severe myocardial damage is often found in clinical practice. Findings from the current case report suggest that CO poisoning can induce coronary artery spasm, resulting in variant angina. Without a CO environment, clinical symptoms are soon relieved after oxygen or hyperbaric oxygen therapy. For those patients with CO poisoning whose heart symptoms are the main manifestation, the nature of CO poisoning should be considered. Hyperbaric oxygen therapy is the primary therapy for the treatment of patient with CO poisoning (especially damage to the central nervous system and heart).[15] Hyperbaric oxygen therapy can improve the oxygen uptake and the availability of oxygen in the body, and then increase the blood oxygen levels, and the partial pressure of oxygen in blood, enhance oxygen diffusing capacity, thereby improving body's hypoxic condition.[16,17] Oxygen therapy and hyperbaric oxygen therapy should be used to improve blood circulation and prevent formation of thrombus to achieve a better curative effect and avoid encephalopathy caused by CO poisoning.

Author contributions

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