Clinical Case Report

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Non-ST elevation myocardial infarction induced by carbon monoxide poisoning

A case report

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Abstract

Rationale: Acute myocardial infarction is a rare complication of carbon monoxide poisoning. there is often no chest pain and other typical manifestations. We report a patient with mild carbon monoxide poisoning who had acute dyspnea as the earliest symptom and was later diagnosed with non-ST elevation myocardial infarction (NSTEMI) and acute left heart failure.

Patient concerns: A 73-year-old woman complained of dizziness and fatigue with shortness of breath after carbon monoxide intoxication.

Diagnoses: This patient had a clear history of carbon monoxide poisoning, acute respiratory distress, bilateral lung dry and moist rale, chest X-ray showed bilateral pulmonary edema, Electrocardiograph indicated general depression of the ST segment of the leads in the chest, cardiac troponin I (CTNI) increased progressively, cardiac ultrasonography indicated abnormal ventricular wall movement, coronary angiography suggested left main trunk and 3-vessel lesions, suggesting diagnosis acute carbon monoxide poisoning, acute coronary syndrome, acute left heart failure.

Interventions: She was treated with a high concentration of oxygen, an inhibitor of platelet aggregation (aspirin plus clopidogrel), an anticoagulant (low molecular weight heparin), an antimicrobial (ceftizoxime), an expectorant (mucosolvan), diuresis (furosemide and spironolactone), and myocardial support (Metoprolol). Coronary angiography and stent placement were performed 8 days later.

Outcome: On the 10th day after onset of the condition, echocardiography was performed, which showed that cardiac function was improved. Mild segmental wall motion abnormality was observed on echocardiography. After 14 days, the patient had recovered well and was discharged without chest tightness, chest pain, dizziness, headache, or unresponsiveness.

Lessons: This case suggests that the symptoms of carbon monoxide poisoning are complex and diverse. It can be manifested as a primary hypoxic symptom, or cause the exacerbation of underlying diseases due to hypoxia. Therefore, patients with carbon monoxide poisoning should actively seek comprehensive cardiac examination to ensure early diagnosis. Whenever necessary, coronary angiography and stent implantation should be performed to improve the likelihood of the patient's survival.

Abbreviations: ACOP = acute carbon monoxide poisoning, CKMB = creatine kinase MB, CTNI = cardiac troponin I, NSTEMI = non-ST elevation myocardial infarction, TIMI = thrombolysis in myocardial infarction.

Keywords: carbon monoxide poisoning, non-ST elevation myocardial infarction

1. Introduction

The mechanism of carbon monoxide poisoning is that the affinity of carbon monoxide and hemoglobin is 250 to 300 times higher than that of oxygen and hemoglobin, and carboxyhemoglobin is very stable and not easily dissociated, resulting in hypoxia of the whole body.[1] The symptoms of mild carbon monoxide poisoning are slight and can be quickly relieved. Moderate to severe carbon monoxide poisoning can cause damage to multiple organs including the brain, heart, lung, liver, kidney, muscle, and other organs.[1,2] The central nervous system and the cardiovascular system require high levels of oxygen and are very sensitive to hypoxia. Neurological damage is well known, and myocardial damage is also very common, occurring in more than one-third of patients with moderate to severe carbon monoxide poisoning.[2,3] There have been many reports that carbon monoxide poisoning can be accompanied by myocardial infarction,[4–6] which is common in patients with moderate to severe carbon monoxide poisoning. In this case study, we report a patient with mild carbon monoxide poisoning-induced severe non-ST elevation myocardial infarction (NSTEMI). Acute left heart failure was the earliest symptom, suggesting that carbon monoxide poisoning can be either an etiology or an inducement, resulting in the exacerbation of a patient's underlying diseases.

2. Case presentation

The patient was a female, 73 years old, admitted to hospital owing to dizziness and fatigue with shortness of breath for 3

Editor: N/A.

The authors report no conflicts of interest.

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Medicine (2019) 98:15(e15151)

Received: 6 November 2018 / Received in final form: 21 February 2019 / Accepted: 14 March 2019

http://dx.doi.org/10.1097/MD.0000000000015151

hours. Three hours before admission, the patient began to show symptoms including dizziness, fatigue, shortness of breath, vomiting stomach contents once, acid reflux, and heartburn. The patient was exposed to indoor stove heating during the onset, with poor ventilation. The other occupants of the same room showed identical symptoms and attended the emergency department of the hospital. This patient had a history of hypertension for 12 years with systolic blood pressure up to 170 mmHg and no specified diastolic pressure record. The patient routinely takes Norvasc to control her blood pressure when it is 130/80 mmHg. The patient has had diabetes for 14 years. Metformin is regularly used for treatment. Before-meal blood glucose was controlled at 7 mmol/L, and postprandial blood glucose was not monitored. Physical examination: blood pressure 120/80 mmHg, conscious, precise speech, shortness of breath, respiratory frequency at 30 beats per minute, pupils equal and equally reactive to light, rough breath sounds in bilateral lungs, audible wet and dry rales, heart rate 110 beats/minute, arrhythmia, class 4 limb muscle strength, normal muscle tone. Emergent examination showed carboxyhemoglobin 17%, electrocardiogram with the wild range ST-segment depression (Fig. 1), emergent cardiac troponin I (CTNI) 4.75 ng/mL, creatine kinase MB (CKMB) 1494 ng/mL, monitored oxygen saturation 90%. After giving oxygen via oxygen mask (oxygen flow 10L/min), emergent blood analysis showed: lactic acid 8.7 mmol/L, pH 7.196, partial pressure of carbon dioxide 34.6 mmHg, partial pressure of oxygen 82.4 mmHg, oxygen saturation 93.5%, actual bicarbonate 13.5 mmol/L, blood glucose 22.89 mmol/L, urine ketone bodies (–). Chest radiography suggested pulmonary edema (Fig. 2). According to the above characteristics, the patient was diagnosed with acute carbon monoxide poisoning (ACOP), acute coronary syndrome, acute left heart failure, lactic acidosis, high-risk grade 3 hypertension, and type II diabetes. The patient was admitted as an inpatient for further treatment.

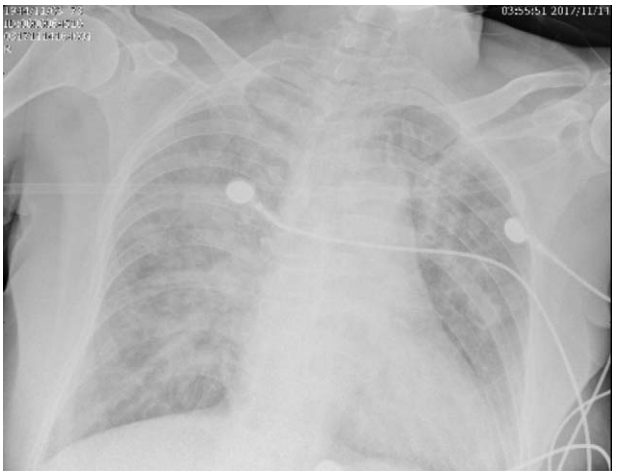

Figure 2. The pulmonary edema shown on chest radiography.

After admission, the re-examination results showed that myocardial enzymes CKMB and CTNI were becoming progressively elevated. Twenty-four hours after the onset, CTNI had increased to 69.42 ng/mL, and brain natriuretic peptide had reached 1113 pg/mL. The echocardiography results indicated segmental wall motion abnormalities (reduced amplitude of motion of lower left ventricular wall, and posterior wall basal and apical segments), reduced left ventricular function, and ejection fraction 50%. The patient was diagnosed with NSTEMI and acute left heart failure. She was treated with a high concentration

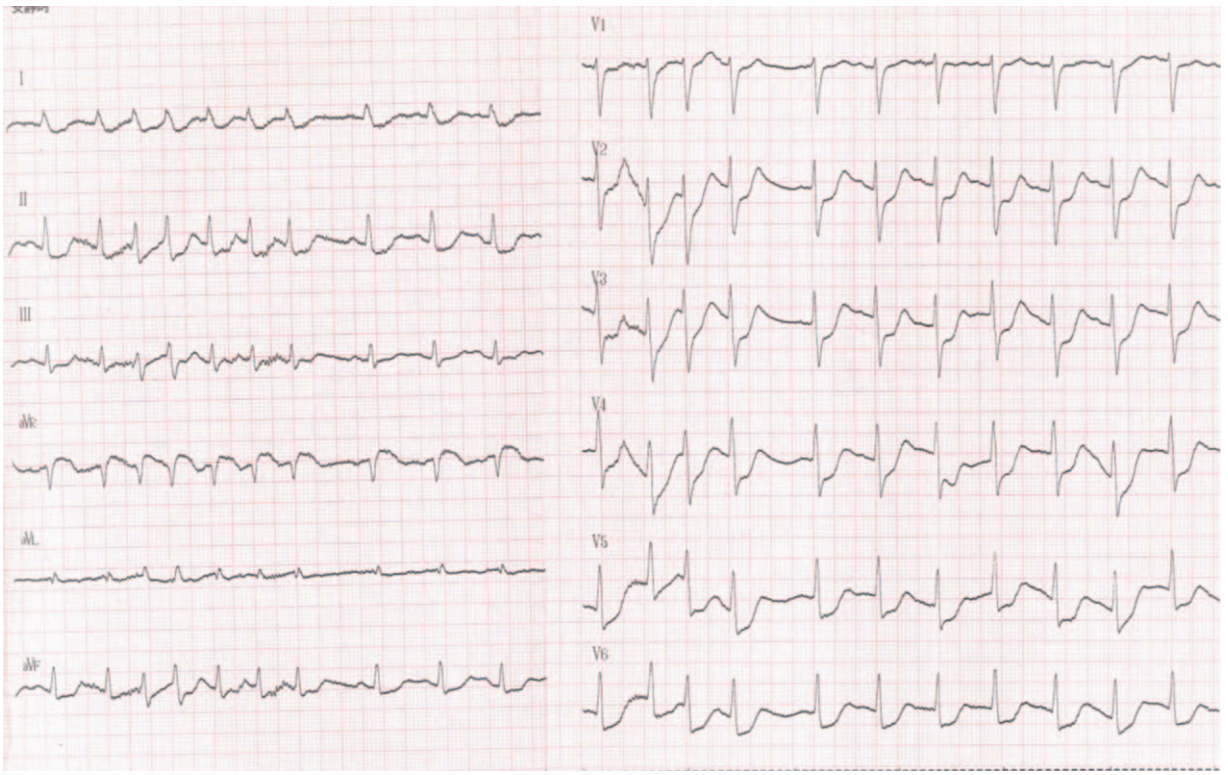

Figure 1. The wild range ST segment depression shown by echocardiography.

of oxygen, an inhibitor of platelet aggregation (aspirin plus clopidogrel), an anticoagulant (low molecular weight heparin), an antimicrobial (ceftizoxime), an expectorant (mucosolvan), diuresis (furosemide and spironolactone), and myocardial support (Metoprolol). On the second day after the onset, the wheezing symptoms began to be relieved, and troponin I decreased to 39 ng/mL with a continuous decrease afterward. After 8 days, the CTNI had decreased to 0.2 ng/mL, and symptoms of wheezing, dizziness, and fatigue were relieved. The patient was transferred to the department of cardiology to undergo coronary arteriography. The result indicated that the coronary blood supply was right dominant. The right canal opening had developed normally, as had the left coronary opening. The left main coronary artery showed 50% localized stenosis, and the thrombolysis in myocardial infarction (TIMI) blood flow was grade III. In the near-middle segment of the left anterior descending artery, 80% diffuse stenosis was observed, and the TIMI flow was grade III. A 90% stenosis was seen in the opening-near segment of the diagonal branch 1, with grade III flow in the TIMI. The obtuse marginal branch I showed 90% localized stenosis with a TIMI grade III (Fig. 4). An 80% localized stenosis was observed at the right main coronary artery opening, and 70% diffuse stenosis was observed in the proximal right coronary artery. A 70% to 95% diffuse stenosis was seen in the right and middle right coronary arteries, with a grade III TIMI (Fig. 3). The conclusion from angiography was: Coronary heart disease in the left main artery + 3 branch lesions (afflicting the left anterior descending artery + left circumflex + right coronary artery).

During the surgical operation, the right coronary artery was treated and 3 stents were implanted (Fig. 5). The rest of the blood vessels were not treated temporarily. On the 10th day after onset of the condition, echocardiography was performed, which showed that cardiac function was improved. Mild segmental wall motion abnormality was observed on echocardiography. After 14 days, the patient had recovered well and was discharged without chest tightness, chest pain, dizziness, headache, or unresponsiveness.

Written informed consent was obtained for publication of this case report from the patient.

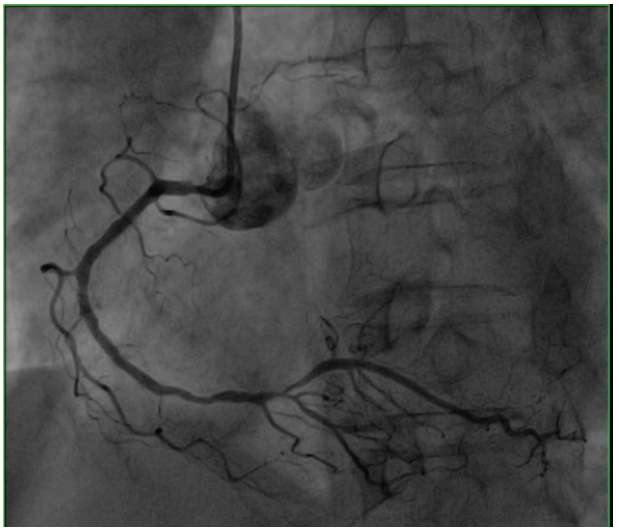

Figure 3. An 80% localized stenosis was observed in the opening of the right main coronary artery. A 70% diffuse stenosis was observed in the proximal right coronary artery, and 70% to 95% diffuse stenosis was observed in the middle and distal right coronary arteries.

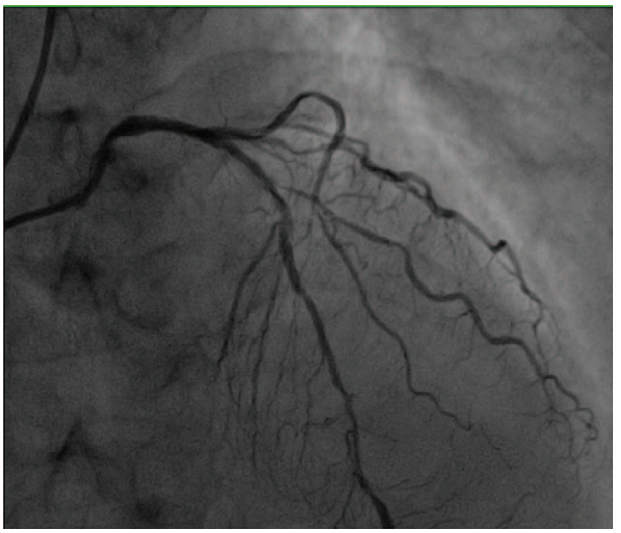

Figure 4. A 50% localized stenosis was seen in the left coronary artery, and 80% diffuse stenosis was observed in the near-middle portion of the left anterior descending branch. A 90% segmental stenosis was seen in the opening of the diagonal branch 1, and 80% to 90% segmental stenosis was seen in the near-middle of the left circumflex branch. A 90% segmental stenosis can be observed in the opening of obtuse marginal branch 1.

The Ethics Approval/Institutional Review Board (IRB) is not needed because this is not a case of new intervention is performed.

3. Discussion

ACOP not only leads to severe disturbance of consciousness, brain edema, pulmonary edema, and shock but also causes severe myocardial damage. It has been reported that more than one-third of moderate-to-severe carbon monoxide poisoning is associated with myocardial damage.[2] The clinical characteristics of such heart disease is that myocardial damage often occurs 1 to 7 days after poisoning. A higher chance of myocardial damage is associated with more severe poisoning, and the earlier the time of occurrence, the more severe the degree of damage. Manifestations of the heart

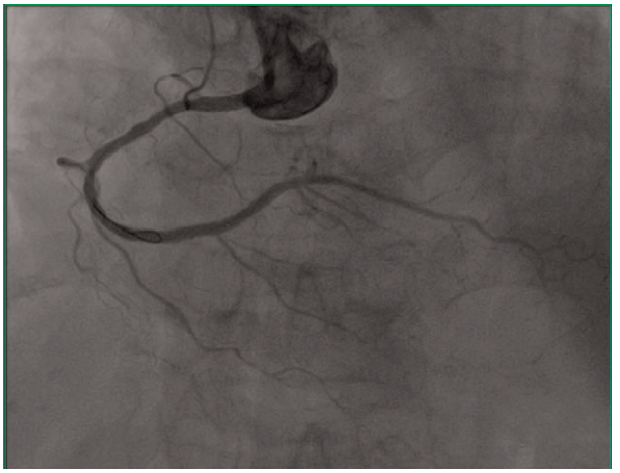

Figure 5. Surgical intervention on the right coronary artery and the implantation of 3 stents during the coronary angiography significantly improved the blood flow.

damage include angina, myocardial infarction, heart failure, and arrhythmias which include ventricular premature beats, atrial fibrillation, bradycardia, and atrioventricular block.[2] The main reason for the tissue damage caused by carbon monoxide poisoning is hypoxia. Furthermore, carbon monoxide is more detrimental to the myocardium than to other tissues because the myocardial oxygen demand is higher than that of peripheral tissues at rest.[7] The binding of carbon monoxide to hemoglobin is 250 to 300 times greater than that of oxygen, while the dissociation curve of oxygen and hemoglobin shifts to the left, which causes hypoxia in tissues.[8] Moreover, carbon monoxide can damage cytochrome C oxidase and reduce glutathione and has a direct toxic effect on the mitochondrial respiratory cascade.[9,10] Adenosine triphosphate (ATP) production by myocardial cells is inhibited, resulting in anaerobic metabolism, cellular hypoxia, lactic acidosis, and apoptosis. Meanwhile, carbon monoxide can induce coronary artery spasm and slow blood flow-induced thrombosis, increase vascular permeability, and accelerate platelet aggregation. All these factors lead to myocardial damage.[11]

Satran et al[3] reported cases of myocardial damage after carbon monoxide poisoning. The first type of patient is the relatively young patient who has no cardiovascular risk factors. When severe carbon monoxide poisoning occurs, left ventricular motor function is likely to decrease, whereas coronary angiography shows a normal condition. The decreased left ventricular motor function may improve gradually with treatment. The second type of patient is the senior patient who has cardiovascular risk factors and may potentially have severe coronary artery diseases. Regional wall motion abnormality occurs at the time of carbon monoxide poisoning, which causes hypoxia to induce myocardial infarction. The reason is that carbon monoxide poisoning can break the weak balance between oxygen supply and demand, resulting in myocardial infarction. It is usually difficult to diagnose these 2 types of patient on the basis of only clinical symptoms, myocardial enzymology, and echocardiography. Therefore, the possibility of myocardial infarction is relatively high for senior patients with carbon monoxide poisoning, especially in the presence of cardiovascular risk factors. In this regard, coronary angiography should be performed at an early stage to facilitate precise diagnosis.[12]

It should be noted that the degree of carbon monoxide poisoning may not be directly proportional to the degree of myocardial damage. In most cases, a more severe degree of carbon monoxide poisoning leads to serious myocardial damage and high troponin values. However, in rare cases, mild carbon monoxide poisoning may also lead to severe myocardial injury. The case reported herein is an example: the patient was a senior patient, and there were multiple cardiovascular risk factors (hypertension and diabetes), which have the potential to induce severe coronary artery disease. Mild carbon monoxide poisoning led to severe myocardial infarction in this patient.

The patient showed dyspnea as the main symptom, without typical chest pain, when she was admitted to hospital. This indicates that the myocardial infarction induced by carbon monoxide poisoning may not produce typical symptoms. The hypoxia caused by carbon monoxide poisoning can cause damage to multiple organs, such as non-cardiogenic pulmonary edema, and the patient may show symptoms of chest tightness and shortness of breath, which should be differentiated from cardiogenic pulmonary edema caused by myocardial infarction. Therefore, it is necessary to conduct early diagnostic testing carefully, and to perform dynamic monitoring of indicators of myocardial damage, such as troponin, creatine kinase, and to undertake echocardiography, for precise diagnosis in the early stage.

For the treatment of carbon monoxide poisoning, Henry et al[13] recommended that the patient inhale 100% oxygen until the symptoms disappear and the carboxyhemoglobin level drops to 5% to 10%. If the cardiovascular system is affected by poisoning, it is recommended that the carboxyhemoglobin should fall below 2%. Under normal pressure, nasal cannula oxygenation takes a long time to improve hypoxia. Recently, hyperbaric oxygen has been utilized for the early treatment of ACOP. Compared with standard oxygen therapy, hyperbaric oxygen therapy can rapidly dissociate carboxyhemoglobin and promote carbon monoxide elimination. Meanwhile, hyperbaric oxygen reduces the secretion of inflammatory factors, increases mitochondrial function, inhibits lipid peroxidation, and reduces leukocyte adhesion to damaged microvascular walls.[14] Experiments on non-human animals have demonstrated that hyperbaric oxygen administration reduced the infarct size in the ischemic rabbit heart during reperfusion[15]; nevertheless, other studies have reported that hyperbaric oxygen had no beneficial effects on infarct size in dogs.[16] At present, there are no more reports on hyperbaric oxygen therapy for carbon monoxide poisoning combined with myocardial infarction. Further clinical studies are needed.

This patient has a good short-term prognosis owing to timely and effective treatment. In regard to the long-term prognosis, Henry et al[13] conducted a prospective cohort study of 230 patients with moderate to severe carbon monoxide poisoning from 1994 to 2005, among whom 37% had troponin I levels $\geq$0.7 ng/mL or creatine kinase $\geq$5.0 ng/mL and/or changes on echocardiography. During the average of 7.6 years' follow-up, 54 patients died (24%), and the mortality rate of patients with myocardial damage was significantly higher than that of those without myocardial damage. Therefore, for patients with carbon monoxide poisoning combined with myocardial damage, diagnosis, and treatment should be performed at an early stage of myocardial damage to reduce the long-term mortality rate.

Acknowledgments

The authors thank the patient for agreeing with our report and for providing a detailed medical history.

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Methodology: Hongshi Wang.
Project administration: Lin Yang.
Supervision: Zhuo Li.
Writing – original draft: Yu Gao.
Writing – review & editing: Yu Gao.

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