Case Report

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A Case of Acute Carbon Monoxide Poisoning Resulting in an ST Elevation Myocardial Infarction

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Carbon monoxide (CO) is a well-known chemical asphyxiant, which causes tissue hypoxia with prominent neurological and cardiovascular injury. After exposure to CO, several cardiac manifestations have been reported, including arrhythmias, acute myocardial infarction, and pulmonary edema. However, an ST elevation myocardial infarction (STEMI) due to CO poisoning is a very rare presentation, and the treatment for STEMI due to CO poisoning is not well established. Here, we report a rare case of STEMI complicated by increased thrombogenicity secondary to acute CO poisoning and complete revascularization after antithrombotic treatment. **(Korean Circ J 2012;42:133-135)**

KEY WORDS: Carbon monoxide; Myocardial infarction.

Introduction

Carbon monoxide (CO) is known as a silent killer, because it is an odorless, colorless, and non-irritating gas. Myocardial infarction related to CO poisoning has been frequently reported in the literature; however, an ST elevation myocardial infarction (STEMI) due to coronary occlusion is a very rare presentation.[1)]

Although an increased tendency for thrombogenesis during CO poisoning has been reported,[2)] the precise mechanism and treatment of STEMI related to CO poisoning remain uncertain. Here, we report a rare case of acute STEMI complicated by increased thrombogenicity secondary to acute CO poisoning and complete revascularization after anti-thrombotic treatment.

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Case

A 47-year-old male presented to the emergency department with prolonged chest pain, which developed during underground mining. Upon arrival at the emergency department, his vital signs were blood pressure, 158/119 mm Hg; heart beat, 68/min; respiratory rate, 12/min; body temperature, 36.5°C; and O_2 saturation, 100%. Initial laboratory data revealed the following: carboxyhemoglobin (COHb), 2.6%; troponin-I, 3.06 ng/mL; myoglobin, 321 ng/mL; and creatine kinase (CK)-MB, 22.9 ng/mL. Electrocardiography (ECG) revealed an ST elevation in leads II, III, and aVF (Fig. 1). Transthoracic ECG, which was performed when the patient arrived at hospital, demonstrated hypokinesia of the inferior wall and preserved systolic function (ejection fraction=50%). The patient was diagnosed with STEMI and underwent emergency coronary angiography.

Coronary angiography revealed total occlusion of the posterior descending (PD) branch of the right coronary artery (RCA) with a large occlusive thrombus burden (Fig. 2). Because the thrombotic occlusive lesion was far from the distal site of the RCA, we decided to closely observe the patient in the coronary care unit and administer anti-thrombotic medications including heparin, aspirin, and clopidogrel. An electrocardiogram obtained 8 hours after admission showed normalization of the ST segment elevation, Q wave, and T waves in leads II, III, and aVF (Fig. 3). Cardiac enzymes were elevated, with a CK-MB of 153.9 ng/mL and troponin I of 30.3 ng/mL on the first admission day. Three days after admission, a follow-up coronary angiography was normal (Fig. 4). The patient was discharged

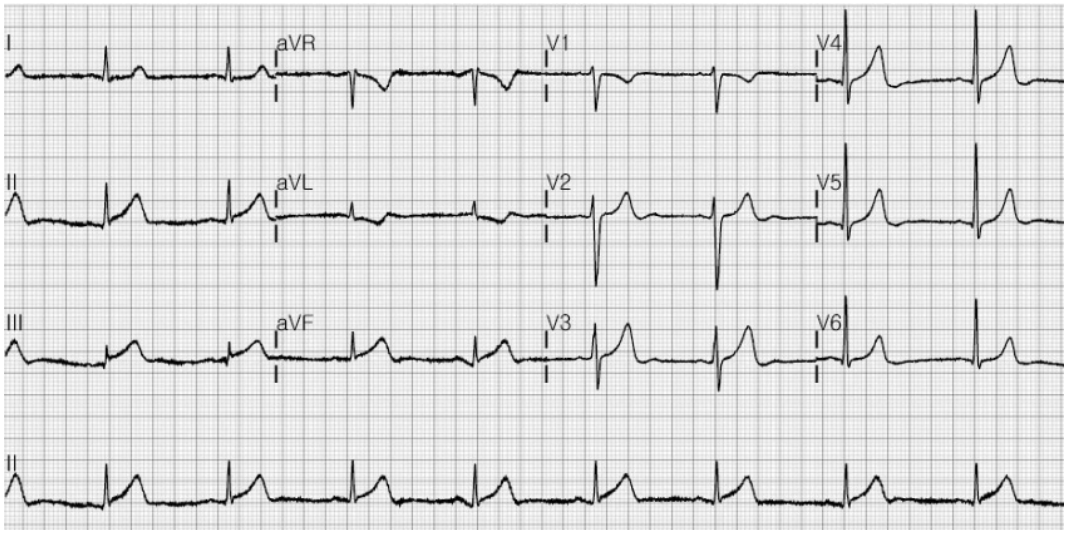

Fig. 1. Electrocardiogram showing ST elevation in leads II, III, and aVF at admission.

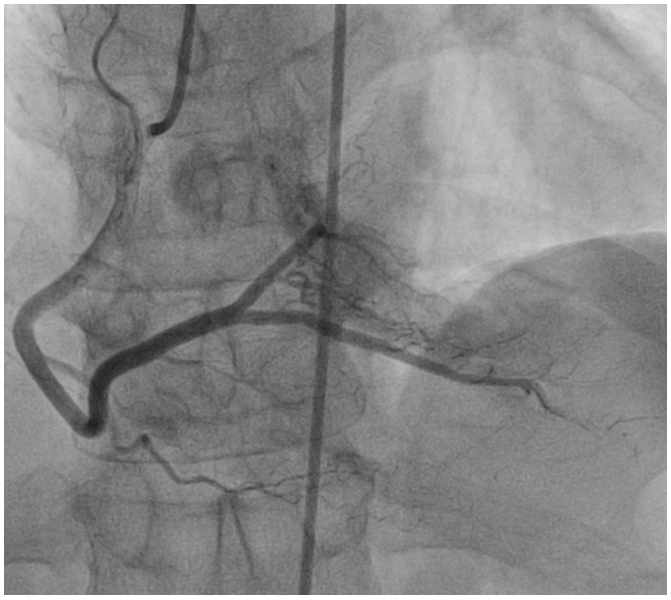

Fig. 2. Coronary angiogram showed total occlusion of the posterior descending branch of the right coronary artery with a large occlusive thrombus burden.

later in a stable condition.

Discussion

Carbon monoxide is one of the leading causes of poisoning-related deaths in the United States. CO binds to hemoglobin with greater affinity than oxygen and forms COHb, which leads to impaired oxygen transport and subsequent tissue hypoxia.[3)4)]

After exposure to CO, several cardiac manifestations have been reported, including arrhythmias and electrocardiographic alterations,[5)] acute myocardial infarction, pulmonary edema, and cardiogenic shock.[6)] Patients with coronary artery disease are more susceptible to CO-induced cardiotoxicity.[7)]

Inhaling even relatively small amounts of CO can lead to hypoxic injury, neurological damage, and possibly death. Additionally, even if the initial COHb level is not very high, acute CO poisoning can still lead to severe cardiovascular complications in high cardiovascular risk cases.[8)] Toxicity also increases due to several factors, including increased activity and ventilation rate, preexisting cerebral or cardiovascular disease, reduced cardiac output, anemia or other hematological disorders, decreased barometric pressure, and high metabolic rate.[9)]

Myocardial ischemic changes often reveal T-wave inversion or ST depression in patients with CO poisoning. However, an ST segment elevation is a rare presentation during CO poisoning. Some cases reported with an ST elevation had normal coronary arteries while undergoing coronary angiography.[1)] However, Hsu et al.[8)] reported a case of CO poisoning complicated with STEMI, which had total occlusion of the left anterior descending artery and underwent primary percutaneous coronary intervention.

The proposed mechanisms of myocardial damage are myocardial stunning as a result of CO poisoning or unmasking of underlying coronary arterial disease by creating a myocardial demand/supply mismatch.[10)] Furthermore, hematocrit, blood viscosity, and platelet function have been implicated as very important pathophysiological mechanisms in patients with acute myocardial infarction but normal coronary arteries. An increasing thrombotic tendency secondary to platelet stickiness and polycythemia has been reported in patients with CO poisoning.[11)]

A COHb level >3% in nonsmokers or >10% in smokers confirms exposure to CO.[12)] In our case, even if the initial COHb level was not very high, emergency coronary angiography revealed total occlusion of the PD branch of the RCA. However, the follow-up coronary

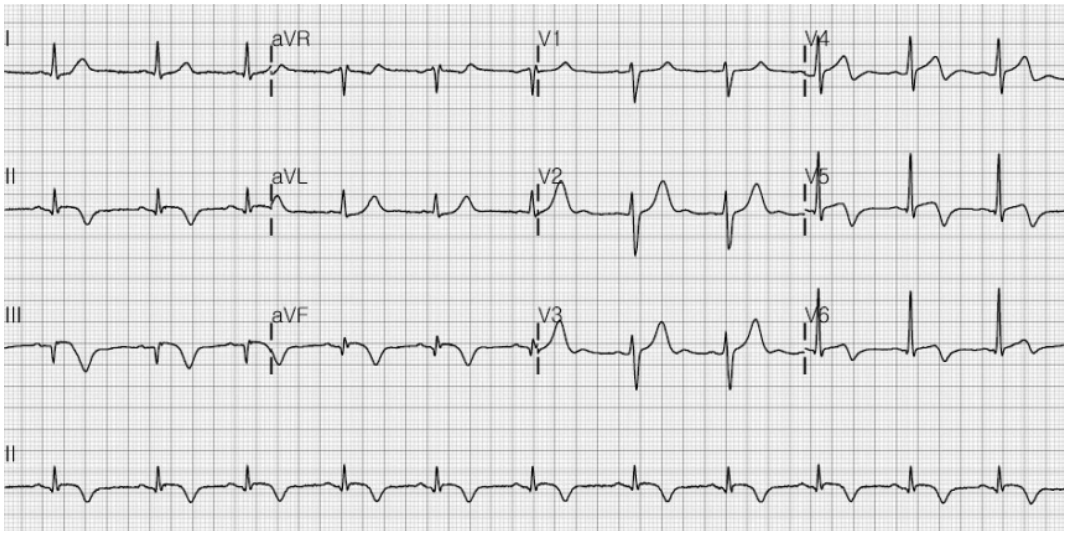

Fig. 3. Electrocardiogram showing Q wave and T inversion in leads II, III, and aVF at discharge.

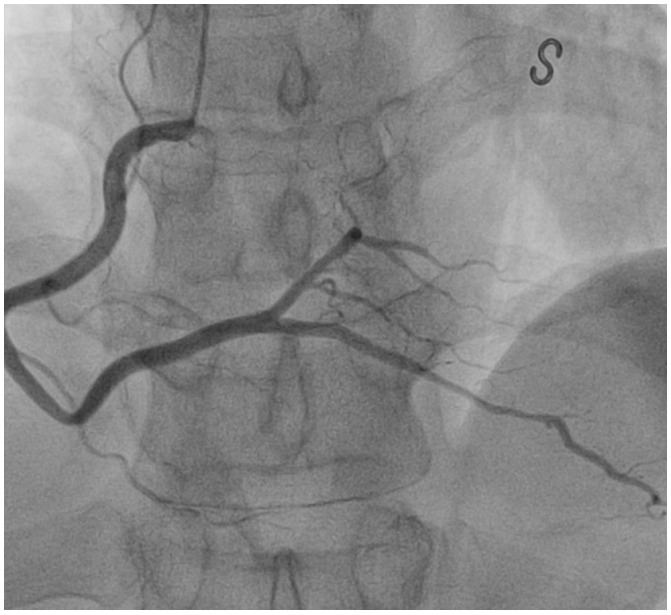

Fig. 4. Follow-up coronary angiogram showed a normal right coronary artery.

angiography revealed a normal coronary angiogram after anti-thrombotic treatment, unlike other cases. The present case was unique in that the mechanism leading to the STEMI in the CO poisoned state was illustrated by coronary angiography, and complete revascularization was performed after anti-thrombotic treatment without percutaneous coronary intervention.

In conclusion, this case highlights that anti-thrombotic treatment is the most essential therapy for CO poisoning complicated by STEMI in some specific cases.

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