ST Elevation Myocardial Infarction Due to Carbon Monoxide Poisoning

Karbonmonoksit Zehirlenmesine Bağlı ST Elevasyonlu Miyokard İnfarktüsü

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Abstract

Carbon monoxide is formed as a result of combustion of any carbon compound and can lead to hypoxia in many organs including the brain and the heart. Carbon monoxide poisoning in the United States is the leading cause of the fatal poisonings. In this study we present a case with no-known accompanying disease in the light of literature where myocardial infarction was developed as a result of carbon monoxide poisoning.

Key Words: Carbon monoxide, Myocardial infarction, Poisoning

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Introduction

The exogenous sources of carbon monoxide (CO), which is colorless, odorless and nonirritating, are carbon-bearing compounds such as manufactured gas, exhaust gas, firewood and coal [1, 2].

Because CO is heavier than air, it can quite easily accumulate, even in environments that are well ventilated [1]. The affinity of hemoglobin for CO is 200 times higher than its affinity for oxygen, which restricts the oxygen-carrying capacity of blood and leads to reduced O2 supply for the tissues [3]. CO can also cause direct cellular injury by forming bonds with enzymes and respiratory pigments, such as myoglobin, cytochrome P450 and cytochrome aa3 [4].

CO intoxication is a fatal condition with early and late-term adverse effects [1, 2]. It is known that organs that need a high oxygen level, including the brain and heart, are easily affected during the intoxication period [2]. The symptoms include nausea, vomiting, concentration and memory impairment, emotional lability, dizziness, paresthesia, weakness, stroke, coma, seizure, chest pain, tachycardia, hypotension and cardiac and respiratory arrest [5].

Though myocardial infarction (MI) is rarely reported after CO intoxication, no distinct ratio is given [6-8]. In this study, a patient with no known prior history of coronary artery disease, who developed MI following CO intoxication, is described, along with a review of the relevant literature.

Case Report

A 44-year-old male patient was sent to our hospital's emergency service for further medical examination after he had been taken to an trauma center with complaints of changes in consciousness. It was thought that he had experienced a minor myocardial infarction. Upon admission to the hospital, his arterial blood pressure was 140/70 mm Hg, his respiratory rate was 20/min, his temperature was 36°C and his O2 saturation level was 94%. His Glasgow coma score (GCS) (E3M5V2) was 10, his pupils were isochoric and his light reflex was bilateral positive during his physical examination. No focal finding, meningeal irritation evidence or pathologic reflex was noted. His heart rate was 110/min during auscultation, and the other system examinations returned normal results. It was determined from his history that he had slept
in a room with a wood burning stove. He had no complaints before the event occurred, and he was found unconscious by his family the following morning. He had no history of chronic illness regarding drug or alcohol use or known coronary artery disease. However, it was noted that he had a 30 pack year history of smoking. A flow of 10 L/min O₂ was applied to the patient with a mask, vascular access was opened and he was monitored in our emergency facility. In the electrocardiogram taken at the previous hospital where he had first been taken, his normal sinus rhythm was 110/min, and there was ST segment elevation in D2, D3 and aVF (Figure 1). In the workups conducted during his admission, the lab values were as follows: pH>7.26 (7.35-7.45), pCO₂: 40 (35-45) mmHg, pO₂: 58 (80-100) mmHg, HCO₃: 14.3 (22-26), carboxyhemoglobin (COHb) level: 32.2% (0.5-1.5), sat O₂: 94.8%, glucose: 152 mg/dL, aspartate transaminopherase: 41 (0-37) U/L, aspartate transaminopherase: 44 (0-41) U/L, creatine kinase (CK): 169 (0-170) U/L, and creatine kinase myocardial band (CK-MB): 29.42 (0-24) U/L. In his follow-ups, the following values were obtained: CK: 1831 U/L and CK-MB: 80.1 U/L. Because there was no troponin kit at the hospital when the patient was referred to our hospital, only CK and CK-MB could be performed. In the ECG, the cardiac sinus rhythm was 110/min, and ST elevation produced an isoelectric error in D2, D3 and aVF (Figure 2). The lung graphy of the case was evaluated as normal. Minimal brain edema was identified in the computed brain tomography image (Figure 3). Because the patient slept in a room with a burning stove, his myocardium and brain were affected by the high COHb level, and he was consequently diagnosed with CO intoxication. Because there is no hyperbaric oxygen treatment center in or around the city in which our hospital is located, the patient was taken to the intensive care unit in our hospital. Intubation was not needed, so an oxygen mask was applied. After approximately 8 hours of monitoring in the intensive care unit, his GCS was determined to be 15, and he was discharged from the hospital without any sequela on the second day of his hospital stay. The patient was advised to apply to a center where coronary angiography was available. Subsequently, the patient was called at his home and invited to the hospital for his follow-up. On the 30th day after the intoxication event, his neurological examination was normal. His echocardiogram, ECG test and brain magnetic resonance imaging observations were reported as normal. He was reminded that he had not undergone a coronary angiography in another center.

Discussion

Carbon monoxide intoxication is a commonly seen health issue all over the world. The number of CO intoxication cases resulting in death are high in the U.S [9]. It is reported that 40,000 people in the U.S. use the emergency services of hospitals each year as the result of CO intoxication; these cases are primarily encountered during the cold seasons [4].

In mild and moderate CO intoxication, headache, nausea, vomiting, vertigo, blurred vision, confusion, syncope, chest pain, shortness of breath, tachycardia and rhabdomyolysis are seen, whereas hypotension, noncardiogenic pulmonary edema, myocardial ischemia, rhythm disorders, seizure, coma, heart and respiratory arrest are observed in

Figure 1. ST segment elevation in D2, D3 and aVF.
severe intoxication [4]. The COHb level is used in the diagnosis of and the treatment follow-up for CO intoxication. In patients whose COHb level is <15-%-20%, it is stated that there is a correlation between mild and medium intoxication findings [3, 10]. The case presented in this report belonged to the severe intoxication group because of the myocardial ischemia findings, the minimal brain edema and a COHb level above 30%.

It has been suggested that electrocardiographic ST segment and T wave changes could be observed in carbon monoxide intoxication [8]. Altay et al. presented a case in which myocardial ischemia developed in spite of no known history of coronary artery disease, precipitated by CO intoxication. Çelik et al., however, have reported that following CO intoxication of a patient with known coronary artery disease, a newly developed left bundle branch block was determined in the patient [7, 8]. The patient in our study had no known coronary artery disease, and after the CO intoxication, ST segment elevation developed in D2, D3 and avF. His cardiac diagnosis tests (echocardiography and ECG with physical effort), which were performed after his discharge from the hospital, were assessed as normal.

In spite of various clinical studies, the best treatment for carbon monoxide intoxication is still a matter of debate. The use of 100% oxygen and/or hyperbaric oxygen is preferred in some patients depending on their symptoms, accompanying diseases and COHb level [2, 7].

The half-life of CO is 320 minutes under normal room conditions, whereas it is 80 minutes when receiving 100% oxygen and 23 minutes under hyperbaric oxygen treatment circumstances [3].
One of the indications for hyperbaric O₂ treatment of CO intoxication is myocardial ischemia findings [10]. Fiorista et al. presented a case in which myocardial ischemia developed following CO intoxication, and no revascularization was applied initially. They reported that the reason for ischemia was the reduction in the O₂ carrying capacity of blood, which developed secondary to tissue hypoxia [11]. Sorodoc et al. published in their study that they did not apply invasive revascularization for the diagnosis and treatment of cases with CO intoxication leading to Q-wave MI. Specific treatment was applied for intoxication, and they reported that their patient completely recovered [12]. Lee D et al. stated that they applied a hyperbaric O₂ treatment to their patient who underwent MI due to CO intoxication, and they reported that the coronary angiography that was carried out later was normal [13]. Because of the high level of COHgb, the case was diagnosed with CO intoxication. Because the electrocardiographic changes were attributed to CO intoxication, no thrombolytic or percutaneous coronary intervention was considered.

In CO intoxication, tissue hypoxia due to the shortage of oxygen delivery and the clinical effects responsible for mitochondrial damage are the most significant factors. In contrast, AMI can occur on account of vasospasm as a result of CO toxicity and atherosclerotic plaque rupture [7, 13, 14]. Therefore, the nonexecution of coronary angiography to identify underlying atherosclerotic heart disease in the presented case can be regarded as a failure in patient management.

In conclusion if there is a coronary artery disease history, CO intoxication is a facilitating factor in myocardial ischemia. However, as has been presented in our study, CO intoxication can also lead to MI, despite no known history of coronary artery disease.

Conflict of interest statement: The authors declare that they have no conflict of interest to the publication of this article.

References