CASE REPORT

Non-ST elevation myocardial infarction induced by carbon monoxide poisoning

Karbon monoksit zehirlenmesinden kaynaklanan ST-yükselmesiz miyokart enfarktüsü

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Summary– Carbon monoxide (CO) poisoning is the most common cause of poisoning-related death in the world. Cardiovascular complications of CO intoxication includes myocardial damage, left ventricular dysfunction, pulmonary edema, and arrhythmias. The carboxyhemoglobin level does not correlate with the clinical severity of CO intoxication. This case report presents a patient with acute myocardial infarction secondary to carbon monoxide poisoning who was successfully treated with coronary bypass surgery.

Özet– Karbonmonoksit (CO) zehirlenmesi dünyadaki zehirlenmeye bağlı ölümlerin en yaygınıdır. CO zehirlenmesinin kardiyovasküler komplikasyonları; miyokart hasarı, sol ventrikül disfonksiyonu, pulmoner ödem ve aritmileri içerir. Karboksihemoglobin seviyeleri, CO zehirlenmesinin klinik ciddiyetiyle korele değildir. Bu yazıda, karbon monoksit zehirlenmesine ikincil akut miyokart enfarktüsü sonrası koroner baypas ameliyatıyla başarılı bir şekilde tedavi edilen bir hastayı sunuyoruz.

Carbon monoxide (CO), commonly called as 'silent killer', is a colorless and odorless gas produced by the incomplete combustion of hydrocarbons.[1] The malfunction or inappropriate use of fire-related appliances like furnaces, stoves, generators, heaters, and fireplaces, as well as gas line leakage, contribute to CO poisoning, especially during the winter months.[2] CO can cause hypoxia by hindering the delivery of oxygen (O_2) to tissues because it binds to hemoglobin with a 250-fold greater affinity than O_2 to form carboxyhemoglobin (COHb).[1,3]

CO poisoning may have various clinical manifestations, ranging from headache, nausea, malaise, dizziness, and syncope, to death. The severity of the symptoms and presentation are related to the concentration of CO and duration of exposure. Cardiovascular complications are common in CO poisoning due to the increased oxygen requirement and high fraction of O_2 extraction by the myocardium. Commonly reported cardiovascular complications include myocardial stunning, acute myocardial ischemia, left ventricular dysfunction, and arrhythmias resulting in permanent myocardial damage.[1] Ischemic findings occur more easily in elderly patients with underlying diseases such as heart disease, lung disease, or diabetes mellitus. Myocardial ischemia may remain undetected because the findings may be masked in patients presenting with respiratory and neurological findings. Mortality and morbidity rates may be reduced by confirmation of ischemic findings and initiation of the appropriate treatment.[4]

Abbreviations:

AST	*Aspartate aminotransferase*
ALT	*Alanine aminotransferase*
CK	*Creatine kinase*
CO	*Carbon monoxide*
COHb	*Carboxyhemoglobin*
ECG	*Electrocardiography*
HBO_2	*Hyperbaric oxygen*
LDH	*Lactate dehydrogenase*
NBO_2	*Normobaric oxygen*
O_2	*Oxygen*
$PaCO_2$	*Arterial partial pressure of carbon dioxide*
PaO_2	*Arterial partial pressure of oxygen*
SpO_2	*Peripheral oxygen saturation*
TTE	*Transthoracic echocardiography*
VES	*Ventricular extrasystole*

Myocardial infarction related to CO poisoning has frequently been reported in the literature.[1,4] This is a

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case report of a patient with mild carbon monoxide poisoning-induced severe non-ST elevation myocardial infarction.

CASE REPORT

A 68-year-old woman was referred to the emergency department with complaints of headache, nausea, vomiting, and agitation. 4-6 hours after the patient's complaints started, she was referred to us after the COHb level was 14% in the arterial blood gas, which was examined at the outer centre. She had a history of controlled hypertension and diabetes mellitus for 2 years and no history of smoking. Upon arrival at the emergency department, her vital signs were as follows: Blood pressure: 102/70 mm Hg, heart rate: 102/minute, respiratory rate: 12/minute, body temperature: 36.5°C, and peripheral oxygen saturation (SpO_2) level: 77%. No pathological findings were found in the physical examination, other than a tendency to sleep. The initial laboratory data revealed COHb: 7.35% (%0.5-1.5), troponin-T: 0.034 ng/mL (0.00-0.03 ng/mL), creatine-kinase (CK)-MB: 6.82 ng/mL (0-2.88 ng/mL), glucose: 351 mg/dL, hemoglobin: 13 g/dL, white blood cell count: 14.300/mm^3, urea: 37 mg/dL, creatinine: 1.1 mg/dL, aspartate aminotransferase (AST): 145 IU/L, alanine aminotransferase (ALT): 32 IU/L, lactate dehydrogenase (LDH): 196 IU/L, pH: 7.39, partial pressure of arterial oxygen (PaO_2): 41.5 mmHg, partial pressure of arterial carbon dioxide ($PaCO_2$): 29.9 mmHg, and SpO_2: 77.6%. Electrocardiography (ECG) showed normal sinus rhythm. The patient was suspected to have CO poisoning, and a face mask providing 10 L/minute of 100% O_2 was administered. The patient was transferred to the intensive care unit with a preliminary diagnosis of CO poisoning.

The treatment of 10 L/minute of 100% O_2 with a face mask was continued. The control blood gas evaluation taken after 30 minutes revealed values of pH: 7.38, PaO_2: 42 mmHg, $PaCO_2$: 30 mmHg, SpO_2: 79%, and COHb: 5.9%. The COHb measurement was 0.9% at the fourth hour of hospitalization. On the second day of hospitalization, the patient had sudden chest pain, tachypnea, and sweating. The SpO_2 level decreased to 75%, blood pressure increased to 220/120 mmHg, and her heart rate was at 130/minute. The patient had bilateral diffuse crackles and rhonchi, and a chest radiograph revealed interstitial edema. Values of troponin-T: 2.18 ng/mL, CK-MB: 125.3 ng/mL, AST: 187 IU/L, and LDH: 389 IU/L were detected. ECG showed normal sinus rhythm with dynamic slight ST segment depressions on inferior and lateral leads with frequent monomorphic outflow tract type premature ventricular complexes (PVCs) (Fig. 1). There was no ST elevation. Transthoracic echocardiography (TTE) revealed a left ventricular ejection fraction of 49% and however dynamic ST-T wave changes were observed on the inferior and lateral ECG leads. Inferior and anteroseptal walls of left ventricular walls were seen to be mildly hypo kinetic, in addition to moderate tricuspid and mitral insufficiency. Pulmonary thromboembolism was ruled out by computerized thoracic pulmonary angiography. Bilevel positive airway pressure was administered in the presence of bilateral pleural effusion and edema. A diagnosis of non-ST segment elevation myocardial infarctian (NSTEMI) was diagnosed and due to the hemodynamic instability early invasive approach was decided. Coronary angiography revealed an osteal 90% critical left anterior descending (LAD) artery lesion with a successive 80% lesion at the level of second diagonal branch, sub-totally occluded LAD diagonal-1 branch, subtotal osteal occlusion on the on the well developed circumflex (Cx) optuse marginalis-1 branch, 80% Cx-posterolateral osteal branch and an osteal spasm on the right coronary artery which was resolved by coronary nitrate infusion. On the 10th day of hospitalization, the patient underwent 4-vessel coronary bypass surgery and was discharged on the 16th postoperative day.

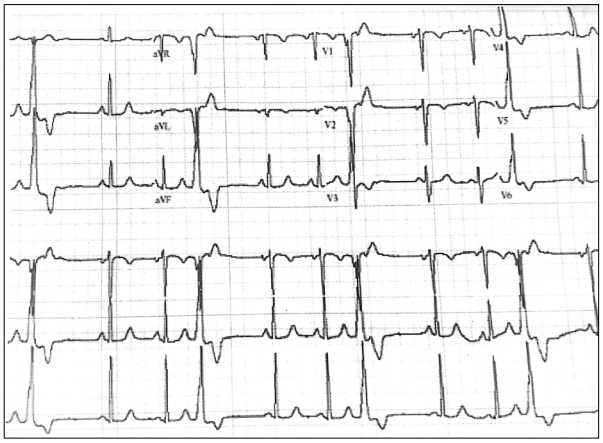

Figure 1. Electrocardiogram taken on the second day of hospitalization with clinical deterioration and chest pain.

Written informed consent was obtained from the patient.

DISCUSSION

CO poisoning may be acute or chronic. Patients usually present with signs of involvement of the central nervous system, but severe cardiac abnormalities may be observed.[1,3,4] In addition to publications indicating a correlation between the level of COHb and the severity of poisoning, there are also reports indicating that this relationship is only present in mild toxicity. It should be kept in mind that the COHb level does not correlate with clinical status, and the toxicity threshold can show individual differences.[1,5,6] Acute CO poisoning is easy to recognize and is clinically urgent. A detailed anamnesis is sufficient for the early diagnosis of CO poisoning. As the half-life of COHb is short, the COHb level should be measured in affected individuals as soon as possible. COHb is used for diagnosis and follow-up. Patients with suspected CO poisoning should be given high-flow oxygen regardless of the saturation value in the pulse oximeter.[7] The current therapy for CO poisoning is 100% normobaric oxygen (NBO_2) or hyperbaric oxygen (HBO_2) (2.5–3 atmospheres). NBO_2 and HBO_2 remove CO from the blood faster by increasing the partial pressure of O_2, which increases the dissociation rate of CO from hemoglobin.[6–9] Elimination of CO in the blood may be accelerated by increasing the O_2 concentration or atmospheric pressure in the breathing air. The half-life of COHb is approximately 4–5 hours in room air, 60 minutes with NBO_2 treatment, and 20 minutes with HBO_2 treatment.[8–10] Although the American College of Emergency Physicians acknowledges HBO_2 as a therapeutic option for CO poisoning, it does not mandate HBO_2 use.[11] Recent practice recommendations by experts in the field of hyperbaric medicine, however, do recommend HBO_2 use for CO poisoning (HBO_2 should be considered for all cases of serious acute CO poisoning, including loss of consciousness, ischemic cardiac changes, neurological deficits, significant metabolic acidosis, or a COHb level >25%). Depending upon the patient's symptoms, comorbidities, and COHb level, NBO_2 and/or HBO_2 are the preferred treatment.[7–11] Almost all patients receive NBO_2 upon rescue or arrival at the emergency department. There is often a significant delay in the delivery of HBO_2 between diagnosis in the field, transportation to a hyperbaric therapy center, and succesive treatment.[8,11] HBO_2 treatment was not considered in our patient because of mild toxicity symptoms, rapid presentation at the hospital after intoxication, and a quick return to normal COHb values with NBO_2 treatment.

Acute CO poisoning may cause severe cardiovascular complications in patients with high cardiovascular risk, even if relatively little CO is inhaled and the COHb level is not very high.[12] Cardiac involvement may occur immediately after exposure to CO, or it may occur even after a few days. After exposure to CO, studies have reported various cardiac symptoms, such as electrocardiographic changes (sinus tachycardia, atrial fibrillation, PVCs, bradycardia), elevated troponin-I, CK-MB fraction, left ventricular dysfunction, acute myocardial infarction, pulmonary edema, and cardiogenic shock. Angina pectoris and myocardial infarction may occur in patients who have ischemic heart disease.[12–15]

History of Satran et al.[15] reported most probably because of a demand-supply mismatch in the coronary circulation of these patients. Satran et al. reported myocardial injury in 37% of patients with moderate to severe CO poisoning and they proposed a baseline ECG together with a serial cardiac biomarker follow-up in all patients admitted with this diagnosis and echocardiogram check in case of abnormal cardiac biomarker results.

However in a study conducted in Turkey, only one of the 40 patients who were followed up with CO poisoning had myocardial damage, and it was concluded that significant myocardial damage and life-threatening cardiac events were less likely to develop in CO-poisoned patients with COHb level below 60% and without any known underlying coronary artery disease.[4] In our patient, we suggest that considering the underlying risk factors like diabetes and hypertension, CO poisoning might be the trigger for the NSTEMI that she suffered.

CO poisoning is common in Turkey. ECG monitoring is important to detect the possibility of silent ischemia, especially in elderly and diabetic patients, even in the presence of mild symptoms, and even after normalization of the COHb level. Therefore, we suggest that ECG follow up, cardiovascular examination should be performed in patients presenting with acute CO poisoning, and in case of previous coronary artery disease and multiple coronary artery disease risk factors, cardiac biomarker follow up might be reasonable

in patients presenting with acute CO poisining.

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Keywords: Coronary bypass; carbon monoxide poisoning; myocardial ischemia.

Anahtar sözcükler: Koroner baypas; karbon monoksit zehirlenmesi; miyokart iskemisi.