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Myocardial damage caused by carbon monoxide poisoning: A case report

Karbonmonoksit intoksikasyonunun neden olduğu miyokardiyal hasar: Olgu Sunumu

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ÖZET

Giriş: Zehirlenmelerin neden olduğu morbidite ve mortalitenin en yaygın sebeplerinden biride karbonmonoksit (CO) zehirlenmesidir. Bu olgu sunumunda CO'e maruz kalan bir hastada gelişen miyokardiyal hasarı sunmayı amaçladık.

Olgu sunumu: Kırk dört yaşında bayan hasta göğüs ağrısı olması nedeni ile dış merkezden acil servisimize sevk edildi. Hasta iki gün önce CO zehirlenmesine maruz kalmış ve dış merkezde tedavi almış. Hastanın özgeçmişinde diyabet, hipertansiyon, hiperlipidemi, sigara kullanma ve kardiyak hastalık öyküsü yoktu. Hastanın çekilen elektrokardiyogramında (EKG) sinüs taşikardisi mevcuttu. Hastanın göğüs ağrısı ve troponin yüksekliği olması üzerine kardiyoloji konsültasyonu istendi. Kardiyoloji tarafından primer perkütan koroner anjiografi yapıldı. Sonucun normal olması nedeniyle CO zehirlenmesine bağlı miyokardiyal hasar düşünüldü. Hastaya 2 gün tedavi verildi ve hasta şifa ile taburcu edildi.

Sonuç: CO zehirlenmesi sonucu acil servise başvuran hastalarda CO-Hb düzeylerinin ölçümü dışında, CO'in zehirlenmesine bağlı miyokardiyal hasar gelişebileceği düşünülerek EKG ve kardiyak enzim takibi de yapılmalıdır.

Anahtar Kelimeler: *Karbon monoksit Zehirlenmesi, Miyokardiyal Hasar, Acil Servis*

ABSTRACT

Introduction: CO is one of the main cause of toxicities which lead to morbidity and mortality. In the present study, we aimed to report a myocardial injury case resulted from CO exposure.

Case Report: 44-year-old female patient admitted to our emergency service from a state hospital with the complaint of the chest pain. The patient had been exposed to carbon monoxide two days ago and had been taken care in the state hospital. The patient has no cardiac problems, diabetes mellitus, hyperlipidemia and smoking history. Her ECG was in the sinus tachycardia pattern. She was consulted to cardiology department for chest pain and positive troponin result. She was intervened percutaneous coronary angiography and no vascular pathology was defined. So, she thought as myocardial injury due to CO exposure. She got therapy in cardiology service for 2 days and discharged after full recovery.

Conclusion: In CO intoxication cases, first CO-Hb level should be analyzed. Additionally, it must be remembered to look for presence of myocardial injury by ECG and cardiac enzymes

Key words: Carbon monoxide poisoning, Myocardial damage, Emergency service

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Introduction

Carbon monoxide (CO) intoxicity is one of the most common cause of morbidity and mortality in the world. Despite the lack of the data, it is reported that CO is responsible from almost 31% of the intoxication cases in Turkey (1). In Turkey, CO poisoning is mostly observed during winter and in windy weather conditions.

Patients with CO intoxication often apply to the hospitals with symptoms involving the central nervous system. However, we also rarely observe severe cardiac abnormalities (2). Since the hemoglobin affinity of the CO is greater than the oxygen, oxygen is released to the tissues in lesser amounts. Cardiac damage is induced by direct toxic effects of CO on both myocardial hypoxia and myocardial mitochondria (3).

In this paper, we aimed to present a myocardial damage case that caused by CO toxicity.

Case Report

A 44-year-old female patient applied to the emergency service with chest pain referred to us by a secondary health care facility. The patient was hospitalized two days ago for carbon monoxide poisoning at another hospital. The patient had no history of diabetes, hypertension, hyperlipidemia, smoking and cardiac disease. Her family did not have a coronary artery disease history. The examination of the patient revealed normal vital signs. There was no pathological physical exam sign. Sinus tachycardia was determined at the electrocardiogram (ECG). Echocardiography (ECHO) showed no obvious pathology. In the blood gas analysis conducted in an external center, carboxyhemoglobin (CO-Hb) level was 11% and other laboratory parameters were normal. On admission to the emergency department, the patient's white blood cell count was 6.1×10^3/mL, hemoglobin level was 13.1g/dL, hematocrit was 39.8%, platelet count was 294×10^3/mL in the whole blood count. When the cardiac enzyme levels analyzed, and the troponin level was 5.6μg/L (0,010-0,023μg/L), the creatine kinase level was 480U/L (29-200U/L) and the creatine kinase-MB level was 132U/L (0-24U/L). In the blood gas analysis, the pH was found as 7.45, the CO-Hb was 1.3% and the pCO2 was 32 mmHg. There was no significant difference on her control ECG. The chest pain was also gone. The 100% oxygen treatment was continued in the emergency department and 300 mg acetyl salicylic acid was administered orally. The patient was previously referred to the cardiology consultation due to the chest pain and elevated troponin. The patient was hospitalized by cardiology department for primary percutaneous coronary intervention. Coronary pathology was not detected after the procedure. Cardiologists diagnosed myocardial ischemia due to carbon monoxide poisoning and treatment of oxygen and acetyl salicylic acid was continued for 2 days in the cardiology department. The patient was discharged when the patient's cardiac enzymes regressed, and normal sinus rhythm was determined at the ECG. No pathological findings were determined at ECG, ECO, and cardiac enzymes after 1-month follow-up.

Discussion

In the present article, a CO induced myocardial damage case was discussed. First myocardial ischemia was considered, because the patient complained chest pain and had elevated levels of cardiac enzymes, and primary percutaneous transluminal angioplasty was implemented. But no coronary pathologies were determined, and CO induced myocardial damage was considered.

Old age, inhalation period, pre-treatment period, presence of coma, presence of metabolic acidosis, increased serum amylase and aspartate aminotransferase levels are considered as poor prognosis markers, however high blood CO-Hb levels were not found to be associated with short and long-term prognosis (4). Thus, the cases are ranked based on the variations in awareness or the presence of a coma and patients with altered consciousness or history are considered as moderate to severe poisoning cases. In certain studies, higher than 40% blood CO-Hb level and myocardial ischemia symptoms or accompanying arrhythmia are defined as moderate to severe poisoning (5). Although in our case, the patient's blood CO-Hb level was 11%, the case was considered as moderate-to-severe poisoning due to lack of awareness history in the patient and presence of myocardial ischemia symptoms.

CO toxicity is a result of the combination of the direct damaging effects of tissue hypoxia and carbon monoxide at the cellular level. CO competes with oxygen to bind to the hemoglobin. The hemoglobin affinity of CO is 200-250 times higher than that of oxygen. Therefore, even minute increases in the environmental CO concentrations could cause toxic CO-Hb. The affinity of CO for myoglobin is very high. CO binding to cardiac myoglobin leads to myocardial depression, hypotension, and arrhythmia. Cardiac dysfunction further increases tissue hypoxia (6). In our case, it was considered that CO was associated with myocardial damage by binding to cardiac myoglobin.

In a study conducted in Turkey, among the 40 patients that were followed due to CO poisoning without a known coronary artery disease, myocardial damage was determined only in one patient (2.5%). In the same study, sinus tachycardia was reported as the most common ECG finding after CO poisoning (22.5% of cases), like our case (7). In the literature, silent myocardial infarction cases due to acute CO poisoning without chest pain were reported as well (8). It was considered that silent myocardial damage due to CO poisoning had developed in the patient due to elevated cardiac enzymes, chest pain and the absence of stenosis in coronary arteries on angiography.

CO-Hb levels are used for diagnosis and follow-up. Use of the 100% oxygen and/or hyperbaric oxygen are preferred in the treatment of the patient's symptoms, associated diseases and CO-Hb level (9, 10). In our case, the patient was treated with acetylsalicylic acid due to the presence of chest pain as a symptom to support 100% oxygen treatment and the patient was discharged into health.

In addition to the measurement of CO-Hb levels in patients that apply to the emergency

department after CO poisoning, EKG monitoring and cardiac enzyme follow-up should therefore be conducted considering possible myocardial damage due to CO poisoning.

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