Case Report

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Rare Inferior Myocardial Infarction Triggered by Carbon Monoxide Poisoning

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Abstract

Carbon monoxide (CO) poisoning is one of the most common causes of death due to poisoning in the world. Cardiovascular complications of CO poisoning includes myocardial infarction, left ventricular dysfunction, cardiogenic shock, and various arrhythmias. Carboxyhemoglobin (COHb) levels do not always correlate with severity of symptoms. In this article, We present a patient who developed a rare ST elevated acute myocardial infarction triggered by CO poisoning and was treated with coronary angiography.

Keywords: CO poisoning, inferior myocardial infarction, ST elevation

Özet

Karbonmonoksit (CO) zehirlenmesi sık karşılaşılan dünyada zehirlenmeye bağlı ölümlerin en yaygın sebeplerindendir. CO zehirlenmesinin kardiyovasküler komplikasyonları miyokard enfarktüsü, sol ventrikül disfonksiyonu, kardiyojenik şok ve çeşitli aritmileri içerir. Semptomların şiddeti ile karboksihemoglobin (COHb) seviyeleri her zaman korele değildir. Bu yazıda, CO zehirlenmesinin tetiklediği ST elevasyonlu akut miyokart enfarktüsü gelişen ve koroner anjiografi ile tedavi edilen bir hastayı sunuyoruz.

Anahtar Kelimeler: CO intoksikasyonu, inferior miyokard enfarktüsü, ST elevasyonu

Introduction

Carbon monoxide (CO); since it is a tasteless, odorless and colorless gas, patients may apply to the emergency service in the late period in cases of poisoning. CO shows 200-250 times higher affinity for binding to hemoglobin than oxygen[1]. The symptoms, signs and prognosis of acute CO poisoning correlate poorly with the level of carboxyhemoglobin (COHb)[2].

Since CO binds to cardiac myoglobin with higher affinity than hemoglobin, myocardial depression and hypotension develop; may further exacerbate tissue hypoxia, leading to myocardial infarction[3]. Even 5-10% increases in COHb levels in people with previous coronary artery disease (CAD) may trigger exercise angina. High levels of COHb may affect the myocardium even in young and healthy individuals[4]. The occurrence of ST elevated myocardial infarction (STEMI) is rare.

In this case report, we aimed to present a rare case of STEMI after acute CO poisoning in a patient without coronary artery disease.

Case

An 87-year-old male patient was brought to the hospital by 112 ambulance service with complaints of headache, nausea and vomiting. He had no history of diabetes mellitus, hypertension, coronary artery disease, and was paraplegic after previous surgery. In the detailed anamnesis, it was learned that they lived in a house with a stove. His blood pressure was 90/70 mmHg, his heart rate was 94/minute, and his respiratory rate was 22/minute. Heart beats were dysrhythmic in cardiovascular system examination. No additional sound or murmur was heard. Pulmonary and other system examinations were normal. ST elevation was detected in DII, DIII,

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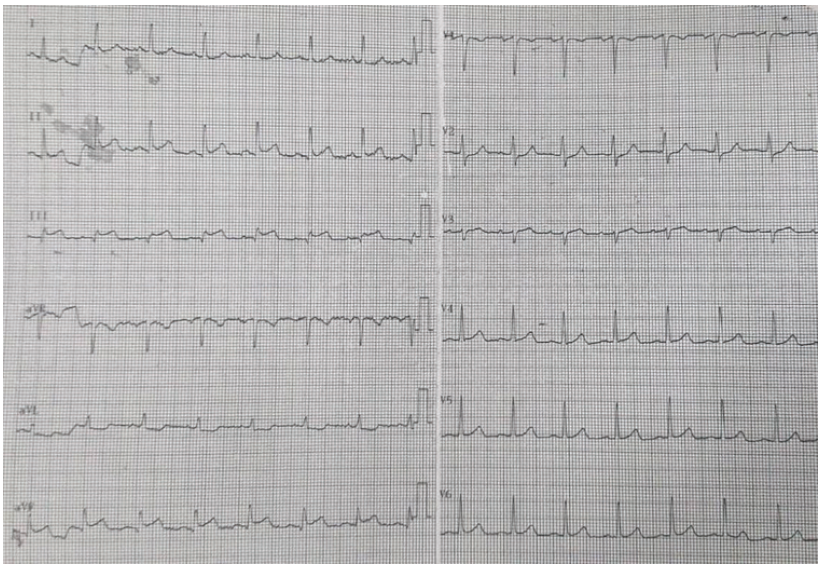

Figure 1: ST elevation in DII, DIII and aVF

aVF in his electrocardiography (ECG) (Figure 1). It was evaluated as acute STEMI. No abnormality was observed except for COHb 22.8%, WBC 19.600/U, glucose: 172mg/dl, creatinine:1.4mg/dl in the first laboratory tests, but cardiac markers were elevated in the follow-up. Troponin I: 41.6 ng/L, CK-MB: 5.7 ng/mL. He was given 10lt/minute O_2 treatment with a reservoir mask and 300 mg tb acetylsalicylic acid. The patient was given 180 mg of ticagrelor. The patient was admitted to the coronary intensive care unit. Coronary artery angiography of the patient revealed complete occlusion in the posterior branch of the right main coronary. The patient was taken to the ward from the intensive care unit on the third day after angiography. The patient recovered clinically and symptomatically and was discharged on the 5th day with full recovery.

Discussion

Early diagnosis is life-saving in CO poisoning. However, the most important factor in the diagnosis is doubt. Detailed anamnesis is important in patients presenting with nonspecific findings. It can present with symptoms ranging from flu-like symptoms to coma and death. The most common symptom is seen in the central nervous system and cardiovascular system, where oxygen use is most intense. The affinity of CO to hemoglobin; it can cause deep tissue hypoxia because it is 200–250 times more than oxygen. At the same time, it directly affects cellular respiration, as it binds to cytochrome oxidase[5].

COHb can be used in the diagnosis and follow-up. Patients with suspected CO poisoning should be given high-flow oxygen, regardless of peripheral oxygen saturation[6]. Our patient also presented with nonspecific findings such as headache, nausea and vomiting, but when the anamnesis was deepened, it was learned that he lived in a house with a stove. The patient was immediately started on oxygen at 10 lt/min with a reservoir mask. The COHb level in the blood gas was found to be high.

While myocardial damage and fibrosis are observed at low dose exposures, it has been reported that fatal arrhythmias can be seen at high dose exposures. It is known that cardiotoxicity in CO poisoning is not only related to hypoxia, but also that CO gas has a direct toxic effect by inhibiting cytochrome oxidase in myocyte mitochondria[7].

Cardiac findings after moderate-severe CO poisoning were examined in a study conducted by Satran et al. It was found that 30% of the patients had ischemic ECG changes, and 35% had myocardial damage in cardiac markers. The mortality of the patients was found to be 5%. With these results, it was suggested that cardiac sequelae are common after CO poisoning and that patients should be followed up with ECG and serial cardiac markers[8].

Dziewierz et al. found inferior STEMI in their patients with a COHb level of 22% in their case report. They reported that acute occlusion of the distal left anterior descending coronary artery was detected in the patient's coronary angiography, and that symptoms regressed after angioplasty[9]. Kim et al. reported complete occlusion in the right main coronary posterior branch in the angiography of the patient in whom they detected STEMI after CO poisoning. They stated that this obstruction disappeared in control angiography after anti-thrombotic therapy[10]. In our patient, although there was no history of coronary artery disease, we detected inferior STEMI. In his angiography, 50-60% occlusion was detected in the right main coronary artery branch. In

patients without coronary artery disease, CO is the mechanism blamed in the pathogenesis of both increasing platelet aggregation and causing vasospasm as a result of its direct effect on the coronary arteries. STEMI is the result of vasospasm[11].

Altay et al. reported a case with previous coronary artery disease in which CO exposure triggered the development of infarction. Çelik et al. also reported that a newly developed left bundle branch block was detected after CO poisoning in a patient known to have coronary artery disease[12,13].

Despite various clinical trials, the best treatment for carbon monoxide poisoning is still a matter of debate. In some patients, the use of 100% oxygen and/or hyperbaric oxygen is preferred according to the symptoms, accompanying diseases and COHb level[12,14]. Our patient was at risk of coronary artery disease because he was old, male and bedridden. We think that CO poisoning also increases cardiac injury. Detection of cardiac injury is among the indications for HBO therapy in CO poisoning patients[15]. However, HBO treatment was not applied since our patient was relieved clinically and symptomatically after angiography after he was admitted to the intensive care unit. He was discharged without any problems during his follow-ups.

Conclusion

Myocardial ischemia is an expected condition in CO poisoning, but STEMI is a rare condition. Even if the patients do not have chest pain or coronary artery disease in their history, ECG and cardiac enzyme follow-up should be performed in the first hour.

References

1. Reumuth G, Alharbi Z, Siamak K, Kim B, Siemers F, Christian P, et al. ScienceDirect Carbon monoxide intoxication: What we know. Burns 2018;6-10.
2. Afolayan JM, Edomwonyi NP, Esangbedo SE. Carbon monoxide poisoning in a Nigerian home: case reports. The Nigerian Postgraduate Medical Journal. 2014;21(2):199-202.
3. Jankowska D, Palabindala V, Salim SA. Non-ST elevation myocardial infarction secondary to carbon monoxide intoxication. Journal of Community Hospital Internal Medicine Perspectives 2017;7(2):130-3.
4. Omaye ST. Metabolic Modulation of Carbon Monoxide Toxicity. Toxicology 2002;180:139-50.
5. Tsai SH, Chu SJ, Hsu CW, Cheng SM, Yang SP. Use and and interpretation of cardiac troponins in the ED. Am J Emerg Med 2008;26;331–41.
6. Metin S, Yıldız Ş, Çakmak T. Demirbaş, Ş. The role of oxygen therapies in carbon monoxide poisoning. TAF Prev MedBull 2011;10:487–94
7. Peers C, Steele DS. Carbonmonoxide: a vital signalling molecule and potent toxin in the myocardium. J Mol Cell Cardiol 2012; 52: 359-65.
8. Satran D, Henry CR, Adkinson C, Nicholson CI, Bracha Y, Henry TD. Cardiovascular manifestations of moderate to severe carbon monoxide poisoning. J Am Coll Cardiol. 2005;45(9):1513-16
9. Dziewierz A, Ciszowski K, Gawlikowski T, Rakowski T, Kleczyński P, Surdacki A, et al. Primary Angioplasty in Patient with St-Segment Elevation Myocardial Infarction in the Setting of Intentional Carbon Monoxide Poisoning. J Emerg Med 2013; 45: 831-4.
10. Kim S, Lim JH, Kim Y, Oh S, Choi WG. A case of acute carbon monoxide poisoning resulting in an ST elevation myocardial infarction. Korean circulation journal 2012; 42: 133-5.
11. Lippi G, Rastelli G, Meschi T, Borghi L, Cervellin G. Pathophysiology, clinics, diagnosis and treatment of heart involvement in carbonmonoxide poisoning. Clin Biochem 2012; 45: 1278-85.
12. Altay S, Bozbay M, Dedemoğlu M, Ayhan E, Eren M, Çakmak HA. Acute myocardial infarction following carbon monoxide poisoning. Cerrahpasa J Med 2009; 40: 144-7.
13. Çelik T, Kurşaklıoğlu H, İyisoy A, Demirkol S, Yüksel Ç, Işık E. Acute myocardial infarction after carbon monoxide poisoning. Gulhane Med J 2005; 47: 67-6.
14. Van Meter KW. Carbon monoxide poisoning. In: Tintinalli JE, Kelen GD, Stapczynski JS editörs. Emergency Medicine: A Comprehensive Study Guide. 6th Edn. New York: McGraw-Hill; p.1238-42.
15. Drinhaus H, Nüsgen S, Hinkelbein J. Guidelines desirable for treatment of carbon monoxide poisoning. Anaesthesist. 2016;65(4):301-2.