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A rare cause of myocardial infarction: acute carbon monoxide poisoning

Miyokard infarktüsünün nadir bir sebebi: Akut karbon monoksit zehirlenmesi

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Introduction

Carbon monoxide (CO) poisoning is a very rare cause of myocardial infarction. Carbon monoxide has a higher affinity for hemoglobin than does oxygen. It attaches to the hemoglobin (Hb) and blocks the capacity to carry oxygen. It has been suggested that COHb causes myocardial infarction by severe generalized tissue hypoxia and a direct toxic effect on the myocardial mitochondria, in patients both with and without preexisting coronary artery disease (CAD). We report a case of 72 years old man who had myocardial infarction after exposure to CO poisoning.

Case report

A 72 years old man was admitted to our emergency department of Isparta State Hospital with altered consciousness as a consequence of acute domestic exposure to CO from stove poisoning. The diagnosis of CO poisoning was based upon history that neighbors gave. He was also suffering from an epigastric pain. There was a history of balloon dilatation and stenting of 70% stenotic lesion in the right coronary artery two months ago. There were also noncritical lesions in the left anterior descending artery (30%) and left circumflex artery (40%) at that time. He was asymptomatic since then.

On physical examination, the arterial blood pressure was 135/80 mmHg and the heart rate was 92 beats/min and irregularly irregular. Auscultation revealed tachyarrhythmia (irregularly irregular sounds) and grade 1/6 systolic murmur along left sternal border. Electrocardiography showed ST-segment elevation in inferior leads (Fig. 1, 2). The blood analysis revealed white blood count: 16000 K/uL, Hb: 10.6 g/dL, hematocrit: 32.1%, creatine kinase: 1305 U/L, creatine kinase-MB: 150 U/L and aspartate aminotransferase: 143 U/L. The arterial blood

gas analysis showed normal values. The serum carboxyhemoglobin level was not measured. The echocardiographic examination performed two months ago at the time of stenting documented presence of grade 2 mitral regurgitation, grade 2 aortic regurgitation and grade 2 tricuspid regurgitation. The patient was using aspirin, clopidogrel, bisoprolol, lisinopril, and atorvastatin. The oxygen treatment was started immediately. Intravenous nitrate was given in high doses as tolerated and ST-segment changes were checked to rule out coronary spasm. No ST-segment changes were observed. Intravenous streptokinase 1500000 IU was given without any complication. The control coronary angiogram showed 30% in-stent restenosis, 70% stenosis in 2nd obtuse marginal branch of circumflex artery and 70% stenosis of the left anterior descending artery.

Discussion

Carbon monoxide has a higher affinity for hemoglobin than does oxygen, it attaches to the hemoglobin of the red blood cells and blocks their capacity to carry oxygen. As a result, elevated blood concentration of CO leads to reduced tissue oxygen delivery. Cardiac toxicity may occur because of myocardial hypoxia or a direct toxic effect of CO on myocardial mitochondria. Such cardiac involvement may occur promptly after exposure, or it may be delayed for several days. Sinus tachycardia and various arrhythmias, including ventricular extrasystoles and atrial fibrillation are common without myocardial damage (1); bradycardia and atrioventricular block may occur in more severe cases. Angina pectoris may be precipitated in patients with and without underlying CAD (2). Myocardial infarction has been reported in patients with underlying CAD (3) as in our case. It has also been reported in patients without preexisting CAD (4-6). It has been assumed that COHb causes myocardial infarction by severe generalized tissue

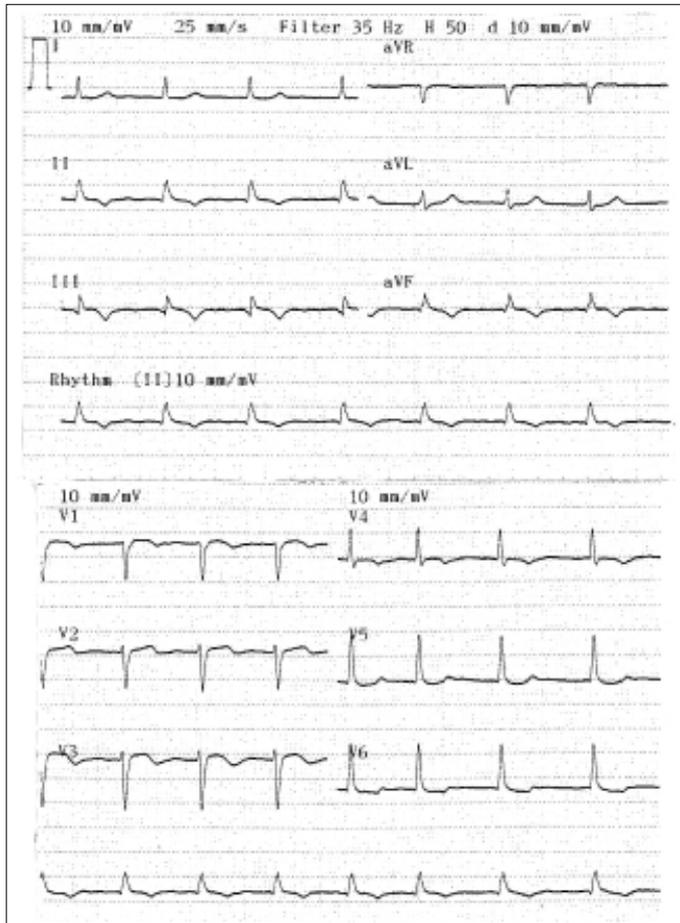


Figure 1. Electrocardiogram before CO poisoning

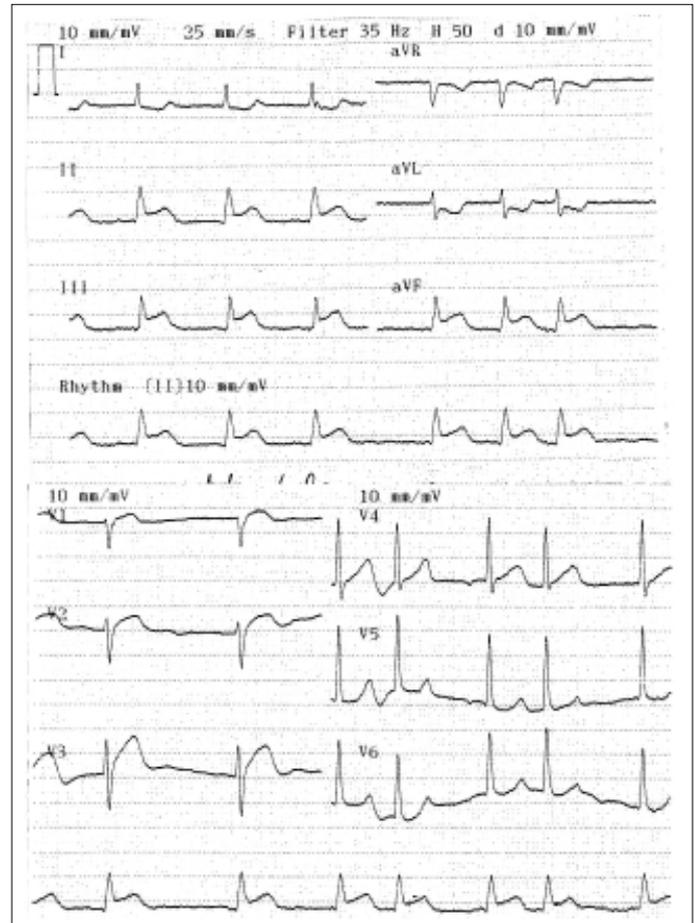


Figure 2. Electrocardiogram at the time of CO poisoning

hypoxia and a direct toxic effect on the myocardial mitochondria (4). Factors such as inadequate myocardial perfusion and increased thrombotic tendency might also contribute to decreased myocardial oxygenation (4). Coronary spasm has also been accused (6). ST-segment and T wave abnormalities are common and transient ventricular dysfunction may occur. Administration of 100% oxygen usually results in rapid recovery. Treatment in a hyperbaric oxygen chamber is also suggested for complete recovery (7).

Interestingly our patient had a history of balloon dilatation and stenting of the right coronary artery two months ago. The effect of acute CO exposure in CAD patients with a history of recent stenting is not known. The patients who underwent balloon dilatation and stenting may be more prone to myocardial ischemia or myocardial infarction in the setting of CO poisoning. The control coronary angiogram one week later showed 30% in stent restenosis and an increase in degree of lesions in circumflex artery and left anterior descending artery. If we had performed coronary angiogram in acute phase of myocardial infarction for primary percutaneous intervention, we would have shown the infarct related artery and state of the stent. Intravenous nitrate was given in high doses as tolerated and ST-segment changes were checked to rule out coronary spasm. No ST-segment changes were observed. Coronary vasospasm, thought it cannot be the major cause of myocardial infarction, but may be a contributing factor. It has been reported that CO can increase thrombotic tendency (4) and can cause thrombus formation in coronary arteries and in heart chambers (8). It has been supposed that acute thrombosis, which is triggered by CO might be the cause of myocardial infarction in patients with underlying CAD in the basis of generalized tissue hypoxia even in the use of aspirin and clopidogrel.

As a result, we consider that careful electrocardiographic and enzymatic monitoring of all patients in the first hours after CO exposure is important for early diagnosis and treatment of this rare complication of CO poisoning especially in preexisting CAD with stenting. This can improve the outcome of these patients, because the typical chest pain may be absent.

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