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Very Late Stent Thrombosis in a Patient Presenting with Acute Carbon Monoxide Poisoning

Selçuk Öztürk[1], Selim Ayhan[2], İbrahim Dönmez[2], Fatma Erdem[2], Alim Erdem[2]

[1]Department of Cardiology, Ankara Research and Training Hospital, Ankara, Turkey
[2]Department of Cardiology, Abant İzzet Baysal University İzzet Baysal Research and Training Hospital, Bolu, Turkey

ABSTRACT

Introduction: Carbon monoxide (CO), which is commonly referred to as the silent killer, can cause deleterious and unwanted cardiac effects. Some of these are arrhythmias, acute myocardial infarction (AMI), cardiogenic shock, heart failure, and pulmonary edema.

Case Report: A 50-year-old man, complaining of dyspnea and chest pain, presented to the emergency room approximately half an hour after exposure to fire smoke. He had a history of anterior myocardial infarction 3 years previously and had been treated with a tacrolimus-eluting stent at that time. On admission, electrocardiography showed ST segment elevation in the leads D1, aVL, and V1–3, and ST segment depression in the reciprocal leads. The patient was transferred to the cardiac catheterization laboratory, and coronary angiography revealed stent thrombosis in the proximal part of the left anterior descending artery, causing 100% occlusion. The patient underwent successful balloon angioplasty and stenting. For the treatment of acute CO poisoning, he was administered oxygen. The patient's condition continued to be stable, and he was discharged from the coronary intensive care unit.

Conclusion: Patients who have undergone coronary stenting, particularly those in whom drug-eluting stents have been implanted, should be carefully investigated for AMI, stent thrombosis, or any other cardiac complications in the emergency room after CO poisoning.

Keywords: Carbon monoxide poisoning, anterior wall myocardial infarction, drug-eluting stent

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Introduction

Carbon monoxide (CO) is known to be an inodorous, colorless, and non-irritating gas. It is commonly known as the silent killer. The toxicity of CO has been known for years, and it is one of the main causes of unintentional poisoning deaths in European countries. The main pathophysiological mechanism is dependent on the capability of CO to bind to hemoglobin with a high affinity, which displaces oxygen and forms carboxyhemoglobin (COHb), in which oxygen cannot be delivered to the cells. The brain and the heart, which are more prone to injury, need more oxygen than the other organs; therefore, neurological and cardiovascular manifestations are the most common results of acute CO poisoning. The cardiac effects of CO poisoning include angina, arrhythmia, cardiomyopathy, cardiogenic shock, and even sudden death (1). Acute myocardial infarction (AMI) secondary to CO poisoning is frequently reported in the literature; however, there are no published data of very late thrombosis of tacrolimus drug-eluting stents with acute CO poisoning (2, 3). Here we describe a case of very late stent thrombosis in a patient with CO poisoning.

Case Report

A 50-year-old man presented to the emergency room approximately half an hour after exposure to fire smoke. On admission, he complained of dyspnea and retrosternal chest pain. Physical examination revealed a heart rate of 90 beats/min, blood pressure of 132/89 mmHg, bilateral rales (crackles) in the lower lung zones, and S1 and S2 with a regular rhythm and without murmurs, S3, or S4. His arterial blood gas analysis results were consistent with metabolic acidosis. Electrocardiography (ECG) showed ST segment elevation in leads D1, aVL, and V1–3 and ST segment depression in the reciprocal leads (Figure 1). Initial labora-

Address for Correspondence:
Selçuk Öztürk, Department of Cardiology, Ankara Research and Training Hospital, Ankara, Turkey
E-mail: selcukozturk85@hotmail.com

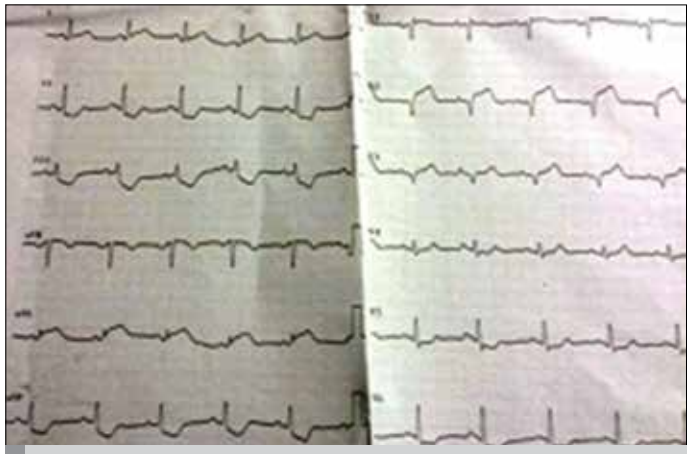

FIGURE 1. Electrocardiogram of the patient showing ST elevation in D1, aVL, and V1–3 and ST depression in the reciprocal leads on admission

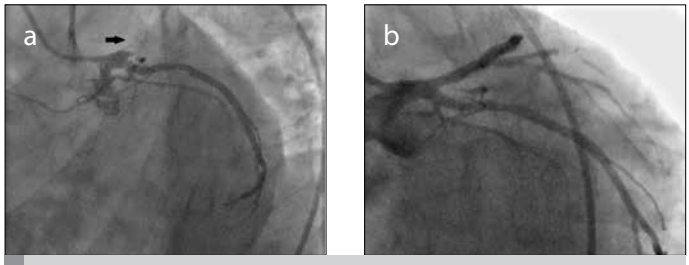

FIGURE 2. a, b. (a) Left coronary angiography demonstrating total occlusion of the proximal part of the left anterior descending artery, with stent thrombosis (black arrow). (b) Left coronary angiography after intervention demonstrating restored thrombolysis in myocardial infarction-3 flow after angioplasty with stenting

tory analyses revealed the following: troponin I level 0.122 ng/mL (normal range: 0-0.06 ng/mL) and creatine kinase-MB level 63.3 U/L (normal range: 0-24 U/L). The COHb level was 20.7%. The remaining blood counts and biochemistry results were within normal ranges. It was learned from his history that the patient had presented with an anterior AMI 3 years previously and had undergone successful percutaneous intervention with a single tacrolimus-eluting stent (3×19 mm) in the proximal part of the left anterior descending artery (LAD), which was found to be an optimal stenting implantation from the hospital records. At that time, he had been discharged from the hospital with dual-antiplatelet therapy consisting of oral administration of clopidogrel 75 mg and acetylsalicylic acid 100 mg, but clopidogrel had been stopped 1 year later. In addition, he had been prescribed atorvastatin, metoprolol, and ramipril. Three months after stenting, he presented to the hospital with chest pain and had been hospitalized with the diagnosis of unstable angina pectoris. Angiography revealed non-critical lesions and no stent re-stenosis at that time. He had been followed up as stable at the cardiology polyclinic since then. The patient received acetylsalicylic acid 300 mg and 600 mg clopidogrel orally and 5000 U unfractionated heparin intravenously in the emergency room. For the treatment of acute CO poisoning, he was administered oxygen with a FiO2 of 100% via a non-rebreather mask. The patient was transferred to the cardiac catheterization laboratory, where coronary angiography revealed stent thrombosis in the proximal LAD, causing 100% occlusion (Figure 2a). He underwent successful balloon angioplasty (3×20 mm) and stenting with a zotarolimus-eluting stent (3×30 mm) (Figure 2b). He was then transferred to the coronary intensive care unit, and medical treatment was continued, including aspirin, clopidogrel, atorvastatin, metoprolol, ramipril, and oxygen therapy via a non-rebreather mask. After stenting, ECG showed resolution of the ST segment elevation and revealed that the patient was symptom-free. The echocardiogram demonstrated septal and anterior wall hypokinesia of the left ventricle, with an ejection fraction of 45%. Follow-up laboratory tests also revealed a peak in the cardiac enzymes (troponin I>25 ng/mL; creatine kinase-MB: 135.4 U/L). The patient's condition continued as asymptomatic and hemodynamically stable, and he was discharged after 4 days. Written informed consent was obtained from the patient.

Discussion

To the best of our knowledge, there are no published data regarding very late thrombosis of a tacrolimus drug-eluting stent in the setting of acute CO poisoning. It is known that CO can cause neurological and cardiovascular complications. Numerous harmful cardiac effects of CO poisoning have been published in the literature, including angina, myocardial injury, arrhythmia, cardiomyopathy, cardiogenic shock, and even sudden death (1), but there is not much evidence about very late stent thrombosis.

Different mechanisms have been demonstrated concerning CO causing myocardial ischemia. CO attaches to hemoglobin with great affinity. This causes COHb formation and impairments in the oxygen transport system, which lead to tissue hypoxia (1, 4). Furthermore, CO also binds to the proteins myoglobin and cytochrome-c oxidase, which are found in the cardiac and skeletal muscles. This situation interrupts muscle oxygen transport and causes consequent hypoxia (3). If CO binds to cytochrome-c oxidase, it results in contractile dysfunction of the myocardial cells (5, 6).

Hypoxic injury, neurological damage, and possible death can occur after inhaling even relatively small amounts of CO, particularly in patients with high cardiovascular risk. Toxicity also increases, depending on the previous history of cerebral or cardiovascular diseases and of conditions that lead to high metabolic rates (7). Our case had a history of stent implantation due to AMI 3 years ago and of a reduced ejection fraction.

CO has prothrombotic potential and may trigger thrombus formation in the arterial and venous systems. CO binds to fibrinogen-bound heme and this situation affects the coagulation cascade. Secondly, an increased aggregation of platelets and polycythemia after CO poisoning can cause thrombus formation. The increase in viscosity, polycythemia, and thrombogenicity can be the cause of myocardial injury, even in the presence of normal coronary arteries (8, 9). In our case, we cannot suggest that the formation of stent thrombosis is due to CO poisoning, but we can speculate that the combination of these events may be related. Dileo et al. reported a 50-year-old patient presenting with ST segment elevated AMI and late stent thrombosis (sirolimus drug-eluting stent) in the setting of CO poisoning. They treated the patient successfully with balloon angioplasty (2). In another case report, Hsu et al. described a patient

presenting to their clinic with AMI secondary to CO poisoning. The patient did not have coronary artery disease previously, and they treated the patient successfully with balloon angioplasty (3).

Conclusion

CO poisoning can cause multiple cardiac complications, and stent thrombosis is one of them, although it is rarely mentioned in the literature. Thus, a history of previous cardiac events may facilitate these incidents. However, cardiac complications may occur in the absence of a cardiac disease history. There are different mechanisms through which CO poisoning may be linked to myocardial ischemia, and we can speculate that thrombus aggregation in the myocardial arteries is the most important one. This report describes a very rare case of CO poisoning with very late thrombosis of a tacrolimus drug-eluting stent. In particular, patients who have undergone coronary stenting, particularly those in whom drug-eluting stents have been implanted, should be carefully investigated for AMI, stent thrombosis, or any other cardiac complications in the emergency room after CO poisoning.

Informed Consent: Written informed consent was obtained from patient who participated in this study.

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