Multivessel Coronary Artery Thrombosis Accompanying Acute Myocardial Infarction: A Case Report

Abdulmecit Afşin 1, Yılmaz Ömür Otlu 2, Mahmut Yılmaz 1, Zeynep Ulutaş 1, Nusret Açıklöz 1

1 İnönü University, Faculty of Medicine, Department of Cardiology, Malatya, Turkey
2 Kars State Hospital, Department of Cardiology, Kars, Turkey

Abstract
Acute ST-segment elevation myocardial infarction (STEMI) complicated with multiple coronary artery thrombosis is a rare clinical situation and is associated with poor prognosis. A 63-year-old male patient was admitted to the emergency department with sudden chest pain that had started three hours ago. The patient was diagnosed with acute inferior STEMI. The emergency coronary angiography showed acute arterial thrombosis in the left anterior descending (LAD), left circumflex (LCx), and the left first branch of the circumflex artery (obtuse margin). Obtuse margin of the lesion was treated with a stent. We decided to infuse tirofiban (glycoprotein 2b/3a inhibition) for the thrombosis in the LAD and CX vessels. After the tirofiban infusion, the coronary angiography showed no thrombi in the coronary arteries. The patient was discharged without further complications.

Key Words: Myocardial Infarction; Coronary Artery Disease; Multivessel Coronary Thrombosis.

Coronary angioplasty and other interventional techniques are widely used in the acute treatment of coronary thrombus. Concurrent multiple coronary artery occlusion is a special type of coronary artery diseases. In this report, we aim to present the case of a patient who underwent coronary angiography in our clinic with a diagnosis of acute myocardial infarction due to total occlusion in the left anterior descending (LAD), left circumflex (LCx), and obtuse margin (OM) arteries.

INTRODUCTION
Coronary thrombus formation is the most important etiological cause for acute myocardial infarction and unstable angina. Obstruction of different coronary arteries by concomitant thrombus is a rare condition (1).

As of now, the mechanism behind simultaneously occluded multiple coronary arteries is not clear. Factors such as hypertension, diabetes, smoking, essential thrombocytosis, coronary spasm, hypercoagulation, and cocaine use have been blamed as the reasons behind this condition (1, 2).

CASE REPORT
A 63-year-old male patient was admitted to the emergency department with chest pain accompanied by sudden tightening that had started 3 hours ago. The patient had a history of smoking 1 pack a day for 20 years. The physical examination did not show any significant signs. ECG revealed that he had normal sinus rhythm, ST elevation in D2, D3 and aVF derivations, and ST depression in V1, V2, and V3 precordial leads (Figure 1).

Figure 1. Patient’s ECG on admission.
The patient was taken to the catheterization laboratory for primary percutaneous coronary intervention. Through the right femoral artery, we performed coronary angiography with standard Judkins technique. The coronary angiography showed that the distal portion of the LAD and LCx, the first branch of the LCx artery, and the OM ostial area were entirely occluded (Figure 2A).

Throughout the same session, we passed a 0.014” guide wire through the lesion in Cx-OM 1. After performing predilatation with a 2,0mm x 20mm Sprinter Legend balloon (Medtronic, Minneapolis, MN, USA), we implanted a 2,5mm x 18mm Integrity bare metal stent (Medtronic) and obtained the TIMI-3 flow (Figure 2B). We then added 0,15 micro gram/kg/mins of tirofiban (glycoprotein 2b/3a inhibitor) infusion to the patient’s medical treatment schedule for 24 hours.

The coronary angiogram following the 24-hour tirofiban infusion revealed that LAD, LCx, and LCx OM-1 coronary arteries were unobstructed (Figure 2C). The white blood cell count at the complete blood count was 9200/mm3 while hemoglobin level was 11,5 g/dL and platelet count was 210,000/mm3, respectively. The prothrombin, factor 5 Leiden, lupus anticoagulant, antithrombin 3, protein C, and protein S values were normal. The ejection fraction was 50%, the lateral wall and inferior wall were hypokinetic in the echocardiography. There were no thrombi in the left ventricle or left atrium. Having observed no complications and regulated his medical treatment during the follow-ups, we discharged the patient on the 6th day of his hospitalisation.

**Figure 2.** Total occlusion in the LAD, LCx, and OM (A); Total occlusion in the distal of the LAD and LCx. TIMI-3 flow in the LCx –OM artery following PTCA (B); TIMI-3 flow in the coronary arteries after the tirofiban infusion (C).

**DISCUSSION**

Acute thrombus was present in more than one coronary artery in our patient. Simultaneous occlusion in multiple coronary arteries is very uncommon in acute myocardial infarction and there are only less than 30 such cases that have been published in the literature (1). Simultaneous occlusion of two or more coronary arteries is associated with poor prognosis and high mortality rates. In this respect, there are studies that report patients presenting with acute cardiogenic shocks (3). Studies conducted with angiography and intravascular ultrasonography have reported that multi plaque rupture is a common phenomenon in acute coronary syndromes and that it can take place in different coronary arteries (4). Another study asserts that acute coronary syndrome in various vessels due to coronary flow disruption caused by the initial plaque rupture is one of the reasons of acute thrombus in multiple coronary arteries (1). In our patient, we observed three simultaneous total occlusion in three coronary arteries through there was no clinical sign of a cardiogenic shock. The coronary angiography performed in emergency conditions showed total occlusion in the distal of the LAD, distal portion of LCx, and in CX OM -1 ostium. In line with the ECG results of our patient, we considered the possibility of a lesion in the CX OM and inserted a guided wire; the wiring was easy since we could penetrate the lesion without any difficulty and then we performed a percutaneous coronary intervention to the thrombosed lesions. We decided to perform a tirofiban infusion for the lesions in the distal of LAD and LCx. After the tirofiban infusion, we observed TIMI-3 flow in the LAD and LCx coronary arteries; there were no significant thrombi or plaques after the application. In addition, the coronary angiography showed that there was no collateral flow from the right coronary artery towards the LAD and LCx which made us think that, rather than the emergence of a chronic occlusion, the patient had an acute occlusion. A recent study proposes that acute coronary syndrome patients presenting with stable conditions along with intracoronary thrombus should be evaluated for percutaneous coronary intervention after glycoprotein 2b/3a inhibitor infusion (5). We could not detect any of the above mentioned etiological factors such as cocaine use, hypercoagulation, essential thrombocytosis, or rheumatic diseases in our patient. But our patient had old age and a smoking history as the risk factors for coronary artery diseases. The significant risk factor in patients who experience acute myocardial infarction with multiple coronary thrombus is smoking (1). It is known that smoking leads to plaque rupture by causing vasospasm in the coronary arteries and stiffness in the arterial walls. It is also commonly agreed that smoking increases thromboxane-A2 emission in coronary arteries whereby leading to vasospasm and platelet aggregation and resulting in platelet aggregation and coronary occlusion (6).
Multiple epicardial coronary thrombosis is a rare but a critical clinical condition in STEMI patients. It requires early diagnosis and treatment. A great number of factors have been implicated in its etiology. In our opinion, the cause of multiple coronary artery thrombosis was smoking in the case of our patient.

REFERENCES