

Case report

Double trouble with acute inferior myocardial infarction and left anterior descending artery thrombi☆



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ABSTRACT

Concomitant occlusion of multiple epicardial coronary arteries is an uncommon finding in patients presenting with ST-segment elevation myocardial infarction. We reported a 50 years-old male patient who presented with inferior myocardial infarction. Coronary angiography revealed simultaneous occlusion of left anterior descending (LAD) and right coronary artery (RCA). The RCA occlusion treated successfully with percutaneous coronary intervention while LAD occlusion treated with antithrombotic and glycoprotein 2B/3A administration. Although multiple coronary thromboses of coronary arteries in the course of myocardial infarction is uncommon, it is crucial to determine proper treatment for these patients.

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Introduction

Acute myocardial infarction (AMI) is caused by disruption of atheromatous plaques and thrombus formation followed by partial or complete occlusion of the epicardial coronary arteries. Multiple ruptured plaques with thrombus formation have been shown in autopsies of >10% of individuals with atherosclerosis who died due to non-cardiac etiologies. Conversely, thrombi are frequently observed at sites other than those of the major culprit lesions in patients died subsequent to acute coronary syndromes.¹ Autopsy series showed thrombotic coronary occlusion of >1 major epicardial artery can be as common as 50% in patients suffering from sudden cardiac death following AMI, this phenomenon is only seen 2.5% of AMI patients undergoing primary percutaneous coronary intervention (PPCI).^{2,3}

Case report

A 47 years old man with a previous history of smoking, acute coronary syndrome and percutaneous coronary intervention admitted to our emergency unit with sternal pain for 2 h. He was hemodynamically stable, his vital signs showed a regular heart rate of 100 beats/min, blood pressure of 131/90 mm Hg and no respiratory distress. Cardiac

auscultation revealed decrease in the intensity of first heart sound. The electrocardiography on arrival revealed sinus rhythm and ST-segment elevation in leads II, III, and aVF (Fig. 1A). Patient received anti-thrombotic medication prior to primary percutaneous coronary intervention. Coronary angiography showed total occlusion of right coronary artery (RCA) and subtotal occlusion of left anterior descending artery (LAD) with a fresh thrombus (TIMI thrombus grade 4). The RCA lesion was crossed using guidewire and subsequent predilatation performed with 2.0 × 15 mm compliant balloon (Invader PTCA, Alvimedica, Assen, Netherlands). This was followed by 3.5 × 32 mm bare metal stent (BMS) (Ephos, Nemed Lxtd, Istanbul, Turkey) implantation and achieved good angiographic result with TIMI-3 flow (Fig. 2). Although the patient had TIMI-3 flow in LAD, following the engagement of left main coronary artery by left Judkins guiding catheter, intracoronary tirofiban bolus dose (Irokocardio International, SARL, Geneva) was administered according to the patient's body weight. The LAD patency continued with a TIMI-3 flow and good myocardial blushing (Fig. 3A). Tirofiban maintenance dose completed over 24 h in the intensive cardiac care unit (ICCU). Addition to tirofiban infusion patient was administered antithrombotic and low molecular weight heparin. Patient had no angina during the ICCU follow-up and ECG showed no changes other than T wave inversion in leads DII, DIII and aVF (Fig. 1B). Patient had no history of hypercoagulopathy states like malignancy or essential thrombocytosis. He also had no history of using amphetamine, cocaine or homeopathic/herbal drugs recently. Transthoracic echocardiography revealed mild hypokinesia in inferior segments with a left ventricular ejection fraction of 45% and mild degree of mitral regurgitation. Following 24th hours, control coronary angiography showed reduced coronary thrombus burden (Fig. 3B). After 72 h, another coronary angiography

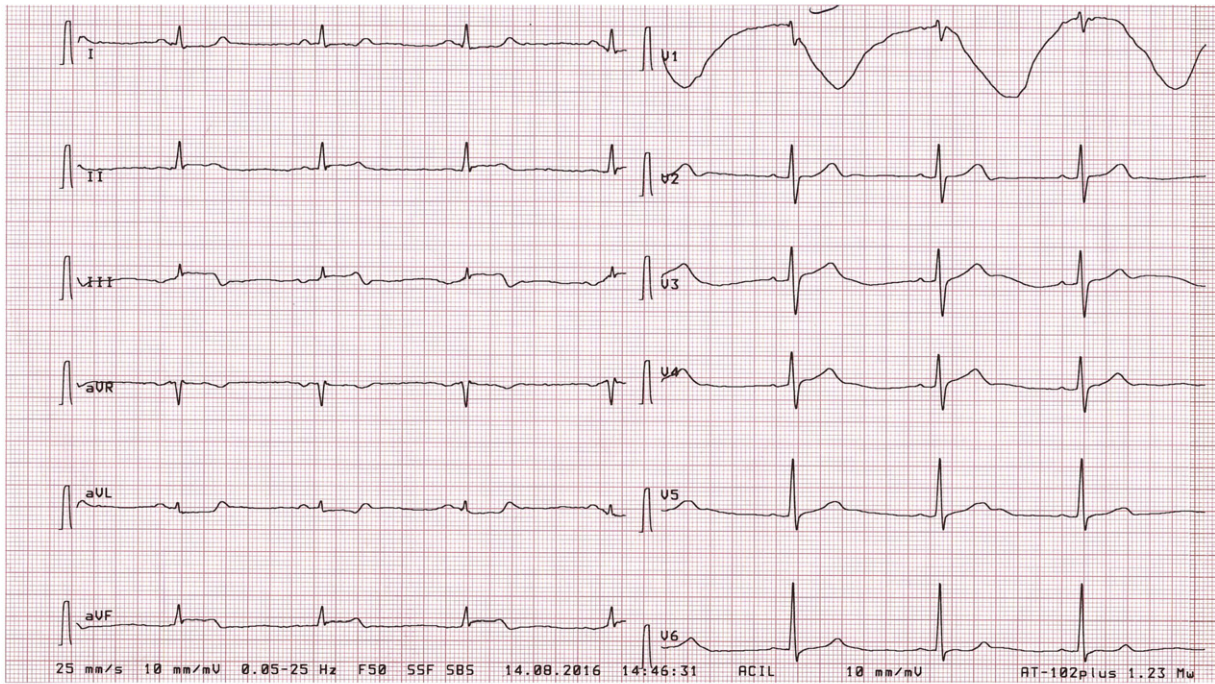
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A



B

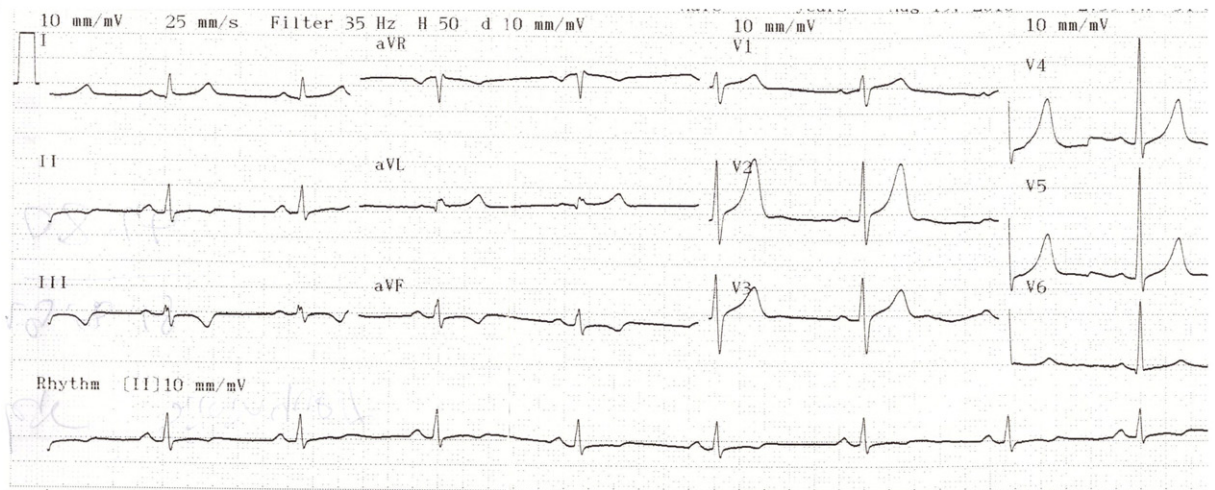


Fig. 1. A: Admission electrocardiography of the patient with ST elevation in DII, DIII and aVF leads and ST depression in DI and aVL. B: Control electrocardiography with negative T waves in DII, DIII and aVF.



Fig. 2. Right coronary artery before and after primary percutaneous coronary intervention.

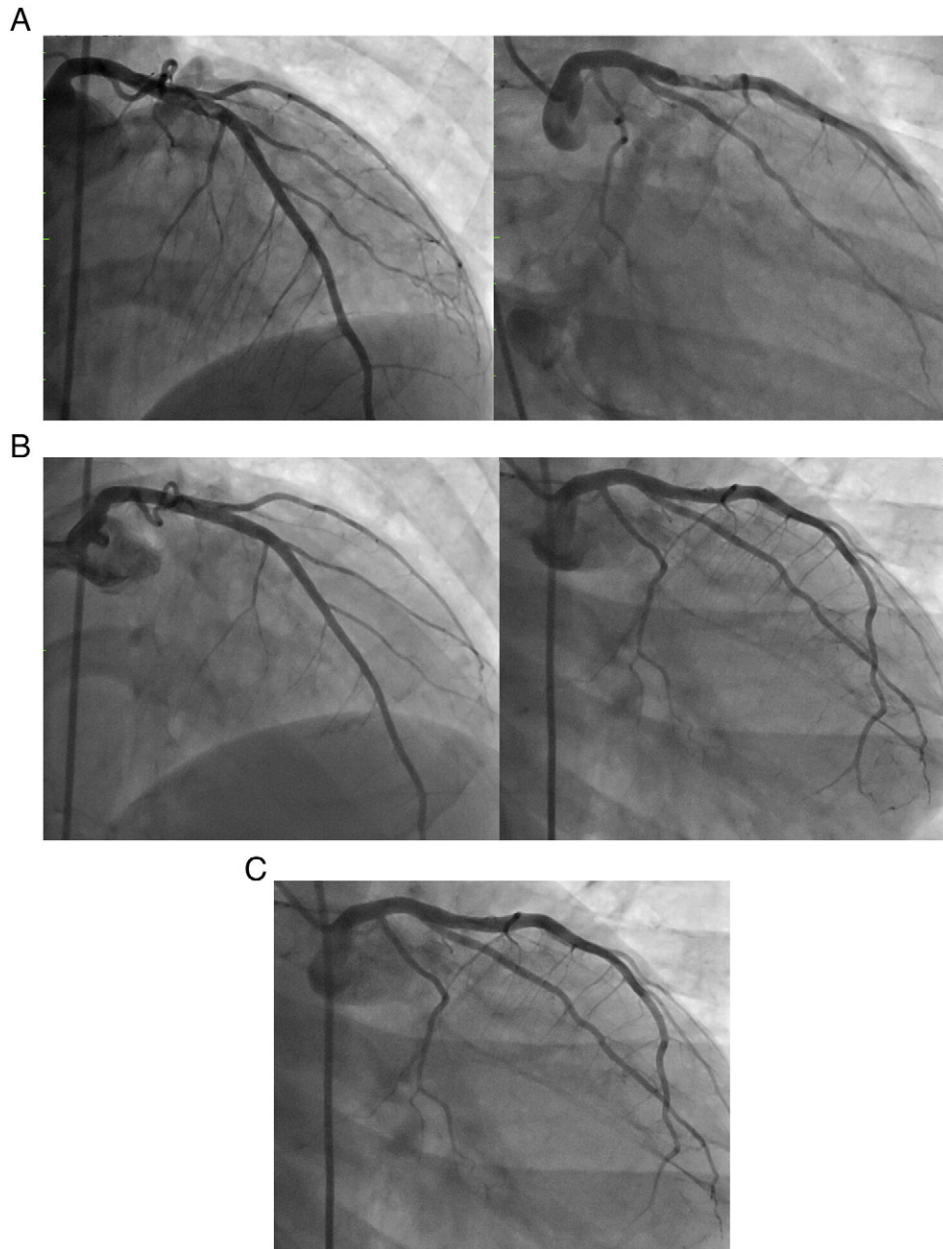


Fig. 3. A: A large thrombus in the proximal left anterior descending artery during primary percutaneous coronary intervention. B: Disappearance of thrombus in the left anterior descending artery in control coronary angiogram.

performed to evaluate coronary thrombus on LAD and no thrombus was observed (Fig. 3C). Patient recovered in a short period and discharged with no symptoms.

Discussion

In this case report, we presented a patient presented with concomitant occlusion of LAD and RCA resulting in acute inferior myocardial infarction. Glycoprotein 2B/3A infusion following primary PCI for RCA resulted in excellent recovery.

Loss of integrity in coronary atheromatous plaques with a subsequent thrombotic occlusion is the main pathological finding in AMI. Coronary artery stenosis in multiple coronary arteries may be commonly seen in patients undergoing PPCI. However, AMI with more than one epicardial coronary artery thrombosis is an infrequent clinical and angiographic finding. The pathophysiology of multiple coronary artery

thrombosis is unclear. Existence of additional unstable plaque formations other than culprit lesion has been shown in 21% of patients with AMI.⁴ Therefore, widespread plaque instability in all coronary arteries can predispose to multiple coronary thromboses. Previous case reports also suggested that cocaine use, hypercoagulable state and essential thrombocytosis⁵ can be held responsible for multiple coronary thromboses. The etiology of multivessel thrombosis should be evaluated with further studies.

Multiple coronary thromboses are usually reported in patients with fatal arrhythmias, acute pulmonary edema and cardiogenic shock. In our case, patient was normotensive with no additional clinical symptoms, probably due to distal TIMI flow of LAD. The incidence of double-vessel coronary thrombosis may account 1.7–4.8% of all PPCI patients. According to the review by Kanei, et al., patients with multiple coronary thromboses had common risk factors including essential thrombocytosis, intravenous cocaine abuse, diabetes mellitus and

smoking. LAD and RCA was involved in 78% and 87% of patients⁶ respectively. In our patient the only risk factor was smoking and thrombotic lesions were in both LAD and RCA.

The optimal management strategy in patients with large coronary thrombus is challenging. The presence of intracoronary thrombus may lead to macro and micro distal embolizations, which causes reduced benefit of PPCI.⁷ Pharmacological agents like glycoprotein 2B/3A inhibitors used as an upstream therapy adjunctive to PPCI to reduce thrombus burden and improve microvascular flow.⁸ In recently published EROSION study, 60 patients with the diagnosis of acute coronary syndrome whose coronary angiography revealed stenosis less than 70% were treated with antithrombotic agents without stenting.⁹ Patients were treated with antithrombotic agents following detection of plaque erosion by OCT. One third of the patients had no thrombus following antithrombotic treatment. Authors concluded that this could be a treatment option for acute coronary syndrome patients.

Conclusion

Simultaneous thrombosis of coronary arteries in the course of AMI is an uncommon entity that is usually clinically accompanied by cardiogenic shock. Management of these patients should be individualized

and the invasive approach should not be considered as the only treatment option.

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