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LETTER TO THE EDITOR

Double Infarction after Acute Multivessel Coronary Artery Occlusion

KEYWORDS
myocardial infarction;
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1. Introduction

Rupture or erosion of a coronary vulnerable plaque followed by acute thrombosis is the most common cause of ST-segment elevation acute myocardial infarction (STEMI). Multiple intrinsic and extrinsic factors can cause plaque instability.[1] In most cases, a single culprit lesion is recognized and treated, while simultaneous multivessel occlusions that provoke an infarction in two different segments of the left ventricle are relatively uncommon.[2] It is unclear whether multiple simultaneous occlusions of coronary arteries represent a special subtype of coronary artery disease or nothing more than an accidental finding.[3] We describe a case of a 56-year-old female who was admitted to our hospital due to anterior STEMI and who suffered an inferior infarction 90 minutes after admission.

2. Case

A 56-year-old female with a past medical history of dyslipidemia and hyperthyroidism under medical treatment, presented to our emergency department with persistent severe retrosternal chest pain that had started 1 hour before presentation. She had no family history of coronary artery disease (CAD) or other risk factors associated with CAD.

The physical examination revealed no signs of acute heart failure, a respiratory rate of 18/min, a regular heart rate of 54/min and blood pressure of 110/70 mm Hg. Her jugular veins were not distended, and her lung fields were clear on auscultation. Her oxygen saturation was 97% on ambient air. Her heart auscultation revealed an S4 along with a mild systolic murmur (1+/6).

Her initial ECG revealed sinus rhythm along with ST-segment elevation in the anterior leads (V1-4, Figure 1), indicating an acute anterior myocardial infarction. She was immediately admitted to the cardiac care unit after receiving per os aspirin (500 mgr), per os clopidogrel (300 mgr) and IV enoxaparin (60 mg/0.6 ml). The patient was subjected to thrombolysis with reteplase in the cardiac care unit and reported no chest pain 60 minutes after its administration. Ninety minutes after the administration of reteplase, the patient reported new onset of retrosternal chest pain, along with ST-segment normalization in the anterior leads, new-onset ST-segment elevation in the inferior leads (II, III, aVF) and reciprocal ST-segment depression in leads I and aVL, indicative of an inferior STEMI (Figure 2). Her clinical condition improved during the following hour and her ECG at 180 minutes after thrombolysis was normal.

A coronary angiography performed 15 hours later revealed a 70–80% stenosis of the proximal left anterior descending (LAD) artery (Figure 3A), along with severe proximal stenosis followed by a total occlusion located at the middle segment of the right coronary artery (RCA) (Figure 4A). The patient underwent angioplasty of the right coronary artery and the successful placement of two DES stents (Promus Element 3.0×20 mm, 2.75×38 mm) (Figure 4B).

Echocardiography of the patient's heart showed an ejection fraction of 40% with moderate hypokinesia of the inferior wall and mild hypokinesia of the middle and apical anterior wall of the left ventricle. To assess the severity of LAD stenosis along with the possible presence of viable hibernating or stunned myocardium in the anterior wall of the left ventricle, the patient underwent a stress echo[4] 4 weeks after discharge from the hospital. The stress echo exhibited a biphasic response of the anterior wall, a finding indicative of hibernating myocardium. As a result, the decision to perform a coronary angioplasty at the site of the

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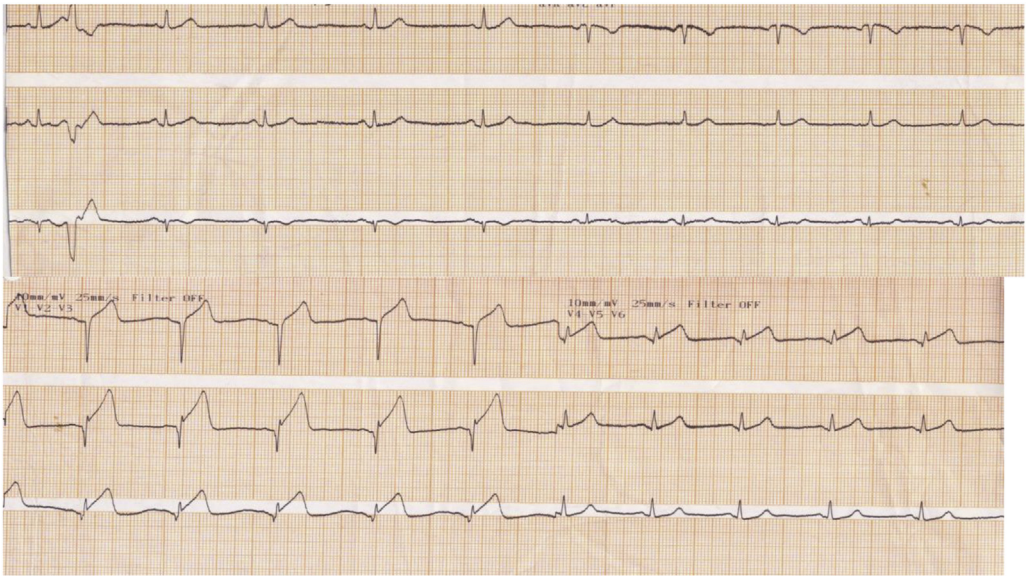

Figure 1 Emergency department ECG.

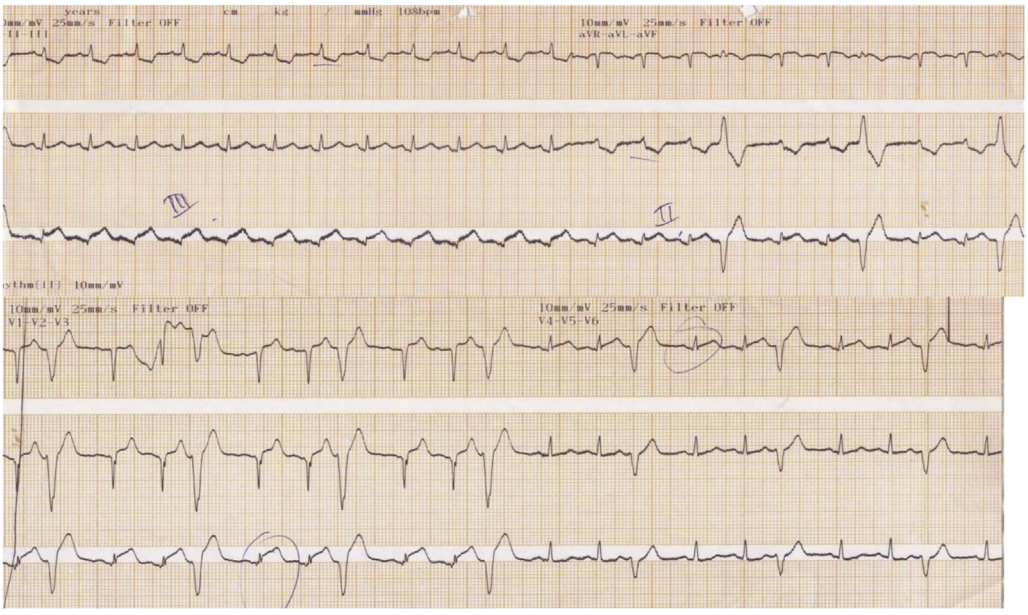

Figure 2 ECG performed 90 minutes after thrombolysis.

previously known proximal LAD stenosis was made. The patient had a successful LAD angioplasty and DES stent deployment (Promus 3.0×16 mm) (Figure 3B). An echocardiogram during her routine follow-up after 3 months revealed a normal ejection fraction (50%) with mild hypokinesia of the inferior wall.

3. Discussion

The exact mechanism causing occlusion in more than one of the coronary arteries is unclear. Multiple factors, both intrinsic and extrinsic, can cause plaque instability, rupture or erosion, leading to acute thrombosis and artery occlusion. Destabilization of multiple atherosclerotic plaques in addition to the one responsible for the acute infarction has been demonstrated in studies, and multiple ruptures in different coronary arteries have been detected in acute coronary syndromes by using intracoronary angioscopy or ultrasonography and multidetector computed tomography.[5,6]

Autopsies of patients who died from acute myocardial infarction show that simultaneous thrombotic occlusion in two or more coronary arteries is not rare, occurring in up to 25-50% of patients.[7] In other cases, however, especially in patients who underwent primary PCI, the same phenomenon has been found in approximately 1.3% to 2.5%.[3,8] This discrepancy is likely due to selection bias; patients with STEMIs and multiple culprit arteries are more likely to

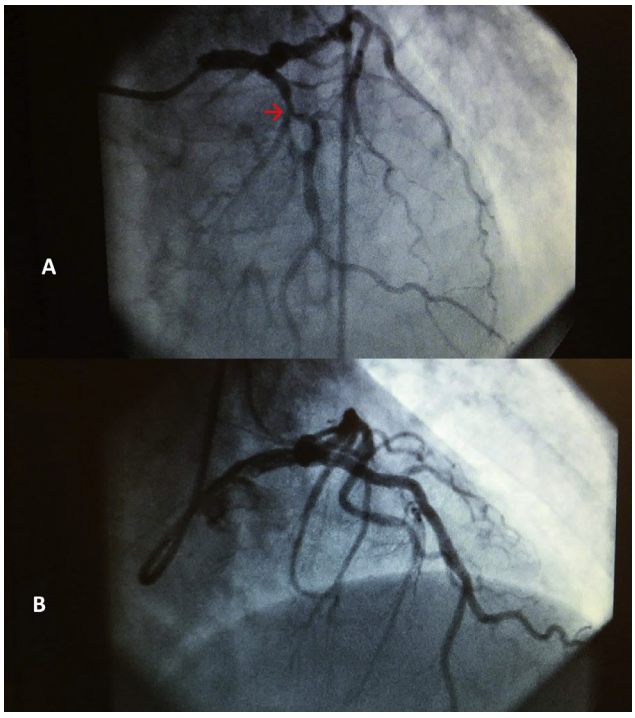

Figure 3 Proximal stenosis of the LAD artery (red arrow) before (A) (LAO-cranial view) and after stenting (B) (RAO cranial view).

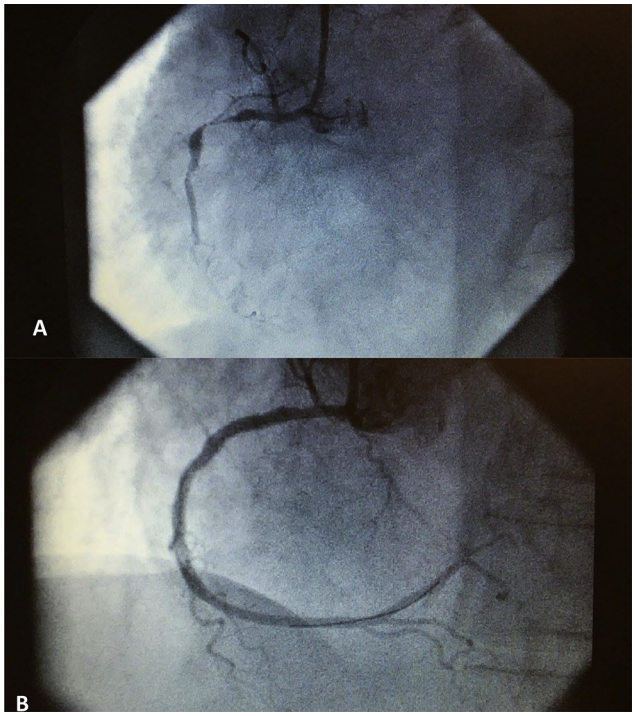

Figure 4 Severe proximal stenosis and total occlusion (mid section) of the RCA before (A) (LAO view) and after stenting (B) (LAO cranial view).

present with sudden cardiac death[9] and not survive long enough to undergo coronary angiography. Approximately 1/3 of patients presented with cardiogenic shock, and nearly 25% of patients suffered life-threatening arrhythmias or required intra-aortic balloon pumps.[3] Goldstein et al.[10] demonstrated that more than 30% of patients with acute myocardial infarction had angiographically detectable thrombi-overlying plaques in nonculprit coronary arteries, an observation associated with adverse clinical outcomes.

Although the causative factors involved in the acute and simultaneous thrombosis of multiple coronary arteries are unclear, possible contributing factors include the following: 1. heightened inflammatory response and catecholamine surge caused by the acute occlusion of one vessel, resulting in a second coronary artery occlusion, 2. hemodynamic instability and hypotension due to the occlusion of a coronary artery, resulting in blood stasis and acute occlusion in another artery with a severe underlying lesion, 3. hypercoagulable state due to malignancy and/or thrombocytosis, 4. coronary embolism and 5. prolonged coronary vasospasm (from Prinzmetal angina or cocaine use).[3]

In a case series of 47 patients with multiple culprit arteries, 19 (40%) had co-morbidities that were potential contributing factors, including a history of cancer, human immunodeficiency virus, cocaine use, coronary artery vasospasm, platelet abnormalities, atrial fibrillation and hypehomocysteinemia.[3]

In our case, we excluded a systematic prothrombotic condition on the basis of an extensive diagnostic work up of hemostatic parameters carried out more than one month after the acute phase. In addition, the possibility of embolism was excluded by the angiographic appearance of intracoronary thromboses, and the paradoxical embolism by the absence of an interatrial shunt on the basis of a complete predischarge echo examination. Our patient did not have any history of Prinzmetal's angina or cocaine use. We believe that in our patient, in addition to the heightened inflammatory response and hemodynamic instability, the interaction between subtotal occlusion of the proximal right coronary artery and the possible obstruction of the first septal branch, which supplies the RCA in a retrograde fashion, is crucial. It is probable that during the acute phase of anterior myocardial infarction, the large first septal branch became totally occluded due to its origin within the culprit lesion of the LAD. The combination of low antegrade and retrograde flow pressure led to stasis and thrombosis of the mid RCA. This scenario matches with the time sequence of infarctions. During the total occlusion of LAD and maybe of the septal branch, ST segment elevation did not exist in the leads of the inferior left ventricle wall, because this wall was supplied in an antegrade fashion through a partially occluded RCA. In the next step and despite LAD revascularization, the circumstances of thrombosis at the mid portion of the RCA had already been completed. Acute and chronic occlusions are confused because they are based only on angiographic findings. In our case, the total occlusion of the mid RCA was acute and not chronic, a fact that was proven by the easy passing of our soft guide wire through the total occlusion. Additionally, the ECG and angiographic findings both supported multiple simultaneous-sequential coronary occlusions.

In summary, simultaneous coronary occlusions in acute myocardial infarction are infrequent. Multiple mechanisms may lead to this situation. Accurate identification of the affected vessels can be achieved with a correct interpretation of both the ECG and coronary angiography.

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