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Case Report

Subacute Anterior Myocardial Infarction due to the Discontinuation of Warfarin in a Patient with Aortic Valve Prosthesis; What is the Best Treatment

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Case Report

Acute Myocardial Infarction (MI) is a common entity in patients due to the atherosclerotic plaque rupture. But there are other suspicious etiologic reasons that lead to coronary embolism, such as intracardiac prosthesis, infective endocarditis, mural thrombus or a cardiac tumor [1-8]. Even if the patient did not use warfarin, the real reason for acute myocardial infarction whether embolism or atherosclerotic plaque rupture should be revealed in order to decide optimal treatment option. Intravascular ultrasonography and optical coherence computed tomography are very useful for diagnosis so which treatment is the best; thrombolysis, angioplasty or stenting. Previous cases of coronary emboli in association with prosthetic mechanical valves have been reported previously, but the treatment is controversial [9-18]. We present a patient who had a mechanical aortic valve and was admitted to the hospital for a subacute anterior ST elevation myocardial infarction due to the discontinuation of warfarin.

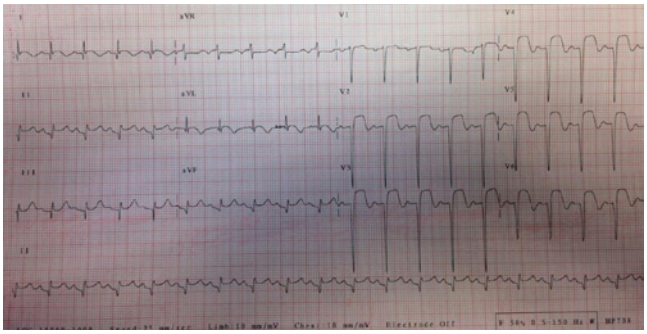

Figure 1: The ECG during admission.

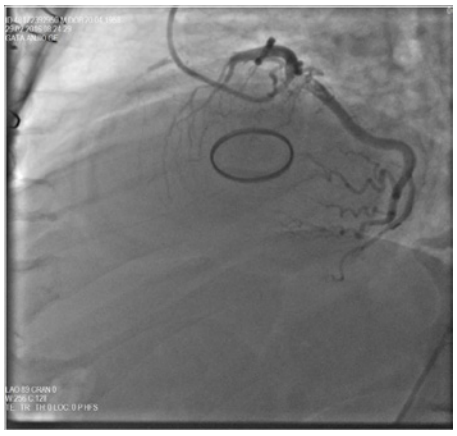

Figure 2: The angiographic images of the patient with total occlusion of the LAD.

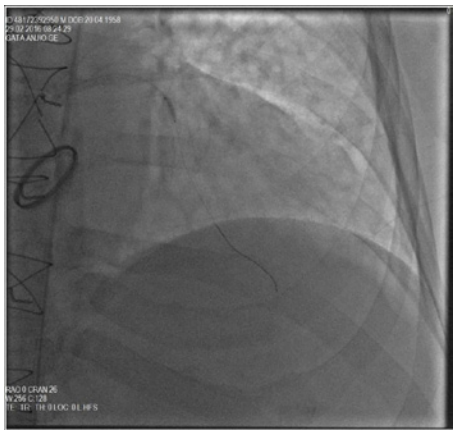

Figure 3: The angiographic images of the patient during balloon inflation.

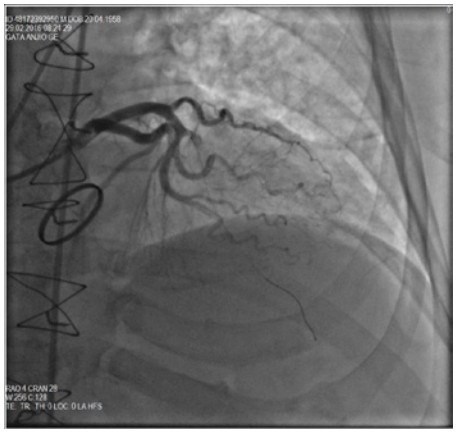

Figure 4: The angiographic images of the patient after first balloon inflation.

A 58-year- old male presented with severe retrosternal chest and back pain radiates to his left arm for 20 hours was admitted to our coronary care unit. His symptoms were resolved on admission. He had previous mechanical aortic valve implantation nine years earlier

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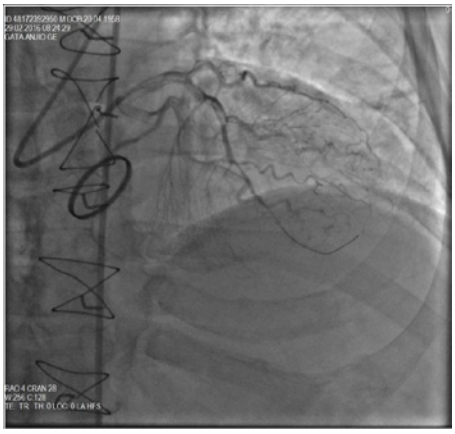

Figure 5: The angiographic images of the patient after second balloon inflation with cranial projection.

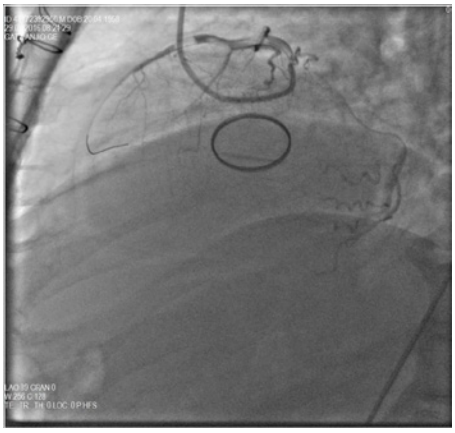

Figure 6: The angiographic images of the patient after second balloon inflation and residue thrombus with cranial projection.

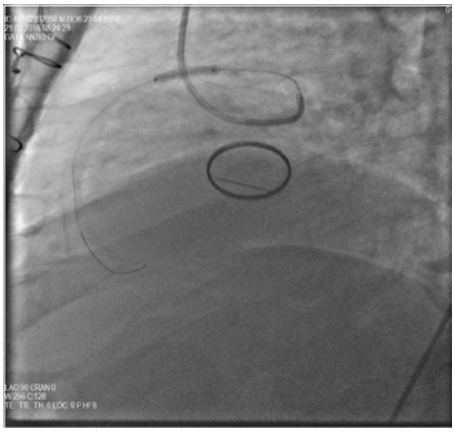

Figure 7: The angiographic images of the patient after second balloon inflation with lateral projection.

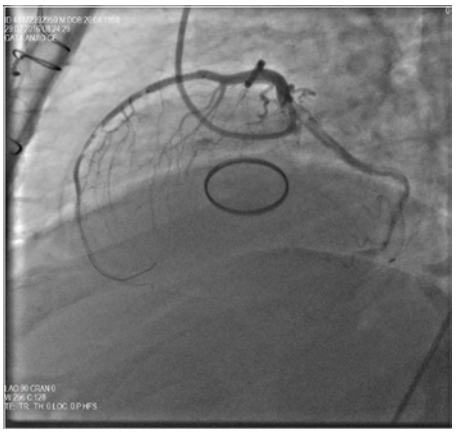

Figure 8: The angiographic images of the patient during stent implantation.

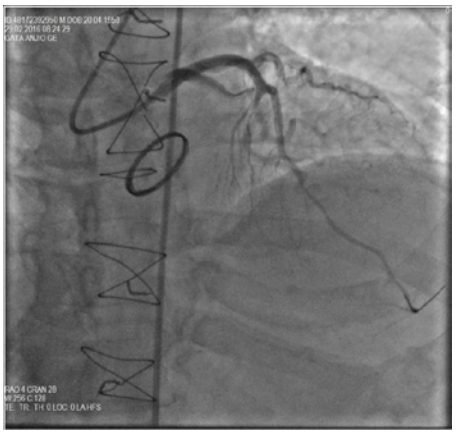

Figure 9: The angiographic images of the patient after stent implantation with lateral projection.

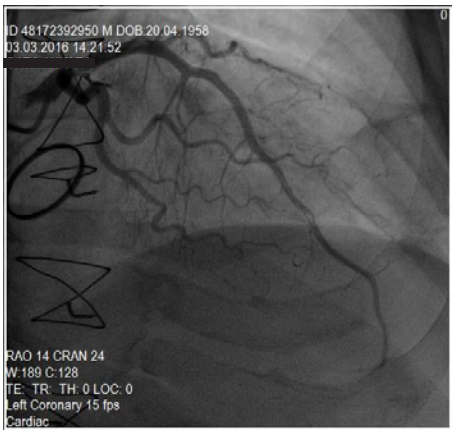

Figure 10: The angiographic images of the patient after stent implantation and residue thrombus in the distal LAD with cranial projection.

because of a rheumatic aortic valve. The patient did not have a history of traditional cardiovascular risk factors except smoking (1 pack a day) and hypertension. No other cormobidities are present. His familial medical history is otherwise unremarkable. Pulse rate was 68/bpm, regular, arterial blood pressure was 110/70mmHg, lungs were clear, breath rate was 18/pm, no murmur on physical examination. Systolic ejection murmur grade 2/6 on the second right parasternal space and a mechanical valve click was heard with the cardiac auscultation. The Electrocardiography (ECG) revealed ST-segment elevation in the precordial leads consistent with subacute anterior Myocardial Infarction (MI); ST segment elevation in V1-6, T inversions and

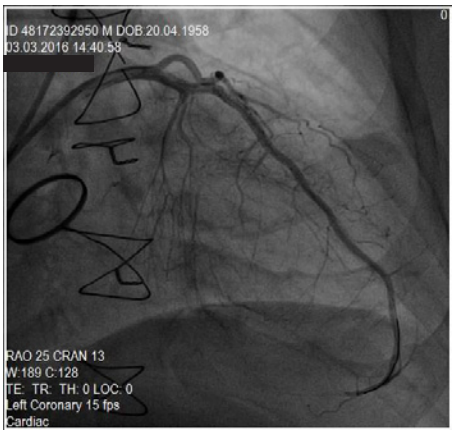

Figure 11: The angiographic images of the patient after stent implantation and no residue thrombus in the distal LAD with cranial projection.

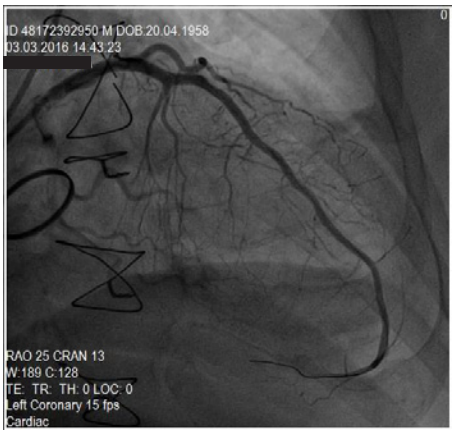

Figure 12: The final result of the distal LAD with cranial projection.

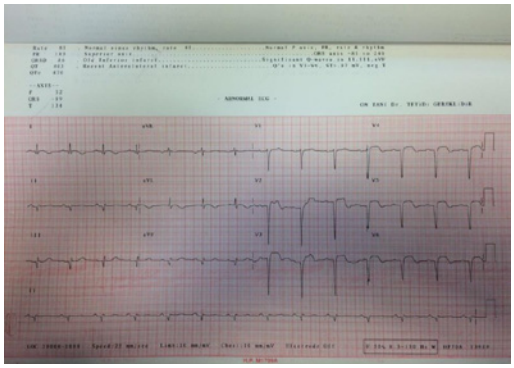

Figure 13: The ECG after stent implantation to the proximal LAD.

pathological Q waves on admission (Figure 1). There was significant elevation of the cardiac markers (troponin T level of 8022 ng/ml and creatinine kinase-MB level of 340 U/L). The patient's INR level was 1.34 reflecting an unprotected and prothrombotic state. Physical findings were not remarkable. The patient was diagnosed to have a subacute anterior ST-Elevation Myocardial Infarction (STEMI). LVEF was 30% and severe akinesia of the anterior and apical wall of the left ventricle was detected on transthoracic echocardiography so we started the dual anti-platelet therapy (aspirin 300 mg +180 mg ticagrelor as for loading dosage), and unfractionated heparin infusion. After that, the patient was transferred to the catheter laboratory and Coronary Angiography (CAG) showed total occlusion of the proximal part of the Left Anterior Descending Artery (LAD) (Figure 2). There were no significant stenosis on the right coronary artery and circumflex artery. We advanced a 0.014 inch floppy guidewire and passed through the lesion, we attempted to aspirate the thrombus three times by using intracoronary catheter aspiration was but was not successful. The occlusion was inflated with 1.5x15 mm and 2.5x15 mm balloons (Figure 3) but they were also unsuccessful due to the severe residue coronary thrombus (Figures 4-7). A drug eluting stent (3.0x15mm) was inserted and TIMI flow was so good but distal embolism developed, which completely not obstructed the lumen (Figures 8-10). The patient was transferred to the coronary care unit and intravenous glycoprotein II b III a inhibitor (abciximab) plus heparin were also started for 24 hours. Control CAG showed the flow was good but there was no thrombus formation in the LAD (Figures 11-12). The ECG was improved (Figure 13). The patient was discharged 7 days later on acetylsalicylic acid, and beta-blockers, and angiotensin-converting enzyme inhibitor therapy under maintenance of warfarin (with an INR value of 3-3.5).

In conclusion, in case of a high thrombus burden causes total occlusion of the coronary vessels, aggressive antithrombotic treatment is feasible in case of low bleeding risk as in our case. So, in case of persistant thrombus formation despite aggresive antitrombotic and anticoagulant therapy, thrombus aspiration, angioplasty and stenting should be preferred rather than conservative approach. Prosthetic mechanical valves requires the use of long-term conventional anticoagulation therapy with warfarin and it is important for the doctors to be lack of awareness of medication of patient who carries significant thromboembolic risk.

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