

# Sub acute 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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Received date: May 23, 2016; Accepted date: June 30, 2016; Published date: June 30, 2016

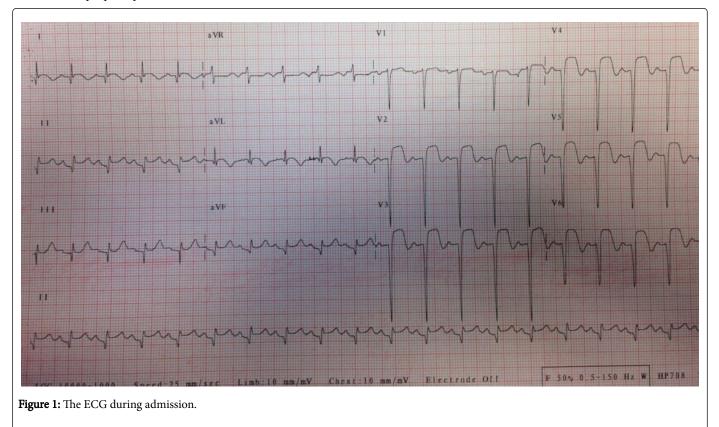
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#### Abstract

Acute myocardial infarction (MI) is a common entity in patients due to the atherosclerotic plaque rupture or coronary embolism. We present a patient who had a mechanical aortic valve and was admitted to the hospital for a sub acute anterior ST elevation myocardial infarction due to the discontinuation of warfarin. In case of persistent thrombus formation despite aggressive antithrombotic and anticoagulant therapy, thrombus aspiration, angioplasty and stenting should be preferred rather than conservative approach.

#### Introduction

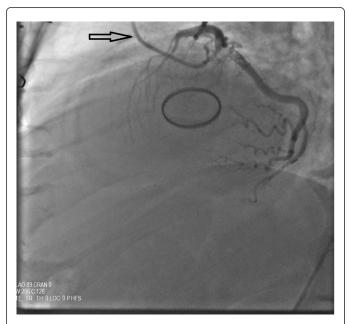
Acute myocardial infarction (MI) is a common entity in patients due to the atherosclerotic plaque rupture. But there are other suspicious etiologic reasons that lead to coronary embolism such as intracardiac prosthesis, infective endocarditis, mural thrombus or a cardiac tumor [1-7]. Even if the patient did not use warfarin, the real reason for acute myocardial infarction whether embolism or atherosclerotic plaque ruptures should be revealed in order to decide optimal treatment option. Intravascular ultrasonography and optical coherens computed tomography are very useful to 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 [8-16]. 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.



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## Case

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.



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

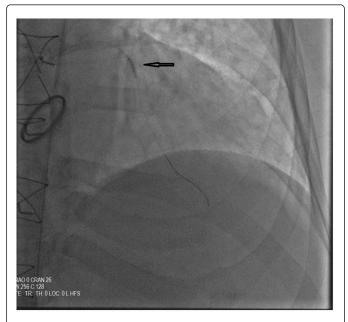


Figure 3: The angiographic images of the patient during balloon inflation.

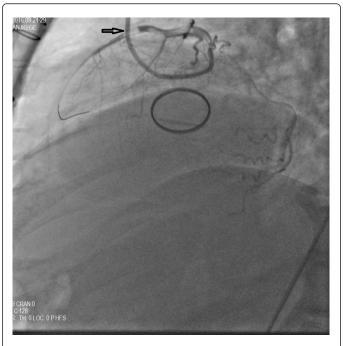


**Figure 4:** The angiographic images of the patient after first balloon inflation.

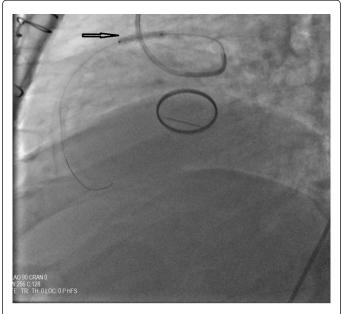


**Figure 5:** The angiographic images of the patient after second balloon inflation with cranial projection.

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**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.

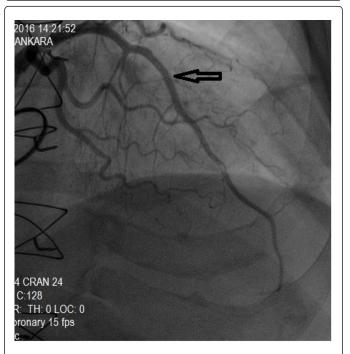
His symptoms were resolved on admission. He had previous mechanical aortic valve implantation nine years earlier because of a rheumatic aortic valve. The patient did not have a history of traditional cardiovascular risk factors except smoking (1 pack a day) and hypertension. No other comorbidities are present. His familial medical history is otherwise unremarkable.Pulse rate was 68/bpm, regular, arterial blood pressure was 110/70 mmHg, 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 sub acute anterior myocardial infarction (MI); ST segment elevation in V1-6, T inversions and 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 sub acute anterior ST-elevation myocardial infarction (STEMI). Left ventricular ejection fraction(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 guide wire 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  $1.5 \times 15$  mm and  $2.5 \times 15$  mm balloons (Figure 3) but they were also unsuccessful due to the severe residue coronary thrombus (Figures 4-7). A drug eluting stent  $(3.0 \times 15 \text{ mm})$  was inserted and TIMI flow was so good but distal embolism developed, which completely not obstructed the lumen (Figure 8-10). The patient was transferred to the coronary care unit and intravenous glycoprotein IIbIIIa 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 and 12). The ECG was improved (Figure 13). The patient was discharged 7 days later on acetylsalicylic acid, and beta-

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blockers, and angiotensin-converting enzyme inhibitor therapy under maintenance of warfarin (with an INR value of 3-3.5).



**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.



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

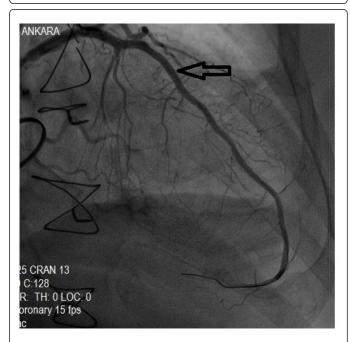
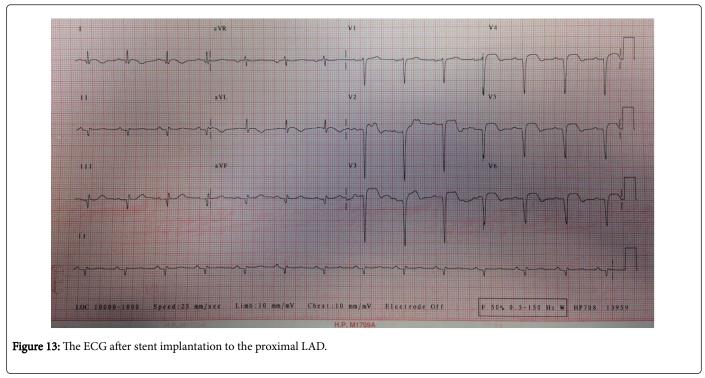


Figure 12: The final result of the distal LAD with cranial projection.



### Discussion

There are many solutions for treatment of coronary embolism. One of them is thrombus aspiration but it may not be enough to track thrombus from coronary vasculature despite several trials with aspiration catheter. The other one is only percutaneous coronary angioplasty without stenting but it may not also obtain optimal angiographic result and TIMI flow. Another choice is stenting in case of all failed interventions. High dosage antiaggregant and anticoagulant therapy are also possible solutions. In case of 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 persistent thrombus formation despite aggressive antithrombotic and anticoagulant therapy, thrombus aspiration, angioplasty and stenting should be preferred rather than conservative approach. A prosthetic mechanical valve 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. The choice of treatment should be based on individual decisions and should be set depending on the clinical and comorbid condition of the patient.

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