

Acute myocardial infarction in a patient with a mechanical aortic valve

Omar F Hassan

FICMS (Cardiology). Cardiologist, Email: omarflaih@gmail.com.

ABSTRACT

A case of an acute myocardial infarction with ST segment elevation in a patient with a mechanical aortic valve prosthesis who is having a sub-therapeutic INR level due to intermittent interruption of his anticoagulant therapy. He was successfully treated with systemic thrombolytic therapy, in order to restore coronary flow in the left anterior descending coronary artery as there were no facilities for primary coronary intervention in our hospital. Review of the literature suggests that in such cases the diagnosis of the embolic origin of the acute coronary syndrome is assumed and can never be proved definitely. Thrombus aspiration must be included in the therapeutic strategy of these patients.

Key words: Acute myocardial infarction, embolism, prosthetic aortic valve.

CASE PRESENTATION

A 52 year-old gentleman, who is known to underwent aortic valve replacement with St. Jude bileaflet mechanical aortic valve since 2007, admitted to hospital because of an anterior acute ST elevation myocardial infarction (STEMI). (figure 1)

The patient was brought to the emergency department 40 minutes after the onset of a typical chest pain. Apart from being cigarette smoker, he did not report any conventional risk factors for coronary artery disease such as diabetes mellitus, hypertension, or hypercholesterolaemia with a negative family history of coronary heart disease. His body mass index was 26 kg/m².

On admission and after a rapid clinical evaluation, ECG trace was done (Figure 1) and a transthoracic echocardiographic examination was performed which revealed dyskinetic apex and akinetic mid-septum of the left ventricle. Left ventricular ejection fraction was 40% and the mechanical aortic valve was functioning normally without any apparent thrombus. The peak of the transvalvular gradient of the aortic valve prosthesis was 23 mmHg with a mean of 16 mmHg. Although he was supposed to have been receiving warfarin, his INR was found to

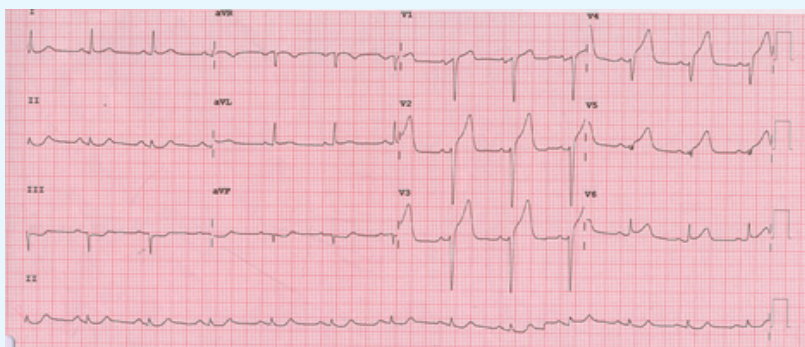
be 1.7. Patient confessed that he was holding warfarin therapy intermittently due to intermittent bleeding from chronic haemorrhoids without referring to any physician.

After the initial bedside assessment, performance of transthoracic echocardiography, and insertion of I.V. line, patient was given glyceryl trinitrate (angised) 0.5 mg sublingually, morphine sulphate 5 mg I.V., aspirin 300 mg, clopidogrel 300 mg, I.V. heparin infusion, and fluvastatin 80 mg.

His initial clinical assessment has showed a middle aged mildly overweight man in pain. His pulse was regular at a rate of 86 beat per minutes, blood pressure was 136/84 mmHg, and JVP was within normal. All peripheral pulses were symmetrically palpable. Chest has shown a thoracotomy scar. The first heart sound and the metallic second heart sounds were distinctly heard without any added sounds or murmurs. Breath sounds were clearly heard over both lung bases without any basal crackles. Abdomen was soft for superficial and deep palpation; tenderness was not elicited. No palpable organs were elicited in the abdomen.

ECG on admission has shown a sinus rhythm at a rate of 80 beat per minutes with ST seg-

Figure 1: Initial 12-lead ECG indicating acute anterior STEMI. The trace shows sinus rhythm at a rate of 80 beat per minutes with ST segment elevation from V2-V5.



ment elevation from V2-V5. Troponin t level was 0.025 ng/ml (normal value is < 0.01 ng/ml) then increased to 2.38 ng/ml over minutes. Chest X ray showed a sternotomy sutures, mild aortic root dilatation, and the ring of the aortic valve prosthesis. Lungs parenchyma looked within normal. Prothrombin time was 17.5 seconds with international normalizing ratio (INR) of 1.7.

The condition was discussed thoroughly with the patient and the available options for therapy were explained. Primary coronary interventions were not available in our hospital; thrombolytic therapy was the only available therapeutic measure. Benefits and risks of thrombolysis were thoroughly discussed with the patient and after taking the consent for the thrombolytic therapy, alteplase (rTPA) was started about 45 minutes from admission to emergency department. Ninety minutes after starting thrombolytic therapy, the patient was free of pain, stable general condition and vital signs, and his new 12- leads ECG trace has shown more than 50 % reduction of ST segment elevation. He was transferred to the ICU for continuous monitoring. His medication there has included I.V. heparin infusion which was titrated according to the partial thromboplastin (PTT) ratio, aspirin 100 mg twice daily, clopidogrel 75 mg once daily, fluvastatin XL 80 mg once daily,

metoprolol 50 mg twice daily and perindopril 5mg once daily. Warfarin was resumed two days later; its dose was regulated according to daily monitoring of INR which was 2.1 on discharge. The patient was discharged home after 6 days with uneventful hospital course. (pre-discharge ECG trace has shown in [figure 2](#))

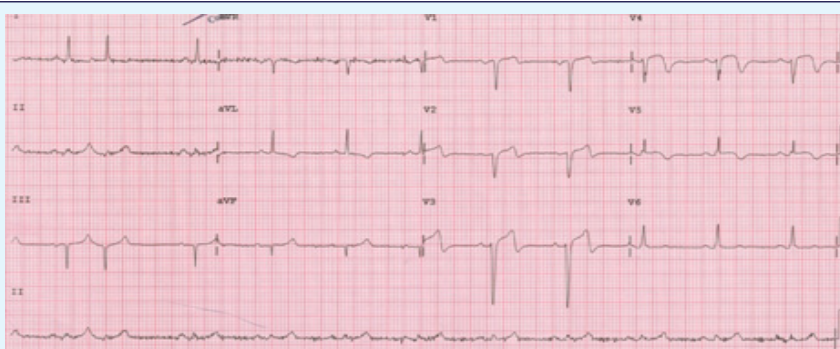
A planned coronary angiography, which was done three weeks later on, has shown a normal result without any critical or non-critical lesions in the left and right coronary systems. ([figure 3](#))

The patient now having regular follow up visits in the cardiology clinic for 7 years. He runs a stable medical condition, did not experience recurrent chest pain and maintain a good functional capacity. Regular monitoring of his ECG and Echocardiography showed no new changes while his INR is kept with the desired therapeutic range of 2-3.

DISCUSSION

Myocardial infarction (MI) due to coronary artery embolization is a rare and potentially lethal complication of prosthetic heart valve thrombosis. Thrombus formation on prosthetic valve appears to be common¹⁻⁴ and the incidence of emboli from such a thrombus has been estimated to be as high as 40% in patients

Figure 2: Predischage 12 leads ECG with complete resolution of ST segment elevation and normalization of the ECG.



surviving 2 years or longer.^{3,4} Fortunately, fatal emboli are much less common, and adequate, well-controlled anticoagulation appears to decrease the incidence of thromboembolism.^{4,5}

Coronary emboli are often suspected in patients with prosthetic valves, but they are not frequently demonstrated.^{2,4} In addition, coronary atherosclerosis and postoperative intimal proliferation of the proximal coronary arteries may lead to myocardial ischemia.³

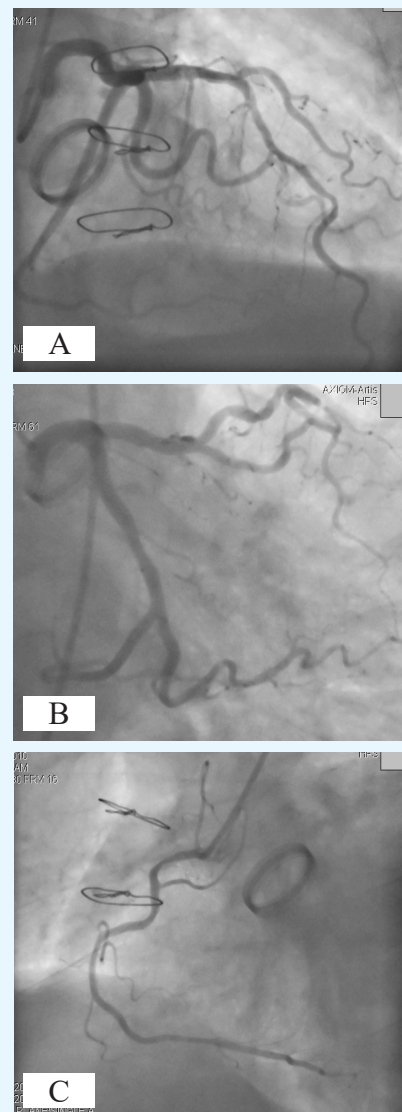
Patients with complications related to embolisation present with signs related to the site of embolization. Stroke syndromes are the most common; however, patients may present with MI, sudden death, or visceral or peripheral embolisation. Systemic embolisation should alert the physician to suspect valve thrombosis or PVE.

Since the advent of prosthetic valvular surgery, another source for coronary emboli has been introduced, and fragments of the prosthetic material or more commonly thrombus formed at the surface of the prosthesis may constitute the embolic material.⁶ In 1964, Bjork and Malers,⁷ on discussing the late results of mitral valve replacement, reported the first case of coronary embolism arising from a mitral prosthesis.

Despite technical improvement in prosthesis design and the development of less thrombogenic material, mechanical valve prostheses still carry a significant thromboembolic risk, which warrants long-term anticoagulation therapy. However, even adequate anticoagulation therapy does not eliminate the risk of thromboembolism in these patients. Type of valve, position and associated conditions affect the risk of thromboembolism.

Patients with caged ball or tilting disk valves have a higher risk as compared to patients with bileaflet valves (2.5 % /year versus 0.5 % /year respectively). A mechanical valve in the aortic position has a lower risk (0.5 % /year) as compared to mitral valve (0.9 % / year) or both (1.2 % / year). From this point of view, our patient had a favorable profile (bileaflet valve, aortic position) to avoid an episode of thromboembolism, but unfortunately he had a myocardial infarction as he was intermittently holding his anti-coagulant therapy and his INR level was in the subtherapeutic level. A case has been reported in the literature of a patient with a metallic aortic valve who was scheduled for stripping of a low-

Figure 3: Coronary angiography of the patient. Picture A & B show The left coronary system angiogram with normal left main, left anterior descending, left circumflex and their epicardial branches. Picture C shows the right coronary artery angiogram showing normal RCA and it's epicardial branches.



er limb varix and had interrupted anticoagulant therapy for only 3 days, who suffered an acute myocardial infarction postoperatively.⁸ On the other hand, another interesting case⁹ of coronary embolism causing myocardial infarction in a patient with mechanical aortic valve prosthesis who had previously quit warfarin for almost a year has also been reported.

In cases of coronary embolization and myocardial infarction, a source of thrombotic material is usually detected.¹⁰⁻¹² It is noteworthy that acute myocardial infarction due to coronary embolism can occur as a result of left atrial thrombus in cases of atrial fibrillation.⁹ Echocardiography is a very useful tool in diagnosing a thrombus on a mechanical valve prosthesis, although differential diagnosis from pannus is usually a difficult challenge. But in some cases,^{13,14} as

in the present case, no apparent thrombus could be detected. In such cases a definite proof of an embolic pathophysiology cannot be documented and rather remains a presumptive diagnosis.

In this case, our assumption that acute myocardial infarction has been caused by an embolism from the mechanical valve prosthesis was based on the presence of predisposing factors for thrombo-embolism like mechanical valve prosthesis and a documented sub-therapeutic anticoagulant therapy in one hand, and the normal coronary angiographic results in a patient with low probability for development of myocardial infarction on the other hand. Argument that failure to detect a thrombus over a prosthetic valve by transthoracic echocardiography can critically jeopardize this assumption, is not very hard to negotiate as the presence of a thrombus is not a necessitate for the diagnosis of embolism as a cause for acute myocardial infarction,^{13,14} in addition, the presence of such a thrombus in a patient who sustains acute myocardial infarction is not a hard proof for a causative relationship with an occluded coronary artery. It is also of importance to say that transthoracic echocardiography is relatively not sensitive to detect all thrombi and transesophageal echocardiography, which was not available at our hospital at the time, may sound a better surrogate for this purpose. From this point of view, we assume that myocardial infarction in our patient could be probably attributed to embolization of microthrombi from the mechanical valve to the left anterior descending artery and we should keep in our mind this causative relationship in any vascular event that may develop in a patient with prosthetic valve with a great deal of attention be paid to primary prevention of this dreadful fate once it happens by maintaining the anticoagulation state in its optimal level.

Treatment of acute myocardial infarction due to embolization includes many therapeutic alternatives, both drugs and interventional procedures. A case has been described of percutaneous transluminal coronary recanalization¹⁵ attempted by infusing 1.2 million units of urokinase which however proved unsuccessful. Nevertheless, repeat coronary angiography performed 40 days later, showed normal coronary arteries. Another case of coronary embolism with suboptimal INR levels, was successfully treated with percutaneous transluminal coronary angioplasty and stenting,¹⁰ whereas in another relevant case an aspiration catheter (Export XT, Medtronic) was only

used and succeeded in restoring coronary flow without stenting.¹⁴ Adjunctive therapy with infusion of a platelet glycoprotein IIb/IIIa inhibitor may be effective in reducing thrombotic burden and eliminating the risk of the no-reflow phenomenon when angioplasty is performed. In these cases, the IIb/IIIa agent should be started as soon as possible even before transferring the patient to the catheterization laboratory. It is noteworthy that coronary embolism in a patient with mitral valve prosthesis¹⁶ has been successfully treated with the IIb/IIIa agent, tirofiban, and half a dose of tissue-type plasminogen activator.

In patients with a presumed embolic coronary event,¹⁷ transesophageal echocardiography (TEE) has been recommended to identify any potential endocardial source of emboli.¹⁷ In the present case, TEE was not performed and this may be a limitation, however, it is known that even TEE has a very low sensitivity in documenting presence of microthrombi on a metallic valve, as increased echogenicity hinders good visualization.

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Abbreviation list: Electro Cardio Graphy (ECG), Intensive Care Unit (ICU), International Normalizing Ration (INR), Intra Venous (I.V.), Jugular Venous Pressure (JVP), Milligram (mg), Millilitre (ml), Millimetre Mercury (mmHg), Myocardial Infarction (MI), Nano Gram (ng), Partial Thromboplastin Time (PTT), Pulmonary Venous Embolization (PVE), Recombinant Tissue Plasminogen Activator (rTPA), ST Elevation Myocardial Infarction (STEMI), Trans-Oesophageal Echocardiography (TEE).

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