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CASE REPORT

Acute Myocardial Infarction Presumably Embolic, in a Patient With a Mechanical Aortic Valve: a Rare Cause of Non-Atherosclerotic Coronary Artery Occlusion

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ABSTRACT

We present a case of an acute myocardial infarction with ST segment elevation in a patient with a mechanical aortic valve prosthesis who had discontinued anticoagulant therapy. We swiftly performed a primary coronary intervention procedure, including thrombus aspiration as well as plain balloon angioplasty (low atmospheres), in order to restore coronary flow in the left anterior descending coronary artery. 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 been proved definitely. Thrombus aspiration must be included in the therapeutic strategy of these patients.

CASE PRESENTATION

A 30-year-old male with a bileaflet mechanical aortic valve (St Jude Medical Standard, 23 mm), implanted for stenosis of a bicuspid aortic valve two years ago, was admitted because of an anterior acute myocardial infarction with ST segment elevation (Figure 1). The patient did not have any conventional risk factor for coronary artery disease; never had he smoked, had no diabetes mellitus or hypertension or hypercholesterolemia, and his body mass index was 28 kg/m^2. He reached the emergency department 90 minutes after the onset of chest pain. On admission, after a rapid clinical evaluation and recording of an ECG (Figure 1), a transthoracic echocardiographic examination was performed which revealed marked hypokinesis of the anterior and septal wall of the left ventricle, left ventricular ejection fraction of 45% and a normally functioning aortic valve without any apparent thrombus (Figure 2A) and with an acceptable transvalvular peak gradient (23 mmHg) for the specific type of metallic aortic valve (Figure 2B). Although he was supposed to have been receiving warfarin, surprisingly, INR was found to be 1. The patient admitted that he had on his own discontinued warfarin 15 days earlier.

The patient was immediately placed on aspirin 500 mg and clopidogrel 600 mg per os. He also received intravenous eptifibatide, a platelet glycoprotein IIbIIIa inhibitor, in a bolus dosage followed by continuous infusion at a maintenance dosage

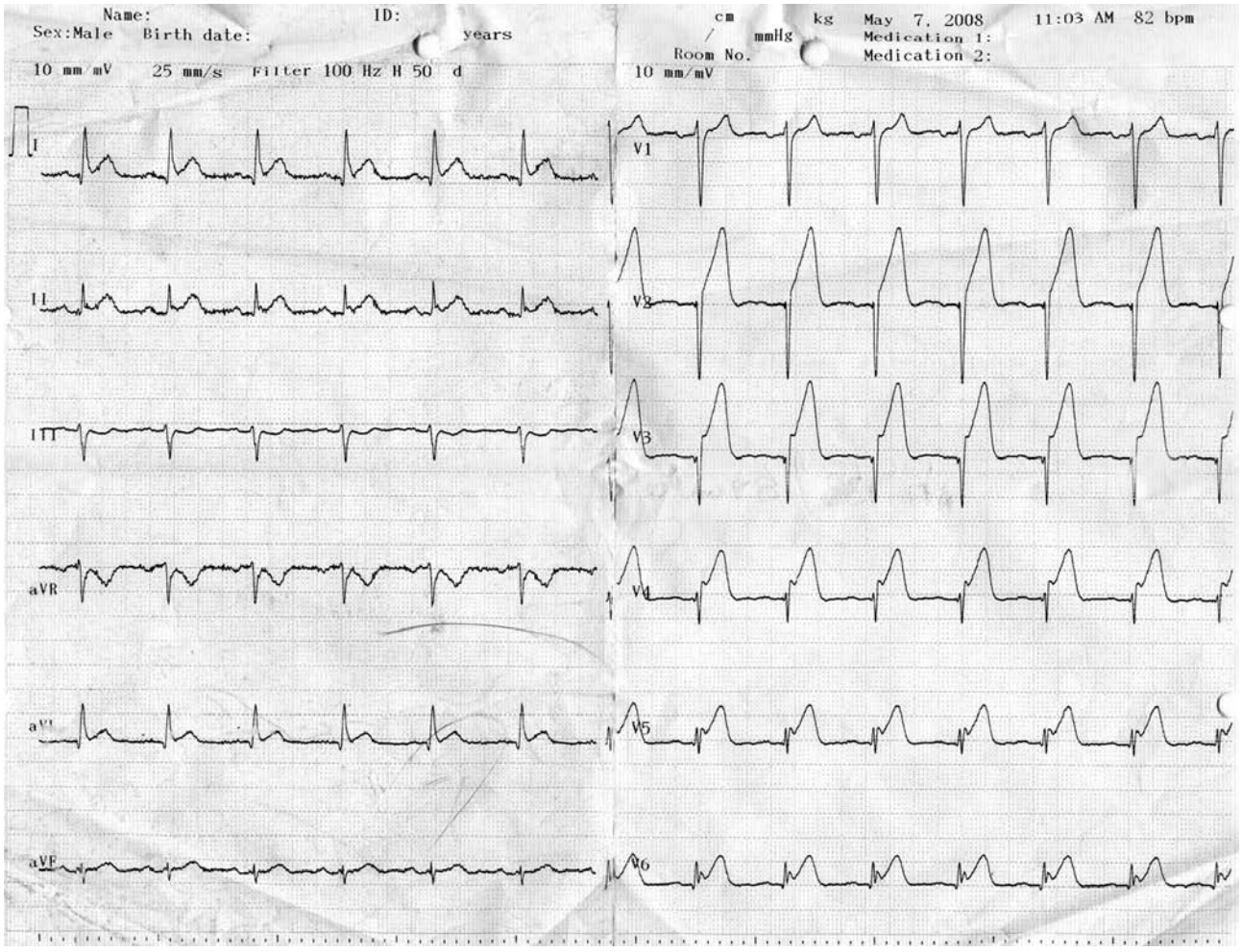

FIGURE 1. Admission 12-lead ECG indicating acute anterior ST elevation myocardial infarction.

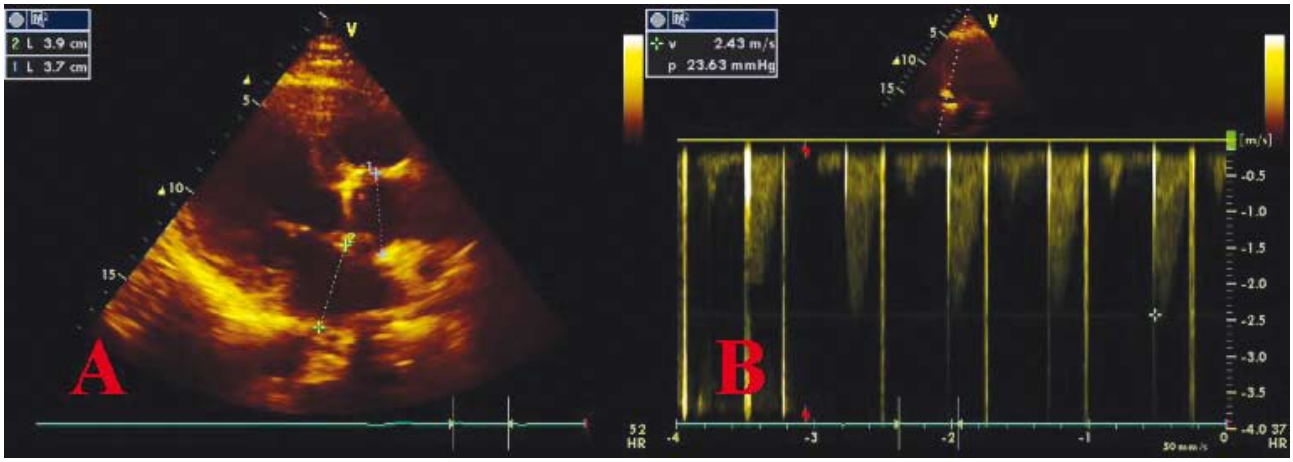

FIGURE 2. Transthoracic echocardiography study (**A**, long axis parasternal view) showing no apparent thrombus on the metallic aortic valve, while a Doppler study (**B**) indicated an acceptable transaortic gradient for the specific type of prosthesis.

before his transfer to the catheterization laboratory 30 minutes later. Fluoroscopy documented normal opening of both leaflets of the mechanical aortic valve (Figure 3). Coronary angiogram showed total occlusion in the mid segment of the left anterior descending coronary artery (LAD) (Figure 4A). After administration of adjusted unfractionated heparin in a bolus infusion (70 IU/kg), we proceeded to perform coronary angioplasty. Initially it was attempted to restore vessel flow with a transcatheter thrombus removal system (Export Catheter, Medtronic). However, thrombus aspiration proved partially successful in restoring LAD flow, and only very small pieces of thrombotic material were recovered (Figure 5). Subsequently, we inflated a small (2 x 10 mm) balloon (Figure 4B) at low pressure (6 atmospheres), relieving the obstruction and achieving a TIMI II flow. Since the obstructive lesion in the LAD was attributed to coronary embolus, no stent was implanted, in order to avoid any restenosis as a result of vessel trauma and also to obviate the need for double antiplatelet therapy which combined with warfarin therapy, necessary for his artificial valve, might significantly increase the bleeding risk. Considering a pathophysiology of partial no-reflow phenomenon, repeated doses of 50 mcg of intracoronary adenosine to a total dosage of 200 mcg were administered. Eventually an open vessel with restored TIMI III flow was obtained (Figure 4C) and the patient had complete relief of anginal symptoms. A door-to-balloon inflation time of approximately 50 minutes was achieved and the total duration of myocardial ischemia was estimated at 140 minutes.

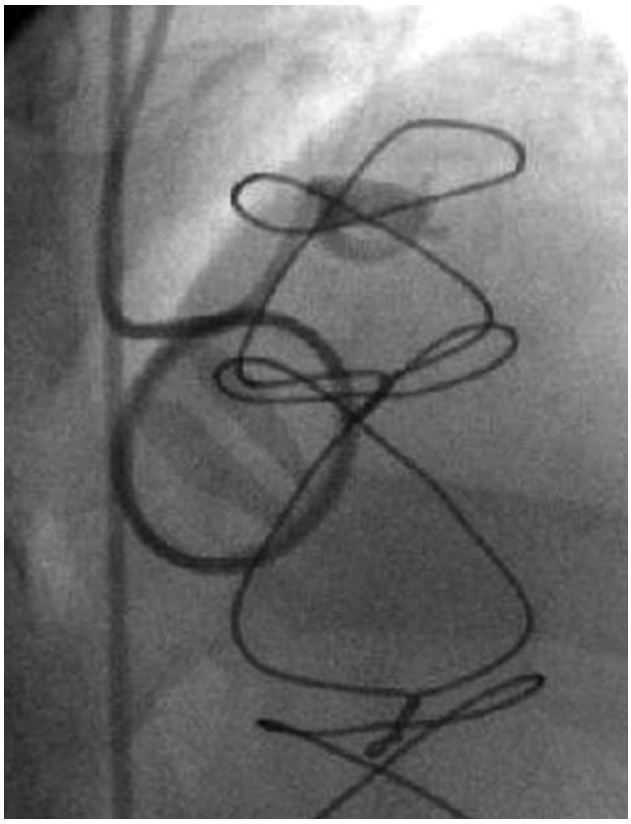

FIGURE 3. Fluoroscopic view of the two leaflets of the aortic prosthesis showed normal opening of the artificial valve.

Twelve hours after sheath removal, the patient was started on warfarin; he was bridged with subcutaneous enoxaparin (1 IU/kg) twice daily until the INR was stabilized at values >2 for the subsequent three days. Eptifibatide continuous infusion was maintained for 24 hours. Postprocedurally, the patient was started and maintained on aspirin 100 mg daily, carvedilol 6.25 mg bid and enalapril 10 mg daily. Serum levels of biochemical markers of myocardial damage were indicative of an extensive myocardial infarction; CK-MB peaked at 115 U/L and the troponin I level at 65 ng/ml. The patient did

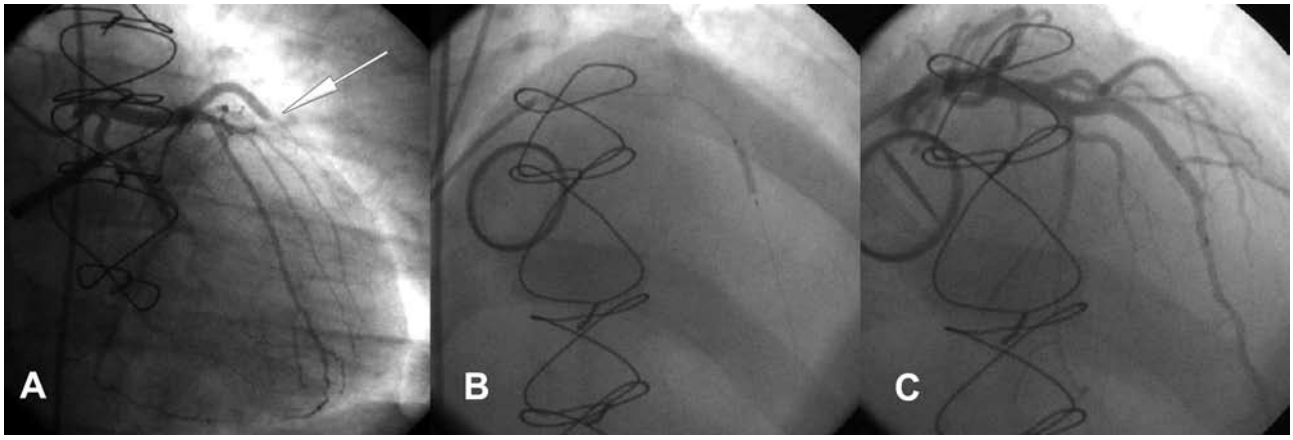

FIGURE 4. Angiographic views indicating the total obstruction of the left anterior descending (LAD) coronary artery (**A**, arrow), which was relieved with use of an aspiration catheter (not shown) and a plain angioplasty balloon (**B**), which restored flow to the LAD (**C**).

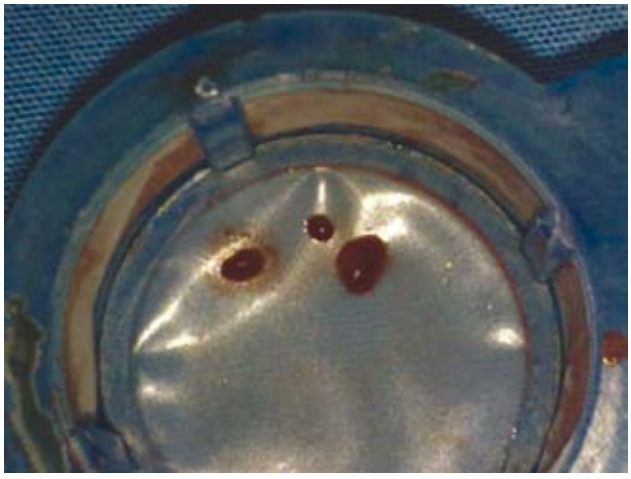

FIGURE 5. Here is shown (within the basket) the thrombotic (red) material which was retrieved with use of the aspiration catheter from the site of the total occlusion of the LAD.

well clinically and was discharged 6 days later. On the day of discharge, an echocardiography study showed that the left ventricular ejection fraction remained at 45% and there was still hypokinesis of the anterior and septal walls.

Due to the absence of any detectable thrombi on the metallic aortic valve by serial transthoracic echocardiographic studies and the fluoroscopic documentation of a normally functioning aortic prosthetic valve, a transesophageal echocardiogram was not performed either at presentation, or after the procedure.

DISCUSSION

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.[2] In 1964, Bjork and Malers,[3] 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 vs 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 15 days after discontinuing anticoagulant therapy. A case has been reported in the literature of a patient with a metallic aortic valve who was scheduled for stripping of a lower limb varix and had interrupted anticoagulant therapy for only 3 days, who suffered an acute myocardial infarction postoperatively.[5] On the other hand, another interesting case[6] 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.[7-9] 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 panus is usually a difficult challenge. But in some cases,[10,11] 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. Although we documented the presence of thrombus at the site of arterial occlusion, this is not proof of the embolic origin of acute myocardial infarction, since the thrombotic process is very common in acute coronary syndromes in patients with atherosclerotic coronary artery disease. Our assumption regarding embolic pathophysiology in this case was based on the predisposing factors for thrombosis (mechanical valve and interruption of anticoagulant therapy), and the low probability for coronary artery disease in this patient. The patient was very young with very low probability for myocardial infarction, but with very high probability for any embolic event since he was not receiving any anticoagulation therapy at all. From this point of view, we considered that myocardial infarction could probably be attributed to embolization of microthrombi from the mechanical valve to the LAD. On the other hand, even in cases with an apparent thrombus formation, this does not constitute proof for a causative relationship with an occluded coronary artery.

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 recanalization[9] 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.[11]

Although thrombus aspiration has been proved to be an effective method[12] in treating acute myocardial infarction due to coronary embolism, in the case presented herein, the aspiration device failed to restore flow in the occluded vessel. Adjunctive therapy with infusion of a platelet glycoprotein IIbIIIa 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 IIbIIIa 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 prosthesis[13] has been successfully treated with the IIbIIIa agent, tirofiban, and half a dose of tissue-type plasminogen activator.

In patients with a presumed embolic coronary event,[14] transesophageal echocardiography (TEE) has been recommended to identify any potential endocardial source of emboli. 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. Removal of microthrombotic material from the LAD supports the hypothesis that acute myocardial infarction in this case was presumably due to embolization from the mechanical aortic valve prosthesis in a non-compliant patient who was not receiving anticoagulation prophylaxis for the preceding 2 weeks. From this point of view, it was considered that further exclusion of a paradoxic origin of such an embolization was not necessary, since additionally, transthoracic echocardiography had not revealed any apparent intracardiac communication.

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