Case Report

Coronary embolism causing acute myocardial infarction in a patient with mitral valve prosthesis: successful management with angioplasty

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Abstract

A 24-year-old male patient with anterior myocardial infarction, caused by embolization from mitral valve prosthesis due to inadequate anticoagulation is presented. The patient underwent cardiac catheterization within 90 minutes of arrival. Angiography showed total occlusion of the left anterior descending coronary artery (LAD) after the second diagonal branch. Thrombus was extracted with export catheter from LAD, and coronary artery perfusion was restored. The pain disappeared completely immediately after this intervention. Transoesophageal echocardiography performed 2 days later revealed no thrombus at the prosthetic valve. In conclusion, this case demonstrated that coronary embolism may occur even without prosthetic valve thrombus or dysfunction with suboptimal International Normalized Ratio levels, and can be successfully treated with coronary angiography with clot extraction with aspiration catheter (Export XT 6F Medtronic) only, without stenting.

Introduction

Coronary embolism has been considered as a rare cause of coronary occlusion. Prosthetic heart valve is an infrequent cause of this condition. The exact incidence of coronary embolization in patients with prosthetic heart valves is not well known. Furthermore, the effective treatment and management of coronary emboli in this setting remains unknown. The purpose of this report is to show the successful management of a case of coronary embolism with primary angioplasty using aspiration catheter (Export XT 6F Medtronic) for extracting the thrombus without ballooning and stenting.

Case Report

A 24-year-old man with mitral valve prosthesis was admitted to our hospital on August 20, 2008 complaining of a sudden onset of severe chest pain that began two hours prior to presentation in the emergency room. He had no history of coronary artery disease, diabetes mellitus, or hypertension. He underwent mitral valve replacement with a Medtronic valve (27mm) in June 2002 for severe mitral stenosis and regurgitation due to rheumatic heart disease. He was not taking anticoagulants regularly and not maintaining the therapeutic international normalized ratio (INR) as he stopped warfarin for the last one month . Upon admission to the hospital, ECG showed ST elevation in leads V1 to V4 and sinus rhythm with heart rate of 100 beats/min. Physical examination revealed an acutely ill, pale, diaphoretic man with a blood pressure of 130/80 mmHg. Examination of the heart showed normal opening and closing clicks of the mitral prosthesis and a fourth heart sound; no murmurs were heard. The lungs were clear to auscultation. From clinical presentation, acute myocardial infarction was suspected. Clotting profile showed international normalized ratio 1.29. Urgent coronary angiography was performed 3 hours after the onset of chest pain. Coronary angiography was normal except total occlusion of distal left anterior descending artery (LAD) with intra luminal defect as shown in Figure 1. Aspiration catheter (Export XT 6F Medtronic) was used to extract a thrombus from the artery and injection Eptifibatide (Glycoprotein IIb/IIIa antagonist) infusion at the rate of 9 ml/hour after two boluses was started and continued for 24 hours. Clot was extracted with aspiration catheter and angiogram after extraction revealed normal LAD artery as shown in Figure 2. TIMI III coronary blood flow was restored. The patient was shifted to the coronary care unit where transthoracic echocardiography with Doppler study was carried out showing normal functioning prosthetic valve with normal left ventricular systolic function; in addition, the patient was treated with aspirin, beta-blocker, and nitroglycerine. On the second day transoesophageal echocardiography (TEE) was done to see the status of prosthetic valve and no thrombus was seen on the valve. Heparin infusion was administered for three days and warfarin was started The patient was discharged on warfarin, aspirin, and beta-blocker therapy after achieving INR of 3.0. The patient was also educated on prophylaxis measures against infective endocarditis and rheumatic fever.

Discussion

Coronary embolism was first described by Virchow in 1856.[1] Since then it has been reported clinically and from postmortem studies, and in 1958 Wengere and Bauer, reviewing the literature, found about 15 well documented cases and several necropsy studies.[2] The most common cause of coronary embolism was bacterial endocarditis, and others included rheumatic heart disease, dilated cardiomyopathy, left atrial myxoma, arrhythmias and myocardial infarction.[3] Since the advent of prosthetic valvular surgery, another source for coronary emboli has been introduced, that is, fragments of the

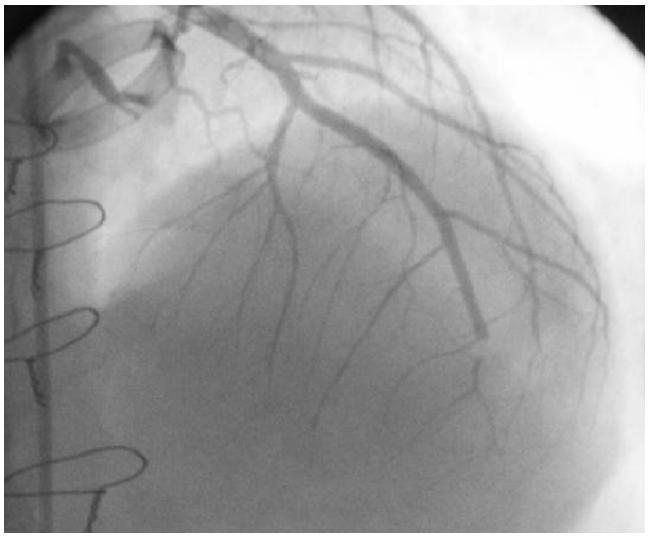

Figure 1: Total occlusion of distal left anterior descending (LAD) artery.

prosthetic material or more commonly thrombus formed at the surface of the prosthesis. In 1964, Bjork and Malers[4] reported the first case of coronary embolism arising from a mitral prosthesis.

The consequences of coronary embolism depend on two major factors: the size of the embolus and the size of the lumen of the artery in which it becomes impacted.[5] The smaller the embolus, the greater is the chance that it will travel distally to a small coronary arterial segment and the less is the

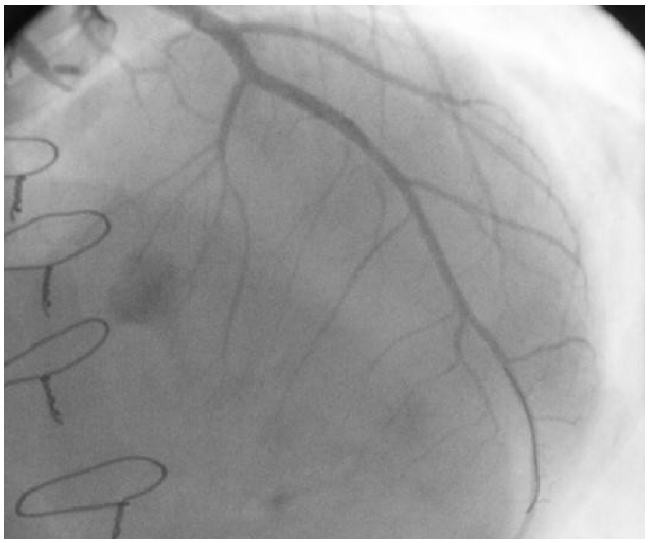

Figure 2: Complete restoration of blood flow in the artery after successful angioplasty.

likelihood of myocardial infarction or fatal arrhythmia.[5]

Currently, there is no clear guideline for the effective treatment and management of coronary emboli. Kamishirado et al[6] reported a case of coronary artery embolism that was not recannulized with 960 000 IU of urokinase. In another case, coronary embolization secondary to aortic valve endocarditis was treated with standard doses of streptokinase and aspirin.[7] The patient survived but sustained a large myocardial infarction and a major gastrointestinal bleeding.

Glycoprotein IIb/IIIa antagonists have been evaluated in many clinical trials. The findings in these trials show that patients undergoing percutaneous transluminal coronary angioplasty (PTCA) benefit from these agents. It has been a common experience of cardiologists that thrombi seen during coronary intervention procedures are reduced or eliminated by this agent, so that it was felt to be logical to employ it in this setting. Prolonged infusion with urokinase was found to be useful in eliminating thrombus from saphenous vein grafts.[8] Recently, it has been reported that a combined regimen of intracoronary urokinase and intravenous abciximab was successful in achieving complete resolution of the emboli in a patient with mitral and aortic valve replacement.[9] We experienced restoration of TIMI III coronary blood flow with only extracting the thrombus with an aspiration catheter (Export XT 6F Medtronic) and continued glycoprotein IIb/IIIa antagonist for 24 hours.

In summary, coronary embolism secondary to prosthetic valve is an uncommon etiology of myocardial infarction. Angioplasty using aspiration catheter without ballooning and stenting and Eptifibatide given intravenously may be an effective treatment of this condition. The outcome looks gratifying.

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