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Acute Coronary Embolism without Valve Thrombosis in a Patient with a Prosthetic Mitral Valve—Successful Percutaneous Coronary Intervention: A Case Report

Mehmet Yazici, MD, Mehmet Kayrak, MD, Yasar Turan, MD, Fatih Koc, MD, Mehmet S. Ulgen, MD

Selcuk University, Cardiology Department, Konya, Turkey

ABSTRACT

We present a 44-year-old female patient with anterior myocardial infarction caused by embolization from mitral valve prosthesis due to inadequate anticoagulation. The patient underwent a cardiac catheterization within the 1st hour of arrival. The angiography showed total occlusion of the left anterior descending coronary artery after the second diagonal branch. Percutaneous transluminal coronary angioplasty and stenting were performed, and coronary artery perfusion was restored. The pain disappeared completely immediately after this intervention. Transthoracic echocardiography shortly after this intervention showed normal prosthetic valve function and no thrombus. Transesophageal 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 percutaneous transluminal coronary angioplasty and stenting.

INTRODUCTION

Coronary thromboembolism is a rare cause of acute myocardial infarction (MI). Several conditions such as dilated cardiomyopathy, chronic atrial fibrillation, infective endocarditis, intracardiac shunts, and hypercoagulable states are reported to cause coronary embolism, but there are only a few cases of coronary embolism in patients with a prosthetic heart valve reported in the literature [Aslam 2002]. Coronary thromboembolism with prosthetic heart valves is very rare, and the optimal management is not clear. We describe a 44-year-old woman with coronary embolus secondary to prosthetic mitral valve.

CASE REPORT

A 44-year-old woman presented to the emergency department complaining of severe chest pain. Shortly after she arrived in the emergency department, ventricular fibrillation occurred. She was defibrillated with 300 joules and intubated. Sinus rhythm returned after resuscitation.

In physical examination she was unconscious. Her blood pressure was 120/70 mmHg and heart rate was 120 bpm. There was no jugular venous distension. The heart was rhythmic and the prosthetic valve clicks were normal. The lungs were clear, and the arterial pulses were equal bilaterally.

Laboratory findings were an International Normalized Ratio (INR) of 1.25 and an activated partial thromboplastine time of 109.7 seconds. Initial creatine phosphokinase was 101 IU/L, creatinine kinase-MB fraction was 1.8 ng/mL, and troponin I level was 0.03 ng/mL. These cardiac markers were elevated on the follow-up period. Electrocardiogram revealed ST segment elevation in leads V1–V4 suggesting acute anterior MI. Heparin, aspirin, intravenous nitroglycerin, and beta-blocker therapies were initiated. Transthoracic echocardiography showed no evidence of intracardiac thrombus. The mitral prosthetic valve was functioning normally.

She had no risk factors such as diabetes mellitus, hypertension, smoking, hypercholesterolemia, menopause, or premature coronary artery disease in the family according to her past medical history. She had a mitral valve replacement with a St. Jude prosthesis (St. Paul, MN, USA) because of rheumatic heart disease 6 years ago. Coronary angiography had been normal. Oral anticoagulant therapy with warfarin had been initiated after the operation, and she continued the therapy to this day. However, the patient had no INR control for the previous 6 months.

The patient underwent a cardiac catheterization within the 1st hour of arrival. The angiography showed total occlusion of the left anterior descending coronary artery (LAD) after second diagonal (Figure 1). The remainder of the coronary vasculature was normal. A guide-wire was passed through the LAD to disrupt the thrombus, followed by balloon inflation (2.0 × 20 mm). Dissection occurred after the balloon inflation, and stenting (2.5 × 12 mm) was performed. The angiogram performed immediately after angioplasty and stenting showed no reflow of the LAD. After intracoronary diltiazem (2.5 mg), nitroglycerin (200 μg), and saline (40cc) administration, TIMI 3 flow was re-established with no evidence of residual stenosis (Figure 2). After this attempt, electrocardiography normalized completely and the pain

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Correspondence: Mehmet Yazici, Selcuk Univ. Meram School of Medicine, Dept. of Cardiology, Konya, Turkey 42080 (e-mail: myazici61@hotmail.com).

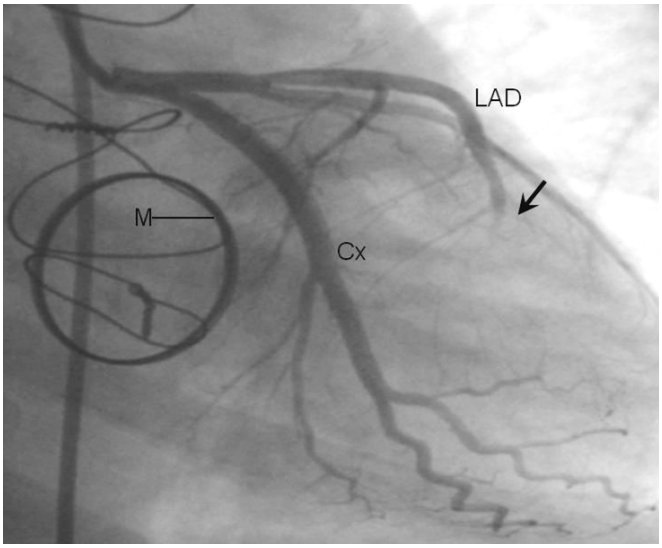

Figure 1. Left anterior descending (LAD) artery occlusion shown in coronary angiography. M indicates mitral prosthetic valve; Cx, circumflex artery; arrow, coronary occlusion.

disappeared. Warfarin dose increased to a target INR level of 3.5.

Transesophageal echocardiography showed no evidence of intracardiac thrombus. The mitral St. Jude prosthetic valve was functioning normally. Results of hypercoagulability evaluation were negative. No event was seen in the hospital care. There were no bleeding complications and no clinical evidence of emboli to other sites. She was discharged on warfarin therapy and tight monitorization of INR was strongly recommended. The patient continued to do well after 4 months and her control INR level was 3.21.

DISCUSSION

The majority of coronary events are related to atherosclerotic coronary artery disease. Less frequently, patients have no evidence of atherosclerotic occlusion on coronary angiography and their MI is related to a nonatherosclerotic process.

The APPROACH study on patients undergoing coronary angiography for acute MI demonstrated that 2.8% of patients had angiographically normal coronary arteries and that these patients have a better prognosis than patients with angiographically verified coronary artery disease [Larsen 2005]. These nonatherosclerotic coronary artery diseases include coronary embolism, vasculitis, dissection, vasospasm, and congenital coronary artery anomalies [Mirza 2003].

Coronary embolism should be suspected when an acute MI occurs in the presence of predisposing conditions for systemic embolism such as valvular heart disease, infective endocarditis, arrhythmias, or intracardiac thrombus [Charles 1983].

Coronary embolism leading to MI was first reported by Virchow in 1856 [Wegner 1958]. Because of the larger caliber of the left coronary system, it is 3 to 4 times more likely to have an embolic event than the right coronary system [Prizel 1978]. If the embolus is small and gets lodged in a large blood vessel, it can be silent and clinically unrecognized. Conversely, a large embolus causing complete occlusion of a coronary artery can lead to fatal MI [Mirza 2003].

Clinical suspicion is very important for diagnosis. The presence of a prosthetic valve replacement represents an independent risk factor for acute coronary syndrome, particularly in the presence of inadequate anticoagulation. Burchart and coworkers confirm that tightly controlled INR values are the most significant predictor of survival following valve replacement surgery [Burchart 2002].

Currently no guideline is present for the effective management of coronary embolism. Several reports have described the intracoronary use of thrombolytics and intravenous administration of abciximab as an adjunct to percutaneous transluminal coronary angioplasty (PTCA) and stenting in patients with coronary embolism [Quinn 1998; Marti 2000]. Immediate PTCA provides a higher rate of patency of the infarction-related artery and lower occurrence of cardiac events as compared to fibrinolysis [Zihlstra 1993]. Recently, several studies have shown that coadjuvant platelet

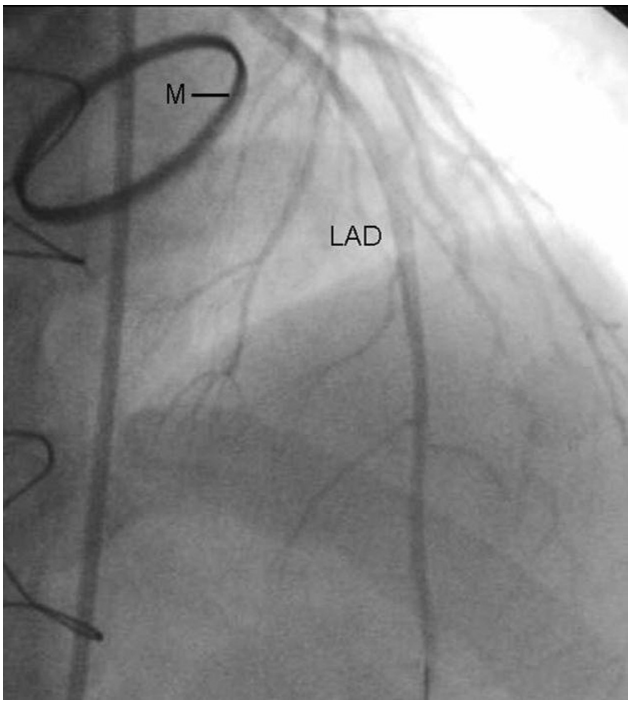

Figure 2. After percutaneous transluminal coronary angioplasty and stenting. LAD indicates left anterior descending artery; M, mitral prosthetic valve.

inhibition with GP antagonists during high-risk, primary, and rescue PTCA reduces the risk of coronary ischemic events [EPIC Investigator 1994; Lefkovits 1996].

We have presented a patient with anterior acute MI caused by embolization from a mitral valve prosthesis due to inadequate anticoagulation. This case demonstrated that coronary embolism may occur even without prosthetic valve thrombus or dysfunction with suboptimal INR levels, and can be successfully treated with PTCA and stenting.

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