

Myocardial infarction related to coronary embolism in a patient with prosthetic mitral valve thrombosis demonstrated by three-dimensional transesophageal echocardiography

Üç boyutlu transözofageal ekokardiyografi ile gösterilmiş protez mitral kapak trombüsü olan bir hastada koroner emboliye bağlı miyokard infarktüsü

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ABSTRACT

The incidence of systemic embolization with mechanical valves is 1% per year. Bacterial endocarditis, valvular heart disease, cardiomyopathies, atrial myxoma, cardiac arrhythmias, and acute coronary syndrome are the causes of coronary embolism. Although coronary embolism due to mechanical valve thrombosis is encountered rarely, it is an important and serious complication. There is no evidence-based effective treatment and management of coronary embolism. Here we report a case of myocardial infarction caused by coronary embolism due to mechanical mitral valve thrombosis.

Keywords: Coronary embolism, myocardial infarction, prosthetic mitral valve thrombosis

ÖZ

Mekanik kapaklarla birlikte sistemik emboli insidansı yıllık %1'dir. Bakteriyel endokardit, kalp kapak hastalığı, kardiyomiyopatiler, atriyal miksoma, kardiyak aritmiler ve akut koroner sendrom koroner embolinin sebeplerindedir. Mekanik kapak trombozuna bağlı koroner emboli nadir görülmesine rağmen, önemli ve ciddi bir komplikasyondur. Koroner embolinin kanıta dayalı etkili bir tedavi ve yönetimi yoktur. Burada mekanik mitral kapak trombozuna bağlı koroner embolinin neden olduğu bir akut miyokard enfarktüsü vakası sunduk.

Anahtar kelimeler: Koroner emboli, miyokard enfarktüsü, protez mitral kapak trombozu

INTRODUCTION

The incidence of systemic embolization with mechanical valves is 1% per year (1). Most cases present with cerebrovascular events. As the incidence of mechanical valve surgery has increased, systemic thromboembolism became an important problem. Although coronary embolism due to mechanical valve thrombosis is encountered rarely, it is an important and serious complication (1). Here, we report a case of myocardial infarction caused by coronary embolism due to mechanical mitral valve thrombosis. Written informed consent was obtained from patient who participated in this study.

CASE PRESENTATION

A 54-year-old woman admitted to emergency unit (EU) with severe retrosternal chest pain radiating to inter-scapular area that had begun two hours ago. Her past medical history had

hypertension for 10 years and she had undergone mitral valve replacement (MVR) 29 St. Jude and tricuspid De Vega annuloplasty for severe mitral regurgitation and moderate tricuspid regurgitation due to rheumatic heart disease 2 years ago. Coronary angiography performed before mitral valve surgery was normal. She was taking warfarin 7.5 mg/day but her international normalized ratio (INR) checked in the EU was below the therapeutic level (INR: 1.15 IU). Electrocardiogram (ECG) showed sinus rhythm, 72/min, first-degree block, left bundle branch block, ST depression, and T wave inversion in lateral and inferior leads. She was monitored and blood samples were drawn 4 hours apart for cardiac markers that showed significant increase. Physical examinations were within normal range except for blood pressure of 170/85 mmHg. Auscultation of the heart showed normal opening and closing clicks of the mitral prosthesis and 2/6 systolic murmur at apex. The lungs were clear to auscultation. High sensitive troponin (hs-troponin)

Figure 1. Coronary angiography shows normal coronary arteries except for hazy appearance in distal circumflex artery

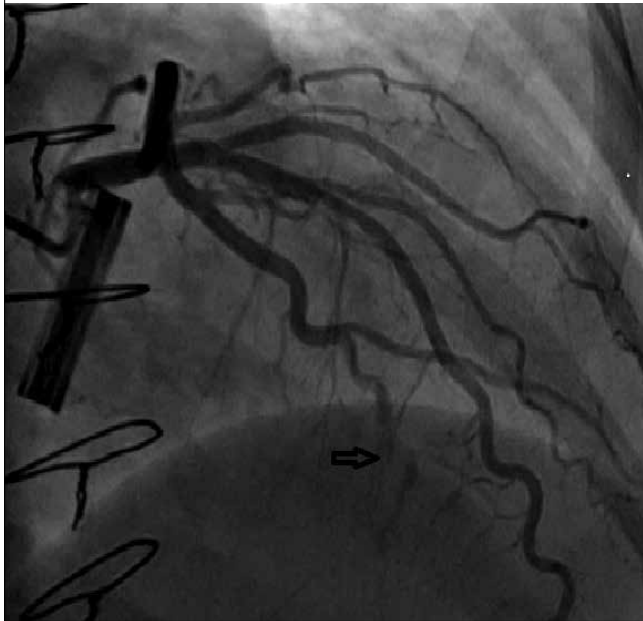
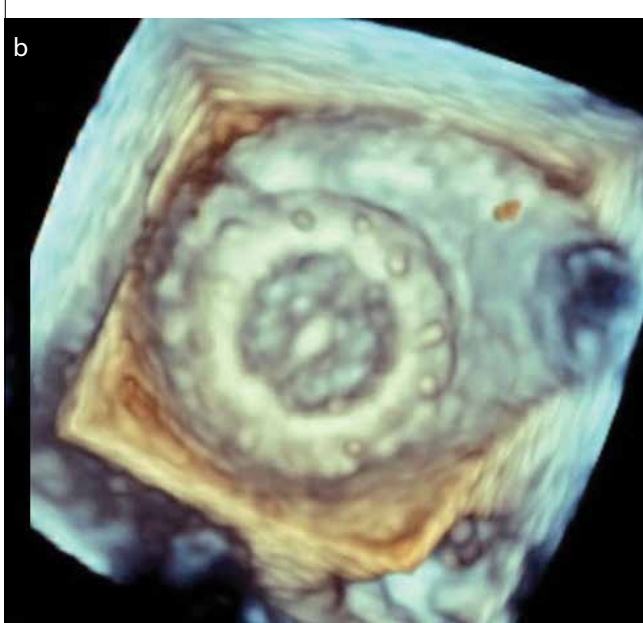
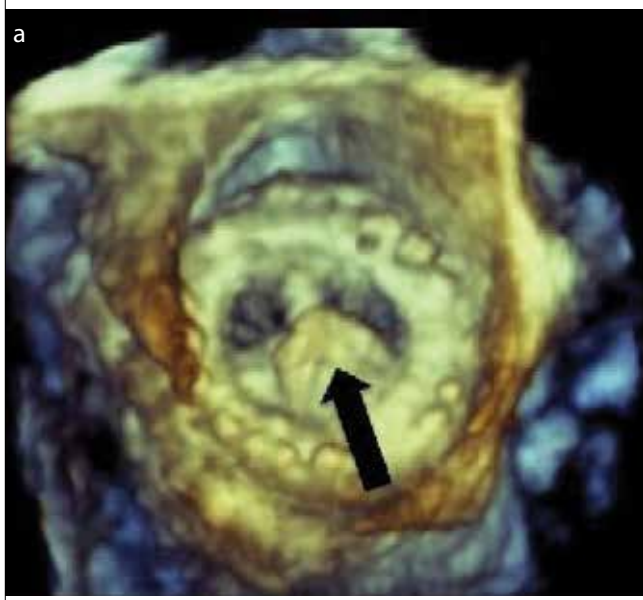


Figure 2. Two-dimensional TEE shows non-obstructing mitral annular thrombosis



Figure 3. a, b. a) Three-dimensional TEE shows non-obstructing mitral annular thrombosis. b) Three months later, control three-dimensional TEE shows normal prosthetic mitral valve with no thrombosis



and creatinine kinase myocardial band (CK-MB) levels were increased (hs-troponin: 216 ng/mL (0-15) and CK-MB: 6 ng/mL (0-5). The patient was admitted to the Coronary Care Unit with diagnosis of non-ST segment elevation myocardial infarction and treatment was started with clopidogrel, acetylsalicylic acid, beta-blocker, angiotensin-converting enzyme (ACE) inhibitor, enoxaparin, and calcium channel blocker (CCB). On the next day, coronary angiography was performed that was normal except for hazy appearance in distal circumflex artery (Figure 1). After coronary angiography, three-dimensional (3D) transesophageal echocardiography (TEE) was carried out showing normal function of prosthetic valve with normal left ventricular systolic function, mild mitral regurgitation, and non-obstructing mitral annular thrombosis (10×12 mm²) (Figures 2 and 3a) without any paravalvular complication suggesting infective endocarditis. There were no signs and symptoms

suggesting infective endocarditis. So, we did not draw blood samples for microorganism culture. Heparin infusion was started for 48 hours, and warfarin was given 10 mg/day. Percutaneous coronary intervention was not performed, and the patient was discharged on warfarin, clopidogrel, acetylsalicylic acid, beta-blocker, ACE inhibitor, and CCB therapy after achieving INR of 2.54 IU. The patient was also educated on prophylaxis measures against infective endocarditis and rheumatic fever. Three months later, control 3D TEE was performed and it showed normal prosthetic mitral valve with no thrombosis and with normal left ventricular systolic function (Figure 3b).

DISCUSSION

Bacterial endocarditis, valvular heart disease, cardiomyopathies, atrial myxoma, cardiac arrhythmias, and acute coronary syndrome are the causes of coronary embolism. Since the introduction of prosthetic valvular surgery, another source for coronary embolism has been introduced. Bjork and Malers (2) published the first case of coronary embolism caused by the mitral prosthetic valve in 1964. Coronary embolism leads to serious problems according to size of the emboli (3). Although smaller embolic materials usually travel to the distal small arterial segments, myocardial infarction and fatal cardiac arrhythmias are less common.

There is no clear guideline for the effective treatment and management of coronary embolism. Kamishirado et al. (4) published a case report of coronary arterial embolism that was not recanalized with 960.000 IU of urokinase therapy. Glycoprotein IIb/IIIa antagonists have been investigated in many clinical studies. The findings in these studies demonstrate that patients undergoing percutaneous transluminal coronary angioplasty benefit from these agents. Coronary thrombi seen during the coronary intervention procedures are reduced or eliminated by glycoprotein IIb/IIIa antagonists. Prolonged infusion with urokinase therapy was found to be useful in eliminating thrombus from saphenous vein grafts. Recently, it has been published that a combined regimen of intracoronary urokinase and intravenous abciximab therapy was successful in achieving fully resolution of the coronary embolism in a patient with mitral and aortic valve replacement (5). Sial et al. (6) demonstrated that TIMI (thrombolysis in myocardial infarction) III coronary blood flow was obtained by extracting the thrombus with an aspiration catheter (Export XT 6F Medtronic) and continued glycoprotein IIb/IIIa antagonist for 24 hours. Yuce et al. (7) showed a similar case of coronary embolism treated with anticoagulation therapy. As in our case, the coronary vessel was not completely occluded with TIMI II antegrade flow, and the thrombus was located in distal circumflex artery with <10% myocardium at risk. So, we preferred medical management with heparin infusion to percutaneous coronary intervention or thrombolytic therapy. The infarction limited itself and hs-troponin peaked at 910 ng/mL and declined gradually. Heparin infusion did not either eliminate the thrombus on mitral valve. But after a course of anticoagulation therapy for about three months, we found that the thrombi were eliminated completely. Although most myocardial infarction in patient taking oral anticoagulation is due to inadequate anticoagulation, it is also possible that myocardial infarction may occur despite normal therapeutic range of INR (8). There is no consensus in the treatment of coronary embolism due to prosthetic valve thrombosis. A low dose (25 mg), slow infusion of tissue plasminogen activator was shown to be effective and safe in a case series of three patients with prosthetic valve thrombosis complicated with coronary embolism and causing non-ST elevation myocardial infarction (9). In another case report, Aykan et al. (10) demonstrated that low dose and prolonged infusion of tissue plasminogen activator was effective in the treatment of coronary embolism related to prosthetic mitral valve thrombosis.

CONCLUSION

Because of the lack of guidelines and the presence of many options in the management and treatment of coronary embolism due to prosthetic valve thrombosis, the decision must be made on individual basis taking into account the size of thrombus, vessel involved, presence of antegrade flow, percentage of myocardium at risk, patient hemodynamics, bleeding risk scores, and the resource available at the institution. 3D TEE is important in determining the precise location and size of prosthetic thrombosis, paravalvular complications in case of endocarditis and therefore may add up to the diagnosis and management of such cases as compared with 2D TEE.

Informed Consent: Written informed consent was obtained from patient who participated in this study.

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