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Case Report

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A Rare Cause of Acute Myocardial Infarction: Coronary Artery Embolism

Selami DEMIRELLI[a1], Sakir ARSLAN[2], Eftal Murat BAKIRCI[3], Arif ARISOY[1], Husnu DEGIRMENCI[1]

[1]*Atatürk University, Department of Cardiology, Erzurum, Turkey*

[2]*Antalya Training and Research Hospital, Department of Cardiology, Antalya, Turkey*

[3]*Bayburt State Hospital, Department of Cardiology, Bayburt, Turkey*

ABSTRACT

A 49-year-old woman with a history of hypertension, diabetes mellitus and aortic valve replacement five years ago, admitted to our outpatient clinic with shortness of breath. Transesophageal echocardiography revealed a 5x2 mm thrombus on the anterior face of prosthetic aortic valve. A thrombolytic therapy (TT) was initiated but it was unsuccessful. In the tenth day of follow-up, she experienced chest pain with ST segment elevations through V2-V6 derivations on her ECG. With the diagnosis of acute myocardial infarction, the patient was taken into the catheterization laboratory. In the coronary angiography, the mid segment of left anterior descending artery was completely occluded by a thrombus. Percutaneous transluminal coronary angioplasty was performed but it was failed. After then TT was started again. The second TT has become successful both in terms of coronary revascularization and resolusion of valve thrombosis.

Key words: *Coronary embolism, Aortic valve prosthesis, Myocardial infarction*

ÖZET

Akut Miyokard İnfarktüsünün Nadir Bir Nedeni: Koroner Arter Embolisi

Beş yıl önce aort kapak replasmanı yapılan, diyabet ve hipertansiyon hikayesi olan, kırk dokuz yaşındaki bayan hasta kliniğimize nefes darlığı şikayeti ile başvurdu. Transözefajial ekokardiyografide aortik protez kapağın ön yüzünde 5×2 mm trombüs ile uyumlu görüntü izlendi. Trombolitik tedavi uygulandı ancak başarısız olundu. Takibin onuncu gününde hastada göğüs ağrısı ile birlikte EKG'de V2-V6 derivasyonlarında ST segment elevasyonu gözlendi. Akut miyokard infarktüsü tanısı ile yapılan koroner anjiyografide, sol inen arter orta segment trombüs ile tıkalıydı. Perkütan translüminal koroner anjiyoplasti denendi ancak başarısız olundu. Hastaya ikinci kez trombolitik tedavi uygulandı. İkinci kez uygulanan trombolitik tedavinin, hem koroner revaskülarizasyon hem de kapak trombozu açısından başarılı olduğu gözlendi.

Anahtar Sözcükler: *Koroner emboli, Protez aort kapak, Miyokard infarktüsü*

Even though many of myocardial infarction occur as a result of atherosclerotic plaque rupture and enclosed thrombus on to it, numerous pathological circumstances other than atherosclerosis may lead to myocardial infarction. Congenital coronary artery anomalies, coronary arteritis and coronary artery embolism are among the causes of nonatherosclerotic myocardial infarction (1).

Occurrence of thrombosis has been reported on left side mechanical prosthesis valves in proportion between 0.5%- 8%, on the other hand the incident ratio has been reported as 20% on tricuspid valve prosthesis (2, 3). Coronary artery embolism connected with prosthesis valve thrombosis is a rare but life-threatening complication. We wanted to report a case of acute anterior myocardial infarction depending on coronary embolus in a patient having aortic prosthesis valve thrombosis.

CASE REPORT

A forty-nine years old, female patient admitted to our clinics due to ever increasing respiratory disorder started before 3 or 4 month ago. The patient was in NHYA functional class III. It was understood from her history revealed that she underwent operation of aortic valve replacement (st jude valve, size 19 Mechanical Valve) five years ago and she has had both hypertension and diabetes mellitus for ten years. Blood pressure, pulse and fever was measured during her physical examination as 120/80 mmHg, 85 beat/min and 36.5 ˚C, respectively. In the course of auscultation, mechanical valve noise in the focus of aorta as well as 4/6° systolic ejection murmur was heard. Other systemic examinations were inherent. Sinus rhythm was seen on electrocardiogram (ECG). In laboratory tests, hemoglobin, hematocrit, white blood cells, erythrocyte sedimentation rate and international

[a] Corresponding Adress: Dr. Selami DEMIRELLI, Atatürk University, Department of Cardiology, Erzurum, Turkey
e-mail: demirelli23@yahoo.com

normalized ratio(INR) were 11gr/dl, 34, 16200 mm^3, 20/h and 2, respectively. The systolic functions of left ventricle (left ventricular ejection fraction (LVEF) was 65%) were assessed as normal on transthoracic echocardiography (TTE). However, aortic valve gradients were found as increased (peak/mean gradient was 83/50 mmHg). On transesophageal echocardiography (TEE), performed for better assessment of prosthesis valve during patient follow-up, a mobile, dimensions of 5x2 mm image matching with thrombosis was seen on anterior of the aortic face of prosthesis valve (Figure 1). Surgical operation was offered to the patient with the diagnosis of obstructive prosthetic aortic valve thrombosis, but the patient did not accept it. Then, thrombolytic treatment (TT) was applied to the reevaluated patient (100 mg rt-PA iv infusion was enforced in the course of 2 hours). On the TTE after 24 hours of TT, no significant changes were found on aortic valve gradients, and thrombosis was still continuing. Consequently, the TT was accepted as unsuccessful. On tenth day of her hospitalisation typical chest pain and ST segment elevation in V2-6 derivations had occurred. With diagnosis of myocardial infarction, the patient was immediately taken into the coronary angiography (CAG). On CAG, the mid segment of left anterior descending artery was completely occluded. Furthermore, a matching image with thrombosis existed in this part (Figure 2a). Other coronary arteries were observed as normal. Percutaneous balloon angioplasty was applied to this part. After the balloon angioplasty, it was monitored that thrombosis was still continuing (Figure 2b). Therefore, it was decided to give TT once again to the patient. For that purpose, the patient was taken into the coronary intensive care unit and applied TT (rt-PA was started with 15 mg bolus and then applied in accordance with 0.75 mg/kg in 30 minutes and 0.5 mg/kg in 60 minutes, as iv infusion). Afterwards, ST segment depressions on the electrocardiogram were more than %50, the patient's angina was relief and the enzyme peak was occured and TT was accepted as successful. On TTE, decrease in aortic valve gradients (peak/mean gradient was 45/22 mmHg), LV wall movement disorder (apical hypokinesis) and slight decrease in the functions of LV (LVEF was 55%) were detected. Since the patient did not accepted the control TEE, the anticoagulant treatment was arranged that INR would be between 3.5 and 4.5 in addition to giving 100 mg aspirin and the patient was discharged. During two years of follow-up, no new coronary incident or valve problem came into existence.

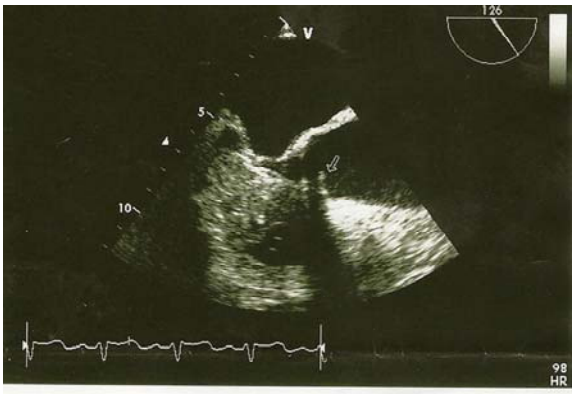

Figure 1. *Transesophageal echocardiography showing thrombosis on prosthesis aortic valve*

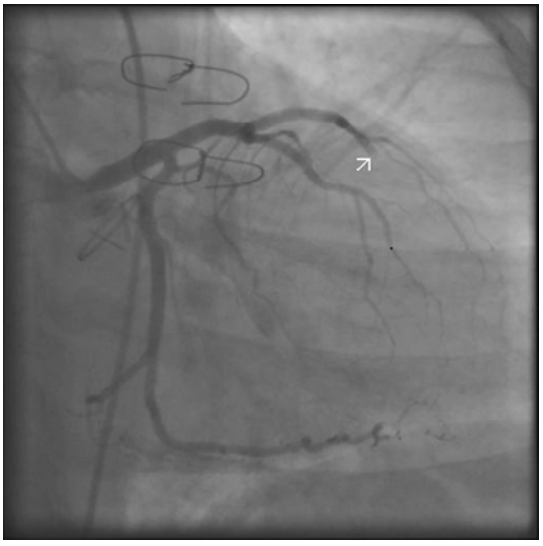

Figure 2a. *Coronary angiography showing thrombosis on left anterior descending artery*

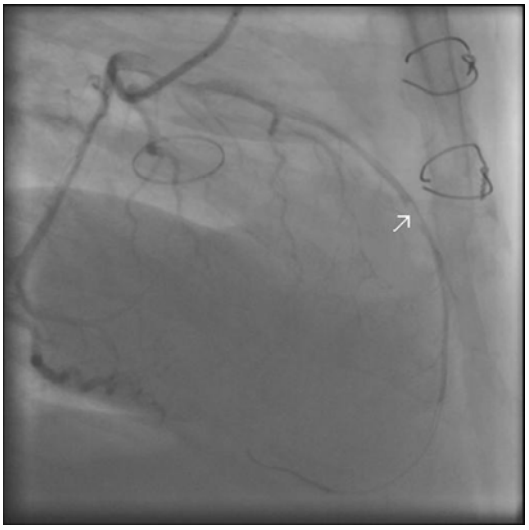

Figure 2b. *Thrombosis continues on left anterior descending artery after percutaneous coronary intervention*

DISCUSSION

Coronary artery embolism is rarely encountered and it arises frequently from cardiac valves (4). The most frequent reasons are septic emboli depend on infective endocarditic and prosthetic valve thrombosis emboli.

Prosthesis valve thrombosis is observed on the left side mechanical prosthesis valves in proportion between 0.5%- 8%. Valve malfunction and atrial fibrillation increase the risk of thromboembolia. In currently used bileaflet prosthesis valves, the risk of thromboembolia is similar with bioprosthesis valves- 1.5%; patient/year (5). Coronary emboli restrain typically distal epicardial and intramural branches of left descending artery (6). Consisted acute occlusion leads to severe myocardial damage since these patients have not any chronic atherosclerosis.

Prosthetic valve obstruction could be treated medically or surgically. All treatment types have high risks. In 2006 ACC/AHA guideline; for treatment of left side mechanical prosthesis valves (MPV) thrombosis having big thrombosis burden, surgical treatment is offered as the first option (7). Thrombolytic treatment, however, is suggested to the patients having small thrombosis burden that their NYHA functional classes are I and II or to the patients having small thrombosis burden that their NYHA functional classes are III and IV, but surgically-high compromised or inappropriate for surgery. The success rate of thrombolytic treatment on left side MPV has been reported between 67% and 90%. As the functional class of our case was class III, it was initially offered surgical treatment to the patient. Yet, the patient did not accept it. Hereupon, it was started to thrombolytic treatment (TT). Surprisingly, coronary embolus has developed on the tenth day of unsuccessful TT. The reason could be that some parts of thrombosis depending on TT have broken to pieces due to dissolving in the course of time. On the other hand, percutaneous intervention for myocardial infarction developing as secondary to coronary embolus has become unsuccessful. On the contrary, TT has become successful. This ineffectiveness may result from that the only existing pathology is thrombosis instead of coronary lesion.

Applied TT because of developing coronary embolus in our patient has become successful both in terms of coronary revascularization and with regard to valve thrombosis. For this reason, trying the same treatment in the cases of unsuccessful TT on valve thrombosis may be considered one more time.

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