Percutaneous effective closure of severe aortic prosthetic paravalvular leak using Amplatzer duct occluder device with the guidance of 3D TEE

**Introduction**

Aortic paravalvular leaks are well-known complications of prosthetic valve replacement. They are asymptomatic and small, but sometimes may cause symptoms due to severe regurgitation or hemolysis. Medical therapy is limited, while operation causes significant mortality. Percutaneous transcatheter closure techniques, potentially offer symptomatic relief. We describe the use of an Amplatzer duct occluder device with the guidance of 3-dimensional transesophageal echocardiography (3DTEE) in a patient with severe aortic prosthetic paravalvular leak resulting in effective closure.

**Case Report**

A 33-year-old man applied to hospital with shortness of breath. He had previous history of aortic valve replacement due to severe aortic stenosis. We detected diastolic murmur along left sternal border on physical examination. According to blood test results, he had anemia (Hemoglobin 10.9 g/dl) and elevated bilirubin levels. Transthoracic echocardiography revealed an increased left ventricular wall thickness (interventricular septum diastolic thickness 13 mm) and normally functioning left ventricle (ejection fraction 58%, diastolic internal diameter 48 mm). However, there was a moderate degree leak related to prosthetic aortic valve, and hence, we decided to perform TEE (Fig. 1). A Philips I33 machine (Andover, USA) equipped with X7-3 probe was used for this purpose, which revealed a posteriorly located moderate to severe aortic paravalvular leak. We measured paravalvular defect diameter as 5 mm by activating 3D zoom and viewing resulting image with grid lines (Fig. 2, 3). We tried to explain possible treatment options to the patient. He opted for percutaneous closure. The procedure was undertaken under general anesthesia. An 8 Fr sheath was placed in the right femoral artery, and then the patient was anticoagulated with heparin. A 0.0035 inch wire was placed across the defect into the left ventricle under 3D TEE guidance. Then, the delivery sheath was advanced over the wire and the Amplatzer duct occluder was positioned again with 3D TEE guidance (Fig. 4). At the 2-month follow-up, he was free of symptoms and TEE showed occluder in a good position with mild degree residual leakage.

**Discussion**

Aortic paravalvular leaks are generally due to inadequate apposition between sewing ring and native aortic valve (1). It develops more commonly in patients with heavy annular calcification and localized infections (2, 3). Although majority of paravalvular leaks (PVL) are asymptomatic, clinically significant paravalvular leaks may develop in up to 5% of patients with prosthetic valves (1) which may cause progressive left ventricular dilatation, congestive heart failure and hemolysis. Surgical reoperation is usually required in such a patient but higher operative mortality and morbidity should also be expected (4). As an alternative, experience related to percutaneous closure of paravalvular leaks has
Acute myocardial infarction associated with Captagon use

Kaptagon kullanımı ile ilişkili akut miyokart enfarktüsü

Introduction

Although acute myocardial infarction (AMI) is usually a disease of older ages, it may be seen in younger ages. In young cases of AMI non-atherosclerotic coronary artery disease (CAD), thrombophilia, illicit drug abuse, premature atherosclerosis must be considered. Illicit drug abuse such as amphetamines and cocaine has been widely recognized as a causative agent in AMI. Abuse of fenethylline as the brand Captagon may also cause AMI. Today, most of the counterfeit Captagon tablets do not contain fenethylline, but combination of substances that mimic the effects of the original molecule.

This report describes a young male who presented with an acute anterior MI after taking a tablet of Captagon and discuss cardiovascular effects of amphetamines and substances that mimic the effects of the amphetamines.

Case Report

A 21-year-old male was admitted to the emergency department of local State Hospital with chest pain. He collapsed during initial examination, and cardiac monitorization showed ventricular fibrillation, and immediate, successful defibrillation was carried out. An electrocardiogram (ECG) showed a widespread ST segment elevation in anterolateral leads with reciprocal changes in inferior leads confirming AMI. He was given 300 mg acetyl salicylic acid, oxygen, unfractionated heparin bolus (5000 units) and an infusion of heparin was initiated. Then, he was transferred to our center for an emergency Percutaneous Coronary Intervention (PCI). On admission, he was confused with having strange behaviors and acting aggressively. Information about the patient’s past medical history was taken from his relatives and revealed no any cardiovascular risk factors except smoking. On physical examination, his blood pressure was 115/70 mmHg, heart rate was 90 beats per minute. Blood tests showed normal liver enzymes, high nitrate level, and normal INR. On the other hand, there was no any disease, and cranial CT was normal. A primary PCI was deferred, so he was agitated and had normalization of ST changes on ECG (Fig. 2).

References