

Mitral valve perforation from aortic insufficiency

Aort yetmezliğinden oluşan mitral kapak delinmesi

A 60-year-old man with a history of endocarditis presented with congestive heart failure New York Heart Association class III symptoms. He did not have any signs or symptoms of microembolic phenomenon and the blood cultures were negative. A transesophageal echocardiogram showed an eccentric jet of mitral regurgitation (MR) from a perforation in the anterior mitral valve leaflet (MVL) (Fig. 1, Video 1). There were no visible vegetations however; there was an eccentric jet of aortic insufficiency (AI) pointing directly against the point of anterior MVL perforation (Fig. 2, Video 2). Thus the primary lesion was AI from previous endocarditis with secondary involvement of the MV. The patient underwent mechanical mitral and aortic valve replacement.

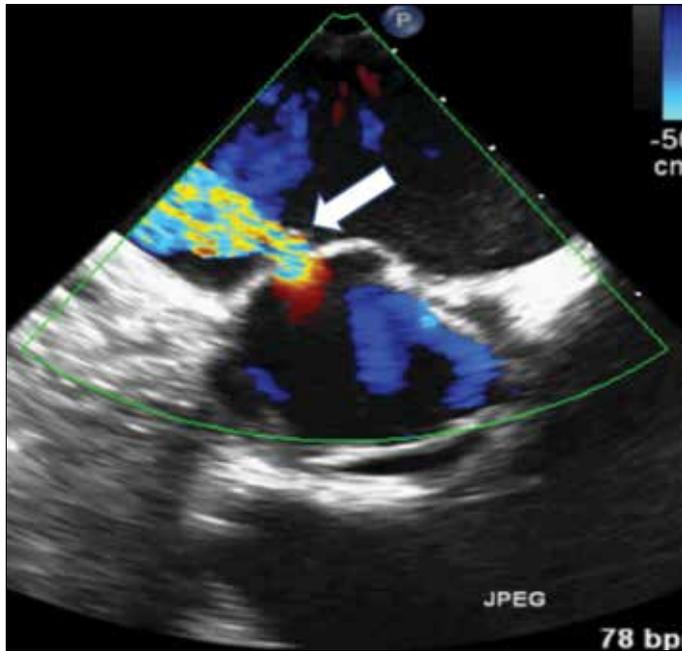


Figure 1. Transesophageal echocardiogram demonstrating an eccentric regurgitant jet (white arrow) through the perforated anterior mitral valve leaflet

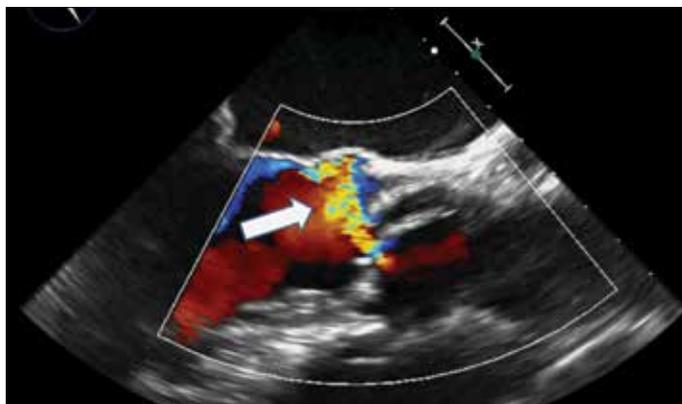


Figure 2. Transesophageal echocardiogram showing aortic regurgitation jet (White Arrow) pointing directly towards the site of perforation of mitral leaflet

Intra-operative cultures of valve tissue were also negative and the pathology was consistent with fibrosis.

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Video 1. Transesophageal echocardiogram demonstrating an eccentric regurgitant jet (white arrow) through the perforated anterior mitral valve leaflet

Video 2. Transesophageal echocardiogram showing aortic regurgitation jet (White Arrow) pointing directly towards the site of perforation of mitral leaflet

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Giant aneurysm with thrombosis refractory to medical therapy due to Kawasaki disease

Kawasaki hastalığı nedeniyle gelişen, tıbbi tedaviye dirençli trombüslü dev anevrizma

A diagnosis of incomplete Kawasaki disease was made in a 10-month-old female infant on the basis of fever, widespread erythematous rash of both lower extremities, swelling in both hands and feet (Fig. 1) and aneurysm with 5.5 mm diameter in the left anterior descending artery (LAD) on echocardiography (Fig. 2). Intravenous immunoglobulin 2 g/kg/day 12 hours of infusion, aspirin 80 mg/kg/day in 4 doses and dipyridamole 6 mg/kg/day in 3 doses was started. After 3 days, a 9 mm giant aneurysm was observed in LAD. Warfarin (0.2 mg/kg/day) was added to the treatment. Thrombus was observed within the giant aneurysm (6.3x7.9 mm) on the 8th day (Fig. 3, Video 1). Unfractionated heparin 100 U/kg bolus followed by 14 units/kg/h infusion and tissue plasminogen activator (t-PA) 0.05 mg/kg/h infusion was initiated. Thrombus disappeared on the fourth day of this treatment (Video 2). Low molecular weight heparin (LMWH) 2 mg/kg/day was initiated while decreasing doses of heparin and t-PA. Thrombus regressed successively with therapy in three times. Subsequently, giant aneurysm reached 11 mm diameter and a new thrombus was noted (Fig. 4, Video 3). Due to high level of troponin I and ST elevation on ECG (V1-V4), t-PA infusion was started again. Clopidogrel 1 mg/kg/dose and propranolol 2 mg/kg/day was added. Patient was referred to cardiac surgery center for coronary artery bypass due to refractory medical therapy.

Kawasaki disease is the most important cause of acquired heart disease in children. Coronary artery aneurysm may develop in about 15-25% of untreated patients. Moreover, patients with giant coronary aneurysm are more likely to encounter myocardial infarction and sudden cardiac death.