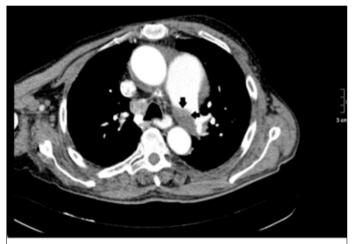


Figure 1 Transthoracic echocardiography view of an aneursym of pulmonary artery together with aneurysm of ascending aorta, pericardial effusion and right ventricular dilatation



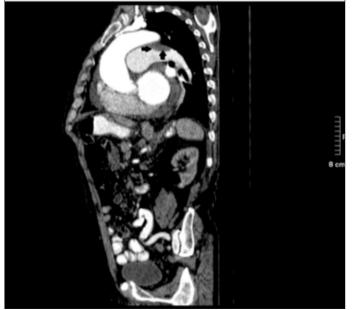


Figure 2 A-B. Multislice CT demonstrated main pulmonary artery of 6.1 cm, left pulmonary artery of 3.3 cm and right pulmonary artery of 3.6 cm. There was a massive (1.3 cm) thrombus in the lumen of the aneurysmatic left pulmonary artery

led atrial fibrillation and right ventricular strain pattern. Transthoracic echocardiography showed PAA together with aneurysm of ascending aorta, pericardial effusion and right ventricular dilatation (Fig. 1). Multislice computerized tomography demonstrated main, left and right pulmonary arteries with diameters of 6.1 cm, 3.3 cm, and 3.6 cm respectively. There was a massive (1.3 cm) thrombus in the lumen of the aneurysmatic left pulmonary artery (Fig. 2A-B). The medical treatment of patient consisted of warfarin 5 mg/day, metoprolol 50 mg/day and furosemid 40 mg/day. The functional capacity of patient showed improvement after treatment and two- year follow-up was uneventful. In our case, pulmonary dilatation developed due to the pressure overload on pulmonary circulation caused by PHT. There is no definitive therapeutic approach for PAA. However, low-pressure aneurysms without PHT are usually treated medically; aggressive surgical management is recommended for patient with high risk of dissection or laceration of high-pressure PAA with underlying PHT.

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Resolution of obstructive prosthetic valve thrombosis after coronary embolism 🔊

Koroner emboli sonrası düzelen tıkayıcı protez kapak trombüsü

Coronary embolism is an uncommon but serious complication of prosthetic valve thrombosis. During the course of prosthetic valves, myocardial infarction (MI) due to coronary embolism can be seen as a presentation or during treatment of valve thrombosis.

A 35-year-old man, with a history of bileaflet mechanical aortic and mitral prosthetic valve replacement 12 years ago, presented with dyspnea. He has not taken warfarin for six months. The patient's INR was measured as 1.3. Transthoracic echocardiographic examination

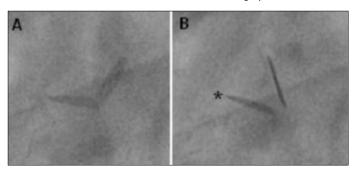


Figure 1. Aortic prosthetic valve, one leaflet (asterisk) is stuck (A-diastole, B-systole)

showed severely increased transprosthetic aortic gradients (mean: 72 mmHg) and normal transprosthetic mitral gradients. Although aortic prosthetic valve could not be adequately visualized, transesophageal echocardiographic examination revealed decreased valve motions. In fluoroscopic examination one leaflet of aortic valve was severely restricted (Fig. 1, Video 1. See corresponding video/movie images at www.anakarder.com). Intravenous heparin and oral warfarin treatment was started. After four days of admission, the patient complained of severe chest pain. His electrocardiogram revealed acute inferior MI. His INR level was 3.6. Coronary angiography revealed occlusion of the

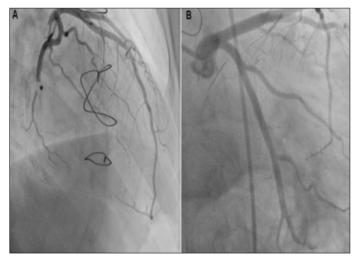


Figure 2. A-Occlusion of the distal circumflex artery with embolus (asterisk), B-Circumflex artery after successful percutaneous transluminal coronary angioplasty

B

Figure 3. Views of normally functioning prosthetic valve after coronary embolism (A-Diastole, B-Systole)

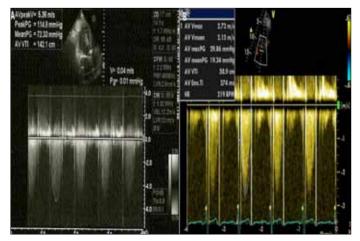


Figure 4. Doppler echocardiographic views of aortic gradients: A-during the valve thrombosis, B-after coronary embolism

distal circumflex artery, which appeared to be an embolus (Fig. 2, Video 2. See corresponding video/movie images at www.anakarder.com). The lesion was treated with balloon angioplasty with successful result. Amazingly, during percutaneous coronary intervention, fluoroscopic imaging showed normal motion of prosthetic aortic valve (Fig. 3, Video 3. See corresponding video/movie images at www.anakarder.com), so further treatment of obstructed aortic valve became unnecessary. Control transthoracic echocardiography revealed decrease in transprosthetic aortic gradients (mean: 19 mmHg) (Fig. 4). The patient had an uneventful recovery and was discharged on warfarin anticoagulation with a therapeutic INR of 3.5 as well as antiplatelet therapy with aspirin and clopidogrel.

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Role of cardiovascular magnetic resonance in the diagnosis of arrhythmogenic right ventricular cardiomyopathy/dysplasia with left ventricular involvement

Kardiyovasküler manyetik rezonans'ın sol ventrikül tutulumlu aritmojenik sağ ventrikül kardiyomiyopati/ displazi tanısındaki yeri

A 55-year-old female patient presented to Rapid Access Chest Pain Clinic with symptoms of chest pain on exertion. Her resting electrocardiogram showed precordial T wave inversion. A presumptive diagnosis of coronary artery disease led to an exercise stress test, which was non diagnostic with pseudo-normalisation of the T waves. Echocardiography revealed mild inferolateral hypokinesia of the left ventricle (LV) with normal ejection fraction (EF) and mild impairment of right ventricular (RV) systolic function. Nuclear myocardial perfusion scan suggested a small inferolateral infarct with some reversible ischemia. The patient was referred for cardiovascular magnetic resonance (CMR) imaging to assess ventricular function and the possibility of myocardial infarction and ischemia as the cause of her symptoms. Cine CMR images revealed abnormalities of both ventricles (Fig. 1 A- B, Video 1- 2. See corresponding video/movie images at www.anakarder.com). The LV was dilated with EF at the lower range of normal (LV EF 58%). There were regional hypokinesia in the inferolateral wall and the apex of LV. Right ventricle was also dilated and systolic function was impaired (RV end-diastolic volume 120 ml/m² and RV EF 48%). There were regional hypokinetic and dyskinetic areas in the RV free and inferior walls. CMR myocardial perfusion study did not show any inducible ischemia. Late gadolinium images revealed subepicardial to mid-wall enhancement at the inferolateral LV wall (corresponding to the proba-