

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

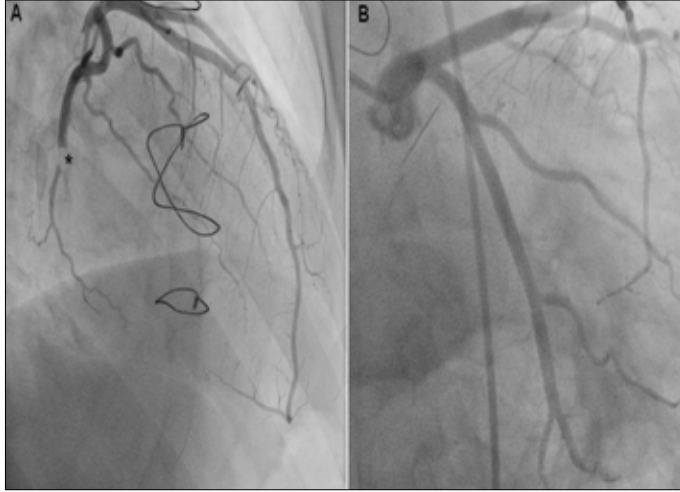


Figure 2. A-Occlusion of the distal circumflex artery with embolus (asterisk), B-Circumflex artery after successful percutaneous transluminal coronary angioplasty

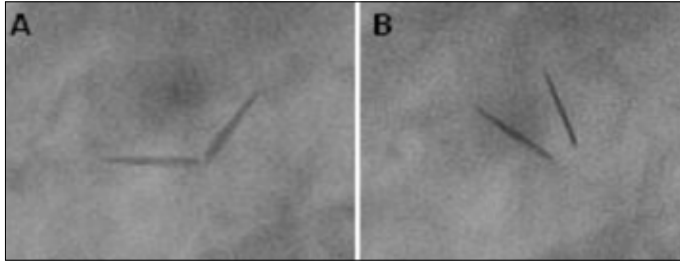


Figure 3. Views of normally functioning prosthetic valve after coronary embolism (A-Diastole, B-Systole)

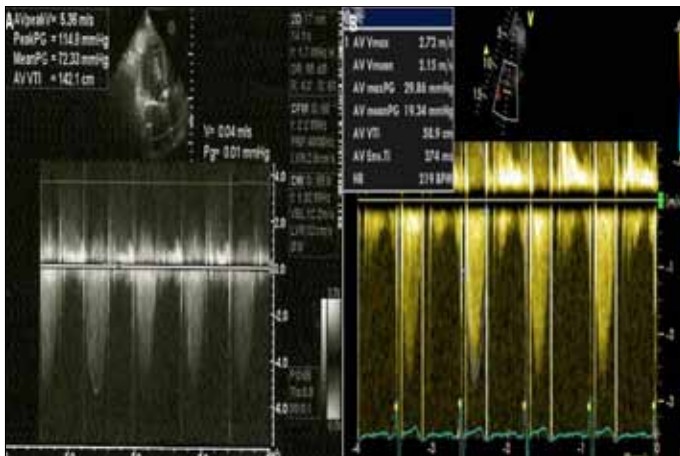


Figure 4. Doppler echocardiographic views of aortic gradients: A-during the valve thrombosis, B-after coronary embolism

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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Available Online Date/Çevrimiçi Yayın Tarihi: 10.01.2012

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doi:10.5152/akd.2012.028

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-

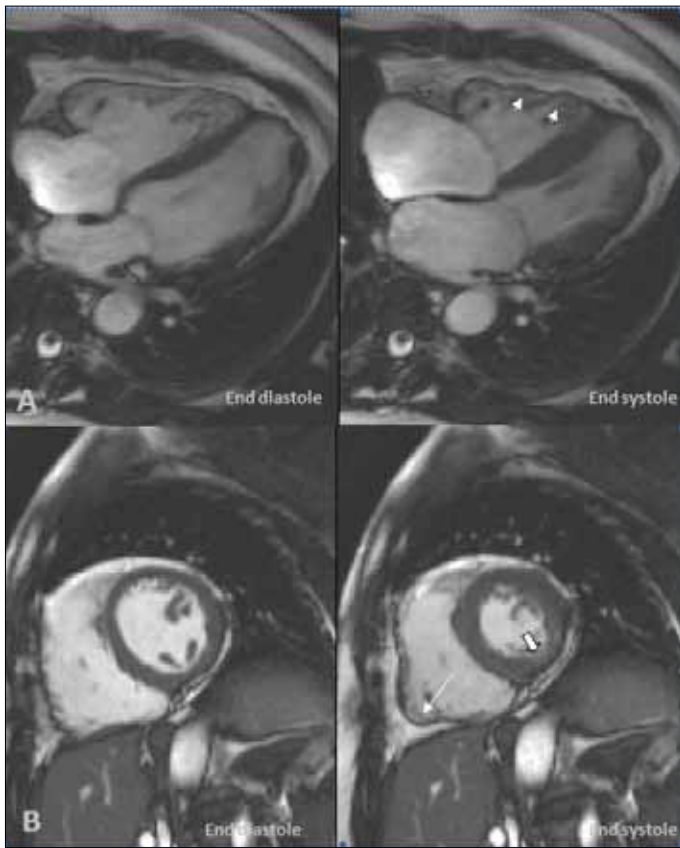


Figure 1. (A) Four-chamber end-diastolic and end-systolic frames showing regional hypokinesia of the lateral wall and apex of LV. RV is dilated and regional areas of hypokinesia at the free wall were noted (arrowheads). (B) Short-axis end-diastolic and end-systolic frames revealing hypokinesia of the inferolateral wall of LV (thick arrow). Dyskinetic area in the RV inferior wall is also seen (thin arrow)

LV - left ventricle, RV - right ventricle

ble small infarct area noted by the nuclear perfusion scan) (Fig. 2). CMR features were consistent with a cardiomyopathic process involving both ventricles, the most likely diagnosis being arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC) with LV involvement. The probability of an infarction was excluded in the absence of a subendocardial scar. The capability of CMR for tissue characterisation and better evaluation of the ventricular function, particularly of the RV enabled to exclude infarction and shed light on the diagnosis of a completely different cardiac pathology in this patient.



Figure 2. Late gadolinium image showing extensive subepicardial to mid-myocardial enhancement -denoting fibrosis- in the inferolateral LV wall (arrow)

LV - left ventricle

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Available Online Date/Çevrimiçi Yayın Tarihi: 10.01.2012

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doi:10.5152/akd.2012.029