

Giant left ventricular pseudoaneurysm detected three years after myocardial infarction

Miyokard infarktüsünden üç yıl sonra saptanan dev sol ventrikül psödoanevrizması

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A 70-year-old male patient presented with symptoms of heart failure three years after having myocardial infarction. Transthoracic echocardiography revealed severely compromised left ventricular systolic function (ejection fraction 20%) and a large left ventricular pseudoaneurysm. The diagnosis was confirmed by computed tomography. The size of the pseudoaneurysm sac was 11x9 cm. At surgery, the pseudoaneurysmal sac was resected and a defect of 3.5x4 cm was detected in the anterolateral wall of the left ventricle. The defect was repaired by the remodeling ventriculoplasty method of Dor. The patient whose general condition improved through intensive medical treatment was discharged with medications for heart failure and coronary artery disease on the 15th postoperative day.

Key words: Aneurysm, false/surgery; heart aneurysm/surgery; myocardial infarction/complications.

Left ventricular (LV) pseudoaneurysm is a rare and extremely fatal complication of acute myocardial infarction.^[1] Because of its propensity to rupture, it has been generally recommended that it should be repaired surgically.^[2,3] A few patients manage to survive this disorder.

In this report, we presented a patient who had a long survival despite the presence of a giant LV pseudoaneurysm.

CASE REPORT

A 70-year-old male patient was admitted to our clinic with symptoms of heart failure (NYHA class II-III). He had a history of myocardial infarction that occurred three years before his admission. He had a complaint of progressively increasing shortness of breath that had developed over the past year. On

Üç yıl önce geçirilmiş miyokard infarktüsü öyküsü olan 70 yaşında erkek hasta kalp yetersizliği semptomları nedeniyle kliniğimize başvurdu. Transtorasik ekokardiyografide ciddi sol ventrikül disfonksiyonu (ejeksiyon fraksiyonu %20) ve büyük bir sol ventrikül psödoanevrizması saptandı. Tanı bilgisayarlı tomografiyle doğrulandı. Psödoanevrizma kesesinin boyutu 11x9 cm olarak ölçüldü. Psödoanevrizma kesesi cerrahi olarak çıkarıldı ve sol ventrikülün anterolateral duvarında 3.5x4 cm ölçülerinde açıklık saptandı. Bu defekt Dor ventriküloplastisi yöntemiyle tamir edildi. Genel durumu yoğun tıbbi tedaviyle düzelen hasta, ameliyattan 15 gün sonra, kalp yetersizliği ve koroner arter hastalığına yönelik tedavi verilerek taburcu edildi.

Anahtar sözcükler: Psödoanevrizma/cerrahi; kalp anevrizması/cerrahi; miyokard infarktüsü/komplikasyon.

examination, he had normal sinus rhythm with 83 beats/min and normal blood pressure with 100/60 mmHg. The first and the second heart sounds were normal. He also had a third heart sound and a low-intensity systolic murmur at the apex. Chest examination showed bilateral crepitant rales in basal lung regions. Electrocardiogram (ECG) showed normal sinus rhythm as well as nonspecific intraventricular conduction delay. Specific biochemical markers of severe myocardial ischemia or acute myocardial infarction (cardiac protein troponin I, creatine kinase isoenzyme MB mass) were found to be within normal limits in three consecutive blood samples. Transthoracic echocardiography revealed a large pseudoaneurysm in the anterolateral wall of the left ventricle, along with severely compromised left ventricular systolic function (ejection fraction 20%) and

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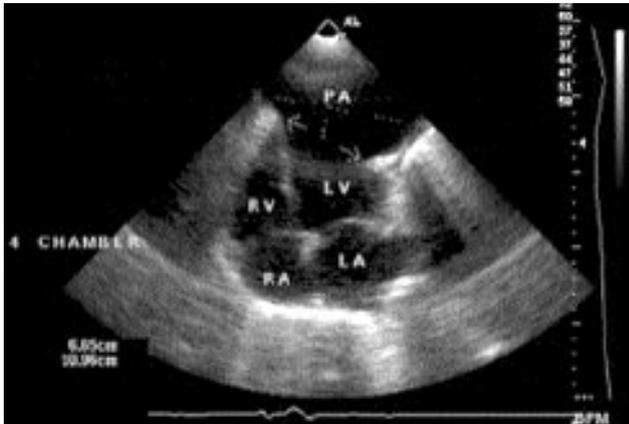


Fig. 1. The appearance of the pseudoaneurysm on a transthoracic echocardiogram. PA: Pseudoaneurysm; RV: Right ventricle; RA: Right atrium; LV: Left ventricle; LA: Left atrium; Arrows: The neck of the pseudoaneurysm.

mild mitral valve regurgitation (Fig. 1). The diagnosis was confirmed by computed tomography. The pseudoaneurysm sac was measured as 11x9 cm, and the pseudoaneurysm was connected to the ventricle by a narrow neck (3.5 cm) (Fig. 2). Coronary angiography revealed occlusion (85%) only in the mid-portion of the left anterior descending coronary artery (LAD). The other coronary arteries were normal. Ventriculography showed a large pseudoaneurysm. The cause of the pseudoaneurysm appeared to be occlusion in the LAD.

The pseudoaneurysmal sac was surgically resected. A defect, 3.5x4 cm in size, was detected in the anterolateral wall of the left ventricle and was repaired by the remodeling ventriculoplasty method of Dor (Fig. 3). As the LAD was not suitable, coronary artery bypass could not be performed.

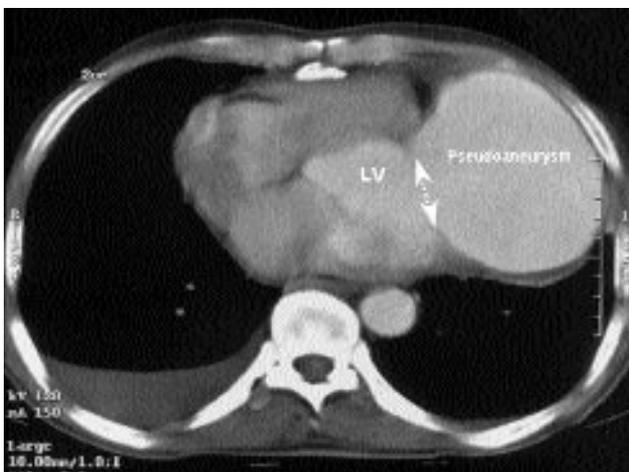


Fig. 2. Pseudoaneurysm and its narrow neck (double head arrow) on a computed tomography scan. LV: Left ventricle.

Histopathological examination of the resected material confirmed the diagnosis of pseudoaneurysm and showed no myocardial elements in the aneurysmal wall. On transthoracic echocardiography performed on the seventh day after the operation, left ventricular dysfunction (ejection fraction 27%) and bilateral pleural effusion were detected. The patient's general condition improved through intensive medical treatment and he was discharged on the 15th postoperative day with medications for heart failure and coronary artery disease.

DISCUSSION

Left ventricular pseudoaneurysm is defined as a rupture of the myocardium. It contains epicardial adhesions or the epicardial wall. It is less common than a true aneurysm. Pseudoaneurysms mostly occur as a complication of myocardial infarction^[4-7] and cardiac surgery,^[8,9] especially after mitral or aortic valve replacement or after surgical repair of true cardiac aneurysms. They also occur after infectious endocarditis^[10] or chest trauma.^[11] Those occurring after myocardial infarction are typically diagnosed within six months after the infarction although intervals as long as 12 years have been reported.^[6] Factors predisposing to cardiac rupture after an acute myocardial infarction include age over 60 years, female sex, previous hypertension, first acute transmural infarct, coexisting pericarditis, high CK-MB levels (≥ 150 IU/l), lack of coronary artery collateral vessels in the infarction area, and thrombolytic therapy given for more than seven hours after the onset of symptoms.^[12]

The contribution of clinical signs and symptoms to the diagnosis of a pseudoaneurysm is very limited. Yeo et al.^[10] reviewed 52 patients with cardiac

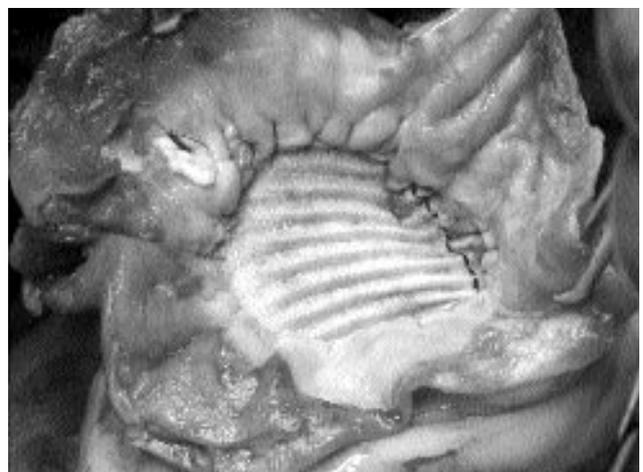


Fig. 3. Intraoperative view of the remodeling ventriculoplasty method of Dor.

pseudoaneurysms and detected no specific signs and symptoms. In their series, pseudoaneurysms were incidentally found in 25 patients (48%). Of the rest, eight patients (15%) had congestive heart failure, seven patients (13%) had chest pain, and several had arrhythmias or systemic embolizations. Only four patients (8%) displayed acute myocardial infarction or cardiac tamponade. Our patient presented with the complaint of shortness of breath secondary to chronic heart failure.

Left ventricular pseudoaneurysms have a high risk for rupture and are generally fatal, so surgical treatment is recommended by most authors as soon as a pseudoaneurysm is diagnosed. However, some authors believe that medical treatment of chronic pseudoaneurysms (>3 months) is not associated with an increased risk for cardiac rupture, and that surgical resection should be considered in patients with ventricular tachycardia, recurrent embolism, or who develop congestive heart failure related to a pseudoaneurysm; in those having other indications for cardiac surgery such as coronary artery bypass surgery or staged repair of congenital heart disease, and in those in whom a pseudoaneurysm is detected within three months after myocardial infarction.^[13] In our patient, surgical treatment was chosen because of the unusually large size of the pseudoaneurysm and the presence of LV dysfunction which was thought to be related to the pseudoaneurysm.

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