

A giant left ventricular pseudoaneurysm like an “hourglass” after silent myocardial infarction

Sessiz miyokard enfarktüsü sonrasında gelişen “kum saati” görünümündeki dev sol ventrikül psödoanevrizması

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ABSTRACT

A 51-year-old man was referred to our institution for dyspnea on exertion and non-productive cough, of three-month history. A silent inferior myocardial infarction was detected incidentally on an ECG that manifested itself as inferior Q waves. Two-dimensional transthoracic echocardiography demonstrated a second large cardiac chamber (6x5 cm) adjacent to the inferior-basal left ventricle wall, with a narrow neck and to-and-fro signals during Doppler US evaluation. Ventriculography showed a pseudoaneurysm, like an hourglass, on the inferior wall of the left ventricle which was approximately 6x5 cm in diameter. Coronary angiography showed triple vessel disease with total occlusion of the circumflex coronary artery. These findings were consistent with left ventricular pseudoaneurysm resulting from an old inferior myocardial infarction. After diagnosis of left ventricular pseudoaneurysm and concomitant coronary artery disease, surgery was recommended. He underwent coronary artery bypass surgery including the repair of the pseudoaneurysm. But, unfortunately the patient died in during the operation. This case illustrates that pseudoaneurysm can develop after silent myocardial infarction with nonspecific symptoms. In addition, if false aneurysm invades adjacent organs such as lungs, then the risk of surgical mortality increases.

Keywords: Myocardial infarction, left ventricular aneurysm, pseudoaneurysm

ÖZ

Elli bir yaşında, erkek hasta, eforla ilişkili nefes darlığı ve 3 aydır devam eden kuru öksürük şikayeti ile kliniğimize başvurdu. Hikayesinde EKG’de Q dalgaları ile kendini gösteren sessiz bir inferior miyokard enfarktüsü mevcuttu. Hasta öncesinde şikayet tarif etmiyordu. İki boyutlu transtoraksik ekokardiyografide, sol ventrikül infero-bazal segmente komşu, dar boyunlu ve doppler incelemesinde ileri-geri akım sinyali veren ikinci bir kalp odacığı (6 cm x 5 cm) saptandı. Ventrikülografi, yaklaşık 6x5 cm çapında, sol ventrikül inferior duvarında, kum saatine benzeyen bir psödoanevrizmayı gösterdi. Koroner anjiyografide, sirkumfleks koroner arterin tam tıkalı olduğu üç damar hastalığı saptandı. Bu bulgular, eski bir inferior miyokard enfarktüsü sonucunda gelişen sol ventrikül psödoanevrizması ile uyumlu idi. Sol ventrikül psödoanevrizması ve eşlik eden koroner arter hastalığı tanısı konduktan sonra hastaya ameliyat önerildi. Hastaya yalancı anevrizma onarımı da dahil olmak üzere koroner arter bypass ameliyatı uygulandı. Ancak, ne yazık ki hasta intraoperatif kaybedildi. Bu vaka, psödoanevrizmanın, semptomsuz seyreden sessiz miyokard enfarktüsü sonrası gelişebileceğini göstermektedir. Ayrıca, psödoanevrizmanın akciğerler gibi komşu organlara invazyon göstermesi cerrahi mortalite riskini arttırmaktadır.

Anahtar kelimeler: Miyokard enfarktüsü; sol ventrikül anevrizması; psödoanevrizma

INTRODUCTION

Left ventricular (LV) pseudoaneurysm develops after rupture of the ventricular wall in an area of pericardial adhesion and organized hematoma¹. It usually occurs several weeks after the myocardial infarction of the inferior or posterolateral left ventricular wall².

Early diagnosis and appropriate treatment are essential for patients with LV pseudoaneurysms. Patients with LV pseudoaneurysms have higher surgical mortality rates. However, surgery is considered as the treatment modality in selected patients³.

The symptoms of a LV pseudoaneurysm are often

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nonspecific, and the diagnosis is generally made incidentally. Congestive heart failure, chest pain and dyspnea are the most frequently reported symptoms. Sometimes, pseudoaneurysms may remain clinically silent, and be discovered during routine investigations³. These patients can be asymptomatic. Our patient, who suffered a transmural myocardial infarction, did not have a previous history of angina pectoris.

Left ventricular pseudoaneurysm is a rare complication of acute myocardial infarction^{2,3}. We describe a patient with silent inferior myocardial infarction, who developed heart failure as well as atypical symptoms like cough, due to a giant pseudoaneurysm of the inferior wall.

CASE REPORT

A 51-year-old male patient was admitted to our institution with dyspnea and non-productive cough. A silent inferior myocardial infarction was detected incidentally on an ECG that demonstrated inferior Q waves. His functional capacity was NYHA class II, and his exercise capacity was greatly impaired during the last three months, and rapidly worsened within the last week.

On physical examination, his blood pressure was measured as 125/80 mmHg and heart rate as 82 bpm. The first and the second heart sounds were normal as well as the chest examination. On physical examination there was no evidence of heart failure.

Hematological and biochemical tests were normal. Serum cardiac enzymes and serum troponin I levels were also within normal ranges. Chest radiography revealed enlargement of the cardiac silhouette.

Transthoracic echocardiography demonstrated normal LV systolic function (ejection fraction 50%) and a second cardiac chamber (7cm X 6 cm) adjacent to the inferior LV wall, with a narrow neck and to-and-fro signals at Doppler evaluation. The pseudoaneurysm communicated with the left ventricle cavity through a narrow neck (Figure 1).

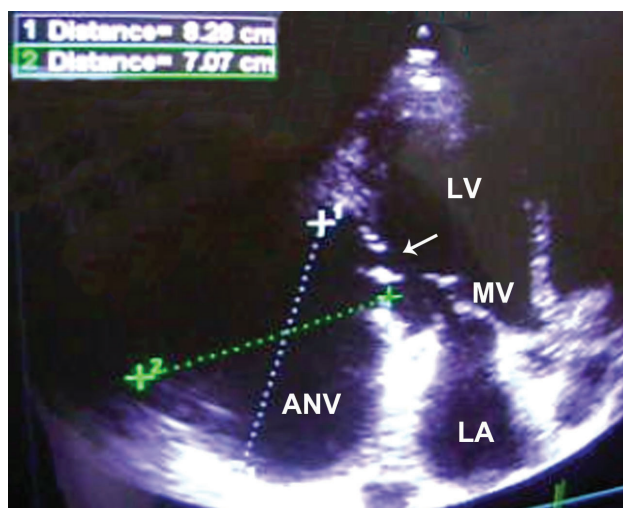


Figure 1. Transthoracic echocardiogram illustrating a postero-lateral left ventricular aneurysm (ANV). Arrow indicate the connection between the left ventricle (LV) and the aneurysm. LA: Left atrium; MV; Mitral valve.

Left ventriculography showed inferior and postero-basal hypokinesis with mild mitral regurgitation. Left ventricular ejection fraction was 50 percent. In addition, a large pseudoaneurysm resembling an hourglass was demonstrated that originated from the inferior wall (Figure 2-3). Coronary angiography showed severe stenosis of the coronary arteries as indicated in parenthesis: (the mid and distal portion the left anterior [70%], and the first diagonal [95%],

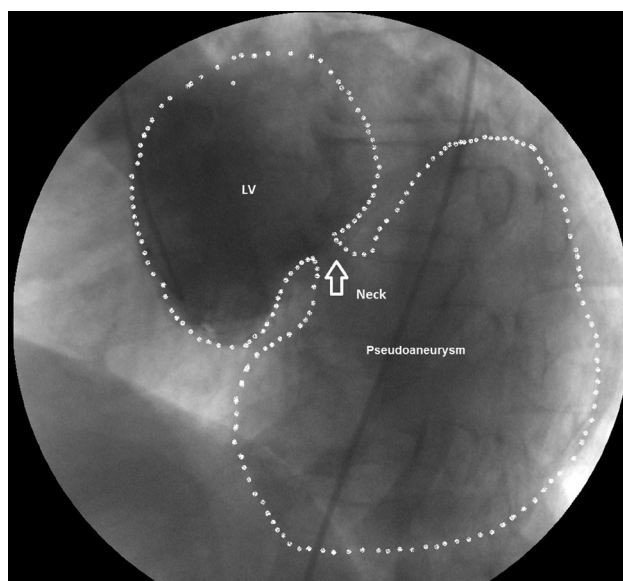


Figure 2. Left ventriculography showing a giant pseudoaneurysm communicating with the left ventricle cavity through a narrow neck like an "hourglass".

descending coronary artery the proximal [40%] and the mid-portion [100 %] of the circumflex artery, the proximal [30%], and mid-portion [70%] of the right coronary artery).

Owing to the presence of significant multivessel coronary artery disease, the patient was referred for coronary artery bypass graft surgery and surgical repair of the pseudoaneurysm.

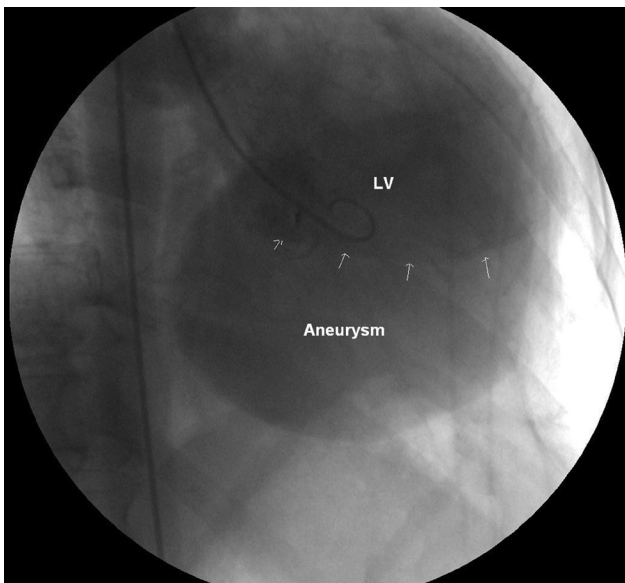


Figure 3. Left ventriculography showing the pseudoaneurysm as a second cardiac chamber originating from the inferior wall of the left ventricle.

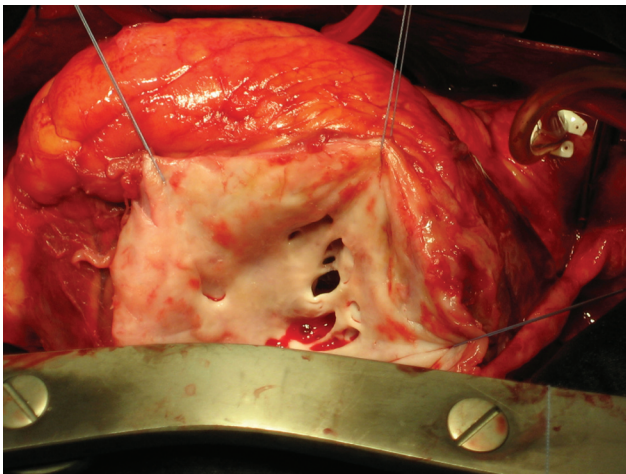


Figure 4. Intraoperative view of the pseudoaneurysm. Notice the rupture in the posterolateral wall of the left ventricle with multiple sieve-like openings beneath the posterior papillary muscle.

The surgery was performed via medial sternotomy incision with the use of extracorporeal circulation, aortic cross-clamping and blood cardioplegia. A large, non-ruptured posterolateral pseudoaneurysm with a dense capsule was seen and there were adhesions between this capsule and the hilus of the left lung as well as the left inferior lobe of the lung. The pseudoaneurysm was resected, and the rupture in the posterolateral wall of the left ventricle with multiple sieve-like openings beneath the posterior papillary muscle was noted (Figure 4). The defect was repaired with a polytetrafluoroethylene patch and the sutures were reinforced with pledgets of the autologous pericardial graft material. The anastomosis of the left internal mammary artery to the left anterior descending artery and venous grafts on first diagonal and posterior descending arteries were performed. After removal of the aortic cross clamp a heavy bleeding occurred from the hilus and parenchyma of the inferior lobe left lung. Unfortunately due to the long cardiopulmonary bypass time it was not possible to wean from the extracorporeal circulation and the patient deceased.

DISCUSSION

In our patient, clinical data, echocardiographic and ventriculographic findings were compatible with the diagnosis of LV pseudoaneurysm. LV pseudoaneurysm typically has a narrow neck and more often located in the inferior and lateral wall segments as seen in our patient^{3,4}. It is believed that LV pseudoaneurysms are rare, but can lead to significant morbidity and mortality³⁻⁵. The most common etiologic factor is myocardial infarction. In addition, previous cardiac surgery, endocarditis, blunt or penetrating trauma may also cause development of a pseudoaneurysm³⁻⁵. It is the result of the rupture of the ventricular free wall, contained by overlying adherent pericardium and organized hematoma¹.

The symptoms of a LV pseudoaneurysm are often nonspecific, and the diagnosis is generally made incidentally²⁻⁵. Sometimes, they may be asymptomatic at the time of the diagnosis³. The most common

clinical presentation of cardiac pseudoaneurysms is characterized by congestive heart failure, chest pain and dyspnea. Patients may also present nonspecific complaints such as cough, altered mental status, and dizziness^{3,4}.

Although, transthoracic echocardiography is a reasonable first diagnostic tool, further imaging studies should be performed. Ventriculography is considered to be the best available test in the diagnosis of LV pseudoaneurysm. Moreover, it is useful in planning and organization of the surgery. In addition, coronary angiography is usually necessary prior to surgery to evaluate the need of concomitant bypass grafting. Transesophageal echocardiography (TEE), cardiac multislice computed tomography and magnetic resonance imaging (MRI) are the other sensitive imaging techniques in the detection of LV pseudoaneurysms^{3,5}. In our patient, we established our diagnosis by echocardiography, and confirmed it with ventriculography.

The management of patients with cardiac pseudoaneurysm is frequently complicated by comorbidities such as significant coronary artery disease and heart failure⁵. Poor prognosis has been described in pseudoaneurysms caused by myocardial infarction and in patients requiring more complex surgery²⁻⁷.

Since pseudo-aneurysms are rarely observed, no randomized controlled trials exist to guide the treatment decision. LV pseudoaneurysms have a high mortality rate especially in those who had not undergone a surgery. Frances et al.³ found high mortality rates (23%) in patients who underwent surgery as well as in those who were treated medically (48%). Left ventricular pseudoaneurysms have a higher risk of rupture and are generally fatal, therefore surgical treatment is recommended by most authors as soon as the diagnosis is established. However, in hemodynamically stable patients, especially with small and chronic (>3 months) pseudoaneurysms, medical treatment and follow-up with echocardiography, cardiac multislice computed tomography or MRI to monitor LV remodeling may be considered³⁻⁶. Although,

randomized controlled data are missing, new data suggest that not all cardiac pseudoaneurysms require immediate surgery⁵. Very narrow communication between the left ventricle and pseudoaneurysm can reduce the likelihood of rupture. Despite this, surgical resection should be considered in patients requiring coronary bypass surgery. In addition, surgery is also recommended in patients having malign arrhythmia, peripheral embolism, or heart failure related to a pseudoaneurysm⁵⁻⁷. Another treatment method is the percutaneous closure of the left ventricular pseudoaneurysm using coils, vascular plugs and septal occluder devices^{8,9}.

Untreated pseudoaneurysm may cause complications such as rupture, thromboembolism, compression of nearby structures, and infection^{5,10}. Pseudoaneurysm may partially reduce stroke volume similar to a true aneurysm and they may also serve as a focus for arrhythmias and result in decreased cardiac output³⁻⁵.

Surgical treatment of a pseudoaneurysm is traditionally performed with primary repair or patch closure of the defect with aneurysmorrhaphy, and appropriate myocardial revascularization^{7,11}. Although, a high operative mortality has been reported, improvements in cardiac surgery techniques and hybrid approaches may have led to a decrease in perioperative mortality⁷. Our patients had multivessel disease and the left lung was invaded by the pseudoaneurysm. These two issues may have an impact on mortality. We assume that, in order to avoid this catastrophic condition, the surgical attention should mainly be focused on the repair of the LV wall, not on the resection of the remaining aneurysmal sac^{3,7,10}.

As a conclusion, in cases with left ventricular pseudoaneurysms, surgical mortality is high especially when the adjacent tissues are invaded. Therefore, different surgical methods or hybrid approaches such as percutaneous interventions, may diminish the morbidity and mortality rates.

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