# OLGU SUNUMLARI
# CASE REPORTS

## Pseudoaneurysm Following High Lateral Myocardial İnfarction: A Case Report

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Male patient with a history of high lateral myocardial infarction experienced 45 days before admission was accepted to our clinic with the initial diagnosis of congestive heart failure. Physical examination revealed 2-3°/6 systolic murmur in the mesocardiac region. Telecardiography revealed a mass of 12x6 cms in the left hilus. Pseudoaneurysm was diagnosed by transthoracic echocardiography and followed by left ventriculography. The symptoms of heart failure disappeared after successful surgical repair. *(Ana Kar Der, 2001; 1: 76-79)*

**Key Words:** Pseudoaneursym, high lateral myocardial infarction, heart failure

### Introduction

Left ventricular psedoaneurysm is formed by blood entering into the pericardial space from the ruptured myocardium, which forms an aneurysmatic dilatation (1,2). Unlike a true aneurysm, the wall of the psedoaneurysm is formed by thrombi and enveloping pericardial tissue with a narrow neck1 (3, 4). The wall of pseudoaneurysm wall lacks myocardial elements (1, 3, 4). Pseudoaneurysm is a rare entity and unless a high suspicion led research is done, is hard to diagnose and without treatment carries a high mortality (1-3). Pseudoaneurysms are seen most frequently following myocardial infarction (55%) (1). Other leading causes are cardiac surgery (33%), trauma (7%) and infective endocarditis (5%) (1). Within the myocardial infarctions, it is generally seen following inferior wall infraction (40%) (1). Although heart failure is rare following high lateral MI, it is not an uncommon clinical outcome in pseudoaneurysm cases (1, 2). We report a case of pseduoaneurysm following high lateral MI.

### Case Report

A 49 years old male patient was admitted to our clinic with the complaints of shortness of breath and palpitations. Patient history revealed 45 days old myocardial infarction. The patient had no complaints until 2 days ago, and his complaints progressively worsened since. Blood pressure was 70/40 mmHg, pulse 110 beats/min. A pansistolic murmur was heard over the mesocardiac region (2-3°/6°). A high frequency rales were heard radiating to the middle zone of the lungs bilaterally. Other system examinations were normal and central venous pressure was 16-18 cm/$H_2O$. On electrocardiogram pathological Q waves, ST elevation of l mm and negative T waves in D1, aVL derivations; ST depression of 1 mm in D2, D3 and aVF; negative T waves in V4-V6 derivations were seen (Fig. 1). CK-MB levels were within normal range and Troponin-T was negative. A mass of 12x6 cms adjacent to the left ventricle was seen on the te-

**Figure 1: 12 lead electrocardiogram illustrates acute high lateral myocardial infarction manifested by ST segment elevation in leads D1 and aVL, and reciprocal ST segment depression in leads D2, D3 and aVF.**

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lecardiography (Fig. 2). Initial diagnosis was pulmonary edema, which required dopamine and dobutamine infusions, and 60 mg of furosemid was given intravenously. When systolic blood pressure exceeded 100 mm Hg, nitroglycerine infusion was started. 24 hours later, dopamine, dobutamine and nitroglycerine infusions were discontinued, and oral nitrates, furosemid, captopril and aspirin were initiated.

**Figure 2: Telecardiogram shows mass in left hilus.**

When the patient was stabilized hemodynamically, transthoracic echocardiography was done. Echocardiography revealed akinesia of the left ventricle late-

**Figure 3: Transthoracic echocardiography demonstrates pseudoaneurysm of the anteroseptal wall of left ventricule in the short axis position.**

ral wall, defect of 7 mm in diameter and a pseudoaneurysm of 47x45 mm adjacent to the defect with pathological flow (Fig. 3). Coronary angiography and

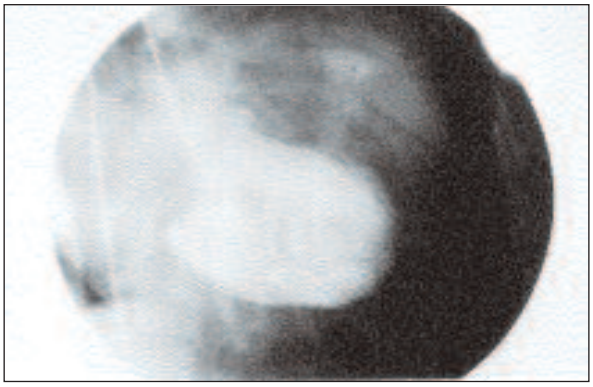

**Figure 4: Left ventriculography shows pseudoaneurysm in the right oblique view.**

left venticulography were done to assess the coronary anatomy and to form an operative plan. Left ventriculography revealed psedoaneurysm adjacent to the left ventricle (Fig. 4) and coronary angiography documented a 60 percent narrowing of left anterior descending (LAD) coronary artery, a 70-80 percent narrowing distal to the second diagonal branch of LAD, a 90 percent narrowing distal to the atrioventricular branch of circumflex (Cx), a 70-80 percent narrowing distal to the obtuse marginal-1 branch of Cx and a 70-80 percent narrowing distal to the sinus nodal branch of right coronary artery. The patient underwent open heart surgery, repair of pseudoaneurysm with teflon felt and coronary artery bypass graft (LIMA-LAD) was done. Postoperative period was uneventful. The patient remained asymptomatic in the 3rd and 6th month follow-ups and control echocardiography showed a complete resolution of lateral wall defect and pseudoaneurysm.

## Discussion

In previous articles (5) pseudoaneurysm have been reported to be seen most frequently following anterior MI, but a recent study of 290 cases (1) reported inferior MI as the most frequent reason for pseudoaneurysm. Pseudoaneurysm following high lateral MI in our patient is rare. Patients with pseudoaneurysms might be clinically asymptomatic (%10), and the most frequently encountered symptoms are those of heart failure (1). In patients with high lateral MI, left ventricle dysfunction is not an expected finding, yet our patient was admitted to our clinic because of congestive heart failure. In almost all of the patients with pseudoaneurysms there are abnormal findings on ECG and telecardiography, but these changes are not specific to pseudoaneurysm (1). In

our patients ECG, ST elevations in DI and aVL derivations, and on his telecardiography a mass of 12x6 cms adjacent to the left ventricle was evident.

TTE is a vital tool for the diagnosis of pseudoaneurysm (85-90%) (1) and using this diagnostic technique we showed the pseudoaneurysm adjacent to the left ventricle lateral wall. The golden standard for the diagnosis of pseudoaneurysm is left ventriculography (1, 6-8). In patients, whom a surgical intervention is planned, coronary angiography is eminent to show the coronary pathology (1, 6, 7). Our patient's cardiac catheterization revealed triple vessel coronary disease in addition to the pseudoaneurysm.

In previous studies, the medical treatment of pseudoaneurysm was shown to have a poor prognosis with a high risk of rupture (30-45%), with death occurring due to cardiac rupture (3, 5, 9, 10). Natarajan et al (11), in a retrospective study between 1984-1993 reported that 66 patients with pseudoaneurysm did not have an increased risk of rupture (in follow up of more than 3 months). Yeo et al (2) reported similar results stating that the risk of rupture did not increase in pseudoaneurysm cases. They pointed out that the cause of mortality tended to be the severity of coronary artery disease and associated medical conditions. In addition surgery was justified in these groups of patients. Rittenhouse et al (12) reported low mortality following early surgery of pseudoaneurysm cases.

In our patient surgery was chosen as the treatment method because the patient was not stable hemodynamically and coronary angiography with left ventriculography was done prior to surgery. Following these procedures, thoracic and cardiovascular surgery department operated the patient. The patient had open heart surgery where teflon felt repair of pseudoanerysm with concomitant coronary artery bypass graft (LIMA-LAD) was done. He was asymptomatic in long-term follow-up period.

## Conclusion

Patients with high lateral MI with progressive heart failure need to be assessed for pseudoaneursyms and in this group of patients surgical intervention is the treatment of choice.

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