

CASE REPORT

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## Left ventricular pseudoaneurysm after myocardial infarction

Received: September 12, 2002 / Accepted: February 15, 2003

**Abstract** In this report, a case of a left ventricular (LV) pseudoaneurysm due to a previous myocardial infarction, which was repaired successfully, is described. A 62-year-old man, with a history of acute anterior wall myocardial infarction 6 months previously, was admitted with the complaints of acute dyspnea and palpitation. Echocardiography revealed an LV aneurysm, and ventriculography showed ventricular dysfunction and an LV pseudoaneurysm. Coronary angiography showed total occlusion of the proximal segment of the left anterior descending artery with a very thin lumen and insufficient retrograde filling. Under cardiopulmonary bypass and beating heart, the pseudoaneurysm was resected and the defect on the ventricular free wall was closed by the remodeling ventriculoplasty method of Dor. Histopathologic examination of the resected material confirmed the diagnosis of pseudoaneurysm. The postoperative course of our patient was uneventful. He was discharged on the ninth postoperative day.

**Key words** Left ventricle · Pseudoaneurysm · Myocardial infarction

### Introduction

The development of a left ventricular (LV) pseudoaneurysm is a rare disorder that usually occurs after transmural myocardial infarction or cardiac surgery.<sup>1–3</sup> Free wall rupture usually results in cardiac tamponade and death. Less frequently, cardiac rupture is contained by adherent pericardium or scar tissue, and pseudoaneurysm of the left ventricle occurs. Thus, unlike a true LV aneurysm, an LV pseudoaneurysm contains no endocardium or myocardium. The natural history of a chronic pseudoaneurysm is not

clearly understood. However, the danger of secondary rupture is real for a large pseudoaneurysm, but uncertain for small ones.<sup>2,4</sup> Although LV pseudoaneurysm is clinically uncommon, the mortality rate is very high due to secondary rupture.<sup>2–6</sup>

In this report, a case of a large LV pseudoaneurysm on the anterior wall due to a previous anterior myocardial infarction, which was repaired successfully, is described.

### Case report

A 62 year-old man, who had experienced an anterior myocardial infarction 6 months previously, was admitted to our clinic with the complaints of acute dyspnea and palpitation. The patient was hospitalized with a diagnosis of congestive heart failure due to LV aneurysm. Chest X-rays showed that the heart was pathologically enlarged, primarily on the left, and exhibited an aberrant contour. An echocardiogram demonstrated left ventricular dysfunction, a large LV aneurysm containing a thrombus, and the ejection fraction (EF) was 30%. Transthoracic echocardiography did not provide more information for evaluation of an anterior ventricular pseudoaneurysm. Contrast left ventriculography showed ventricular dysfunction and an LV pseudoaneurysm. The pseudoaneurysm, containing a thrombus, was connected to the ventricle by a narrow neck, and the contrast medium tended to remain in the pseudoaneurysmal cavity for several beats after injection, consistent with the stagnant flow of blood in the sac (Fig. 1). Coronary angiography showed total occlusion of the proximal segment of the left anterior descending artery (LAD) with a very thin lumen, and its retrograde filling was TIMI grade-1. The other coronary arteries were normal. We considered the cause of the pseudoaneurysm to be left anterior descending artery occlusion. At operation, we found that the pericardium was adherent to the aneurysmal wall. Under cardiopulmonary bypass and beating heart, the heart was gently mobilized from the pericardium and loose adhesions were removed. An approximately 2 × 3-cm piece of the pericardium

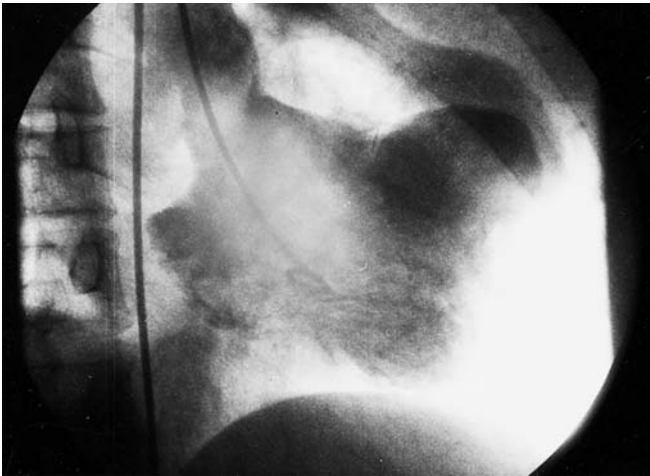
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was left at the side of the pseudoaneurysmal sac. The pseudoaneurysm had a dimension of approximately  $7 \times 6 \times 5$  cm and was found at the anterior region (Fig. 2A). The postinfarction pseudoaneurysmal sac was resected, and a 2-cm defect was seen on the left ventricular wall. An organized thrombus was also found within the cavities of both the pseudoaneurysm and the left ventricle. After the thrombi within the LV cavity were removed (Fig. 2B), the defect on the ventricular free wall was closed by the remodeling ventriculoplasty method of Dor (Fig. 2C, 2D). As the LAD was not suitable, coronary artery bypass was not performed. Histopathological examination of the resected ma-

terial revealed no myocardial elements on the aneurysmal wall, and the diagnosis of pseudoaneurysm was confirmed. The postoperative course was uneventful, the patient was discharged on the ninth postoperative day.

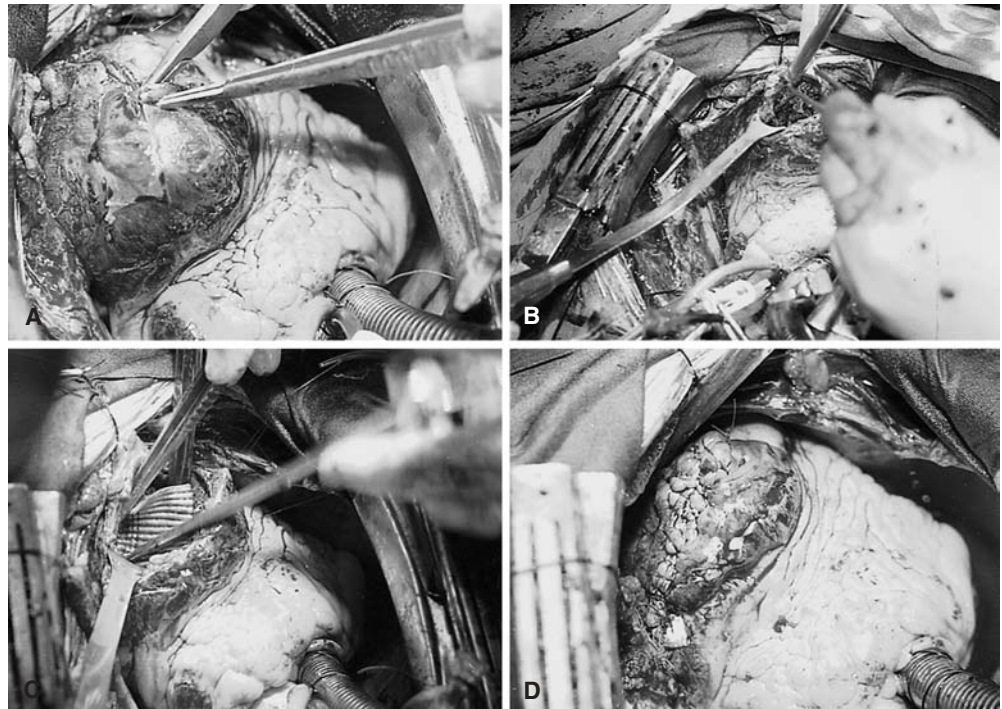
## Discussion

LV pseudoaneurysm develops after transmural myocardial infarction (55%), surgery (33%), trauma (7%), or infection (5%).<sup>2-5</sup> LV pseudoaneurysm formation is a serious complication of myocardial infarction, occurring in approximately 4% of patients with infarction, and has potentially catastrophic consequences. Pathologically, pseudoaneurysms are characterized by a small, narrow-necked channel that connects the ventricle with a larger aneurysmal sac containing blood and thrombus, and lined by fibrous pericardial tissue without any myocardial elements.<sup>7</sup> It has been reported that pseudoaneurysm of the left ventricle generally occurs after inferior myocardial infarction due to occlusion of the circumflex artery.<sup>2,7</sup> The prediction of inferior myocardial infarction, which results in LV pseudoaneurysm, is consistent with the location of pseudoaneurysms on the posterior, lateral apical, or inferior surface of the left ventricle, and not on the anterior surface.<sup>2,7,8</sup> In contrast to LV pseudoaneurysms, only about 4% of true LV aneurysms are located on the posterolateral or diaphragmatic surface. Thus, it has been suggested that a posterior localization is indicative of a pseudoaneurysm rather than a true aneurysm. One of proposed explanations for the relative lack of anterior LV pseudoaneurysm is that anterior rupture may be more likely to result in hemopericardium and death than



**Fig. 1.** Contrast left ventriculogram showing a left ventricular pseudoaneurysm on the anterior wall

**Fig. 2A-D.** Intraoperative view. **A** The left ventricular pseudoaneurysm is seen clearly in the anterior region of the left ventricle. **B** The cavity of the pseudoaneurysm was opened, and thrombi within the LV cavity were removed. **C,D** The defect on the ventricular free wall was closed by the remodeling ventriculoplasty method of Dor



posterior rupture, since the rupture of the anterior wall is rarely compressed by adherent pericardium.<sup>1,2,7,8</sup> A false aneurysm may develop after acute rupture of an infarcted area of the left ventricle when the rupture is rarely compressed by adherent pericardium. Adhesions may already exist at the time of infarction or more frequently develop de novo during the process of rupture.<sup>1</sup> In our case, LV pseudoaneurysm occurred from occlusion of the left anterior descending artery and was located anteriorly. We think that adhesions may have already existed at the time of infarction and the pericardium may have been sufficiently adherent to the epicardium, and thus the anterior myocardial rupture may have resulted in an LV pseudoaneurysm.

LV pseudoaneurysms usually present with symptoms, but these symptoms are not specific for LV pseudoaneurysm. Diagnosis is complicated because the most frequently reported symptoms are heart failure, chest pain, and dyspnea, which are common in patients with coronary artery disease. Our patient had complained of acute dyspnea and palpitation.

The salient feature that distinguishes a pseudoaneurysm from a true aneurysm is the discontinuity of the myocardium around the cavity.<sup>9</sup> The presence of a neck smaller than the aneurysmal cavity, detected by echocardiography and/or contrast left ventriculography, is strongly suggestive of a pseudoaneurysm.<sup>1</sup> We established the diagnosis of LV pseudoaneurysm preoperatively by contrast left ventriculography, and histopathologic examination of the resected material confirmed it.

Unlike true aneurysms, which have a resistant fibrotic wall, pseudoaneurysms initially consist of loose tissues and have an excessively high propensity for secondary rupture. Most investigators have supported surgery as the appropriate treatment for LV pseudoaneurysm since untreated pseudoaneurysms have an approximately 30%–45% risk of rupture.<sup>2</sup> Embolization of thrombotic material, induced by stagnant patterns of blood flow, has also been reported with large pseudoaneurysms (>3 cm in diameter).<sup>2–6</sup> Surgical treatment of the LV pseudoaneurysm, along with myocardial revascularization or mitral valve repair or replacement, improves symptoms in the great majority of patients. In 10%–20% of cases, chronic pseudoaneurysms are discovered incidentally.<sup>2</sup> Because of the uncertainties surrounding their natural history, and the relative safety of surgical repair in this subgroup, the decision to operate should prevail over conservative management in the case of a large or expanding pseudoaneurysm.<sup>1,2</sup> Asymptomatic chronic small pseudoaneurysms (<3 cm in diameter) have a more stable

course, and regular echocardiographic or magnetic resonance assessments could be a reasonable approach in patients who do not require surgical myocardial revascularization or mitral valve surgery. Any increase in size should lead to surgical treatment.<sup>1–6</sup> In the present case, surgical treatment of the large LV pseudoaneurysm was performed without coronary artery bypass grafting because the LAD was not suitable for bypass surgery.

As the mortality rate is very high in patients with ruptured LV pseudoaneurysm, early diagnosis is very important. In patients with a large or expanding small pseudoaneurysm, who require additional cardiac surgical intervention or who have symptoms, early elective surgical repair should be performed. Early diagnosis is very important, and when a diagnosis is made, as in our case, surgical results are satisfactory.

**Acknowledgments** We would like to thank Assistant Professor Fazlı Erdogan, Department of Pathology, for his help with the histopathological examination and Assistant Professor Kemal Erol, Department of Cardiology, for his help with this work.

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