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Case Report

# Left ventricle pseudoaneurysm: Diagnosis by a new murmur

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ABSTRACT

Incomplete rupture of the ventricle free wall can occur after myocardial infarction. This occurs when an organized thrombus and the pericardium seal the ventricular perforation. This can progress to the formation of a left ventricle pseudoaneurysm (LVPA).

A 70-year-old male with an antero-septal ST-elevation myocardial infarction (STEMI) underwent an emergent left heart catheterization which revealed severe three-vessel disease with occluded grafts, non-amenable to re-vascularization, and an apical thrombus. As he was high-risk for repeat coronary artery bypass graft, he was medically managed. Transthoracic echocardiogram (TTE) showed a normal left ventricle ejection fraction (LVEF), apical anterior and inferior wall akinesis, moderate sized apical thrombus, and pericardial thickening. On hospital day 7, examination revealed a new 3/6 to-and-fro murmur that was loudest at the apex. The patient was asymptomatic with normal vital signs. A repeat TTE revealed an apical wall rupture with flow into the pericardial cavity and absence of the apical thrombus. A LVPA was diagnosed and the patient was immediately referred for surgical repair.

This case illustrates the potential for developing LVPA in STEMI patients and the importance of physical examination. If identified early a potential emergent situation in a previously asymptomatic patient can be averted, thereby preventing fatal consequences.

<**Learning objective:** With the growing use of diagnostic testing the importance of physical examination is being lost. However, with an astute cardiac examination, potential complications such as a left ventricular pseudoaneurysm can be identified and promptly managed. In addition, a ventricular pseudoaneurysm must be considered in the differential as a rare complication in post ST-elevation myocardial infarction patients with a new murmur.>



## Introduction

Left ventricular free wall rupture is invariably a calamitous event with the most frequent presentation being sudden hemodynamic collapse and death [1]. In such a scenario, however, a pseudoaneurysm may be considered a blessing in disguise. Left ventricular pseudoaneurysm (LVPA) develops when the rupture is contained by adherent pericardium, overlying fibrin and clot, or scar tissue. This prevents the rapid accumulation of blood in the pericardial space which would have otherwise led to death via the formation of a fatal pericardial tamponade. In contrast to rupture per se, one with a false aneurysm can lead to longer survival, thus providing physicians with crucial hours, days, or rarely even weeks to make the diagnosis [2].

LVPA is clinically uncommon and remains a challenge to diagnose. It can have numerous presentations, ranging from asymptomatic to chest pain and dyspnea [3]. Hence, it merits a high index of suspicion. The importance of making the diagnosis cannot be overstated, since the incidence of a fatal left ventricular rupture from a pseudoaneurysm varies from 19 to 32% [4]. We present a case in which an asymptomatic LVPA was diagnosed by picking up a new murmur on physical examination.

## Case report

A 70-year-old male of Vietnamese origin was referred to the emergency room from an urgent care set up because of chest pain,

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elevated troponin I, and ST segment elevations on electrocardiogram (ECG). He had central, sharp chest pain for one day, which was persistent, non-radiating and 10/10 in intensity. His past medical history was significant for coronary artery disease (CAD) status post three-vessel coronary artery bypass graft (CABG) 10 years previously, poorly controlled diabetes mellitus, hypertension, cerebrovascular accident, and chronic obstructive pulmonary disease. He was non-adherent to his medications and had 40 pack years smoking history. Vital signs and physical examination were within normal limits on admission. ECG revealed ST segment elevations in antero-septal leads with elevated troponin I of 5 ng/ml (Fig. 1). He was immediately taken to the catheterization laboratory. Left heart catheterization revealed high grade 3 vessel disease along with the occluded venous grafts. There was subtotal occlusion of left internal mammary artery (LIMA) to left anterior descending artery with a 95% ostial lesion. In addition, the LIMA was a diminutive vessel and was not amenable to revascularization. The patient was referred for CABG but was deemed too high of a risk for a redo open heart surgery, given his history of uncontrolled diabetes mellitus, strokes, and smoking. Medical management was therefore employed with aspirin, atorvastatin, metoprolol, clopidogrel, and therapeutic dose enoxaparin. Transthoracic echocardiography (TTE) revealed a normal left ventricular ejection fraction (LVEF), akinesis of the anterior apical and inferior walls, moderate sized apical thrombus, and pericardial thickening with a small pericardial effusion. His hospital stay was prolonged by atrial fibrillation. He, however, was improving clinically, was chest pain free and able to ambulate without difficulty. A routine physical examination on the day of discharge (day 7) revealed a new, grade 3/6, continuous to-and-fro murmur, heard best at the apex. The patient remained asymptomatic with normal vital signs. ECG taken immediately did not reveal any new ST-T wave changes. Another TTE was immediately obtained which on color flow Doppler study showed left ventricular free wall rupture at the apex with the free flow to and from the contained pericardial cavity (Figs. 2–4). The patient was diagnosed with LVPA and was immediately referred for surgical repair. Due to comorbidities, the patient was not deemed an open cardiac surgery candidate. This was discussed in detail with the patient. A fully informed consent for a percutaneous procedure with the use of an atrial occlusion device was obtained. The patient underwent percutaneous trans-catheter closure of the LV pseudoaneurysm by atrial septal occlusion device under the guidance of trans-esophageal echocardiography (TEE) and fluoroscopy (Fig. 5). The procedure went well, and TEE post procedure showed minimal flow between the LVPA and the LV (Fig. 6). The patient had an uneventful recovery. However, later in the day, the patient had an unexpected episode of ventricular fibrillation followed by cardiac arrest, which proved to be fatal despite cardiopulmonary resuscitation. An exact cause of the sudden arrhythmia could not be established since an autopsy was not performed.

## Discussion

LVPA differs from a true aneurysm by being a left ventricle free wall rupture that is contained by an adherent layer of overlying pericardium, fibrin, and clot or scar tissue as opposed to a true aneurysm that contains all the cardiac layers. In its pathogenesis, a LVPA is closer to a free wall rupture than to a true aneurysm. However, the similarities in the clinical presentation and the appearance on imaging makes LVPA a challenge to differentiate from a true aneurysm. In our case, previous cardiac insult and surgery possibly resulted in the development of a thick fibrous pericardium. The ST-elevation myocardial infarction then went on to cause wall rupture which was contained by the overlying fibrous pericardium and hence led to the LVPA formation.

The most common cause of a left ventricle pseudo-aneurysm is a transmural myocardial infarction [3,5]. This is followed by patients who have undergone cardiac surgeries or suffered trauma [4]. Rarely it is seen with myocardial abscesses and endocarditis [6]. In post-infarction patients, this entity most commonly arises after occlusion of the right coronary or the left anterior descending artery and hence usually involves the inferior wall [5,7]. Pseudoaneurysms rarely arise from the anterior wall. This is because ruptures of the anterior wall are not well tolerated and generally lead to hemopericardium or tamponade [8]. Moreover, they are seldom compressed by adherent pericardium and hence are fatal before diagnosis can be established [4].

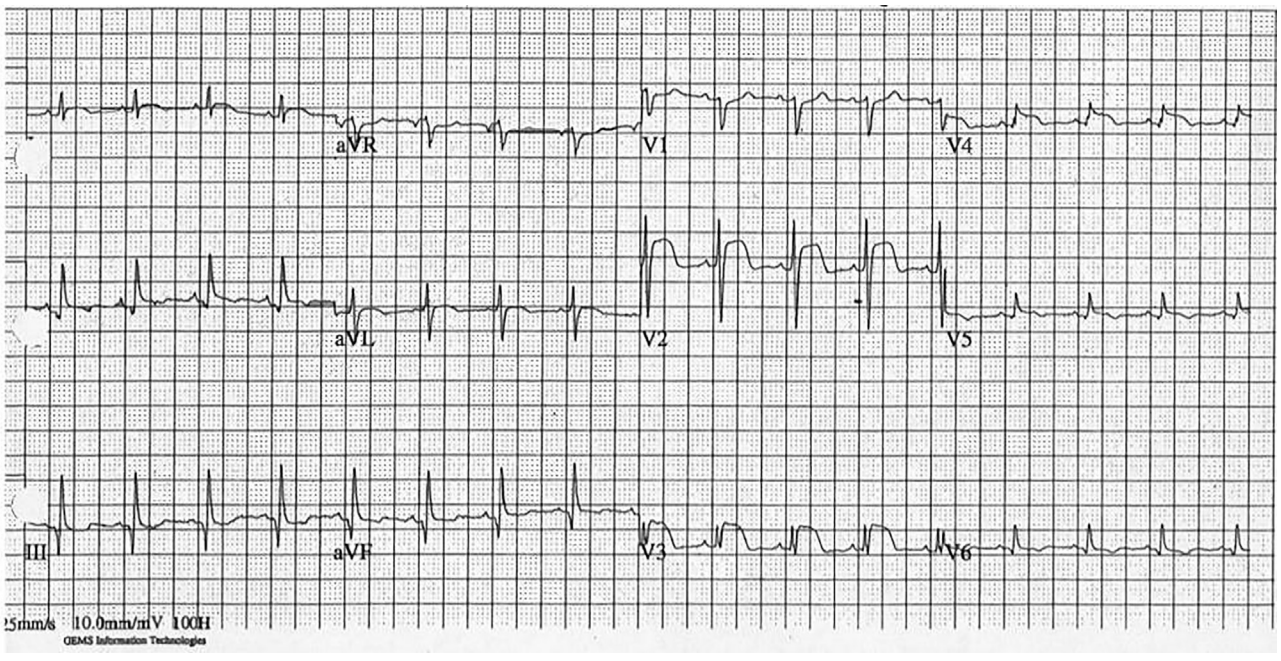


**Fig. 1.** Electrocardiogram: taken initially in the emergency room revealing ST segment elevations in the antero-septal chest leads.

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