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CASE REPORT

Sealing the breach: successful surgical management of post-myocardial infarction left ventricular pseudoaneurysm

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Abstract
Left ventricular (LV) pseudoaneurysm, a rare occurrence, develops when a ruptured ventricle is encapsulated by the pericardium or scar tissue. Unlike free intrapericardial rupture, which often results in cardiac tamponade and fatal outcome, there are instances where the cardiac rupture remains contained, forming a pseudoaneurysm and averting immediate tamponade. We describe a 43-year-old male who underwent successful surgical repair of LV rupture following inferior wall myocardial infarction that resulted in the formation of a large pseudoaneurysm. The diagnostic challenges encountered and the technical difficulties that were seen while performing the repair of the defect are being described. As the diagnosis can be challenging, swift and precise identification of LV pseudoaneurysm is paramount due to significant risk of rupture and mortality. Consequently, surgical intervention is advised as the preferred treatment for pseudoaneurysm repair. The potential dangers of rupture far outweigh the inherent risks associated with surgery, emphasizing the urgency of timely intervention.

Keywords Left ventricular pseudoaneurysm · Myocardial infarction · Left ventricular rupture · Surgical repair

Introduction

Despite advancements in the therapeutic interventions, complications such as the left ventricular pseudoaneurysm (LVPA) can arise, which pose substantial challenges to both patients and clinicians, especially in the case of delayed presentation [1]. LVPA is characterized by a contained rupture of the myocardium with communication to the left ventricular (LV) cavity through a fibrous or thrombotic tract and represents a rare, yet potentially life-threatening, complication after myocardial infarction (MI).

The management of LVPA presents a clinical dilemma due to its association with high rates of morbidity and mortality, including the risk of rupture, thromboembolism, heart failure, and arrhythmias [2]. Surgical intervention is often warranted to prevent catastrophic outcomes; however, the optimal timing and approach remain the subject of debate. Furthermore, the successful repair of a large post-MI LVPA requires a multidisciplinary approach, involving skilled cardiac surgeons, interventional cardiologists, and critical care specialists.

In this case report, we present a patient who experienced a large LVPA following MI and underwent successful surgical repair. Through this case, we highlight the diagnostic challenges, therapeutic considerations, and clinical outcomes associated with the management of this complex cardiac complication. In addition, we discuss the importance of prompt recognition and tailored management strategies in achieving favorable patient outcomes and preserving cardiac function.

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Case report

A 43-year-old male patient presented in the cardiology out-patient department of our hospital with complaints of occasional mild atypical chest pain for the last 1 month. He had a history of rest angina 2 months ago, associated with sweating, for which he took medications from local hospital and the pain was relieved after 24 h. He was a known case of systemic hypertension on irregular medications. The electrocardiogram (ECG) showed left axis deviation and poor progression of R wave with Q waves in leads II, III, and aVF (Fig. 1). Chest X-ray showed cardiomegaly, and transthoracic echocardiography (TTE) showed left ventricular ejection fraction (LVEF) of 40–45%, mild circumferential pericardial effusion, regional wall motion abnormality in the inferior and infero-septal regions, and LV aneurysm arising from the inferior wall. Coronary angiography revealed a 100% occlusion of the proximal right coronary artery (RCA); other coronary arteries were normal. Subsequent cardiac magnetic resonance imaging (MRI) showed a dilated LV with a well-defined lobulated cavity with altered signal intensity seen in continuity with the inferior wall of the LV at mid-level of size 6.5 × 5.6 cm. There was contrast going from LV into the aneurysm cavity. Cardiac computed tomography (CT) scan showed a partially thrombosed pseudoaneurysm of size 7.7 cm × 6.4 cm × 7.2 cm arising from the inferior wall of the basal to mid-LV cavity with a diffusely thin and dyskinetic adjacent wall. As the patient was asymptomatic but had a large LV pseudoaneurysm, surgical repair of the aneurysm, possibly with RCA bypass graft surgery, was planned. Preoperative complete blood count, liver function tests, kidney function tests, and coagulation profile were in normal range. He was started on once daily tablet ramipril 2.5 mg, tablet metoprolol succinate 50 mg, tablet furosemide 20 mg, and tablet spironolactone 25 mg.

On the day of surgery, after applying standard American Society of Anesthesiologists (ASA) monitoring, a large-bore intravenous line and a radial arterial line were placed under local anesthesia. The patient was induced with injection fentanyl 10 mcg/kg, injection thiopentone 1 mg/kg, and injection vecuronium. After intubation, a 7.5-Fr central venous catheter was placed in the right internal jugular vein, and a 5-Fr arterial sheath was placed in the left femoral artery. A mean arterial pressure (MAP) of 60–70 mm Hg was maintained before cardiopulmonary bypass (CPB). Transesophageal echocardiography (TEE) showed a large 5.0 × 5.9 cm pseudoaneurysm with regional wall motion abnormality in the inferior wall (Fig. 2A) and also a tiny ostium secundum atrial septal defect (ASD) with a left-to-right shunt which was undiagnosed previously (Fig. 2B).

From a surgical point of view, the right femoral vessels were kept exposed in case an emergency peripheral cannulation was required due to pseudoaneurysm rupture. Midline sternotomy was done. The pericardium was opened and harvested. Systemic heparinization was done. Standard aorta bicaval cannulation was done when activated clotting time

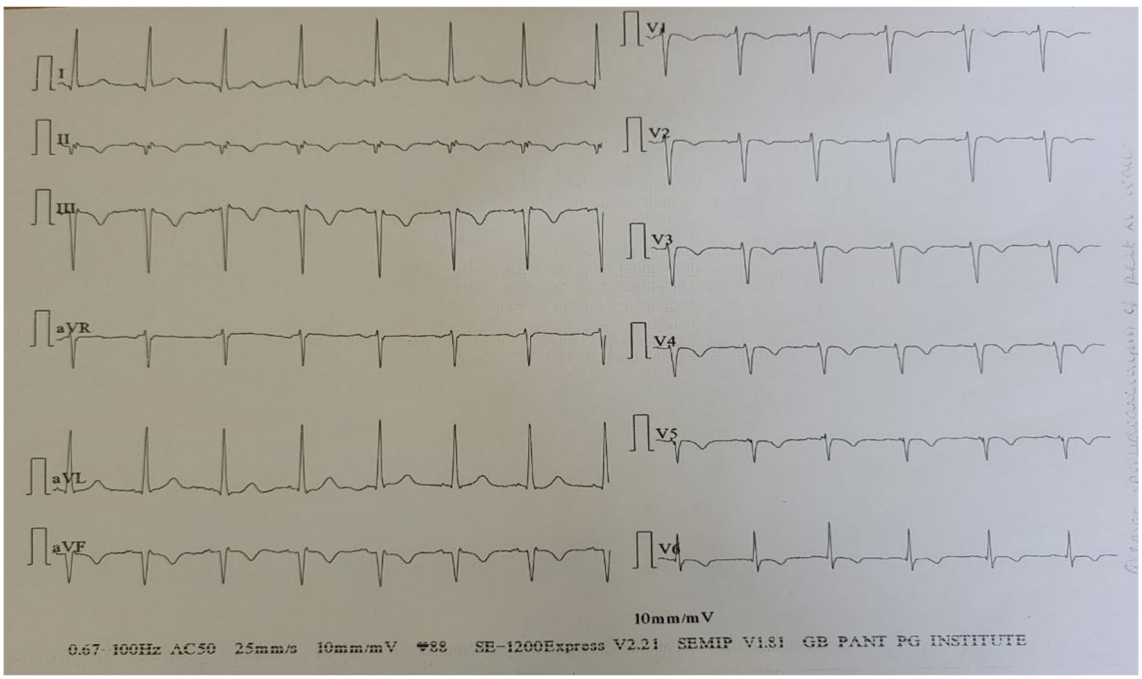

Fig. 1 Twelve lead electrocardiogram showing left axis deviation and poor progression of R wave and Q waves in II, III, and aVF

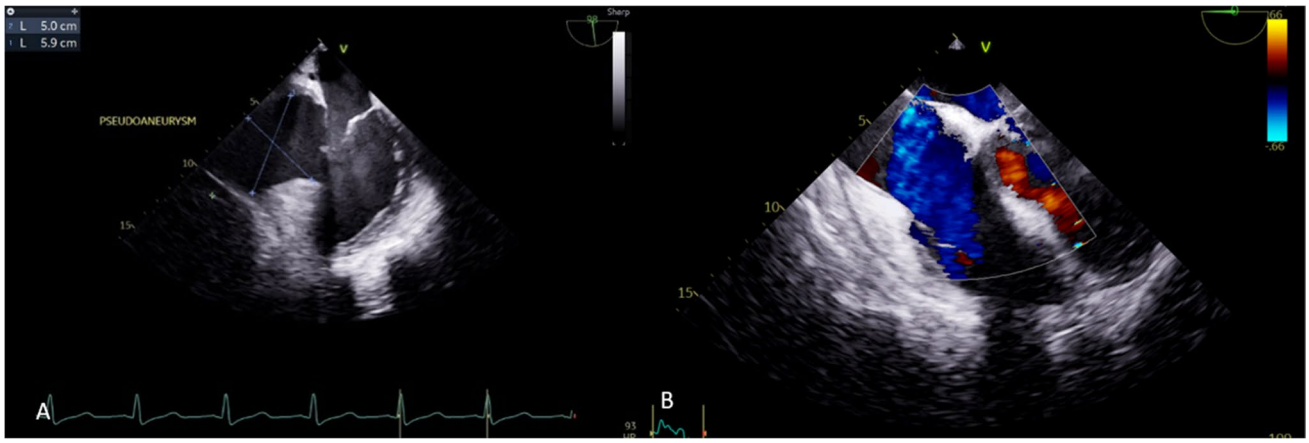

Fig. 2 **A** Transesophageal echocardiography image showing large left ventricular pseudoaneurysm. **B** Transesophageal echocardiography image showing tiny ostium secundum atrial septal defect

was > 400 ms. The cardioplegia needle was inserted in aortic root and antegrade cardioplegia (Del Nido- 1700 ml + 500 ml repeated after 60 min of aortic cross-clamp time) was given. The aortic cross-clamp was applied and the heart was cooled to 27 °C. Intraoperatively dense adhesions were found on the epicardium; the RCA was occluded. A LV pseudoaneurysm of size 6 × 6 cm was found on the inferior surface of the heart. The aneurysmal portion of the LV was opened in the midline, and the defect was repaired with Dacron, along with autologous pericardial composite patch (endoventricular circular patch plasty repair) (Fig. 3) with 4–0 polypropylene interrupted sutures without use of any sealant. The RCA was not graftable. The secundum ASD was closed by primary closure. Rewarming and deairing were done and aortic cross-clamp was removed. The heart activity resumed to normal sinus rhythm. Cardiopulmonary bypass time was 183 min and aortic cross-clamp time was 123 min. The patient was weaned from cardiopulmonary bypass with injection adrenaline, injection levosimendan, injection milrinone, and injection noradrenaline infusion. Injection protamine was given and decannulation was done. The hemostasis was achieved and chest was closed in layers over two mediastinal drains. The patient was shifted to intensive care unit in stable condition. The patient was extubated on postoperative day 1 and further recovery was uneventful. The patient was discharged on day 10 in hemodynamically stable condition. After 1 month postoperatively, he is asymptomatic with an improvement in LVEF to 45% and near complete pseudoaneurysm thrombosis.

Discussion

LV aneurysms and pseudoaneurysms represent critical complications stemming from MI, demanding precise diagnosis and swift intervention to avert potentially fatal outcome. While true aneurysms entail areas of thinned myocardium that are dyskinetic and involve the full thickness of the ventricular wall, pseudoaneurysms occur from a rupture in the ventricular free wall, contained by the adherent pericardium. Notably, true aneurysms typically exhibit a narrower neck compared to their diameter and are predominantly found in the posterior and lateral LV wall segments [3].

The diagnosis of these conditions can be daunting because they are often asymptomatic or presentation is with nonspecific symptoms, as was in the present case. Angina, the most prevalent symptom of LV aneurysm/pseudoaneurysm, has been attributed to volume overload in the LV and a resultant increase in oxygen consumption. Any functional mitral regurgitation can exacerbate ventricular overload, progressively leading to the onset of heart failure. Dyspnea, the second most frequent symptom, occurs because of a combination of systolic and diastolic dysfunction. Other symptoms include syncope (due to arrhythmias) and symptoms related to hypotension and hypoperfusion. To differentiate between LV pseudoaneurysms and true aneurysms, a comprehensive array of imaging modalities can be employed. TTE, TEE, LV angiography, MRI, CT scan, and radionuclide scanning all play pivotal roles in this diagnostic endeavor. Cardiac MRI and cardiac CT allow for visualization of the heart in any plane, making it possible to see segments that are difficult to assess with echocardiography. The high spatial resolution and superior tissue characterization of cardiac MRI make it particularly suitable for evaluating ventricular pseudoaneurysms and distinguishing them from true aneurysms. In such cases, the use of late gadolinium enhancement is especially valuable for identifying the location and transmural extent of prior infarcts. However, not all patients can undergo cardiac MRI due to contraindications such as implanted cardiac devices or claustrophobia, or due to a lack of availability. Cardiac CT, on the other hand, offers high spatial resolution

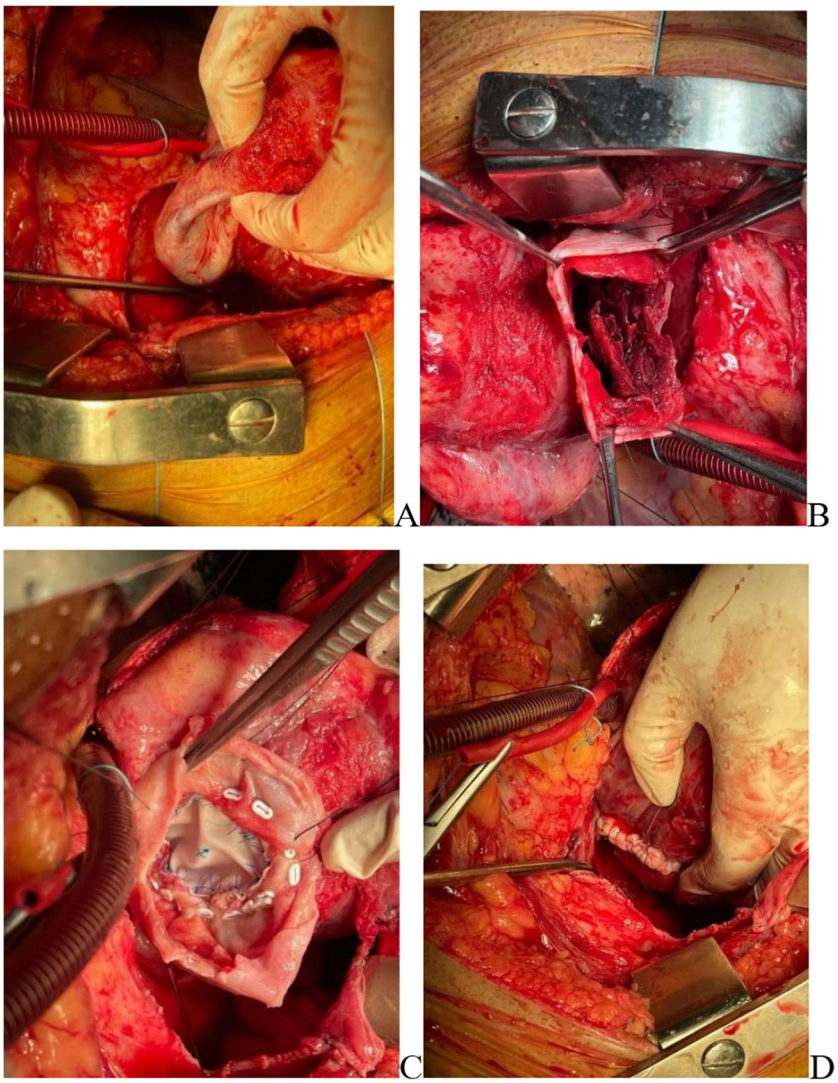

Fig. 3 **A** Left ventricular (LV) pseudoaneurysm before opening the left ventricular cavity. **B** LV pseudoaneurysm after opening the left ventricular cavity. **C** Composite patch seen after suturing the defect. **D** LV closed after the composite patch repair

and provides excellent visualization of the LV myocardium, coronary arteries, and bypass grafts [4]. The echocardiography permits the comparison of the diameter of the orifice or neck of the aneurysm with maximum cavity diameter thus providing valuable insights. Additionally, the detection of turbulent flow by pulsed Doppler echocardiography at the neck of the cavity, or within the cavity itself, can indicate the presence of a pseudoaneurysm. Besides overall cardiac function, regional wall motion abnormality, valve regurgitation, and pericardial effusion can be determined. MRI stands out for its ability to provide detailed visualization of the entire heart, enabling clear differentiation between various structures such as pericardium, myocardium, thrombus, and epicardial fat [4–6].

Despite the diagnostic challenges, prompt and accurate identification of pseudoaneurysm is imperative, as there is substantial risk of rupture and mortality [7]. Therefore, surgical intervention is the treatment of choice for pseudoaneurysm repair, as the potential consequences of fatal rupture outweigh the associated surgical risks [5]. The choice to proceed with surgical intervention in our case hinged on two primary considerations: the size of the pseudoaneurysm and the absence of baseline imaging predating hospitalization. Larger pseudoaneurysms inherently pose a heightened risk of rupture, thus warranting closer scrutiny and potentially necessitating prompt surgical management. Additionally, the absence of pre-hospitalization imaging deprives clinicians of valuable comparative data, further underscoring the need for surgical intervention to mitigate the risk of adverse outcomes associated with pseudoaneurysm rupture.

LV pseudoaneurysm poses a serious challenge to anesthetists because of its feared complications like congestive cardiac failure, arrhythmia, thromboembolism, and acute LV failure in the postoperative period [8]. Induction of anesthesia can be done with either high doses of opioids, ketamine, or etomidate depending upon the LVEF. Cardiac depressant drugs should be avoided; inhalational agents should be used cautiously, and histamine-releasing neuromuscular

drugs should be avoided. It is advised to have boluses of medications such as epinephrine, norepinephrine, and nitroglycerine ready to avoid sudden changes in blood pressure before repair. The MAP should be maintained between 60 and 80 mm Hg in such cases of LV pseudoaneurysm repair because spikes in afterload can lead to rupture of the aneurysmal sac, which can be catastrophic [8]. Cerebral oximetry is also required to ensure adequate cerebral perfusion during induction, cardiopulmonary bypass, and any resuscitation periods.

Conclusion

We describe a large LV pseudoaneurysm arising from RCA territory, which is an uncommon complication of MI. However, timely diagnosis and intervention can prevent catastrophic sequelae, even in large-size pseudoaneurysms, as is exemplified in this case report.

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Data Availability Raw data can be provided to interested parties on reasonable request.

Declarations

Informed consent Written informed consent was obtained from the patient.

Conflict of interest None.

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