# **CASE REPORT**

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# A simplified approach to managing a complex infected left ventricular pseudoaneurysm



# Abstract

**Background** Left ventricular pseudoaneurysm (LVP) is a rare but life-threatening condition resulting from acute myocardial infarction, trauma, bacterial infection, or previous cardiac operations. Diagnosis can be challenging as LVPs remain asymptomatic or present with nonspecific clinical symptoms. Early diagnosis is crucial to prevent rupture and recurrent septicemia. Various imaging techniques can aid in diagnosis, including transthoracic echocardiography (TTE), transesophageal echocardiography, computed tomography angiography, and cardiac magnetic resonance imaging.

**Case report** : A 72-year-old man with a history of coronary artery bypass grafting presented with episodes of recurrent fever. An infected LVP was diagnosed using TTE and thoracic Computed tomography (CT) angiography. The patient underwent removal of the infected hematoma with excision and repair of the pseudoaneurysm via left anterior thoracotomy with peripheral cannulation. The neck of the pseudoaneurysm was repaired with a Dacron patch. Post-operative TTE showed no residual pseudoaneurysm tissue, and the patient recovered well.

**Conclusion** Our experience with the anterior thoracotomy approach with peripheral cannulation in specific cases of infected LVPs has yielded promising results. However, it is crucial to recognize that this approach may not be universally suitable.

**Keywords** Pseudoaneurysm, Ventricular pseudoaneurysm, Case report, Management, Left ventricle pseudoaneurysm, Infected left ventricle pseudoaneurysm

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# Introduction

Ventricular pseudoaneurysm or false aneurysm is a rare but life-threatening disorder resulting from acute myocardial infarction, trauma, bacterial infection, and previous cardiac operations [1, 2]. It also can be a rare complication of left ventricular aneurysm repair [3]. They can be infected and cause recurrent septicemia [4]. The diagnosis of left ventricular pseudoaneurysm (LVP) could be suspected in any patient with a history of cardiac surgery who presents with pulsatile anterolateral chest wall mass. If the patient has a fever and recurrent sepsis, an infected LVP is more probable. False aneurysms are more likely to occur posteriorly and laterally than true



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aneurysms and tend to rupture [5]. Due to the high rates of morbidity and mortality associated with LVPA and the ongoing debate regarding the optimal approach, its management poses a significant clinical dilemma [2]. The surgical approach is usually through a median sternotomy [1, 6], but the anterolateral thoracotomy is also reported [1, 6].

We report a rare case of this infected LVP treated at our center, aiming to highlight the diagnostic challenges, the simplified surgical approach, and the clinical outcomes in managing this complex cardiac complication. The present study has been reported per the SCARE criteria [7].

# **Case presentation**

Written informed consent was obtained from the patient to publish this case report and accompanying images.

A 72-year-old man was referred to our center due to dyspnea and a fever of unknown origin. The patient had a history of recurrent fever for months and dyspnea on exertion (Function class II). He had a history of myocardial infarction and coronary artery bypass graft operation 11 years ago. He had hyperlipidemia but did not have hypertension or diabetes. The patient had no history of rheumatologic or immunosuppressive conditions, and previous assessments excluded the possibility of tuberculosis and brucellosis. The patient received broad-spectrum IV antibiotics for each fever episode and was on oral antibiotics after admission.

On examination, the patient was ill but not toxic, and blood pressure was 140/80, heart rate 88/min, respiratory rate 18/min, and oral temperature 36.8 °C. O2 Sat was 97% in room air. The electrocardiogram findings showed the heart rate was 75 beats per minute with a normal sinus rhythm and a normal axis. T-wave inversion was observed in leads I and V2 to V6. Furthermore, a Q-wave was identified in lead III and lead aVF. The other physical



**Fig. 1** Apical 4-chamber view of transthoracic echocardiogram showing apical akinesia with discontinuity and large pseudo aneurysmal formation in the apex

findings were unremarkable except for a palpable heave on the left ventricular apical area.

Laboratory data were erythrocyte sedimentation rate (ESR) 66, C-reactive protein (CRP) 12.2, procalcitonin 0.4, white blood count high 6800 (Neutrophils 70%), Hemoglobin 12, platelet count 257, and blood culture positive for Staphylococcus Aureus (Coagulase Positive) on 10th day of admission, it was sensitive to Amikacin, Cefepime, Cefoxitin, Ciprofloxacin, Clindamycin, Doxycycline, Erythromycin, Gentamycin, Linezolid, Methicillin, Vancomycin, Oxacillin, Cloxacillin and it was resistance to Penicillin-p. After 14th days of antibiotic therapy (vancomycin, gentamycin, and Cloxacillin), the blood culture was taken and negative. We used 2D transthoracic echocardiography (TTE) to measure LV and LA size, with volume-based estimates for EF following Simpson's method. TTE revealed that the left ventricular (LV) chamber size was severely enlarged, and no evidence of left ventricular hypertrophy was noted. The systolic function was severely reduced, with an estimated ejection fraction (EF) of 15-20%. The left atrium exhibited standard dimensions; no abnormalities were observed in the right ventricle or right atrium. The mitral valve was evaluated and found to be normal. The tricuspid aortic valve appeared normal, with no signs of aortic insufficiency or stenosis. Both the ascending and descending sections of the aorta exhibited normal flow patterns. The pulmonary valve and septum (intraventricular and intra-atrial) were normal. The inferior vena cava size was normal, and the pericardium appeared normal in imaging. TTE revealed severe LV enlargement (LVEDVI=129 ml/m2), severe LV systolic dysfunction (EF=15-20%), akinesia and loss of tissue in apical, anteroseptal, mid-anterior, and midinferior segments, a large echo-free space (3.7\*9.5 cm) around the apex and lateral side of LV with thickened and calcified margins containing septations which connect to apical-lateral wall of LV through a narrow neck (2.5 cm) with circumferential, thick mural thrombosis in the mentioned cavity, suggestive of LVP (Fig. 1) (movie 1).

The CT angiography revealed a patent left main coronary artery, and both the left internal mammary artery (LIMA) to the left anterior descending (LAD) artery and the saphenous vein graft (SVG) to the right coronary artery (RCA) was patent with the acceptable post-anastomotic flow. Two occluded SVG stumps were observed in the ascending aorta. The left anterior descending artery exhibited stenosis with calcified and noncalcified plaques. Additionally, the first diagonal branch (D1) displayed a mixed proximal plaque, while D2 and D3 were patent. The left circumflex artery showed diffuse disease, and the proximal and mid-portion of the right coronary artery exhibited plaque. However, the distal portion had no calcified plaque and mild stenosis after the SVG and right ventricular branch, and both the posterior



Fig. 2 (a, b) Four chamber and long axis two chamber Maximum Intensity Projection demonstrates large defect and pseudoaneurysm (white arrow)



Fig. 3 (a, b) Volume Rendering Technique shows pseudoaneurysm (white arrow), Saphenous Vein Graft (SVG) (orange arrow), and occluded SVG (yellow arrow)

descending artery and posterior ventricular branch were patent.

Thoracic CT angiography revealed a contrast-filled irregular border 105&102\*64 mm outpouching connected to LV apex with a tortuous neck with maximum and minimum dimensions of 26- and 8-mm extension of sub-endocardial calcification up to the neck of mentioned pseudoaneurysm and small luminal thrombosis was visible (Figs. 2 and 3). In coronary CT angiography, two SVG grafts to the first diagonal and first obtuse marginal branches were occluded with poor distal run-off. LIMA to LAD and SVG to RCA grafts were patent with good downstream vessel run-off. The operation took place 20 days later. Following the administration of heparin, cannulation of the left femoral vein and artery occurred, initiating cardiopulmonary bypass. Hypothermia was induced to 30 °C, and a left anterior thoracotomy was performed approximately 5 cm below the nipple, at the level of the 6th intercostal space (Fig. 4). A large LV pseudoaneurysm was identified, exhibiting significant adhesion to the surrounding tissues. Following the release of these adhesions, a temporary ventricular fibrillation was



Fig. 4 Incision, 5 cm below the left nipple



Fig. 5 Dacron patch closure of the neck of the pseudoaneurysm

induced, and the pseudoaneurysm was opened longitudinally (video 2). All infected clots and fibrotic tissues were removed (video 3), and the neck of the pseudoaneurysm was repaired with a piece of Dacron patch (Fig. 5). The patient was weaned off from CPB after 95 min easily. Post-operative TTE revealed no residual pseudoaneurysm tissue, and LV EF was increased to 35% (video 4). Also, the tissue culture after 72 h and 21 days were negative. Post-operative antibiotic therapy was maintained with vancomycin and gentamicin for 7 days. The patient experienced a favorable intensive care unit course, leading to a transfer to the ward after two days. Subsequently, five days later, they were discharged. Over the past four years postoperatively, our case has been under continuous observation, and we are pleased to report that the patient remains problem-free and in good health.

# Discussion

Ventricular pseudoaneurysm or false aneurysm is a rare and life-threatening condition. This outpouching happens when cardiac rupture contains pericardium, scar tissue, or thrombus, and there is no myocardial tissue [8]. It results from acute myocardial infarction, trauma, bacterial infection, or previous cardiac operations [1]. Patients with LVP can be completely asymptomatic or have nonspecific clinical presentations such as chest pain, arrhythmia, dyspnea, or symptoms of heart failure, acute MI, syncope, tamponade, and embolism that can delay their diagnosis. Also, they can be infected and cause recurrent septicemia [4, 8]. The diagnosis of LVP should be suspected in any patient with a history of cardiac surgery, especially with these nonspecific symptoms. Also, it may be presented with fever, draining fistula, recurrent sepsis, or even pulsatile anterolateral chest wall mass. In the literature review, Staphylococcus aureus was the most commonly reported causative microorganism, responsible for 52% of cases, excluding MRSA. Similarly, in our case, Staphylococcus aureus was identified as the cause of the infection [9]. False aneurysms are more likely to occur posteriorly and laterally, while true aneurysms occur in apical and anteroseptal walls [10]; however, in this case, there was a large pseudoaneurysm formation in the apex and lateral of LV.

Pseudoaneurysm aneurysms tend to rupture up to 45%, so early diagnosis and therapy are essential [3, 5, 11]. LVP diagnosis is by using noninvasive imaging techniques, including TTE, transesophageal echocardiography (TEE),

computed tomography angiogram, and cardiac magnetic resonance imaging [3]. The accuracy of TTE and TEE in providing a conclusive diagnosis is 26% and 75%, respectively [12]. The hallmarks of a pseudoaneurysm on echocardiography include a narrow neck and wide apex, as we detected in our patient, too [8, 13, 14]. In a case series of 23 patients with infectious LVP, echocardiography was the most commonly used imaging tool for diagnosis [9]. Ventricular angiography and Contrast ventriculography have helped diagnose LVP [12, 14]. For the identification of LVPs, multimodality imaging with various axes is advised, which also aids in defining margins and designing a patient's surgical plan [15].

Surgical repair of an LVP with patch closure is strongly recommended as a first option, particularly for patients with symptomatic or acute conditions, and an operation should be done as soon as possible [3, 5, 16]. However, conventional treatment may be appropriate in specific situations [17]. The surgical approach can be median sternotomy or thoracotomy. The selection process should account for the LVP's location, preoperative surgery, lung function, and the potential need for concurrent procedures [18]. Median sternotomy is typically performed using peripheral cannulation and deep hypothermia and may require total circulatory arrest [1]. While it offers complete access to the heart, especially in cases requiring revascularization, it has potential drawbacks, including the risk of cardiac chamber injuries, significant bleeding, and the need for extensive dissection during re-operative surgery. It's also important to note that surgeries of this nature tend to have longer durations. The anterior thoracotomy with peripheral cannulation we used in our patient approach has several advantages. It significantly reduces the need for extensive surgical dissection, eliminates the risk of aorta and right heart chamber injury, and maintains the integrity of previous coronary graft conduits, lessens the probability of sternal wound infection and dehiscence, and also diminishes the risk of peripheral thromboembolization, primarily due to reduced manipulation of the heart [18]. In our case, because of the location of LVP and the surgeon's decision to perform the procedure via left thoracotomy due to better accessibility. This approach, rather than the more traditional sternotomy, allowed for optimal exposure and repair of the pseudoaneurysm, demonstrating an innovative and patient-specific surgical strategy.

To prevent recurrent infections in the management of LVP, early identification of an infectious pseudoaneurysm is essential, as it significantly reduces the risk of complications, including recurrence. After that, delayed surgical intervention increases the likelihood of infection due to the prolonged exposure of necrotic or damaged cardiac tissue, which creates a breeding ground for bacteria. It is critical to remove all infected and necrotic tissue

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during surgery. Strict adherence to sterile surgical techniques is equally important to prevent the introduction of new pathogens. The use of biocompatible materials, such as pericardial patches, is recommended for repair, as proper closure minimizes the risk of infection by sealing off areas where bacteria could potentially invade or persist. Another critical component in avoiding recurrent infection is antimicrobial therapy. Targeted antibiotic treatment should be initiated preoperatively and postoperatively based on microbial cultures and sensitivities. Prolonged antibiotic administration, often extending over several weeks or even months, may be necessary to ensure complete eradication of the infection. Postsurgical monitoring is also vital. Patients must be closely observed for any signs of infection, as early detection and intervention can prevent recurrence and improve overall outcomes [2, 9].

In conclusion, this case report underscores the complexities associated with infected LVPs in redo surgery. It demonstrates the potential advantages of the anterior thoracotomy approach with peripheral cannulation in select cases. While our experience yielded favorable results, it's important to note that this approach is not universally applicable and may require careful patient selection. Further research and clinical studies are essential to evaluate this surgical technique's broader applicability and long-term outcomes.

Abbreviations

CPB	Cardiopulmonary bypass
CRP	C-reactive protein
CT angiography	Computed tomography angiography
EF	Ejection fraction
ESR	Erythrocyte sedimentation rate
LAD	Left anterior descending
LIMA	Left internal mammary artery
LVEDVI	Left ventricular end-diastolic volume index
LVP	Left ventricular pseudoaneurysm
LV	Left ventricle
MI	Myocardial infarction
RCA	Right coronary artery
SVG	Saphenous vein graft
TTE	Transthoracic echocardiogram
TEE	Transesophageal echocardiography

# **Supplementary Information**

The online version contains supplementary material available at https://doi.or g/10.1186/s13019-024-03194-9.

Supplementary Material 1	
Supplementary Material 2	
Supplementary Material 3	
Supplementary Material 4	
Supplementary Material 5	

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Not applicable.

#### Author contributions

YT, HP, and SH were involved in the study concept or design. SB, YT, MSh, HKh, and HP were involved in data collection. SB and MSh were involved in writing the paper under the supervision of YT, HKh, HP, and SH. All of the authors approved this version to be published. All authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part are appropriately investigated and resolved.

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# Data availability

The datasets and materials generated during the course of this case report are available upon reasonable request. Researchers interested in accessing the data for further validation may contact the corresponding author at Yasertolouei@yahoo.com.

# Declarations

## Ethics approval and consent to participate

This case report has received ethical approval from the Rajaie Cardiovascular, Medical and Research Center Ethics Committee. The study design complies with the ethical standards outlined in the Declaration of Helsinki.

## **Consent for publication**

Uploaded.

#### **Competing interests**

The authors declare no competing interests.

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