

Case Report

A Rare Case of Undiagnosed Post-Myocardial Infarction Left Ventricular Apical Pseudoaneurysm

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ABSTRACT

Introduction: Mechanical complications following acute myocardial infarction (MI) are associated with very high morbidity and mortality. Left ventricular (LV) pseudoaneurysms constitute a rare complication after MI. Considered a contained rupture of the LV free wall, an LV pseudoaneurysm is more prevalent in older age, the female sex, hypertension, and inferior and lateral wall MI. Echocardiography, computed tomography, and cardiac magnetic resonance are considered good noninvasive imaging modalities for the diagnosis of LV pseudoaneurysms.

Case: A 39-year-old man with a history of anterolateral MI 18 months earlier, coronary stent insertion, and implantable cardioverter-defibrillator implantation presented for follow-up, but he was incidentally diagnosed with LV pseudoaneurysm in transthoracic echocardiography, which was confirmed by cardiac computed tomography.

Discussion: Pseudoaneurysms must be diagnosed because of their high likelihood of rupture. However, as their clinical presentation is not specific, they are occasionally diagnosed incidentally. Clinicians should, therefore, always look for them in post-MI patients' echocardiography. (*Iranian Heart Journal 2023; 24(1): 91-96*)

KEYWORDS: LV pseudoaneurysm, Myocardial infarction, TTE, CMR, Cardiac CT

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Mechanical complications following acute myocardial infarction (MI) have been associated with very high morbidity and mortality.¹ Left ventricular (LV) pseudoaneurysms constitute a rare complication reported in fewer than 0.1% of all patients with MI.² Echocardiography, computed tomography (CT), and cardiac magnetic resonance (CMR) are considered good noninvasive

imaging modalities for diagnosing pseudoaneurysms.³

We herein present a rare case of a missed LV pseudoaneurysm diagnosed 18 months after an MI in a relatively asymptomatic young patient.

Case Presentation

A 39-year-old man with a family history of ischemic heart disease was referred to the heart failure clinic in our hospital. The

patient had a history of anterolateral MI 18 months previously when presenting with ventricular fibrillation to a local hospital, where after electrical cardioversion, electrocardiography (ECG) showed anterolateral MI and in bedside transthoracic echocardiography (TTE), a left ventricular ejection fraction (LVEF) of 20% to 25%, akinesia in all the apical segments and the anterior and anteroseptal walls, and moderate ischemic mitral regurgitation. After fibrinolytic therapy, he was referred to a percutaneous coronary intervention-capable hospital, where stenting with a 3.0×28 XIENCE Alpine drug-eluting stent was successfully performed on his totally cutoff left anterior descending artery (LAD) from the mid-part. After a few hours, he developed frequent episodes of nonsustained ventricular tachycardia, which was subsequently terminated with amiodarone administration. After 7 months, the patient visited his cardiologist complaining of progressive dyspnea. At the time, he had dyspnea at rest and S3 and rales in the cardiopulmonary physical examination. In echocardiography, he had an LVEF of 20% to 25% and the same regional wall motion abnormalities in the LAD territory. Consequently, he received a single-chamber implantable cardioverter defibrillator (Medtronic).

In our clinic, the patient was asymptomatic at rest and just had dyspnea on exertion (New York Heart Association functional class I). Additionally, he had a blood pressure of 126/82 mm Hg and a heart rate of 75 beats per minute. Electrocardiography showed an old anterolateral MI (Fig. 1). In echocardiography, the LV was moderately enlarged and had severe systolic dysfunction (LVEF=20%) and akinesia. Further, loss of tissue in the LAD territory, normal size and mild systolic dysfunction of the right ventricle, enlargement of the left atrium, moderate mitral regurgitation, mild-to-

moderate tricuspid regurgitation, and a systolic pulmonary artery pressure of 30 mm Hg were reported. A more precise evaluation of the LV in the off-axis views illustrated discontinuity of the myocardium in the apex and a large space full of thrombi adjacent to the LV apex with no flow in and out in color Doppler study, suggestive of a thrombosed pseudoaneurysm not reported in the previous echocardiography studies (Fig. 2 & 3 and Video 1).

Chest X-ray showed an abnormal contour of the LV apex (Fig. 4). Next, ECG-gated cardiac CT angiography (due to the previous cardioverter-defibrillator implantation and CMR contraindications), followed by multiplanar reconstruction and volume-rendering reconstruction, depicted the aneurysmal dilatation of the LV apex with mural thrombosis. The acute angle of the aneurysm border and the extension up to the visceral pericardium were suggestive of a pseudoaneurysm sealed by the pericardium (Fig. 5).

Due to the chronic total occlusion of the cavity by thrombosis with fibrotic adhesion to the pericardium diagnosed 18 months after an MI without any visible flow in the cavity in echocardiography and cardiac CT in a relatively asymptomatic patient and the high surgical risk because of severe LV systolic dysfunction, a conservative strategy was recommended to the patient. Aspirin was substituted with rivaroxaban (15 mg once a day) to reduce probable thromboembolism, and the doses of β -blockers and angiotensin-converting-enzyme inhibitors were raised. He was advised to reduce stress and avoid hard work. He was also scheduled for a follow-up visit 3 months later to evaluate his symptoms and hemodynamic state and to investigate any progression in the pseudoaneurysm size or any feature suggesting its impending rupture.

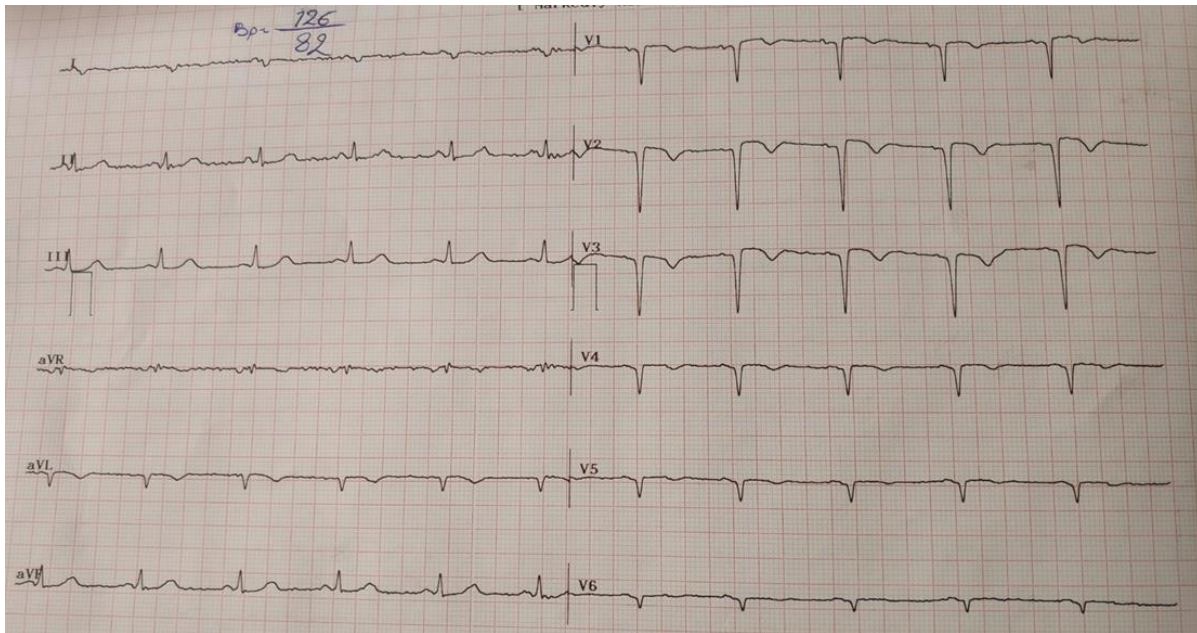


Figure 1: The electrocardiogram shows an old anterolateral myocardial infarction.

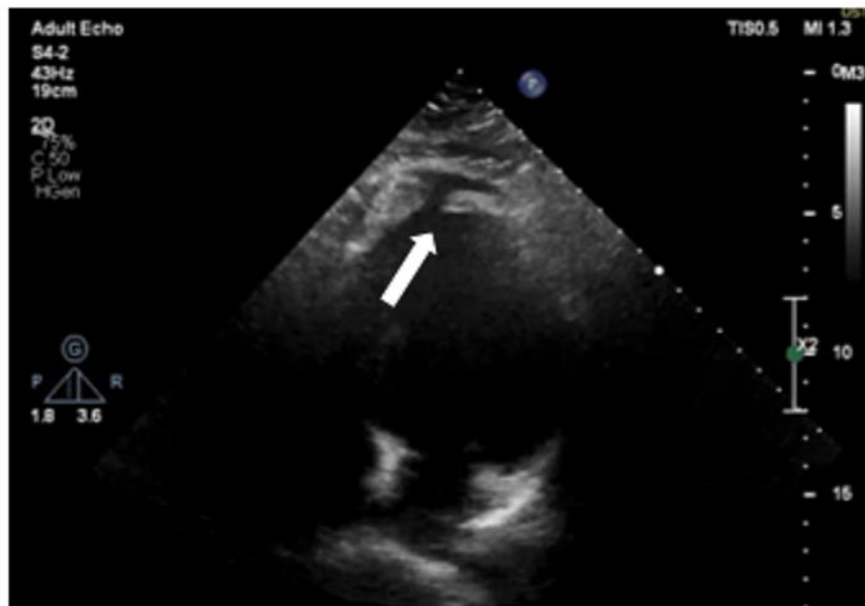


Figure 2: There is a defect in the left ventricular (LV) apex connecting the LV to a large space full of thrombi, suggestive of an LV apical pseudoaneurysm (the arrow).

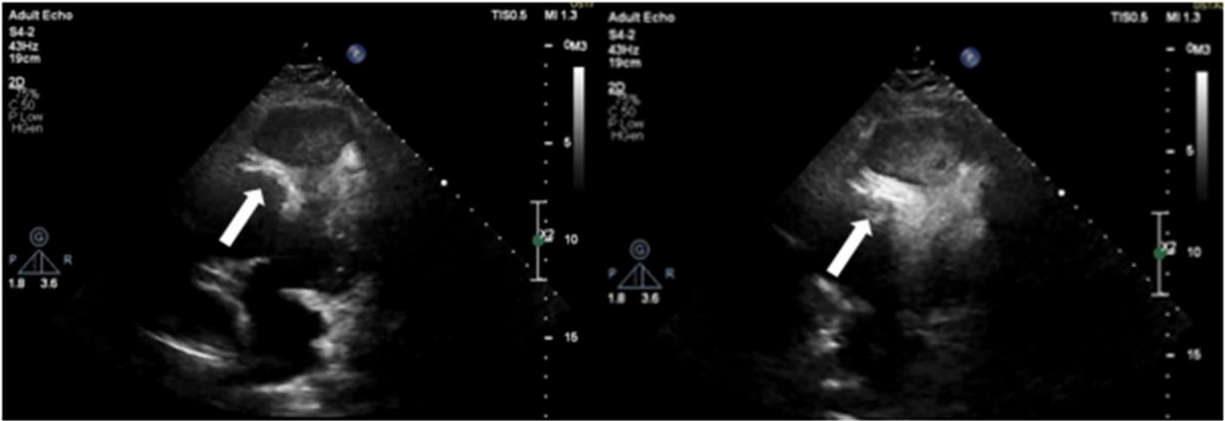


Figure 3: The images show a large thrombosed left ventricular (LV) pseudoaneurysm connected to the LV via a narrow neck (the arrows).



Figure 4: The chest X-ray shows the abnormal contour of the left ventricular apex (the arrow).

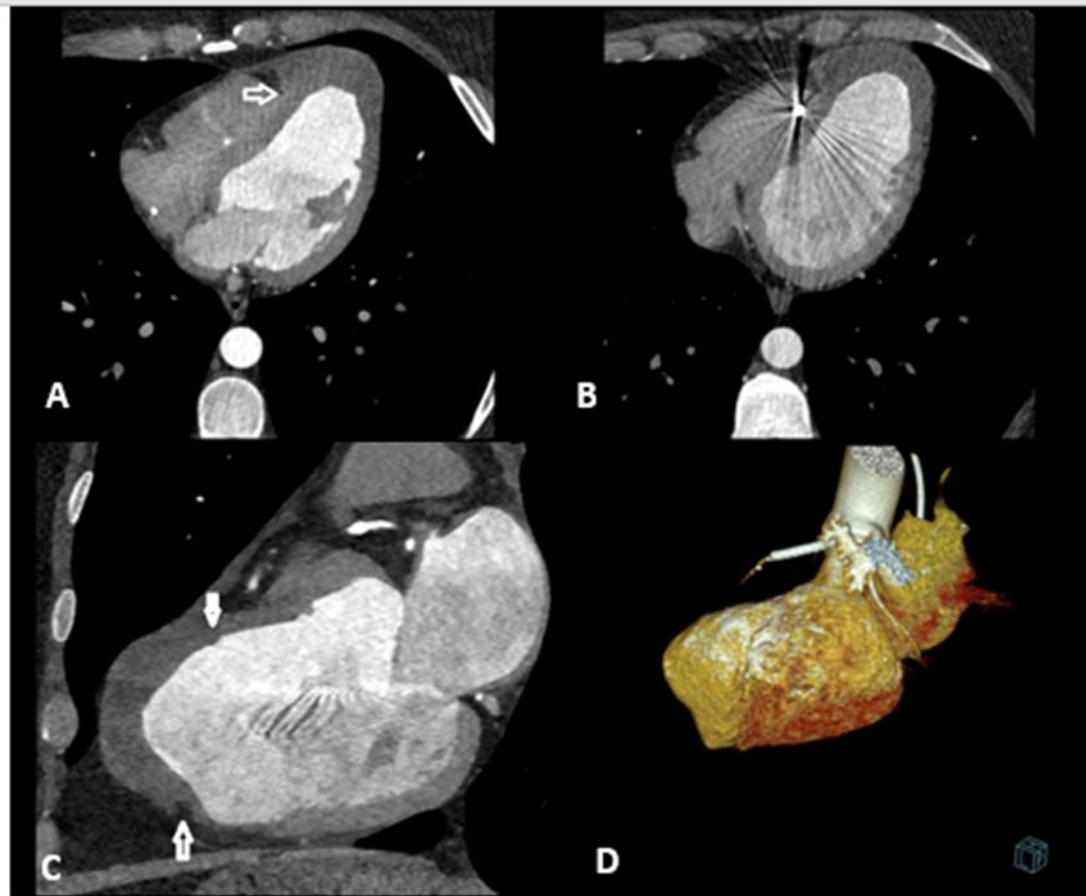


Figure 5: A and B) The axial view shows the aneurysmal dilatation of the left ventricular (LV) apex and mural thrombosis. The acute angle of the aneurysm is suggestive of a pseudoaneurysm (the arrow). **C)** The aneurysmal dilatation of the LV in the reconstructed 2-chamber LV is presented here. The acute angle of the aneurysm (the arrow) can be seen. **D)** The image shows the volume-rendering reconstruction of the LV and the aneurysmal dilatation of the LV.

DISCUSSION

A pseudoaneurysm is a contained rupture of the LV free wall and usually represents a complication of an acute MI.⁴ The risk factors for LV pseudoaneurysms after MI are older age, the female sex, hypertension, and inferior and lateral wall MI.⁵ Most pseudoaneurysms are located in the inferoposterior or inferolateral regions.⁴ The clinical presentation of patients is not specific and can mimic MI or heart failure, and patients could be asymptomatic (12%). Therefore, cardiac imaging plays a crucial role in the diagnosis.⁶ ECG and chest X-ray are nonspecific, but TTE has increased the number of diagnosed

LV pseudoaneurysms and is routinely used for the initial assessment in diagnosing LV pseudoaneurysms and also determining the extent of infarction.⁷ A distinguishing echocardiographic feature of a pseudoaneurysm is the narrow neck, with a ratio of the neck diameter to the maximum aneurysm diameter of less than 0.5 mm. Spectral and color Doppler imaging demonstrates a characteristic flow in and out of the pericardial cavity at the site of the tear within the pseudoaneurysm.⁴ CMR is a noninvasive modality for diagnosing LV pseudoaneurysms, detecting thrombi, and assessing myocardial viability. It provides a morphological definition of an LV

pseudoaneurysm's location, extension, and relation to the adjacent structures without the risk of radiation exposure. Moreover, it has a significant role in differentiating between LV true aneurysms and LV pseudoaneurysms, with a sensitivity of 100% and a specificity of 83%.⁸ Cardiac CT is another imaging modality used for diagnosing cardiac aneurysms; nonetheless, its limited temporal resolution, contrast use, and radiation risk have rendered it less favorable.⁹ Surgery is the first-line treatment associated with significantly lower mortality than conservative therapy.⁵ Still, there is disagreement concerning the best treatment of chronic cases given the evidence suggesting that patients with an LV pseudoaneurysm who remain hemodynamically stable can be managed medically, and it is sensible to take into account the hemodynamic and functional status as well as the comorbidity profile of each individual. Accordingly, the surgical repair of chronic pseudoaneurysms can be reserved for symptomatic patients, those diagnosed recently (<3 mon), and those with large (>3 cm) and progressively expanding pseudoaneurysms.¹⁰

We herein describe a young patient with an incidentally diagnosed LV pseudoaneurysm by TTE and cardiac CT 18 months after an anterolateral MI at a follow-up visit, indicating that we should always keep in mind MI mechanical complications and look for them in every visit, even months after the initial event. Additionally, diagnosing pseudoaneurysms by TTE is not without challenges since they can be missed easily. Thus, we should always use all standards and off-axis views to find them, especially when significant LV regional wall motion and akinetic walls are present.

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