Radiology Case Reports 19 (2024) 4242–4247

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Case Report

Cardiac MRI in the diagnosis and management of left ventricular pseudoaneurysms with previous myocardial infarction: A report of two cases ☆

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A R T I C L E   I N F O

Article history:
Received 19 June 2024
Revised 27 June 2024
Accepted 3 July 2024

Keywords:
Pseudoaneurysm
Cardiac magnetic resonance imaging
Left ventricular false aneurysm
Left ventricle
Case report

A B S T R A C T

A left ventricular pseudoaneurysm, an ominous consequence of acute myocardial infarction, poses a significant threat to patient well-being. Prompt and accurate diagnosis is crucial for improving patient outcomes. This report describes diagnostic imaging findings for identifying left ventricular pseudoaneurysms, emphasizing the critical role of cardiac magnetic resonance imaging alongside other modalities. We present two cases of patients with a history of myocardial infarction who presented with palpitations, chest pain, and shortness of breath. Initial 2D echocardiography in both patients revealed aneurysmal dilation of the left ventricle. Cardiac MRI was then performed, confirming the diagnosis in both cases.

Introduction

Left ventricular pseudoaneurysms are rare but serious complications that arise from contained myocardial wall ruptures. Unlike true aneurysms, which involve all layers of the heart wall, pseudoaneurysms are contained by a fragile combination of pericardium, thrombus, hematoma, and scar tissue. This distinction is critical, as the lack of structural integrity in pseudoaneurysms significantly increases the risk of rupture. While myocardial infarction (MI) and cardiac surgeries are recognized as primary causes, a significant portion of cases, approximately 48%, are detected incidentally in asymptomatic individuals [1]. For those who do experience symptoms, the clinical presentation is often nonspecific, encompassing heart failure, chest pain, syncope, and arrhythmias. It is important to emphasize that while free rupture into the pericardial space typically leads to fatal cardiac tamponade, in some instances, the rupture is contained, leading to pseudoaneurysm formation. This underscores the critical need for prompt diagno-

☆ Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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https://doi.org/10.1016/j.radcr.2024.07.007

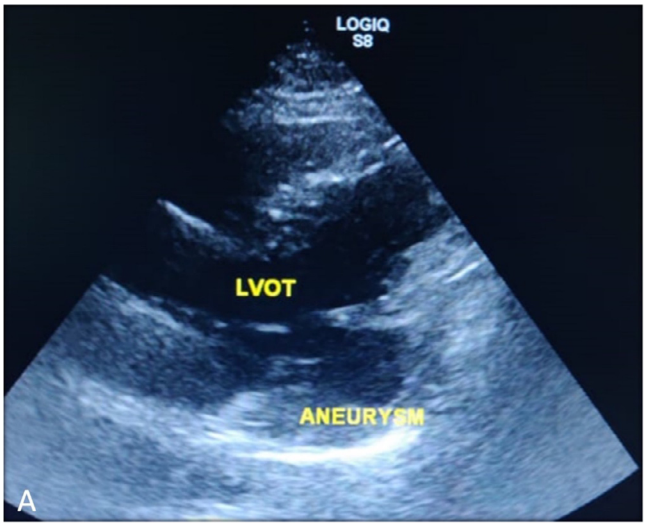

Fig. 1 – Transthoracic echocardiogram (2D, LVOT view) demonstrating an aneurysmal outpouching originating from the inferior wall of the left ventricle.

sis and intervention in suspected cases, as these pseudoaneurysms can rapidly expand, significantly increasing the risk of rupture [2].

Cases

Case 1

A 60-year-old female presented with a 12-year history of palpitations, chest pain, and dyspnea. She had a prior history of myocardial infarction of which details were not available. Physical examination revealed stable vital signs. A chest radiograph was obtained and demonstrated no significant abnormalities. Due to persistent palpitations, a 2D echocardiogram was performed. Transthoracic echocardiography, specifically the left ventricular outflow tract (LVOT) view, revealed an outpouching originating from the inferior wall of the left ventricle, measuring approximately 3 × 2 cm (Fig. 1). This outpouching displayed paradoxical systolic expansion, raising suspicion for a possible pseudoaneurysm.

To further characterize the finding, a computed tomography (CT) scan of the chest was obtained. Both coronal (Fig. 2A) and sagittal (Fig. 2B) reconstructions confirmed the presence of a well-defined outpouching arising from the inferolateral wall of the left ventricle, suggestive of a pseudoaneurysm.

Cardiac magnetic resonance imaging (CMR) was subsequently performed to evaluate myocardial perfusion and viability. Steady-state free precession (SSFP) bright-blood imaging in the short-axis view demonstrated the outpouching originating from the basal inferior and basal inferolateral wall of the left ventricle, measuring 3.1 × 2.6 cm (Fig. 3). Notably, this outpouching exhibited a narrow neck with a neck-to-mouth ratio of 0.5 and demonstrated prominent systolic expansion, further supporting the diagnosis of a pseudoaneurysm rather than a true ventricular aneurysm. Additionally, areas of wall thinning and hypokinesis were observed in the surrounding myocardium.

Short-axis views on delayed gadolinium enhancement (LGE) sequences, specifically phase-sensitive inversion recovery (PSIR) (Fig. 4A) and magnitude inversion recovery (Fig. 4B) images, revealed delayed near-transmural (>75% wall thickness) contrast enhancement. This enhancement pattern involved the apico-inferior, apico-lateral, basal, and mid-inferolateral and inferior segments of the myocardium, corresponding to the watershed zone between the RCA and LCX territories. Late enhancement was also prominent in the inferolateral wall of the left ventricle and extended to the periphery of the aneurysmal wall.

While surgery was recommended as the definitive treatment, the patient opted for medical management which included angiotensin converting enzyme inhibitors for afterload reduction, warfarin for anticoagulation and β-blocker as an antianginal medication. Some literature also supports this approach for post-infarction pseudoaneurysms, particularly small ones. A study by Moreno et al. [3] on 9 patients with post-infarct pseudoaneurysms treated conservatively showed an 88.9% survival rate at both 1 and 4 years, with no cases progressing to rupture. However, these patients faced a significant risk of ischemic stroke, with rates of 10% at 1 year rising to 33% at 4 years, underscoring the importance of anticoagulation therapy in nonsurgical management approaches.

The patient was discharged in stable condition on medical therapy, with instructions for careful monitoring and regular follow-ups. At the 6-month check-up, an echocardiogram showed no progression of the pseudoaneurysm, which remained unchanged in size. The patient reported only occasional shortness of breath during physical activity.

Case 2

A 45-year-old male presented with a 3-month history of palpitations and episodic chest pain. His past medical history was significant for a myocardial infarction 2 years prior. Transthoracic echocardiography demonstrated a dilated left atrium and left ventricle, with hypokinesis of the inferior and infero-septal walls. Grade II left ventricular diastolic dysfunction (LVDD) was also present. Of note, a large aneurysmal dilatation was visualized in the mid and basal infero-septal wall. Left ventricular ejection fraction (LVEF) was estimated to be around 30%.

Cardiac magnetic resonance imaging (CMR) was performed for definitive diagnosis and further evaluation. Steady-state free precession (SSFP) white-blood images (Fig. 5) revealed a large defect encompassing the basal inferior segment (segment 4) and extending into the mid-ventricular inferior segment (segment 10) of the left ventricle. This defect measured 4.9 × 4.5 cm (short-axis × long-axis). The left ventricular cavity was in direct communication with a large aneurysmal outpouching, measuring 10.0 × 6.5 cm (short-axis × long-axis), contained by the pericardium. The dependent portion of this aneurysmal outpouching contained a layered thrombus with a maximum thickness of 17 mm, which was non-enhancing on first pass perfusion imaging.

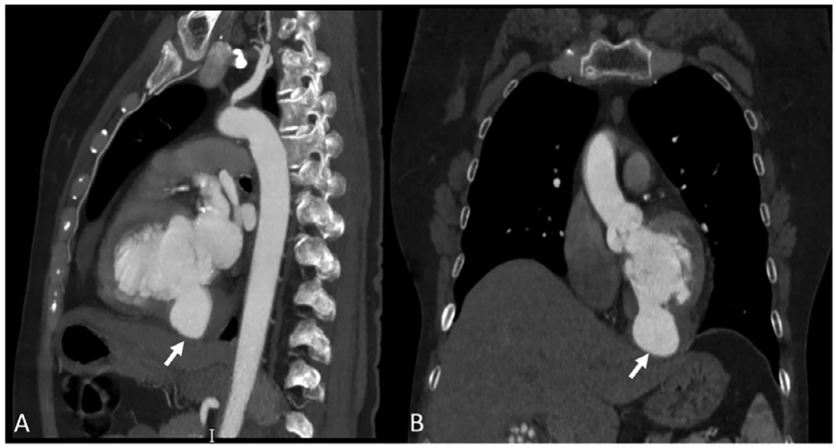

Fig. 2 – Cardiac CT images. (A) Coronal reconstruction. (B) Sagittal reconstruction. Both views demonstrate an outpouching (arrow) arising from the inferolateral wall of the left ventricle, consistent with a pseudoaneurysm.

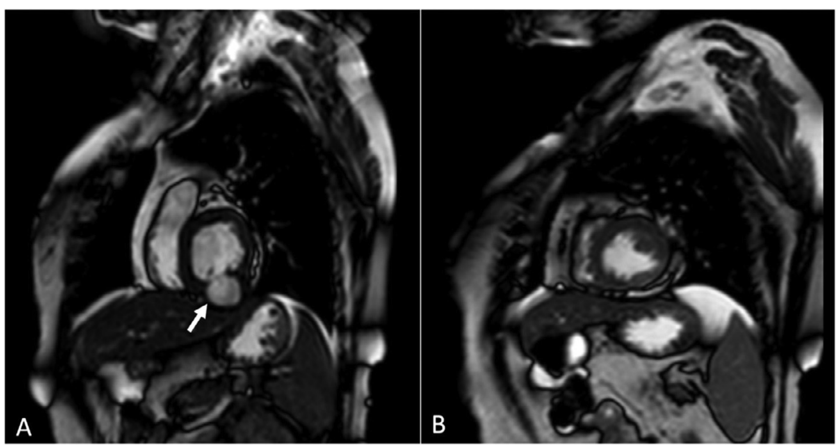

Fig. 3 – Cardiac MRI, SSFP bright-blood imaging (short-axis view). The image shows the outpouching (arrow) originating from the basal inferior and basal inferolateral wall of the left ventricle, notable for a narrow neck.

Late gadolinium enhancement (LGE) sequences (Fig. 6) demonstrated transmural delayed enhancement of the adjacent basal inferolateral segment (segment 5), suggesting prior infarction in this territory. Endocardial and subendocardial enhancement were also observed in the mid-ventricular inferior and inferolateral segments (segments 10 and 11). Notably, marked delayed enhancement of the surrounding pericardium was present.

Left ventricular (LV) volumes were calculated both with and without the inclusion of the aneurysmal sac. Excluding the aneurysm, LV end-diastolic volume index (LVEDVi) and LV end-systolic volume index (LVESVi) were 165.94 mL/m^2 and 114.35 mL/m^2, respectively. Including the aneurysm, LVEDVi and LVESVi were 260.43 mL/m^2 and 219.27 mL/m^2, respectively. Left atrial (LA) volume was also enlarged, measuring 63.72 mL, with an LA volume index (LAVI) of 41.11 mL/m^2. Mild pericardial effusion was noted with a maximum pericardial thickness of 10.4 mm. LVEF calculated by CMR was 31.09% when excluding the aneurysm and 25.80% when including the aneurysmal sac.

Given the pseudoaneurysm's considerable dimensions and the significant risk of unexpected rupture, which could lead to sudden pericardial tamponade or systemic embolism, the patient was advised surgical intervention. The proposed procedure involved an urgent aneurysmectomy using a double-layer heterologous pericardial patch, along with necessary coronary artery bypass surgery. Despite this recommendation, as the patient's condition was stable and they had no other notable health issues, they opted for self-referral to specialized center for further management.

Discussion

Cardiac pseudoaneurysms, a rare complication following transmural myocardial infarctions, have a prevalence rate of 0.2% to 0.3% [4]. They are particularly associated with extensive ischemic heart disease, though they can also occur after cardiac surgery, chest trauma, and infections [2]. This condi-

Fig. 4 – Cardiac MRI, delayed gadolinium enhancement, short-axis views. (A, B) Phase-sensitive inversion recovery (PSIR) image. (C, D) Magnitude inversion recovery image. Both images demonstrate delayed near-transmural contrast enhancement in the inferolateral wall and extending to the periphery of the aneurysmal wall formed by pericardium (arrows).

tion develops when the overlying pericardium contains a ventricular free wall rupture. The progression often leads to rupture and sudden death due to tamponade. Risk factors include the use of corticosteroids, NSAIDs, and the presence of arterial hypertension [5]. These pseudoaneurysms are often silent, detected incidentally during routine imaging or postmortem [1]. Clinical examination may range from normal to showing heart failure signs due to progressive enlargement of pseudoaneurysmal cavity, possibly including a new murmur at auscultation. Some patients may present with ventricular arrhythmias or thromboembolic complications due to thrombus expulsion from the aneurysmal sac to the ventricular cavity [5].

LV pseudoaneurysms are predominantly observed in the posteroinferior wall and basal segments rather than the apical segments. The scarcity of anterior LV pseudoaneurysms may be attributed to the higher risk of hemopericardium and mortality associated with anterior ruptures compared to posterior ones [1,6]. Also, the recumbent position of most hospitalized patients can lead to an inflammatory response in the posterior pericardium, resulting in pericardial adhesions and the formation of a posterior LV pseudoaneurysm [2,6].

Distinguishing between true and false aneurysms is paramount for appropriate management, with true aneurysms typically managed medically unless complications such as heart failure or arrhythmias arise, whereas pseudoaneurysms demand urgent surgical intervention due to their lack of myocardial wall, rendering them prone to rupture. Pseudoaneurysm and true aneurysm can be differentiated using multimodal approach involves utilizing various imaging techniques for cardiac assessment, including transthoracic echocardiogram (TTE), trans-esophageal echocardiogram (TEE), cardiac magnetic resonance (CMR) imaging, and cardiac computed tomographic angiography (CCTA).

The TTE is widely accessible, cost-effective, and easily obtainable at the bedside, providing assessments of hemodynamics, structure, and function. However, it is reliant on operator skill, offers limited tissue characterization, and may overlook off-axis findings. TEE, available in most medical centers, offers excellent anatomical and functional images but is more invasive, time-consuming, and requires patient cooperation. In TTE, a pseudoaneurysm is characterized by a narrow neck with a neck to maximal diameter ratio of 0.25–0.5, and turbulent flow detected by pulsed Doppler at the neck of the outpouching. Conversely, a true aneurysm shows a wide neck with a neck to maximal diameter ratio of 0.9–1.0 [7].

CMR imaging provides superior structural and tissue characterization, particularly for scars, thrombi, and masses, with

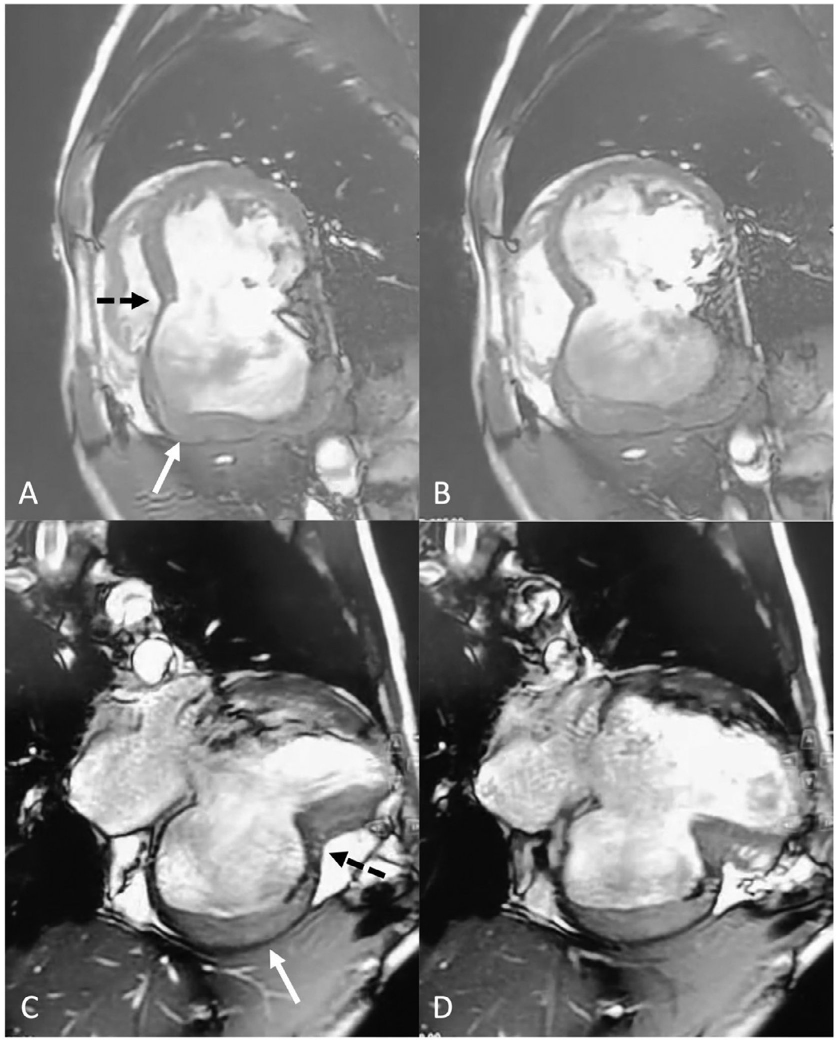

Fig. 5 – Cardiac MRI using steady-state free precession (SSFP) white-blood imaging. (A) Short-axis view and (C) 2-chamber view during systole demonstrates a large defect (black dashed arrow) encompassing the basal inferior and mid-ventricular inferior segments of the left ventricle. The left ventricular cavity communicates with a large, pericardially contained aneurysmal outpouching (white arrow) containing a layered thrombus. (B) and (D) show the corresponding views in diastole.

unique information from delayed enhancement imaging as to identify the location and transmural extent of previous infarcts. In CMR imaging, pseudoaneurysms display a narrow neck to maximal diameter ratio of 0.25–0.5, an abrupt transition between healthy and scarred myocardium. A reduction of more than 50% in the wall thickness of the aneurysm sac, measured 1 cm from the aneurysmal neck and referred to as the "Myocardial cut-off sign," has demonstrated high sensitivity (91%) and specificity (97%) for diagnosing ventricular pseudoaneurysms [8]. This is clearly demonstrated in our Case 2 as well (Fig. 6). There is enhancement noted of the overlying pericardium on delayed enhancement imaging, which possibly results from blood released during the immediate aftermath of cardiac rupture causing pericardial chemical irritation, which in turn causes an inflammatory response and pericardial neovascularization [6,9]. True aneurysms, on the other hand, exhibit a wide neck to maximal diameter ratio of 0.9–1.0, a smooth transition between healthy and scarred myocardium, and non-enhancing pericardium post-MI in delayed enhancement imaging. CMR's role extends beyond diagnosis to assessing myocardial viability, identifying thrombus formation, epicardial fat and evaluating the relationship between the aneurysm and adjacent structures such as heart valves. Nevertheless, CMR is time-consuming, expensive, and necessitates both a cooperative patient and specialized expertise.

CCTA offers rapid image acquisition, wide imaging windows, 3D capabilities, and precise coronary artery anatomical information but involves radiation exposure and does not provide hemodynamic data. CCTA is especially useful for pseudoaneurysm associated with prosthetic endocarditis [10].

Untreated ventricular pseudoaneurysms carry a significant rupture risk of 30% to 45% due to the absence of the

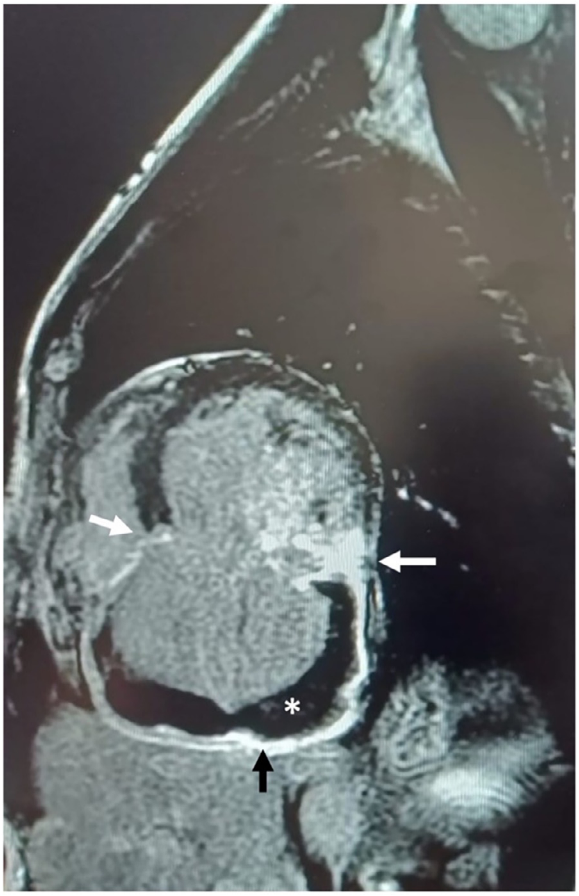

Fig. 6 – Cardiac MRI, late gadolinium enhancement image (short-axis view). Transmural delayed enhancement is present in the basal inferolateral segment (white arrows). Layered thrombus is noted in the pseudoaneurysm (*). Marked delayed enhancement is also present in the surrounding pericardium (black arrow).

myocardial wall, necessitating urgent surgical intervention [2]. In contrast, true aneurysms, less prone to rupture, are typically managed medically, with surgery reserved for complicated cases. For patients with large, symptomatic pseudoaneurysms, proactive surgical management is crucial, which itself is challenging and related with significant morbidity [11]. However, a conservative approach is suggested for asymptomatic patients, particularly those with smaller, chronic pseudoaneurysms discovered incidentally and posing high surgical risk [1,3,12]. Percutaneous device closure serves as an alternative, especially for patients with smaller lesions or those requiring repeat cardiac surgery [13]. The best therapeutic strategy requires a careful evaluation of the benefits and drawbacks of surgical intervention.

Patient consent

Written informed consent were obtained from the patients for publication of their cases.

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