WWW.IJRPR.COM

International Journal of Research Publication and Reviews

Journal homepage: www.ijrpr.com ISSN 2582-7421

Voluminous Left Ventricular Aneurysm Following Myocardial Infarction: From Clinic to Surgery: A Case Report

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ABSTRACT:

The management of myocardial infarction has significantly improved over the past few decades, leading to notable improvements in patient prognosis and a reduction in both short- and long-term complications. However, the severity of this condition lies in its complications, particularly mechanical ones, which can occur in the first few hours or in the weeks following the infarction. One such complication is left ventricular (LV) aneurysm, which predominantly occurs in transmural myocardial infarctions. This results in an area of scarring that forms a thin-walled pocket with a wide, protruding neck. This region of severe dyskinesia no longer participates in systolic contraction and is at risk of rupture. Diagnosis is based on both echocardiography and cardiac MRI. Therapeutic decisions depend on the size of the aneurysm and its impact, with surgical treatment being necessary in several situations. The presence of a left ventricular aneurysm is associated with a significantly reduced survival rate, making it crucial to manage it effectively, both diagnostically and therapeutically.

We present the case of a patient who developed an extensive anterior myocardial infarction, complicated a few weeks later by a large apical left ventricular aneurysm that required surgical intervention.

KEY WORDS: Myocardial infarction, Ventricular aneurysm, Dor technique, Myocardial patch.

Consent: The authors confirm that written consent for the submission and publication of this case, including the images, was obtained from the patient, in accordance with the guidelines of the Committee on Publication Ethics (COPE).

Publication Ethics: In accordance with COPE guidelines.

Conflict of Interest: No conflict of interest declared.

Funding: No funding declared.

Availability of Data and Materials: Data sharing is not applicable to this article, as no datasets were generated or analyzed during this study.

LIST OF FIGURES:

INTRODUCTION:

Myocardial infarction can be a severe condition, associated with life-threatening complications. These complications must be promptly identified to ensure that appropriate and early treatment is initiated, thereby reducing morbidity and mortality. These complications can be mechanical in nature, such as left ventricular (LV) wall rupture, LV aneurysm, mechanical mitral regurgitation, and ventricular septal defect. In this article, we will focus on LV aneurysm, detailing the various aspects of its diagnostic and therapeutic management in a patient admitted to the cardiovascular surgery department of the Mohamed V Military Training Hospital in Rabat.

CLINICAL CASE:

We report the case of a 69-year-old patient with the following cardiovascular risk factors: hypertension managed for 3 years with calcium channel blockers, type II diabetes for 5 years poorly controlled with oral anti-diabetic agents (latest HbA1c: 7.2%), complicated by diabetic neuropathy and nephropathy, and dyslipidemia for 2 years treated with statins. The patient had been hospitalized one month prior for the management of an extensive anterior myocardial infarction (MI) related to thrombotic occlusion of the middle anterior interventricular artery, which was successfully treated with balloon dilation.

The patient had intermediate angiographic disease complicated by a flare-up of left ventricular (LV) failure, which was controlled with medical treatment, resulting in a favorable clinical outcome.

The patient was admitted for the investigation of a rapidly progressive worsening of baseline dyspnea to NYHA stage IV, associated with orthopnea and increased lower limb volume, without chest pain. Clinical examination revealed a hemodynamically stable patient with clear signs of congestive heart failure: bilateral lower limb edema up to the knees, hepatomegaly with hepatalgia, and bilateral crackles extending to mid-field on pulmonary auscultation. The electrocardiogram showed signs of electrical LV hypertrophy, a flattening of the anterosepto-apical R wave, with persistent non-significant ST segment elevation in the same territory (same appearance on previous tracings).

Chest X-ray showed cardiomegaly (TIA: 0.65 with a subdiaphragmatic peak), bilateral alveolar-interstitial syndrome, and bilateral pleural effusion. Biological findings were consistent with iron-deficiency anemia, chronic renal failure with an estimated glomerular filtration rate (GFR) of 33 ml/min/1.73 m², and slightly elevated troponin levels.

Transthoracic echocardiography revealed a non-dilated left ventricle with concentric hypertrophy due to hypertension. Segmental kinetics revealed akinetic-like abnormalities of the apex and adjacent segments, the inferoseptal wall, and the medial segments of the anterior, lateral, and anteroseptal walls. Systolic function was significantly impaired with an ejection fraction estimated at 16% at standard body position. The left ventricular apex was difficult to assess in its entirety due to the presence of a giant aneurysm, with no possibility of ruling out the presence of a thrombus. In addition to left ventricular function abnormalities, echocardiography revealed dilated atria with minimal mitral and tricuspid regurgitation.

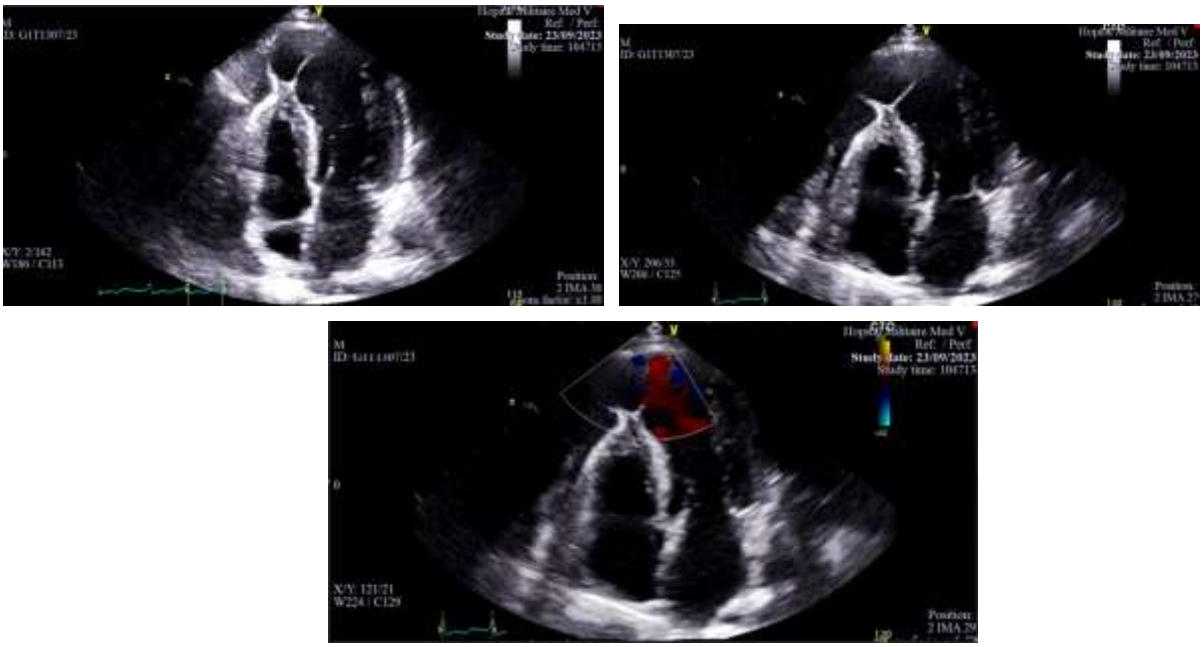

Figure 1: Echocardiographic images in 4-chamber apical view in two-dimensional mode and color Doppler highlighting the apical aneurysm of the left ventricle.

Cardiac MRI confirmed the presence of a giant apical aneurysm measuring 71 mm at the neck and 67 mm in depth, with a residual cavity volume estimated at 85 ml/m², containing a thrombus measuring 7 mm x 10 mm, as well as a small pericardial effusion.

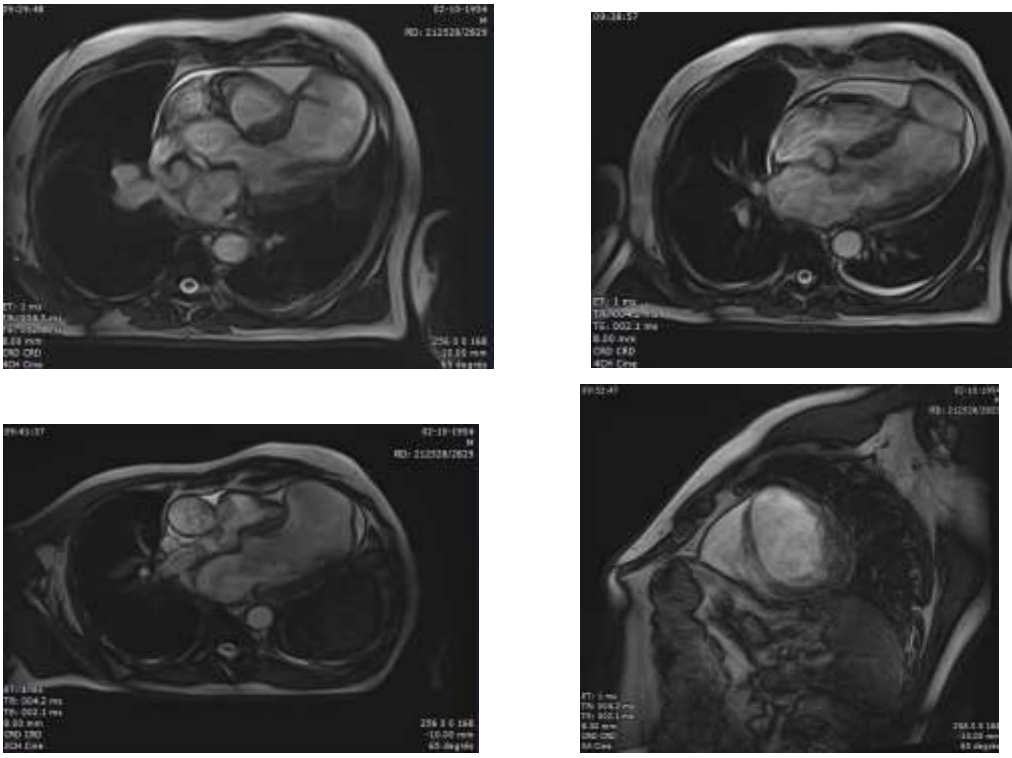

Figure 2: SSFP MRI images in 3-chamber, 4-chamber, and short-axis views at the apical level showing the apical aneurysm of the left ventricle, measuring 71 mm at the neck and 67 mm in depth.

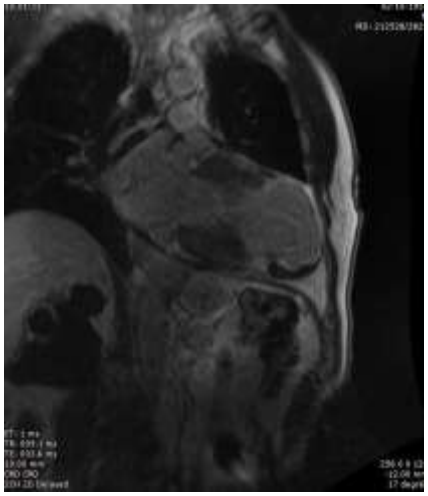

Figure 3: Late MRI sequences in a 2-chamber view showing late enhancement of the walls of the aneurysmal sac with the presence of a small thrombus measuring 7 mm x 10 mm.

After a complete preoperative workup, the therapeutic decision was to perform aneurysm excision using the Dor beating-heart technique to avoid myocardial ischemia. The procedure was performed as follows:

After stable anesthetic induction, a median sternotomy and pericardotomy were carried out, revealing non-dilated heart chambers with a giant aneurysm at the apex of the left ventricle adhered to the pericardium. After heparinization, the aneurysm was freed from pericardial adhesions. The heart was dislocated, the apex explored, and the apical aneurysm opened longitudinally in the beating heart. The thrombus in the aneurysm sac was removed, and a double 4/0 Prolene suture was placed around the aneurysm neck to exclude this portion from the left ventricle. Resection and closure of the neck were performed with a 4/0 Prolene suture after a gas purging maneuver. The excess aneurysmal sac was resected, and the defect was covered with a 4/0 Prolene Dacron patch using Dor's technique. The weaning off from extracorporeal circulation was achieved under Dobutamine.

Post-operative follow-up was straightforward, with echocardiographic evidence of the patch at the apex, persistent segmental kinetic abnormalities, systolic dysfunction, and spontaneous left ventricular contrast. Longitudinal function of the right ventricle was impaired, with a dry pericardium.

DISCUSSION:

Myocardial infarction is a serious and common condition that significantly increases morbidity and mortality. It can lead to a number of mechanical, rhythmic, embolic, and inflammatory complications. Ventricular aneurysm is one of the most frequent complications, first diagnosed in 1951, but its incidence has declined significantly with the advent of coronary reperfusion. In the CASS coronary bypass study, 7.6% of patients had a left ventricular aneurysm out of a total population of 15,000 patients.

Ventricular aneurysm can develop in the early phase, between 48 hours and 2 weeks after myocardial infarction. It refers to a well-defined transmural scar zone, taking the form of a pocket with a thinned wall due to the absence of cardiomyocytes, and a smooth ventricular inner surface due to the complete disappearance of the fine trabeculations characteristic of the left ventricular cavity. This protrusion communicates with the rest of the left ventricle via a wide neck. Having lost its contractile function due to necrosis, it protrudes outside the left ventricle even during systole. The expansion of this fragile necrotic zone likely occurs as a result of myocardial stretching, causing it to bulge further with each contraction.

This complication is primarily observed in extensive transmural infarctions (85-90%). The two main factors favoring its formation in the early phase are: total occlusion of the anterior interventricular artery and the acute nature of the lesion, which prevents the development of the collateral network. In addition to these two factors, the adjacent myocardium must retain normal kinetics, and there must be no reperfusion, arterial hypertension, ventricular dilation, or parietal thinning. This explains why 85% of left ventricular aneurysms occur in the apical and anteroseptal walls, compared to 5-10% in the inferior and lateral walls. This preferential location is probably explained by the fragility of the apical region.

However, aneurysms can also develop in the late phase, between 2 and 4 weeks after the ischemic event, due to ventricular remodeling and the replacement of myocytes by fibrous tissue.

Other etiologies can cause ventricular aneurysms, including infectious, traumatic, post-partum causes, and certain diseases such as sarcoidosis and Chagas disease. Idiopathic cases are very rare.

The endoventricular appearance of the aneurysm, which may be lined by a thrombus, differentiates it from a false aneurysm, which is a ventricular rupture contained within a partitioned pericardium and is even rarer.

The clinical presentation correlates with the size of the aneurysm. Smaller aneurysms are often asymptomatic and typically only become evident when complications arise or during routine follow-up echocardiography. In contrast, the clinical presentation of large aneurysms can range from isolated functional signs to severe arrhythmias or even sudden death. Symptoms may include fatigue, dyspnea on exertion or at rest, chest pain, palpitations or syncope, signs of right and/or left heart failure, stroke, or signs of visceral or limb thromboembolism.

Clinical examination may reveal a murmur of mitral insufficiency, a bifocal auscultation at the apex, or signs of heart failure.

The suggestive electrocardiographic tracing shows the persistence of ST-segment elevation beyond 3 weeks after acute ischemia, with Q-waves indicating necrosis and T-wave inversion or, at a minimum, low amplitude in the same territory. These signs are characteristic if the tracing remains stable without modification. Another sign, known as Goldberger's wave, is the presence of a large R wave in AVR. Electrical abnormalities are not correlated with the size of the aneurysm. The arrhythmias observed may indicate complications arising from the aneurysm.

Chest X-rays, part of the routine examination, may reveal cardiomegaly, aneurysmal deformation of one of the arches of the heart, and, in chronic cases, rare calcifications.

Imaging is key to the diagnosis. Echocardiography offers sensitivity and specificity of around 90%. Thanks to apical incidences, echocardiography can be used to search for ventricular aneurysms, as it provides a better characterization of the left ventricular cavity, and because aneurysms are frequently located anteriorly and apically.

The aneurysm appears as a thin-walled protrusion in a territory of severe dyskinesia, communicating with the left ventricle via a wide neck. Echocardiography is not only used to locate and determine the shape and measurements of the aneurysm but also to differentiate it from a false aneurysm, which has a narrow neck. The deformation persists in both diastole and systole and is characterized by oscillatory motion on pulsed Doppler that varies with inspiration and expiration. The operator must systematically look for the presence of a thrombus at this level and check for a potential mitral valve leak, which may be associated. Echocardiography also assesses altered systolic function and segmental kinetics of the left ventricle in relation to ischemic heart disease.

Other imaging techniques can complement this examination in cases of diagnostic doubt or for a better preoperative assessment of the aneurysm, notably MRI, cardiac angioscanner, and, more rarely, left ventriculography.

In this context, magnetic resonance imaging is a reliable, high-performance, non-invasive means of identifying and assessing the resectability of ventricular aneurysms and detecting intra-aneurysmal thrombosis. It is also the gold standard for assessing ischemic heart disease and, above all, for studying myocardial viability in the affected territory, as well as guiding the indication for revascularization in certain patients.

Computed tomography (CT scan) is another reliable imaging modality for this condition, offering better visualization of the aneurysm wall and the presence of mural thrombus, though it carries the risk of contrast-induced nephropathy.

Left ventriculography has become the "gold standard" and the most accurate test for diagnosing and locating the site of a left ventricular aneurysm. It shows a large area of dyskinesia or akinesia, generally in the anterosepto-apical walls.

Ventricular aneurysms can be complicated by ventricular arrhythmias, manifested by palpitations or even syncope, and may lead to sudden death. This is due to the transformation of myocardial tissue into fibrous tissue, creating a focus of arrhythmia between normal and pathological myocardium. The ventricular aneurysm is a major contributor to the formation of wall thrombi at this site through blood stasis.

The thrombus may fragment or detach, causing embolism to various organs, most notably leading to a stroke.

The most dreaded complication of aneurysms is rupture, which can lead to cardiac tamponade. This is more common in early, immature aneurysms. Mature aneurysms are less likely to rupture, as they are composed of dense fibrous tissue.

As a result, this condition is associated with high cardiac mortality, reaching around 67% at 3 months and 80% at 1 year, respectively.

In clinical studies, prognosis depends on aneurysm size and whether it is symptomatic. In the study by Mourdjinis et al., the 5-year mortality rate was 50% overall, but only 10% in patients with small aneurysms.

In the prospective CASS study, the 4-year cumulative survival rate for patients with left ventricular aneurysms was 71%. Mortality rates were influenced by the extent of coronary disease and the degree of left ventricular dysfunction, highlighting the importance of intervening before potentially fatal complications develop.

Management of left ventricular aneurysms involves both medical and surgical approaches.

Medical treatment is recommended for all aneurysms. Surgery is not indicated for small, asymptomatic aneurysms, which can be closely monitored for safety, with a survival rate of 90% over the course of 5 years.

Management may include optimizing risk factors for coronary artery disease through the use of an angiotensin-converting enzyme (ACE) inhibitor, or within the first 24 hours post-infarction, to inhibit ventricular remodeling. Angiotensin II receptor antagonists and anti-aldosterone medications can also have similar effects. The introduction of beta-blockers is crucial at this stage.

Treatment of acute coronary syndrome with thrombolytics or angioplasty can help prevent the formation of a ventricular aneurysm.

Corticosteroids and non-steroidal anti-inflammatory drugs (NSAIDs) should not be used in the first few days after a myocardial infarction, as they can interfere with myocardial healing and promote aneurysm formation.

Regarding anticoagulation, due to the increased risk of mural thrombus formation within the aneurysm, it is generally advisable to initiate anticoagulant therapy (warfarin) for the first three months following myocardial infarction. The duration of anticoagulation may be extended if the thrombus is large, friable, or protruding into the ventricular cavity, or if there is documented systemic embolism beyond the initial three months. It may also be continued in cases of severely impaired left ventricular function, especially in the absence of high bleeding risk.

In patients with large, symptomatic left ventricular aneurysms, medical treatment remains the same, with specific treatment of complications and surgical management as needed.

The indications for surgery are primarily refractory heart failure, persistent angina, arrhythmia unresponsive to medical treatment, and the presence of embolic events. It is important to note that whenever cardiac surgery is indicated in a patient with a ventricular aneurysm, the aneurysm must be addressed.

The indication for aneurysm resection due to isolated arrhythmia (circumferential ventriculotomy, cryoablation of the arrhythmogenic zone) has largely been replaced by the placement of an implantable defibrillator, given the lower mortality risk.

Jatène-Dor endoventriculoplasty involves performing an aneurysmectomy with myocardial reconstruction using a patch to restore the left ventricle's morphology under extracorporeal circulation and a balanced anesthetic technique. This procedure may be combined with coronary bypass surgery and the surgical treatment of complications such as associated mitral regurgitation, when indicated. When combined with bypass surgery, systolic function is gradually restored.

The goal of this surgery is to eliminate the fibrotic tissue, which serves as the site for arrhythmias, and to restore normal ventricular size while preserving the left ventricular morphology. The size of the left ventricle after surgery is crucial for optimal outcomes, highlighting the importance of pre- and postoperative three-dimensional imaging, with an ideal telediastolic volume averaging 150 mL after correction.

Jatène-Dor circular patch endoventriculoplasty provides satisfactory results, ensuring complete exclusion of the aneurysm, including at the level of the interventricular septum.

Surgical treatment improves the contractile function of the left ventricle by restoring ventricular tension during systole, eliminating the akinetic zone, and reducing parietal stress, especially when combined with coronary bypass surgery.

Operative mortality and long-term survival rates are highly dependent on the quality of revascularization. However, literature reports a perioperative mortality rate of 3 to 6.5% in isolated Dor surgery, and 3 to 23% in cases involving additional procedures. The average 5-year survival rate rarely exceeds 30%, though it remains higher than that of the equivalent non-operated population.

Anatomopathological examination after surgery confirms that the aneurysm wall is thin, no more than 5 mm thick, and consists solely of fibrous tissue with a few necrotic cardiomyocytes. The presence of calcification characterizes a true ventricular aneurysm, which often contains a thrombus lining the wall, with both old and recent thrombotic elements.

CONCLUSION:

Despite major advances in the management of myocardial infarction, mechanical complications particularly left ventricular aneurysm remain serious and potentially life-threatening events. This complication, often associated with extensive transmural infarctions, requires thorough imaging evaluation and an appropriate therapeutic approach. Management can be either medical or surgical, depending on the aneurysm's size, associated symptoms, and overall risk. The case presented underscores the importance of close post-infarction monitoring and a multidisciplinary approach to optimize survival and improve patients' quality of life. A deeper understanding of diagnostic and therapeutic strategies is therefore essential to improving the prognosis of this condition.

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