

Giant Left Ventricular Aneurysm as a Complication in a Patient with Acute Myocardial Infarction: Case Report

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ABSTRACT

INTRODUCTION: Giant ventricular aneurysm is a rare but highly relevant clinical entity due to its impact on cardiac function and its high morbidity and mortality. A giant ventricular aneurysm is considered when the diameter of the aneurysmal sac exceeds 5 cm. Left ventricular (LV) aneurysms are generally a consequence of mechanical complications caused by severe ischemic injury of the myocardium secondary to myocardial infarction (MI) and are associated with heart failure, arrhythmias and rupture. They are generally located apically and anteriorly, with significant mortality.

The incidence is low, and the diagnosis is usually made late through suggestive findings on chest x-ray or transthoracic echocardiogram .

CASE PRESENTATION: We present the case of an 85-year-old male who was admitted to the emergency room with severe chest pain 48 hours prior to arrival. An electrocardiogram diagnosed STEMI, with a finding of subendocardial lesion and inferior wall necrosis, angina intensity 8/10. Since there was no catheterization laboratory available, he was thrombolized with alteplase and presented criteria for reperfusion . A chest x-ray showed dilation in the middle and apical third of the left ventricle. An echocardiogram showed a biplane left ventricular systolic function of 28% with an image suggestive of a left ventricular aneurysm.

A chest CT scan showed a finding compatible with a giant left ventricular aneurysm measuring 65 mm x 85 mm (2.5 x 3.3 in) and the presence of a laminar thrombus.

CONCLUSION: Establishing an early diagnosis is vital to avoid complications. Treatment may be medical in patients with mild symptoms, but in those with severe heart failure or complex arrhythmias, surgery (such as surgical resection or aneurysm repair) may be necessary. A thorough evaluation and a multidisciplinary approach are recommended for the management of these patients, tailoring treatment to the individual characteristics of each case.

KEYWORDS: ventricular aneurysm, giant ventricular aneurysm, laminar thrombus, left ventricular thrombus

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INTRODUCTION: Giant ventricular aneurysms are a rare and complex clinical entity characterized by abnormal, localized dilation of the cardiac ventricle walls. Although ventricular aneurysms are generally well recognized, giant aneurysms, generally defined as those with a diameter greater than 5 cm, present a diagnostic and therapeutic challenge due to their size, potential for serious complications, and the difficulty of effective treatment. These lesions can arise as a sequela of a myocardial infarction, although they can also present in the absence of ischemic history. Management of giant ventricular aneurysms ranges from conservative treatment to complex surgical interventions and is closely related to comorbidities and patient prognosis.

This paper aims to review the clinical features, diagnosis, and therapeutic options, as well as highlight the need for multidisciplinary management for early identification, especially in patients with significant infarcts, and to optimize treatment strategies.

CASE PRESENTATION

An 85-year-old male patient with a history of active smoking for 79 years (smoking index of 39 pack-years) and no chronic degenerative diseases.

He came to the emergency department with a 48-hour history of oppressive chest pain accompanied by adrenergic symptoms. He self-medicated with analgesics without improvement. His symptoms worsened, prompting his admission.

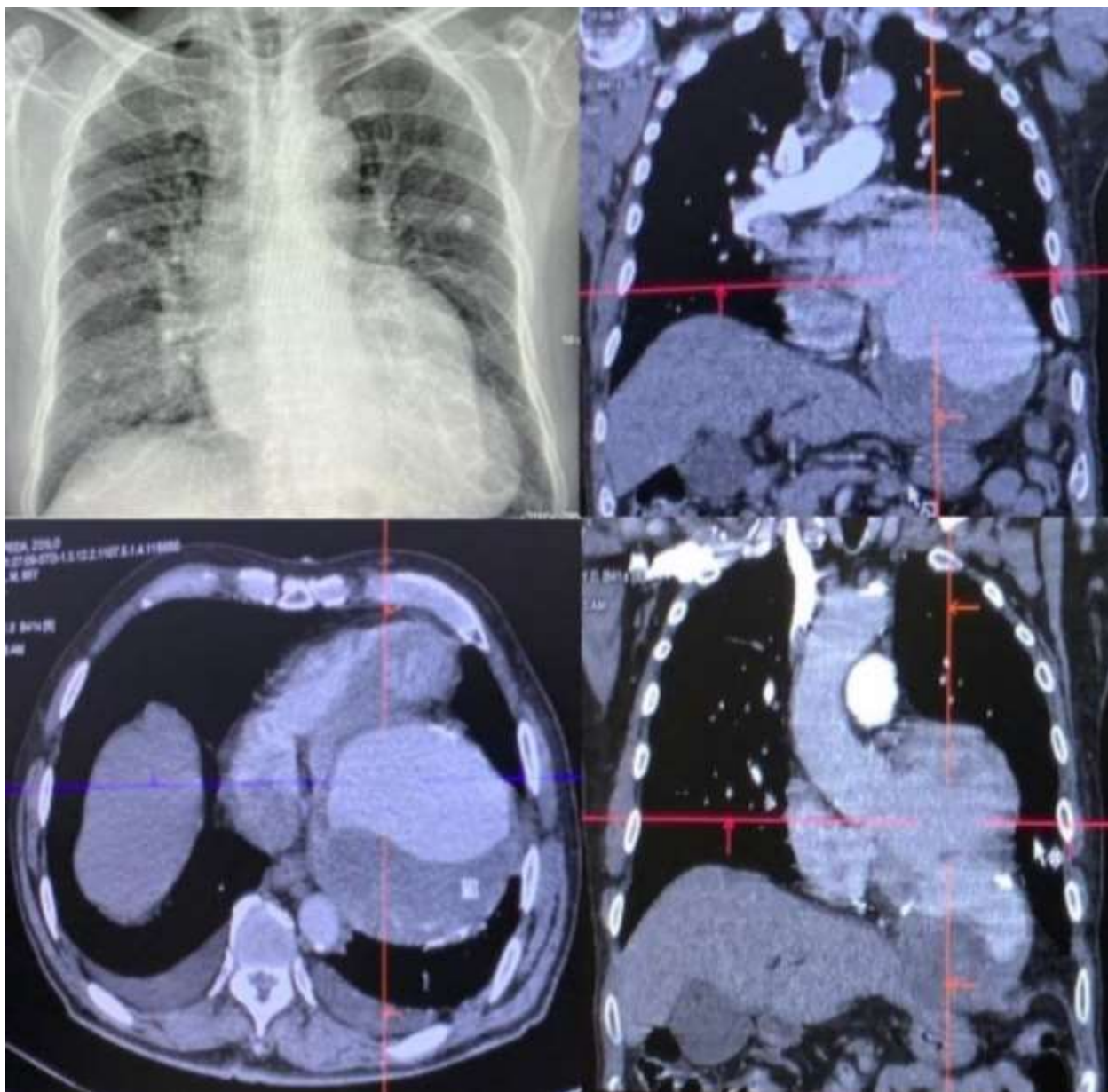
The electrocardiogram showed findings consistent with subendocardial injury and inferior wall necrosis, leading to a diagnosis of ST-segment elevation myocardial infarction (STEMI) inferior wall. Pain intensity was rated 8/10 on the visual analog scale. Due to the lack of immediate access to a catheterization laboratory, thrombolysis with alteplase at a reduced dose was initiated, achieving clinical and electrical criteria for reperfusion (remission of pain and accelerated idioventricular rhythm).

A chest X-ray revealed dilatation in the middle and apical third of the left ventricle. An echocardiogram showed reduced left ventricular systolic function of 28%, with an image suggestive of a left ventricular aneurysm. A computed tomography scan subsequently confirmed the presence of a giant left ventricular aneurysm, measuring 65 x 85 mm (2.5 x 3.3 inches), associated with a laminar thrombus.

Physical examination revealed a visible and palpable, diffuse, and enlarged apical impulse displaced downward and laterally toward the eighth intercostal space in the anterior axillary line. There was a protomesosystolic murmur in the mitral focus radiating toward the posterior axillary line, low-intensity heart sounds, and the presence of a fixed left S4.

biomarker curve showed positive and decreasing values. Acute kidney injury was also documented. During the hospital stay, the patient remained clinically asymptomatic, with no evidence of arrhythmias.





DISCUSSION

Giant ventricular aneurysms are a rare but serious complication of myocardial infarction, characterized by abnormal dilation of the ventricular wall involving a significant portion of the affected ventricle, with a diameter greater than 5 cm. They often develop weeks to months after a transmural myocardial infarction, and their pathogenesis is associated with extensive myocardial injury compromising the integrity of the ventricular wall (Yusuf et al., 2022).⁷

A left ventricular aneurysm is a delimited, fibrotic, and thin scar that causes dilation of the cavity; the wall is thin, whitish, and fibrous with smooth endocardium.

Histologically, initial changes in the ventricular wall include muscle necrosis and inflammatory reaction, replacing the myocardium with scar tissue, and in later stages a thin, white fibrous scar is found.

The aneurysm scar contains hyalinized fibrous tissue, smooth, nontrabeculated endocardium, and contains a thrombus in 50% of cases. Its incidence in thrombolized

patients ranges from 7% to 19% in those with Q-wave infarctions.

Risk factors include anterior wall infarction, delayed or incomplete reperfusion, and absence of extensive collateralization. Less common causes include congenital diseases, nonvascular disorders such as Chagas disease, tuberculosis, trauma, or idiopathic causes. Five-year survival is 12%.

5-30% of extensive transmural myocardial infarctions develop a left ventricular aneurysm, 70-85% are located in the anterior or apical walls and are associated with total occlusion of the left anterior descending coronary artery (LAD) and the absence of collateralization, 10-15% affect the lower basal walls.

88% of aneurysms with dyskinesia originate in anterior infarcts and 12% in the inferior surface.

The mechanism behind giant ventricular aneurysm usually involves fibrosis of the cardiac wall after a myocardial infarction, which compromises the contractile capacity of the

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affected area. Over time, the infarcted area distends, forming a cavity that fills with blood. This increases the end-diastolic volume and decreases the ejection fraction, leading to impaired ventricular function. As the aneurysm grows, it can lead to serious complications, including congestive heart failure, arrhythmias, and in extreme cases, rupture (Salam et al., 2021). Clinically, they present with a diffuse apical impulse displaced to the left of the midclavicular line, with a third or fourth heart sound.

The diagnosis of giant ventricular aneurysm is usually made by identifying the outlined area of thin, scarred myocardium without functional myocardium using imaging techniques, with echocardiography and cardiac magnetic resonance imaging being the methods of choice. Echocardiography allows visualization of ventricular wall deformity and the presence of thrombi in the aneurysm cavity, while magnetic resonance imaging offers a detailed and accurate assessment of aneurysm characteristics, extension, ventricular function, contractility, and viability of hibernating myocardium (Piers et al., 2022).⁴

In more advanced cases, where the aneurysm is associated with secondary mitral or tricuspid regurgitation due to ventricular dilation, cardiac catheterization may be necessary to assess hemodynamic function (Schultz et al., 2023).⁶ The differential diagnosis is made with ventricular pseudoaneurysm, the difference being a rupture of the free wall.

The treatment of giant ventricular aneurysms remains a challenge. Medical management includes control of heart failure, the use of drugs that reduce the burden on the heart, such as angiotensin-converting enzyme inhibitors (ACE inhibitors), beta-blockers, and anticoagulants to prevent thrombus formation within the aneurysm, and management of arrhythmias. However, surgery remains the definitive treatment for aneurysms that significantly compromise ventricular function or are symptomatic. Surgical indications include congestive heart failure, angina, and stroke. pectoris, systemic embolism, malignant ventricular arrhythmias, progressive dilation of the left ventricle with increased mitral regurgitation or decreased contractility. (Gupta et al., 2024).² Currently, surgical resection of giant ventricular aneurysms may include graft placement or the use of a ventricular assist device in cases of advanced heart failure. Surgical repair improves patient survival and quality of life, although mortality rates associated with this intervention remain significant (Liu et al., 2023).

CONCLUSION

This case report highlights the rare but potentially catastrophic complication of a giant left ventricular aneurysm (LVA) developing in the aftermath of an acute myocardial infarction (AMI), underscoring the critical importance of early recognition, comprehensive diagnostic evaluation, and timely multidisciplinary intervention in optimizing patient outcomes. The pathophysiological cascade leading to

aneurysmal formation involves extensive transmural necrosis, progressive ventricular remodeling, and mechanical stress on the infarcted myocardium, ultimately resulting in a thinned, dyskinetic segment that paradoxically expands during systole. The hemodynamic consequences of such a massive structural derangement are profound, including reduced stroke volume, impaired cardiac output, and elevated ventricular wall stress, which predispose to life-threatening complications such as ventricular arrhythmias, systemic thromboembolism, and progressive heart failure.

The diagnostic approach to this condition requires a high index of suspicion, particularly in patients presenting with persistent ST-segment elevation, unexplained heart failure, or recurrent ventricular arrhythmias post-AMI. Advanced imaging modalities, particularly cardiac magnetic resonance imaging (CMR) with late gadolinium enhancement, play an indispensable role in characterizing the extent of myocardial scarring, assessing residual viable myocardium, and evaluating for the presence of mural thrombus. Echocardiography remains the initial diagnostic tool of choice, revealing the aneurysmal dilation, altered ventricular geometry, and potential mechanical complications such as mitral regurgitation secondary to papillary muscle displacement or ventricular dyssynchrony.

Therapeutic decision-making in such cases presents a formidable challenge, requiring careful consideration of the patient's hemodynamic stability, comorbidities, and surgical risk profile. While medical management with guideline-directed heart failure therapies—including beta-blockers, angiotensin-converting enzyme inhibitors, and aldosterone antagonists—forms the cornerstone of treatment to mitigate adverse remodeling, the presence of a giant LVA often necessitates surgical intervention due to the inherent risks of rupture, refractory arrhythmias, or thromboembolic events. Surgical strategies, including linear aneurysmectomy, endoventricular patch repair (Dor procedure), or even ventricular assist device implantation as a bridge to transplantation, must be tailored to the individual patient's anatomical and functional status. The timing of surgical intervention remains controversial, with some advocating for early repair to prevent further ventricular deterioration, while others emphasize the importance of allowing myocardial scar maturation to facilitate secure surgical reconstruction.

This case also illustrates the critical role of long-term surveillance in post-infarction ventricular aneurysm patients, given their predisposition to late complications such as aneurysm expansion, recurrent ventricular tachycardia, or progressive ventricular dysfunction. The integration of advanced heart failure therapies, including consideration for implantable cardioverter-defibrillator (ICD) placement for primary prevention of sudden cardiac death and the potential role of cardiac resynchronization therapy in selected patients with dyssynchrony, remains an essential component of comprehensive post-treatment care.

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Giant left ventricular aneurysm represents a dramatic and clinically significant complication of acute myocardial infarction that demands a multidisciplinary approach encompassing cardiology, cardiac imaging, electrophysiology, and cardiothoracic surgery expertise. This case reinforces the importance of early revascularization in AMI to prevent such catastrophic remodeling and highlights the evolving role of advanced imaging in risk stratification and surgical planning. Future directions in management may include refined techniques for ventricular reconstruction, novel percutaneous approaches to exclude aneurysmal segments, and targeted therapies to modulate post-infarction remodeling processes. Ultimately, the management of this condition epitomizes the intricate balance between medical optimization and timely mechanical intervention in structural heart disease, serving as a compelling reminder of the complex sequelae that can arise from acute coronary syndromes. Continued reporting and analysis of such cases remain vital to advancing our understanding of optimal therapeutic strategies and improving outcomes for patients with this serious complication of ischemic heart disease. (Piers et al., 2022).

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