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"Left Ventricular Aneurysm: A Late and Underrecognized Complication of Acute Myocardial Infarction — A Case Report"

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ABSTRACT

Background:
Left ventricular aneurysm is an uncommon but clinically relevant late complication of acute myocardial infarction in the contemporary reperfusion era. It is associated with adverse ventricular remodeling, systolic dysfunction, functional mitral regurgitation, and increased risk of heart failure, arrhythmias, and thromboembolic events. Early recognition and appropriate risk stratification are essential to guide management and avoid unnecessary invasive procedures.

Case presentation:
We report the case of a 71-year-old man with a history of ST-segment elevation myocardial infarction two years prior, managed conservatively without documented reperfusion. He presented with progressive exertional dyspnea and reduced exercise tolerance. Transthoracic and dobutamine stress echocardiography revealed a true chronic left ventricular aneurysm involving the mid anterolateral, mid inferolateral, and basal inferior segments, associated with reduced left ventricular ejection fraction and moderate functional mitral regurgitation, without evidence of contractile reserve or inducible ischemia. Given the absence of myocardial viability and high procedural risk, a conservative, optimized medical approach was adopted.

Conclusion:
This case highlights left ventricular aneurysm as a late and underrecognized complication of myocardial infarction. Advanced echocardiographic evaluation was crucial in confirming irreversible myocardial injury, guiding individualized management and preventing unnecessary invasive interventions.

Keywords: Left Ventricular Aneurysm, Acute Myocardial Infarction, Ventricular Remodeling, Stress Echocardiography, Functional Mitral Regurgitation

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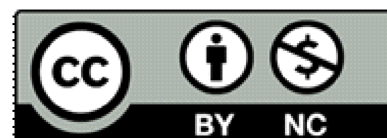

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INTRODUCTION

Acute myocardial infarction (AMI) remains a major cause of morbidity and mortality worldwide despite significant advances in reperfusion strategies and pharmacologic therapy. Although early reperfusion has reduced the incidence of mechanical complications, late structural sequelae continue to occur in a subset of patients. One of the most important of these complications is left ventricular (LV) aneurysm, a localized area of ventricular wall thinning and dyskinesia that typically develops after transmural myocardial infarction. LV aneurysms are most commonly associated with anterior wall infarctions due to occlusion of the left anterior descending artery and are characterized by permanent ventricular deformation resulting from infarcted myocardial tissue replaced by fibrous scar. The reported incidence of LV aneurysm has declined significantly in the modern reperfusion era but remains clinically relevant, particularly in patients in whom appropriate reperfusion therapy was delayed or not given.[1]

The pathophysiology of LV aneurysm formation is closely related to post-infarction ventricular remodeling. Following myocardial ischemia and necrosis, inflammatory as well as reparative mechanisms lead to thinning of the infarcted myocardium which causes expansion of the affected ventricular wall. With time this area loses contractile capacity and becomes akinetic or dyskinetic thereby forming a true aneurysm which is largely composed of scar tissue. This structural alteration in affected ventricular wall increases wall stress in adjacent myocardial segments which contributes to progressive ventricular dilation and systolic dysfunction. The formation of a left ventricular aneurysm in such patients is associated with important hemodynamic disturbances, including a reduction in left ventricular ejection fraction and diminished cardiac output. In addition, the presence of an akinetic segment of the ventricular wall can predispose patients to complications such as functional mitral

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regurgitation, which may occur due to displacement of the papillary muscles and dilation of the mitral annulus. These pathophysiological changes may substantially contribute to the progression of heart failure and increase the likelihood of unfavorable long-term cardiovascular outcomes.[2]

Patients with left ventricular aneurysm may present with a wide range of signs and symptoms. Some patients remain asymptomatic and only have mild ventricular dysfunction. Others may develop overt heart failure, angina, ventricular arrhythmias, or systemic thromboembolism. In many patients LV aneurysms remain undetected for many years and may be discovered incidentally during imaging studies performed for unrelated indications. The presence of a ventricular aneurysm also increases the risk of mural thrombus formation secondary to blood stasis within the dyskinetic ventricular segment. This dyskinetic ventricular portion is known to increase the risk of thromboembolic events as well as ventricular arrhythmias.[3]

Advances in cardiovascular imaging have improved the detection and characterization of post-infarction ventricular aneurysms. Transthoracic echocardiography remains the primary diagnostic modality because of its broad availability, non-invasive nature, and capacity to assess ventricular morphology and systolic function in real time. Typical echocardiographic findings include a thin, dyskinetic ventricular wall demonstrating paradoxical systolic expansion. Additional imaging modalities such as stress echocardiography, cardiac magnetic resonance imaging (MRI) and computed tomography may further provide insights into myocardial viability, scar burden and the presence of intracavitary thrombus. Dobutamine stress echocardiography may assists in analysing contractile reserve and identifying viable myocardium that may benefit from early revascularization.[4]

Despite improvements in diagnostic techniques and therapeutic strategies, left ventricular aneurysms remain an underrecognized late complication of myocardial infarction. This is especially seen in patients who did not receive timely reperfusion therapy or who were managed conservatively during the acute event. Data describing the clinical course and decision-making process in patients presenting years after an untreated or incompletely treated myocardial infarction remain limited.[5] This report describes a patient with a chronic left ventricular aneurysm identified two years after a myocardial infarction. The case focuses on the diagnostic role of stress echocardiography and the therapeutic decision-making process in the absence of demonstrable myocardial viability.

CASE PRESENTATION

A 71-year-old man presented to the outpatient cardiology clinic with progressive exertional dyspnea and declining exercise tolerance over several months. He had a prior history of ST-segment elevation myocardial infarction (STEMI) two years back. The initial myocardial infarction had been managed conservatively and reperfusion therapy was given. The patient had multiple age-related cardiovascular risk factors,. At presentation, the patient reported increasing fatigue and shortness of breath on exertion, corresponding to New York Heart Association (NYHA) functional class II–III symptoms. He denied current chest pain, syncope, palpitations or history of prior thromboembolic events.

On physical examination, the patient was hemodynamically stable. Blood pressure was within therapeutic targets and cardiac rhythm was regular. On auscultation a holosystolic murmur best heard at the cardiac apex with radiation toward the axilla was present. This was suggestive of functional mitral regurgitation. There were no signs of pulmonary congestion, peripheral edema or signs of systemic venous congestion. Given the history of prior myocardial infarction and progressive symptoms transthoracic echocardiography was performed. Baseline echocardiography demonstrated moderate left ventricular dilation with a reduced left ventricular ejection fraction (LVEF) of approximately 35%. Additional findings on echocardiography included extensive regional wall motion abnormalities that were not restricted to a single coronary artery distribution. Moderate functional mitral regurgitation was observed secondary to systolic leaflet restriction associated with adverse ventricular remodeling (Figures 1).

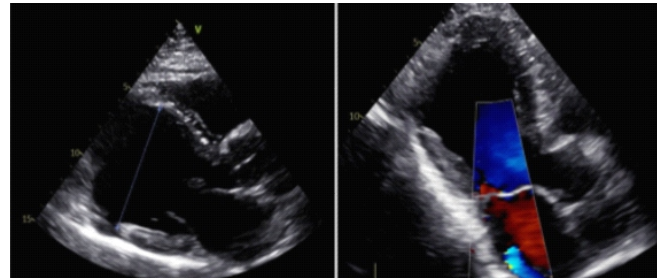

Figure 1 :- Transthoracic echocardiography showing adverse left ventricular remodeling and moderately dilated left ventricle (Left) Color Doppler image demonstrating moderate mitral regurgitation with a systolic regurgitant jet directed into the left atrium. (Right).

To further assess myocardial viability and ischemia dobutamine stress echocardiography was performed. The study demonstrated a true chronic left ventricular aneurysm involving the mid-anterolateral, mid-inferolateral and basal inferior segments. The affected myocardial segments exhibited persistent akinesia without contractile reserve while the remaining viable segments showed compensatory hyperkinesia during pharmacologic stress. Importantly, no inducible ischemia or evidence of myocardial viability was detected. Under stress conditions, there was also worsening of mitral regurgitation severity. This indicated a significant hemodynamic impact of the aneurysmal remodeling (Figures 2).

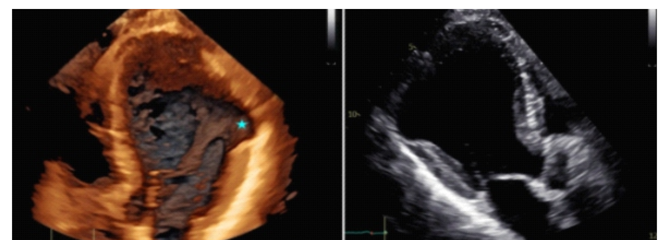

Figure 2:- Dobutamine stress echocardiography showing a chronic left ventricular aneurysm with adverse remodeling. (Left) Rendered echocardiographic view demonstrating outward bulging of the aneurysmal left ventricular segment. (Right) Conventional grayscale echocardiographic view confirming the aneurysmal cavity with persistent akinesia of the involved segments.

These findings were consistent with a left ventricular aneurysm secondary to adverse ventricular remodeling following myocardial infarction which was managed conservatively. The case was subsequently discussed in a multidisciplinary cardiology team meeting. Considering the absence of viable myocardium, the chronic nature of the infarction, and the patient's elevated risk for contrast-induced nephropathy, it was determined that invasive coronary angiography or revascularization would be unlikely to provide clinical benefit.Therefore, the patient was managed with an optimized medical therapy strategy,

including guideline-directed heart failure management and regular clinical follow-up. Surveillance was planned to monitor ventricular function, progression of functional mitral regurgitation and potential complications related to the ventricular aneurysm such as arrhythmias or thromboembolic events.

This case highlights the importance of advanced echocardiographic assessment in identifying irreversible myocardial damage particularly in cases with history of myocardial infarction which were managed conservatively without reperfusion therapy.

DISCUSSION

Left ventricular aneurysm (LVA) is a well-recognized mechanical complication of acute myocardial infarction (AMI), resulting from transmural myocardial necrosis followed by progressive fibrotic remodeling and ventricular wall thinning. Although historically reported in up to 10–35% of patients following transmural infarction, the incidence has markedly declined in the contemporary era due to early reperfusion strategies such as primary percutaneous coronary intervention and thrombolysis. Nevertheless, LVA continues to be encountered in patients who experience delayed diagnosis or managed conservatively after myocardial infarction as illustrated in the present case.[6]

Pathophysiologically, LVA develops when infarcted myocardium undergoes irreversible scar formation thereby leading to thinning as well as outward bulging of the ventricular wall during systole and diastole. This structural abnormality leads to adverse ventricular remodeling and increased wall stress in the remaining viable myocardium. The resulting hemodynamic consequences may include heart failure, ventricular arrhythmias, thromboembolic complications, and functional mitral regurgitation, as seen in this case. The association between post-infarction remodeling and ventricular aneurysm formation was described by Sutton et al who demonstrated that geometric distortion of the left ventricle play a significant role in the subsequent development of chronic ventricular aneurysms.[7]

Clinically, most left ventricular aneurysms develop after large anterior wall myocardial infarctions particularly those involving the left anterior descending coronary artery. However, aneurysms involving other myocardial segments may also occur if there is extensive multivessel ischemia or delayed reperfusion. In the present case, the aneurysm involved the mid anterolateral, mid inferolateral, and basal inferior segments. This reflects extensive post-infarction remodeling rather than a single coronary territory distribution. Such patterns have been described in patients with long-standing ischemic cardiomyopathy and chronic ventricular remodeling.[8]

Echocardiography remains the primary imaging modality for diagnosis of left ventricular aneurysm. Transthoracic echocardiography allows visualization of ventricular wall thinning, dyskinetic motion as well as chamber dilation. It can also reliably evaluate left ventricular function and associated complications such as mitral regurgitation or intracavitary thrombus. As highlighted by Vlodaver et al distinguishing a true ventricular aneurysm from other structural abnormalities such as pseudoaneurysm is of critical importance because the natural history and management strategies differ substantially in both of these cases.[9]

Stress echocardiography in cases of LVA can provide additional prognostic value by assessing myocardial viability and contractile reserve. Allman et al conducted a meta-analysis to evaluate whether myocardial viability testing predicted survival benefit from revascularization in patients with coronary artery disease and left ventricular dysfunction. For this purpose, the authors undertook a study comprising 24 viability studies involving 3,088 patients, using thallium imaging, FDG imaging, or dobutamine echocardiography. The study found that, in patients with viability, revascularization reduced annual mortality from 16% to 3.2%, whereas in those without viability outcomes were similar with revascularization or medical therapy. On the basis of these findings, the authors concluded that viability testing identified patients most likely to benefit from revascularization therapy.[10]

In the present case dobutamine stress echocardiography showed no contractile reserve or inducible ischemia, indicating that the affected myocardium was chronic irreversible scar tissue. This finding helped avoid unnecessary coronary angiography and revascularization attempts.

The case also demonstrated moderate functional mitral regurgitation caused by adverse ventricular remodeling and papillary muscle displacement which can worsen heart failure and prognosis. Overall, the case underlines that left ventricular aneurysm remains an important late complication of myocardial infarction and stress echocardiography in these cases remain essential for guiding individualized management decisions.

CONCLUSION

Left ventricular aneurysm remains an important late complication of acute myocardial infarction particularly in patients who did not receive timely reperfusion therapy. It also demonstrates the value of advanced echocardiographic assessment, especially dobutamine stress echocardiography, in defining aneurysm morphology, assessing contractile reserve, and identifying absence of myocardial viability. In the present patient, these findings supported a non-invasive management strategy and avoided unnecessary coronary angiography or revascularization.

Conflict Of Interest : None Declared.

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Author Contribution :MR and **DG** - contributed to patient management, data collection, and manuscript drafting; **JF** and **LV** - participated in data compilation, interpretation, and literature review; **JO** and **SF** - assisted with manuscript revision and data validation; **EA** - supervised the study and approved the final manuscript.

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