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Improvement of Severe Ischemic Mitral Regurgitation after Surgical Reconstruction of a Post-Infarction Left Ventricul Araneurysm without Mitral Valve Intervention: A Case Report

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Abstract **Case Report**

Left ventricular aneurysm (LVA) is a serious late mechanical complication of myocardial infarction and may be associated with ischemic mitral regurgitation (IMR), leading to or worsening severe left ventricular systolic dysfunction. We report the case of a 64-year-old man with diabetes and active smoking who was admitted with New York Heart Association (NYHA) class IV dyspnea. Transthoracic echocardiography and cardiac magnetic resonance imaging revealed a post-myocardial infarction left ventricular aneurysm, severe systolic dysfunction with a left ventricular ejection fraction of 30%, and severe ischemic mitral regurgitation. Coronary angiography demonstrated three-vessel coronary artery disease with chronic total occlusion of the right coronary artery. The patient underwent complete myocardial revascularization combined with surgical left ventricular reconstruction without mitral valve intervention. The postoperative course was uneventful, with clinical improvement and regression of mitral regurgitation to grade I. This case highlights the potential role of surgical ventricular reconstruction combined with coronary artery bypass grafting in restoring left ventricular geometry and hemodynamics, with secondary improvement of functional ischemic mitral regurgitation.

Keywords: left ventricular aneurysm; ischemic mitral regurgitation; myocardial infarction; surgical ventricular reconstruction; coronary artery bypass grafting.

INTRODUCTION

Cardiovascular diseases account for approximately one-third of deaths worldwide and remain a major cause of morbidity and mortality [1]. In 2020, an estimated 244 million people were living with ischemic heart disease, which was responsible for approximately nine million deaths worldwide [2]. Mechanical complications following myocardial infarction are relatively uncommon but can be associated with substantial morbidity and mortality. Among these, left ventricular aneurysm (LVA) may coexist with ischemic mitral regurgitation (IMR), further contributing to left ventricular systolic dysfunction.

Left ventricular aneurysm is a serious late complication of myocardial infarction, with a reported incidence ranging from 3% to 38%. It results from adverse healing and remodeling of the infarcted myocardium, leading to wall thinning and the formation of a dyskinetic ventricular segment that exhibits paradoxical systolic expansion. Its clinical significance is related particularly to complications such as ventricular arrhythmias and systemic thromboembolism [3,4].

Secondary mitral regurgitation may develop as a consequence of ischemic myocardial injury [5], and significant mitral regurgitation has been reported in up to 30% of patients following myocardial infarction [6]. Left ventricular remodeling alters ventricular and mitral annular geometry, displaces the papillary muscles, and increases leaflet tethering, resulting in impaired leaflet coaptation despite the absence of primary structural mitral valve disease [5].

Surgical management remains an important therapeutic option in selected patients with postinfarction left ventricular aneurysm and significant ischemic mitral regurgitation. We report the case of a patient with a post-infarction left ventricular aneurysm associated with severe ischemic mitral regurgitation and severe systolic dysfunction who underwent surgical ventricular reconstruction and coronary revascularization without mitral valve intervention.

CASE PRESENTATION

1. Patient Information

A 64-year-old man, an active smoker with a 40-pack-year smoking history and a 10-year history of type 2 diabetes mellitus complicated by diabetic nephropathy, was admitted with New York Heart Association (NYHA) class IV dyspnea associated with orthopnea.

His symptoms had begun three months before admission with exertional angina, followed by progressively worsening dyspnea, initially NYHA class II and subsequently progressing to class IV, with the development of orthopnea. He was initially hospitalized in the cardiology department and was subsequently referred to our cardiovascular surgery department for further management. He reported no history of syncope or sensorimotor neurological deficits.

2. Clinical Findings

On admission, the patient was hemodynamically stable, with a blood pressure of 112/64 mmHg and a heart rate of 80 beats/min. Cardiac auscultation revealed a holosystolic murmur at the mitral area without radiation. The remainder of the physical examination was unremarkable.

3. Diagnostic Assessment

Electrocardiography showed sinus rhythm, T-wave inversion in the inferior leads, and right bundle branch block. Figure 1

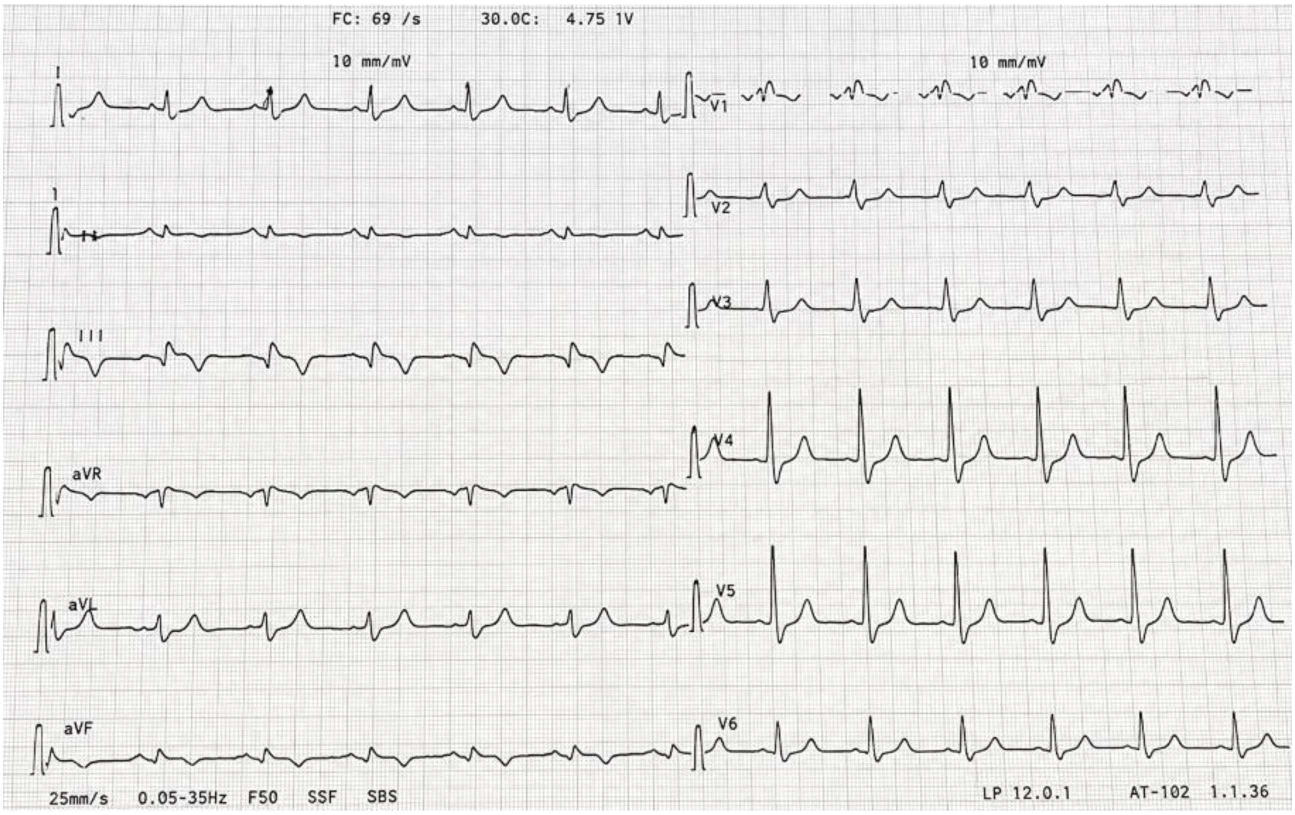

Figure 1: Twelve-lead electrocardiogram showing sinus rhythm, T-wave inversion in the inferior leads, and right bundle branch block

Chest radiography showed no cardiomegaly, with hilar vascular prominence and no focal pulmonary parenchymal abnormalities.

Laboratory investigations were otherwise unremarkable, except for a mild elevation in cardiac troponin levels to 1.3 times the upper limit of normal.

Transthoracic echocardiography revealed a mildly thickened mitral valve with preserved leaflet mobility and significant leaflet tethering, resulting in grade III ischemic mitral regurgitation. The mitral annular diameter was 37 mm, with a tenting height of 18 mm and a tenting area of 2.8 cm². The effective regurgitant orifice area was 30 mm², with a regurgitant volume of 45 mL. The left ventricle was dilated and non-hypertrophied, with global hypokinesia and akinesia of the basal and mid-lateral segments and the basal inferoseptal segment. An aneurysmal formation of the inferior wall with a suspected associated thrombus was also identified. Left ventricular systolic function was severely impaired, with a left ventricular ejection fraction (LVEF) of 34%. The right ventricle was normal in size with preserved systolic function. Figure 2

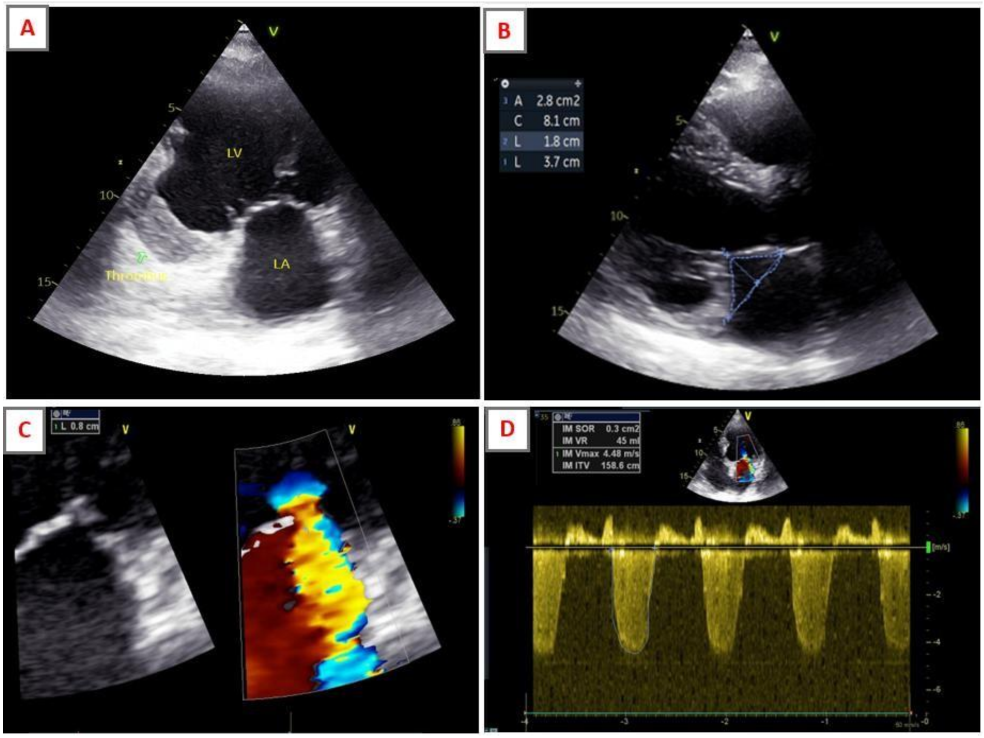

Figure 2: Transthoracic echocardiographic findings

(A) Apical two-chamber view showing a post-infarction mural thrombus attached to the aneurysmal inferior wall of the left ventricle. **(B)** Parasternal long-axis view demonstrating significant mitral leaflet tethering, with a tenting area of 2.8 cm². **(C, D)** Color Doppler and continuous-wave Doppler imaging demonstrating a secondary mitral regurgitation jet.

Coronary angiography demonstrated severe multivessel coronary artery disease, including significant distal left main and left anterior descending artery disease and severe left circumflex artery stenosis. Most notably, the dominant right coronary artery showed a chronic total occlusion of its proximal segment, with collateral filling from the contralateral coronary circulation.

Cardiac magnetic resonance imaging confirmed the echocardiographic findings, demonstrating an inferior left ventricular aneurysm measuring 53 × 42 mm and containing a mural thrombus measuring 42 × 15 mm. The LVEF was 30%. Figure 3

Figure 3: Cardiac magnetic resonance imaging findings.

Late gadolinium enhancement (LGE) sequence demonstrating a 53 × 42 mm inferior wall left ventricular aneurysm with a broad neck, containing a 42 × 15 mm mural thrombus

4. Diagnosis:

Post-myocardial infarction posterior left ventricular aneurysm with mural thrombus, severe ischemic mitral regurgitation, and left ventricular systolic dysfunction.

5.. Therapeutic Intervention

Surgical exploration confirmed an aneurysmal lesion of the posterior left ventricular wall. A longitudinal ventriculotomy was performed, followed by complete removal of the left ventricular thrombus and scar tissue. Inspection of the left ventricular cavity revealed preserved mitral papillary muscles without structural abnormalities. Left ventricular reconstruction was then performed using a biological patch, followed by closure of the ventriculotomy. Figure 4

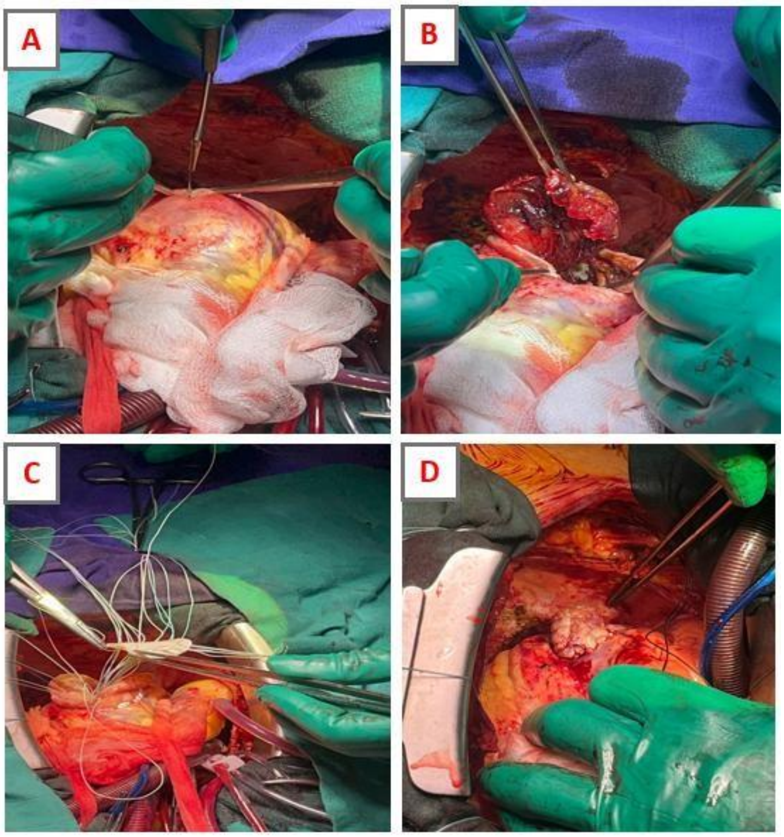

Figure 4: Surgical steps of left ventricular aneurysm repair

(A) Surgical exposure and incision of the left ventricular aneurysm. **(B)** Excision of the intraventricular thrombus. **(C)** Left ventricular reconstruction using a biological patch. **(D)** Final appearance of the reconstructed left ventricle.

Complete myocardial revascularization was subsequently performed. No mitral valve intervention was undertaken because intraoperative assessment revealed no structural abnormality of the mitral apparatus. The aortic cross-clamp time was 126 minutes, and the total cardiopulmonary bypass time was 159 minutes. The patient was successfully weaned from cardiopulmonary bypass with dobutamine support at 5 μg/kg/min.

5. Follow-up and Outcomes

The immediate postoperative course was uneventful. The patient remained hemodynamically stable and was comfortable at rest, with a heart rate of 87 beats/min and a blood pressure of 100/62 mmHg. The surgical wound was clean, and the sternum was stable.

Follow-up transthoracic echocardiography demonstrated marked improvement in ischemic mitral regurgitation from grade III to grade I, with a proximal isovelocity surface area radius of 4 mm, an effective regurgitant orifice area of 10 mm², and a regurgitant volume of 10 mL. The left ventricle remained dilated, with global hypokinesia and persistent severe systolic dysfunction (LVEF, 30%). The patient was discharged home in stable clinical condition on postoperative day 11.

DISCUSSION

Left ventricular aneurysm and ischemic mitral regurgitation are both consequences of adverse left ventricular remodeling following myocardial infarction. This remodeling involves complex mechanical and neurohormonal mechanisms. Increased systolic wall stress promotes stretching and expansion of the infarcted myocardium, while hemodynamic impairment activates neurohormonal pathways, particularly the sympathetic nervous system and the reninangiotensin-aldosterone system. Together, these mechanisms contribute to progressive remodeling of both infarcted and non-infarcted myocardium.

Ischemic mitral regurgitation is primarily a ventricular rather than a valvular disease. Postinfarction left ventricular remodeling alters ventricular geometry, resulting in papillary muscle displacement, increased leaflet tethering, annular dilatation, and impaired leaflet coaptation despite structurally preserved mitral leaflets. This mechanism was particularly relevant in our patient, who had significant mitral leaflet tethering, with a tenting height of 18 mm and a tenting area of 2.8 cm², supporting the functional mechanism of the severe mitral regurgitation.

Transthoracic echocardiography remains a key imaging modality for the diagnosis and assessment of left ventricular aneurysms. A true ventricular aneurysm typically appears as a thin-walled, broad-necked outpouching involving the infarcted territory and exhibiting dyskinetic or paradoxical systolic expansion. These characteristics are important for differentiating a true left ventricular aneurysm from a pseudoaneurysm [4,7].

Echocardiography also provides essential information regarding left ventricular function, associated mitral regurgitation, and intracavitary thrombus.

Cardiac magnetic resonance imaging provides complementary information by allowing accurate assessment of ventricular volumes, regional wall motion, myocardial tissue characteristics, and intracavitary thrombus [8]. In our patient, cardiac magnetic resonance imaging confirmed the inferior left ventricular aneurysm and associated mural thrombus and demonstrated severe left ventricular systolic dysfunction.

The therapeutic strategy in our patient consisted of surgical left ventricular reconstruction combined with complete coronary revascularization, without mitral valve intervention. Surgical ventricular reconstruction aims to exclude or resect scarred and dyskinetic myocardium, reduce left ventricular volume, and restore a more physiological ventricular geometry [9]. Surgical techniques have progressively evolved from simple linear aneurysmectomy toward ventricular reconstruction techniques using patch exclusion to restore ventricular shape and function [10].

An important feature of our case was the decision not to perform concomitant mitral valve surgery. Intraoperative examination showed no structural abnormality of the mitral apparatus, supporting the functional nature of the regurgitation. Restoration of left ventricular geometry through surgical ventricular reconstruction, together with myocardial revascularization, may reduce papillary muscle displacement and leaflet tethering, thereby improving mitral valve coaptation. This mechanism may explain the marked postoperative reduction in mitral regurgitation observed in our patient, from grade III to grade I, despite the absence of direct mitral valve intervention.

The potential benefit of surgical ventricular reconstruction in appropriately selected patients has been evaluated in previous studies. Yang *et al.,* evaluated 140 patients with chronic myocardial infarction and severe left ventricular dysfunction, including 70 patients who underwent coronary artery bypass grafting (CABG) combined with surgical ventricular reconstruction (SVR) and 70 who underwent isolated CABG. During a mean follow-up of approximately 10 years, the CABG plus SVR group had a significantly lower rate of rehospitalization for heart failure than the isolated CABG group (4.3% vs. 19.1%, $p=0.007$), although no significant difference in mortality was observed [11]. Furthermore, previous studies comparing coronary revascularization alone with revascularization combined with mitral valve surgery in patients with ischemic mitral regurgitation have not consistently demonstrated a survival benefit associated with concomitant mitral valve intervention [12-14]. These findings, together with the postoperative improvement observed in our patient, highlight the importance of assessing the mechanism of ischemic mitral regurgitation and left ventricular geometry when determining whether direct mitral valve intervention is necessary.

Conclusions

Post-myocardial infarction left ventricular aneurysm associated with severe ischemic mitral regurgitation reflects advanced adverse ventricular remodeling and may result in severe left ventricular systolic dysfunction. In appropriately selected patients, surgical ventricular reconstruction combined with complete myocardial revascularization may restore left ventricular geometry and lead to substantial improvement in functional ischemic mitral regurgitation, even without direct mitral valve intervention.

Careful multimodality imaging, particularly echocardiography and cardiac magnetic resonance imaging, is essential for defining ventricular anatomy, assessing the mechanism of mitral regurgitation, and guiding the surgical strategy. Appropriate patient selection remains critical to achieving favorable clinical outcomes.

Competing Interests

The authors declare no competing interests.

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