Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Cardiology

Ventricular Septal Rupture and Ventricular Aneurysm Complicating Myocardial Infarction: A Case Report

Sana Nehame, MD[1]*, Salma Nafidi, MD[1], Joumana El Masrioui, MD[1], Saloua El Karimi, MD, PhD[1], Mustapha El Hattaoui, MD, PhD[1]

[1]Department of Cardiology, Arrazi Hospital, Mohammed VI University Hospital Center (CHU), Faculty of Medicine and Pharmacy, Cadi Ayyad University, Marrakech, Morocco

DOI: https://doi.org/10.36347/sjmcr.2026.v14i05.011 | **Received:** 21.02.2026 | **Accepted:** 17.04.2026 | **Published:** 05.05.2026

***Corresponding author:** Sana Nehame, MD
Department of Cardiology, Arrazi Hospital, Mohammed VI University Hospital Center (CHU), Faculty of Medicine and Pharmacy, Cadi Ayyad University, Marrakech, Morocco

Abstract | **Case Report**

Aims: The objective is to report a rare case of a double post-infarction mechanical complication (ventricular septal rupture [VSR] and ventricular aneurysm) and to discuss the impact of the restrictive nature of the shunt as well as the surgical "timing paradox" on the patient's survival. ***Case presentation :*** A 70-year-old diabetic female patient was admitted on the 8th day of a neglected inferior myocardial infarction (MI), which presented as global heart failure. Transthoracic echocardiography revealed a 7 mm basal inferoseptal VSR, with a restrictive left-to-right shunt (gradient at 4.69 m/s), associated with an inferoseptal ventricular aneurysm. Coronary angiography confirmed the proximal occlusion of the right coronary artery. Following medical stabilization, the patient underwent a delayed surgical strategy including an aneurysmectomy, septal closure using an exclusion technique, and coronary artery bypass grafting (CABG). ***Discussion:*** The patient's paradoxical survival up to the 8th day is explained by the anatomical restriction of the shunt and the preservation of the left ventricular ejection fraction. This delayed presentation favored a fibrous consolidation of the rupture margins, facilitating surgical repair and optimizing the postoperative prognosis. ***Conclusion:*** Therapeutic success relies on precise echocardiographic diagnosis and a multidisciplinary approach to define the ideal surgical timing to transform the prognosis of these extremely severe complications.

Keywords: Myocardial infarction; Ventricular septal rupture; Ventricular aneurysm; Mechanical complications.

1. INTRODUCTION

Myocardial infarction (MI) remains a major cause of global morbidity and mortality. Although the advent of primary angioplasty has reduced the incidence of mechanical complications to less than 1% [1,2], their occurrence remains a formidable prognostic turning point. Among these, post-infarction ventricular septal defect (VSD) is rare (0.2 to 0.3%) but extremely severe, with in-hospital mortality often exceeding 50% [3].

Concurrently, ventricular aneurysm generally develops away from the acute phase in extensive infarctions. The synchronous association of a VSD and a ventricular aneurysm is exceptional; it often characterizes delayed management, which promotes early cavitary remodeling and major tissue weakening [4].

2. CASE PRESENTATION

2.1. Medical History

The 70-year-old female patient presented with several cardiovascular risk factors: poorly controlled type 2 diabetes of 15 years' duration on oral antidiabetics, dyslipidemia on statins, and a history of ischemic stroke 15 years ago complicated by residual right hemiplegia.

2.2. History of Present Illness

The patient was admitted to the emergency department for the management of a post-inferior myocardial infarction complicated by global heart failure. The interview revealed the onset, eight days prior to admission, of atypical chest pain associated with intense and continuous epigastralgia, neglected by the patient and relieved by gastric antacids and analgesics. The subsequent clinical course was marked by the progressive worsening of exertional dyspnea, initially NYHA class II, progressing to class IV with orthopnea, associated with bilateral lower extremity edema.

2.3. Clinical Examination

Upon admission, the patient was conscious and hemodynamically stable. Cardiac auscultation revealed a pan-systolic murmur radiating like the spokes of a wheel (*en rayons de roue*). Bilateral basilar crackles were noted, indicating pulmonary congestion, associated with signs of right ventricular failure: jugular venous distension and bilateral edema extending up to the mid-calf. No peripheral signs of shock were present.

2.4. Paraclinical Investigations

Electrocardiogram (Figure 1): Sinus rhythm, normal axis, narrow QRS complexes. Mild ST-segment elevation in the inferior leads with necrotic Q waves confirming the inferior territory. Peripheral microvoltage.

Laboratory findings: Hepatic cytolysis, spontaneous prothrombin time at 60%, and high-sensitivity troponin elevation to more than 1,000 times the normal limit.

Transthoracic echocardiography (TTE) (Figures 2 and 3): Left ventricular ejection fraction (LVEF) preserved at 54%; akinesia of the basal and mid inferior wall; thinned and aneurysmal inferoseptal wall extending from the basal to the mid segments, with a 16 mm neck and 16 mm excursion; 7 mm basal inferoseptal muscular VSD with a restrictive left-to-right shunt (maximum continuous-wave Doppler gradient: 4.69 m/s; estimated systolic pulmonary artery pressure at 84 mmHg); moderate right ventricular systolic dysfunction; mild circumferential pericardial effusion.

Coronary angiography (Figure 4): Total thrombotic occlusion of the proximal segment of the right coronary artery (RCA), associated with intermediate lesions of the proximal left anterior descending (LAD) artery.

2.5. Management and Outcome

The patient was referred to cardiovascular surgery after medical stabilization. The intervention combined a coronary artery bypass graft (CABG) of the RCA, a ventricular aneurysmectomy with resection of the pathological inferoseptal wall, and a septal closure using an exclusion technique.

The postoperative course was uneventful. A follow-up TTE on the 7th postoperative day confirmed the absence of a residual aneurysm, the absence of a residual VSD, and a mild pericardial effusion. The patient was extubated early.

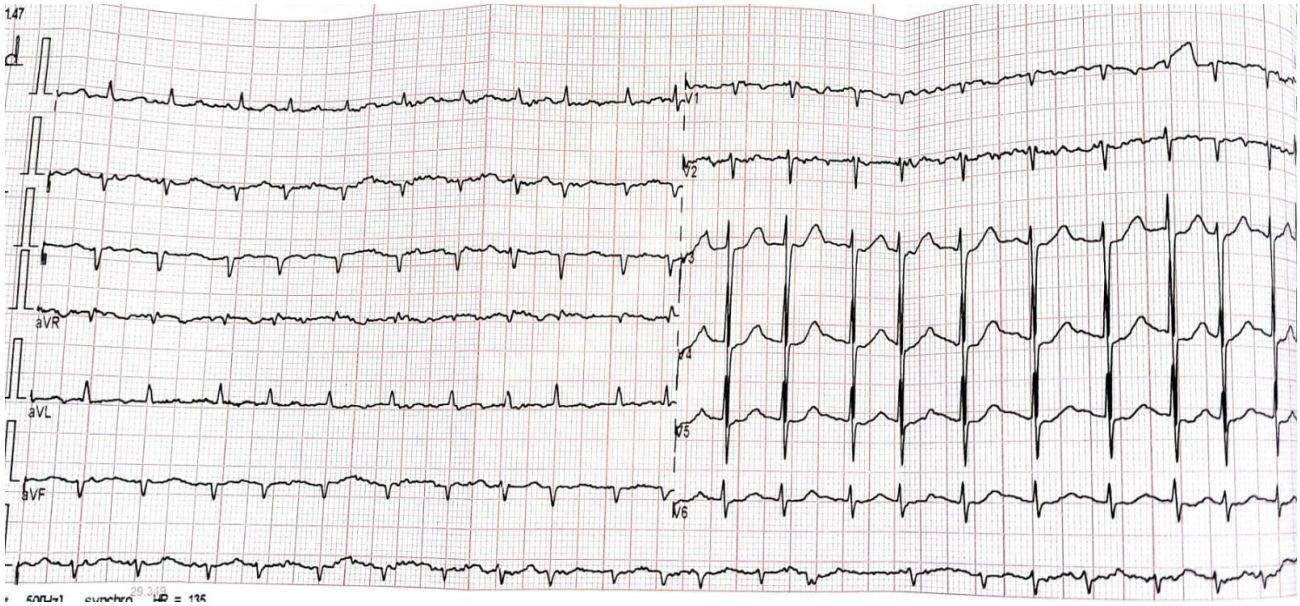
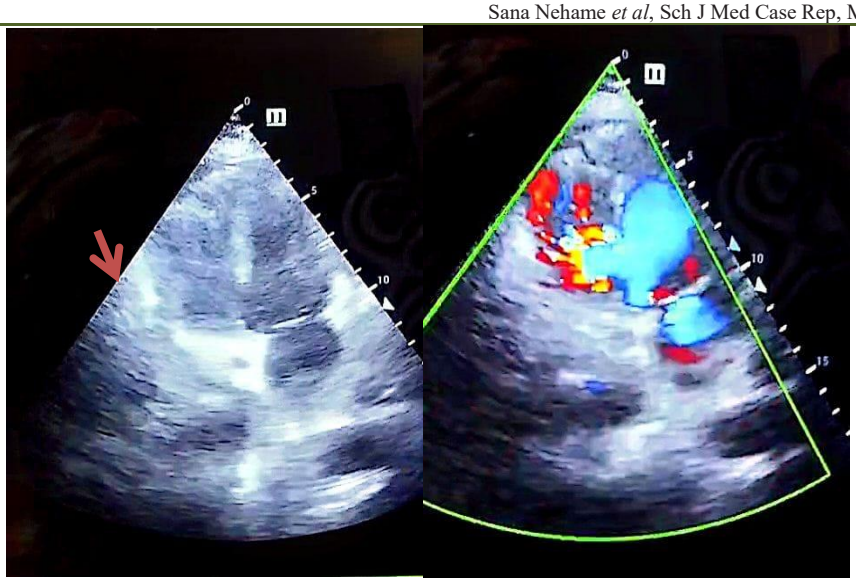


Figure 1: Electrocardiographic tracing demonstrating subtle ST-segment elevation and necrotic Q waves in the inferior territory, associated with peripheral microvoltage



Figures 2 and 3: Transthoracic echocardiography demonstrating an inferoseptal wall aneurysm complicated by a muscular ventricular septal defect (VSD), with Doppler evaluation of the left-to-right shunt

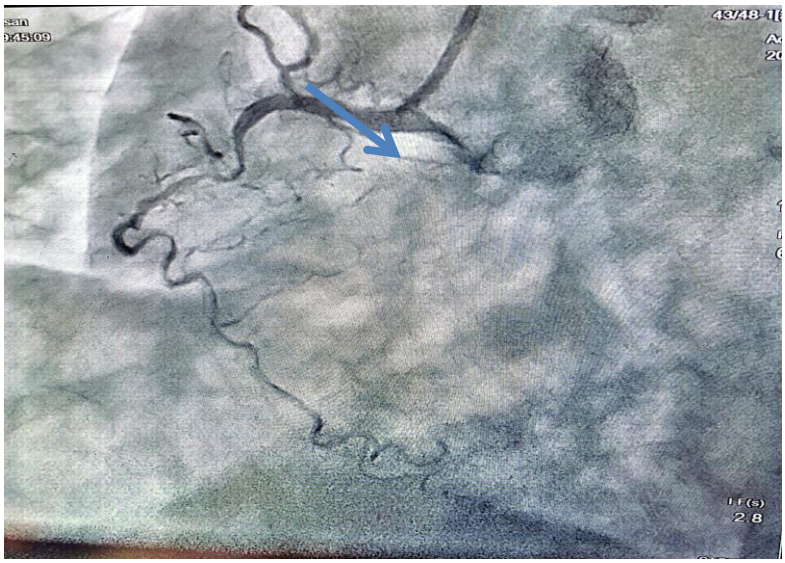


Figure 4: Coronary angiography revealing a thrombotic occlusion of the proximal segment of the right coronary artery

3. DISCUSSION

3.1. Epidemiology and Risk Factors

The incidence of VSR has dropped from 1–2% to less than 0.3% [1] due to early reperfusion, but its mortality remains critical (40–80%). Delayed revascularization remains the primary risk factor, as persistent occlusion inevitably leads to transmural necrosis [2].

The common denominator in the majority of contemporary mechanical complications is the delayed revascularization of the culprit artery [3]. Persistent total coronary occlusion leads inevitably to extensive transmural necrosis, predisposing the patient to parietal rupture [5, 6].

The clinical history of our patient illustrates this paradigm: atypical symptoms (dominant epigastralgia) of an inferior MI ignored for eight days. A presentation without classic chest pain is frequently associated with diabetes, in which autonomic neuropathy alters pain perception and promotes diagnostic delay [7]. Furthermore, the clinical profile—advanced age, female sex, long-standing diabetes, and lack of ischemic preconditioning—represents exactly the population at very high risk for septal rupture. Diabetes plays a fundamentally deleterious role by inducing endothelial dysfunction, aggravating the extent of necrosis, limiting collateral development, and qualitatively hindering scar fibrosis [4].

3.2. Pathophysiology: Mechanisms of Paradoxical Survival

Septal dehiscence typically occurs between the 3rd and 5th day post-infarction, during a phase of matrix metalloproteinase (MMP) release that prematurely

degrades interstitial collagen, rendering the myocardium friable before reparative fibrosis begins [7]. Inferior MIs generate basal inferoseptal damage, which is anatomically more complex due to its proximity to the mitral subvalvular apparatus [8].

The uniqueness of our observation lies in the association of a septal rupture with concomitant aneurysm formation. Two mechanisms explain the patient's paradoxical survival. On one hand, the systolic expansion of the necrotic wall, governed by Laplace's law, acted as a compliance chamber, cushioning the kinetic force of the flow and moderating the impact of cavitary pressures on the septal breach. On the other hand, the anatomical restriction of the septal defect drastically limited the trans-septal flow, causing a progressive volume overload rather than sudden biventricular collapse, thereby allowing the pulmonary circulation time to develop adaptive post-capillary pulmonary hypertension [9].

3.3. Role of Imaging

Transthoracic echocardiography (TTE) confirms its status as the gold-standard examination [10]. It allows for an exhaustive morphological and hemodynamic mapping: visualization of the septal breach, continuous-wave Doppler flow analysis characterizing the restrictive shunt, evaluation of right ventricular dysfunction, and quantification of pulmonary pressures. Preoperative coronary angiography completed this diagnostic workup by confirming the ischemic territory and allowing for the planning of revascularization [3].

3.4. Management: The Challenge of Surgical Timing

While guidelines suggest early surgery to prevent multiorgan failure, data from the Society of Thoracic Surgeons (STS) registry show that mortality drops from 50% to 20% if the intervention is delayed in a stabilized patient [11, 12].

During the acute phase, the fragility of necrotic tissue makes suturing perilous. In our patient's case, the delay in management allowed for cicatricial fibrosis, providing a solid anchor for prosthetic sutures [13].

The surgical procedure combined resection of the aneurysmal tissue with tension-free septal repair. The concomitant performance of CABG proved crucial: the restoration of adequate arterial flow to adjacent viable territories conditioned right ventricular recovery, enabling cardiorespiratory weaning and the regression of pulmonary pressures [14]. The favorable outcome confirms the validity of a cautious temporization strategy when hemodynamics allows, and highlights the central role of collaborative decision-making within the Heart Team.

4. CONCLUSION

This observation highlights the life-saving "buffering" effect of the inferoseptal aneurysm, where the restriction of the shunt allowed for survival despite a delayed diagnosis. Echocardiography remains the key examination here for hemodynamic assessment.

This case primarily demonstrates the paradox of surgical timing: initial stability allows the procedure to be deferred to benefit from cicatricial fibrosis, ensuring the strength of the sutures. A collaborative decision within the Heart Team is therefore essential to optimize this timing and secure the combined repair.

Patient Consent: Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Conflict of Interest: The authors declare that they have no competing interests.

Funding: This research received no external funding.

Authors' Contributions: All authors contributed equally to the manuscript and approved the final submitted version.

REFERENCES

1. Elbadawi A, Elgendy IY, Mahmoud K, *et al.,* Temporal Trends and Outcomes of Mechanical Complications in Patients with Acute Myocardial Infarction. JACC Cardiovasc Interv. 2019;12(18):1825-1836.
2. Jones BM, Kapadia SR, Smedira NG, *et al.,* Ventricular septal rupture complicating acute myocardial infarction: a contemporary review. Eur Heart J. 2014;35(31):2060-2068.
3. Byrne RA, Rossello X, Coughlan JJ, *et al.,*2023 ESC Guidelines for the management of acute coronary syndromes. Eur Heart J. 2023;44(38):3720-3826.
4. [4] Moreno PR, Fuster V, Valentin V. New aspects in the pathogenesis of diabetic atherothrombosis. J Am Coll Cardiol. 2004;44(12):2293-2300.
5. BS, Granger CB, Birnbaum Y, *et al.,* Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. GUSTO-I Trial Investigators. Circulation. 2000;101(1):27-32.
6. Hochman JS, Buller CE, Sleeper LA, *et al.,* Cardiogenic shock complicating acute myocardial infarction — etiologies, management and outcome: a report from the SHOCK Trial Registry. J Am Coll Cardiol. 2000;36(3 Suppl A):1063-1070.
7. Canto JG, Shlipak MG, Rogers WJ, *et al.,* Prevalence, clinical characteristics, and mortality among patients with myocardial infarction presenting without chest pain. JAMA. 2000;283(24):3223-3229.

8. Spinale FG. Myocardial matrix remodeling and the matrix metalloproteinases: influence on cardiac form and function. Physiol Rev. 2007;87(4):1285-1342.
9. Hutchins GM. Rupture of the interventricular septum complicating myocardial infarction: pathological analysis of 65 patients. Am Heart J. 1979;97(2):165-173.
10. Zamorano JL, Badano LP, Bruce C, *et al.,* EAE/ASE recommendations for the use of echocardiography in new valvular heart disease. Eur J Echocardiogr. 2011;12(4):257-277.
11. Arnaoutakis GJ, Zhao Y, George TJ, *et al.,* Surgical repair of ventricular septal defect after myocardial infarction: outcomes from the Society of Thoracic Surgeons National Database. Ann Thorac Surg. 2012;94(2):436-444.
12. Ronco D, Matteucci M, Kowalewski M, *et al.,* Surgical Treatment of Postinfarction Ventricular Septal Rupture. JAMA Netw Open. 2021;4(10): e2128309.
13. David TE, Armstrong S. Surgical repair of postinfarction ventricular septal defect by infarct exclusion. Semin Thorac Cardiovasc Surg. 1998;10(2):105-110.
14. Paparella D, Guida P, Caparrotti S, *et al.,* Ventricular septal rupture complicating acute myocardial infarction: is concomitant coronary artery bypass grafting beneficial? Interact Cardiovasc Thorac Surg. 2020;31(2):189-196.