Cureus
Part of SPRINGER NATURE

Open Access Case Report

A New Murmur After Successful Reperfusion: Post-infarction Ventricular Septal Rupture Diagnosed by Bedside Examination

Review began 09/15/2026
Review ended 11/01/2026
Published 11/09/2026

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DOI: 10.7759/cureus.119377

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Abstract

Post-myocardial infarction ventricular septal defect (PIVSD) is an uncommon but devastating mechanical complication of acute myocardial infarction. We describe the case of a 63-year-old man with cardiovascular risk factors and prior coronary artery disease (CAD) who presented 7 hours after symptom onset with an anterior ST-segment elevation myocardial infarction (STEMI) and underwent percutaneous coronary intervention (PCI). Despite a stable clinical course, a new systolic murmur appeared 72 hours later, prompting repeat transthoracic echocardiography, which demonstrated a ventricular septal defect with associated left ventricular aneurysmal change. Following multidisciplinary Heart Team discussion, the timing of surgical repair was individualized: given the patient's sustained hemodynamic stability and the defect's anatomical characteristics, a deferred approach was considered acceptable in this specific setting, under close clinical and echocardiographic surveillance. Surgical management subsequently consisted of apical amputation. This case illustrates that delayed mechanical complications may occur despite favorable early revascularization, and emphasizes the value of vigilant clinical monitoring, timely imaging reassessment, and patient-tailored decision-making regarding the timing of repair, which should integrate hemodynamic status, defect anatomy, tissue characteristics, and overall clinical condition.

Categories: Cardiology, Emergency Medicine, Internal Medicine
Keywords: acute myocardial infarction, delayed surgical repair, left ventricular aneurysm, mechanical complication, new systolic murmur, percutaneous coronary intervention, ventricular septal rupture

Introduction

Post-infarction ventricular septal defect (PIVSD), also referred to as post-infarction ventricular septal rupture (VSR), is a rare but life-threatening mechanical complication of acute myocardial infarction. Although its incidence has markedly declined with early reperfusion therapy, from approximately 1%-2% in the pre-reperfusion era to 0.25% in contemporary series, it remains associated with substantial morbidity and mortality [1,2]. PIVSD results from transmural ischemic necrosis and rupture of the interventricular septum, producing an acute left-to-right shunt, and typically develops three to seven days after acute myocardial infarction. Its clinical presentation is highly variable, ranging from a newly detected harsh systolic murmur in an otherwise stable patient to pulmonary edema, low cardiac output, biventricular failure, or cardiogenic shock [3].

Early recognition is essential, as conservative management alone is associated with extremely poor outcomes. In the absence of definitive repair, mortality remains extremely high, exceeding 90% in some medically treated series [4]. Transthoracic echocardiography with color Doppler is the first-line imaging modality for confirming the defect, assessing shunt direction and hemodynamic significance, and evaluating ventricular function. In selected stable patients, cardiac magnetic resonance imaging may provide complementary anatomical and tissue characterization by defining infarct extent, myocardial viability, and associated aneurysmal remodeling.

Management remains challenging and requires prompt hemodynamic assessment and multidisciplinary decision-making. Initial stabilization may include medical therapy and, in unstable patients, temporary mechanical circulatory support as a bridge to definitive closure. Although definitive closure is usually required, the optimal timing and modality remain debated, particularly the balance between urgent repair and delayed intervention, as well as the choice between surgical and transcatheter closure. These decisions must be individualized according to hemodynamic status, defect anatomy, tissue quality, associated ventricular remodeling, and institutional expertise [5,6].

We report the case of a 63-year-old man in whom PIVSD was detected on day 3 after an initially uncomplicated course following successful percutaneous coronary intervention (PCI), through a newly detected systolic murmur. This case combines several features: an initially stable presentation, a small restrictive apical defect associated with aneurysmal remodeling, and an individualized decision to delay surgical repair under close clinical and echocardiographic surveillance, with cardiac magnetic resonance

imaging guiding the surgical strategy. It emphasizes the need for continued clinical vigilance after myocardial infarction, even when reperfusion appears successful, and illustrates the value of repeated physical examination, prompt Doppler echocardiography, and multimodality imaging in individualizing the timing and modality of repair.

Case Presentation

A 63-year-old man with a history of hypertension, dyslipidemia on statin therapy, former smoking, and known coronary artery disease presented with an extensive ST-segment elevation myocardial infarction. His cardiovascular history was notable for a non-ST-segment elevation myocardial infarction in 2019, treated with drug-eluting stent implantation in the mid left anterior descending artery. He presented seven hours after symptom onset with typical prolonged ischemic chest pain occurring at rest, radiating to the left upper limb, and associated with nausea and vomiting. On admission, he was hemodynamically stable, with a heart rate of 112 beats/min, blood pressure of 100/60 mmHg, and preserved peripheral oxygen saturation. There were no clinical signs of peripheral hypoperfusion or overt left- or right-sided heart failure.

The admission electrocardiogram showed extensive ST-segment elevation involving leads V1-V6, I, aVL, II, and aVF, with hyperacute anterior T waves (Pardee waves), anterior pathologic Q waves consistent with evolving necrosis, and reciprocal ST-segment depression in lead aVR (Figure *1A*).

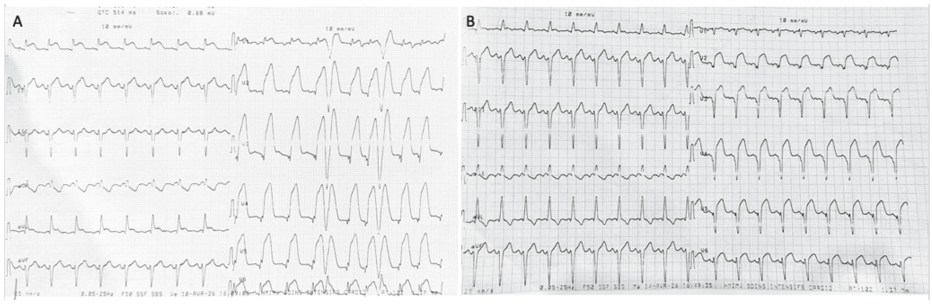


FIGURE 1: Electrocardiographic findings before and after reperfusion

Initial 12-lead electrocardiogram showing extensive ST-segment elevation in the anterior, lateral, and inferior leads (A);(B) follow-up EKG showing persistent anterior ST-segment elevation after reperfusion.

Despite successful reperfusion, anterior ST-segment elevation persisted on follow-up electrocardiograms, suggesting extensive transmural myocardial injury and possible evolving left ventricular aneurysmal remodeling (Figure *1B*). Initial laboratory findings are summarized in Table *1*.

Parameter	Value	Reference range
Hemoglobin (g/dL)	15.9	13-17
White blood cell count (/mm³)	14,500	4,000-10,000
Platelet count (mm³)	254,000	150,000-400,000
Prothrombin time (%)	72	70-100
Sodium (mmol/L)	141	135-145
Potassium (mmol/L)	3.4	3.7-5.3
Chloride (mmol/L)	107	95-110
Alkaline reserve (mmol/L)	22	21-28
Urea (g/L)	0.29	0.15-0.45
Creatinine (mg/L)	8	5.7-11.1
Estimated GFR (mL/min/1.73m²)	75	-
C-reactive protein(mg/L)	55	<5
Troponin I (ng/L)	697 to 81750	2-16
HbA1c (%)	5.5	<5.7
Total cholesterol (g/L)	1.08	<1.50
LDL cholesterol (g/L)	0.40	<1.55
HDL cholesterol (g/L)	0.47	0.40-0.60
Triglycerides (g/L)	1.05	0.60-1.50
TSH (mIU/L)	0.24	0.27-4.20
Free T4 (ng/dL)	1.1	0.93-1.70

TABLE 1: Initial laboratory and metabolic findings with reference ranges

HDL, high-density lipoprotein; LDL, low-density lipoprotein; TSH, thyroid-stimulating hormone

Initial bedside transthoracic echocardiography performed prior to coronary angiography revealed moderately reduced left ventricular systolic function with anterior regional wall motion abnormalities and did not identify any mechanical complication. Following initial stabilization, the patient was taken to the catheterization laboratory. Coronary angiography showed a thrombotic occlusion of the mid-left anterior descending artery at the site of the previously implanted drug-eluting stent (Figure *2A*), as well as a non-significant lesion of the intermediate branch. Successful primary percutaneous coronary intervention (PCI) of the mid-left anterior descending artery was achieved with implantation of a 3 × 22 mm drug-eluting stent (Figure *2B*).

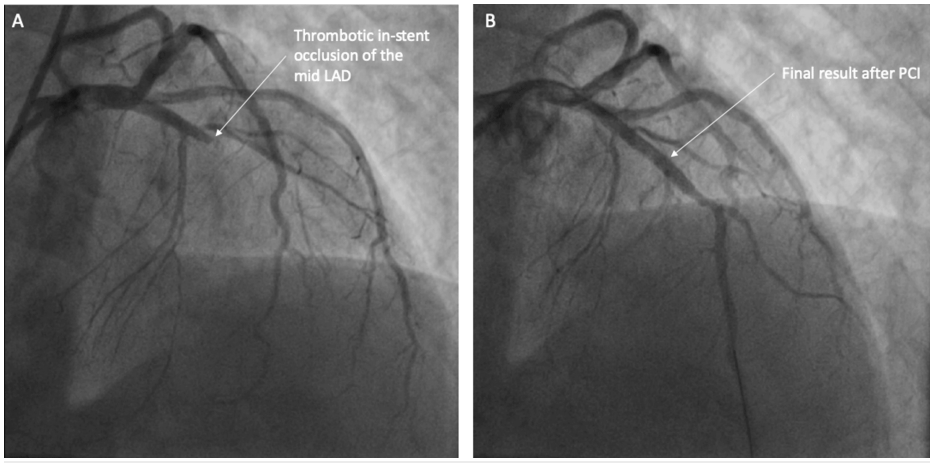


FIGURE 2: Coronary angiography of the culprit lesion and the final result after revascularization

(A) Thrombotic occlusion of the mid-left anterior descending artery at the site of the previously implanted drug-eluting stent.

(B) Final angiographic result after successful primary percutaneous coronary intervention with drug-eluting stent implantation.

The patient remained clinically stable during the early post-procedural course and was deemed suitable for transfer from the intensive cardiac care unit to the cardiology ward 72 hours after angioplasty. Shortly after transfer, repeat physical examination disclosed a new systolic murmur, maximal over the mitral area and radiating broadly across the precordium. In the context of recent ST-segment elevation myocardial infarction, this finding was highly suggestive of a mechanical complication and led to urgent repeat transthoracic echocardiography.

Repeat transthoracic echocardiography showed a non-dilated left ventricle with akinesia of the mid anteroseptal and apical segments, associated with compensatory basal hyperkinesia and reduced systolic function. An apical inferoseptal aneurysm was identified, complicated by a restrictive ventricular septal defect measuring approximately 4 mm, with a left-to-right shunt and a maximal Doppler velocity of 4.4 m/s (Figures *3*, *4*) (Videos *1*, *2*).

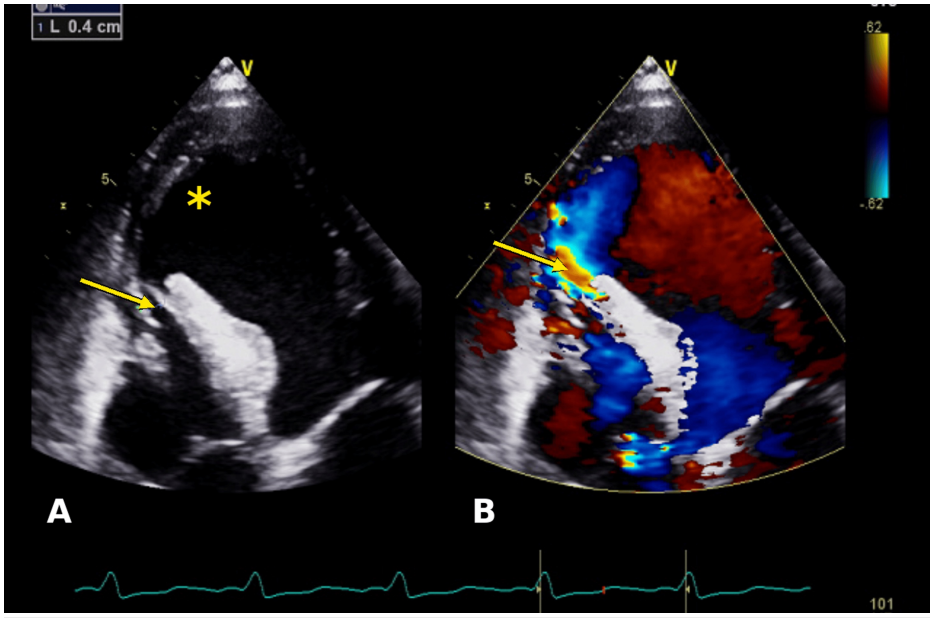


FIGURE 3: Apical four-chamber view showing a ventricular septal defect with left-to-right shunt and apical aneurysm

(A) Two-dimensional imaging showing a ventricular septal defect measuring 0.4 cm (left arrow) and aneurysmal dilatation of the left ventricular apex (asterisk). (B) Color Doppler imaging demonstrating left-to-right shunt flow across the septal defect (right arrow).

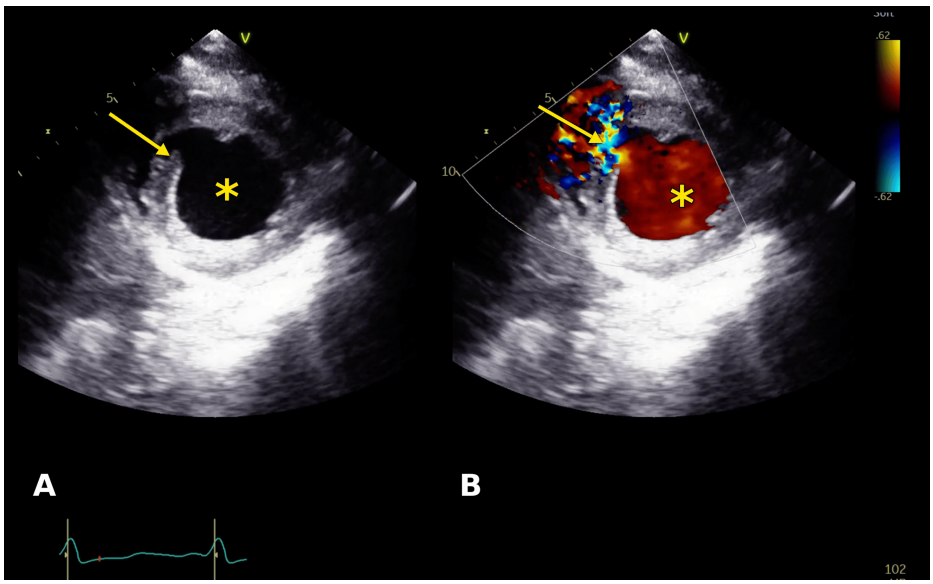
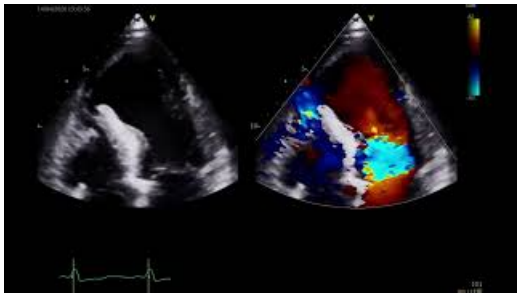


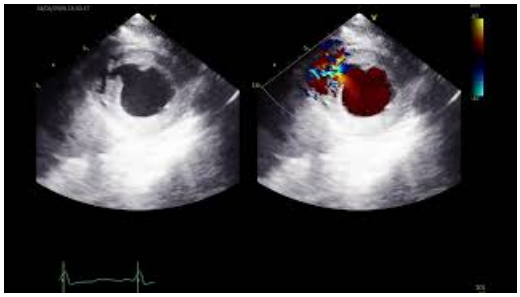
FIGURE 4: Parasternal short-axis color Doppler view showing the location of the ventricular septal defect

(A) Two-dimensional imaging showing the aneurysmal cavity (asterisk) and its communication (arrow). (B) Color Doppler imaging demonstrating turbulent mosaic flow at the level of the defect (arrow) and filling of the aneurysmal cavity (asterisk).



VIDEO 1: Apical four-chamber view showing ventricular septal defect with left-to-right shunt

View video here: https://youtu.be/0pGxhw5LhKk



VIDEO 2: Parasternal short-axis color Doppler view showing the location of the ventricular septal defect

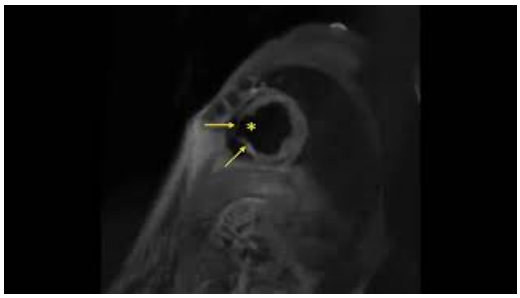
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Although transthoracic echocardiography established the diagnosis, cardiac magnetic resonance imaging was performed to further delineate the anatomical features of the septal defect and associated left ventricular aneurysmal remodeling. It confirmed the presence of an apical inferoseptal ventricular septal defect arising from an aneurysmal segment (Figure *5*) (Video *3*), as well as the absence of myocardial viability in the left anterior descending artery territory.



FIGURE 5: Cardiac magnetic resonance imaging, short-axis T2-weighted view, showing the ventricular septal defect and left ventricular aneurysm

Image from the T2-weighted sequence plane demonstrating discontinuity of the interventricular septum consistent with the ventricular inferoseptal defect (horizontal arrow), thinning of the adjacent septal wall (oblique arrow), and the left ventricular aneurysm (asterisk).



VIDEO 3: Short-axis T2-weighted cardiac magnetic resonance sequence showing the apical Inferoseptal ventricular septal defect and left ventricular aneurysm

Sequential short-axis slices from base to apex. On the two consecutive slices showing the defect, arrows indicate the discontinuity of the interventricular septum and the thinned adjacent inferoseptal wall, and the asterisk marks the left ventricular aneurysm. These slices are held on screen at each pass. The annotated slice corresponds to Figure 5.

View video here: https://youtu.be/KsQH0K_OCTQ

Following Heart Team discussion, surgical repair was indicated. The timing of repair was individualized: given the patient's sustained hemodynamic and respiratory stability, a delayed surgical strategy was considered acceptable, with repair scheduled approximately two weeks after diagnosis. During this period, intra-aortic balloon pump support was considered but deemed unnecessary, in the absence of cardiogenic

shock or refractory congestion. Medical therapy was optimized with intravenous loop diuretics for decongestion (furosemide 60 mg twice daily) and cautious afterload reduction with low-dose ramipril (1.25 mg twice daily), together with dual antiplatelet therapy (aspirin 100 mg/day and clopidogrel 75 mg/day) and high-intensity statin therapy (atorvastatin 80 mg/day), under close clinical, biological, and echocardiographic surveillance. Clopidogrel was discontinued five days before surgery, while aspirin was continued. Clinical monitoring included continuous electrocardiographic and non-invasive arterial blood pressure monitoring, together with daily assessment of urine output and signs of congestion. Serial transthoracic echocardiography was performed every 72 hours, showing a stable defect size of 0.4 cm, a stable left-to-right shunt, a mild decline in left ventricular systolic function related to the attenuation of compensatory basal hyperkinesis, preserved right ventricular systolic function, and stable systolic pulmonary artery pressure. No clinical or echocardiographic deterioration requiring earlier intervention was observed. Concomitant coronary artery bypass grafting was not performed, as the culprit left anterior descending (LAD) artery had been successfully revascularized by PCI, the intermediate branch lesion was non-significant, and cardiac magnetic resonance imaging demonstrated transmural late gadolinium enhancement (>50% of wall thickness) involving 5 of 7 segments of the LAD territory, with wall thinning and akinesia, indicating non-viable myocardium with a low likelihood of functional recovery after revascularization. Surgical management consisted of ventricular septal defect repair using the apical amputation technique described by Daggett [7]. The immediate postoperative course was uneventful. The patient remained clinically stable and was subsequently maintained on guideline-directed medical therapy.

Discussion

PIVSD is an uncommon but devastating complication of acute myocardial infarction whose incidence has substantially declined with modern reperfusion strategies. However, this reduction in prevalence has not been paralleled by a comparable improvement in outcomes, especially when the defect is complicated by cardiogenic shock. Its clinical expression is highly variable and depends on the size, location, morphology, and hemodynamic impact of the defect. While large defects may rapidly lead to pulmonary edema, biventricular failure, cardiogenic shock, or cardiac arrest, smaller restrictive defects may initially present with subtle findings. Therefore, a newly detected systolic murmur after myocardial infarction should be considered a key warning sign and should prompt immediate transthoracic echocardiography [3,4].

In our case, the new systolic murmur was the clinical trigger for repeat imaging, despite preserved hemodynamic stability after successful reperfusion. Transthoracic echocardiography rapidly confirmed the diagnosis by demonstrating a restrictive left-to-right shunt arising from an apical inferoseptal defect within an aneurysmal segment, while also assessing defect location, shunt severity, and left ventricular systolic function. Cardiac magnetic resonance imaging added incremental value by delineating the aneurysmal remodeling and documenting non-viable myocardium in the left anterior descending artery territory. This combined anatomical and tissue characterization was important for therapeutic planning.

The management of PIVSD requires an individualized multidisciplinary Heart Team approach, integrating cardiologists, cardiac surgeons, interventional cardiologists, and anesthesiologists [2,8]. Surgical repair remains the reference treatment, but transcatheter closure has emerged as an alternative in selected patients, particularly those at high surgical risk or with residual defects after surgery. In a recent meta-analysis, surgical closure was associated with lower short-term mortality and a lower need for reintervention than transcatheter closure, whereas long-term mortality did not differ significantly between the two approaches [9]. The feasibility of device closure depends strongly on defect size, morphology, location, and the quality of the surrounding tissue [9]. In our patient, the apical location of the defect and its association with aneurysmal remodeling favored surgical repair, as a device would not have addressed the aneurysm. The apical amputation technique described by Daggett was considered particularly suitable [7], as it allows resection of the aneurysmal apical segment and exclusion of the septal communication in a single procedure.

The optimal timing of repair remains debated. Patients with cardiogenic shock refractory to medical therapy require urgent intervention, although temporary mechanical circulatory support may be used as a bridge to restore hemodynamic stability and allow preoperative optimization. Intra-aortic balloon pump counterpulsation remains the most commonly used strategy, mainly through afterload reduction and reduction of left-to-right shunting, whereas venoarterial extracorporeal membrane oxygenation or other advanced devices may be considered in refractory cases [6,10]. In hemodynamically stable patients, delayed repair is increasingly adopted [5]. In the Society of Thoracic Surgeons database, operative mortality was 54.1% when repair was performed within 7 days of myocardial infarction, compared with 18.4% beyond 7 days [4], and the UK national surgical audit of 1,010 patients reported an operative mortality of 61.3% when repair was performed within 24 hours of infarction [11]. However, these observational data must be interpreted with caution, as they are subject to survival and selection bias: the most unstable patients undergo early surgery, while those who die before delayed repair are not captured in surgical series [4]. The apparent benefit of delayed surgery may therefore partly reflect patient selection rather than a true effect of timing. The multicenter CAUTION study reported an early surgical mortality of 40.4%, unchanged over two decades, and described the association between delayed surgery and survival with caution [12]. Accordingly, the timing of repair should be individualized according to hemodynamic status, defect anatomy, tissue characteristics, and the overall clinical condition of the patient [5]. In our patient, sustained hemodynamic

stability and a small restrictive defect allowed repair approximately two weeks after diagnosis, under close clinical and echocardiographic surveillance, with no deterioration requiring earlier intervention.

This case illustrates several practical lessons. First, PIVSD may develop despite timely reperfusion and an initially uncomplicated course, so daily clinical examination, including auscultation, remains essential in the first days after myocardial infarction. Second, multimodality imaging is central at each step of management. Serial echocardiography turns a delayed strategy into active surveillance of defect size, shunt severity, biventricular function, and pulmonary pressures, with a low threshold for earlier repair if any of these deteriorate. Combined with cardiac magnetic resonance imaging, it also individualizes the surgical decision, as defect location and associated aneurysm guided the choice of surgical over device closure and the repair technique, while the absence of viability in an already revascularized LAD territory supported the decision not to perform bypass grafting. Finally, in apical defects associated with aneurysmal remodeling, apical amputation addresses both lesions in a single procedure. Despite advances in reperfusion, imaging, and surgical care, PIVSD remains associated with high mortality, and its management should rest on individualized Heart Team decision-making rather than a uniform strategy.

Conclusions

Post-infarction ventricular septal defect remains a rare but highly lethal mechanical complication of myocardial infarction. This case highlights that PIVSD may occur even in initially stable patients, emphasizing the importance of careful bedside examination even after reperfusion. Prompt echocardiographic confirmation, close monitoring, and early multidisciplinary discussion are essential to guide definitive repair, with delayed surgery representing a reasonable option in selected stable patients, provided that the timing of repair is individualized.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Kaoutar Maliki

Acquisition, analysis, or interpretation of data: Kaoutar Maliki, Khadija Dekkak, Amal Hsain, Mohamed Malki

Drafting of the manuscript: Kaoutar Maliki

Critical review of the manuscript for important intellectual content: Khadija Dekkak, Amal Hsain, Mohamed Malki

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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