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Case Report

Delayed Surgical Repair of Post-Infarction Apical Ventricular Septal Rupture Bridged with Intra-Aortic Balloon Pump Support: A Case Report with 1-Year Follow-Up

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ABSTRACT

Post-infarction ventricular septal rupture (VSR) is a rare but life-threatening mechanical complication of acute myocardial infarction (MI). We report a 52-year-old male who presented with acute inferior wall ST-elevation MI complicated by cardiogenic shock and a 12-mm apical VSR. Initial serum lactate was 4.8 mmol/L, peaking at 5.7 mmol/L preoperatively, with oliguria, progressive renal dysfunction (peak creatinine 3.0 mg/dL), and hepatic injury (peak AST 1312 U/L; ALT 965 U/L). Coronary angiography revealed 100% distal occlusion of a dominant right coronary artery. Intra-aortic balloon pump (IABP) support was initiated on admission with concurrent vasopressor therapy. Delayed surgical repair was performed on hospital day 8 following recurrent hypoperfusion despite temporary stabilization. Total IABP duration was 11 days. Postoperative echocardiography demonstrated trivial residual shunt with left ventricular ejection fraction (LVEF) of 35%. At 1-year follow-up, the patient remained asymptomatic (NYHA class I) with LVEF 40% and no clinically significant residual shunt. This case illustrates a pragmatic application of serial physiological and end-organ perfusion assessment to help individualize timing of delayed surgical repair in a patient with post-infarction VSR bridged by prolonged IABP support.

Keywords: Ventricular Septal Rupture; Myocardial Infarction; Cardiogenic Shock; Intra-Aortic Balloon Pump; Delayed Surgical Repair

Introduction

Ventricular septal rupture (VSR) is an uncommon but catastrophic mechanical complication of acute myocardial infarction (MI), occurring in less than 1% of patients in the contemporary reperfusion era [1–3]. Despite advances in reperfusion therapy, surgical techniques, and mechanical circulatory support (MCS), mortality remains extremely high, particularly in patients presenting with cardiogenic shock and multiorgan hypoperfusion [3–5]. Surgical repair remains the definitive treatment; however, the optimal timing of intervention continues to be controversial.

Early surgical repair is often unavoidable in patients with refractory shock or rapidly progressive hemodynamic collapse but is associated with substantial operative mortality due to friable necrotic myocardium and risk of patch dehiscence [4–7]. Conversely, delayed repair may permit infarct maturation and improved tissue integrity for surgical reconstruction, although prolonged delay risks worsening end-organ dysfunction and death in unstable patients [5–7]. Reported survival advantages associated with delayed surgery must also be interpreted cautiously because of significant survivor and selection bias within observational studies [4–6].

Mechanical circulatory support is frequently used to stabilize patients before definitive repair. Intra-aortic balloon pump (IABP) counterpulsation remains the most widely available support modality and may reduce left ventricular afterload and left-to-right shunt fraction [8]. More advanced support strategies, including Impella and venoarterial extracorporeal membrane oxygenation (VA-ECMO), can provide greater hemodynamic support but are associated with higher cost, increased complexity, vascular complications, and variable institutional availability [9]. Percutaneous closure techniques have also emerged as potential alternatives or bridging strategies in selected patients but may be limited by acute tissue friability, irregular defect morphology, and large shunt burden during the early infarction phase [10].

Although delayed repair supported by temporary MCS has been previously described, there remains limited literature detailing how serial physiological and end-organ perfusion parameters may practically inform timing of intervention in unstable patients. Rather than proposing a novel management paradigm, this report describes a pragmatic application of serial clinical, biochemical, and hemodynamic monitoring to guide timing of delayed surgical repair in a patient with post-infarction apical VSR complicated by cardiogenic shock. The case is additionally notable because the apical septal rupture occurred following isolated distal dominant right coronary artery occlusion, an uncommon anatomical pattern compared with the classic anterior MI-associated apical VSR.

Case Presentation

A 52-year-old hypertensive man presented six hours after onset of severe retrosternal chest pain associated with diaphoresis and progressive dyspnea. On admission, he was tachycardic (heart rate 110 bpm), hypotensive (blood pressure 90/60 mmHg; mean arterial pressure [MAP] 58–60 mmHg), tachypneic (respiratory rate 28/min), and oliguric (<0.4 mL/kg/h) with cool extremities and clinical evidence of systemic hypoperfusion. Oxygen saturation was 92% on supplemental oxygen via face mask. Electrocardiography demonstrated inferior ST-elevation myocardial infarction.

Initial laboratory investigations revealed hs-troponin I 38,420 ng/L, peaking at >50,000 ng/L on hospital day 1. Serum lactate was 4.8 mmol/L on admission and subsequently peaked at 5.7 mmol/L preoperatively. Serum creatinine was 3.0 mg/dL, AST 386 U/L, and ALT 241 U/L at presentation, with subsequent peak AST and ALT values of 1312 U/L and 965 U/L, respectively, consistent with worsening hypoperfusion-related hepatic injury.

Transthoracic echocardiography demonstrated a 12-mm apical ventricular septal rupture within the inferoapical septum with irregular margins and significant left-to-right shunting on color Doppler imaging. Mild right ventricular systolic dysfunction was present with estimated pulmonary artery systolic pressure of approximately 45 mmHg. Initial left ventricular ejection fraction (LVEF) was approximately 50% with inferoapical hypokinesia. Serial echocardiography during hospitalization demonstrated persistent shunting without significant increase in defect size or evidence of progressive right ventricular failure. Formal Qp/Qs quantification was not feasible because of ongoing hemodynamic instability.

Coronary angiography demonstrated 100% occlusion of the distal dominant right coronary artery without significant left coronary artery disease. The anatomical distribution suggested wrap-around inferoapical septal supply from the dominant distal right coronary circulation, explaining the atypical occurrence of apical VSR in the setting of inferior MI. Primary percutaneous coronary intervention (PCI) was deferred despite early presentation because the culprit lesion was distal with limited myocardial salvage

potential and an established mechanical complication had already developed. No delayed PCI was performed. Concomitant coronary artery bypass grafting (CABG) was not indicated because there was no surgically significant residual coronary artery disease.

Invasive arterial and central venous monitoring were established on admission. Norepinephrine infusion was initiated with a peak dose of 0.18 μg/kg/min. An intra-aortic balloon pump (IABP) was inserted via the right femoral artery within hours of admission using 1:1 counterpulsation augmentation along with intravenous unfractionated heparin anticoagulation. No vascular access complications, limb ischemia, major bleeding, or device-related complications occurred during support.

Following IABP insertion, hemodynamics improved with MAP increasing to 70–75 mmHg and gradual reduction in vasopressor requirement. Norepinephrine dose decreased from 0.18 μg/kg/min to 0.03 μg/kg/min over the subsequent stabilization period. Urine output improved from <0.4 mL/kg/h to approximately 1.0–1.2 mL/kg/h. Serum lactate decreased from 4.8 mmol/L to 1.8 mmol/L, while creatinine improved from 3.0 mg/dL to 1.5 mg/dL. Oxygenation remained stable on supplemental oxygen without requirement for invasive mechanical ventilation prior to surgery.

On hospital day 7, recurrent hemodynamic deterioration developed with rising lactate (2.6 mmol/L progressing to 5.7 mmol/L preoperatively), increasing vasopressor requirement, worsening renal function, declining urine output, recurrent hypotension (MAP 58–62 mmHg), and progressive end-organ dysfunction despite ongoing IABP support. Following multidisciplinary discussion, urgent surgical repair was performed on hospital day 8.

Intraoperative findings confirmed a large apical septal rupture with partially organized infarcted myocardium involving the inferoapical septum. Repair was performed via median sternotomy under cardiopulmonary bypass through a left ventriculotomy approach. A Dacron patch was secured using interrupted pledgeted sutures. Cardiopulmonary bypass time was 112 minutes and aortic cross-clamp time was 74 minutes. Infarct exclusion technique was not required because tissue quality at the defect margins was considered adequate for patch anchoring.

Postoperatively, the patient recovered uneventfully. IABP support was successfully weaned and removed on postoperative day 3, resulting in a total IABP duration of 11 days. Serum lactate decreased to 1.9 mmol/L with progressive improvement in renal and hepatic function. Postoperative transthoracic echocardiography demonstrated trivial residual left-to-right shunt with LVEF 35%. The reduction in postoperative LVEF compared with initial presentation was attributed to infarct evolution, ventriculotomy-related myocardial injury, and postoperative ventricular remodeling following repair.

The patient was discharged on aspirin, clopidogrel, atorvastatin, metoprolol succinate, ramipril, spironolactone, and furosemide.

At 1-month follow-up, he remained asymptomatic (NYHA class I) with LVEF 38% and trivial residual shunt on echocardiography. At 1-year follow-up, he continued to be NYHA class I with LVEF 40%, stable ventricular remodeling, mild residual inferoapical hypokinesia, and no clinically significant residual shunt.

Written informed consent for publication of clinical details and images was obtained from the patient. A completed CARE checklist is provided as supplementary material.

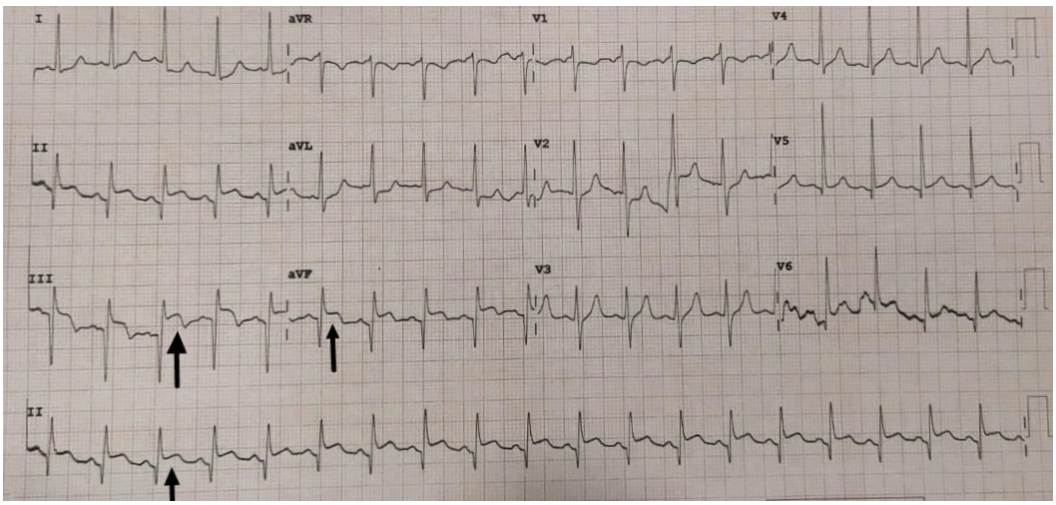


Figure 1. Twelve-lead electrocardiogram (25 mm/s, 10 mm/mV) showing sinus tachycardia with ST-segment elevation in leads II, III, and aVF and reciprocal ST depression in leads I and aVL, consistent with acute inferior wall ST-elevation myocardial infarction.

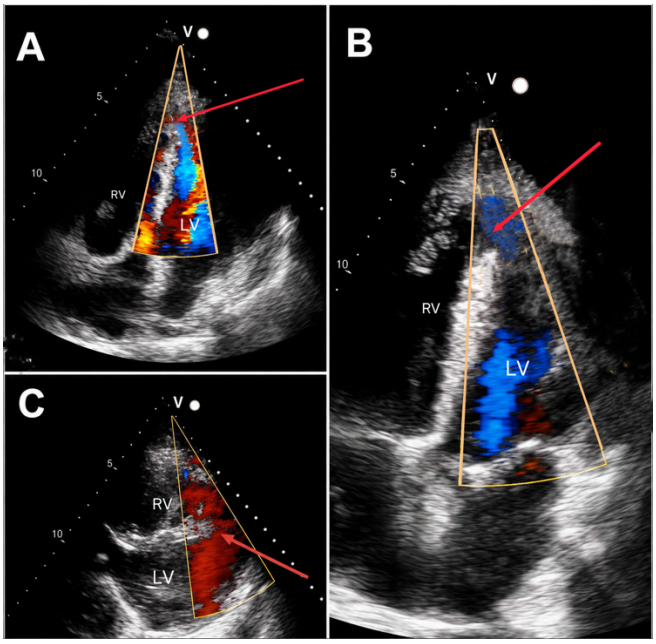


Figure 2. Transthoracic echocardiography with color Doppler demonstrating a large apical ventricular septal rupture with significant left-to-right shunting (arrows) in apical four-chamber (A, C) and parasternal short-axis (B) views.

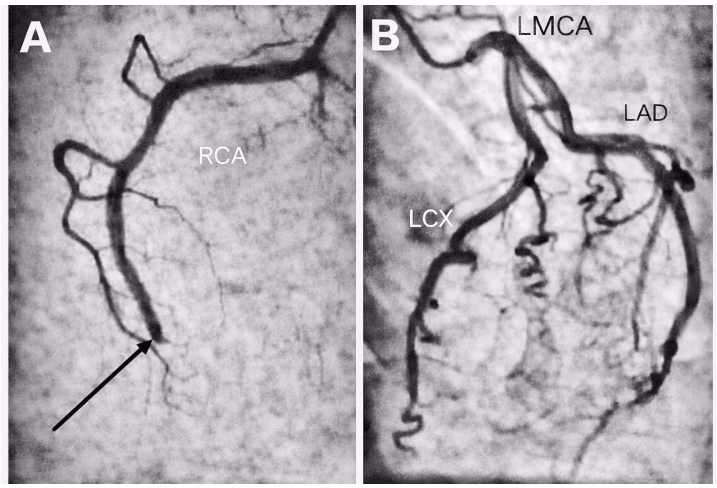


Figure 3. Coronary angiography. (A) Right coronary artery angiogram demonstrating 100% distal occlusion (arrow). (B) Left coronary angiogram showing normal left main, left anterior descending, and left circumflex arteries without significant stenosis.

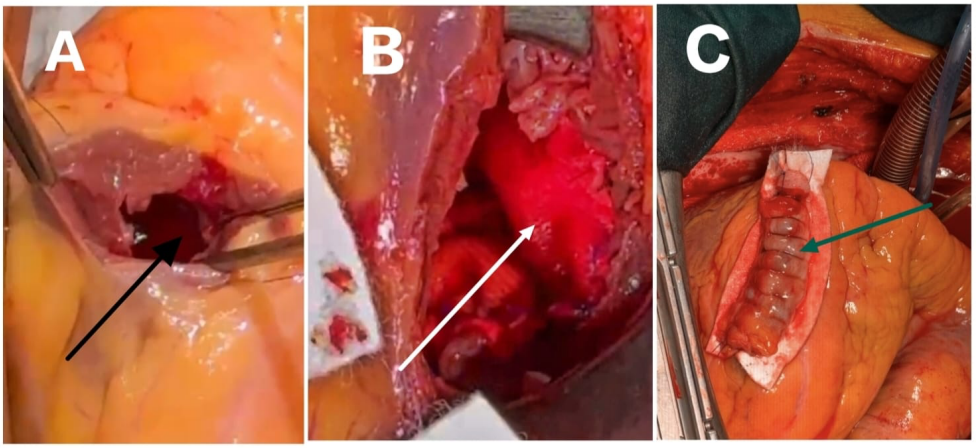
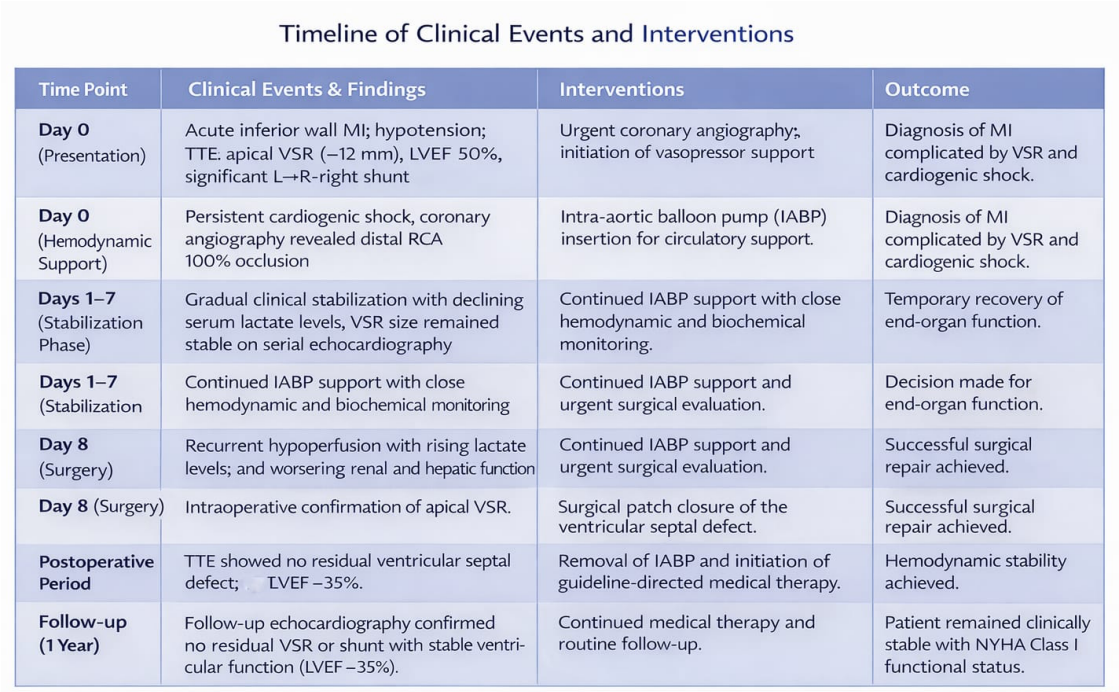


Figure 4. Intraoperative findings and surgical repair of post–myocardial infarction apical ventricular septal rupture. (A) Left ventriculotomy exposing a large apical septal rupture (arrow). (B) Patch closure of the septal defect using a Dacron patch secured with interrupted pledgeted sutures (arrow). (C) Completed repair following closure of the left ventriculotomy.



Timeline of Clinical Events and Interventions

Time Point	Clinical Events & Findings	Interventions	Outcome
Day 0 (Presentation)	Acute inferior wall MI; hypotension; TTE: apical VSR (–12 mm), LVEF 50%, significant L→R-right shunt	Urgent coronary angiography; initiation of vasopressor support	Diagnosis of MI complicated by VSR and cardiogenic shock.
Day 0 (Hemodynamic Support)	Persistent cardiogenic shock, coronary angiography revealed distal RCA 100% occlusion	Intra-aortic balloon pump (IABP) insertion for circulatory support.	Diagnosis of MI complicated by VSR and cardiogenic shock.
Days 1–7 (Stabilization Phase)	Gradual clinical stabilization with declining serum lactate levels, VSR size remained stable on serial echocardiography	Continued IABP support with close hemodynamic and biochemical monitoring.	Temporary recovery of end-organ function.
Days 1–7 (Stabilization	Continued IABP support with close hemodynamic and biochemical monitoring	Continued IABP support and urgent surgical evaluation.	Decision made for end-organ function.
Day 8 (Surgery)	Recurrent hypoperfusion with rising lactate levels; and worsering renal and hepatic function	Continued IABP support and urgent surgical evaluation.	Successful surgical repair achieved.
Day 8 (Surgery)	Intraoperative confirmation of apical VSR.	Surgical patch closure of the ventricular septal defect.	Successful surgical repair achieved.
Postoperative Period	TTE showed no residual ventricular septal defect; LVEF –35%.	Removal of IABP and initiation of guideline-directed medical therapy.	Hemodynamic stability achieved.
Follow-up (1 Year)	Follow-up echocardiography confirmed no residual VSR or shunt with stable ventricular function (LVEF –35%).	Continued medical therapy and routine follow-up.	Patient remained clinically stable with NYHA Class I functional status.

Figure 5. Summarised timeline of clinical events, findings, interventions, and outcomes in a patient with acute inferior wall myocardial infarction complicated by apical ventricular septal rupture**.**

Table 1. Serial biochemical, hemodynamic, and end-organ perfusion parameters during intra-aortic balloon pump support and delayed surgical repair for post-infarction ventricular septal rupture. Trends in serum lactate, renal and hepatic function, urine output, vasopressor requirement, and mean arterial pressure were used to assess systemic perfusion status and guide timing of surgical intervention.

Hospital Day	Serum Lactate (mmol/L)	hs-Troponin I (ng/L)	Serum Creatinine (mg/dL)	AST (U/L)	ALT (U/L)	Urine Output (mL/kg/h)	Norepinephrine (μg/kg/min)	MAP (mmHg)	Clinical Status	Management Decision
Day 0 (Admission)	4.8	38,420	3.0	386	241	<0.4	0.18	58–60	Cardiogenic shock with hypoperfusion and acute kidney injury	Norepinephrine initiated; IABP inserted
Day 1	3.9	>50,000	2.8	1312	965	0.4	0.16	62–65	Persistent hypoperfusion with marginal hemodynamic improvement	Continued mechanical support
Day 2	2.8	-	2.4	-	-	0.6	0.12	68–70	Early lactate clearance with improving renal perfusion	Deferred surgery; continued stabilization
Day 3	2.2	39,600	2.0	440	210	0.8	0.08	70–72	Improved systemic perfusion	Continued IABP support

Hospital Day	Serum Lactate (mmol/L)	hs-Troponin I (ng/L)	Serum Creatinine (mg/dL)	AST (U/L)	ALT (U/L)	Urine Output (mL/kg/h)	Norepinephrine (μg/kg/min)	MAP (mm Hg)	Clinical Status	Management Decision
Day 4	1.9	-	1.7	-	-	1.0	0.05	72–75	Hemodynamic stabilization with partial organ recovery	Multidisciplinary review; delayed repair strategy
Day 5–6	1.8	-	1.5	150	132	1.0–1.2	0.03	72–75	Temporary stabilization phase	Continued support with close monitoring
Day 7	2.6	20,880	1.9	228	205	0.7	0.08	65–68	Early recurrence of hypoperfusion	Heightened monitoring
Day 8 (Preoperative)	5.7	18,450	2.6	565	318	<0.5	0.15	58–62	Recurrent cardiogenic shock with worsening organ dysfunction	Urgent surgical repair
Postoperative Day 1	3.4	-	2.3	348	220	0.7	0.10	68–70	Improving perfusion and organ recovery	Intensive care monitoring
Postoperative Day 3	1.9	8,640	1.8	172	148	1.0	Off vasopressors	72–75	Stabilization following surgical repair	IABP removal and continued monitoring

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; hs-Troponin I, high-sensitivity troponin I; IABP, intra-aortic balloon pump; MAP, mean arterial pressure; NR, not repeated.

Discussion

Post-infarction ventricular septal rupture (VSR) remains one of the most lethal mechanical complications of acute myocardial infarction despite advances in reperfusion therapy, cardiac surgery, and mechanical circulatory support (MCS) [2–6,10]. Although delayed surgical repair has been associated with lower operative mortality in observational studies, these findings are influenced by significant survivor and selection bias, as patients surviving long enough to undergo delayed intervention are often more hemodynamically stable than those requiring emergent surgery [4–7].

This case illustrates a pragmatic, physiology-guided approach to timing of delayed repair rather than proposing a new management framework. Serial assessment of lactate levels, urine output, vasopressor requirements, mean arterial pressure, and end-organ function helped identify a temporary stabilization period during prolonged IABP support before recurrent deterioration prompted definitive surgical intervention.

IABP was selected because it was immediately available, rapidly deployable, and capable of reducing afterload and left-to-right shunt fraction while providing sufficient temporary hemodynamic support. Although advanced MCS strategies such as Impella or venoarterial extracorporeal membrane oxygenation (VA-ECMO) may provide greater circulatory support, these modalities are associated with higher cost, increased technical complexity, and greater resource utilization [8,9]. In the present case, institutional resource availability, local expertise, and clinical stability achieved with IABP favored continued balloon pump support rather than escalation to more advanced devices.

Percutaneous closure was not considered suitable because the defect was located within acutely infarcted, friable myocardium with irregular apical morphology and significant shunt burden during

ongoing cardiogenic shock. The risk of device instability, residual shunting, and extension of necrotic tissue was considered high in the acute setting, making surgical repair the preferred definitive treatment strategy.

An additional notable aspect of this case is the occurrence of an apical VSR following isolated distal dominant right coronary artery occlusion. Apical septal rupture is classically associated with anterior myocardial infarction involving the left anterior descending artery, whereas inferior infarction more commonly produces basal septal defects [10]. In this patient, wrap-around distal right coronary circulation likely supplied the inferoapical septum, providing a plausible anatomical explanation for this less common presentation.

The reduction in left ventricular ejection fraction (LVEF) from approximately 50% at presentation to 35% postoperatively likely reflected infarct evolution, ventriculotomy-related myocardial injury, and postoperative ventricular remodeling. Partial recovery of LVEF to 40% at 1-year follow-up with NYHA class I functional status suggested subsequent ventricular adaptation after successful repair.

Compared with previously reported delayed-repair cases, this report provides detailed longitudinal physiological, biochemical, hemodynamic, operative, and follow-up data demonstrating how serial perfusion markers may assist individualized clinical decision-making during prolonged IABP-supported stabilization. However, this single-case experience should be considered illustrative and hypothesis-generating rather than representative of a standardized treatment strategy.

A few limitations should be acknowledged. Hemodynamic assessment was based primarily on serial clinical evaluation and biochemical perfusion markers, as advanced invasive hemodynamic monitoring modalities, including pulmonary artery catheter–based cardiac output assessment, and advanced mechanical circulatory support options were limited by local resource availability. Formal Qp/Qs shunt quantification was not feasible during the acute phase because of ongoing hemodynamic instability. Furthermore, the observational nature of a single-case report inherently limits the ability to draw definitive conclusions regarding optimal timing of repair or broader applicability of this management approach.

Conclusion

This case demonstrates successful delayed surgical repair of post-infarction apical ventricular septal rupture following temporary stabilization with prolonged intra-aortic balloon pump support. Serial assessment of hemodynamic and end-organ perfusion parameters assisted clinical decision-making regarding timing of intervention in a resource-limited setting. This report illustrates a practical individualized management approach; however, broader conclusions regarding optimal timing strategies require further validation in larger studies.

Learning Points

- Prolonged intra-aortic balloon pump support may provide temporary hemodynamic stabilization in selected patients with post-infarction ventricular septal rupture.
- Serial assessment of lactate, urine output, vasopressor requirement, and end-organ function may assist individualized timing of surgical repair.
- Recurrent hemodynamic or biochemical deterioration during mechanical support may indicate the need for definitive surgical intervention.

List of Abbreviations

Abbreviation	Full Form
ALT	Alanine Aminotransferase
AST	Aspartate Aminotransferase
CABG	Coronary Artery Bypass Grafting
CARE	CAse REport Guidelines
CPB	Cardiopulmonary Bypass
ECG	Electrocardiography
IABP	Intra-Aortic Balloon Pump
LVEF	Left Ventricular Ejection Fraction
MAP	Mean Arterial Pressure
MCS	Mechanical Circulatory Support
MI	Myocardial Infarction
NYHA	New York Heart Association
PCI	Percutaneous Coronary Intervention
Qp/Qs	Pulmonary-to-Systemic Flow Ratio
RCA	Right Coronary Artery
RV	Right Ventricle / Right Ventricular
STEMI	ST-Elevation Myocardial Infarction
VA-ECMO	Venoarterial Extracorporeal Membrane Oxygenation
VSR	Ventricular Septal Rupture

Supplementary Materials

Supplementary file available via: https://www.jebm.org/suppfile/751/jebm003-suppl.-mat.-Complete-CARE-checklist-for-case-report-vsr.jpeg

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Ethics Approval and Consent to Participate: Institutional Review Board approval was waived for this case report, as it describes a single patient managed as part of routine clinical care, in accordance with institutional and national ethical guidelines.

Consent for Publication: Written informed consent for publication of this case report and accompanying images was obtained from the patient's legally authorized representative. All identifying information has been anonymized.

Availability of Data and Materials: The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing Interests: The authors declare that they have no competing interests.

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Authors' Contributions: ZRA conceived the study and drafted the manuscript. ZAH contributed to clinical management and manuscript revision. AK and SJ assisted with surgical data acquisition and interpretation. YL and MH supervised the study and critically revised the manuscript.
All authors read and approved the final manuscript.

Disclosure of AI Use: The authors declare that no artificial intelligence (AI) or AI-assisted technologies were used in the preparation, writing, analysis, interpretation, or editing of this manuscript.

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