CASE REPORT

From Cardiac Arrest to Definitive Repair: Delayed VSR Closure and CABG in Post-Infarction Ventricular Septal Rupture

ABSTRACT

Background: Post-myocardial infarction ventricular septal rupture (VSR) complicated by cardiac arrest and cardiogenic shock stage E by SCAI (Society for Cardiovascular Angiography and Interventions stage) is a catastrophic event with prohibitive surgical mortality. While immediate repair is fraught with risks due to tissue friability, optimal timing remains debated. We highlight a "stabilize-then-delay" strategy that utilizes the Intra-Aortic Balloon Pump (IABP) as a crucial hemodynamic bridge to facilitate delayed definitive repair.

Case: We report a case of a patient presenting with post-MI VSR and single-vessel disease who suffered cardiogenic shock (SCAI stage E) complicated by cardiac arrest on January 22, 2026. Following resuscitation, IABP support was immediately initiated as a bridge to surgery, allowing for significant hemodynamic stabilization and successful extubation on January 27, 2026. This stability permitted a strategic 12-day delay from the arrest event to surgery. Definitive repair, consisting of VSR closure and coronary artery bypass grafting (CABG), was performed on February 3, 2026. The patient achieved early postoperative extubation and successful IABP weaning. However, a residual shunt was noted on postoperative day two, highlighting the complexity of tissue management even with delayed intervention.

Discussion: This case demonstrates that an IABP-mediated "stabilize-then-delay" strategy was feasible even in extreme conditions such as stage E SCAI shock. In the shift from emergency surgery to stable, semi-elective surgery, IABP serves as a crucial bridge, enabling organ recovery before repair.

Conclusion: In selected patients who stabilize after profound shock, temporary mechanical circulatory support may provide time for end-organ recovery before surgical VSR repair. The risks of deterioration during the waiting period and postoperative residual shunting require continued individualized assessment.

Keywords: bridge to surgery; cardiac arrest; intra-aortic balloon pump; myocardial infarction; ventricular septal rupture.

INTRODUCTION

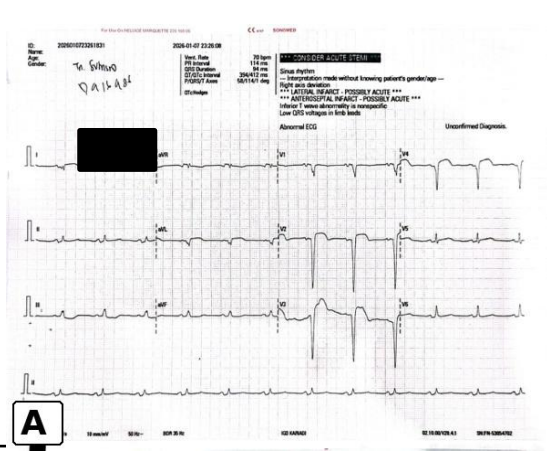
Post-myocardial infarction ventricular septal rupture (post-MI VSR) remains one of the most lethal mechanical complications of acute myocardial infarction (AMI).[1] Despite advancements in percutaneous reperfusion strategies, the incidence of VSR persists at approximately 0.2% to 0.3%, carrying a dismal prognosis. Without surgical intervention, mortality rates exceed 90%, driven largely by rapid hemodynamic deterioration leading to profound cardiogenic shock and multiorgan failure.[2] However, even with surgical repair, operative mortality remains notoriously high, ranging from 40% to 50% in various registries, particularly when the patient presents in advanced stages of shock.[3]

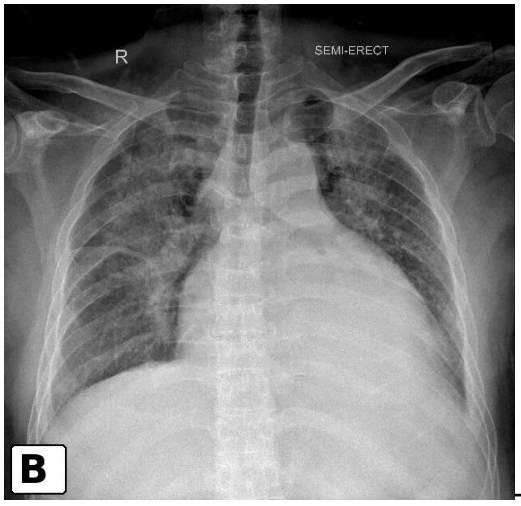
The management of post-MI VSR poses a complex clinical dilemma regarding the optimal timing of surgery. Early intervention, while necessary to correct the hemodynamic burden, is often compromised by the presence of necrotic and friable myocardial tissue, which holds sutures poorly and increases the risk of recurrent septal defects.[4] Conversely, delaying surgery allows for myocardial fibrosis and scar formation, facilitating a more secure repair, but exposes the patient to the high risk of death while waiting. This "timing dilemma" is most critical in patients presenting with SCAI Stage E cardiogenic shock (Extremis), defined by circulatory collapse and cardiac arrest, where immediate survival is uncertain.[5]
To address this, a "stabilize-then-delay" strategy has emerged as a crucial paradigm. This approach relies heavily on mechanical circulatory support (MCS) to maintain systemic perfusion and reduce the left-to-right shunt, thereby "bridging" the patient to a safer surgical window. The Intra-Aortic Balloon Pump (IABP) serves as the first-line modality in many centers, reducing left ventricular afterload and improving coronary perfusion. While the benefits of delayed surgery are well-documented in stable patients, successful bridging in patients recovering from cardiac arrest (SCAI Stage E) is less frequently reported and presents unique challenges regarding organ preservation and neurological recovery.[6]

Successful bridging in individuals recovering from cardiac arrest and SCAI Stage E shock remains rarely described. In this report, we present a case of post-MI VSR complicated by cardiac arrest and SCAI Stage E shock. We describe the successful utilization of an IABP-assisted "stabilize-then-delay" strategy, which allowed for a 12-day period of hemodynamic optimization and organ recovery prior to definitive surgical repair.

CASE

A 59-year-old male presented with a history of typical chest pain commencing on January 2, 2026. He was initially admitted to a regional hospital and diagnosed with an extensive anterior ST-elevation myocardial infarction (STEMI). On the fifth day post-onset, transthoracic echocardiography (TTE) revealed a VSR, prompting referral to the tertiary center for further management.





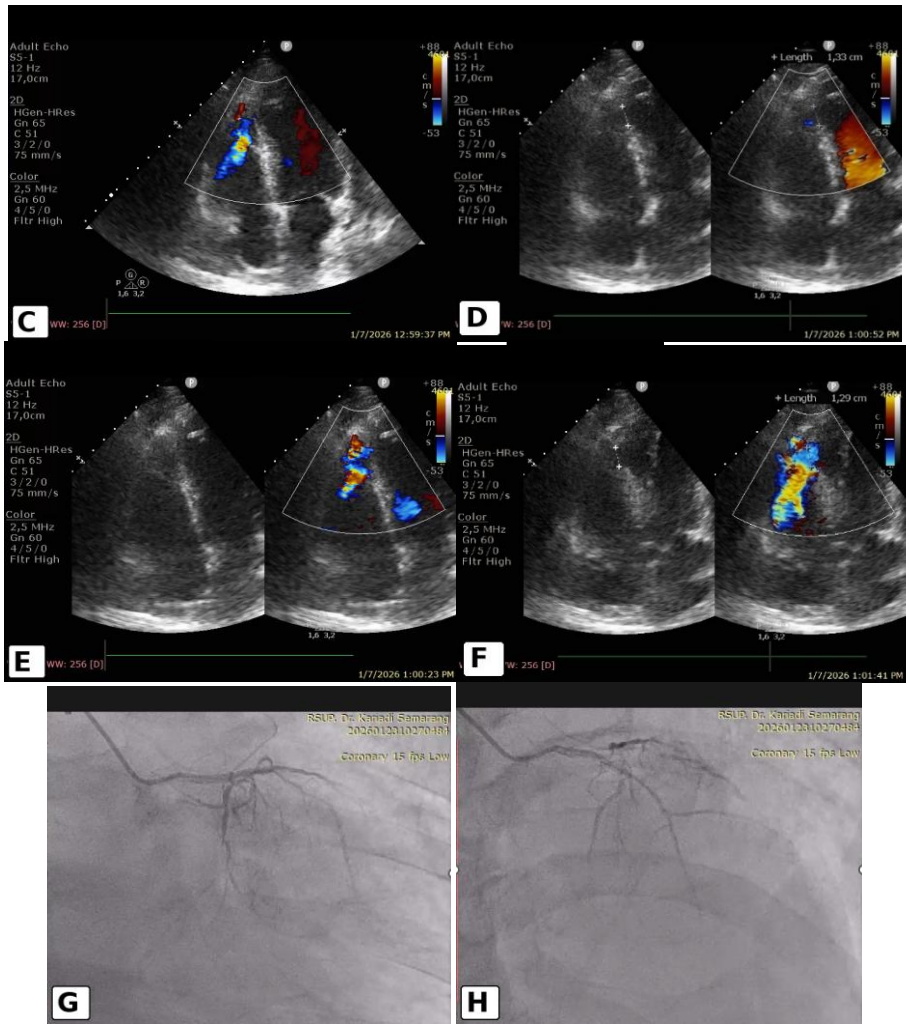


Figure 1. Admission electrocardiography and preoperative investigations. **(A)** Twelve-lead electrocardiogram demonstrating anterior ST-segment elevation in leads V1–V4. **(B)** Chest radiograph showing cardiomegaly and bilateral pulmonary opacities consistent with pulmonary congestion. **(C)** Color Doppler transthoracic echocardiography demonstrating flow across the apical interventricular septum. **(D)** Echocardiographic measurement of a septal defect of approximately 1.33 cm. **(E)** Additional color Doppler view of the left-to-right septal shunt. **(F)** Additional echocardiographic measurement of approximately 1.29 cm in another imaging plane. **(G–H)** Coronary angiographic views demonstrating severe (90%) mid-LAD stenosis with reduced distal flow; no other significant epicardial coronary stenosis was reported.

Upon admission (January 7, 2026), the patient was in a state of cardiogenic shock. TTE confirmed an apical VSR with a defect size of 13–17 mm and a left-to-right shunt (Qp:Qs 1.3), alongside an akinetic anterior wall and a left ventricular ejection fraction (LVEF) of approximately 52%. Initial laboratory findings indicated reduced kidney function (Creatinine 1.6 mg/dL, eGFR 44.5), leukocytosis (13,200/µL), and elevated liver enzymes (SGOT 532 U/L, SGPT 321 U/L). Chest X-ray showed cardiomegaly with signs of pulmonary edema and pneumonia.

On January 22, 2026, the patient's condition deteriorated precipitously into cardiac arrest (SCAI Stage E). Cardiopulmonary resuscitation (CPR) was performed for 30 minutes, achieving return of spontaneous circulation (ROSC). The patient was intubated and placed on mechanical ventilation. To support the failing hemodynamics, an IABP was urgently inserted on January 23, 2026.

Concurrent coronary angiography performed on January 23 revealed single-vessel disease with 90% stenosis in the mid-left anterior descending (LAD) artery, identified as the culprit vessel. The right coronary artery (RCA) and left circumflex (LCx) were normal. Following the implementation of the "stabilize-then-delay" strategy utilizing IABP support (1:1 ratio, 100% augmentation), the patient showed remarkable hemodynamic improvement. The IABP effectively reduced afterload and supported coronary perfusion, allowing for the weaning of vasoactive agents.

The patient was successfully extubated on January 27, 2026, five days after the cardiac arrest event. By February 2, 2026 (pre-operative assessment), significant organ recovery was observed. Serum creatinine normalized from 1.6 mg/dL to 0.9 mg/dL, and liver enzymes returned to normal levels (SGOT 23 U/L, SGPT 26 U/L). Leukocytosis resolved, and electrolyte imbalances were corrected. Twelve days post-cardiac arrest (February 3, 2026), the patient was cleared for definitive surgery. The pre-operative TTE showed a persistent apical VSR with haemodynamic status left ventricular outflow tract velocity time integral (LVOT VTI) 11.6 cm, Stroke Volume (SV) 35.8 ml, Cardiac Output (CO) 3.75 L/min, Inferior Vena Cava (IVC) 23/18 mm, systemic vascular resistance (SVR) 1237 dyne × sec/ cm^5, Qp:Qs of 1.42 and a mild circumferential pericardial effusion. Hemodynamics were stable on minimal inotropic support (norepinephrine 0.04 mcg/kg/min, dobutamine 2 mcg/kg/min) with IABP assistance, maintaining a mean arterial pressure (MAP) of 72 mmHg. The surgical plan consisted of VSR closure and coronary artery bypass grafting (CABG) to the LAD.

Upon arrival in the operating room, standard American Society of Anesthesiologists (ASA) monitoring was applied, including 5-lead ECG with continuous ST-segment analysis, pulse oximetry, and non-invasive blood pressure. Given the anticipated hemodynamic instability, invasive monitoring was established prior to induction consist of left radial arterial catheter for continuous blood pressure monitoring and serial arterial blood gas analysis; central venous catheter (CVC) at left subclavia vein cannulation with triple-lumen catheter because the right internal jugular vein had installed previously, introducer sheath placement followed by pulmonary artery catheter (PAC) insertion for advanced hemodynamic assessment (CVP, PAP, SvO_2, and cardiac output monitoring as indicated), and also core temperature monitoring.

Considering the recent myocardial infarction, VSR physiology, and prior cardiac arrest, a hemodynamically stable induction strategy was implemented. Preoxygenation with 100% oxygen was performed. Induction was carried out in a titrated manner using fentanyl 150 mcg, propofol 50 mg, and rocuronium 40 mg intravenously. Norepinephrine and dobutamine infusion were continued and titrated to maintain MAP ≥65–70 mmHg, aiming to prevent sudden decreases in SVR and excessive shunt augmentation. Endotracheal intubation was performed without complications. Mechanical

ventilation was adjusted to maintain normocapnia ($PaCO_2$ 35–40 mmHg) and moderate PEEP to avoid a significant reduction in preload. After hemodynamic stabilization, a transesophageal echocardiography (TEE) probe was supposed for intraoperative assessment, but intraoperative TEE was unavailable due to technical limitations.

In the pre-cardiopulmonary bypass phase, anesthesia was maintained with a low concentration volatile anesthetic combined with supplemental fentanyl dosing. Neuromuscular blockade was maintained as required. Systemic heparinization was performed before cannulation, targeting activated clotting time (ACT) >480 seconds.

Following aortic and venous cannulation, cardiopulmonary bypass was initiated. Mechanical ventilation was discontinued during full bypass, as was the IABP. Anesthesia was maintained using a volatile anesthetic delivered via the oxygenator. Moderate hypothermia was employed. Surgical procedures included patch closure of the VSR and single-vessel CABG to the affected coronary artery using the great saphenous vein.

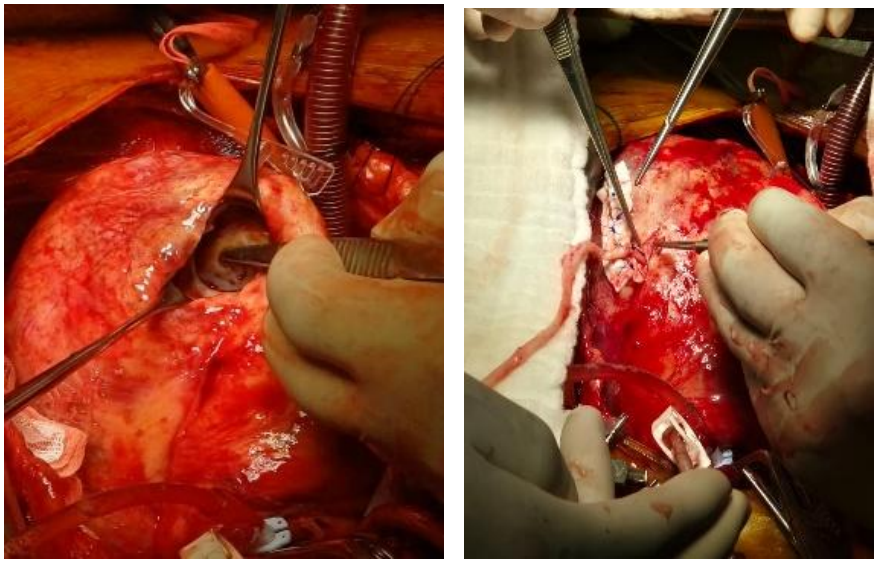


Figure 2. Closure of VSR and vein graft attachment.

During CPB, perfusionists carefully controlled hematocrit maintained above 22–24%, serial lactate monitoring, tight glycemic control, correction of electrolyte abnormalities, particularly potassium and ionized calcium. Rewarming to normothermia was achieved prior to separation from CPB. After 51 minutes, the aortic cross-clamp was removed, and the cardiac rhythm resumed spontaneously. Inotropic support was re-initiated in anticipation of post-ischemic myocardial stunning and possible low cardiac output syndrome using dobutamine 5 mcg/kg/min, and the patient was successfully weaned from

CPB with IABP re-assistance. CPB time took 63 minutes.

Protamine was administered gradually under close hemodynamic monitoring, then packed red cell (PRC) was administered about 242 ml, thrombocyte concentrate (TC) 137 ml, and fresh frozen plasma (FFP) 139 ml to secure hemostasis. Coagulation parameters were monitored with an ACT target near baseline. Ventilation was resumed with a lung-protective strategy, maintaining normocapnia and adequate oxygenation. Hemodynamics remained stable with low-to-moderate inotropic support. Urine output was adequate, core temperature normalized, and lactate levels showed a downward trend.

The patient was transferred to the intensive cardiac care unit (ICCU) intubated and sedated, with continuous arterial pressure monitoring, central venous access, ongoing inotropic support, and stable MAP >65 mmHg. The postoperative plan included gradual inotrope titration, ventilator weaning once hemodynamically stable, and serial echocardiographic evaluation to monitor ventricular function and surgical repair integrity. Intravenous paracetamol and continuous morphine were given intravenously for postoperative analgesia.



Figure 3. Patient's hemodynamic Hemodynamic Progression after CPB weaning.

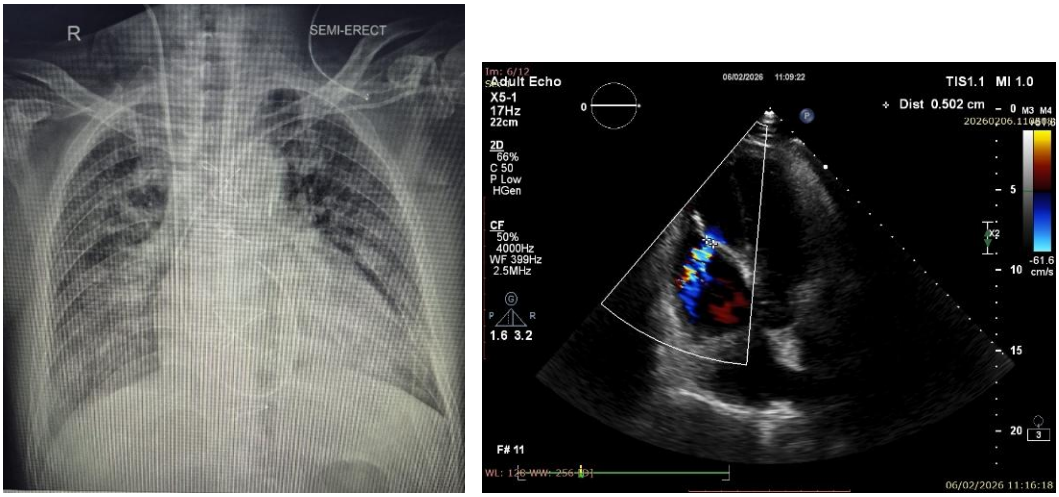


Figure 4. Post-operative Imaging.

In the ICCU, initial postoperative TTE demonstrated patch closure without a significant residual shunt. Hemodynamic measurements included LVOT VTI 15 cm, stroke volume 57 mL, cardiac output 5.2 L/min, IVC dimensions 19/14 mm, and SVR 1323 dyn·s·cm^{-5}. The patient was extubated approximately six hours after surgery. On postoperative day two, repeat TTE identified a small residual flow jet at the site of septal repair. The subsequent examination reported LVOT VTI 14.1 cm, stroke volume 57 mL, cardiac output 5.2 L/min, IVC dimensions 19/14 mm, and SVR 1323 dyn·s·cm^{-5}. In view of his clinical stability, the residual shunt was managed initially with medical optimization and echocardiographic surveillance.

Residual shunting was managed using a physiology-directed strategy focused on afterload optimization and comprehensive neurohormonal modulation. Reduction of systemic vascular resistance was prioritized to limit left-to-right shunt fraction and improve effective forward flow, achieved through carefully titrated vasodilator therapy while maintaining adequate coronary perfusion pressure.[7] Guideline-directed medical therapy was initiated and optimized, including renin–angiotensin–aldosterone system inhibition and mineralocorticoid receptor antagonism, with selective beta-adrenergic blockade introduced once hemodynamic stability was achieved. Volume status was meticulously controlled to prevent exacerbation of right ventricular volume overload and pulmonary congestion. Hemodynamic parameters and end-organ perfusion were closely monitored to guide gradual de-escalation of mechanical circulatory support. Serial echocardiographic evaluation was performed to assess shunt magnitude, ventricular function, and pulmonary pressures, enabling dynamic adjustment of therapy. This structured approach allowed clinical stabilization without immediate re-intervention while preserving the option for staged closure should the residual defect become hemodynamically significant.[8]

The patient was subsequently weaned from IABP support. Episodes of irregular tachycardia were treated with amiodarone, and he was ultimately transferred from the ICCU to the ward.

DISCUSSION

Post-infarction VSR is one of the most fatal mechanical complications of AMI, with operative mortality ranging from 42.9% to 80% in patients presenting with cardiogenic shock. The prognosis in these cases is exceptionally poor, particularly in patients classified as SCAI Stage E (Extremis), which is characterized by circulatory collapse and cardiac arrest, where survival has historically been very rare.[1,9] In this case, the patient presented with a highly severe condition consisting of an anterior STEMI, cardiac arrest, and multiorgan dysfunction. A critical condition in SCAI Stage E does not always lead to a poor outcome. Adequate, rapid, and precise hemodynamic resuscitation is the primary key to achieving a favorable outcome.[10]

The optimal timing of surgical VSR repair remains controversial. In the Society of Thoracic Surgeons National Database (n=2,876), observed operative mortality was 54.1% when repair occurred within seven days of myocardial infarction and 18.4% when repair occurred later. These observations must not be interpreted as proof that delaying surgery reduces mortality, because patients able to survive until later repair represent a selected population and the comparison is vulnerable to survivorship bias. Biologically, postponement may permit infarct tissue consolidation and improve suture retention, but ongoing shunting and abrupt deterioration may necessitate urgent intervention. Thus, contemporary consensus statements and practice guidelines support individualized treatment decisions based on hemodynamic stability, VSR anatomy, and availability of temporary circulatory support.[11,16,31]

Published reports illustrate the range of stabilization periods before VSR repair. Miyajima et al. performed surgery 21 days after myocardial infarction in a patient maintained in stable condition with IABP support. Baillot et al. reported repair after 19–27 days of IABP-assisted circulatory support in three patients, all of whom survived to discharge. Guo et al. described repair after 20 days of IABP support. Conversely, Morton et al. documented fatal recurrent rupture despite an approximately 10-day delay before surgery. These reports underscore that a fixed waiting interval cannot be recommended and that clinical stability during the bridge remains essential.[32, 33, 34, 35]

The selection of intra-aortic balloon pump (IABP) over more advanced mechanical circulatory support devices such as Impella or veno-arterial extracorporeal membrane oxygenation (VA-ECMO) in this case was guided by a combination of physiological appropriateness, resource availability, and observed clinical response. Although patients with Stage E cardiogenic shock (per SCAI classification) may benefit from more potent circulatory support, current evidence does not uniformly demonstrate a survival advantage of routine escalation to devices such as VA-ECMO or Impella, particularly in the context of post–myocardial infarction mechanical complications.[12] IABP, while providing more modest hemodynamic augmentation, offers effective afterload reduction and diastolic coronary perfusion support, which are physiologically advantageous in ventricular septal rupture by potentially reducing left-to-right shunt fraction and improving myocardial oxygen supply–demand balance.[13]

From a pragmatic standpoint, the availability of advanced devices such as Impella and VA-ECMO remains limited in many centers, and their use is associated with increased procedural complexity, bleeding risk, and vascular

complications.[12] In contrast, IABP is widely accessible, rapidly deployable, and associated with a more favorable safety profile. Importantly, in this patient, initiation of IABP resulted in a demonstrable improvement in hemodynamic stability, allowing successful bridging to definitive surgical repair. This positive response supports the concept of tailored escalation, where initial stabilization with a less invasive modality may be sufficient, reserving more aggressive support for refractory cases.[14]

Therefore, the use of IABP in this scenario reflects a rational, physiology-driven, and resource-conscious approach. Rather than representing a compromise, it underscores that in selected patients—including those with advanced shock—adequate stabilization can be achieved with IABP when combined with timely surgical intervention and close hemodynamic monitoring.[14,15]

In the present case, definitive repair was performed on February 3, 2026, 32 days after the reported onset of myocardial infarction symptoms on January 2, 12 days after cardiac arrest on January 22, and 11 days after IABP placement on January 23. The 12-day interval therefore refers specifically to the post-arrest stabilization period and should not be confused with the interval from infarction or first recognition of VSR. During this period, the patient underwent recovery of renal and hepatic function and improvement in clinical status. Although additional tissue maturation may have favored surgical repair, direct confirmation of septal fibrosis was not available. The decision to proceed was based on repeated assessment of stability and operative feasibility rather than a predetermined duration of IABP support.[16,31]

Percutaneous closure with an occluder device is an alternative to surgery in selected patients with post-infarction VSR, particularly when operative risk is prohibitive and imaging demonstrates favorable anatomy. Devices used have included muscular ventricular septal defect occluders, the dedicated Amplatzer post-infarction muscular VSD occluder (AMVO family), and, in selected off-label circumstances, atrial septal occluders (ASO). Important limitations include friable necrotic margins, insufficient apical rim, serpiginous or multiple defects, and proximity to valvular structures, each of which may compromise anchoring or lead to residual shunting and device-related complications. In this case, angiography confirmed a severe 90% mid-LAD lesion with distal underflow requiring consideration of coronary revascularization. Combined surgical VSR closure and LAD bypass grafting allowed treatment of both lesions during one operation. Although the apical defect measured approximately 13–17 mm on TTE, the available documentation did not establish that transcatheter closure was anatomically impossible; accordingly, surgery is described as the definitive strategy selected rather than the only technically feasible option.[16,31]

The IABP plays both a mechanical and metabolic role. Mechanically, counter-pulsation reduces left ventricular afterload, decreases the interventricular pressure gradient, and thereby reduces the left-to-right shunt fraction.[17] This unloading effect increases forward systemic flow while limiting pulmonary overcirculation. Further hemodynamic insight can be derived from the relatively low left-to-right shunt fraction observed in this case. The Qp:Qs ratio was initially 1.3 and increased modestly to 1.42 prior to surgical intervention, values that are comparatively low for post-infarction

ventricular septal rupture. This limited shunt magnitude likely reflects effective afterload reduction achieved with pharmacological medications and intra-aortic balloon pump (IABP) support, which decreases left ventricular systolic pressure and thereby attenuates transseptal flow. The controlled shunt fraction may have played a pivotal role in maintaining systemic perfusion and preventing rapid hemodynamic deterioration, allowing the patient to tolerate a 12-day delayed surgical strategy aimed at improved tissue integrity.[18] In contrast, patients with large shunt fractions (e.g., Qp:Qs >2.5) typically experience severe pulmonary overcirculation, right ventricular overload, and refractory cardiogenic shock, often necessitating urgent intervention and demonstrating limited tolerance to prolonged preoperative optimization even with mechanical circulatory support. Therefore, in this case, the combination of a relatively small shunt burden and effective IABP-mediated afterload modulation was likely a key determinant of preoperative stability.[19]

Improved systemic perfusion during mechanical support was accompanied by recovery of end-organ function. Serum creatinine decreased from 1.6 mg/dL at admission to 0.9 mg/dL at the February 2 preoperative assessment, while AST (SGOT) decreased from 532 to 23 U/L. End-organ dysfunction at the time of cardiac surgery can increase perioperative risk, making stabilization clinically relevant. In this patient, IABP support served as a bridge not only to definitive repair but also to observable renal and hepatic recovery.[20]

Post-MI VSR presents challenging hemodynamic issues. The magnitude of the left-to-right shunt is influenced by PVR, pulmonary vascular resistance (PVR), and biventricular function. An excessive reduction in PVR can exacerbate the shunt and pulmonary overcirculation, whereas excessive afterload can impair cardiac output due to decreased left ventricular (LV) function. Therefore, anesthetic management is focused on several aspects, such as maintaining balanced PVR, preserving coronary perfusion pressure, protecting right ventricular (RV) function, avoiding tachycardia, and preventing sudden preload fluctuations.[1,21]

Norepinephrine was titrated to maintain coronary perfusion pressure, followed by the addition of low-dose dobutamine when contractility was inadequate. Fluid administration was performed conservatively to avoid worsening pulmonary congestion. Induction required meticulous control, as a sudden drop in SVR could trigger a fatal hemodynamic collapse. A controlled induction strategy emphasizing hemodynamic stability and readiness for vasopressor escalation is crucial in a post-cardiac arrest myocardium with limited reserve.[21,22]

Although TEE is a highly valuable adjunct examination in structural heart surgery, intraoperative management in this case relied on physiologically driven monitoring. Hemodynamic assessment was guided by several factors: arterial waveform morphology, pulse pressure development during CPB (Cardiopulmonary Bypass) flow reduction, central venous pressure trends, and systemic perfusion indicators. Surgical assessment and meticulous de-airing techniques become increasingly important in the absence of real-time imaging.[23]

Weaning from CPB is the most critical intraoperative phase. Following VSR closure, sudden changes in ventricular loading conditions can unmask RV dysfunction that had previously adapted

to chronic volume overload. Furthermore, myocardial stunning and post-infarct LV dysfunction can limit forward flow. Gradual reduction of CPB flow with strict monitoring of pulsatility and systemic pressure stability is essential. Continuous IABP support combined with inotropic therapy facilitates ventricular unloading and myocardial recovery while avoiding excessive afterload stress.[24,25]

Postoperative evaluation revealed a residual shunt. Such findings are not uncommon due to the friable necrotic septal tissue and the ongoing remodeling process. Most importantly, a residual VSR does not always indicate surgical failure. Its clinical relevance in such conditions depends on several factors: the size of the shunt, RV tolerance, pulmonary vascular resistance, and global ventricular function.[23,26]

In SCAI Stage E survivors, the primary goal of the initial procedure is survival and hemodynamic stabilization, rather than anatomical perfection. The successful weaning of the IABP and early extubation in the patient suggest that the residual defect was hemodynamically tolerable, validating a survival-first strategy over immediate re-intervention. Early postoperative TTE is crucial for assessing defect size, shunt fraction, biventricular function, and pulmonary pressures. Clinical and hemodynamic integration allows for the differentiation between transient low cardiac output syndrome and shunt-mediated compromise.[27,28]

For small, well-tolerated residual defects, conservative management with optimal afterload reduction and guideline-directed medical therapy for ischemic cardiomyopathy is the appropriate course of action. Mechanical support can be weaned as systemic perfusion stabilizes. Moderate to large residual shunts associated with persistent instability may require staged re-intervention. Immediate reoperation on acutely infarcted myocardium carries an exceptionally high risk of mortality; furthermore, delayed surgical revision or transcatheter device closure following septal maturation may yield superior outcomes in selected patients.[21,23]

Long-term management in these cases focuses on optimizing heart failure therapy, serial echocardiographic surveillance, monitoring for RV dysfunction, and surveillance for pulmonary hypertension. The prognosis in this case depends on the residual ventricular function and the hemodynamic burden of the persistent shunt. Some small residual defects remain clinically stable, whereas larger ones can accelerate the progression of chronic heart failure. Coronary artery bypass grafting (CABG) performed concurrently addresses the underlying ischemic substrate of the septal rupture and may enhance long-term myocardial recovery. However, this combined procedure increases CPB duration and inflammatory burden, thereby emphasizing the importance of meticulous myocardial protection and structured postoperative care.[29,30]

CONCLUSION

In this single case of post-infarction VSR complicated by cardiac arrest and SCAI stage E shock, IABP-supported stabilization was followed by delayed surgical septal repair and concomitant LAD bypass grafting. The 12-day post-arrest stabilization interval was associated with improved end-organ function before surgery, although a small residual shunt developed postoperatively. This observation supports consideration of an individualized bridge-to-repair strategy only when adequate perfusion and close surveillance can be maintained.

The experience should not be generalized to all patients with severe cardiogenic shock: persistent instability, increasing shunt burden, or end-organ deterioration may require earlier intervention or escalation of circulatory support.

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