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Delayed Closure in the Successful Treatment of Postinfarction Ventricular Septal Rupture

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Abstract

Ventricular septal rupture (VSR) is a rare but highly lethal mechanical complication of acute myocardial infarction (AMI). Surgical repair is the gold standard treatment; conservative management is associated with an estimated 90% mortality within two months. We report the case of an 81-year-old male patient with post-infarction VSR who underwent percutaneous coronary intervention and was subsequently admitted to the cardiovascular intensive care unit for hemodynamic stabilization in preparation for delayed surgical closure. Twenty-one days after the event, the patient underwent successful VSR repair using a bovine pericardial patch, with favorable postoperative evolution.

Abbreviations: Ventricular Septal Rupture (VSR), Angiotensin II Receptor Antagonists (ARAs), Electrocardiogram (ECG), Left Anterior Descending Artery (LAD), Thrombolysis in Myocardial Infarction 3 (TIMI 3), Acute Myocardial Infarction (AMI), Ventricular Assist Devices (VADs).

Keywords: Post-infarction mechanical complications, Acute myocardial infarction, Ventricular septal rupture, Surgical repair of ventricular septal rupture.

Introduction

Ventricular septal rupture (VSR) is a rare yet highly lethal mechanical complication of acute myocardial infarction (AMI). Surgical intervention is considered the gold standard, although it carries a high mortality rate. Early surgical repair is generally recommended in cases of hemodynamic instability; however, the optimal timing for intervention remains undefined. Due to the low incidence of VSR, most data come from cohort studies or national registries.

Case Presentation

We report the case of an 81-year-old male referred for management of ST-segment elevation myocardial infarction (STEMI). His medical history included ten years of systemic arterial hypertension treated with angiotensin II receptor antagonists (ARAs); no other chronic-degenerative conditions were reported.

The current illness began one day prior to admission at 6:00 PM, with oppressive chest pain (10/10 on the visual analog scale), accompanied by vagal symptoms including diaphoresis and nausea. He presented to the General Hospital at 7:00 PM with the following vital signs: blood pressure 188/83 mmHg, heart rate 96 bpm, respiratory rate 22 breaths/min, oxygen saturation 96%. The initial ECG showed ST-segment elevation in leads V1–V4. He received aspirin, clopidogrel, atorvastatin, and enoxaparin. He was transferred to our institution under the infarct protocol, arriving 5 hours and 50 minutes after symptom onset, hemodynamically stable, with 5/10 anginal pain and no additional symptoms.

Repeat ECG confirmed an anterior infarct, and auscultation revealed a holosystolic murmur at the apex. Transthoracic echocardiography revealed a 5 mm apical VSR, left ventricular ejection fraction (LVEF) of 45%, and segmental wall motion abnormalities including septoapical, lateroapical, inferoapical, and anteroapical akinesis; inferoseptal hypokinesis and mid-anterior and mid-anteroseptal akinesis.

Coronary angiography revealed two-vessel disease: 99% subocclusive stenosis in the mid-left anterior descending (LAD) artery and a 75% focal lesion in the distal posterior descending artery. The LAD was treated with a 3x38 mm XIENCE Sierra stent, achieving final TIMI 3 flow. The right coronary artery was later treated with a 3x38 mm Resolute Onyx stent, also achieving TIMI 3 flow.

The patient was admitted to the cardiovascular intensive care unit. Follow-up echocardiography revealed a 15 mm interventricular septal defect with a left-to-right shunt and a pressure ratio of 3:1. A multidisciplinary surgical board accepted the patient for delayed surgical closure using a pericardial patch, planned for three weeks post-AMI.

On day 21 post-VSR, the patient developed hemodynamic deterioration consistent with cardiogenic shock and refractory heart failure. Vasopressors were initiated, and a hemodynamic assessment confirmed decreased peripheral vascular resistance, warranting urgent surgical intervention.

A median sternotomy was performed. After arterial and bicaval cannulation, total cardiopulmonary bypass and aortic cross-clamping were initiated. Right ventriculotomy was performed parallel to the interventricular septum, revealing a 3x2 cm defect (Figure 1). A bovine pericardial patch was placed (Figure 2) and secured with 4-0 prolene U-stitches with pledgets (Figure 3). A sandwich ventriculoplasty was completed using 2-0 prolene with pledgets (Figure 4). Intraoperative findings included grade III global cardiomegaly, macroscopic infarct on the anterior and inferior walls, and a fenestrated 3x2 cm apical VSR.

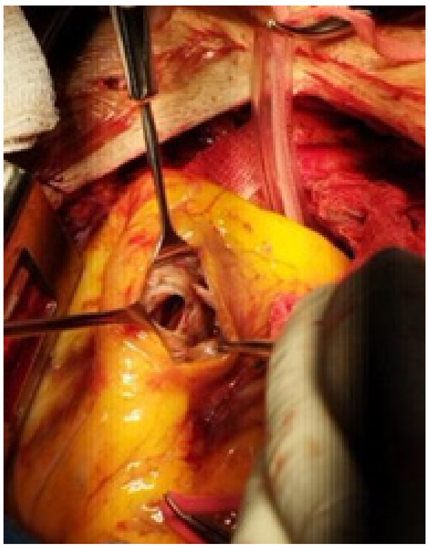

Figure 1: Defect in the interventricular septum

Figure 2: Placement of bovine pericardial patch, 3×2 cm defect

Figure 3: Closure with U-shaped sutures with pledgets

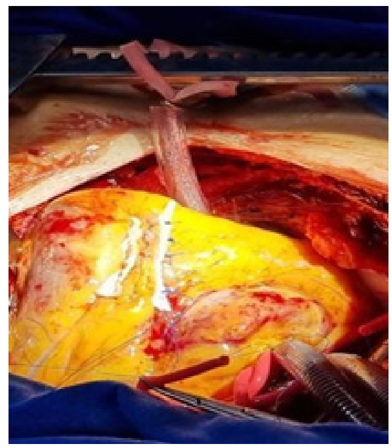

Figure 4: Ventricular repair using the sandwich technique with pledgets.

Postoperatively, the patient was admitted intubated and on vasoactive support. He was extubated at 24 hours and weaned off vasopressors at 48 hours. At 72 hours, he was transferred to the general ward hemodynamically stable, afebrile, without ventilatory or vasopressor support. Follow-up echocardiography showed a hemodynamically insignificant residual shunt, LVEF of 32%, and moderate myocardial damage due to ischemic cardiomyopathy. The patient had a favorable recovery and was discharged home without complications.

Discussion

VSR is the most common mechanical complication of AMI and is associated with extremely high mortality. It occurs in approximately 0.21% of STEMIs and 0.04% of non-STEMIs.

Risk factors for post-AMI VSR in the reperfusion era include advanced age, female sex, absence of smoking, hypertension, right ventricular infarction, and extensive myocardial infarction.

According to Elbadawi et al., using the National Inpatient Sample (NIS) database (2003–2015), among 13,767 patients with mechanical complications of AMI, 10,344 (75%) had VSR, while others developed acute mitral regurgitation due to papillary muscle rupture (19%) or free wall rupture (6%).

STEMI-related VSR is more likely to be associated with transmural infarction, a prerequisite for rupture. Most cases occur between days 2–6 after infarction, although onset can vary. The mean time to presentation was one day in the GUSTO-I trial, four days in the AHA mechanical complication registry, and 16 hours in the SHOCK trial.

The clinical course varies depending on infarct size, shunt severity, and associated right ventricular failure. The most critical determinant of outcome is the development of heart failure and cardiogenic shock, both related to the extent of necrosis and left-to-right shunting.

Surgical closure remains the definitive treatment. Repair involves excising necrotic tissue, patch closure of the defect, and revascularization. Without surgery, 90% of patients die within two months.

Arnaoutakis et al., using the Society of Thoracic Surgeons (STS) National Database (1999–2000), analyzed outcomes in 2,876 patients undergoing surgical repair for post-AMI VSR. Overall 30-day in-hospital mortality was 42.9%, decreasing significantly with delayed repair (54.1% mortality within 7 days vs. 18.4% beyond 7 days). Risk factors for operative mortality included age, female sex, shock, inferior infarct, intra-aortic balloon pump use, dialysis, mitral regurgitation, emergency surgery, and timing of repair. Mortality was highest (60%) among those operated within the first 24 hours.

The CAUTION international multicenter retrospective cohort (2001–2019) analyzed 475 patients undergoing VSR repair. Major findings: (1) early mortality was 40.4%; (2) age, preoperative cardiac arrest, percutaneous revascularization, and need for IABP or ECMO were independently associated with mortality; (3) longer intervals from AMI to VSR and from VSR to surgery were associated with improved survival; (4) recurrent VSR was not linked to increased mortality.

The optimal timing for definitive repair remains controversial. ACC/AHA guidelines recommend emergency surgery regardless of hemodynamic status. Individualized decision-making is necessary: early repair is favored in stable patients with favorable anatomy; delayed repair is preferred in those with complex anatomy or friable tissue.

Pathophysiologically, VSR results from transmural necrosis and subsequent tissue rupture. Classical rupture involves coagulative necrosis with neutrophil infiltration, weakening the septum over 3–5 days. Early rupture (<24h) may result from intramural hematoma dissection or hemorrhage in ischemic myocardium.

Early repair involves operating on friable myocardium, increasing the risk of extending the rupture and leading to higher mortality. Delayed repair allows collagen deposition (which peaks after day 7), tissue remodeling, and firm scar formation, providing better anchorage for sutures and reducing dehiscence risk.

Surgical technique typically begins with myocardial revascularization for optimal protection. The most widely used method today is the David technique, involving infarct exclusion with an endocardial patch.

Anterior VSRs are approached by parallel incision to the LAD. A pericardial or synthetic patch is sewn with U-stitches and pledgets to the non-infarcted right ventricular tissue, excluding the entire infarcted septum. True apical ruptures may be treated by amputating the apex and primary closure. Posterior VSRs are approached via posterior left ventricular wall ventriculotomy parallel to the posterior descending artery; a patch is sewn to the left ventricular face of the non-infarcted septum with primary closure, patch repair, or infarct exclusion depending on wall involvement.

When opting for delayed repair, VADs can bridge patients to surgery by reducing preload and afterload and improving coronary perfusion. Veno-arterial extracorporeal membrane oxygenation (VA-ECMO) offers additional benefits—avoiding sternotomy, providing oxygenation support, and being easily reversible—facilitating stabilization, organ protection, antiplatelet washout, and surgical planning.

In selected cases (<1.5 cm defects, subacute phase, poor surgical candidates), percutaneous closure devices may offer a minimally invasive alternative. However, in a series of 29 cases, 41% experienced device-related complications, and 30-day survival was only 35%, with higher mortality among those in cardiogenic shock.

Conclusion

Post-infarction VSR remains a severe condition with challenging medical and surgical management. Despite advances in infarct care and surgical techniques, early mortality rates have remained largely unchanged. Evidence suggests improved prognosis with delayed repair; however, further prospective studies are needed to optimize both surgical and preoperative strategies.

At our institution, based on literature and institutional experience, the management algorithm includes coronary care unit admission, medical optimization, ventricular support, and surgical repair after 14 days post-event. In unstable patients, mechanical circulatory support is considered, with surgical correction performed after 7 days, yielding favorable outcomes.

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Conflicts of interest

The authors declare no conflicts of interest.

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