CASE REPORT

Post Anterior STEMI Ventricular Septal Rupture: When is the Right Time to Perform Surgical Repair? A Case Report

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ABSTRACT

Ventricular septal rupture (VSR) after acute myocardial infarction (MI) is a rare yet fatal complication. Although surgical repair is essential, the optimal timing remains controversial. We report a case of ST-Elevation Myocardial Infarction (STEMI) complicated by VSR. Fibrinolytic therapy was initially considered successful; however, the patient developed worsening dyspnoea. Further evaluation confirmed an apical VSR by echocardiography. The patient received intensive monitoring and supported with an Intra-Aortic Balloon Pump (IABP). Surgical repair was performed on day 26. Hemodynamic initially improved postoperatively, but the condition deteriorated again on the ninth day after surgery, and the patient ultimately died. This case underscores that while surgical repair is the preferred definitive treatment for VSR, the timing of intervention is critical. Proper timing requires balancing surgical risks with tissue readiness and hemodynamic stability. The interval between VSR detection and surgical repair plays a pivotal role in determining patient survival.
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INTRODUCTION

Ventricular septal rupture (VSR) following acute myocardial infarction (AMI) remains deadly, despite its rare incidence. VSR occurred in approximately 1-2% of MI cases prior to reperfusion therapy, with a declining trend post-reperfusion availability (1). However, current VSR mortality rates remain high at around 80% and have been exhibiting little change over decades (2). Surgical repair is the primary treatment for VSR. Nevertheless, controversy exists regarding the optimal timing (3), where "early repair" may be defined as within the first week after diagnosis (often 24–72 hours) and "delayed repair" as beyond one to two weeks, each with distinct risks and benefits (4).

CASE REPORT

Patient Main Symptom and Medical History

A 63-year-old man was referred to the emergency room of Soetomo General Hospital due to VSR post-STEMI. He suffered from chest pain and dyspnea for seven days, with the VAS score was seven of ten. The cardiovascular risk factors of this patient were smoking and diabetes mellitus. In the previous hospital, the patient received thrombolysis therapy 9.5 hours after the chest pain onset, leading to a gradual decrease in chest pain as shown in Table I. However, the dyspnea remained and Fowler's position (semi-sitting) was preferred. After two days of intensive observation in the ICU, the shortness of breath worsened. The patient was then referred to Soetomo General Hospital.

Table I: Timeline of Patient Clinical Presentation

Day	Clinical events
Day 1	The patient was diagnosed with anterior wall STEMI and received thrombolysis 9.5 hours after the onset.
Day 2	Thrombolysis was considered successful due to resolution of chest pain and ST-segment changes in ECG. Meanwhile, the patient still suffered from mild dyspnea.
Day 6	The patient experienced worsening dyspnea. Physical examination revealed pansystolic murmur and the ECG shows new onset RBBB. The TTE confirmed the diagnosis of VSR.
Day 8	The patient was referred to Soetomo General Hospital. Initial management with vasopressor and inotropic. Coronary angiography revealed multiple vessel total occlusion.
Day 15	Hemodynamic instability occurred. Transthoracic Echocardiography (TTE) showed a decreased CO, CI, and LVOT VTI along with the increase of RAP.
Day 26	The patient received the surgical repair and CABG. During the induction, IABP was placed because of the hemodynamic instability. The patient also experienced VF twice; after the sternotomy and decannulation.
Day 27	Patient were monitored in the intensive care unit
Day 30	The patient passed away

Initial Assessment and Work Up

Physical Examination

The patient presented with intact alertness, with a blood pressure of 113/84 mmHg, respiratory rate of 32 x/minute, heart rate of 104 bpm supported by norepinephrine. Oxygen saturation was 100% with a non-rebreathing mask at 10 lpm. Jugular vein pressure was elevated. Chest auscultation revealed vesicular breath sounds with basal rales bilaterally along with a pansystolic murmur. Both extremities showed warm and dry peripheral perfusion.

Additional Examination

Initial electrocardiography (ECG) before the thrombolytic revealed ST elevation in several leads (Figure 1). After thrombolysis, it changed into qS pattern with an inverted T wave, suggesting recent anterior AMI (Figure 2). During sudden worsening symptoms, the ECG depicted a new onset of RBBB. Meanwhile, the chest X-Ray revealed cardiomegaly and pulmonary edema. Echocardiography examination confirmed a defect in the interventricular septum (IVS) at the apex with a L to R shunt (Figure 3). Angiography was performed and the result was triple vessel diseases.

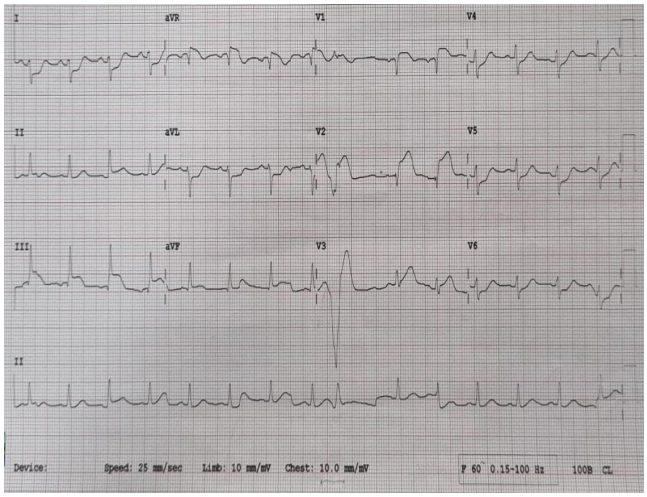

Figure 1: ECG Pre-Thrombolytic

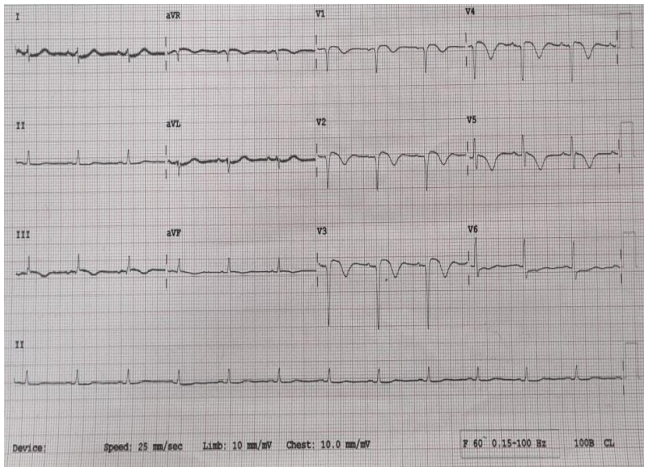

Figure 2: ECG Post-Thrombolytic

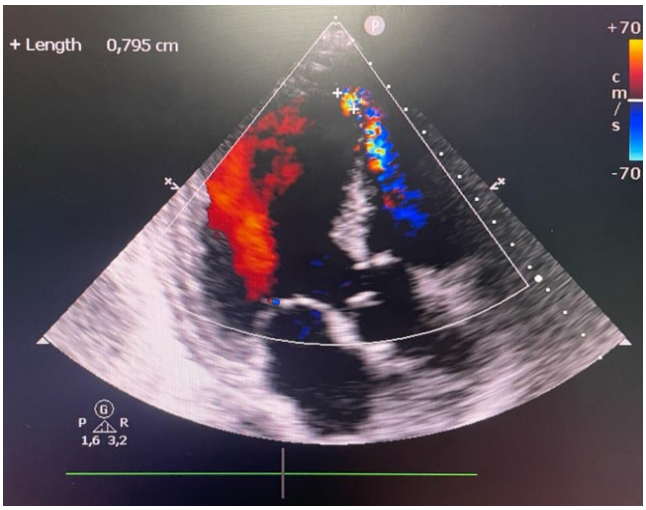

Figure 3: Transthoracic Echocardiography

Early Treatment

Upon arrival at the emergency room, the patient had already received oxygen supplementation 10 lpm via non-rebreathing mask, with infusion of norepinephrine and dobutamine via syringe pump. The patient's fluid intake was restricted to 1500 mL/day and the patient also received a furosemide infusion. Additionally, the patient received aspirin, clopidogrel, fondaparinux, and atorvastatin as acute myocardial infarction treatment.

Hemodynamic Monitoring

Initially, the patient presented with a high SVR, low CO, and low CI, requiring 200 nanograms/kg/minute of norepinephrine. The norepinephrine dose was then decreased, accompanied by the introduction of dobutamine. These adjustments resulted in a decrease in SVR, increase in CO, and an increase in CI, leading to sustained hemodynamic improvement. Following the intervention, during the anesthesia induction, the patient required the intra-aortic balloon pump (IABP) to provide additional hemodynamic support.

Surgical Management of VSR

Surgery was performed on day 26 after the STEMI onset. During the sternotomy, an extensive fibrosis scar on the anterior ventricular wall was found. Coronary arteries evaluation showed diffuse calcification at LAD, LCx, and RCA. Left ventriculostomy revealed multiple VSRs at the apex with each diameter of 1.0 cm and fibrotic edges. The patient underwent CABG with Saphenous Vein Graft (SVG) to the right posterolateral coronary (RPL) artery, sequential into distal LCx. Then, the VSR was repaired with a 0.6 mm polytetrafluoroethylene (PTFE) patch with 12 pledged sutures.

Outcome

After the successful surgical repair, the patient hemodynamic was initially improved. However, on the 5th day post-surgery, it deteriorated. The dose of norepinephrine was then increased, along with the administration of milrinone, adrenaline, and vasopressin.

The patient was also diagnosed with acute kidney injury, necessitating renal replacement therapy. The patient died on the 9th day post-surgery due to septic shock and multiple comorbidities.

DISCUSSION

Pathophysiology of VSR in AMI

About two-thirds of VSRs occur in the anterior septum, often causing apical defects, while inferior or lateral infarctions commonly cause basal defect. VSRs result from physical shear stress, leading to necrosis and subsequent rupture of the ventricular septum. This creates left-to-right shunting, increasing pulmonary blood flow, and causing cardiogenic shock (4,5). In this patient, significant risk factors—namely diabetes mellitus and a history of smoking—may have contributed to extensive coronary artery disease progression and tissue fragility, thereby amplifying the likelihood of mechanical complications such as VSR and influencing the final outcome (1).

Hemodynamic Management of VSR

Unpredictable hemodynamic deterioration occurs in many patients in days or weeks following the VSR (2). Therefore, initial hemodynamic management is essential. A fterload must be decreased by administering vasodilators to reduce the left-to-right shunt. Meanwhile, vasopressors may increase afterload thus worsening the shunt. Mechanical circulatory support (MCS) could also play an important role. IABP is commonly used for short-term support as it reduces afterload and shunt, as well as facilitates coronary perfusion (1).

Controversies in VSR repair

VSR patients should receive surgical repair or closure except in the cases where there is no evidence of cardiogenic shock, with optimal perfusion and cardiac output; minimal signs of congestive heart failure; minimal vasopressor use; and normal kidney function (5). The time interval between VSR detection and its repair is the most determining factor in the patient's survival, considering VSR maturation and hemodynamic adaptation. Generally, delayed elective VSR repair is more favorable for the hemodynamically stable patients, considering tissue fragility. On the other hand, patients with unstable hemodynamics, especially resistant to medical therapy, required immediate closure (2,3,5). Additionally, percutaneous closure devices such as Amplatzer, have been proposed in certain cases with smaller defects or high surgical risk (4).

In this case, surgery was performed on day 26, which falls into the delayed repair window, potentially allowing some degree of infarct healing. Despite a transient decline in hemodynamic status on day 15 of treatment, subsequent pharmacological optimization resulted in clinical improvement, thereby averting the necessity for immediate surgical intervention. Current evidence lacks consensus on a definitive "golden period" for VSR repair. In this case, delayed elective surgical repair is advised to facilitate scar tissue formation, which may enhance procedural feasibility and technical outcomes. However, the patient deteriorated postoperatively, possibly due to extensive myocardial damage, comorbidities, and secondary complications like septic shock. Earlier surgery might have been riskier given the fragility of the necrotic tissue, whereas waiting longer could have posed additional hemodynamic risks. In addition, this patient had received thrombolytic therapy 9.5 hours after symptom onset, which underscores the importance of intensive post-thrombolysis monitoring.

CONCLUSION

Surgical repair is superior to medical therapy for post-MI VSR, offering better outcomes and reliability compared to transcatheter repair with its associated complications. The timing of VSR repair significantly impacts patient survival, favoring delayed repair for better tissue readiness. However, immediate closure is required for patients with unstable hemodynamics, particularly those resistant to aggressive medical management.

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