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Case Report

Unveiling strategies in acute cardiac care for ventricular septal rupture following acute myocardial infarction: Lessons from cases

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ABSTRACT

Background: Ventricular septal rupture (VSR) following acute myocardial infarction (AMI) is drastically decreasing in the reperfusion era but mortality remains high. VSR correction is the definitive treatment and using mechanical support to delay closure is an attractive option despite data on success being limited.
Case Illustration: A 60-year-old man presented with late presentation of anterior STEMI complicating hemodynamic deterioration. Echocardiography showed apical VSR 11-14 mm L-R shunt. Patient was given adequate fluids, multiple inotropic agents, and IABP insertion, then a successful PPCI procedure was performed immediately. IABP was maintained for hemodynamic stabilization and patient was scheduled for interventional closure. Unfortunately, the patient worsened due to cardiogenic shock and passed away on the 5th day of admission.
In another case, a 61-year-old man came to our hospital also with a late presentation of anterior STEMI but stable in hemodynamics. Echocardiography showed apical VSR 9-11 mm L-R shunt. Coronary angiography showed CAD three vessel disease with critical stenosis at LAD. In hospital's heart team discussion, patient was planned to be performed VSR closure percutaneously and continue with PCI procedure. Both procedures were performed successfully. Patient was improved and discharged on 20th day of admission.
Conclusion: Rapid diagnosis and prompt treatment are the keys to optimal management of VSR complicating late presentation STEMI. Mechanical circulatory support and correction of VSR are required to optimize patient outcomes despite VSR is still a challenging case.

1. Introduction

Ventricular septal rupture (VSR) is a rare but potentially lethal mechanical complication that can occur after acute myocardial infarction (AMI). In the absence of reperfusion therapy, around 1-3% of patients with AMI will develop VSR. With the reperfusion treatment era, the occurrence of post-MI VSR has decreased to a range of 0.2–0.8%.[1,2] Usually, VSR becomes apparent within three to five days following myocardial infarction. Non-HLA gene polymorphisms have been shown to influence the immune response in viral infections such as dengue, and similarly, these polymorphisms may also impact the body's response to post-myocardial infarction complications like VSR.[3] Even with optimal medical treatment, the mortality rate for this mechanical complication still stands at approximately 40%.[4] Studies have shown that early intervention in managing severe conditions, such as cirrhosis or rheumatoid arthritis, significantly impacts mortality rates, a concept that is similarly applicable in the management of VSR. For example, research highlights the importance of timely prognostic interventions in patients with cirrhosis, which can help reduce mortality, a principle that aligns with the urgent management required for VSR. Additionally, early cardiovascular interventions have been shown to improve outcomes in patients with rheumatoid arthritis, emphasizing the need for rapid intervention in cases of VSR.[5,6]

The article presents two case studies of VSR, one with an unstable patient who required multiple inotropic drugs and intra-aortic balloon pump (IABP), and another with a stable patient who underwent successful VSR closure through a transcutaneous procedure. This report provides valuable information regarding the timing of occurrence, potential risks, clinical features, outcomes of VSR that complicated AMI, and acute cardiac care strategies.

2. Case Presentation

Case 1

A 60-year-old man who came to the emergency room (ER) Saiful Anwar Hospital Malang was referred from another hospital complaining of crescendo angina that began 4 days prior to admission, as well as shortness of breath and cold sweats. The risk factors are hypertension, diabetes mellitus type II, and active smoking. Physical examination showed shock condition with blood pressure 105/55 mmHg on inotropic drugs norepinephrine 0.5 mcg/kgbw/min and dobutamine 10 mcg/kgbw/min. A pansystolic murmurs were heard around the lower left sternal border (LLSB) without Carvallo's sign. The auscultation also heard bilateral rales indicating congestive sign. The ECG showed deep pathological Q wave with ST-elevation at segments V1-V4. The chest x-ray showed cardiomegaly with left ventricular hypertrophy (LVH) configuration with congestive pulmonum. The patient was diagnosed with late-onset ST-elevation myocardial infarction (STEMI) Killip IV. The patient was then performed endotracheal intubation for airway and breathing support and then transported to the catheterization lab for primary percutaneous coronary intervention (PCI) with the support of multiple inotropic drugs.

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Because hemodynamics was deteriorating, intra-aortic balloon pump (IABP) implantation was performed first. Primary PCI was performed via femoral access. Angiography showed two-vessel disease with total occlusion in the proximal left anterior descending (LAD) with no collateral vessels found. No complications were found when wiring and ballooning were carried out. A drug-eluting stent 3.0 mm x 25 mm was implanted in the proximal-mid LAD but only obtained a TIMI flow 2. The patient was transferred to the cardiovascular care unit (CVCU) for further management. The heparinization was continued for at least 5 days. Trans-thoracal Echocardiography (TTE) examination showed an ejection fraction (EF) of 29%, normal heart dimensions with LVH concentric, and an 11-14 mm VSR in the apical segment with a left-to-right shunt.

The patient's condition worsened further during post-primary PCI treatment in the CVCU. Cardiogenic shock and acute pulmonary edema occurred, necessitating the use of multiple inotropic medications with norepinephrine, dobutamine, and epinephrine support up to maximal doses. Patient was still with ventilator support and IABP support. The patient finally passed away on the fifth day of admission due to cardiogenic shock.

Case 2

A 61-year-old man who came to ER Saiful Anwar Hospital Malang also was referred from another hospital complaining of crescendo angina that began 2 weeks prior to admission. The risk factors are hypertension and active smoking. Patient had stable hemodynamics with BP 107/70 mmHg without support. A pansystolic murmurs were heard around the LLSB without carvallo's sign. Auscultation of pulmonary examination was within normal limits. The ECG showed deep pathological Q wave with ST-elevation at segments V1-V4 back to the baseline. The chest x-ray showed cardiomegaly with LVH configuration. The patient was diagnosed with late-onset STEMI Killip 1. TTE examination showed EF of 45%, normal heart dimensions with LVH eccentric, and a 13-18 mm VSR in the apical segment with a left-to-right shunt. Echocardiography showed sufficient rim to deploy a device Diagnostic coronary angiography showed critical stenosis at proximal LAD with TIMI Flow 3.

Heart team discussion with a multidisciplinary approach involved cardiologists, thoracic surgeons, and anesthesiologists. The discussion concluded that VSR closure will perform percutaneously and continue with PCI procedure with a backup from the thoracic surgical team. VSD occluder then was implanted well to VSR area. After that, the PCI procedure was performed. A drug-eluting stent 3.0 mm x 28 mm was implanted in the proximal-mid LAD with TIMI flow 3 results and no residual stenosis. Patient then followed up until 20 days of care. The patient was improved and discharged on 20th day of admission.

3. Discussion

VSR is a rare and severe complication of late-onset STEMI associated with high mortality rates. The incidence of VSR was 1–2% in the pre-thrombolysis era, with a subsequent decline following the introduction of thrombolytic therapy.[7,8] Anterior infarctions are more likely to cause apical defects, while inferior or lateral infarctions typically result in defects at the septal and posterior wall boundaries. This leads to the formation of a left-to-right shunt. The clinical presentation of this mechanism varies, including hemodynamic instability, infarction, ischemia, and right ventricular volume overload. Hemodynamic instability occurs in the majority of patients within a few days to weeks. Additionally, genetic factors, such as non-HLA gene polymorphisms, have been shown to influence the immune response in various medical conditions, including post-myocardial infarction complications like VSR.[9] The willingness to pay for medical interventions, including those for VSR, can be influenced by socioeconomic factors, as similarly observed in the acceptance of dengue vaccines.[10] This highlights the importance of considering socioeconomic determinants in designing effective healthcare interventions.[11] Furthermore, polymorphisms in ACE and bradykinin B2 receptor genes can affect how patients respond to ACE inhibitors, which are commonly prescribed for managing complications like VSR post-AMI.[12]

Table 1. Summary of Cases

Characteristics	Case 1	Case 2
Age (years)	60	61
Sex (M/F)	M	M
Risk Factors		
Hypertension	+	+
Diabetes	+	-
- Current Smoking	+	+
- History of CVA	-	-
Onset	4 days	14 days
Physical Examination		
BP/HR (mmHg/bpm)	80/60, 120	107/70, 65
Cardiogenic shock	+	-
Congestive Condition	+	-
ECG	ST elevation V1-V5	ST elevation V1-V4
	Deep Q wave V1-V5	Deep Q wave V1-V4
Infarct Location	Anterior	Anteroseptal
Hs Troponin	16689	120
Echocardiography		
- EF (%)	38	52
- VSR finding	Apicoseptal 11-14 mm	Apicoseptal 13-18
Inotropic drugs	Multiple	-
IABP Insertion	+	-
DCA	CAD 3VD+LM Disease	CAD 3VD
Culprit lesion	Acute total occlusion proximal LAD	Critical stenosis proximal LAD
Reperfusion therapy	Primary PCI	PCI
VSR Closure	-	Transcutaneous Closure
Concomitant disease	Pneumonia, Septic	Pneumonia
In-Hospital Mortality	Passed away at day 5	-

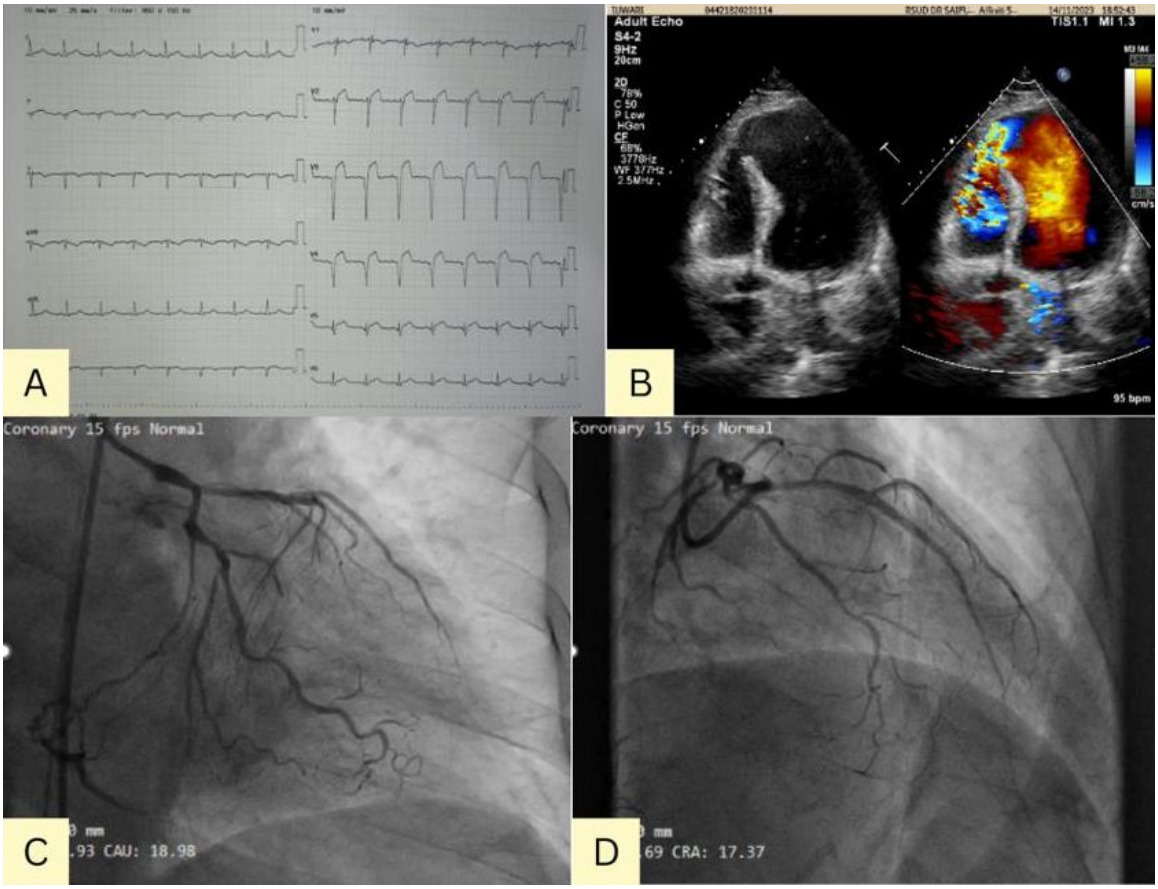

Figure 1. (A) ECG showed deep pathological Q wave with ST elevation at anteroseptal leads. (B) TTE showed apical VSR with a left-to-right shunt. (C) Angiography showed total occlusion at LAD. (D) Post-PCI angiography showed LAD flow improved. ECG: electrocardiography; TTE: trans-thoracal echocardiography; VSR: ventricular septal rupture; LAD: left anterior descending; PCI: percutaneous coronary intervention.

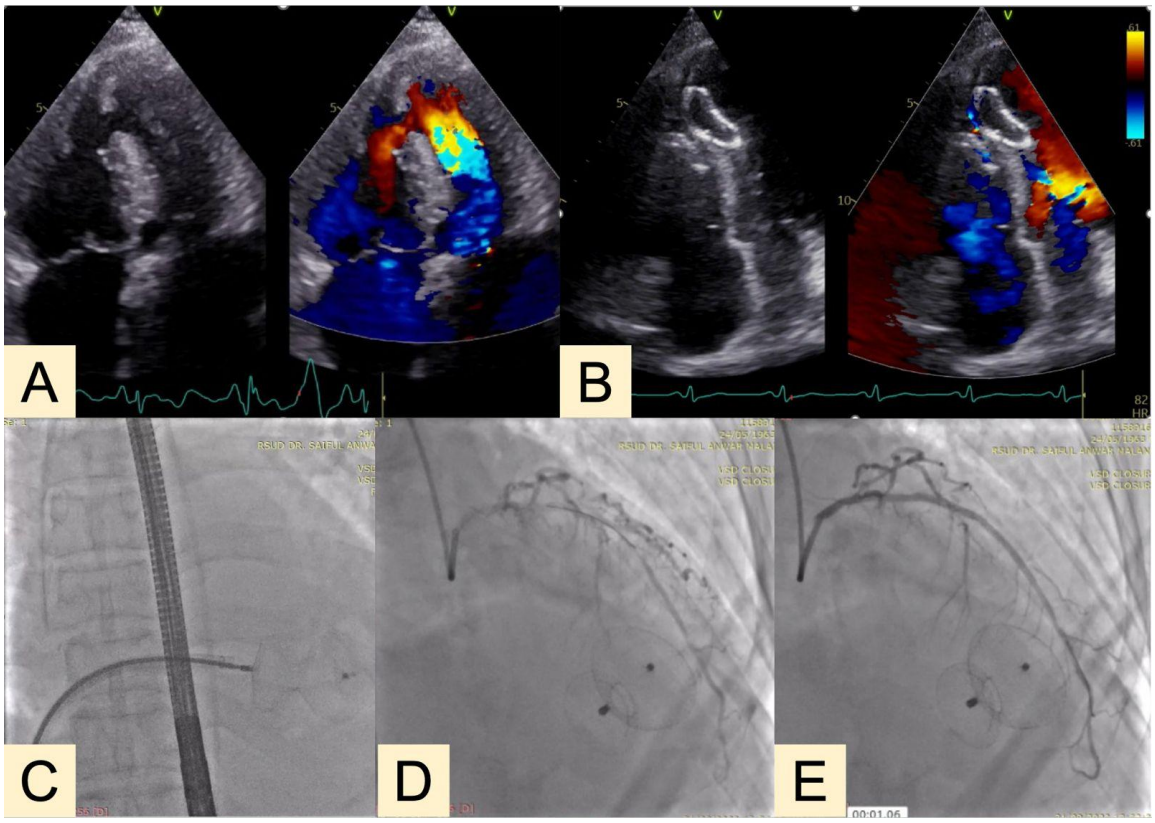

Figure 2. (A) TTE showed apical VSR with a left-to-right shunt. (B) Post-transcatheter VSR closure showed occluder in situ. (C) Fluoroscopy during transcatheter VSR closure. (D) PCI procedure on critical stenosis at LAD. (E) Post PCI showed stenotic lesions improved. TTE: trans-thoracal echocardiography; VSR: ventricular septal rupture; LAD: left anterior descending; PCI: percutaneous coronary intervention.

To enhance patient outcomes, it is essential to promptly diagnose and intervene surgically in cases of rapid deterioration during the acute phase of a VSR following a myocardial infarction. Echocardiography, a portable imaging technology, is highly sensitive and specific. It is effective for assessing the risk of VSR and determining the need for surgical or percutaneous correction of the septal rupture.

IABP can enhance coronary blood flow while simultaneously lowering ventricular wall stress and myocardial oxygen demand.[13] Additional mechanical support options, such as extracorporeal membrane oxygenation (ECMO) and percutaneous left ventricular assist devices (LVAD) like Impella, are also utilized, although empirical data on their efficacy remains limited. These mechanical support systems are anticipated to help maintain hemodynamic stability when the timing of surgical correction is strategically delayed.[14]

Although surgical repair is the definitive treatment for VSR, delaying closure with full mechanical support remains an attractive option despite limited data on its success. Percutaneous closure of the defect may also be a valuable therapeutic alternative.[15]

In the first case, VSR was suspected upon the patient's arrival at the emergency department, though it was only confirmed during treatment in the CVCU after primary PCI. The diagnosis of VSR was often established after primary PCI in patients with late-onset STEMI and cardiogenic shock (Killip IV), considering the necessity to achieve door-to-wire crossing time in this condition. Echocardiography evaluation showed a VSR with an 11-14 mm shunt from left to right and a reduction in the left ventricular ejection fraction to 29%, necessitating continuous hemodynamic monitoring and therapeutic adjustment. Optimal shock management is a priority following primary PCI, including the use of IABP and inotropic agents, alongside standard dual antiplatelet therapy and high-intensity statins for STEMI.[13,16] Anticoagulation therapy was also continued post-primary PCI due to low flow in the LAD coronary artery after stenting. The optimal management of VSR in STEMI was planned with a heart team discussion, considering surgical VSR closure as the primary option.

Surgical intervention is the definitive treatment; however, early postoperative mortality presents a significant challenge. Mortality rates varied based on the timing of surgery. Patients who underwent surgery within the first 7 days had a mortality rate of 54.1% compared to 18.4% if the surgery was delayed beyond 7 days.[17] Improved outcomes with delayed closure are related to changes in infarction, increased myocardial tissue stability, and potential biases, where early surgeries were performed in patients with hemodynamic instability and circulatory disturbances. Therefore, clinicians must weigh the benefits and risks for each patient and decide on the timing of surgery to avoid further clinical deterioration. VSR management involves various disciplines, but approaches may vary by individual. In the case discussed, the patient experienced deterioration due to cardiogenic shock, resulting in death on the fifth day.

In the second case, the patient presented with late-onset STEMI with an onset of 2 weeks and stable hemodynamics. According to ESC guidelines, late-onset STEMI with stable hemodynamics does not require immediate PCI. The patient, after a physical examination and TTE, exhibited mechanical complications of VSR. In this context, heart team discussions are crucial for finding the best multidisciplinary approach for optimal management. The heart team concluded that for a patient with stable hemodynamics, VSR located at the apical, a sufficient rim for device deployment, and a hospital with experienced interventional cardiologists, VSR closure by device followed by PCI was the appropriate strategy. The procedure was successful, and the patient improved and was discharged, continuing regular follow-up at the cardiology outpatient clinic.

4. Conclusion

VSR is a lethal mechanical complication of late-onset STEMI, characterized by its rarity and associated with high mortality and morbidity. Rapid diagnosis and prompt treatment are the keys to optimal management of VSR complicating late presentation STEMI. Immediate revascularization, using mechanical circulatory support, and correction of VSR are required to optimize patient outcomes.

5. Declaration

5.1 Ethics Approval and Consent to participate
Patient has provided written informed consent prior to involvement in the study.

5.2. Consent for publication
Not applicable.

5.3 Availibility of data and materials
Data used in our study were presented in the main text.

5.4 Competing interests
Not applicable.

5.5 Funding Source
Not applicable.

5.6 Authors contributions
Idea/concept: INA. Design: INA. Control/supervision: SA, IP, VY, AF, WK. Data collection/processing: INA. Analysis/interpretation: INA, SA, IP, VY, AF, WK. Literature review: INA, SA, IP, VY, AF, WK. Writing the article: INA. Critical review: SA, IP, VY, AF, WK. All authors have critically reviewed and approved the final draft and are possible for the content and similarity index of the manuscript.

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