



## Case Report

**A Patient with Acute Myocardial Infarction Who Experienced Mortality due to Ventricular Septal Rupture: A Case Report**Romi Ermawan<sup>1\*</sup><sup>1</sup>Medical Faculty of Mataram University, Mataram, Indonesia.

## ARTICLE INFO

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## ABSTRACT

Ventricular septal rupture (VSR) is one of the rare complications of acute myocardial infarction (AMI). Although the incidence decreased in percutaneous coronary intervention (PCI) era, the mortality rate remained extremely high. We report a case of an AMI patient who developed a post-fibrinolytic VSR, which was confirmed by echocardiography. Although rescue PCI had been performed, the clinical condition did not improve because he was also having coronary slow flow (CSF). Then he fell into cardiogenic shock and acute lung edema, and died. The main key to dealing with VSR is to reduce afterload so that the left-to-right bypass flow can be reduced in order to maintain the adequate LV stroke volume. In addition to pharmacological therapy, mechanical supportive therapy and correction of VSR both surgically or transcatheterly are required. However, AMI patients with VSR still have a poor prognosis even with the optimal treatment.

**Introduction**

Acute myocardial infarction (AMI) is still a major cause of morbidity and mortality worldwide. In patients with coronary artery disease, AMI occurs when an atherosclerotic plaque ruptures, forming a thrombus that partially or completely occludes a coronary artery, reducing blood flow to the myocardium <sup>[1]</sup>. Ventricular septal rupture (VSR) is a rare complication of AMI with a high mortality rate <sup>[2]</sup>. In the fibrinolytic era, the incidence of VSR in AMI patients reached 1 - 3%, while it fell to 0.2% in the percutaneous coronary intervention (PCI) era <sup>[3]</sup>. However, this situation has had no impact on the mortality rate, which has remained high over the

last decade and is estimated to be between 41 - 80% <sup>[4]</sup>.

VSR is a shortcut that occurs in the interventricular septum as a result of myocardial necrosis, resulting in flow from the left ventricle (LV) to the right ventricle (RV), causing volume overload in the RV and eventually causing hemodynamic instability, pump failure, and death <sup>[3]</sup>. This mechanical complication was more common in anterior wall AMI (60%) than in inferior wall AMI (40%). The VSR was more commonly found at the anterior wall's apex <sup>[1]</sup>. In this article we will present a complicated case of an AMI patient who died as a result of a VSR.

## Case Presentation

A 57-year-old man arrived at a hospital complaining of chest pain that began 2 hours prior, as well as severe cold sweats, nausea, and shortness of breath. The risk factor is hypertension in the absence of any medication. Physical examination revealed that the blood pressure was 150/90 mmHg, the heart rate was 110 bpm, the respiratory rate was 28 times per minute, and the axillary temperature was 36.6°C. Other physical examinations found no abnormalities. An extensive anterior acute myocardial infarction was discovered during the ECG examination (Figure 1). The patient was diagnosed with ACS STEMI - extensive anterior AMI and hypertension. Because he was not in the PCI-capable hospital, he received fibrinolytic therapy in the emergency room using Streptokinase before being transferred to the ICU. There were no abnormalities found on the chest x-ray, but laboratory results revealed total cholesterol 210 mg/dL, triglycerides 359 mg/dL, HDL-C 35.7 mg/dL, LDL-C 135 mg/dL, and uric acid 10.6 mg/dL.

Despite the administration of 12.5 mcg transdermal Fentanyl and repeated intravenous injections of Morphine, the patient still felt heaviness in his chest during ICU treatment. The hemodynamics were relatively stable, but a new holosystolic murmurs were heard around the left parasternal - 4th intercostal space, indicating a new ventricular septal rupture (VSR), which was discovered 20 hours after the STEMI. Furthermore, the patient was transferred to a PCI-capable hospital for rescue percutaneous coronary intervention (PCI) and advanced intensive care.

On the third day of treatment following STEMI in the cardio-vascular care unit (CVCU) of PCI-capable hospital, the patient's condition was deteriorating, blood pressure fell to 95/55 mmHg, heart rate increased to 120 bpm, respiratory rate was 32 times

per minute, and Sp.O<sub>2</sub> was 93 - 95% (by nasal cannula 4 L/min). The patient underwent echocardiography examination, which revealed the ejection fraction (EF) by biplane of 29%, normal heart dimensions (LVIDd 4.3 cm), apical ballooning in the LV, pseudonormal diastolic LV function, TAPSE 2.2 cm, akinetic in the anteroseptal and anterior segments, cardiac index 1.91 L/min.m<sup>2</sup>, estimated PCWP 19.75 mmHg, estimated RAP 20 mmHg, and discovered a 6 mm wide VSR in the apical segment of interventricular septum with a left-to-right shunt. The patient was then transported to the cathlab for rescue-PCI with the support of a Norepinephrine pump 0.1 mcg/kg/min.

Rescue-PCI was performed via radial access and resulted in single vessel disease with total occlusion in the proximal LAD with no collateral vessels found. Direct stenting method was used to place a 3.5 mm x 25 mm DES in the proximal LAD, but only obtained a TIMI flow 1 (Figure 2). The procedure was then followed by intracoronary flushing with a saline-heparin solution and the additional administration of 1,000 units of heparin intravenously. The patient was then transferred to the CVCU for further intensive treatments. Because the flow of the distal stent area was still sub-optimal, the Enoxaparin 2 x 0.6 cc was administered subcutaneously and planned to be continued for at least 5 days.

The patient's condition deteriorated further during the post-rescue-PCI treatment at the CVCU. Cardiogenic shock and acute lung edema developed (Killip 4), and the patient required Norepinephrine support up to 0.3 mcg/kg/min and Dobutamine pump up to 10 mcg/kg/min. A 15 L/min non-rebreathing mask oxygen supplementation had also been used on the patient, and a central venous catheter had been inserted. The patient finally died of asystole on the fourth day of post-STEMI treatment before being placed on a mechanical

ventilator. The ECG was documented on the monitor prior to the patient's death as a new complete right bundle branch block (RBBB).

## Discussion

VSR is a complication that can develop following a transmural infarction and can affect any segment of the interventricular septum [5]. According to the SHOCK trial registry, the median interval from AMI to VSR was 16 hours. There are two types of VSR: simple and complex. The straight bypass flow between the LV and RV occurs at the same point between the two cavities in the simple type. In anterior wall AMI, the simple type is more common. While the complex type has a tortuous bypass flow and a path that spreads far from the origin of the infarct, it is more common in inferior wall AMI and is accompanied by bleeding and disruption of myocardial tissue [1].

Age > 65 years, women, chronic renal disease, and never having an AMI before are all risk factors for VSR. Although VSR is more common in anterior wall AMI, if it involves the inferior wall, the prognosis is much worse because it is associated with RV dysfunction and biventricular failure. On cardiac auscultation, the VSR bypass flow produces a loud, harsh new systolic murmur. Bed-side echocardiography is required to confirm the presence of a VSR and to describe the size, borders, and velocity of blood flow through the VSR, as well as to describe ventricular function to guide appropriate treatments [3]. Echocardiography has a sensitivity of around 40%, and when combined with the Doppler ultrasound feature, the sensitivity can reach nearly 100%, making it an appropriate diagnostic modality for determining VSR after AMI [4].

In this case, a 6 mm VSR with onset 20 hours after STEMI was discovered and confirmed by Doppler ultrasound echocardiography as a simple left-to-

right shunt type. These findings were consistent with clinical anterior wall AMI. The profile of LV systolic function, which was characterized by a decrease in ejection fraction and cardiac index, and abnormal diastolic function, which began to deteriorate with pseudonormal LV and an increase in PCWP, indicated the risk of further hemodynamic disturbances. The LV was dilated and akinetic, with an apical ballooning appearance, indicating that the infarcted area was the most fragile site.

According to the GUSTO-I study, the most common cause of VSR was occlusion of the left anterior descending (LAD) artery. The study also concluded that patients who underwent surgical correction had a higher survival rate than those who only received pharmacological therapy (47% versus 94%) [4]. Within hours to days after VSR, patients may experience unpredicted hemodynamic disturbances. Several multicenter studies, including the Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries trial and the SHOCK registry, reported mortality rates of 94% and 96%, respectively [5].

Initially, fibrinolytic therapy had been administered to the patient, but due to prolonged chestpain symptom, persistent T-segment elevation on the ECG, and no reperfusion arrhythmias during fibrinolysis, it was concluded that fibrinolytics had failed. With a finding of persistent total occlusion in the LAD, the patient underwent a rescue PCI in less than 24 hours, and a stent had been implanted in the LAD. Interestingly, the distal flow after procedure was only TIMI flow 1, revealed that despite successful mechanical reperfusion, coronary flow remained low. The side branches of the LAD vessels appeared to have been occluded, and the surrounding myocardium appeared to have been severely destroyed. As a result, coronary wash out was disrupted, resulting in the retention of dye in the epicardial coronary vessels. This is

known as coronary slow flow, and it predicts a poor PCI outcome.

Coronary slow flow (CSF) is defined by delayed dye opacity filling the coronary arteries in the absence of epicardial coronary vessel stenosis [6]. According to several studies, the prevalence of CSF in patients undergoing elective PCI were 2 - 3.2%, while it rose to 30 - 40% in patients undergoing primary PCI. The presence of CSF was correlated to an increased risk of death and a poor prognosis. Endothelial dysfunction, microvascular ischemia and edema, distal embolization, and reperfusion injury are possible underlying mechanisms, but the etiology is complex and not fully understood [7].

The patient in this case had a poor outcome, which even resulted in death. The selection of fibrinolytic therapy might contribute to suboptimal outcomes and even provoked VSR. Becker and colleagues conducted a meta-analysis on the survival benefits of fibrinolytic therapy, which had also a controversial side effect. Based on their large registry of 350,000 AMI patients, it was concluded that fibrinolytic therapy increases the incidence of cardiac wall rupture, particularly in the first 24 - 48 hours. They also pointed that the bleeding complications were a possible cause of the rupture, and recommended further research on these findings [2].

The main key to dealing with VSR is to reduce afterload so that the left-to-right bypass flow can be reduced in order to maintain the adequate LV stroke volume. In addition to pharmacological therapy, mechanical supportive therapy and correction of VSR are required, both surgically or by using transcatheter [8]. If the systolic blood pressure remains higher than 90 mmHg, vasodilator therapy such as nitroglycerine or nitroprusside, should be initiated as soon as possible. Inotropes may be required to maintain cardiac output. These

treatments are critical in managing hemodynamic stability until the patient is ready for further correction. If pharmacological therapy is ineffective, intra-aortic balloon pump (IABP) can be used [1].

IABP has the ability to increase coronary flow while decreasing ventricular wall stress and oxygen demand. Other mechanical supportive therapies, such as extracorporeal membrane oxygenation (ECMO) and percutaneous left ventricular assist device (LVAD) like Impella, can also be used, though there isn't much data. When surgical correction is postponed until the correct time, mechanical supportive therapy is expected to be able to maintain hemodynamic stability [5]. However, a meta-analysis conducted by Matteuci and colleagues regarding mechanical supportive therapy in the form of insertion of an intra-aortic balloon pump (IABP) showed interesting results. The odds mortality of patients with preoperative or perioperative IABP were higher than those without IABP (OR = 3.48; 95% CI, 3.01-4.02;  $P < .001$ ;  $I^2 = 0\%$ ), with a mortality rate of 46.5% (1,633 of 3,515 patients) compared to 22.4% (352 of 1,570 patients) [5].

According to the GUSTO - I study, when surgical correction was performed, patients who had their surgery delayed fared better than those who had surgery within the first 7 days (mortality rate 18.4% versus 54.1%) [4]. The American College of Cardiology (ACC) guidelines recommend performing surgery as soon as possible regardless of the patient's hemodynamic condition, whereas the European Society of Cardiology (ESC) guidelines recommend deferring surgery if the patient responds to medical therapy [8]. However, regardless of the best correction time, it is still unable to reduce the early postoperative mortality rate (19 - 66%) [2]. The precise timing of surgical correction is still a point of contention today. In fact, a meta-analysis conducted by Shafiei and

colleagues revealed that regardless of the time chosen for VSR correction, the mortality rate remained high (Table 1) <sup>[1]</sup>. The longer correction interval had been linked to improved survival, which was associated with the evolution of more stable infarcted tissue, whereas infarcted myocardium in the acute phase was still fragile and thus difficult to suture by surgeons, carrying a higher risk of tearing and postoperative residual shunt <sup>[5]</sup>. It appears that the best time for surgical correction is after the necrotic tissue has recovered from fibrosis, whereas histologically, connective tissue proliferation begins in the third week after AMI <sup>[9]</sup>.

Percutaneous VSR closure can be used as an alternative treatment to surgery. Schlotter and colleagues's meta-analysis revealed that the percutaneous procedure could be performed with a high success rate, but the mortality rate remained high, particularly in patients with cardiogenic shock. The overall mortality rate of percutaneous VSR closure was nearly identical to that of surgical VSR repair, which ranged from 36 - 81% <sup>[10]</sup>.

In this case, the patient's treatment was limited to fibrinolytic therapy and rescue PCI. Mechanical supportive therapy such as IABP, as well as VSR correction both transcatheter or surgically, were not feasible due to limited facilities and human resources. So, if the patient's condition worsened, there wasn't much that can be done. The new RBBB pointed to an acute RV failure due to VSR. However, according to all data presented previously, even if optimal treatment is administered in accordance with the existing literatures, AMI patients with VSR still have a poor prognosis.

## Conclusion

In this paper, we present the case of an AMI patient who developed VSR complications and died as a result. VSR complications have poor outcomes and a high mortality rate, especially if the treatment is

sub-optimal. However, the optimal treatment, including mechanical supportive therapy, and surgical or transcatheter VSR correction, should be pursued because there is still hope of lowering morbidity and mortality.

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