# Conservative Management for Anterior STEMI Complicated by Ventricular Septal Rupture

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## ABSTRACT

Ventricular septal rupture is a rare complication of acute myocardial infarction but with a very high mortality, most often caused by severe hemodynamic failure. Conservative treatment is very inefficient with over 90% mortality rate; surgery is recommended as definitive treatment. A case of 67-year-old woman with chest pain since 1 day accompanied by shortness of breath and diaphoresis. Cardiac auscultation showed a 3/6 systolic murmur without thrill. ST elevation at the anterior lead was found in ECG. Echocardiography detected a rupture in interventricular septal with left-to-right shunt. The patient was diagnosed with anterior ST elevation myocardial infarction (STEMI) complicated by ventricular septal rupture. A conservative management was given without intra-aortic balloon pump placement and surgical operation due to patient's rejection. One month post treatment, the patient exhibited a relatively stable hemodynamic with moderate to severe activity limitation.

**Keywords:** Conservative management, hemodynamic pertubation, myocardial infarction, ventricular septal rupture

## ABSTRAK

Ruptur septum ventrikel merupakan komplikasi infark miokard akut yang jarang, namun mortalitasnya sangat tinggi, paling sering karena kegagalan hemodinamik berat. Penanganan konservatif sangat tidak efisien dengan tingkat kematian lebih dari 90 %; intervensi bedah direkomendasikan menjadi tatalaksana definitif. Seorang wanita 67 tahun mengeluh nyeri dada sejak 1 hari disertai sesak nafas dan diaforesis. Auskultasi jantung menunjukkan bising sistolik 3/6 tanpa *thrill*. EKG mendapatkan elevasi segmen ST di sadapan anterior. Pada ekokardiografi ditemukan ruptur septum ventrikel dengan *left-to-right shunt*. Pasien didiagnosis infark miokard akut elevasi ST anterior dengan ruptur septum ventrikel. Manajemen konservatif tanpa pemasangan *intra-aortic balloon pump* ataupun tindakan operatif karena pasien menolak. Satu bulan pasca-perawatan, hemodinamik relatif stabil dengan keterbatasan aktivitas sedang-berat. **Nanda Eka Sri Sejati, Habibie Arifianto, Irnizarifka. Tatalaksana Konservatif untuk STEMI Anterior dengan Komplikasi Ruptur Septum Ventrikel**

**Kata kunci**: Hemodinamik, infark miokard, ruptur septum ventrikel, terapi konservatif

## BACKGROUND

Ventricular septal rupture (VSR) is a rare complication of acute myocardial infarction (AMI). Before the era of reperfusion therapy, the incidence of VSR ranged from ~ 1-2% and decreased to ~ 0.17- 0,31% after reperfusion therapy.[1,2] The mortality rate due to these complication is still very high. Moreyra, et al,[3] through data study of the Myocardial Infarction Data Acquired System (MIDAS) reported that patients with AMI complicated by VSR had an increased risk of hospital mortality 7 times greater than without VSR. During 18 years observation, in-hospital mortality rate of this complication tends to be stationary at 50%. Deaths in the first 30 days reported from *SHould we emergently revascularize Occluded Coronaries in cardiogenic shocK* (SHOCK) and *Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries* (GUSTO-I) trial was 87% and 74%, respectively.[4,5] Meanwhile, Poulsen, et al, reported that overall mortality at 30 days, 1 year, and 5 years consecutively were 62%, 72%, and 95%. Without surgical intervention, the mortality rate at 30 days increases to 100%, while with surgery was 39%.[6] The highest cause of death from AMI complicated by VSR was cardiogenic shock, early-onset VSR, and low left ventricular function. Other causes include multiple organ failure, ventricular arrhythmia and cerebral infarction.[7]

Most studies reported consistently that older woman is an independent risk factor for VSR.[3-5] Other factors also known to increase the incidence of VSR are history of heart failure and chronic kidney disease; history of diabetes mellitus (DM) and hypertension is less associated with the incidence of VSR.[2]

## CASE

A 67-year-old woman presented with severe chest pain since 1 day ago. This complaint arose after the patient experienced an event that provoked severe heart palpitation 2 days ago. The complaint was accompanied by shortness of breath, body weakness, trembling, cold sweat, nausea, and vomiting. History of similar complaints, heart disease, DM, and hypertension was denied.

The patient was conscious with blood pressure 100/70 mmHg and pulse 78 times/min. The

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3/6 systolic murmurs was found without thrill. The laboratory tests showed an increase in leukocytes, neutrophils, urea, creatinine, and cardiac enzyme (**Table 1**).

ECG examination showed an ST elevation at the anterior leads (**Figure 1**). A rupture of interventricular septal followed with left-to-right shunt was also found during echocardiography (**Figure 2A**). The parasternal long axis and apical chamber view detected mild tricuspid regurgitation, pulmonary regurgitation, pulmonary hypertension, and left ventricular aneurysm (**Figure 2B**) with left ventricular ejection fraction (LVEF) 26,9 % (Simpson method).

The diagnosis was anterior STEMI complicated by VSR. The patient was treated with routine antiplatelet, intravenous heparin, ACE inhibitor, beta blocker, statin, and furosemide.

## DISCUSSION

VSR is a condition due to perforation of the interventricular septum thus forming a shunt between the two ventricles. The main etiology of this condition is myocardial infarction extending to transmural/full-thickness infarction. Both anterior and inferior infarct has a relatively similar frequency to cause VSR.[3] In GUSTO-1 study; VSR is more common in patients with anterior infarct (70%) than inferior (29%). Blockages is most commonly found in the left anterior descending artery (64%) and the right coronary artery (28%).[5]

The pathogenic process of rupture changes over time. Coagulation of the necrotic site is just begining during the first 24 hours. The process of rupture commences from neutrophils invasion into the infarct tissues. The neutrophil will subsequently undergo apoptosis and release lytic enzyme.[1] This enzyme causes disintegration of the necrotic myocardial tissues. Early rupture occurs in infarct with large intramural hematoma that dissect into tissues. Fibrotic tissue remodeling will happen after several weeks if the patients remain alive.[1]

Rupture generally occurs in the first week after infarction and rarely over than 2 weeks.[1,8] The study showed the incidence of VSR follow a bimodal distribution, which is higher in the first 24 hours as well as the third to fifth day after infarction.[1] Based on the SHOCK study, there was a difference in the onset of VSR between patients with and without thrombolysis. The median time of VSR in patients who received thrombolysis was 13 hours, while without thrombolysis was 6 hours. [4]

**Table 1.** The laboratory profiles

| Test | Result | Unit |
|---|---|---|
| Leukocyte | 15,03 | $10^3$/µl |
| Eritrocyte | 3,82 | milion/µl |
| Hemoglobin | 10,2 | g/dL |
| Trombocyte | 220 | $10^3$/µL |
| Lymphocyte | 13,6 | % |
| Monocyte | 6,1 | % |
| Neutrophil | 80,1 | % |
| Eosinophil | 0,1 | % |
| Basophil | 0,1 | % |
| Blood Glucose | 144 | mg/dL |
| Ureum | 96 | mg/dL |
| Creatinine | 3,30 | mg/dL |
| Hs Troponin I | 8550,2 | ng/L |

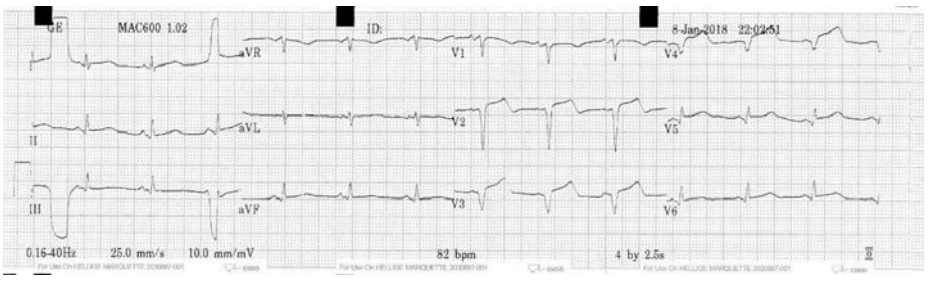


**Figure 1.** ECG of the patient showed qs wave and ST elevation in $V_1$-$V_4$

Most VSR will result in severe hemodynamic perturbation. Rupture causes left-to-right shunt accompanied by volume overload of the right ventricle (RV), increased pulmonary blood flow, and secondary volume overload of the left atrium and left ventricle. The ongoing process will cause the left ventricular systolic function to decrease. The body will perform negative feedback by increasing systemic vascular resistance through peripheral vasoconstriction.[1] However, this mechanism actually causes the shunt flow becomes stronger. Over time, the left ventricle may experience pump failure so the systolic pressure will decrease.[1]

In the SHOCK, MIDAS and global registry of acute coronary events (GRACE) trials, the risks of heart rupture include old age, female sex, stroke history, congestive heart failure, and history of chronic kidney disease. In contrast, patients with a history of DM, hypertension, hyperlipidemia, infarction, and smoking were lower in cardiac rupture incidence.[4,5,9] DM and hypertension become a major risk factor for myocardial infarction, however in the context of cardiac rupture, DM and hypertension play a reverse role. In case of hypertension, myocardial concentric hypertrophy is thought to be a protective factor against rupture. While collateral circulation resulted from angiopathy in DM provide an alternative blood supply to the ischemic septum, thus preventing rupture.[3]

The patient is a 67 year-old woman without history of DM and hypertension. The onset of infarction was about 36 hours. The patient did not undergo reperfusion therapy because the time is beyond the advisable period according to ACCF/AHA 2013 recommendation.[10]

The main principle of conservative management of VSR is afterload reduction to achieve effective LV stroke volume by reducing left-to-right shunt pharmalogically or mechanically. Sodium nitroprusside usually used as pharmalogical approach due to its beneficial effect in afterload reduction by its flexibility to be titrated or emergently discharged. Mechanical afterload reduction can be achieved with IABP placement.[2] IABP is proven to be effective in afterload reduction and cardiac output augmentation. Clinical and survival rate improvement are seen in IABP group compare with non IABP. Its instatallaion

is beneficial even in hemodynamic stable patient to minimize unpredicted fatal complication.[11]

During observation, the patient showed a relatively stable hemodynamic. The patient had LVEF of 26.9% with a septal perforation diameter of 15.6 mm and left ventricular aneurysm dimension of 42.1 x 47.2 mm . According to Noguchi, et al, the majority of non-survivors of VSR patients had an average ejection fraction of 25.6 ± 6.7%. The average perforated septum diameter performed by operative management is 21 ± 8 mm. As a comparison, Su, et a,l[8] presented their cases of VSR with EF > 40 % and perforated septal diameter of ± 9.5 mm. The two of the three subjects die in the first 48 h even the data show better parameter than our case, indicating the survival rate of VSR patient influenced by other existing factors that contribute to hemodynamic stability.

A survival rate of patients with VSR without surgical intervention is very low. Nevertheless, few cases had ever been reported to survive for more than 5 years. Kamishirado, et al,[12] reported a case of a man with VSR who was able to survive for up to 6 years with conservative therapy. When autopsy performed, aneurysm was found with a diameter of 25 x 30 mm. Ventricular aneurysm is considered to be a factor that may contribute to nonlethal hemodynamic in patients who do not undergo surgery. The flow and pressure characteristics of aneurysm contractility differ from the normal ventricle. This contractile pattern decreases the LVEF thus reducing the degree of left-to-right shunt.[12] Rothfled, et al,[13] reported a patient of AMI complicated by VSR who survived for up to 17 years due to high pulmonary artery resistance. The patient had Eisenmenger syndrome with severe pulmonary hypertension. The left-to-right shunt had undergone reversion. The patient only complains of tightness in moderate to severe activity. This patient is also known to suffer from mild pulmonary hypertension. However, these slight changes may reduce the RV overload, decreasing the left-to-right shunt, and minimizing exessive pulmonary blood flow; thus prevent rapid deterioration of hemodynamic even without support from IABP.

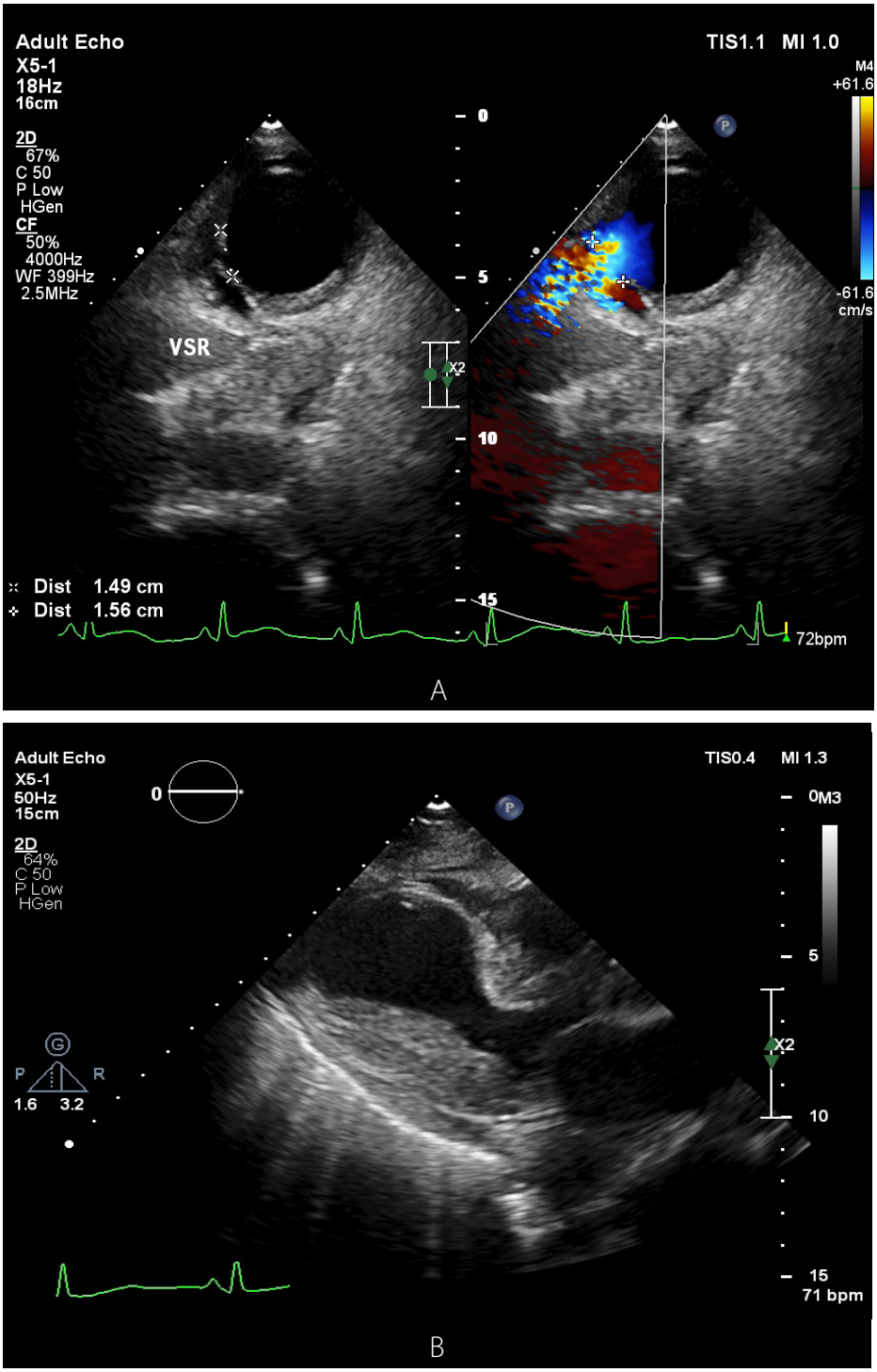


**Figure 2. A)** The echocardiography showed rupture of the interventricular septum with left-to right shunt flow **B)** Ballooning at the apical portion of the left ventricular wall with apical akinesis motion and hypokinetis septal base confirm the left ventricular aneurysm.

Systolic heart failure with pulmonary hypertension in our patient was likely a contributing factor for reduction of left-to-right shunt severity, thereby reducing lethal hemodynamic perturbation under VSR. According to Su, et al[8], the mortality of patients with rupture is strongly determined by the patient's specific conditions including age, cause, sex and comorbidities. Hemodynamic stability at the time of diagnosis of VSR may also become a predictor of patient survival. Our patient was relatively stable with conservative treatment without IABP, however we do not recommend to perform conservative management without IABP and




highly suggest definitive correction with surgical operation.

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