Case report

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ST-Elevation myocardial infarction patient with ventricular septal rupture complication: A Case Report

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Abstract

Ventricular septal rupture (VSR) is an uncommon but very significant mechanical complication of acute myocardial infarction (AMI), with typically severe hemodynamic effects. Until surgical closure of the defect and revascularization of the coronary bypass surgery graft (CABG), the patient at Wahidin Sudirohusodo Hospital with VSR reports sequelae of MI with stable hemodynamic condition. ***Clin Ter 2024; 175 (1):1-6 doi: 10.7417/CT.2024.5025***

Keywords: *ventricular septal rupture, acute myocardial infarction, coronary artery bypass graft*

Introduction

Ventricular septal rupture (VSR) is a rare but very significant mechanical complication of acute myocardial infarction (AMI) that often results in severe hemodynamic effects. Rapid detection and effective therapy of these problems are expected to increase patient survival. In some modern studies, the introduction of a reperfusion approach to the treatment of AMI, involving thrombosis and percutaneous coronary intervention, has reduced the incidence of VSR. Before reperfusion treatment, 1-3% of patients with AMI had VSR problems. In more recent studies, the incidence of VSR is decreasingly rare, affecting between 0.17 and 0.34% of patients with IMA.[1]

Despite the fact that the prevalence of VSR after IMA has decreased with the age of reperfusion, the mortality rate remains quite high. According to a study by Shi Tai et al., 52% of the patients died during the first two weeks after VSR post-IMA. The long-term prognosis of patients who survive 2 weeks and have effective percutaneous closure tends to improve.[2]

According to research from the Multinational Global Registry of Acute Coronary Events (GRACE), the death rate for patients with VSR with cardiogenic shock is 100%.3 The prognosis for conservatively treated VSR is quite bad and the fatality rate exceeds 80%.[1]

The following describes a patient at Wahidin Sudirohusodo Hospital with ventricular septal rupture due to complications of acute myocardial infarction and stable hemodynamic condition until surgery to repair the defect and revascularization with CABG.

Case reports

HBG, a 55-year-old man of Bugis ethnicity, was admitted to the hospital on 19 November 2021 after a cardiologist at Andi Sulthan Daeng Radja Bulukumba Hospital referred him with a diagnosis of STEMI extensive anterior wall and ventricular septal rupture. Currently, the patient continues to complain of chest pain that he has been experiencing for the last three days prior to hospitalization, pain that feels like pressure behind the sternum, pain lasting more than twenty minutes, pain radiating to the shoulder and left arm, and pain accompanied by cold sweat. Rest does not alleviate discomfort. There was no history of chest pain in the past. There is no shortness of breath. There was no fever and no nausea or vomiting. Good appetite. No complaints about defecation or urination. With a 30-year history of hypertension, the patient routinely takes 5 mg of amlodipine. Histories of smoking twelve cigarettes daily. There is no history of type 2 diabetes. Currently, the patient is receiving aspilet 80 mg, clopidogrel 75 mg, fondaparinux 2.5 mg subcutaneous, atorvastatin 40 mg, isosorbide dinitrate 5 mg sublingual, furosemide 2.5 mg / hour / syringe pump and dobutamine 7 mcg/KgBW/minute/ syringe pump.

Physical examination

On physical examination, he appeared to be very ill, with adequate nutrition and compos mentis. Blood pressure 100/70 mmHg with inotrope, pulse 84 beats per minute, respiration 18 beats per minute, axillary temperature 36.7 C, SpO_2 99.9% with nasal cannula 3 liters per minute, VAS

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3/10. Body mass 65 kg, height 165 cm, and BMI 23.9 kg/m^2. The conjunctiva and sclera are neither pale nor icteric. On examination of the neck DVS R+2 cm H2O, neither the lymph nodes nor the adenoids were enlarged. On chest examination, the chest appears symmetric, is not tender, and no tumor mass can be felt. Right and left vocal fremitus are identical and the bronchovesicular breath sounds have a fine bilateral rhonchi base. Regular heart sounds of S1 and S2 are accompanied by a grade 3/6 systolic murmur at the apex. The left heart border is located two fingers laterally to the left midclavicular line. Examination of the abdomen revealed no abnormalities. No abnormalities were observed in the extremities.

Electrocardiographic examination revealed sinus rhythm, heart rate 88 beats/minute, axis -90°, p wave 0.08 seconds, PR interval 0.16 seconds, QRS complex duration 0.06 seconds, R wave in the lead aVL + S wave in lead V3 > 28 mm, elevation of qST in leads V1-V6 with conclusion of sinus rhythm, heart rate 88 beats/minute, axis -90°, left atrial enlargement, left ventricular hypertrophy, extensive anterior wall.

From laboratory tests, Hs Troponin I (4555.1 ng/l) and other laboratory results were within normal parameters.

Table 1. Complete blood count (19/11/2021)

Parameter	Results	Unit	Reference
WBC	8,300	10^3/uL	4.00 – 11.00
HGB	14,7	g/dL	13.0 – 16.0
HCT	42	%	40.0 – 50.0
MCV	86	fL	80.0 – 100.0
MCH	30	Pg	27.0 – 34.0
MCHC	35	g/dL	31.0 – 36.0
PLT	387,000	10^3/uL	150 – 450
NEUT	73,1	%	50.0 – 70.0
LYMPH	19,2	%	20.0 – 40.0
MONO	6,7	%	2.00 – 8.00
EOSINOPHIL	1.0	%	1.00 – 3.00
BASOPHIL	0.0	%	0.00 – 0.10

Table 2. Blood chemistry (11/19/2021)

Parameter	Results	Unit	Reference
RBS	126	mg/dl	140
Urea	38	mg/dl	10-50
Creatinine	1.14	mg/dl	< 1.3
SGOT	26	U/L	< 38
SGPT	26	U/L	< 41
PT	11,4	second	10 – 14
INR	1.10		
APTT	21,9	second	22 - 30
Sodium	139	mmol/l	136 – 145
Potassium	4.0	mmol/l	3.5 – 5.1
Cloride	112	mmol/l	97 – 111

Table 3. Immunoserology (19/11/2021)

Parameter	Results	Unit	Reference
Hs Troponin I	**4555,1**	**ng/l**	**17-50**
HBsAg	Non Reactive	COIs	< 0.13 (Non Reactive)
Anti-HCV	Non Reactive	COIs	< 1.00 (Non Reactive)

Laboratory

At Andi Sulthan Daeng Radja Hospital, a chest X-ray revealed cardiomegaly with pulmonary congestion and bilateral pleural effusion. On MSCT Scan Thoracic examination without contrast at Wahidin Sudirohusodo Hospital of the Cardiac Center, left pneumonia, bilateral paratracheal and subcarina lymphadenopathy, cardiomegaly with signs of pulmonary congestion, atherosclerosis of the aortae and coronary branch, and bilateral pleural effusion resulting in collapse of the inferior lobes of both lungs were observed.

From the examination of the transthoracic echocardiography, the left ventricular systolic function decreased with the ejection fraction 38% (TEICH), while the right

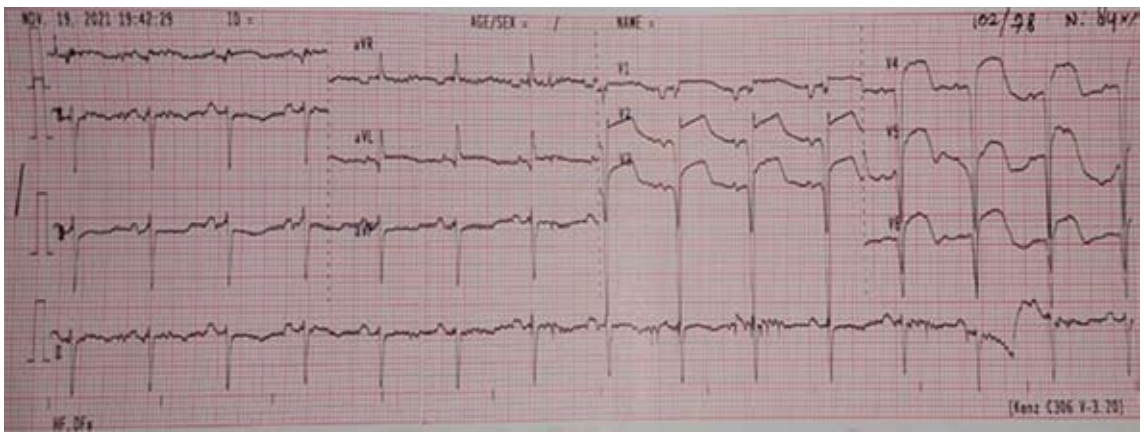

Fig. 1. Electrocardiogram in the emergency room of the wahidin sudirohusodo hospital (11/19/2021).

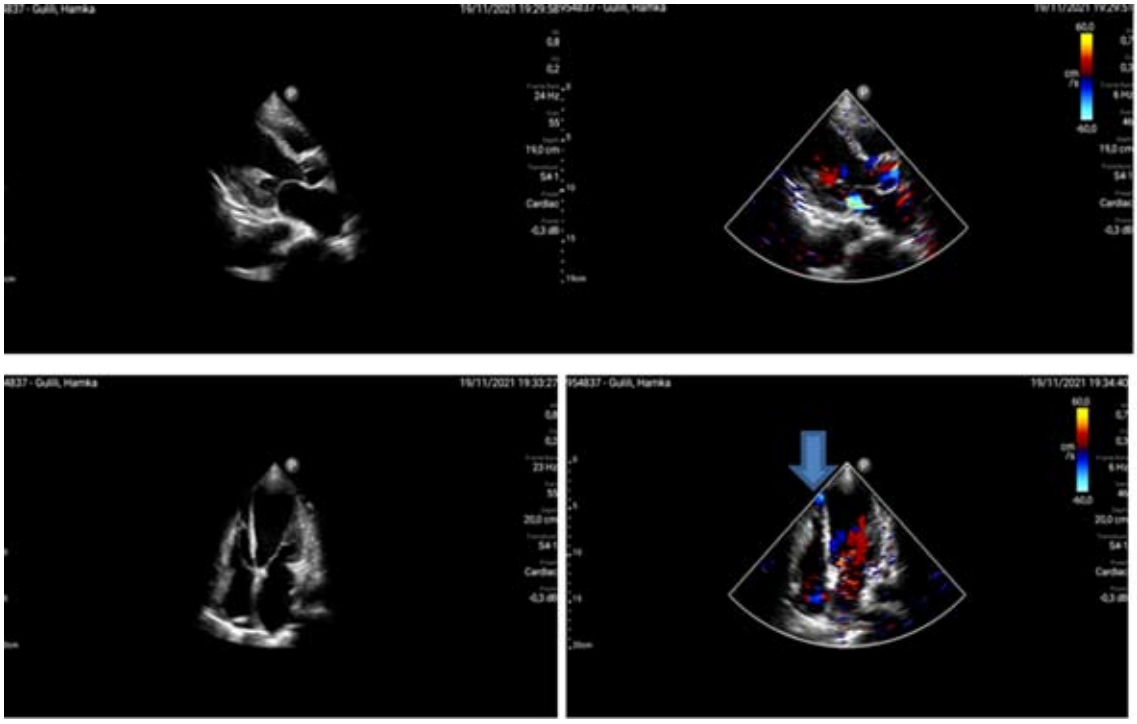

Fig. 2. Transthoracic echocardiography in the emergency room of Dr. Wahidin Sudirohusodo Hospital's Cardiac Center on November 19, 2021.

ventricular systolic function was normal with TAPSE 1.8 cm, mild mitral regurgitation, segmental akinetic and hypokinetic, intraventricular septal defect on the apical side suspected of rupture of the ventricular septum, the left atrium and left ventricle appear dilated, eRAP 3 (IVC Exp 1.4/IVC Insp 0.4).

ST Elevation Myocardial Infarction Extensive Anterior Wall Onset > 48 hours KILLIP IV (TIMI) Risk score 6; 16.1% risk of all-cause mortality at 30 days) and Ventricular septal rupture was diagnosed based on the patient's medical history, physical examination, and supporting examinations. As a treatment, the patient received an intravenous infusion of 0.9% sodium chloride 500 ml/24 hours, aspilet 80 mg/24 hours/oral, clopidogrel 75 mg/24 hours/oral, atorvastatin 40 mg/24 hours/oral, Dobutamine 5 mcg/KgBW/minute/syringe pump, fondaparinux 2.5 mg/24 hours/subcutaneous, and furosemide 5 mg/hour/syringe pump. The patient was scheduled for CVCU care, primary PCI strategy, a comprehensive echocardiographic examination, and cardiothoracic surgery consultation for coronary artery bypass graft (CABG) and ventricular septal rupture (VSR) closure.

On the second day of hospitalization in the emergency room, chest pain complaints were reported to have decreased. Subsequently, the patient was transferred to the CVCU. On the second day of hospitalization in the CVCU, the patient underwent a complete echocardiogram on 22 November 2021, which revealed a 0.4 cm intraventricular septal defect in the apical region with a left-to-right shunt. On the same day coronary angiography was performed, proximal total occlusion in left anterior descending disease was identified and it was determined that 1 vessel disease (VD) was determined. On the third day of hospitalization in the CVCU, the patient's hemodynamic status was stable enough to discontinue dobutamine. After 8 days in the CVCU, the patient was transferred to regular care.

The patient's condition remained stable between the ninth and sixteenth day of hospitalization. One week prior to scheduled surgery, dual antiplatelet therapy (DAPT) was discontinued. The patient's condition remained stable from the seventeenth to the twenty-fourth day of hospitalization.

On the 25th day of hospitalization or the 28th day after the appearance of myocardial infarction, the patient underwent coronary artery bypass graft (CABG) and closure of the ventricular septal rupture (VSR). The CVCU treated the patient after a successful operation. Throughout the CVCU, the patient did not experience hemodynamic abnormalities. Before being transferred to regular care, the patient was treated for four days in the CVCU. After the closure of the VSR, an echocardiography was scheduled for the patient. The patient was allowed to be discharged after 12 postoperative days.

Discussion

*The patient had a severe anterior STEMI and the infarct-related artery was probably the LAD. The septal branch, which supplies the anterior 2/3 of the septum, is one of the LAD's branches. The echocardiogram revealed an apical VSR. In this case, the culprit was most likely the proximal LAD.*The LAD and PDA branches provide blood flow to the septum. In rare cases, the LCX may be the source of blood flow. Infarcts are often widespread and transmural due to total obstruction of the coronary arteries that feed

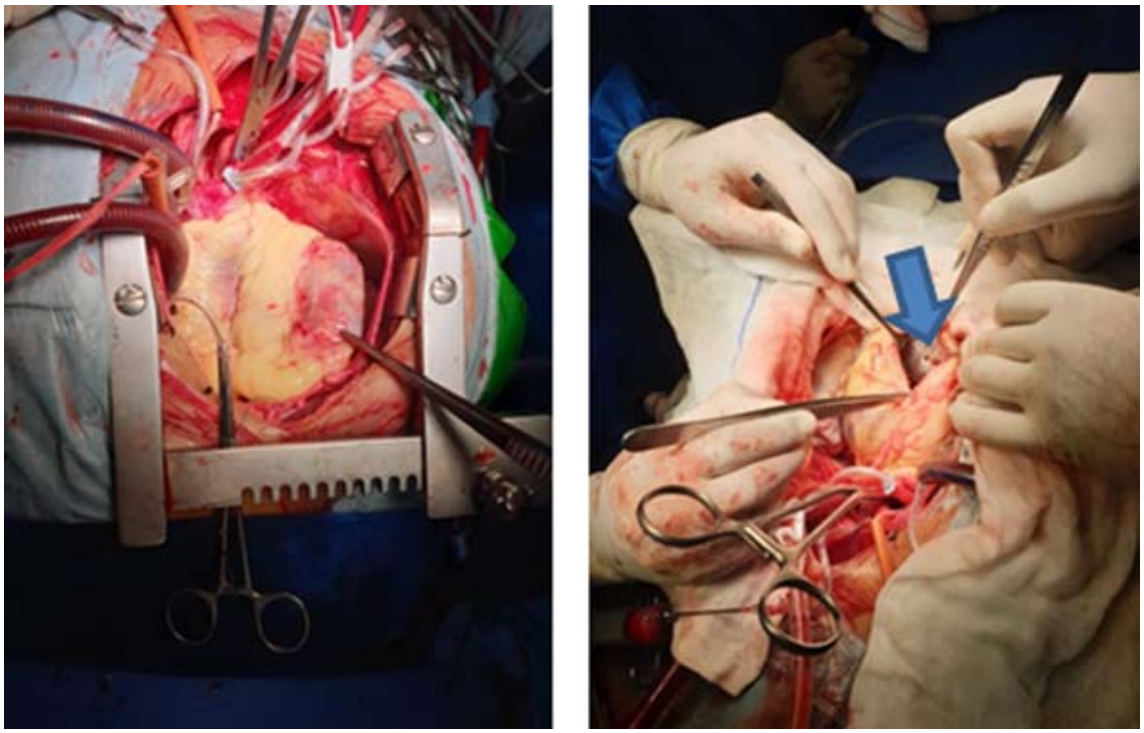

Fig. 3. Thoracotomy revealed a left ventricular aneurysm (left) and a 1 cm ventricular septal rupture (right).

that portion of the septum. Almost two-thirds of the VSR is located in the anterior septal wall, while approximately one-third is located in the inferior or posterior wall. During the autopsy, the offending coronary artery was almost completely blocked and did not have collaterals. Infarct-related arteries are often left anterior descending (LAD) (64%) and often completely blocked (55%). Rarely can several septal perforations develop.[4] VSR may also occur in conjunction with other mechanical problems, such as ventricular aneurysm, free wall rupture, or papillary muscle rupture.[5]

In this 55-year-old male patient, the presence of extensive anterior STEMI and low LV systolic function (EF 38%) increased the risk of VSR events. As a result of extensive myocardial ischemia and necrosis, systolic function is severely altered. According to pathological studies, when a large myocardial infarction occurs, a progressive necrotic process results in ventricular rupture. Transthoracic and Doppler echocardiography is crucial for diagnosing the size and hemodynamic impact of the VSR and for excluding other causes of hemodynamic instability. Using color Doppler, the VSR image on echocardiography reveals a disrupted ventricular septum with a left-to-right shunt. Transesophageal echocardiography may be useful if transthoracic echocardiography is difficult or if there is an inferior myocardial infarction when transthoracic echocardiography is planned. percutaneous closure. Occasionally, when undiagnosed hemodynamic instability occurs in the catheterization room, left ventriculography with evidence of a left-to-right shunt can help confirm the presence of VSR with the presence of contrast agent from the left ventricle to the right ventricle, while right heart catheterization with oximetry can detect an increase in oxygenation levels in the right ventricle.[6]

Our patient had a extensive anterior Killip IV and acute decompensated heart failure due to a STEMI with extensive anterior extension. This is consistent with the findings in the literature that VSR is more prevalent in acute myocardial infarctions affecting the anterior myocardial wall. Furthermore, the presence of cardiogenic shock (grade Killip IV) and heart failure in this patient can be considered independent risk factors for VSR. There are several independent risk factors for VSR in patients with acute myocardial infarction, including older age, female sex, a history of previous stroke, chronic kidney disease, and congestive heart failure. Patients with VSR are more likely to present with ST segment elevation, positive early cardiac biomarkers, cardiogenic shock, cardiac arrest, higher Killip class, and a longer time to initial balloon inflation or thrombolytic therapy. Interestingly, patients who experience VSR generally have a history of hypertension, diabetes, smoking, or a previous myocardial infarction. The incidence of VSR depends on the type of myocardial infarction (MI), with STEMI patients having a higher incidence rate of 0.9% compared to patients with non-ST-segment elevation myocardial infarction (NSTEMI) (0.17%) and unstable angina (0.25%).[7]

The patient has type II subacute VSR, which is characterized by erosion of the infarcted myocardium, neutrophil infiltration, and coagulative necrosis. The condition that results is a thinning and weakened septum. Although it is common within three to five days after onset of a myocardial infarction. According to the characteristics of the VSR defect, the patient had a simple rupture, which means that the defect occurred at the same level on both sides of the septum. Ventricular septal defect defects can be divided into two categories, simple and complex ruptures, based on the characteristics of the damage. In simple defects, the defect

is located at the same level on both sides of the septum, typically in the apical VSR. On the contrary, complex defects are frequently observed in inferior infarctions, which are characterized by the formation of a serpiginous route through a hemorrhagic and necrotic septum.[8,9]

On physical examination, a left-to-right shunt was detected as a systolic murmur in the LSB. On echocardiography, the appearance of a ruptured ventricular septum supports the diagnosis of VSR with a left-to-right shunt. Once the link between the left and right ventricles occurs through the VSR, oxygenated blood flows from the high-pressure left ventricle to the low-pressure right ventricle.[4] Therefore, there is a left-to-right shunt, volume, and pressure overload in the right ventricle. Relative systemic and pulmonary vascular resistance establishes the degree of shunt or shunt fraction (Qp/Qs).[1]

Hemodynamically, VSR will result in a left-to-right shunt, leading to volume overload and pressure in the right cardiac chambers, increased blood volume pumped to the lungs, and volume overload in the left heart. The size of the shunt is based on the VSR, systemic and pulmonary vascular resistance, and systolic function of the LV and RV. In individuals with massive anterior infarction, left ventricular systolic function decreases, causing a decrease in cardiac output that is balanced by an increase in systemic vascular resistance to maintain normal blood pressure. Increased systemic resistance will increase the degree of left-to-right shunt, which will subsequently increase volume overload in the right ventricle and eventually the left ventricle.[6]

Clinical manifestations include heart failure, cardiogenic shock, and, in rare cases, asymptomatic. The left-to-right shunt creates a loud systolic murmur that is often audible throughout the precordium and is strongest at the left sternal border (LSB). This murmur is sometimes accompanied by a thrill. However, in individuals with poor cardiac output and cardiogenic shock, thrills are uncommon and murmurs are difficult to detect due to decreased turbulent blood flow through the defect. Due to increased blood flow to the right ventricle, a loud pulmonary component (S2), tricuspid regurgitation, and an S3 heart sound are occasionally heard. During the beginning of the murmur, the patient may experience an abrupt change in hemodynamics.[6]

For the treatment of cardiogenic shock, this patient received an inotropic agent in the form of dobutamine at a dose of 5 mcg/KgBB/minute/syringe pump. The patient's hemodynamic condition improved until the third day of treatment, so inotropes were no longer administered the next day. Giving medical therapy to VSR can be considered, with the main goal of therapy being to reduce the afterload so that the left-to-right shunt can be reduced and the LV stroke volume increases. For medical treatment, the patient received low doses of dobutamine (≤5 μg/kg-1/min-1) with a stimulatory effect on β1 and β2. β1 stimulation of the heart will increase cardiac contractions with less chronotropic activity. In the vascular, β2 stimulation causes mild vasodilation, thus reducing the afterload.[10]

In cardiogenic shock, inotrope administration increases myocardial oxygen consumption, the risk of ventricular arrhythmias, and expansion of infarction, but hypotension alters myocardial perfusion, resulting in an increase in left ventricular end-diastolic pressure (LVEDP), an increase in myocardial oxygen demand, and a further decrease in coronary perfusion gradient. Thus, the hemodynamic benefits of inotropic therapy typically outweigh its risks when used as a bridge to further action.[10]

This patient underwent elective surgery 28 days after the onset of myocardial infarction. Based on the data, it was determined that patients who underwent elective VSR repair for more than 21 days had a low mortality rate of 10%. The presence of a relatively small defect size contributes to hemodynamic stability and reduces the risk of mortality. Calvert et al. reported increased mortality in elderly patients, women and those with larger defect, delayed revascularization, and more advanced disease severity (cardiogenic shock, inotrope use, and increased creatinine).[11]

The definitive treatment for post-infarction VSR is surgical closure. In the 2017 European Society of Cardiology

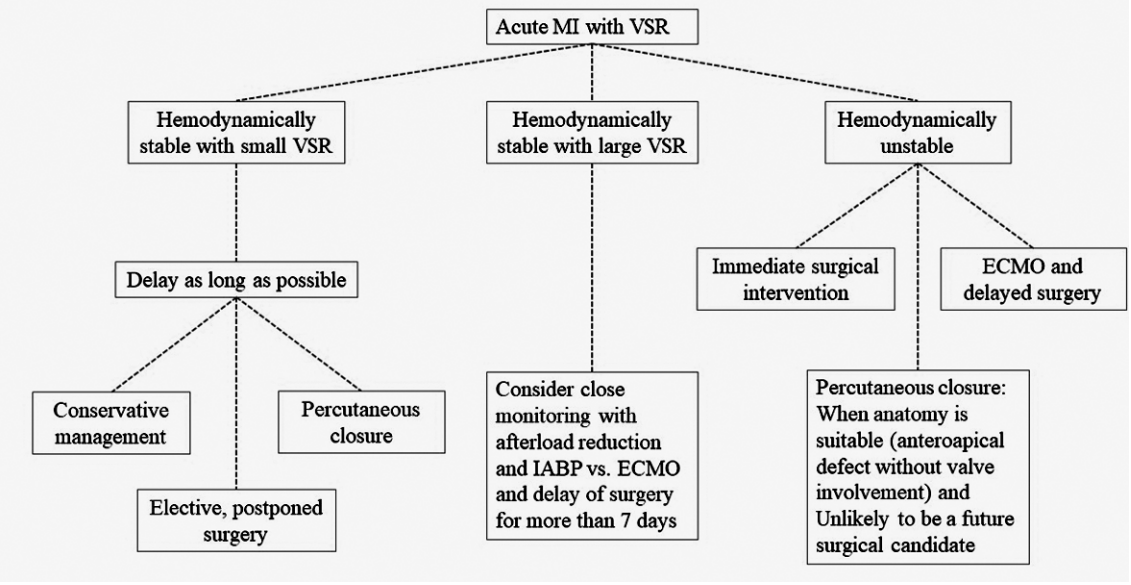

Fig. 4. Management of acute ventricular septal rupture.[1]
MI = Myocardial Infarction; VSR = Ventricular Septal Rupture; IABP = Intra-Aortic Balloon Pump; ECMO = Extracorporeal membrane oxygenation

(ESC) guidelines, there is also no consensus on optimal surgical timing. Earlier surgery is associated with a high mortality rate and the risk of recurrent ventricular rupture, while delayed surgery makes septal repair easier, but there is a risk of extensive rupture and even death during the waiting period.[12]

Consequently, the optimal time for surgery must be determined by the patient's condition and hemodynamic status. In patients who are hemodynamically stable, surgical intervention may be delayed.[1]

Following infarction, metalloproteinase activity and tissue disintegration reached a peak on the seventh day, collagen deposition began between the second and fourth days, and necrotic myocytes were completely replaced by collagen 28 days later. Therefore, delay in surgery can promote effective tissue healing by allowing fragile tissue to strengthen and differentiate from the healthy tissue surrounding it. In this situation, close observation in the intensive care unit may be necessary for tissue healing.[12]

Conclusions

A patient with severe anterior STEMI was reported to have a VSR complication, was hospitalized with cardiogenic shock, and was treated until he recovered. After many days of therapy, his hemodynamic status was determined to be stable and he underwent surgery to close the VSR defect and CABG revascularization. After surgery, the patient's condition was stable and he was discharged from the hospital.

Conflict of Interest

All the authors have no conflicts of interest or financial ties to disclose.

Authors' Contributions

The authors have made equal contributions.

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