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Case Report

Long Term Survival of Ventricular Septal Rupture (VSR) Closure Concomitant with CABG in Post-Acute Myocardial Infarction Patient

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ABSTRACT

Ventricular Septal Rupture (VSR) is a serious complication of acute myocardial infarction. This incidence continues to decline with the thrombolytic and PCI era, but the mortality rate is still high. VSR occurs most frequently after anterior AMI. VSR can occur in the apical, mid or basal of the septum. The most common location is in the apical portion of the septum. The timing of VSR closure surgery depends on the state of the patient's hemodynamic and VSR diameter. Various studies have suggested that VSR closure concomitant with Coronary Artery Bypass Grafting (CABG) provide a better outcome.

Introduction

Ventricular septal rupture (VSR) is one of the mechanical complications of Acute Myocardial Infarction (IMA). The incidence is 1-2% in the pre-thrombolytic era, 0.17% in those undergoing Primary Coronary Intervention (PCI) and 0.2% in those who undergo thrombolytic. The mortality rate of VSR patients who do not get thrombolytic or PCI with surgical therapy is 45% and without surgery is 90%.[1]

Other data showed that mortality rate in 30 days and 1 year when VSR patients undergone surgical therapy is 53% and 47%. Whereas if not performed surgically by 6% and 3% [2]. VSR most often occurs in apical region of the interventricular septum (60%). Basal defects provide a poorer prognosis [2]. Various literature states surgical closure is the main therapeutic option for VSR. The optimal time recommended for this closure is still on debate. Timing of surgery based on the patient's hemodynamic condition and the diameter of the defect.[1]

Case Presentation

A hypertensive man, 53 years, with a body mass index (BMI) of 22.08 kg/m2, came to outpatient heart clinic of Dr. Soetomo general hospital. This patient was a referral from another general hospital with a diagnosis of VSR after the acute coronary syndrome, referred for further management. His chief complaint was shortness of breath.

Shortness of breath was felt since 15 days ago when walking activities 15-20 meters or drinking a lot. From that day on, sufferers sleep with 2 pillows. Eating, bathing and changing clothes can still be done alone. Coughing is also sometimes felt especially at night, no phlegm and no coughing of blood. 20 days ago, the patient experienced typical chest pain, then was taken to another general hospital (24 hours after onset of chest pain). Patients were treated at referral hospital for 11 days, 5 of which were admitted to the ICU with no revascularization strategy. VSR was diagnosed at 4th day being treated at the ICU. In his first outpatient clinic control, he was given a reference to Dr. Soetomo General Hospital.

This patient in compos mentis awareness, 105/70 mmHg blood pressure, regular heart rate 84bpm, 24 times of the respiratory rate, axilla temperature of 36.8oC and oxygen saturation of 98% without oxygen support. There is no anemia, no jaundice, no cyanosis, and no elevated in jugular venous pressure. On a cardiac examination, a single S1 and S2 heart sound were found, there was a systolic murmur grade IV/VI in apex and did not radiate, no gallop and no extrasystole. Lungs Examination obtained vesicular breath in both lung fields, minimal rales in the base of the left lung, and no wheezing. The abdominal examination found that the liver was not palpable and bowel sounds were within normal limits. The extremities are warm and no edema.

Chest x-ray represents cardiomegaly with 59% Cardio-Thoracic Ratio (CTR). Electrocardiography (ECG) showed sinus rhythms 86 bpm, normal frontal and horizontal axis, left atrial abnormalities, recent acute inferior myocardial infarction and slow progressions of R in V1-V3. Laboratory findings showed creatinine 1.30 mg/dL, SGOT 152/L, SGPT 229 U/L. Other parameters are within normal limits.

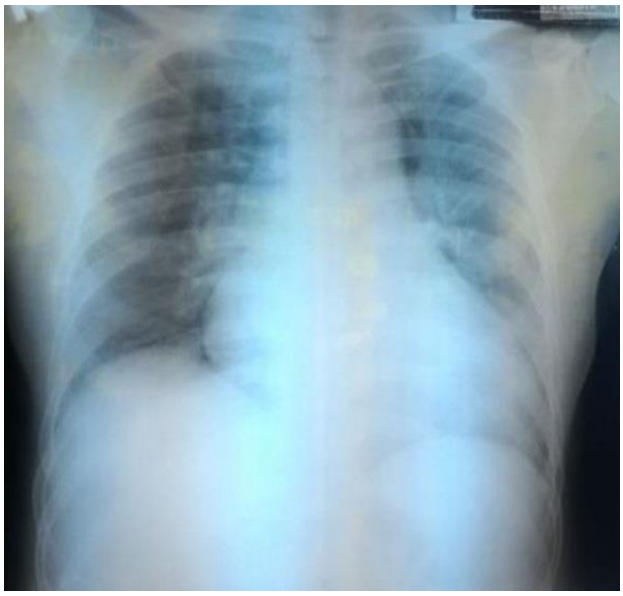

Figure 1. Chest X-Ray on admission showing cardiomegaly

From echocardiography examination, we obtained mild MR, cardiac dimensions within normal limits. There is no thrombus or vegetation intracardiac. Left Ventricle (LV) systolic function was decreases with Ejection Fraction (EF) by teich 58%, by MOD A4C 50%, By MOD A2C 54%, by biplane 52%. LV diastolic function was pseudonormal and normal RV systolic function. Segmental LV analysis was hypokinetic at inferoseptal (BM), septal (A) and inferior (A) segment. The other ones were normokinetic. We also found concentric Left Ventricular Hypertropy (LVH) and VSR emerging at midapical septal portion with diameter range from 1.5 to 2.3 cm. Colour Doppler represent LV to Right Ventricle (RV) flow. Hemodynamic analysis was obtained pulmonary capillary wedge pressure (PCWP) parameters 16.97 mmHg, systemic vascular resistance (SVR) 1855.44 dynes.sec/cm5, mean pulmonary artery pressure (mPAP) 29.05 mmHg, pulmonary vascular resistance (PVR) 312.73 dynes.sec/cm5, left ventricle cardiac output (LVCO) 3.09 L/min, left ventricle cardiac index (LVCI) 2.04 L/min.m2 and 10 mmHg right atrial pressure (RAP) estimation.

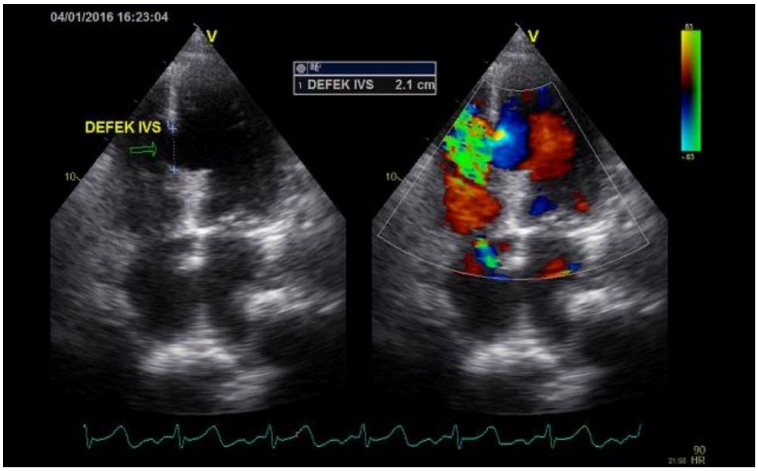

Figure 2. Trans thoracic echocardiography with mid-apical VSR

Patients were diagnosed with CAD Recent inferior IMA (20th day), VSR (16th day), New York Heart Association (NYHA) class II and controlled hypertension. Patients received optimal management with orally furosemide, spironolactone, atorvastatin, nitrate, bisoprolol, Ramipril, aspirin and clopidogrel.

On the 3rd hospitalization day, coronary angiography examination was performed. It showed significant stenosis (70%) in the Left Main Coronary Artery (LMCA), significant stenosis in the osteal to proximal the Left Anterior Descendent (LAD) artery by 90%, significant stenosis (80%) was seen in mid LAD after the D1 branch, significant stenosis (85%) appears distal to LAD after the D2 branch. Significant stenosis in (80%) the osteal of the Left Circumflex (LCx), significant stenosis (85%) distal Right Coronary Artery (RCA) before the Posterior Descending Artery (PDA) branch, significant stenosis in the PDA of 90%, and significant stenosis (70%) distal RCA after PDA branch at. Left heart catheterization was performed before and after measurement of Left Ventricular End Diastolic Pressure (LVEDP). The LVEDP before was 20 mmHg, after LVEDP was 24 mmHg. It also represents apical VSR with 0.6 cm in diameter. The result if this examination suggested surgical closure concomitant with CABG. Expert forum discussions were held to maximizing the management of this patient.

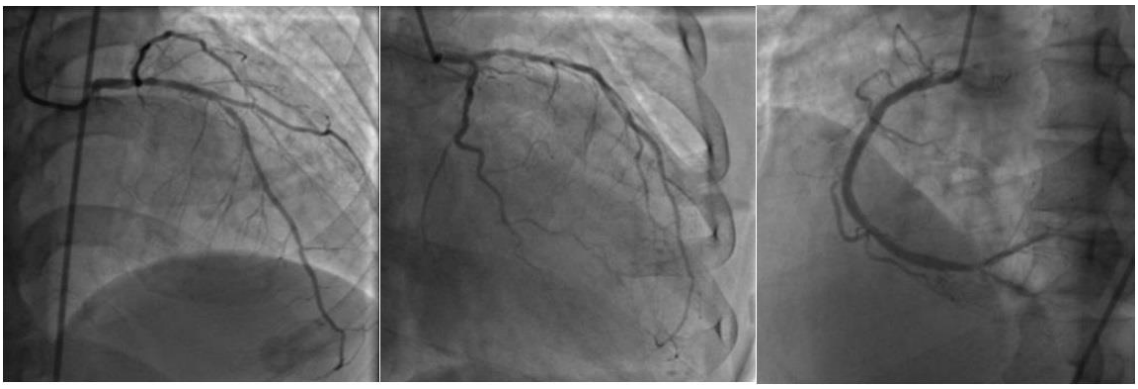

Figure 3. Diagnostic coronary angiography represents CAD triple vessel disease (TVD) with left main disease

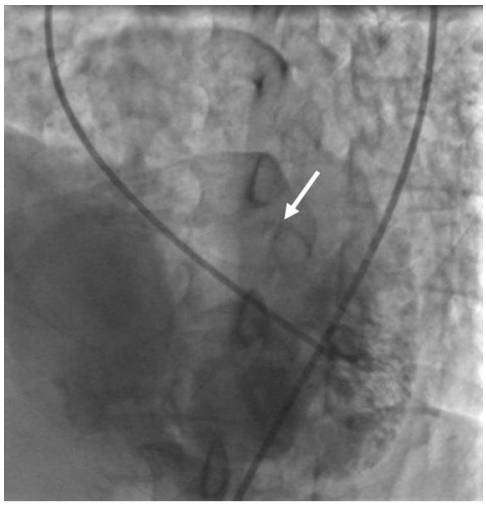

Figure 4. Ventriculography results indicate VSR. White arrows shows VSR location.

Surgery is performed on the 25th hospitalization day. During the procedure, we found cardiomegaly, sufficient contractility, proximal calcification in LAD and LCx, scar and aneurismatic at mid-RCA and PDA. VSR size is 3x2 cm. Then CABG was performed with 3 anastomoses, SVG - RPDA; SVG – distal LCx; SVG - mid LAD, LV aneurysm excision, VSR closure performed with Goretex patch with sutures 4-0 Prolene pledged primary sewing.

We performed echocardiography evaluation on 6th day after surgery. Many changes were found: LV systolic function (EF by teich 59%, EF by MOD A4C 50%, EF by MOD A2C 53%, EF by biplane 53%), moderate pericardial effusion at inferior (1.8 cm), posterior (1.4 cm), left lateral (1.3 cm) and no residual flow was seen in the VSR site.

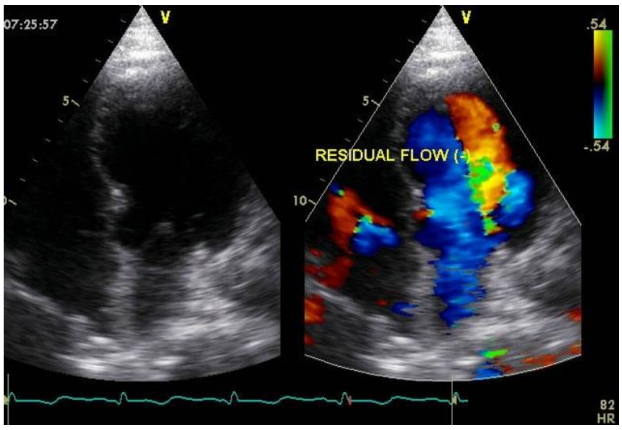

Figure 5. Trans Thoracic echocardiography evaluation post-surgery (no residual flow at VSR site)

This patient was discharged from hospital on the 12th day after surgery with good condition, and had control at outpatient heart clinic of Dr Soetomo hospital on the 3rd day after discharge, then went

on to continue of care at the nearest health center on the island (Bawean). Patients routinely control and take medication. 4 months after surgery the patient has returned to undergo hoeing and fishing. In 1 year observation, the patient returned to control at Dr.Soetomo hospital with good condition and we conducted echocardiographic evaluation with good results. We observed his condition for 3 years. Until now, he was in his optimal condition.

Discussion

VSR is one of the serious mechanical complications of acute myocardial infarction. Various risk factors for patients with Acute Myocardial Infarction (IMA) that can experience VSR are age, female sex, history of previous strokes, chronic kidney disease, and chronic heart failure. The interesting one is that patients who experience VSR often have no history of hypertension, diabetes mellitus, previous smoking history and previous history of IMA.[1]

VSR mostly occurs in the anterior, inferior and lateral segment of IMA. Anterior IMA often causes apical defect and inferior or lateral IMA often causing a basal defect at the septal border with the posterior wall.[1]

From the autopsy, it was found that the anterior VSR was associated with 33% left ventricular infarction and 10% right ventricular infarction, while the posterior VSR is associated with 20% left ventricular infarction and 33% right ventricle. This is related to a sudden increase in volume and pressure in the right ventricle and often causes right heart failure.

The conventional mechanism for VSR includes coagulation necrosis of tissues that experience ischemia characterized by neutrophil infiltration, which causes the myocardial septum to become thin and brittle. This sub-acute process takes 3-5 days. A rupture that occurs within 24 hours after IMA is usually caused by dissection of intramural hematoma or bleeding in the myocardium that is experiencing ischemia. This occurs because of increased shear stress at the boundary of the infarct area, combined with hypercontractile, which dilutesthe segment of the myocardium. This usually occurs in inferior infarcts with VSR occurs in the basal septum, with hyperdynamic in the middle part of the septum perfused by LAD.[1]

Becker and Van Mantgem make the VSR classification into 3 types. Type I, rupture occurs suddenly without the thinning of the septum layer. Type II, myocardial infarction is thinning first before septal rupture and is covered by thrombus. Type III, perforation occurs in an aneurysm that has occurred before. There are other morphological classifications of VSR, simple type and complex type. Simple type usually occurs in anterior IMA, while complex type often occurs in inferior IMA [3]. Clinical manifestations of VSR appear on average 2-4 days after AMI and vary from hemodynamic stable to shock, depending on the size of the defect, presence of right ventricular infarction, ongoing right ventricular ischemia, right ventricular stunning due to volume overload [1-2]. Clinical symptoms arise due to the presence of acute right heart failure, pulmonary hypertension and worsening cardiac output which often manifests as shock and or discordant of various organs. 66% of patients who survived the acute phase will develop into ventricular aneurysms.[2]

Suspicion of VSR when a systolic murmur is found in the precordium, harsh with thrill, audible flow on the right side, the pulmonary component of the heart II hardens, gallop, or tricuspid regurgitation [4]. Electrocardiogram pattern may appear branch block due to damage to the conduction pathway in the septum [2]. From the examination of two-dimensional TTE, interventricular septal tears and blood flow across the septum with color Doppler are

found. Also there is dilation of the right ventricle and pulmonary hypertension. Pericardial effusion is found when ventricular wall rupture occurs [1]. TEE is used if there is a high suspicion of VSR but from TTE there is no or unclear defect [2]. From ventriculography, there is a contrast flow from the left ventricle to the right ventricle. When using a pulmonary artery catheter, VSR is suspected if there is a sudden increase in O2 saturation of mixing venous, which describes the flow from left to right [1]. MRI of the heart is not the first choice diagnostic tool. Cardiac MRI provides more value in the results of catheterization and echocardiography which still raises many questions or for assessing postoperative residual flow.[2]

In this patient, there was a history of inferior IMA without revascularization with VSR complication which occurred on the 4th day after IMA. Risk factors for VSR after IMA in these patients are age, hypertension and smoking history. In this patient VSR was in mid-apical septal. The patient came to Dr. Soetomo outpatient heart clinic on 20th day after IMA with stable hemodynamic condition. There are 2 VSR closure techniques, surgery and percutaneous closure. Surgery technique includes Daggett and David procedures. The Daggett procedure uses 1 or 2 patches to close the VSR and sewn into the right and left ventricles. In the other hand, David's procedure excludes infarction with all sutures placed in the left ventricle. In acute conditions, the infarcted myocardium becomes weak and fragile. At that time closing the defect by sewing is not a good choice because it can cause a rapid rupture. The successfulness of the VSR closure is also determined by infarcted tissue and using appropriate patch sizes [1]. Posterior VSR closure technique is challenging. Specific maneuvers are needed, such as the heart being raised so that it can place patches on the posterior side [1]. Furukawa H. et al., reported that the Dagget technique is a simpler, faster, more effective and reliable technique for VSR closure operations.[5]

Percutaneous closure can be done when defect diameter <15 mm. Inferior or posterior defects often have only a few edges to place the tool. Closing the defect in the basal is more difficult. In placing the tool, it must be known well about the size of the defect, shape and edges. Previously TEE and the catheterization can be performed.[1-2]

Many studies reported that VSR closure concomitant with CABG has better results in mortality reduction and increasing in life expectancy compared to VSR closure which is not concomitant with CABG. Barker et al. Reported survival rates in 30 days, 1 year, 2 years, and 4 years in patients who carried out VSR closure operations concomitant with CABG of 96.2%, 91.6%, 88.8% and 82.8%. On the contrary, patients who did not concomitant with CABG reached to 79.1%, 58.8%, 49.1% and 32.2%.[3]

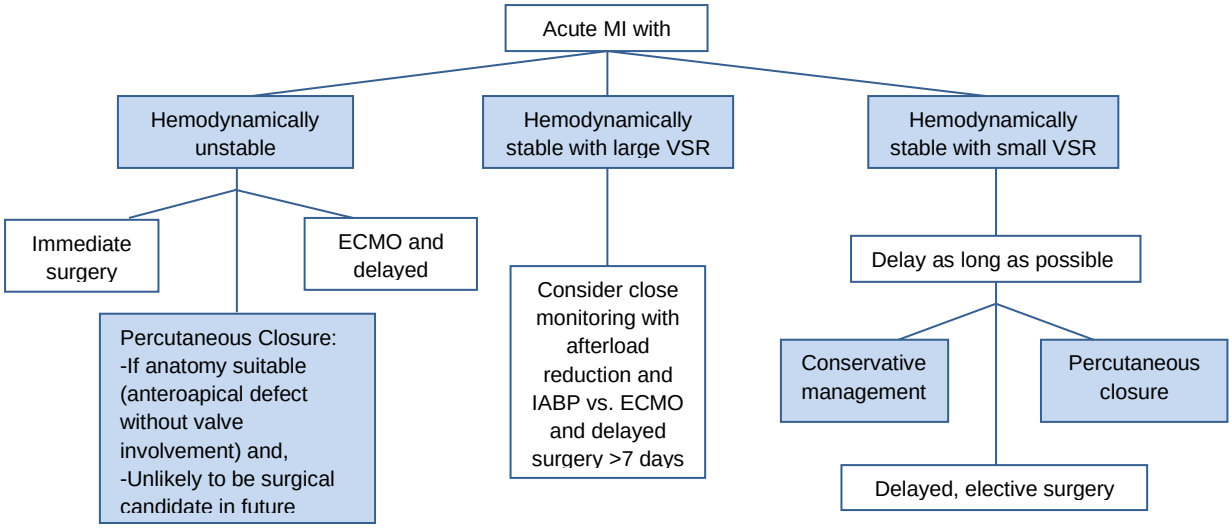

Figure 6. A multidisciplinary approach for managing acute ventricular septal rupture

Conclusion

We have been reported male, 53 years old, farmers and fishermen, with a history of hypertension and smoking, was referred to Dr. Soetomo general hospital diagnosed with an VSR after IMA (20th day) to get further management. We performed anamnesis, physical examination and various investigations. As a result of multidisciplinary discussions, patients are planned to carry out VSR closure surgery concomitant with CABG. The surgery is carried out on the 25th day of hospitalization or the 41st day after being diagnosed with VSR. We conducted appropriate management and echocardiographic evaluation. Patients routinely control and take medication. At 3 year of observation period, patient in good condition and back to his activity like before.

Acknowledgment

There is no conflict of interest.

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