Cardiovascular Cardiometabolic Journal (2022) 2, 100-111
E-ISSN: 2722-3582 , P-ISSN: 2746-6930
DOI: 10.20473/ccj.v3i2.2022.100-111

Case Report

Ventricular Septal Rupture (VSR) in Post-Acute Anterior Myocardial Infarction Patients: A Case Series

Ken Christian Kawilarang[1*], I Kadek Herry Hermawan[1], Febriyanti Hartono[2]
[1]*Intern GP, dr. Ramelan Navy Hospital, Surabaya.*
[2]*Cardiologist, dr. Ramelan Navy Hospital, Surabaya.*

ARTICLE INFO

Article history:
Submitted 22 June 2021
Reviewed 25 May - 07 June 2022
Revised 02 - 17 August 2022
Accepted 20 September 2022
Available online 30 September 2022

**Corresponding author:*
kenchristiank@gmail.com

Keywords:

Cardiogenic Shock
STEMI
Venctricular Septal Rupture

ABSTRACT

Case Summary: We present 2 cases of post-STEMI VSR. They had anterior AMI without reperfusion therapy because of patient delay and limited resources. Thereafter apical VSR occurs on 5th day. VSR was suspected because of sudden hemodynamic deterioration and new holosystolic murmur (Grade III/VI) at left parasternal line of fourth-fifth ICS. Then it was confirmed by echocardiography. Both patients underwent hemodynamic stabilization while waiting for surgical therapy. Unfortunately, they deceased because of cardiogenic shock accompanied by other comorbidities. **Discussion:** VSR causes shunt from left to the right ventricle, which overloads the RV, leading to cardiogenic shock. The peak incidence of VSR in the first 24 hours or 3-5 days post-AMI. This fits both of our cases. VSR occurs in total coronary occlusion, which then causes coagulation necrosis in the ischemic area, characterized by neutrophils that trigger apoptosis and lytic enzymes, so that septum becomes thin, brittle then ruptures. VSR is most common in anterior AMI and the defect is at apical septum, as in our patients. Definitive therapy for VSR is surgical closure, but the best timing is still under debate. This article is expected to make us more aware of post-AMI VSR, so we can quickly diagnose and promptly treat the patients with multidisciplinary approach. This is very important to optimize patient's outcomes even in limited resources area.

Introduction

Acute Myocardial Infarction (AMI) still remains one of the leading causes of death worldwide[1]. In myocardial infarction, mechanical complications can occur, although the incidence is relatively rare (<1% cases). One of the mechanical complications is cardiac rupture, which consists of free wall rupture (0.52%), ventricular septal rupture (0.17%), and papillary muscle rupture (0.26%).[2]

This case series will discuss about ventricular septal rupture (VSR) in post-acute anterior MI Patients.

Ventricular septal rupture (VSR) is one of the fatal mechanical complications of AMI. Although it is rare, it has a very high mortality and morbidity rate[3–5]. The incidence of VSR in the pre-reperfusion era ranges from 1-2%. Currently, after

various methods of reperfusion are available, the incidence of VSR is decreasing, ranging from 0.17 to 0.31% [3,4,6]. However, the mortality rate is still very high. For VSR patients who did not undergo surgery or device closure ranged from 46% - 90%. Even after surgery, it is still high at around 40%.[7]

The purpose of this article is to expand our knowledge about post-infarction VSR in our work environment. Thus, we can know the clinical course, predictive factors and risk factors of VSR to be able to speed up the diagnosis even in places with limited resources. In addition, prompt treatment with multidisciplinary approach is also needed to provide better outcomes and decrease mortality rate,

Case Presentation

Case 1

A 62-year-old man came to the ER, dr. Ramelan Navy Hospital Surabaya. The patient was referral from another hospital. He was only hospitalized for 4 hours in the previous hospital, then he was referred for further treatment at a more advanced health facility. He came with chief complaint of left-sided chest pain. Pain was felt 17 hours before going to our hospital while he was cleaning the house. The pain persisted. It was like being crushed by heavy weight and radiating to his left arm. He also complained nausea and diaphoresis. There was no dyspnea. According to the patient, he had never felt a similar symptom before. He had no previous history of heart disease, but he had a history of diabetes mellitus, which was not well controlled. He denied history of hypertension, dyslipidemia and smoking.

On physical examination when he first came to the ER, the patient's consciousness was good, compos mentis (GCS 4-5-6), BP 150/90 mmHg, HR 75 bpm, RR 20x/min, axillary temperature 36.7°C and oxygen saturation 99% with room air. There were no signs of anemia, jaundice and cyanosis. Besides that, JVP was not increase. On cardiac examination, there were single S1 and S2, with regular heart rhythm and no murmurs and gallops heard. On lungs examination, vesicular breath sounds in both lung fields, with minimal rales at the basal of both lungs, without wheezing. Abdominal and extremities examinations were within normal limits. There was no edema. Chest X-ray showed cardiomegaly, with CTR 60%. ECG showed sinus rhythm, 78 bpm, normal frontal and horizontal axis, ST elevation with pathological Q at V1-V5, indicating recent anterior AMI (Figure 1). Laboratory examination showed HB 8.9 g/dL, HCT 26.9%, random blood glucose 187 mg/dL, HbA1C 13.1, creatinine 2.03 mg/dL. Other lab tests were within normal limits.

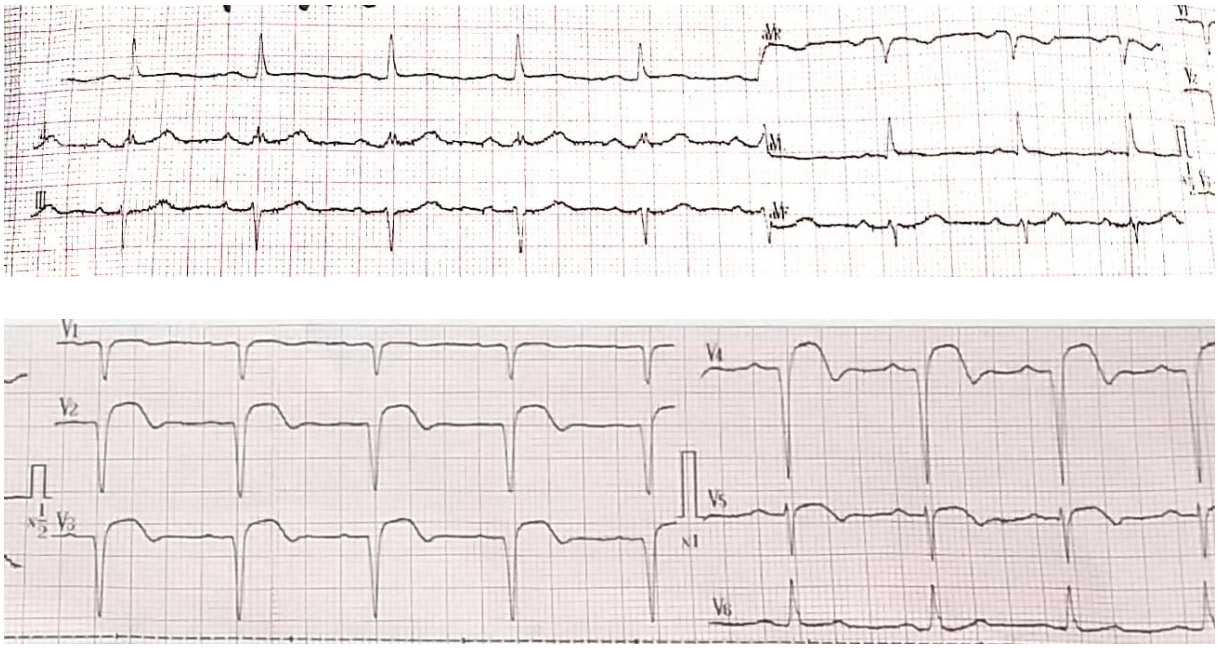

Figure 1. ECG on admission showing recent anterior AMI

The patient received pharmacological therapy, including loading doses of aspirin and clopidogrel, then continued with enoxaparin injection, ISDN, furosemide, bisoprolol and atorvastatin. After sometime, his clinical condition gradually improved, the complaints slowly reduced and the condition became relatively stable.

Unfortunately, on the 5th day of hospitalization, the patient's clinical condition worsened and became severe dyspnea. There was a decrease in consciousness (GCS 3-3-5), BP 79/54 mmHG, HR 100 bpm, RR 30x/min, SpO2 96% with oxygen supplementation 10 lpm using NRM, axillary temperature 36.6°C. The patient was in cardiogenic shock. Latest cardiac examination, found a new holosystolic murmur at the left parasternal line of fourth intercostal space. Grade III/VI murmur, not accompanied by thrill. Thus, leading to suspicion of a VSR complicating AMI. On the lung examination, there were fine crackles in both lung fields. After that, immediately carried out a chest x-ray. The results revealed cardiomegaly with cephalization leading to pulmonary edema (Figure 2).

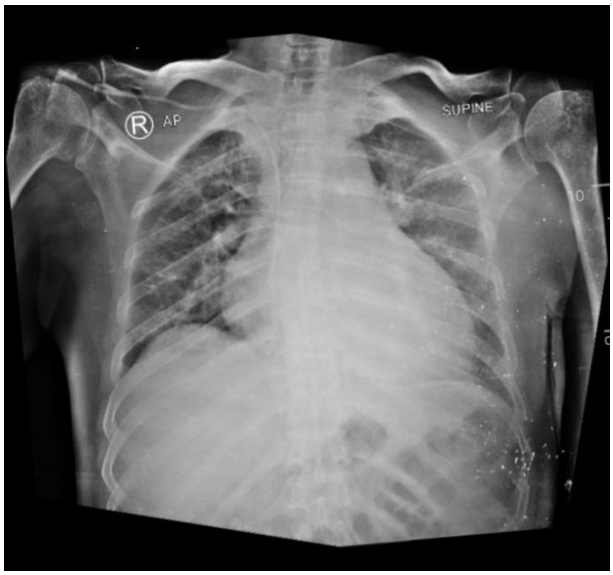

Figure 2. Chest x-ray 5th day of hospitalization cardiomegaly and pulmonary edema

The next step to confirm the presence of VSR was an echocardiography examination. The results showed a 0.6 cm VSR at apical septal segment with flow from LV to RV, Decreased LV systolic function, 29% ejection fraction, Decreased LV diastolic function (grade II). There were Akinetic all apical segments with aneurysm at apical septal, and akinetic at mid anterolateral segments. Another segment was normokinetic. In addition, there were mild TR, mild PR, Good RV contractility and Low probability PH (Figure 3).

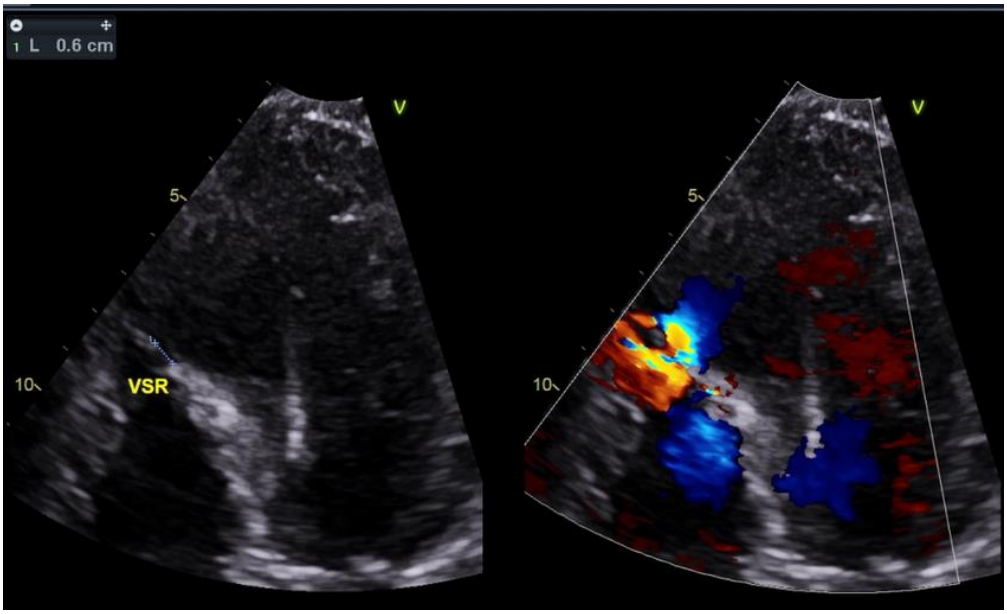

Figure 3. Echocardiography (apical 4-chamber view) showing VSR (0.6 cm) at apical septal segment

The patient was diagnosed with recent anterior AMI, VSR, acute pulmonary edema, cardiogenic shock, chronic kidney disease and diabetes mellitus. To overcome this condition, he was given norepinephrine, dobutamine, furosemide and several other pharmacological therapies to stabilize his hemodynamic.

The patient's condition after receiving various therapies was improved. He was compos mentis (GCS 4-5-6) and reduced shortness of breath. Vital signs were relatively stable despite still using inotropes and other supportive drugs. After discussions between cardiologist, thoracic and cardiovascular surgeons, and other specialists who were involved in treating the patient, he was finally planned to undergo VSR closure surgery 2 weeks after the onset of AMI.

Unfortunately, on the 14th day of hospitalization, the patient experienced another deterioration. There were several comorbid that worsen his condition. He had urinary tract infection, worsening renal function (ACKD), metabolic acidosis. So then he had decreased consciousness (GCS 1-1-1), BP 55/34 mmHg, HR 50 bpm, RR 12x / min, oxygen saturation 60% with JR 15 lpm, and cyanosis. The patient deceased despite all the medical efforts.

Case 2

The second patient came to the ER one day after the first patient was hospitalized and also a referral from the same hospital. Prior to being referred, she had been hospitalized for 3 days. She was referred with the diagnosis of Recent AMI anterior, cardiogenic shock, suspected VSR, acute heart failure, acute kidney injury, paroxysmal AF. A 51-year-old female with chief complaint of left-sided chest pain in the last 5 days before going to the ER. It was like a burning sensation and radiated to the left shoulder. She complained of dyspnea that was getting worse and still persists even though she was at rest. She also complained of diaphoresis. Chest pain and shortness of breath still persisted. Previously, she had never experienced similar symptoms. She was 95 kg with a BMI of 37.1 and classified as grade II obesity. She had a history of dyslipidemia. History of previous heart disease, diabetes mellitus and hypertension were denied by her.

On physical examination when she first came to the ER, she was compos mentis (GCS 4-5-6), BP 83/56 mmHg, HR 86 bpm, RR 28x/min, axillary temperature 36.8°C and oxygen saturation 96% with oxygen supplementation 15 lpm using NRM. There were no signs of anemia, jaundice and cyanosis. Besides that, JVP was not increase. On cardiac examination, there were single S1 and S2, with regular heart rhythm. There was a new holosystolic murmur at the left parasternal line of fourth intercostal space and also at the midclavicular line of fifth intercostal space. Grade III/VI murmur, not accompanied by thrill.

Therefore, it was suspected that VSR occurred due to complications from AMI. On lung examination, vesicular breath sounds were found in both lung fields, with rales at the bases of both lungs, without wheezing. On abdominal examination was within normal limits. On extremities examination, all extremities were cold, CRT >2 seconds and there were pitting edema at lower extremities. Chest x-ray showed cardiomegaly (Figure 4). ECG showed sinus rhythm, 88 bpm, Normal frontal and horizontal axis, ST elevation with pathological Q at V1-V6, indicating the presence of recent anterior AMI (Figure 5). Laboratory examination showed creatinine 2.5 mg/dL. Other lab tests were within normal limits. Echocardiography examination had not been done on the patient because the portable echo equipment was broken, while the condition was not transportable to be examined in echocardiography room.

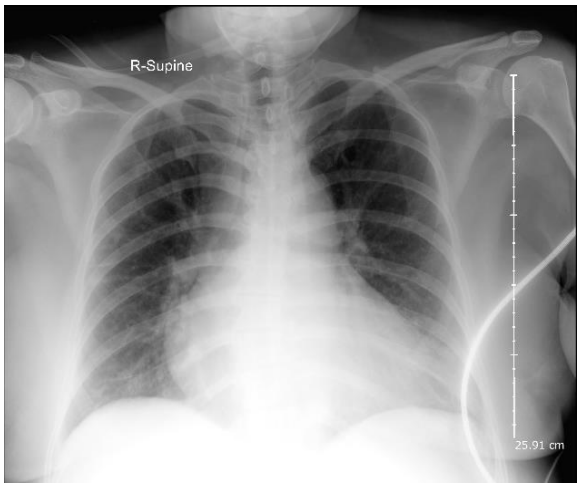

Figure 4. Chest x-ray on admission showing cardiomegaly

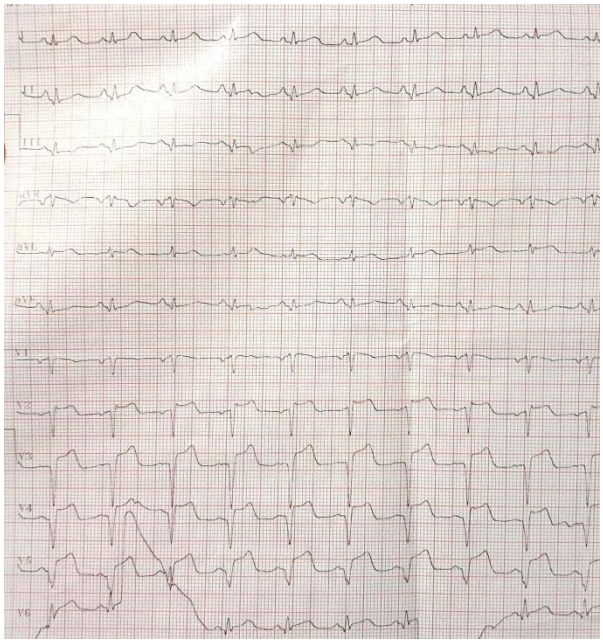

Figure 5. ECG on admission showing recent anterior AMI

The patient received pharmacological therapy. These include dopamine, aspirin, clopidogrel and atorvastatin. After it was given for hemodynamic stabilization, her clinical condition gradually improved and became relatively stable. Nevertheless, the next day, her condition worsened again, she was getting more dyspnea, experiencing desaturation and worsening peripheral perfusion. Renal function deteriorated rapidly and the patient also developed urinary tract infection, metabolic acidosis, recurrent heart rhythm disturbances and recurrent hypoglycemia. She underwent emergency hemodialysis because of worsening renal function. However, her condition was still getting worse. The patient deceased on the 3rd day of hospitalization at our hospital.

Table 1. Summary of the comparison of the two patients

	Patient 1	Patient 2
Sex	Male	Female
Age	62 y.o	51 y.o
BP on admission (mmHg)	150/90	83/56
HR (bpm)	75	86
History of MI	-	-
Diabetes	+	-
Hypertension	-	-
CHF	-	-
CKD	+	-
Smoking	-	-
ECG	Sinus rhythm, 78 bpm, Normal axis, ST elevation with pathological Q at V1-V5	Sinus rhythm, 88 bpm, Normal axis, ST elevation with pathological Q in V1-V6,
Location of infarction	Anterior AMI	Anterior AMI
Delay in coming to the hospital from the onset of symptoms	14 hours	2 days
Reperfusion therapy	-	-
Murmur	Holosystolic murmur at the left PSL of 4th ICS (Grade III/VI); thrill (-)	Holosystolic murmur at the left PSL of 4th ICS & MCL of 5th ICS. (Grade III/VI); thrill (-)
Echocardiography	0.6 cm VSR at apical septal segment with flow from LV to RV; EF 29%; Decreased LV diastolic function (grade II). Akinetic all apical segments with aneurysm at apical septal, and akinetic at mid anterolateral segments.	-
Onset of VSR	5th day post-AMI	5th day post-AMI
Location of VSR	Apical septal segment	Suspicion at apical septal segment
Cardiogenic shock	+	+
Live or decease	Deceased on 14th day of hospitalization	Deceased on 3rd day of hospitalization

Discussion

VSR is perforation of the interventricular septum, resulting in shunt from left to right ventricle [1,6]. It is fatal mechanical complications of AMI. Although it is rare, it has a very high mortality and morbidity rate [3–5]. Currently the incidence is decreasing due to reperfusion therapy [1,2,8,9]. However, the mortality rate is still very high, despite development of surgical therapy. VSR is often caused by delays in the diagnosis and treatment of AMI, especially in developing countries such as Indonesia due to limited resources and patient's knowledge [10]. This is also experienced by both of our patients. Especially in the second patient who just dared to go to the hospital after 2 days of experiencing chest

pain. Both patients were also unable to receive reperfusion therapy either invasively or pharmacologically due to limited resources and patients delay. This delay has also been extended because we are still in the era of the Covid-19 pandemic.

The peak of VSR incidence has a bimodal peak. It occurs 24 hours after AMI, and 3-5 days, with an average of 1-14 days after AMI [1,6]. This is match with our cases, as VSR in both patients occurred on 5th day post AMI. Etiology of VSR is a total occlusion of the coronary arteries that supply interventricular septum, resulting in transmural myocardial infarction (full thickness infarction) [3,6,11]. This can occur at any anatomic location of the interventricular septum. In the GUSTO-1 study, VSR was more common in anterior AMI (70%), than in inferior AMI (29%) [6,7]. The most common blockages occurred in the left anterior descending artery (64%) and right coronary artery (28%). VSR in anterior AMI tends to occur at the apical of septum, whereas inferior AMI is often associated with perforation of the basal septum and has a poorer prognosis [1,8]. In both of our cases, there were anterior AMI and suspicion of VSR att the apical septum, which in the first case was confirmed by echocardiography. Thus, the two cases match with the most common cases according to previous studies.

Several risk factors for VSR in AMI patients include old age, female sex, history of stroke, CKD and CHF. VSR is more common in people who have never had an AMI and previous reperfusion therapy. Both of our patients had some of these risk factors [4,7,8,12]. They are more than 50 years old, have never had AMI and reperfusion therapy. The first patient also had a history of CKD. This is in line with several previous studies which showed a higher incidence of VSR in this group. In people who have experienced MI or chronic ischemia, it will trigger the formation of new blood vessels (collateral circulations). This has a protective effect in case of total coronary artery occlusion. In addition, chronic ischemia can "prepare" the myocardium by forming myocardial fibrosis (preconditioned). So maybe our patient has no collateral or fewer collateral vessels and no myocardial fibrosis, but they need to be proven by further objective examination.

The influence of hypertension and diabetes on the incidence of VSR is still being debated until now. According to several studies, diabetic patients will experience impaired angiogenesis or formation of collateral vessels caused by endothelial dysfunction and several other mechanisms. Therefore, there is a higher risk of VSR. But another study said that diabetes has no effect on the incidence of VSR. In a limited study said that CAD patients with hypertension will have more collateral than non-

hypertension patient. In addition, there is cardiac hypertrophy and interstitial fibrosis (preconditioned heart) which is more resistant to damage due to MI so that the incidence of VSR is lower [1,13]. But there are different studies suggesting that high blood pressure during MI will increase the likelihood of VSR because this condition puts more stress on the weakening septum. Possibly, this happened in both of our cases, because their blood pressure was high at the beginning of MI.[1,13–15]

Non-smokers (as in our two patients) will have a higher incidence of post-infarction VSR than smokers. This is because in smokers often cause the formation of collateral circulation. But it is also still being debated and needs further investigation.[1,10,13,16]

The mechanism of VSR is presence of coagulation necrosis of the ischemic tissue and characterized by neutrophil infiltration which can cause the septum to become thin and brittle. Neutrophils will trigger apoptosis and release of lytic enzymes. It causes disintegration of necrotic myocardial tissue. This sub-acute process takes 3-5 days, which explains why VSR often occurs on 3rd – 5th day post MI. Meanwhile, septal rupture that occurs within 24 hours post AMI is usually caused by dissection of an intramural hematoma or bleeding in the ischemic myocardium. This dissection occurs because of increased tension in the area of infarction and hypercontractility that weakens the myocardium. It usually occurs in inferior infarcts with a VSR at the basal septum.[1,3,6,17]

Rupture of the interventricular septum causes a left to right shunt followed by RV overload, increased pulmonary blood flow which in turn causes LA and LV overload. If the process continues, it causes a decrease in the LV systolic function. Furthermore, the body will give negative feedback by increasing systemic vascular resistance through peripheral vasoconstriction. However, this mechanism can actually cause the shunt flow become more severe. Over time, pump failure occurs and causes cardiogenic shock.[1,6,7]

The clinical manifestations of VSR are diverse. Generally, patients with VSR experience acute HF and cardiogenic shock, but there are asymptomatic patient, although this is very rare [2,8]. Therefore, all patients with AMI should be evaluated clinically, especially in patients with sudden hemodynamic deterioration and presence of a new, harsh, loud holosystolic murmur heard best at the lower left sternal border and may be accompanied by a thrill.[1,3,9,10]

Clinical manifestations also depend on size of the septal defect, ongoing ischemia, presence of RV infarction, RV stunning due to volume overload, pulmonary hypertension and worsening cardiac output [1,8]. Other physical examinations obtained in patients with VSR are the presence of 3rd heart sound, prominent 2nd heart sound (pulmonary

component), signs of pulmonary edema, RV and LV failure and also cardiogenic shock.[1,17]

On chest x-ray, there were no specific signs indicating VSR. There may be LV enlargement and acute lung edema [1]. ECG can show the location of infarction, thus predicting the location of VSR. You can also find branch block due to damage to conduction pathway in the interventricular septum [3,8,18]. The gold standard for diagnosing VSR is transthoracic echocardiography (TTE), because it has high specificity and sensitivity. From the echocardiography, we can determine the presence of interventricular septal tears/ruptures, location of the rupture, size of the defect and blood flow/shunt at the septum. It can also evaluate the function of right and left ventricles, and estimate which coronary arteries are involved. Echocardiography can also predict prognosis of the patient. Because it is very useful, it is recommended to perform echocardiography in all AMI patients, especially those with sudden hemodynamic instability and suspicion of VSR.[2,6,11,17]

Management of VSR requires rapid diagnosis, aggressive oxygenation and hemodynamic stabilization of the patient with medication, mechanical support and surgery [1,10]. Pharmacological therapy is given to stabilize hemodynamics before definitive therapy is taken.[10]

The principle is to reduce afterload with intravenous nitroglycerin or other vasodilators and also reduce preload with intravenous diuretics. This can improve the condition of acute heart failure. Decreased afterload can also reduce left-to-right shunt so that LV stroke volume is more effective [6,10,17]. In addition, Inotropic is expected to reduce afterload and increase cardiac output thereby increasing tissue perfusion [10,14]. Mechanical support systems such as IABP (intra-aortic balloon counter pulsation), ECMO (extracorporeal Membrane Oxygenation), LVAD (left ventricular assist device) can be used to assist bridging therapy before definitive therapy.[1,10,19]

Definitive therapy for VSR patients is surgical closure/repair of the septum accompanied by CABG. Transcatheter closure of the septal defect can be an alternative therapy. Surgical closure should be performed on all patients, even if the patient is hemodynamically stable, because the septal defect can increase in size at any time [1,2,10,13]. Even though, the exact time for surgical therapy is still much debated until now [2,4,5,10].

Several studies suggest immediate surgical closure without considering hemodynamic status of the patient to avoid worsening of patient's hemodynamics, because in VSR, the hemodynamic deterioration occurs very quickly. Furthermore, it can causes cardiogenic shock, increased pulmonary venous pressure, decreased renal function, fluid imbalance and other serious

complications such as infection, respiratory distress syndrome, renal failure and many others.[2–5]

Another conflicting opinion is the recommendation to postpone surgical intervention, because several studies show that surgery will provide optimal results if performed within 2-6 weeks from the onset of AMI when fibrotic tissue has formed. It helps maintain the suture, whereas if surgical procedure is performed during acute phase, infarcted myocardium is still weak and fragile, so the sutures are easily ruptured. The outcome of surgery is strongly influenced by size of the rupture, hemodynamic status and patient's surgical risk.[8,10,11,15]

In both of our patients, due to limited resources and other considerations, surgical therapy was planned 2 weeks after the onset of AMI. Prior to that, hemodynamic stabilization and treating acute heart failure with appropriate pharmacological therapy were carried out, so that surgical therapy gave optimal results for the patient. However, unfortunately both of our patients deceased, due to cardiogenic shock. In addition, also accompanied by many other comorbidities, such as rapid deterioration of kidney function, urinary tract infection, acid-base imbalance and heart rhythm disturbance. The second patient had a worse clinical presentation due to a longer delay in AMI treatment, and more severe comorbidities than the first patient.

Conclusion

VSR is a fatal mechanical complication of AMI, which is rare and has a very high mortality and morbidity rate. Therefore, every doctor, especially in limited resources area like our hospital, should be aware of post-infarction VSR, especially in AMI with sudden deterioration of hemodynamic status. Early diagnosis and prompt treatment involving multidisciplinary approach are needed, especially before being referred for surgical therapy which is the definitive therapy for VSR. The timing of surgical therapy and the surgical technique must be individualized for each patient in order to provide optimal outcomes and decrease mortality.

Acknowledgement

There is no conflict of interest.

References

1. Zipes DP. Braunwald’s Heart Disease. J Chem Inf Model. 2019;53(9):1689–99.
2. Michelis I, Kontonasakis I, Tsakliadou C, Kouvelas N. Ventricular septal rupture presented with chronic heart failure symptoms: A case report. Eur Hear J - Case Reports. 2019;3(2):1–5.
3. Kinkel CR, Janette S, Arias SP. Ventricular Septal Rupture Complicating Acute Myocardial Infarction: Interesting Case and Review. Cardiovasc Thorac Surg [Internet]. 2019;4(1):1–5. Available from: www.symbiosisonlinepublishing.com

4. Shah SH, Shah MA. Post-myocardial infarction ventricular septal rupture in a patient with large secundum atrial septal defect: A case report. Eur Hear J - Case Reports. 2019;3(2):10–4.
5. Wang X, Nie F, Ye N, Liu X, Yang S, Guo F, et al. Successful occlusion of ventricular septal rupture in myocardial infarction under the guidance of echocardiography. J Cardiothorac Surg. 2019;14(1):3–7.
6. Eka N, Sejati S, Ari H. Conservative Management for Anterior STEMI Complicated by Ventricular Septal Rupture. 2019;46(3):204–7.
7. Pradana AD, Widodo J. Acta Cardiologia Indonesiana Ventricular Septal Rupture following Anterior Acute Myocardial Infarction : a Challenge to a Sub-District Physician. 2020;7(1):48–53.
8. Hernawati IE. Long term survival of ventricular septal rupture (VSR) closure concomitant with CABG in post-acute myocardial infarction patient. Cardiovasc Cardiometabolic J. 2020;1(2):50.
9. Giuliani ER, Danielson GK, Pluth JR, Odyniec NA, Wallace RB. Postinfarction ventricular septal rupture. Surgical considerations and results. Circulation. 1974;49(3):455–9.
10. Wahjono A, Anjarwani S. Manajemen Ruptur Septum Ventrikel Pasca Infark Miokard Akut di Rumah Sakit tanpa Fasilitas Bedah Ventricular Septal Rupture Management as Acute Myocardial Infarction Complication at Hospital without Surgical Facility. 2017;29(3):271–5.
11. Khazi FM, Al-Safadi F, Karaly Y, Siddiqui NR, Al-Zamkan B, Aljassim O. Management issues during postinfarction ventricular septal defect and role of perioperative optimization: A case series. Ann Card Anaesth. 2019;22(1):30–4.
12. Amorosi NM, White A. Case of ventricular septal rupture following acute myocardial infarction. Ultrasound. 2020;28(3):196–201.
13. Suryono S, Naesilla N, Roestanajie J, Ariyanti D, Wulandari P. Ventricular Septal Rupture in ST-Elevation Myocardial Infarct in Low Resource Setting: A Case Series 226: 226–36.
14. Morimura H, Tabata M. Delayed surgery after mechanical circulatory support for ventricular septal rupture with cardiogenic shock. Interact Cardiovasc Thorac Surg. 2021;31(6):868–73.
15. Kumar S, Choudhury A, Velayudam D, Kiran U. Peri-operative challenges in post myocardial infarction ventricular septal rupture: A case series and review of literature. Saudi J Anaesth. 2014;8(4):546–9.
16. Campos ID, Bonnefoy-Cudraz E, Pozzi M, Hayek A. Postmyocardial infarction ventricular septal rupture as a potentially fatal complication: When time is the best medicine. BMJ Case Rep. 2020;13(9):1–3.
17. Pramadita AM, Sungkar S, Sugiri, Rifqi S. Medica Hospitalia. 2018;5(1):59–65.

18. Yıldırım A, Avcı S, Kızılgöz B, Vural A, Altınbaş ME, Kahraman F. Ventrıcular Septal Rupture Secondary to Inferıor Myocardial Infarctıon. Emerg Med – Open J. 2017;3(2):27–9.
19. Dulai R, Hline A, Javaid MR, Akhtar J. Delayed ventricular septal rupture complicating anterior wall myocardial infarction. BMJ Case Rep. 2016;2016:1–4.