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to Ejection Fraction correlated with Global Longitudinal Strain Value in Predicting
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Coronary Intervention in Acute myocardial Infarction

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Infarct in Low Resource Setting: A Case Series

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Pedoman Tatalaksana Takiaritmia Supraventrikular (TaSuV)

👁 173

Principles of the 3Rs and ARRIVE Guidelines in Animal Research

👁 109

Elevasi Segmen ST Variasi Normal

👁 97

Penilaian Fungsi Diastolik Ventrikel Kiri Dengan Ekokardiografi

👁 77

Fibrilasi Atrium Pada Penyakit Hipertiroidisme Patogenesis dan Tatalaksana

👁 75

INFORMATION

For Readers

For Authors

For Librarians

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Editorial Office

Heart House, Jalan Katalia Raya No. 5, Kota Bambu Utara

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Telephone: +62 21 5681149, Fax: +62 21 5684220

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Search

HOME / ARCHIVES

Vol 41 No 4 (2020): Indonesian Journal of Cardiology: October - December 2020



PUBLISHED: 2021-03-24

CLINICAL RESEARCH

Epicardial Fat Thickness as a Diagnostic Marker of Coronary Lesions in Stable Angina Pectoris Patients

Ulzim Fajar, Mefri Yanni, Muhammad Syukri

PDF

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A Significant Addition of Left Ventricular Fractional Shortening to Ejection Fraction correlated with Global Longitudinal Strain Value in Predicting Major Acute Cardiovascular Event in patients with Acute Coronary Syndrome

Bertha Gabriela Napitupulu, Harris Hasan, Nizam Z Akbar, Andre Pasha Ketaren, Zainal Zafri, Ali Nafiah Nasution

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High Coronary Collateral Circulation Increases Left Ventricular Reverse Remodeling Event in Patients with Chronic Ischaemic Heart Disease Underwent Coronary Artery Bypass Surgery

Ignatius Faizal Yuwono, Udin Bahrudin, Misbah Hari Cahyadi, Ilham Udin, Yan Herry, Sodikur Rifqi, Sahal Fatah, Hardian Hardian, Mochamad Arif Nugroho, Susi Herminingsih

 **PDF**

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Incidence and outcome of no flow after primary percutaneous coronary intervention in acute myocardial infarction

Goutam Datta, Sandipan Sarkar

 **PDF**

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CASE REPORTS

Ventricular Septal Rupture in ST-Elevation Myocardial Infarct in Low Resource Setting: a case series

Suryono Suryono, Naesilla Naesilla, Jarwoto Roestanajie, Dwi Ariyanti, Pipiet Wulandari

 **PDF**

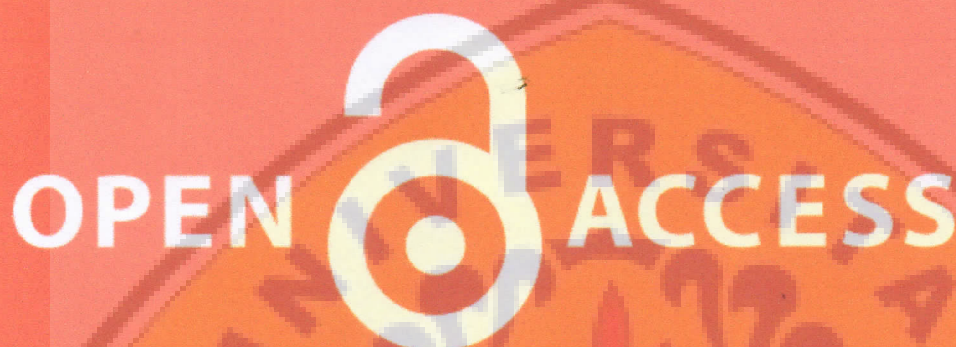
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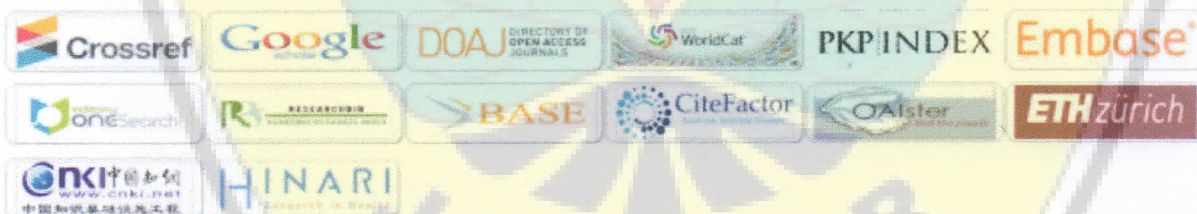
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Pedoman Tatalaksana Takiaritmia Supraventrikular (TaSuV)

👁 173

Principles of the 3Rs and ARRIVE Guidelines in Animal Research

👁 109

Ventricular Septal Rupture in ST-Elevation Myocardial Infarct in Low Resource Setting: A Case Series

Suryono Suryono¹, Naesilla Naesilla², Jarwoto Roestanajie¹,
Dwi Ariyanti¹, Pipiet Wulandari¹

Abstract

Background: The mortality rate caused by myocardial infarct (MI) escalates in the presence of ventricular septal rupture (VSR). This article aimed to describe several clinical features that might be valuable to be explored as predictors of VSR.

Case Illustration: We found and presented six documented post-infarction VSR out of 1,613 MI cases between January 2015 to December 2019. The cases consisted of 4 males and 2 females aged ranging from 40-63 years old. Two patients had hypertension, one had diabetes, while the other two had both diabetes and hypertension. All cases were diagnosed as ST-elevation MI (STEMI) based on electrocardiography and positive troponin result. None of the patients received reperfusion therapy for STEMI. All of the patients developed VSR between 2nd-3rd day since onset and had cardiogenic shock eventually. Despite the treatment, only one patient survived.

Conclusion: Based on the cases, these factors were potential to be explored as VSR predictors: advanced age, anterior location of the infarct, infranodal conduction disorder, no reperfusion treatment, high systolic blood pressure on admission, and no smoking history.

¹ Department of Cardiology,
Dr. Soebandi General Hospital,
Faculty of Medicine, University of
Jember, Jember, Indonesia

² Kaliwates General Hospital,
Jember, Indonesia

Correspondence:

Naesilla, Naesilla,
General Practitioner, Kaliwates
General Hospital, Jember, Indonesia
Email: liemnaesilla@gmail.com

(Indonesian J Cardiol. 2020;41:226-236)

Keywords: myocardial infarct, mechanical complications, predictors, ventricular septal rupture

Introduction

Myocardial infarct (MI) remains one of the leading causes of death worldwide. WHO projected that 80% of non-communicable disease death occur in lower-middle income countries, including South East Asia.¹ Unequal availability and poor affordability of treatments, delay presentation, and ethnicity may contribute to severity of the disease.^{2,3}

One of myocardial infarct (MI) mechanical complications includes cardiac rupture. It can be classified into: free wall rupture (FWR), ventricular septal rupture (VSR), and papillary muscle rupture (PMR).⁴⁻⁶ In this modern era, newer treatments and technology (e.g. percutaneous coronary intervention

(PCI)) could prevent and even cure these complications thus its prevalence nowadays is less than 1%.⁷ Non-urban areas rarely have these privileges. The morbidity and mortality escalate drastically with the presence of cardiac ruptures even in the well-equipped health centers. This article aimed to enrich our knowledge and analyze the post-infarction VSR cases in our local area to find any predictive factors to speed up diagnosis making regardless of the limited resources.

Case Illustrations

We found six documented VSR out of 1.613 MI cases in our town between January 2015 to December 2019 from three hospitals. All the cases will be summarized in Table 1.

Table 1. Summary of the cases¹

Characteristics	I	II	III	IV	V	VI
Age (years)	40	58	48	51	56	63
Sex (F/M)	M	M	M	M	F	F
Risk Factors						
Hypertension	+	+	+	-	-	+
Diabetes	+	-	+	-	+	-
Current Smoking	-	-	-	-	-	-
Previous MI	-	-	-	-	-	-
Physical examination						
BP/HR (mmHg/bpm)	170/90	150/90	160/93	100/60	95/60	180/90
ECG	102	112	105	100	100	72
ST-elevations in leads V1 – V6	ST-elevations in leads V1 – V6	ST-elevations in leads V1 – V6	ST elevations in leads II, III, aVF, V1-V6	ST elevations in leads V2-V6, I, AVL	ST elevations in leads V1-V4, ST depression in leads I, aVL, II, III, aVF, Complete RBBB	ST elevations in leads V2-V6, I, and depression in leads aVL
ECG	Complete RBBB	Left axis deviation				
Infarct location	Extensive anterior	Extensive anterior	Extensive anterior	Anterior (+)	Anteroseptal (+)	Anterior (+)
Biomarker	(+)	(+)	(+)			
Echocardiography						
EF (%)	42	47	48	33	58.5	54.72
Diameter (cm)	0.80	1.28	0.98	1.77	1.02	0.90
Cardiogenic shock	+	+	+	+	+	+
VSR finding since onset	3 rd	3 rd	2 nd	2 nd	3 rd	3 rd
Reperfusion therapy	-	-	-	-	-	-
Mortality	+	+	-	+	+	+

¹ F = Female, M = Male, MI = Myocardial infarction, BP = Blood Pressure, HR = Heart rate, RBBB = Right Bundle Branch Blok, EF = Ejection Fraction, VSR = Ventricular Septal Rupture, PCI = percutaneous coronary intervention

Case 1

The first patient was a 40-year-old man admitted to a hospital due to epigastric pain, chest tightness, and profuse vomiting for the first time started an hours ago. He had a personal medical history of uncontrolled hypertension and diabetes without history of smoking. His father also had hypertension. His initial blood pressure (BP) was 170/90 mmHg, heart rate (HR) was 102 bpm, and respiratory rate (RR) was 26x/min. Oxygen saturation was 96% in room air. Physical examination revealed to be normal (no additional lung/heart sound – murmur or gallop). A 12-leads electrocardiogram demonstrated ST-elevations in leads V1 – V6 and wide QRS in the leads V1 – V3, suggesting extensive anterior STEMI and complete right bundle branch block (RBBB) Figure 1A. Chest X-ray showed cardiomegaly. The patient and his family refused both thrombolysis and primary PCI due to the financial reason (no universal health coverage). He received loading dose (LD) of aspirin 320 mg and clopidogrel 300 mg, atorvastatin 40 mg, and nitroglycerin IV started at 10 mcg/min. His condition seemed to be improved.

Two days after, attending cardiologist found a fresh holosystolic murmur in his left parasternal border of ICS IV. He started having respiratory distress. Echocardiogram revealed a simple apical VSR sized 0.8 cm, left-to-right shunt, left ventricular thrombus, and left ventricle (LV) dysfunction (ejection fraction (EF) = 42%) Figure 1B. Unfractionated heparin was given, to reduce the risk of emboli. Despite the therapy, a few hours later, he reported right-sided hemiplegia and began to lose his consciousness (GCS 2-2-4). Non contrast head CT-scan showed an embolic stroke in his left cerebral hemisphere. His general condition deteriorated as cardiogenic shock was developed shortly after. Intravenous norepinephrine and followed by dobutamine was administered. No further intervention was performed due to family absolute refusal of PCI/reperfusion/cath/transfer. He went into cardiac arrest and after immense resuscitation effort, he was pronounced dead.

Case 2

The patient was 58 years old man presenting a typical chest pain and dyspnea since a day prior. He had a history of hypertension but denied any history of diabetes and

smoking. Physical examination revealed his initial BP was 150/90 mmHg, HR was 112 bpm, RR was 25x/min, normal oxygen saturation (SO₂), and warm dry extremities. Electrocardiogram showed ST elevations in leads V1 – V6 with left axis deviation (Figure 1C). He was diagnosed extensive anterior STEMI. He received LD of aspirin 320 mg, clopidogrel 300 mg, atorvastatin 40 mg, and nitroglycerin IV started at 10 mcg/min. The patient himself refused PCI/reperfusion/cath/transfer due to economic issues.

In early second day of admission, his BP was unstable resulting on postponing nitroglycerin IV. Yet, the BP continued decreasing (and remained at 70/50 mmHg and HR 130 bpm), oxygen saturation of 92%, with cold clammy extremities. He had a cardiogenic shock. Physical examination revealed a new left parasternal border murmur, indicating VSR. Emergency echocardiography confirmed an apical septal rupture, 1.28 cm, with clear left-to-right shunt Figure 1D. He had O₂ 8 lpm (via face mask). Intravenous norepinephrine was started at 0.05 mg/kg/min up titrated on the patient's response and combined with dobutamine. However, the patient remained in shock. Several hours later, he had a cardiac arrest and did not respond to resuscitation.

Case 3

A 48-year-old man complained of chest pain with increasing intensity for 3 hours before admission. Upon arrival in the emergency room (ER), the vital signs were BP of 160/93 mmHg, RR of 25x/min, SO₂ of 92% in room ambient, with warm clammy extremities. According to his family, he had a history of hypertension and diabetes mellitus. He had no smoking habit. Chest x-ray found cardiomegaly with absence of pulmonary edema signs. His electrocardiogram showed normal sinus rate, 105 bpm, and ST elevations in leads II, III, aVF, V1-V6 suggesting anteroinferior STEMI Figure 2A. He, similar to previous cases, refused PCI/reperfusion/cath/transfer due to financial problem, despite extensive education. He agreed on applying to national health coverage. He received standard therapy: LD of aspirin 320 mg, clopidogrel 300 mg, atorvastatin 40 mg, and nitroglycerin IV started at 10mcg/min.

The following day, he developed cardiogenic shock (BP 80/60 mmHg) and audible pansystolic murmur was found in his parasternal border of ICS IV. Emergency echocardiography confirmed simple type VSR sized

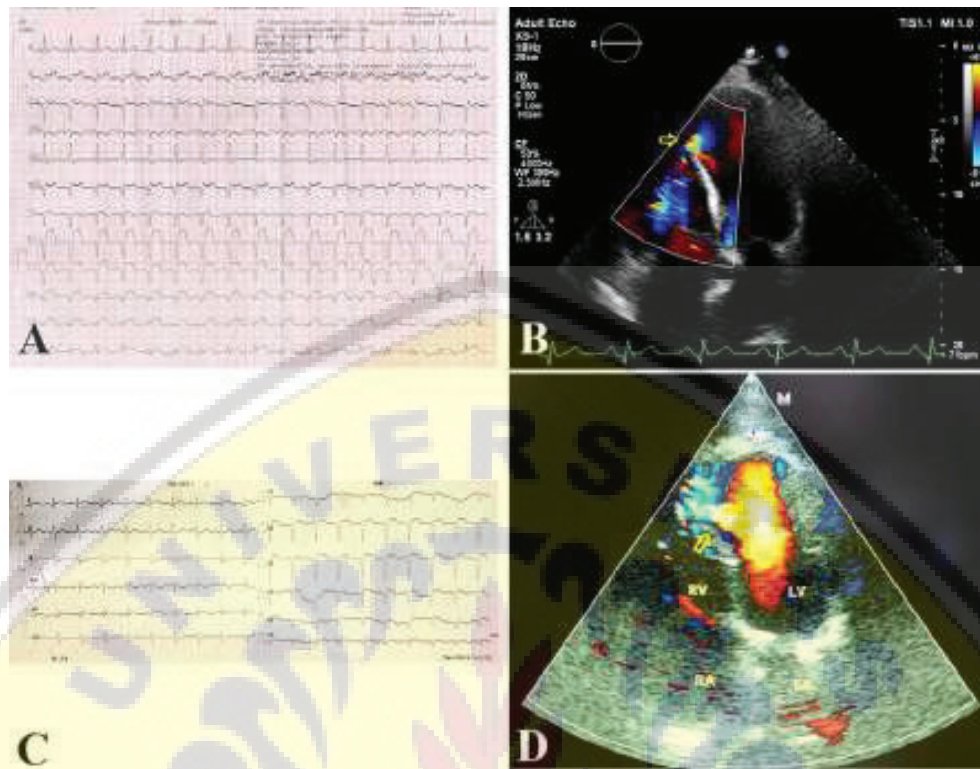


Figure 1. Twelve-leads ECG and echocardiogram for cases 1 and 2. A. ECG for case 1 showing extensive anterior STEMI and complete RBBB B. Echocardiography found a simple apical VSR (left) and echogenic mass at the apex of LV (right). C. ECG for case 2 showing extensive anterior STEMI D. Echocardiography confirmed apical VSR and clear left to right shunt (yellow arrow).

0.98 cm and left ventricular ejection fraction of 48% Figure 2B. He was directly admitted to ICU. His hemodynamic status was gradually improved only with vasopressor. No transfer/intervention/surgery was done to correct the lesion. He continued stabilizing in the follow-up period, and was discharged home on the day 7th since admission. Coronary angiogram showed non-significant stenosis at proximal right coronary artery and left circumflex artery; however critical stenosis was found at proximal left anterior descending (LAD) artery.

Case 4

The fourth case concerns a 51-year-old man who came to the ER with debilitating chest pain for 14 hours. No history of diabetes/hypertension/smoking was found. Clinical assessment at admission: BP was 100/60 mmHg, slightly increased RR (25x/minutes), and heart rate of 100bpm. His SO_2 was 94%. He had a clear chest auscultation. Electrocardiography presented ST elevations in leads V2-V6, I, AVL Figure

2C. CXR found no evidence of pulmonary edema. He received oxygen supplementation via nasal cannula and LD of aspirin 320 mg, LD of clopidogrel 300 mg, and atorvastatin 40 mg. Nitroglycerin IV was given cautiously with close vital signs monitoring.

The patient showed signs of cardiac failure thirty hours later. His BP dropped to 80/50 mmHg. Auscultation revealed apical pansystolic murmur. Transthoracic echocardiography found reduced left ventricular EF (33%) with anterior, anteroseptal hypokinesia and a 1.77 cm rupture of the ventricular septum Figure 2D. He developed cardiogenic shock. The patient and family decided against the referral plan. Despite a high dose of inotropic and vasopressor therapy, he remained unstable and suffered a cardiac arrest.

Case 5

A 56-year-old woman sought medical help for worsening chest tightness and out of breath for 2 days. She had these complaints for almost a month, but

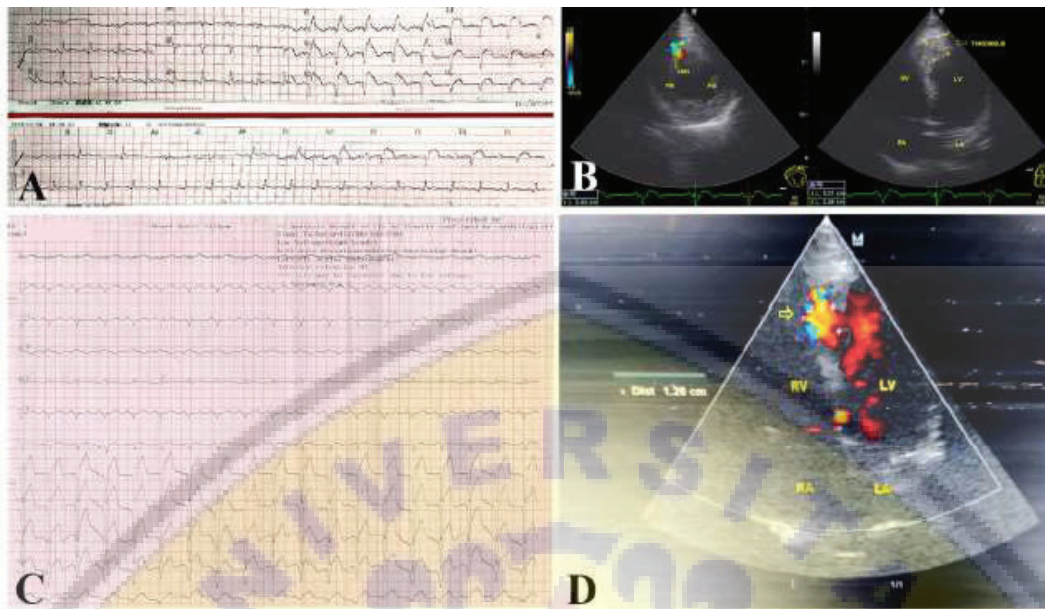


Figure 2. Twelve-lead ECG and echocardiogram for cases 3 and 4. A. ECG for case 3 presenting anterior inferior STEMI B. Four-chamber view echocardiography found a simple apical VSR (yellow arrow). C. ECG for case 4 presenting anterior STEMI D. Echocardiography revealed a VSR sized 1.77 cm (yellow arrow).

she ignored the symptoms and never got it evaluated by medical professionals. Other symptoms presented were nausea and cold sweat. She had a history of diabetes mellitus, but no prior history of hypertension or smoking. At admission, her BP was 95/60 mmHg, HR and RR were normal (100 bpm and 28x/min, respectively), with cold limbs. Chest auscultation found no additional sounds (murmur/gallop/rales). CXR suggested a cardiomegaly. Initial electrocardiogram showed ST elevation in leads V1-V4, ST depression in leads I, aVL, II, III, aVF, and complete RBBB, suggestive of anteroseptal STEMI Figure 3A. She was treated with antiplatelet, anticoagulant, and nitrate.

The next day, her condition worsened and she developed new audible murmur (BP fell to 70/50 mmHg). Bedside echocardiography presented a VSR sized 1.02 cm in the apical septum and anterior, anteroseptal, inferolateral akinetic wall Figure 3B. The left ventricular EF was 58.5%. She required vasopressor (norepinephrine 0.3 mcg/kg/min) and inotropic (dobutamine 10 mcg/kg/min) support, but her condition remained unstable and was recommended to have definitive treatment via surgery. Her family asked specifically not to transfer the referral hospital due to financial problem. She died two hours later.

Case 6

The final case demonstrated a woman aged 63-year-old who had progressive chest pain 12 hours prior to arrival. She denied any history of the similar symptoms previously. She was hypertensive without history of diabetes mellitus, smoking, nor taking any medications. Her admission BP was 180/90 mmHg, HR was 72 bpm, and RR of 25x/min. Physical examination found no abnormalities. CXR revealed a cardiomegaly and her electrocardiogram showed normal sinus rhythm with ST-elevation in leads V2-V6, I, and aVL Figure 3C. The patient refused to be taken for percutaneous coronary intervention or having thrombolytic. Standard therapy consisted of aspirin, clopidogrel, nitrate, and atorvastatin was given.

On the third day since onset, her BP dropped to 80/60 mmHg, her heart rate increased to 139 bpm. A 3/6 murmur was prominent in her left parasternal border and basal rales was found in both lungs. She had emergency echocardiography revealing decreased LV function (EF = 54.72%), akinetic mid-anteroseptal and anterior wall, and hypokinetic basal-anteroseptal, and most importantly a simple anteroseptal mid to apical VSR sized 0.9 cm causing left-to-right shunt Figure 3D. The confirmed diagnosis was extensive anterior with

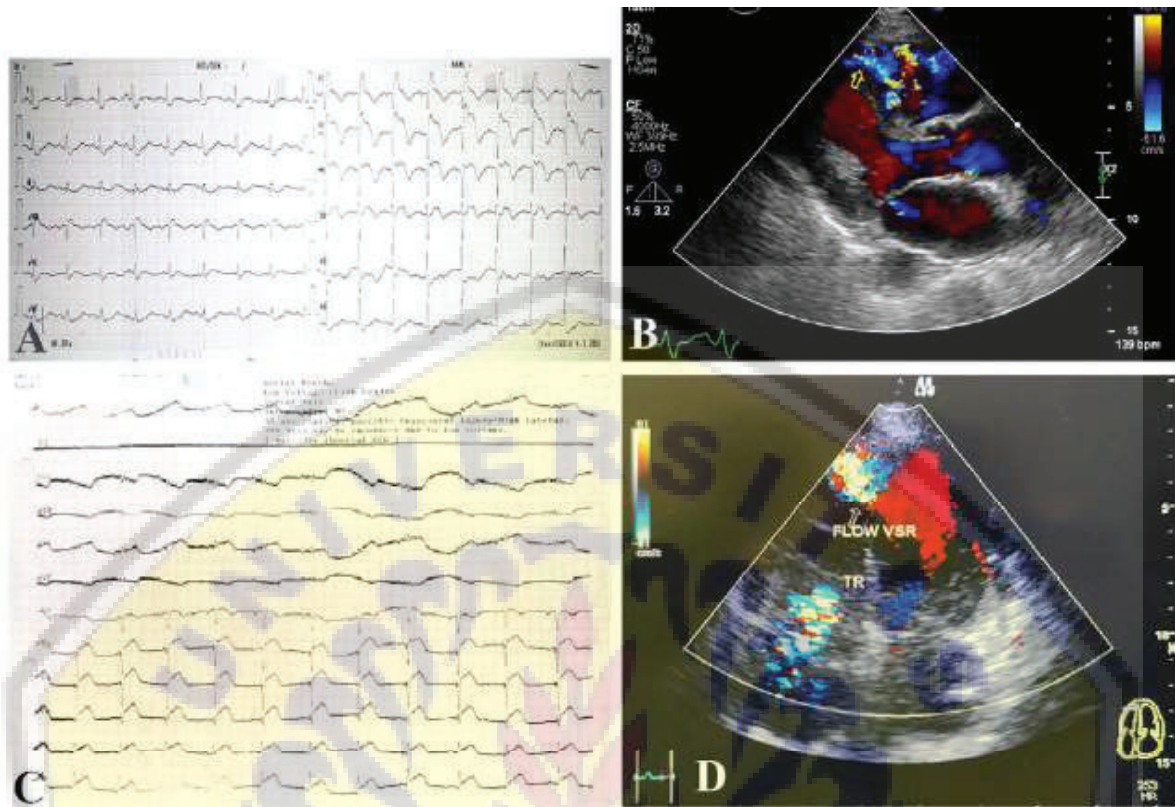


Figure 3. Twelve-lead ECG and echocardiogram for cases 5 and 6. A. ECG for case 5 showing anteroapical STEMI and complete RBBB B. Echocardiography showed a simple VSR in the apical septum (yellow arrow). C. ECG for case 6 showing anterior STEMI D. Echocardiography confirmed a simple anteroapical mid to apical VSR and left-to-right shunt (yellow arrow).

VSR. Unfortunately, the patient did not survive

Discussion

Six cases of VSR were identified in three main hospitals. We observed that 4 out of 6 patients were male. Four out of six had hypertension, one third had diabetes, while no patient had a history of prior or current smoking. This study also found some important findings: all patients had STEMI, most VSRs occurred within the first three days, and female patients were older than the male patients, and most importantly, no patients had received reperfusion therapy.

Currently, reperfusion therapy reduces the incidence of VSR post-MI. However, reperfusion therapy was not accessible equally throughout some areas in Indonesia. A previous study showed that in Jakarta, the capital city of Indonesia, the proportion of non-perfused

STEMI patients was relatively high (62.8%) despite having numerous referral hospitals with 24/7 PCI service.³ Delay in the prehospital setting, including case presenting delay and public lack of knowledge and awareness on heart attack, is still problematic. Historical data from the non-thrombolysis era observed the incidence of VSR was around 1-2%^{4,7,8} which reduced to less than 0.5% in reperfusion era.^{9,10}

Only few studies had briefly described the exact pathogenesis and pathophysiology of postinfarction VSR. Neglected non-reperfused transmural infarction leads to cell necrosis. Relatively thin necrotic septum attracts neutrophils infiltration that provokes apoptosis causing the rupture.^{10,11} A previous study suggested that inflammation arises to aid the heart healing and scar formation throughout myocardial infarction event. However, excessive inflammation could result in adverse events such as degradation of extracellular matrix

protein. It yielded several mechanical complications such as heart failure, damage of collagen, reduce tensile strength, and finally the rupture itself.⁴ This process starts within 24 hours after infarction and requires 3 to 5 days until rupture.^{7,11,12} Another study stated that the process would take different periods depending on the type of ruptures (based on Becker Classification). Type I and II occur within 48 hours after MI. Type III, as the most common type, could vary between 3 to 10 days.⁴ As for this study, all of the patients presented a new finding murmur on the 2nd-3rd day since the chest pain onset.

As presented above, most of the patients were older than 50 years old (5 out of 6 patients). It supported previous studies that older or advanced age acted as one of the most important predictors of VSR.^{7,9,13-15} As the patient aged, the risk of VSR also increased as estimated STEMI risk increased by 41% per 5-year increase of age.¹⁷ Of note, STEMI patients in South-East Asia, specifically Indonesia, tend to be younger than patients from other parts of the world (age group of < 70 years).¹⁸⁻²⁰

In this series, more males experiencing VSR than females. The current finding was contradictory to several studies showing that females were more likely to have VSR.^{7,9,12,14,16,21} Men had been observed of having more considerable amount of collateral hence had more protection and represented a MI events²², considering collateral vessels affected VSR pathogenesis.^{12,23} However, this finding should be interpreted carefully as the gender could be confounded by the age factor and other comorbidities. Females with STEMI typically were older than male counterparts as seen in the cases above. A study found in both genders mice, the total collagen amount was similar, but males were more fragile due to a 70% reduction on cross-linked collagen.²⁴ It was also important to point out that higher mortality rates in women may also be affected by the delay presentation and diagnosis compared to men²⁵, and also by other cultural and financial factors, resulting in cases in female resurfaced.

As mentioned before, most studies agreed that the key to VSR laid on collateral circulation. Collaterals provide a protective factor in severe total occlusion state by limiting the infarct size and prevent deterioration of left ventricle function.^{26,27} Chronic ischemia also stimulate the formation of new vessels. The presence of

prior chronic ischemia could 'prepare' the myocardium by forming myocardial fibrosis (preconditioned).¹³ Most patients with VSR provided no collaterals or grade I (faint filling) (56% and 26%, respectively).²⁸ Another suggested that septal ruptures were unprotected by collateral circulation.²³ This case series exhibited all patients admitting that there was no prior symptoms of MI, speculating they might have fewer collateral vessels. However, the data was highly unreliable since no angiography was done as confirmation.

A limited study described that hypertensive patients with CAD would have more extensive collateral circulation compared to non-hypertensive.²⁹ Diabetes disrupt the formation of collateral coronary vessels.³⁰ It was suggested that diabetes and high blood glucose level interferes endothelium function in angiogenesis through several mechanisms.³¹ However, the effect of hypertension and diabetes on collaterals coronary vessels were still controversial. Newer study discovered no difference between diabetic and non-diabetic CAD patients³² and hypertension might inversely relate to collateral circulation.³³ Hypertensive patients were at lower risk for having VSR as the cardiac hypertrophy and interstitial fibrosis (preconditioned heart) would be more resistant to the damage MI caused.³⁴ As for current cases, most of our patients (4 out of 6) had a history of hypertension while only one third had diabetes. Therefore, both diabetes and hypertension roles in VSR is still debatable.

Nevertheless, these cases showed an interesting finding. It was shown that 4 out of 6 patients had higher BP on admission. Several studies had pointed out that high BP at the case presentation increased the likelihood of VSR as this condition put more stress on weakening septum.^{4,7,11,13} However, case IV and V could not be concluded whether they had high initial BP as they presented in late-onset and pre-shock conditions.

As presented above, no subject had history of past or current smoking. It is being said that patients without current smoking history were more likely to have post-infarct VSR.⁷ SHOCK trial also found that patients with smoking history were less likely to have post infarction VSR.¹² A study had observed that current smoking was positively related to the presence of collateral circulations.³⁵ Yet, no prior study had directly discussed or evaluated the role of smoking on post-infarction VSR, thus further investigations are warranted.

All the subjects of this study had ST-elevations. Several studies had described the correlation between STEMI and VSR. GRACE study found that heart rupture (FWR and VSR) occur more frequently in subjects with STEMI, therefore it stated as a risk factor.⁹ Patients who develop VSR tend to have ST-segment elevation. ST-elevation depicted transmural ischemia resulting in the weakening and rupture of the septal wall.^{10,36} Two out of 6 patients had RBBB. The existence of RBBB as infranodal conduction particularly in anterior MI may indicate an extensive myocardial necrosis event. RBBB was correlated to the occlusion of the left descending coronary artery, proximal to its first septal branch, causing septal ischemia, the predisposition of VSR.³⁷ Septal/left descending infarct was also found to correlated to distal conduction.³⁸ RBBB found to be associated to higher mortality and higher incidence in patients with anterior MI, related to the infarct size.^{37,39}

There were also on-going debates about which location is more prone to VSR. Several studies suggested that anterior MI was one of the independent risk factors of VSR (and other cardiac ruptures).^{5,7,16,21} Contrarily, another study observed the likelihood between inferior and anterior infarct was not statistically different⁸, whilst the other found inferior MI was more likely to have VSR.¹⁵ This study showed most of the patients had anterior involvement. Several studies had found that inferior MI tend to have worse outcome and higher mortality rate than other locations as it was correlated to complex type of VSD. This type could lead to extensive hemorrhage^{10,36}. It is confirmed that mortality risk is higher for patients with inferior-basal defects than for those with anterior-apical defects.⁴⁰ The lack of pure inferior MI resulting VSR in this study might blurred from undetected death cases on STEMI. It is worth to mention that case III was the only case with anterior and inferior STEMI as the angiography showed that this patient had wrapped LAD. A study suggested that VSR will be more likely to occur on anterior MI when the infraction extends to inferior wall as the septum only received blood supply from this artery. This study also mentioned that distal LAD artery occlusion to the first septal branch will caused the ST-segment elevation which supported this finding.⁴¹

VSR progresses into a more severe state in the following days. This progression is caused by the LV ischemia and pump failure and also LV overload,

increased pulmonary flow and venous return due to left-to-right shunt. The sudden increase in right side flow overburdens the right ventricle, leading to significant heart failure.^{42,43} However, as far as we know, no study had investigated the correlation of the VSR diameter and the outcome, considering even the small defect would affect the whole heart system that already been interfered by preceding ischemia.

Five out of six patients did not survive. Previous study had observed that patients with VSR would have a higher mortality rate than non-VSR MI patients.⁷ In line with this case series, some studies had observed several predictors for mortality in VSR within 2 weeks, including shock, advanced age, and female sex.⁴⁴ They concluded that advanced age and female sex were associated with both VSR occurrence and mortality. Another found that survivors were significantly younger and had earlier repair surgery.⁴⁵ The high mortality rate would be reduced with surgical intervention (47% vs 94% 30-days mortality; 53% vs 97% at 1 year). Another study proposed that VSR patient 30-days survival rate who have been treated medically vs surgically were differ significantly (0% vs 71%).⁴⁶ Interestingly, the subject who survived the initial admission would have a good prognosis, as the mortality rate did not differ between 30 days and 1 year.⁷ The fourth patient was exemplary in our study, being currently in good health status, despite having VSR and post-cardiogenic shock. This finding emphasizes that appropriate and precise treatment through the critical time and shock should be established and investigated further.

Conclusion

In summary, there were several clinical features that need to be explored, including : older age (5/6), anterior location of the infarct (6/6), infranodal conduction disorder (2/6), no reperfusion treatment (6/6), high systolic blood pressure on admission (4/6), and no current smoking history (0/6). Further investigation of the exact pathophysiology and the natural history of VSR is the key to the prevention and management of the disease. Also, future studies need to confirm any causal association of these factors on VSR and even further establish the risk stratifications.

Acknowledgement

We would like to thank the radiologists, medical record staffs, and other medical teams for their valuable contributions in data collection for this article.

Publication Approval

All authors read and approved the final manuscript.

Conflict of interest

None.

Sources of funding

This paper received no specific grant from any funding agency, commercial or not-for-profit sectors.

Ethical Clearance

Not Applicable .

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