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Isolated Right Ventricular Infarction without Right Ventricular Dysfunction: A Case Report

Tristi L. Wening[1], Abigail Siagian[1], Rismarini Asanti[1], Sidhi Laksono[1,2]*

[1]Department of Cardiology and Vascular Medicine, Siloam Diagram Heart Hospital, Depok, Indonesia; [2]Department of Cardiology and Vascular, Faculty of Medicine, Universitas Muhammadiyah Prof Dr Hamka, Tangerang, Indonesia

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***Correspondence:** Sidhi Laksono, Faculty of Medicine, Universitas Muhammadiyah Prof. Dr. Hamka, Tangerang, Indonesia/Jl. Raden Patah No. 01, Tangerang, Banten 13460, Indonesia. Tel: +62 811-1585-599. E-mail: sidhilaksono@uhamka.ac.id
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Abstract

BACKGROUND: Myocardial infarction is the leading cause of the occlusion of the coronary artery. Meanwhile, right ventricular myocardial infarction (RVMI) is usually associated with inferior left ventricular infarction in 10–50% of cases. We presented isolated RV infarcts which are rare cases that happen in only 3% of total myocardial infarction.

CASE PRESENTATION: We presented a case of a 67-year-old man with sudden chest pain, shortness of breath, and a history of diabetes mellitus. From his 12 lead electrocardiogram, there is no specific ST elevation yet elevation in V3R and V4R and elevated troponin I highly sensitive value. The patient developed junctional bradycardia and early percutaneous coronary intervention backup temporary pacemaker showed total occlusion in the proximal right coronary artery (RCA)

CONCLUSION: The RVMI commonly occurs in the dominant RCA that also supplies the sinoatrial node and atrioventricular node. Therefore, due to its location that passed through the RCA, bradycardia to complete heart block may happen. Isolated right ventricular (RV) infarction may happen because either RV marginal as the predominant location is occluded, or non-dominant RCA occluded by thrombus. RV infarction may impair RV contractility causing a decrease in RV stroke volume and this condition leads to hypotension, shock, and severe hemodynamic derangement. Meanwhile, acute proximal RCA occlusions do not result in significant RV necrosis, one of the reasons is collateral flow from the left coronary system further protects from RV dysfunctions in our patient.

Introduction

Myocardial infarction is the leading cause of mortality worldwide caused by coronary artery occlusion. Right ventricular myocardial infarction (RVMI) is usually associated with inferior left ventricular infarction. The incidence of right ventricular (RV) infarction occurring alongside inferior wall infarction varies from 10 to 50%. Meanwhile, acute myocardial infarction involving only RV infarction is a rare condition, occurring only in 3% of cases of total myocardial infarction. RV infarction may result in a reduction of RV function and is associated with hemodynamic compromise in 25–50% of cases. The RVMI also contributes to increasing the risk of death, arrhythmia, and cardiogenic shock, thus increasing the mortality rate. However, RV coronary perfusion has a unique characteristic that might contribute to its relative resistance to ischemic dysfunction and injury.

Here, we present a case of isolated RV infarction which manifests as bradyarrhythmia without significant hemodynamic compromise. The patient underwent coronary angiography and was found to have three-vessel diseases with total occlusion in the proximal right coronary artery (RCA) as the culprit vessel.

Case Illustration

A 67-year-old man was admitted to our emergency department with sudden chest pain and shortness of breath 5 h before hospital admission. Chest pain radiated to his left arm and his back accompanied by sweating and nausea. The patient was already given sublingual nitrate by his family, but the symptoms did not improve. His previous medical history was significant for diabetes mellitus. He came to our hospital about a month ago and was scheduled to have an elective coronary angiogram, but the symptoms got worse, so he decided to come to our hospital.

His vital blood pressure of 162/94 mmHg, pulse is 58 ×/min, respiratory rate is 22 ×/min, and oxygen saturation is 94% room air. His electrocardiogram (ECG) showed mild ST elevation in V1, hyperacute T V2-V3, inverted T in III, aVF, and ST elevation in V3R and V4R (Figure 1) with elevated troponin I highly sensitive value 573.00 ng/dl (normal range 29.0 ng/dl). The patient was given a loading dose of 160 mg aspilet and intravenous omeprazole 30 mg. The patient then stabilized and was transferred to the intensive care unit (ICU).

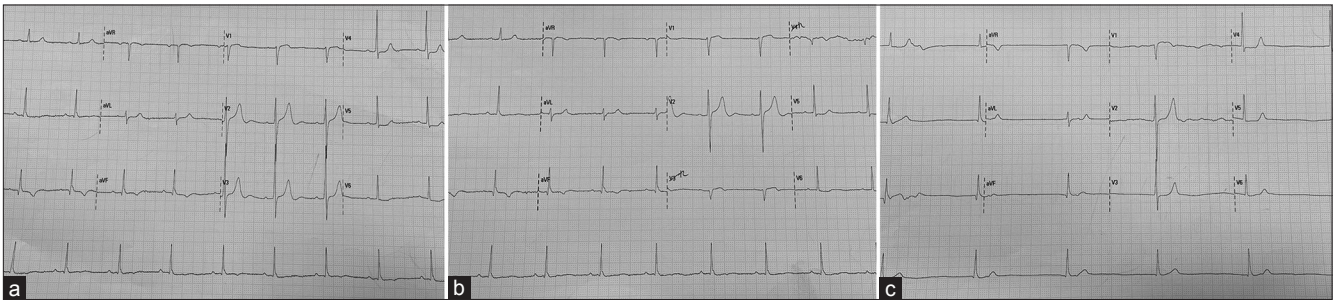

Figure 1: Electrocardiography (ECG) examination: (a) Lead ECG while the patient arrived Emergency Department showed T inverted in lead II and III, and mild V1 elevation. (b) The right-sided ECG leads show ST-segment elevation >1 mm in V3R and V4R. (c) The patient developed junctional bradycardia during observation in the intensive care unit

During observation in ICU, the patient developed junctional bradycardia with a heart rate of 33 bpm (Figure 1). He was given intravenous dopamine 5 mcg/kg/min. No hemodynamic compromise was shown during observation. Early percutaneous coronary intervention with temporary pacemaker backup was done. Moreover, the result is a total occlusion in the proximal RCA. A temporary pacemaker lead through the right femoral vein was inserted into apical RV with setting HR 80 Bpm output 2 mA and sensitivity 2 mV, predilatation was performed with sapphire II 2.0 × 15 mm balloon up to 14 atm. Supraflex cruz 3.0 × 40 mm drug-eluting stent/DES was then placed up to 16 atm in ostial until mid-part. Thrombolysis in myocardial infarction grade 3 flow was achieved (Figure 2).

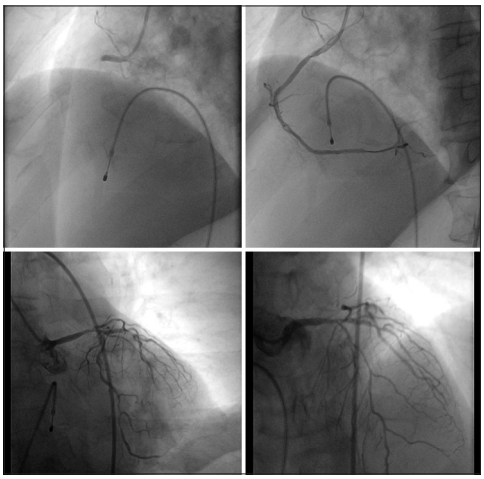

Figure 2: The early percutaneous coronary intervention showed total occlusion of the proximal right coronary artery (RCA), 40% stenosis in the distal part of the left main artery, 95% stenosis in the proximal until the distal left artery descending, and 90% stenosis of the left circumflex artery. 1 stent was placed in the ostial until the mid-part of the RCA

After that, the patient was transferred to the ICU. After 2 days with a temporary pacemaker, the patient developed a sinus rhythm, and a stable heartbeat was maintained. After 24-h observation, the post-off temporary pacemaker patient moves to the general ward, and after observation for 2 days, the patient developed impending acute lung edema, so we decided to transfer the patient back to ICU treated with furosemide drip 5 mg/h and cedocard 10 mg/h. The patient was stabilized in the ICU for 2 days and in the general ward for 3 days. After that, the patient was discharged 7 days after admission.

Discussion

RV infarction associated with inferior ST-segment myocardial infarction contributes to 50% of cases of inferior STEMI, which have higher mortality and morbidity rates. Isolated RVMI is a rare case occurring in 3% of all myocardial infarctions and is mostly caused by an iatrogenic process related to the heart involving procedure [1]. RV infarction commonly occurs when there is occlusion of the dominant RCA proximally to the major right ventricle branches causing disruption of blood flow to the RV free wall [2]. From the majority of cases of isolated RV infarct, the prognosis is good.

The right ventricle receives blood supply mostly from RCA and it also supplies the sinoatrial node. RCA gives multiple blood supplies to the anterior RV and runs to the acute marginal branch (AM) and posterior descending artery (PDA) which supplies the atrioventricular node in 90% of patients. Due to its location passed through the RCA, bradycardia to complete heart block may happen in patients with RV infarct. The patient developed junctional bradycardia while observed in ICU. Isolated RV infarction may happen because either RV marginal branch as a predominant location is occluded or a non-dominant RCA occluded by thrombus.

The diagnosis of RV infarction is confirmed by the presence of ST elevation in the right-side ECG Lead (V3R and V4R). ST elevation in V4R more than 1 mm has 100 % of sensitivity and 87% of specificity in determining the diagnosis of RV infarction [3]. In inferior STEMI, RV infarction is suggested by findings such as elevation of V1 lead accompanied by the depression of V2 lead, isoelectric ST segment in lead V1 with marked V2 ST depression, and ST-segment elevation

in lead III which are higher than the lead II ST-segment elevation [4]. RV infarction leads to decreased RV function and reduces the blood flow from the venous system to the heart and to the heart causing the triad of RV dysfunction such as clear lung field, hypotension, and jugular vein distention. RV infarction also has symptoms commonly seen in the left ventricular infarction including chest pain, shortness of breath, nausea, and cold sweat.

In this case, the patient came with chest pain specifically leading to angina but his first 12 lead ECG showed mild elevation in V1 lead accompanied by inverted t in lead II and lead III, so we performed the right-sided and posterior ECG. His right-sided ECG showed an elevation of V3R and V4R lead, accompanied by elevated troponin level and leukocytosis that confirmed the diagnosis of RV infarction. In this patient, the diagnosis of RV infarction was quite challenging at first for the absence of clinical appearance related to ventricular dysfunction nor the problem with hemodynamics despite the administration of a preload-reducing agent such as nitrate that he had taken previously.

RV infarction may impair RV contractility causing a decrease in RV stroke volume leading to diminished LV filling and drop in cardiac output. This condition may further result in hypotension and shock. An associated increase in RV filling pressure can further cause increased pressure in the right atrium. This might be a stimulus for the secretion of atrial natriuretic factor resulting in further vasodilatory and diuresis effects, thereby worsening the clinical condition of the patient.

Even though RV infarction might present with severe hemodynamic derangement, many acute proximal RCA occlusions do not result in significant RV necrosis [5]. This phenomenon might be seen in our patient, where RV infarction was seen on ECG without signs of RV dysfunction. This relative resistance to ischemic dysfunction and injury might be explained by some of RV perfusion characteristics such as perfusion throughout the entire cardiac cycle caused by lack of mechanical inhibition to perfusion during contraction, lower baseline oxygen demand due to lower developed systolic pressure, and less wall stress, and higher oxygen extraction reserve [6]. Furthermore, we propose the collateral flow from the left coronary system further protects us from RV dysfunction in our patient [7].

Conclusion

Isolated RV infarction is a rare condition and challenging to diagnose. The RV infarction will lead to dysfunction of the right ventricle and impairment of right ventricle contractility. RV infarction might present with mild to severe hemodynamic derangement. Bradycardia is associated with RCA occlusion and might be the only sign of RV infarction. Not all patients will develop the symptoms of right ventricle dysfunction for in some cases the collateral flow from the left coronary system protects the right ventricle.

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