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Case Report

Ventricular tachycardia and acute myocardial infarction induced by coronary artery spasm in patient without coronary artery disease

Gabrielle Alexander Kartawan[1]*, Tiffany Alexander Kartawan[2]

[1]Faculty of Medicine, Udayana University, Indonesia
[2]Faculty of Medicine, Airlangga University, Indonesia

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***Correspondence:**
Dr. Gabrielle Alexander Kartawan,
E-mail: gabriellekartawan@gmail.com

ABSTRACT

Coronary artery spasm, marked by coronary vasoconstriction, is one of the etiologies of myocardial ischemia, often presenting as vasospastic angina. Vasospastic angina is diagnosed when angina which predominantly occurs at rest, is accompanied by ST-segment changes in ECG, or in the setting of borderline ECG changes, a positive provocation test through coronary angiography is required. Although coronary artery spasms could manifest in wide clinical settings, the occurrence of ventricular arrhythmias and acute myocardial infarction solely caused by spasms without evidence of prior coronary artery disease is rare. This case report is about a 46-year-old man who presented with ventricular tachycardia and acute myocardial infarction that later was found to be secondary to coronary vasospasm observed directly through coronary angiography. We aim to emphasize the importance of coronary artery spasms as the etiology of malignant ventricular arrhythmias and acute myocardial infarction manifestation. Optimization in treatment and prevention shall reduce future life-threatening complications of coronary artery spasms.

Keyword: Coronary artery spasm, Vasospastic angina, Vasospasm, Ventricular tachycardia, Acute myocardial infarction

INTRODUCTION

Coronary vasospasm is defined as an abnormal contraction of the coronary artery, resulting in flow reduction, and producing myocardial ischemia. Variant angina which is diagnosed when ST-segment changes are present during angina attack is considered a type of vasospastic angina.[1] Unlike angina on effort that occurs secondary to increased myocardial oxygen demand, vasospastic angina is not always preceded by increased heart rate or blood pressure, showing the hallmark of angina symptoms occurring predominantly at rest and maintained effort tolerance.[1-3]

Manifestations of vasospastic angina are associated with a wide variety of clinical settings, from stable angina, acute coronary syndrome (ACS), syncope, life-threatening arrhythmias, and even sudden cardiac death.[4] Although the prevalence of major adverse cardiovascular event (MACE) including death and MI in vasospastic angina is difficult to define, 3-year MACE reported to be 1-37%.[5,6]

The most common arrhythmia during vasospastic angina crisis is ventricular arrhythmias; its prevalence is related to the duration of the episodes, degree of ST-segment elevation, presence of ST-T wave changes.[7] The prognosis of vasospastic angina which manifests as ACS, ventricular arrhythmias, or aborted sudden cardiac death has been reported to be poor.[8,9] Early diagnosis and management of vasospastic angina in the presence of the above-mentioned life-threatening manifestations are significantly important.

CASE REPORT

A 46-year-old man was admitted to the emergency department, complaining of chest pain that had been felt since approximately 12 hours before. He felt tightness and

could localize the pain just at the center of his chest, not radiating anywhere else, accompanied by cold sweat. The chest pain was subsided by rest. He claimed about the history of similar chest pain around 2 years before when he was also admitted to the hospital, but he couldn't elaborate further information about the diagnosis and treatment he received back then. He is an active smoker. Another past medical history was denied.

Upon examination, he was alert, BP 134/80 mmHg, HR 218 bpm, RR 38, SpO_2 99% room air. Bedside monitor show ventricular tachycardia. Other physical examinations were unremarkable. Using midazolam as sedation, electrical cardioversion was delivered in 100 J and the rhythm converted to sinus with a rate of 102 bpm. He was first assessed with VT and ACS and directly planned for urgent angiography. Loading dose of ticagrelor and aspirin was given, along with bolus of 150 mg amiodarone, then continued with maintenance dose. Hs-cTn T increased at 3-fold URL. Other laboratory examinations were unremarkable.

In the catheterization laboratory, occlusion of 70% in right coronary artery (RCA) was found (Figure 1). PCI was decided but just minutes after a guidewire was introduced, the angiographic image showing the occluded area was relieved and then became fully dilated (Figure 2).

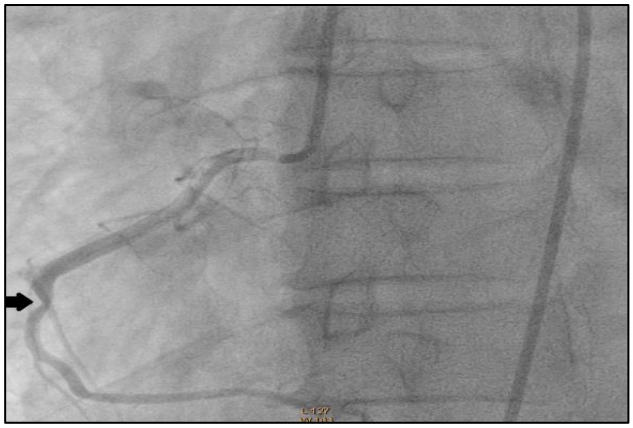

Figure 1: Occlusion of 70% RCA.

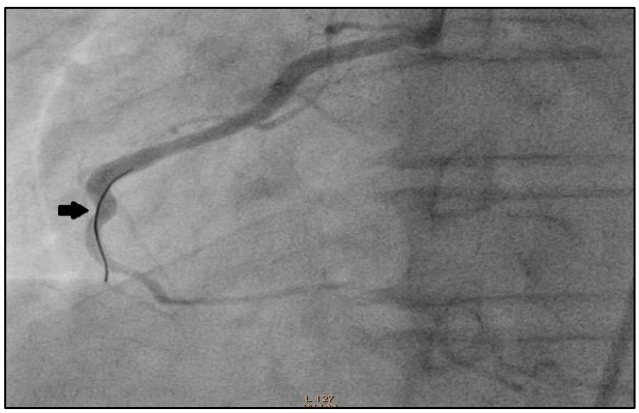

Figure 2: Fully dilated RCA.

The patient was finally diagnosed with acute myocardial infarction with cardioverted ventricular tachycardia due to coronary vasospasm. He was treated conservatively with pharmacological treatment: clopidogrel, oral amiodarone, and diltiazem. Smoking cessation and lifestyle changes was advised. The patient was discharged on 3rd day without any report of chest pain or arrhythmia.

DISCUSSION

Diagnosis

Based on the European society of cardiology (ESC) in 2018, the term acute myocardial infarction should be used when there is an acute myocardial injury with clinical evidence of acute myocardial ischemia and with detection of a rise and/or fall of cardiac troponin value with at least one value above 99th percentile URL and at least one of:[10] Symptoms of myocardial ischemia, new ischemic ECG changes, development of pathological Q waves and imaging evidence of new loss of viable myocardium or new regional wall motion abnormality

For type 2 MI, there must be evidence of an imbalance between myocardial oxygen supply and demand unrelated to acute atherothrombosis. Coronary spasm is included in type 2 MI, with its remark on oxygen demand-supply mismatch.[10]

On the other side, the diagnosis of vasospastic angina is made by detecting ischemic changes in ECG during an angina attack; or in the case of borderline or no clear ischemic changes in ECG, an additional examination is required with a drug-induced provocation test during cardiac catheterization or hyperventilation test.[1,2]

In the case reported, the manifestations of angina tended to be mimicking ACS as it was subsided by rest (angina on effort), along with the presentation of VT as ventricular arrhythmias are more commonly occurs in the association with ACS and coronary artery disease (CAD). VT or VF developed in 6% of ACS patients within the first 48 hours after onset of symptoms. Prompt coronary angiography and if indicated, followed by revascularization, are recommended in ACS patients presenting with life-threatening ventricular arrhythmias.[11] The result of high-sensitivity cTn at 3-fold URL supported that there was cardiomyocyte injury due to prolonged ischemia resulting from vasospasm. Based on the aforementioned criteria of MI, it is safe that this case falls into type 2 MI with increased hs-cTn T, symptoms of myocardial ischemia, ischemic ECG changes, and evidence of coronary artery spasm without acute atherothrombosis in angiography.

Type 2 MI patients are further divided into a patient with or without CAD. In patients with CAD, coronary spasms could be a cause of the rupture of vulnerable plaques. Spasms cause endothelial cell derangement and fibrous cap rupture, resulting in exposure of the plaque protrusion to the vascular lumen, and inducing thrombus production.[1]

Ventricular arrhythmia can occur early or later after acute MI. Sudden death secondary to sustained VT or VF in MI accounts for about 50% of all death in high-risk patients. The early or acute phase accounts for up to 48-72 hours, which is a time of very dynamic ischemia and reperfusion. Acute ischemia causes cellular hypoxia, resulting in intracellular acidosis, cell swelling, and calcium overload. These conditions trigger early and late after depolarization. Furthermore, re-entry involving the infarct scar tissue might sustain monomorphic VT. In acute myocardial ischemia with no previous scar, zones of slow conduction and block may create conditions for re-entry.[11-13]

A study by JCS association, 2.5% of patients with vasospastic angina survived out-of-hospital cardiac arrest (OHCA) which includes life-threatening arrhythmias and revealed that history of OHCA is a strong predictor of cardiovascular events.[14]

Diagnosis of vasospasm as a cause of myocardial ischemia or myocardial infarction often requires a provocation test.[1] In this presented case, we were lucky enough to have found the evidence of vasospasm directly through coronary angiography without provocation test or even administration of NTG.

Management

The mechanism of vasospasm is thought to be multifactorial. Several proposals of the pathophysiology include vascular smooth muscle cells hypercontractility, endothelial dysfunction, autonomic nervous system activity, and oxidative stress. Triggers include mental stress, smoking, alcohol consumption, consumption of beta-blockers, and sympathomimetic agents. Management of vasospasm targets these mechanisms.[1,15]

Management of lifestyle and correction of risk factors consist of smoking cessation, BP control, maintenance of ideal body weight, correction of impaired glucose tolerance, correction of dyslipidemia, and avoidance of mental stress and vasospastic agents.[1,15]

Calcium channel blockers (CCB) and nitrates remain the frontline pharmacotherapy. CCB suppress calcium inflow into vascular smooth muscle cells are highly effective in preventing coronary spasm. Both non-DHP CCB and DHP-CCB have been reported to be effective. They work on peripheral arteries to induce vasodilation and on the myocardium via inhibition of Ca influx through L-type calcium channels.[1,15,16]

Nitrates could be administered either sublingually, spraying in the oral cavity, or intravenous administration during an attack. Long-acting nitrates (LAN) could prevent episodes of coronary spasm.[1] Nitrate is metabolized to NO, activates cGMP and promotes vasodilation; it also reduces ventricular filling pressures through venodilation and decreases myocardial oxygen demand. Nitrates have a different mechanism of action than CCB, therefore, a combination of CCB and LAN is effective for reducing vasospastic angina, although CCB is typically preferred over LAN due to potential nitrate tolerance.[15,16]

The use of beta-blockers is limited to vasospastic angina with CAD with significant stenosis, concomitantly with first-line vasodilator agents. Monotherapy of beta-blockers for vasospastic angina without significant stenosis of coronary artery is not advised (class III).[1]

The use of antiplatelet in vasospastic angina without concomitant CAD is still conflicting. A high dose of aspirin blocks the production of prostacyclin, a potent vasodilator. A low dose of aspirin blocks thromboxane A2, a potent vasoconstrictor, therefore it appears to be safe and may be effective in preventing acute attacks.[15,16] Study by Mori et al about the impact of antiplatelet on patients with vasospastic angina, including low-dose aspirin and P2Y12 inhibitors, reported that antiplatelet therapy had no marked impact on MACE.[17] Coronary artery spasms are often accompanied by hypercoagulation, decreased fibrinolytic activity, and activation of platelets and adhesion molecules, resulting in a thrombophilic state in ACS.[1] Therefore, antithrombotic therapy might be useful in the prevention and treatment of coronary artery spasms.

Coronary spasms could lead to sudden cardiac death related to ventricular arrhythmias. ICD implantation might have a role in high-risk patients surviving life-threatening arrhythmias. A study by Matsue et al demonstrated a high recurrence rate of ventricular arrhythmias in vasospastic angina patients and revealed the clinical implications of ICD in a high-risk population. Predicting the risk of recurrent arrhythmias is difficult, indicating ICD therapy would be appropriate for all patients resuscitated from ventricular arrhythmias even if the symptoms are well controlled by medication.[8,15,19]

In this case, the use of ICD should be considered since the patient had a history of resuscitated life-threatening VT.

CONCLUSION

Coronary artery vasospasm could be the etiology of acute myocardial infarction and life-threatening malignant ventricular arrhythmia, even without concomitant significant coronary artery disease. Comprehensive management with lifestyle changes, avoidance of risk factors and triggers, along with optimal medical therapy with CCB and nitrates as first-line options are important. The use of low-dose antiplatelets should be considered because coronary artery spasms are often accompanied by several natures including hypercoagulation, decreased fibrinolytic activity, and activation of platelets and adhesion molecules. Implantation of ICD should be considered in a patient who survived life-threatening arrhythmias to prevent sudden cardiac death.

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