

A rare cause of cardiogenic shock: variant angina

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Abstract: Variant angina (VA) is a clinical syndrome caused by spontaneous vasospasm of the epicardial coronary artery which is characterized by episodes of angina. Endothelial dysfunction and neurohormonal hyperactivity are important factors in pathogenesis of VA. Although patient prognosis is good, VA may be one of the reasons of sudden cardiac death (SCD) in case of persistent ST segment elevation and malignant arrhythmias. Therefore, early treatment of VA is crucial for prevention of malignant arrhythmias and SCD. In this case report we describe a case of VA presented with cardiogenic shock and malignant ventricular arrhythmia.

Keywords: cardiogenic shock, coronary vasospasm, variant angina

Introduction

Variant angina (VA) is a clinical syndrome caused by spontaneous vasospasm of epicardial coronary artery which is characterized by episodes of angina usually at rest, between midnight and early morning. Although patient prognosis is good, VA may be one of the reasons of sudden cardiac death (SCD) in case of persistent ST segment elevation and malignant ventricular arrhythmias. In this case report we describe a case of VA presented with cardiogenic shock and malignant ventricular arrhythmia.

Case presentation

A 58-year-old woman was presented to the emergency department with severe chest pain that had begun 30 minutes ago. On admission, she was unconscious, her blood pressure was 70/40 mmHg and heart rate was 50 beats per minute (bpm). While she was being monitored, cardiac arrest occurred due to ventricular fibrillation. She was immediately cardioverted with 360 J. Electrocardiography revealed 45 bpm nodal rhythm, 2 mm ST segment elevation in inferior leads (D₂, D₃, aVF) and 2–3 mm ST segment depression in anterior leads (aVL, V₂₋₅) (Figure 1).

The patient was transferred urgently to the coronary angiography (CAG) laboratory. CAG

revealed normal left coronary arteries and diffuse critical stenosis of the right coronary artery (RCA) (Figure 2a–b). When a 0.014 inch floppy guide wire was inserting in the RCA, prominent coronary vasospasm was revealed (Figure 2c). After intracoronary nitroglycerine (100 µg) was injected, control images revealed non-critical coronary artery plaques in the RCA (Figure 2d).

The patient had a history of hypertension for 9 years. She had no family history of cardiovascular disease or SCD. She was on medical treatment with perindopril 5 mg daily. The patient had no history of smoking, alcohol or drug abuse. Transthoracic echocardiography revealed normal left ventricular systolic function (ejection fraction: 70%) with hypokinesis of inferior wall and mild mitral regurgitation. Although optimal medical therapy was given, the patient during hospitalization [diltiazem 240 mg by mouth (po) and isosorbid-5-mononitrat 60 mg po], severe episodes of angina repeated. An intracardiac defibrillator (ICD) was inserted for secondary prevention of malignant arrhythmias. The patient was discharged 10 days later. She was evaluated a month later with no complaint of chest pain. ICD records were also within normal range during controls.

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Figure 1. Surface 12-leads electrocardiography shows 45 beats per minute (bpm) nodal rhythm, 2 mm ST segment elevation in inferior leads (D₂, D₃, aVF) and 2–3 mm ST segment depression in anterior leads (aVL, V₂₋₅).

Discussion

Variante angina was first described by Prinzmetal in 1959 [Roguin, 2008; Prinzmetal *et al.* 1959]. He described this type of angina as a disease of spontaneous vasospasm of major coronary arteries due to increased vessel tonus. VA is usually seen in patients between the ages of 35 and 50, especially in women. CAG reveals normal findings in about 70% of VA episodes. Although its pathogenesis is unclear, endothelial dysfunction and neurohormonal hyperactivity are important factors in the pathogenesis of VA [Prinzmetal *et al.* 1959]. Previous studies have shown that several drugs [Acikel *et al.* 2010] and cardiac arrhythmias [Wang *et al.* 2008] can trigger the VA. Malignant ventricular arrhythmias such as ventricular tachycardia and ventricular fibrillation may cause of VA in about 5–15% of cases. Previous studies showed that QT dispersion and abnormal ventricular repolarization are important factors for malignant arrhythmias [Myerburg *et al.* 1992; Parchure *et al.* 2001]. VA may also cause of myocardial infarction in case of coronary thrombosis [Maseri *et al.* 1978]. Early treatment of VA is crucial for prevention

of malignant arrhythmias and SCD. Nitrate and calcium channel blocker therapy are two essential drugs for termination of the acute angina episodes. Beta-blockers and acetylsalicylic acid should be avoided because they can trigger coronary vasospasm [Lanza *et al.* 2007]. The role of ICD for prevention of SCD in patients with VA is controversial. However, patients who are symptomatic with optimal medical therapy should be considered for ICD implantation [Matsue *et al.* 2012].

In conclusion, our case report has several important learning points: a) all physicians performing coronary angiography should keep in mind that some of the stenotic images may be due to coronary vasospasm which need to be tested with intracoronary nitrate; (b) VA might also be cause of cardiogenic shock and SCD; and (c) acute angina episodes and malignant arrhythmias may continue even when optimal medical therapy is given. Therefore, physicians should keep in mind that ICD implantation may be required for secondary prevention of malignant arrhythmias in those patients.

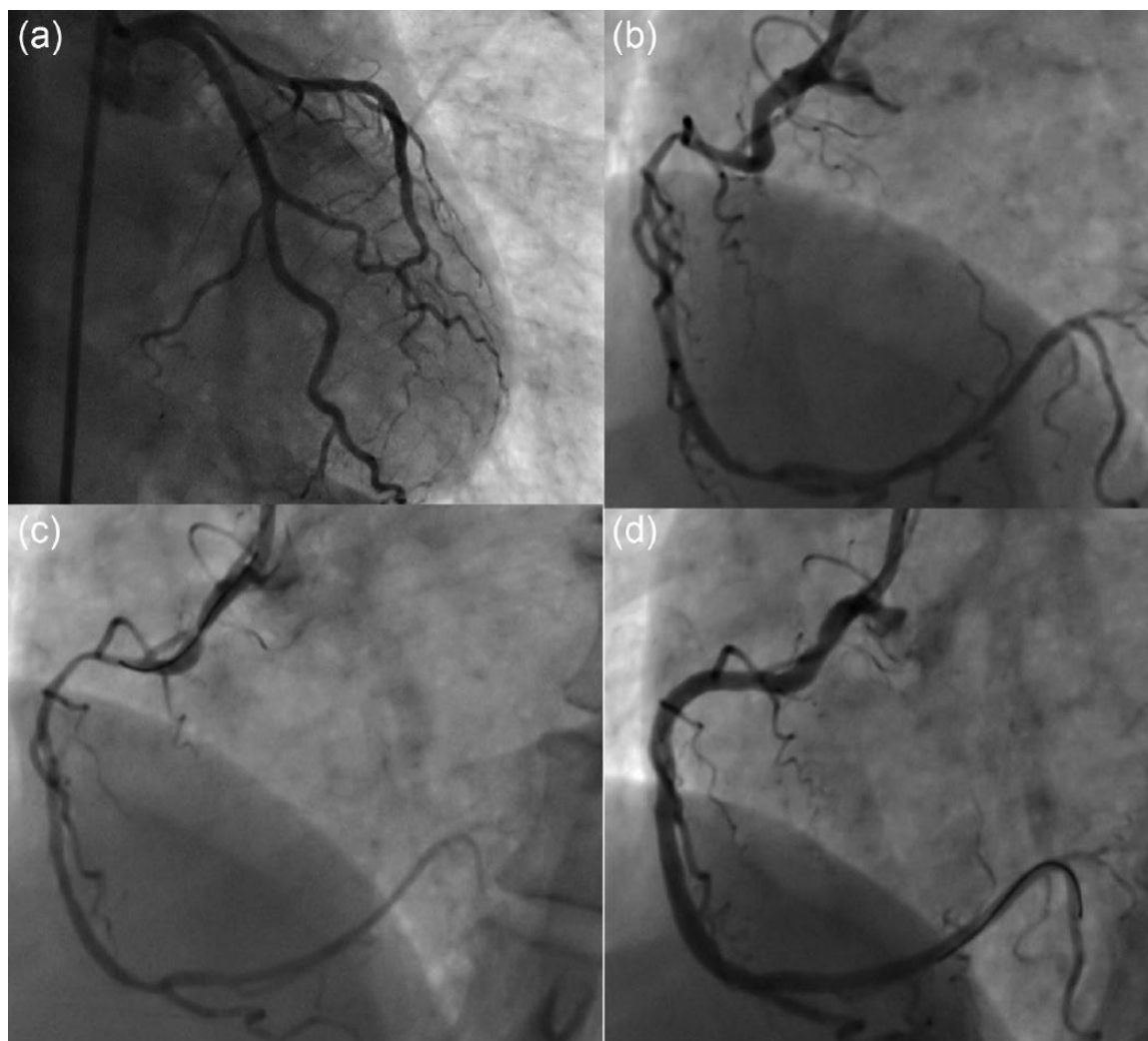


Figure 2. Coronary angiography shows: (a) normal left coronary arterial system; (b) diffuse critical stenosis of the right coronary artery; (c) prominent coronary vasospasm after guide wire is inserted; and (d) non-critical coronary plaques after intracoronary nitroglycerine injection.

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Conflict of interest statement

The authors declare no conflicts of interest in preparing this article.

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