Spontaneous and Simultaneous Multivessel Coronary Spasm Causing Multisite Myocardial Infarction, Cardiogenic Shock, Atrioventricular Block, and Ventricular Fibrillation

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A 57-year-old Taiwanese man with a past history of variant angina developed simultaneous anterior and inferior myocardial infarction, atrioventricular block, cardiogenic shock, and eventually ventricular fibrillation. Left coronary angiography revealed simultaneous occlusion of the left anterior descending and the left circumflex coronary arteries, which was relieved by intracoronary administration of isosorbide dinitrate. This is the first report of such a case in the English-language medical literature. (Circ J 2009; 73: 1961–1964)

Key Words: Acute myocardial infarction; Cardiac arrest; Implantable cardiac defibrillators; Variant angina

Coronary artery spasm (CAS) is a well-documented etiology of variant angina, which generally occurs when a patient is at rest and is usually transient. However, in some cases prolonged CAS can lead to myocardial infarction (MI), malignant arrhythmias, sudden cardiac death, and even cardiac rupture. Spontaneous and simultaneous multivessel CAS presenting with multisite MI, atrioventricular block, acute lung edema, cardiogenic shock and ventricular fibrillation (VF) has never previously been reported in a single case.

Case Report

A 57-year-old Taiwanese man with a previous history of hypertension, type 2 diabetes mellitus, and variant angina (treated by amlodipine 5 mg before sleep with good compliance) presented to the emergency department with severe chest pain and cold sweating in the early morning hours. Surface electrocardiography (ECG) on admission showed atrioventricular block and widespread ST-segment elevations in the anterior and inferior leads (Figure 1, ECG #1). Chest radiography showed bilateral pulmonary edema (Figure 2A): heart rate was 44 beats/min, blood pressure was 72/44 mmHg, and he was breathing at a rate of 32/min, with oxygen saturation approximately 80% on room air. While he was on nasal oxygen (3 L/min), blood gas analysis revealed significant metabolic acidosis with pH 7.33, PaO2: 56 mmHg, PaCO2: 23.3 mmHg, HCO3: 12.1 mmol/L and base excess −13.8. He was intubated and put on mechanical ventilation, inotropic support, and intra-aortic balloon pumping. Blood tests obtained immediately after arrival showed troponin-I of 2.12 ng/ml (normal <0.03 ng/ml). Blood creatine kinase (CK)-MB fraction level (CK: 334.0 IU/L [normal, 32–180 IU/L]; MB: 35.0 IU/L [normal <10.8 IU/L]) on arrival was elevated, with the peak level (CK: 1,225.5 IU/L; MB: 132.4 IU/L, respectively) reached at 20 h after admission. Transthoracic echocardiography showed global akinesis and impaired left ventricular function, with an ejection fraction (EF) of 28%. At 10 min after arrival, he developed VF (Figure 1, ECG #2). He was successfully resuscitated without apparent neurological sequelae by immediate defibrillation. Immediately after defibrillation, surface ECG revealed partial resolution of the ST-segment elevation, and sinus tachycardia with restoration of atrioventricular conduction (Figure 1, ECG #3). Emergency coronary angiography showed simultaneous CAS of the entire left anterior descending artery (including the diagonal branches) and the left circumflex (LCX) artery (including obtuse marginal branches), with almost complete obliteration of the lumen of each vessel (Figures 3A, B). After intra-coronary administration of isosorbide dinitrate, there was normalization of the coronary artery diameters (Figures 3C, D) and ECG abnormalities (Figure 1, ECG #4). The non-dominant right coronary artery was normal (Figures 3E, F). The angina did not recur after administration of intravenous nitrates for 2 days, with normalization of CK-MB and troponin I in the following days. On the 5th hospital day, he received an implantable cardioverter defibrillator (ICD) and was started on oral administration of amlodipine at a dose of 5 mg OD before sleep, diltiazem at a dose of 240 mg before sleep, and long-acting nitrates. Chest radiography (Figure 2B) on the 3rd of admission showed improvement of pulmonary edema. Predischarge echocardiography revealed a left ventricular EF of 55% with normal wall motion. During a follow up period of 30 months, the patient was free of symptoms.

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Figure 1. (ECG#1). Surface ECG demonstrating diffuse ST-segment elevation in the inferior and precordial leads and complete atrioventricular block with junctional escape rhythm at 44 beats/min. (ECG#2) Shortly after arrival at the emergency department, ventricular fibrillation developed. (ECG#3) Immediately after DC shock, the surface ECG shows partial resolution of ST-segment elevation and restoration of atrioventricular conduction. (ECG#4) The final ECG shows complete resolution of ST-segment elevation after intracoronary administration of isosorbide dinitrate. ECG, electrocardiogram.
Discussion

Prolonged CAS without underlying coronary artery stenosis leading to acute MI has been widely reported, but to our knowledge, simultaneous multiple CAS causing multisite MI, atrioventricular block, acute lung edema, cardiogenic shock, and VF has not been reported in the English-language medical literature. In the present case, the spontaneous and simultaneous spasm in the left coronary arterial system was rare because it was generalized (involving the whole length of both arteries, including the branches), and intense (causing almost complete obliteration of the vessel lumen). The territory involved in the spasm was equivalent to an acute left main occlusion. Consequently, the patient...
developed lung edema, cardiogenic shock and VF that required defibrillation, mechanical ventilation, inotropic support, and intra-aortic balloon pumping. Whether the non-dominant right coronary artery was also involved during the generalized spasm is unknown. The LCX in the present patient was a relatively large artery, and may account for the ST-segment elevation in the inferior ECG leads.

Prognosis of patients with MI caused by prolonged CAS in the absence of significant coronary artery disease appears to be generally good.\textsuperscript{5,6} In many cases, vasospasm responds to calcium-channel blockers and nitrates, but life-threatening rhythm disorders, including both bradyarrhythmias (such as atrioventricular block) and tachyarrhythmias (such as VF), may complicate transmural MI caused by prolonged CAS, possibly resulting in cardiac arrest or sudden death. Given that the present patient developed multivessel CAS and VF while being treated with amlodipine, which supported the finding by Meune et al\textsuperscript{17} of frequent residual spasm despite treatment with high-dosage calcium-channel blockers in a prospective study on out-of-hospital cardiac arrest, ICD may be helpful for preventing recurrent fatal events. Simcha et al reported that a patient with variant angina complicated by VF was at life-long risk for sudden death when exposed to myocardial ischemia.\textsuperscript{8} Moreover, as reported previously,\textsuperscript{9,10} multivessel CAS, evidenced by diffuse ST-segment elevation, is also associated with an increased risk of life-threatening cardiac events. The present case highlights the fact that, although uncommon, when it occurs multivessel CAS may increase the risk of life-threatening cardiac events (particularly VF) and there should be careful consideration of ICD implantation. Indeed, medical control of CAS using calcium-channel blockers and/or nitrates remains crucial in these patients to avoid angina recurrence, painful ICD discharges and possible clinical deterioration consequent to recurrent and prolonged attacks of myocardial ischemia, as evidenced by the recent case report of persistent CAS that eventually resulted in death of the patient despite ICD placement.\textsuperscript{11} In conclusion, multivessel CAS may lead to multisite MI, atrioventricular block, acute lung edema, cardiogenic shock, and VF in a patient without significant coronary or other structural heart diseases. Such patients need to be aggressively treated with vasodilators, and require careful consideration of ICD placement.

**Disclosure**

None.

**References**