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Case Report

# Out-of-hospital cardiac arrest related to coronary arterial spasm in three elderly patients with no obstructive coronary artery disease

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ABSTRACT

Coronary spastic angina (CSA) is relatively more common in young people than in elderly people. Here, we present three cases of elderly male patients who experienced out-of-hospital cardiac arrest (OHCA) likely due to coronary spasm-induced ventricular fibrillation (Vf) from 2013 to 2016. After defibrillation, emergency coronary arteriography demonstrated severe coronary vasospasm that resolved following intracoronary infusion of nitroglycerin in the right coronary arteries in all three patients, with no organic obstructive lesion in the coronary arteries after nitroglycerin infusion. Case 1 was a 74-year-old patient with a past history of unstable angina and no organic obstructive lesion on coronary arteriography. He was administered oral amlodipine, isosorbide mononitrate, and nicorandil. He survived an OHCA and underwent implantable cardioverter defibrillator (ICD) implantation on day 57. Case 2 was a 71-year-old patient without prior CSA, who suddenly lost consciousness during a break after tennis. Vf was reversed to sinus rhythm by defibrillation in the ambulance. He died of multi-organ failure on day 7. Case 3 was a 66-year-old patient diagnosed with multi-vessel CSA by coronary arteriography with acetylcholine provocation test. He survived an OHCA associated with inferior acute myocardial infarction, rejected ICD implantation, and has not had a chest pain attack or syncope since discharge.

<**Learning objective:** This article reports a case series of out-of-hospital cardiac arrest (OHCA) likely due to coronary spastic angina (CSA) in the presence of non-obstructive coronary artery disease in elderly patients. Although CSA is associated with an increased risk of OHCA, little is known regarding clinical risk factors, the effectiveness of implanted defibrillators for the secondary prevention of cardiac arrest, or the long-term prognosis of elderly CSA patients who survive OHCA.>



## Introduction

Patients with coronary spastic angina (CSA) are often younger and exhibit fewer classic cardiovascular risk factors except for smoking [1]. In general, patients with CSA who respond to calcium channel blockers have a good prognosis; consequently, the 5-year-survival rate of CSA is more than 90% [2,3]. In a Japanese registry study including 1429 patients with CSA [4], 5.9% reached the primary major adverse cardiovascular event (MACE) combined endpoint [cardiac death, nonfatal myocardial infarction, unstable angina, heart failure, or implantable cardioverter defibrillator (ICD) shock] during a median follow-up of 32 months [4]. Patients with CSA who have obstructive coronary artery disease have a poorer prognosis [4,5].

Here, we present three cases of out-of-hospital cardiac arrest (OHCA) likely due to coronary spasm in the presence of non-obstructive coronary artery disease in men older than 65 years. We consider the prognosis of elderly CSA patients and the association of CSA with OHCA.

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*Case 1*

A 74-year-old male patient with history of unstable angina was admitted to our hospital for emergency cardiac catheterization. Coronary angiography (CAG) indicated no organic stenosis in any coronary artery. Although an acetylcholine provocation test was not performed, the patient was diagnosed with CSA based on chest pain characteristics. The patient was administered oral 5 mg of amlodipine, 40 mg of isosorbide mononitrate, and 15 mg of nicorandil per day.

Six months after the previous admission, he was found lying on the stairs in cardiac arrest at 8:00 AM, and his family called an ambulance. He was a current smoker. Electrocardiogram (ECG) showed ventricular fibrillation (Vf), and after successful defibrillation at 8:10 AM, he was transported to a nearby hospital.The arrival time was not recorded. ECG at that time did not show ST-T change, but a second ECG performed at 11:00 AM showed ST elevation in leads II, III, and aVf. He was referred to our hospital, where, on arrival at 12:45 PM, ECG again showed Vf and that lasted until 1:20 PM. Immediately after defibrillation, blood pressure was 64/34 mmHg, and pulse was 128 beats/min. ECG showed sinus tachycardia; ST-segment elevation in leads II, III, and aVf; and ST-segment depression in leads V1–V3 (Fig. 1a). Four hours after admission, ECG showed ST-segment recovery in leads II, III, aVf, and V1–3 (Fig. 1b). Emergency CAG on admission showed no significant stenosis of the left coronary artery (LCA) (Fig. 1c), and diffuse coronary vasospasm of the right coronary artery (RCA), particularly its distal segment. Coronary vasospasm resolved following intracoronary infusion of nitroglycerin and sodium nitroprusside (Fig. 1d,e). There was no significant stenosis of the coronary artery. On hospitalization day 57, the patient underwent ICD implantation. At the time of discharge on day 70, he had no neurological deficit and was free from all angina symptoms. His most recent prescription regimen is as follows: 8 mg of benidipine, 15 mg of nicorandil, 100 mg of amiodarone, and 10 mg of nitroglycerine tape per day. He has not suffered from Vf since discharge.

*Case 2*

A 71-year-old male patient was playing tennis and suddenly lost consciousness during a break, at 11:08 AM. There was no bystander cardiopulmonary resuscitation. An ambulance was called at 11:10 AM. On arrival of the ambulance at 11:17 AM, ECG showed Vf. Pulseless electrical activity continued after automated external defibrillator (AED) shock. After transport to a nearby hospital, Vf recurred, and electrical defibrillation was performed. Vf disappeared at 11:20 AM. ECG showed ST elevation in lead aVR and III; R wave decrease in leads V1–3; andST-segment depression in leads I, II, aVL, and V4–6. He was transported to our hospital by ambulance at 11:37 AM.

The patient had dyslipidemia and hypertension, but was not prescribed any medication for these conditions. He had experienced no chest pain episodes. He was a current smoker, having smoked 20 cigarettes per day for 41 years.He was intubated,and had blood pressure of 108/73 mmHg, pulse of 72 beats/min, and oxygen saturation of 98% (10 L/min $O_2$). ECG showed sinus rhythm with pulse of 57 beats/min; ST elevation in lead aVR, III; R wave decrease in leads V1–3; and ST-segment depression in leads I, II, aVL, and V4–6 (Fig. 2a). Emergency CAG on admission showed diffuse coronary vasospasm of the RCA, particularly its distal segment, which resolved following intracoronary infusion of nitroglycerin and sodium nitroprusside (Fig. 2c,d). Left coronary angiography was then performed, and no significant stenosis was observed (Fig. 2e). Five hours after admission, ECG showed ST-segment recovery in leads aVR and III, ST-segment depression in leads V5–6 and negative T wave in leads II, III, and aVf (Fig. 2b). On day 3, he was still in a coma and his blood pressure decreased, with systolic blood pressure of 60–80 mmHg and hypouresis. An echocardiogram showed diffuse hypokinetic wall motion especially in the broad anterior wall, with ejection fraction of 35%. We started dobutamine, dopamine, and noradrenaline, but hypotension continued. The serial changes in serum creatine kinase levels were 1922 IU/L on admission, 6070 IU/L on day 2, 5640 IU/L on day

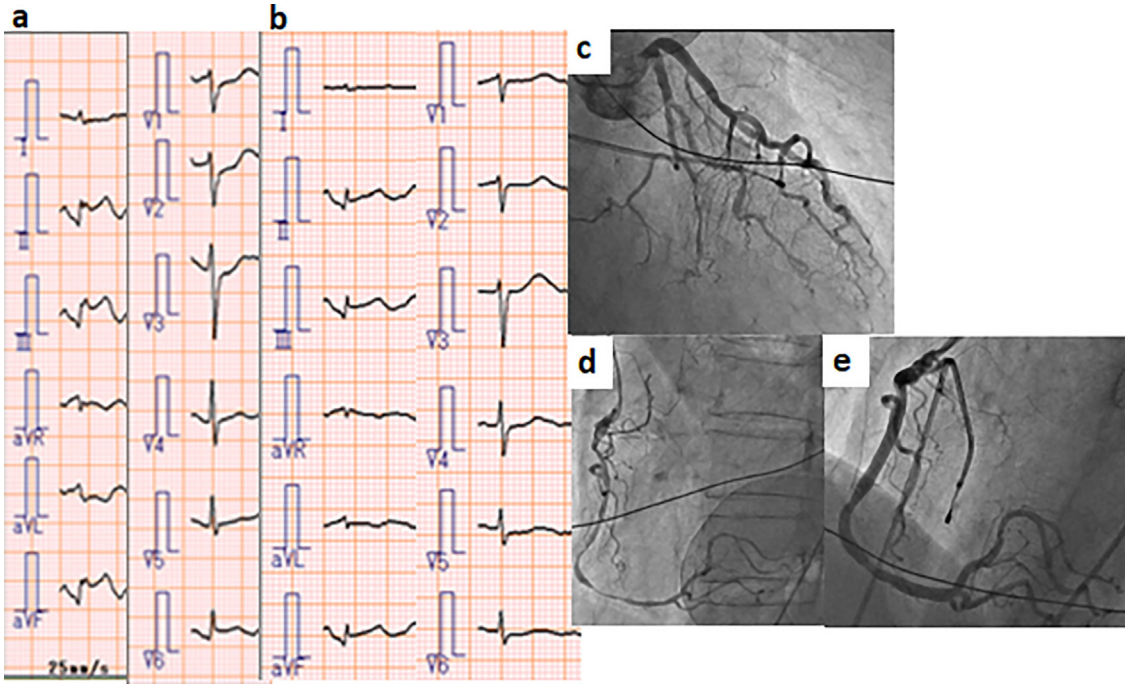


**Fig. 1.** (a) Electrocardiogram (ECG) of Case 1 on admission showed ST-segment elevation in leads II, III, aVf, V5, and V6 and ST-segment depression in leads V1–V3. (b) ECG after relief of ST elevation. (c) Results of coronary angiography (CAG) in Case 1 showing normal left coronary artery. (d) CAG in Case 1 showing severe diffuse vasospasm of the right coronary artery. (e) The vasospasm was relieved after intracoronary infusion of nitroglycerin and sodium nitroprusside.

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