

[CASE REPORT]

Cardiogenic Shock due to Pulseless Electrical Activity Arrest Associated with Severe Coronary Artery Spasm

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Abstract:

A 75-year-old man was admitted to our hospital for follow-up coronary angiography. Just after starting coronary angiography, his electrocardiogram showed ST-segment elevation in the V1-6, I, II, and aVF leads, and he fell into catastrophic cardiogenic shock. His left coronary arteriogram showed proximal total obstruction in the left anterior descending artery and proximal subtotal occlusion in the left circumflex artery. Because pulseless electrical activity arrest was recognized, cardiopulmonary support was started. After more than 15 minutes' cardiac massage, his blood pressure gradually returned to baseline. During the cardiogenic shock due to pulseless electrical activity arrest, neither ventricular fibrillation nor ventricular tachycardia was recognized.

Key words: cardiogenic shock, coronary spasm, pulseless electrical activity arrest, aborted sudden cardiac death

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Introduction

Coronary artery spasm may be involved in the genesis of various cardiac disorders, such as acute coronary syndrome, sudden cardiac death, unstable angina, serious fatal arrhythmia, transient heart failure, and atypical chest symptoms. Serious fatal arrhythmia such as ventricular fibrillation or tachycardia is often observed in patients with aborted sudden cardiac death due to coronary spasm. Implantable-cardioverter defibrillator (ICD) was found to be effective in suppressing the next serious fatal event in patients with coronary spastic angina as well as structural heart disease (1). However, some patients with pulseless electrical activity (PEA) arrest due to coronary spasm die after the implantation of ICD (2-6). We herein report a case of spontaneous severe coronary spasm that was resuscitated from catastrophic cardiogenic shock due to severe spasm accompanying PEA arrest.

Case Report

A 75-year-old man was admitted to our hospital for

follow-up coronary angiography after the implantation of a drug-eluting stent (Ultimaster 3.0×38 mm & Xience Alpine 2.5×23 mm) into the left anterior descending artery approximately 1 year earlier. He had undergone coronary angiography four times, including coronary intervention three times, before this admission. He had quit smoking 10 years earlier and had hypertension, dyslipidemia and diabetes mellitus. He had no typical chest pain but some atypical chest discomfort at rest during daily life. He had not been using sublingual nitrates when he complained of atypical chest discomfort. He had ventricular fibrillation treated with direct current possibly due to a catheter wedge in the right coronary artery during follow-up coronary angiography nine years earlier. He was medicated with antiplatelets (ticlopidine 200 mg), aspirin (100 mg), angiotensin-receptor blockers (telmisartan 20 mg), statins (atrovastatin 10 mg) and beta-blockers (carvedilol 2.5 mg). Cardiac thallium scintigraphy showed slight partial redistribution on the anterior lesion.

We started coronary angiography at 9:00 AM without premedication. We used the same local anesthetic drug (xylocaine injection polyamp 1%; AstraZeneca, Osaka, Japan) on the four previous occasions. His electrocardiogram (ECG)

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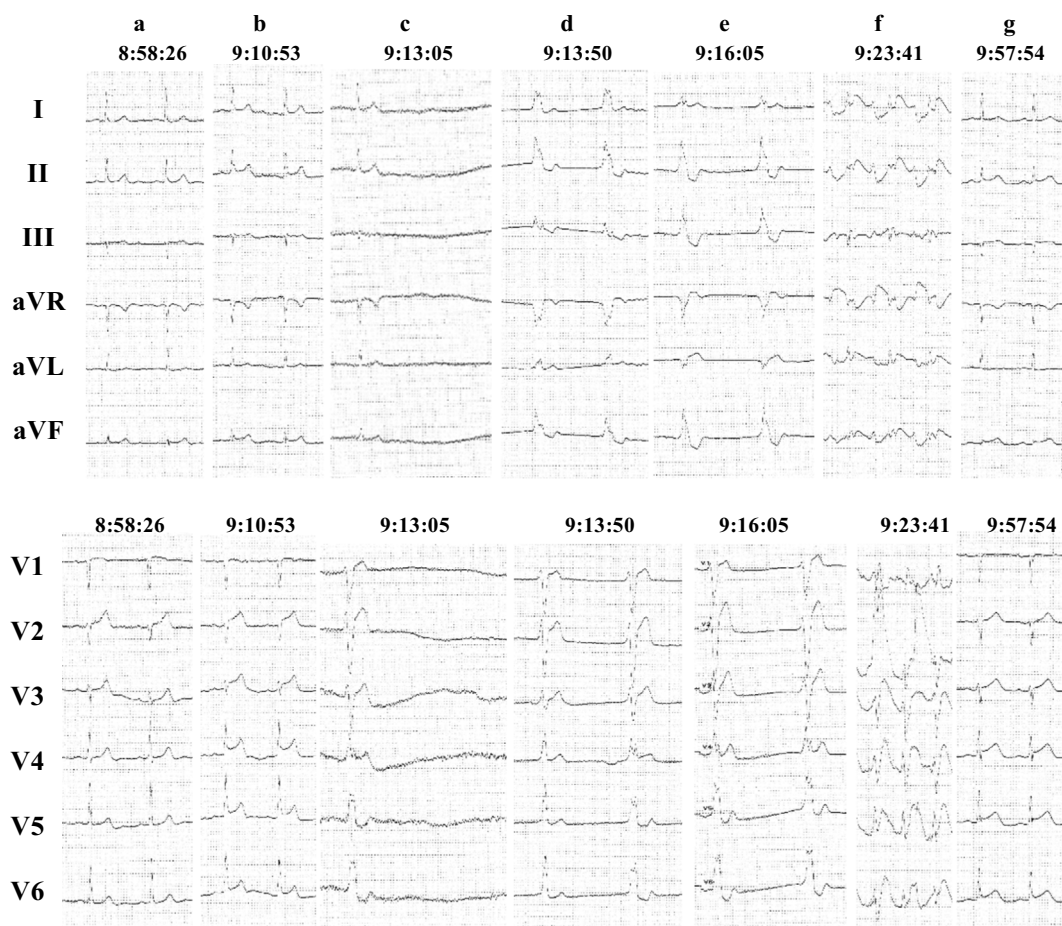


Figure 1. Serial changes in the ECG findings during cardiogenic shock due to severe spasm.

had no significant ST-T changes, and chest symptoms were not recognized (Fig. 1a). His blood pressure was 140/80 mmHg, and his heart rate was 80/min (Fig. 2a). ST-segment elevation in the V1-6, I, II, and aVF (Fig. 1b) leads and a decreased blood pressure (60/40 mmHg) (Fig. 2b and c) were observed after the insertion of a diagnostic catheter (5-Fr Terumo outlook™ JL 3.5, Terumo, Tokyo, Japan) into the ascending aorta.

We performed left coronary angiography first. His left coronary angiogram showed total obstruction at the proximal left anterior descending artery and subtotal occlusion at the proximal left circumflex artery, as shown in Fig. 3a. Contrast medium into the left coronary artery still remained for a few minutes (Fig. 3b). He lost consciousness and fell into cardiac arrest (Fig. 1c, Fig. 2d and e).

Cardiac massage was promptly performed. However, despite the administration of adrenaline/etilefrine as well as nitroglycerine (Fig. 2c, e and g), he did not recover from the cardiogenic shock, and the PEA arrest continued (Fig. 2f and g). Wide QRS changes were observed on ECG during the shocks (Fig. 1d and e). After more than 15 minutes' cardiac massage, his blood pressure gradually increased (Fig. 2h). His left coronary arteriogram showed no fixed stenosis (Fig. 3c and d) and his right coronary artery had no stenosis (Fig. 3e). Tachycardia, possibly due to the administration of adrenaline, was recognized (Fig. 1f). His

blood pressure recovered to the baseline value after 18 minutes (Fig. 2i), although ST-segment elevation was observed in the V4-6, II, III, and aVF leads even after 40 minutes (Fig. 1g). We inserted an intra-aortic balloon to improve the coronary flow and prevent severe spasm for one night. The intravenous administration of nitroglycerine and nicorandil was started to prevent a second catastrophic event. Under additional medications of diltiazem R (200 mg twice daily), benidipine (8 mg twice daily), and nitrate tape (40 mg), he had neither chest pain nor ischemic ECG changes on monitoring.

He was discharged in good condition after two weeks' admission.

Discussion

In this article, we reported a case of PEA arrest due to severe coronary artery spasm. Spontaneous severe multiple spasm caused catastrophic cardiogenic shock without complications of fatal ventricular tachycardia or fibrillation. Multiple spasms may lead to severe ischemia associated with serious life-threatening ventricular arrhythmia. However, the present case showed no ventricular fibrillation or tachycardia during cardiogenic shock due to severe spasm. Contrast medium stayed in the left coronary artery for a prolonged period of time. We administered the intracoronary

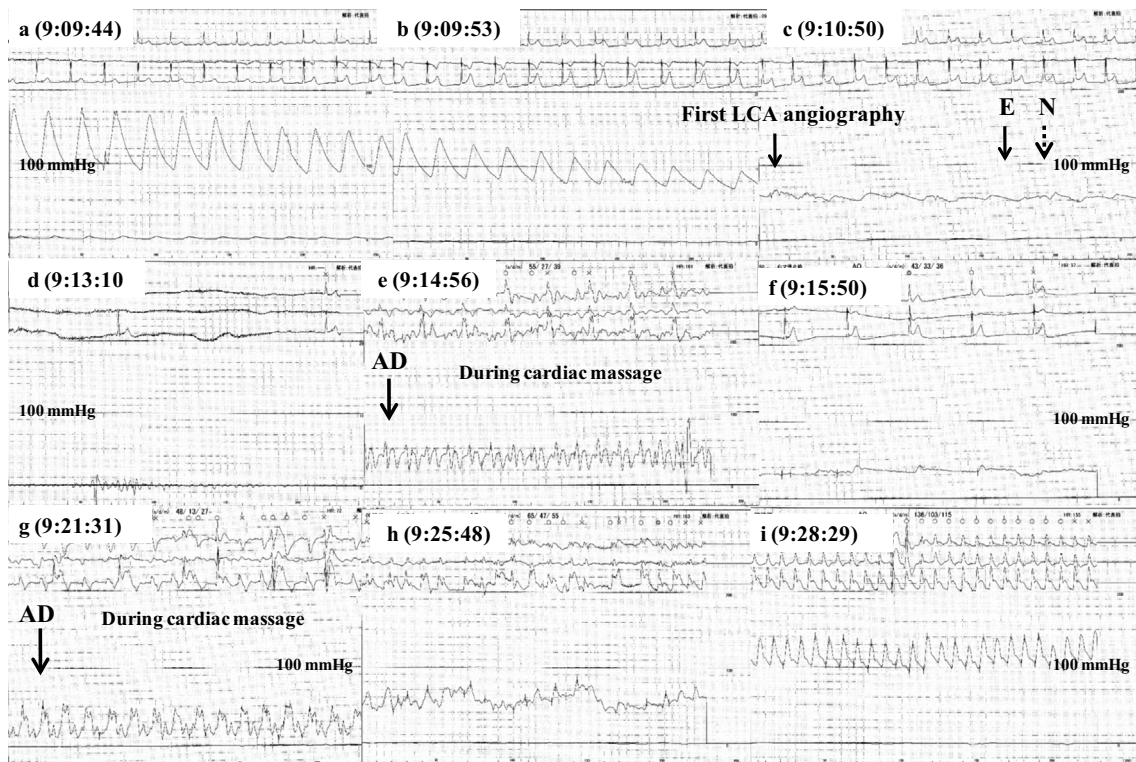


Figure 2. Serial changes in the ECG findings and blood pressure during cardiogenic shock due to severe spasm.

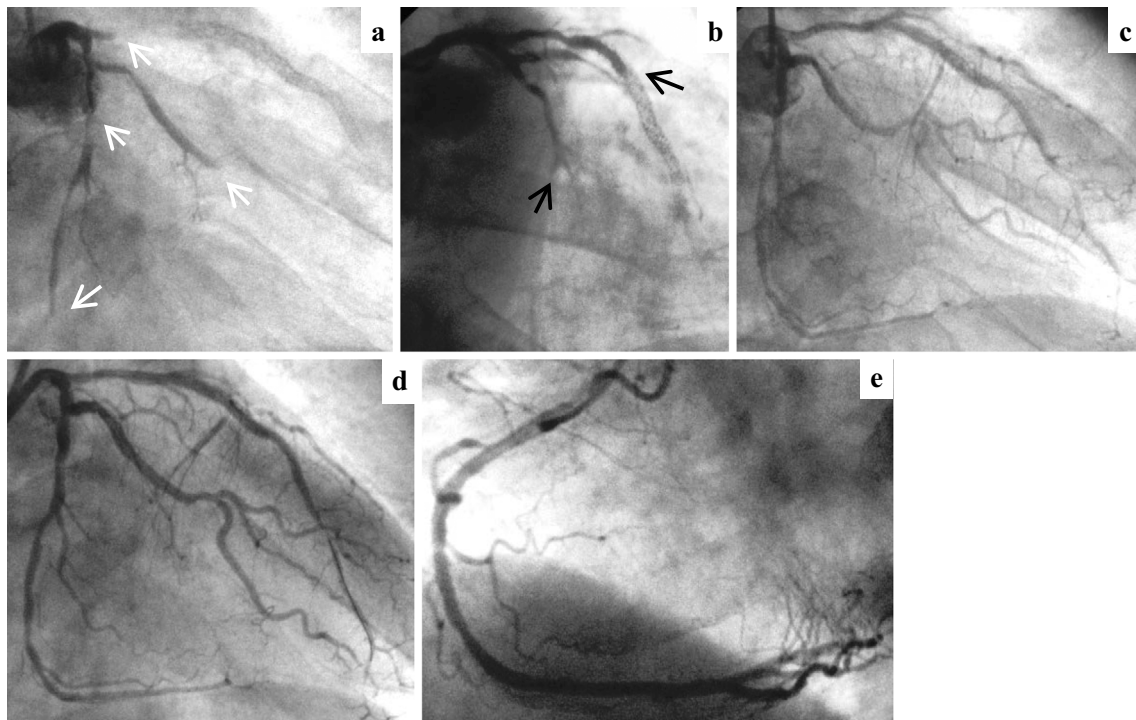


Figure 3. Coronary arteriograms during cardiogenic shock. a: Total and subtotal spasm were observed at the proximal left anterior descending artery or proximal left circumflex artery. b: Contrast medium was observed at the proximal left coronary artery. c: Severe coronary artery spasm was relieved after 15 minutes' cardiopulmonary support. d: No fixed stenosis was found after the insertion of a drug-eluting stent. e: Normal coronary arteriogram in the right coronary artery.

injection of nitroglycerine into the left coronary artery. PEA arrest. Triple-vessel severe spasms can cause the heart to suddenly stop beating. However, the coronary flow was not recognized because of to suddenly stop beating. Despite continuous cardiac mas-

sage, the blood pressure did not increase. Multiple severe spasms may freeze the cardiac muscle in an instant. Prolonged continuous cardiac massage for 15 minutes was effective for successfully resolving catastrophic cardiogenic shock due to severe spasm.

Even if an ICD had been implanted in this case, whether or not this patient might have avoided such catastrophic event is debatable. Cardiac pacing alone might not restore frozen myocardium to viable muscle. Indeed, some patients with coronary spasm die regardless of the implantation of an ICD. In these patients, PEA arrest may lead to unexplained death after the implantation.

The present patient had never complained of resting angina, but he did report some atypical chest discomfort. We observed the severe multiple coronary spasm by chance on follow-up coronary angiography. This might have been his first severe attack. However, the same attack outside of a hospital may have led to unexplained sudden cardiac death. In the real world, there may be some patients who experience typical spasms without severe chest symptoms during their daily life. The administration of a beta-blocker might have been the cause of his atypical chest discomfort.

The right coronary artery showed no spasm after the severe spasm in the left coronary artery was relieved, although ST-segment elevation in the inferior leads continued for more than 40 minutes. However, we deduced that severe triple-vessel spasm occurred spontaneously in this case, suddenly stopping all coronary flow. This may have led to the flash-freezing of the entire myocardium. Ventricular fibrillation did not occur in this case, as the coronary flow of the three major coronary arteries stopped all at once. Myocardial ischemia due to coronary artery spasm may lead to the occurrence of lethal ventricular arrhythmia like acute coronary syndrome, while some cases of triple-vessel severe spasm can cause PEA. The broad extent of ischemia may cause PEA or asystole without lethal ventricular arrhythmias. However, the precise mechanism involved may differ among patients, ischemia of the sinus node artery or atrioventricular node artery can influence the occurrence of PEA or asystole.

Matsue et al. reported that 5 patients reached the endpoints, including 4 patients with appropriate therapy by ICD and 1 patient with PEA, during the 2.9-year follow-up period among 23 patients with an ICD inserted for lethal arrhythmia and vasospastic angina. All four patients with ventricular fibrillation were treated appropriately by ICD and resuscitated. One patient presented to the hospital with sudden cardiac arrest after chest pain, and electrocardiography at the emergency department showed PEA. An intracardiac electrogram showed no ventricular arrhythmia, and this patient was successfully resuscitated (7). ICD implantation was shown to be useful for preventing the next life-threatening ventricular arrhythmia, such as ventricular fibrillation or tachycardia, in patients with coronary spasm as well as structural heart disease. In previous report, however, one refractory patient died of ventricular fibrillation regardless of

appropriate ICD shocks (8).

Eschaliel et al recommended the ergonovine tests be performed before the implantation of ICD in patients with aborted sudden cardiac death due to possible coronary spasm (9). When the ergonovine test was negative under the optimum treatment conditions, they recommend individual discussion for ICD implantation; if the test was positive, they recommend ICD implantation in all cases, even if the optimum treatment was being administered. We also reported the findings of sequential spasm provocation tests in these near-miss patients under the optimum treatment conditions (10). If a positive provoked is not noted on a sequential spasm provocation test under optimum medical therapy in patients with aborted sudden cardiac death due to coronary spasm, cardiologists and patients are free to select a course of medical treatment without ICD implantation. However, if a single spasm provocation test, such as an acetylcholine test or ergonovine test, was positive under optimum treatment conditions, cardiologist should select implant an ICD. The indications of ICD implantation in patients with aborted sudden cardiac death due to coronary spasm have not been established. Further studies are necessary to investigate the mechanism underlying aborted sudden cardiac death due to coronary artery spasm.

Learning objective

Life-threatening ventricular tachyarrhythmia is often observed in patients with aborted sudden cardiac death due to coronary spasm. An implantable-cardioverter defibrillator has proven effective for suppressing the next fatal arrhythmia. Pulseless electrical activity arrest is a mechanism underlying sudden cardiac death due to coronary spasm after the implantation of such a defibrillator. Multiple severe spasms can therefore cause the heart to freeze in a flash.

The authors state that they have no Conflict of Interest (COI).

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