Case Report

Unexpected Lethal Complication of Ventricular Fibrillation in Symptom Free Variant Angina Pectoris

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We report an unexpected sudden cardiac death due to variant angina complicated by ventricular fibrillation occurring during routine ambulatory electrocardiographic monitoring. The patient had one previous episode of ventricular fibrillation before the lethal event. He had no significant coronary artery disease and was asymptomatic throughout his illness. In clinical practice, when an episode of ventricular fibrillation is noted, one should be aware of the risk of sudden cardiac death, even if the patient's vasospastic angina is relatively stable and asymptomatic.
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Key words: Sudden death, Silent myocardial ischemia, Ambulatory electrocardiographic monitoring, Implantable cardioverter-defibrillators

Introduction

Patients with vasospastic angina (variant angina) typically have angina symptoms at rest and show ST-segment elevation on the electrocardiogram (ECG). The symptoms and ST-segment elevation are caused by coronary artery spasm. Generally, isolated coronary spasm not related to coronary artery obstruction has a favorable prognosis.[1–3] However, ventricular arrhythmia is frequently recognized as a complication of variant angina.[4] Variant angina without fixed coronary obstruction may rarely trigger lethal ventricular arrhythmia.[1,2] For patients with variant angina, medical treatment with calcium channel antagonists with and without nitrates appears to offer a good prognosis. The prognosis for patients with vasospastic angina complicated by lethal ventricular arrhythmias and/or having refractory anginal symptoms is also reported to be relatively good. However, a recent report shows that ventricular fibrillation complicating vasospastic angina identifies a patient population at risk for sudden cardiac death, and that these patients should be considered for implantable cardioverter-defibrillators (ICD).[5,6] Thus, the necessity for more aggressive anti-arrhythmic treatments such as an ICD implant is currently the subject of much debate. We report a patient who had a poor prognosis

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with symptom-free variant angina pectoris complicated by ventricular fibrillation. A more aggressive treatment strategy including ICD implantation might have been effective in this case.

Case Report

A 60-year-old man presented with symptoms of lower abdominal discomfort of about two weeks duration. He visited the outpatient clinic of the gastroenterology section of the National Defense Medical College Hospital Internal Medicine department in April 2003. He denied any sleep disorders. His past history was unremarkable except for a light heat stroke three years earlier. His family history revealed that his mother had angina pectoris. With respect to coronary risk factors, he had smoked one pack daily for 35 years, and there was a family history of coronary artery disease. The physical examination of the patient in the outpatient clinic was unremarkable (height 167 cm, body weight 65 kg, blood pressure 137/72 mmHg, pulse rate 82 beats/min and regular). The laboratory findings were also normal. His chest X-ray (cardiothoracic ratio = 48%) and electrocardiogram (ECG) were normal (**Figure 1**). Thus, ECG showed no findings suggesting idiopathic ventricular fibrillation, Brugada, long QT and/or short QT syndromes.

Upper gastrointestinal tract and colonic endoscopies revealed cancer of the stomach and of the transverse colon. Abdominal computed tomography showed no signs of abdominal invasion and no liver or lymph node metastases. He was referred to the First Department of Surgery in our hospital for radical resection of the stomach and colon. General anesthesia with nitrous oxide, oxygen, and sevoflurane, combined with epidural anesthesia was planned for his first operation in June 2003, but ventricular fibrillation developed during the induction of anesthesia. Induction of anesthesia consisted of 5 mg vecuronium bromide and 70 mg propofol given intravenously. Immediately after intra-tracheal intubation following induction of anesthesia, incessant ventricular tachycardia developed and was followed by ventricular fibrillation. DC defibrillation was necessary to restore sinus rhythm. The first operation was abandoned due to inability to find the cause of ventricular fibrillation. The monitoring ECG (modified bipolar CM5 lead) showed no significant ischemic changes and the anesthesiologist noted no problems related to the induction of anesthesia and intubation.

The cardiology section was asked to consult on the case and perform further evaluation. Two-dimensional echocardiography showed normal findings, and an exercise treadmill stress test was negative for ischemia with 6 METs. Ambulatory ECG monitoring (NASA lead) revealed marked ST-segment elevation (**Figure 2**) at night accompanied by atypical angina symptoms, such as lightheadedness. The CM5 lead, which was identical to the ECG

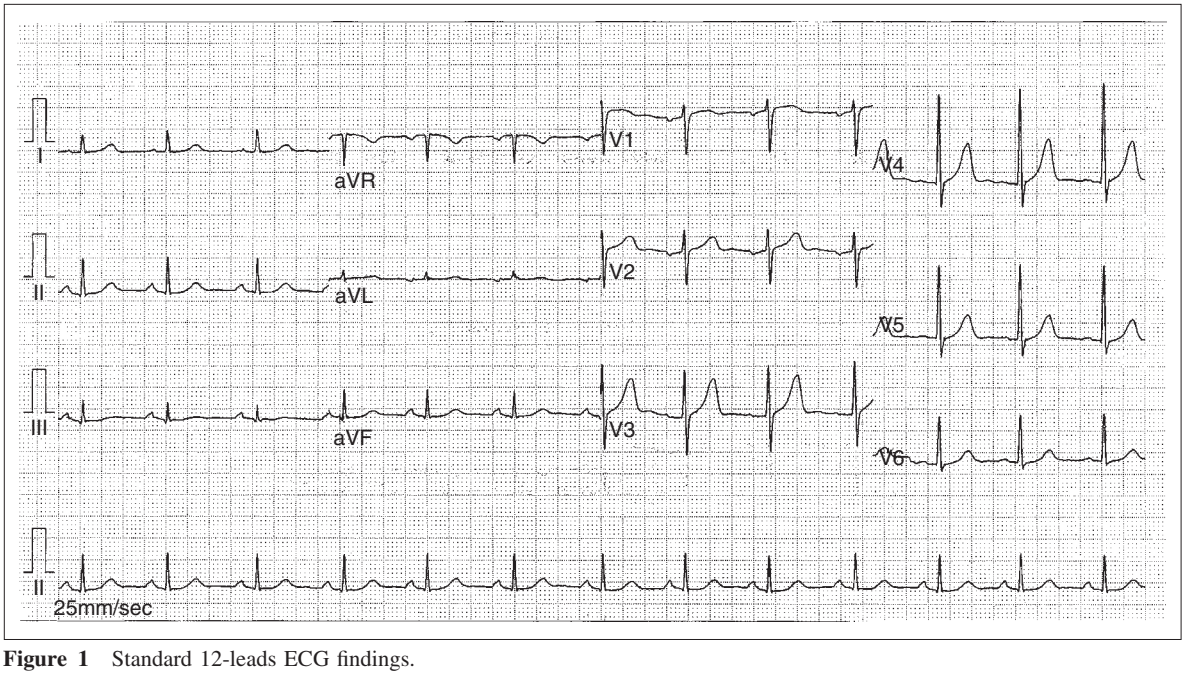

Figure 1 Standard 12-leads ECG findings.
No signs of idiopathic ventricular fibrillation, Brugada, long QT and/or short QT syndrome were observed.