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Case Report

Vasospastic angina resulting in sudden cardiac arrest, initially misdiagnosed as a psychiatric disorder



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ABSTRACT

A 51-year-old-woman with a history of ablation therapy due to Wolff–Parkinson–White syndrome had been suffering from ambiguous chest pain, prompting investigation by several cardiologists. After being dissatisfied with a psychiatric disorder diagnosis, she was admitted to our hospital for further investigation. She lost her consciousness due to a sudden cardiac arrest shortly after admission. A provocation test indicated vasospastic angina associated with a diffuse spastic pattern of her left anterior descending artery.

<Learning objective: This case demonstrates that implantation of a cardioverter defibrillator may be avoided if the angiographic pattern of the vasospasm is recognized, the condition is correctly diagnosed, and appropriate medications are prescribed.>

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Introduction

The number of patients suffering from vasospastic angina, or Prinzmetal angina, is relatively low compared with the number experiencing exertional angina. Although some aspects of the disease mechanism remain unclear [1], the pathogenesis of coronary artery spasm is reported to be different from that of atherosclerotic stenosis; patients with vasospastic angina do not typically have significant coronary stenosis angiographically. This makes the non-invasive diagnosis of the disease difficult unless an attack occurs during an electrocardiogram (ECG) [1,2]. Thus, there is the dangerous possibility of ignoring the seriousness of the symptoms, because, in many cases, true coronary function abnormalities are not evident. As a result, pharmacological and non-pharmacological coronary spasm provocation tests, during coronary angiography, have been established as useful diagnostic tools for vasospastic angina [3].

We treated a patient who was eventually diagnosed with vasospastic angina using a pharmacological provocation test. She presented with frequent ambiguous chest pain, initially diagnosed as a psychiatric disorder by several cardiologists, and finally collapsed because of ventricular fibrillation.

Case report

A 51-year-old woman underwent catheter ablation due to Wolff–Parkinson–White syndrome several years prior to this admission. Two months prior to this admission, she began suffering from intermittent ambiguous chest pain that was not correlated with exertion and dyspnea. The frequency and strength of the pain gradually worsened with the simultaneous occurrence of severe dizziness. She had met with another doctor because she developed faintness after taking a sublingual nitroglycerin tablet after experiencing chest pain. Although she was prescribed nicorandil (60 mg/day) and diltiazem hydrochloride (100 mg/day) at the hospital, her chest pain continued. She underwent cardiac ultrasonography, 24-h continuous ECG monitoring, and multislice coronary computed tomography. However, there were no significant findings that could explain her symptoms. Additionally, as she had a personal ECG machine, she recorded ECGs many times when she experienced chest pain, but abnormalities were not recorded. Until then, she had not undergone the pharmacological provocation test in order to make the differential diagnosis about vasospastic angina, mostly because her way of complaining symptoms was judged to be over-exaggerated.

Finally, the patient visited our hospital after being diagnosed with a psychiatric disorder. The patient was admitted for a thorough examination of the mechanism of her symptoms. Upon admission, all medicines were discontinued for 11 days prior to a pharmacological provocation test, using the catheter technique,

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and 24-h continuous ECG monitoring was implemented; we were deeply skeptical about her having vasospastic angina. Early the following morning (07:05), she called a nurse because of chest pain; the cardiac monitor indicated a short run of ventricular tachycardia. Both her symptoms and the arrhythmia disappeared within a couple of minutes, causing no vital sign abnormalities (Fig. 1a). Shortly thereafter (07:17) the monitor showed another episode of ventricular tachycardia, and the patient lost consciousness. Her blood pressure was unmeasurable and ventricular fibrillation was ascertained, on the monitor, following a period of sustained ventricular tachycardia (Fig. 1b). Cardiopulmonary resuscitation was performed immediately, with intravenous administration of epinephrine (1 mg) until the first electrical defibrillation (260 J) was performed; it failed. A second defibrillation was performed after the

administration of another dose of epinephrine (1 mg), resulting in conversion to a sinus rhythm. The patient then began continuous venous administration of nicorandil (2 mg/h) with benidipine hydrochloride (4 mg, orally) every 8 h. This was followed by oral administration of both nicorandil (15 mg/day) and benidipine hydrochloride (12 mg/day). A coronary angiogram, performed on post-admission day 12, did not show any significant stenotic abnormalities except for a hypoplastic right coronary artery (Fig. 2a and b).

A pharmacological provocation test was performed with an intracoronary administration of acetylcholine. Immediately after the injection of acetylcholine (20 µg), the patient's left anterior descending artery and diagonal branches became diffusely spastic (Fig. 2c), with significant ST-segment depression in the II, III, aVF,

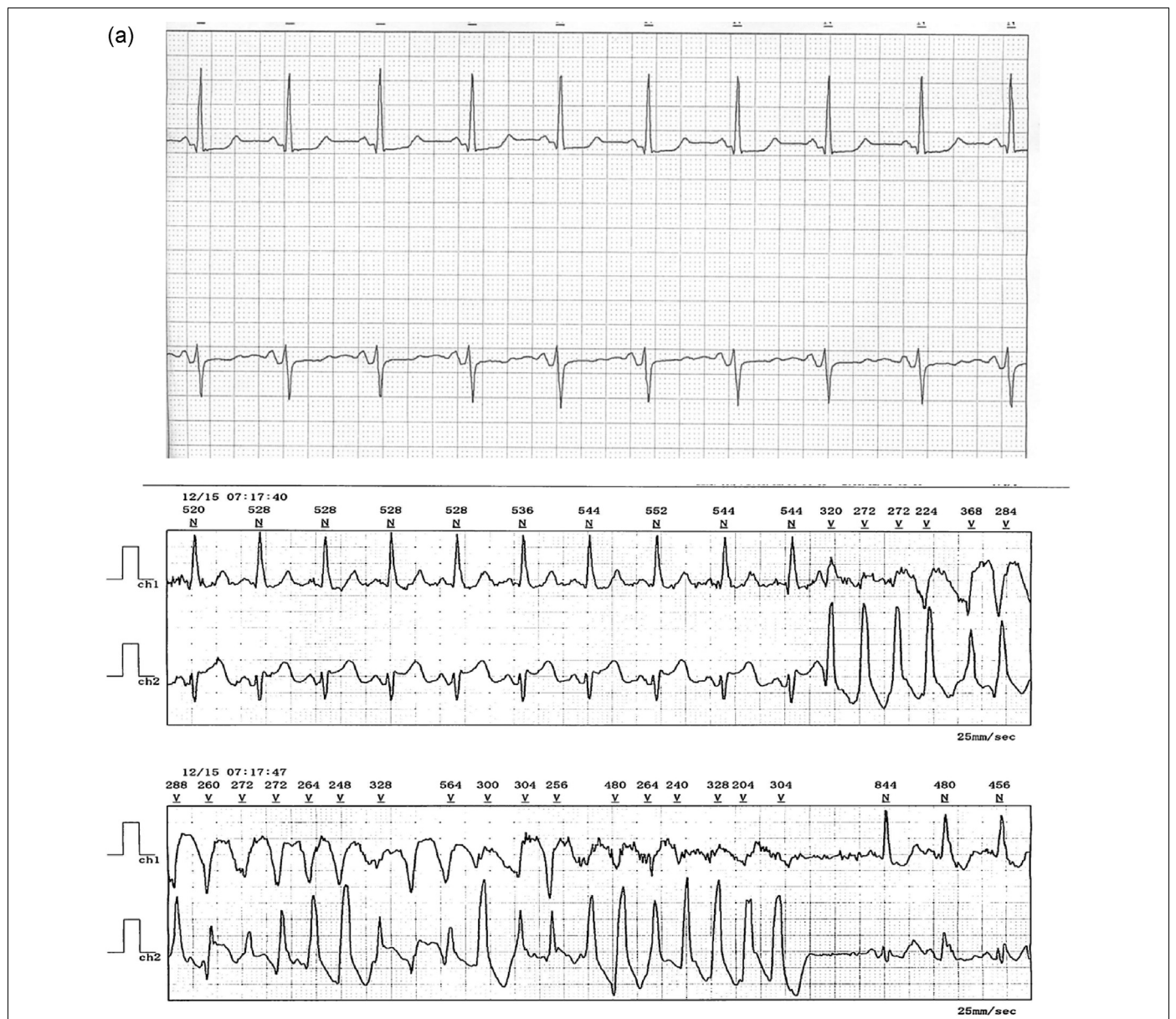


Fig. 1. (a) 24-h continuous electrocardiogram. (Upper) Just before the initial chest pain. (Lower) At the time of the initial chest pain complaint, after admission. There was a mild ST-segment elevation in the lower channel followed by non-sustained ventricular tachycardia. (b) 24-h continuous electrocardiogram. (Upper) Just before ventricular tachycardia. (Lower) At the time of loss of consciousness, following admission. There was ventricular fibrillation after a short period of ventricular tachycardia.

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