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Recurrent Ventricular Fibrillation in a Patient with Variant Angina Treated with an Implantable Cardioverter Defibrillator—a Case Report

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Abstract: In selected patients suffering from variant angina, an implantable cardioverter defibrillator (ICD) can be helpful for preventing sudden death. We herein report a case of a 51-year-old woman suffering from variant angina, who experienced recurrent episodes of ventricular fibrillation and promptly cardioverted. She developed ventricular fibrillation despite therapy with calcium-channel blockers. Coronary angiography demonstrated no atherosclerosis. Thereafter, an ICD was implanted and oral calcium-channel blockers and nitrates were prescribed. No other episodes of angina or ventricular arrhythmia were documented during the following 3-month follow-up.

Key words: Variant angina, Ventiricular fibrillation, Implantable cardioverter difibrillator

Introduction

Vasospastic angina is the presentation of sudden focal severe spasm of the coronary arteries predominantly occurring at rest.[1] Most of these patients respond to nitrates and calcium-channel blocker therapy. However, variant angina is a rare diagnosis in sudden cardiac-death patients and vasospastic angina without obstructive lesions is usually as a separate entity with a generally non-fatal prognosis.

We describe a patient with variant angina, who was resuscitated from recurrent episodes of ventricular fibrillation (VF), and thereafter received an automatic implantable cardioverter defibrillator.

Case Report

We present a 51-year-old female with recurrent episodes of variant angina and serious complications of coronary artery spasm including ventricular fibrillation, complete atrioventricular block and a non-Q wave myocardial infarction. She had suffered from morning attacks of angina at rest for 5 years. The patient's cardiac risk factors included mild hypertension, mild diabetes mellitus and smoking. Lipid profiles showed the total cholesterol, triglyceride, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol levels to be 165, 77, 37 and 113 mg/dl, respectively. In 1997, she had her first syncopal attack for a short period after

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experiencing chest pain. After being admitted to Fukuoka University Chikushi-Hospital, a 12-lead ECG at rest showed a normal sinus rhythm with an infrequent ventricular premature beat. No ST segment change was observed in any leads. Coronary angiography without acetylcholine revealed diffuse severe spasm including severe stenosis of the left circumflex and the right coronary arteries. Vasospasm was relieved by the intracoronary administration of isosorbide mononitrate. Thereafter, she was treated with calcium-channel blockers and nitrates. In 1998, when the administration of diltiazem tablets was discontinued, she collapsed due to ventricular fibrillation. The fibrillation was successfully defibrillated. On the early morning in April 28, 2003, she was hospitalized, because of severe chest pain, syncope and ventricular fibrillation. She had again forgotten to take her medication. The patient with fibrillation was successfully defibrillated. ECG demonstrated an impressive and marked ST segment elevation in Ⅱ, Ⅲ and aVF, and a complete atrioventricular block after defibrillation (Fig. 1A). Coronary angiography revealed no stenotic lesions, but a part of the apical-posterior and septal wall showed hypokinesis on left ventriculography. An electrophysiological study was performed using the standard technique under treatment with diltiazem. To induce ventricular tachyarrhythmia, 3 additional stimuli and rapid pacing at a cycle length of up to 200 ms were used. Under baseline conditions, VF was induced by applying 3 additional stimuli to the right ventricular apex (Fig 2), and an external defibrillator was required.

The patient was discharged after the implantation of a cardioverter defibrillator (Medtronic, GEM Ⅱ DR 7273) programmed with a rate threshold of 188 beats/min. The concurrent therapy at discharge included diltiazem (200 mg b.i.d.), isosorbide mononitrate (40 mg b.i.d.) and thereafter she was counseled on the dangers of smoking, and was encouraged to quit.

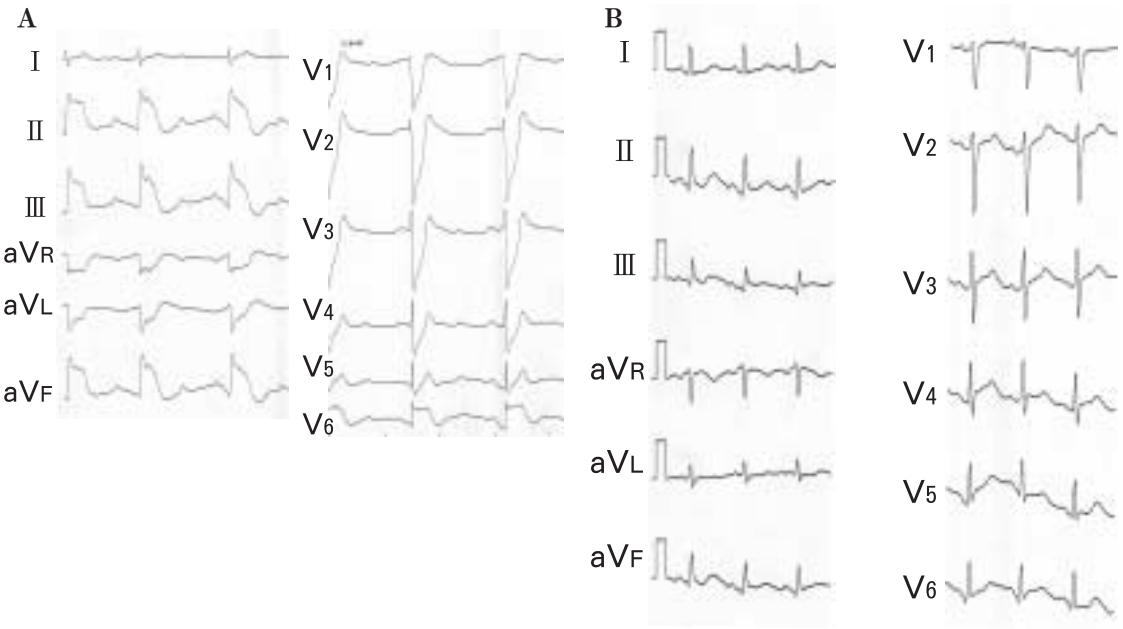

Fig. 1. An electrocardiogram just after resuscitation reveals a marked ST-T elevation especially in leads Ⅱ, Ⅲ and aVF, and a complete AV block (A). An electrocardiogram one hour after resuscitation (B).

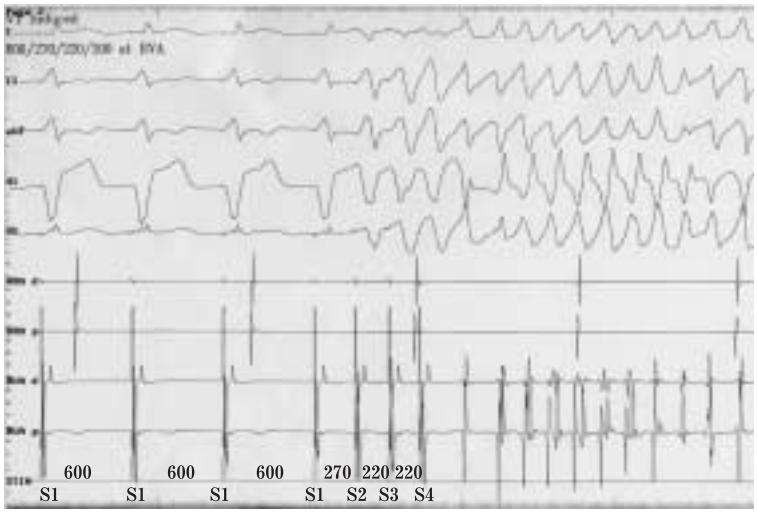

Fig. 2. An electrophysiological study showing the induction of ventricular fibrillation (VF). Under baseline conditions, 3 additional stimuli applied to the right ventricle induced VF, which required external defibrillation.
HRA, high right atria; RVA, right ventricular apex; d, distal; p, proximal; STIM, stimulation

Discussion

The incidence of coronary artery spasm is higher in Japanese than in western people.[2] ST-segment elevation can be reproduced on exercise stress in only a few patients with vasospastic angina (30%). The most dangerous complication in patients with variant angina is cardiac arrest. Despite treatment with calcium-channel blockers and nitrates, persistent or recurrent episodes of angina are frequently observed, in contrast, complications such as myocardial infarction or sudden death are rare.[3] The incidence of sudden death was lower in patients treated with calcium-channel blockers (up to 5%) than in groups without the routine use of calcium-channel blockers (up to 11%).[4] The simultaneous ST elevation in the inferior and anterior leads has been reported to be a marker for sudden cardiac death.[5]

In this case, ECG demonstrated an impressive and marked ST segment elevation in Ⅱ, Ⅲ and aVF after resuscitation, and the continuance of arterial occlusive spasm was believed to be responsible for VF. However, VF was inducible under treatment with diltiazem, probably due to the arrhythmogenic substrate of LV after a non-Q wave myocardial infarction. Diltiazem may therefore not have been sufficient to prevent VF in this case. This report underlines the possible failure of calcium channel blockers for preventing sudden cardiac-death recurrences in such patients, and also supports similar findings reported by others.[6,7]

References

1) Yasue, H., Kugiyama, K.: Coronary spasm: clinical features and pathogenesis. Intern. Med., 36: 760-765, 1997.
2) Nobuyoshi, M., Abe, M., Nosaka, H., Kimura, T., Yokoi, H., Hamasaki, N., Shindo, T., Kimura, K., Nakamura, T., Nakagawa, Y., et al.: Statistical analysis of clinical risk factors for coronary artery spasm: identification of the most important determinant. Am. Heart. J. 124: 32-38, 1992.
3) Bory, M., Pierron, F., Panagides, D., Bonnet, J. L., Yvorra, S., Desfossez, L.: Coronary artery spasm in patients with normal or near

normal coronary arteries. Long-term follow-up of 277 patients. Eur. Heart J., 17 : 1015-1021, 1996.
4) MacAlpin, R. N. : Cardiac arrest and sudden unexpected death in variant angina : complications of coronary spasm that can occur in the absence of severe organic coronary stenosis. Am. Heart J., 125 : 1011-1017, 1993.
5) Yasue, H., Takizawa, A., Nagao, M., Nishida, S., Horie, M., Kubota, J., Omote, S., Takaoka, K., Okumura, K. : Long-term prognosis for patients with variant angina and influential factors. Circulation, 78 : 1-9, 1988.
6) Lacroix, D., Kacet, S., Lekieffre, J. : Vasospastic angina without flow-limiting coronary lesions as a cause for aborted sudden death. Int. J. Cardiol., 43 : 247-249, 1994.
7) Seniuk, W., Mularek-Kubzdela, T., Grygier, M., Grajek, S., Cieslinski, A. : Cardiac arrest related to coronary spasm in patients with variant angina : a three-case study. J. Intern. Med. 252 : 368-376, 2002.

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