

[CASE REPORT]

Ventricular Fibrillation Induced by Coronary Vasospasm in a Patient with Early Repolarization and Hyperthyroidism

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Abstract:

Vasospastic angina (VSA) has been recognized as a cause of ventricular fibrillation (VF) degenerating into sudden cardiac death. We experienced a case of VSA with hyperthyroidism in which VF was provoked with an augmented J-wave amplitude in the inferior leads. The patient underwent insertion of an implantable cardioverter-defibrillator for the secondary prevention of VF in addition to taking Ca-channel antagonists. He has shown no recurrence of fatal arrhythmia or anginal attack for a follow-up period of one year.

Key words: ventricular fibrillation, early repolarization, vasospastic angina, hyperthyroidism

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Introduction

Vasospastic angina (VSA) has been recognized as a cause of ventricular fibrillation (VF) degenerating into sudden cardiac death (1). In addition, it was reported that J-wave [early repolarization (ER)] was associated with life-threatening ventricular tachyarrhythmia in patients with VSA (2-4). In contrast, hyperthyroidism is known to augment coronary vasoconstriction and induce VF (5).

We present a case of VSA with hyperthyroidism in which VF was provoked with an augmented J-wave amplitude in the inferior leads.

Case Report

A 79-year-old man was hospitalized for the investigation of a benign mass in the colon. During colonoscopy, he suffered from cardio-pulmonary arrest due to VF, which was terminated by an external defibrillator (Fig. 1). Electrocardiogram (ECG) immediately after the resuscitation exhibited ER with a prominent J-wave and ST segment elevation in leads II, III, and aVF, although complete right bundle block (CRBBB) coexisted (Fig. 2B). A similar J-wave on ECG had been observed at a medical examination two years ear-

lier (Fig. 2A).

He had no family history of electrocardiographic abnormality, heart diseases, or sudden death. Transthoracic echocardiography revealed no abnormal findings. Coronary angiography showed no organic stenosis, and left ventriculography demonstrated a normal LV function. Laboratory data showed that he had no electrolyte imbalance; however, he had hyperthyroidism caused by uncontrolled Grave's disease (FT3 >30.0 pg/mL, FT4 3.39 ng/dL, TSH <0.03 µU/mL, and positive TSH receptor antibody). Thiamazole, steroids, and subsequently propylthiouracil were administered for his hyperthyroidism.

On the first night after admission, spontaneous progression of ST segment elevation (STE) and simultaneously accentuated J-wave amplitude in leads II, III, and aVF on 12-lead ECG was documented (Fig. 2C). Although he had no significant chest pain, these findings suggested he had acute coronary ischemia induced by vasospasm, which led to an augmented amplitude of J-wave. After bolus injection of isosorbide dinitrate (ISDN) followed by continuous intravenous infusion of ISDN, the STE returned to the normal range, and the J-wave amplitude in the inferior leads decreased (Fig. 2D).

After the thyroid function was controlled (FT3 3.77 pg/mL, FT4 1.59 ng/dL, TSH 0.03 µU/mL), an acetylcholine

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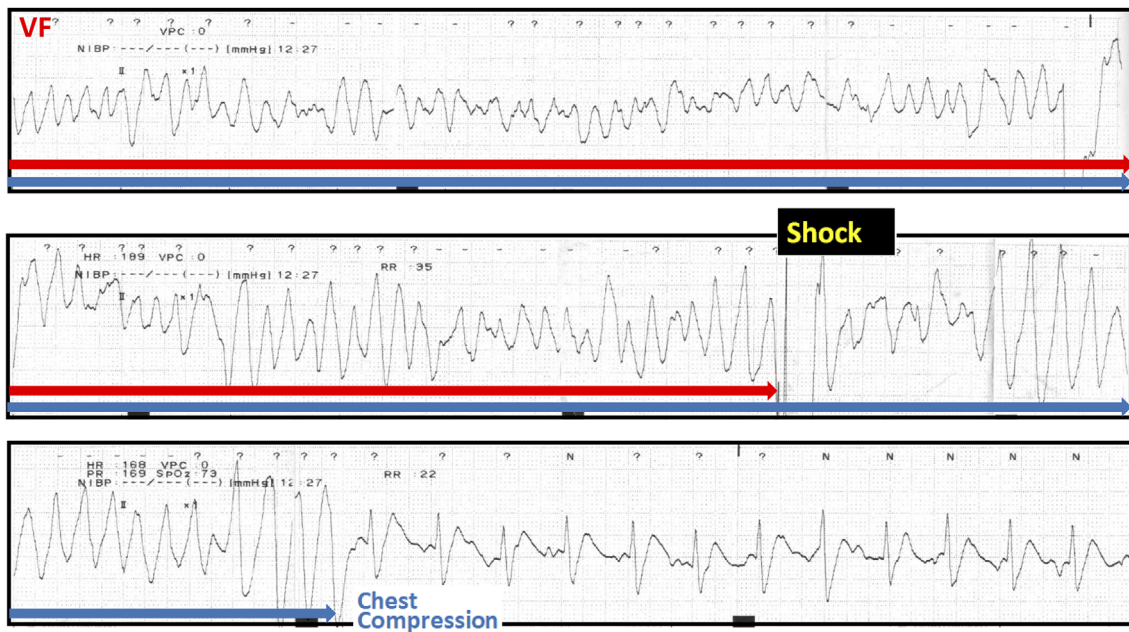


Figure 1. ECG monitoring during colonoscopy. ECG revealed ventricular fibrillation (VF) which was successfully terminated by an external defibrillator. ECG: electrocardiogram

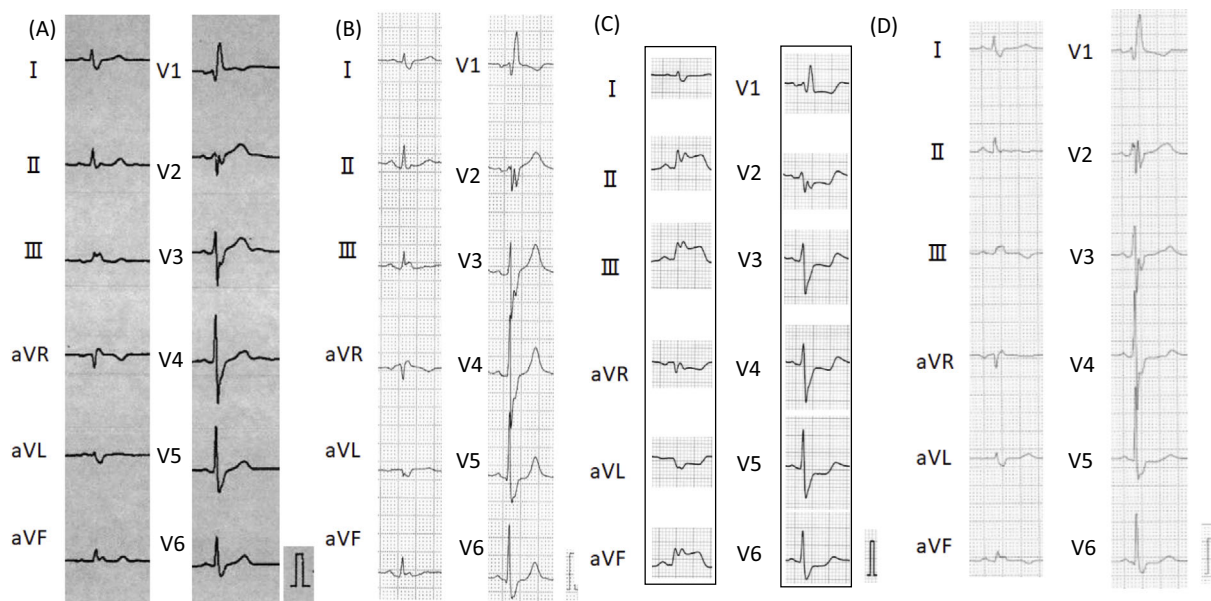


Figure 2. A series of 12-lead electrocardiogram (ECG) tracing in the present case. (A) ECG recorded at a medical examination 2 years before admission. Notched type J-wave are visible in leads II, III, and aVF. (B) ECG with J-wave in the inferior leads immediately after termination of VF by direct current shock. (C) ECG with augmented amplitude of J-wave together with ST segment elevation in the inferior leads on the first night after admission. (D) ECG with decrease of J-wave amplitude after infusion of ISDN. ISDN: isosorbide dinitrate

(ACh) provocation test was performed with discontinuation of venous infusion of ISDN 10 hours before the study. After a quadripolar electrode catheter was inserted into the right ventricular apex, incremental doses of ACh were injected into the left coronary artery (20, 50, and 100 μ g) and the right coronary artery (20, 50, and 70 μ g). As a result, coronary vasospasm was provoked in three vessels by the intra-

coronary injection of ACh, accompanied by chest discomfort and ECG changes similar to those during spontaneous anginal attack (Fig. 3). In particular, ECG during focal spasm in the right coronary artery showed the development of J-wave and ST segment elevation similar to a spontaneous attack. He was diagnosed with vasospastic angina (VSA) exhibiting multivessel coronary vasospasm. After the administration of

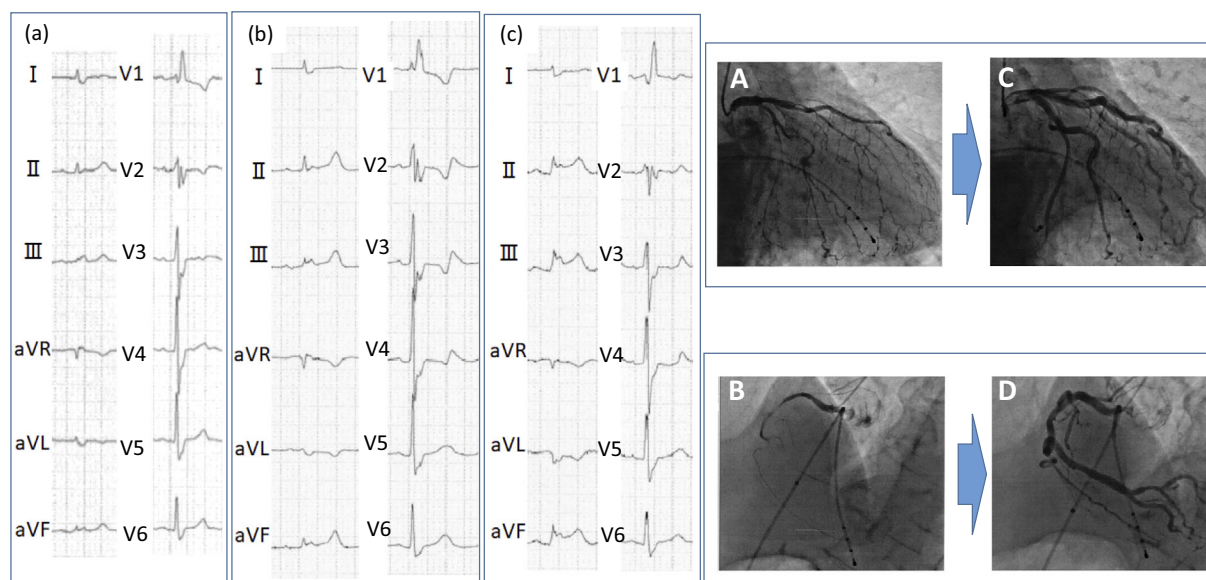


Figure 3. (A-D) The induction of spasm in both the left and right coronary arteries by the intra-coronary injection of acetylcholine (ACh). (A) Severe diffuse spasm was provoked in the left descending and left circumflex arteries after ACh 100 μ g injection. (B) Severe focal spasm was provoked in the proximal right coronary artery after ACh 70 μ g injection. Right panel: (C, D) After the intra-coronary infusion of isorbidine dinitrate, all three vessels were released from spasm. (a, b) ECG during the ACh provocation test. (a) ECG before the injection of ACh. (b) ECG showed biphasic T wave changes in leads V2-4 when diffuse spasm was provoked in the left coronary artery. (c) Augmentation of J-waves together with ST segment elevation in the inferior leads was observed when total occlusion due to focal spasm in the proximal portion of the right coronary artery was induced by ACh.

ISDN, an electrophysiological study was performed at the right ventricular apex with up to three extrastimuli. However, ventricular tachyarrhythmia could not be induced. The arrhythmogenicity was also examined by microscopic T wave alternans (TWA) during a euthyroid state by the modified moving average method (MMA-TWA) using ambulatory ECG monitoring with the MARS software program, ver. 7.03 (GE Healthcare, Milwaukee, USA). This patient exhibited a high maximum TWA (68 μ V), suggesting the presence of repolarization abnormalities.

The patient underwent insertion of an implantable cardioverter-defibrillator for the secondary prevention of VF in addition to taking Ca-channel antagonists (initial dose of Nifedipine 20 mg/day increased to a final dose of 40 mg/day) after his hyperthyroid state was controlled. He has shown no recurrence of fatal arrhythmia or anginal attack during the one year of follow-up.

Discussion

Life-threatening ventricular arrhythmias triggered by acute myocardial ischemia, including VSA, have been recognized as a cause of a sudden cardiac death (SCD) due to VF (1). It has been reported that the appearance or progression of ER is sometimes observed during acute myocardial ischemia in patients with coronary artery disease. Furthermore, VSA patients with ER, especially its day-to-day variation, develop VF more frequently than those without ER, even during the

asymptomatic phase (2, 3). On the other hand, Inamura et al. reported the VSA patients with fatal arrhythmic events showed a high incidence of ER and positive for TWA during the symptom-free period, which exhibits potentially depolarization and repolarization abnormalities (4).

Thyroid hormone is reported to positively modulate Ito in rat and rabbit ventricular myocytes (6). When Ito is augmented, a transmural voltage gradient between the endocardium and epicardium during the early phase of ventricular repolarization appears to produce the ER pattern. However, in our case, the J-waves had already been observed at a medical examination before admission. We therefore suspected that thyroid hormone did not directly affect the arrhythmogenicity through the development of such a transmural voltage gradient. However, it is known that hyperthyroidism induces coronary vasospasm. The first case of hyperthyroidism-associated vasospastic angina was reported in 1979, and since then, several reports had been published. In a retrospective analysis, Choi et al. reported that, among 325 patients with VSA, 8 had hyperthyroidism (5). In our case, hyperthyroidism might have induced myocardial ischemia due to vasospasm, subsequently leading to the augmented amplitude of the J-wave associated with the development of fatal tachyarrhythmia.

The definition of J-wave coexisting with CRBBB is unclear at present. In particular, in patients of Brugada syndrome, ER may coexist with CRBBB, although whether or not ER associated with coronary artery spasm is accompa-

nied by CRBBB is unknown. According to the consensus report for J-wave syndromes (7), the end QRS notch (J-wave) in the inferior leads in the present case meet the criteria, as the QRS duration (measured in the leads in which a notch or slur is absent) was <120 ms. Furthermore, the morphology of the QRS complex manifesting end QRS notching, especially in the inferior leads, was not observed in the ECG in this case showing a typical CRBBB. In addition, the augmentation of the J-wave amplitude and the dynamic changes in the J-point elevation in three inferior leads were provoked by acute myocardial ischemia due to coronary spasm (Fig. 2). Thus, the development of end QRS notching with coexisting CRBBB during the ischemic state reflects the manifestation of arrhythmogenicity.

This patient developed VF triggered by coronary spasm along with hyperthyroidism and showed ER in the inferior leads and high levels of TWA during the euthyroid state. The multivessel coronary vasospasm provoked in the present case has been shown to indicate a high risk of sudden cardiac death in patients with VSA (8). Therefore, patients with multivessel coronary vasospasm require appropriate medical therapy, including a Ca-channel blocker to control vasospastic attack. These patients may need intensive medical therapy to combat recurrence of coronary vasospasm in the future.

The implantation of an ICD in patients with fatal arrhythmias due to coronary spasm remains controversial. However, ICD therapy should be considered in patients, especially those showing multivessel coronary vasospasm, who are at a high risk for recurrence of VF triggered by coronary spasm despite appropriate medical therapy (9). Furthermore, the presence of ER and a high value of TWA, which is a significant risk factor for life-threatening ventricular arrhythmia in VSA patients, helps confirm the indication of ICD implantation for the secondary prevention of VF in this patient (2-4).

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

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