Case Reports

Early-Morning Type Ventricular Fibrillation With J Waves Effectively Cured by Oral Administration of Long-Acting Disopyramide

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Summary

A 67-year-old man who had cardiopulmonary arrest (CPA) at home was admitted to our institution. His spontaneous circulation was restored by bystander cardiopulmonary resuscitation (CPR) performed by his wife and an automated external defibrillator (AED). J waves were observed in the inferior leads of an electrocardiogram. We performed an implantable cardioverter defibrillator (ICD) implantation. After the ICD implantation, appropriate shocks due to ventricular fibrillation were observed on interrogation of the ICD at a frequency of twice a month. Most VF events occurred in the early morning between 1:00 to 6:00, and ventricular premature contractions (VPCs) were detected just before the occurrence of VF. Since the VF events always occurred in the early morning, we started long-acting disopyramide (150 mg/day, before bedtime), which has a muscarinic receptor blocking action. As a result, he has not received any appropriate ICD shocks for more than two years. (Int Heart J 2015; 56: 459-461)

Key words: Disopyramide, Idiopathic ventricular fibrillation, J wave syndrome, Implantable cardioverter defibrillator, Ventricular premature contraction

Ventricular fibrillation (VF) is an arrhythmia that causes sudden cardiac death and can easily occur in patients with structural heart disease. It is well known that VF may occur in patients with a normal heart and J waves on the 12-lead electrocardiogram (ECG). Some studies have reported that J waves on the 12-lead ECG are often detected in healthy subjects with VF events. In addition, coronary spastic angina (CSA) is also known as a heart disease that causes VF without any structural myocardial injury. Here we present a J wave syndrome patient who had frequentVF events in the early morning showing J waves in the inferior leads of the 12-lead ECG and the VF events were successfully cured by the oral administration of long-acting disopyramide.

Case Report

A 67-year-old man who had cardiopulmonary arrest (CPA) at home was admitted to our institution. His spontaneous circulation had been restored by bystander cardiopulmonary resuscitation (CPR) performed by his wife with an automated external defibrillator (AED) (Figure 1). He had no history of previous cardiovascular or other major diseases. Structural heart disease was not detected by echocardiography, but J waves were observed in the inferior leads of the 12-lead ECG (Figure 2). In the 24-hour Holter ECG recording with 12 leads, a circadian variation of the J waves was observed. The J waves were augmented in a lower heart rate, particularly in the early morning. We performed a signal-averaged ECG, but ventricular late potentials were not detected in this case. Blood test results and chest X-rays were normal. In addition, he did not experience any syncopal episodes and did not have a family history of sudden cardiac death. We confirmed that no Brugada-like electrocardiogram pattern was detected using the class Ic antiarrhythmic drug pilsicainide. We performed an electrophysiological study and programmed stimulation for VF induction (2 cycle lengths [600 and 400 msec] of up to double stimulation), but VF was not induced by extra-stimulation.

Left coronary artery spasms were induced by acetylcholine loaded coronary angiography (Figure 3). Therefore, we started a calcium channel blocker (benidipine) and then implanted an ICD. After the ICD implantation, appropriate shocks due to VF were observed at a frequency of twice a month (Figure 4). All VF events occurred in the early morning between 1:00 to 6:00, and ventricular premature contractions (VPCs) were detected just before the occurrence of VF. However, he did not feel any symptoms associated with coronary spasms such as chest pain, and the Holter ECG recordings did not detect any significant ST-T changes or frequent VPCs. The occurrence of VF was frequently recorded on the ICD, but it was not recorded on the virtual surface ECG. Additionally, we performed many Holter ECGs, however, no VF was ever detected. Since VF events always occurred in the early morning, we started long-acting disopyramide (150 mg/day) before bedtime, which has a muscarinic receptor blocking action, in addi-
tion to administration of a calcium channel blocker. As a result, he has not experienced any appropriate ICD shocks for more than two years.

**Discussion**

In this patient, J waves were observed in the inferior leads of the 12-lead ECG. In healthy subjects, some studies have reported that J waves on 12-lead ECG are observed in 3-6%.1-3) In idiopathic VF patients, J waves are frequently detected in 31-60%.2,3) According to a report that evaluated J wave characteristics in young athletes and healthy subjects,4) the diagnostic value is high when both; 1) a J-point elevation of 0.1 mV or more, and 2) a J-point elevation in the limb leads, are detected. In addition, it is known that there is a high risk of arrhythmic mortality when J waves of 0.2 mV or more are present.1,4) In our case, the average J waves were 0.25 mV in lead II, depending on the heart rate, and therefore, we considered him as a high risk patient.

In 1992, Aizawa, et al5) reported that a notch in the late part of the QRS complex was observed in the inferior and V3-5 leads and VPCs can lead to VF. Haïssaguerre, et al2) described the clinical characteristics of 206 patients resuscitated from VF with J wave syndrome. J wave syndrome has certain characteristics, such as a high association with being male, often occurs during sleep, and has a frequent VF recurrence rate. VPCs were detected just before the occurrence of VF in this patient, and as in the report by Aizawa, et al5) VPC bigeminy from a left ventricular outflow tract origin was revealed in the 12-lead ECG during the course, but the relationships to the VF

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**Figure 1.** ECG strips recorded on an AED. A: Documentation of VF converting to sinus rhythm just after an electroshock (black arrow). B: Documentation of remarkable J waves after an appropriate shock.

**Figure 2.** Recording of the baseline 12-lead ECG. Notch-type J waves in the inferior leads are shown (arrows).

**Figure 3.** Findings of the coronary angiography pre- and post-acetylcholine loading; Baseline coronary angiography of the right coronary artery (A) and the left coronary artery (C) showing no lesions. Coronary angiography after the acetylcholine loading of the right coronary artery (B) and left coronary artery (D) showing segmental spasms.

**Figure 4.** An ECG strip of VF initiation on an ICD. The same VPCs occurred (stars) and then VF was induced.

**Figure 5.** The time course of the limb leads of the 12-lead ECG. The height of the J waves changed with the heart rate (arrows) chronologically.
events were unknown. We considered catheter ablation therapy targeting the VPCs, but we hesitated because there was no repeatability of the VPCs in the frequent Holter recordings. In another study by Aizawa, et al\textsuperscript{6} and in our patient, the height of the J waves was highly dependent on the heart rate (Figure 5). We estimated that an elevation of the J waves just before the VF was closely related to bradycardia. Shinohara, et al\textsuperscript{7} analyzed the relevance between medications and the autonomic nerve system activity in a J wave syndrome patient. According to their report, the J waves were increased by parasympathetic (vagal) nervous tension, β-blocker administration, and bradycardia. In contrast, the J waves were decreased by β-agonist administration (isoproterenol), antiarrhythmic drug administration (quinidine, disopyramide), and atrial pacing in order to prevent bradycardia. Among them, isoproterenol was also effective in suppressing VF.\textsuperscript{8} On the other hand, amiodarone, which blocks multiple channel currents, did not have an effect.\textsuperscript{9} Among anti-arrhythmic drugs, disopyramide is a drug with a potent muscarinic receptor blocking action. In addition, disopyramide has a mild I\textsubscript{Na} blocking action and early dissociation from the Na\textsuperscript{+} channels, and fewer side effects as compared to quinidine. We selected long-acting disopyramide (half-life of 7 - 8 hours), which is able to maintain stable blood levels. Moreover, disopyramide is expected to sufficiently suppress VPCs because of its membrane stabilizing action on cardiomyocytes. Since the VF always occurred in early morning, we started long-acting disopyramide (150 mg/day) before bedtime. As a result, he has not experienced any appropriate ICD shocks for more than two years.

Left coronary artery spasms were induced by acetylcholine loaded coronary angiography in this patient. Therefore, it is necessary to distinguish J wave syndrome from CSA. A recent study reported that J waves were observed in approximately 20% of CSA patients and were associated with cardiovascular events.\textsuperscript{10} J wave syndrome and CSA patients have some similarities, such as they are highly associated with being male and often occur during sleep. The diagnosis of J wave syndrome in this patient was acceptable, but we could not completely deny the possibility of CSA.

**Conclusion:** We experienced a structurally normal patient, in which the oral administration of long-acting disopyramide successfully prevented frequent VF events with ICD shocks showing J waves on the 12-lead ECG. Dysopyramide may be effective not only for atrial fibrillation, but also for patients with idiopathic VF, including J wave syndrome.

**References**