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A case of a concealed type of Brugada syndrome with a J wave and mild ST-segment elevation in the inferolateral leads

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Abstract We report a patient with a concealed type of Brugada syndrome. The electrocardiogram in the emergency department revealed atrial fibrillation with an almost normal ST segment. Slight electrocardiogram abnormalities of the J wave and mild ST-segment elevation appeared in the inferolateral leads a few days later. Although the ST segment in the right precordial leads, including that recorded from the high intercostal space recording sites, was completely normal, a drug challenge test using pilsicainide revealed a coved-type ST-segment elevation only in a modified V2 lead placed 1 or 2 intercostal spaces higher.

Keywords: Idiopathic ventricular fibrillation; Brugada syndrome; Concealed type; Na channel blocker; Pilsicainide

Introduction

A diagnosis of Brugada syndrome is not difficult when a patient has a history of ventricular fibrillation (VF), and the coved-type ST-segment elevation is continuously observed in the right precordial leads with (incomplete) a right bundle branch block pattern.[1] However, it has recently been reported that the electrocardiographic phenotype of Brugada syndrome was sometimes intermittently observed.[2,3] In such cases, it is sometimes difficult to discriminate between Brugada syndrome and idiopathic VF (IVF). We experienced a case of a concealed type of Brugada syndrome with inferolateral J waves and ST-segment elevation in the baseline electrocardiogram (ECG). The typical coved-type ST-segment elevation then appeared only after the administration of the pure Na channel blocker, pilsicainide, when recorded from 1 to 2 intercostal spaces above the standard V2 right precordial lead position.

Case report

A 29-year-old man lost consciousness and collapsed suddenly in his factory during the morning. He had been in good health until the day of the event and did not have a family history of sudden cardiac death. About 8 minutes after he collapsed, the ambulance crew arrived, and cardiopulmonary resuscitation was performed immediately. The ECG recorded by the ambulance crew revealed VF, and several direct current countershocks were delivered. The ECG obtained at the emergency department exhibited atrial fibrillation (AF) with a rapid ventricular response without any ST-segment elevation or QT prolongation in any of the leads (Fig. 1). The AF converted to sinus rhythm spontaneously on his fourth day in the hospital. When he transferred to our department for further examination of his VF, he had recovered to a point similar to the condition before his collapse. The baseline ECG during sinus rhythm showed almost normal findings except for a J wave with mild ST-segment elevation in the inferior leads and leads V5-6 (Fig. 2A). The chest x-ray, echocardiograms, and cardiac magnetic resonance imaging were normal. Although there was no apparent structural heart disease, late potentials on the signal-averaged ECG were considered positive; the filtered QRS duration was 140 ms, duration of the low amplitude signals of less than 40 μV was 42 ms, and root-mean-square voltage of the signals in the last 40 ms was 13 μV. The coronary arteries were intact, and intracoronary administration of acetylcholine could not induce coronary spasms.

Because the presence of a J wave might suggest a variant type of Brugada syndrome, a pharmacological challenge test using the pure Na channel blocker, pilsicainide, was

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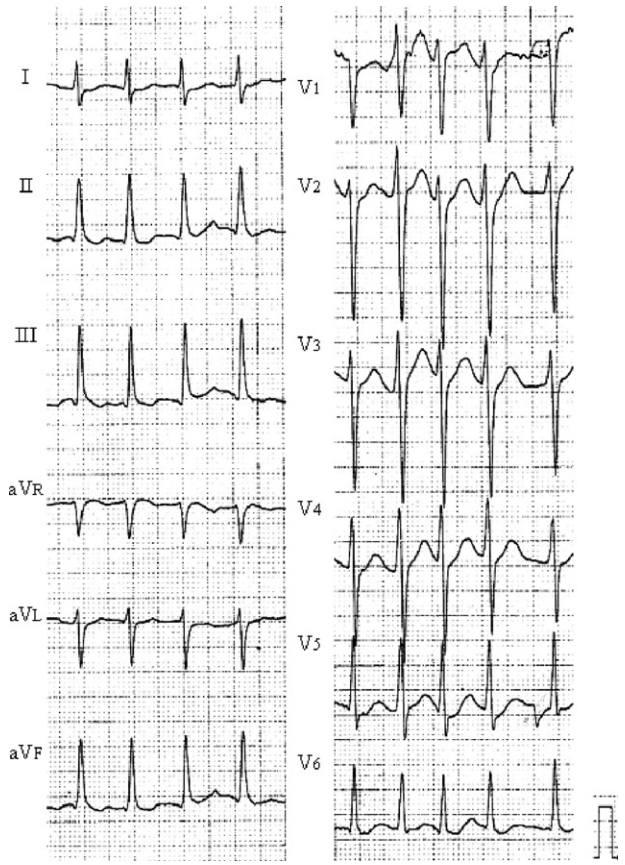

Fig. 1. An ECG recording just after arrival at our emergency department. The ECG showed AF with a rapid ventricular response.

performed. After the administration of pilsicainide (1 mg/kg per 10 minutes), the ECG revealed a saddleback-type ST-segment elevation in lead V2 and coved-type ST-segment elevation at 1 and 2 intercostal spaces higher than lead V2. In contrast, the ST-segment elevation with a J wave in the inferior leads and leads V5-6 normalized after the pilsicainide (Fig. 2B). The ST-segment elevation in the high right precordial leads nearly normalized 1 hour after the administration (Fig. 2C and D). After obtaining an informed consent, an electrophysiological study (EPS) was performed. In the EPS, the maximum sinus node recovery time was mildly prolonged at 3040 ms. The minimum Wenckebach period was 150 beats/min, and the HV interval was 45 ms. VF was induced twice by triple extrastimuli (basic cycle length, 400 ms; S1S2, 220 ms; S2S3, 200 ms; S3S4, 200 ms) applied at the RV outflow tract under baseline conditions. The VF was suppressed by an isoproterenol infusion. After the administration of 10 mg (0.1 mg/kg) of propranolol, VF was induced again by double extrastimuli (basic cycle length, 400 ms; S1S2, 230 ms; S2S3, 180 ms) applied at the RV outflow tract.

From these findings, we diagnosed a patient with a concealed type of Brugada syndrome and recommend ICD therapy. However, the patient and his family refused the implantation of an implantable cardioverter defibrillator (ICD). Therefore, we taught his family cardiopulmonary resuscitation and recommended they purchase an automated external defibrillator. He has been asymptomatic without any antiarrhythmic therapy for 20 months.

Discussion

In this case, VF was documented in the ambulance, but the imaging and biochemical examination did not show any structural heart disease, myocarditis, cardiomyopathy, or drug intoxication. Ischemic heart disease was excluded because of the absence of any stenosis or coronary artery spasms. The surface ECG did not exhibit any long QT or coved-type ST-segment elevation in the right precordial leads or any ventricular preexcitation, such as Wolff-Parkinson-White (WPW) syndrome. Thus, we made a differential diagnosis to rule out the unexplained VF produced by a primary electrical disease, just as was recently reported by Krahn et al.[4]

It was very important to discriminate between Brugada syndrome and other IVF, because antiarrhythmic agents such as Na channel blockers, which are sometimes intoxicative in Brugada syndrome, may be used in some cases of IVF. The use of an Na channel blocking agent challenge test, such as with flecainide, pilsicainide, and ajmaline, is thought to be one method to discriminate Brugada syndrome from other forms of IVF.[4-6] Furthermore, ECG recording from the high right precordial lead positions is also another method to unmask the concealed form of Brugada syndrome.[6,7]

According to a previous report, the prevalence of the Brugada sign among patients diagnosed with IVF was approximately 20%.[8] In the present case, no Brugada sign, neither the coved-type nor the saddleback-type ST-segment morphologies, were observed in the baseline ECG, even when recorded just after the VF episode. However, there were slight ECG abnormalities noted with inferolateral J waves and mild ST-segment elevation a few days later. We paid close attention to those slight ECG abnormalities because Kalla et al[9] and Takagi et al[10] reported cases in which spontaneous VF with J waves and ST-segment elevation in the inferior leads occurred. They pointed out the similarities of the clinical characteristics to those of Brugada syndrome, which is known as a J wave–associated VF or variant of Brugada syndrome. In a drug challenge test, pilsicainide accentuated the ST-segment elevation only in the right precordial leads, particularly when recorded from the 1 and 2 higher intercostal spaces, whereas the inferolateral J waves and ST-segment elevation were normalized rather than accentuated. Therefore, the clinical significance of the ECG abnormalities, the inferolateral J waves at baseline in our case, was uncertain.

According to the second consensus report, a coved-type ST elevation should be observed in at least 2 precordial leads, but in the present case, it was only observed in the leads placed higher than lead V2, and it accounted for a false-positive in the drug challenge test.[6,11] Therefore, we performed an EPS to assess the substrate of the VF and confirmed the inducibility of the VF. As a matter of course, because the signal-averaged ECG revealed positive late potentials, we also carefully checked for the possibility of the presence of arrhythmogenic right ventricular cardiomyopathy. However, there were no acceptable data for suggesting the presence of arrhythmogenic right ventricular cardiomyopathy in the present case. We also made a