

[CASE REPORT]

Idiopathic Ventricular Fibrillation Manifesting Delta-wave during Hypothermia Treatment

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Abstract:

We herein report a case of a 53-year-old man who survived cardiac arrest due to ventricular fibrillation (VF). When admitted to the hospital, his 12-lead electrocardiogram did not show Brugada-like ST elevation, early repolarization or delta-wave, in any leads. During the treatment of hypothermia, the manifestation of delta-wave was documented, which disappeared after the cessation of this treatment. A cardiac evaluation showed no structural heart disease, and electrophysiology studies did not demonstrate conduction via accessory pathway. Although the etiology of VF could not be determined, the most probable diagnosis was idiopathic VF. The patient was fitted with an implantable cardioverter-defibrillator.

Key words: ventricular fibrillation, hypothermia, delta wave, intermittent Wolff-Parkinson-White syndrome, implantable cardioverter-defibrillator

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Introduction

Wolff-Parkinson-White (WPW) syndrome generally has a good prognosis (1). However, several case studies have reported sudden cardiac death (SCD) as the first manifestation of WPW syndrome (2, 3). The shortened effective refractory period of accessory pathway during atrial fibrillation may cause ventricular fibrillation (VF) (4).

Furthermore, during the treatment of hypothermia, non-specific electrocardiogram (ECG) findings, such as prominent J wave, prolongation of the QT interval, sinus bradycardia, and atrio-ventricular block, have been observed (5). However, a case showing transient delta-wave during hypothermia treatment has never been reported.

We herein report a 53-year-old man who survived cardiac arrest due to VF and showed transient manifestation of delta-wave during hypothermia treatment.

Case Report

A 53-year-old man was admitted to our hospital after he experienced sudden cardiac arrest. He had no history of

SCD in the family or a history of syncope. He suddenly developed abnormal respiration and subsequent convulsions while working at his desk at his office. His colleague noticed this abnormality and immediately called emergency medical services and performed cardiopulmonary resuscitation. Emergency medical services arrived 20 minutes later. Automated external defibrillator (AED) identified VF, which was successfully defibrillated, and sinus rhythm was restored (Fig. 1A). He was intubated and transferred to the intensive-care unit of our hospital.

An ECG on admission showed sinus tachycardia with ST depression in the inferolateral leads (Fig. 1B), findings that were not very different from those of his previous ECG (Fig. 1C) taken at a medical checkup. Because of the reduced level of consciousness, therapeutic hypothermia was initiated with a surface-cooling device. During therapeutic hypothermia, neither early repolarization patterns in the inferolateral leads nor other critical arrhythmias were observed. However, delta-wave was observed, which suggested the existence of a left posterior accessory pathway (body temperature: 34.0 °C, Fig. 2A).

After the patient was rewarmed, the delta-wave disappeared and did not reappear at all (Fig. 2B). He gained con-

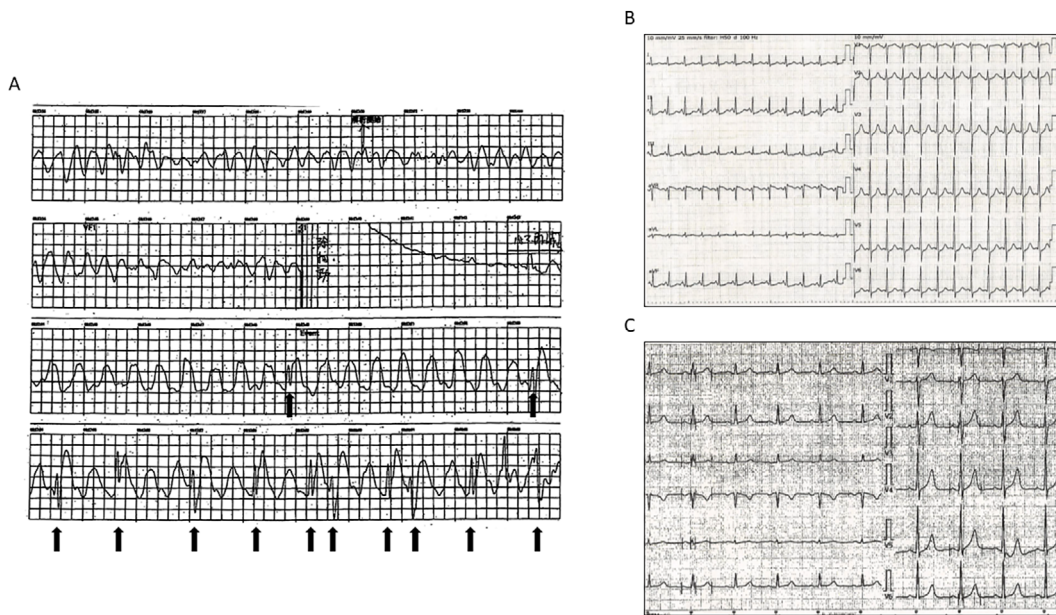


Figure 1. A: VF recorded in the automated external defibrillator. The return of spontaneous circulation was subsequently documented after cardioversion, and the heart rhythm gradually recovered to sinus rhythm (arrow). B: The 12-lead ECG on admission showed sinus tachycardia with ST depression in the inferolateral leads. C: The 12-lead ECG of the patient taken one year ago during a medical checkup.

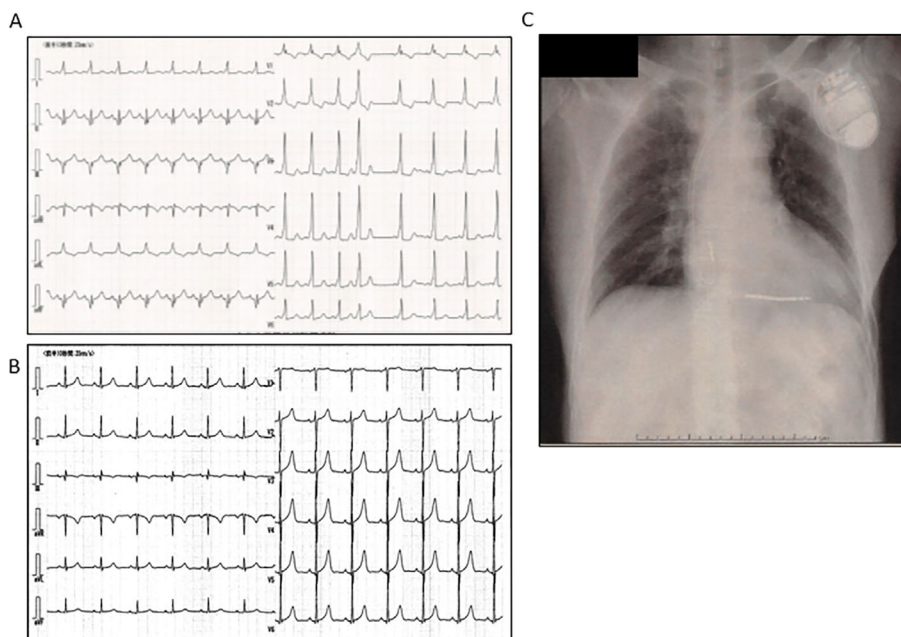


Figure 2. A: Delta-wave was observed during hypothermia treatment. B: After the patient was rewarmed, the delta-wave disappeared and did not reappear at all. C: Chest X-ray after ICD implantation.

sciousness, so cardiac evaluations, including coronary angiography with acetylcholine provocation test, echocardiography, cardiac computed tomography and magnetic resonance imaging, a biopsy of the cardiac muscle, and myocardial scintigraphy, were performed. The results of these tests showed no signs of organic structural heart disease. The signal-averaged ECG was also negative, and a repeated Hol-

ter ECG showed no significant arrhythmias. A pharmacological stress test using pilsicainide was performed to rule out Brugada syndrome. However, coved- or saddleback-type ST-segment elevation in precordial leads was not induced.

An electrophysiology study was conducted. A control intracardiac electrocardiogram (ICE) is shown in Fig. 3A. Retrograde ventriculo-atrial conduction was not observed by

right ventricular pacing (Fig. 3B). Antegrade atrio-ventricular conduction, via an accessory pathway, was also never observed, even after repeated intravenous injection of adenosine (20 mg) (Fig. 4). Therefore, we were unable to perform catheter ablation targeting the accessory pathway. The VT/VF induction test was negative as per the protocol, following the Guidelines of Japanese Circulation Society (6). An implantable cardioverter-defibrillator (ICD) was placed in the patient (Fig. 2C).

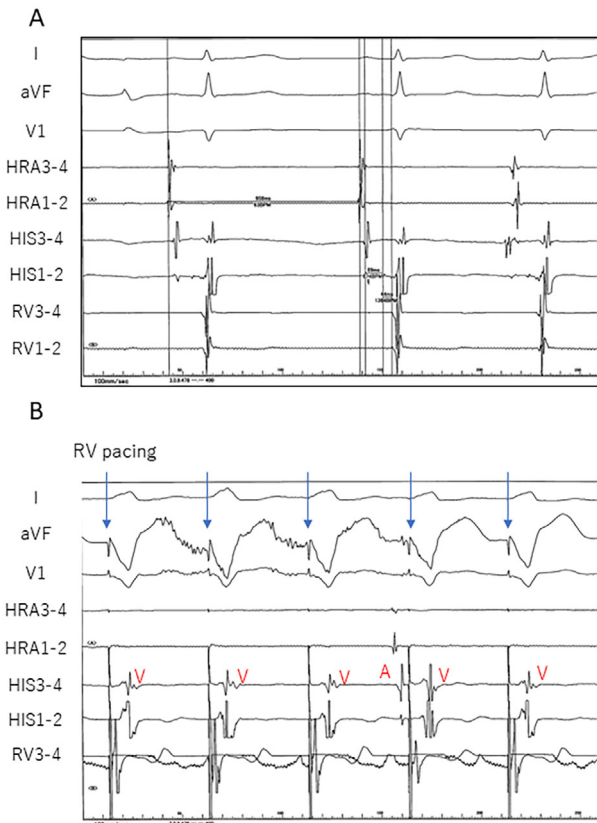


Figure 3. A: Control intracardiac electrocardiogram (ICE). B: ICE during right ventricular pacing. Ventriculo-atrial conduction over the atrio-ventricular node or accessory pathway was not observed.

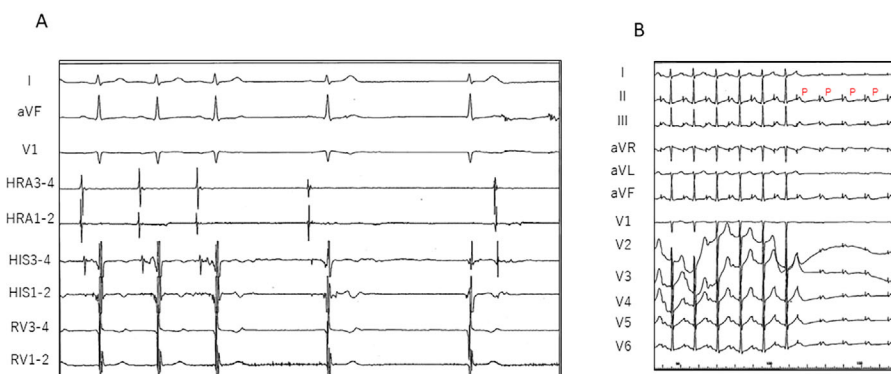


Figure 4. ICE immediately after the rapid intravenous injection of adenosine. A: Sinus bradycardia and junctional rhythm followed the adenosine injection. B: Right atrial pacing failed to reveal any antegrade atrio-ventricular (AV) conduction over an accessory pathway during adenosine injection-induced AV node block.

During 30 months of follow-up, he has had no episodes of VF, paroxysmal atrial fibrillation, or supraventricular tachycardia without any drug treatments.

Discussion

Antz et al. (7) reported that ICD implantation is generally not recommended for resuscitated patients with WPW syndrome who have a normal left ventricular function and no ECG abnormalities suggesting additional electrical disease, as catheter ablation is effective in eliminating accessory pathways. In fact, ICD implantation is not recommended for such patients even in the Guidelines of Japanese Circulation Society (8). In the present case, the etiology of VF was not conclusive. First, we considered the possibility that a rapid ventricular response through an accessory pathway during atrial fibrillation might have caused VF. However, delta-wave was not seen at all after the cessation of hypothermia treatment. In addition, even an electrophysiology study using adenosine showed no evidence of conduction via an accessory pathway. However, there was a possibility that delta-wave might have appeared during hypothermia by chance. Hence, based on these findings, we considered the second possibility of idiopathic VF.

It is difficult to speculate about the mechanism underlying the manifestation of delta-wave during hypothermia. Atrio-ventricular nodal conduction, which depends on calcium channels, may be intensively disturbed during hypothermia. It is quite unlikely that hypothermia directly unmasked the accessory pathway, as accessory pathway conduction is dependent on sodium channels (9). A previous study reported that Ca^{2+} currents tend to be more suppressed during hypothermia than other ion channels (10), which is considered one of the factors responsible for the emphasis on the J-wave during hypothermia. However, prominence of J-wave during hypothermia was not observed in the present case, although this might have been masked by the presence of ventricular preexcitation, as previously reported by us (11).

In conclusion, none of the present patient's 12-lead ECGs

recorded after hypothermia treatment showed any signs of Brugada syndrome or early repolarization syndrome. Thus, his condition was diagnosed as idiopathic VF. More attention should be paid to electrogram recordings of ICD in the future, as they might provide essential evidence for determining the exact etiology of VF.

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

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