

CASE REPORT

Revelation of early repolarization by eliminating accessory pathway in manifest Wolff–Parkinson–White syndrome: A case report

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Email: m_nauchi@tobu.saiseikai.or.jp**Abstract**

A 23-year-old male with manifest Wolff–Parkinson–White syndrome presented with a first occurrence of ventricular fibrillation (VF). Initially, we anticipated the occurrence of atrial fibrillation, causing rapid antegrade conduction over the accessory pathway and, thus, resulting in hemodynamic deterioration. Electrophysiological study revealed that the atrioventricular accessory pathway was located at the mid-septum. After eliminating the pathway, a J-point elevation was revealed in the inferior and lateral leads. In addition, program ventricular stimulation induced VF, and the administration of isoproterenol suppressed VF. In our case, VF occurrence can be attributed to early repolarization syndrome and ventricular preexcitation-modified J-point elevation.

KEYWORDS

early repolarization syndrome, J wave syndrome, preexcitation syndrome, Wolff–Parkinson–White syndrome

1 | INTRODUCTION

Sudden cardiac death can occur in patients with preexcitation syndrome, such as Wolff–Parkinson–White (WPW) syndrome.¹ In such patients, atrial fibrillation (AF) can occur and cause rapid antegrade conduction over the accessory pathway, eventually resulting in hemodynamic deterioration.

Early repolarization (ER) is a common electrocardiogram (ECG) pattern that is characterized by the J-point and ST segment elevations in two or more contiguous leads.² Häissaguerre et al reported that patients with ER pattern have a history of idiopathic ventricular fibrillation (VF).³ The condition associated with the presence of ER pattern in the inferior and/or lateral leads is known as the ER syndrome (ERS). Furthermore, WPW syndrome can modify the presence of ER.⁴

2 | CASE REPORT

A 23-year-old male with a typical “type C” WPW syndrome (Figure 1A) was healthy since his childhood. He neither had any syncope episode nor had a family history of sudden cardiac arrest; however, he experienced an episode of out-of-hospital cardiac arrest for the first time. He was a sailor by profession, and his colleague found him lying at 5:50 AM. ECG recorded using an automated external defibrillator (AED) revealed VF. Subsequently, he received two shocks using AED and VF spontaneously terminated after the second shock (Figure 2A). In addition, after termination of VF, wide QRS tachycardia occurred, which we presume was AF by the accessory pathway (Figure 2B). He was then hospitalized. Thereafter, he did not report any neurological disturbance. Accordingly, amiodarone was initiated for preventing VF, and he was referred to our hospital for intensive examination and treatment.

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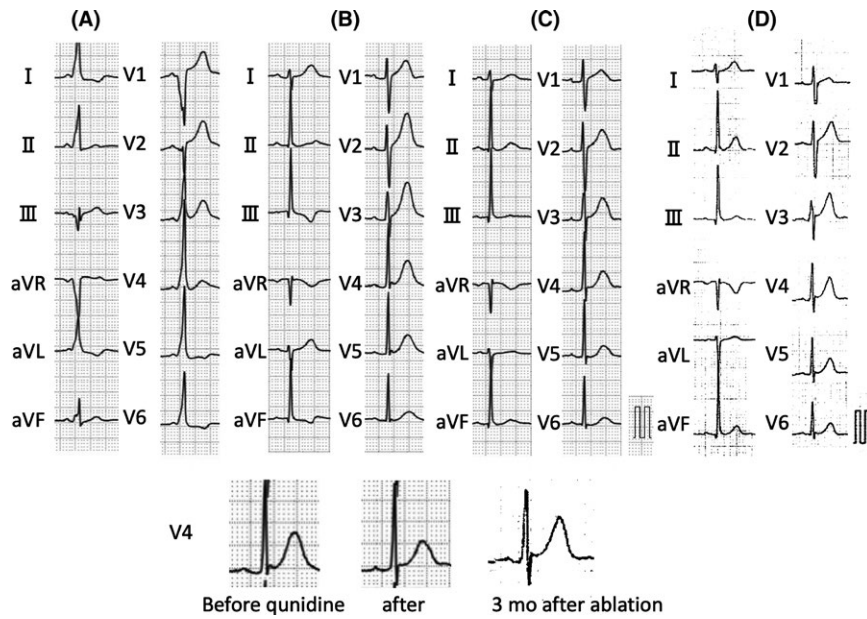


FIGURE 1 Change of 12-lead ECG A, before ablation. B, J-point elevation was slurred in the inferior leads and notched in the lateral leads. C, After quinidine administration, the notched J-point elevation in V5 became smaller. D, 3 months after ablation without quinidine. J-point elevation remained. Inverted T wave in lead III (Fig. 1B) was changing to normal gradually through Fig 1C to D. ECG: electrocardiogram

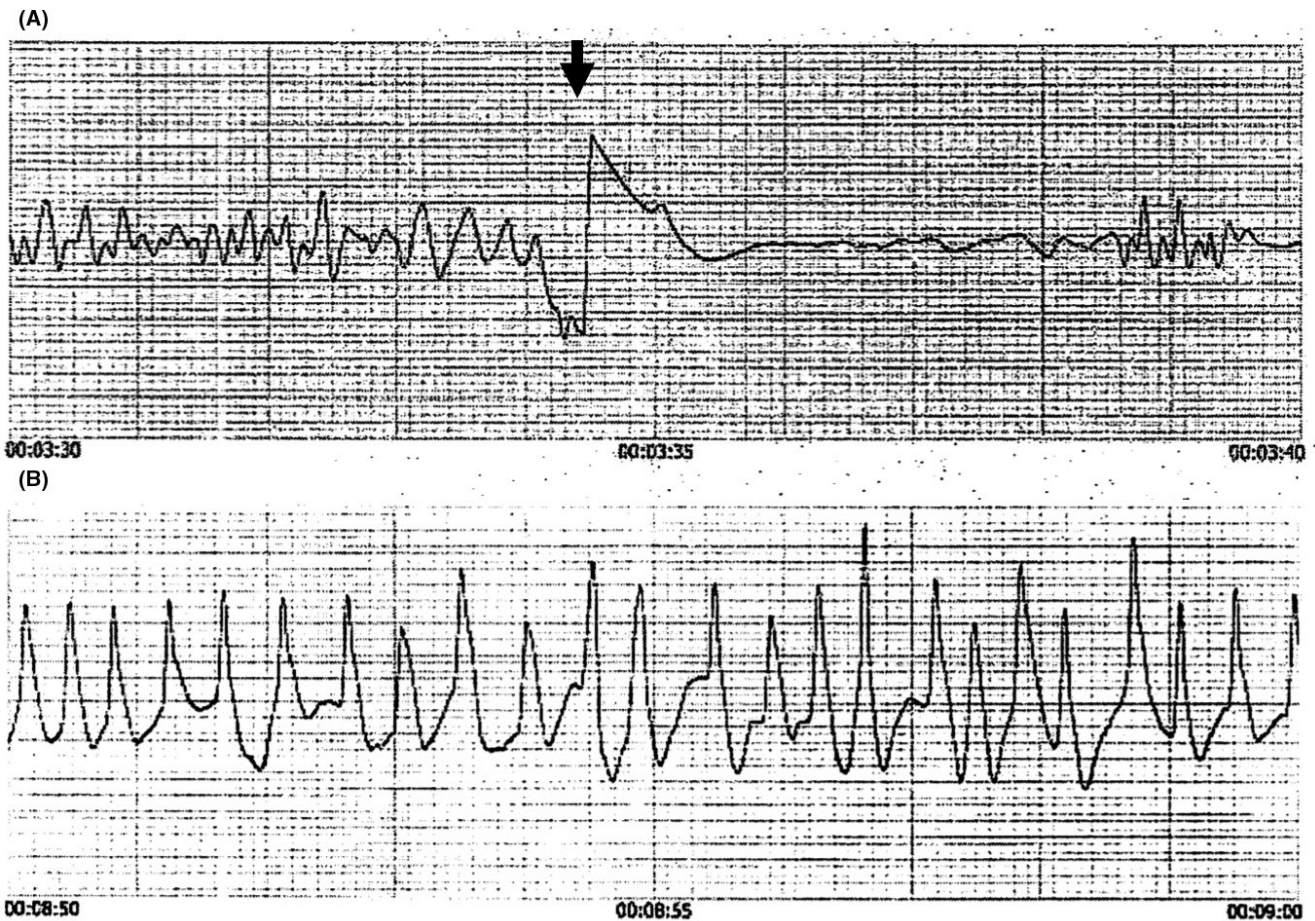


FIGURE 2 ECG connected to AED. A, Second shock (arrow) from AED defibrillated ventricular fibrillation. B, ECG after defibrillation spontaneously changed to atrial fibrillation. AED: automated external defibrillator; ECG: electrocardiogram

The results of echography and coronary angiography were normal, and acetylcholine administration did not provoke coronary artery spasm. Therefore, we performed electrophysiological study (EPS) and catheter ablation. EPS revealed that the accessory pathway of the atrioventricular (A-V) tract was not multiple and was located at the atrioventricular midseptum. In addition, the minimum cycle length of antegrade 1:1 conduction of accessory pathway was 430 ms. The antegrade effective refractory period (ERP) of the accessory pathway was a coupling interval of 340 ms during pacing at a basic cycle length of 600 ms. The Ventriculoatrial (V-A) conduction blocking cycle length was 860 ms. Retrograde V-A conduction had a decremental property. Earliest retrograde atrial activation site was recorded in his bundle electrogram and V-A conduction was considered as via to A-V node. Furthermore, AF was not induced. Although amiodarone could have affected the results, these findings suggested the low risk of life-threatening arrhythmias.

After eliminating the accessory pathway by catheter ablation of the right-side approach, the delta waves disappeared, the A-V conduction decremented, and the retrograde earliest atrial activation site was observed on his bundle electrogram. Adenosine triphosphate blocked the A-V and V-A conduction. We assumed that the tract was completely eliminated. Thereafter, J-point and ST segment elevations were revealed in the inferior and lateral leads (Figure 1B). Considering the possibility of ERS, we performed VF inducibility test. Briefly, VF was induced by the right ventricular outflow tract using two extra-stimuli (pacing cycle length, 400 ms; first coupling interval, 210 ms; and second coupling interval, 200 ms and 190 ms). After intravenously administering isoproterenol (ISP), the ER pattern became unclear, and VF was not induced by any program stimulation.

After EPS, the intravenous administration of pilsicainide (1 mg/kg) infusion exhibited no remarkable change in his ECG. Thus, we concluded that VF was not induced by preexcitation syndrome but by ERS. Furthermore, we prescribed quinidine (300 mg/day), following which the J-point elevation became obscure (Figure 1C). He received an implantable cardioverter defibrillator at another hospital. 3 months later, these changes remained (Figure 1D).

3 | DISCUSSION

Sudden cardiac death can occur in patients with WPW syndrome. According to EPS, a high risk of sudden death occurs when the ERP of the accessory pathway is <250 ms,¹ and a rapid A-V conduction can cause VF. Although the patient took amiodarone for about 20 days, the findings in EPS were not related to high risk.

The prevalence of ER pattern in patients with WPW syndrome before and after successful ablation has been previously reported.⁴ Takahashi et al reported a case with "intermittent" WPW syndrome and VF in which the J-point elevation wave presented in only the inferior leads after the accessory pathway disappeared.⁵ On the

contrary, our case is different with regards to the "manifest" WPW syndrome, J-point elevation in both lateral and inferior leads, and programmed stimulation induced VF. In this case, ERS was first recognized only after ablation. In fact, the ER pattern might have been masked by the presence of delta waves.

Although the mechanism of ER remains unclear, the hypothermic J waves on ECG reflect an increased dispersion of repolarization caused by a disproportionate abbreviation of the epicardial action potential compared with that of the endocardium. In addition, the presence of a transient potassium outward current (I_{to}) is seemingly associated with ERS and Brugada syndrome (BrS). However, some overlap exists between ERS and BrS. Medications, such as quinidine and ISP, that are efficacious in BrS have also proven efficacy for ERS.³ In this case, programmed ventricular stimulation induced VF and the administration of ISP suppressed it.

This case further supports the opinion that sudden death in patients with WPW syndrome can be not only caused by AF but also by ERS.

CONFLICT OF INTEREST

The authors declare no conflict of interests for this article.

REFERENCES

1. Lundqvist CB, Scheinman MM, Aliot EM, et al. ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias. Executive summary: A report of the American college of cardiology/American heart association task force on practice guidelines and the European society of cardiology committee for practice guidelines. *Circulation* 2003;108:1871-909.
2. Priori SG, Wilde AA, Horie M, et al. Executive summary: HRS/EHRA/APHRS expert consensus statement on the diagnosis and management of patients with inherited primary arrhythmia syndromes. *Heart Rhythm*. 2013;10:e85-108.
3. Haïssaguerre M, Derval N, Sacher F, et al. Sudden cardiac arrest associated with early repolarization. *N Engl J Med*. 2008;358:2016-23.
4. Mizumaki K, Nishida K, Iwamoto J, et al. Early repolarization in Wolff-Parkinson-White syndrome: Prevalence and clinical significance. *Europace*. 2011;13:1195-200.
5. Takahashi N, Shinohara T, Hara M, Saikawa T. Wolff-Parkinson-White syndrome concomitant with idiopathic ventricular fibrillation associated with inferior early repolarization. *Intern Med*. 2012;51:1861-4.

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