Sodium channel blockade unmasked Brugada electrocardiographic pattern in a patient with complete right bundle branch block and early repolarization in the lateral leads

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Key Clinical Message
Early repolarization syndrome (ERS) and Brugada syndrome (BrS) share many electrocardiographic and clinical features, and recently have been collectively grouped as J wave syndrome. However, the effects of sodium channel blockers on the J waves differ greatly between ERS and BrS.

Keywords
Brugada syndrome, early repolarization, sodium channel blocker.

Introduction
Early repolarization syndrome (ERS) and Brugada syndrome (BrS) share many electrocardiographic and clinical features, and recently have been collectively grouped as J wave syndrome [1, 2]. However, the effects of sodium channel blockers on the J waves differ greatly between ERS and BrS [2]. Sodium channel blocker is extremely effective for deciding the therapeutic strategy in a patient after cardiopulmonary arrest due to ventricular fibrillation (VF) complicated by complete right bundle branch block (CRBBB) and early repolarization (ER) in the lateral leads.

Case Report
A 64-year-old man experienced VF at home early in the morning. He was resuscitated with an automated external defibrillator and was admitted to our hospital. He had no family history of sudden cardiac death and a genetic study including for SCN5A, was negative. Echocardiogram revealed normal left ventricular ejection fraction (LVEF = 75%) without any local asynergy or valvular heart disease and cardiac MRI showed no evidence of delayed enhancement. A 12-lead electrocardiogram (ECG) revealed CRBBB with ER in the lateral leads (Fig. 1A). QRS morphologies of right precordial leads (V1, V2) in third intercostal spaces were almost similar to those in fourth intercostal spaces (Fig. 1B). A drug challenge test was performed with pilsicainide, a pure sodium channel blocker (1 mg/kg body weight in 10 min), to clarify the mechanisms of the idiopathic VF. An intravenous administration of pilsicainide after 4 min, unmasked a coved-type ST-segment elevation in the inferior leads and spontaneous frequent premature ventricular contractions that finally resulted in VF (Fig. 2). Pharmacological intervention with pilsicainide revealed the features of BrS, a coved-type ST-segment elevation, and therefore, the administration of quinidine with an implantable cardioverter defibrillator (ICD) implantation was started. Early repolarization in the
Figure 1. (A) A 12-lead electrocardiogram before the administration of quinidine. Right precordial leads (V1, V2) in fourth intercostal spaces. The 12-lead electrocardiogram shows complete right bundle branch block with early repolarization in the lateral leads. (B) A 12-lead electrocardiogram before the administration of quinidine. Right precordial leads (V1, V2) in third intercostal spaces. QRS morphologies of right precordial leads (V1, V2) in third intercostal spaces were almost similar to those in fourth intercostal spaces. (C) A 12-lead electrocardiogram after the administration of quinidine and an implantable cardioverter defibrillator implantation. An atrial pacing rate of 80 ppm was observed and the early repolarization in the lateral lead disappeared on the 12-lead electrocardiogram.
lateral leads disappeared after taking oral quinidine and atrial pacing at 80 ppm (Fig. 1C), and he had received no appropriate ICD shocks due to ventricular arrhythmias within 3 years after the implantation.

Discussion

A high incidence of an ER pattern in the inferolateral leads has been reported in patients with idiopathic VF [3–5]. Implantable cardioverter defibrillator implantations are recommended in patients with ERS who have survived a cardiac arrest, however, the medical therapy for ERS is still challenging [6], and on the other hand, quinidine is effective for preventing recurrence of ventricular arrhythmias in BrS [7].

Early repolarization syndrome and BrS share many characteristics including an adult onset, male preponderance, cardiac events at rest or during sleep, and rare ventricular arrhythmias during Holter ECG monitoring, and have now been collectively grouped as J wave syndrome; however, the effects of a sodium channel blockade on the J waves differ greatly between ERS and BrS [1]. After an injection of a sodium channel blocker, the J waves in BrS become more prominent, while those in ERS either do not change or become attenuated. The Brugada Consensus Report proposed that a sodium channel blockade is required to diagnose BrS in the absence of a coved-type ST-segment elevation [2]. Moreover, some reports have demonstrated that the J waves in BrS are concealed by CRBBB [8, 9]. In this case, the 12-lead ECG also exhibited CRBBB, which masked the typical coved-type ST-segment elevation. Therefore, the intravenous administration of a sodium channel blockade was extremely useful for clarifying the underlying disease of the idiopathic VF and quinidine with high-rate atrial pacing was able to prevent any VF recurrence with the disappearance of the ER in the lateral leads for 3 years.

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Conflict of Interest

None declared.

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