Recording of isolated very delayed potentials on the right ventricular epicardium in a patient with Brugada syndrome

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Introduction

Epicardial mapping revealed the existence of abnormal potentials on the right ventricular outflow tract (RVOT) in patients with Brugada syndrome.[1] It is reported that tissues of the RVOT epicardium from open-chest biopsy and autopsy showed the existence of fibrosis and fatty infiltration associated with increased interstitial collagen and decreased connexin 43.[2] Although the abnormal potentials can be explained by repolarization abnormality[3] of delayed upstroke of the action potential dome and concealed phase 2 reentry,[4] these abnormal potentials are associated with epicardial tissue fibrosis[2] and include the mechanism of delayed conduction.

We experienced a case of Brugada syndrome and frequent ventricular fibrillation (VF) episodes. The patient had abnormal epicardial delayed potentials that have been reported previously and also an unusual isolated very delayed potential (IVDP) that was reproducibly recorded 400–500 ms after the last ventricular activation on the epicardium of the RVOT.

Case report

The patient was a 24-year-old man without any organic heart disease. He experienced syncope after drinking alcohol at night and visited a hospital. The rhythm was atrial fibrillation, and an electrocardiogram (ECG) did not show any other abnormalities. A doctor therefore injected pilsicainide, a pure sodium channel blocker, to terminate the arrhythmia. VF occurred spontaneously during the injection of pilsicainide and was successfully terminated by cardioversion. The ECG was typical type 1 ECG (Figure 1A, Left panel), and he was diagnosed as having Brugada syndrome. Gene analysis showed that the patient did not have SCN5A mutation.

After implantation of an implantable cardioverter-defibrillator, frequent appropriate shocks to terminate VF were documented despite medical treatment (bepridil at 200 mg/day, cilostasol at 100 mg/day, and continuous infusion of isoproterenol). We therefore planned emergent ablation to suppress the drug-refractory VF attacks.

Under general anesthesia, 2 vascular sheaths were placed in the right femoral vein for a quadripolar catheter and an ablation catheter (Navistar ThermoCool SmartTouch; Biosense Webster, Diamond Bar, CA) with an 8F irrigated D-curve in the right ventricle (RV). We then performed endocardial RV voltage mapping using an electroanatomic mapping system (CARTO 3; Biosense Webster, Diamond Bar, CA). However, we found very few abnormal low-voltage potentials on the endocardial surface of the RV free wall. Then we performed epicardial mapping via a subxiphoid approach. A 7F decapolar catheter (DECANAV; Biosense Webster, Diamond Bar, CA) was advanced into the pericardial space via an epicardial access through an Agilis (St. Jude Medical, Minnetonka, MN) sheath under electroanatomic and fluoroscopic guidance. We performed electroanatomic mapping on the epicardial surface of the RV. Epicardial voltage mapping revealed very extensive abnormal potentials, especially from below the pulmonary artery valve to above the tricuspid annulus (Figure 1B). Clustering of prolonged split potentials, local abnormal ventricular activity, and complex fractionated ventricular signals were noted on the epicardium of the RVOT as well as the mid free wall. The total number of mapping points using a decapolar catheter and an ablation catheter was 272. We could find the variable activation time according to shortening of coupling intervals of extrastimuli. Moreover, IVDPs appeared about 400–500 ms after the last ventricular activation captured by the

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KEY TEACHING POINTS

- Epicardial voltage mapping revealed very extensive multiple abnormal potentials, especially from below the pulmonary artery valve to above the tricuspid annulus.
- Isolated very delayed potentials (IVDPs) on the right ventricle epicardium appeared 500 ms after the last stimulus beats. The appearance of IVDPs was associated with the last stimulus beats, and the response of the IVDPs to ventricular pacing was unusual.
- Turning on the electricity for ablation immediately eliminated local abnormal potentials within 1–5 seconds. After the ablation, ventricular fibrillation attacks were completely suppressed.

extrastimuli. IVDPs were discrete low-voltage potentials with 1–3 sharp spikes, and there were no electrical activities between the last ventricular activation by extrastimuli and IVDPs. This potential appeared by ventricular pacing and did not reflect any potential on the body-surface ECG (Figure 2). The interval between the stimulus (St) and IVDPs was slightly reduced by shortening of the coupling interval of extrastimuli, but it was later gradually prolonged. When the extrastimulus captured local ventricular activation with a significant delay, the appearance of IVDPs was also delayed in parallel (Figure 3). Programmed electrical stimulation and rapid pacing from the endocardium of the RV apex and RVOT and the epicardium of the RVOT failed to induce VF. We performed radiofrequency catheter ablation targeting these fractionated and late potentials using the vector of the ablation catheter. Turning on the electricity for ablation immediately eliminated local abnormal potentials within 1–5 seconds. Extensive ablations (power of 30–35 W, temperature of $<42^{\circ}C$, 30 seconds/point) were carried out along the area of the RV epicardium having the abnormal potentials. The total number of ablations was 41 points and ablation time was 26 minutes. The ST elevation in V1-V2 leads decreased and the VF attacks were suppressed after the ablation (Figure 1A, Right panel). The patient, after the electrophysiological study, has remained asymptomatic, with no further occurrence of ventricular tachycardia/VF episodes for 15 months.

Discussion

We presented a case of Brugada syndrome in which frequent VF episodes occurred. Epicardial mapping of the RV showed extended delayed potentials that have been reported previously.[1,5,6] Moreover, the patient had IVDPs on the RV epicardium that appeared 400–500 ms after the last St

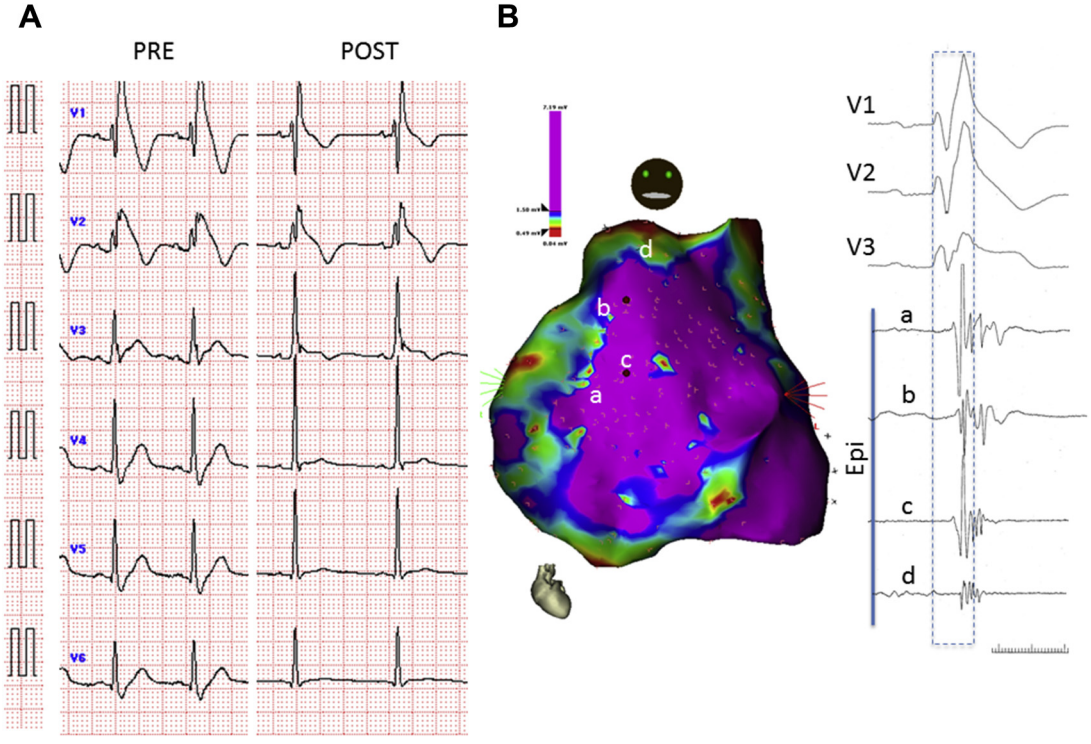

Figure 1 **A:** Left panel: Twelve-lead electrocardiogram (ECG) showing sinus rhythm and typical coved-type pattern with Brugada syndrome. Right panel: The ST elevation of V1 and V2 lead was reduced after the ablation. **B:** Multiple abnormal potentials were found on the epicardium at the right ventricular outflow tract: (a) high-voltage + low-frequency delayed potential, (b) double potential, (c) high voltage + fragmentation, (d) low voltage + fragmentation.