Epsilon wave disappearance by catheter ablation for ventricular arrhythmia from the left ventricular outflow tract

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Introduction

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is pathologically characterized by fibrofatty replacements.[1] It is reported that the subtricuspid/peritricuspid area and the left ventricular inferolateral wall may be the only affected regions in the early stages of ARVC.[2] This lipomatous or lipofibromatous composition causes delayed excitation. This late conduction through diseased myocardium is described as an epsilon wave, which is included in the revised Task Force diagnostic criteria.[3] Herein, we report a case of ARVC with epsilon wave, in which radiofrequency catheter ablation (RFCA) on left ventricular outflow tract (LVOT) eliminated the epsilon wave.

KEY TEACHING POINTS

- Early activation in premature ventricular complexes (PVCs) and a low-amplitude electrogram during sinus rhythm were not revealed in the right ventricular outflow tract.
- Radiofrequency catheter ablation targeting frequent PVCs from the left ventricular outflow tract eliminated the epsilon wave.
- Epicardially extending myocardial strands affected by the remodeling of arrhythmogenic right ventricular cardiomyopathy may be the cause of the epsilon wave.

Case report

A 26-year-old Japanese man had a mild dilatation and dysfunction of the right ventricle (RV) on a medical checkup and was subsequently referred to our hospital. Cardiac magnetic resonance (CMR) imaging showed regional RV akinesis in the anterior wall, a mild increase in RV end-diastolic volume /body surface area ratio of 107.7 mL/m^2, and a reduced RV ejection fraction (RVEF) of 41.6%, but without late gadolinium enhancement (Figure 1). Electrocardiography (ECG) showed an epsilon wave and inverted T waves in the V_1 and V_2 leads, as well as an incomplete right bundle branch block (RBBB) (Figure 2A). Although the 12-lead ECG showed frequent ventricular extrasystoles, the Holter monitoring did not show $\geq$500 ventricular extrasystoles per 24 hours. No relatives were previously diagnosed with ARVC or had sudden cardiac death. Endomyocardial biopsy and genetic testing were not performed. Chest computed tomography with contrast showed no abnormalities in the coronary arteries and lung field, nor dilated pulmonary artery. In the Revised Task Force criteria,[3] 1 major and 2 minor criteria were satisfied, and the diagnosis of ARVC was confirmed. As the patient did not show left ventricular dysfunction or any history of ventricular tachyarrhythmia, we instructed him to avoid high-intensity endurance exercises and followed him annually without medical or implantable cardioverter-defibrillator therapy.[4]

Four years later, the patient complained of palpitation, and the Holter ECG showed an increased burden of premature ventricular complexes (PVC) (10%–16%). Repeat CMR imaging showed a further decrease in the RVEF (29.4%), whereas the RV end-diastolic volume / body surface area ratio was unchanged and late gadolinium enhancement was not observed. As the pharmacological treatment with metoprolol did not reduce the PVC frequency, we performed RFCA.

The 12-lead ECG with PVC revealed a left bundle branch block morphology with a northwest axis deviation and QS

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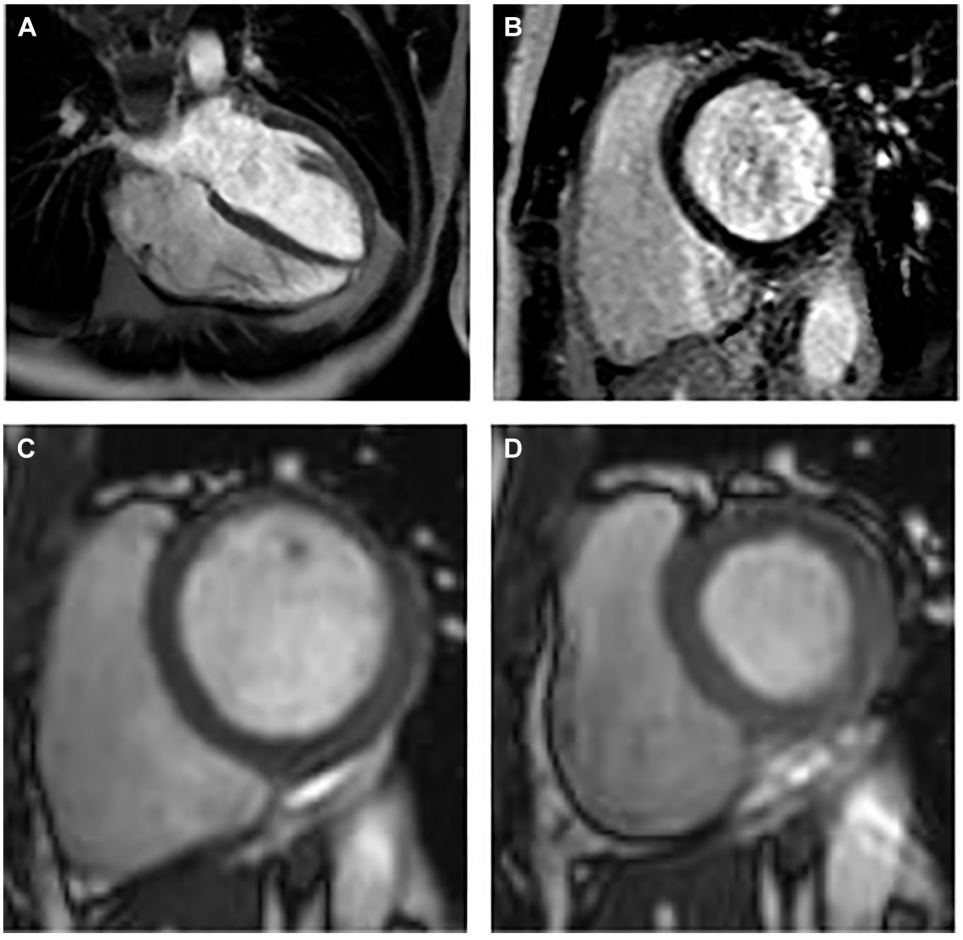

Figure 1 Cardiac magnetic resonance imaging showing a mild increase in the right ventricular end-diastolic volume without late gadolinium enhancement (**A** and **B**). Short-axis views in the end-diastole (**C**) and end-systole (**D**) showing regional right ventricle akinesis in the anterior wall.

morphology in the V_1 lead, with notching on the downward deflection (Figure 2A), suggesting its origin from the right coronary cusp–left coronary cusp (RCC–LCC) commissure.[5] Anatomically, the RCC–LCC commissure abutted the mid-septal aspect of the RV outflow tract (RVOT); we initially performed mapping in the RVOT, which did not reveal early activation in PVC or low-amplitude electrogram during sinus rhythm. Substrate mapping of the entire RV was not performed. Next, we mapped the aortic root and observed a discrete potential activated 60 ms earlier than the QRS onset in the RCC–LCC commissure (Figure 3A, red point). The RF application in this region only decreased the PVC frequency, but did not eliminate the PVC. Further mapping in the LVOT beneath the aortic cusp showed a discrete potential 83 ms earlier than the QRS onset (Figure 3A, blue point). Additionally, a delayed activating potential was observed during sinus rhythm (Figure 3A, white arrow), although without a late potential. The RF application at this point eliminated the PVC completely. Furthermore, the delayed activating potential almost disappeared. Interestingly, the 12-lead ECG showed a clear alteration of the notching at the end of the QRS complex in leads III, aVf, and V_1–V_3 (Figure 2B). Of note, the QRS morphology during sinus rhythm showed complete RBBB because of catheter contact during the mapping in the RVOT. An ECG after RFCA without complete RBBB revealed the epsilon wave disappearance (Figure 3B, right panel). CMR imaging after 4 months showed no improvement in RVEF and RV mild dilation. Repeat ECG recordings consistently showed the absence of epsilon waves. The Holter ECG after RFCA showed only a few PVCs (0.2%), and the patient was free from palpitation for ˃6 months, with no epsilon wave reappearance.

Discussion

To the best of our knowledge, this is the first report showing that ablative therapy from left-side outflow tract modified the epsilon waves.

Epsilon wave is prominent in the right precordial leads and manifests as a low-amplitude potential between the end of the QRS complex and the beginning of the T wave.[6] Epsilon waves are believed to represent delayed activation of the RV free wall, and their presence reflects more diffuse RV involvement. The current case differs in 2 points. First, we observed delayed activation not in the RVOT, but in the LVOT opposite to the septal aspect of the RVOT. Second, epsilon wave was observed in a patient with structurally moderate RV remodeling. With the fact that the delayed potential was observed in the anteroseptal LVOT and the epsilon

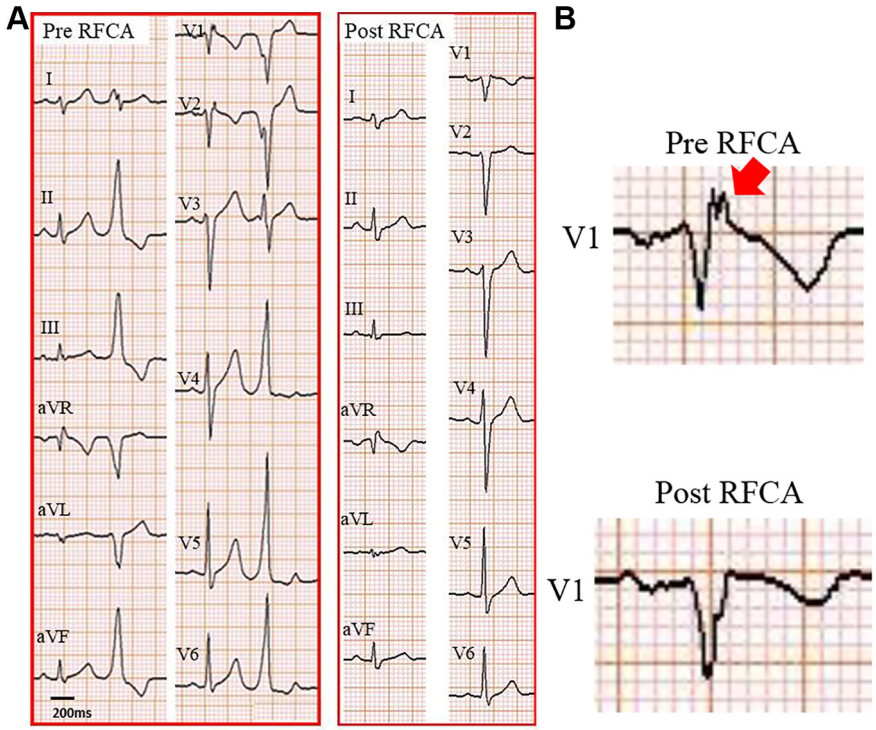

Figure 2 **A:** Twelve-lead electrocardiography (ECG) before (*left*) and after (*right*) radiofrequency catheter ablation (RFCA). **B:** Magnified ECG in the V^1 lead before (*top*) and after (*bottom*) RFCA.

waves disappeared by ablation on this area, we believe that delayed activated myocardium was present in the epicardial side of the septal LVOT, and that ablative intervention electrically isolated these myocardia or modified the conduction pattern in this area, leading to the disappearance of the epsilon waves.

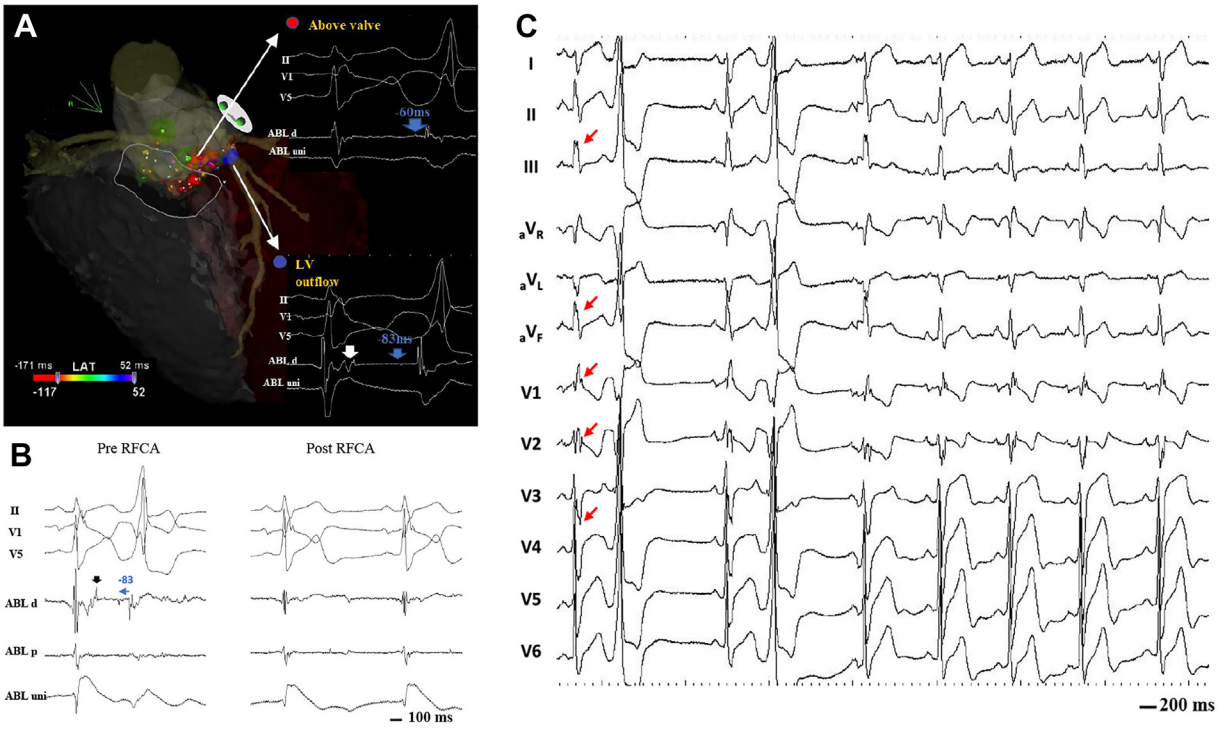

Figure 3 **A:** Activation map for the premature ventricular complex (PVC) in the aortic root and left ventricular outflow tract (LVOT) and intracardiac electrograms. **B:** Intracardiac electrograms before and after ablation. Of note, a sharp delayed activating potential (*black arrow*) during sinus rhythm before ablation almost disappeared after radiofrequency application. **C:** Twelve-lead electrocardiography during ablation at the LVOT (*blue point* in Figure 2A). The radiofrequency application eliminated the PVC and changed the notching of QRS waves during sinus rhythm (*red arrows*).

ARVC is a progressive disease, and patients do not always show all diagnostic manifestations at the initial assessment. Kirubakaran and colleagues[7] reported the early-stage ARVC patients without structural remodeling and that these patients have limited scarring in the epicardial RV outflow. Epsilon waves are known as a maker of the depolarization disorder of ARVC.[6] However, it is of note that ARVC also shows various ECG changes related to the depolarization disorder: terminal activation delay and RBBB, in addition to Epsilon waves.[8] Actually, in our case, low-amplitude signals in leads V_1–V_2 were observed at the same timing of the terminal of the QRS complex, and one might define the ECG change as atypical RBBB patterns.[9] Tanawuttiwat and colleagues[8] analyzed the association between these depolarization disorders and the anatomical location with scarring and delayed activation, in which they showed the latest activated area was located in the basal RVOT and free wall dominantly in the epicardial side. Recent review article by Anderson and colleagues[10] described that myocardial strands extend distally beyond the anatomic ventriculoarterial junction toward the nontubular junction. Importantly, these myocardial strands are opposite to the LVOT in the anteroseptal direction, and therefore we speculate that these epicardially extending myocardial strands were affected by the remodeling of ARVC, showing the epsilon waves in our case.

There have been several reports describing the modification of the QRS complex in ARVC patients. Nogami and colleagues[11] reported that ablation eliminated isolated delayed components in the intracardiac electrograms, which corresponded to the modification of the signal averaged electrocardiogram. Recently, Lee and colleagues[12] reported a case in which extensive low-voltage area and scar over the RVOT and body were demonstrated, and epicardial ablation suppressed the epsilon wave. On the contrary, Caldwell and colleagues[13] stated that the extensive ablation for RVOT produced local delayed activation and manifested epsilon waves. In contrast to these above cases, it is interesting that our case showed that ablation from anteroseptal LVOT modified the epsilon waves in an ARVC patient with structurally moderate RV remodeling.

Conclusions

The epsilon wave disappeared after RFCA, which eliminated the PVC originating from LVOT. Epicardially extending myocardial strands affected by the remodeling of ARVC may be the cause of the epsilon wave.

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