Case Report

Ablation of Idiopathic Ventricular Tachycardia with a Left Bundle-Branch Block Morphology Originating from the Pulmonary Artery

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We successfully performed radiofrequency catheter ablation (RFCA) in 2 cases involving patients with idiopathic ventricular tachycardias (VTs) and premature ventricular contractions (PVCs) originating from the pulmonary artery (PA). The QRS morphology of the VTs and PVCs in the two cases exhibited a left bundle-branch block (LBBB) morphology with an inferior axis. Activation and pace mappings were performed in the right ventricular outflow tract (RVOT) and above the pulmonary valve to determine the origin of the VTs and PVCs. In both cases, the earliest ventricular activation was recorded in the PA above the pulmonary valve. Applications of radiofrequency current at those sites in the PA resulted in the elimination and noninducibility of the VT and PVC. During the follow-up, the VT or PVC did not recur without any antiarrhythmic drug administration.
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Key words: Pulmonary valve, Premature ventricular contractions, Activation mapping, Pace mapping, CARTO

Introduction

Radiofrequency catheter ablation (RFCA) is an established curative therapy for idiopathic ventricular tachycardias (VTs) or symptomatic premature ventricular contractions (PVCs) with a left bundle-branch block (LBBB) morphology originating from the outflow tract (OT) in structurally normal hearts. Most of those arrhythmias have their origin located on the septal side of the right ventricular outflow tract (RVOT). However, some originate from the free wall of the RVOT, left ventricular outflow tract (LVOT) or aortic sinus cusp (ASC). Additionally, several reports have indicated that some idiopathic VTs originate from the pulmonary artery (PA) and can be ablated successfully in the PA. In this report, we discuss 2 cases with idiopathic VTs originating from the PA.

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Case Report

Case 1

A 38-year-old woman with no previous cardiac history visited the hospital because of palpitations associated with dizziness. Physical examination revealed no abnormalities. The 12-lead surface ECG revealed normal sinus rhythm with frequent PVCs. The PVCs had an LBBB morphology, inferior-axis, QS pattern in lead I, and R/S ratio >1 in lead V4 (**Figure 1**(A)). The R-wave amplitude of the PVCs in the inferior leads were 1.9 mV(II), 1.9 mV(III), and 2.1 mV(aVF). Twenty-four-hour Holter monitoring revealed a repetitive nonsustained ventricular tachycardia (NSVT) of up to 12 beats with the same morphology as the PVCs. We suspected an idiopathic VT originating from the anterior-septal side of the RVOT. The patient was then referred to our institution for an electrophysiologic study and RFCA. Her laboratory data were normal, and the presence of structural heart disease was negated by echocardiography. Coronary angiography did not reveal any significant stenosis of the left or right coronary arteries.

Electrophysiologic study and ablation procedure

After obtaining informed consent the procedure was performed with the patient under local anesthesia. Three catheters were inserted via the right femoral vein into the right atrium, His-bundle region, and RVOT under fluoroscopic guidance. One catheter was inserted via the internal carotid vein into the coronary sinus (CS). During the study, the presence of frequent spontaneous clinical VTs or PVCs was observed, so we did not use programmed stimuli to induce clinical VT or PVCs. While the mechanisms of those VTs and PVCs were not ascertained, we thought that triggered activity was the possible mechanism for those VTs and PVCs as is the usual case with idiopathic VTs with an LBBB morphology. At first we performed activation and pace mapping of the RVOT during the spontaneous clinical VT or PVCs. An endocardial activation preceding the QRS complex of the PVC by 30 msec

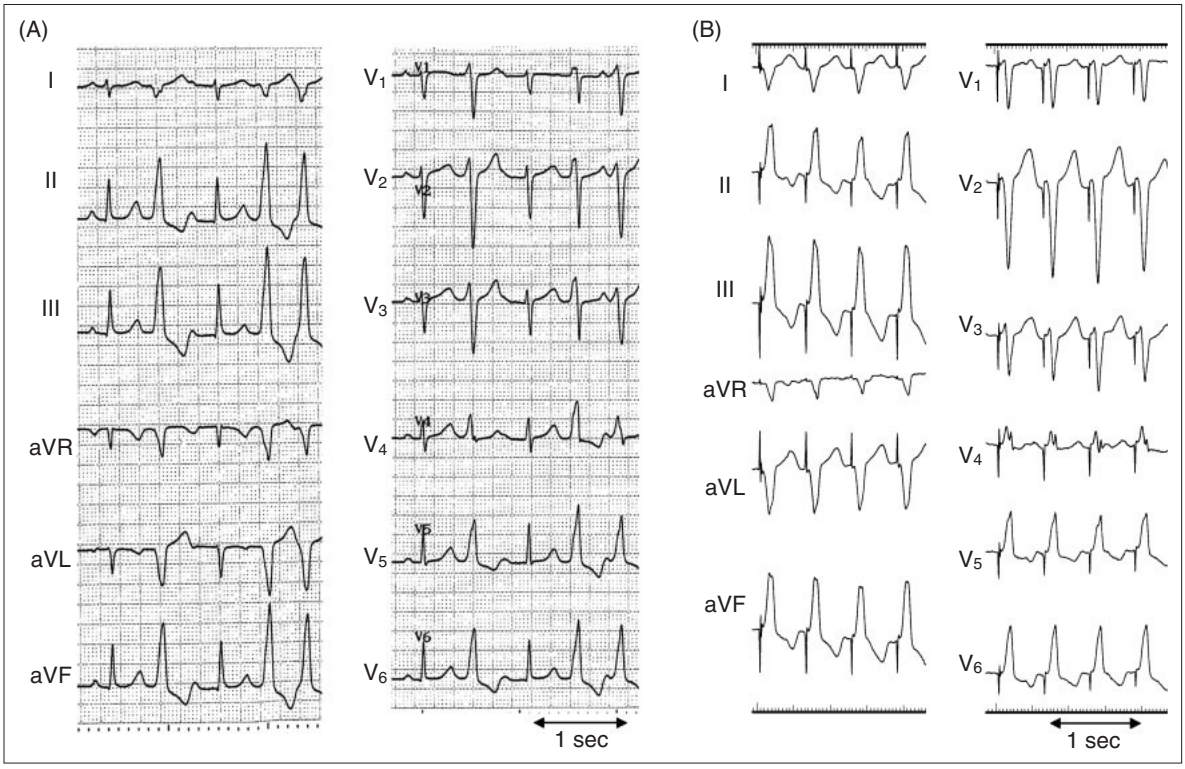

Figure 1

(A) A surface 12-lead ECG during sinus rhythm with spontaneous premature ventricular contractions (PVCs). The QRS morphology during the PVCs exhibited an inferior axis, left bundle branch block, QS pattern in lead I, and R/S ratio >1 in lead V4. The R-wave amplitude of the PVCs in leads II, III, and aVF was 1.9 mV, 1.9 mV, and 2.1 mV, respectively.

(B) A surface 12-lead ECG showing pace mapping from the anterior-septal side of the right ventricular outflow tract (RVOT) just under the pulmonary valve. The R-wave amplitude in leads V2 and V3 during pace mapping were lower than those of the clinical VT or PVCs.