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CLINICAL CASE REPORT

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Dynamic Changes of QRS Morphology of Premature Ventricular Contractions During Ablation in the Right Ventricular Outflow Tract

A Case Report

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Abstract: Electrocardiographic characteristics can be useful in differentiating between right ventricular outflow tract (RVOT) and aortic sinus cusp (ASC) ventricular arrhythmias. Ventricular arrhythmias originating from ASC, however, show preferential conduction to RVOT that may render the algorithms of electrocardiographic characteristics less reliable. Even though there are few reports describing ventricular arrhythmias with ASC origins and endocardial breakout sites of RVOT, progressive dynamic changes in QRS morphology of the ventricular arrhythmias during ablation obtained were rare.

This case report describes a patient with symptomatic premature ventricular contractions of left ASC origin presenting an electrocardiogram (ECG) characteristic of right ventricular outflow tract before ablation. Pacing at right ventricular outflow tract reproduced an excellent pace map. When radiofrequency catheter ablation was applied to the right ventricular outflow tract, the QRS morphology of premature ventricular contractions progressively changed from ECG characteristics of right ventricular outflow tract origin to ECG characteristics of left ASC origin.

Successful radiofrequency catheter ablation was achieved at the site of the earliest ventricular activation in the left ASC. The distance between the successful ablation site of the left ASC and the site with an excellent pace map of the RVOT was 20 mm.

The ndings could be strong evidence for a preferential conduction via the myocardial bers from the ASC origin to the breakout site in the right ventricular outflow tract. This case demonstrates that ventricular arrhythmias with a single origin and exit shift may exhibit QRS morphology changes.

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Abbreviations: ASC = aortic sinus cusp, ECG = electrocardiogram, IVTs = idiopathic ventricular tachycardias, LBBB = left bundle branch block, LVOT = left ventricular outflow tract, PVCs = premature ventricular complexes, RFCA = radiofrequency catheter ablation, RVOT = right ventricular outflow tract.

INTRODUCTION

Idiopathic ventricular arrhythmias, including premature ventricular complexes (PVCs) and idiopathic ventricular tachycardias, mainly originate from the right ventricular outflow tract (RVOT), with a small part of them originating from the left ventricular outflow tract (LVOT).[1] Electrocardiographic characteristics can be useful in differentiating between idiopathic RVOT and aortic sinus cusp (ASC) ventricular arrhythmias.[1,2] The ventricular arrhythmias originating from the ASC, however, show preferential conduction to the RVOT, which may render the algorithms using the electrocardiographic characteristics less reliable.[3–5] This case report describes a patient with symptomatic PVCs of the left ASC origin presenting an electrocardiographic characteristic of RVOT before ablation.

Case Presentation

The patient was a 50-year-old Chinese woman with a one-year history of frequent palpitation. The baseline electrocardiogram (ECG) revealed frequent monomorphic PVCs (Fig. 1A). The QRS morphology of the PVCs was characterized by a RS pattern in lead I, a monomorphic R pattern in leads II, III, aVF, and V4-V6, a RS pattern in leads V1 and V2, a QS pattern in leads aVR and aVL, and left bundle branch block pattern (LBBB) with inferior axis and precordial transition in lead V3. The R-wave amplitude in leads III, and aVL were greater than that in leads II, and aVR, respectively. R-wave duration index[1] (calculated as a percentage by dividing the QRS complex duration by the R-wave duration in lead V1) was 46%, and R/S-wave amplitude ratio in lead V1 was 36%. The ECG characteristics suggested the PVCs origin from the RVOT or the left ASC. The preoperative 24 hour of ambulatory Holter monitoring showed isolated monomorphic PVCs and the PVC burden was 25,124 (23.7%). Recently, Sardu et al[6] found that metabolic syndrome is associated with a poor outcome in patients affected by outflow tract premature ventricular contractions treated by catheter ablation. In the current report, the patient had no metabolic syndrome. No obesity was observed (body mass index: 23.4). Laboratory testing showed normal C-reactive protein, electrolytes, cardiac troponin T, and brain natriuretic peptide. No hypertension, dyslipidemia, and diabetes were found. The chest x-ray film was normal. Echocardiography revealed normal ventricular function and no structural abnormalities. Exercise electrocardiogram testing was negative. The arrhythmia was resistant to beta-blockers (metoprolol) and other antiarrhythmic medications. Therefore, the patient was

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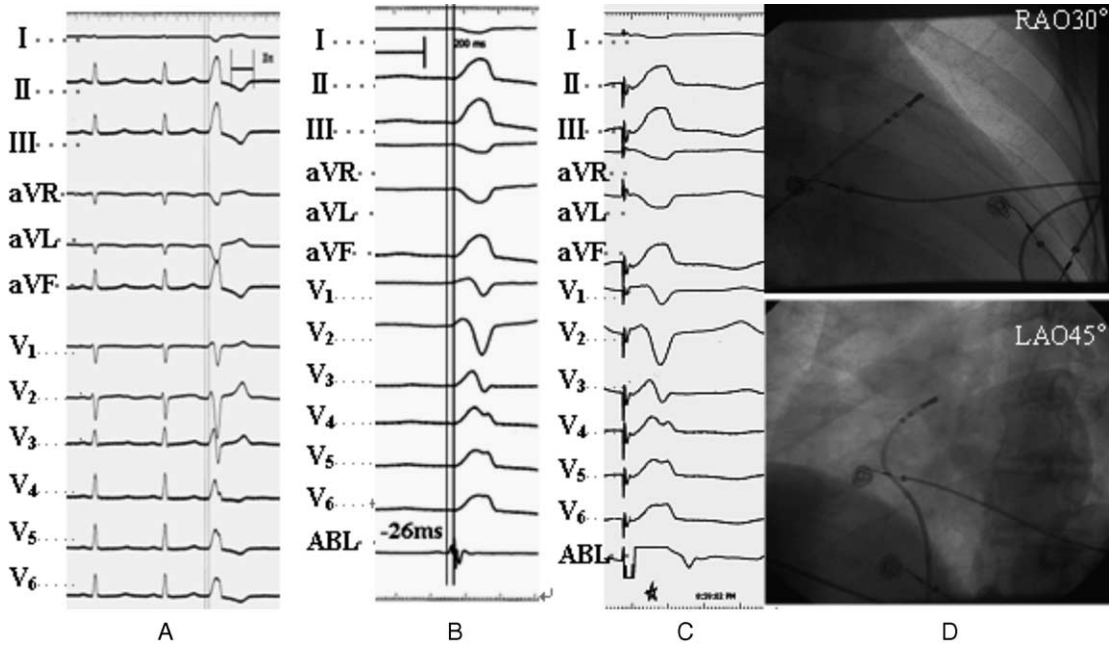

FIGURE 1. Recordings obtained at the RVOT ablation site. A, Electrocardiogram characteristic. B, The local ventricular activation time recorded at the RVOT ablation site that preceded the onset of the QRS complex was 26 milliseconds. C, Excellent pace map at the RVOT ablation site. D, The fluoroscopic position of the RVOT ablation site. RVOT = right ventricular outflow tract.

referred for radiofrequency catheter ablation (RFCA) of the symptomatic, drug-refractory PVCs.

To avoid the adverse effect of antiarrhythmic drugs on the electrophysiologic study and ablation, metoprolol was discontinued at least 5 half-lives before the procedure. After informed written consent was given, the electrophysiologic study and RFCA for the ventricular arrhythmias were performed using conventional fluoroscopically guided mapping, as we reported previously.[7] Under the fluoroscopic guidance, a 7F quadripolar catheter was inserted percutaneously into the

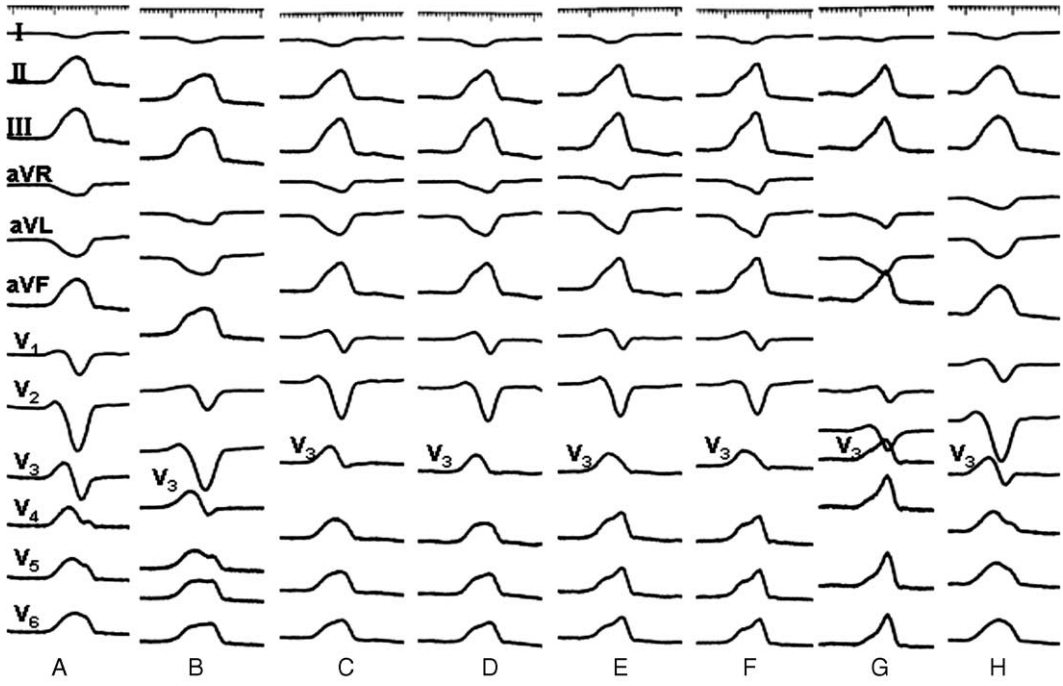

FIGURE 2. Serial ECG recordings obtained at the RVOT ablation site demonstrating progressively altered QRS morphology of PVCs during ablation. A, ECG characteristic of PVCs before ablation. B-G, Dynamic changes in QRS morphology of the PVCs during ablation. H, ECG characteristic of PVCs after ablation similar to the QRS morphology before ablation. ECG = electrocardiogram.

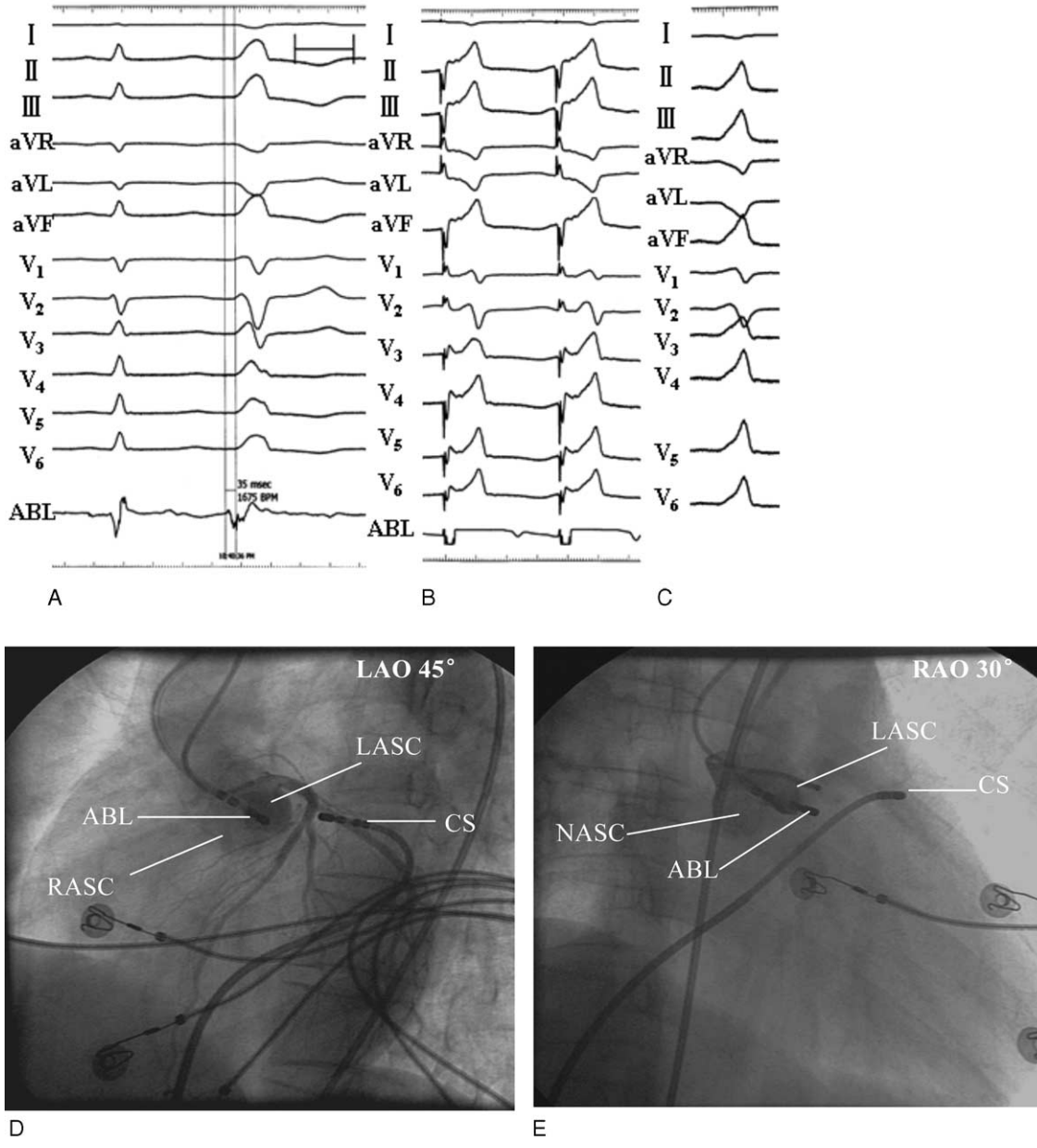

FIGURE 3. Recordings obtained at the left ASC ablation site. A, The local ventricular activation time recorded at the successful ablation site that preceded the onset of the QRS complex was 35 milliseconds. B, Excellent pace map at the successful ablation site. C, The electrocardiogram characteristic of premature ventricular complex occurring during the ablation in the right ventricular outflow tract. D-E, The fluoroscopic position of the successful ablation site.

right femoral vein and positioned at the RVOT. Activation mapping and pace mapping were firstly performed in RVOT. The local ventricular activation time recorded at the antero-septal site of RVOT that preceded the QRS complex onset was 26 milliseconds during the clinical PVCs (Fig. 1B). Pacing from the site exhibited a pace map (Fig. 1C) with a perfect match (10/12) to the clinical PVCs. The r wave in lead V1 during pacing was smaller than that during clinical PVCs (Fig. 1). The interval from the pacing stimulus to the onset of the QRS complex (St-QRS) was 5 milliseconds during pacing from the RVOT. Although a single RF application at the site (Fig. 1D) with a target temperature of 55 °C and maximum power output of 50 W could not terminate the spontaneous PVCs, an interesting phenomenon was that dynamic changes in QRS morphology of the PVCs occurred during ablation (Fig. 2A–H). The R wave with rounded and blunt apex in leads II, III, aVF, V4-V6 before ablation gradually changed to a sharp R wave with an increased amplitude in these leads, R-wave amplitude progressively increased and S-wave amplitude decreased progressively in leads V1 to V3, and the precordial R-wave transition shifted from lead V3 to V2 during ablation. When RF application was stopped after 50 seconds, the PVCs returned to baseline values as the QRS morphology before ablation (Fig. 2H). Because RF ablation failed at the earliest RVOT site, the pace mapping and

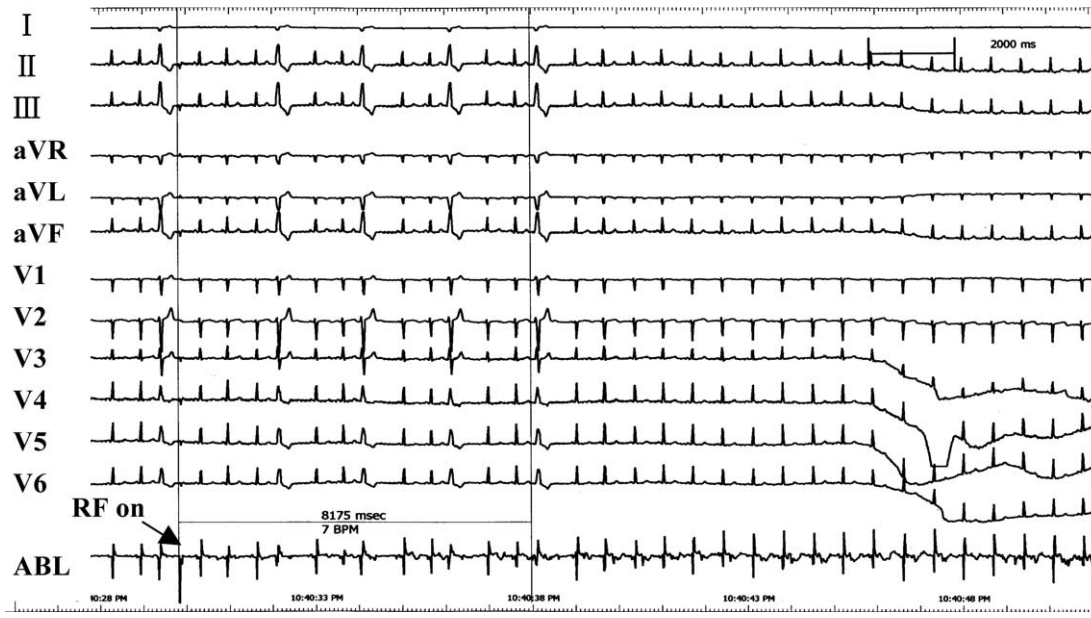

FIGURE 4. Termination of premature ventricular complexes within 8 seconds during radiofrequency application at the left aortic sinus cusp site.

activation mapping did not indicate a focus inside the great cardiac vein, and the altered QRS morphology during ablation strongly suggested a left ASC origin, a new attempt was undertaken to ablate the PVCs from left ASC. Activation mapping was performed in the left ASC using an ablation catheter via the femoral artery during the PVCs. The earliest local ventricular activation preceding the QRS complex onset by 35 milliseconds was recorded at the left ASC during PVCs. The QRS morphology obtained by pacing at the site completely matched (12/12) the PVC occurring during the ablation in the RVOT (Fig. 3A–C). A longer St-QRS interval (42 milliseconds) was observed during pacing from the left ASC than during pacing from the RVOT. An attempt was undertaken to ablate the PVCs at the site. The left main coronary artery was cannulated as a marker and for protection (Fig. 3D–E). A single RF application at the site using a target temperature of 55 °C and maximum power output of 40 W successfully eliminated the PVCs within 8 seconds (Fig. 4). Further ablation around this point was 60 seconds in duration. Thereafter, ventricular stimulation with or without isoproterenol infusion failed to induce any ventricular arrhythmias. The distance between the successful ablation site in the left ASC and the site with an excellent pace map in the RVOT was 20 mm. Holter ECG monitoring revealed no ventricular arrhythmias after the procedure. No complications occurred. The patient has been asymptomatic during 11 months of follow-up.

DISCUSSION

Electrocardiogram characteristics to differentiate PVCs with LBBB and inferior axis arising from either the RVOT or the ASC have been well described.[1,2] Ouyang et al[1] described that the value for an R-wave duration index was 50% and for an R/S wave amplitude index was 30% in lead V1 strongly suggest that the ASC origin in patients is with a typical LBBB morphology and an inferior axis. Recently, Yoshida[2] found ''cardiac rotation-corrected'' transitional zone index might be a more useful marker for differentiating RVOT origin from ASC origin. In our patient the surface ECG, however, was difficult to precisely localize the site of origin of the PVCs. Although there have been a few reports describing ventricular tachycardias (VTs) with ASC origins and endocardial breakout sites in the RVOT,[3–5] serial dynamic changes in QRS morphology of the PVCs obtained in the current study were rare. The progressively altered QRS during ablation with a single origin in the current case may be associated with multiple exit sites from the site of origin because of the close proximity of the left ASC to the septal RVOT. Previous reports have also shown that changes in QRS morphology following RFCA may require additional ablation at a different portion of the outflow tract to cure the outflow tract VT.[3–5,8–10] Yamada et al[5] reported a 50-year-old man with the altered QRS morphology of PVCs originating from the LVOT during mapping and ablation and suggested that preferential conduction to multiple exits may cause ventricular arrhythmias with LVOT origins to exhibit variable ECG. Andrea et al[9] reported a 62-year-old man that the VT morphology abruptly changed and its exit shifted to the left ASC during RFCA in the RVOT. Yamada et al[10] reported a rare case of idiopathic ventricular tachycardia originating from the right ASC with QRS alternans and demonstrated that VT with a single origin and multiple exits in the ASC may exhibit QRS alternans. In our patient, pacing at RVOT reproduced an excellent pace map. When RFCA was applied in RVOT, the QRS morphology of PVCs progressively changed from ECG characteristics of RVOT origin to ECG characteristics of left ASC origin. Successful RFCA was achieved at the site of the earliest ventricular activation in the left ASC. A longer St-QRS interval was observed during pacing at the successful ablation site within the ASC than during pacing from the RVOT. These ndings could be strong evidence for a preferential conduction via the myocardial bers from the ASC origin to the breakout site in the RVOT.[3] The pace map from the successful ablation site within the left ASC was identical to the PVC occurring during the ablation in RVOT, which suggested there should also be

myocardial bers traveling from the left ASC origin to the left ventricle.[3] Elimination of, or interference with, the original exit by radiofrequency energy delivery at the ablation site of RVOT may facilitate opening of a different exit for PVCs, resulting in a fusion of multiple QRS morphologies from multiple exits. Therefore, a careful analysis of the ECG changes during ablation at ventricular outflow septum may be useful for identifying the successful ablation site.

CONCLUSIONS

This case demonstrates that ventricular arrhythmias with a single origin and exit shift may exhibit QRS morphology changes.

CONSENT STATEMENT

Ethical approval was obtained from the Ethics Committee of the Second Affiliated Hospital of Wenzhou Medical University. Written informed consent was obtained from the patients for publication of this case report and any accompanying images.

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