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Original Article

Premature ventricular complexes originating in an inaccessible site in the left ventricular summit successfully terminated by the ablation of adjacent sites with the guidance of CT reconstruction: a case report and literature review

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Abstract: Ventricular arrhythmias originating in the outflow tract are easily mapped and ablated, but those originating in the endocardium and epicardium of the left ventricular summit can pose challenges. We report a 72-year-old woman with premature ventricular complexes whose surface electrocardiogram suggested an anterior septum endocardial origin rather than an epicardial origin; however, electrophysiological mapping localized the origin to the epicardium of the summit of the left ventricle. Radiofrequency ablation cannot be delivered directly to epicardial foci, so radiofrequency ablation was performed on the adjacent structures, especially in the anterior septum of the right ventricular outflow tract, with three-dimensional computed tomography reconstruction of heart used for guidance. The patient has not had a recurrence for 6 months now. Although it is difficult to ablate premature ventricular complexes originating in the left ventricular summit, they can be eliminated successfully by ablating all the adjacent sites and electrically isolating the origin site. Three-dimensional heart computed tomography reconstruction in association with detailed activation and pace mapping achieve successful ablation.

Keywords: Premature ventricular complexes, ventricular tachycardia, left ventricular summit, computed tomography reconstruction, radiofrequency ablation

Introduction

Arrythmias such as premature ventricular contractions (PVCs) and ventricular tachycardia (VT) originating in the outflow tract are usually easily mapped and ablated [1, 2], but those originating in the left ventricular (LV) summit-especially in the "Bermuda triangle" of the heart-can pose challenges. The "Bermuda triangle" refers to the area that lies between the distal coronary sinus (CS; i.e., the origin of the great cardiac vein [GCV]), the posterior right ventricular outflow tract (RVOT), and the left coronary cusp (LCC) [3-5]. The closer the arrhythmogenic focus is to the center of the triangle, the harder it is to identify and ablate [6]. It is important to make a careful preoperative evaluation of LV summit anatomy, where adjacent parts are more prone to indirect damage by three-dimensional computed tomography (CT) reconstruction. PVCs and VT in the LV summit are commonly eliminated by ablating the LCC, endocardial LV summit, or the GCV, but rarely the RVOT [7-9].

Case presentation

A 72-year-old female patient was referred to our hospital after experiencing recurrent palpitations for 20 years. The patient suffered from hypertension and coronary heart disease and underwent percutaneous coronary intervention with stenting. A twenty-four-hour Holter recording revealed frequent monomorphic PVCs with a total of 14,243 beats. No abnormality was found using transthoracic echocardiography. The morphology of the PVCs on the electrocardiogram is shown in **Figure 1**. Tall R-waves in

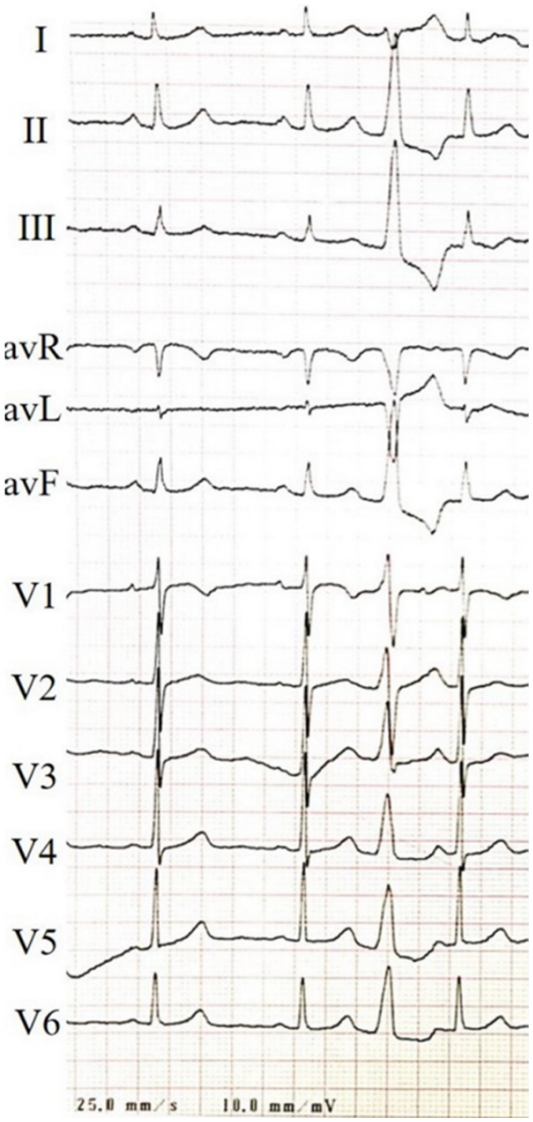

Figure 1. Surface electrocardiogram of the PVC. Tall R-waves in the inferior leads with an LBBB configuration in lead V_1, an R/S transition in lead V_3, and an rS in lead I. Q_{aVL}>Q_{aVR}, MDI = 0.52.

the inferior leads, with left bundle branch block (LBBB) configurations in lead V1 and R/S transition at lead V3, suggested that the focus was either in the left ventricular outflow tract (LVOT) or the RVOT. No obvious abnormality was found in the laboratory examination. A three-dimensional reconstruction of the cardiac anatomical structure by CT (**Figure 3A** and **3B**) shows the anatomical relationship between the RVOT, the LVOT, and the LV summit.

After written informed consent was obtained, the patient was transferred for electrophysiology and catheter ablation. A 6-F quadripolar catheter and an 8-F Smarttouch® contact-force ablation catheter (Biosense Webster Inc., Diamond Bar, CA) were inserted into the right femoral vein and advanced into the distal coronary sinus (CS) and right ventricle, respectively. A three-dimensional electroanatomic system (3D-EAS) (CARTO® 3; Biosense Webster, Diamond Bar, CA, USA) was used for the activation and pace mapping. The site of earliest activation (35 ms before the onset of the QRS complex) was found to be in the anteroseptum of the RVOT (**Figure 2A**). Radiofrequency ablation of this site immediately-but transiently-suppressed the PVCs, suggesting that the PVCs arose from the adjacent sites. The 8-F Smarttouch ablation catheter was inserted into the LOVT using a retrograde method via the right femoral artery. The earliest activation site (with the precedence of 25 ms) of the LV was mapped to the LCC, and the endocardial LV summit was opposite the LCC; however, the radiofrequency ablation from the LCC only achieved a transient suppression of the PVCs (**Figure 2B**, **2C**). The quadripolar catheter was placed in the distal GCV for activation (36 ms before QRS complex onset) and pacing mapping. The paced QRS morphology from the GCV was almost identical to the target PVC, but the paced QRS morphology from the site of the earliest activation in the RVOT and the LCC was very different (**Figure 2A**, **2C**, **2D**). Therefore, it was reasonable to assume that the PVCs arose from the epicardial LV summit close to the distal GCV. The distal end of the GCV was not accessible by the ablation catheter after repeated attempts because of the small size of the middle segment of the CS (**Figure 3B**). However, the CT reconstruction indicated that the site of earliest activation abutted the anteroseptum of the RVOT. Consequently, PVCs were eliminated successfully by enhanced ablation (35 W, 42°C, 300 s with a saline infusion at a rate of 17 ml/min) delivered to the anteroseptum of the RVOT, which was located nearly opposite the GCV early activation site (**Figure 3C**, **3D**). Over 6 months of follow-up, the patient has not had palpitations or Holter evidence of recurrence.

Discussion

A 12-lead electrocardiogram (ECG) acquired during the VT/PVCs provides several clues that help localize the site of origin of the arrhythmia. The R/S transition in the precordial leads can

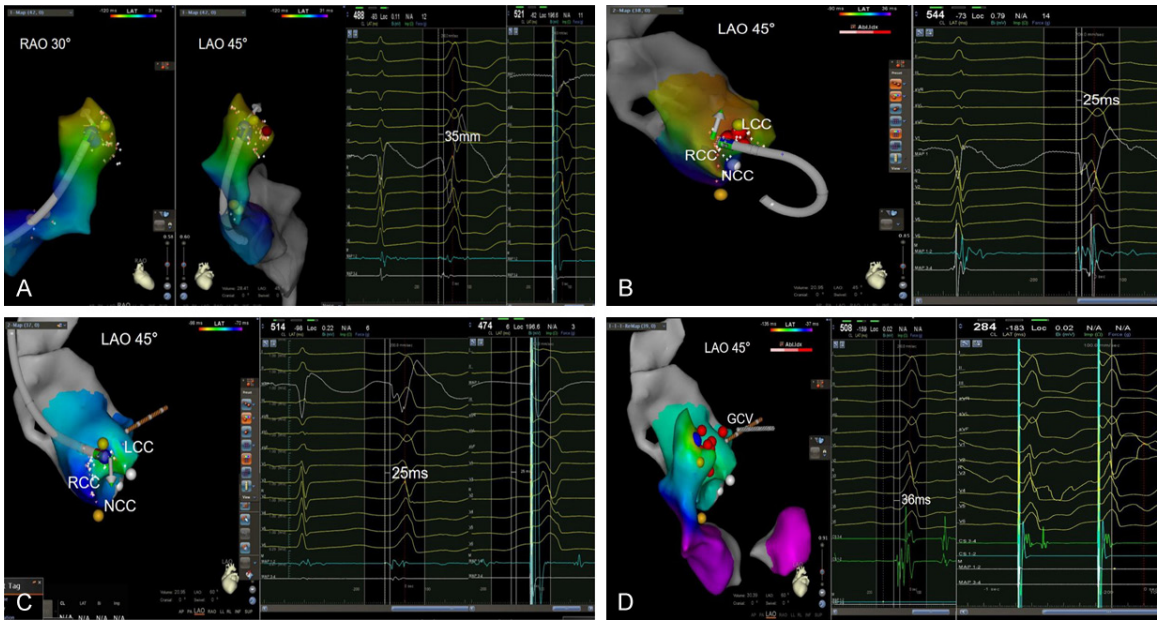

Figure 2. Activation and pacing mapping in the RVOT (A), LV (B), aortic root (C), and GCV (D). The endocardial and epicardial early activation sites are located at the RVOT anteroseptum and distal GCV with the precedences of 35 ms and 36 ms, respectively. The QRS morphology from the GCV pacing was almost identical to the target PVC (D), but the QRS morphology from the RVOT pacing (at the site of the earliest activation) (A) and LCC (C) was very different. RVOT, right ventricular outflow; LV, left ventricular; GCV, great cardiac vein; PVCs, premature ventricular complexes; LCC, left coronary cusp; RCC, right coronary cusp; NCC, non-coronary cusp.

help determine whether the arrythmia originates in the RVOT or LVOT: R/S transition occurring later than V_4 suggests an origin in the RVOT, but the R/S transition occurring earlier than V_2 suggests an origin in the LVOT [10]. The transition at lead V_3 could be explained by the R/S conversion ratio of lead V_2 when the PVC conversion ratio in the LVOT origin is >0.6 [11].

Ouyang et al. used the amplitude and width of the R wave in lead V_1 to identify the site of origin of PVC [12]. They reported that when the height of the R in lead V_1 is >30% of the amplitude of the QRS complex, and the width >50% of the width of the QRS complex, the PVCs arise in the LVOT. In another report [10], a V_2S/V_3R index (S wave amplitude of V_2/R wave amplitude of V_3) of <1.5 had an 82.6% sensitivity, a 91.9% specificity, and an 86% predictive power for identifying arrhythmogenic foci in the LV. The accuracy of the initial r wave surface area of >12 in lead V_1 or V_2 was 92.9% and 78.3% for the LVOT and the RVOT, respectively. Siontis et al. [13] reported that the maximum deflection index (MDI) of ≥0.55 had a sensitivity of 100% and a specificity of 98.7% in the epicardium. Because the most superior part of the LV ostium is the LV summit, tall R-waves with an RBBB or LBBB configuration are typically seen in lead V_1, along with an early transition (earlier than V_3). The site of origin of VT may be the epicardium or endocardium of the LV summit. The QRS morphology in lead V_1 can help in deciding the mapping strategy. A focus in the GCV or anterior interventricular vein (AIV) or an accessible lateral area will cause an RBBB configuration in lead V_1, but an origin closer to the midline will cause an LBBB pattern, with an early transition (earlier than V_3). Another useful finding is the "V_2 pattern break"-an abrupt loss of R in lead V_2 relative to V_1 and V_3-which suggests that the site of origin is in the LV septum close to the left anterior descending (LAD) artery; anatomically, this area lies beneath lead V_2 [14]. ECG findings indicating an origin within the GCV or an accessible area include the following: an aVL/aVR Q-wave ratio >1.1, an S-wave in V_5-V_6, and a transition zone earlier than V_1 [15]. An R-wave ratio >1.25 in lead III/II and an aVL/aVR Q-wave ratio >1.75 indicates a more accessible lateral origin and therefore is an indication for a transpericardial approach [4]. In this case, the PVCs on the electrocardiogram showed the R/S transition at V_3, an R/S conversion ratio of 0.63 at V_2, an R-wave with an amplitude of 41.2% of the QRS and a width of 55% of the QRS complex,

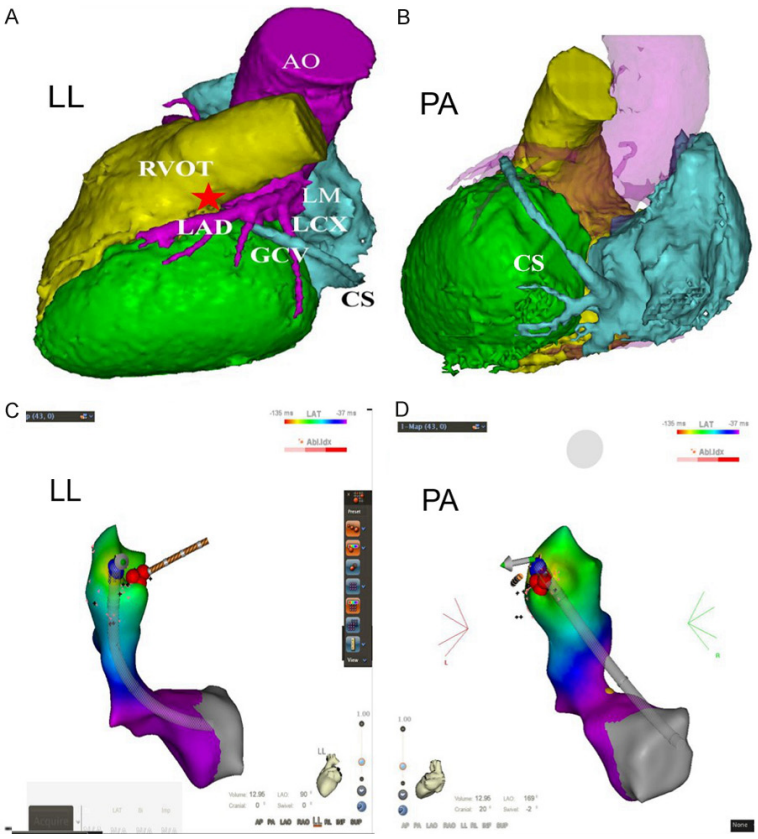

Figure 3. The three-dimensional reconstruction of the cardiac CT and the successful ablation are shown using the CARTO 3 system. A. The anatomical relationship between the RVOT, LVOT, and the LV summit, and the successful ablation site at the anteroseptum of the RVOT (pentagram). B. The small size of the middle segment of the CS. C, D. The successful ablation site at the anteroseptum of the RVOT located nearly opposite the distal GCV in different positions. LL, left lateral; PA, posteroanterior; RVOT, right ventricular outflow tract; LVOT, left ventricular outflow tract; AO, aorta; LM, left main coronary artery; LAD, left anterior descending; LCX, left circumflex; GCV, great cardiac vein; CS, coronary sinus.

and an rS configuration in lead I. The V_2S/V_3R index was 1.2, indicating that the PVC originated in the LVOT LCC. The MDI index was 0.52, and the R/S conversion did not occur earlier than V_3, which suggested an endocardial origin. The aVL/aVR Q-wave ratio of 1.27 and the R/S conversion in V_3, with absent S-waves in V_5 and V_6, did not support the possibility of a PVC origin in GCV/AIV.

Based on the abovementioned positioning criteria, the electrocardiogram morphology suggested that the PVCs may have arisen from the epicardium of the LCC rather than the RVOT. However, the PVCs originating in the distal GCV were confirmed, and they were assigned to the epicardial LV summit through an electrophysiological study and were eliminated successfully at the anteroseptum of the RVOT where the endocardial early activation site preceded the LCC early activation site by 10 ms and was located nearly opposite the GCV early activation site (**Figure 3**). Therefore, the morphology of the PVCs originating from the LV summit could vary. Thus, it should be considered that PVCs can arise from this area when the anterior septum is first localized as the PVC origin by electrocardiography. The cause of the misguided electrocardiographic localization may be the preferential conduction between the PVC origin and the RVOT anteroseptum.

Activation mapping is used to localize the site of origin. It can be used in combination with pacing mapping. The earliest activation site may only be the exit of the PVC, but the real origin point may be in the epicardium with superior conduction to the RVOT or LVOT, so the earliest activation site should be located in the RVOT or LVOT using activation mapping. Pacing mapping can better reflect the real site of origin. In the epicardium, particularly the GVC, pacing mapping is important. In this case, the distal GCV pacing mapping, which was a good match with ECG morphology, indicated that the origin was in the LV summit epicardium. Although activation mapping showed the site of the earliest activation to be in the RVOT, the pacing mapping did not match at the RVOT, indicating that the RVOT was only the exit of the target PVCs.

The ablation of the PVCs that originate in the LV summit involves an ablation of the adjacent areas, including the RVOT, the LCC, and beneath the aortic valve [8, 16-19] epicardium, including ablation with saline perfusion, increasing saline perfusion velocity, and ethanol ablation in the GCV [9, 20, 21] and the left atrial appendage [6]. In this study, RVOT and LCC ablation were effective, and they did eliminate all the

PVCs. The activation mapping of the CS catheter in the distal GCV was not ahead of the QRS complex more obviously than in the RVOT, but the QRS morphology from the GCV pacing was almost identical to the target PVC, but the QRS morphology from the RVOT and LCC pacing was very different. Thus, it is reasonable that the PVCs arose from the epicardial LV summit. The ablation catheter could not reach the distal end of the GCV after repeated attempts due to the small size of the middle segment of the CS, which could be recognized by three-dimensional CT reconstruction. Fortunately, the GCV early activation site was anatomically abutting the anteroseptum of the RVOT through CT reconstruction revealing the adjacent structure in the relationship with the summit. Thus, the PVCs were eliminated successfully using enhanced ablation delivered to the anteroseptum of the RVOT, which was located almost opposite the GCV early activation site. Therefore, it is advisable not to terminate ablation quickly when encountering LV summit PVCs. Preoperative three-dimensional CT reconstruction, which can reveal the adjacent anatomical structure of the LV summit, is important for efficient mapping and ablation. Detailed mapping from the endocardial and epicardial myocardium can help identify the most effective and safest site for the radiofrequency ablation of an arrhythmogenic focus in the LV summit.

Conclusion

The surface electrocardiogram morphology of PVCs originating from the LV summit is varied. The possibility of a summit origin should be considered when localizing PVCs in the anterior septum of the outflow tracts. Although it is difficult to ablate PVCs originating in the LV summit, they can be eliminated successfully by ablating the site of origin or by completely isolating all the exits as identified by a three-dimensional cardiac CT reconstruction in combination with careful activation and pacing mapping.

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Disclosure of conflict of interest

None.

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