COMPLEX CASE STUDY

Ablation of Ventricular Arrhythmias Originating from the Right Coronary Cusp–Left Coronary Cusp Commissure

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Introduction

Ventricular tachycardia (VT) and ventricular premature depolarizations (VPDs) are known to originate from the aortic sinus of Valsalva (ASOV) and are typically described as originating from the individual right coronary cusp (RCC) or left coronary cusp (LCC) sinuses.1–5 Several recent publications have noted ventricular arrhythmias (VAs) originating from the RCC–LCC commissure.6–7 These VAs have distinct electrocardiographic and electrophysiologic features. This case report describes an example of a VPD originating from the RCC–LCC commissure, highlighting the important features and the mapping strategy utilized in ablation of aortic cusp VAs in this location.

Case presentation

A 73-year-old man presented to his primary physician with palpitations and dyspnea on exertion. His electrocardiogram (ECG) revealed frequent VPDs in a pattern of bigeminy. His cardiac work-up included the following: an echocardiogram revealed an ejection fraction (EF) of 30% with a left ventricular (LV) end-diastolic diameter of 5.6 cm; a 24-h Holter revealed 39% ventricular ectopy; and a cardiac catheterization was not significant for obstructive coronary artery disease. The patient was started on verapamil and experienced fatigue. He underwent an electrophysiology (EP) study/ablation at an outside hospital, and the clinical VPD was mapped to the right ventricular outflow tract (RVOT). Ablation at this site suppressed the VPD but did not eliminate it. His follow-up cardiac studies revealed no improvement in his EF and a high VPD burden on verapamil. He was subsequently referred for repeat EP study/ablation.

The clinical VPD had a left bundle morphology with a precordial transition at lead V3 and a characteristic QS morphology with notching on the downward deflection in lead V1 (Figure 1). VPDs that have a precordial transition at lead V3 can originate from either the RVOT or the LV outflow tract (LVOT).7–8 The first step was to perform detailed activation mapping in the RVOT. A three-dimensional (3D) electroanatomical map (CARTO, Biosense Webster, Diamond Bar, CA) was constructed with a 7-Fr, 4-mm tip Navistar catheter (Biosense Webster). The site of earliest activation (30–35 ms pre-VPD) was the midseptal RVOT (Figure 2). Pacemapping at this site was not a good match, with a later precordial transition (V4) than the clinical VPD. Thus, the next step was to map the LVOT, specifically the aortic cusp region.

Prior to mapping the aortic cusp region, an 8-Fr intracardiac echocardiography (ICE) probe (Acuson AcuNav catheter, Siemens, New York, NY) was placed in the left femoral vein and advanced across the tricuspid annulus into the RV with the transducer placed at the base of the RVOT to obtain short-axis echocardiographic views of the aortic cusp region. Each cusp was identified and its relation to the coronary vasculature was noted. ICE imaging was used to accurately identify catheter tip location, to characterize the anatomy of the ASOV region, to assess the distance from the coronary vasculature, to monitor radiofrequency (RF) lesion formation, and to assess for complications.

A detailed activation map of the clinical VPD was created in the aortic cusp region. This electroanatomical
Figure 1: 12-lead electrocardiogram of clinical ventricular premature depolarization (VPD), which originated from the RCC-LCC commissure. Note the QS morphology in lead V1 with notching on the downward deflection and precordial transition at lead V3. RCC: right coronary cusp; LCC: left coronary cusp.

Figure 2: (a) Activation map of clinical ventricular premature depolarization (VPD) in the right ventricular outflow tract (RVOT). (b) The site of earliest activation was the midseptal RVOT (30–35 ms pre-VPD).
map, created in conjunction with ICE imaging to confirm precise anatomy and catheter location, was merged with a preprocedure computed tomography (CT) angiogram of the ASOV and LV. Three cusp points, confirmed by catheter location on ICE, were tagged and used to register the CT scan to the electroanatomical map. The area of earliest activation was marked on the electroanatomical map and confirmed on ICE. The site of earliest activation (80 ms pre-VPD) was the RCC–LCC commissure. At this site, bipolar electrograms recorded in sinus rhythm revealed distinct late potentials (Figure 3). ICE imaging confirmed the site of origin at the RCC–LCC commissure and was used to confirm that the catheter tip was a safe distance from the coronary vasculature prior to RF ablation (RFA) at the RCC–LCC margin (Figure 4). The first RF lesion terminated the clinical VPD and it was no longer seen. A second VPD (VPD 2) was seen, but it was infrequent. VPD 2 was mapped anterior to the aortic valve. The patient subsequently had a sustained VT that was identical to VPD 2. The site of earliest activation of this VT was also anterior to the aortic valve, close to the site of successful ablation of the clinical VPD (Figure 5). During activation mapping, the sustained VT terminated with exit block (Figure 6). A perfect pacemap was obtained at this site for the VT. This pacemap had a long stimulus to QRS interval and was obtained at threshold pacing (Figure 7). RFA at this site terminated VPD 2 and the sustained VT. Burst pacing and isoproterenol failed to elicit any further VAs.

The patient was seen in follow-up in the outpatient EP clinic. A 6-month transthoracic echocardiogram (TTE) revealed an EF of 45–50%, and a 24-h Holter revealed 1.4% VPDs.

![Figure 3:](image1.png)

**Figure 3:** (a) Activation map of clinical ventricular premature depolarization (VPD) in the aortic cusp region. The activation map of the cusp region was merged with a computed tomography angiogram. (b) The site of earliest activation was the RCC–LCC commissure (80 ms pre-VPD). The anatomic location was confirmed by intracardiac echocardiography. Black arrow denotes late potential in sinus rhythm that transitioned to presystolic activation during the clinical VPD. RCC: right coronary cusp; LCC: left coronary cusp; NCC: non-coronary cusp. (Figure 3a reprinted from reference 7, with permission from Elsevier.)

![Figure 4:](image2.png)

**Figure 4:** A representative example of an intracardiac echo (ICE) image of the aortic cusp region from a separate case. The ablation catheter tip (white arrow) is located at the RCC–LCC commissure. R: right coronary cusp; L: left coronary cusp; LA: left atrium; PA: pulmonary artery. (Reprinted from reference 7, with permission from Elsevier.)
Figure 5: (a) Activation mapping localized both the sustained ventricular tachycardia (VT) and ventricular premature depolarization (VPD) 2 anterior to the aortic valve, close to the site of successful ablation of the clinical VPD. (b) Early activation noted on the ablation catheter (M1–M2) during sustained VT.

Figure 6: At the site of earliest activation, ventricular tachycardia (VT) terminates with exit block (there is no subsequent QRS on the surface electrocardiogram following the local bipolar electrogram on Carto D).
Discussion

This case highlights the important electrocardiographic and electrophysiologic features of VAs originating from the RCC–LCC commissure. In this case, the site of origin of the clinical VPD was the RCC–LCC commissure. The ECG morphology of the clinical VPD was consistent with the predominant ECG pattern of VAs originating from the RCC–LCC commissure: a QS morphology in lead V1 with notching on the downward deflection and precordial transition at lead V3. Mapping at the RCC–LCC commissure revealed distinct late potentials in sinus rhythm.

Initial activation mapping in the RVOT revealed electrograms that were 30–35 ms pre-VPD in the mid-septal region. RFA was not performed at this site. Activation mapping in the RCC–LCC commissure, opposite to the midseptal RVOT, revealed presystolic activity that was 80 ms pre-VPD. The distance between the site of earliest activation in the RVOT and RCC–LCC commissure was small, emphasizing the importance of detailed mapping prior to performing ablation. The second VPD that was seen after ablation of the clinical VPD had a “w” morphology in lead V1 and a precordial transition at lead V2. This VPD and an identical sustained VT were mapped anterior to the aortic valve. VPDs with a “w” morphology in lead V1 can originate from the RCC–LCC commissure or the LCC.

Yamada et al6 published a paper on five patients with VAs originating from the junction of the left and right coronary sinus of Valsalva. The authors described a QRS pattern in leads V1–V3 from VAs originating from the RCC–LCC junction. They used aortic angiography, electroanatomic mapping, and limited echocardiography in their study. Our group recently published a study identifying the electrocardiographic and electrophysiologic characteristics of VAs originating from the RCC–LCC commissure.7 We reported on 19 patients with VAs originating from the RCC–LCC commissure. In 15 of 19 VAs, the ECG revealed a QS morphology in lead V1 with notching on the downward deflection, and in 4 of 19 VAs, the ECG revealed a “w” pattern in lead V1. At the site of earliest activation, 13 of 19 patients in our series manifested late potentials during sinus rhythm with reversal of activation during the clinical VPD/VT.

The origin of the RCC–LCC VAs is not fully understood. The apical junction of the RCC–LCC commissure is contiguous with the interleaflet triangle and is traditionally composed of fibrous tissue. The basal junction of the RCC–LCC margin is coincident with the distal parts of the left ventricle.9–11 A recent review by Yamada et al12 emphasizes the LV ostium as the origin of these VAs. Hasdemir et al13 published a study demonstrating ventricular myocardial extensions into the pulmonary artery and aorta beyond the ventriculoarterial junction. The late potentials in sinus rhythm may represent a tract of slow conducting fibers that are activated after the ventricles and may correlate to the depolarization of ventricular myocardial extensions seen in previous studies.

It is possible that the LV ostium served as the origin of the clinical VPD that was ablated at the RCC–LCC

![Figure 7](image-url)

Figure 7: (a) 12-Lead electrocardiogram of ventricular tachycardia (VT). Note the “w” morphology in lead V1 and precordial transition at lead V2. (b) A perfect pacemap for the sustained VT was obtained at the site of earliest activation. This pacemap had a long stimulus to QRS interval.
commissure. Given that the site of origin of VPD 2 and the sustained VT were anterior to the aortic valve, it is possible that ablation of the clinical VPD altered the exit of the focus, resulting in VPD 2 and the sustained VT.

In this case, a combination of electroanatomical mapping and ICE imaging was used to confirm anatomic location, catheter tip position and contact, distance to coronary vasculature, and to monitor RF delivery. ICE was used to assess the distance to coronary vasculature in place of coronary arteriography. Operators less experienced with ICE should utilize coronary arteriography prior to RFA in the aortic cusp region to ensure a safe distance from the coronary vasculature.

References