RHYTHM DISORDERS AND ELECTROPHYSIOLOGY

CASE REPORT: CLINICAL CASE

Bipolar Ablation From the Anterior Interventricular Vein to the Left Ventricle



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ABSTRACT

Ventricular tachycardia from the left ventricular summit can be challenging for catheter ablation due to difficult accessibility and proximity to coronary arteries. This paper presents a case of premature ventricular contraction-induced ventricular tachycardia from the left ventricular summit that was ablated using bipolar radiofrequency ablation from the anterior interventricular vein and adjacent left ventricular endocardium. (JACC Case Rep 2024;29:102457) Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 37-year-old man with dilated cardiomyopathy and a secondary prevention implantable cardioverterdefibrillator (ICD) presented to the electrophysiology (EP) clinic for evaluation for EP study and catheter ablation after having recurrent premature ventricular contractions (PVCs) and ventricular tachycardia (VT). The patient reported having felt palpitations and the sensation of his ICD pacing him in the days prior to presentation. Vital signs showed a heart rate of 94

LEARNING OBJECTIVES

- To understand the clinical challenges associated with catheter ablation of ventricular tachycardia originating from the LVS.
- To describe the anatomic considerations when performing ablations near the LVS, including the relationship with coronary arteries.
- To explain the indications and methodology for using bipolar radiofrequency ablation in the treatment of ventricular tachycardia.

beats/min and blood pressure of 93/54 mm Hg. Physical examination was notable for no significant peripheral edema and normal heart and lung sounds with occasional ectopy.

PAST MEDICAL HISTORY

The patient was diagnosed with nonischemic cardiomyopathy and was under the care of a heart failure and cardiac transplant team. Despite being on guideline-directed medical therapy, the patient still had severely reduced ejection fraction (<30%). He had a prior episode of ventricular arrhythmia resulting in the placement of a secondary prevention single-chamber ICD 9 years prior. He had also undergone a prior attempt at VT ablation from the anteroseptal right ventricular outflow tract (RVOT) and adjacent left ventricular outflow tract that was not successful in suppressing his ventricular arrhythmias.

DIFFERENTIAL DIAGNOSIS

At this time, the diagnoses under consideration were as follows: 1) increased frequency of PVCs or

Manuscript received November 13, 2023; revised manuscript received April 22, 2024, accepted May 2, 2024.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

ABBREVIATIONS AND ACRONYMS

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AIV = anterior interventricular vein

EP = electrophysiology

ICD = implantable cardioverter-defibrillator

LVS = left ventricular summit

PVC = premature ventricular contraction

RVOT = right ventricular outflow tract

VT = ventricular tachycardia

sustained VT resulting in initiation of tachytherapies from his device; and 2) supraventricular tachycardia with aberrant conduction resulting in inappropriate device therapies.

INVESTIGATIONS

A transthoracic echocardiogram revealed a severely reduced left ventricular ejection fraction of 27%, which persisted despite adherence to guideline-directed medical therapy, including a beta-blocker, angiotensin-converting enzyme inhibitor, and aldosterone receptor antagonist. Review of his defibrillator intracardiac electrograms suggested there was a consistent PVC trigger for his VT

and his PVC burden was measured at 20% on an external cardiac monitor.

MANAGEMENT

The patient presented to the EP laboratory in sinus rhythm with frequent PVCs of 2 dominant morphologies: PVC 1 was a left bundle, rightward, inferiorly directed PVC with V4 transition, and PVC 2 was a right bundle, rightward, inferiorly directed (LBRI) PVC. Ventricular stimulation with 400 drive train and double extrastimuli induced VT with the same LBRI V₄ transition as PVC 1. Electroanatomic mapping of the left ventricle and right ventricle with the Carto mapping system (Biosense Webster, Inc) demonstrated normal bipolar and unipolar voltage. Activation mapping of PVC 1 was performed indicating that the earliest activation was at the septal aspect of the RVOT. Pace mapping in the RVOT demonstrated >90% match at the anteroseptal RVOT to PVC 1. Unipolar radiofrequency ablation in this area, with up to 40 W for 60-second lesions, did not result in suppression of the PVC. Next, a DecaNav catheter (Biosense Webster, Inc) was used to map PVC 1 from the basal to mid-anterior interventricular vein (AIV) and was found to be 25 milliseconds pre-QRS complex with a 98% pace match (Figure 1A).

To evaluate proximity to nearby coronary vessels, coronary angiography was performed and demonstrated that the left anterior descending artery coursed 1 to 2 mm medial to the AIV (Figure 1B), and the site of earliest activation in the AIV was at least 5 mm away from the left coronary artery. Using a SMARTTOUCH SF open irrigation ablation catheter (Biosense Webster, Inc), a linear set of ablation lesions was performed from the proximal AIV back through the earliest activation area with 10 to 15 W of power for up to 60 seconds' duration. These ablations resulted in transient suppression of PVC 1 and complete suppression of PVC 2.

The endocardial surface of the left ventricle immediately opposite the earliest AIV site produced a pacemap match of 90%. Unipolar ablation lesions with the ablation catheter in the adjacent left ventricular endocardium was done with 40 W for 60second lesions, but this only temporarily suppressed the VT.

We decided to perform bipolar radiofrequency ablation with 2 SMARTTOUCH SF open irrigation catheters from the AIV and left ventricular endocardium because the sites of earliest activation between the AIV and the left ventricular endocardium were approximately 15 mm apart. Bipolar radiofrequency ablation with 15 W for 120 to 300 seconds was then performed with one ablation catheter in the AIV and the other in the left ventricle at the respective sites of earliest activation (Figures 1C and 1D). Ablation was delivered first with the AIV catheter as the active catheter. The polarity of the ablation was then changed to make the ablation catheter in the AIV the inactive or grounding catheter and the left ventricular catheter the active catheter. This resulted in a more successful ablation of the ventricular arrhythmia. The average ablation impedance within the AIV was 228 ohms with an average decrease of 22 ohms when ablating unipolar with the irrigated ablation catheter. Bipolar ablation yielded an average initial impedance of 224 ohms with a 26 ohm decline during ablation when the irrigated catheter in the AIV was the active catheter. When bipolar ablation was switched to make the irrigated catheter in the AIV the inactive catheter, the initial ablation impedance was 139 ohms and decreased 10 ohms with ablation. After bipolar ablation with both ablation polarities, VT was no longer inducible, and the procedure was concluded.

DISCUSSION

The left ventricular summit (LVS) is the most superior region of the left ventricle bounded on the epicardial surface by the bifurcation of the left main coronary artery and inferiorly by the first septal perforator branch.¹ PVCs and ventricular arrhythmias arising from an intramural region near the LVS have been targeted by both endocardial and epicardial approaches.^{2,3} One of the limitations of standard unipolar radiofrequency ablation in successfully targeting intramural ventricular arrhythmias is limited lesion depth. In the LVS region, attempting



contractions; RV = right ventricle.

standard unipolar radiofrequency ablation of an intramyocardial substrate from an endocardial or epicardial approach can be challenging due to the tissue thickness and presence of epicardial fat, respectively.⁴ Strategies that take advantage of nearby coronary venous vessels (eg, ablation from the

great cardiac vein or AIV) can be met with impedance limitations or risk of injury to proximal coronary arteries.⁵ Use of bipolar radiofrequency ablation has been reported as an alternative method to deliver intramyocardial lesions to previously inaccessible intramural foci.^{6,7} 4

Because PVCs and VT arising from the LVS region can be challenging to ablate as previously mentioned, successful ablation may require use of different modalities and strategies. Some of these strategies include use of bipolar radiofrequency ablation, coronary vein injection of ethanol, and ablation from adjacent structures.⁶ Limitations to success have been shown to be related to insufficient ablation lesion depth before risking overheating or steampops.⁸ Bipolar ablation allows for deeper lesion creation without the same risk to surrounding structures in unipolar ablation due to its ability to focus the radiofrequency energy to a smaller radius of tissue.⁷ The bipolar ablation has been shown to be more effective when there is an interelectrode distance between 10 and 20 mm.⁸ In a study by Shapira-Daniels et al,⁹ myocardial lesion depth with unipolar ablation ranged from 4.8 to 6.1 mm compared with the 10.5 \pm 1.4 mm depth seen with bipolar ablation. With the average left ventricular myocardial thickness in men around 6.3 mm and epicardial fat thickness adding approximately another 5.3 mm, unipolar ablation may not be able to reach deep midmyocardial foci regardless of endocardial or epicardial approach.⁹ Nguyen et al⁸ also demonstrated that bipolar ablation can be effective in creating transmural lesions when the 2 catheters are up to 15 mm apart, as was seen in this patient.

If limitations due to elevated impedance in coronary sinus vessels are encountered, the ablation technique could have been modified to place an 8-mm nonirrigated catheter in the AIV, which would have decreased the ablation impedance.¹⁰ Work looking at the biophysics of bipolar ablation techniques has also shown that techniques using externally irrigated catheters in a perpendicular orientation to the myocardium or half normal saline irrigation can allow for larger and deeper lesions without increased adverse events.¹¹

In this case, after ablation from the adjacent endocardial structures was unsuccessful, we turned to bipolar ablation because of the need for a deeper lesion and limitations in unipolar ablation as mentioned previously.

FOLLOW-UP

Bipolar radiofrequency ablation in the region near the LVS resulted in clinical improvement and reduction in ventricular arrhythmias for this patient. The patient was seen periodically in heart failure and EP outpatient clinics, and device interrogations showed no recurrence of sustained ventricular arrhythmias at 1-year postablation. The patient's left ventricular systolic function improved but did not normalize after ablation of his summit PVCs. The etiology of the patient's PVCs and associated cardiomyopathy remains unknown. A cardiac magnetic resonance was subsequently performed that showed no evidence of infiltrative cardiomyopathy. However, the symptomatic and clinical freedom from ventricular arrhythmias without procedural complications supports the position that bipolar radiofrequency ablation can be both a safe and durable management strategy when standard ablation techniques are ineffective.

CONCLUSIONS

PVCs and VT originating from the LVS and adjacent areas may pose challenges for ablation, often necessitating a combination of strategies for effective treatment. This case described a situation where bipolar radiofrequency ablation was successfully used to ablate VT of intramural origin in the LVS from the AIV and left ventricular endocardium. In the appropriate patient, use of bipolar radiofrequency ablation represents a valuable addition to the armamentarium of techniques to conquer the summit.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS bipolar ablation, left ventricular summit, premature ventricular contraction, radiofrequency ablation, ventricular tachycardia