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CASE REPORT

CLINICAL CASE

Ventricular Tachycardia Ablation Through a Recanalized Surgically Plicated Left Ventricular Apical Aneurysm

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ABSTRACT

Left ventricular apical aneurysms are associated with scar-related ventricular tachycardia (VT) in hypertrophic cardiomyopathy patients. We present a patient with apical hypertrophic cardiomyopathy who underwent combined epicardial and endocardial VT ablation of a left ventricular apical aneurysm, necessitating repeat endocardial VT ablation through a recanalized surgical plication. (J Am Coll Cardiol Case Rep 2024;29:102299) © 2024 The Authors. 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 64-year-old man with prior cardiac history of hypertrophic cardiomyopathy (HCM) with apical aneurysm, cardiac arrest with implantable cardioverter-defibrillator (ICD) insertion and prior ventricular tachycardia (VT) ablations, and prior surgical plication of the left ventricular (LV) aneurysm was referred for repeat VT ablation after experiencing 3 shocks.

PAST MEDICAL HISTORY

The patient presented initially with cardiac arrest in 2013, at which point HCM with an apical aneurysm was diagnosed on transthoracic echocardiogram. At this time, coronary angiography demonstrated ectatic but otherwise normal coronary arteries. During that admission he underwent dual-chamber ICD implantation for secondary prevention. Subsequently in 2018, he had 2 separate episodes of VT (200 and 190 beats/min), the first requiring ICD shock and the second terminating spontaneously. He was started on Sotalol 80 mg twice daily, which was up-titrated to 120 mg twice daily for recurrent episode of atrial fibrillation. Despite this, 3 episodes of VT were recorded, all of which were terminated by antitachycardia pacing from the ICD. At this point, the patient was referred for his first catheter ablation.

LEARNING OBJECTIVES

- To be able to identify the arrhythmic risk associated with LV apical aneurysms in patients with HCM.
- To understand how catheter ablation of VT reduces arrhythmia recurrence in HCM patients.

A combined endocardial and epicardial ablation was performed. The bipolar voltage maps demonstrated a large apical aneurysm consisting of scar with mid-myocardial obstruction in the LV (**Figure 1**). Two VTs were induced with morphologies suggestive of an LV apical exit. The apical scar had abundant late and fractionated potentials, which were targeted with radiofrequency ablation from the endocardium and

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ABBREVIATIONS AND ACRONYMS

HCM = hypertrophic cardiomyopathy

ICD = implantable cardiac defibrillator

LV = left ventricular

VT = ventricular tachycardia

epicardium. At the conclusion of the procedure, VT was noninducible.

Two weeks after VT ablation, the patient was admitted with atrial fibrillation with a rapid ventricular response and underwent cardioversion. Six months later, because of progressive valvular disease, he underwent mitral and tricuspid valve repair, biatrial cryomaze, left atrial appendage clipping, and linear plication of the apical LV aneurysm using a nonabsorbable polypropylene monofilament suture reinforced with felt. The patient was discharged on amiodarone 200 mg daily, later titrated down to 100 mg daily with sotalol discontinued. Six months after surgery, he had multiple episodes of VT that were treated successfully with antitachycardia pacing. After a multidisciplinary discussion, the team decided that a second catheter ablation should be performed. Because the patient had prior cardiac surgery, percutaneous epicardial ablation would not be possible because of pericardial adhesions; therefore, an endocardial VT ablation was attempted. In the electrophysiology laboratory, VT could not be induced, and the LV endocardium was mapped using a force-sensing, 8-F unidirectional ablation catheter advanced transeptal through a deflectable sheath. The LV apex was carefully probed for any residual aneurysm with the assistance of intracardiac echocardiography guidance; however, none was identified (**Figure 2**, Video 1). The patient did well for 3 years when he had VT recurrence and experienced 3 ICD shocks.

DIFFERENTIAL DIAGNOSIS

Recurrent VT originating from the intramyocardial or epicardial LV vs from the surgically plicated LV apical aneurysm.

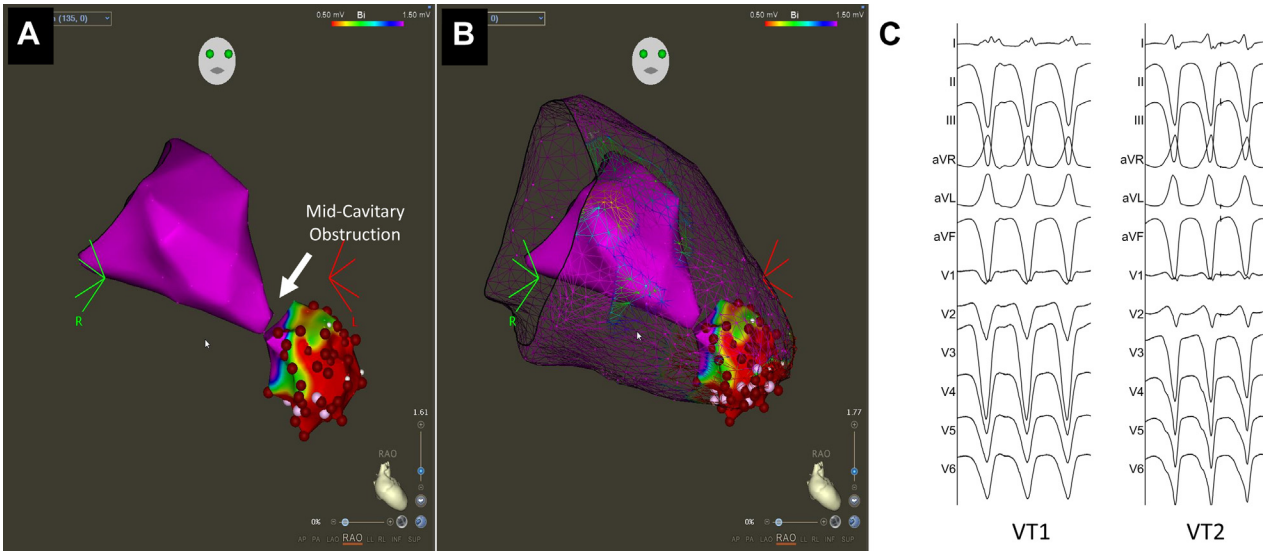
INVESTIGATIONS

Because the LV aneurysm was surgically plicated and endocardial ablation of aneurysm would not be possible, an epicardial ablation with a surgical subxyphoid window was considered. However, a contrast-enhanced transthoracic echocardiogram suggested that the surgically plicated LV apical aneurysm was accessible (**Figure 3**).

MANAGEMENT

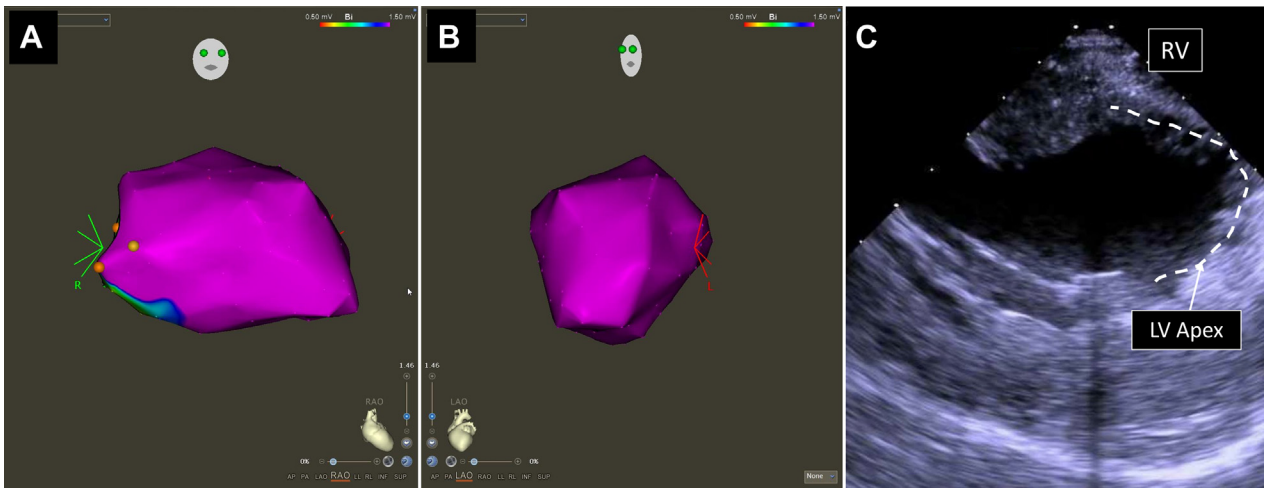
A repeat endocardial VT ablation was performed. This time, using identical techniques as the previous ablation, LV apical aneurysm was accessed with the ablation catheter and subsequently a multielectrode

FIGURE 1 Voltage Maps of Left Ventricle and Epicardium



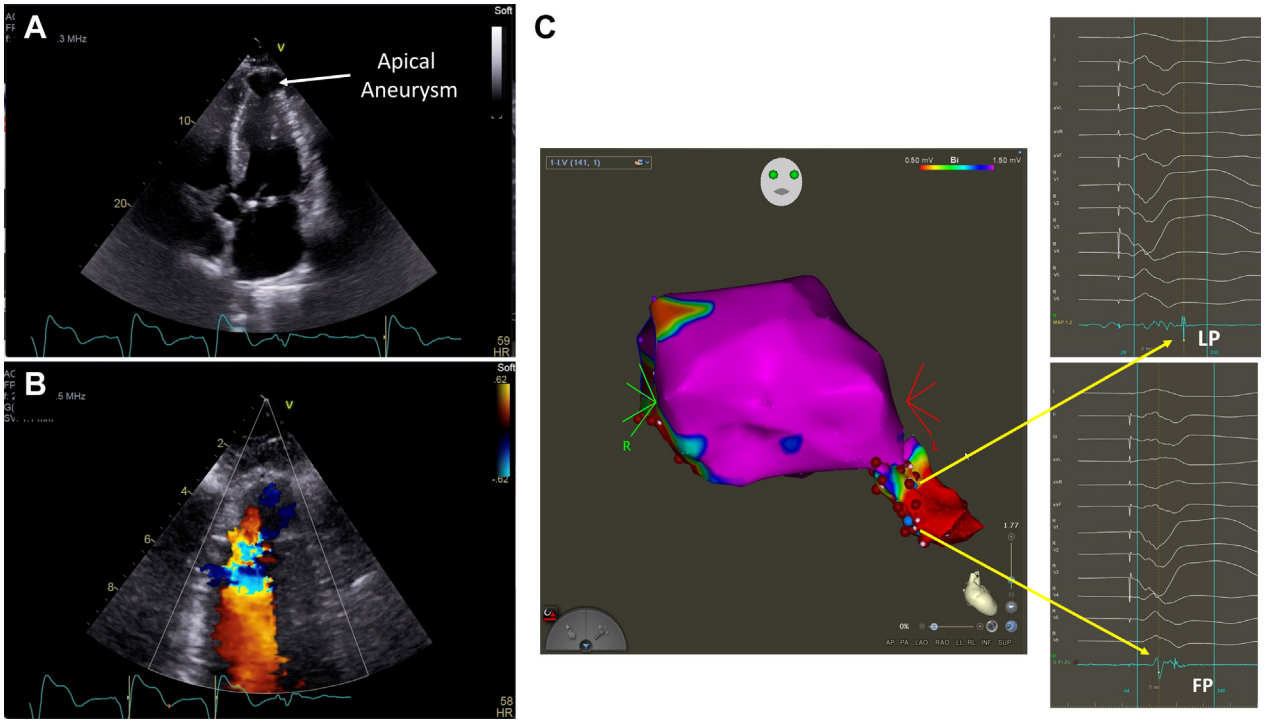
(A) The left ventricular endocardium was mapped and is shown in the right anterior oblique (RAO) projection. The purple represents normal myocardium (bipolar voltage >1.50 mV) and the red represents scar (<0.5 mV). There is large apical aneurysm that is largely scar. Areas of fractionated electrograms (white dots), late potentials (pink dots), and sites of ablation (red dots) are shown. The patient had a midcavitary obstruction. (B) The epicardial voltage map (mesh) is shown overlayed on the left ventricular endocardial voltage map (RAO projection). The epicardial scar was smaller with a few fractionated potentials. Ablation was also done on the epicardial surface. (C) Two morphologies of ventricular tachycardia were induced, both suggestive of a left ventricular apical exit.

FIGURE 2 **LV Endocardial Voltage Map Following Surgical Plication of LV Apical Aneurysm**



(A) The left ventricular (LV) endocardium in right anterior oblique (RAO) projection has normal voltage (purple area) and the previously seen aneurysm is no longer seen. (B) The LV endocardium in left anterior oblique (LAO) projection. (C) Intraprocedural intracardiac echocardiography imaging performed from within the right ventricular (RV) apex, facing posteriorly at the left ventricle, demonstrating no evidence of apical aneurysm.

FIGURE 3 **Transthoracic Echocardiogram and Voltage Maps Showing Recanalization of the Plicated of Apical Aneurysm**



(A) Transthoracic echocardiogram shows the apical aneurysm. (B) Color Doppler shows flow to the apex. (C) A repeat ventricular tachycardia ablation procedure was performed, and the left ventricular endocardial voltage map (right anterior oblique projection) is shown. The apical aneurysm is now accessible with an ablation catheter. Areas of fractionated electrograms (FP) (pink dots) and late potentials (LP) (blue dots) along with their characteristic electrograms are shown (arrows). These abnormal electrograms were targeted with radiofrequency ablation (red dots).

catheter. Although no VT was induced, arrhythmogenic substrate involving late and fractioned electrograms was readily identified and homogenized. The patient was discharged on Amiodarone 200 mg daily.

DISCUSSION

LV apical aneurysms in HCM patients are a major risk factor for ventricular arrhythmias and sudden cardiac death.[1] Catheter ablation of VT in HCM patients have been shown to be an effective approach to decrease VT recurrence.[2,3] In these patients, apical aneurysms are a common finding, occurring in up to 32% of patients, and a combined endocardial and epicardial approach is often required (~41% of the time) to treat them successfully. In our patient, the apical aneurysm and scar was a result of midcavitary obstruction and was responsible for the VT. Catheter ablation using radiofrequency energy was performed, targeting late and fractionated electrograms (ie, substrate modification) to render the VT noninducible after the initial procedure.

Given that the patient had worsening MR, he underwent cardiac surgery with MV and TV repair, biatrial cryomaze, left atrial appendage clipping, and linear plication of the LV apical aneurysm. There are numerous techniques for surgical LV reconstruction, including linear repair as was done in this patient. However, these techniques do not reduce ventricular arrhythmias unless they are supplemented with surgical techniques aimed at destroying or isolating the arrhythmic tissue—such as subtotal endocardial scar resection or endocardial cryoablation.[4] This was successfully demonstrated in a series of 44 patients with apical aneurysms resulting from HCM undergoing surgical repair, 17 (39%) had a history of ventricular arrhythmias in the 2 years prior to their surgery.[5] These patients underwent surgical *resection* of the aneurysm with a 9% incidence of surgical complications. As a result of removal of the arrhythmogenic substrate, only 2 patients had VT periprocedurally, and 2 more patients experienced ventricular arrhythmias 4 and 9 years postoperatively. Although excision of the aneurysm combined with the complex surgical procedure being conducted in our patient may not have been possible, it is worth noting that suture-plication of the aneurysm threatened the potential for future percutaneous access and endocardial VT-ablation.

FOLLOW-UP

The patient remained without VT for 4 months after the second procedure, at which point he had a spontaneous intracerebral bleed on apixaban and ultimately died.

CONCLUSIONS

This case demonstrates the following: 1) the arrhythmic risk associated with apical aneurysms in HCM patients; 2) that catheter ablation is possible in this patient population to decrease VT recurrence; and 3) that the arrhythmic risk is not reduced following linear plication of the apical aneurysms unless other surgical techniques are also used to destroy or modify the arrhythmic tissue.

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Dr Dukkipati has served as a consultant for Biosense Webster; has received payment for equity in Farapulse from Boston Scientific; and has equity in Manual Surgical Sciences. Drs Freilich and Musikantow have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS epicardial ablation, endocardial ablation, hypertrophic cardiomyopathy, ventricular tachycardia

APPENDIX For a supplemental video, please see the online version of this paper.