When to sear, when to burn, and when to chop: The art of substrate modification

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Introduction

Recurrent ventricular tachycardia (VT) in the setting of left ventricular (LV) aneurysm and scar can be challenging to control, particularly in cases with midmyocardial or epicardial circuits. Strategies for substrate modification in VT with LV aneurysm can include endocardial and epicardial ablation as well as surgical resection in cases refractory to catheter ablation. Here, we present a case of a left ventricular apical aneurysm and VT that was refractory to medical therapy and recurred after endocardial and epicardial ablations. Our patient underwent surgical aneurysmectomy and endocardial resection with resolution of her VT.

Case report

A 54-year-old female patient who was a former marathon runner presented with a 2-year history of recurrent exercise-induced monomorphic VT. She initially presented to an outside hospital 2 years prior for symptomatic VT. Computed tomography angiogram (CTA) at that time demonstrated anomalous coronary arteries with the left anterior descending (LAD) and circumflex arteries arising from the right coronary artery. The LAD had a truncated course and terminated without supplying the apex. CTA also showed evidence of prior inferior-apical infarction with an apical aneurysm and mural thrombus in the LV apex. Coronary angiogram performed at that time demonstrated anomalous coronary arteries with no atherosclerotic coronary artery disease. Timing and etiology of her apical infarction were unclear. Troponins were normal at the time of presentation. Given that there was no obstructive coronary artery disease and no targets for revascularization, no coronary interventions were performed. Her LV apical thrombus was treated with warfarin and treatment later transitioned to apixaban. She received a dual-chamber implantable cardioverter-defibrillator (ICD) for secondary prevention and was placed on amiodarone and metoprolol.

Over the next 2 years, the patient had over 10 appropriate ICD shocks for monomorphic VT. Her ICD was programmed with 3 VT detection zones with the following therapies: (1) slow VT zone for heart rates of 130–180 beats per minute (bpm) (Antitachycardia pacing [ATP] ×6: Burst ×3, Ramp ×3); (2) Fast VT zone for rates of 180–230 bpm (ATP ×6: Burst ×3, Ramp ×3, followed by 41.0 J DC shock ×6); (3) VF zone for rates greater than 230 bpm (41.0 J DC shock ×8, ATP during charging). Electrocardiography showed monomorphic VT at a cycle length of 530 ms and right bundle branch block configuration with transition in V_3 (Figure 1A). There was left axis deviation and a superior axis, compatible with an apical inferoseptal LV exit.

Work-up involved a transthoracic echocardiogram, which showed an LV ejection fraction of 40%–45% with akinesis and thinning of the mid inferolateral wall and apex. A repeat cardiac CTA showed a right dominant

KEY TEACHING POINTS

- Strategies to prevent phrenic nerve injury during epicardial ablation include infusion of saline into the pericardial space, intrapericardial balloon inflation, and use of a catheter to manually deflect the pericardium and phrenic nerve from the epicardial surface.
- Strategies for substrate modification in ventricular arrhythmias can include endocardial and epicardial ablation as well as surgical intervention.
- Recurrence of ventricular tachycardia (VT) can occur after percutaneous VT ablation and is frequently associated with midmyocardial or epicardial circuits.
- Surgical aneurysmectomy with endocardial resection can be effective for reducing VT in cases of recurrent VT refractory to medical therapy and catheter ablation.

KEYWORDS Ventricular tachycardia; Phrenic nerve deflection; Epicardial ablation; Apical aneurysm; Aneurysmectomy
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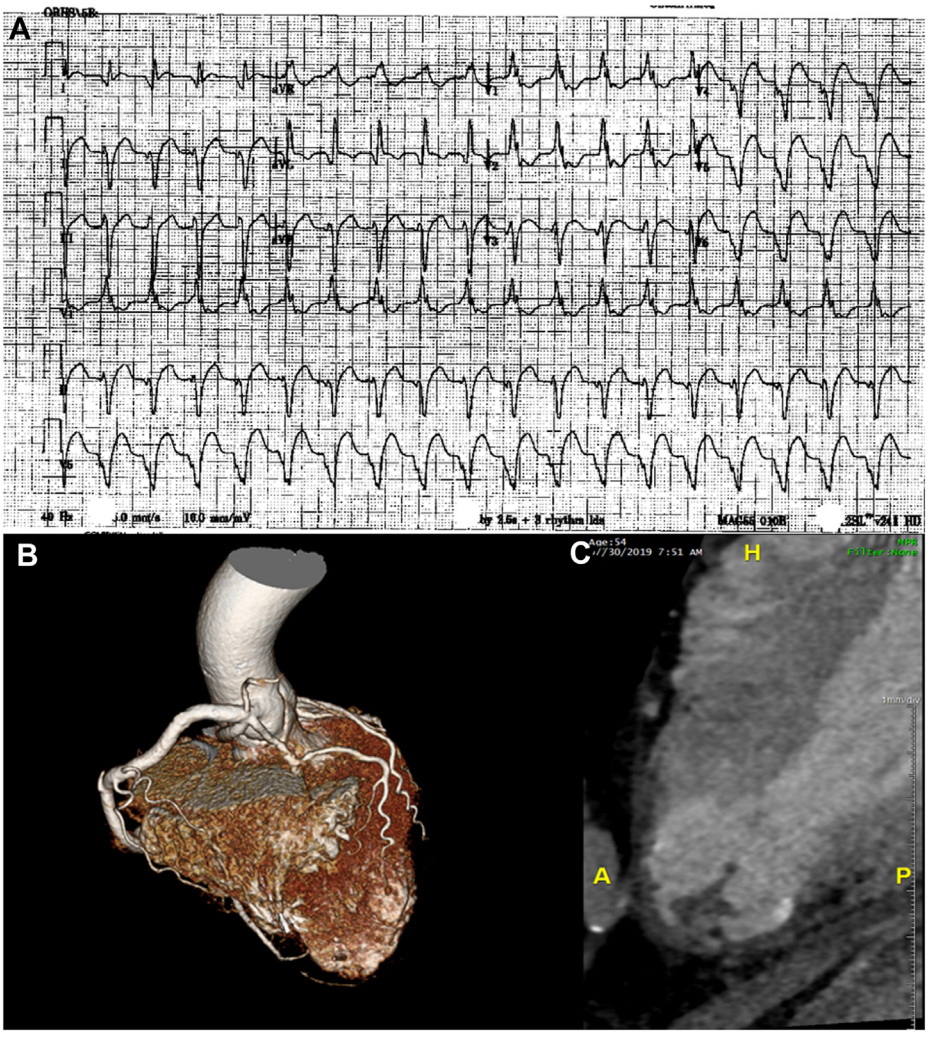

Figure 1 **A:** Electrocardiogram showing monomorphic ventricular tachycardia at cycle length of 530 ms. There is a right bundle branch block configuration with transition in V_3, left axis deviation, and a QR pattern in aVL compatible with an inferoseptal apical left ventricular exit. **B:** Cardiac computed tomography angiogram (CTA) demonstrating anomalous left anterior descending and circumflex arteries arising from proximal right coronary artery. **C:** Cardiac CTA showing apical aneurysm with apical thrombus.

coronary system with no obstructive coronary disease (Figure 1B). The LAD and circumflex arteries each arose separately from the proximal right coronary artery. The LAD passed anterior to the left ventricular outflow tract and terminated at the mid ventricle without supplying the LV apex. The origin of the LAD was oval and not slit-like, and there was no intramural segment. CTA showed severe myocardial thinning of the mid and apical inferolateral walls, consistent with a large remote myocardial infarction. An apical aneurysm was present with a 16 mm thrombus in the LV apex (Figure 1C). Her LV thrombus was unchanged in size despite 2 years of anticoagulation and was felt to be a laminated mural thrombus. Positron emission tomography / computed tomography cardiac viability study with stress showed apical scar without evidence of myocardial ischemia. The etiology of her apical infarction was unclear and thought to be secondary to congenitally truncated LAD or possible remote embolic event. There were no atherosclerotic lesions or targets for revascularization.

The patient was taken for endocardial substrate ablation. Despite the apical thrombus being laminated, a Sentinel® Cerebral Protection System (Boston Scientific, Marlborough, MA) was deployed in the bilateral carotid arteries for stroke prevention (Figure 2A). LV access was achieved via transseptal puncture using a steerable Agilis® sheath (Abbott, Plymouth, MN, USA). An irrigated Thermocool® SmartTouch™ bidirectional ablation catheter with D/F curve (Biosense Webster, Inc, Irvine, CA, USA) was used to map the LV endocardium, confirming an apical aneurysm with dense scar at the apex (Figure 2B). In sinus rhythm, deceleration zones were noted in the inferolateral apex, and compatible pace maps were seen in the inferoseptal apex. Nonsustained monomorphic VT was induced, which was consistent with her clinical VT. Entrainment maneuvers were not performed owing to concern for apical thrombus dislodgement. Endocardial substrate ablation was performed circumferentially at the neck of the aneurysm and the inferolateral wall of the LV. Ablation was performed

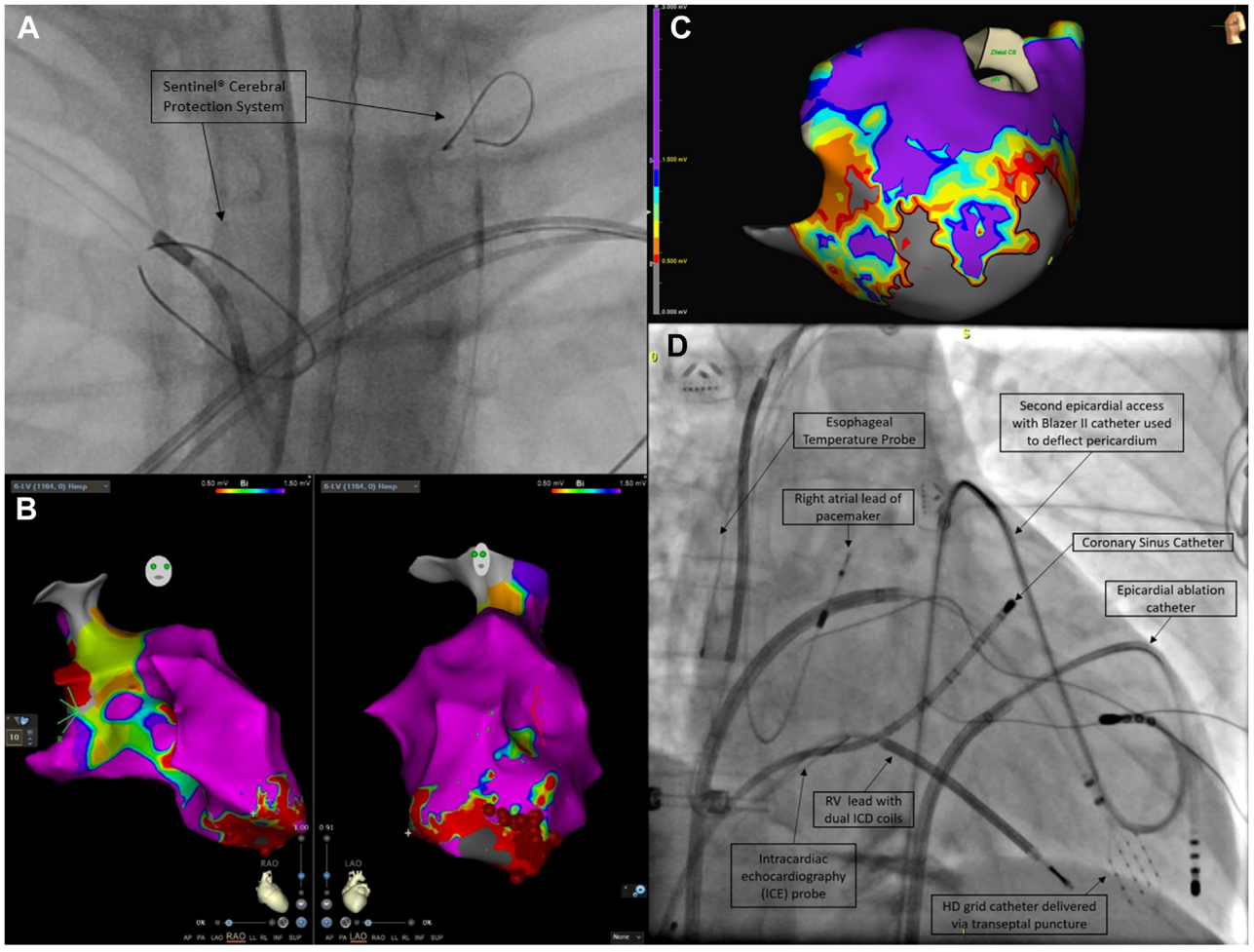

Figure 2 **A:** Fluoroscopic image showing Sentinel Cerebral Protection System (Boston Scientific, Marlborough, MA) deployed for stroke prevention. **B:** Right anterior oblique (RAO) and left anterior oblique (LAO) views showing voltage map of left ventricular endocardial substrate during endocardial ablation. Map shows apical inferolateral and inferior outpouching and scar. Red dots represent ablation lesions performed at the scar borderzone. **C:** Left lateral view of the left ventricular epicardium demonstrating apical scar and the borderzone. **D:** Fluoroscopy during epicardial ablation. A Blazer II catheter (Boston Scientific, Marlborough, MA) was inserted through second epicardial abscess and used to deflect away the pericardium, with subsequent loss of phrenic nerve capture. ICD = implantable cardioverter-defibrillator; RV = right ventricular.

with power at 25–40 W for 60–120 seconds, targeting impedance drops of 5–10 ohms. Optimal ablation was defined as lack of capture with high output pacing at 20 mA at 2 ms pulse width. At the end of the procedure, VT was noninducible.

The patient did well for approximately 1 year and then had a recurrence of monomorphic VT, with 8 shocks in the prior 6 months. These occurred despite high doses of amiodarone, mexiletine, and metoprolol. Her VT was faster, with a cycle length of 470 ms. Electrocardiography was similar to the prior VT, with subtle differences in notching patterns. QRS duration was increased with a slurred initial deflection, suggesting a possible intramyocardial or epicardial exit. Consequently, the patient was offered repeat radiofrequency ablation with both endocardial and epicardial approaches.

Prior to ablation, a Sentinel Cerebral Protection System was again deployed for stroke prevention. Epicardial voltage mapping performed using an HD Grid mapping catheter showed apical lateral and inferior scar with late and very late potentials (Figure 2C). Pace mapping helped identify the exit at the epicardial inferolateral apex. Ablation was performed with power set at 25–40 W for 60–120 seconds targeting impedance drops of 5–10 ohms. Ablation was limited by the presence of phrenic nerve stimulation in the area of interest. A second epicardial access was performed, and dextrose 5% solution (D5W) was infused into the pericardial space in an attempt to displace the parietal pericardium and deflect the phrenic nerve for ablation. The patient had a drop in blood pressure with D5W infusion, requiring the removal of fluid from the pericardial space. A standard curve Blazer™ II catheter (Boston Scientific, Marlborough, MA) was then inserted into the pericardial space and used to mechanically deflect the pericardium and phrenic nerve (Figure 2D). This allowed for ablation around the epicardial

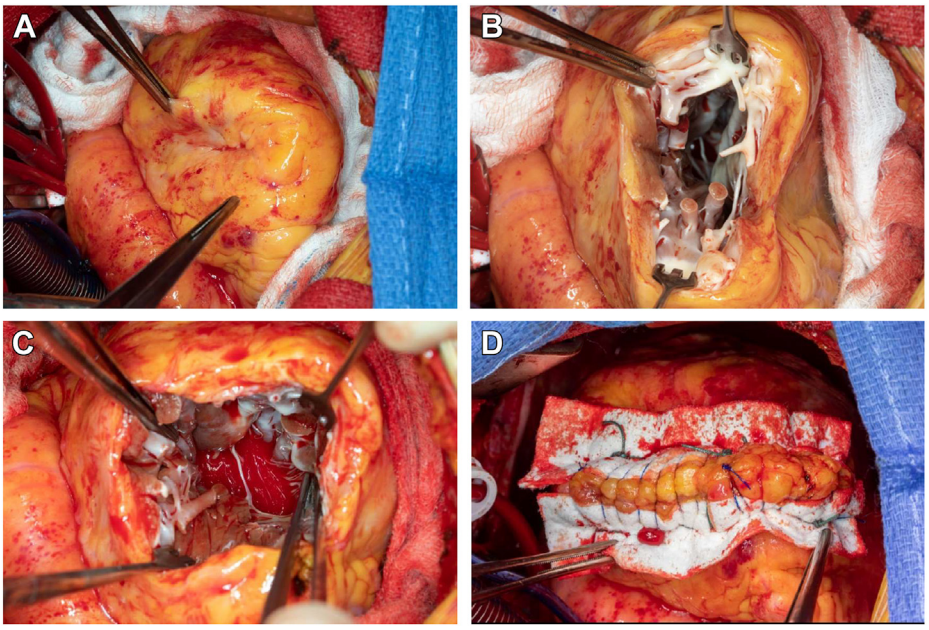

Figure 3 Surgical resection of apical aneurysm. **A:** Sternotomy after pericardium resection showing left ventricular (LV) apex with aneurysm. **B:** Incision into apical aneurysm with view into LV cavity. **C:** LV cavity after aneurysm resection. **D:** Closure of LV incision after aneurysm resection.

scar, after which VT was noninducible. The phrenic nerve was intact with good phrenic capture at the end of the case.

Unfortunately, the patient had a recurrence of a similar VT a few months later. Her VT was less frequent, but her quality of life was significantly impaired. A repeat coronary angiogram again showed no atherosclerotic coronary artery disease. There was concern for possible ischemia from her anomalous circumflex artery. Instantaneous wave-free ratio (iFR) was performed at rest and with supine bicycle exercise to assess the functional significance of her anomalous coronary arteries. No evidence of myocardial ischemia was found.

The decision was then made to undergo surgical aneurysmectomy and subendocardial resection in order to decrease the scar burden and nidus for VT. Left ventricular apical aneurysm was excised with removal of the apical thrombus. Surgery was successful (Figure 3A–3D) and her postoperative course was uneventful. Our patient has had no ICD therapies or recurrence of VT for more than 2 years after surgery.

Discussion

This case highlights several creative strategies that can be useful during ablation and substrate modification for ventricular arrhythmias. First, a Sentinel Cerebral Protection System was deployed for stroke prevention during endocardial ablation in the setting of an LV apical thrombus. Safety and feasibility of these devices for VT ablation have been demonstrated in small case series, and further studies are needed to define the benefits of these devices during VT ablation.[1,2] During epicardial ablation, care was taken to deflect the phrenic nerve to allow for epicardial mapping and avoid nerve injury. Injury to the phrenic nerve can occur with endocardial or epicardial ablation and can lead to phrenic nerve palsy and permanent diaphragmatic paralysis.[3] The right phrenic nerve travels along the lateral surface of the superior vena cava and over the pericardium of the right atrium.[4] Injury can occur during ablation involving the superior vena cava, right atrium, or right upper pulmonary vein.[5] The left phrenic nerve travels over the left atrial appendage and along the pericardium of the LV.[4] This nerve can be injured during ablations involving the lateral LV.[5] High-output pacing can be used to identify sites close to the phrenic nerve prior to ablation.[4,5] There are several techniques for displacing the nerve to allow ablation near the nerve course. Injection of saline into the pericardial space can deflect the parietal pericardium and phrenic nerve.[5] Limitations of this technique include development of tamponade physiology and hemodynamic compromise, as was noted in our case. Intrapericardial balloon inflation can also be used to separate the phrenic nerve from the epicardium. This technique was considered for our patient; however, given the hemodynamic compromise after saline injection, there was concern that balloon inflation may have a similar effect. This case demonstrates a third technique for phrenic nerve protection in which a second epicardial access can be placed, and a catheter can be used to mechanically deflect the phrenic nerve.[5] The advantages of using a second steerable ablation catheter to displace the phrenic nerve were increased maneuverability and a smaller profile compared to balloon inflation.

The present case also demonstrates strategies for substrate modification in ventricular arrhythmias. Both endocardial

and epicardial ablations were performed in this patient, with eventual recurrence of her VT. Ultimately, our patient underwent surgical resection of her apical aneurysm, highlighting aneurysmectomy as an effective treatment for VT refractory to catheter-based therapies. Surgical excision of LV aneurysms was first described as therapy for drug-resistant VT in the late 1950s.[6] In 1979, Josephson and colleagues[7] presented a surgical technique in which intraoperative activation mapping was used to guide subendocardial resection at the aneurysm border, a technique that became known as the "Pennsylvania peel." This publication demonstrated that VT frequently originates from the subendocardium at the aneurysm border, a location that was not typically resected at that time.[7] Later, surgical cryoablation was used as an adjuvant to surgical aneurysm resection with endocardectomy, particularly when VT was localized to difficult-to-resect regions such as papillary muscles or midmyocardial tissue.[8,9]

The success of intraoperative activation mapping, endocardial resection, and surgical cryoablation in treatment of VT played a role in driving the emergence of electrophysiology and catheter-based ablation techniques in the early 1990s.[9] Catheter ablation is now first line as therapy for recurrent drug-resistant sustained VT.[10] Despite the high success rate of percutaneous VT ablation, recurrence of VT is not uncommon, occurring in as much as 30% of patients within 2 years.[11,12] These refractory cases often have midmyocardial or epicardial circuits, making percutaneous ablation less effective.[13] In VT refractory to percutaneous ablation, surgical management including surgical cryoablation and surgical aneurysmectomy with endocardial resection remain therapeutic strategies that should be considered.[13]

Conclusion

This case highlights the complexities of substrate modification in patients with VT. Surgical resection of an apical aneurysm can be effective for reducing VT in cases refractory to medical therapy and catheter ablation.

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