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Use of Computed Tomography Delayed Enhancement Imaging and a Ventricular Assist Device in Ablation of Recurrent Ventricular Tachycardia

DAVID CESARIO, MD, PhD, JEROLD SHINBANE, MD, GILBERT ESSILFIE, MD, SANDEEP TALWAR, MD, MICHAEL CAO, MD, JABI SHRIKI, MD, LESLIE SAXON, MD and MARK CUNNINGHAM, MD

Cardiovascular Thoracic Institute, Keck School of Medicine, University of Southern California, Los Angeles, CA

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Introduction

Ventricular tachycardia (VT) in the setting of ischemic cardiomyopathy can often be mapped to postinfarct myocardial scars. Within the scarred myocardium, slow or aberrant conduction can facilitate reentrant VT. Patients with extensive myocardial scarring can have VT that is refractory to treatment with implantable cardioverter-defibrillator (ICD) devices and medical management. Studies have shown that electroanatomic mapping of VT circuits and catheter ablation is the treatment of choice when ICD and medical therapy fail to adequately control VT.

We present a 74-year-old male with recurrent VT status post attempted catheter ablation. The patient’s past medical history was significant for ischemic cardiomyopathy, coronary artery disease (CAD) status post coronary artery bypass surgery, sick sinus syndrome, atrial fibrillation status post atrioventricular (AV) nodal ablation, and biventricular ICD placement. Catheter ablation initially produced a significant decrease in the patient’s VT burden, but VT episodes returned within a few months of the ablation. The patient was intolerant of amiodarone secondary to neurotoxicity. Medical management with both dronedarone and dofetilide failed to control VT, prompting attempts at remapping and ablation. During the electrophysiology study, the patient went into VT storm and became too unstable hemodynamically to complete the procedure. He required a total of 9 external shocks and was given boluses of lidocaine and amiodarone intravenously before sinus rhythm was restored. The patient subsequently underwent installation of a HeartMate II (Thoratec Corporation, Pleasanton, CA), which provided hemodynamic support for a successful epicardial intraoperative ablation procedure at the time of his ventricular assist device (VAD) placement.

Extensive myocardial scarring and advanced ischemic cardiomyopathy can facilitate rapid recurrent VT. Under such conditions, the myocardium can have a very low threshold for triggering VT. This makes electroanatomic mapping of VT substrate virtually impossible. Delineation of VT ablation targets using cardiac computed tomography (CT) has been previously demonstrated, and was useful in defining the scar tissue associated with the recurrent VT. However, we were initially unable to pursue even a substrate-based ablation strategy in this case, secondary to the frequent occurrence of hemodynamically unstable VT and eventual VT storm. We were eventually able to pursue a substrate-based epicardial ablation at the time of surgery after we had the patient on left ventricular assist device (LVAD) placement.

Case study

The patient is a 74-year-old male with a history of CAD status post coronary artery bypass grafting surgery. He
also has a history of ischemic cardiomyopathy, paroxysmal atrial fibrillation, paroxysmal ventricular tachycardia, and sick sinus syndrome. The patient was status post AV nodal ablation, biventricular ICD placement, and attempted catheter ablation for VT. His medications include spironolactone (Aldactone), warfarin (Coumadin), digoxin, furosemide (Lasix), lisinopril, metoprolol, mexiletine, dronedarone (Multaq), omega-3 (Omacor), sublingual nitroglycerin, and aspirin.

Electroanatomic mapping revealed a rapid VT with left bundle branch block (LBBB) morphology, which was mapped to a large anteroseptal scar. He underwent catheter ablation at an outside facility with the placement of multiple radiofrequency ablation lesions along the scar perimeter. The patient tolerated the procedure well and initially had significantly fewer episodes of VT. However, 3 months after the ablation procedure, the patient experienced two new episodes of VT within a 3-week period. During these episodes the patient reported experiencing significant lightheadedness and rapid palpitations but denied frank syncope or angina (Figure 1 shows a typical VT episode from ICD interrogation). Additionally, he complained of worsening dyspnea on exertion to the point where he was short of breath after walking 10 feet.

The patient subsequently experienced two more episodes of VT with ICD shocks and was admitted for further evaluation and management. During his admission, cardiac catheterization showed a normal left main artery, and total occlusion of the left anterior descending (LAD), circumflex, and right coronary arteries (RCA). A saphenous vein graft (SVG) to his LAD diagonal had 20–30% stenosis and the SVG to his circumflex and RCA were both totally occluded. No revascularization targets were identified.

As the patient had an ICD in place contraindicating cardiac magnetic resonance imaging (MRI) delayed enhancement imaging, 64-slice CT delayed enhancement imaging was performed and revealed a transmural inferolateral infarct extending from the basal to the mid-inferolateral wall (Figure 2). There was also evidence of lipomatous metaplasia within the lateral wall due to the chronicity of the infarct. A transesophageal echocardiogram showed no evidence of left atrial appendage thrombus.

The patient failed therapy with dofetilide, and we attempted an electrophysiology study to map the VT for ablation.

The baseline RR interval was 800 ms and it was a paced complex. Pacing was done from the right ventricular apex with a single extra stimulus, which induced a rapid VT with a cycle length of 300 ms and LBBB morphology with upright QRS complexes in leads II, III, and AVF. Subsequent pacing from the free wall of

Figure 1: Implantable cardioverter-defibrillator (ICD) interrogation showing a typical ventricular tachycardia episode. Ventricular tachycardia cycle length was 274 ms, and the arrhythmia was successfully terminated by a 10-J shock from the ICD.

Figure 2: Cardiac computed tomography delayed enhancement image revealing a large scar on the lateral border of the left ventricle.
the right ventricular outflow tract (RVOT) produced rhythms very similar to the clinical VT. Touching the catheter anywhere in the RVOT induced VT. The patient went into VT storm and became hemodynamically unstable, with VT episodes resulting in no measureable blood pressure on our arterial line. The patient required multiple external shocks (a total of 9), and a bolus of amiodarone and lidocaine to maintain sinus rhythm (Figures 3 and 4). The procedure was terminated due to the patient’s hemodynamic instability and he was evaluated by cardiothoracic surgery for VAD support for ablation.

The patient was subsequently taken to surgery. After administering general endotracheal anesthesia, the right atrium was accessed through the femoral vein. Very early in the surgery, the ICD fired due to another VT episode. These events prompted turning off the ICD, and cardiopulmonary bypass was initiated.

A HeartMate II was installed in the usual manner with a left ventricular apical cannula and outflow tract to the aorta. Two areas of well-defined inferolateral scars were identified, which correlated to the areas identified by preprocedural CT scan. These scars were circumscribed with an AtriCure (West Chester, OH) bipolar pen, avoiding damage to the epicardial surface or injury to the coronary vessels. A thickened calcified region of pericardium over the anterior aspect of the right ventricle was excised and submitted to pathology. The patient was weaned off cardiopulmonary bypass and the HeartMate II was initiated (Figure 5 shows chest X-ray with VAD in place). Chest tubes were placed, sternal wires were used to reapproximate the sternum, and the layers of fascia were closed in the usual manner.

**Discussion**

**Severe CAD and VT recurrence status post ablation**

The patient’s history of significant CAD and ischemic cardiomyopathy are characteristic of advanced structural heart disease that could be formidable to treat because of the chronicity and extensive scarring.

The high incidence of VT in structural heart disease and its predominance in patients with CAD is well recognized. Although VTs in this setting have been well studied, there is still a significant recurrence rate after ablation. Large infarct scars are often associated with multiple reentrant VT circuits, not all of which can be identified and characterized by electroanatomic map-

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**Figure 3:** Spontaneous initiation of monomorphic ventricular tachycardia with cycle length of 323 ms.

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ping. Also, in some instances the critical VT zone is located in the epicardium or intramural regions, making VT circuits difficult to access. These and other factors may account for the recurrence of VT after ablation. Extensive characterization of scar tissue is, therefore, a prerequisite to improve the success of ablation.

CT delayed enhancement imaging of myocardial scars for ablation

VT in the setting of structural heart disease and CAD can be unstable or hemodynamically poorly tolerated, making electroanatomic mapping very difficult. In our patient’s case, attempts to map the VT triggered VT storm with hemodynamic instability, thus preventing successful mapping. Under such circumstances, alternative characterization is required to define the VT substrate for ablation.

Imaging of cardiac tissue with MRI provides excellent detail of cardiac pathology, including viability of cardiac tissue and spatial distribution of ischemic damage in
postinfarct scars. The presence of implanted metal devices, however, precludes using MRI and makes CT imaging an attractive alternative. Reports of CT-guided ablation of VT substrate have demonstrated that the delineation of myocardial scars by CT with contrast is very well correlated with scar topography defined by both positron emission tomography (PET) imaging and electroanatomic mapping.

In the case of our patient, CT imaging of cardiac scars identified several areas of scarred myocardium consistent with the patient’s extensive history of heart disease. These images were well correlated with the scars that were seen and circumscribed with the AtriCure bipolar pen during surgery. Although CT/PET imaging of VT substrates has been reported in previous literature, this case further demonstrates its utility in evaluating lesions for ablation.

Using a VAD for hemodynamic support during ablation
The use of VAD as a bridge to heart transplantation or destination therapy has been shown to improve survival and functional status of patients with advanced heart failure. Using VAD for hemodynamic support in a variety of cardiac procedures has also been demonstrated with a low incidence of complications and low operative mortality. This made using a VAD for hemodynamic support an attractive treatment strategy in this case. The HeartMate II VAD was installed in the usual manner without complication, providing the hemodynamic stability needed for VT ablation. Although VT ablation in patients with VAD has been demonstrated, to the best of our knowledge, this is the first case report of an LVAD installed to provide hemodynamic stability concurrently with the ablation procedure.

Conclusion
Severe CAD can cause chronic myocardial ischemia and extensive scarring leading to ischemic cardiomyopathy. Such extensive scarring can precipitate recurrent VT. In this setting ICD and medical management are the first-line therapies to control dysrhythmia and reduce the risk of cardiac arrest. Refractory and unstable VT, however, necessitates catheter ablation as an adjunctive therapy for managing VT. This case demonstrates the use of CT cardiac imaging as an alternative to electroanatomic mapping and installing an LVAD for hemodynamic support in a patient with unstable VT. The CT imaging and LVAD provided a means to VT ablation in a case of severe unmappable VT that would have been formidable to treat otherwise.

References