Teaching Rounds in Cardiac Electrophysiology

Three-Dimensional Delayed-Enhanced Cardiac MRI Reconstructions to Guide Ventricular Tachycardia Ablations and Assess Ablation Lesions

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Voltage mapping is the primary tool for identifying sites for substrate-guided ventricular tachycardia (VT) ablation, but there are limitations in its application. Delayed-enhanced cardiac MRI (DE-CMRI) can facilitate VT ablations by providing complementary detail about myocardial scar location and geometry. We present the case of a patient with recurrent VT with left bundle branch block (LBBB)-right-side inferior axis morphology and a challenging voltage map.

Case

The patient was a 76-year-old man with a history of hypertension, type 2 diabetes mellitus, atrial fibrillation, and newly diagnosed dilated nonischemic cardiomyopathy (ejection fraction, 30%). He was transferred to our facility because of multiple episodes of monomorphic VT with a heart rate of 160 beats/min and an LBBB-right-side inferior axis morphology (Figure 1) consistent with an origin in the right ventricular outflow tract (RVOT). He required 2 episodes of electric cardioversion and amiodarone therapy, but the VT was refractory to amiodarone, so the patient underwent a VT ablation procedure.

CMRI with a 1.5-T Siemens Avanto scanner was performed in multiple anatomic planes using T1-weighted and cine steady-state free precision sequences. Gadolinium-enhanced sequences to evaluate early myocardial perfusion and delayed myocardial enhancement were also performed, using 0.1 mL/kg gadobenate dimeglumine. DE-CMRI demonstrated midmyocardial scar in the anteroseptal region of the RVOT (Figure 2), which raised the possibility of a substrate-mediated VT.

Reformatted 2D slices using Amira software allowed the reconstruction of 3D models of the right ventricle (RV) and left ventricle (LV) anatomy with embedded myocardial scar. These were exported as 3D Carto-readable mesh files and allowed the identification of mapping point positions on the corresponding 2D images. MRI surfaces were uploaded into the clinical CartoMERGE system (Biosense).

RV and LV voltage maps were created using the CartoMERGE system and a 3.5-mm Navistar cooled-tip catheter (Biosense Webster) with a filling threshold of 15 mm. The 3D DE-CMRI reconstructions were coregistered as previously described, using the commercial visual alignment and surface registration algorithms. Standard clinical voltage criteria were used to define scar (<0.5 mV), border zone (0.5–1.5 mV), and normal (>1.5 mV) myocardium. A bipolar endocardial RV and LV voltage map revealed no endocardial scar (Figure 3).

Using programmed electric stimulation, VT was easily inducible, with a tachycardia cycle length of 418 ms (LBBB-right-side inferior axis morphology). Activation mapping demonstrated earliest activation in the high septal outflow tract but was limited because of self-termination. The normal LV and RV voltage maps and 12-lead VT morphology raised the question of a more benign RVOT tachycardia mechanism occurring coincidentally in this patient with newly diagnosed nonischemic cardiomyopathy. However, the integrated scar substrate in the antero-septal RVOT suggested a scar-mediated reentrant VT and directed the pace mapping to the area of midmyocardial delayed enhancement, where a 12/12 pace map match was found (Figure 4). Ablation at the superior border of the septal scar (30–50 W; total ablation time, 297 s) resulted in noninducibility of the VT with programmed electric stimulation of up to triple extrastimuli both on and off isoproterenol as well as burst pacing.

Postablation DE-CMRI performed 24 hours later as part of a research study demonstrated ablation lesions as distinct areas of nonreflow reaching into the area of previously observed midmyocardial MRI scar (Figure 5). Two days after the VT ablation, the patient had an implantable cardioverter-defibrillator (ICD) implanted for secondary prevention. During a 6-month follow-up period, the patient remained off antiarrhythmic drugs and has not experienced any episodes of ventricular arrhythmia.

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Discussion and Teaching Points

RVOT Tachycardia

RVOT tachycardia is the predominant form of idiopathic VT, and the cellular mechanism is believed to be triggered by c-AMP-mediated delayed afterdepolarization activity induced by catecholamines. Patients generally do not have structural heart disease. In 90% of cases, it manifests as repetitive monomorphic nonsustained VT or paroxysmal exercise-induced sustained VT. The morphology of RVOT VT is monomorphic with an LBBB pattern and a left- or right-side inferior frontal plane axis. There appears to be an equal male-to-female distribution, and the majority of patients are aged 30 to 50 years, although the age range can span from 6 to 77 years. This condition has an excellent prognosis with no requirement for ICD implantation. Although patients with no or infrequent symptoms may require no treatment, the treatment of choice for VT-associated symptoms, such as presyncope or syncope, is radiofrequency ablation. Successful ablation rates are in the 90% to 95% range, with minimal complication rates. Alternatively, antiarrhythmic agents, such as β-blockers, calcium channel blockers, and class I and III antiarrhythmic agents, can be used. Although the prevalence of

Figure 1. Clinical ventricular tachycardia with left bundle branch block-right-side inferior axis morphology.

Figure 2. Preablation 2D delayed-enhanced cardiac MRI. Preablation short-axis views from base to apex, showing delayed gadolinium enhancement corresponding to midseptal scar in the anteroseptal area of the right ventricular outflow tract (arrows).
c-AMP-triggered RVOT-type VT in patients with a normal voltage map and cardiomyopathy is not known, the presence of a cardiomyopathy should always raise strong concerns about a scar-mediated reentrant VT.

CMRI Detection of Scar-Related VT Substrate

CMRI is considered the current gold standard to image cardiac fibrosis or scarring. An excellent correlation between CMRI scar and histological examination has been shown in canine studies. CMRI allows for high-resolution evaluation of the RV anatomy, tissue characterization, and wall motion abnormalities to rule out other structural abnormalities, such as arrhythmogenic right ventricular cardiomyopathy.

In the present patient, although the 12-lead morphology, VT origin, and normal voltage map were compatible with a triggered outflow tract tachycardia, the cardiomyopathy and presence of myocardial scar on DE-CMRI as well as induction with programmed electric stimulation indicated a substrate-related reentrant mechanism. It is important to point out that the midmyocardial scar was not detected by bipolar voltage mapping from the RV or LV, which may have been due to the myocardial thickness because endocardial layers of ≥2-mm thickness frequently result in bipolar voltage values of >1.5 mV.1

The reconstructed and integrated DE-CMRI scar directed the pace mapping to the area of midmyocardial scar and resulted in the rapid identification of the best 12/12 pace map site. Importantly, it provided a rationale to ablate in an area of preserved voltage because the underlying scar substrate had been confirmed.

CMRI Characterization of Ablation Lesion Relative to Scar Substrate

The matched location of CMRI substrate and the successful ablation site confirmed the likely substrate-based reentrant mechanism. Additionally, the visualization of the ablation extending into the area of scar provided confirmation that a sufficiently large ablation lesion to alter the scar substrate had been created and that scar modification had been performed. Such a combination of intramyocardial scar and normal voltage might provide an explanation for the location of ~4% of successful ablation sites within healthy myocardium as defined by voltage mapping.6

Recently, Ilg et al6 demonstrated the feasibility of visualizing ablation lesions with DE-CMRI as delayed enhancement in human myocardium without any preexisting fibrosis. However, the visualization of ablation lesions in areas of fibrosis might be more difficult, with DE-CMRI akin to

Figure 3. Three-dimensional voltage map of the right and left ventricles demonstrates normal endocardial voltage (red, <0.5 mV; rainbow, 0.5–1.5 mV; purple, >1.5 mV).

Figure 4. Twelve-lead surface ECG shows the clinical VT with corresponding 12/12 pace match (left bundle branch block right inferior axis QRS morphology). This corresponded to the site of successful ablation.
trying to image a white lesion within white myocardium. A possible solution could be the more pronounced degree of nonreperfusion created by the ablation lesion. Ablation results in a complete loss of cellular and vascular architecture. Although residual vascularity in scar-related fibrosis results mostly in a white enhancement pattern, ventricular ablation lesions demonstrate initially a dark area of nonreflow, as has been shown in animal studies. This case demonstrates that such an approach might be feasible in human cases.

**Take-Home Message**
Although an LBBB inferior axis VT morphology and normal voltage map is consistent with a triggered RVOT VT mechanism, the presence of structural heart disease always requires assessment of scar-mediated VT. DE-CMRI is capable of detecting myocardial fibrosis not identified by voltage mapping (pointing toward a reentrant mechanism), defining the ablation target, and providing visual feedback about the ablation success.

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**References**

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EDITOR’S PERSPECTIVE

This teaching rounds case makes several interesting points. First, ventricular tachycardias (VTs) that originate from scar in the superior septum or outflow region can have an electrocardiographic appearance mimicking idiopathic VT. Second, a normal bipolar voltage map, as defined by an electrogram amplitude $>1.5$ mV, does not exclude the presence of scar deep into the endocardium. Third, it illustrates how imaging to detect scar can potentially help to guide an ablation procedure and may at some point be useful for assessing ablation lesions. These points are amplified herein.

Most sustained monomorphic VTs in structural disease are due to scar-related reentry.2 Rarely is an idiopathic VT found in a patient with structural heart disease, such as an idiopathic right ventricular outflow tract VT in a patient with hypertrophic cardiomyopathy.3 Although idiopathic VT cannot be absolutely excluded in this case, the proximity of the successful ablation site to the scar and inducibility by programmed stimulation favor a scar-related VT. Assessment of entrainment would have been interesting if it were possible. Entrainment would further support scar-related reentry rather than idiopathic VT, which usually behaves in a manner more consistent with triggered automaticity.

Despite its association with a scar, the electrocardiographic appearance of the VT is consistent with idiopathic VT. Electrocardiographic features of left bundle branch-block-like VTs that are associated with scar rather than idiopathic VT include transition after V4, notched downslope in V1 or V2, and a prominent onset of S wave in V1 of $>0.09$ mV. The present VT lacks these features. The case exemplifies the limitation of electrocardiography, which by itself is not a sufficiently reliable indication that VT is not associated with scar. An apparently idiopathic VT usually warrants additional imaging to provide further reassurance that structural disease is absent.

The current bipolar voltage map was normal by present standards, another factor that supports idiopathic VT rather than scar-related VT. A bipolar amplitude of $>1.5$ mV is now widely accepted as the lower limit of normal.4 It is important to recognize, however, the limitations of voltage mapping. Marchlinski and coworkers5 codified the 1.5-mV standard from mapping studies using catheters with 4-mm-tip electrodes separated by 1 mm from a smaller ring electrode. Signals were filtered at 10 to 400 Hz on an electroanatomic mapping system. They mapped 4 right and 4 left ventricles in 6 subjects, finding that 95% of electrograms had an amplitude of $>1.55$ mV in the left ventricle, whereas the corresponding cut point of 95% of electrograms in the normal right ventricle was $>1.44$ mV. Marchlinski et al suggested a 1.5-mV threshold for clinical use. This value has held up remarkably well in different studies for identifying scar in animal models of infarction and in humans, despite some differences in recording systems and recording catheters (eg, 3.5-mm rather than 4-mm distal electrode). It is apparent, however, that this is a specific but has limited sensitivity.

MRI is beginning to define some of these limitations.6-8 Wijmaaalen and coworkers6 compared voltage maps with MRI in patients with prior myocardial infarction. They found excellent correlation of scar, in general, but several patients with marked discrepancies had nontransmural areas of scar that were not appreciated by voltage maps. Dickfeld and colleagues7 found that a bipolar endocardial voltage map with a threshold of 1.5 mV will not detect areas of intramural and epicardial scar that does not extend to within 2 mm of the endocardium. More recently, Hutchinson and coworkers9 exploited the presence of greater far-field signal content in unipolar recordings and developed voltage criteria for unipolar, minimally filtered electrograms that suggest the presence of epicardial, and potentially intramural, scar. Whether a unipolar voltage map would have detected the intramural scar in the present case is not known. Further studies correlating imaging with electrogram characteristics should continue to refine the ability to detect intramural and epicardial scar to help guide ablation. Detection of epicardial scar is particularly relevant when arrhythmogenic right ventricular cardiomyopathy is suspected because the electrophysiological abnormalities in this disease often are more extensive in the epicardium than in the endocardium, and epicardial ablation often is required. MRI is a powerful tool that allows visualization of scar and potentially of radiofrequency lesions. The ability to integrate MRIs with electroanatomic mapping systems in the electrophysiology laboratory will continue to provide insights into the pathophysiology of arrhythmias.7

Unfortunately, many patients who could benefit from this approach have implantable cardioverter-defibrillators (ICDs) that preclude imaging at many centers. When it can be performed, the presence of an ICD degrades the image quality of some regions of the heart.9 Thus, additional methods for imaging scar are of interest.10,11

The present patient likely had a scar-related VT. The etiology of scar is not known, but a myopathic process or infiltrative disease, such as sarcoidosis, is possible. Although no VT was inducible after ablation, he received an ICD. Guidelines recommend ICD implantation for “patients with structural heart disease and spontaneous sustained VT, whether hemodynamically stable or unstable.”12 Data that ICDs improve the rate of mortality in patients with VTs are possible. Although no VT was inducible after ablation, he received an ICD. Guidelines recommend ICD implantation for “patients with structural heart disease and spontaneous sustained VT, whether hemodynamically stable or unstable.”12

References


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