COMPLEX CASE STUDY

Substrate Ablation for Post-Infarction Ventricular Tachycardia

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ABSTRACT. More than 70% of patients with post-myocardial infarction ventricular tachycardia (VT) will have one or more unmappable morphologies induced. Traditional entrainment cannot be performed for these arrhythmias, most commonly due to hemodynamic instability. Substrate ablation allows the operator to effectively eliminate unmappable VT during sinus rhythm by deducing the anatomic relationship of the VT circuit to the patient’s scar. The methods for determining the residual conductive channels within the healed infarction require a detailed assessment of bipolar electrogram amplitude and morphology, as well as sinus rhythm pacing within the scar. Commonly, a combination of linear ablation connecting unexcitable structures or targeting VT exit sites, and focal ablation of abnormal electrograms, is used.

KEYWORDS. myocardial infarction, radiofrequency ablation, ventricular tachycardia.

Introduction

Substrate-based ablation techniques are commonly required to effectively eliminate post-infarction ventricular tachycardia (VT). The characterization of VT using traditional entrainment pacing requires that the induced morphology be “mappable,” thus exhibiting all of the following features: 1) a uniform, single QRS morphology; 2) reproducible inducibility with programmed ventricular stimulation; and 3) hemodynamic stability during VT.1 Unmappable VT lacks one of these three characteristics and, thus, is not amenable to conventional entrainment mapping. Approximately 70% of patients with post-infarction VT referred for catheter ablation will have at least one, and often multiple, unmappable VT morphologies induced, with three-quarters causing hemodynamic embarrassment.1,2 This case report describes an example of ablation of unmappable post-infarction VT, highlighting important concepts related to substrate characterization and ablation strategy.

Case presentation

A 57-year-old man with remote anterior infarction presented with multiple implantable cardioverter-defibrillator (ICD) shocks for monomorphic VT. His arrhythmias were previously well controlled with dofetilide. His rhythm on emergency evaluation is shown in Figure 1. An echocardiogram revealed evidence of prior anteroseptal myocardial infarction with a left ventricular (LV) ejection fraction of 15% and an LV end-diastolic diameter of 6.8 cm. He was subsequently referred for VT ablation.

He was brought to the electrophysiology laboratory where two distinct VT morphologies (VT1: right bundle, left superior axis, and VT2: right bundle, right inferior axis) with identical cycle lengths (360 ms) were induced with double extrastimuli (Figure 1); VT1 matched his clinical arrhythmia. An echocardiogram revealed evidence of prior anteroseptal myocardial infarction with a left ventricular (LV) ejection fraction of 15% and an LV end-diastolic diameter of 6.8 cm. He was subsequently referred for VT ablation.

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The first step was to create a detailed substrate map of the LV during sinus rhythm. This revealed a large area of low bipolar voltage involving the anteroseptal LV and extending from the base to the apex (Figure 2). Numerous sites within the low bipolar voltage region demonstrated fractionated and late potentials.

Next, the voltage threshold values of the bipolar map were decreased from the reference values of 0.5–1.5 mV
until two putative conducting channels were identified (Figure 3). Bipolar pacing (10 mA, 2 ms pulse duration) within the resulting low voltage region (<0.38 mV) failed to produce diastolic ventricular capture. Pacing along the border of the scar was then performed to approximate the exit sites for the induced VTs. The sites at the border of the voltage-derived conducting channels were initially assessed. Pacing at the mid-inferior septum

Figure 1: The patient’s spontaneous clinical ventricular tachycardia (VT; right bundle, left superior axis 360 ms) and the two VT morphologies induced with programmed stimulation (VT1: right bundle, left superior axis 360 ms; VT2: right bundle, right inferior axis 360 ms) are shown. VT1 matched the patient’s clinical arrhythmia.

Figure 2: A detailed electroanatomic map is shown from the right anterior oblique (RAO; left) and left anterior oblique (LAO; right) projections and reveals a large anteroseptal scar extending from the left ventricular base to the apex.
Figure 4) produced a QRS complex, which mimicked VT1; pacing at the basal anterior scar border (Figure 4) mimicked VT2. Pacing deeper within the scar while decreasing the pacing output (Figure 5) revealed shifting of ventricular activation from the mid-inferior septal LV (VT1 exit) to the basal superior LV (VT2 exit). This shift was thought to reflect the contribution of a larger virtual electrode with high output pacing, and more discrete capture of local myocardium within the conducting channel at lower outputs. The prolongation of the stimulus-QRS, coincident with shifting of the site of ventricular activation, suggested significant marked slow conduction in the orthodromic limb of the channel with antidromic conduction block in the opposite direction. Thus, it was hypothesized that VT1 and VT2 propagated in opposite directions within a single conducting channel (Figure 6). A linear ablation strategy was designed to transect the putative conducting channels, thereby connecting larger regions of unexcitable scar. Next, linear lesions were constructed from the border of the normal myocardium (adjacent to the pacing-derived VT exit sites) and extended into the dense scar. Lesions were delivered with a 3.5 mm externally irrigated ablation catheter (Navistar Thermocool, Biosense Webster, Diamond Bar, CA) from 30 to 50 W, to a maximum temperature of 40°C for 60 s. The acute efficacy of each lesion was documented by a marked reduction in local electrogram amplitude and/or an increase in local bipolar pacing threshold. After completion of the linear lesion sets, the patient had no inducible VT with programmed ventricular stimulation, with up to triple extrastimuli to refractoriness from both the right ventricle (RV) and LV. The patient’s postoperative course was unremarkable, and he was discharged on the second day on low-dose β-blockers.

Discussion

When planning a strategy for substrate-based ablation of post-infarction VT, several fundamental concepts must be considered. First, VT circuits are composed of surviving conductive corridors of myocardium within the healed scar, which are bounded by unexcitable barriers. These barriers can be fixed (e.g. valve annuli) or functional, and are defined either by a low bipolar voltage amplitude or by a lack of diastolic capture with bipolar or unipolar pacing. Lowering the “abnormal” voltage threshold on electroanatomic maps can also identify channels of relatively preserved conduction through which the VT may propagate. The resulting VT circuits often have large dimensions, with lengths averaging 20–30 mm and widths of 10–15 mm. Often, multiple VT morphologies may share a single conductive channel. The VT circuits propagate in a perpendicular fashion relative to the scar border, with the exception of peri-mitral circuits that are oriented parallel to the valve annulus. The infarct border zone, defined by a bipolar voltage amplitude of 0.5–1.5 mV, is a critically important region, as more than 50% of VT exit sites and nearly 20% of VT isthmus sites are located there. The VT exit site can be approximated with threshold pacing at the

Figure 3: The patient’s substrate map is shown with the standard endocardial voltage limit of 1.5 mV (left). The voltage threshold is then reduced until continuous corridors of relatively preserved voltage are seen through the scar (right). At a threshold of 0.38 mV, two putative conducting channels (white lines) are visualized.
**Figure 4:** Pacing during sinus rhythm is performed at the infarct border and within the voltage-determined channels. The left panel shows a reasonable pacemap match for VT1 from the mid-inferior septum. The right panel shows an excellent pacemap match for VT2 from the basal anterior left ventricle.

**Figure 5:** Pacing during sinus rhythm is performed from the middle of the infarct, near the intersection of the two voltage-derived conducting channels. With higher output pacing, ventricular activation occurs at the mid-inferior septum and mimics VT1. As the pacing output is decreased, the stimulus-QRS interval increases, ventricular activation shifts to the basal anterior left ventricle, and the morphology mimics VT2.
interface of the infarct border zone and normal myocardium. Finally, sites within the low voltage region demonstrating electrograms with activation after completion of QRS complex in sinus rhythm (late potentials, LP) are commonly found at proximal or central sites within the re-entrant circuit, as well as at sites where VT is terminated with ablation. However, LP are commonly absent at VT exit sites and are often present at a significant minority of bystander sites. Importantly, the paced QRS morphology from critical isthmus sites demonstrating activation in sinus rhythm >40 ms after QRS completion often does not fully replicate the morphology of the spontaneous VT; this is likely due to antidromic capture of circuit elements during pacing.

The substrate ablation strategy for post-infarction VT often incorporates several of the aforementioned concepts. Commonly, linear lesion sets are created to transect the conductive corridors through which the VT propagates. This may be achieved by connecting two non-conductive barriers such as regions of dense scar or valve annuli. Alternatively, the linear lesion can be constructed from the pacing-derived VT exit site at the scar border zone and can extend either perpendicular or parallel to the edge of the scar. Often, multiple perpendicular linear lesions are required to disrupt all potential circuits within the scar. Additionally, focal ablation targeting specific sites that demonstrate isolated diastolic electrograms can be utilized. Regardless of the strategy chosen, the short-term results for substrate ablation of post-infarct VT are similar, with a 1-year recurrence rate of 20–30%.

References