Case Report

Catheter Ablation of the Mitral Isthmus for Ventricular Tachycardia Associated with Myocardial Infarction

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Mitral isthmus ventricular tachycardia (VT) involves a reentrant circuit with a critical isthmus of conduction bounded proximally by the mitral valve and distally by the remote inferior infarct scar. Successful catheter ablation requires placement of a lesion that transects the isthmus and thus prevents wave-front propagation. We report two cases of mitral isthmus-dependent VT. Electroanatomic mapping revealed a VT isthmus in each case, and linear lesions placed from the edge of the inferior infarct scar (as determined on a three-dimensional electroanatomic voltage map) to the base of the mitral valve eliminated the VT. Electroanatomic mapping can be used to identify isthmus boundaries and thus guide successful ablation.

Key words: ventricular tachycardia, myocardial infarction, perimital isthmus, ablation


Introduction

Catheter ablation of ventricular tachycardia (VT) that occurs late after myocardial infarction is becoming increasingly successful due to improved techniques for localizing critical components of the reentrant circuit1–4). Computer modeling and animal and human studies have revealed a narrow isthmus of viable and slowly conducting tissue that forms a critical part of the VT reentrant circuit3). This zone of slow conduction can be bounded by infarcted tissue or a fixed anatomic barrier, such as a valve annulus5,6). In patients with remote inferior infarction, mitral isthmus VT, for which the critical zone of slow conduction is an isthmus of tissue bounded by the infarct distally and the mitral valve proximally, has been described5). Such VT can have left or right bundle branch block morphology, depending on the direction of wavefront propagation around the mitral valve. Ablation at the inferior isthmus eliminates VT of either morphology.

Vulnerable regions of the VT circuit can be localized during endocardial catheter mapping by means of fractionated electrograms, mid-diastolic electrograms, or entrainment with concealed fusion3,7). Entrainment with concealed fusion suggests pacing at a site within the reentrant circuit, but it can also occur at bystander sites near the reentrant circuit that are not critical to it3,8). Further refinements in pacing criteria to establish the location of the catheter in a zone that is critical to the reentrant circuit include comparison of the degree of stimulus-to-QRS delay during concealed entrainment with that of the electrogram-to-QRS interval during tachycardia3). Additionally, a postspacing interval equal to the VT cycle length suggests catheter location within the reentrant circuit3).

Despite these improved techniques, many VTs cannot be ablated successfully, and clinical success rates remain limited compared to those for ablation of other arrhythmias9,10). Various factors may result in failure to successfully ablate the critical slow conduction zones, including inadequate lesion depth and inadequate lesion width11). To abolish reentry successfully, the ablation lesion must fully transect the critical isthmus to prevent wavefront propagation. Herein, we describe two patients with mitral isthmus VT. A linear lesion placed from the edge of the inferior infarct (as determined on the three-dimensional [3D] electroanatomic voltage map) to the base of the mitral valve eliminated the tachycardias.

Case Reports

Case 1: A 70-year-old man suffered inferior myocardial infarction in 2001. He did well through 2009. Starting in January 2010, he began to experience palpitations lasting approximately 30 minutes, and in July 2011, he was transported to Nihon University Hospital by ambulance because of sustained palpitations. Twelve-lead ECG upon admission showed sustained VT at 280 beats/min with a right bundle branch block pattern and superior axis (Fig. 1); the VT was treated with cardioversion. Transthoracic echocardiography revealed a left ventricular ejection...
fraction of 52% with severe hypokinesia of the left ventricular inferoposterior region. The patient underwent electrophysiologic study and radiofrequency ablation.

**Case 2:** A 60-year-old man suffered inferior myocardial infarction in 1994. He underwent coronary artery bypass surgery for three-vessel disease in 2011, and he received an implantable cardioverter defibrillator (ICD) for recurrent VT (Fig. 2) in 2012. Ten months later, he presented with frequent ICD shocks secondary to VT and was therefore brought to the electrophysiology (EP) laboratory for VT ablation. Transthoracic echocardiography showed a left ventricular ejection fraction of 34% with akinesia of the left ventricular posterior septum to the inferolateral region and dyskinesia of the mid-inferior-apex region.

**Electroanatomic mapping**

Electroanatomic mapping was performed with a CARTO Mapping System (Biosense-Webster, Diamond Bar, CA, USA), as previously described. Briefly, the system consists of an external ultra-low magnetic field emitter (location pad) that is placed beneath the patient and a NAVI-STAR TERMOCOOL® mapping/ablation catheter (Biosense-Webster), which incorporates an integrated passive magnetic field sensor to permit localization of the catheter. The 7.5-French location mapping catheter has electrodes capable of recording unipolar and bipolar electrograms that are associated with a point in 3D space relative to a location reference patch electrode placed on the body surface. When the mapping catheter is moved along the endocardium,
**Fig. 3** Electroanatomic map of the case 1.
*Left panel:* 3D left ventricular voltage map viewed from a posterolateral position.
*Right panel:* Local bipolar electrogram recorded from the viable tissue shows late ventricular potential (LP, arrow).
*Middle panel:* Morphology of the paced 12-lead ECG recorded from this site was a 94% match to that of the clinical VT morphology.

**Fig. 4** Electroanatomic map of the case 2.
*Left panel:* 3D left ventricular voltage map viewed from the posterolateral position.
*Right panel:* Three VT morphologies and paced QRS morphologies during sinus rhythm.

**Fig. 5** Suggested VT1, VT2 and VT3 activation patterns in case 2.
multiple points can be catalogued and used to create a map depicting either local activation or voltage superimposed on the 3D cardiac structure. We used the CARTO Mapping System in conjunction with a conventional electrophysiologic workstation (BARD LABSYSTEM™ PRO EP Recording System, Bard Electrophysiology, Lowell, MA, USA). Conventional pacing techniques including comparison of stimulus-to-QRS with electrogram-to-QRS timing and postspacing intervals were used in conjunction with voltage and activation time electroanatomic maps to identify critical regions within the arrhythmia circuit.

Electrophysiologic study

Both patients were brought to the EP laboratory in the post-absorptive state. The following catheters were inserted percutaneously from the femoral veins: a 6-French quadripolar catheter to the right ventricular apex; a 6-French octopolar catheter to the His-bundle region; a 6-French deflectable quadripolar catheter to the right high atrium. A 7-French open lumen decapolar catheter to the coronary sinus was inserted from the right internal jugular vein.

Case 1: Clinical VT at a cycle length of 240 ms was induced by double ventricular premature stimuli with infusion of 5 μg/min isoproterenol. Hemodynamic deterioration prevented activation mapping. Therefore, the NAVI-STAR TERMOCOOL® mapping/ablation catheter was advanced to the left ventricle (LV) via retrograde aortic approach, and a left ventricular endocardial voltage map was created (Fig. 3). On this bipolar voltage map, regions with voltage < 0.5 mV, which likely represented infarcted tissue and/or aneurysm, were depicted in red, as shown in Fig. 3, left panel. The LV is viewed from a posterolateral position in Fig. 3, left panel. Note the area of low voltage, but viable, myocardium (colored orange-yellow) bounded by the inferior infarct apically and the mitral valve basally. The local bipolar electrogram, recorded from the mid-inferolateral LV, was characterized by a right bundle branch block pattern and superior axis. The activation wavefront of VT1 was directed mainly toward the inferior septum; therefore, the QRS morphology was characterized by a left bundle branch block pattern and superior axis. The activation wavefront of VT3 was thought to be directed mainly toward the anterolateral LV; therefore, the QRS morphology was characterized by a right bundle branch block pattern and inferior axis. The VT2 activation wavefront was thought to be directed toward both the inferior septum and anterolateral LV; therefore, the QRS morphology was characterized by a right bundle branch block morphology, but the QRS axis showed an intermediate pattern between VT1 and VT3. Ablation with 40–45 watts/60 sec at each point under 30 mL/min saline irrigation was performed at sites with good pace maps (Pace 1, Pace 2 and Pace 3, Fig. 4, left) and also to the delayed and fragmented potentials located at the mitral annulus and within the scar tissue to the mitral annulus. After ablation, no VT was induced with triple ventricular premature stimulation from the right ventricular apex or the outflow tract under isoproterenol infusion. Clinically, there has been no recurrent tachycardia and ICD discharge for 2 years.

Discussion

Mitral isthmus VT occurs in patients with previous inferior myocardial infarction via a reentrant circuit parallel to the mitral valve annulus that contains a critical zone of slow conduction bounded by the infarct and the valve annulus. Friedman et al. described the first case in which a linear lesion was placed between the edge of the infarct and the valve annulus to transect the isthmus after focal lesions...
failed to abolish the VT, and they were the first to use electroanatomic mapping to define the mitral isthmus circuit boundaries\textsuperscript{14}. Although catheter ablation may fail due to inadequate mapping or limited lesion formation, the inability to induce clinical VT and the presence of hemodynamically unstable tachycardias also have contributed significantly to failure of the procedure\textsuperscript{15}. The use of late potentials to guide ablation has been disappointing\textsuperscript{16,17}. Localizing circuit boundaries to direct ablation, however, appears promising. A 3D CARTO voltage map has been used to define circuit boundaries and successfully guide VT ablation. de Chillou et al. reported that detailed 3D electroanatomic mapping is helpful in reconstructing postinfarct VT circuits and in defining the characteristics of their related protected isthmi, and the wide ranging isthmus width values support the need for linear radiofrequency lesions to eliminate the reentrant substrate of postinfarct VTs\textsuperscript{18}. They reported that linear radiofrequency ablation performed across the most accessible part of the isthmus prevented the recurrence of tachycardia in 19 patients (90%) who were followed up for 16 ± 8 months\textsuperscript{19}. Marchlinsky et al. used a different technique, creating linear lesions from dense scar tissue to anatomic boundaries or from dense scar tissue to normal endocardium (applied radially from the abnormal tissue)\textsuperscript{20}. Of 16 patients with drug-refractory VT and frequent ICD shocks with unmappable arrhythmias in whom this technique was applied, 13 (81%) remained free of VT at a median follow-up time of 7 months. They defined scar or abnormal tissue by an amplitude < 0.5 mV.

An ICD was not implanted after ablation in our case 1. It was recently reported that patients with well-tolerated sustained monomorphic VT, structural heart disease, and a left ventricular ejection fraction of > 30% undergoing primary VT ablation without a back-up ICD had a very low arrhythmic death rate, and recurrences were generally non-fatal\textsuperscript{20}.

Limitations
We did not confirm complete mitral isthmus block by mapping the mitral isthmus activation sequence during pacing from adjacent tissue to the ablation line.

Conclusions
Mitral isthmus VT may require a linear lesion for creation of a nonconductive barrier between the inferior infarct and the mitral valve annulus, which form the boundaries of the isthmus of slow conduction critical to VT maintenance. Electroanatomic mapping can be used to define these boundaries and to guide ablation. The two cases described herein further support the concept of anatomically based mapping of VT occurring late after myocardial infarction.

References
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