Ablation of Ventricular Tachycardias With Left Ventricular Apical Endocardial and Epicardial Exit Sites in a Patient With Nonischemic Cardiomyopathy

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ABSTRACT

In this report, we describe a case of ventricular tachycardia (VT) ablation performed in a patient with an implantable-cardioverter defibrillator (ICD) and recurrent VTs with different morphologies and 2 exits, one from the endocardium and another from the epicardium.

A 76-year-old woman with a history of congestive heart failure and with moderate to severe aortic insufficiency had recurrent VT, which became incessant. The patient had received ICD implantation 3 years ago. The problematic VT exhibited a right bundle branch block (RBBB) configuration and superior axis deviation. Two different VT morphologies were seen during programmed right ventricular (RV) stimulation. The first VT was very similar to the clinically documented VT while the second showed a wider QRS complex compared to the first VT. After creating a 3-dimensional voltage map of the left ventricle (LV), we performed pace-mapping along the border zone where multiple isolated delayed potentials were recorded between the low and normal voltage areas. The sites showed well-matched pace-maps gathered in the apical region, where we deployed a linear ablation lesion along the border zone. After endocardial linear ablation, the first VT was not re-induced, but the second VT, which was suspected to exit from the epicardium, was sustained. We placed an irrigating ablation catheter in the apical area facing the endocardial ablation lesion by transcutaneous pericardial puncture. We identified an ablation site that showed an M-shaped local electrogram (EGM) spanning the diastolic interval of the second VT. However, entrainment mapping was not possible at that site due to pacing inability. We attempted ablation based on thermal mapping. Within 7.0 seconds of radiofrequency (RF) energy application at that site, the VT was terminated. The patient continues to be in a stable condition at this time, approximately 6 months following the procedure.

Key words: • endocardium • epicardium • nonischemic cardiomyopathy • RF ablation • ventricular tachycardia

Introduction

Sustained monomorphic ventricular tachycardia (VT) with structural heart disease is often associated with areas of ventricular scarring comprised of
surviving myocytes and fibrotic tissue. After myocardial infarction (MI), scarring involving the endocardium is typically evident, and most re-entry circuits causing VT can be ablated from the endocardium. Sustained monomorphic VT also occurs in dilated cardiomyopathies (DCM) that are not associated with coronary artery disease, although at a lower frequency. Re-entry within the myocardium is the most common cause, although bundle branch re-entry and focal VT also occur. Catheter ablation for VT due to myocardial re-entry in DCM is generally thought to be more difficult than ablation in patients with previous myocardial infarction. In some cardiomyopathies, such as Chagas disease, the presence of epicardial re-entry circuits that cannot be ablated with an endocardial approach contributes to this difficulty.

Recently, a method of plotting low-amplitude regions of scarring on 3-dimensional anatomic reconstructions of the ventricle has been successfully used to mark infarct regions and dense unexcitable scarring that serves as a conduction block in these regions causing VT. The locations of low-amplitude bipolar electrograms (EGM) correlate well with the location of the infarct scars in animal models. In this report, we demonstrate the relationship between monomorphic VTs and areas of low-amplitude scarring through 3-dimensional electroanatomic ventricular mapping of the endocardium, and VTs to have 2 different exits, one from the endocardium and another from the epicardium.

Case Report

A 76-year-old woman with a history of CHF, who has been treated at our institution from March 2009, received a dual chamber implantable cardioverter-defibrillator (ICD) because her left ventricular (LV) ejection fraction was 34% with fast inducible VTs.

At the start of the year 2013, the patient complained of worsening palpitations, which resulted in frequent anti-tachycardia pacing (ATP) and ICD shocks. These episodes became more frequent, and her 12-lead electrocardiogram (ECG) documented a sustained monomorphic VT that exhibited a right bundle branch block (RBBB) configuration and superior-axis deviation (Figure 1). A 12-lead ECG during sinus rhythm revealed a left axis deviation, indicating a left anterior hemiblock.

In May 2013, a radiofrequency (RF) catheter ablation procedure was planned as her VT became incessant despite the use of medications including amiodarone, which she continued to take up until the procedure.

Electrode catheters were positioned in the high right atrium (HRA), right His bundle (HIS), and right ventricular apex (RVA). At baseline, the patient exhibited normal AH (75 ms) and HV (35 ms) intervals with a sinus cycle length of 934 ms. The clinical VT was easily induced by pacing from the RVA. The VT was entrained from the RV pacing and exhibited constant fusion. Two different forms of VTs were induced (Figure 2). Both VTs were noted to have a similar RBBB pattern and axis. The first VT was morphologically similar to the clinically documented VT; the second VT was faster and wider in the duration of QRS. The LV was mapped by a 4-mm tip ablation catheter, which was deployed through the left atrium by transseptal atrial puncture using a Mullin sheath, and a 3-dimensional voltage map of the LV was created. A well-matched pace-map with a stimulus–QRS interval of 20 ms was obtained at the LV interior wall near the apex, implying that the exit site of the first VT was located nearby. After creating a 3-dimensional map of the LV, the clini-
cal VT was not reproducibly induced and RF catheter ablation was performed along the border zone at the LV apical region (Figure 3). After linear ablation of the endocardial aspect, the first VT could not be re-induced. However, the second VT was still re-inducible. The wider QRS duration of the second VT led us to speculate that it was coming from the epicardium.

We performed transcutaneous pericardial puncture to place the ablation catheter in the pericardial space (Figure 4). During epicardial mapping near the bottom of the LV apex, roughly facing the area of the endocardial linear lesion, we were able to identify a site that showed an unusual M-shaped local EGM. The local EGM resembled a far-field potential because of the lack of high frequency potential, but the duration of local EGM seemed to span the diastolic interval of the VT. Because pacing was not possible

Figure 1. (A) and (B). ECG of clinically documented VT. The slightly different QRS morphologies between the figures of the two VTs were noted, especially in lead 1; one with an rS vs. another with a qR wave.
from the epicardial mapping catheter, we decided to attempt thermal mapping. Within 7.0 seconds of RF energy application at that site, the VT was terminated (Figure 5). We delivered RF energy at that site for 60 seconds. Following this, there were no more inducible VTs. The patient continues to remain stable at this time, 6 months after the ablation procedure.

Discussion

This case defines the substrate causing VT in patients with DCM and supports myocardial fibrosis as an important factor. As acknowledged above, myocardial re-entry was the most common cause of sustained VT. In this case, the re-entry circuits were closely related to regions of low-amplitude EGMs, consistent with scarring, and in agreement

Figure 2. (A) and (B) Two different VTs were induced during the procedure. The first VT showed similar morphology to the clinically documented VT. The second VT shared a similar axis to the first VT but showed a wider QRS.
with the findings of previous studies.\textsuperscript{3, 10}

In studies of explanted hearts, de Bakker et al.\textsuperscript{11} found unexcitable fibrosis creating regions of conduction block and surviving myocardium creating potential re-entry circuit paths after infarction and in DCM. Slow conduction through muscle bundles...
separated by interstitial fibrosis can create a zig-zag path, producing slow conduction that promotes re-entry.

The cause of fibrosis in cardiomyopathy (CMP) is not well defined. Scattered regions of replacement fibrosis are commonly seen at autopsy, but confluent regions of scarring are not common. Sustained monomorphic VTs in DCM are usu-

Figure 5. (A) This figure showed the local EGM of success site from epicardial ablation catheter. The local EGM seemed to be far field potential but interestingly, the M shaped local EGM spanned the diastolic interval of VT. (B) The RF energy delivery on the spot of Figure 5-A could terminate the VT within 7 seconds.
ally caused by re-entry associated with low-voltage areas consistent with scarring. In the patients with nonischemic CMP, the scar areas involved in the reentrant VT path are often known to be adjacent to a valve annulus; they extend deep into the endocardium, and can be transmural or greater in extent on the epicardium than on the endocardium.

Although the arrhythmic substrate in patients with myocardial re-entry VT in DCM has several similarities to that in patients with previous infarction, low voltage areas of scarring observed in DCM were frequently adjacent to a valve annulus, as is often the case in VT after inferior wall infarction.\textsuperscript{13, 14} The annulus sometimes seems to form a border for an isthmus in the re-entry path.

It is interesting to speculate that the formation of a long channel, or isthmus, along an annulus contributes to the formation of re-entry circuits that can support sustained monomorphic VT. Pacing demonstrated slow conduction in these regions with long S-QRS delays during pace mapping and entrainment. However, in this present patient who had no history of MI, the echocardiogram interestingly showed apical aneurysm formation which was not noted at the time of ICD implantation, indicating the plausible interrelation between ICD lead placement in the RV apex and the worsening of apical wall motion. Even though the mechanism of apical aneurysm formation in this patient was uncertain, the mapping study showed that the VT exit site was adjacent to the apical region.

The success rate of endocardial ablation for nonischemic CMP was lower than that of post-infarct VT\textsuperscript{2}. Re-entry circuits deep in the endocardium and in the epicardium appear to be a likely explanation. Epicardial mapping led to successful ablation in more than half of the patients in whom it was attempted. The successful ablation sites were again associated with low-amplitude regions. Pacing in these regions also showed evidence of slow conduction. Interestingly, the region of low amplitude was strikingly larger in the epicardium than at the endocardium. The importance of epicardial re-entry circuits in CMP was demonstrated by Sosa \textit{et al},\textsuperscript{6} for patients with Chagas disease, in whom approximately 70% of VTs were epicardial in origin. Recently, Hsia \textit{et al},\textsuperscript{10} used limited epicardial mapping via the coronary venous system to demonstrate epicardial involvement in the re-entry circuits in 3 of 19 patients with CMP unrelated to Chagas disease.

In terms of ECG criteria for prediction of an epicardial origin of VTs, several ECG markers need to be emphasized. Activation from an epicardial origin produces a widening of the initial part of the QRS complex, visible on a conventional surface ECG as a pseudo delta wave. The presence of a Q wave in the limb leads also suggests an epicardial origin of VTs. The wide QRS duration of VT could be a marker of epicardial origin as well. The wider QRS of the second VT compared to the first VT led us to speculate on the possibility of an epicardial origin of VT in this patient, which was confirmed by the epicardial abolition of the VT.

Although safe epicardial ablation has been reported by others,\textsuperscript{6, 15} in this present case, about 600 mL of blood drained through the pericardial sheath for a day. Fortunately, the bleeding stopped spontaneously. Prudent precautions must be taken to avoid coronary artery and phrenic nerve injury. We performed coronary angiography while the ablation catheter was on a target site to assess the distance to the coronary artery and also attempted epicardial pacing to detect proximity to the left phrenic nerve, which, however, was not
Because it is desirable to achieve pericardial access before systemic anticoagulation for endocardial LV mapping, performing epicardial mapping before LV endocardial mapping in DCM is a reasonable consideration. This approach must be balanced, however, by anticipated risks and the experience of the team with the epicardial approach, because many VTs can be ablated from the endocardium.

Conclusions

The patient described in this case report, however, exhibited a VT originating from the apical region, which showed aneurysmal change, indicating a possible connection to ICD lead placement. Combined endocardial and epicardial mapping approaches are likely to improve the success of ablation.

References


