Ventricular Tachycardia Originating from the Epicardium Identified by Intracoronary Mapping Using a PTCA Guidewire

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Ventricular Tachycardia Identified by Intracoronary Mapping. **We report a case in which endocardial radiofrequency (RF) ablation guided by unipolar intracoronary mapping using a guidewire was an effective therapy for treating VT originating from an epicardial site of the left ventricle. A 55-year-old man with dilated cardiomyopathy was referred for treatment of refractory sustained monomorphic VT. The earliest unipolar electrogram with a near-perfect pace map was identified at segment #8 of the left anterior descending artery utilizing a guidewire through a microtube catheter during the VT. VT was terminated by an endocardial RF application at an upper midventricular septal site that did not have a good pacemap or the earliest electrogram, and that was the most adjacent anatomical endocardial site to the guidewire tip. No VT was clinically observed during a follow up period of 14 months.** *(J Cardiovasc Electrophysiol, Vol. 17, pp. 670-673, June 2006)*

ventricular tachycardia, microtube catheter, guidewire, radiofrequency ablation, left anterior descending artery

Introduction

In the majority of sustained monomorphic ventricular tachycardias (VTs) involved in dilated cardiomyopathy (DCM), VT with a reentrant circuit is the most common type; however, bundle branch reentrant VT and focal VT also occur.[1,2] It is generally thought to be more difficult to ablate VT associated with DCM than that after a myocardial infarction, due to the reentrant circuit involving the epicardium. In some cardiomyopathies such as Chaga's disease, an epicardial approach was needed to abolish VT with an epicardial reentrant circuit.[3] We here first report a case in which endocardial radiofrequency catheter ablation (RFCA) guided by unipolar intracoronary mapping using a percutaneous transluminal coronary angioplasty (PTCA) guidewire was an effective technique for treating VT originating from an epicardial site of the left ventricle.

Case Report

A 55-year-old man who previously underwent a Batista's operation and mitral valvuloplasty for DCM was referred for treatment of symptomatic VT refractory to antiarrhythmic drugs. An ECG recorded during sinus rhythm exhibited first-degree AV block with complete right bundle branch block (Fig. 1A). Clinical VT at a rate of 170 beats/minute exhibited a QS pattern in all the precordial leads and an unsettled axis (Fig. 1B). The coronary angiogram demonstrated no significant stenosis, and left ventriculography demonstrated a grossly abnormal, severely dilated chamber with diffuse severe hypokinesis. The estimated LV ejection fraction was 31%, and mild mitral regurgitation was noted.

An electrophysiological (EP) study with a standard recorder was performed after the withdrawal of the antiarrhythmic drugs under documented informed consent. A sustained polymorphic VT with a cycle length of 280 msec was easily induced without the intravenous infusion of isoproterenol, but the clinical VT was not. The sustained polymorphic VT, which was hemodynamically unstable, was immediately converted to sinus rhythm by electrical cardioversion. After an implantable cardioverter defibrillator (ICD) was implanted, the clinical VT could be suppressed by 200 mg/day of amiodarone. However, the amiodarone was discontinued due to the development of interstitial pneumonitis. An arrhythmic event involving an electrical storm with incessant VT occurred 2 months after the cessation of the amiodarone. Therefore, RFCA was performed with a conventional mapping system. Sustained monomorphic VT (cycle length of 350 msec) was induced by an intravenous isoproterenol infusion (0.5–4 μg/minute). VT entrainment was not observed during any of the sustained VT episodes. The VT could not be induced by programmed ventricular stimulation or ventricular burst pacing. It could be terminated by a bolus injection of adenosine triphosphate (10–40 mg) acutely in the electrophysiology laboratory.

The earliest endocardial ventricular electrograms (Vs) recorded during the VT preceded the QRS onset by 26 msec and 20 msec at the right and left midventricular septum, respectively. However, nine RF applications delivered at those sites failed to abolish the VT. The pace mapping at those sites did not display a perfect match. Pace mapping at other endocardial ventricular sites did not display a match. The origin of the VT was thought to be located in the deep myocardium of the ventricular septum. A 0.014-inch guidewire was inserted into a 2.0 F microtube catheter and was positioned in the left anterior descending artery (LAD) and septal branch artery to try and perform intracoronary chemical ablation. VT was not terminated by an injection of 3 mL of cold saline through the microtube catheter in the third septal branch artery, but was in segment #8 of the LAD. Therefore, we tried to perform epicardial unipolar mapping. The distal 5 mm of the tip of the guidewire extending from the end of the microtube catheter was used for the mapping. The

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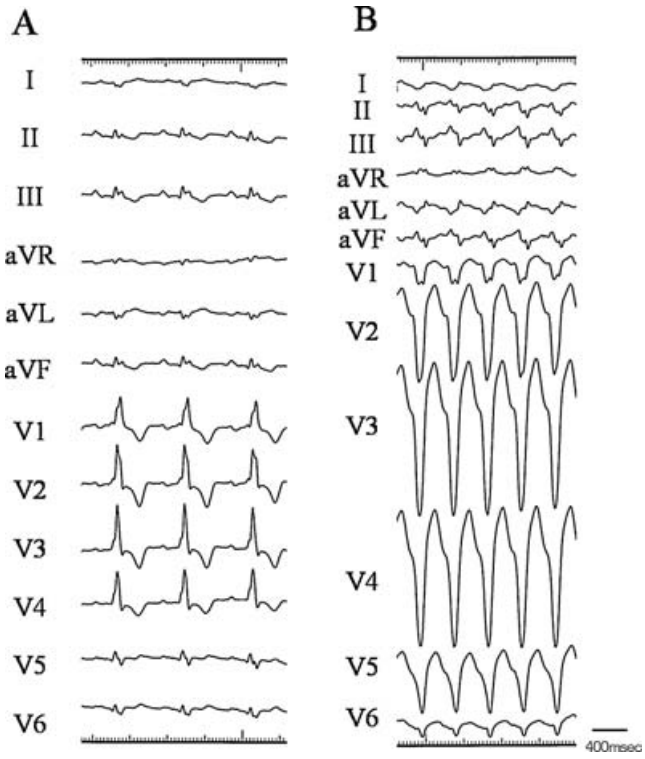

Figure 1. *A: Twelve-lead electrocardiogram during sinus rhythm. B: Clinical ventricular tachycardia at a rate of 170 beats/minute. The QRS morphology of the VT revealed a QS pattern in all the precordial leads and extreme axis deviation.*

earliest unipolar electrogram with a QS morphology recorded from the guidewire tip placed through the microtube catheter during VT preceded the QRS onset by 33 msec (Fig. 2A). No VT entrainment using intracoronary unipolar pacing was observed during the sustained VT. The site was identified to be located in segment #8 of the LAD where a good pace map was obtained, and not in the septal branch artery (Fig. 2C). An earliest electrogram with a good pace map could not be obtained (Fig. 2D) from the upper midventricular septal endocardial sites just beneath the guidewire tip, but the distance between the guidewire tip and endocardial site was anatomically very short (Fig. 3B). An RF application with a 50 W setting was carefully delivered at this endocardial site. During the delivery of the RF application, we checked for coronary artery stenosis using angiography. VT was terminated by an endocardial RF application guided by the guidewire mapping after 3.7 seconds (Fig. 4). Several additional applications were delivered. Only nonsustained VT lasting less than 5 seconds was induced by burst ventricular pacing, under an intravenous isoproterenol infusion (0.5–4 μg/minute). No significant stenosis of the LAD was detected by the coronary angiography after the RFCA. No VT was clinically observed during a follow-up period of 14 months after the RFCA.

Discussion

The key findings of the intracoronary mapping and successful RFCA in the present case were as follows: (1) The endocardial RFCA by only endocardial activation mapping failed to abolish the VT; (2) due to a previous surgery, pericardial mapping and intracardiac venous mapping could not be performed; (3) unipolar intracoronary mapping utilizing a PTCA guidewire was performed to get the epicardial EP information and the critical site was determined by (i) activation mapping by identifying the site of the earliest activation during VT, (ii) pace mapping, (iii) response to the injection of cold saline, and (iv) endocardial RFCA was finally performed by the anatomical guidance, and the successful site was close to the critical site determined by intracoronary mapping.

RFCA is a well-accepted treatment modality for VT refractory to antiarrhythmic drugs. However, the efficacy of RFCA for DCM is limited. In the majority of DCM cases, the circuit of the VT has been reported to involve the epicardial portion.[4,5] To ablate the VT, a cooled tip or irrigation ablation catheter system or epicardial approach might be required,[3] but the efficacy of those methods has not yet been established.

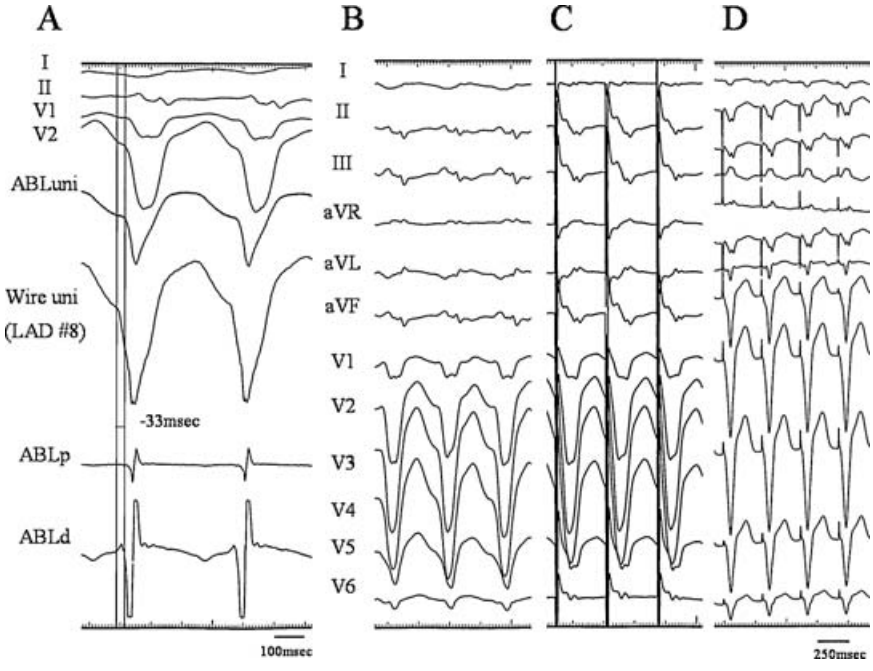

Figure 2. *The ECG recordings during an EP study are shown. A: An intracardiac electrogram recorded at the earliest activation site. During the VT, the local unipolar electrogram at segment #8 had a QS morphology, which preceded the onset of the QRS complex by 33 msec. B: Clinical ventricular tachycardia. C: A pace map using high output pacing with a near-perfect match at the site of segment #8. D: A pace map from the successful endocardial ablation site. Note that the morphology of the pace map does not match that of the clinical ventricular tachycardia.*

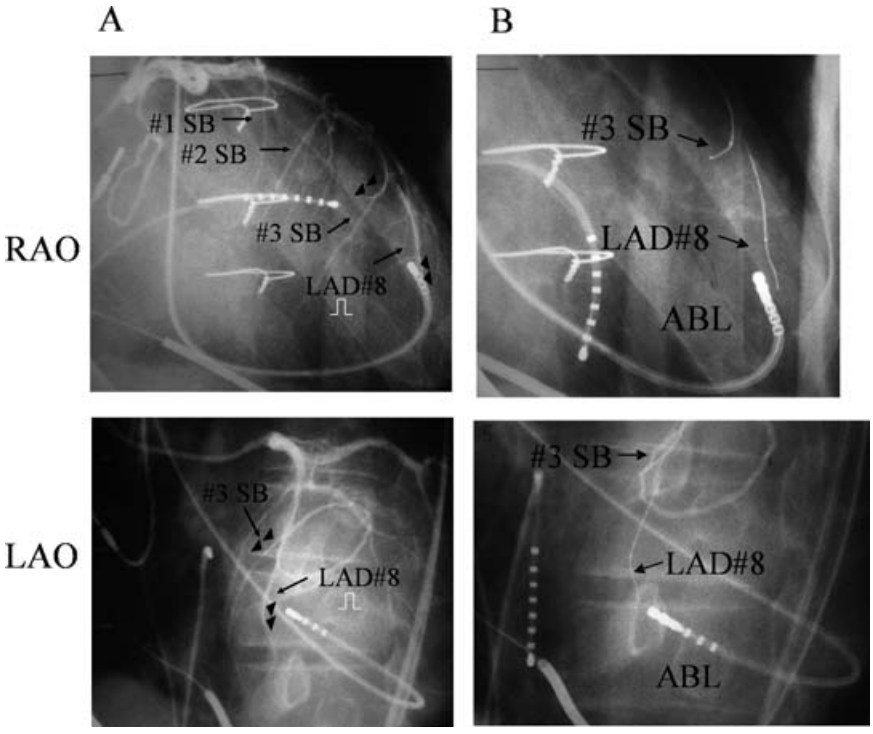

Figure 3. *A: Unipolar intracoronary mapping using a guidewire placed through a microtube catheter in the third septal branch artery and segment #8 of the LAD. The arrowheads indicate the tip of the guidwire. B: The location of the ablation catheter placed at the successful site on the endocardial surface of the upper midventricular septum nearest to segment #8 where the earliest unipolar electrogram with a good pace map was obtained. ABLp and ABLd = proximal and distal electrode recording pairs of the ablation catheter; uni = unipolar electrogram; LAD = left anterior descending artery; SB = septal branch.*

Endocardial RFCA with an 8-mm-tip catheter utilizing intracoronary mapping guidance with the tip of a PTCA guidewire through a microtube catheter was useful for abolishing VT originating from an epicardial site on the left ventricle. Pace mapping and activation sequence mapping from the endocardium was ineffective in successfully ablating the VT. For a successful ablation**,** it was useful to anatomically locate the endocardial site, which was the closest distance to the epicardial origin. In this article we report a new approach with the first use of a guidewire tip for intracoronary mapping of VT.

In this case, the mechanism of VT was nonreentry with a focal origin, which is rare in DCM. The site of the epicardial origin was located adjacent to the LAD. The delivery of the RF application from the endocardium reached the epicardial origin most adjacent to the guidewire tip. This technique might not be effective for VT with a reentrant circuit frequently observed with cardiac disease. However, when the slow conduction zone of the reentrant circuit is located in the myocardium close to a coronary artery, entrainment pace mapping might identify the reentrant circuit isthmus in order to achieve a successful ablation.

The successful ablation sites for VT observed in DCM using the epicardial approach are reported to be located adjacent to the coronary arteries and veins, irrespective of the mechanism of the VT.[6] Endocardial RFCA guided by unipolar intracoronary or intracoronary sinus vein mapping using a guidewire might be an effective therapy for some cardiac diseases with VT originating from epicardial sites located on the left ventricle. In addition, in contrast to intracardiac vein mapping, unipolar intracoronary mapping also might be a possible technique to obtain information on the ventricular septal electrophysiology through the septal branch artery. Although the importance of recording the electrograms from the great cardiac vein to the anterior interventricular vein was reported to be useful for identifying the successful catheter ablation sites of idiopathic VT originating from the epicardial aspect of the left ventricle,[7] the anterior interventricular vein usually runs close to the LAD, and the risk of a potential complication might be higher with unipolar intracoronary mapping using a PTCA guidewire than with intracardiac vein mapping. Coronary angiography might be required during or after the RFCA because the delivery of the RF application from the endocardium might injure the coronary artery or vein adjacent to the epicardial origin.[6]

Limitations

In this case it was difficult to position a microcatheter in the cardiac venous system in order to perform intracardiac vein

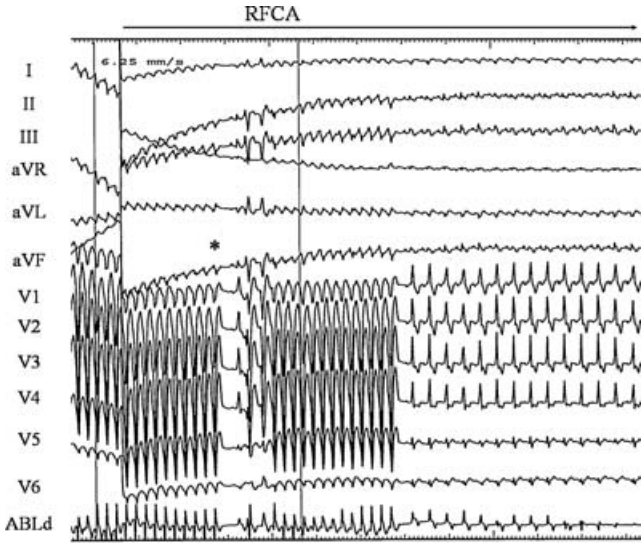

Figure 4. *Elimination of the ventricular tachycardia 3.7 seconds after initiating the delivery of the radiofrequency current.*

mapping due to the patient undergoing a previous surgery, and the pericardial approach also could not be performed because of the pericardial adhesions from the previous surgery. We would like to think that the pericardial and/or venous mapping strategy would be the first choice, with the coronary approach as the final option because ablation near a coronary artery may result in an infarction.

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