Catheter Ablation of Ventricular Tachycardia Using 3-dimensional Mapping System in Arrhythmogenic Right Ventricular Dysplasia (ARVD) Patients Refractory to Antiarrhythmic Drugs

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ABSTRACT
Ventricular tachycardia (VT) is a common complication in patients with arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D). Implantable cardioverter defibrillator (ICD) therapy can rescue VT. However, incessant VT may be associated with electrical storm. We report a patient with ARVD who presented with drug-refractory ventricular tachycardia. VT was eliminated by radiofrequency catheter ablation using a 3-dimensional mapping system.

Key words: ■ ventricular tachycardia ■ arrhythmogenic right ventricular dysplasia ■ catheter ablation

Introduction
Arrhythmogenic right ventricular dysplasia (ARVD) is a progressive, genetically determined, infiltrative myocardial disease with characteristic structural and functional abnormalities primarily involving the right ventricle (RV). At a later stage, it may also affect the left ventricle (LV). The most common affected areas are the infundibulum, apex, and the inferoposterior subtricuspid areas, collectively known as the triangle of dysplasia. The most common cause of sustained monomorphic VT associated with structural heart disease is reentry related to ventricular scars. Multiple VTs with different QRS morphologies can be caused by multiple exits from the same scar region or changes in activation remote from the circuit that are caused by functional regions of block. Peritricuspid ventricular reentry is a frequent mechanism of VT in patients with ARVD that can be identified by detailed 3-dimensional (3D) electroanatomical mapping. Electrical storm is defined as the occurrence of 3 or more episodes of sustained VT separated by 5 minutes, during a 24-h period, or the presence of incessant VT (defined as persistent, sustained VT or continuous episodes of VT separated by brief periods of normal rhythm). We performed radiofrequency catheter ablation (RF ablation) for VT in patients with ARVD using a 3D mapping system (Carto-3) to relieve frequent ICD shock.
Case

A 48-year-old man was referred to our hospital in May 2005 for palpitation associated with sustained VT that was terminated by direct current (DC) cardioversion. The patient had never been hospitalized before the event. Physical examination was normal, as were 12-lead ECG and chest X-ray. Two-dimensional echocardiography presented normal left ventricular function and right ventricular dilation and hypokinesia. The coronary angiogram was also normal. He was discharged against medical advice to undergo ICD implantation.

Cardiac magnetic resonance imaging (MRI) revealed a fibrofatty infiltration in the RV in November 2008. Electrocardiogram (Figure 1) showed epsilon wave. The patient underwent single-lead ICD implantation, with the ICD programmed for VVI pacing at a rate of 50 bpm and with antitachycardia pacing as the first option, low-energy cardioversion as the second, and high-energy cardioversion as a subsequent intervention in case of failure of the previous options. The patient, however, experienced frequent ICD shocks and hospitalization since the ICD was implanted. He was admitted to replace the ICD battery and receive a new atrial lead to better discriminate between supraventricular tachycardia (SVT) and VT in July 2011. He was admitted to receive RF ablation to eliminate VT, which causes ICD shock.

Electrocardiogram during VT (Figure 2) showed two different QRS morphologies with the same axis. A left bundle branch block-like configuration in lead V1 indicated an exit in the RV or interventricular septum. A dominant R-wave in V1 indicated an exit in the LV. According to the morphology of VT, the exit site may have been the pericristusid valve area.

Substrate mapping delineates the likely arrhythmogenic substrate during a stable sinus or paced rhythm. This method often allow the
Figure 2. Surface electrogram showed two different morphology of VTs. (A) The morphology of VT was a left bundle branch block pattern and left axis deviation. (B) The morphology of VT was a right bundle branch block pattern and left axis deviation.
identification of exits and channels without mapping during VT, facilitating ablation in patients with multiple, unstable VTs. Substrate mapping usually can be performed by 3-dimensional mapping system (Figure 3). Both endocardial mapping and epicardial mapping should be done because ARVD progresses from epicardium to endocardium. Isolated potentials after the QRS complex during sinus or paced rhythm may be an isthmus.

Two VTs were induced with programmed electrical stimulation. During slow VT induction, vital signs were stable, pace mapping and entrainment mapping (C, D in Figure 4) were done. After ablation, the patient no longer experienced ICD shock. Nonsustained VT was often noted, but it was terminated by antitachycardia pacing.

Figure 3. Contact voltage mapping during sinus rhythm showed the extensive scar area locating right ventricle. Areas with bipolar voltage >0.5 mV are represented in purple. Endocardial sinus rhythm bipolar voltage maps were shown in (A), (B) and (C) and epicardial maps were shown in (D), (E) and (F). Tricuspid valve was represented by blue dots, double potentials by white dots and mid-diastolic potentials by pink dots. The sites which could not be captured were represented by yellow dot and the sites which were perfectly matched by green dot. Ablation points were represented by red dots.
Discussion

Scar-related RV tachycardias occur in ARVD patients and reentry circuits are often adjacent to the scar. The most commonly affected areas are the infundibulum, apex, and the inferoposterior subtricuspid areas. Patients with ARVD have exhibited a good acute success rate, but the recurrence rate is >70% during follow-up exceeding 1 year, suggesting disease progression. Catheter ablation of VT plays an important role in reducing VT episodes in patients with ICDs and controlling incessant VT and electrical storms. Techniques for epicardial mapping and ablation have improved outcomes for patients with arrhythmias that are not endocardial in origin (Figure 4).

References

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Figure 4. (A) endocardial late potential, (B) epicardial late potential, and (C) slow VT are initially induced during programmed ventricular electrical stimulation. The cycle length of VT was 417 ms; the (D) post-pacing interval was 415 ms.


