

When to go epicardially during ventricular tachycardia ablation? Role of surface electrocardiogram

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Abstract: Early recognition of ventricular tachycardias (VTs) with epicardial circuits is crucial. Surface electrocardiogram (ECG) suggesting an epicardial origin could guide ablation procedures and increase success rates. A 35-year-old female patient with VT treated by combined epicardial and endocardial ablation approach is presented in this report, and the role of surface electrocardiogram and timing of epicardial access is discussed.

Keywords: ventricular tachycardia, ablation, epicardial, endocardial, surface electrocardiogram

Introduction

Radio frequency (RF) catheter ablation of ventricular tachycardia (VT) ablation is a challenging procedure with limited success rates. Presence of epicardial circuits is an important cause of these failed endocardial ablations [1]. According to data of a multicenter study, 17% of VT ablations involved epicardial mapping [2]. Therefore, characteristics of VT and underlying heart disease should be considered before deciding the ablation strategy. Twelve-lead surface electrocardiogram (ECG) during arrhythmia could be helpful to identify the origin of VT. In this report, we discuss the role of surface ECG over a case of VT treated by combined epicardial and endocardial approach.

Case Report

A 53-year-old female with nonischemic cardiomyopathy and left ventricular (LV) systolic dysfunction had undergone cardiac resynchronization therapy with implantable cardioverter defibrillator (CRT-D). Three months after the implantation, she experienced palpitations. ECG during symptoms revealed wide QRS tachycardia with a cycle length of 480 ms. CRT-D recordings revealed 84 VT episodes. Although most of them were

nonsustained, 24 of them were terminated by antitachycardia pacing (ATP). Because of frequent VT episodes and failure of antiarrhythmic therapy, she underwent electrophysiological study.

Sustained VT was induced easily by programmed ventricular stimulation. Morphology and cycle length of arrhythmia were same as clinical VT (*Fig. 1*). Right bundle branch block (RBBB)-like configuration, wide QRS duration (337 ms), and pseudo-delta wave (149 ms) were suggesting epicardial origin. Therefore, we decided to start ablation procedure by epicardial mapping. After epicardial access via subxiphoid approach, hemodynamically stable sustained clinical VT was induced. Electro-anatomical mapping system (CARTO™, Biosense Webster Inc., Diamond Bar, CA, USA) with a 7.5 French, 3.5-mm-tip, open irrigated ablation catheter (Navistar ThermoCool™, Biosense Webster Inc.) was used for activation mapping. Earliest activity was recorded at basal lateral LV. At this location, classic entrainment with concealed fusion and best post-pacing interval (480 ms) was demonstrated (*Fig. 2*). High-output pacing and coronary angiography were performed to confirm safe ablation site. During ablation (40 W, 22 mL/min external irrigation flow), morphology of the VT changed. QRS duration became narrower (190 ms), and pseudo-delta waves disappeared (*Fig. 3*). These findings were suggest-

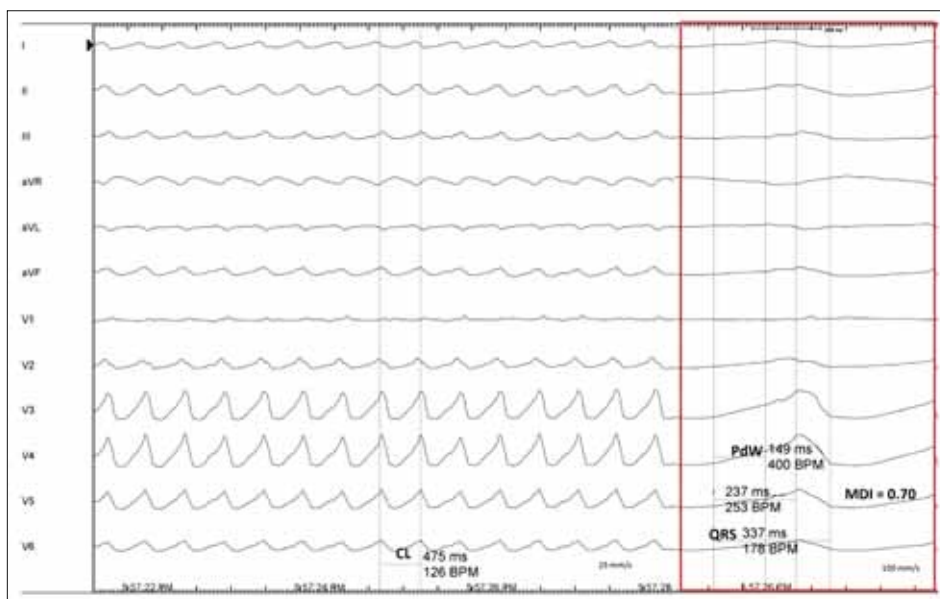


Fig. 1. Twelve-lead surface ECG of first VT (epicardial). CL: cycle length; PdW: pseudo-delta wave: interval measured from the earliest ventricular activation to the onset of the earliest fast deflection in any precordial leads; MDI: interval measured from the earliest ventricular activation to the largest amplitude deflection in each precordial leads (taking the lead with shortest time)/QRS duration

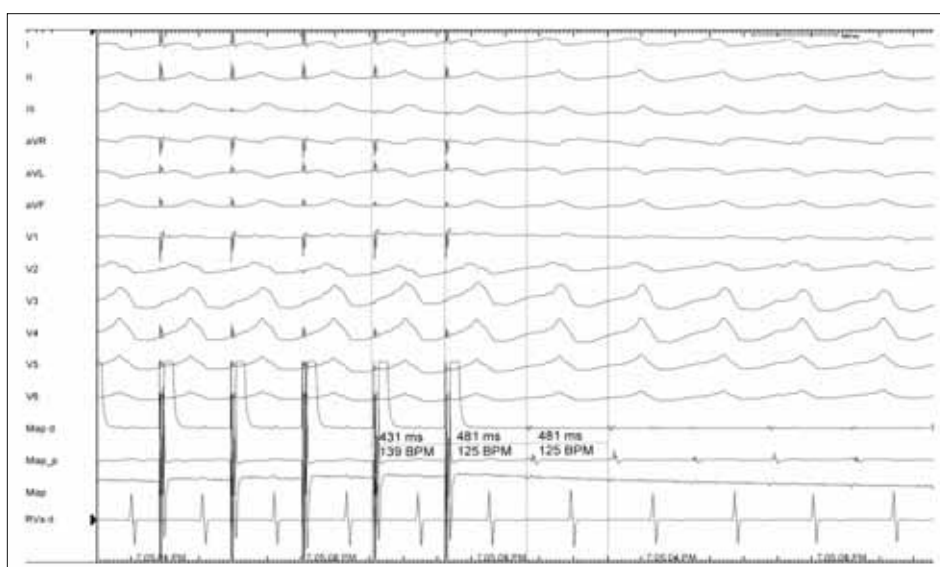


Fig. 2. Cardiac tracings presenting classic entertainment with concealed fusion and best post-pacing interval. Map: mapping catheter; Rva: right ventricular catheter

ing endocardial origin, and no early activation point was found during epicardial mapping of second VT (VT2).

Therefore, we decided to perform endocardial mapping. LV was accessed by retrograde trans-aortic approach. Endocardial activation mapping located earliest activity at basal lateral wall of LV, adjacent to successful ablation site of epicardial VT (Fig. 4). RF applications at this site terminated VT2. Thereafter, programmed ventricular stimulation up to three extra stimuli failed to induce any tachyarrhythmia. During more than 3 months

of follow-up, patient had no symptoms and monthly ICD interrogations have not recorded any ventricular arrhythmic events.

Discussion

Early recognition of epicardial origin is crucial to avoid prolonged and unsuccessful endocardial ablation. Achieving an epicardial access before systemic antico-

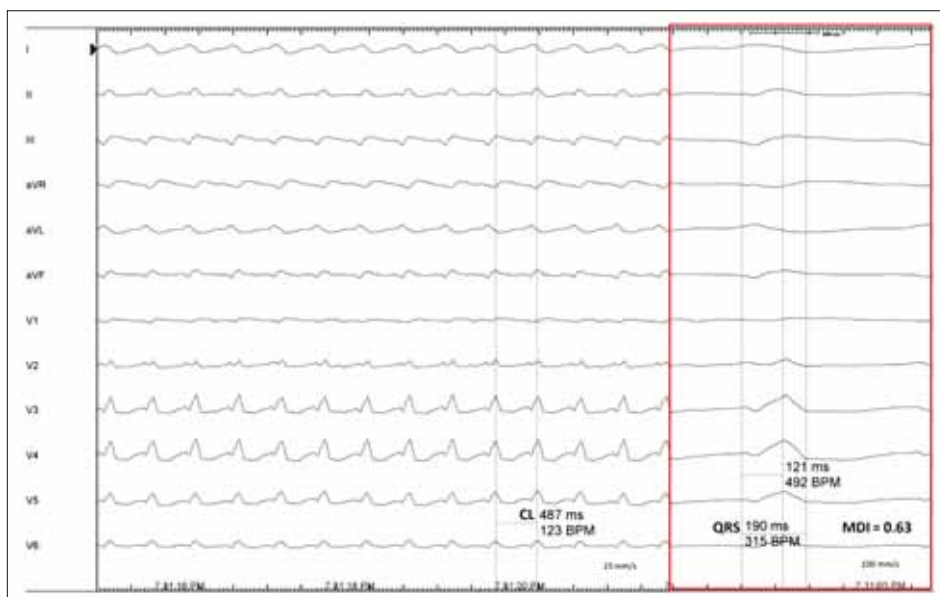


Fig. 3. Twelve-lead surface ECG of second VT (endocardial). Abbreviations are same as in the Fig. 1

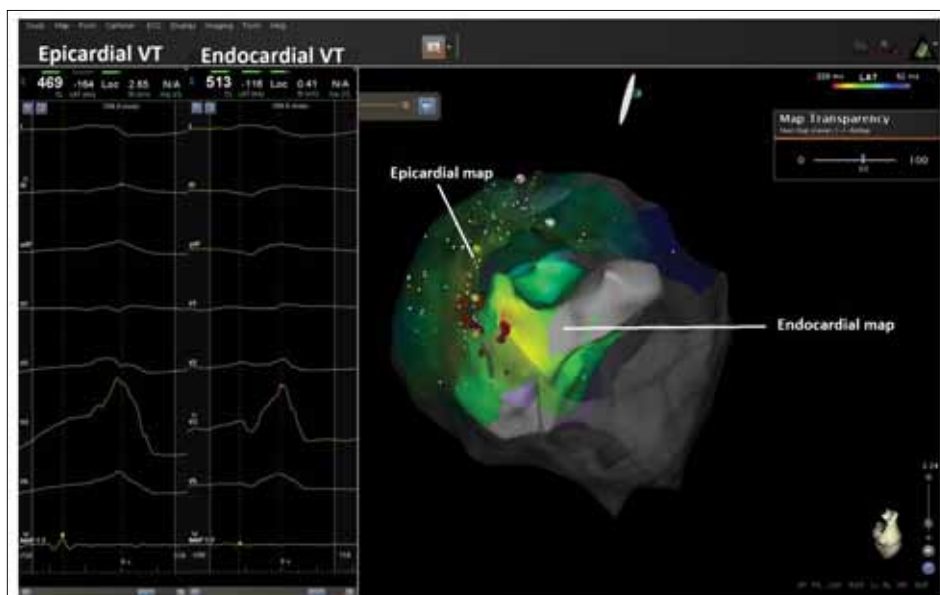


Fig. 4. Epicardial and endocardial activation maps of VTs and successful ablation sites (red dots) adjacent to each other

agulation is also safer. Epicardial approach could be an option for patients who have intra-ventricular thrombi or aortic prosthetic valve, especially epicardial VT is suspected.

Unfortunately, it is hard to define a certain ECG criteria for epicardial VT. Different cutoff values and morphology criteria were published according to patient groups with different heart diseases and normal hearts [3–6]. Furthermore, some of these studies only focused on locations where most of VTs originate. Therefore, ECG criteria suitable for underlying heart disease should be used. Vallès et al. suggested a 4-step algorithm for identifying endo/epicardial origin in patients with

nonischemic cardiomyopathy (sensitivity 96%, specificity 93%) [6]. According to this algorithm, presence of q waves in inferior leads excludes epicardial origin (step 1). Pseudo-delta wave ≥ 75 ms (step 2), maximum deflection index (MDI) ≥ 0.59 (step 3), and presence of q waves in lead I (step 4) were suggesting epicardial origin. First 3 steps had high specificity, and the last step was the most accurate. During epicardial VT, no q wave was observed neither in inferior leads nor in lead I. Pseudo-delta wave duration was 149 ms, and MDI was 0.70. Although successful ablation sites of both VTs were adjacent to each other, QRS configurations and intervals were different. There were q waves in inferior leads of

endocardial VT. Pseudo-delta waves disappeared, and MDI was 0.63. Difficulty to create transmural lesion on LV might be responsible from this morphological shifting. Therefore, combined epicardial and endocardial approach seems feasible for patients with suspected epicardial VTs. Not only ECG findings but history of patient is helpful as well. Failure of endocardial ablation could reflect an epicardial origin [7]. Prevalence of epicardial VT is higher in patients with chagas disease, nonischemic cardiomyopathy, and arrhythmogenic right ventricular cardiomyopathy. Magnetic resonance imaging may also help identifying an epicardial substrate [8].

In conclusion, surface ECG suggesting an epicardial origin could guide ablation procedures and increase success rates. However, it should be considered that morphology and interval are variable depending on substrate and location of VT.

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