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Case Report

Identifying the true origin of sustained monomorphic ventricular tachycardia associated with dilated-phase hypertrophic cardiomyopathy: A case of successful catheter ablation

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ABSTRACT

This case report describes sustained monomorphic ventricular tachycardia (VT) caused by a large epicardial scar, related to dilated-phase hypertrophic cardiomyopathy mimicking VT originating from the apical septum. VT resolved with epicardial catheter ablation. The exit of the VT circuit suggested that a 12-lead electrocardiogram can be remote with respect to the critical isthmus in this case. In patients with structural heart disease, it is difficult to identify the VT reentrant circuit by surface electrocardiography, which shows only the exit site. VT originating in the epicardium should be considered, even if the suspected origin is another ventricular site.

1. Introduction

Dilated-phase hypertrophic cardiomyopathy (DHCM) is a rare subset of hypertrophic cardiomyopathy (HCM) [1]. Data available on sustained monomorphic ventricular tachycardia (SMVT) associated with DHCM is scarce. We report a rare case of SMVT caused by a large epicardial scar related to DHCM that mimicked ventricular tachycardia (VT) originating from the apical septum. The SMVT was resolved by catheter ablation. A detailed analysis of the electrophysiological findings is presented.

2. Case report

A 49-year-old man was admitted to our hospital for SMVT treatment. He was diagnosed with DHCM at the age of 45 years in the referring hospital. His chief complaint was palpitations, and he was conscious and hemodynamically stable during the VT. A 12-lead electrocardiogram (ECG) during sinus rhythm revealed an unusual R wave progression in the precordial leads and a tall R wave in aVR (Fig. 1A, left panel). An rS pattern was observed in lead V1, and a QS pattern in leads V2–V6; northwest axis deviation in the 12-lead ECG (Fig. 1A, right panel) suggested that the VT originated from the ventricular apical septum. The VT cycle length (VTCL) was prolonged to 630 ms following intravenous amiodarone infusion. Cardiac computed tomography imaging demonstrated a thick left ventricular (LV) wall (~20 mm), and the LV ejection fraction was reduced to 40% (Fig. 1B). Delayed contrast-enhanced cardiac magnetic resonance (CMR) imaging showed an enlarged delayed-enhanced region in the apical septum and lateral wall (Fig. 1C). The VT recurred immediately following cardioversion, and amiodarone administration failed to resolve it. Therefore, we performed an electrophysiological study and catheter ablation after obtaining written informed consent. Endocardial activation mapping of the right (RV) and left ventricles during VT using an electroanatomic map (EAM) (CARTO3, Biosense Webster Inc., Diamond Bar, CA, USA) showed a focal activation pattern, wherein the site of earliest activation was located in the apical septum of both ventricles (Fig. 2A). However, the VT mechanism was considered as reentry because manifest entrainment was observed by burst pacing during VT. No diastolic potential was observed during the VT in the endocardium of either ventricle. Despite the remote location of the earliest activation sites in the RV and LV (the

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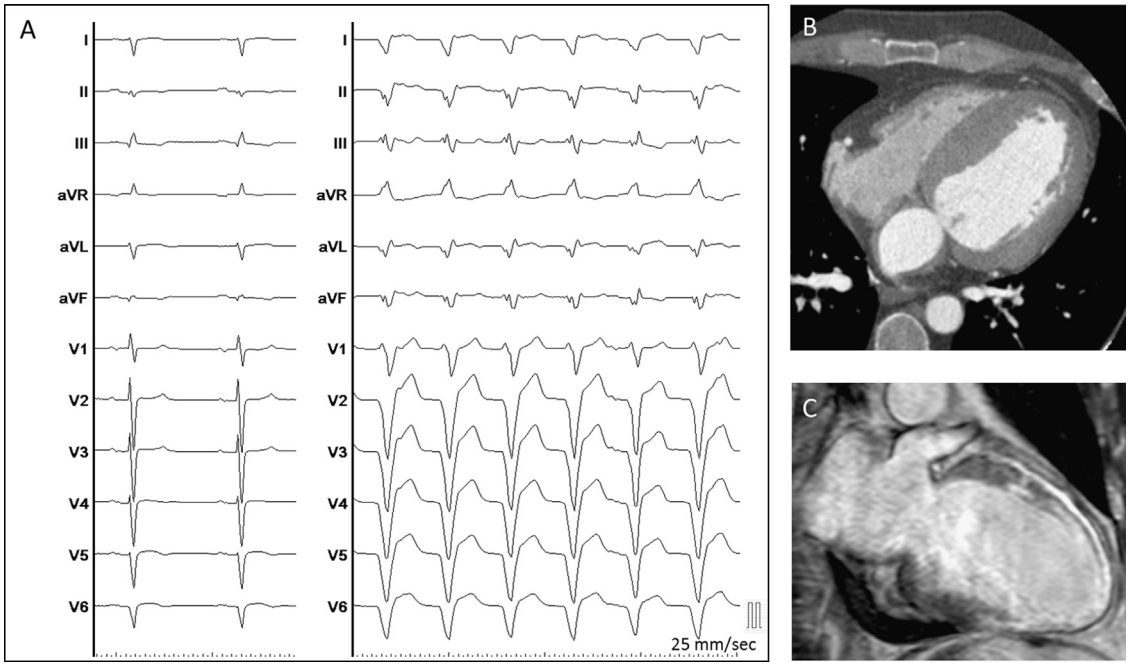

Fig. 1. (A) A 12-lead electrocardiogram during sinus rhythm (left panel) and VT (right panel). (B) Computed tomography imaging. The thickness of the LV wall is ~20 mm. (C) Cardiac magnetic resonance imaging showing delayed enhancement in the LV anterior and inferior wall.

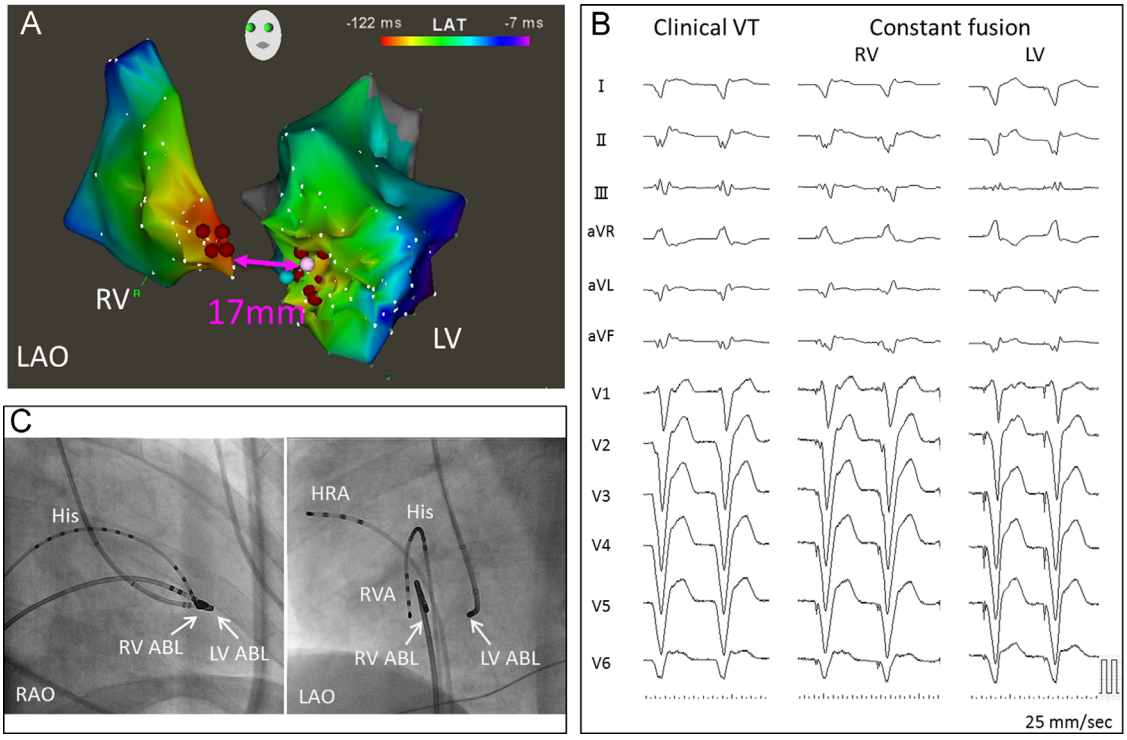

Fig. 2. (A) Activation maps of the RV and LV endocardium during VT. Note the focal activation pattern at the apical septum. The distance between the sites of earliest activation in the RV and LV is 17 mm. (B) Comparison of QRS morphologies during clinical VT and during pacing from the earliest activation sites in both ventricles during entrainment maneuver. The three QRS morphologies are almost identical. (C) Two ablation catheters in the RV and LV for bipolar ablation.

distance measured on the EAM was 17 mm), QRS morphologies during pacing from the earliest activation sites were almost identical to those observed during VT (Fig. 2B). Furthermore, the postpacing intervals (PPI) minus the VTCL following entrainment pacing from the RV and LV were 30 ms and 28 ms, respectively. Radiofrequency (RF) energy delivered (Navistar ThermoCool, Biosense Webster Inc.) at the earliest RV site did not affect the VT, whereas RF energy delivered at the earliest LV site resulted in temporary VT termination (maximum power of 50 W). However, VT recurred a few minutes after removing the RF energy delivery. We suspected that the VT circuit was located in a deep layer of the ventricular septum. Therefore, bipolar ablation between the RV and LV was attempted. Two irrigated catheters (LV: Navistar ThermoCool, RV: Cool Path Duo, St. Jude Medical) were placed at the contralateral site of the earliest activation in each ventricle (Fig. 2C), and RF energy was delivered between the two catheters (maximum power of 35 W). However, bipolar ablation did not completely resolve the VT. As a result, we hypothesized that the