Journal of Arrhythmia 31 (2015) 406–409

Contents lists available at ScienceDirect

Journal of Arrhythmia

journal homepage: www.elsevier.com/locate/joa

Case Report

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

Hiroshi Kawakami, MD[a], Takayuki Nagai, MD, PhD[a,*], Akira Fujii, MD[a], Teruyoshi Uetani, MD, PhD[a], Kazuhisa Nishimura, MD, PhD[a], Katsuji Inoue, MD, PhD[a], Jun Suzuki, MD, PhD[a], Kazuhiro Satomi, MD, PhD[b], Takafumi Okura, MD, PhD[a], Jitsuo Higaki, MD, PhD[a], Akiyoshi Ogimoto, MD, PhD[a]

[a] Division of Cardiology, Department of Cardiology, Pulmonology, Hypertension and Nephrology, Ehime University Graduate School of Medicine, Shitsukawa, Toon, Ehime 791-0295, Japan
[b] Department of Cardiology, Tokyo Medical University Hachioji Medical Center, Tokyo, Japan

ARTICLE INFO

Article history:
Received 5 April 2015
Received in revised form
27 May 2015
Accepted 8 June 2015
Available online 14 July 2015

Keywords:
Bipolar ablation
Epicardial ablation
Dilated-phase hypertrophic cardiomyopathy
Ventricular tachycardia

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

* Corresponding author. Tel.: +81 89 960 5303; fax: +81 89 960 5306.
E-mail address: nagait@m.ehime-u.ac.jp (T. Nagai).

http://dx.doi.org/10.1016/j.joa.2015.06.003
1880-4276/

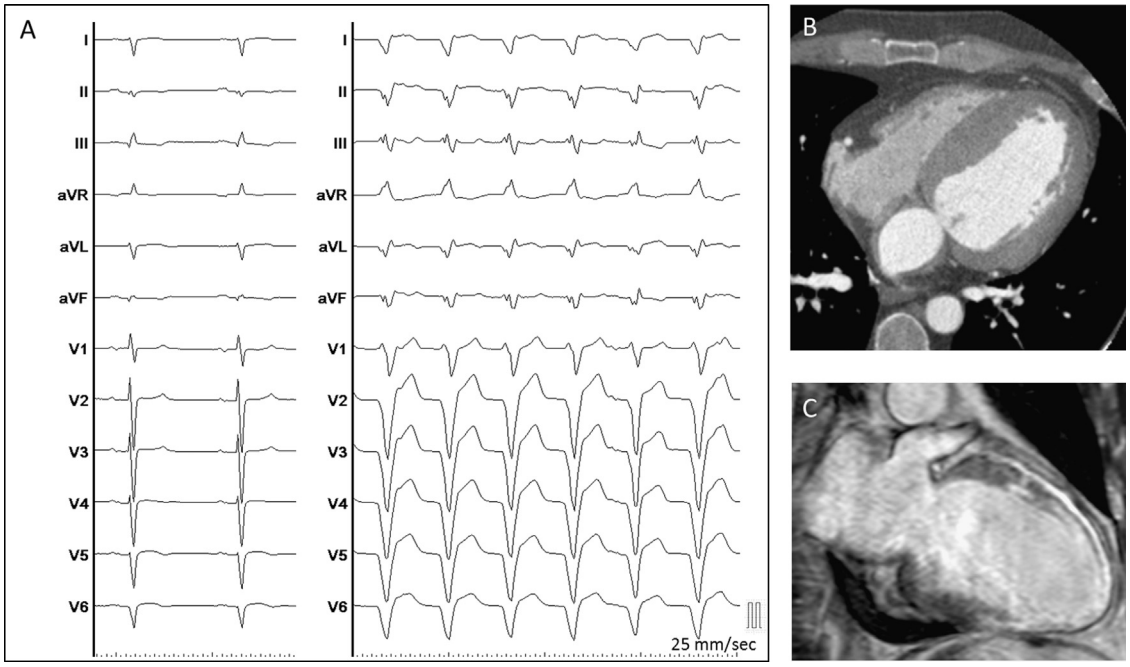

Fig. 1. (A) A 12-lead electrocardiogram during sinus rhythm (left panel) and VT (right panel). (B) Computed tomography imaging. The thickness of the LV wall is ~20 mm. (C) Cardiac magnetic resonance imaging showing delayed enhancement in the LV anterior and inferior wall.

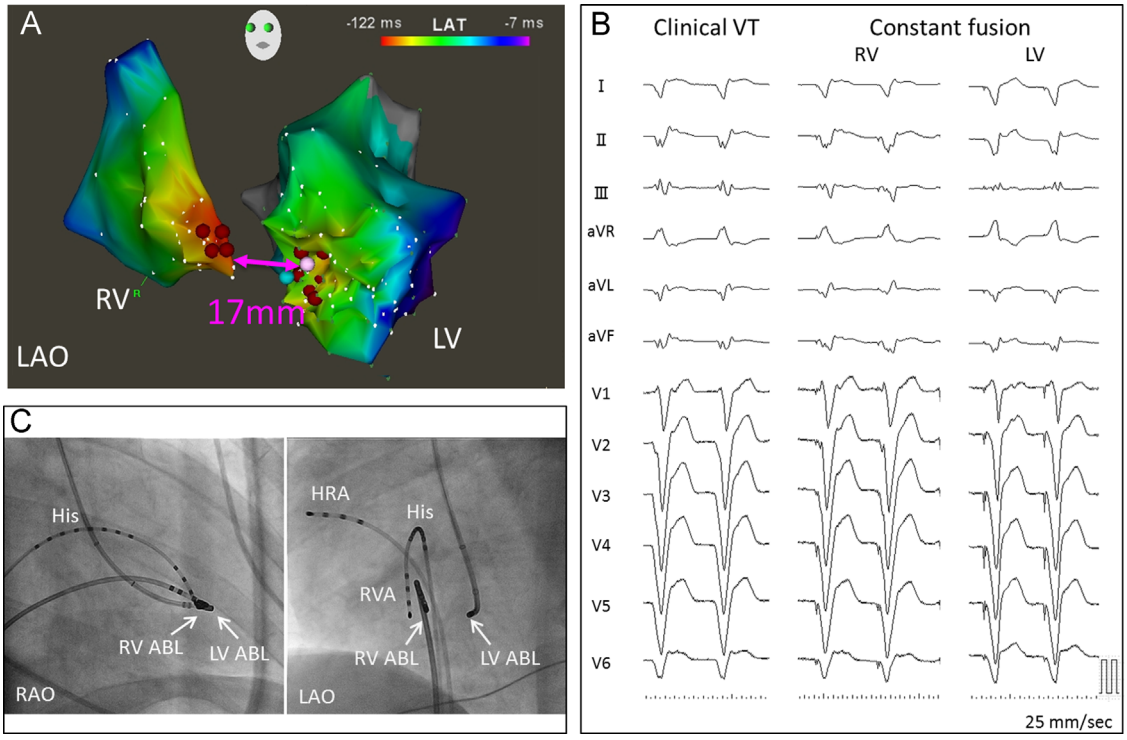

Fig. 2. (A) Activation maps of the RV and LV endocardium during VT. Note the focal activation pattern at the apical septum. The distance between the sites of earliest activation in the RV and LV is 17 mm. (B) Comparison of QRS morphologies during clinical VT and during pacing from the earliest activation 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

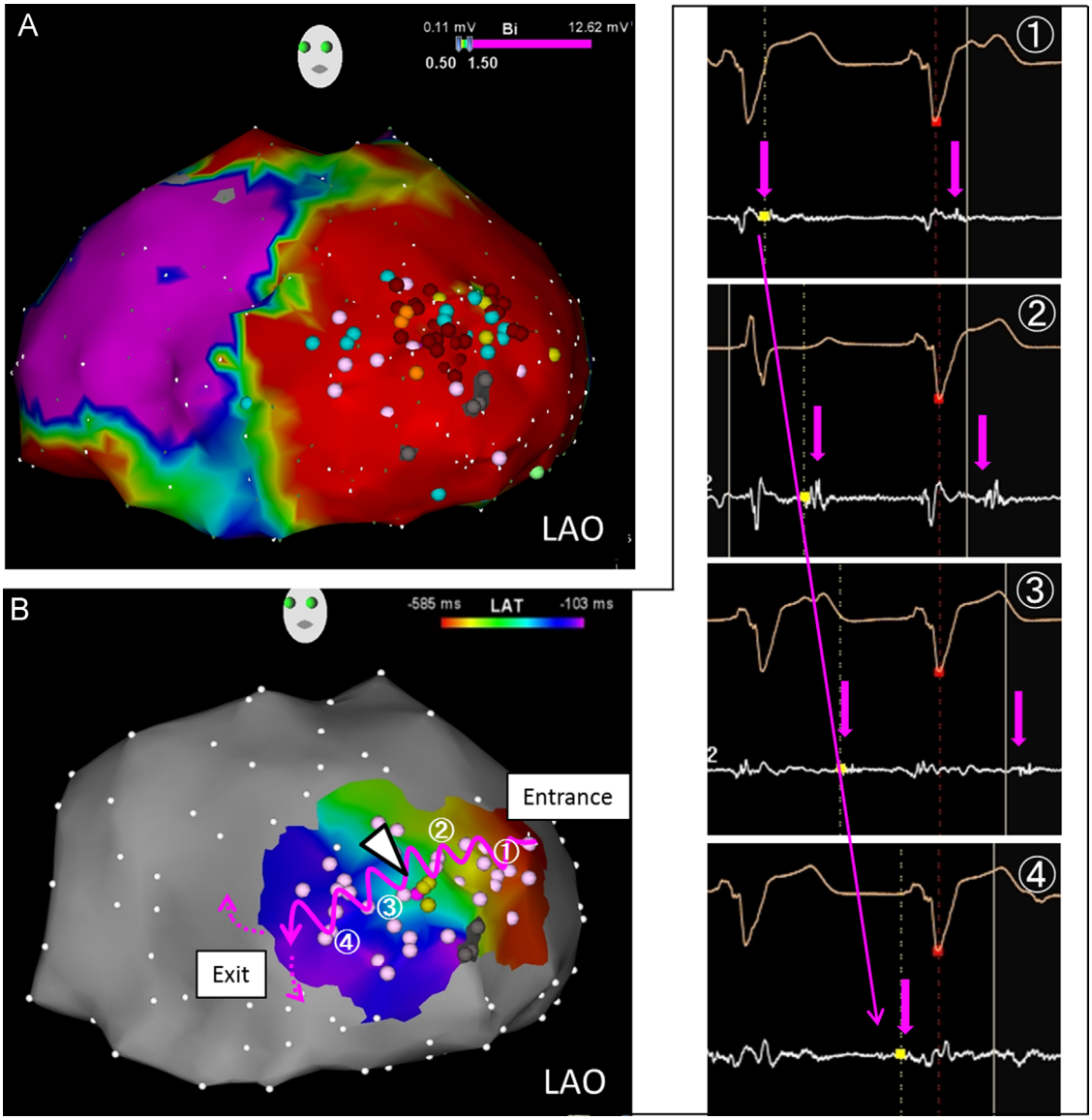

Fig. 3. (A) Voltage map of the epicardium based on electroanatomic mapping during sinus rhythm. A wide low-voltage area (< 1.5 mV) was identified in the LV epicardium. Isolated delayed potentials were recorded in this area during sinus rhythm. (B) Activation map of diastolic potentials constructed during VT. Diastolic potentials were recorded continuously (right panel (1)–(4)). Note that the critical isthmus of the VT is very clear (left panel).

main VT circuit may be located in the epicardium, and performed epicardial voltage mapping using a subxiphoid puncture. A wide low-voltage area (< 1.5 mV) was identified on the lateral epicardial wall during sinus rhythm (Fig. 3A), and diastolic potentials were recorded during the VT in this area. We performed detailed mapping of the diastolic potentials using EAM, and the critical isthmus was revealed on the anterolateral epicardial wall (Fig. 3B). At the center of the critical isthmus (Figs. 3B arrowhead, and 4A), concealed entrainment was observed (PPI = VTCL, stim-QRS interval = egm-QRS interval) (Fig. 4B). The initial RF energy (maximum power of 35 W) was delivered at this site, which completely resolved the VT after 21 s (Fig. 4C). We added bonus applications at an adjacent site. Subsequently, VT could not be induced by programmed electrical stimuli. No complications were observed following the procedure, and the patient was discharged 5 days after catheter ablation.

3. Discussion

This report describes successful SMVT ablation in a patient with DHCM. Previous studies have described successful ablation of SMVT in patients with nondilated-phase HCM [2–5]. However, there are few reports describing SMVT associated with DHCM. Ueda et al. reported clinical and electrophysiological characteristics in patients with SMVT associated with DHCM [6]. The authors state that the SMVT circuits in DHCM patients are more likely to be located either at the basal septum or at the basal anterior to the anterolateral LV. However, in the present case, the VT was suspected to originate from the ventricular apical septum, based on surface ECG morphology. In addition, the QRS morphologies during entrainment pacing were almost identical for both ventricles, despite the long distance between the pacing sites. These findings suggest that both pacing points were close to the exit of the VT circuit. Thus, the reentry circuit was suspected to be located in the deep layer of the apical septum. However, we were not able to eliminate the circuits by the ablation of the earliest site of both ventricles or by bipolar ablation. Finally, epicardial ablation revealed that the critical isthmus spanned from the anterolateral

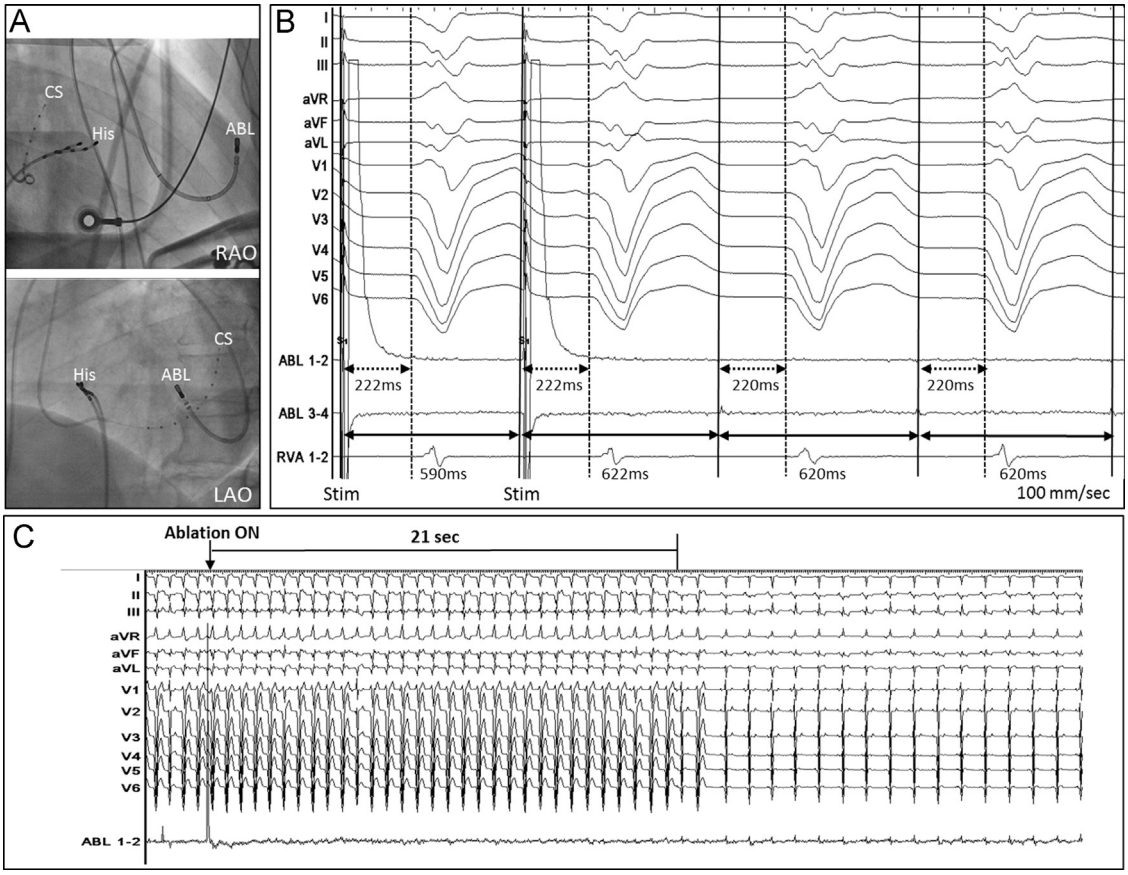

Fig. 4. (A) Fluoroscopic images showing catheter positions at the successful ablation site on the epicardium during VT. This site is at the center of the critical isthmus of the VT circuit (arrowhead in Fig. 3B). (B) Concealed entrainment was observed (PPI=VTCL, stim-QRS interval=egm-QRS interval). (C) VT was terminated in 21 s and subsequently could not be induced.

wall to the exit site, which was also confirmed by entrainment. The location of the critical isthmus was consistent with the site described in a previous study [6]. A major concern in this case was the long distance between the suspected exit based on the QRS morphology in the 12-lead ECG and the critical isthmus of the SMVT. This highlights the limitation of surface ECG, which only shows the exit site, in terms of identifying the reentrant circuit. Previous studies reported intramural or epicardial reentry circuits in some patients with HCM [2,3,6]. The extent of fibrosis is one of the important factors in an arrhythmic substrate, and delayed contrast-enhanced CMR provides information regarding the extent and distribution of fibrosis [7,8]. The extent of fibrosis may be significantly greater in DHCM patients than in individuals with nondilated-phase HCM. In the present case, CMR showed diffuse delayed enhancement in the LV septum and apical and lateral wall. The surface ECG morphology during sinus rhythm was unusual because of a large extent of DHCM-associated fibrosis. The VT circuit may have been surrounded by extensive scar tissue. Therefore, the 12-lead ECG in this case suggested that the exit of the VT circuit was located at a large distance from the critical isthmus. These arrhythmogenic lesions tend to be epicardial or deeply intramural rather than endocardial, which may explain the difficulty related to ablation in patients with DHCM. The 12-lead ECG is useful for predicting the origin of tachyarrhythmia [9]; however, its accuracy is low in patients with structural heart diseases [10]. The possibility of VT originating in the epicardium should be considered, even if its suspected origin is another ventricular site.

Conflict of interest

The authors declare no conflict of interest related to this study.

References

[1] Maron BJ. Hypertrophic cardiomyopathy: a systematic review. J Am Med Assoc 2002;287:1308–20.
[2] Santangeli P, Di Biase L, Lakkireddy D, et al. Radiofrequency catheter ablation of ventricular arrhythmias in patients with hypertrophic cardiomyopathy: safety and feasibility. Heart Rhythm 2010;7:1036–42.
[3] Dukkipati SR, d'Avila A, Soejima K, et al. Long-term outcomes of combined epicardial and endocardial ablation of monomorphic ventricular tachycardia related to hypertrophic cardiomyopathy. Circ Arrhythm Electrophysiol 2011;4:185–94.
[4] Inada K, Seiler J, Roberts-Thomson KC, et al. Substrate characterization and catheter ablation for monomorphic ventricular tachycardia in patients with apical hypertrophic cardiomyopathy. J Cardiovasc Electrophysiol 2011;22:41–8.
[5] Wong KC, Qureshi N, Jones M, et al. Epicardial ablation of monomorphic ventricular tachycardia in a case of hypertrophic cardiomyopathy with apical aneurysm. Europace 2013;2:296.
[6] Ueda A, Fukamizu S, Soejima K, et al. Clinical and electrophysiological characteristics in patients with sustained monomorphic reentrant ventricular tachycardia associated with dilated-phase hypertrophic cardiomyopathy. Europace 2012;14:734–40.
[7] Rickers C, Wilke NM, Jerosch-Herold M, et al. Utility of cardiac magnetic resonance imaging in the diagnosis of hypertrophic cardiomyopathy. Circulation 2005;112:855–61.
[8] Leonardi S, Raineri C, DeFerrari GM, et al. Usefulness of cardiac magnetic resonance in assessing the risk of ventricular arrhythmias and sudden death in patients with hypertrophic cardiomyopathy. Eur Heart J 2009;30:2003–10.
[9] Waxman HL, Josephson ME. Ventricular activation during endocardial pacing: I. Electrocardiographic patterns related to the site of pacing. Am J Cardiol 1982;50:1–10.