Clinical Case Reports

Open Access

CASE REPORT

Endocardial and epicardial focal activation pattern due to microreentry ventricular tachycardia in a patient with cardiac sarcoidosis

Hiroshi Kawakami, Ikutaro Nakajima, Mitsuru Wada, Kazuhiro Satomi & Kengo Kusano

Division of Arrhythmia and Electrophysiology, Department of Cardiovascular Medicine, National Cerebral and Cardiovascular Center, Suita, Japan

Correspondence
Ikutaro Nakajima, Division of Arrhythmia and Electrophysiology, Department of Cardiovascular Medicine, National Cerebral and Cardiovascular Center, 5-7-1 Fujishiro-dai, Suita, Osaka, 565-8565 Japan.
Tel: +81-6-6833-5012; Fax: +81-6-6872-7486;
E-mail: ikutaro0817@icloud.com

Funding Information
No sources of funding were declared for this study.

Received: 11 September 2016; Revised: 11 March 2017; Accepted: 14 March 2017

Clinical Case Reports **2017; 5(6): 829–832**

doi: 10.1002/ccr3.947

Key Clinical Message

Majority of ventricular tachycardias (VTs) are caused by scar-related macroreentry in patients with cardiac sarcoidosis. These VTs usually exhibit the reentry circuit on electroanatomic maps. We here describe a rare case of VT that exhibits a focal activation pattern on the electroanatomic map despite reentrant VT.

Keywords

Cardiac sarcoidosis, epicardial ablation, focal activation pattern, microreentry, ventricular tachycardia.

Introduction

Lethal ventricular tachycardias (VTs), particularly sustained monomorphic VT, are among the most important clinical manifestations in patients with reduced systolic function due to cardiac sarcoidosis. Some recent studies have reported that radio frequency catheter ablation (RFCA), by both endocardial and epicardial approaches, is an effective treatment for VT [1, 2]. According on these reports, the majority of VTs originating from the epicardium are caused by scar-related macroreentry. These VTs usually exhibit the reentry circuit on electroanatomic maps. We, herein, describe a rare case of VT that exhibits a focal activation pattern on endocardial and epicardial surfaces despite the reentrant mechanism in a patient with cardiac sarcoidosis.

Case Presentation

A 66-year-old female with cardiac sarcoidosis was admitted to our hospital due to VT storm. A 12-lead electrocardiography during atrial pacing rhythm and VT is shown in Figure 1. The left ventricle (LV) was suspected as the origin of VT based on the presence of a QRS complex with a right bundle branch block-type morphology and inferior axis deviation. Although she was prescribed antiarrhythmic drugs, including amiodarone, and an implantable cardioverter–defibrillator (ICD) was previously implanted at another hospital, she developed a VT storm that resulted in multiple ICD shocks. Her transthoracic echocardiogram demonstrated a reduced LV ejection fraction of 30% and dual ventricular aneurysms on the LV lateral wall and LV apex. We performed an electrophysiological study and RFCA the day after her admission. Ventricular mapping and ablation were performed with a 3.5-mm ThermoCool open-irrigation tip catheter (Biosense Webster, Inc., Diamond Bar, CA) using an electroanatomic map (CARTO3; Biosense Webster, Inc.). First, endocardial voltage mapping during sinus rhythm or atrial pacing rhythm was performed, which revealed low-voltage (bipolar voltage <1.5 mV) areas in both LV aneurysms (Fig. 2A). Clinical VT (cycle length was 430 msec) was reproducibly induced

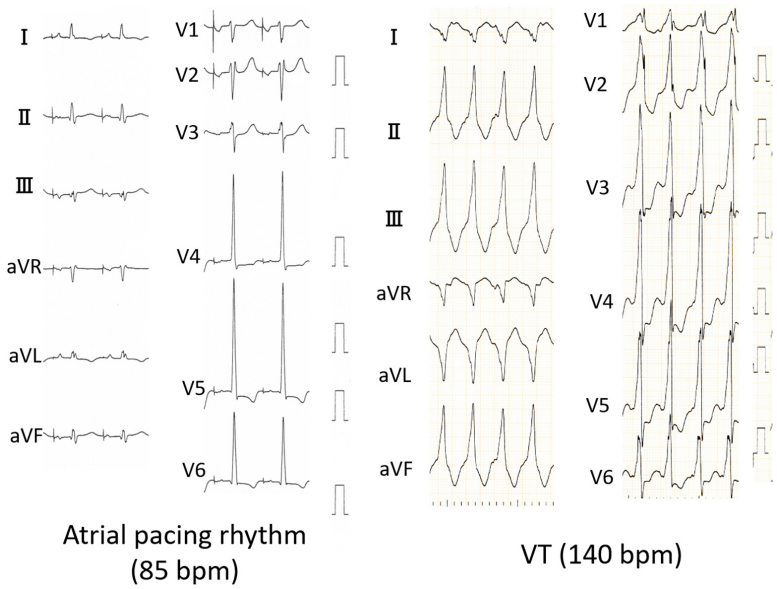

Figure 1. Twelve-lead electrocardiography during pacemaker rhythm (atrial pacing) and ventricular tachycardia.

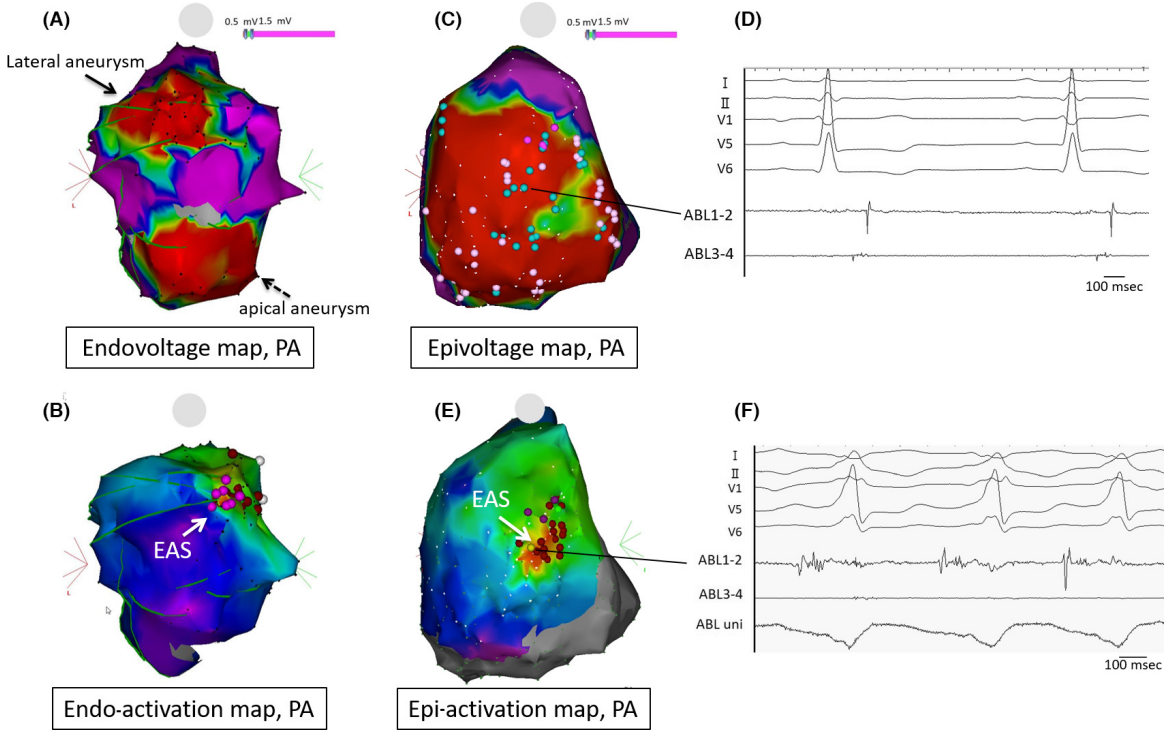

Figure 2. (A, C) Voltage maps of the left ventricular (LV) endocardium (A) and epicardium (C) during sinus rhythm. The black arrow shows the LV lateral aneurysm, and the dotted arrow shows the LV apical aneurysm. Low-voltage (<1.5 mV) areas on the endocardium were consistently found in the aneurysms on the lateral wall and apex of the LV (A). In contrast to the low-voltage area on the endocardium, a wider low-voltage area was found on the epicardium (C). (B, E) Activation maps of the LV endocardium (B) and epicardium (E) during a clinical ventricular tachycardia (VT). Note the focal activation pattern at the endocardial and epicardial site. The white arrow shows the earliest activation site (EAS), and the yellow dot shows the ablation site that successfully terminated the VT. (D, F) Local potential at the earliest activation site on the epicardium. Isolated delayed potentials were obtained around the successful ablation site during sinus rhythm (D). The local potential at the epicardial earliest activation site during VT (F). ABL1-2 demonstrates very fractionated potentials and continuous potentials during the diastolic phase. ABL, ablation catheter; LAO, left anterior oblique; PA, posteroanterior.

by programmed stimulation (burst and extrastimuli) from the right ventricular apex, which was hemodynamically tolerated. The endocardial activation map during the VT suggested a focal activation pattern and that the site of the earliest activation was in the LV lateral aneurysm (Fig. 2B). However, the VT mechanism was considered reentry because manifest entrainment was observed with burst pacing during the VT. The delivery of radio frequency (RF) energy (maximum power, 50 W) at the earliest endocardial activation site was ineffective in terminating the VT. As the critical circuit of VT was thought to be in the epicardium, electroanatomic mapping of the epicardium was subsequently performed. The pericardial space was accessed via a standard percutaneous subxiphoid puncture, as previously described [3]. A wider low-voltage area was found in the epicardium than in the endocardium (Fig. 2C), and isolated delayed potentials were recorded (Fig. 2D). Clinical VT was induced again, followed by activation mapping of the epicardium (VT cycle length was slightly prolonged to 530–540 msec), which exhibited a focal activation pattern too (Fig. 2E). The earliest activation site was located on the surface of the LV lateral aneurysm and was thought to be opposite of the earliest endocardial activation site. At that site, remarkable fragmented and mid-diastolic potentials were recorded during the VT, and the duration of fragmented potentials accounted for >50% of the VT cycle length (Fig. 2F). Entrainment pacing revealed concealed fusion, and the postpacing interval (PPI) was equal to the VT cycle length (Fig. 3A). VT terminated just after delivery of the RF energy (30 W) (Fig. 3B). Additional RF energy was applied near that site. Finally, no further ventricular arrhythmias could be induced by programmed electrical stimulation with isoproterenol infusion. During a follow-up of 6 months, a second ablation was needed for another macroreentrant VT related to the apical aneurysm. However, there was no recurrence of the present VT.

Discussion

Most VTs in patients with nonischemic heart disease, including cardiac sarcoidosis, occur via macroreentry mechanisms associated with scaring of the endocardium

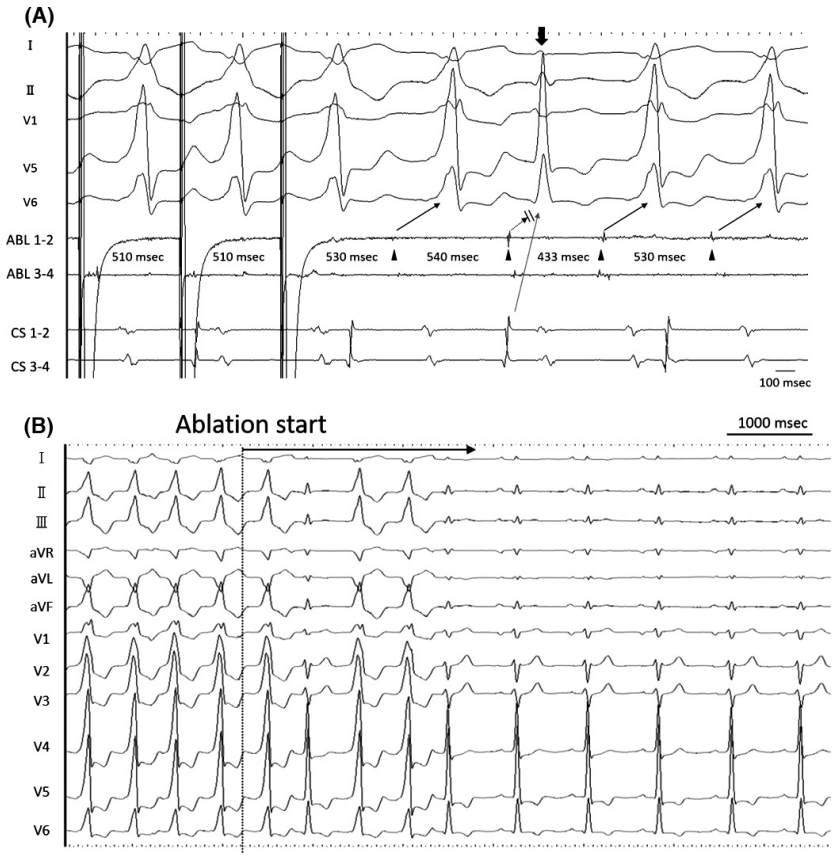

Figure 3. (A) Entrainment pacing revealed concealed fusion, and the postpacing interval (PPI) was equal to the VT cycle length. Arrow heads indicated mid-diastolic potentials. The fusion of normal atrioventricular conduction beat and VT was recorded accidentally (large arrow). In addition, mid-diastolic potential was reset by the sinus rhythm beat. (B) VT was eliminated just after delivery of radio frequency energy at the epicardial earliest activation site (yellow dot in Fig. 2F). ABL, ablation catheter; CS, coronary sinus.

or epicardium [4, 5]. Activation maps often exhibit large reentry circuits around the scar areas in these VTs. On the other hand, scar-related VT exhibiting focal activation pattern is rare, and there have been no previous studies that have investigated the detailed mechanisms underlying focal activation pattern in patients with cardiac sarcoidosis. In the present case, activation mapping during VT exhibited a focal activation pattern on the endocardial and epicardial surface, and the site of the earliest activation was located in the ventricular aneurysm on the left ventricular lateral wall (Fig. 3B and E). Although the activation map during VT suggested a focal VT, such as Purkinje-related tachycardia caused by focal mechanism including abnormal automaticity, the focal mechanism was considered unlikely because the essential substrate was located on the epicardium and there were no preceding Purkinje potentials during sinus rhythm and VT at the successful RFCA site. Furthermore, constant and progressive fusion was observed, and findings of resetting VT were recorded (Fig. 3A). Therefore, we diagnosed the mechanism of the clinical VT was reentry despite focal activation pattern. The most interesting point is the reason why VT exhibits a focal activation pattern despite reentrant VT. A remarkable delayed conduction was confirmed at the site of the earliest activation on the epicardium. This finding might suggest that a very strong conduction delay existed in a local area. The duration of this delayed conduction accounted for more than half of VT cycle length at the site. We suspected the substrate for this VT might exist in a very small area of the epicardium and intramural myocardium. Therefore, VT exhibits a focal activation pattern on the endocardial and epicardial surfaces despite reentrant VT.

Conclusion

We here describe a rare case of VT exhibiting a focal activation pattern on the endocardial and epicardial surfaces despite reentrant VT in a patient with cardiac sarcoidosis. The circuit existed in a very local area with a pronounced conduction delay around the circuit, which was considered to be the cause of the observed focal pattern.

Authorship

HK: wrote the first draft of the manuscript. All authors participated in the concept and design and in drafting and critically revising the manuscript. All authors approved the manuscript as submitted.

Conflict of Interest

None declared.

References

1. Koplan, B. A., K. Soejima, K. Baughman, L. M. Epstein, and W. G. Stevenson. 2006. Refractory ventricular tachycardia secondary to cardiac sarcoid: electrophysiologic characteristics, mapping, and ablation. Heart Rhythm 3:924–929.
2. Jefic, D., B. Joel, E. Good, F. Morady, H. Rosman, B. Knight, et al. 2009. Role of radiofrequency catheter ablation of ventricular tachycardia in cardiac sarcoidosis: report from a multicenter registry. Heart Rhythm 6:189–195.
3. Lim, H. S., F. Sacher, H. Cochet, B. Berte, S. Yamashita, S. Mahida, et al. 2014. Safety and prevention of complications during percutaneous epicardial access for the ablation of cardiac arrhythmias. Heart Rhythm 11:1658–1665.
4. Soejima, K., W. G. Stevenson, J. L. Sapp, A. P. Selwyn, G. Couper, and L. M. Epstein. 2004. Endocardial and epicardial radiofrequency ablation of ventricular tachycardia associated with dilated cardiomyopathy: the importance of low-voltage scars. J. Am. Coll. Cardiol. 43:1834–1842.
5. Wissner, E., W. G. Stevenson, and K. H. Kuck. 2012. Catheter ablation of ventricular tachycardia in ischaemic and non-ischaemic cardiomyopathy: where are we today? A clinical review Eur. Heart J. 33:1440–1450.