



Contents lists available at ScienceDirect

## Indian Pacing and Electrophysiology Journal

journal homepage: [www.elsevier.com/locate/IPEJ](http://www.elsevier.com/locate/IPEJ)

## Ventricular tachycardia based on cardiac sarcoidosis with a narrow QRS complex, ablated on the left ventricle free-wall

Hiroshi Imada <sup>a</sup>, Koji Fukuzawa <sup>b, \*</sup>, Yu Izawa <sup>a</sup>, Kunihiro Kiuchi <sup>b</sup>, Ken-ichi Hirata <sup>a, b</sup><sup>a</sup> Division of Cardiovascular Medicine, Department of Internal Medicine, Kobe University Graduate School of Medicine, Kobe, Japan<sup>b</sup> Section of Arrhythmia, Division of Cardiovascular Medicine, Department of Internal Medicine, Kobe University Graduate School of Medicine, Kobe, Japan

## ARTICLE INFO

## Article history:

Received 1 March 2021

Received in revised form

11 May 2021

Accepted 28 May 2021

Available online 3 June 2021

## Keywords:

Ventricular tachycardia

Catheter ablation

Cardiac sarcoidosis

Narrow QRS complex

## ABSTRACT

A septuagenarian female with cardiac sarcoidosis suffered from drug refractory ventricular tachycardia (VT) requiring multiple implantable cardioverter-defibrillator shocks. The QRS complex during the VT was very similar to that during sinus rhythm although the QRS width during the VT (142 ms) was relatively wider than that during sinus rhythm (107 ms). The VT exit was located on the ventricular septum close to the His-bundle recording region. However, the critical pathway of this VT was detected on the anterior free wall of the left ventricle (LV), and a radiofrequency application at that site could terminate the VT. No Purkinje potentials were recorded there during the VT or sinus rhythm. According to the electrophysiological study, 3-D mapping, and the response to the ablation, the critical circuit of the VT was surrounded by a protected area of scar associated with cardiac sarcoidosis. As a result, the VT circuit was connected to the basal septal area close to the His-Purkinje system as an outer loop of the VT circuit. This unique trajectory of the VT might have caused a similar QRS morphology to that of sinus rhythm, and the relatively narrow QRS complex despite the critical isthmus was located on the anterior free wall of the LV.

Copyright © 2021, Indian Heart Rhythm Society. Production and hosting by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

## 1. Introduction

Ventricular arrhythmias are one of the serious problems associated with cardiac sarcoidosis [1]. Generally, ventricular tachycardia (VT) associated with the His-Purkinje system can exhibit a narrow QRS complex [2]. Purkinje-related VT is usually a bundle branch reentry VT, intra-fascicular reentry VT, or focal automaticity from the His-Purkinje system. The important electrophysiological feature of Purkinje-related VT is a Purkinje potential followed by the VT-QRS onset [3].

Here, we report a case of VT with a narrow QRS complex, which exhibited a unique electrophysiological feature; the earliest site was on the ventricular septum near the His-bundle (HB), however, the successful ablation site was on the left ventricular (LV) free wall where no Purkinje potentials were observed.

## 1.1. Case report

A septuagenarian female with cardiac sarcoidosis was referred for catheter ablation of an electrical storm involving a monomorphic VT refractory to amiodarone. Anti-tachycardia pacing from an implantable cardioverter-defibrillator (ICD) failed to terminate her VT, so ICD shocks were sometimes delivered while she was conscious.

The twelve-lead ECG during sinus rhythm and the VT are presented in Fig. 1. The VT cycle length (VTCL) was 447 ms (134bpm), and the QRS duration was 142 ms (Fig. 1B). Other than the QRS width, the QRS morphology and axis of the VT were very similar to that during sinus rhythm (Fig. 1A/B).

## 1.2. The 1st and 2nd sessions

Each electrophysiological study (EPS) was performed by using a CARTO3 system (Biosense Webster Inc., Diamond Bar, CA, USA). After a femoral and internal jugular venous access, multiple electrode catheters were positioned in the coronary sinus (CS), right ventricular apex (RVA), and HB. An irrigated-tip catheter (THERMOCOOL SMARTTOUCH, Biosense Webster Inc., Diamond Bar, CA,

\* Corresponding author. Section of Arrhythmia, Division of Cardiovascular Medicine, Department of Internal Medicine, Kobe University Graduate School of Medicine, 7-5-2 Kusunoki-chou chuou-ku, Kobe, Japan.

E-mail address: [kfuku@med.kobe-u.ac.jp](mailto:kfuku@med.kobe-u.ac.jp) (K. Fukuzawa).

Peer review under responsibility of Indian Heart Rhythm Society.

USA) was inserted via the femoral artery.

The baseline A-H and H-V intervals were 170 ms and 60 ms. The amplitude of the local bipolar electrograms on the basal septum of the RV and mid-basal septum-anterior wall of the LV was less than 1.5mV. A good pace map match was obtained (11/12) in the RV close to the HB. The local bipolar voltage of the good pace map site was within normal range and no delayed potentials were observed there. A perfect pace map could not be obtained anywhere in either ventricle. An activation map of the clinical VT revealed that the earliest area was observed in the basal septal area of both ventricles with a focal pattern. The mapped CL did not cover the VTCL, and no delayed or diastolic potentials could be found, however, her VT was defined as reentry. It later emerged that we were not able to find the critical part of the VT during the 1st or 2nd sessions. Entrainment pacing at the earliest site demonstrated manifest fusion and the post pacing interval (PPI) was equal to the VTCL. These findings indicated that this area was thought to be an outer loop of the circuit. Since entrainment pacing from the RVA exhibited manifest fusion and the PPI – VTCL was 52 ms, a BBR-VT was unlikely [4]. When pacing was applied from the atrium during the VT (supplement data of Figs 1), the QRS morphology and H-V interval (=60 ms) during pacing were exactly the same as that during sinus rhythm. No fused QRS morphology during atrial pacing or the VT was observed. Therefore, atrial pacing could not entrain the VT. Atrial pacing unmasked a His bundle (HB) potential, and the HB potential was buried within the ventricular electrogram during the VT. Note that the ABL catheter was positioned in the HB potential recording region around the tricuspid annulus. These findings suggested that her VT was not a His-Purkinje-related VT. Radio-frequency (RF) energy was applied to the earliest area during VT while being careful to avoid damage to the AV conduction system, however, these two sessions were unsuccessful.

### 1.3. The 3rd session

Then, again she suffered from an electrical storm of VT requiring a 3rd session with the same system as previously. In this session, the distal tip of the CS catheter could be positioned near the bifurcation of the anterior interventricular vein. Further, a multi-

electrode catheter was inserted via a *trans*-septal approach, which was successfully positioned along the HB and left posterior fascicular recording region at the basal septal LV outflow tract (LVOT).

The QRS complex during sinus rhythm became a right bundle branch block pattern (QRS widths = 142 ms) after the 2nd session (Fig. 1C). Although less than the original QRS complex during sinus rhythm (Fig. 1A), the QRS morphology during sinus rhythm in the 3rd session comparatively resembled that of the VT (Fig. 1B/1C). The A-H and H-V intervals were 165 ms and 61 ms at the beginning of the session. The same clinical VT (CL 444 ms) was induced by double extra-stimuli from the RVA. Just as in the previous sessions, the His-Purkinje potential did not precede the QRS during the VT (Fig. 2A/2B, open triangles). Further, the earliest area was also observed on the basal septum close to the HB recording area (Fig. 3B, white arrowheads). However, surprisingly unlike that in the previous two sessions, a mid-diastolic potential (MDP) could be found on the free wall of the basal anterior LV (MDP-site, Figs. 2A and 3A), which was about 40mm away from the earliest site (Fig. 3B, yellow arrows indicate MDP-site). The MDP was also observed on the CS distal electrode (Fig. 2A, white arrows). Further, looking back at the electrograms from CS1-2 before the VT induction, a delayed potential, which could account for a critical part of the VT circuit, was recorded (Fig. 2C-1, orange triangle). The distance between the tip of the ablation catheter and CS catheter was estimated to be 5mm. The wall thickness in this area was very thin and late gadolinium enhancement on cardiac magnetic resonance imaging was positive in that area (Fig. 3C/3D). Entrainment pacing from the MDP-site resulted in a concealed entrainment phenomenon (Fig. 2A). An RF application at the MDP-site could terminate the VT after 11 seconds (Fig. 2B). No His-Purkinje potentials were identified at this site either during the VT or sinus rhythm. RF applications were delivered around this area (power setting max 35 W, irrigation flow 30ml/min, temperature limit 43 °C, and max duration 124sec). No VT was inducible by programmed stimulation up to triple extrastimuli with an isoproterenol infusion at the end of the session. During more than 36 months of follow-up, she has been free from any VT.

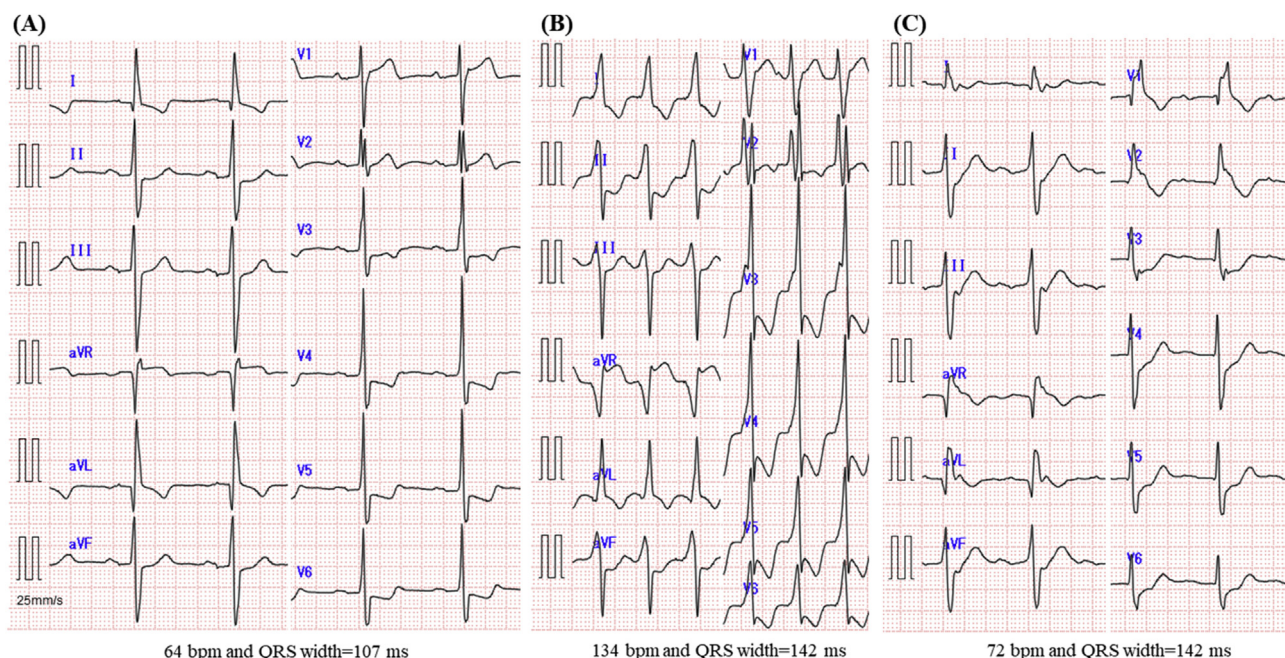
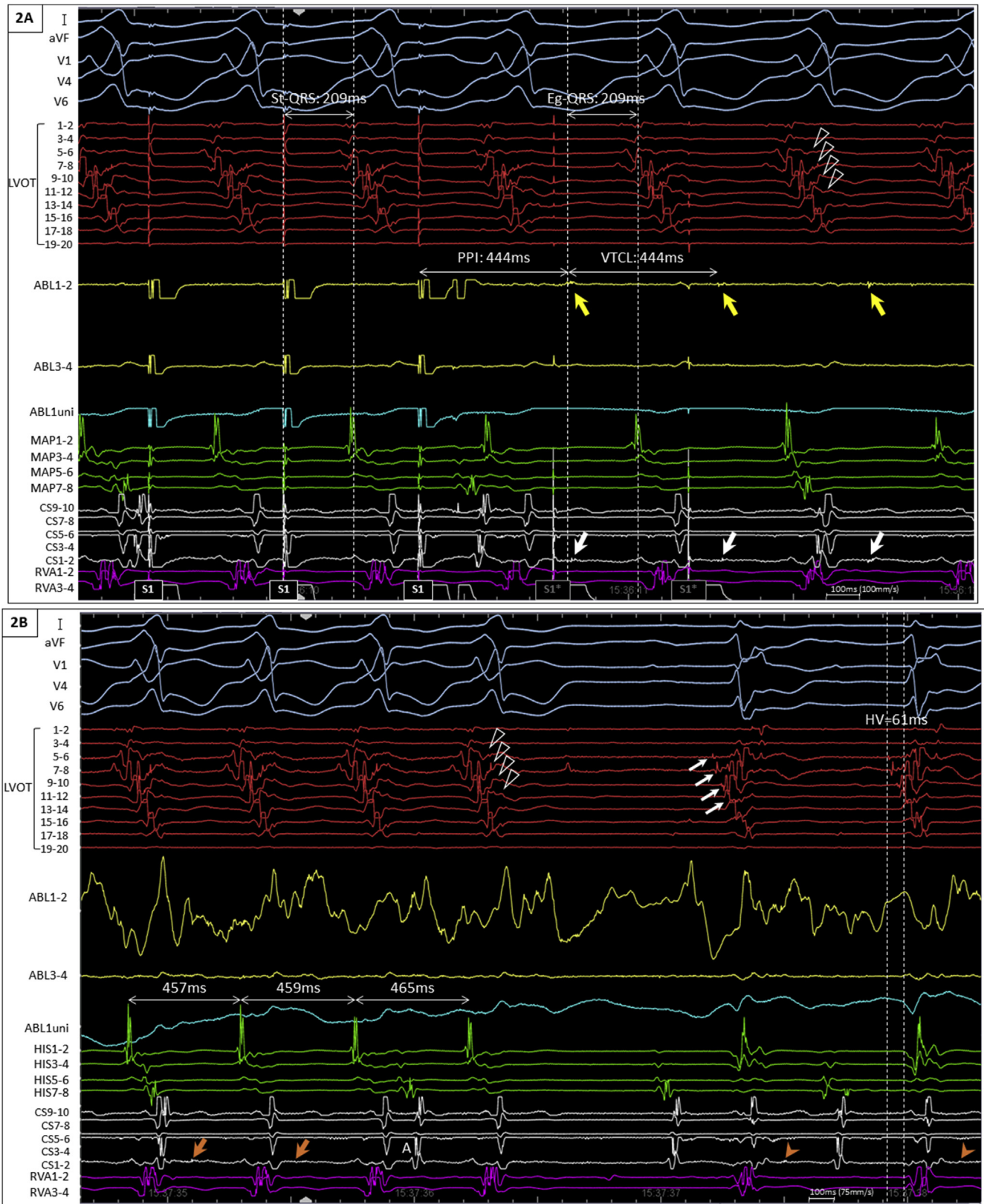


Fig. 1. The 12-lead electrocardiogram of sinus rhythm (A, C) and ventricular tachycardia (B).





**Fig. 2. The intracardiac electrograms are present.** (2A) An MDP was identified on the anterior free wall of the basal LV (yellow arrows). The post pacing interval was the same as the VTCL. The QRS morphology was identical to that of the VT. The interval from the stimulus to QRS (St-QRS) was equal to that from the local electrogram to QRS (Eg-QRS). Importantly, the MDP was also recorded by the CS distal electrode (white arrows). The sequence of the His-fascicular potential during the VT was downwardly (open triangles), the same as during sinus rhythm (white arrows in Fig. 2B/2C). (S1\*: The last 2 stimuli failed to capture). (2B) The VTCL increased and then the VT terminated. The mid-diastolic potential was observed during the VT at CS1-2 electrode (orange arrows) and it disappeared soon after. (2C-1) The intracardiac electrograms during the 3rd session before the VT induction are presented. The late potential was recorded in the electrograms from CS1-2 (2C-1, orange triangle). Note that the ABL catheter was positioned in the HB potential (white arrowhead) recording region on the basal septum of the LV.

The HB and left posterior fascicular potentials were recorded by the LVOT catheter (white arrows indicate the HB-left posterior fascicular potential). (2C-2) After the 1st RF application, the late potential was no longer observed (orange open triangle). At that time, the ABL catheter was positioned at the successful ablation site.

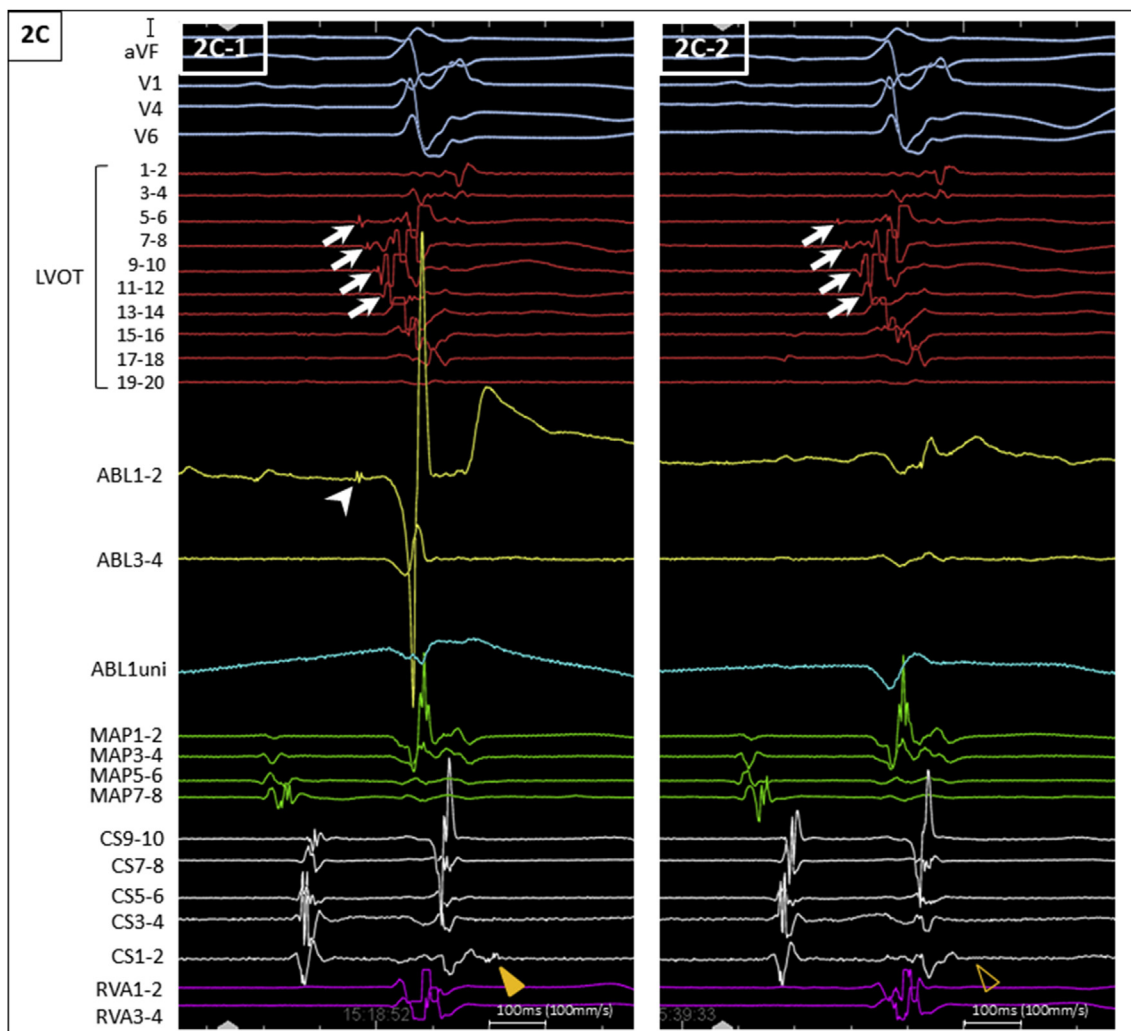


Fig. 2. (continued).

## 2. Discussion

### 2.1. VT with a relatively narrow QRS in cardiac sarcoidosis

Naruse et al. analyzed VT associated with cardiac sarcoidosis. Six of 37 VTs (16.2%) were classified as Purkinje-related VTs, in which Purkinje potentials were identified during sinus rhythm and always preceded the QRS onset by at least >20 ms at the successful ablation site. A QRS duration of <170 ms could identify a Purkinje-related VT with a high sensitivity and specificity [3].

### 2.2. The mechanism of the relatively narrow QRS VT

Stephen P Page et al. reported a case of ischemic VT with a narrow QRS arising from an area of scar near the base of the left posterior septum. They found an MDP at the successful ablation site, but no Purkinje potentials were recorded there. Their proposed VT circuit was within a protected area of scar and the excitation directly exited via an HB fiber [5].

In our case, we speculated that the VT circuit was located within the anterior LV free wall and basal septum in a protected area of scar

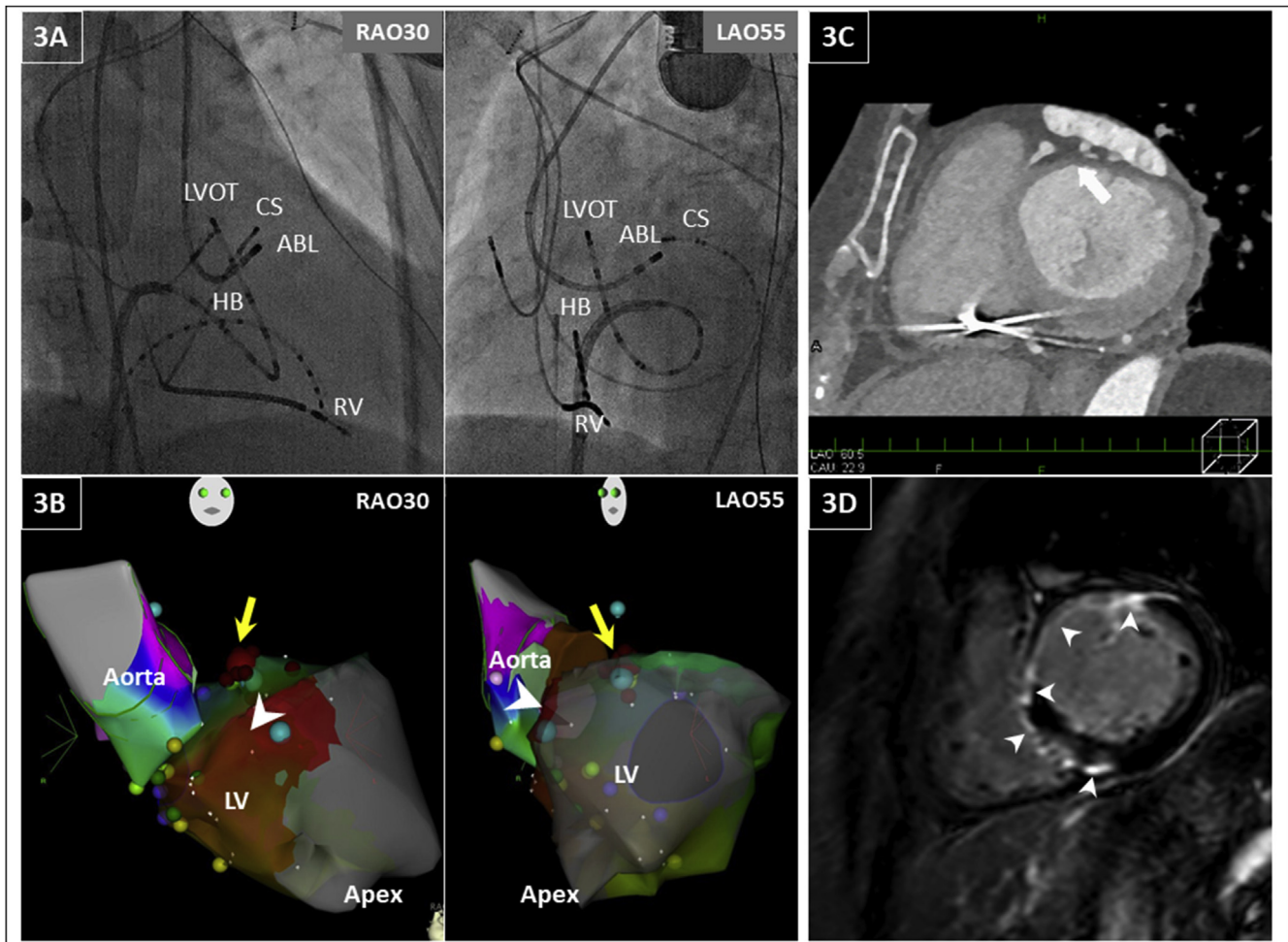
(Fig. 3C/3D) associated with cardiac sarcoidosis, and the circuit was connected to the basal septal region close to the His-Purkinje system. This unique trajectory of the VT might cause a similar QRS to that of sinus rhythm, and the relatively narrow QRS complex despite the critical isthmus was located on anterior free wall of the LV.

## 3. Conclusions

The true critical VT circuit was located far from the pre-operatively presumed region. We learned again the limitations of surface ECG localization of scar related VT. For the success of the VT ablation, knowledge of how to map both ventricles comprehensively with adequate prior preparation and without any pre-conceptions is essential, which is considerably required especially when a target cannot be identified in a pre-operatively presumed region.

### Declaration of competing interest

The Section of Arrhythmia is supported by an endowment from Abbott JAPAN and Medtronic JAPAN. KH chairs the Section, and KF



**Fig. 3.** Fluoroscopic views, electro-anatomical maps, and cardiac images (3A) The fluoroscopic views of the catheters are presented. The ABL catheter was located at the MDP-site (successful ablation point). (3B) Three-dimensional maps of the VT are shown. The yellow arrows show the successful ablation point (white arrowheads indicate the earliest area of the LV, just opposite the RV septum near the HB). (3C) The cardiac computed tomography image focusing around the successful ablation site (white arrow indicates the successful ablation site) is presented, and the LV wall thickness around this area was thin (approximately 5mm). (3D) A cardiac magnetic resonance image obtained before the 1st session is presented. The late gadolinium enhancement was positive (arrowheads) at the endocardial site in the basal anterior and basal antero-septal region. It was also positive in the mid-layer of the septal and epicardial region of the inferior wall.

and KK belong to the Section. However, all authors report no conflict of interest for this manuscript's content.

### Acknowledgments

We would like to thank Mr. John Martin for his linguistic assistance.

### Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.ipej.2021.05.008>.

### References

- [1] Bargout R, Kelly RF. Sarcoid heart disease: clinical course and treatment. *Int J Cardiol* 2004;97:173–82.
- [2] Koplan BA, Soejima K, Baughman K, Epstein LM, Stevenson WG. Refractory ventricular tachycardia secondary to cardiac sarcoid: electrophysiologic characteristics, mapping, and ablation. *Heart Rhythm* 2006;3:924–9.
- [3] Naruse Y, Sekiguchi Y, Nogami A, et al. Systematic treatment approach to ventricular tachycardia in cardiac sarcoidosis. *Circ Arrhythm Electrophysiol* 2014;7:407–13.
- [4] Merino JL, Peinado R, Fernandez-Lozano I, et al. Bundle-branch reentry and the postpacing interval after entrainment by right ventricular apex stimulation: a new approach to elucidate the mechanism of wide-QRS-complex tachycardia with atrioventricular dissociation. *Circulation* 2001;103:1102–8.
- [5] Page SP, Watts T, Yeo WT, Mehl D. Ischemic ventricular tachycardia presenting as a narrow complex tachycardia. *Indian Pacing Electrophysiol J* 2014;14:203–10.