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## Case Report

# PVCs with multiple exits and single site of origin in the outflow tract: What is the mechanism?



Muthiah Subramanian, Sachin Yalagudri, Daljeet Kaur Saggu, Vickram Vignesh Rangaswamy, Calambur Narasimhan\*

AIG Hospitals, Gachibowli, Hyderabad, India

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## ABSTRACT

A 40 year old man with frequent PVCs with two different morphologies was referred for catheter ablation. Although initial mapping in the RVOT revealed fragmented potentials 20ms earlier than PVC2 onset with a good pace map score, ablation at this site was unsuccessful. Subsequent mapping in the LCC/NCC junction revealed that local ventricular activation preceded QRS onset by 30 and 28 ms for PVC1 and PVC2, respectively. Altering the pacing output at this site produced QRS morphologies similar to PVC1 (low output, 6mA) and PVC2 (high output, 15mA) with better pace map scores compared to RVOT. During high-output pacing, there was an increase in stim-QRS latency with decremental conduction. Ablation at this site was successful and suppressed both PVCs.

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## 1. Introduction

Complex anatomy of myocardial extensions may contribute to variable exit sites in the circumference of the outflow tracts. Specialized myocardial fibers can contribute to preferential conduction from the aortic sinus cusps to the right ventricular outflow tracts [1]. We report a case of a PVC originating from the aortic cusp preferential conducting to two exits in the outflow tract, exhibiting two different morphologies of PVCs.

## 2. Case report

A 40-year-old man, who was symptomatic with palpitations that were refractory to medical therapy, was referred for catheter ablation. A transthoracic echocardiogram and cardiac magnetic resonance imaging revealed no structural heart disease. Electrocardiogram revealed premature ventricular contractions (PVCs) with two different morphologies and coupling intervals (PVC1–630ms, PVC2–540ms, Fig. 1 A). Although both PVCs had left bundle branch with inferior axis morphology, PVC1 was narrower (QRS duration = 140ms, V2 transition) compared to PVC2 (QRS

duration = 190ms, V4 transition) and had an earlier precordial transition.

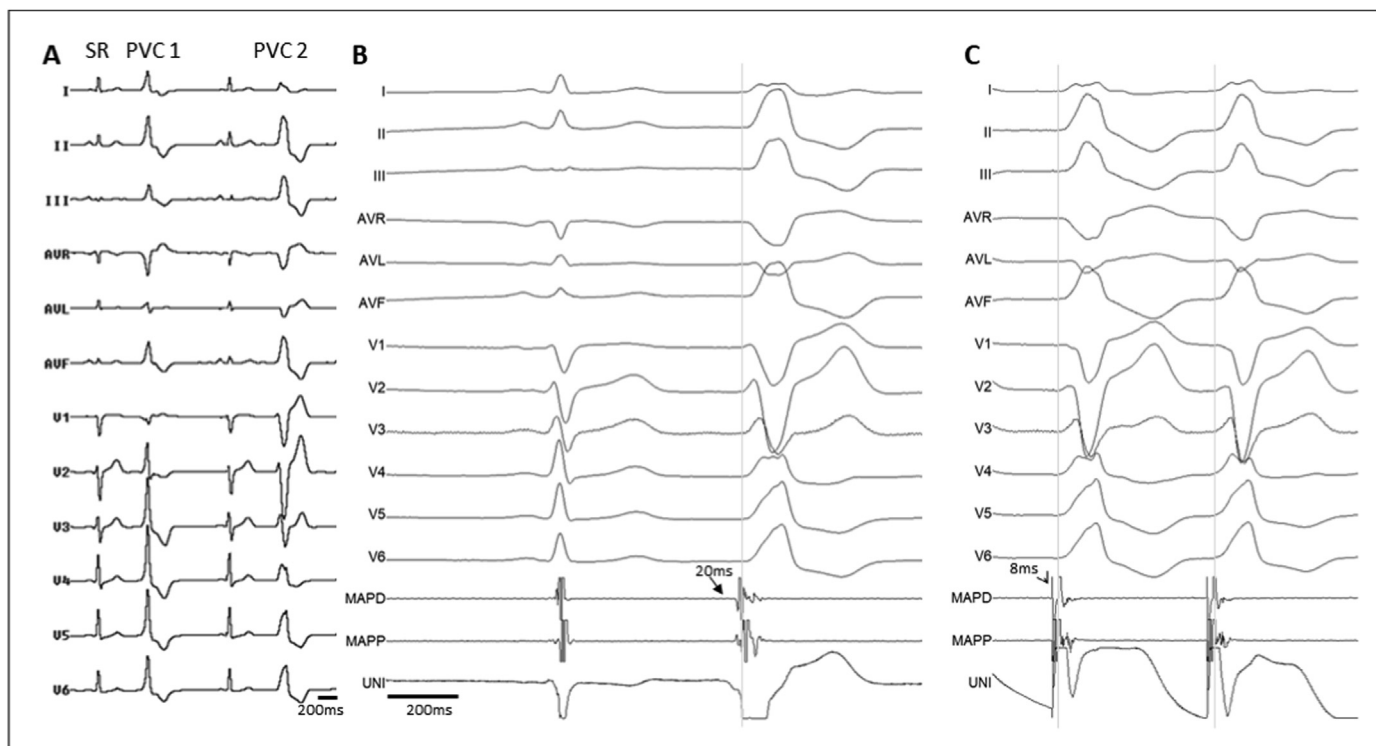
The electroanatomical mapping (EAM) and ablation was performed using a three dimensional EAM system (Carto3; Biosense Webster, Diamond Bar, CA) with 7.5F irrigated-F curve catheter (Navistar Themocool; Biosense Webster, Diamond Bar, CA). PVC1 and PVC2 were both present spontaneously during the electrophysiological study. Activation and pace mapping was initially performed in the right ventricular outflow tract (RVOT) and pulmonary cusps (Fig. 1B and C). With regards to PVC2, the earliest activation site was in the posterior septum, where local ventricular activation preceded QRS onset by 20ms. The unipolar electrogram at this site showed a QS pattern. Pace mapping at this site produced a QRS morphology with a pace-map score of 10/12 for PVC2. The pacing stimulus-QRS (S-QRS) interval of 8 ms. A radiofrequency (RF) application using a power of 30 W up to 43°C failed to suppress PVCs.

At this point, the options in this case include using a higher energy in the RVOT, mapping of the coronary venous system or the aortic cusp. As there not any suppression of PVCs using a power of 30 W up to 43°C, a higher energy was not attempted. Mapping of distal CS and anterior interventricular vein junction was performed but the activation during PVC2 was not early. Hence, further mapping of the coronary venous system was not performed. Hence, we proceeded with activation and pace mapping of the aortic cusp (Fig. 2A–D).

\* Corresponding author. Asian Institute of Gastroenterology, Gachibowli, Hyderabad, 500032, India.

E-mail address: [calambur@hotmail.com](mailto:calambur@hotmail.com) (C. Narasimhan).

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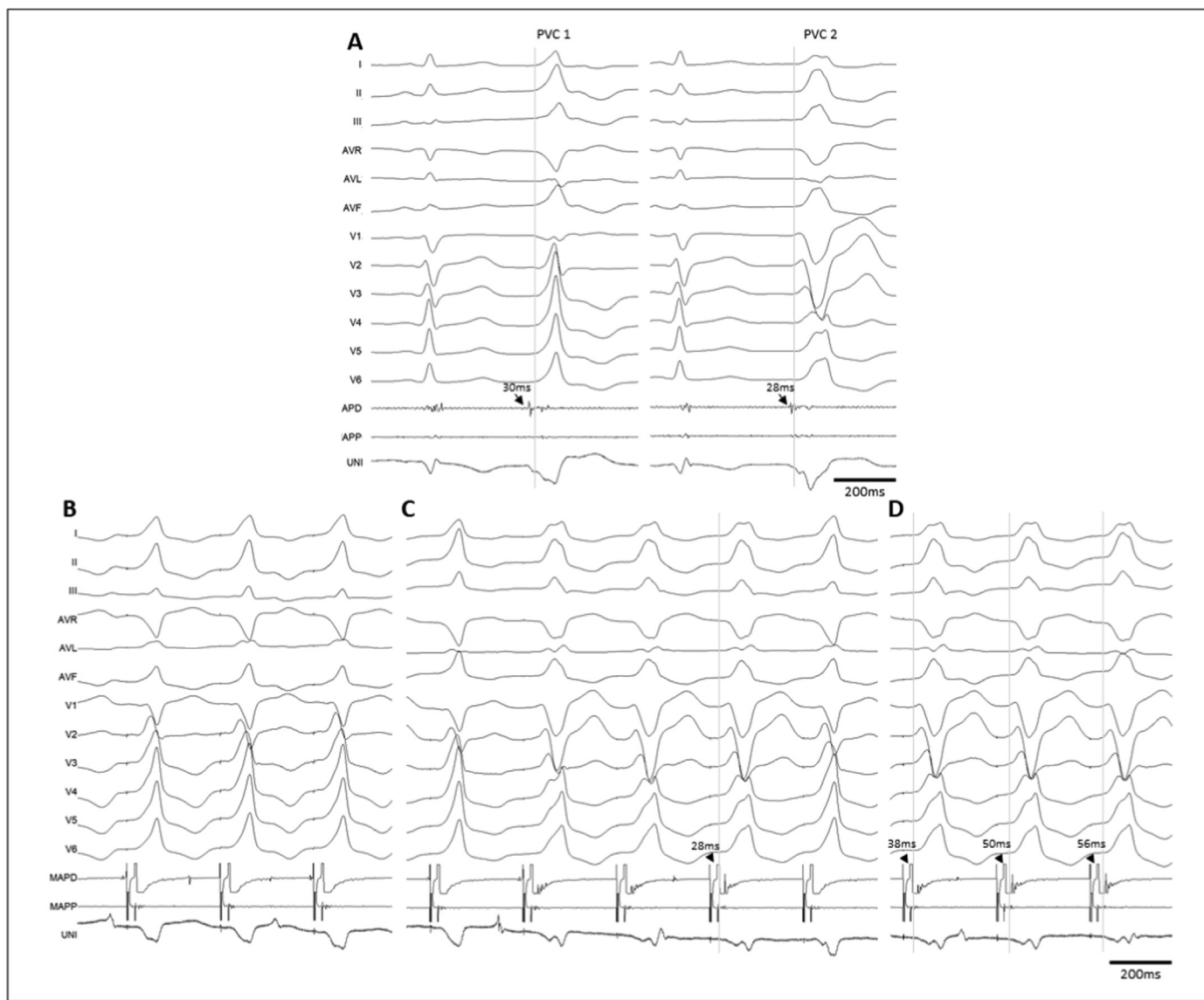
**Fig. 1.** 12 Lead ECG with Activation and Pace Mapping of PVC 2 in the RVOT. A: Twelve lead electrocardiogram recorded during the procedure showing sinus rhythm (SR) and premature ventricular contractions (PVC) 1 and 2. B: Intracardiac electrograms recorded during activation mapping of PVC2 at the RVOT posterior septum. The black arrow indicates the local ventricular potential preceding the QRS onset by 20 ms. The unipolar electrogram showed a QS pattern. C: Pace mapping at the RVOT posterior septum revealed a pace map score of 10/12 for PVC 2. The pacing stimulus to QRS interval was 8ms (arrowhead). MAPD, MAPP (the distal and proximal electrode pairs of the mapping catheter); UNI (the distal unipolar electrode of the mapping catheter).

Activation mapping in the non and left coronary cusp (LCC/NCC) junction revealed that local ventricular activation preceded QRS onset by 30 and 28 ms for PVC1 and PVC2, respectively. The unipolar electrograms at this site for both PVCs showed a QS pattern. Pacing with an output of 6mA (fixed pulse width of 2ms) at the LCC/NCC junction produced QRS morphologies only similar to PVC1 with a pace map score of 10/12. Pacing with an output of 9mA at the same location produced QRS morphologies similar to both PVC 1 and PVC2 with pace map scores of 10/12 and 10/12, respectively. Interestingly, pacing with an output of 15mA at the same site produced QRS morphologies similar to only PVC 2 with a pace map score of 11/12 and a S-QRS interval of 38ms. Subsequent pacing at the same output showed a decremental conduction (38,50,56 ms). Ablation at this site in the LCC/NCC junction using a power of 30 W up to 43°C suppressed both PVCs within 5 seconds of starting energy (Fig. 3). The lesion was further consolidated for total of 60 seconds. The ectopy did not recur during a waiting time of 30 minutes including an isoproterenol challenge. At 18 months of follow up, the patient remains asymptomatic without any recurrence of PVCs.

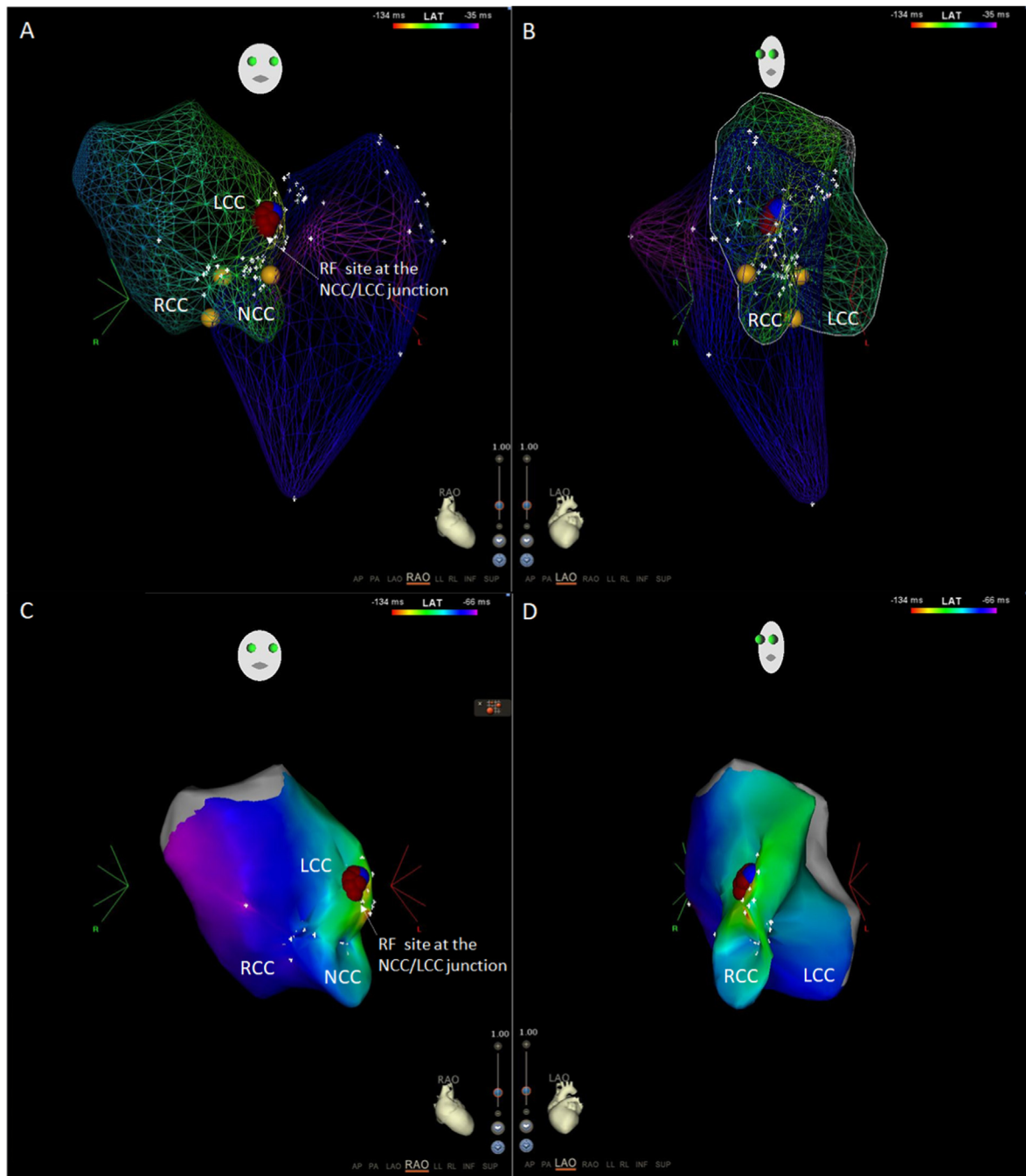
In this case, a PVC originating from the aortic cusp had preferential conduction to two exits in the outflow tract and exhibited two different morphologies of PVCs. Outflow tract anatomy and electrophysiological properties of the surrounding myocardium may explain this observation. Parts of the right and left coronary aortic leaflets are related to the ventricular septum and left ventricular free wall, respectively [2]. In these areas, ventricular myocardium extends beyond the semilunar valves, enclosing muscle at the cusps of the aortic sinuses. Although PVCs can be ablated either above or below the aortic valve, it is the myocardium

of the LV ostium that is often the target for ablation [3]. These myocardial extensions can vary in course (oblique or longitudinal), location (endocardial or epicardial), or continuity with underlying ventricular musculature. In addition, myocardial hypertrophy, fibrosis, and interposed adipose tissue have been described within these myocardial extensions [4]. The complex anatomy of these extensions may contribute to variable exits across the circumference of the aortic cusps.

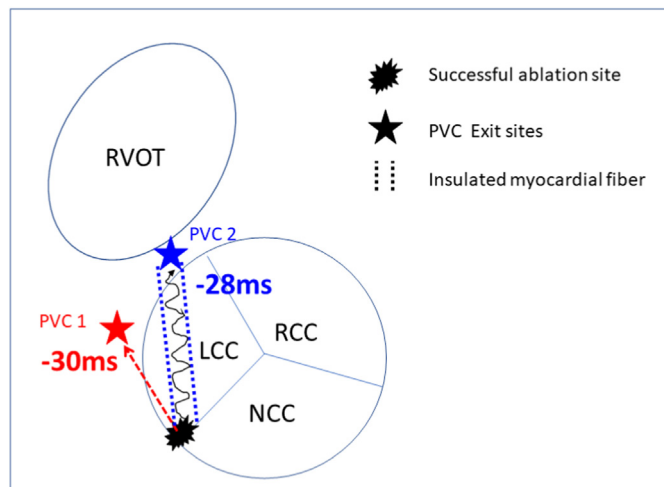
A breakout site, suggested by an excellent pace map, that is remote from the VA origin can be explained by the involvement of preferential conduction through specialized myocardial fibers. These fibers can contribute to preferential conduction from the aortic sinus cusp to the RVOT [5–7]. These myocardial fibers travel between the site of origin and the site of breakout in the larger mass of the myocardium. As hypothesized by Yamada et al., preferential conduction via myocardial fibers in this case is supported by the significantly longer stim-QRS interval when pacing from the aortic cusp compared to the RVOT [1]. In addition, this case demonstrated two novel properties of these myocardial fibers. First, pacing at a higher output from within the aortic cusp yielded a closer match to the QRS morphology of PVC2 than pacing from within the RVOT. Pacing at lower outputs from the same location diminished the preferential conduction of PVC 2 from the aortic cusp to the RVOT. These findings suggest that an insulated myocardial fiber travelling from the origin in the aortic cusp to the breakout site in the RVOT might exist (Fig. 4). Such a myocardial fiber may only be selectively captured with a higher pacing output. If preferential conduction is suspected, pacing at different outputs can be used to selectively capture the responsible fibers. Second, pacing at a higher output in the aortic cusp revealed decremental



**Fig. 2.** Activation and Pace Mapping of PVC 1 and PVC 2 in the Aortic Cusp. A: Intracardiac electrograms recorded during activation mapping of PVC 1 and PVC 2 at the junction of the left coronary and right coronary cusp (LCC/NCC junction). The black arrow indicates the local ventricular potential preceding the QRS onset by 30 and 28 ms for PVC 1 and PVC 2, respectively. B: Pacing at a 6mA output at the LCC/NCC junction revealed a 10/12 pace-map for PVC 1. C: Pacing at 9mA at the same site revealed QRS complexes with morphology similar to PVC 1 (5th QRS complex, pace map 10/12) and PVC 2 (4th QRS complex, pace map 10/12). The first three paced complexes represent fusion complexes. The pacing stimulus to QRS (S-QRS, black arrowhead) for PVC2 was 28ms. D: Pacing at 15mA at the same site revealed QRS morphology similar to only PVC 2. Pace-map score was 11/12 with a S-QRS interval of 38, 50 and 56 ms, suggestive of decremental conduction. Refer to Fig. 1 for other abbreviations.



**Fig. 3.** Three-dimensional electroanatomical CARTO map of the LV and RV outflow tracts are shown as a mesh in RAO 30 (A) and LAO 30 (B). The non (NCC), left (LCC), and right (RCC) coronary cusps are labelled. The yellow tags represent the His and red tags represent the ablation points. The earliest activation site of PVC1 and PVC2 (shown in Fig. 2) was at the NCC/LCC junction (C and D). This was the site of successful radiofrequency ablation and is shown in LAO (A and C).



**Fig. 4.** Diagram showing the origin and presumed preferential conduction paths of PVC 1 and PVC 2. With regards to PVC2, we hypothesized that preferential conduction occurred via an insulated myocardial fiber from the origin in the aortic cusp and the exit in the RVOT septum.

conduction with longer S-QRS intervals. This may support the presence of slow conduction within these myocardial fibers. Preferential conduction of arrhythmias originating from the aortic cusp may be explained by a combination of structural and functional properties unique to myocardial fibers in this location.

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### Declaration of competing interest

None.

### References

- [1] Yamada T, Murakami Y, Yoshida N, et al. Preferential conduction across the ventricular outflow septum in ventricular arrhythmias originating from the aortic sinus cusp. *J Am Coll Cardiol* 2007;50:884–91.
- [2] Ouyang F, Fotuhi P, Ho SY, et al. Repetitive monomorphic ventricular tachycardia originating from the aortic sinus cusp: electrocardiographic characterization for guiding catheter ablation. *J Am Coll Cardiol* 2002;39:500–8.
- [3] Yamada T, Litovsky SH, Kay GN. The left ventricular ostium: an anatomic concept relevant to idiopathic ventricular arrhythmias. *Circ Arrhythm Electrophysiol* 2008;1:396–404.
- [4] Hasdemir C, Aktas S, Govsa F, et al. Demonstration of ventricular myocardial extensions into the pulmonary artery and aorta beyond the ventriculo-arterial junction. *Pacing Clin Electrophysiol* 2007;30:534–9.
- [5] Kanzaki Y, Morishima I, Awaji Y, Kato R. Preferential conduction travelling from the left coronary cusp to the right ventricular outflow tract via the right coronary cusp of the aorta. *Indian Pacing Electrophysiol J* 2015;15:165–7.
- [6] Yazaki K, Enta K, Watarai M, et al. Successful elimination of premature ventricular contractions by ablation of origin and preferential pathway. *Clin Case Rep* 2018;6:52–5.
- [7] Yamada T, Platonov M, McElderry HT, Kay GN. Left ventricular outflow tract tachycardia with preferential conduction and multiple exits. *Circ Arrhythm Electrophysiol* 2008;1:140–2.