Ablation of parahisian ventricular focus

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Introduction

Idiopathic premature ventricular contractions (PVCs) are a common arrhythmia, sometimes presenting as highly symptomatic and drug-resistant ectopy and more rarely as a reduction in left ventricular function.[1] Common sites of PVCs include the right and left ventricular outflow tracts, the mitral annulus, and the aortic cusps. In addition, the right ventricular septum in the vicinity of the His bundle has been reported to be a site of idiopathic PVCs.[2–4] Radiofrequency (RF) application at the His-bundle region has the potential risk for atrioventricular conduction disturbance. We report the case of a patient with idiopathic PVCs that was ablated in a location recording the largest amplitude His potential.

Case report

A 17-year-old boy with highly symptomatic PVCs and failed ablation at another hospital was referred for ablation. Holter monitoring demonstrated frequent single and paired monomorphic PVCs with 22773/111645 beats (20%) per day. Echocardiography demonstrated normal left ventricular function. Twelve-lead ECG of the clinical PVCs showed left branch block pattern, inferior axis, and precordial transition in leads V_2–V_3 (Figure 1A). After obtaining informed consent, electrophysiologic study and ablation were a performed via right femoral venous and arterial access. A steerable quadripolar catheter (2-5-2 mm, Xtrem,

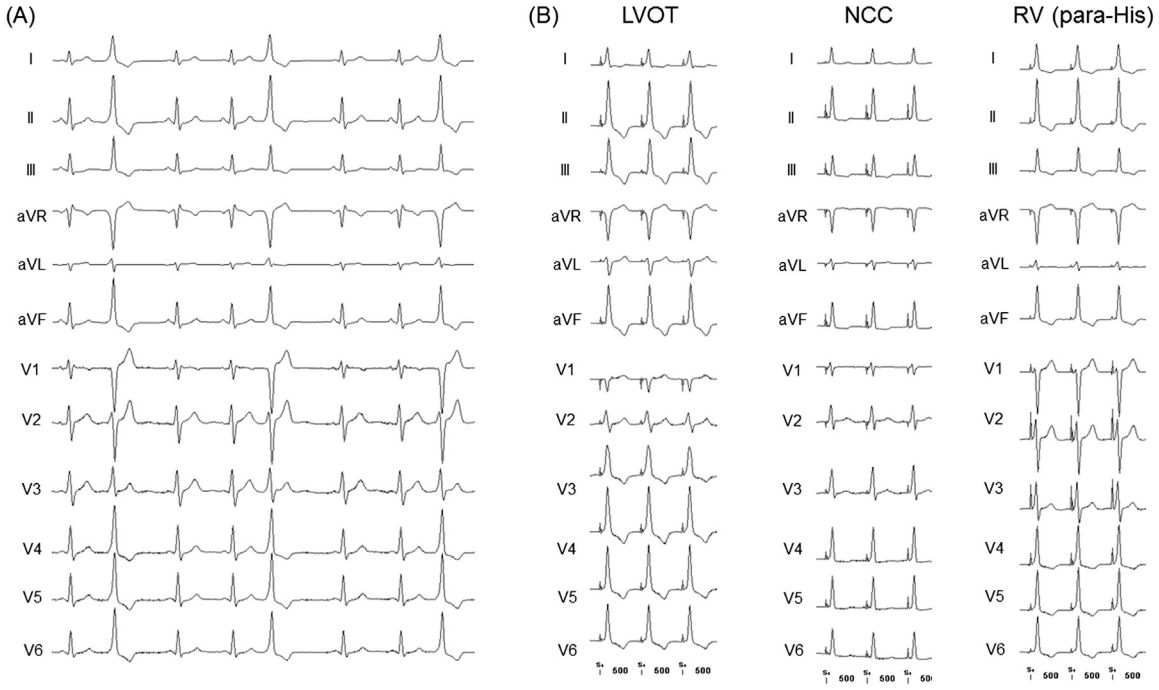

Figure 1 **A:** Twelve-lead ECG of the baseline rhythm. The morphology of premature ventricular contractions exhibited inferior axis, left bundle branch block pattern with rsR pattern in aVL and early precordial transition in leads V_2–V_3. **B:** Pace-mapping from the left ventricular outflow tract, aortic noncoronary cusp, and right ventricular His region (see Figure 2 for pacing sites). The best pace-mapping was obtained in the parahisian region. LVOT = left ventricular outflow tract; NCC = noncoronary cusp; RV = right ventricle.

KEYWORDS Ventricular premature contraction; His bundle; Radiofrequency ablation

ABBREVIATIONS **LVOT** = left ventricular outflow tract; **NCC** = noncoronary cusp; **PVC** = premature ventricular contraction; **RF** = radiofrequency (Heart Rhythm Case Reports 2015;1:64–67)

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KEY TEACHING POINTS

- Mapping of premature ventricular contractions (PVCs) originating from the parahisian region should be performed biventricularly, including the aortic coronary cusp. The precocity, QS pattern in the unipolar electrogram of the ablation catheter, and good pace-mapping can help in finding a successful ablation site.
- Ablation at the parahisian region has a risk of atrioventricular conduction injury. Radiofrequency application in this region should be started with low power energy and discontinued immediately if accelerated junctional beats or atrioventricular dyssynchrony is observed.
- In the present case, location of the PVCs toward the relatively protected distal end of the His bundle allowed for ablation without the development of heart block. We adopted a strategy of low power ablation, with subsequent power incrementation, at the site of earliest ventricular activation, immediately adjacent the His bundle. Using this strategy, we could eliminate the PVCs without atrioventricular conduction block.

ELA Medical, Montrouge, France) was positioned in the right ventricular basal septum. The left ventricle was accessed by transseptal puncture (BRK needle and Agilis sheath, St. Jude Medical, St. Paul, MN), and the aortic cusp was mapped via a retrograde approach with a 3.5-mm irrigated-tip ablation catheter (NaviStar ThermoCool, Biosense Webster, Diamond Bar, CA). Mapping of the PVCs was initially performed in the left ventricular outflow tract (LVOT) and around the aortic annulus. The noncoronary cusp (NCC) was identified as being located posteriorly in both the right anterior oblique and left anterior oblique fluoroscopic views, without assistance of angiography or ultrasound imaging. The LVOT and NCC sites demonstrated local PVC activation preceding QRS onset by 30 and 32 ms, respectively, with small-amplitude His potentials in these regions (0.03 and 0.04 mV, respectively). Good pace-mapping was obtained on the NCC (Figures 1B, 2A, and 2B). However, RF applications performed at these sites, with a maximum of 40 W, had no impact on the PVCs. During mapping in the right ventricle, right bundle block was produced by mechanical trauma due to the steerable sheath and persisted thereafter. Mapping revealed earliest PVC activation on the tricuspid annulus adjacent to the His bundle. PVC activation preceded QRS onset by 42 ms, and there was a coexistent large-amplitude (0.31 mV) His potential of similar amplitude to the atrial electrogram. The ablation catheter recorded distal-to-proximal His-bundle activation during PVCs (Figure 2C). A good pace-map was obtained in this area (Figure 1B). Initially, several ablations were attempted at a site slightly remote from the maximal His potential with the aim of minimizing the risk of complications. However, ablation there achieved only a transient effect. Having discussed with the patient and family in detail the risks of further ablation, including the probability of right bundle block, and taking into account the highly symptomatic nature of the PVCs, further ablation centered on the site with high-amplitude His potential and earliest ventricular activation. RF application at this site using the steerable sheath durably abolished PVCs after 4.5 seconds at energy increasing from 5 to 10 W (temperature limit 50ºC; Figure 3). Subsequently, RF energy was cautiously increased up to 26 W during 4 minutes at the same site, scrutinizing for the occurrence of junctional rhythm. There was no further atrioventricular conduction disturbance with ablation at the site of the large His potential; AH and HV intervals remained at 56 and 42 ms preablation and postablation. PVC inducibility was assessed with 12 mg adenosine but produced no effect. PVCs remained totally absent (0/113692 beats) on repeat Holter monitoring performed 6 weeks postprocedure, with persistence of right bundle branch block.

Discussion

This case demonstrates idiopathic PVCs originating in a parahisian location that was successfully ablated using slow incrementation of RF energy to avoid atrioventricular conduction injury. Ablation of idiopathic parahisian PVCs has been described previously.[2,5,6] However, our case demonstrates successful ablation at a location with a higher-amplitude His potential on the distal bipole of the ablation catheter than previously reported. Dick et al[7] described the relationship between the amplitude of the His potential and the distance from the His bundle during open heart surgery, albeit using a catheter with half the bipole separation of the ablation catheter used here. The amplitude of the His potential was recorded as 0.38–1.13, 0.27–0.86, and 0.2–0.44 mV at 1, 2, and 3mm from the His bundle, respectively, indicating that the origin of PVCs in our case was within 2–3 mm of the His bundle.[7] At the site of successful ablation, we recorded a His potential that was similar on the distal bipole of the ablation catheter compared to the proximal bipole, but we ablated distally toward the distal end of the His bundle. Whereas the proximal His bundle is relatively unprotected from RF energy, the distal bundle is surrounded by a fibrous body that may help protect it from injury during RF application,[8] as has been reported during ablation of parahisian anteroseptal accessory pathways.[9] Special care to avoid inadvertent atrioventricular conduction block should be taken during RF application, including (1) stopping RF at the onset of accelerated junctional beats[9,10] and (2) avoiding ablation over the proximal His bundle, as indicated by an atrioventricular electrogram amplitude ratio of 1:1–1:2 with His potential,[11] the presence of His and atrial potentials on the distal ablation bipole, and the absence of ventricular potential on the proximal bipole.[12] We contend that in our case, location of the PVCs toward the relatively protected