Right-sided substrate eliminated by transmural ablation from the left atrial septum in a patient with atrioventricular nodal reentrant tachycardia



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Introduction

Atrioventricular nodal reentrant tachycardia (AVNRT) is the most common supraventricular arrhythmia in humans, is more common in women than in men (2-fold women-tomen ratio), and is rare in pediatric patients ($\sim 3\%$).¹ Although catheter ablation is highly effective, the precise anatomical substrate and nature of the fast and slow pathways are still unknown, and several case reports and studies noted that ablation in the left atrium (LA) was required when conventional right-sided ablation failed to eliminate typical or atypical AVNRT (~1%).²⁻⁶ Although the left inferior extension of the slow pathway is considered a main reason for indispensable ablation of the inferior (posterior) part of the mitral annulus (MA), we herein report a male pediatric patient in whom critical substrate of AVNRT was considered present in the right atrium (RA) (Koch's triangle), but left septal ablation was indispensable for modification of the slow pathway without injury to the superior input of the atrioventricular (AV) node and fast pathway, which is known to have substantial anatomical variability.7

Case report

A 13-year-old boy without structural heart disease was referred to our institution for electrophysiological study with possible catheter ablation for the sudden onset and termination of palpitations. A tachycardia was induced by programed stimulation and the diagnosis of slow/fast form of AVNRT was made. Although ablation between the coro-

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KEY TEACHING POINTS

- Ablation in the left atrium is required when conventional right-sided ablation fails to eliminate typical or atypical atrioventricular nodal reentrant tachycardia (AVNRT) (~1%).
- Although left inferior extension of the slow pathway is considered a main reason for indispensable ablation of the inferior (posterior) part of the mitral annulus, the reasons and mechanisms for failure of right-sided ablation seem not to be focused on and may not have been adequately discussed in previous studies.
- Even if a leftward extension of the slow pathway does not exist, left atrial ablation may be effective in eliminating the substrate of AVNRT to overcome the close proximity between the fast and slow pathways within the right atrium and the high risk of atrioventricular block.

nary sinus (CS) ostium and tricuspid annulus (TA) at the level of the CS ostium and roof of the proximal CS musculature was performed, AVNRT was still inducible. Because of the patient's young age and our reluctance to risk the creation of AV block, no further attempts were made. The patient was maintained on oral bisoprolol or flecainide for 5 years, and a redo procedure was performed under CARTO guidance (Biosense Webster Inc, Diamond Bar, CA) owing to recurrence of palpitations.

Four quadripolar or decapolar catheters were placed in the RA appendage, CS, His bundle, and right ventricular apex. Programmed atrial stimulation with single extrastimuli using a drive cycle length of 400 ms induced supraventricular tachycardia with an atrio-Hisian jump (Figure 1A and 1B). The diagnosis of slow/fast form of AVNRT was confirmed

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Figure 1 Atrioventricular conduction curve and induction of atrioventricular nodal reentrant tachycardia (AVNRT). A: Programed atrial stimulation with single extrastimuli using a drive cycle length of 600 ms revealed an atrio-Hisian jump. B: AVNRT was reproducibly induced by programed stimulation. C: Atrioventricular block was observed during the tachycardia. CS = coronary sinus; HB = His bundle; RA = right atrium; RV = right ventricle.

by a V-A-V response after ventricular overdrive pacing, ventriculoatrial (VA) linking,⁸ no advancement or delay of His bundle timing or tachycardia termination upon delivery of scanned single premature ventricular contractions (Supplemental Figure 1), and observation of 2:1 AV block during the tachycardia (Figure 1C). Junctional tachycardia was unlikely based on the delta H-A interval (H-A interval during ventricular pacing minus H-A interval during tachycardia) of -13 ms (Supplemental Figure 2).⁹

An electrogram obtained near the TA at the level of the CS ostium (Figure 2) revealed dull and sharp potentials in the atrial component (A/V ratio of 0.15), so-called slow pathway potential (Figure 3A). Radiofrequency (RF) ablation was first attempted at this point with an open-irrigated catheter (ThermoCool STSF, Biosense Webster) at a power setting of 25–30 W, at which the risk of AV block is relatively low (yellow tags in Figure 2), and repetitive junctional beats were induced and stably maintained. However, subsequent ablation (red tags) was immediately stopped 7 seconds after RF-on owing to junctional rhythm with a VA block (Figure 3B). Although this site was 22 mm away from the His bundle recording site

(black tags in Figure 2) and was located on the ventricular side of the TA (A/V ratio of 0.15), the risk of AV block was considered high. In contrast, ablation at the roof of the CS ostium did not induce any junctional beats or VA block (yellow tags). Unintentionally, AV block was also induced by a bump phenomenon during mapping using an ablation catheter around the mid septal area of the RA (white tag in Figure 2), suggesting the close proximity of the input of the AV node and fast pathway to the right inferior extension of the slow pathway. At this time, AVNRT was still easily inducible, and we decided to perform transseptal puncture and inserted a deflectable sheath (Agilis; Abbott, Saint Paul, MN) into the LA. First, the posteroseptal area in the MA, which is just opposite to the roof of the CS ostium, was mapped and ablated (purple tags in Figure 2), but no junctional beats occurred. Then, the left-sided mid (superior) septal area was mapped, and finally, a site opposite to the site with AV block by the bump phenomenon in the RA (8 mm in distance from each other; black arrows in Figure 2) was ablated at a power setting of 30-35 W. The atrial electrogram at this site (A/V ratio of 0.58) (Figure 3D) was less sharp than



Figure 2 Fluoroscopic imaging (A) and 3-dimensional map (B). A: Right anterior oblique (RAO) and left anterior oblique (LAO) views show catheter positions at successful ablation of the slow pathway from the left atrial septum. B: The red tags indicate sites at which junctional rhythm with ventriculoatrial block occurred during right atrium (RA) ablation (Figure 3B), the yellow tags indicate sites at which no junctional rhythm occurred during RA ablation, the purple tags indicate sites at which no junctional rhythm occurred during left atrium (LA) ablation, and the large blue tag indicates the site at which junctional rhythm occurred (Figure 3E) and the slow pathway was successfully modified by LA ablation. Black tags indicate His bundle recording sites, and a white tag indicates a site where atrioventricular block occurred during mapping (bump). ABL = ablation catheter; CS = coronary sinus; HB = His bundle; LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein; RV = right ventricle; SVC = superior vena cava; TV = tricuspid valve.

that in the RA (Figure 3A). Of note, it took a long time, nearly 14 seconds, until junctional beats appeared (Figure 3E), suggesting the transmural effect of RF energy delivery on the right-sided slow pathway. After this application, AVNRT was never induced again, although an atrio-Hisian jump and single slow/fast atrial echo complexes were observed during isoproterenol infusion at 1 mcg/min. The PR interval did not significantly change before and after ablation (136 ms). The patient has been free from any palpitations for 6 months.

Discussion

Prior studies and case reports have described the critical role of left atrial ablation in AVNRT resistant to right-sided ablation.^{2–6} The prevalence of LA ablation in this retrospective cohort was approximately 1%. Although the details of LA ablation were commonly reported in these previous studies and case reports, the reasons and mechanisms for failure of right-sided ablation seemed not to be focused on and may not have been adequately discussed. The left inferior (posterior) extension of the slow pathway is commonly believed to be a mechanism in difficult cases resistant to right-sided ablation, and ablation of the roof of and inside the CS, which is not rarely effective in the elimination of AVNRT, can transmurally eliminate the left-sided nodal extension. Of interest, Kilic

and colleagues³ reported that ablation between the CS and TA did not induce any junctional beats in all 9 patients requiring LA ablation, whereas CS ablation did induce junctional beats in half of them. However, we felt that critical substrate of AVNRT was present within the RA in a persistent manner in the present patient because of the following findings: (1) discrete slow pathway potentials (Figure 3A) were observed more clearly on the TA side (ventricular side) than on the CS side (septal side) in the RA; (2) ablation at those sites characterized by slow pathway potentials reproducibly and stably induced junctional beats, but ablation in the roof of the CS close to a common route of the left inferior extension did not induce any junctional beats; (3) the slow pathway potentials were sharper and more discrete in the RA ablation site than in the LA ablation site; (4) the common successful ablation site in the LA side (ie, the posteroseptal aspect of the MA) was not critical in this case; (5) mid (more superior) septal ablation in the LA required longer-duration RF energy delivery until junctional beats occurred than did right-sided ablation, suggesting transmural lesion formation affecting the substrate localized in the RA; and (6) complete elimination of the slow pathway could not be achieved as an endpoint, although complete noninducibility was achieved.

Moreover, AV block and VA block were induced by the ablation or bump phenomenon around the common



Figure 3 Intracardiac electrograms during the redo procedure. A: A sharp slow pathway potential (*red arrow*) recorded near the tricuspid annulus. B: Junctional beats and ventriculoatrial block (VAB) induced by ablation (light red tag in Figure 2). C: Atrioventricular block (AVB) occurred during mapping at the right septal area (white tag in Figure 2). D: Intracardiac electrograms during successful radiofrequency (RF) ablation. Dull slow pathway potential (*red arrow*) recorded in the left mid septum during sinus rhythm (blue tag in Figure 2). E: Junctional beats begin 14 seconds after RF ablation.

anatomical area of the slow pathway. This suggested the close proximity of the input of the AV node and fast pathway to this area⁷ and is a limitation of right-sided ablation. Taken together, we thought that critical substrate of AVNRT was

present within the RA, but LA ablation was required to modify it without injury to the AV node and fast pathway in this patient. A recent study by Katritsis and colleagues² reported that LA ablation alone (without right-sided ablation)



Figure 3 (continued).

eliminated AVNRT in all 11 prospectively included patients. Although the data provided here are neither direct nor specific evidence supporting the localized circuit within the RA, this case may support a wider range of indications for LA ablation of AVNRT with a poor response to RA ablation.

Conclusion

Even if a leftward extension of the slow pathway does not exist, LA ablation may be effective in eliminating the critical substrate of AVNRT to overcome both the close proximity between the fast and slow pathways within the RA and the high risk of AV block.

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Appendix Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2022. 05.011.

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