Atypical Fast-Slow Atrioventricular Nodal Reentrant Tachycardia
Using a Slow Pathway Extending to the Superoanterior Right Atrium

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Summary

We report a case of atypical fast-slow atrioventricular nodal reentrant tachycardia (AVNRT) using a slow pathway variant extending to the superoanterior right atrium. The AVNRT diagnosis was confirmed by using standard electrophysiologic criteria that exclude a diagnosis of atrial tachycardia and atrioventricular reentrant tachycardia. The earliest atrial activation during tachycardia was found in the superoanterior right atrium adjacent to the tricuspid annulus, where the first delivery of radiofrequency energy terminated and eliminated the inducibility of the tachycardia.

Key words: Ablation, Electrophysiologic study

Recently, we reported the presence of superior slow pathway (SP) that is a variant of the SP originating from compact node and extending superiorly outside of Koch’s triangle.1) We report a case of atypical fast-slow atrioventricular nodal reentrant tachycardia (AVNRT) utilizing a variant of a SP extending to the superoanterior right atrium (RA).

Case Report

An 80-year-old woman with a history of multiple episodes of paroxysmal supraventricular tachycardia underwent electrophysiologic studies and a catheter ablation procedure. During the tachycardia, the 12-lead electrocardiogram showed a long RP tachycardia with biphasic P waves in leads II, III, and aVf. Atrial extrastimulation elicited no dual anterograde atrioventricular (AV) nodal conduction. During ventricular burst stimulation and ventricular single extrastimulation, retrograde conduction occurred via a fast pathway (FP) with a short ventriculoatrial (VA) interval, and the earliest atrial activation site in the region of the His bundle (HB) was observed. However, ventricular double extrastimulation reproducibly represented retrograde conduction via a pathway other than the FP, as evidenced by the difference in the atrial activation sequence followed by the initiation of a long RP tachycardia documented previously with an initial V-A-V activation sequence (Figure 1A and B). The atrial activation during the tachycardia was earlier in the HB recording than in the coronary sinus recordings; this finding is identical to the atrial activation immediately after ventricular double extrastimulation. Ventricular overdrive pacing during the tachycardia represented two types of response: a reproducible termination of the tachycardia without premature atrial capture (Figure 1C) and VA dissociation (Figure 2A). During the ongoing tachycardia, a second-degree AV block occasionally developed (Figure 2B). Atrial differential entrainment pacing was unsuccessful owing to the development of an AV block during the pacing. During isoproterenol infusion, retrograde conduction via the FP was also observed, and ventricular overdrive pacing induced the tachycardia probably following double atrial response.2) However, retrograde conduction via the slow pathway (SP) was not reproducible during ventricular burst pacing with the tachycardia cycle length, and ventricular entrainment capturing the atria with a 1:1 ratio was unsuccessful. A 4 mg bolus injection of adenosine triphosphate reproducibly terminated the tachycardia after a last ventricular event following the incremental prolongation of the atrial cycle length. The verapamil sensitivity of the tachycardia was not evaluated. Accordingly, when making a differential diagnosis of this long RP tachycardia, the exclusion of atrial tachycardia (AT) was determined by observing the V-A-V activation sequence on ventricular induction3) and the termination of the tachycardia by ventricular pacing without atrial capture4) (Figure 1). The exclusion of AV reentrant tachycardia using a slowly conducting accessory pathway was determined by the observations of VA dissociation during the ventricular burst stimulation of the tachycardia5) and the second-degree AV block during the tachycardia6) (Figure 2). This finding confirms the diagnosis of fast-slow AV nodal reen-
Figure 1. Intracardiac recordings of tachycardia induction with double extrastimulation from the RVA (A), VA conduction curve, and tachycardia termination with burst stimulation from the RVA (C). A: The first two-paced stimuli at an S1-S1 cycle length of 600 ms capture the atria (A1) with a short VA interval and the earliest site of atrial activation at the distal HB electrogram consistent with retrograde conduction over an FP. The S2 with an S1-S2 coupling interval of 400 ms also captures the atria (A2) via the FP with a decremental conduction delay in the VA interval. Immediately after the S3 at an S2-S3 coupling interval of 310 ms, retrograde conduction over the variant of SP appears (A3) (indicated by the dotted arrow) with a long VA interval and is followed by tachycardia initiation. Although not shown, single extrastimulation from the RVA did not induce tachycardia, thus further supporting that the A3 is not caused by the A2 but is attributed to the S3. This represents a V-A-V activation sequence that excludes the diagnosis of AT. The earliest atrial activation on the A3 occurs at the HB electrogram but differs from that during retrograde conduction via the FP, and the interval between the onset of atrial deflection in the HBE and the HRA (indicated by the horizontal bidirectional arrows) is shorter during retrograde conduction via the SP variant than via the FP, thus suggesting that the atrial breakthrough of the SP variant is superior to that of the FP. After the second atrial cycle of the tachycardia, the alternans of the tachycardia cycle length that depends on the alternans of the AV interval is visible with a fixed VA interval. B: Horizontal and vertical axes indicate S2-S3 and S3-A3 intervals in ms, respectively. The VA conduction curve consists of S3-A3 intervals with (indicated by the closed circles) or without tachycardia induction (indicated by the open circles). C: Tachycardia is sustained without spontaneous termination. Immediately after the fourth stimulus of the RV overdrive pacing of the tachycardia at an S-S CL of 360 ms, the tachycardia is terminated with an end of ventricular deflection; this finding is consistent with a retrograde conduction block in the SP variant. All recordings were obtained at baseline. I, II, and V1 indicates the surface electrocardiogram, HRA1-HRA2 indicates distal high RA, HBE1-HBE2 to HBE3-HBE4 indicates a distal to proximal HB region, and CS9-CS10 to CS1-CS2 indicates proximal to distal CS recording.
try tachycardia (AVNRT) using the FP as the antegrade limb and the SP variant as the retrograde limb of the reentry circuit. AV block development during the ongoing AVNRT could be explained by the anterograde block at the level of the lower common pathway (LCP) within the AV node.\textsuperscript{1,6} The responses during the ventricular burst simulation of the tachycardia seemed to depend on the retrograde conductivities at the LCP: when the retrograde wavefront passed via the LCP and was blocked at the SP, a termination of the tachycardia without atrial capture could occur; however, VA dissociation could occur when the retrograde wavefront was blocked at the LCP.\textsuperscript{2,5} Therefore, AV block during the tachycardia and VA block during the ventricular pacing of the tachycardia could co-exist according to LCP conductivities.\textsuperscript{7} Retrograde conduction via the FP during the ventricular pacing of the tachycardia also seemed to be masked according to LCP conductivities. Even if the retrograde wavefront passed via the LCP and penetrated into the FP, it might collide with the antegrade wavefront within the FP. An apparently fixed VA interval during the tachycardia accompanied by the alternans of the tachycardia cycle length was consistent with the AVNRT diagnosis (Figure 1A).\textsuperscript{8} These variabilities in the tachycardia cycle length may be attributable to the decremental properties of AV nodal pathways.\textsuperscript{9} Furthermore, retrograde conduction via the SP variant and decremental properties were reproducibly unmasked via ventricular double extrastimulation (Figure 1A and B).

The 3D activation mapping of the tachycardia revealed the earliest site at 11:30 in the free wall (FW) of the right atrium (RA) adjacent to the tricuspid annulus,\textsuperscript{10} where the first delivery of radiofrequency (RF) energy terminated the tachycardia following the development of an accelerated ectopic atrial complex and eliminated its inducibility (Figure 3). No recurrence of tachycardia was observed during a three-month follow-up.

**Discussion**

The tachycardia in the present case was characterized by the site of the earliest atrial activation in the superoanterior FW of the RA adjacent to the TA. This eccentric atrial activation during tachycardia with long RP intervals has been considered a sign that supports a diagnosis of adenosine-sensitive AT or verapamil-sensitive AT originating from the vicinity of the AV node\textsuperscript{11-15} or AV reentrant tachycardia (AVRT) incorporating slowly conducting AP.\textsuperscript{16} The detection of fragmented atrial potential at the HB region and the earliest site of atrial activation during the tachycardia might suggest microreentry as the mechanism of tachycardia. However, we believe that the standard criteria we applied confirmed the diagnosis of AVNRT by successfully excluding a diagnosis of AT and AVRT. The reproducible findings of a V-A-V activation sequence on the ventricular induction and the termination of the tachycardia by ventricular pacing without atrial capture (Figure

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**Figure 2.** Intracardiac recordings of the second-degree AV block during the ongoing tachycardia (A) and VA dissociation during RV overdrive pacing (B). A: The HB electrogram is indicated by H. B: During the RV overdrive pacing of the tachycardia at an S-S CL of 370 ms, VA dissociation is present, excluding AVRT. The numbers between atrial EGM at the high RA and between the pacing stimuli at the RVA indicate the CL in ms. All recordings were obtained at baseline. The other abbreviations are the same as those given in Figure 1.
and are anatomically continuous with the inferior extensions of the human retroaortic node behind the noncoronary aortic cusp of Valsalva.1) Inoue17) anatomically discovered superior extensions of the human compact AV node, which is hypothesized to be involved in “superior SP” conduction. Furthermore, several investigators suggested that the AV rings of nodal-like myocytes surrounding the tricuspid and mitral annuli converge in the retroaortic node behind the noncoronary aortic cusp and are anatomically continuous with the inferior extensions of the AV node, thus forming figure-of-eight rings of nodal and transitional cells.18,19) This continuity between the embryological development of the AV ring and the AV node from an identical origin, namely, the so-called embryonic AV canal.20,21) In humans, although the presence of the AV ring22) and a retroaortic node in the interatrial septum just behind the noncoronary aortic cusp23) has been histologically confirmed, the anatomic connection of the retroaortic node to the sinuatrial node has not been histologically confirmed, and the superior extensions to the retroaortic node, superior AV ring tissue, or both that forms the SP remains to be clarified. On the basis of our findings, we hypothesize that a primitive substrate of the SP variant in the present case was composed of the AV ring in the superoanterior RA, retroaortic node, and nodal-like cells present between the retroaortic node and superior nodal extensions.

To treat this AVNRT by RF ablation, we targeted the earliest site of atrial activation during the tachycardia as recorded at HBE1-HBE2 and ABL1-ABL2. A: Approximately 1 s after the onset of RF delivery, the tachycardia terminated with a last atrial activation in the ablated region (ABL1-ABL2) developed before returning to the sinus rhythm. B: The distance between the successful site (purple tag and unfilled arrow) and the nearest HB (yellow tags) measured was 16.7 mm. The other abbreviations are the same as those given in Figure 1.

Figure 3. Intracardiac recordings during tachycardia immediately before (A) and after RF delivery (RF on) to the successful ablation site (B). Fluoroscopic views of the position of the catheters in the RAO and LAO projections (C and D, respectively) showing the site of successful ablation (white arrows). CARTO activation maps of the RA during the tachycardia in the LAO views (E). A: ABL1-ABL2 and ABL3-ABL4 are the distal and proximal bipoles of the ablation catheter, respectively. The atrial electrogram at ABL1-ABL2 preceded the onset of the P wave by 45 ms, and its amplitude was greater than that of the ventricular electrogram. Fragmented atrial potential was recorded at HBE1-HBE2 and ABL1-ABL2. B: Approximately 1 s after the onset of RF delivery, the tachycardia terminated with a last ventricular electrogram, consistent with a retrograde conduction block over the SP. Ectopic atrial cycles (asterisks) with the earliest activation in the ablated region (ABL1-ABL2) developed before returning to the sinus rhythm. E: The distance between the successful site (purple tag and unfilled arrow) and the nearest HB (yellow tags) measured was 16.7 mm. The other abbreviations are the same as those given in Figure 1.
lar to the case of a typical SP, might be a reliable indicator of the heating effect on SP variants.

Conclusion
To the best of our knowledge, this is the first report on the successful ablation of an atypical fast-slow AVNRT using the SP variant extending to the superoanterior RAFW along the TA.

Disclosure
Conflicts of interest: None.

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