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Shift in the retrograde atrial activation sequence after radiofrequency catheter ablation in left variant atypical atrioventricular nodal reentrant tachycardia☆

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ABSTRACT

The role of left AV nodal (SVN) connections in the genesis of "left-variant" atypical atrioventricular nodal reentrant tachycardia (AVNRT) and those with multiple retrograde pathways remain unclear. We describe an unusual case of "left-variant" atypical AVNRT, where change in the retrograde earliest atrial activation site (REAAS) at the coronary sinus (CS) following radiofrequency catheter ablation (RFCA) was observed. Our observation suggests that the REAAS, that is, the left AVN connections, could participate in the formation of the reentrant circuit of "left-variant" atypical AVNRT. Furthermore, its atrial breakthroughs involved as a circuit of SVT could be (functionally) multiple.

Case presentation

A 49-year-old man with supraventricular tachycardia (SVT) was referred for radiofrequency catheter ablation (RFCA). His baseline ECG and ECG at SVT are shown in Fig. 1A and B. No structural heart disease was observed on transthoracic echocardiography. An electrophysiological study (EPS) was performed after obtaining written informed consent. The baseline conduction intervals were normal. Atrial extrastimuli showed a continuity in the atrio-ventricular (AV) conduction curve and could not induce SVT. Ventricular extrastimuli in the right ventricular apex (RVA) revealed the decrement ventriculoatrial (VA) conduction with the retrograde earliest atrial activation site (REAAS) at the coronary sinus (CS) 3–4. SVT was reproducibly induced by a single extrastimuli in the RVA followed by a "V–A–V" sequence without the VA jump-up (Fig. 1C). AV reentrant tachycardia (AVRT) involving the accessory pathway (AP) could be excluded on the following findings; (1) Left ventricular (LV) extrastimulus during His refractoriness was given without change in atrial timing activation. However, no His recording was available at that moment. His refractoriness was calculated taking in account SVT-cycle length (CL), basal His-ventricular interval (Fig. 2), (2) Administration of adenosine during ventricular pacing resulted in VA block, and (3) Entrainment from the RVA during SVT showed a stimulus-atrial–VA interval, a PPI–TCL, and a corrected PPI–TCL were 133 ms, 189 ms, and 187 ms, respectively [1]. AT could be excluded on the following findings; (1) Delta VA interval (defined as last captured ventricular electrogram to the REAAS after over drive pacing during the SVT) between the different atrial pacing sites was less than 14 ms [2], and (2) Ventricular overdrive pacing during the SVT entrained the tachycardia, and the tachycardia resumed with a "V–A–V" sequence after the last RVA pacing. On the basis of the above-mentioned findings, a diagnosis of atypical AVNRT was made. RFCA was applied to the right posteroseptal site. Although junctional rhythm occurred, the SVT remained inducible. Therefore, RFCA was applied at the REAAS (CS 3–4) transseptally, which resulted in the shift of the REAAS to the CS 7–8 (Fig. 3B). Subsequent RFCA at the REAAS (CS 7–8) eliminated the retrograde VA conduction, and resulted in the non-inducibility of SVT.

Commentary

The change in the REAAS during atypical AVNRT in which successful ablation was accomplished at the right posteroseptal site was reported [3]. However, the change in the REAAS observed in the previous report was noticed only during ventricular pacing. Therefore, whether the

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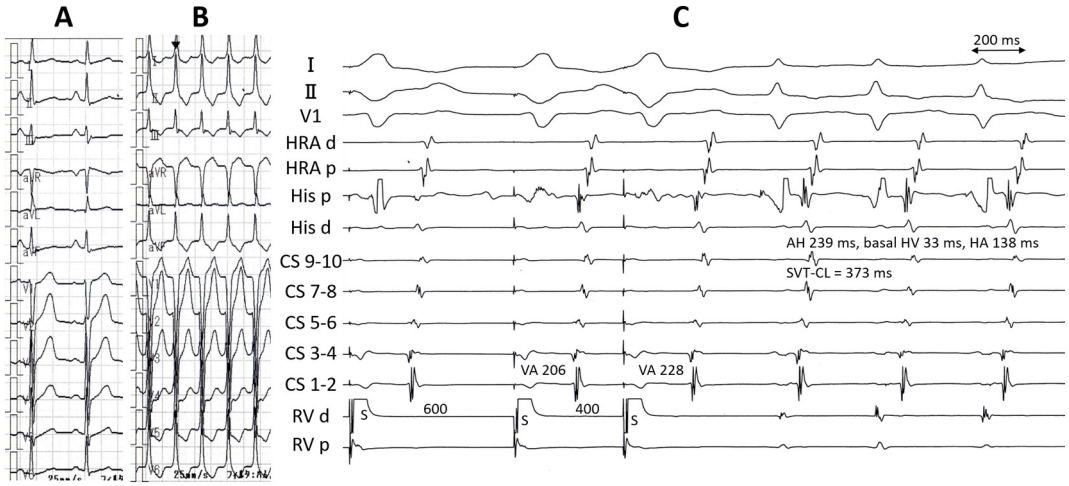

Fig. 1. (A) ECG at sinus rhythm. (B) ECG at SVT. (C) Intracardiac recordings at the initiation of the SVT. AH, atrio-His interval; CL, cycle-length; CS, coronary sinus; HRA, high right atrium; HV, His-ventricular interval; RVA, right ventricular apex; S, stimulus; SVT, supraventricular tachycardia; VA, ventriculoatrial interval; d, most distal pair; p, most proximal pair.

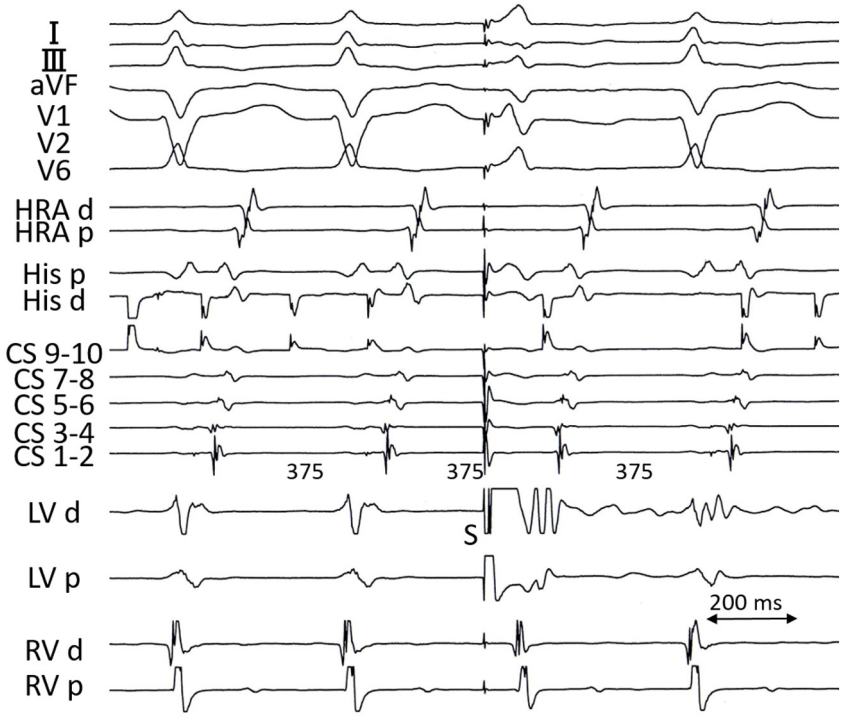

Fig. 2. Left ventricular extra stimuli during SVT when the His bundle was refractory could not reset the atrial electrograms. No His recording was available at that moment. His refractoriness was calculated taking in account TCL, basal HV. LV, left ventricular, other abbreviations as in Fig. 1.

REAAS was involved in the circuit of the AVNRT is unclear. Hwang et al. reported a similar case [4] but did not mention the catheter position of the CS and of the P-wave morphology on the surface ECG before and after the change in the REAAS. Although Nam et al. reported 2 cases in which multiple exits of the retrograde pathway might exist [5], they did not provide any figure to illustrate these cases. In our case, RFCA applied to the right posteroseptal site did not result in the non-inducibility of SVT. Furthermore, RF applied to the REAAS resulted in the elimination of retrograde VA conduction and in the non-inducibility of SVT. Therefore, our observations suggested that the REAAS, that is, the AVN connections to the LA, is involved in the formation of the reentrant circuit of "left-variant" atypical AVNRT. Furthermore, its atrial breakthroughs involved as a circuit of AVNRT could be (functionally) multiple.

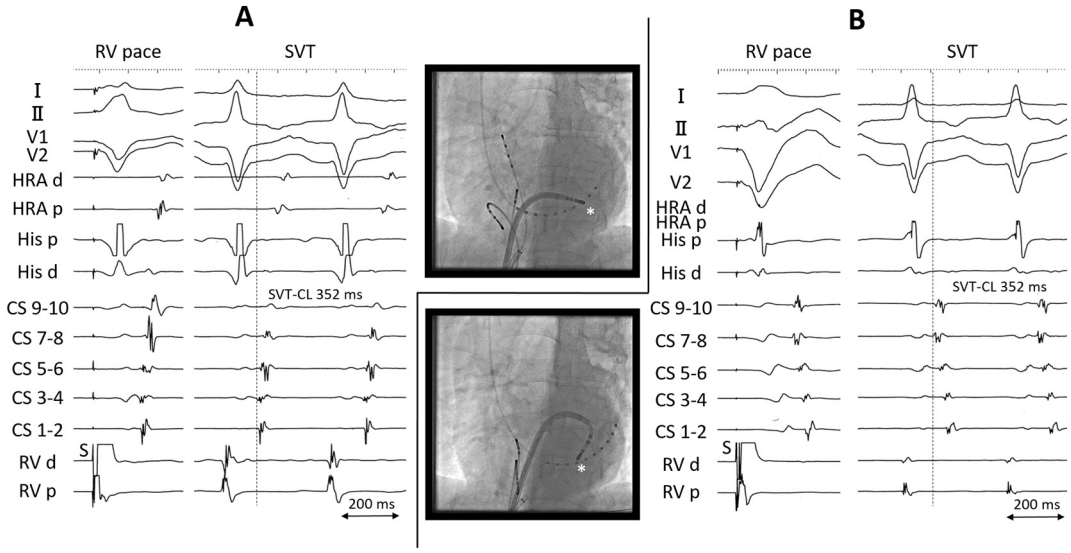

Fig. 3. (A) Left panel: The REAAS during RVA pacing is in the CS 3–4. Right panel: Recordings during SVT showing the REAAS in the CS 3–4, similar to the pattern in the left panel. (B) Left panel: The REAAS during RVA pacing is in the CS 7–8. Right panel: Recordings during SVT showing that the REAAS is in the CS 7–8, similar to the pattern in the left panel. The fluoroscopy image shows the position of the CS catheter, which was not displaced during the event. The retrograde P wave during SVT on the surface ECG slightly changed, although the SVT cycle length was unchanged, which suggests that the intra-atrial activation sequence had changed. The asterisk shows the REAAS. Abbreviations as in Fig. 1.

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