EP CASE REPORT

Regular Supraventricular Tachycardia with Irregular Atrial Activations

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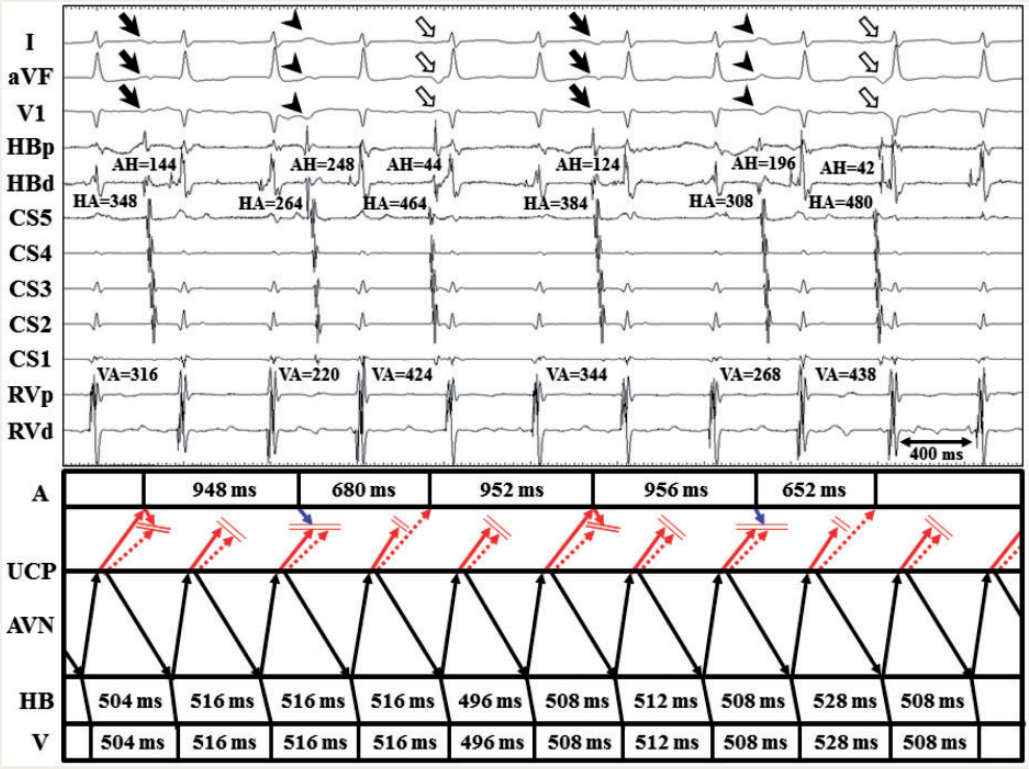
A 19-year-old woman developed a recurrent supraventricular tachycardia (SVT) 3 months after catheter ablation of a typical slow-fast type atrioventricular nodal re-entrant tachycardia (AVNRT). During the procedure, the slow pathway was mapped and ablated anterior to the bottom of the coronary sinus (CS) ostium. She was referred to our institute and underwent electrophysiological testing a year after the first ablation. At baseline, the atrio-His (AH) and His-ventricular (HV) intervals were normal. Rapid ventricular pacing revealed irregular atrial activations with variable ventriculoatrial (VA) intervals and P wave morphologies (Supplementary material online, *Figure-1*). Atrial extrastimulation induced a regular SVT following a sudden prolongation of the AH interval. During the SVT, VA block occurred with irregular atrial activations (*Figure 1*). A premature atrial contraction (PAC) that occurred during the His-refractory period advanced the following His bundle (HB) activation during the SVT (Supplementary material online, *Figure-2*). A premature ventricular stimulus delivered during the His refractory period did not reset the following HB activation during the SVT. Rapid ventricular pacing could terminate the SVT.



The differential diagnosis of this kind of SVT consisted of AVNRT with an upper common pathway, nodofascicular or nodoventricular tachycardia, and junctional tachycardia with retrograde block.[1,2] In this case, a nodofascicular or nodoventricular tachycardia using a concealed nodofascicular or nodoventricular pathway as the retrograde limb was excluded because a premature ventricular stimulus delivered during the His refractory period did not reset the SVT. A junctional tachycardia was unlikely for two reasons. First, programmed atrial stimulation could reproducibly induce the SVT and ventricular pacing could terminate the SVT. Second, a PAC occurring during the His-refractory period reset the following HB activation during the SVT. Therefore, an AVNRT with an upper common pathway was the most

likely mechanism of the SVT in this case. Catheter ablation targeting the slow pathway was performed anterior to the roof of the CS ostium, resulting in a stable junctional rhythm with an intact AV conduction. Thereafter, no SVTs were induced on or off an isoproterenol infusion.

In this case, there was VA block as well as an eccentric VA association unlike a Wenckebach type VA block (*Figure 1*). Three different atrial activations were recorded during the SVT, and the same atrial activations were recorded during ventricular pacing. During the two atrial activations (atrial activation-1; arrowhead and atrial activation-2; solid arrows), the atrial activations in the HB region were earlier than those in the proximal CS while during the other atrial activation (atrial activation-3; open arrows) that in the proximal CS was earlier than that in the HB region. Although the atrial activation was not recorded in the high right atrium, atrial activation-1 was consistent with a sinus beat. Atrial activation-1 was antegrade, but did not reset the SVT. Considering the VA intervals and atrial activation sequences, atrial activations-2 and 3 should have been retrograde. It has been reported that there are anterior and posterior inputs from the atria to the atrioventricular node.[3] Therefore, atrial activation-2 was likely to have conducted retrogradely through the anterior pathway, and atrial activation-3 through the posterior pathway.

Supplementary material is available at *Europace* online.

References

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