A long RP tachycardia with the earliest atrial activation at the His bundle region : What is the mechanism?

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Abstract

A 72-year-old female with frequent palpitation was referred for radiofrequency ablation. The baseline 12-lead electrocardiogram and echocardiography results were normal. At baseline, the atrio-His (AH) and His-ventricular (HV) intervals were 90 and 41 ms, respectively. Dual atrioventricular (AV) nodal physiology or ventriculoatrial (VA) conduction was not observed during programmed atrial and ventricular stimulation. After isoproterenol infusion, VA conduction became decremental and concentric, with the earliest atrial activation seen at the His bundle (HB) region during ventricular pacing. A supraventricular tachycardia with a long RP interval (SVT) was induced by atrial extra-stimulation, without any jump-up in the AH interval. During the SVT, the AH and HV intervals were 180 and 180 ms, respectively, and the earliest atrial activation was recorded in the HB region (Figure 1A). During the SVT, transient 2:1AV conduction was observed (Figure 1B). Ventricular overdrive pacing at a pacing cycle length (CL) of 360 ms was performed during the SVT with a CL of 390ms (Figures 2A and B). Based on these observations, what is the mechanism of this tachycardia?

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EP ROUNDS

Case presentation

A 72-year-old female with frequent palpitation was referred for radiofrequency ablation. The baseline 12-lead electrocardiogram and echocardiography results were normal. At baseline, the atrio-His (AH) and His-ventricular (HV) intervals were 90 and 41 ms, respectively. Dual atrioventricular (AV) nodal physiology or ventriculoatrial (VA) conduction was not observed during programmed atrial and ventricular stimulation. After isoproterenol infusion, VA conduction became decremental and concentric, with the earliest atrial

activation seen at the His bundle (HB) region during ventricular pacing. A supraventricular tachycardia with a long RP interval (SVT) was induced by atrial extra-stimulation, without any jump-up in the AH interval. During the SVT, the AH and HV intervals were 180 and 180 ms, respectively, and the earliest atrial activation was recorded in the HB region (Figure 1A). During the SVT, transient 2:1AV conduction was observed (Figure 1B). Ventricular overdrive pacing at a pacing cycle length (CL) of 360 ms was performed during the SVT with a CL of 390ms (Figures 2A and B). Based on these observations, what is the mechanism of this tachycardia?

Commentary

The differential diagnosis of a long RP tachycardia, with the earliest atrial activation at the HB region, includes atypical atrioventricular (AV) nodal reentry tachycardia (AVNRT), atrial tachycardia (AT), and orthodromic reciprocating tachycardia (ORT) using a slowly conducting AV accessory pathway or a concealed nodofascicular/nodoventricular (NF/NV) bypass tract (BT). The persistence of the SVT with a 2:1 AV block excluded the possibility of an ORT with a slowly conducting AV accessory pathway and a concealed NF/NV BT. As shown in Figure 2A, ventricular burst pacing did not accelerate the atrium to the pacing cycle length, and the SVT terminated without conduction to the atrium after the third fully-paced QRS complex. This finding is consistent with AVNRT and ruled out the possibility of an AT. Figure 2B shows the response to ventricular burst pacing with a delayed timing during the SVT. The SVT terminated without conduction to the atrium after the first fused paced QRS complex. The fused-paced QRS complex indicates a His-refractory timing. Therefore, this response to the ventricular pacing indicates the presence of a concealed NF/NV BT, but not necessarily its participation in the tachycardia circuit. The responses to these pacing maneuvers can be explained as follows: when ventricular burst pacing with a delayed timing was performed during the SVT (Figure 2B), the first His-refractory ventricular pacing impulse was conducted over the NF/NV AP and penetrated the slow pathway (SP). The conduction block in the SP resulted in tachycardia termination owing to the decremental properties of the SP. When ventricular burst pacing with an earlier timing was performed during the SVT (Figure 2A), the connection of the NF/NV BT to the SP was blocked, but the SVT persisted. The timing of the third ventricular pacing impulse being earlier than the His-refractory period resulted in tachycardia termination without conduction to the atrium, due to the conduction block in the SP via the right bundle branch-His conduction. These findings established the diagnosis of an atypical AVNRT, with a bystander NF/NV BT inserting into the retrograde SP.

We performed electroanatomical mapping to identify the earliest site of atrial activation during the SVT, and radiofrequency ablation was performed in the noncoronary aortic cusp of Valsalva just superior to the HB region, thereby eliminating the inducible tachycardias (Figure 3). Kaneko et al. reported that the mechanism of an atypical AVNRT, incorporated a superior-SP, located above Koch's triangle as the retrograde limb.[1] Thus, we diagnosed the SVT as an atypical AVNRT, with a bystander NF/NV BT inserting into the retrograde superior SP.

NF/NV BTs are rare accessory pathway variants connecting the AV node to the right bundle branch or right ventricle. Most of NF/NV BTs are reportedly inserted into the SP; hence, concealed NF/NV BT-associated supraventricular tachycardias can be eliminated by the ablation of the right or left inferior extensions of the AV node, regardless of their mechanism.[2,3] In this case, the nodal pathway of the NF/NV BT is the superior SP, because the successful SP ablation site was the noncoronary aortic cusp of Valsalva just superior to the HB region. Termination of the SVT without conduction to the atrium after the fused paced QRS complex favors an NV BT over an NF BT, because the collision site between antidromic and orthodromic wavefronts during the QRS fused period occurs in the ventricular myocardium. The present case highlighted that the precise mechanism of AVNRT, in which NVBT was attached to the superior SP, could be elucidated by adjusting the timing of ventricular burst pacing during tachycardia.

References

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FIGURE LEGENDS

Figure 1(A) Intracardiac electrogram during the tachycardia with the earliest atrial activation at the His bundle region. (B) Intracardiac electrogram during the tachycardia with 2:1 atrioventricular conduction. I, II, V1 = surface electrogram; HRA d and HRS p= distal and proximal high right atrium; His d and His p = distal and proximal His bundle region; CS d and CS p = distal and proximal coronary sinus; RV d and RV p = distal and proximal right ventricle; H = His bundle electrogram.**Figure 2**Intracardiac electrograms during ventricular burst pacing at a pacing cycle length of 360ms during the tachycardia with a cycle length of 390ms.

With an early timing.

With a delayed timing.

The numbers indicate the cycle lengths in ms. VP = ventricular pacing. The other abbreviations are as in Figure 1.**Figure 3**

Intracardiac electrogram at a successful ablation site. Radiofrequency ablation in the noncoronary aortic cusp of Valsalva just superior to the His bundle region terminated the tachycardia.

(B) Fluoroscopy image at a successful ablation site.

ABL = ablation catheter. The other abbreviations are as in Figure 1.