INTERESTING ELECTROCARDIOGRAM

COMPLEX CASE STUDY

A Supraventricular Tachycardia with A Challenging Diagnosis

ABDEL J. FUENMAYOR, MD

Arrhythmia and Electrophysiology Section, Cardiovascular Research Institute, University of The Andes, Mérida, Venezuela

ABSTRACT. We present the case of a young woman who suffered from paroxysmal episodes of tachycardia in spite of treatment with carvedilol and verapamil. In sinus rhythm the electrocardiogram (ECG) showed a 110-ms PR interval and normal QRS complexes without delta waves. The ECG recorded during the tachycardia displayed a regular, narrow complex, 210 bpm tachycardia without discernible P waves. In the electrophysiological study, in sinus rhythm, the AH interval was 58 ms and the HV 11 ms. A supraventricular tachycardia was induced with one extra-stimulus and presented the same morphology as the spontaneous clinical arrhythmia. Atrial activation was central. Electrophysiological maneuvers were applied to elucidate the correct physiopathological diagnosis and, finally, radiofrequency ablation eliminated the tachycardia. The electrophysiological maneuvers and traces are discussed.

KEYWORDS. electrocardiogram, atrioventricular, high right atrium.

Case presentation

A 21-year-old woman had been suffering from palpitations since her adolescence. Treatment with both carvedilol and verapamil had failed, and she had more than three episodes of tachycardia each month. The electrocardiogram (EKG) in sinus rhythm displayed a PR interval of 110 ms and a QRS complex of 80 ms, but did not display any delta waves. During the arrhythmia, the ECG showed a regular, narrow QRS complex tachycardia with a cycle length of 285 ms and without discernible P waves. After granting informed consent, the patient underwent an electrophysiological study so that an ablation could be performed.

In the electrophysiological study, in sinus rhythm, the AH interval was 58 ms and the HV interval 11 ms. The atrioventricular (AV) node conduction curve evidenced an AV nodal jump. The tachycardia was initiated by a premature atrial stimulus that, in the first beat, conducted to the AV node with an A2H2 interval of 200 ms and an HV interval of 11 ms that was identical to the HV recorded in sinus rhythm. From the second beat on, the tachycardia cycle length was 240 ms, the VA interval at the His recording was 25 ms, the AH was 200 ms, and the HV remained at 11 ms. The first atrial activation was always recorded with the His catheter (Figure 1). A ventricular stimulus delivered when the His bundle was refractory did not advance the next atrial depolarization (Figure 2). Ventricular stimulation at a cycle length that was 30 ms shorter than the tachycardia cycle length induced the resetting of the tachycardia (atrial–atrial timing perturbation > 10 ms) outside the transition zone (Figure 3). By applying shorter S2 coupling intervals during programmed atrial stimulation, atrial fibrillation was induced, and the HV interval did not prolong during the atrial fibrillation (Figure 4). After performing slow pathway ablation, the AV nodal jump disappeared, and the AV node effective refractory period increased by 40 ms. However, the AH and HV interval remained short, and the tachycardia was still inducible with the same pattern of atrial activation (Figure 2). This second tachycardia was slower and also began with an A2H2 interval of 200 ms and an HV interval of 11 ms. From the second beat on, the tachycardia cycle length was 310 ms, the VA interval at the His recording was 25 ms, the AH was 275 ms, and the HV remained at 11 ms. A ventricular stimulus delivered when the His bundle was refractory did not advance the next atrial depolarization in this second...
tachycardia either. While the patient was in sinus rhythm, we performed ventricular pacing and obtained a detailed mapping of the area where the earliest atrial activation was recorded during the tachycardia. During ventricular pacing, the earliest atrial activation was found to be at a spot 5 mm posterior to the proximal pair of electrodes of the His recording catheter. We interpreted this difference in the earliest atrial activation as an activation through an accessory pathway.

After explaining the AV node block risk to the patient and her relatives, they consented to having the ablation performed. We decided to apply radiofrequency at this spot during sinus rhythm by progressively increasing the power from 10 watts on. A standard, non-irrigated, 4-mm-tip ablation catheter was used for ablation. When the power reached 20 watts, the HV interval was prolonged to 30 ms. The radiofrequency ablation was sustained at 20 watts for 30 s more. Thereafter, the tachycardia was no longer inducible. After performing the procedure, the AH remained identical, but the HV continued to be 32 ms (Figure 5). After performing the ablation, antiarrhythmic drugs were withdrawn, and the patient has remained free of arrhythmia during a 24-month follow-up period. Let us analyze what the mechanism of the arrhythmia could be.

**Discussion**

The differential diagnosis of a regular, narrow QRS complex tachycardia with a central V-A activation pattern consists of an AV nodal reentrant tachycardia, an orthodromic AV reentrant tachycardia mediated by a septal accessory pathway, or an atrial tachycardia. Our patient presented evidence of a dual AV nodal physiology, and the electrophysiological maneuvers results (ventricular stimulus during His refractory period and ventricular pacing entrainment) did not support the diagnosis of a septal accessory pathway-mediated tachycardia.\(^1,2\) As the ECG of the patient did not show delta waves, we initially interpreted the short HV interval as a failure to record the His spike, and we believed that we were recording a right bundle branch spike. Indeed, the short HV interval could be the result of a fascicular–ventricular bypass tract, but in our case we did not observe any pre-excitation as has been described elsewhere with this type of accessory pathway.\(^3\) We then decided to ablate the tachycardia as an AV nodal slow–fast reentrant tachycardia. During the ablation we observed the usual signs of successful ablation of the slow AV nodal pathway (a junctional rhythm, prolongation of the AV nodal refractory period,
and disappearance of the dual AV nodal physiology); however, the tachycardia was still inducible, and the HV remained short. We then suspected the existence of an atrio-hisian accessory pathway because the difference between atrial activation during the tachycardia and the one obtained during ventricular pacing (during ventricular pacing, the earliest atrial activation was 5 mm posterior to the place where the earliest atrial activation was recorded during the tachycardia) suggested that the tachycardia could be mediated by an atrio-hisian pathway with the atrial insertion near the fast AV nodal pathway. Delivering radiofrequency at the earliest atrial activation position found during ventricular pacing resulted in prolongation of the HV interval to the low range of HV normal values and rendered the tachycardia non-inducible. These results suggest an antidromic atrio-hisian accessory pathway-mediated tachycardia. Indeed, after ablating in the right atrium, the HV prolonged to a normal value and this is not consistent with the recording of a right bundle which is not possible to ablate from the right atrium. Another explanation could be a parahisian atrial tachycardia but, had this been the case, we would not have observed an HV prolongation after performing an atrial ablation, as occurred in our patient, and we never observed any A-A-V response during the electrophysiological maneuvers.

We did not observe any HV prolongation during atrial fibrillation (Figure 4). This finding does not support the diagnosis of an atrio-hisian accessory pathway-mediated tachycardia according to what has been described.3 Along the same line, the AH interval during the tachycardias described above are longer than the AH observed during sinus rhythm, and the AH of the first beat of the tachycardia is shorter than the ones following. Both the persistence of the short HV and the relatively long and variable AH during tachycardia suggest that the bypass tract that mediated this antidromic tachycardia could have had decremental conduction properties.

Our case presents evidence of a supraventricular tachycardia mediated by an accessory pathway located as the bypass tracts described by Thomas James.4 The bypass tract produced a short PR and AH intervals, a very short HV interval, and no ECG evidence of pre-excitation. These characteristics have been described elsewhere for the atrio-hisian accessory pathway; however, in our particular case, the pathway also

Figure 2: Electrocardiogram(ECG) and intracardiac recordings at a speed of 100 mm/s during the tachycardia that was induced after performing a slow AV nodal pathway. The first beat of the tachycardia has an AH interval that is shorter than that of the subsequent beats. The activation sequence remains the same as in the first tachycardia. H: His bundle potential; V: ventricular potential; A: atrial potential. From top to bottom II and AVF surface ECG recordings, high right atrium (HRA), His proximal, middle and distal and coronary sinus recordings from proximal (9–10) to distal (1–2).
Figure 3: Ventricular stimulation at a cycle length that was 30 ms shorter than the tachycardia cycle length induced the resetting of the atrial intervals outside the transition zone. The tachycardia cycle length is 280 ms (left). Ventricular pacing at a cycle length 30 ms shorter than the tachycardia cycle length produce A-A shortening to the stimulation cycle length outside the transition zone. Recording description and speed as in Figures 1 and 2.

Figure 4: During atrial fibrillation the HV interval is not prolonged. Recording description and speed as in Figures 1 and 2.
displayed conduction properties that do not fit into what has been previously described for atrio-hisian bypass tracts. In the context of an arrhythmia like the one described above, the conventional electrophysiological maneuvers used to differentiate AV nodal reentrant tachycardias from orthodromic AV reentrant tachycardias do not seem to allow us to reach an accurate diagnosis.

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References