Case Report

Simple Maneuver to Unmask a Nonmanifest Accessory Atrioventricular Pathway: A Case Report

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ABSTRACT

We present the case of a 58-year-old patient admitted for wide QRS tachycardia (200 bpm). There was no delta wave on a 12-lead surface electrocardiogram during sinus rhythm. During the electrophysiological study, stimulation of the high right atrium and the proximal coronary sinus resulted in decremental anterograde conduction through the atrioventricular node, whereas pacing at the right ventricular apex led to retrograde conduction through the atrioventricular node. Conduction through an anterograde accessory pathway was revealed during pacing at the distal site of its atrial insertion. Some nonmanifest accessory pathways may be unmasked by differential pacing maneuvers.

RESUMÉ

Nous présentons le cas d’un patient de 58 ans admis en raison d’une tachycardie à QRS large (200 bpm). Aucune onde delta n’a été observée à l’électrocardiogramme de surface à 12 dérivations durant le rythme sinusal. Durant l’étude electrophysiologique, la stimulation de l’oreillette droite haute et du sinus coronaire proximal a entraîné une conduction antérograde décrémentielle passant par le nœud auriculoventriculaire, tandis que la stimulation à l’apex ventriculaire droit a entraîné une conduction rétrograde passant aussi par le nœud auriculoventriculaire. La stimulation d’une voie accessoire antérograde à distance de son point d’insertion dans l’oreillette a révélé une conduction.

Some accessory pathways (APs) are manifest as seen in Wolff-Parkinson-White syndrome, but many are concealed with only retrograde conduction present. Some rare APs are not even revealed by the usual maneuvers used for electrophysiological (EP) studies. Past publications have reported that conduction through APs may be blocked depending on the direction of wave-front propagation during atrial pacing.

We report a rare case of an anterograde “latent,” left lateral AP only revealed when pacing in the coronary sinus (CS) close to its atrial insertion, in a patient initially admitted for wide QRS symptomatic tachycardia.

Case

A 58-year-old patient was admitted to our intensive care unit for wide QRS tachycardia (200 bpm) that was poorly tolerated hemodynamically (Fig. 1A). Because ventricular tachycardia was initially suspected in the emergency department, and because clinical tolerance was poor, one single external cardioversion was delivered. The surface electrocardiogram was normal immediately after restoration of the sinus rhythm, although the presence of a short PR interval and the lack of septal q waves suggested pre-excitation. An EP study was conducted after informed consent had been obtained from the patient. Three multipolar catheters were inserted percutaneously and advanced under fluoroscopic guidance. The catheters were positioned as follows: high right atrium (HRA), His bundle electrogram, and CS. Programmed electrical stimulation was performed using atrial and ventricular trains of 8 S1 (cycle length of 600 and 400 milliseconds) followed by up to 3 extra-stimuli (not shorter than 200 and 150 milliseconds for the ventricle and the HRA, respectively). Pacing at the HRA and middle CS (dipole 5-6) led to decremental anterograde conduction through the atrioventricular (AV) node (AVN), with no jumps or pre-excited beats at baseline, even after administration of isoproterenol, until AV nodal refractoriness was reached (220-210 milliseconds) (Fig. 1B). Pacing at the CS also resulted in decremental and concentric retrograde conduction through the AVN with no jumps until refractoriness was reached at 240 milliseconds. We were not able to induce AV re-entry tachycardia (AVRT) or supraventricular tachycardia (SVT) at baseline or on isoproterenol with conventional pacing maneuvers at the HRA, middle CS, or ventricular apex. We applied bursts and
also pacing trains at 600 and 400 milliseconds cycle length completed with a single automatic decremental extra-stimulus (−10 milliseconds) from 360 to 200 milliseconds (unless the refractory period was reached). During continuous monitoring in the EP room, we recorded only 2 pre-excited beats (due to premature atrial contractions originating from the left atrium) with morphologic characteristics almost identical to the initial wide QRS tachycardia (right axis deviation, negative polarity in lateral leads, positivity in inferior leads, right bundle branch block pattern with a positive delta wave in V1) (Fig. 1C). We then decided to stimulate the left atrium at the distal tip of the CS catheter (dipole 1–2). Pacing at 2 V for 2 milliseconds did not capture the left ventricle but unmasked a permanent pre-excitation with the same morphology as the initial wide QRS tachycardia (Fig. 1D), whereas pacing more proximally (dipole 5–6) did not. The injection of isoproterenol allowed us to measure the anterograde refractory period of the AP at 280 milliseconds. The pacing maneuvers facilitated ablation of the AP using a 4-mm-tip nonirrigated catheter placed along the mitral annulus through a trans-septal puncture close to the distal part of the CS catheter where the pacing was delivered. The tip of the ablation catheter at the successful site recorded the following parameters: AV interval 32 milliseconds; V-Delta 12 milliseconds. The pre-excitation disappeared after 4 seconds of radiofrequency ablation at 40 W (total duration 120 seconds) (Fig. 1E). The ablation procedure was continued for a further 60 seconds using the same parameters for consolidation. CS pacing maneuvers, repeated after a waiting period of 30 minutes, no longer triggered pre-excitation. However, we detected atrial vulnerability with numerous premature atrial contractions originating at the left upper pulmonary vein. We therefore concluded that the initial wide QRS tachycardia could have been due to atrial tachycardia originating in the left atrium. At 1-year follow-up, the patient was free of symptoms.

**Discussion**

To our knowledge, this is a rare observation of a possible (not proven) antidromic AVRT due to an exclusively anterograde nonmanifest left lateral AP difficult to unmask by standard EP maneuvers.

At the time of the procedure, our main goal was to ablate this unusual AP. We think that it would have been impossible to ablate the AP without stimulating the atria near the AP insertion (distal CS). We therefore came up with this simple method, which we think may be of interest to other electrophysiologists. Even though the most realistic hypothesis of competition between the AVN and the AP is likely, other possible mechanisms should be mentioned.

Baseline 12-lead surface electrocardiogram showed a short PR interval with the absence of septal q waves, which may suggest pre-excitation. However, the absence of q waves in lateral leads is unusual for a left lateral AP. The apparent differences after the loss of pre-excitation may also suggest competition between the AVN and the AP. However, the absence of changes during incremental atrial pacing (long PR interval until the Wenckebach period) may support another hypothesis. Pacing within the middle CS may have resulted in the AVN “winning the race” and thus explain the lack of obvious pre-excitation. In contrast, pacing closer to the AP (distal CS) may help to reveal a greater degree of pre-excitation, but could also help to unmask an AP that is dependent on the wave-front direction. Interestingly, we believe that this AP may have had an oblique insertion. Although such a phenomenon has rarely been described, we concluded that the conduction block through the AP was dependent on the direction of atrial wave-front propagation (Fig. 2, A and B). Goldberger et al. reported 2 patients with latent decremental AP in which anterograde conduction was only manifest during AVRT. The authors suggested that the cause was the oblique course of the AP with its atrial insertion located anterior to the ventricular insertion. In these cases, the latent AP conduction may also have been due to the direction of atrial wave-front propagation associated with an impedance mismatch. This phenomenon was also very well described by Aiba et al.

Unfortunately, we did not use adenosine and we did not perform all of the pacing maneuvers needed (alternative ventricular pacing sites or pacing from the atrial His region) to definitely prove direction-dependent AP conduction. A waveform originating from the right atrium during antidromic AVRT may not necessarily suggest an oblique AP. However, the atrial vulnerability from the left upper pulmonary vein that we recorded shortly after AP ablation may support the hypothesis of an initial atrial tachycardia with pre-excitation. However, this will remain unproven as we did not induce sustained SVT.

Another study showed that some concealed left-sided APs can only be revealed with left ventricular pacing. In fact, pacing close to the ventricular insertion of the AP may help to promote retrograde AP conduction rather than VA nodal conduction. In our case, this technique was unnecessary as we found a simpler technique, which consisted of pacing close to the atrial AP insertion inside the CS. We did not use IV adenosine, which would have helped us toprecise the mechanism (oblique vs AVN competition) but not to realize the ablation.

In summary, AP ablation can sometimes be tricky, especially when there is no or only intermittent pre-excitation, no retrograde conduction via the AP, and no inducible SVT. In our case, we believe that the AP that we unmasked was responsible for the initial antidromic AVRT. APs can be unmasked easily by differential pacing maneuvers beyond the atrial insertion of the AP. We think that our novel technique may be of interest to reveal and then to ablate certain particular or rare APs.
Figure 1. (A) Twelve-lead ECGs. Wide QRS tachycardia at 200 bpm with a right axis, positivity in AVR. (B) HRA burst pacing reaching the anterograde Wenckebach period at 210 milliseconds on isoproterenol; no pre-excitation is visible. Bottom: EGMs showing pacing at the HRA site. (C) Sinus rhythm, normal axis, short PR, with 2 pre-excited beats. Bottom: EGMs with primoactivation of the distal CS (CS 1-2) during the pre-excited beats. (D) Pacing at the distal CS (dipole 1-2) helped to reveal the anterograde conduction through the AP. AP, accessory pathway; AVR, atrioventricular re- entry; CS, coronary sinus; ECG, electrocardiogram; EGM, electrogram; HRA, high right atrium.
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