A rare case report of variable degrees of atrioventricular block during atrioventricular nodal reentrant tachycardia

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Introduction

Typical atrioventricular nodal reentrant tachycardia (AVNRT) is the most common regular supraventricular arrhythmia in humans; the anatomical site of the circuit and the nature of the pathways involved has been described for a long time. Complete atrioventricular (AV) block during AVNRT is a rare but well-known condition.[1,2] To date, the site of the 2:1 block has not been well characterized. Usually, the presence or absence of a His bundle potential (HBP) in blocked beats is helpful in determining whether the block is occurring within or below the AV node. Vagal maneuvers as well as atropine can be used to distinguish the level of block.[3,4] The absence of an HBP in blocked beats or a recorded amplitude potential that is smaller than in conducted beats during a 2:1 AV block during AVNRT may be explained by a proximal block of the His bundle. In contrast, a block in the distal portion of the His bundle could result in an HBP with an amplitude similar to that of conducted beats.[5]

We present the case of a patient with typical slow-fast AVNRT and transient high-degree AV block with temporarily no escaped beats. We hypothesized that the origin of the patient's block was functional.

Case report

A 64-year-old woman underwent an electrophysiological study for frequent paroxysmal episodes of abrupt and regular palpitations. Both physical examination and transthoracic echocardiography were normal. There was no family history of cardiac arrhythmias or cardiac disease. Blood tests, including thyroid function, NT-proBNP, electrolytes, troponin, and D-dimer, were normal. Baseline 12-lead surface electrocardiogram (ECG) was normal.

KEY TEACHING POINTS

- High-degree atrioventricular (AV) block during atrioventricular nodal reentrant tachycardia (AVNRT) is very rare; 2:1 AV block is more frequent.
- Various degrees of AV block may be the consequences of a vagal phenomenon.
- The shorter the A-A intervals during AVNRT, the higher the degree of the AV block may suggest a functional mechanism.

During the electrophysiological study at baseline, after informed consent was obtained from the patient, AH and HV intervals were 120 ms and 35 ms in sinus rhythm, respectively. There were no AV conduction abnormalities, seeing as the antegrade AV nodal Wenckebach occurred with a cycle length (CL) of 400 ms and the 2:1 AV nodal block for a CL of 350 ms. A typical AVNRT was induced during atrial stimulation (6 paced beats [S1] at 500 ms CL + S2 at 340 ms), with an AH jump of 52 ms (Figure 1). The arrhythmia was characterized by a variable CL (from 368 to 412 ms). A right ventricular premature stimulation at His refractoriness did not reset the subsequent A-A intervals, confirming that there was no concealed accessory pathway (Figure 2A). Surprisingly, a few seconds after AVNRT induction, we recorded a sudden Luciani-Wenckebach (LW) periodicity in the AV relationship (Figure 2B), followed by a 1:1 conduction as the last A-A interval increased to 412 ms, and then a sudden high-degree AV block (Figure 2C). The changes that one can observe in "A on V" sequences (with A being recorded initially before and then after V) are in favor of a typical LW period during AVNRT. Then the AV conduction partially resumed in a 2:1 AV block fashion before spontaneously converting to a 1:1 AV conduction (Figure 3A). Upon measurement, we found that the shorter the A-A intervals (and therefore the AVNRT cycle length), the higher the degree of the AV block: for example, AVNRT with 1:1 conduction at 412–416 ms

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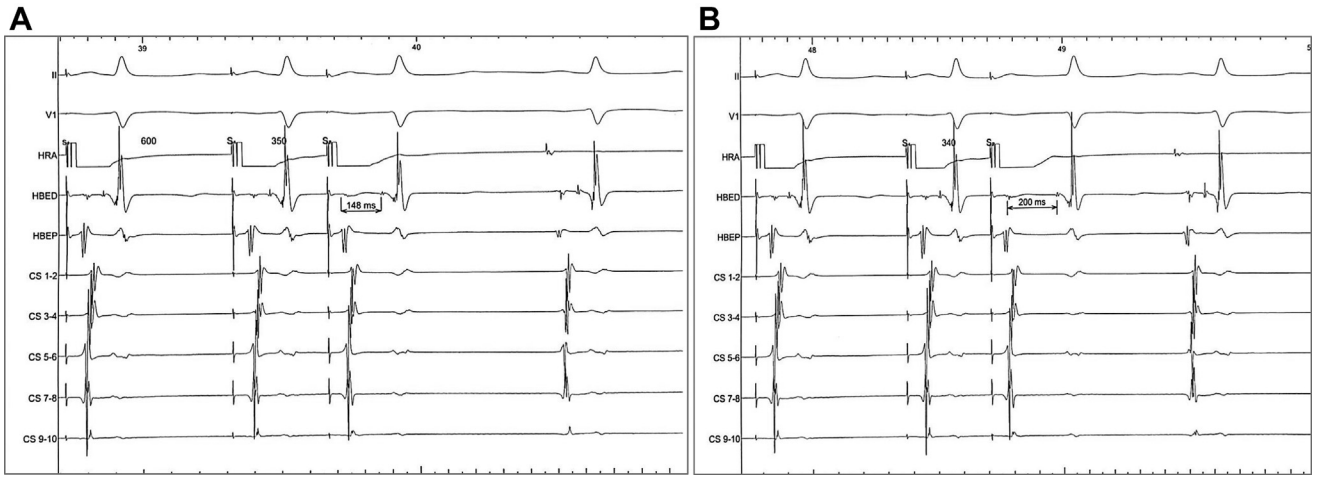

Figure 1 Electrocardiogram and intracavitary tracings. **A:** After 6 paced beats (S1) at 600 ms + S2 at 350 ms, atrioventricular (AV) conduction through the fast pathway with AH interval measured at 148 ms. **B:** After 6 paced beats (S1) 600 ms + S2 at 340 ms, AV conduction through the slow pathway (jump of 52 ms) with AH interval measured at 200 ms, without nodal echo. CS = coronary sinus; HBED = His bundle electrogram, distal; HBEP = His bundle electrogram, proximal; HRA = high right atrium.

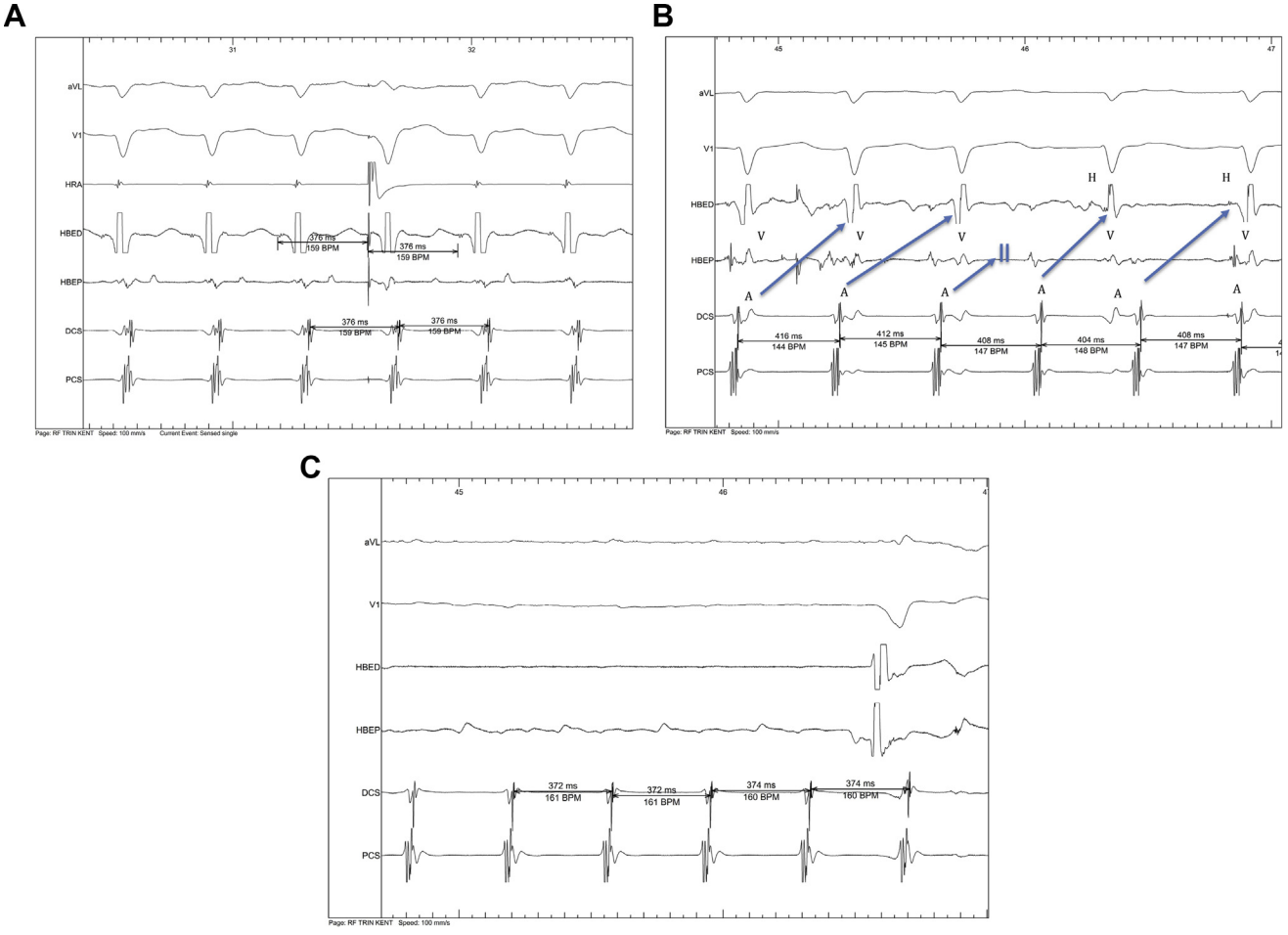

Figure 2 Electrocardiogram and intracavitary tracings. **A:** Right ventricular premature stimulation (HRA lead was placed on the right ventricular apex), exactly at His refractoriness, did not reset the subsequent A-A and H-H intervals. **B:** Atrioventricular nodal reentrant tachycardia (AVNRT) with a sudden typical Luciani-Wenckebach periodicity in the AV relationship. A-A intervals were slightly shorter (from 416 to 408 ms). **C:** AVNRT with a high-degree nodal AV block. A-A intervals are even shorter at this time (372–374 ms). A = atrium potential; DCS = distal coronary sinus; H = His bundle potential; HBED = His bundle electrogram, distal; HBEP = His bundle electrogram, proximal; HRA = high right atrium; PCS = proximal coronary sinus.

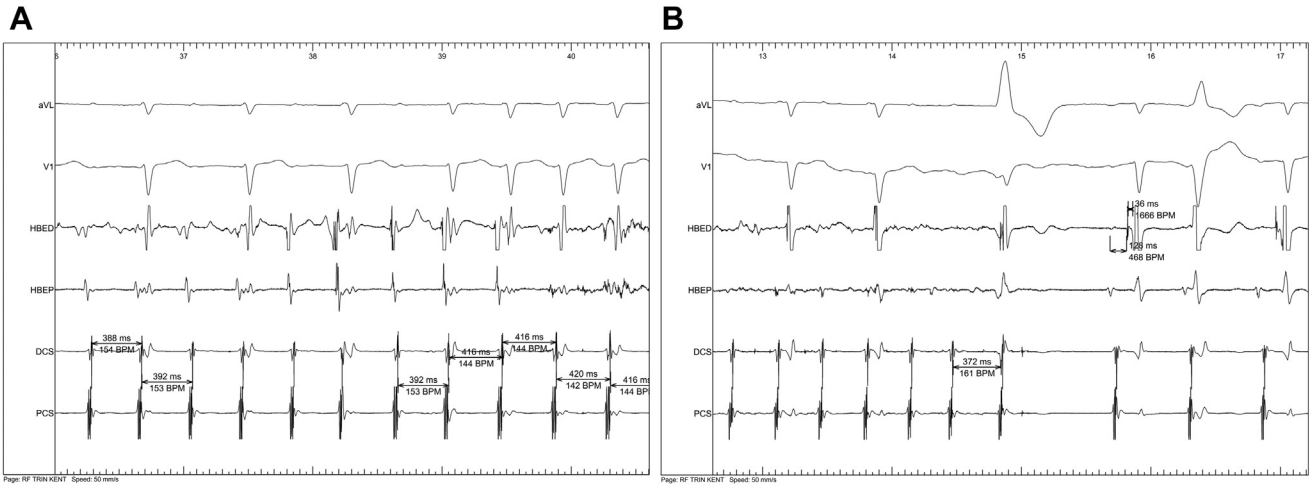

Figure 3 Electrocardiogram and intracavitary tracings. **A:** Atrioventricular (AV) conduction partially resumed in a 2:1 AV nodal block before converting spontaneously to a 1:1 AV conduction. Interestingly, the A-A intervals were prolonged immediately before restoration to 1:1 AV conduction (from 392 to 416 ms). **B:** A 2:1, then 3:1, AV conduction block with a ventricular premature beat that stop the tachycardia with normal AV conduction in sinus rhythm (AH: 128 ms, HV: 36 ms). DCS = distal coronary sinus; H = His bundle potential; HBED = His bundle electrogram, distal; HBEP = His bundle electrogram, proximal; HRA = high right atrium; PCS = proximal coronary sinus.

CL, 2:1 AV conduction at 404–408 ms, and high-degree block at 372–374 ms. We were also able to record a 3:1 AV conduction block at 372 ms CL with a premature ventricular beat that stopped the tachycardia and restored a perfectly normal AV conduction in sinus rhythm (AH: 128 ms, HV: 36 ms) (Figure 3B). No HBP was recorded when ventricular beats were missing. Following successful slow pathway cryoablation (located at the anterior part of the coronary sinus ostium), tachycardia was no longer inducible. Once the ablation was completed, we did perform atrial stimulations at different cycles to ensure that the AV conduction was normal. We observed an LW period at 395 ms and 2:1 nodal block at 385 ms (which was the same as it was before ablation), but no complete AV block. All the surface ECGs that were done during the hospital stay and before discharge were normal. During the 12 months of follow-up, the patient remained free of symptoms and was not required to take antiarrhythmic agents. We did not find any conduction trouble on the ECG during the follow-up.

Discussion

Occurrence of a 2:1 AV block during AVNRT is a well-known phenomenon. It is thought to occur in about 4%–10% of induced AVNRT episodes.[5] Although ablation in the AV junctional area has a high success rate in AVNRT, the exact localization of the tachycardia circuit is not well established. The notion of a lower common pathway (LCP), corresponding to AV nodal tissue between the reentrant circuit and the His bundle, has been utilized to explain the phenomena of AV block without recording of a His electrogram as well as retrograde Wenckebach periodicity during AVNRT.[6,7] Over the past decade, the question of whether or not AV nodal tissue is sandwiched between the AVNRT circuit and the His bundle has been debated. Jackman and colleagues[8] have reported observations suggesting that there is no LCP in typical AVNRT. Conversely, Anselm and colleagues[6] hypothesized that an LCP might exist because simultaneous impulse propagation from the lower part of the circuit to the atrium and the His bundle through AV nodal tissue during AVNRT have been recorded. It has been suggested that the LCP could be present in up to 78% of patients.[6] However, Ching Man and colleagues[5] reported that AV block during AVNRT without recording activation of the His bundle could also be explained by proximal intra-Hisian block. The authors conclude that the lack of a response to atropine and the consistent conversion of 2:1 block to 1:1 conduction by a ventricular extrastimulus may indicate that a proximal and functional Hisian block could exist (regardless of the presence or absence of an HBP in blocked beats). The HBP is absent in blocked beats in 40% of patients with 2:1 AV block. In the remaining patients, the amplitude of the recorded HBP may range from rudimentary (which makes it sometimes very difficult to record) to large. Therefore, irrespective of the presence of a recorded HBP, the level of the block can be located at the LCP or at the proximal portion of the His bundle.[5] This has been shown at the onset of very fast AVNRT, which may expose the His-Purkinje tissue to long–short periods and can lead to functional phase 3 block.[7] In our case, we think that the absence of a detectable HBP in the blocked beats does not add to the proof for the existence of an LCP, as it can be explained by either a proximal intra-Hisian block or a block in the LCP.

In this observation, we found that the shorter the A-A intervals (and therefore the AVNRT cycle length), the higher the degree of the AV block. This may suggest that the AV block was functional, whether it was located in the nodal tissue or proximally in the intra-Hisian area. Unfortunately, we were not able to perfuse atropine in order to clarify the mechanism. Rapid atrial pacing after ablation did not induce a complete AV block; therefore, we think that this could have

been the consequences of a vagal phenomenon during AVNRT.

Conclusion

Here we describe a rare case of spontaneous high-degree AV block occurring during an induced typical AVNRT. The shortened A-A intervals that preceded each episode of block and the prolonging of A-A intervals immediately before AV restoration were in favor of a functional mechanism. The absence of HBP recorded during the AV block may have been the result of a nodal or proximal Hisian block.

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