Clinical Case Report

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Case report: an unstable wide QRS complexes tachycardia after ablation of a poster-septal accessory pathway

What is the mechanism?

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Abstract

Introduction: Differentiation of wide QRS complex tachycardia required repeated electrophysiological stimuli and mapping. However, instability of tachycardia would increase the difficulty in differential diagnosis.

Symptoms and Clinical findings: In this paper, we reported a wide QRS tachycardia following ablation of an atrioventricular reentrant tachycardia participated by a poster-septal accessory pathway. Limited differentiation strategy was performed because the wide QRS tachycardia was self-limited and with unstable hemodynamics. We analyzed the mechanism of the wide QRS tachycardia by only 4 beats ventricular overpacing. On the basis of the last ventricular pacing, an atypical atrioventricular nodal reentrant tachycardia was confirmed.

Intervention and Outcomes: After slow-pathway modification, the wide QRS tachycardia was eliminated.

Conclusion: It was an atypical atrial-ventricular node reentrant tachycardia with right bundle branch block. Reasonable analysis based on electrophysiological electrophysiologic knowledge was the basis of successful diagnosis and treatment.

Abbreviations: AP = accessory pathway, AVN = atrioventricular node, AVNRT = atrioventricular nodal reentrant tachycardia, AVRT = atrioventricular reentrant tachycardia, EP = electrophysiology, FP = fast pathway, LB = left bundle branch, PPI = postpacing interval, PSVT = paroxysmal supraventricular tachycardia, RB = right bundle branch, SP = slow pathway, TCL = total circle length, VT = ventricular tachycardia, WCT = wide QRS-complex tachycardia.

Keywords: catheter ablation, paroxysmal supraventricular tachycardia, poster-septal accessory pathway, wide QRS tachycardia

1. Introduction

Wide QRS-complex tachycardia (WCT) may be a ventricular tachycardia (VT) or a supraventricular tachycardia (SVT) with intraventricular conduction disturbance or with a pre-excitation. Despite the existence of several established electrophysiology (EP) protocols for differentiation of WCTs, it required repeated inducibility and stability of WCT. Lots of factors would increase the difficulty of differentiation of WCT such as hemodynamic instability, self-limitation, or multi-morphology. In this paper, we reported a wide QRS tachycardia following ablation of an atrioventricular reentrant tachycardia (AVRT). Because of self-limitation and hemodynamic instability, there are only limited clues for differential diagnosis.

2. Case presentation

A 56-year-old man with regular narrow QRS complex tachycardia without any structural heart disease was referred to our center for electrophysiological study (EPs) and catheter ablation. This clinic operation was approved by the ethics committee of Zhejiang Province People's Hospital. Standard informed consent was given before the EP study. A baseline electrophysiological test demonstrated extraordinary antegrade and retrograde conduction. During ventricular stimuli, the earliest atrial activation occurred from the CS 9,10 region with no decremental property.

The clinical tachycardia (total circle length, TCL = 370 ms) was repeatedly inducible after atrial S1S2 stimulation (450/300 ms) with A-H jumped 160 ms. The earliest retrograde atrial activation occurred from the $CS_{9,10}$ region during the tachycardia (see Fig. 1A and B) V-A-V sequence was observed after ventricular overdrive pacing during tachycardia. Ventricular extrastimuli during His refractory period terminated the tachycardia (see Fig. 1C).

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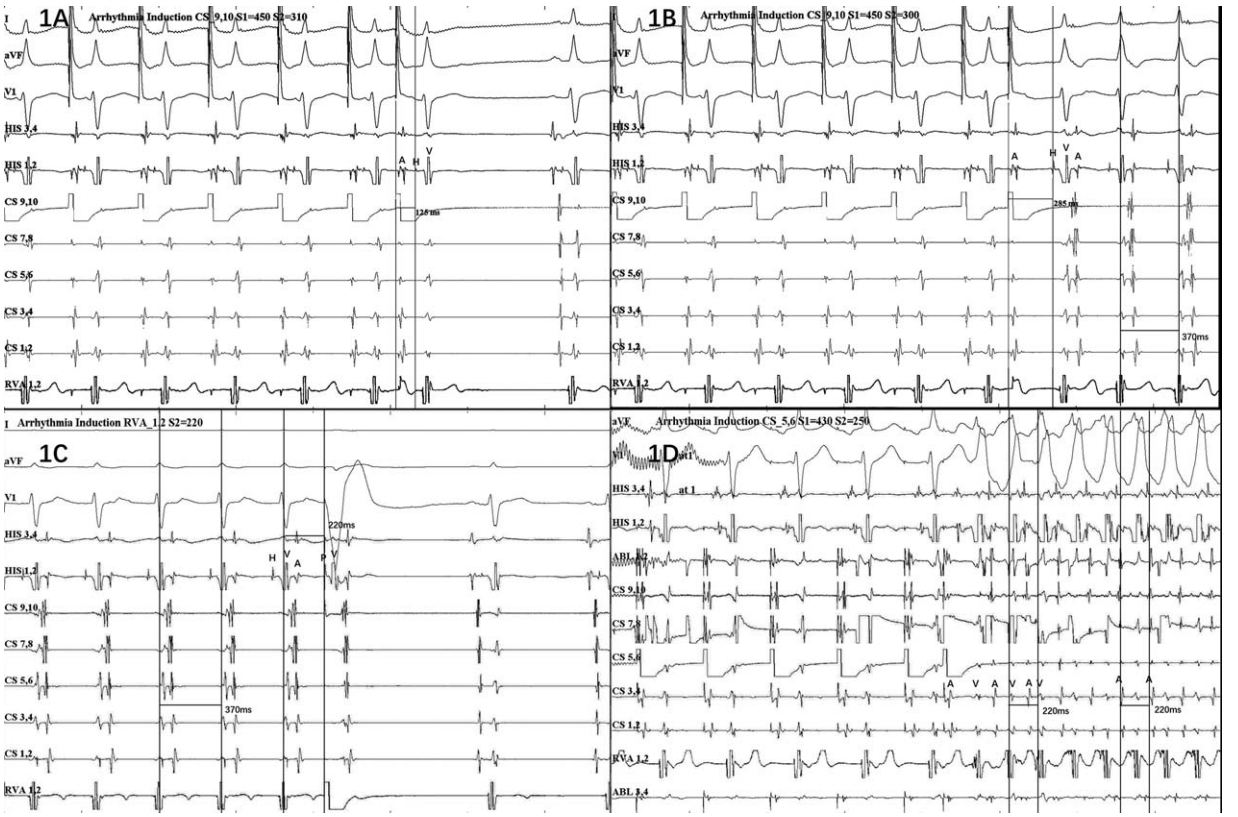

Figure 1. (A and B) (both speed 100 mm/s) showed the S1S2 atrial program stimuli during EP study. After atrial S1S2 stimuli in 450/310 ms, the A-V interval was 125 ms. When atrial S1S2 stimuli in 450/300 ms, A-V interval jumped to 285 ms, and tachycardia (TCL = 370 ms) was induced. The earliest A was from CS9,10 region. (C) (speed 100 mm/s) showed RS2 stimuli during his bundle effective refractory period (ERP) terminated tachycardia. (D) (speed 100 mm/s) showed a wide QSR tachycardia was induced by atrial S1S2 stimuli after AP ablation. The TCL of tachycardia was 220 ms. The earliest A wave was from his region and V: A was 1:1.

Ablation was performed in and around the CS orifice but did not affect the VA retrograde conduction; then, ablation at the MV annulus was performed via retrograde way. No clinical tachycardia was induced by programmed stimuli after ablation. Isoproterenol was used intravenously and programmed stimuli was repeated. A wide QRS complex tachycardia (TCL = 220 ms) was induced by atrial S1S2 stimuli (430/250 ms) with complete right bundle branch block morphology and 1:1 VA conduction (see Fig. 1 D); the earliest A wave was from His region. What is the mechanism of the tachycardia?

VT was first suspected and ruled out. The WCT was induced by supraventricular stimulation, which has reduced the possibility of ventricular arrhythmias. During differential diagnosis (ventricular overdrive-pacing, S1S1 200 ms), hemodynamic instability occurred and the tachycardia terminated spontaneously. Only 4 beats of ventricular stimuli were given. After ventricular overdrive-pacing, the CRBBB WCT turned to a narrow QRS tachycardia, which was described as Wedensky phenomenon. VT was excluded from the diagnosis.

A narrow QRS tachycardia with long RP interval could be atrial tachycardia (AT), atypical atrial ventricular node reentrant tachycardia (AVNRT), or a AVRT with slow access pathway. Only the last ventricular pacing captured atrium, and after that, a V-A-V pattern was observed. The possibility of AT was ruled out (see Fig. 2).

Was it an atypical AVNRT with slow pathway retrograde conduction or a AVRT with post-septal slow AP retrograde conduction? We draw conclusions from the following measurements: First, on $CS_{9,10}$, the V-A interval of tachycardia and the last ventricular pacing was 138 and 168 ms, respectively; Second, the H-A interval of tachycardia and the last ventricular pacing were almost the same. On the basis of these evidence, we inferred that the tachycardia was an atypical AVNRT (the mechanism of this case is described in Fig. 3). After slow pathway modification, no tachycardia could be induced again.

3. Discussion

AVRT combined with AVNRT was uncommon but not rare in clinical practice.[1,2] In most patients with AVRT, tachycardia is re-entrant utilizing the normal AV node pathway for antegrade conduction and an anomalous accessory pathway (AP) for retrograde conduction.[3,4] If a patient with AP also had dual A-V nodal pathways, then several re-entrant loops might be possible and make diagnosis difficult.

In this case, we demonstrated such multi-reentrant loops. At beginning, EPs demonstrated tachycardia participated by poster-septal AP in retrograde conduction and SP in anterograde conduction. After AP ablation, a WCT was induced. Now, we knew it was an atypical AVNRT.

The diagnosis of WCT was the difficult point of the case, because it was self-terminated and hemodynamic unstable. During WCT, only 4 beats of ventricular pacing were performed. Fortunately, the last ventricular pacing successfully captured

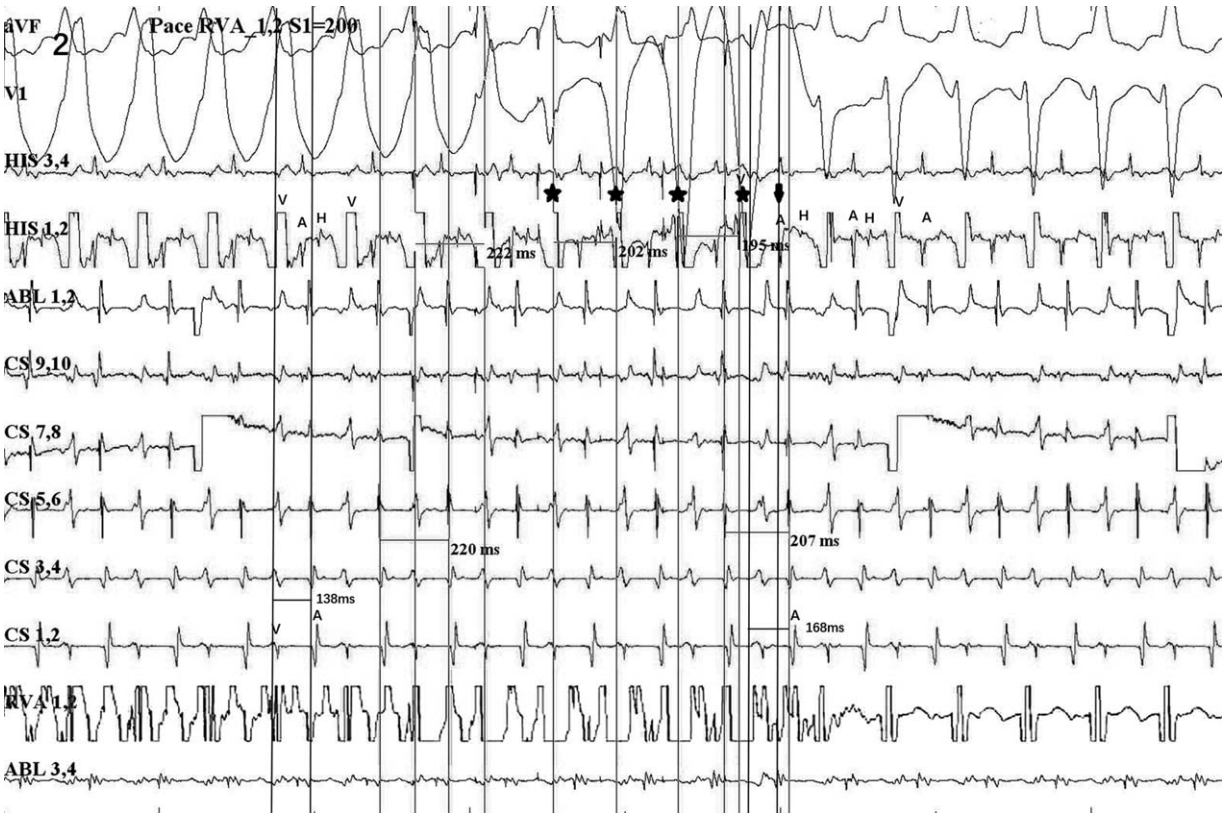

Figure 2. (Speed 100 mm/s) showed ventricular overpacing (S1S1 = 200 ms, shown as black stars) during wide QRS tachycardia (TCL = 220 ms). The continuous 3 beats of ventricular pacing did not capture atria. The last one successfully accelerated atria (A-A interval changed from 220 to 195 ms, shown as black arrow). After pacing, a V-A-V sequence was observed and tachycardia changed into narrow QRS. The V-A interval of tachycardia and the last ventricular pacing was 138 and 168 ms, respectively, and H-A interval of tachycardia and the last ventricular pacing were 158 and 163 ms, respectively (not shown in the figure).

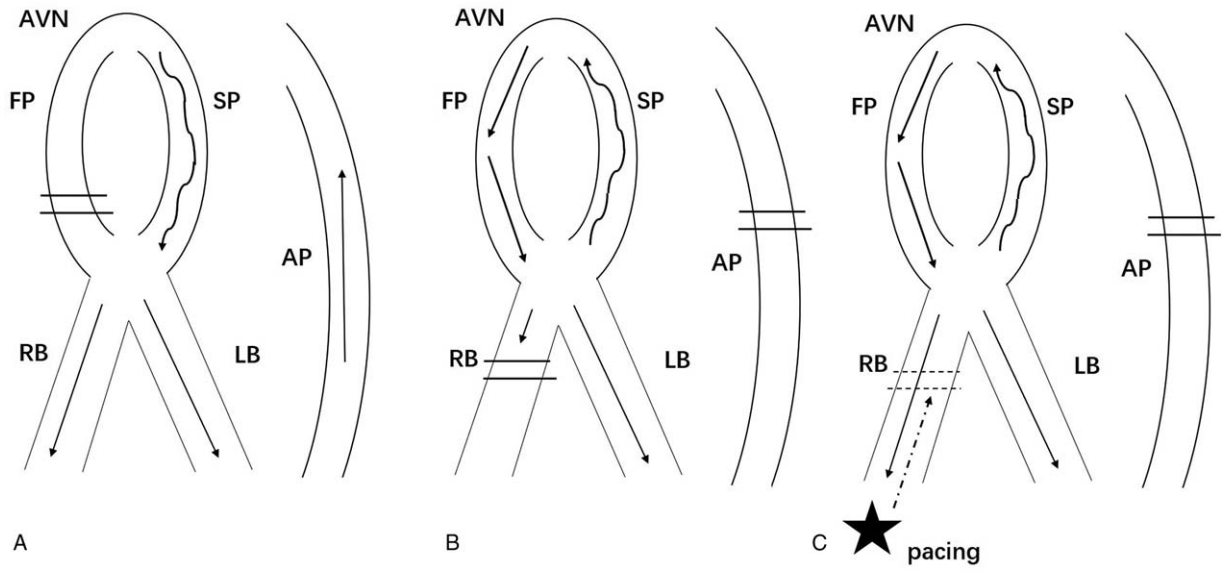

Figure 3. The mechanisms of this complicated tachycardia case. (A) The SP-AP reentrant tachycardia (TCL = 370 ms), which was the first induced tachycardia in the paper; (B) The second FP-SP reentrant tachycardia (TCL = 220 ms), which showed a CRBBB morphology because of distal right bundle branch functional blocked (black double solid lines); (C) Ventricular pacing (black dotted arrow) facilitated the conduction of distal right bundle branch, downregulated the activation threshold of blocking position (black double dotted lines). Thus, the superior activation could pass through (black solid arrow) and QRS of tachycardia turned to narrow. AP = access pathway, AVN = atrioventricular node, FP = fast pathway, LB = left bundle branch, RB = right bundle branch, SP = slow pathway.

atria. This was the prerequisite for the subsequent correct diagnosis.

As mentioned above, we found evidence to support our diagnosis, which considered the WCT was an atypical AVNRT:

1. The V-A intervals during tachycardia and ventricular pacing were 138 and 188 ms, respectively. In AVRT, the V-A intervals during both tachycardia and ventricular pacing are equal to the conducting time of AP; but in AVNRT, the atrium and ventricle are activated in parallel, while during entrainment, their activation is in series.[4] Thus, the differences between V-A intervals indicated the mechanism of AVNRT.
2. H-A interval during pacing (163 ms) was almost equal to H-A interval during tachycardia (158 ms), which was proved to be a strong evidence of AVNRT too.[5] In AVRT, H-V-A was activated in series, but in pacing, the activation sequence changed into H-A, which meant that H-A in pacing would be shorter than in tachycardia. On the contrary, in AVNRT, H-A in pacing would be almost equal to H-A in tachycardia.

There was a trap that should be mentioned. In this case, only the last ventricular pacing captured atria. It was important to notice the change of A-A interval during ventricular pacing. It was misdeemed that all of the 4 beats ventricular pacing were successfully captured atria; after successful entrainment, postpacing interval (PPI)-TCL<100 ms (280 to 220 ms) was the evidence of AVRT.[3] As only the last ventricular pacing captured atria, it could not be considered as a successful entrainment, which was the basic condition of PPI measurement.

4. Conclusion

In this case, the later WCT was difficult to be diagnosed, because differential protocols were not possible due to unstable hemodynamics and self-termination. This case highlighted the value of ventricular overdrive pacing as a simple and useful method to differentiate AVNRT from AVRT. Just 1 beat of ventricular pacing that can accelerate atrium could provide valuable evidence for clinical practice. On the contrary, it also highlighted the importance of understanding of basic EP concept as entrainment. Mistaken understanding of entrainment would lead to a wrong diagnosis.

Author contributions

Conceptualization: H. Wang.
Data curation: H. Wang.
Formal analysis: H. Wang.
Methodology: X. Che.
Writing – original draft: H. Wang.
Writing – review & editing: X. Che.

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