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A regular wide QRS complex tachycardia with fusion beats?

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1 | CASE PRESENTATION

A 40-year-old man with a 1 year history of sudden onset rapid palpitations was referred to the Emergency Room by general practitioner after documenting self-terminating wide QRS complex tachycardia (Figure 1A). The tachycardia exhibits a typical right bundle-branch block (RBBB) morphology and right axis, and occasionally QRS complexes (asterisks) show subtle morphological changes that are much more apparent in lead V3 (Figure 1A). Baseline 12-lead electrocardiogram (Figure 1B) and echocardiography were normal. He was not taking any medication. What is the most likely mechanism of the tachycardia? How the patient should be managed?

2 | DISCUSSION

The electrocardiogram in Figure 1 shows a regular wide QRS complex tachycardia at 187 bpm and QRS duration of 125 ms On the other hand, the baseline electrocardiogram shows normal sinus rhythm and narrow QRS complexes with a PR interval of 200 ms and no ventricular preexcitation.

There are three potential mechanisms to consider in wide QRS complex tachycardia:

1. Ventricular tachycardia (VT).
2. Supraventricular tachycardia (SVT) with aberrant conduction caused by either preexisting bundle-branch block or functional bundle-branch block induced by the fast heart rate.
3. Preexcited tachycardia (SVT with an atrioventricular (AV) conduction over an accessory pathway).

Because the tachycardia exhibited a typical RBBB morphology and right-axis deviation, there are only two possibilities to consider: VT with the onset of ventricular activation near the left anterior fascicular Purkinje system (idiopathic left anterior fascicular VT)[1] and SVT with aberrant conduction. Both entities can be easily mistaken since the QRS complex morphology of idiopathic fascicular VTs can resemble supraventricular QRS morphology as they also mainly occur in patients with structurally normal heart and they rapidly engage the His-Purkinje network. Distinguishing these entities is crucial for appropriate management.

There are numerous criteria and algorithms for differentiation of wide QRS complex tachycardias. Among all electrocardiographic features, AV dissociation is considered a hallmark for VT.[2] Nevertheless, its electrocardiographic absence does not rule out VT. When the atrial activity is observed and ventriculoatrial dissociation or second-degree ventriculoatrial block is present (atrial activity independent and/or slower of ventricular activity), it strongly suggests a ventricular origin of the tachycardia. If the atrial activity is not easily observed, the next step in the search for possible AV dissociation is to see if there are morphological changes in the QRS complexes. These changes are caused by partial (fusion) or complete (capture) depolarization of the ventricular myocardium by a sinus beat, which remains dissociated during VT and is conducted antegrade over the normal conducting system. In the present case, fusion beats (Figure 1A, asterisks) might explain the morphological changes in some QRS complexes during tachycardia; therefore, his referring physician considered idiopathic left anterior fascicular VT as the most likely diagnosis. Although idiopathic fascicular VTs frequently respond to verapamil and have good prognosis, catheter ablation offers curative therapy. However, in this case, since the morphology of the marked beats is identical to all the others, this should never be considered as fusion beats. Actually, this is electrical alternans where only the amplitude of the QRS changes, not the morphology, and indeed, the HV interval remained constant.

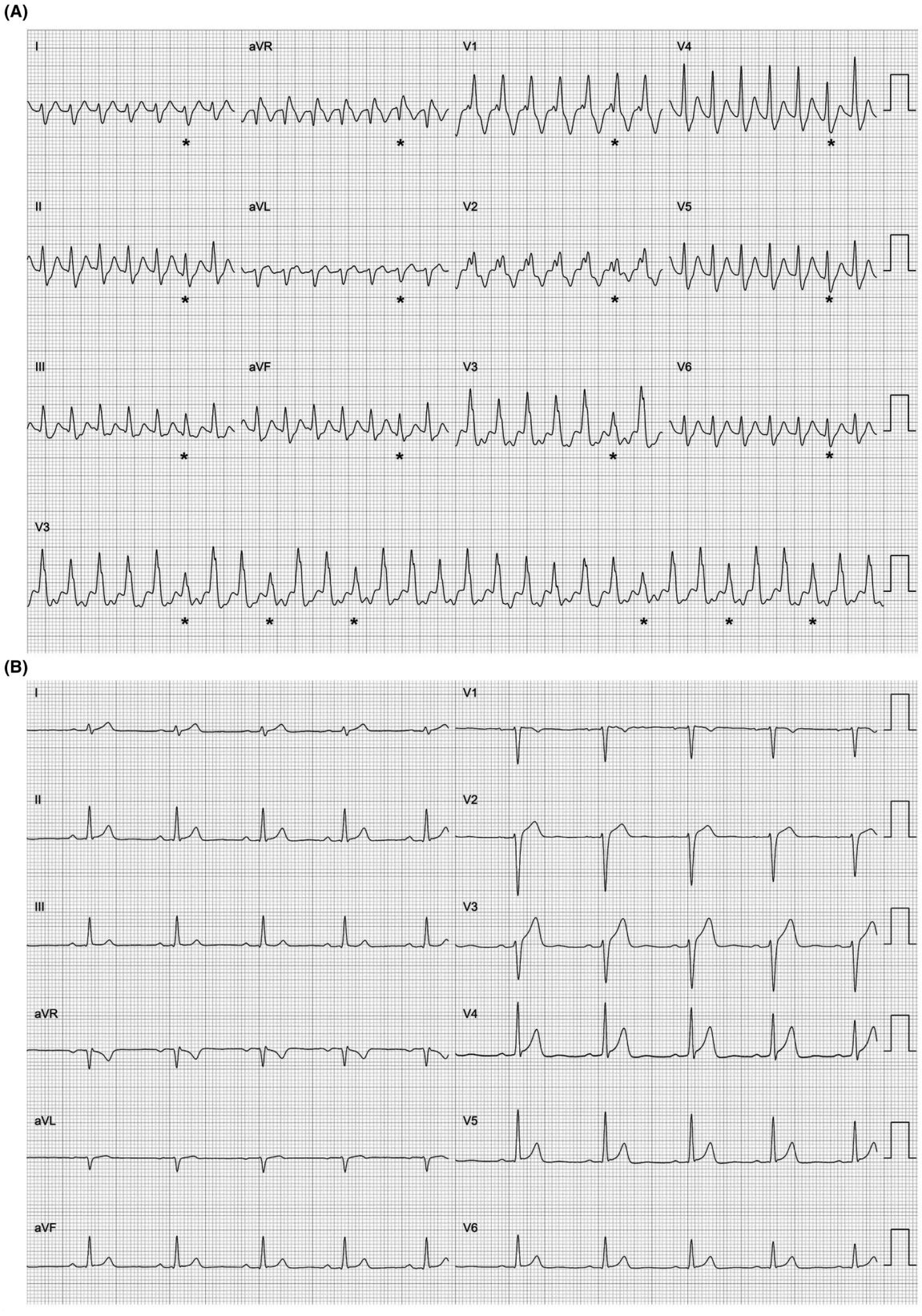

FIGURE 1 A, 12-lead and rhythm electrocardiographic strip from lead V3 of the clinical tachycardia. The electrocardiogram shows a wide QRS complex tachycardia at a rate of 187 bpm with right bundle-branch block morphology and right axis. Subtle changes in QRS amplitude for some QRS complexes are observed (asterisk). B, Baseline 12-lead electrocardiogram of the patient. Normal sinus rhythm at a rate of 62 bpm is shown. Atrioventricular and intraventricular conduction are normal with no ventricular preexcitation

The patient was referred to us for electrophysiological study and catheter ablation. Unexpectedly, the induced tachycardia was an AV nodal reentrant tachycardia (AVNRT) with aberrant conduction, showing second-degree ventriculoatrial block (Figure 2, tachycardia of the right side) because of the possible presence of an upper common pathway above the upper turnaround site of the tachycardia.[3] His-refractory ventricular premature depolarizations were delivered during tachycardia to prove the presence of a nodofascicular/nodoventricular accessory pathway as an alternative mechanism of the observed phenomenon: neither resetting with His bundle advancement, resetting with His bundle delay, nor tachycardia termination were observed, being the three possible phenomena that would prove its presence. In addition, the typical (slow/fast) and the atypical (fast/slow) AVNRT forms with 1:1 AV relationship were also induced (Figure 2). Heart rate was quite similar in all three forms of induced tachycardia (185-190 bpm). When atrial activation occurred before ventricular activation to some degree (as in the middle panel in Figure 2 or the third beat in the right panel in Figure 2), electrical alternans happened. Small changes in ventricular filling and geometry related to the greater or lesser contribution of atrial contraction in each beat of each form of induced tachycardia might therefore explain the QRS alternans in some beats (beats marked with an asterisk

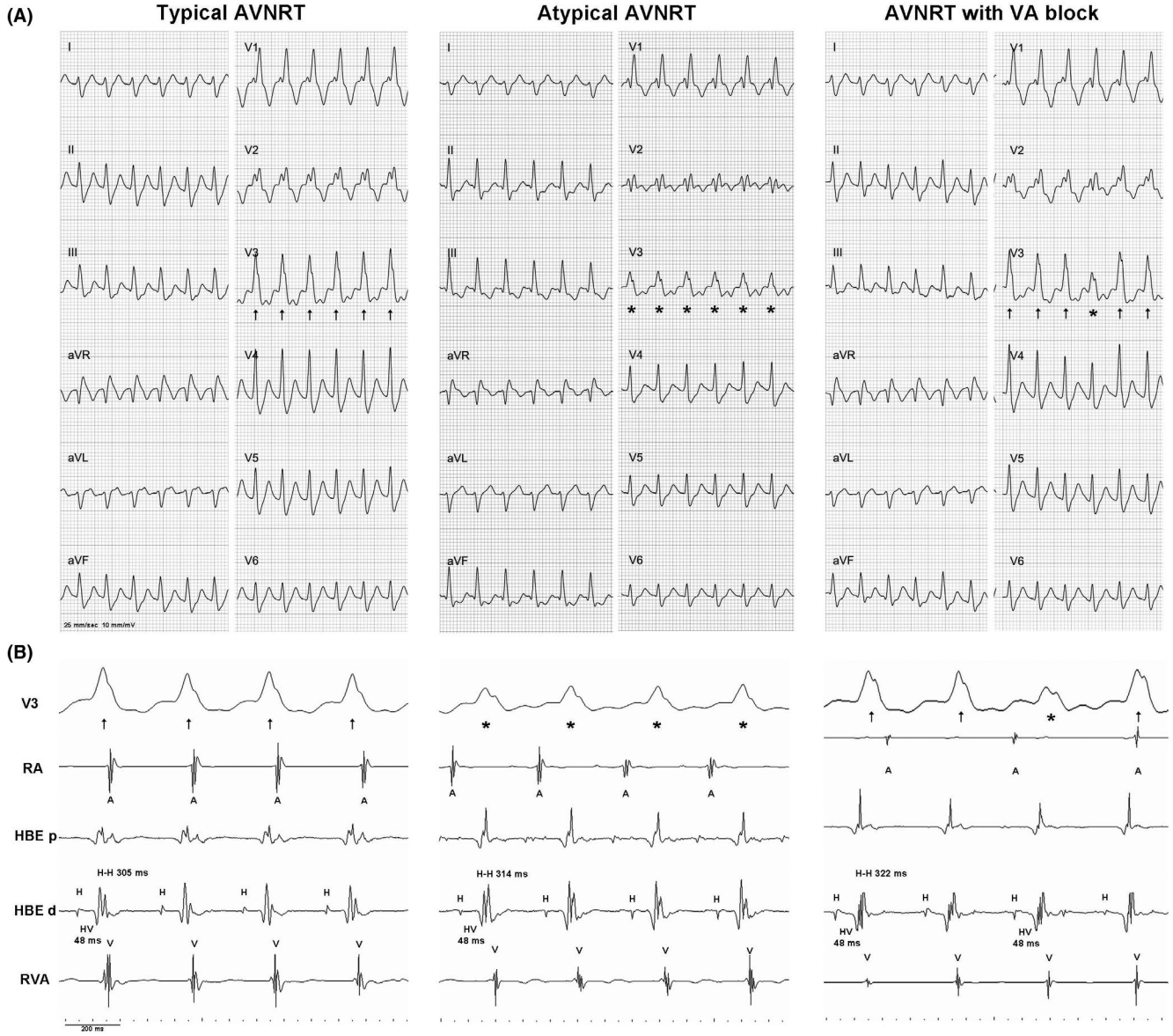

FIGURE 2 Twelve-lead electrocardiogram (A), and electrocardiographic lead V3 and intracardiac electrograms (B) of the three forms of atrioventricular nodal reentrant tachycardia (AVNRT) induced. Differences in QRS amplitude between tachycardias are most evident in lead V3. For the clinical tachycardia (AVNRT with VA block), it is observed that whenever the VA interval is short (as in the typical (slow/fast) AVNRT), QRS amplitude (black solid arrows) is identical to that of the typical AVNRT. By contrary, whenever the VA interval is long (as in the atypical (fast/slow) AVNRT), the next QRS complex (black asterisk) is identical to that of the atypical AVNRT. A, atrial electrogram; H, His recording; HBE p and HBE d, His-bundle electrogram proximal and distal; RA, right atrium; RVA, right ventricular apex; V, ventricular electrogram; VA, ventriculoatrial

in Figures 1 and 2). Successful slow pathway ablation rendered the tachycardia noninducible.

CONFLICT OF INTEREST

The authors declare no conflict of interests for this article.

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