Mahaim pathway tachycardia versus bystander ventricular tachycardia: A distinction without a difference

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Introduction

Decremental accessory pathways (APs) have long been the focus of considerable interest because of their unusual and complex modes of presentation as well as for their proclivity for participating in antidromic reciprocating tachycardia (ART) or to act as passive bystanders in supraventricular tachycardia.[1] Initially described by Mahaim as fibers originating from the atrioventricular (AV) node and inserting into the basal ventricular myocardium, decremental APs, often referred to generically as "Mahaim" pathways, are now classified into at least 3 subtypes: (1) long AV APs that insert into the right bundle branch (atriofascicular) or anterior right ventricular myocardium, (2) short AV APs that insert into peritricuspid ventricular muscle, and (3) nodoventricular (NV) or nodofascicular (NF) pathways that are linked to the AV node and usually emerge from the slow AV nodal pathway.[2–5] With some exceptions, NV/NF pathways are right-sided and, when associated with a regular wide complex tachycardia (WCT), may show AV dissociation, since the atria are not integral to the circuit, making AV dissociation a hallmark for differentiating this form of ART from other forms of decremental AP-mediated ART.

We present a case of a patient who had presumed NV-dependent ART with AV dissociation. However, during electrophysiologic evaluation, we demonstrate that the tachycardia originated from an intra-Mahaim pathway focus, highlighting the potential of decremental APs to develop rapid de novo arrhythmias that may masquerade as ART, passive bystanders, or ventricular tachycardia.

KEYWORDS Arrhythmia; Ablation; Electrophysiology; Supraventricular tachycardia; Ventricular tachycardia
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Case report

A 41-year-old woman with a history of a right-sided AP ablated in China at age 19 presented to an outside hospital with recurrent palpitations and a regular WCT. The arrhythmia terminated with adenosine (6 mg). One week later, she presented to our hospital with a similar tachycardia. The tachycardia had a left bundle branch block pattern with a left superior axis and a cycle length of 280 ms. In the emergency room, she was given intravenous amiodarone, which terminated tachycardia. An electrocardiogram in sinus rhythm showed no evidence of preexcitation. Her subsequent work-up, including an echocardiogram and cardiac magnetic resonance imaging, was normal.

During electrophysiologic study, baseline AH and HV intervals were 65 ms and 37 ms, respectively. Dual AV nodal pathways were demonstrated. Rapid pacing from the proximal coronary sinus resulted in a QRS morphology that reproduced the patient's clinical arrhythmia. Retrograde conduction was concentric and adenosine resulted in ventriculoatrial block. During atrial pacing at a cycle length of 370 ms, conduction proceeded initially over the fast AV nodal pathway, resulting in a narrow QRS complex. However, when conduction abruptly switched to the slow AV nodal pathway (AH increased from 105 to 194 ms), the QRS complex showed fusion for 1 beat (Figure 1A). All subsequent beats were fully preexcited as the AH interval further increased and the His bundle potential was displaced into the ventricular electrogram. Incremental atrial pacing resulted in progressive prolongation of the stimulus-delta interval, findings consistent with a decremental AP.

In the absence of preexcitation, right ventricular apex activation preceded tricuspid annulus (TA) ventricular activation (Figure 1B). However, this relationship reversed with the onset of preexcitation, coincident with a shift in conduction from the fast to the slow AV nodal pathway. (Figure 1B). Greater degrees of preexcitation caused progressively earlier TA ventricular activation relative to the right ventricular apex.

Atrial pacing during concurrent infusion of isoproterenol (2 μg/min) consistently induced WCT with AV dissociation

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KEY TEACHING POINTS

- Rapid tachycardia can originate from an intranodoventricular (NV) focal source. The arrhythmogenic mechanism is due to triggered activity.
- This form of NV tachycardia can masquerade as antidromic reciprocating tachycardia with atrioventricular dissociation or as a passive bystander pathway in atrioventricular nodal reentry.
- The distinction among these 3 entities is challenging, but the algorithm outlined in the Table facilitates the diagnosis. In addition, adenosine termination of a presumed NV-related tachycardia (antidromic or passive bystander) that precedes conduction block in the NV pathway or slow atrioventricular nodal pathway is consistent with a diagnosis of an intra-NV tachycardia.

(Figure 2A and B). The QRS morphology of the clinical tachycardia and the conducted QRS complexes during atrial pacing were identical. The tachycardia terminated with rapid ventricular pacing or adenosine. Of note, fusion beats were observed during induction of tachycardia with atrial pacing and during tachycardia (the latter were due to spontaneous atrial beats) (Figure 2A). Although fusion beats during atrial induction were due to parallel conduction over the NV pathway and AV node, such a mechanism for producing fusion beats cannot occur during ART, owing to collision of anterograde and retrograde wave fronts within the AV node–His-Purkinje system. These data therefore provide incontrovertible evidence that the tachycardia was not due to ART.

Also informative was the differential timing of the response of the Mahaim pathway and tachycardia to adenosine. Immediately following termination of tachycardia with adenosine, AV node conduction prolonged between the first and second sinus beats, although conduction still proceeded over the NV pathway through activation of the slow AV nodal pathway (Figure 3A). During the third sinus beat, conduction blocked in both the Mahaim pathway and AV node; however, by the fourth sinus beat fast AV nodal pathway conduction recovered and the impulse proceeded over the His-Purkinje system, not the Mahaim pathway. Conduction over the Mahaim pathway was therefore linked to conduction over the slow AV nodal pathway. Since tachycardia terminated before conduction block occurred in the slow AV nodal pathway or NV pathway, adenosine's effects on tachycardia occurred *independently* of its effects on the AV node, thus eliminating ART involving an NV pathway or AV nodal reentry with bystander conduction as possibilities. An alternative interpretation is that the tachycardia was due to NV-dependent ART and that adenosine terminated tachycardia by blocking conduction in the retrograde limb, ie, retrograde fast AV nodal pathway. However, this alternative scenario is unlikely, since the anterograde slow AV nodal pathway is notably more sensitive to adenosine than the retrograde fast AV nodal pathway.[6–8] Therefore, in response to adenosine, NV-dependent ART would be expected to terminate in the anterograde limb (slow AV nodal pathway), not the retrograde limb. Accordingly, because of persistence of conduction over the slow AV nodal and NV pathways following termination of tachycardia, and because of the presence of fusion beats during tachycardia, we deduce that the tachycardia had an intra-Mahaim pathway origin and that termination of tachycardia with adenosine was due solely to its direct effects on the Mahaim pathway.

Activation maps were performed during atrial pacing and tachycardia to identify the earliest site of ventricular activation. Both maps localized the ventricular insertion site to the posteroseptal TA. Ablation at this site during tachycardia terminated the arrhythmia within 2 seconds. Although anterograde dual pathways were present post-ablation, AP conduction was not.

Discussion

Our *initial* observations, which included the presence of a decremental NV pathway, AV dissociation during WCT, and linkage of the NV pathway to the slow AV nodal pathway, suggested the possibility of NV-mediated ART (which was atypical, since the pathway inserted at the base of the right ventricle) (Figure 1A and B). Also consistent with this diagnosis is that the morphology of the tachycardia was reproduced with atrial pacing and by pacing at the pathway's ventricular insertion site (Figure 3B). Despite these findings, the presence of fusion beats during tachycardia suggested other potential mechanisms for the patient's WCT. This includes reentrant ventricular tachycardia originating from ventricular muscle contiguous to the pathway's insertion site. However, this is an unlikely explanation, since the tachycardia was sensitive to adenosine, a finding that virtually rules out ventricular reentry.[9] Focal triggered activity originating from the ventricular aspect of the TA annulus is another possibility[10]; however, this is improbable, as it would require a circumstance whereby conduction over the patient's Mahaim pathway exactly replicated the morphology of an unrelated focal tricuspid annular ventricular tachycardia, which also originated at the Mahaim pathway's precise exit site (Figure 2A).

Another consideration is tachycardia originating from within the Mahaim pathway. Although automaticity is known to originate from Mahaim pathways, these arrhythmias usually occur in response to catecholamine stimulation or ablation, are transient, occur at substantially slower rates than that observed in the present study, are not inducible with programmed stimulation, and transiently slow but fail to terminate in response to adenosine.[11,12] Therefore,