Pseudo–Atrial Fibrillation, Rare Manifestation of Multiple Anterograde Atrioventricular Nodal Pathways

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In patients with dual or multiple atrioventricular (AV) nodal pathways manifesting nonreentrant tachycardia or unusual forms of AV nodal reentry, paroxysmal atrial fibrillation is often misdiagnosed and patients may erroneously be considered for pulmonary vein isolation. Multiple anterograde slow AV nodal pathways, identified by >1 discontinuity in the anterograde AV nodal conduction curve, are not rare in patients with slow-fast AV nodal reentrant tachycardia (AVNRT). However, only 1 slow AV nodal pathway is usually involved in anterograde conduction during tachycardia. It was reported that patients with multiple anterograde slow AV nodal pathways presented with different tachycardia cycle lengths. For the first time, 2 patients with AVNRT in which maintenance of tachycardia was strictly dependent on participation of 3 different anterograde slow AV nodal pathways in an uniquely alternating sequence are reported. In both patients, a single application of radiofrequency energy in the posterior aspect of Koch's triangle eliminated simultaneously all evidence of anterograde slow pathway conduction. These findings implied that functional differences in a determined circuit based on nonuniform anisotropy rather than anatomically distinct pathways form the electrophysiologic basis for this rare variant of AVNRT. In conclusion, particularly in patients with lone atrial fibrillation who are potential candidates for pulmonary vein isolation, careful analysis of the surface electrocardiogram during irregular supraventricular tachycardia and invasive electrophysiologic examination helps identify rare arrhythmia mechanisms that can be cured by slow pathway ablation alone. **(Am J Cardiol 2007;100:154–156)**

The most common form of atrioventricular (AV) nodal reentrant tachycardia (AVNRT) is the typical slow-fast variant, in which conduction proceeds anterogradely over the slow and retrogradely over the fast AV nodal pathway.[1] In most patients with typical AVNRT, dual AV nodal pathways can be identified by a discontinuous response of AV nodal conduction time to progressively premature atrial extrastimuli ("jump").[1,2] Multiple discontinuities in the anterograde AV nodal conduction curve can occasionally be observed and are present in up to 6% of patients with slow-fast AVNRT.[3–5] However, only 1 of these anterograde slow pathways is usually operative in 1 and the same tachycardia. Exceptionally, different slow pathways are involved in anterograde conduction during AVNRT, leading to a sudden change or fluctuations in tachycardia cycle length that may mimic atrial fibrillation.[5–7] We report 2 patients with AVNRT using 3 distinct anterograde slow AV nodal pathways in a uniquely alternating sequence. This very rare finding was observed in only 2 of thousands of patients with AV nodal reentry referred to 2 high-volume centers for catheter ablation during the last decade.

Case Descriptions

Patient 1 was a 39-year-old man without structural heart disease who had a history of exercise-induced irregular palpitations. Baseline 12-lead electrocardiogram was normal, and Holter and exercise testing did not record arrhythmias. At electrophysiologic study, atrial extrastimulus testing showed quadruple anterograde AV nodal pathways identified by 3 discontinuities in the anterograde AV nodal conduction curve. Intravenous isoproterenol was required to facilitate induction and maintenance of the clinical tachycardia (Figure 1). This narrow QRS complex tachycardia could mistakenly be diagnosed as atrial fibrillation because the rhythm was irregular and P waves were not clearly detectable. However, careful analysis of RR intervals showed that the tachycardia was regularly irregular. There was a rhythm in the sequence of the QRS complexes giving rise to an image of group beating triplets. Three different cycles repeated themselves each time in the same order with exactly the same RR intervals. This ruled out a random occurrence in the setting of atrial fibrillation. Spontaneous tachycardia termination clarified the underlying mechanism (Figure 2). The fixed and short 1:1 ventriculoatrial conduction ruled out an atrial arrhythmia with Wenckebach-type AV block. Observations that changes in ventricular cycle length preceded changes in atrial cycle length, and tachycardia that terminated spontaneously with an atrial electrogram that blocked in the AV node essentially excluded atrial tachycardia. Accessory pathway–mediated orthodromic tachycardia could be excluded by the coincident atrial and ventricular activation. Thus, slow-fast AVNRT using 3 distinct anterograde slow pathways in an

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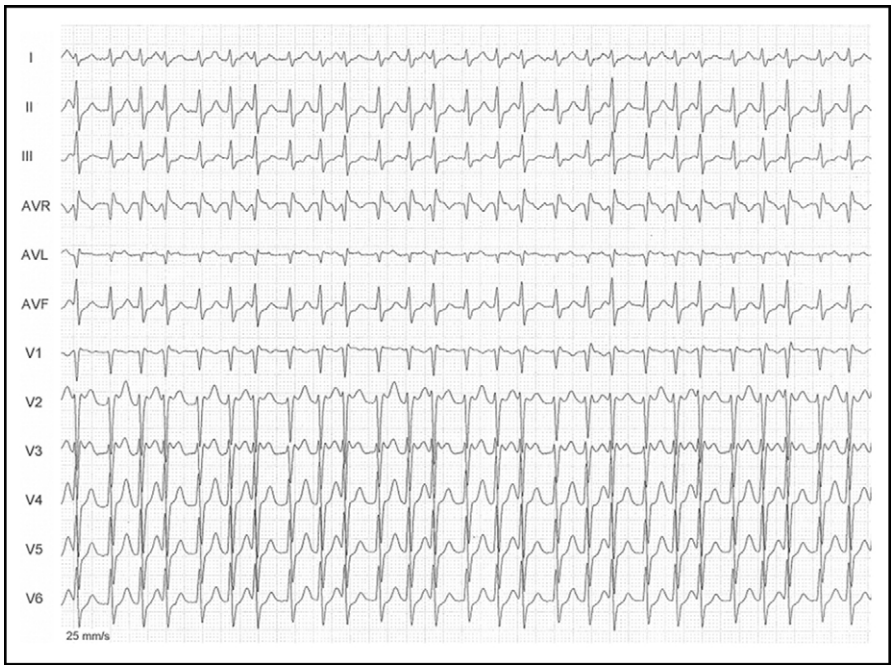

Figure 1. Patient 1: a 12-lead electrocardiogram during AV nodal reentrant tachycardia using 3 different anterograde slow AV nodal pathways in an alternating fashion. Note that this narrow QRS complex tachycardia has a regularly irregular rhythm, giving rise to an image of group beating triplets. Three different cycles repeat themselves each time in the same order with exactly the same cycle lengths (360-320-260 ms). No P waves can be clearly identified.

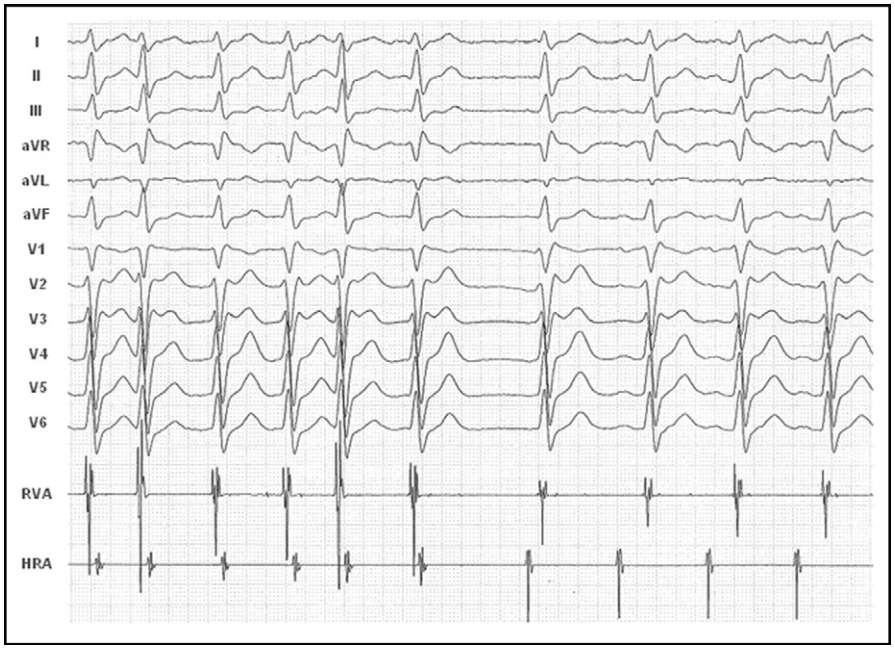

Figure 2. Patient 1: a 12-lead electrocardiogram and intracardiac recordings from the high right atrium (HRA) and right ventricular apex (RVA) during spontaneous termination of the tachycardia. Paper speed 50 mm/s.

alternating fashion was by far the most likely arrhythmia mechanism.

The same phenomenon was observed in patient 2, a 46-year-old woman with a structurally normal heart who presented to the emergency department with a recurrent episode of fast and irregular palpitations. The electrocardiogram showed an irregular narrow QRS complex tachycardia without discernable P waves, for which atrial fibrillation was initially misdiagnosed. Again, careful analysis of RR cycles showed a regularly irregular rhythm with a pattern of repetitive cycle-length triplets (Figure 3). Intravenous injection of adenosine terminated the tachycardia (Figure 3). At electrophysiologic study, atrial extrastimulus pacing showed 3 discontinuities in the anterograde AV nodal conduction curve and reproducibly induced the clinical arrhythmia, that is, AV nodal reentry using alternately 3 slow pathways for anterograde conduction (Figure 3). In both patients, a single application of radiofrequency energy in the posterior aspect of Koch's triangle eliminated all evidence of anterograde slow pathway conduction and rendered tachycardia noninducible.

Comments

We report our observations of 2 patients with a similar mechanism of a very rare variant of AVNRT. Major electrophysiologic characteristics of the 2 unique cases included (1) the presence of quadruple anterograde (1 fast and 3 slow) AV nodal pathways, (2) dependency of AV nodal reentry on participation of 3 anterograde slow pathways in an uniquely alternating sequence, and (3) simultaneous elimination of all 3 slow pathways with a single application of radiofrequency energy in the posterior aspect of the triangle of Koch. In a large study of 500 patients with AVNRT, 1% had quadruple anterograde AV nodal pathways.[5] In most patients, only 1 "clinical slow pathway" was operative during slow-fast AVNRT, and no patient had 3 different slow pathways involved in 1 and the same tachycardia. This implies that different conduction properties of most additional slow pathways were not appropriate to interact with the retrograde fast pathway in a way that allowed reentry or sustained tachycardia to occur. However, multiple AV nodal pathways provide potential loops available for the development of other (slow-slow) variants of AV nodal reentry. In our patients, slow-fast AVNRT with an exceptional alternans pattern of repetitive cycle-length triplets was the only inducible tachycardia, indicating that maintenance of AV nodal reentry was strictly dependent on participation of all 3 slow pathways in its unique alternating sequence. Moreover, a single application of radiofrequency energy in the posterior aspect of Koch's triangle eliminated simultaneously all evidence of anterograde

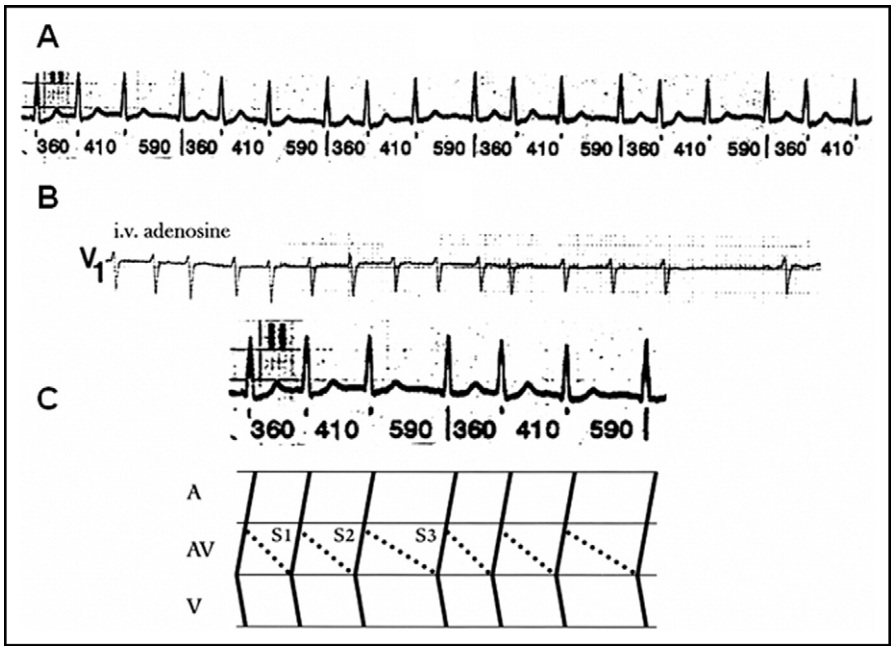

Figure 3. Patient 2. *(A)* Surface lead II shows an irregular narrow QRS complex tachycardia without evident P waves. Careful analysis of RR intervals shows that the tachycardia is regularly irregular with an alternating sequence of 3 different cycles (360-410-590 ms). *(B)* Termination of AVNRT after intravenous (i.v.) adenosine recorded on surface lead V1. *(C)* Surface lead II and ladder diagram show the postulated tachycardia mechanism of AV nodal reentry using retrogradely the fast AV nodal pathway and anterogradely 3 distinct slow AV nodal pathways: S1, S2, and S3 *(dotted lines)*. Paper speed 25 mm/s.

slow pathway conduction. These findings imply that functional differences in a determined circuit based on nonuniform anisotropy may form the arrhythmic substrate in our patients. However, one cannot exclude the possibility of 3 anatomically distinct slow pathways with an upper common pathway or closely spaced insertion sites, allowing simultaneous elimination of all slow pathway conduction by a single ablation lesion. Of great importance, the discussed variant of AV nodal reentry can be erroneously diagnosed as atrial fibrillation if one does not carefully analyze the available electrocardiographic information. In the present patients, the repetitive triple cycle-length pattern should raise a suspicion of a supraventricular tachycardia other than atrial fibrillation. Unfortunately, the rhythm of an AVNRT using >1 slow pathway anterogradely is not always regularly irregular, and varying RR intervals with possibly intermittent aberrant conduction can mimic atrial fibrillation.[7] It is well known that other uncommon manifestations of dual or multiple AV nodal pathways can lead to variable RR intervals, such as nonreentrant double response[8,9] and upper or lower common pathway block during AVNRT.[10] Additionally, arrhythmias related to dual or multiple AV nodal pathways may serve as a potential trigger for atrial fibrillation. In a recently reported series,[11] >4% of patients referred for atrial fibrillation ablation had inducible AVNRT. In these patients, slow pathway ablation alone was associated with an excellent outcome. All these findings emphasize the importance of carefully examining the electrocardiogram of an irregular arrhythmia, including consideration of mechanisms other than atrial fibrillation, and performing a complete electrophysiologic study in patients referred for pulmonary vein isolation, particularly young individuals without risk factors ("lone atrial fibrillation").

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