ARRHYTHMIA OF THE MONTH

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Adenosine-Induced Tachycardia: What is the Mechanism?

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Case Presentation

A 24-year-old woman presented with recurrent episodes of paroxysmal palpitations. She had a documented narrow QRS tachycardia, which was responsive to adenosine. The sinus rhythm ECG was normal. Clinical examination and echocardiogram revealed a structurally normal heart. Adenosine was administered in sinus rhythm to help identify the mechanism of the narrow QRS tachycardia. A bolus of adenosine 12 mg induced self-terminating tachycardia (Fig. 1). What is the mechanism of the tachycardia?

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Commentary

Adenosine is known to terminate paroxysmal supraventricular tachycardia by producing block in the AV node. Adenosine recently has been reported to help differentiate between AV nodal reentrant tachycardia (AVNRT) and AV reciprocating tachycardia (AVRT) when used during sinus rhythm.[1] In patients with AVNRT, the administration of adenosine in sinus rhythm may demonstrate a jump in the PR interval, suggestive of dual AV node pathways. This jump may be associated with an AV nodal echo or the initiation of slow-fast AVNRT. In patients with an accessory pathway, adenosine in sinus rhythm may reveal preexcitation. In either case, transient AV block also may be seen with adenosine.

In our patient, adenosine was used to identify the mechanism of narrow QRS tachycardia. After adenosine,

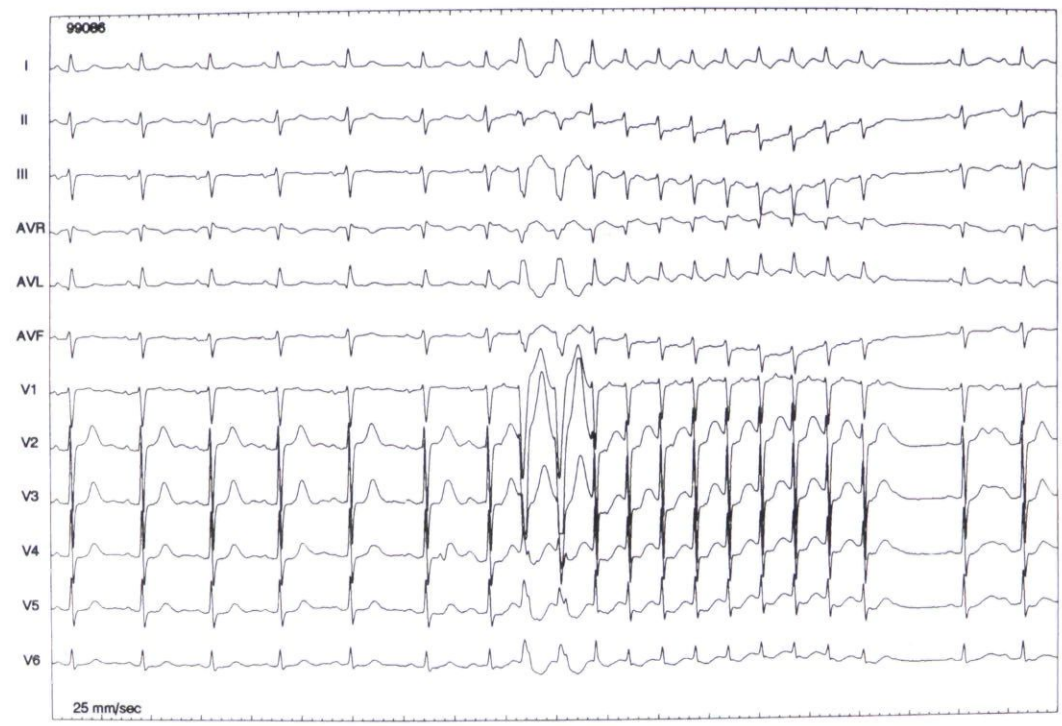

Figure 1. *Twelve- lead ECG recorded during intravenous bolus injection of adenosine 12 mg.*

there was prolongation of the PR interval (Fig. 1), but there was no "jump." After the seventh QRS complex, there is a premature atrial depolarization followed by the initiation of tachycardia, which terminates after a P wave, making atrial tachycardia very unlikely. The atrial echo beat is inverted in leads I and aVL and subsequently has the same morphology during tachycardia (well identified after the wide QRS complexes). This suggests a left lateral origin of the atrial activation sequence. The initial QRS complexes show left bundle branch block morphology, with a longer RR interval compared with those during the narrow QRS complexes. The left bundle branch block aberrancy with slower tachycardia rate is suggestive of an ipsilateral accessory pathway-mediated orthodromic tachycardia.[2]

This phenomenon of tachycardia initiation with adenosine was reproducible. Electrophysiologic study with intracardiac electrograms recorded from the His-bundle and coronary sinus region confirmed the presence of a left lateral accessory pathway and orthodromic tachycardia (Fig. 2). Successful radiofrequency ablation of the left lateral accessory pathway was performed. After ablation, there was no VA conduction, and no tachycardia was induced despite administration of adenosine and isoprenaline.

References

1. Belhassen B, Fish R, Glikson M, et al: Noninvasive diagnosis of dual AV node physiology in patients with AV nodal reentrant tachycardia by administration of adenosine-5'-triphosphate during sinus rhythm. Adenosine for differentiation. Circulation 1998;98: 47-53.
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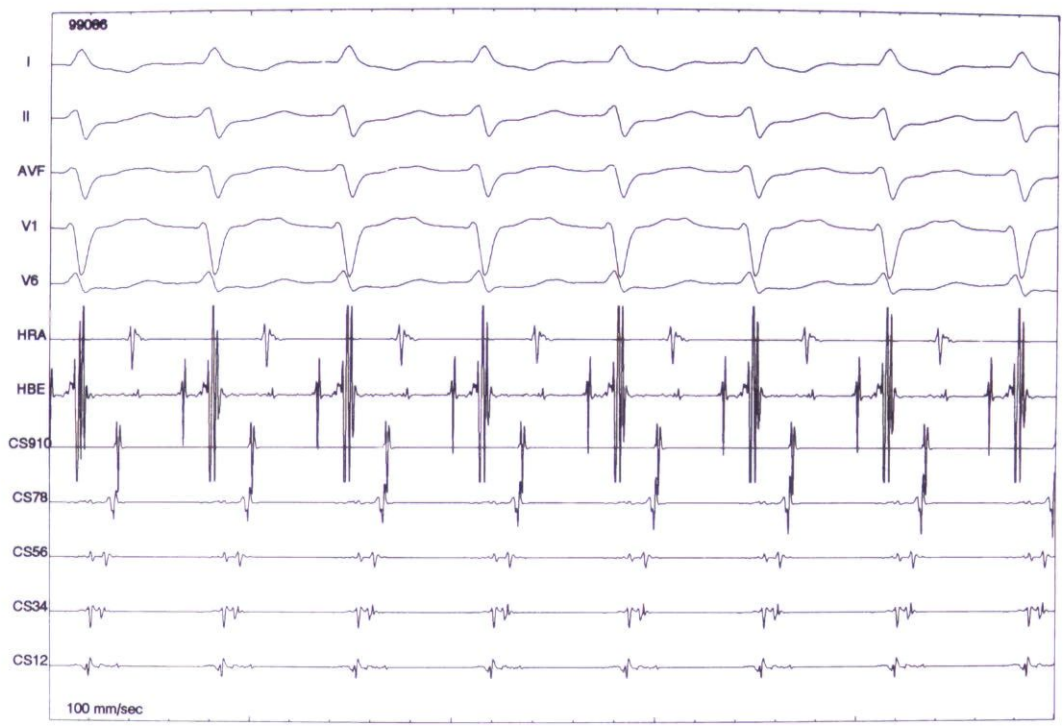

Figure 2. *Surface ECG and intracardiac electrograms recorded during tachycardia. Arrangements of the leads are from top to bottom are surface leads I, II, AVF, V1, and V6, and intracardiac electrograms from the high right atrium (HRA), His-bundle region (HBE), and coronary sinus proximal to distal (CS910 to CS12). Note the eccentric retrograde atrial activation sequence confirming left lateral accessory pathway-mediated orthodromic AVRT.*