ARRHYTHMIA OF THE MONTH

Section Editor: Fred Morady, M.D.

Adenosine-Responsive Wide QRS Complex Tachycardia: What is the Mechanism?

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Case Presentation

A 45-year-old man presented with an acute anterior wall myocardial infarction and underwent successful percutaneous transluminal angioplasty of the left anterior descending coronary artery. Twenty-four hours later, a wide QRS tachycardia at a rate of 108 beats/min that had a QRS duration of 150 msec was noted by telemetric monitoring. There were no symptoms associated with the tachycardia. A 12-lead ECG was recorded (Fig. 1). The tachycardia terminated after infusion of 12 mg of adenosine (Fig. 2). What is the mechanism of this adenosine-responsive wide QRS complex tachycardia?

Commentary

The ECG shown in Figure 1 demonstrates a wide QRS complex tachycardia at a rate of 108 beats/min that has a right bundle branch block configuration and right-axis deviation. There is a P wave immediately after each QRS complex (best seen in lead V_3), indicating either 1:1 AV conduction or 1:1 VA conduction (Fig. 1). The differential diagnosis at this point includes a supraventricular tachycardia (AV nodal reentrant tachycardia, orthodromic reciprocating tachycardia, atrial tachycardia, or automatic junctional tachycardia) with a rate-related or underlying right bundle branch block, and ventricular tachycardia.

The response to adenosine shown in Figure 2 establishes the mechanism of this tachycardia.

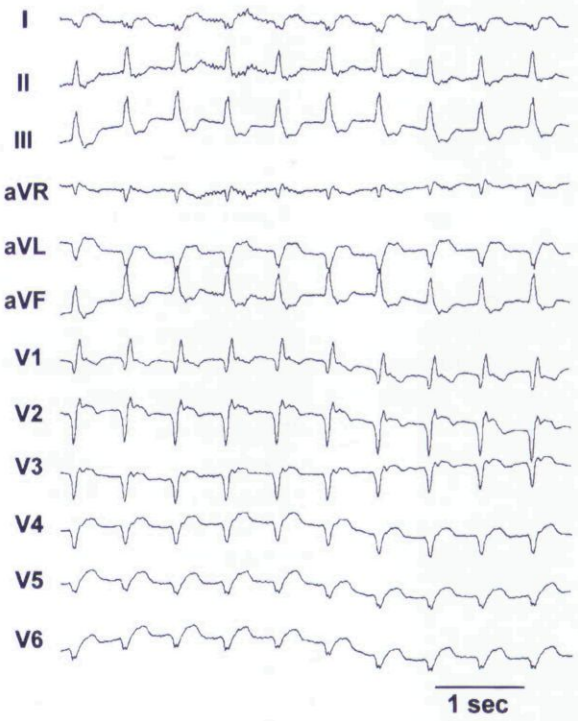

Figure 1. *ECG recorded at the onset of the wide QRS complex tachycardia.*

After the administration of adenosine, the 1:1 AV relationship is replaced by AV dissociation (again best seen in lead V_3). This excludes orthodromic reciprocating tachycardia and atrial tachycardia and narrows the list of possibilities to AV nodal reentrant tachycardia (probably a very rare response to adenosine), automatic junctional tachycardia, and ventricular tachycardia. Note that there is a supraventricular capture beat just before the tachycardia terminates, and that this QRS complex is narrow. This indicates that the wide QRS complexes recorded during the tachycardia cannot be attributed to a rate-related

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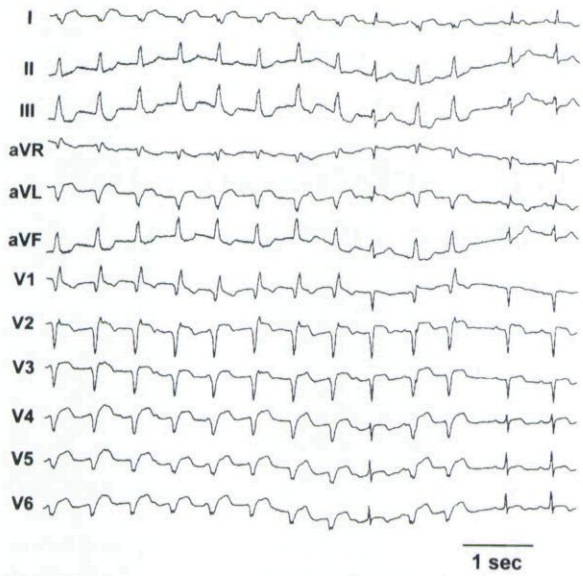

Figure 2. *ECG recorded several seconds after infusion of 12 mg of adenosine.*

or underlying right bundle branch block. Therefore, the tachycardia is a ventricular tachycardia.

Adenosine-sensitive ventricular tachycardia primarily is due to cyclic adenosine monophosphate-mediated triggered activity.[1,2] In patients with coronary artery disease, monomorphic ventricular tachycardia usually is caused by reentry. In this patient, ventricular tachycardia probably was caused by ischemia- or reperfusion-induced triggered activity.

References

1. Lerman BB, Belardinelli L, West GA, et al: Adenosine sensitive ventricular tachycardia: Evidence suggesting cAMP-mediated triggered activity. Circulation 1986;74: 270-280.
2. Lerman BB: Response of nonreentrant catecholamine-mediated ventricular tachycardia to endogenous adenosine and acetylcholine. Evidence for myocardial receptor-mediated effects. Circulation 1993;87:382-390.