

Challenges in Clinical Electrocardiography

Narrow Complex Tachycardias—Therapeutic and Diagnostic Role of Adenosine

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A patient in their 40s presented to the emergency department with sudden-onset rapid palpitations at rest. The patient recalled similar episodes in the past that had terminated spontaneously. The patient's heart rate was 210 beats/min at presentation, and blood pressure was 110/70 mm Hg. Carotid sinus massage was attempted to terminate the tachycardia but to no effect. Intravenous adenosine, 12 mg, followed by rapid saline flush was administered while recording the 12-lead electrocardiogram (ECG) in the emergency department (Figure 1).

Question: Based on the response to adenosine, what is the likely mechanism of tachycardia?

Interpretation and Clinical Course

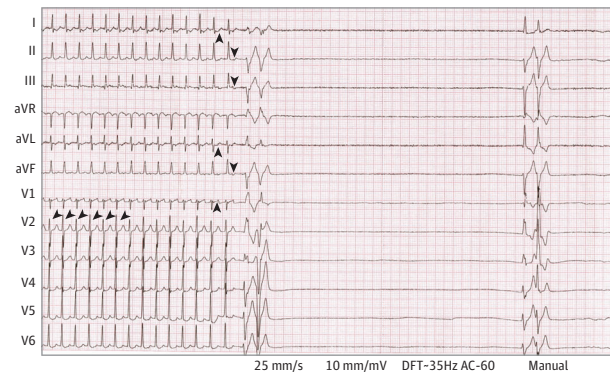
The ECG (Figure 1) tracing on the left showed a regular narrow complex tachycardia (NCT) at the rate of 190 beats/min with 1:1 atrioventricular (AV) conduction and a short RP interval along with QRS alternans. Inverted P waves were seen in the inferior leads away from the QRS complex, which were more apparent near the termination of tachycardia. There were positive P waves in lead V₁ and negative P waves in leads I and aVL, suggesting atrial depolarization progressing from the left to the right atria. This made typical AV nodal reentry tachycardia (AVNRT) unlikely,¹ as the atrial activation began at the AV node and progressed to both the atrium simultaneously in AVNRT. Hence, the retrograde P waves were either embedded into QRS in the surface ECG or manifested as pseudo-R waves in lead V₁ and pseudo-S waves in inferior leads. Progressive PR prolongation before termination of tachycardia (Figure 1) suggested AV nodal block as the mechanism of termination. Tachycardia terminated after the P wave followed by a sinus pause (Figure 1). This ruled out focal atrial tachycardia because it usually terminates with a QRS complex.

The second ECG was recorded after the termination of the tachycardia, which confirmed the mechanism of tachycardia. The first 4 sinus beats after the pause were seen with manifest preexcitation (Figure 2). As the effect of adenosine on the AV node subsided, AV nodal conduction fused with that of the accessory pathway (Figure 2). The presence of preexcitation after termination along with the negative P waves in leads I and aVL during tachycardia was suggestive of orthodromic AV reentrant tachycardia (AVRT) with retrograde conduction through a left-sided accessory pathway. The patient underwent successful radiofrequency ablation of the left posterior accessory pathway.

Discussion

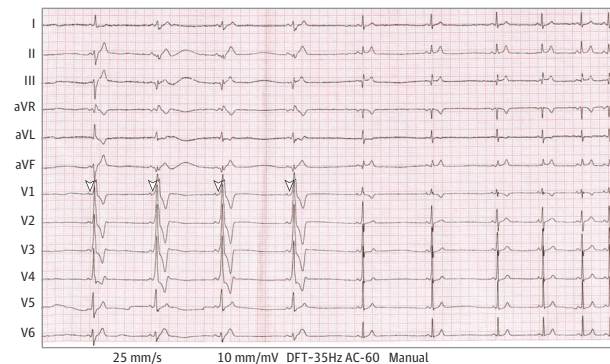
Narrow complex regular tachycardia is a common ECG finding in patients presenting to the emergency department with palpitations. Accurate identification of the rhythm and the mechanism of arrhythmia is imperative, as the success and complication rates after radiofrequency ablation are different. The morphology of the retrograde P wave and the response of the arrhythmia to adenosine play an important role in identifying the rhythm abnormality. Ergo,

Figure 1. 12-Lead Electrocardiogram (ECG) Demonstrating the Termination of a Regular Narrow Complex Tachycardia by Intravenous Administration of Adenosine



Inverted P waves are seen in the inferior leads away from the QRS complex, which are more apparent near the termination of tachycardia (vertical down arrowheads). Retrograde P waves falling on the ST segment (vertical up arrowheads) are negative in leads I and aVL and positive in lead V₁. QRS alternans is also seen in the initial part of the ECG, best seen in lead V₁ (angled arrowheads). Termination is followed by ventricular premature complexes in couplet and a long sinus pause.

Figure 2. Electrocardiogram After Termination of a Narrow Complex Tachycardia With Intravenous Adenosine



The sinus rhythm resumed and unmasked the latent preexcitation for the first 4 beats (white arrowheads). Then the atrioventricular node also resumed the physiological conduction.

adenosine acts not only as a therapeutic drug but also a diagnostic tool.

In patients with typical AVNRT, the retrograde P wave is very close to the QRS complex, thus producing short RP tachycardia with RP interval less than 70 milliseconds. Also, as the atrial depolarization travels from the AV node and above, P waves in inferior leads

are negative, described as pseudo-S waves, and are positive in lead V₁ and termed as pseudo-R waves.²

In orthodromic AVRT, antegrade conduction occurs through the AV node, and retrograde conduction occurs through the accessory pathway. The RP interval is short owing to fast retrograde conduction through the accessory pathway but is usually more than 70 milliseconds, and the morphology of the retrograde P wave depends on the location of the accessory pathway.¹ In the present case, retrograde P-wave morphology became apparent near the termination of tachycardia and helped in localizing the accessory pathway. The P waves were negative in I and aVL and positive in V₁, suggesting the depolarization progression from the left to the right atrium or a left-sided accessory pathway.¹ Atrioventricular reentrant tachycardia with antegrade conduction over the accessory pathway produces wide complex tachycardia known as antidromic reentrant tachycardia with the presence of delta waves.

Continuous ECG recording at the time of administration of adenosine is invaluable to note the response of tachycardia at termination. The mode of termination can help to distinguish the various forms of NCT. Adenosine blocks the anterograde-conducting slow pathway in typical AVNRT and the AV node in orthodromic AVRT. Termination of tachycardia with a P wave is suggestive of AV node-dependent tachycardia, such as typical AVNRT or AVRT with orthodromic conduction.³ In atypical AVNRT, adenosine usually terminates the tachycardia by blocking the conduction in the retrograde slow pathway, thus producing a QRS complex at the end of tachycardia. By inducing AV nodal block, adenosine can also unmask the preexcitation and aid in the diagnosis of accessory pathway.⁴

Up to 50% of focal atrial tachycardias are responsive to adenosine, and those who do respond are referred to as having adenosine-sensitive atrial tachycardia. These are generally located at the perinodal/perianular region and are similar to the AV node

electrophysiologically. The termination of atrial tachycardia occurs by blocking conduction at the site of origin of the atrial tachycardia, not at the AV node. Therefore, the PP interval will prolong, and tachycardia will terminate with a QRS complex even before the effect of adenosine on the AV node manifests.⁵

The full therapeutic and diagnostic potential of adenosine can only be achieved if administered properly. Its half-life is 10 seconds owing to its rapid metabolism by endothelial and red blood cells. For the electrophysiological effects to manifest, adenosine should be administered rapidly, and the administering syringe should not contain blood. The minimum initially recommended dose is 6 mg intravenously, which can be increased by 6 mg to a maximum of 18 to 24 mg. Continuous ECG recording during administration of adenosine should always be performed.⁶ Also, adenosine can rarely cause atrial fibrillation, and in patients with manifest preexcitation at baseline, it can lead to ventricular fibrillation because AV nodal block will lead to rapid antegrade accessory pathway conduction.⁷ Therefore, a defibrillator should be kept ready while administering adenosine.⁸

Take-home Points

- A retrograde P wave and its morphology should always be considered in patients presenting with NCT.
- Continuous ECG monitoring is strongly recommended while administering adenosine.
- Termination of an NCT with a P wave is suggestive of AV node-dependent tachycardias, such as typical AVNRT or AVRT.
- Termination of an NCT with a QRS complex is suggestive of adenosine-sensitive focal atrial tachycardia.
- Adenosine can unmask preexcitation in the beats after tachycardia termination by creating AV nodal block, leading to accessory pathway conduction.

ARTICLE INFORMATION

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