Induction of atrioventricular nodal reentry tachycardia with intravenous adenosine

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ABSTRACT

Adenosine, used to terminate paroxysmal supraventricular tachycardia (SVT), is often useful in understanding the mechanism of tachycardia. This case report describes induction of SVT with adenosine in a 36-year-old man presenting with recurrent palpitations. After a short run of conduction via both slow and fast pathways, SVT was induced following a long PR interval. The long PR interval resulted by conduction via the slow pathway due to the preferential conduction block by adenosine over fast pathway. The notching at the terminal part of QRS during antegrade slow pathway conduction and during tachycardia indicated activation of the atrium via retrograde fast pathway. This electrocardiographical feature confirmed the mechanism of the tachycardia as atrioventricular nodal reentrant tachycardia.

Keywords: adenosine, atrioventricular nodal reentrant tachycardia, supraventricular tachycardia

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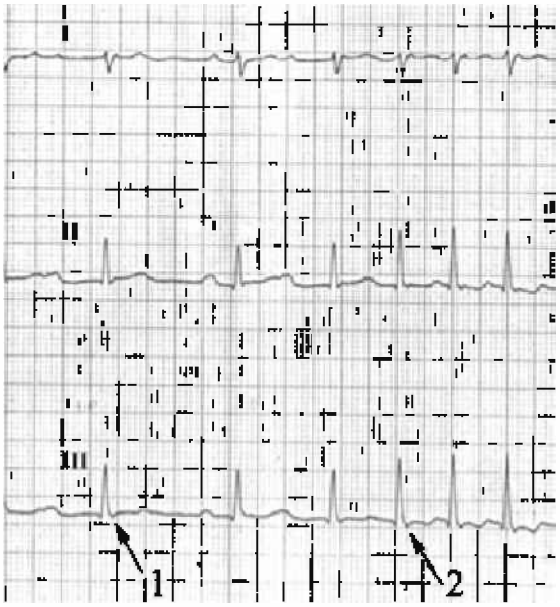

Fig. 1 ECG strip shows that intravenous adenosine injection led to: First PQRST showed PR interval prolongation due to antegrade conduction down the slow AV nodal pathway with AV nodal echo conducted retrogradely via the fast AV nodal pathway (arrow 1). Second PQRST showed normal PR interval due to antegrade conduction down the fast AV nodal pathway. Third PQRST showed PR interval prolongation due to antegrade conduction down the slow AV nodal pathway with AV nodal echo conducted retrogradely via the fast AV nodal pathway and initiation of AVNRT (arrow 2 showing retrograde P as pseudo S during AVNRT).

INTRODUCTION

Adenosine is often used intravenously to terminate supraventricular tachycardia (SVT). It is also being increasingly used during sinus rhythm as a bedside diagnostic tool to detect the substrate that may be responsible for the SVT, such as dual atrioventricular node physiology, atrioventricular nodal or atrioventricular echo beat. The nature of the tachycardia is confirmed when an injection of adenosine reveals the underlying substrate and then gives rise to tachycardia.

CASE REPORT

A 36-year-old man had recurrent palpitations of sudden onset, lasting for minutes to hours, for many years. There was no haemodynamic compromise. He had undergone aortic and mitral prosthetic valve replacement seven years ago for rheumatic valvular disease. The electrocardiogram (ECG) during episodes of palpitation showed SVT, with narrow QRS complexes at a rate of 144 per minute. The P waves were not visible during tachycardia. Basal ECG did not show any preexcitation. The mechanism of tachycardia was thought to be due to either atrioventricular nodal reentrant tachycardia (AVNRT) or atrioventricular reciprocating tachycardia (AVRT) via a concealed accessory pathway.

An intravenous bolus of 12 mg adenosine during sinus rhythm resulted in conduction via slow pathway (long PR interval) and fast pathway (short PR interval) alternately (Fig. 1). This was followed by SVT. During the slow pathway conduction, as well as during SVT, there was a pseudo S wave at the end of QRS waveform indicating retrograde P wave conducted via fast pathway (Fig. 1). Hence, the mechanism of tachycardia was

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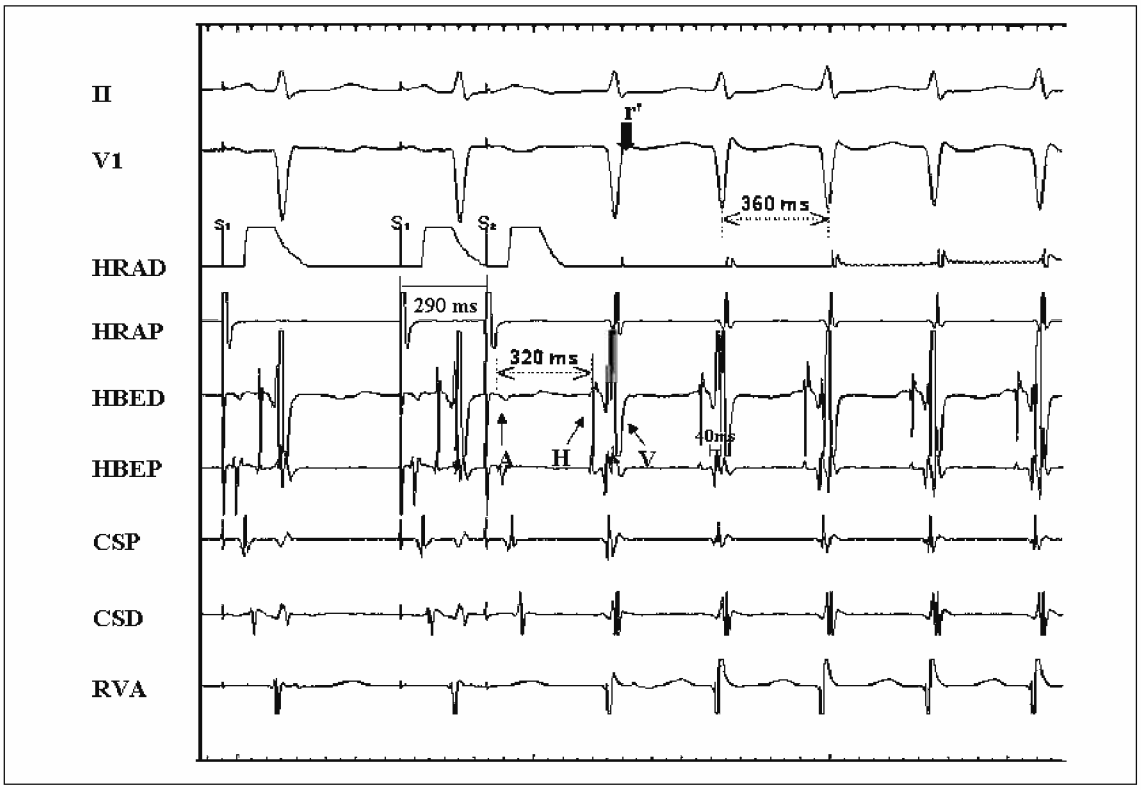

Fig. 2 ECG strips shows that the programmed atrial extrastimulus at a coupling interval of 290 ms (S1S2) gave rise to an AH jump (AH is 390 ms following S2) followed by initiation of supraventricular tachycardia with a cycle length of 360 ms and VA interval of 40 ms. The earliest A is seen in the HBEP region. Note the pseudo r' in V1 from the third QRS onwards during tachycardia. The pseudo r' is due to retrograde P wave conducted via fast pathway. The first two channels are surface ECG leads II and V1. The rest of the channels are intracardiac bipolar electrograms. [HRAD: High right atrium distal; HRAP: High right atrium proximal; HBED: His bundle distal; HBEP: His bundle proximal; CSP: Coronary sinus proximal; CSD: Coronary sinus distal; RVA: Right ventricle distal; A: Atrial electrogram; H: His bundle potential; V: Ventricular electrogram].

diagnosed to be dual AV node physiology with slow-fast type of AVNRT.

During the electrophysiological study, the clinical tachycardia could be easily induced by programmed atrial stimulation with atrial extrastimulus. Programmed atrial extrastimulation at a critical coupling interval gave rise to an AH jump (conduction via slow pathway), followed by induction of SVT (Fig. 2). The nature of induction of tachycardia (following an AH jump), the earliest atrial activation at His bundle and the VA interval of only 40 ms indicated the SVT as a slow-fast type of AVNRT. Radiofrequency ablation of the slow pathway was performed successfully.

DISCUSSION

In addition to its major therapeutic effect in terminating paroxysmal SVT, adenosine has gained importance as a bedside diagnostic tool in regular narrow or wide QRS tachycardia.[1-3] Precise non-invasive diagnosis of the mechanism of narrow QRS tachycardia helps in proper counselling to the patient regarding an electrophysiological study and radiofrequency ablation. Criteria for dual AV nodal physiology are: (1) increment or decrement of PR interval by 50 ms or more in two consecutive sinus beats, and (2) appearance of an AV nodal echo beat. The criterion for diagnosing concealed accessory pathway is the appearance of AV reentrant echo beat. Presence of dual AV node physiology alone in ECG does not explain the nature of tachycardia, since a concealed accessory pathway mediated AVRT can also coexist with a dual AV node physiology. The nature of tachycardia is confirmed only when the dual AV node physiology initiates the tachycardia.

This case report shows that induction of SVT following a PR jump with AV nodal reentrant echo beat and the morphology of the narrow QRS tachycardia was similar to the clinical tachycardia. The differential action of adenosine on fast and slow pathways of AV node helps to unfold the dual AV nodal physiology.[4] At lower serum level, adenosine blocks fast pathway selectively. In a series of 97 patients having AVNRT and AVRT, intravenous bolus of adenosine initiated SVT in only three patients although the other criteria fulfilling dual AV nodal physiology or AV bypass tract were seen in many patients.[2]

Okumura et al reported reproducible induction of slow/fast AVNRT with intravenous bolus injection of adenosine triphosphate in a patient in whom dual AV nodal physiology according to electrophysiological criteria was not shown by single atrial extrastimulation.[5] Such a low incidence of initiation of SVT is possibly due to rapid pharmacokinetics of adenosine in the body. However, at a specific serum concentration, adenosine may block only antegrade conduction over fast pathway leading to conduction over slow pathway and a reentrant echo beat retrogradely via fast pathway, which then passes again via the antegrade slow pathway to establish the tachycardia.

In conclusion, intravenous adenosine is commonly used to terminate an episode of SVT. It may at times help in determining the possible mechanism of tachycardia by showing PR jump or atrial echo beat. In this case, adenosine injection unfolded the dual AV node physiology and initiated the SVT, which resembled the clinical tachycardia and confirmed the diagnosis.

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