

Letter to Editor

A Caveat during Adenosine Challenge

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To the Editor:

Adenosine is a nucleoside purine analog that causes transient atrioventricular (AV) block; when patients are appropriately selected based on presenting rhythm, it has proven to be the best available drug to treat re-entrant paroxysmal supraventricular tachycardias.¹ A second role of this medication is to allow a better characterization of atrial activity by slowing the ventricular response. Nevertheless, an aspect to be considered before adenosine administration is the probability of the underlying rhythm being atrial flutter, since this agent has the ability to facilitate 1 to 1 conduction in some patients, independently of the dose provided.² In this situation, the most dreaded consequence is degeneration of the arrhythmia into ventricular flutter or fibrillation and cardiac arrest.³ We report an adverse event in a patient who received adenosine in an attempt to clarify the etiology of a supraventricular tachycardia.

A 77 year old female with a history of rheumatoid arthritis presented to our institution

complaining of profound fatigue for one day. Her electrolytes, complete blood count, TSH and serial troponins were all within normal limits. Initial ECG (Fig 1) revealed the presence of a narrow-complex supraventricular tachycardia, initially interpreted as sinus tachycardia at 129 beats per minute.

However, given the paucity of change in heart rate over time and abnormal morphology of T waves, an alternative diagnosis was entertained. In order to better characterize the underlying atrial activity, the patient was given 6 mg of IV adenosine with no response; a 12 mg dose was then administered, resulting in transient slowing of ventricular response, high-grade AV block and evidence of flutter waves (Fig 2 a); shortly afterwards, enhanced 1: 1 atrioventricular conduction was observed at a rate of 260 beats per minute (Fig 2b) and persisted for 7 seconds; occasional premature atrial complexes were observed (Fig 2c) before the patient unexpectedly converted to normal sinus rhythm.



Fig-1: 12-lead ECG on presentation displays a narrow complex, regular tachycardia at 130 beats per minute. It was read initially as sinus tachycardia; however, abnormal notching of some T waves (arrows) suggested an alternative diagnosis

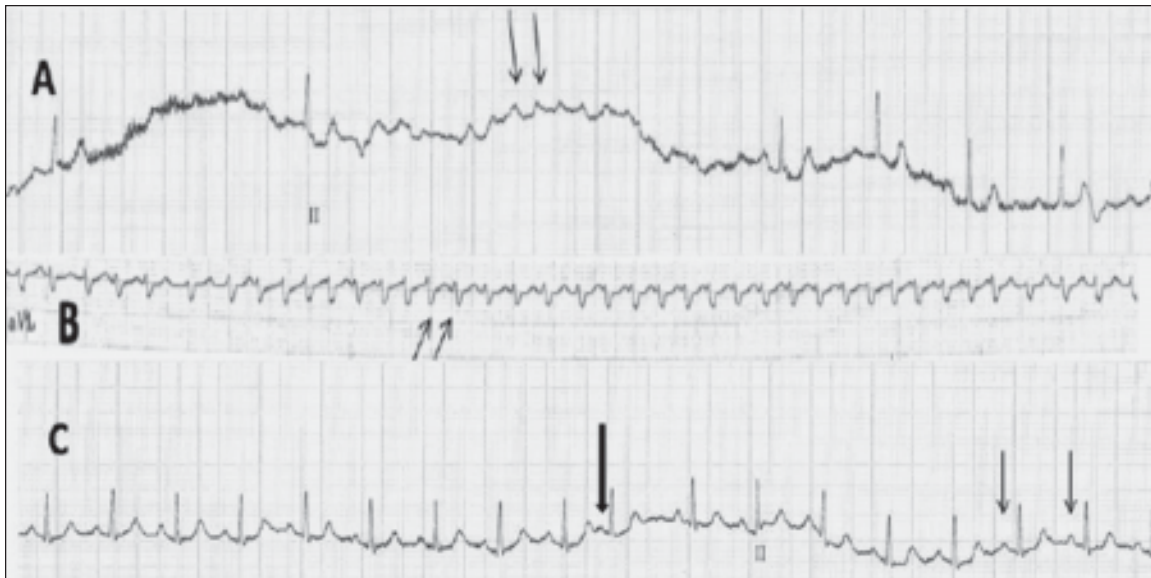


Fig 2: **A:** Rhythm strip (lead II) after administration of 12 mg IV of adenosine, showing high-grade AV block and flutter waves (arrows). **B:** Shortly after slowing of ventricular response, enhanced 1 to 1 conduction occurred at a rate of 260 beats per minute, lasting 7 seconds (lead AVL). **C:** Briefly after accelerated ventricular response, there was spontaneous slowing of heart rate, with some evidence of atrial premature complexes (bold arrow), before the patient regained normal sinus rhythm (thin arrows indicate P waves) at a heart rate of 93 beats per minute.

Adenosine was likely responsible for the enhanced conduction seen in this patient. The mechanism proposed is an increase in adrenergic tone after a brief period of atrioventricular block, secondary to stimulation of carotid body chemoreceptors.⁴ This becomes an important issue if we consider that as many as 10% of hospitalized patients who are given adenosine for diagnostic and therapeutic purposes could present with atrial flutter.⁵ Thus, the favorable effects of adenosine rely on its direct negative dromotropic properties, but its “paradoxical” secondary chronotropic effect needs to be deliberately acknowledged in patients with possible atrial flutter; we underscore the importance of having a controlled, structured setting with advance cardiac life support capabilities when administering this agent for diagnostic purposes.

References:

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