CASE REPORT

Adenosine-induced tachycardia acceleration: an unusual proarrhythmia

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SUMMARY

Adenosine is an effective agent for termination of most re-entrant supraventricular arrhythmias involving the atrioventricular node and often also used as a diagnostic agent for wide QRS tachycardias. Adenosine terminates 90–99% of re-entrant supraventricular tachycardias but it may rarely accelerate tachycardias. Adenosine-induced tachycardia acceleration is a rare phenomenon, as only a handful of cases have been described in the literature. We present a case of a 36-year-old man with a narrow complex, short RP tachycardia at a rate of 165 bpm and an initial blood pressure of 110/78 mm Hg. A bolus of 12 mg of adenosine resulted in slowing of the tachycardia to 150 bpm for 2–3 s, followed by acceleration of the tachycardia to 185 bpm that lasted for approximately 20 s and returned to baseline at 165 bpm. The main mechanism of adenosine-induced acceleration may be the secondary sympathetic stimulation, which may be preceded by transient bradycardia and/or hypotension.

BACKGROUND

Supraventricular tachycardias (SVTs) are very common and house officers and primary care physicians are often required to treat it. Intravenous adenosine is used as the first-line drug for rapid termination of SVTs and it is usually effective. This case demonstrates transient tachycardia slowing, followed by tachycardia acceleration, as an unusual 'side effect' of adenosine.

Adenosine is an endogenous purine nucleoside with a half-life <10 s; it is commonly used for emergency treatment of narrow complex tachycardias as well as being a diagnostic tool for wide complex tachycardias.^{1–2} Glatner *et al*³ have reported the electrophysiological effects of adenosine on SVT and the electrophysiological effects of adenosine on atrial and ventricular myocardium has been well described.⁴ Adenosine causes slowing and termination of SVTs in 99% of patients when administered via a central vein and 90% when administered peripherally.^{5–7} Occasionally, there may be no response to adenosine, but acceleration of an SVT has only rarely been reported and not well studied or understood since it is so rare. This case elucidates a mechanism of tachycardia slowing and acceleration.

CASE PRESENTATION

A 36-year-old man with a history of recurrent palpitations had sudden onset of symptoms at rest.

Figure 1 A short RP tachycardia in the baseline state, at a rate of 165 bpm.

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He did not have any other associated symptoms. The patient did not respond to intravenous boluses of 6, 12 and 12 mg of adenosine administered by emergency medical technicians in the ambulance. The patient was given a 10 mg bolus of intravenous diltiazem and was started on a drip at 5 mg/min in the ambulance and brought to the emergency department. The initial ECG, obtained approximately 20–25 min later, showed a narrow complex tachycardia with a short RP interval, at a rate of 165 bpm (figure 1), and an initial blood pressure of 110/78 mm Hg. A bolus of 12 mg of adenosine was repeated,

resulting in slowing of the tachycardia to 150 bpm for 2–3 s (figure 2), followed by acceleration of the tachycardia to 185 bpm (figure 3) that lasted for approximately 20 s and returned to baseline at 165 bpm. The patient's haemodynamic status remained unchanged throughout. All tachycardias were of the short RP type, suggesting atrioventricular (AV) nodal re-entry tachycardia (AVNRT).

OUTCOME AND FOLLOW-UP

The patient underwent an electrophysiological study where a narrow QRS complex, short RP tachycardia was induced and

Figure 2 The tachycardia slowed for 2–3 s, to a rate of 150 bpm after 12 mg of adenosine.

Figure 3 Tachycardia accelerated to 185 bpm for 20 s and then returned to baseline at 165 bpm.

Figure 4 Baseline 12-lead ECG.

the effect of adenosine was reproduced. The differential diagnosis of a short RP tachycardia includes typical AVNRT, orthodromic AV re-entry (accessory pathway mediated SVT) as well as atrial tachycardia with a long AV interval. In this case, the diagnosis of typical AVNRT was proven by electrophysiological manoeuvres.

Figure 4 shows a baseline 12-lead ECG and has normal sinus rhythm without evidence of delta waves.

Figure 5 shows a narrow QRS complex tachycardia at a rate of 165 bpm. It was obtained during the electrophysiological study. It reproduced the tachycardia observed in the emergency department.

Figure 6 shows intracardiac electrograms of the same tachycardia and a retrograde activation (VA, ventriculoatrial) from

the ventricle to the atrium. Tachycardia cycle length (TCL)=AV interval (ms)+VA interval (ms). Tachycardia rate=60 000/TCL; AV=280 ms; VA=80 ms and TCL=360 ms.

Figure 7 was obtained after adenosine administration peripherally, followed with a 10 mL saline bolus to reproduce the observation in the emergency department. Tachycardia slowed, as the retrograde conduction slowed (VA). AV=210 ms; VA=190 ms and TCL=400 ms.

Figure 8 was obtained 20 s later. It shows that the retrograde conduction has returned to the baseline state (VA=80 ms) and antero-grade conduction has accelerated (AV=250 ms). The shortening is attributed to secondary sympathetic activation because when adenosine had its initial effect of slowing retrograde VA activation (VA increased from 80 to 190 ms), the AV interval was 280 ms.

Figure 5 A narrow QRS complex tachycardia at a rate of 165 bpm induced during electrophysiological study.

Figure 6 Intracardiac electrograms of the induced tachycardia (AV, atrioventricular; TCL, tachycardia cycle length; VA, ventriculoatrial).

DISCUSSION

Antiarrhythmic drugs (AAD) and devices may be proarrhythmic. Proarrhythmia is defined as the occurrence of a new rhythm disturbance or worsening of an existing one.⁸ Bradycardias are the most common form of proarrhythmia. Torsade de pointes (TdP), a polymorphic ventricular tachycardia, is the most dreaded proarrhythmic tachycardia, as it may lead to syncope or sudden death.

In general, AADs can terminate or slow re-entrant arrhythmias by either increasing refractoriness or slowing the conduction velocity. The antiarrhythmic effect of AADs is mediated by blocking ion channels, prolonging the action potential duration (APD) of cells or slowing conduction in the re-entrant circuit. AADs do not shorten the APD of cells or accelerate conduction in a re-entrant circuit; adenosine is unique in this respect. Adenosine shortens APD in atrial tissue, which may accelerate conduction in the

Figure 7 Intracardiac electrograms of the tachycardia obtained after adenosine administration via a peripheral vein (AV, atrioventricular; TCL, tachycardia cycle length; VA, ventriculoatrial).

Figure 8 Intracardiac electrograms obtained 20 s later (AV, atrioventricular; TCL, tachycardia cycle length; VA, ventriculoatrial).

atrium.⁴ We have reported patients with atrial fibrillation and manifest Wolff-Parkinson-White syndrome developing ventricular fibrillation after adenosine administration, due to enhancement of conduction via the accessory pathway.⁹ Adenosine's electrophysiological effects may be classified as primary or secondary.

Primary effects of adenosine

The primary effects of adenosine in the sinoatrial node and atrial myocardium are mediated through the activation of a specific outward potassium current, Ach/adenosine-regulated potassium current.¹ As a result, there is (1) hyperpolarisation and resultant slowing of the sinus node, (2) depression of action potential in the AV node, (3) mild inhibition in inward calcium current (I_{Ca}) in atrial myocytes and (4) suppression of I_{Ca} current in ventricular myocytes, leading to negative dromotropic, chronotropic and inotropic effect. These mechanisms explain how adenosine terminates SVTs. In addition to this, adenosine also shortens atrial APD, thereby reducing the effective refractory period in atrial tissue, and accelerating atrial flutter and fibrillation.⁴

Secondary effects of adenosine

There can be secondary heart rate acceleration after adenosine-induced bradycardia or hypotension due to peripheral vasodilation. Our patient presented with AVNRT at a rate of 165 bpm, which accelerated to 185 bpm seconds after administration of 12 mg of adenosine in the absence of any drop in blood pressure and only mild bradycardia for 2–3 s. Increase in heart rate may have been mainly due to reflex increase in sympathetic activity, corroborated by studies showing increase in sympathetic nerve traffic in carotid body chemoreceptors after adenosine administration.¹⁰

Effect of adenosine in AVNRT

The re-entrant circuit in AVNRT consists of an anterograde slow pathway and a retrograde fast pathway. The direct effect of adenosine on both of these pathways is to increase refractoriness

and slow conduction—thus, adenosine's direct effect is to terminate or slow the tachycardia by blocking conduction in either the slow and/or the fast pathway, not accelerate it.³ The secondary sympathetic stimulation, which generally follows tachycardia slowing and/or hypotension, can accelerate conduction in the anterograde slow pathway, which is more sensitive to adrenergic stimulation. Since adenosine terminates SVT in 90–99% of patients, tachycardia acceleration may only be appreciated in a subset of those in whom adenosine does not terminate the SVT; this is likely the reason for the rarity of this phenomenon.^{5–7}

Learning points

- ▶ Rapid intravenous bolus of adenosine terminates 90–99% of supraventricular tachycardias (SVTs) by slowing conduction in the slow or the fast pathway in the atrioventricular (AV) node.
- ▶ Adenosine may rarely accelerate SVTs—only a handful of cases have been reported in the literature.
- ▶ The main mechanism of tachycardia acceleration may be secondary sympathetic stimulation, which may be preceded by transient bradycardia and/or hypotension and accelerates conduction in the slow pathway.

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