Adenosine-induced worsening of supraventricular tachycardia

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SUMMARY

An approximately 20-year-old to 30-year-old patient presented with a haemodynamically stable supraventricular tachycardia. The patient was managed with intravenous adenosine primarily, with two bolus doses of 6 and 12 mg. This, however, caused a rare paradoxical surge of tachycardia with mild haemodynamic compromise. The patient further required a combination of Metoprolol and Verapamil administration to slow down and reverse the arrhythmia. Following this the patient remained stable with no further episodes till discharge.

BACKGROUND

Adenosine is the most common drug used to reverse a haemodynamically stable supraventricular tachycardia (SVT). It is used extensively across most practices, especially in the emergency room (ER). The most common concern with the use of adenosine however is for bradycardia and the possibility of tachyarrhythmia is not generally considered. There have only been a few publications discussing this and that is why this case presentation maybe of importance. I want to highlight the real possibility of this situation which may occur in multiple clinical practices (ER, Cardiology, OR, etc) and may cause possible critical complications.

CASE PRESENTATION

A patient underwent elective surgery under general anaesthesia and had an uneventful procedure. During the recovery period the patient developed a narrow complex tachycardia with no significant haemodynamic compromise. The patient's heart rate was 146 bpm (figure 1) with a blood pressure of 120/70 mm Hg. The ECG showed a narrow complex, short RP tachycardia (RP<40 ms). The patient was administered a bolus dose of 6 mg of adenosine intravenous, followed by another dose of 12 mg bolus for no response. At this point the heart rate increased to 194 bpm (figure 2) and blood pressure dropped to 96/70 mm Hg. The patient was then managed with intravenous metoprolol 2.5 mg in two repeated doses, which reduced the heart rate considerably. However, the arrhythmia continued. Intravenous verapamil 2.5 mg over 2 min was then administered and a 10 mg infusion over 30 min was continued. With this the rhythm reverted back to sinus (figure 3).

OUTCOME AND FOLLOW-UP

The patient's haemodynamics remained stable during the period of observation in the coronary care unit and in the wards. There were no further recurrences in hospital. The patient was discharged

in a stable condition and followed up after 2 weeks. There has been no recurrence or any fresh complaints since discharge.

DISCUSSION

Adenosine, an endogenous nucleoside, is known to depress sinus node as well as atrioventricular node function. Adenosine shortens atrial action potentials, reducing the effective refractory period and thus promoting the development of atrial flutter and fibrillation. In addition, adenosine causes a reflex increase in circulating catecholamine levels and sympathetic nerve traffic by sympathetic stimulation in the carotid body chemoreceptors. This results in a transient sinus tachycardia and atrial or ventricular ectopy and these ectopics may provoke the reinitiation of re-entrant arrhythmias.¹

Adenosine infusions increase heart rate and systolic blood pressure which could be due to adenosine mediated activation of an autonomic reflex mechanism.²

The mechanism of worsening of atrial arrhythmias following adenosine administration can be multifactorial.³

1. Adenosine could favour conduction through the accessory pathway (AP) by slowing conduction over the aortic valve node and decreasing concealed retrograde conduction into the AP by normally conducted beats.
2. Adenosine may shorten the AP effective refractory period (APERP) directly, leading to an increased ventricular rate.
3. Adenosine may induce a reflex increase in adrenergic tone brought about by its peripheral vasodilatory effect.
4. Ventricles are more arrhythmogenic under catecholamine stimulation.
5. Adenosine induced activation of a reflex autonomic mechanism.

There have been case reports published describing adenosine causing ventricular arrhythmias, however a worsening of SVT with the use of adenosine has not been published.

This clinical case scenario, demonstrated that adenosine is capable of causing transient increase in the ventricular rate. This maybe due to its direct action of modifying the electrophysiological properties of the AP or indirectly by enhancing catecholamine activity or reflex autonomic mechanism. However, unlike intravenous calcium channel blockers, which frequently lead to significant haemodynamic deterioration, the effects of adenosine are brief.

During an enhanced AP activity with adenosine, the presentation may be as a broad or a narrow complex tachycardia depending on the location of

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Figure 1 ECG at presentation.

the pathway and the former may be difficult to differentiate from a ventricular tachycardia, if sudden haemodynamic compromise develops where immediate action will be necessary.

Adenosine remains the drug of choice for treating SVT and hence it is important to keep in mind various possible proarrhythmic complications that could arise with adenosine

Figure 2 ECG postadenosine 6 and 12 mg bolus.

Figure 3 ECG after reverting to normal sinus rhythm.

administration and backing up with a Direct Current cardioversion, kept on standby in all cases.

Competing interests None.

Patient consent Obtained.

Learning points

- ▶ Adenosine though short acting and the drug of choice for supraventricular tachycardia (SVT) is not always benign.
- ▶ Tachyarrhythmia is a rare but critical side effect of adenosine.
- ▶ β -blockers may be useful in adenosine induced catecholamine surge effects.
- ▶ It is important to anticipate and be prepared for this uncommon side effect of adenosine prior to its administration for SVT.

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