Haemodynamic deterioration after treatment with adenosine

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Abstract
A 26 year old woman presented with a narrow complex tachycardia with a rate of 210 beats/min. Adenosine converted this to atrial fibrillation with a rate of 280 beats/min with associated haemodynamic deterioration that needed electrical cardioversion.

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Adenosine is now the first line treatment for narrow complex tachycardias. It has largely replaced verapamil, which may accelerate pre-excited atrial fibrillation and depress ventricular function. Adenosine is considered to be a relatively safe drug with a short half life and a low incidence of proarrhythmia. We describe a case in which adenosine converted a narrow complex tachycardia to rapid pre-excited atrial fibrillation and resulted in considerable haemodynamic deterioration.

Case report
A 26 year old woman presented to the accident and emergency department of the referring hospital with sustained palpitation for one and a half hours. She had had palpitation for several years. There was no family history and she did not take any medication. On arrival at the casualty department she was haemodynamically stable with a blood pressure of 140/80 mm Hg. An electrocardiogram showed a regular narrow complex tachycardia with a rate of 210 beats/min (fig 1). Carotid sinus massage had no effect. An intravenous bolus of 3 mg of adenosine ended the tachycardia but within three beats this was replaced by an irregular broad complex tachycardia with a ventricular rate of 280 beats/min. The configuration was suggestive of pre-excited atrial fibrillation.

Figure 1 12 Lead electrocardiogram on admission showing a regular narrow complex tachycardia.
Figure 2  Electrocardiogram recorded when adenosine was given showing end of the tachycardia for three beats then an irregular broad complex tachycardia (pre-excited atrial fibrillation).

Figure 3 12 Lead electrocardiogram after electrical defibrillation showing a short PR interval and delta waves compatible with Wolff-Parkinson-White syndrome.

atrial fibrillation (fig 2). The casualty officer could not end this with two further boluses of 6 and 9 mg adenosine. During this time the patient became pale and clammy, her blood pressure dropped to 100/60 mm Hg, and she was thus cardioverted under sedation. 50 J had no effect; 100 J produced ventricular fibrillation that was defibrillated with 300 J. A 12 lead electrocardiogram after this showed a sinus tachycardia with a short PR interval and delta waves consistent with Wolff-Parkinson-White syndrome with a left free wall accessory pathway (fig 3). Electrophysiological studies confirmed the diagnosis and the pathway was successfully treated with radiofrequency ablation.

Discussion
Adenosine has been found to be equally effective or more effective than verapamil at ending induced junctional tachycardia and has become established as a first line treatment for such arrhythmias. Also, it is used in the diagnosis of broad complex tachycardia. The main advantage of adenosine as a therapeutic and diagnostic agent is its short duration of action with a half life of only a few seconds. Side effects including facial flushing, dyspnoea, choking sensation, chest tightness, and bradycardia are common but transient and rarely a clinical problem. Adenosine is thus widely considered to be a safe drug.

There is no previous report showing a clear correlation between adenosine and the development of atrial fibrillation with haemodynamic deterioration. Adenosine has, however, been reported to accelerate the rate of atrial flutter. This is probably secondary to activation of the sympathetic nervous system produced by adenosine stimulating chemoreceptors in the carotid body.

In this case, adenosine produced rapid
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atrial fibrillation immediately after a transient reduction in heart rate. The likely mechanism for this was a reduction in the atrial refractory period, which has been shown to occur in humans. Although the same group showed that pacing induced atrial fibrillation was facilitated by adenosine11 the clinical incidence is scant. In a multicentre trial comparing adenosine with verapamil three of the 208 patients treated with adenosine developed atrial flutter or fibrillation at 14 min, 90 s, and 45 s after treatment. This incidence was not significantly different from the placebo group (two of 22) or the verapamil group (none of 58).

Pre-excited atrial fibrillation may result in ventricular fibrillation by rapid antegrade conduction along an accessory pathway. The usual treatment is direct current cardioversion and further adenosine was inappropriate in this case. With increasing use of adenosine this uncommon side effect may be seen more often and doctors should be aware of the possibility. This emphasises that resuscitation facilities should always be immediately available when adenosine is given.

1 Adenosine for acute cardiac arrhythmias. Drug Ther Bull 1993;31:49-50.