

Adenosine-induced non-sustained polymorphic ventricular tachycardia

M. ROMER AND R. CANDINAS

Division of Cardiology, Department of Internal Medicine, University Hospital, CH-8091 Zürich, Switzerland

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Adenosine has become widely used because of its diagnostic and therapeutic value in the emergency management of arrhythmias. It produces transient heart block by slowing conduction through the AV node and thus terminates supraventricular tachycardias that involve the atrioventricular node. Bradyarrhythmias of short duration are common side effects of the use of this drug. Premature atrial and ventricular beats have also been reported. The very short half-life and lack of serious adverse effects generally lead to the consideration that adenosine is a safe drug. We describe a 56-year-old woman with a supraventricular tachycardia. To terminate this rhythm disorder intravenous adenosine was given. Interruption of the supraventricular tachycardia was followed by non-sustained polymorphic ventricular tachycardia.

Introduction

Adenosine is a rapidly metabolized endogenous purine nucleoside that has recently become very useful for the treatment and diagnosis of cardiac arrhythmias. It is effective in terminating most supraventricular tachycardias in which the AV node forms part of the reentrant circuit. Additionally, it may unmask an underlying atrial arrhythmia by slowing AV node conduction. Due to the short half-life of adenosine its clinical side effects last less than 1 min and serious haemodynamic adverse effects are absent. Therefore, adenosine has become the preferred drug, compared to verapamil or beta-blockers, for terminating supraventricular tachycardias^[1]. Previously published review articles^[2,3] mentioned that short runs of non-sustained ventricular tachycardias seldom occur after administration of adenosine. To our knowledge, only one study originally reported this rare arrhythmic phenomenon but did not give a detailed description^[4]. In the present case we describe the occurrence of a polymorphic non-sustained ventricular tachycardia after adenosine was given to terminate an episode of supraventricular tachycardia.

Case report

A 56-year-old woman was admitted because of a recurrence of a paroxysmal supraventricular tachycardia. The ECG recording on several occasions between tachycardia episodes had always showed sinus rhythm with normal PR and QTc intervals. Drug therapy comprised verapamil 40 mg once daily and phenobarbital 20 mg. On admission, the patient complained of

chest tightness and palpitations. Blood pressure was 110/80 mmHg, heart rate 180 beats \cdot min⁻¹, and physical examination showed no evidence of heart failure. The ECG indicated the presence of AV nodal reentrant tachycardia, and since the vagal manoeuvres failed to terminate the tachycardia, adenosine was given as a 6 mg i.v. bolus. Immediately after successful termination of the supraventricular tachycardia, a polymorphic non-sustained ventricular tachycardia of 10 complexes (180 beats \cdot min⁻¹) occurred (Fig. 1). Simultaneously the patient complained of transient lightheadedness and chest discomfort. Blood pressure was not monitored. After resolution of the tachycardia the repeated 12-lead ECG and a two-dimensional echodoppler study showed no abnormality. Routine laboratory tests were within normal limits. Neither a bicycle exercise test nor an ambulatory electrocardiogram demonstrated any ventricular arrhythmias. The patient refused to be rechallenged with another bolus of adenosine while in stable sinus rhythm.

Discussion

Sinus bradycardia and bradyarrhythmias (transient sinus arrest, complete heart block or asystole) may follow adenosine administration. Premature isolated atrial and ventricular complexes are often observed after adenosine-induced termination of tachycardias^[1]. The rare occurrence of short runs of ventricular tachycardias has been quoted^[2,3], but no serious consequences of this proarrhythmic effect have been reported^[4]. Despite extensive research on the cardiac and cardiovascular effects of adenosine^[3], the pathophysiological mechanism of the premature ventricular contractions that develop after adenosine-induced termination of a supraventricular tachycardia remains unknown. A direct myocardial proarrhythmic effect seems unlikely because, in contrast to what is observed

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Correspondence: Markus Romer, MD, Division of Cardiology, Department of Internal Medicine, University Hospital, Rämistrasse 100, CH-8091 Zürich, Switzerland.



Figure 1 Polymorphic non-sustained ventricular tachycardia following the termination of the supraventricular tachycardia after adenosine was given. Paper speed is $25 \text{ mm} \cdot \text{s}^{-1}$.

in supraventricular tissue, no direct effect of adenosine (e.g. receptor-mediated activation of potassium channels) has been observed in ventricular myocytes^[3]. However, adenosine activates autonomic reflex mechanism (by chemoreceptor stimulation) and increases sympathetic tone^[5]. Additional factors, such as the unpleasant side effects of adenosine and the preceding supraventricular tachycardia may contribute to raising catecholamines levels and facilitating runs of ventricular extrasystole.

Conclusion

It is known that runs of ventricular premature beats can initiate more severe and even fatal ventricular tachyarrhythmias, especially in patients with organic heart disease. Whatever may be the underlying mechanisms of adenosine-induced ventricular extrasystole, our case illustrates a serious proarrhythmic effect of adenosine. It has to be emphasized that potentially

lethal tachyarrhythmias may follow intravenous adenosine. Thus, instruments for resuscitation, including a DC-cardioverter, are mandatory when this drug is administered.

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