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Supraventricular Tachycardia in a Human Immunodeficiency Virus-Infected Man

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Abstract

Although supraventricular tachycardias and human immunodeficiency virus infections are common diseases by themselves, a combination is not so common. Such a patient was encountered recently and described in this case report.

Because misdiagnosis of tachyarrhythmias is not uncommon and may lead to inappropriate therapy - frequently resulting in acute clinical deterioration or even death, a discussion of management of supraventricular tachyarrhythmias in general was included. The recent introduction of adenosine into clinical use provides an effective agent in, and revolutionizes, the management of patients with supraventricular tachyarrhythmias. Its application, both for diagnostic and for therapeutic purposes, was discussed in some details in this report.

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Key Words

■ **Supraventricular tachycardia** ■ **Viruses (human immunodeficiency virus infection)**
■ **Drug therapy** ■ **Adenosine**

INTRODUCTION

Recent estimates have suggested that in the United States as many as 5,000 patients per year may have cardiac complications resulting from human immunodeficiency virus (HIV) infection[1]. Although ventricular arrhythmias have been reported in HIV patients due either to the disease itself or to the toxic effects of the various therapeutic agents employed, very little information is available with regard to supraventricular tachycardias in these patients. As a matter of fact, no mention was made of this arrhythmic complication in either the latest edition of a well-known cardiology text[2] or a recent review of cardiac involvement in acquired immunodeficiency syndrome (AIDS)[3]. This presentation reports a case of supraventricular tachycardia in an HIV-infected patient and discusses its management.

CASE REPORT

A consulting cardiologist was asked to see a 28-year-old HIV-infected man for the diagnosis and treatment of a supraventricular tachycardia which the patient developed following hospital admission for abdominal pain, nausea and vomiting the day before. The tachycardia did not respond to 2 consecutive intravenous injections of diltiazem.

Past medical history was significant for cytomegalovirus retinitis for which he had received ganciclovir (Cytovene®). His CD4 +lymphocyte (CD=clusters of differentiation) count had been as low as 19/μl.

Physical examination revealed a cachectic man in moderate cardiorespiratory distress. Blood pressure was 120/80 mmHg. Pulse rate was 210 beat/min. Cardiac examination revealed no murmurs or gallops. Lungs were clear to auscultation and percussion. Abdomen was soft and diffusely tender with diminished bowel sounds.

Electrocardiogram (**Fig. 1−A**) revealed a supraventricular tachycardia at a rate of 210 beat/min with 1: 1 atrioventricular conduction, a normal QRS duration, and nonspecific ST-T changes. After carotid sinus massage and Valsalva maneuver failed to abolish the tachycardia, an intra-

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venous bolus injection of 6mg adenosine resulted in prompt termination of the supraventricular tachycardia (**Fig. 1−B**). A 12-lead electrocardiogram following the termination of the tachycardia was essentially normal except for rare premature atrial contractions (**Fig. 1−C**). Echocardiography did not show any pericardial effusion.

DISCUSSION

Cardiac arrhythmias in HIV-infected patients

As HIV infection is a systemic disease, cardiac involvement is not uncommon[4]. Although cardiomyopathy and pericardial effusion are the 2 most common abnormalities associated with HIV infection[4], a variety of cardiac arrhythmias and possible arrhythmia-related events have been described in patients with HIV infections[5]. The arrhythmias may be the results of a combination of factors, including myocarditis, cardiomyopathy and pericarditis; proarrhythmias from medications such as ganciclovir (Cytovene®)[6] and pentamidine (NebuPent®)[5]; and cardiac sympathetic activation[5]. Although ventricular tachyarrhythmias are more important and potentially lethal, supraventricular tachycardias can often be distressing, though seldom fatal, to the patient.

Management of supraventricular tachyarrhythmias

Distinguishing the 2 commonest types of supraventricular tachyarrhythmias - paroxysmal atrial tachycardia and atrial flutter - is important in planning appropriate therapeutic strategies. Although the atrial rate is slower in paroxysmal atrial tachycardia than in atrial flutter, the overlap in heart rates with these 2 types of supraventricular tachyarrhymias, especially if the atrial flutter is associated with varying degrees of atrioventricular block, makes this feature not very helpful in establishing a definitive diagnosis.

Various vagal maneuvers, including eyeball pressure (**Fig. 2**) and carotid sinus massage (**Fig. 3**) will either terminate the paroxysmal atrial tachycardia or not affect the tachycardia at all. On the other hand, carotid sinus massage will increase the atrioventricular block in atrial flutter, thereby revealing the flutter waves (**Fig. 4**).

Diagnostic and therapeutic uses of adenosine for supraventricular tachyarrhythmias

Since the first short clinical report in 1983[7] on the use of adenosine to terminate some reentrant supraventricular tachyarrhythmias, subsequent and larger studies[8,9] have confirmed its efficacy. In atrioventricular nodal reentrant tachycardia, adenosine usually terminates the arrhythmias by producing block during anterograde conduction over the slow atrioventricular nodal pathway. Other mechanisms for termination, including retrograde fast pathway block and termination by atrial or ventricular extrasystoles, may also contribute to the termination. In typical cases of atrioventricular reciprocating tachycardia, adenosine also terminates the arrhythmia by blocking anterograde atrioventricular nodal conduction. Consistent with the known pharmacokinetic properties of the drug, the tachycardia terminates within 15 to 25 sec after injection (**Fig. 1−B**). Negative effects on blood pressure are not observed, because most of the adenosine administered is metabolized before it reaches the peripheral arterioles. Adenosine should be given as a rapid bolus injection through a large peripheral vein, followed by a saline flush. It is best administered according to an incremental protocol, thereby minimizing the incidence of side effects[10]. Adenosine is cleared from the circulation so rapidly that no accumulation of the drug occurs between multiple doses given at intervals of more than 30 sec. For adult patients the dose may start at 6mg, followed by 12mg if the first dose is ineffective.

Side effects occur commonly with intravenous adenosine injection but are generally benign. They are well tolerated by patients with supraventricular tachycardia but less well by normal subjects[10]. The most commonly reported side effects are facial flushing, chest pain or pressure, and dyspnea. Theophylline attenuates the chest pain suggesting that a direct effect on adenosine receptors in involved. Adenosine should be used with caution is patients with a history suggestive of reversible airway obstruction and should be avoided in patients known to have asthma.

Rarely, several seconds of sinus bradycardia, sinus arrest or atrioventricular block may be seen when adenosine terminates episodes of supraventricular tachycardia (**Fig. 5**), especially if the interval of the incremental dosing schedule is too short. In most patients, this has no clinical significance. However, in a patient at risk for bradycardia-dependent arrhythmias, polymorphic ventricular tachycardia may result[9]. Therefore, it should be emphasized that, as with all antiarrhythmic agents,

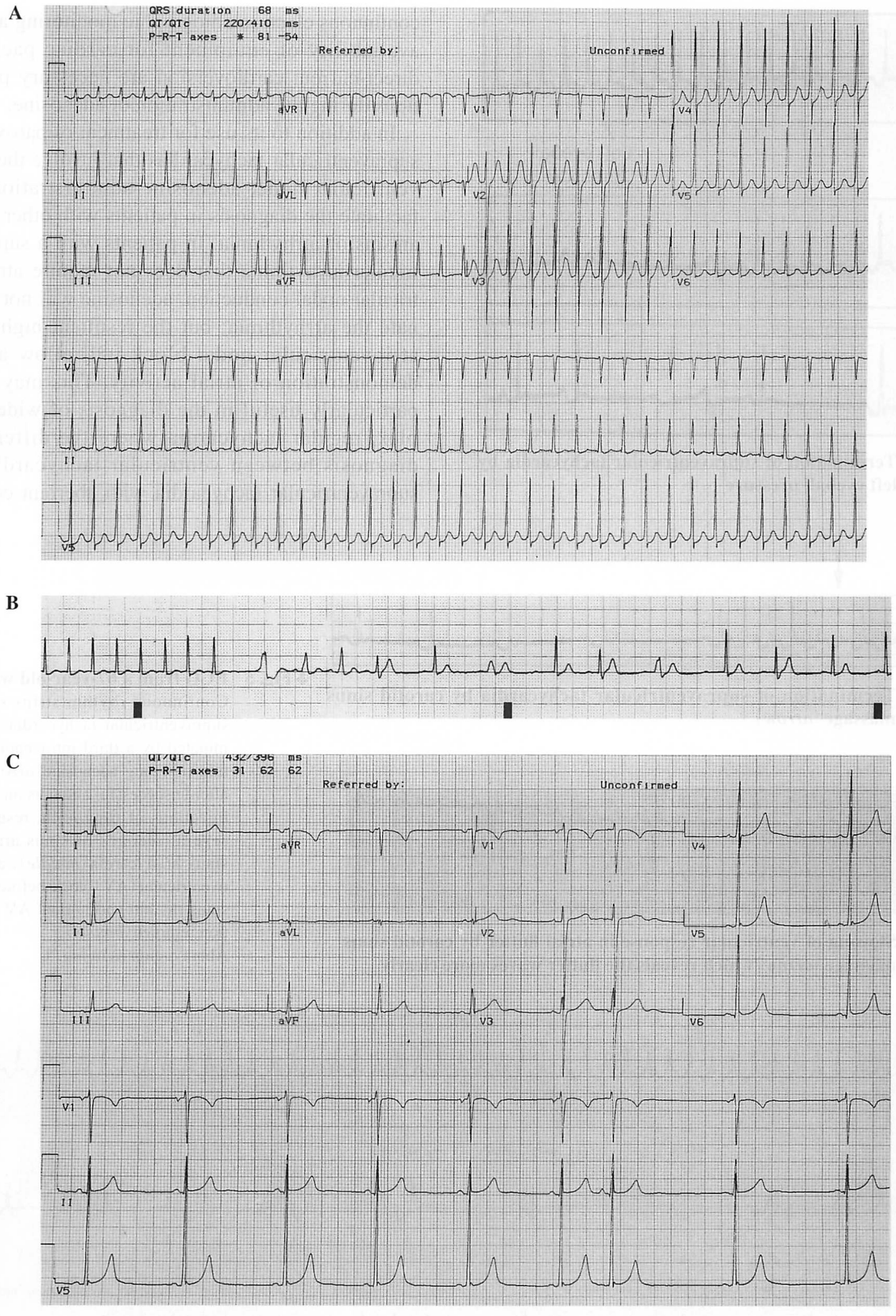


Fig. 1 Electrocardiograms (ECG) from a 28-year-old man with HIV infection
Supraventricular tachycardia (*A*) was promptly terminated by an intravenous injection of 6 mg adenosine (*B*) which restored the normal sinus rhythm (*C*). Injection of adenosine was given a few seconds before the start of ECG recording in *B*.

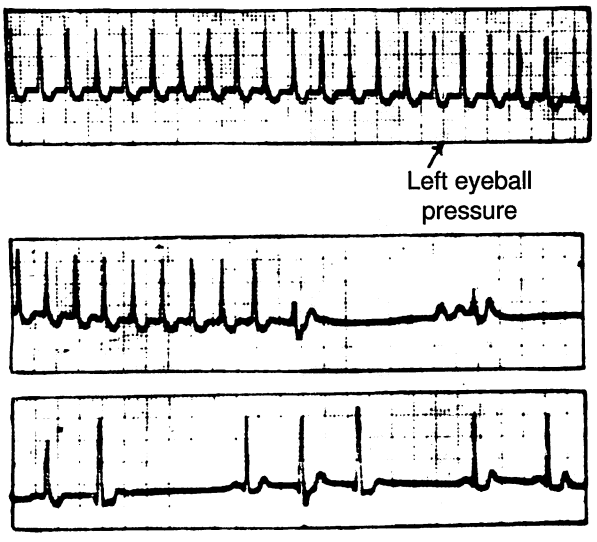


Fig. 2 Termination of supraventricular tachycardia by left eyeball pressure

continuous electrocardiographic monitoring and the availability of equipment for cardiac pacing or direct-current cardioversion are necessary precautions during the administration of adenosine.

In addition to its use for treatment of paroxysmal supraventricular tachycardias that involve the atrioventricular node, adenosine administration may facilitate the diagnosis in patients with other mechanisms of arrhythmia. In patients with a supraventricular arrhythmia that does not require atrioventricular nodal conduction, adenosine will not terminate the arrhythmia; but the resultant high-grade atrioventricular nodal block will allow a clear demonstration of atrial activity. This may prove particularly useful in the diagnosis of wide-complex, regular tachycardia where the differential diagnosis between ventricular tachycardia and supraventricular tachycardia with aberrant conduc-

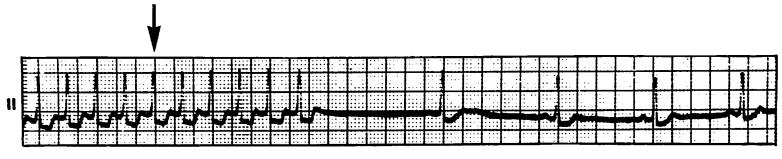


Fig. 3 Termination of supraventricular tachycardia by carotid sinus massage (*arrow*)

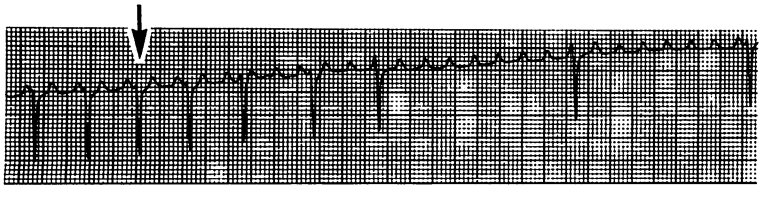
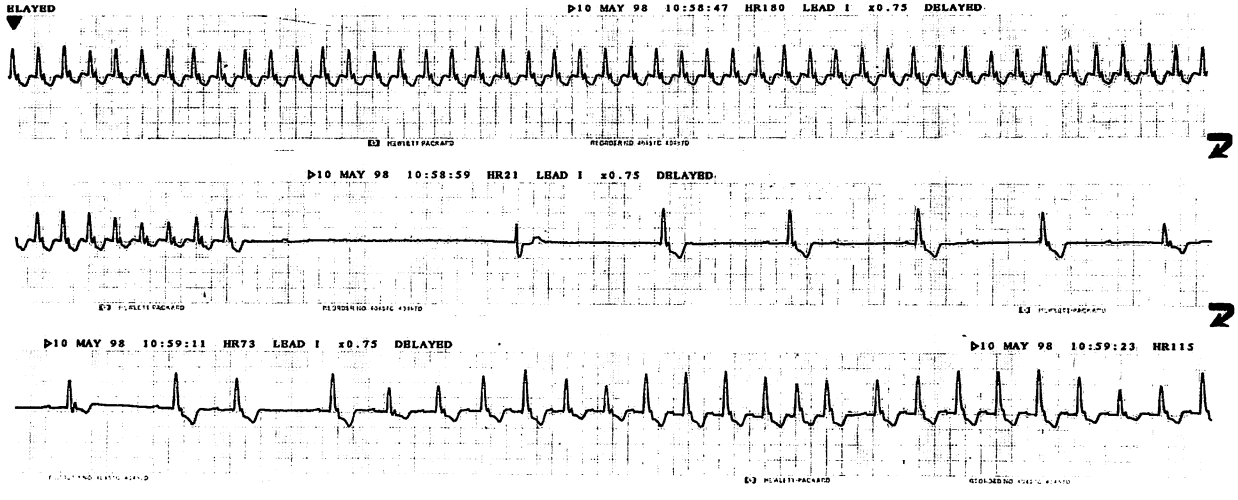


Fig. 4 Slowing of ventricular response in atrial flutter by carotid sinus massage (*arrow*) which reveals the flutter waves more clearly

⬇Fig. 5 ECG from a 67-year-old woman
Continuous rhythm strips showing supraventricular tachycardia (*top*) terminated by a third intravenous injection of 12 mg adenosine (*arrowhead*), after unsuccessful attempts on 2 earlier injections of 6 mg each, resulting in several seconds of sinus arrest and sinus bradycardia (*middle*), and atrioventricular (AV) block before normal sinus rhythm and normal AV conduction returned (*bottom*).
Abbreviation as in Fig. 1.



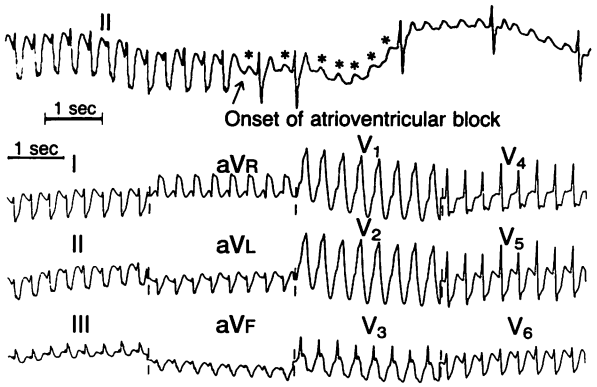


Fig. 6 Differential diagnosis of wide-complex, regular tachycardia by intravenous administration of adenosine

Upper panel shows the unmasking of atrial flutter in a 32-year-old man with dilated cardiomyopathy and a spontaneous episode of wide-complex, regular tachycardia (Lead II). Fifteen seconds after intravenous administration of 12 mg adenosine, the ventricular rate slowed, revealing flutter waves (*asterisks*) occurring at a rate identical to that of the original wide-complex tachycardia. Note that at the slower ventricular rate the QRS complexes are narrow, suggesting that the aberrant conduction to the ventricles was rate-related.

Lower panel shows the 12-lead electrocardiogram of the arrhythmia in the same patient that would be considered diagnostic of ventricular tachycardia. The response of the spontaneous arrhythmia to intravenous adenosine provided the correct diagnosis. The patient was taking disopyramide when these electrocardiograms were recorded (Reproduced by permission of Camm and Garratt)[10].

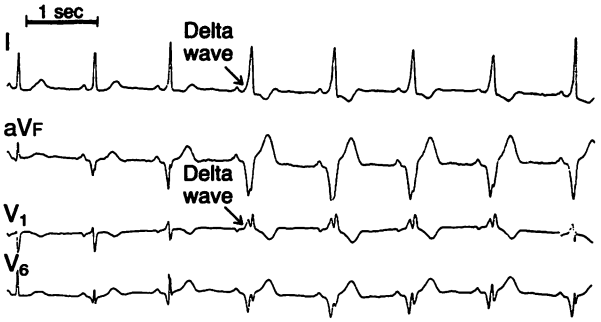


Fig. 7 Unmasking of latent preexcitation by 10 mg adenosine given intravenously during sinus rhythm in a patient with an accessary connection in the left posteroseptal position

The first QRS complex appears normal, after which there is the progressive appearance of a delta wave in all 4 leads shown (Reproduced by permission of Camm and Garratt)[10].

tion remains uncertain after analysis of the 12-lead electrocardiogram (**Fig. 6**)[10]. In fact, no patients with ventricular tachycardia had hemodynamic deterioration after the administration of adenosine.

Adenosine may also have a diagnostic role when administered during sinus rhythm to patients with a documented history of paroxysmal supraventricular tachycardia. Adenosine may reveal latent preexcitation in a number of such patients by slowing or blocking conduction to the ventricles through the atrioventricular node, thus exposing conduction by an accessory pathway (**Fig. 7**)[10]. Patients with latent preexcitation, as opposed to those with intermittent preexcitation, may be at risk for rapid ventricular rates if atrial fibrillation develops.

要　約

上室頻拍やヒト免疫不全ウィルス感染それ自体は日常よくみられる疾患であるが，その両者の合併はそうありうるものではない．最近そのような1例を経験したので報告した．

頻脈性不整脈の誤診は稀なことではないが，不適切な治療となってしばしば急激な臨床症状の悪化，更には死を招くことがあるので，上室頻脈性不整脈に対する対処法を概説した．

近年，adenosineの臨床応用が行われるようになり，上室頻脈性不整脈例の革新的な治療が可能となった．本報告では診断と治療に対する本剤の適用についてやや詳しく論じた．

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