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Case Report

Adenosine triphosphate-induced life-threatening arrhythmia

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ABSTRACT

A 68-year-old woman with idiopathic dilated cardiomyopathy presented with a wide QRS complex regular tachycardia five days after mitral valve replacement. Adenosine triphosphate (ATP) was administered to make the correct diagnosis; however, tachycardia eventually transitioned to ventricular fibrillation, which required cardioversion. Although ATP is considered a relatively safe drug, it can cause unexpected, life-threatening arrhythmias. Careful monitoring and preparation are advised during ATP administration in the event of a regular wide QRS complex tachycardia in patients with irritable conditions.

Learning objective: Adenosine triphosphate (ATP) is considered a safe drug that is often used to manage wide QRS complex tachycardia. Herein, we present a case of regular, wide QRS complex tachycardia in a patient who underwent mitral valve replacement. Tachycardia degenerated into ventricular fibrillation soon after ATP administration, probably because of sympathetic overdrive secondary to the ATP infusion. It is advisable to use ATP with caution, especially in irritable cases such as in the early post-cardiac surgery period.

Introduction

It is often difficult to distinguish between supraventricular tachycardia with aberrant conduction and monomorphic ventricular tachycardia (VT). Adenosine triphosphate (ATP) is often used as a first-line drug in hemodynamically stable patients. When administered under appropriate conditions, a rapid intravenous bolus injection of ATP can suppress atrioventricular nodal conduction and terminate supraventricular tachycardia by creating transient atrioventricular block [1]. ATP (and adenosine) administration can be harmful in cases of pre-excited atrial fibrillation (AF) since they can cause ventricular fibrillation (VF). Moreover, apart from pre-excitation syndromes, previous reports have shown that ATP administration can induce other serious events, such as tachycardia acceleration in atrial flutter [2], torsades de pointes associated with QT prolongation after adenosine-induced atrioventricular block [3], and degeneration of monomorphic VT to VF in cases of dilated cardiomyopathy [4]. ATP-induced complications have also been reported in patients without concomitant factors [5]. It is easy to estimate the mechanism by which life-threatening tachyarrhythmia develops in cases of pre-excited AF or adenosine-related bradycardia-induced QT prolongation. However, it is difficult to determine the underlying mechanisms that cannot be attributed to these factors. Most studies have suggested that the catecholamine surge induced by adenosine may be involved in the induction of life-threatening tachyarrhythmia [2,4,5]. Herein, we report a case of ATP-induced VF that supports this hypothesis.

Case report

A 68-year-old woman was diagnosed with dilated cardiomyopathy that required repeated hospitalizations (New York Heart Association class III). Transthoracic echocardiography revealed a left ventricular end-diastolic diameter of 83 mm and a left ventricular ejection fraction of 29 %. Furthermore, left ventricular basal wall thinning and mitral annular dilatation induced severe functional mitral regurgitation. The patient was treated with adequate doses of beta blockers, angiotensin-converting enzyme inhibitors, mineral corticoid receptor antagonists, and tolvaptan. Despite optimal medical treatment, the symptoms of heart failure did not improve sufficiently and the brain natriuretic peptide level increased to approximately 600–800 pg/mL. The patient was admitted to our hospital with acute decompensated heart failure several days after AF onset. Treatment with inotropic agents was required even after sinus rhythm was restored. Because percutaneous mitral valve repair with mitral clip was not suitable for a short posterior leaflet, we opted to replace the mitral valve with a 31 mm prosthetic valve, followed by the Maze procedure.

After the operation, the severity of mitral regurgitation reduced to grade 1+. However, postoperative left ventricular ejection fraction

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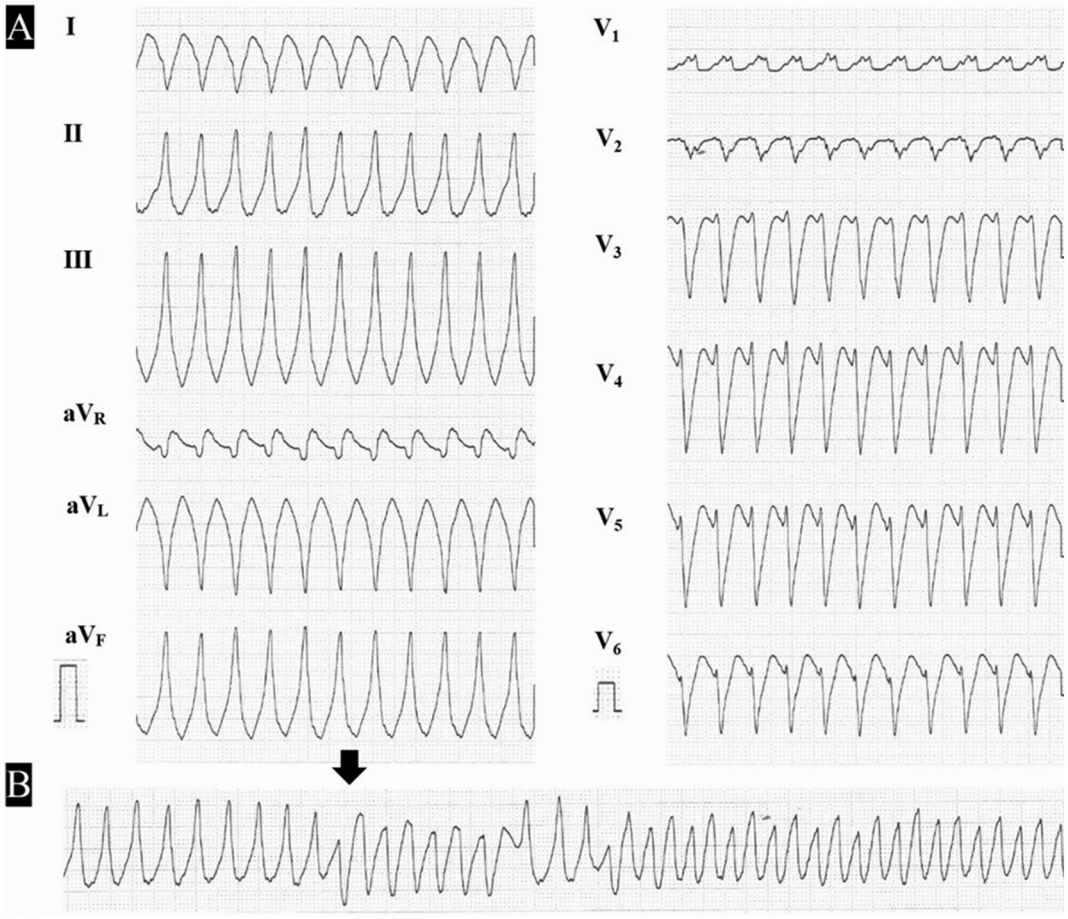

Fig. 1. (A) Twelve-lead electrocardiogram showing wide QRS regular tachycardia with a right bundle block pattern and inferior axis. (B) Conversion to ventricular fibrillation after adenosine triphosphate administration. Arrows indicate the point of sudden transition to ventricular fibrillation. Note that shortening of the ventricular tachycardia cycle from 300 ms to 280 ms occurred immediately before conversion to ventricular fibrillation.

decreased to 20 %. Her blood pressure was 90/60 mmHg with the use of inotropic agents. Five days after the operation, the patient presented with regular wide QRS complex tachycardia (heart rate, 180 bpm) and was conscious of a systolic blood pressure of >70 mmHg. On 12-lead electrocardiography (ECG), the QRS complex showed a right bundle block pattern with an inferior axis (Fig. 1A). Although the QRS morphology during tachycardia (not resembling that of sinus rhythm of complete right bundle branch block; Fig. 2) suggested possible VT rather than supraventricular tachycardia, the diagnosis of VT was not definitive since neither atrioventricular dissociation nor fusion of the QRS complexes (Dressler

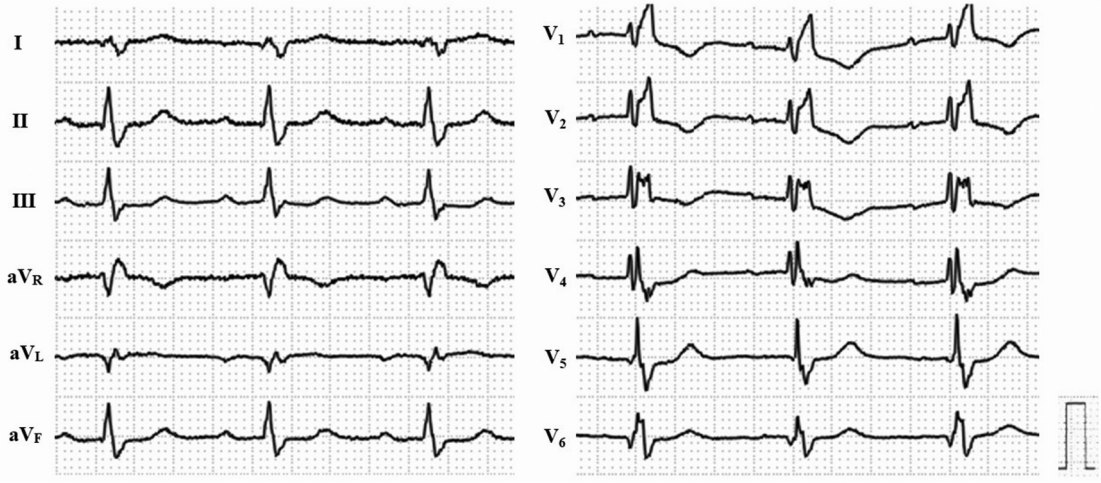

Fig. 2. Twelve-lead electrocardiogram during sinus rhythm showing complete right bundle branch block.

beats) was confirmed. Therefore, ATP (10 mg intravenous (IV) bolus) was administered intravenously to treat the differential diagnosis. Immediately after ATP administration, the patient's heart rate increased unexpectedly (from 186 to 210 bpm) and eventually transitioned to polymorphic VT, degenerating into VF (Fig. 1B), which required repetitive electrical shocks. After restoration of sinus rhythm, intravenous amiodarone was administered. Since arrhythmia was considered to be VT, the patient received cardiac resynchronization therapy with a defibrillator to prevent sudden cardiac death.

Discussion

Differentiating monomorphic wide QRS complex tachycardias is a common problem in clinical settings. Accurate diagnosis of wide QRS tachycardia is not only essential for acute arrhythmia management, but also for assessing the need for additional treatment, including optimizing medical therapy and/or implantable cardioverter-defibrillators for improved prognosis. Although ECG remains a cornerstone diagnostic tool for wide QRS tachycardia, accurate diagnosis is often challenging.

ATP can be used as a diagnostic tool to discriminate monomorphic VT from aberrant supraventricular tachycardia in hemodynamically stable patients. ATP is almost always effective in terminating paroxysmal supraventricular tachycardias in which the atrioventricular node forms part of the reentrant circuit. However, VT does not cease after ATP administration except in the ATP-sensitive type, which is caused by triggered activity or abnormal automaticity. ATP has a short half-life (<10 s), and is considered safe. Transient side effects, including sweating, mild headaches, respiratory distress, nausea, and vomiting, may occur, but are rarely life-threatening. Ertan et al. reported that after adenosine administration in 52 patients with supraventricular tachycardia, 47.8 % of the patients exhibited high proarrhythmic effects such as premature ventricular contraction and non-sustained VT [6]. Non-sustained VT was observed in 17.8 % of the cases. Tan et al. reported that adenosine (average dose 9.7 mg) was used in the emergency department for supraventricular tachycardia in 127 patients over a 5-year period. Among them, ventricular arrhythmia was induced in approximately two-thirds of the patients but ceased spontaneously without transition to persistent VT or VF [7]. However, when ATP (and adenosine) is administered to patients with wide QRS tachycardia and organic heart disease to differentiate supraventricular tachycardia from VT, the risk of adverse drug reactions may increase. Lerman et al. examined the effects of intravenous adenosine on reentrant VT by administering adenosine to 31 patients with VT due to structural heart disease. None of the patients experienced hemodynamic collapse after adenosine administration, suggesting that adenosine has neither protective nor harmful effects on reentrant VT due to structural heart disease [8]. Only one case of VT transitioning to VF after adenosine administration has been previously reported [4]. The authors speculated that the catecholamine surge after adenosine-induced vasodilation may have contributed to VF degeneration, although the precise mechanism remains unknown. In our case, the tachycardia cycle length was shortened despite the unchanged QRS morphology immediately after ATP administration. Accelerating tachycardia may be a predisposing factor for the VT to VF transition. ATP is known to have a direct electrophysiological effect and can shorten the action potential duration in the atria but not in the ventricle [9]. Furthermore, it did not affect the tachycardia cycle length in reentrant ventricular tachycardia. Therefore, the mechanism of the shortened tachycardia cycle length immediately after ATP administration might be a consequence of increased conduction velocity due to sympathetic activation. Newly developed VT in the setting of post-operative heart failure may result in the release of endogenous catecholamines. Under these circumstances, intravenous ATP may further enhance the catecholamine surge. This is a possible mechanism of VT cycle length shortening immediately after ATP injection, which resulted in the VF provocation. We believe that our case provides important insights into the mechanism by which ATP (and adenosine) induce VF in patients with reentrant VT.

In this patient, there was no evidence to clearly deny that the arrhythmia was supraventricular, despite the possibility of VT. Even if a definitive diagnosis of VT is made, secondary prevention of sudden cardiac death using an implantable cardioverter-defibrillator is not indicated because it could not be denied that VT is due to transient factors. We decided to implant a cardioverter defibrillator according to the criteria for primary prevention of sudden cardiac death in patients with non-ischemic cardiomyopathy based on the Japanese Circulation Society (JCS) guidelines (receiving optimal medical therapy, New York Heart Association class II or greater symptoms, and left ventricular ejection fraction ≤35 %) [10]. ATP and adenosine are considered safe and useful drugs for discriminating between aberrant supraventricular conduction and monomorphic VT. However, caution should be exercised when administering ATP and adenosine owing to the possibility of inducing life-threatening ventricular arrhythmias in patients with structural heart disease.

Patient permission/consent statement

Written informed consent was obtained from the patient.

Declaration of competing interest

The authors declare that there is no conflict of interest.

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