

# Failure of Adenosine to Convert a Subtype of Supraventricular Tachycardia

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## Introduction

Paroxysmal supraventricular tachycardia (SVT) is the most common significant arrhythmia encountered in pediatric practice (1). It is well tolerated by most infants and children, but potentially, if persistent, can lead to heart failure and cardiomyopathy. Adenosine is the drug of choice in children with hemodynamically stable SVT or hemodynamically unstable SVT with ready open vascular access (2). In a study conducted in an emergency department, intravenous administration of adenosine led to successful cardioversion in 72% of pediatric patient-events with a presumed diagnosis of SVT (3).

The permanent form of junctional reciprocating tachycardia (PJRT) is a rare (< 1%) subtype of SVT which occurs predominantly in infants and children. It is often incessant and tends to be refractory to pharmacological therapy (4). Older children may require radiofrequency ablation of the accessory pathways (5).

We report on a four-week-old male infant who presented to our emergency department with SVT which did not convert to steady sinus rhythm with adenosine owing to the presence of PJRT. The purpose of this illustrative case is to increase awareness among physicians of the possible presence of PJRT in cases of SVT resistant to adenosine, which mandates a different approach. The management of SVT as well as the diagnosis and treatment of PJRT are discussed.

## Case Report

G.B., a four-week-old male infant, was referred by his pediatrician to the Emergency Department of the Schneider Children's Medical Center of Israel because of tachycardia, noted on physical examination. The perinatal course has been uneventful until five days prior to presentation, when a rash consistent with varicella was noted. No symptoms, such as vomiting, excessive sweating, or feeding problems, were reported. Physical examination revealed a well-appearing, well-nourished white infant with blood pressure 76/60 mmHg, heart rate 230 beats/min, respiratory rate 45 breaths/min, oxygen saturation 100%, and temperature 37.3° C rectally. There were no signs of dyspnea or heart failure. Heart examination revealed a rapid, regular pulse with no murmurs or extra heart sounds, and there was good peripheral perfusion. The physical examination was otherwise unremarkable. The chest x-ray showed mild cardiomegaly with normal lung vascularity.

On laboratory evaluation, hemoglobin was 10.5 g/dl and hematocrit 32%. The white blood cell count was 7700/mm<sup>3</sup> and platelet count 238,000/mm<sup>3</sup>. Electrolytes, glucose and creatinine were within normal range. The 12-lead ECG showed a rate of 220 beats/min, a narrow QRS complex, inverted P waves in leads II, III, AVF, and a PR interval shorter than the RP interval (**Fig. 1**).

SVT was diagnosed and initial treatment was started. This included oxygen administration by mask and establishment of IV access followed by two attempts to convert the rhythm to sinus using the ice bag maneuver (diving reflex). After the ice

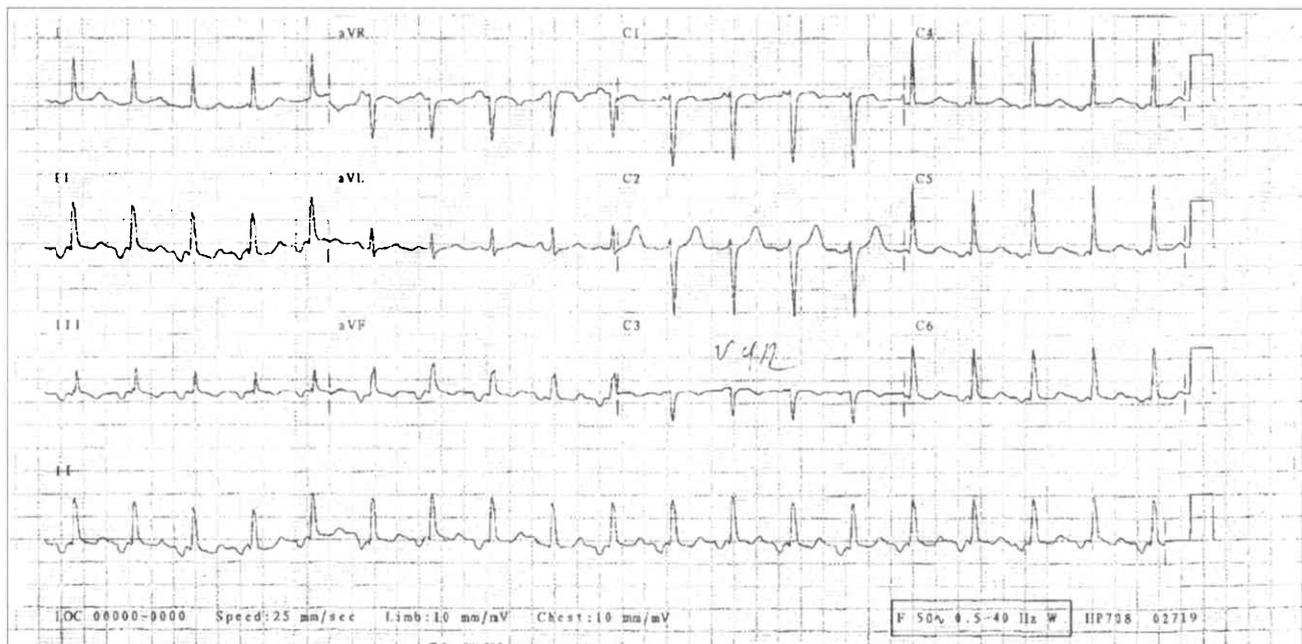


Fig. 1: Twelve-lead ECG demonstrating typical findings of permanent junctional reciprocating tachycardia: inverted P waves in leads II, III, and AVF, and an RP interval which is longer than the PR interval.

bag maneuver failed, IV adenosine boluses were administered. However, doses of 0.05 mg/kg, 0.1 mg/kg and 0.15 mg/kg given at 2-3- minute intervals caused only few seconds of asystole followed by a return to the SVT rhythm, with no change in the initial pattern. The patient remained hemodynamically stable throughout this treatment. At this stage we consulted a pediatric cardiologist who suggested the possible diagnosis of PJRT based on the electrocardiographic criteria and the clinical response to adenosine. A bedside echocardiogram revealed a normal heart anatomy but poor ventricular contractility with a shortening fraction of only 13% during the tachycardia. Owing to the lack of response to adenosine, we attempted to slow down the heart rate with IV digoxin (0.03 mg/kg total digitalization dose over 6 hours); this too failed. Six hours later, IV amiodarone was started, 0.010 mg/kg/min loading dose followed by 0.005 mg/kg/min maintenance dose. Six hours after the initiation of amiodarone, the rhythm converted to sinus, and a repeated echocardiogram showed improvement of the shortening fraction (30%). Amiodarone was gradually tapered off and replaced with sotalol, a class III anti-arrhythmic drug with fewer side effects than amiodarone. Digoxin was continued.

During the next three days the infant remained in sinus rhythm, and repeated echocardiography showed a normal left ventricular function. Digoxin and sotalol were administered for six months and then discontinued. The clinical course, growth rate and findings on repeated echocardiography and Holter monitoring were all normal and remained so until the last follow-up visit at age 1 year.

### Discussion

Our report describes a case of a narrow complex tachycardia which did not convert to a steady sinus rhythm with adenosine therapy. The lack of response was attributed to the nature of the specific type of SVT known as PJRT.

SVT is comprised of two types of arrhythmia mechanisms: 1. That caused by atrioventricular reentry at the sinoatrial node, atrium, atrioventricular junction or an accessory pathway; and 2. That caused by enhanced automaticity in the atrium or

atrioventricular node. Reentry between the atrium and the ventricle is the most common mechanism in pediatric patients (1,6).

PJRT is so named because it was originally considered an atypical form of atrioventricular node reentry. However, later electrophysiological, surgical and anatomic reports confirmed the presence of one or more accessory atrioventricular pathways (5). These pathways conduct only retrogradely with decremental properties and usually have a coronary sinus ostial or a right posteroseptal location (4). Other less common sites have also been reported (9). Children and infants with PJRT may be asymptomatic during the tachycardia or they may develop secondary cardiomyopathy (10). Occasionally - more commonly in young patients - they present with heart failure. The mean tachycardic cycle length increases significantly with age, resulting in slower tachycardic rates at older ages (4). The surface electrocardiogram shows a narrow QRS complex, a retrograde P wave which is usually negative in leads II, III, AVF and in the left lateral leads, and an RP interval that is longer than the PR interval during the tachycardia. During sinus rhythm the electrocardiogram is normal, with no evidence of delta waves. In our case, the electrocardiographic pattern was typical of PJRT. It should be pointed out, however, that a definite diagnosis of PJRT can be made only by electrophysiologic studies, because atypical AV node reentry tachycardia with 1:1 conduction and atrial tachycardia of low atrial origin may be associated with an identical ECG pattern. In our case, the age of the patient, the incessant nature of the tachycardia, and the inability of adenosine to terminate the tachycardia (implying a reentry mechanism) clinically supported the diagnosis of PJRT.

Adenosine is an endogenous nucleoside which, when given in an intravenous bolus dose, slows down conduction through the atrioventricular node by activating the outward potassium current in the cells of the atrium, sinus and atrioventricular nodes (6). It has almost no effect on ventricular tachycardia, because ventricular myocytes are mostly insensitive to it. The half-life of adenosine in the plasma is very short, and therefore side effects are minimal and short-lasting (flushing, chest pain, and dyspnea). The very short half-life of the drug combined with its relative safety and the lack of interference with other cardiac medications give adenosine important advantages over other antiarrhythmic drugs. Adenosine has been shown to brake atrioventricular-reentry SVT in 72-90% of cases (3,8), and is therefore recommended by the American Heart Association as the drug of choice. This property can also serve as a diagnostic tool for the mechanism (reentry vs. ectopic) and site (atrial vs. atrioventricular) of the tachycardia. However, in PJRT, owing to the incessant pattern, adenosine may either stop the tachycardia, causing asystole (as in our case), or convert the tachycardia to sinus rhythm, but only for a few seconds.

Therapy for PJRT is guided by the clinical course. Medical treatment with amiodarone, procainamide, flecainide, propafenone, propranolol, sotalol, quinidine and verapamil, either alone or in combination, has been reported (5) and should be tried first. Unfortunately, pharmacological management is almost always unsuccessful (4). In a recent report on 21 patients, the tachycardia persisted despite multiple antiarrhythmic medications except in one patient who was treated (as in our case) with amiodarone (4). When medical therapy fails, radiofrequency ablation of the accessory pathways, performed during electrophysiologic study in the catheterization laboratory, is the treatment of choice (4,5). Candidates for radiofrequency ablation should be older than one year and weigh more than 10 kg. Dorostkar et al (4) recommended deferring radiofrequency ablation in small children with PJRT as the heart rate usually slows with age. However, being that the tachycardia is associated with both an infrequent spontaneous resolution and a variable expression of impaired ventricular function, and that radiofrequency ablation is safe and effective, we believe that electrophysiologic studies with radiofrequency ablation should be considered in patients of suitable age and weight on the appearance of symptoms related

to the tachycardia and/or impaired ventricular function. It may be performed earlier in the presence of life-threatening cardiomyopathy.

In summary, adenosine is very effective for the treatment of children with SVT presenting to the emergency department. Its failure to convert the SVT may be due to a ventricular origin of the arrhythmia or to the presence of rare subtypes of SVT such as PJRT. Treatment of PJRT is dictated by patient age and clinical condition, and does not include adenosine.

We trust this case will increase the awareness of pediatricians and emergency physicians to the entity of PJRT, which if suspected, can be managed and treated appropriately.

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