

Paroxysmal Supraventricular Tachycardia in the Rural Setting- Case Reports and Review

Thiraviam P. Ashok, Sanjay Sharma, Mahesh Jain

Abstract

A case report of three Omani children age ranging from one month to ten years managed in a district hospital. All three children reverted to normal sinus rhythm after I.V. adenosine and are under follow up. A detailed review of management of Paroxysmal Supraventricular Tachycardia is discussed. Treatment options include no treatment, vagal manoeuvres, long term drug therapy, radiofrequency catheter ablation, and surgery. Recurrence may occur and patients have to be educated about home management and when to seek medical help.

Keywords: PVST children, treatment options, home management.

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From the Department of Child Health, Ibra Hospital, North Sharqiah Region, Oman.

Address correspondence and reprint request to: Dr. Thiraviam Palanikumar Ashok, Department of Child Health, Ibra Hospital, P.O. Box 3, P.C. 400, North Sharqiah Region, Oman.

E-mail: ashoktp7@omantel.net.om

Supraventricular Tachycardia in the Rural Setting

Case 1

In June 2004 a 6-year-old Omani boy walked into our Paediatric Out Patient Department (OPD) with history of having palpitations since morning. His pulse and heart rate were difficult to count. He was immediately shifted to Intensive Care Unit (ICU) and urgent electrocardiogram (ECG) was taken which showed narrow complex tachycardia with heart rate of 194 per min and absence of P waves (figure 1- Top Strip). His blood pressure was 100/73 mmHg. He was comfortable at rest with no distress and not in cardiac failure. We tried carotid sinus massage, which failed to convert Supraventricular Tachycardia. He was not able to perform valsalva manoeuvre. Adenosine Injection 50 microgram/kg was given as I.V. push followed by a saline flush. Immediately his heart rate dropped down to 113/min. His general condition became stable and he slept well with some sedation. The ECG after conversion to sinus rhythm did not show pre-excitation. Chest X-ray and Echocardiography showed normal findings.

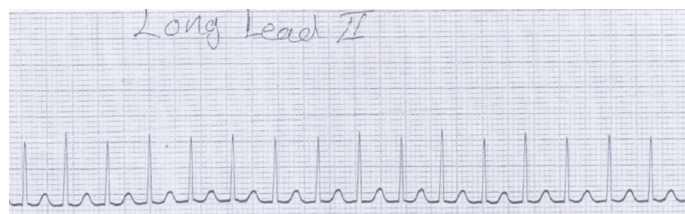


Figure 1: ECG before Adenosine

Case 2

A 10 years old boy presented in January 2004 with complaints of palpitations since morning. His heart rate was 214/min. There was no cardiac murmur and he had been asymptomatic before this. An ECG was done which showed heart rate of above 200/min with

narrow QRS complexes and absence of P waves. (Figure 1 – bottom strips). There was no response to valsalva manoeuvre. Adenosine Injection 1.3 mg was given as a rapid bolus followed by rapid saline flush. Immediately his heart rate came down to 120/min. He was alert and stable. Chest x-ray did not reveal any cardiomegaly. The ECG did not show any evidence of preexcitation. He was sent to SQU cardiology department for cardiac evaluation and his ECHO report did not reveal any cardiac anomaly. 24 hour Holter ECG was normal. He was advised to try valsalva manoeuvre if the symptoms recurred and to contact the hospital if that fails. He was not advised any long-term medication. The child is on follow up. There has been no recurrence till date.

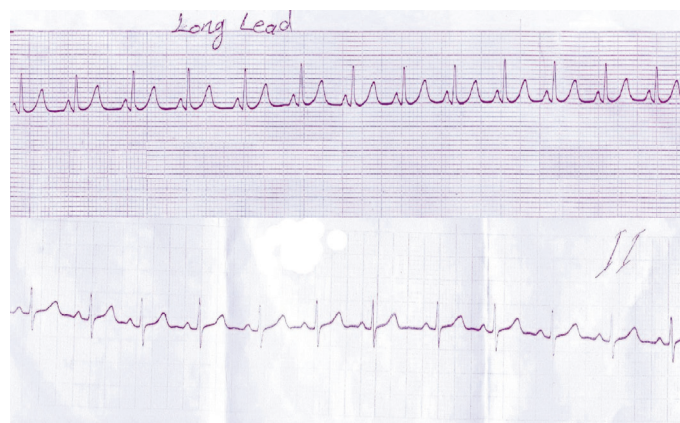


Figure 2: ECG after Adenosine

Case 3

In September 2002 an infant presented to us with history of fever for one day and mild respiratory distress. His peripheral circulation

was adequate and he was fully alert. ECG showed a heart rate of 300/min. with narrow complex. A diagnosis of PSVT was made. Ice pack application on face was tried twice, each for 15 seconds at an interval of 5 minutes with no effect. Adenosine injection was given intravenously followed by a saline flush. The heart rate dropped to around 160/min immediately and after that the baby remained stable. The Basal ECG did not show any pre-excitation. Chest x-ray did not reveal any cardiomegaly. There was no cardiac murmur. After consultation with a Cardiologist, the baby was given oral digoxin for six months. Echocardiography did not reveal any abnormality. There has been no recurrence on follow-up. All these three cases were diagnosed and managed at Ibra Hospital, which is one of the secondary-care hospitals in North Sharqiah region, Oman.

Discussion

Supraventricular Tachycardia can be defined as a sustained, accelerated, non-sinus cardiac rhythm originating above the level of the atrioventricular (AV) junction. Paroxysmal Supraventricular Tachycardia (PSVT) is the most common symptomatic tachyarrhythmia in childhood. The initial onset of this arrhythmia may occur in utero, within the first few weeks of life, or may first occur in adolescence. Although, infants and children are more often affected. SVT using an accessory connection is the most common form of SVT in childhood and accounts for at least 80% of SVT in infancy, decreasing to about 45% of SVT in adulthood. Atrioventricular nodal re-entry tachycardia occurs rarely in infancy, initially occurring in five- to ten-year-old children, and accounting for at least half of SVT in adults. Primary atrial tachycardias account for 10 to 15% of clinical SVT at all ages.¹

Generally infants are more likely to 'outgrow' their SVT. Lundburg showed that up to 70% of infants with SVT do not relapse when treatment was discontinued at the age of one year,² while Perry and Garson reported a recurrence rate of 78% in children aged > 5 years at the time of first episode.³ The presence or absence of structural heart disease did not influence the outcome.⁴ However the presence of WPW syndrome on the surface ECG did indicate a chance for recurrent episodes and even sudden death in symptomatic patients.³

Specific electrocardiogram (EKG) characteristics of paediatric PSVT are:

- Heart rate of greater than 220 beats per minute in infants and greater than 180 beats per minute in children or adolescents.
- Sudden onset and abrupt termination of the arrhythmia.
- Narrow QRS complex (< .08 - .12 seconds)
- Rate is regular with no beat-to-beat variation in rhythm.

- P wave may not be identifiable (if it is, however, the P wave axis is usually abnormal)
- A rate-related phenomenon of 1-8 mm. ST depression, which is not necessarily a sign of myocardial ischemia

The clinical presentation of an infant or child with PSVT will differ depending on the duration of the arrhythmia and the presence /absence of an underlying heart defect or myocardial dysfunction. The fast rate of PSVT may cause a decrease in ventricular diastolic filling time, stroke volume, coronary artery perfusion and cardiac output. Clinical features include some or all of the following: irritability, pallor, lethargy, vomiting, poor feeding, tachypnea, and sweating. If the rate is very high or persistent the child will develop cardiovascular collapse and shock or congestive cardiac failure.

Older children may present with chest pain, dizziness, palpitations, or a syncopal episode.

Known precipitating factors include fever, emotional upset, smoking, caffeine, stimulant and sympathomimetic drugs. But in many instances no precipitating factor can be identified.

In a small number of patients—those with Wolff-Parkinson-White syndrome, in which preexcitation is evident on the electrocardiogram—there is a small but significant risk of cardiac arrest during their tachycardia. Cardiac arrest is due to rapid conduction of atrial fibrillation over the accessory connection to the ventricle, precipitating ventricular fibrillation. Because most episodes of cardiac arrest due to Wolff-Parkinson-White syndrome occur in the first three decades of life, paediatric patients appear to be at the highest risk for this complication. Of most concern is the fact that in almost half of patients suffering cardiac arrest, the initial sustained arrhythmia is the cardiac arrest. Therefore, young patients with Wolff-Parkinson-White identified on ECG should undergo full evaluation to identify individuals who are at risk sudden death.

Treatment of a child with PSVT will depend on a number of factors that include age, severity, and frequency of episodes, underlying heart status, mechanism of SVT and risk stratification.

Treatment strategies for SVT

1. Acute therapy of SVT -options

- *Vagal manoeuvres.
- *Pharmacological conversion--Adenosine.
- *Electrical cardioversion

2. Long-term treatment - options

- *No treatment,
- *Chronic or periodic medications,
- *Catheter ablation,
- *Surgery.

With the advent of adenosine in the physician's pharmaceutical armory, the ease and effectiveness of vagal stimulation is being forgotten. Eyeball pressure and carotid sinus massage is not advisable in children.

- Performing a Valsalva manoeuvre in the supine position, which may be more successful than if the child is standing. (ask the child to blow forcefully through an obstructed straw)
- Physically induced gagging, coughing, retching, or vomiting.

Diving reflex- This is the most effective method of vagal stimulation in the infant or child. There are two techniques:

1. Facial cooling: fill a polythene bag with cold water and ice cubes, place it on the face so that the forehead, eyes, nose, mouth and cheeks are covered; leave for 15 seconds and monitor ECG.
2. Immersion (infants): wrap the trunk and limbs in a towel and immerse head and face completely in a washbasin full of iced water – leave for 5 seconds – do not cover the nose or mouth – the baby becomes temporarily apnoeic. Return to sinus rhythm often occurs at once. It is important to begin trying these manoeuvres immediately, as the longer the PSVT persists, the more difficult it becomes to convert.

Immediate arrangements to an emergency department should be made for all paediatric patients who have no prior history of SVT, patients with known PSVT histories who are symptomatic, or patients who are haemodynamically unstable.

The treatment of choice for symptomatic patients with a stable blood pressure is IV administration of adenosine.⁵ The advantage of this drug is that it is relatively safe, has limited side effects, and has a short half-life of 10 to 15 seconds. It is antagonised by methyl xanthines. (Theophylline and caffeine) (May need higher doses) It is successful in 90 % of cases in restoring sinus rhythm although SVT may recur because of short half-life. The initial dose is 50 to 100 mcg/kg (maximum dose is 300mcg/kg) Due to the extremely short half-life, it should be administered as a rapid bolus over 1 to 2 seconds and as close to the heart as possible, preferably into the antecubital vein using a three-way connection and should be immediately followed with a 2-3 ml. bolus of normal saline. If there is no rhythm change within 2 minutes, a second dose should be administered at twice the first dose. Further doses can be given with increments of 50 mcg/kg at 2-min. intervals until sinus rhythm is restored or the max. Single dose of 300 mcg/kg is reached. Adenosine can also be given by the intraosseous route.^{6,7}

There is a period of cardiac asystole lasting 5–15 seconds followed by return of sinus rhythm. However episodes of junctional and ventricular complexes may be seen during the period of asystole. There is also risk of immediate recurrence of SVT.

The overall incidence of side effects is around 1%. Usual side

effects are confined to autonomic disturbances like a feeling of impending doom, excessive salivation, abdominal pain, vomiting, flushing and headache. Occasionally adenosine can precipitate bronchospasm in a predisposed individual.⁸ More recently, major side effects of the drug have also been reported, such as apnoea, prolonged asystole, accelerated ventricular rhythm, atrial fibrillation and wide complex tachycardia.⁹ Therefore resuscitation equipment should be kept ready before administering adenosine.

These arrhythmias are self-limiting and usually resolve without intervention when the adenosine becomes deactivated. Other drugs can also be tried if adenosine fails (after discussion with cardiologist). Intravenous verapamil is commonly used to terminate PSVT in adults, but are used with greater caution in the paediatric patient. It has caused cardiovascular collapse, profound bradycardia, and death in infants. Intravenous Flecainide (2 mg/kg over 20 min.) is also a very effective treatment in children in case of failure to respond to adenosine. For the patient who is haemodynamically compromised, or in whom pharmacological conversion has failed, synchronised cardioversion (0.5-1.0 Joules/kg) is the treatment of choice.

Recurrences are treated again similar to the initial attack –vagal manoeuvres and adenosine in escalating doses. Frequent recurrences may necessitate therapy with digoxin (in the absence of WPW syndrome) or propranolol (in the presence of WPW syndrome).

The age of the patient and the type of SVT are major determinants for choosing optimal chronic therapy. The presence of preexcitation on electrocardiogram prompts an evaluation for stratification of risk of cardiac arrest plus the avoidance of digoxin or verapamil therapy. The symptoms during tachycardia and frequency of episodes, as well as the sophistication of the patient's caretaker, also are important guides to treatment.

1. No treatment option: May be considered for older children who can recognise and communicate the onset of SVT, have infrequent, well-tolerated episodes of SVT not associated with pre-excitation and those who can try Valsalva manoeuvre to abort the attack or prefer an infrequent visit to emergency room over the chronic medication.
2. The options include digoxin, betablockers, flecainide, amiodarone and some newer drugs. Therapy with any of the second line drugs is to be planned in consultation with a Paediatric Cardiologist.
3. Since 1988, radiofrequency catheter ablation has been used as definitive therapy for most forms of supraventricular tachycardia and some forms of ventricular tachycardia. Using intracardiac catheters, radiofrequency energy is used to desiccate a small, well-circumscribed area of cardiac tissue thought to be essential to the arrhythmia circuit, such as the accessory connection.

In symptomatic patients with recurrent tachycardia over the age of five years, radiofrequency catheter ablation has become the treatment of choice. Patients presenting with tachycardia in this age group are not likely to "outgrow" their tachycardia and are not likely to tolerate or remain compliant with daily medications. In addition, some asymptomatic patients with Wolff-Parkinson-White syndrome who are thought to be at increased risk for cardiac arrest may be referred for ablation. Ablation is performed in-patients between two to five years of age when SVT is refractory to anti-arrhythmic medications or when there is concern over potential drug side effects during long-term administration.

4. The advent of catheter ablation techniques largely eliminated the traditional role of a surgical therapy for accessory-connection mediated tachycardia. In contrast, catheter ablation procedures have poor success rates in patients with atrial re-entry tachycardia following surgical repair of congenital heart disease; mortality from tachycardia in these patients is about 20%. Reasons for these poor results include distorted anatomy, extensive scarring, and the inability of current catheter techniques to deliver lesions of sufficient depth in thickened and hypertrophied atrial tissue. Many of the problems limiting the transcatheter technique could be overcome with a direct surgical approach employing a combination of incisions and cryoablation lesions. At the same time, residual haemodynamic abnormalities creating atrial or ventricular hypertension could be corrected. The long-term management and follow up should always be in consultation with cardiologist.
5. Discharge education for the patient and family;
 - A careful review of the precipitating factors of PSVT must be done. The direct relationship between the avoidance of these factors and the decrease in the number of PSVT episodes must be stressed to the family and understood by them.

- Teach how to count pulse rate and identify SVT recurrence.
- Home management techniques (simple vagal techniques) should be taught to all members of the family.
- Additional education for the family members should be encouraged, e.g. CPR and First Aid courses.
- Emergency numbers should be reviewed with the parent.
- Need for follow up with cardiologist as agreed.

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