Paroxysmal Supraventricular Tachycardia In Infants And Children: A Therapeutic Challenge

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Introduction

Paroxysmal supraventricular tachycardia (PSVT) is the most prevalent and potentially life threatening tachyarrhythmia seen in children, having an incidence of 0.1 to 0.4%[1]. General practitioners, Pediatricians, Paediatric Cardiologists and Obstetricians should be aware that the condition in difficult to diagnose due to paucity of symptoms and can present in the fetus, in infants and in children. Management of this entity has changed over the last decade and the purpose of this entity has changed over the last decade and the purpose of this presentation is to review the changing trends in therapy.

PSVT is a clinical diagnosis for a number of dysrhythmias that appear identical on the electrocardiogram (ECG) but have different underlying pathogenetic mechanisms. The different mechanisms appear to have an age dependent distribution and include firstly PSVT with accessory atrioventricular (AV) connection (which is common in the young), secondly primary atrial tachycardia and thirdly, AV nodal reentry[2]. Fifty per cent of the patients have an idiopathic etiology, 20% have metabolic disturbances (fever, respiratory infections caused by respiratory syncytial virus, sympathomimetic use, drug intoxication, fluid and electrolyte disturbance, acid-base imbalance), 20% have structural heart disease (Ebstein's anomaly, congenitally corrected transposition of great arteries, tricuspid atresia, intraatrial surgical baffles) and 10% involve Wolf Parkinson White (WPW) syndrome[3,1].

The clinical findings depend upon the duration of the arrhythmia, age of the patient and the presence of heart defect, so that a child may be asymptomatic or desperately sick with cardiogenic shock or severe congestive heart failure[12]. A 12 lead ECG is required for the diagnosis. Typically the ECG findings include tachycardia of 220-250 beats/min, narrow QRS complexes, variable P wave morphology and nonspecific ST-T changes (Fig. 1).

Management of acute PSVT

Management is dependant upon the severity of hemodynamic decompensation (Figure 2). When associated with shock, unconsciousness or severe congestive failure, immediate termination of the arrhythmia is necessary. PSVT in small infants is also an emergency because the exact duration of the tachyarrhythmia is unknown. Synchronized Direct Current (DC) cardioversion is done at an energy dose of 0.25 to 2.0 J/Kg, which can be doubled if necessary upto a total maximum dose of 10 J/Kg,[12].

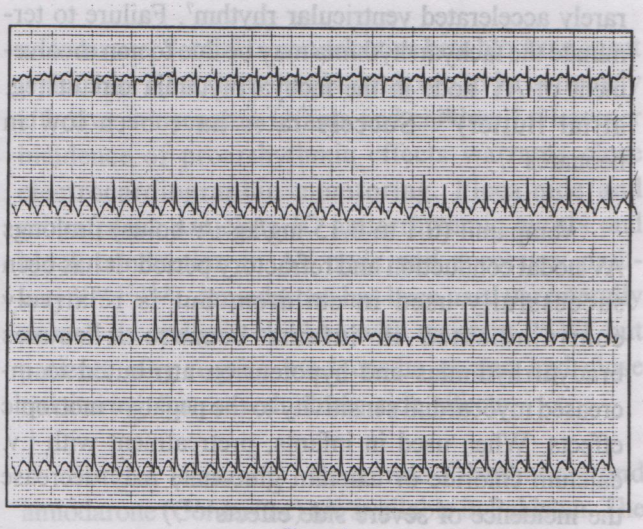

Figure 1.
Electrocardiogram of paediatric acute PSVT

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If the patient is stable, nonpharmacological conversion may be tried. Vagal stimulation often can interrupt reentrant rhythm by slowing conduction in the atria and AV node[4]. In infants, a useful vagal maneuver is the "diving reflex" which is elicited by placing an ice cool cloth or a glove filled with ice slurry over the forehead and nose for 30 seconds. This procedure is successful in 33-62% infants[5,6]. In older children, eliciting a gag reflex or having them stand on their head with support or a Valsalva maneuver can stimulate reflex vagal activity by raising blood pressure in the carotid bulb. Carotid massage and ocular pressure are potentially dangerous and therefore contraindicated.

Because vagal maneuvers frequently fail, pharmacotherapy is the mainstay of treatment. Adenosine is the drug of choice for all types of PSVT with a success rate of 80-100%[7,3]. It is an ultra short acting endogenous purine nucleoside, having a half life of 15 secs[3,8]. It slows AV nodal conduction possibly by parasympathetic stimulation, or stimulation of purinergic receptors or via some other mechanism. It restores sinus rhythm quickly and safely in WPW and other PSVTs and also in wide complex tachycardiacs[5,9]. It is injected as a rapid intravenous (i.v.) bolus of 0.05-0.35 mg/kg, preferably in the central circulation, followed by a saline flush. In emergency situations with no i.v. access intraosseous adenosine given rapidly as 1 mg/kg bolus is equally effective. Adenosine interacts with theophylline and dipyridamole, the former antagonizing and the later potentiating the effects of adenosine. Adverse effects are rare and include flushing, headaches, bronchospasm, apnoea, nausea and rarely accelerated ventricular rhythm[7]. Failure to terminate the tachyarrhythmia may be due to non involvement of AV node in the tachyarrhythmia circuit (eg. atrial flutter)[10]. Success rates have been low in neonates[11].

Verapamil (0.1 to 0.15 mg/kg i/v bolus) prolongs AV nodal conduction and refractory period. It has been successfully used in children, but is relatively contraindicated in infants because it causes bradyarrhythmias and hypotension attributed to increased myocardial sensitivity to the negative inotropic effects. When used in infants, pretreatment with i.v. calcium chloride or calcium gluconate may decrease the incidence of severe side effects[3,12].

Historically, rapid digitalization (20-50 ug/Kg given i.v.) had been advocated as the first therapy of choice, terminating PSVT in 40-50% of patients in half to two hours. The problem of slow onset of therapeutic effect can be overcome in some instances by repeating vagal maneuver after digitalization[7]. It is contraindicated in WPW syndrome, but has a definite place in post conversion prophylaxis.

Overdrive atrial (endocardial or epicardial) or transesophageal pacing at a rate faster than the arrhythmia may capture the atrium and interrupt the reentrant circuit. It can be used in stable patients unresponsive to pharmacotherapy as well as in unstable patients who do not respond to DC cardioversion. Transcutaneous pacing is not routinely used in children[3,7].

Long Term Management

It depends upon the age, natural history, underlying heart defect and the risks and benefits of each treatment option. The two major approaches for children are pharmacotherapy and radiofrequency catheter ablation.

A "natural history" data of paediatric PSVT is not available because almost all patients are treated. But known facts are that infant with PSVT have structurally normal heart and that PSVT does not recur in 40-70% of them when medications are stopped after 1-2 years. In contrast, children with onset of PSVT after 5 years of age have a 78% incidence of continued episodes. Thus infants are more likely to outgrow their PSVT[7]. No difference has been found in the recurrence rate in patients with congenital heart defect. Non pharmacological treatment is infrequently recommended and includes educating the patient or the family about vagal maneuvers. Franklin et al[13]in their retrospective review of 65 infants observed that PSVT did not recur in 20 (74%) out of 27 infants who did not receive prophylactic treatment, success rate being the same as with digoxin. In another series, Weindling et al[14]did not find recurrences in all the six infants who were not treated whereas 30% of children on digoxin or propranolol had recurrences. Placebo controlled multicenter studies would throw more light on the issue.

Table I

COMMON ANTIARRHYTHMIA DRUGS USED FOR LONG-TERM MANAGEMENT OF CHILDREN WITH PSVT

Drug	Oral dose	Side Effects
Digoxin	Loading dose: 40µg/kg/d	Nausea/Vomiting, visual disturbances, bradycardia, AV Block, junctional tachycardia, ventricular tachycardia, ventricular fibrillation, possible accelerated AV conduction of accessory pathway (contraindicated in WPW Syndrom)
Verapamil	1-3mg/kg/dose q8hr.	Hypotension, constipation, dizziness, headache, rash, sinus and/or AV node dysfunction, possible accelerated AV conduction of accessory pathway (contraindicated in WPW Syndrome)
Propranolol	1-3mg/kg/dose q6-8hr.	Decreased myocardial contractility, systemic hypotension, symptoms of CHF, bradycardia, fatigue, emotional disturbances, nightmares
Procainamide	1.5-3. 5mg/dose q4hr.	Hypotension, lupus, rash, anorexia, nausea, vomiting, dizziness, depression
Quinidine	7.5-10.0mg/kg/dose q6hr. (q8-12hr. if long acting form used)	Prolonged QT interval, torsade de pointes, nausea, vomiting, diarrhoea, rash, headache
Sotalol	1-2mg/kg/dose q12hr.	Sinus bradycardia, QT prolongation, ventricular extrasystoles, AV block, hypotension or hypertension
Flecainide	1.5-3mg/kg/dose q12hr. or 50-100 mg/m^2/dose q 12hr.	AV block, bradycardia, bundle-branch block, dizzinesss, visual disturbances, nausea, fatigue, wide-QRS tachycardia, ventricular tachycardia
Propafenone	2-3mg/kg/dose q6-8 hr., max 20mg/kg/day	Dizziness, nausea, constipation, heart failure, proarrhythmia, 1°AV block, Hepatotoxicity, agranulocytosis, ANA positive
Amiodarone	Loading dose: 10-20 mg/kg/day for 5-7 days	Bradycardia, AV Block, exacerbation of arrhythmia, long QT/torsades de pointes, hypothyroidism, abdominal pain, pulmonary fibrosis, photosensitivity.

MA/aa/PAROXYS

Pharmacotherapy: (Table I)

Digoxin has been the drug of first choice for PSVT prophylaxis for years. On long term basis drug therapy can be continued for one to two years and thereafter if recurrences occur, used either as continuous or "pulsed" form of therapy, the latter involving treatment of acute episode only[7]. However, there is no consensus on the form of medical therapy in 40-60% infants who have recurrences while receiving digoxin[15]. Previously therapeutic options (2nd line of drugs) included verapamil, propranolol, procainamide and quinidine.

Newer drugs for paediatric use are now available and include flecainide, sotalol, propafenone and amiodarone. Sotalol, a non selective beta blocking agent with Class III activity has been shown to be extremely effective in neonates, infants and children[18]. However incidence of proarrhythmic effects warrants close electrocardiographic monitoring. Flecainide and propafenone are class IC antiarrhythmic agents and block conduction in AV node and accessary pathway.[15,16,17,19,20]. Amiodarone has been used parenterally as well as orally in children with resistant PSVT but patients may require bradycardia pacing and close monitoring for proarrhythmic side effects.

Sotalol (Sotacor), flecainide (Tambacor) and amiodarone (Cordarone) are available in Pakistan. The experience with the newer drugs is limited and their

serious side effects makes their use restricted to resistant and life threatening PSVT.

Radiofrequency catheter ablation is an alternative non pharmacological treatment for drug refractory tachyarrhythmias. Drug refractoriness is defined as frequent recurrence after a trial of two or three drugs[22,23]. Experience with radiofrequency ablation in infants is limited. However, it has a great promise for older children with WPW syndrome and other PSVT and hopefully would be soon available in Pakistan.

Management of fetal tachyarrhythmia:

PSVT in inutero causes fetal hemodynamic compromise, non immune hydrops and fetal death. Fetal echocardiography is extremely useful in the diagnosis and differential diagnosis of fetal tachycardia and in the evaluation of response to drug therapy. Intravenous digoxin is given to the mother because oral digoxin has no role in acute management, but can be used for prophylaxis. Fetal digoxin levels are 60-100% of maternal serum levels[5]. Other alternatives for chronic management include verapamil, flecainide or amiodarone, alone or in combination with digoxin. Experience with the 2nd line of drugs during pregnancy is limited[17]. The mother and the fetus should be closely monitored by a perinatologist, neonatologist and paediatric cardiologist.

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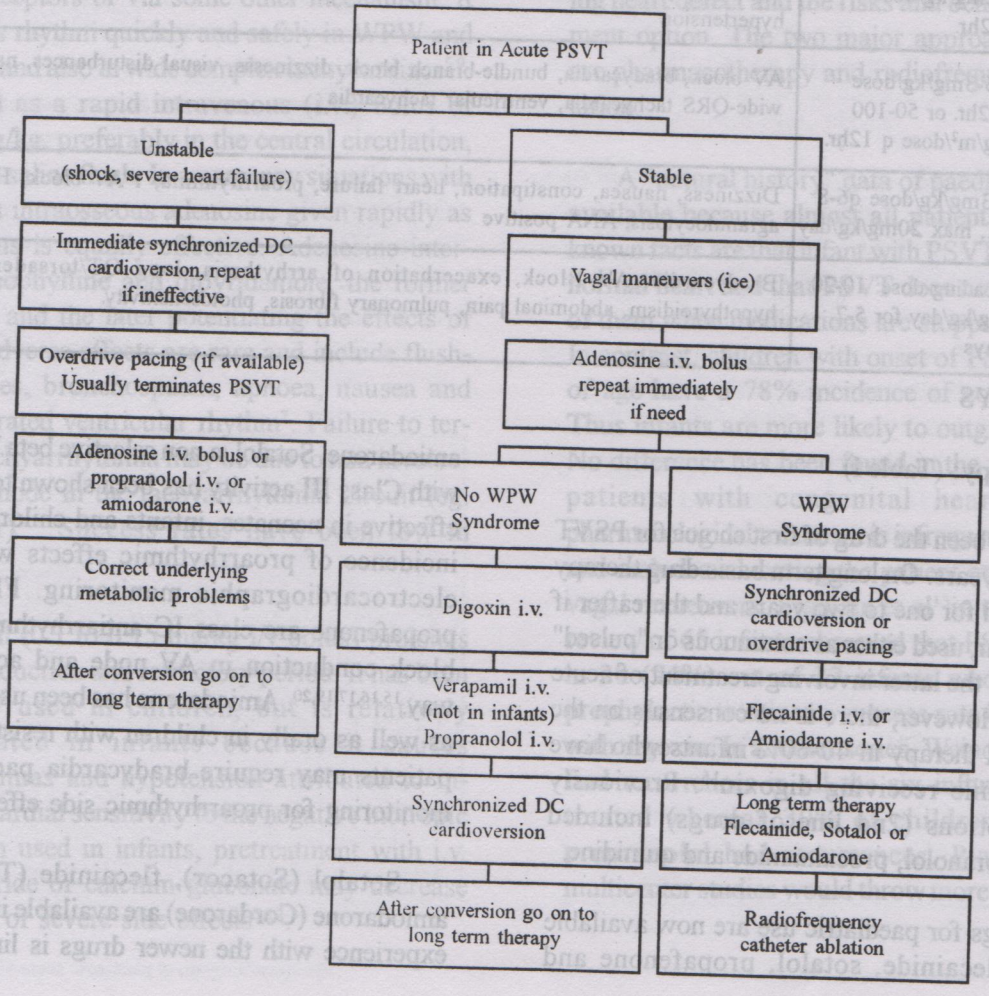
Figure 2.

Algorithm for therapeutic approach to acute Paediatric PSVT



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