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Supraventricular tachycardia in children: a report of three cases, diagnosis and current management

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تسرع القلب الفوق بطيني في الأطفال: التشخيص وأساليب العلاج في تقرير لثلاث حالات

ب. فينوجوبالان ، أ. شكيل ، ع. العامري ، س. جايا

الملخص: في هذا التقرير نستعرض ثلاث حالات لأطفال عمانيين مصابين بتسرع القلب الفوق بطيني متطرقين إلى التشخيص وأساليب العلاج. الفحص السريري وتخطيط القلب يساعدان في تشخيص هذا النوع الشائع من اضطراب نبضات القلب. في الحالات الطارئة يعالج المصاب بعقار " الأدينوسين " ولكن العلاج على المدى الطويل مازال موضوعاً خاضعاً للنقاش.

ABSTRACT: The article presents three Omani children with supraventricular tachycardia and discusses the diagnosis and management. Clinical features along with ECG help diagnosis of this common paediatric arrhythmia. Acute management has been facilitated with the introduction of adenosine. However, longterm management continues to be a topic for debate.

KEY WORDS: tachycardia, supraventricular, pathophysiology, case report, diagnosis, drug therapy, Oman, child

Supraventricular tachycardia (SVT) is the most common cardiac arrhythmia in children requiring therapy. This arrhythmia is usually the manifestation of an accessory conduction pathway that allows the atrioventricular impulses to re-enter the normal pathway, thus completing a circuit and stimulating atrium and ventricle at a fast rate. Infants and young children generally present with poor feeding and tachypnea, while palpitation and chest discomfort are prominent symptoms in older children. Electrocardiogram (ECG) shows a narrow complex tachycardia at a rate >220 per minute, and together with the clinical picture, helps in making a firm diagnosis in the majority of patients. Recognition is made difficult by the non-specific symptoms and the often self-limiting nature of the disorder and long-term management continues to be a topic for debate.[1]

However, in recent years, there have been new insights into the natural history of and the mechanisms responsible for supraventricular tachycardia in infants and children. With advances in antiarrhythmic therapy, there are now many therapeutic options. In this article we present three recently encountered cases of SVT from Oman and discuss the diagnosis, as well as a framework that may help to choose the appropriate treatment for the infant or child with SVT.

CASE 1

A 5-year-old Omani girl was admitted with fever and cough of 2 days. She was stable, pulse rate was >200/minute; the rest of cardiovascular system was normal. Chest examination showed evidence of pneumonia in the right lower lobe. ECG revealed narrow complex tachycardia at 230/min thus confirming SVT (Figure 1a), and there were abnormal P waves following the QRS complexes. Since carotid sinus massage, Valsalva maneuver and application of ice packs to the face failed to change the heart rate, intravenous bolus of adenosine 0.1 mg/kg was given followed by a push of 10-ml normal saline. Rhythm reverted to sinus in a few seconds and a simultaneously running ECG documented the change. A 12 lead ECG at that time failed to show any pre-excitation pathway. The child was started on digoxin, and antibiotics were commenced for pneumonia. Thyroid function tests yielded normal results. The child was discharged on maintenance digoxin and advised follow up. She discontinued digoxin and did not come for

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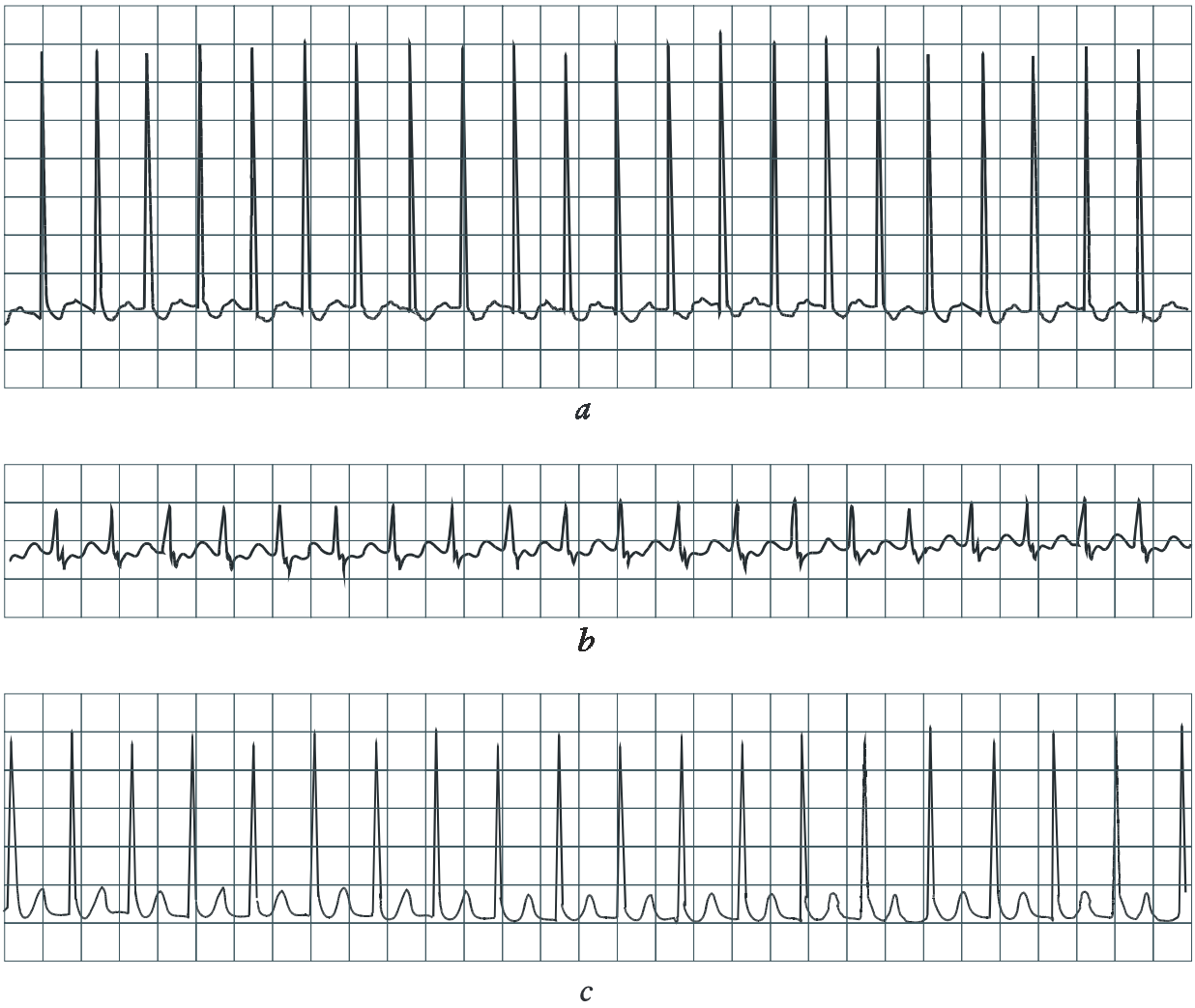

FIGURE 1. *ECG (lead II) of cases 1 (figure 1 a), 2 (figure 1 b) and 3 (figure 1 c) respectively, showing SVT*

follow up. Two months later she was readmitted with recurrence of SVT. This time also the rhythm reverted to sinus with adenosine and the parents were counselled on compliance to medication.

CASE 2

A 9-year-old Omani girl operated for congenital heart disease (atrial and ventricular septal defects) at the age of 7 years was admitted with chest pain and palpitation of 2 days' duration. The significant abnormality on examination was the pulse rate which was >200/min. She was not in heart failure. ECG confirmed SVT (Figure 1b). Abnormal P waves following QRS complexes were present. Vagal maneuvers failed and adenosine was given. Rhythm reverted to sinus. The 12-lead ECG was normal except for the incomplete right bundle branch block that was secondary to her cardiac surgery. The 24-hour ECG Holter record showed multiple episodes of self limiting SVT and the child was commenced on digoxin. Three months later she was readmitted with SVT and this time she required 3 doses of adenosine to arrest SVT. She was commenced on oral amiodarone and discharged home. She did not have any recurrence of symptoms or SVT episode on repeat Holter record 2 months later.

CASE 3

A 4½ year-old Omani girl was admitted with epigastric pain of one day duration. Her father, a hospital employee, had noted the fast heartbeat and brought the child for evaluation. The only abnormality on examination was the pulse rate which was >200/min. ECG confirmed SVT (Figure 1c) and she responded to adenosine. There was no evidence of preexcitation on 12 lead ECG. She was discharged home after counselling her parents on vagal maneuvers and was advised to report to hospital if symptoms recurred and could not be controlled.

DISCUSSION

SVT is a rapid, paroxysmal regular tachyarrhythmia that commonly involves the atrioventricular (AV) conduction system and an accessory AV pathway. This

is the most frequent sustained dysrhythmia in children. Infants and young children are more commonly affected; however a child may experience the first episode at a higher age also.[2] All our patients were older than 4 years at presentation.

MECHANISM OF ORIGIN

Ko[3] has demonstrated that in 90% of infants and about 50% of older children, an AV re-entrant pathway initiates SVT. This accessory pathway normally conducts impulses from atrium to ventricle, giving rise to the delta wave on the surface ECG, as in Wolff-Parkinson-White syndrome (WPW syndrome). When the accessory pathway is non-conducting and does not appear on surface ECG, it is referred to as concealed. SVT is triggered when for some reason the accessory pathway is refractory to the impulse it receives from the atrium, but later conducts the impulse in the reverse direction from ventricle to atrium, in turn initiating a second quick forward impulse that reenters the ventricle via atrioventricular node and normal conduction pathway (figure 2). This sets up a circular movement of electrical impulses from atrium to ventricle through the normal pathway and then in the reverse direction through the accessory pathway. The ventricular response is quick and heart rates of 300/ min are not uncommon. The initiating event may be an episode of premature supraventricular or ventricular beat, sinus pause or sinus tachycardia.[4] The second common mechanism of SVT in children involves an accessory pathway in or around the atrioventricular node (AV nodal reentry). Ectopic atrial tachycardia, atrial flutter and junctional tachycardia are the other varieties of SVT. Atrial flutter forms a significant proportion of foetal and 5–10% of neonatal tachycardias.

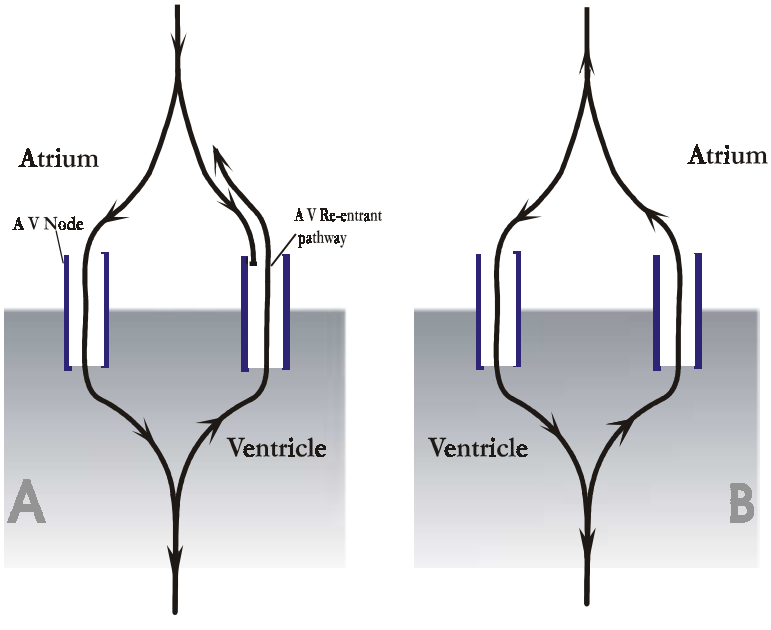

FIGURE 2. *Initiation of atrioventricular (AV) re-entrant tachycardia*

DIAGNOSIS

Regardless of mechanism of origin, SVT has a common mode of presentation. Symptoms are non-specific in infants and young children and include poor feeding, tachypnea, irritability and excessive crying.

TABLE 1

Distinguishing between supraventricular and sinus tachycardias

Criterion	Supraventricular Tachycardia	Sinus Tachycardia
Heart rate	>220/minute	<180/minute
Heart rate variability	No marked variation	Marked variation present
Surface ECG	P wave absent or abnormal if detected	P wave normal if detected
Identifiable cause	Not obvious	Obvious (eg. sepsis, fever)

Palpitation and chest discomfort are more often complained of in older children,[5] as in our case 2. Epigastric pain was the presenting complaint in one of our patients (case 3). Patients usually manifest acutely without any identifiable precipitating factor; however, one of our patients (case 1) had pneumonia at the time of admission. Diagnosis of SVT is based on history, physical examination and ECG. Tachycardia is obvious on examination, but is at times difficult to differentiate from sinus tachycardia secondary to septicaemia or pneumonia (table 1). The elevated heart rate and the narrow QRS complexes are most helpful in this respect. It is to be remembered that 10% of SVT cases have a wide QRS secondary to aberrant conduction. P waves are generally not visible on the surface ECG in SVT and if present are abnormal. These abnormal P waves can be better appreciated if a rhythm strip is recorded at 50 mm/second rather than the usual 25 mm/ second.

MANAGEMENT

TREATMENT OF ACUTE EPISODE

Sustained SVT in children requires intervention, because of the risk of haemodynamic deterioration. In infants it is often a medical

emergency, as they go rapidly into a state of shock. Vagal stimulation by carotid sinus massage, Valsalva maneuver, application of ice cubes to the face (diving reflex), or a combination of these is attempted initially in the stable child and these have a role even in the unstable patient while awaiting more definitive therapy.[6] Eyeball pressure should never be used because of the risk of injury to the eye.

Adenosine is the universal drug of choice in all patients with SVT.[7] Adenosine is an adenine nucleoside that acts by inducing transient block of atrioventricular node, thereby interrupting the re-entrant pathway. It has an extremely short half-life (10–15 seconds) and is effective in aborting an attack in most of the patients. The most important point to be remembered is the mode of administration. Adenosine should be administered as a bolus into a good venous access in the upper limb, using a three-way connection. The initial dose is 0.1 mg/kg and repeat doses 0.2 mg/kg with a maximum of 0.3 mg/kg. Adenosine loaded syringe and a second syringe with 3–5 ml normal saline are both connected to the three-way. The physician administers the adenosine as a bolus push and just as he completes it, an assistant pushes in the saline, after changing the direction of the three-way. A running ECG rhythm strip monitors the effect of adenosine reaching the heart. There is a period of cardiac asystole lasting 5–15 seconds followed by return of sinus rhythm (figure 3). However episodes of junctional and ventricular complexes may be seen during the period of asystole, as was evident in all three of our patients. There is also risk of immediate recurrence of SVT. Usual side effects are confined to autonomic disturbances like a feeling of impending doom, excessive salivation, abdominal pain, vomiting, flushing and headache (10–25%). Occasionally adenosine can precipitate bronchospasm in a predisposed individual.[8] More recently, major side effects of the drug have also been reported, such as apnea, prolonged asystole, accelerated ventricular rhythm, atrial fibrillation and wide complex tachycardia.[9] Therefore resuscitation equipment should be kept ready before administering adenosine. Adenosine is less effective in patients receiving aminophylline.

In critically ill patients intravenous access is not easily obtained and direct current electrical cardioversion has to be resorted to. The recommended dose of energy is 0.5 J/kg to 2 J/kg. Paediatric paddles are used for infants; children >10kg require adult-size paddles.[10] If condition permits, the child should be sedated and paralysed necessitating insertion of an intravenous line. This line could as well be used for adenosine injection. Thus it is clear that the option of using adenosine exists for all patients except for the extremely sick who are treated by immediate electrical cardioversion without sedation and paralysis.

IMMEDIATE STEPS AFTER CONVERSION TO SINUS RHYTHM

The child requires continued monitoring to identify and treat recurrences. A 12 lead ECG is taken to look for evidence of pre-excitation (WPW Syndrome) and good intravenous access maintained. Recurrences are treated in the same manner with adenosine using increasing doses and taking care to optimise the mode of administration. Even if vagal manoeuvres failed initially, at times they may be effective during recurrence. Frequent recurrences may necessitate therapy with digoxin (in the absence of WPW syndrome) or propranolol (in the presence of WPW syndrome). Though intravenous verapamil is another option, it is not recommended in infants due to the risk of hypotension and shock.[11] All these drugs act by producing AV block. They have longer lasting effects than adenosine and repeat doses can be administered orally. However side effects are more common and patients on these medications require more intensive monitoring. Resistant or frequently recurring SVT is one of the few indications for use of parenteral digoxin. The loading dose is 30 mcg/kg and is given in three divided doses as infusion over 20 minutes each at an interval of 8 hours. The initial dose is usually half the total dose (15 mcg/kg) and the subsequent doses one fourth (7.5 mcg/kg). ECG and serum potassium should be monitored during therapy. The maintenance dose is

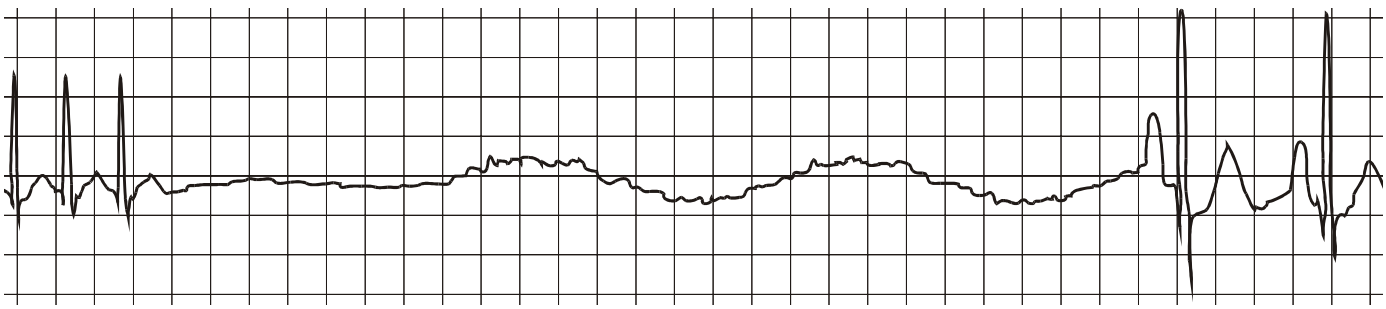

FIGURE 3. *ECG showing the typical response to intravenous bolus of adenosine in patients with supraventricular tachycardia*

10 mcg/kg daily in one or two divided doses administered orally. In selected patients when all other measures fail, there is a role for transesophageal atrial pacing to terminate the tachyarrhythmia.

LONG TERM MANAGEMENT

Long-term therapy with antiarrhythmic medication is so widely used in children with SVT that it is difficult to get data on the pure natural history. Generally infants are more likely to 'outgrow' their SVT. Lundburg[12] showed that up to 70% of infants with SVT do not relapse when treatment was discontinued at the age of one year, while Perry and Garson[13] reported a recurrence rate of 78% in children aged >5 years at the time of first episode. The presence or absence of structural heart disease did not influence the outcome.[5] However the presence of WPW syndrome on the surface ECG did indicate a chance for recurrent episodes and even sudden death in symptomatic patients.[13] The three available long-term treatment options are discussed below.

1. No-treatment option

Franklin[14] and Weidling[15] have shown good results with this option. The success rate with digoxin or propranolol or a combination was similar, indicating that they might have acted only as placebo. Controlled multicentre studies are needed to provide meaningful data on this issue. However the 'no-treatment option' is not recommended in infants and young children who have difficulty in communicating the problem to their carers and hence stand the risk of heart failure and shock. It may be suitable for older children who can recognize the problem early and attempt vagal manoeuvres to terminate the episode. Simple reassurance may be all that is required in some instances.

2. Long-term antiarrhythmic drug therapy

Digoxin is the traditional drug used to prevent SVT in children. Rarely deaths have been reported in patients on digoxin. In the case of patients with WPW syndrome propranolol is recommended. Flecainide is used for resistant cases. Patients with myocardial dysfunction need amiodarone. Therapy with any of the second line drugs (table 2) is to be planned in consultation with a Paediatric Cardiologist.

3. Radiofrequency (RF) catheter ablation

The major advantage of this mode of therapy is the prospect of a cure. Data on RF catheter ablation of accessory pathway in SVT has shown an initial success rate of 94%[16] and a freedom from recurrence of 85%, 77% and 66% at 1,2 and 3 years respectively after the procedure.[17] However it can be performed only in specialized centres and there is a major complication rate of 2.9% even in the best of centres.[18]

TABLE 2

Drugs useful for long-term management of SVT

Drug	Oral maintenance dose	Major side effects
Digoxin	5 microgram/kg q12hr	Nausea, vomiting, heart block, tachyarrhythmias (atrial and ventricular)
Propranolol	1–3 mg/kg q6–8hr	Heart failure, hypotension, bronchospasm, nightmares
Verapamil	1–3 mg/kg q8hr	Hypotension, skin rash, heart block, tachyarrhythmias (atrial and ventricular)
Flecainide	1.5–3 mg/kg q12hr*	Heart block, tachyarrhythmias (atrial and ventricular)
Amiodarone	5 mg/kg q24hr**	Heart block, tachyarrhythmias (atrial and ventricular), hypo/hyperthyroidism, corneal microdeposits, pulmonary fibrosis

* 1–2 mg/kg q6–8 hour in infants, due to shorter half-life in infancy

** loading dose 5 mg/kg q8 hr for first week, q12 hr for 2nd week

SUMMARY

We have presented three children with SVT and discussed the diagnosis and management options. Intravenous bolus of adenosine is the treatment of choice to terminate an acute episode that does not respond to vagal stimulation (Valsalva manoeuvre, application of ice packs to face and/or carotid sinus massage). Infants and young children need long-term drug therapy, while older children with single episode of SVT and absence of WPW syndrome may be followed up without medication ('no treatment' option). In resistant cases RF catheter ablation of the accessory pathway has to be considered.

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