## *Brief Reports*

# Neonatal Supraventricular Tachycardia

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Arrhythmia in neonatal period are not uncommon, but often there is a delay in their recognition and management. An entirely different set of criteria are necessary for recognition of arrhythmias in the neonatal period(l). Here, we discuss the clinical features, electrocardiographic (ECG) findings and response to treatment in 4 neonates with supraventricular tachycardia (SVT). In none of them, the referring physician suspected the diagnosis.

## Case Reports

The clinical profile, ECG findings and therapy of these 4 patients are summarized in *Table I.*

*Clinical Profile:* All infants were male and in the age range 14 to 27 days. Three were referred with a diagnosis of pneumonia and the fourth one for feed intolerance. None had any congenital cardiac anomaly; one patient had underlying myocarditis.

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The duration of symptoms before hospitalization ranged between 24 to 48 hours. At admission all the four babies had a heart rate of more than 200/min and signs of congestive cardiac failure (CCF), namely, tachypnea, hepatomegaly, and profuse sweating.

*Electrocardiographic Findings:* A complete 12 lead surface electrocardiogram was obtained in all the babies. The mean heart rate at admission was 271 beats per min (bpm). P waves were visible on the surface ECG in 2 of 4 patients *(Fig. 1A)* and in other two it was present in form of a pointed T-wave or a notch on the T-wave *(Fig. IB).* The QRS complex was of normal duration and configuration in all, and continued to be normal after restoration of normal sinus rhythm.

*P-QRS Relationship:* P-waves had normal relationship with QRS complex and atrioventricular conduction was 1:1 in all.

*Short Term Treatment:* At admission, all babies received synchronized direct current (DC) cardioversion because of compromised hemodynamic status. The energy dose ranged from 1-3 joules/kg. Two babies required a single DC-cardioversion where as other two required multiple doses. This was successful in terminating SVT within 30 min to 4 hours in all.

*Long Term Treatment:* After DC cardioversion, three patients were started on digoxin. Patient 3 received enalapril for CCF due to myocarditis and evidence of global hypokinesia on echocardiography.

*Recurrence and Outcome:* SVT recurred in patient 3 with underlying myocarditis on the third hospital day. He died of ventricular fibrillation on the fourth hospital day. Other 3 babies did not have any recurrence and were well on follow up

**TABLE I**–*Clinical Profile of 4 Patients with Supraventricular Tachycardia (SVT)*

| Parameters | 1 | 2 | 3 | 4 |
|---|---|---|---|---|
| Age at onset | 14 days | 18 days | 27 days | 18 days |
| Sex | Male | Male | Male | Male |
| Predisposing factor | None | None | Myocarditis | None |
| Family History of SVT | No | No | No | No |
| Presenting symptoms | Cough, resp. distress, cyanosis | Reggurgitation of feeds | Dry cough, lethargy, excessive cry | Fast breathing, poor feeding |
| Heart rate of admission | >220/min | >200/min | >220/min | >200/min |
| Congestive heart failure | + | + | + | + |
| ECG in SVT | | | | |
| QRS rate | 256/min | 300/min | 260/min | 270/min |
| P wave | Absent | Present | Absent | Present |
| Prolonged QRS | Absent (0.07 sec) | Absent (0.06 sec) | Absent (0.07 sec) | Absent (0.08 sec) |
| Chest X-ray (CTR) | Cardiomegaly (CTR 0.65) | Normal | Cardiomegaly (CTR 0.7) | Normal heart |
| Echocardiogram | Normal study | Normal study | Dilated LV, global hypokinesia, Myocarditis | Normal study |
| Treatment | | | | |
| Immediate | DC cardioversion 1J/kg NSR | DC cardioversion 1J/kg NSR | DC cardioversion 1J/kg→2J/kg | DC cardioversion 2J/kg→3J/kg |
| Long term | Oral digoxin | Oral digoxin | NSR 3J/Kg, Enalapril for global hypokinesia | NSR, Oral digoxin |
| Follow up | Asymptomatic up to 1 year | In NSR at 1, 3, 6 months | Recurrence of PSVT after discharge, died of V. fib. | In NSR at 1, 3 months; V. tach after discharge |

V. Tach - Ventricular tachycardia, NSR - Normal sinus rhythm, V. Fib - Ventricular fibrillation, CTR - Cardiothoracic ratio.

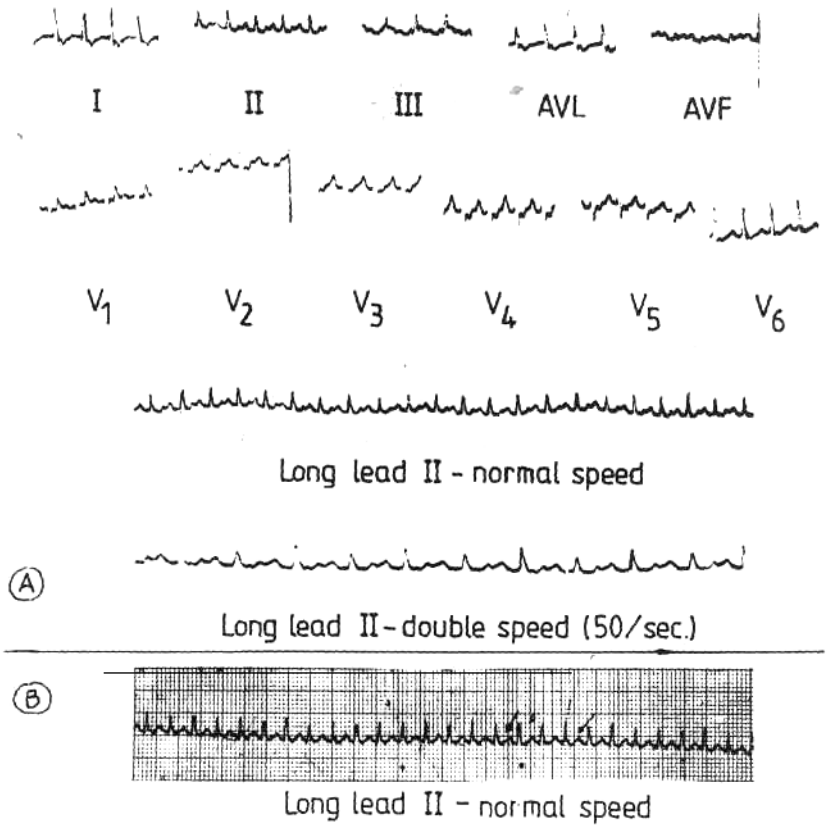


*Fig. 1. (A) ECG in patient 2 showing SVT with clearly visible P-wave, especially on long lead-II taken at a fast rate (50/sec). (B) ECG in long lead-II in patient 4 showing pointed and notched T-waves suggestive of P-waves superimposed on T-wave.*

visits after 1, 3 and 6 months.

## Discussion

Supraventricular tachycardia (SVT) is the most common tachyarrhythmia in infants, with or without underlying heart disease. An estimate in 1967 quoted an incidence of 1 in 25,000(2). It appeared by 1 month of age in 18% of all infants with SVT(3).

*SVT is defined* as tachycardia resulting from an abnormal rhythm originating from structures in heart proximal to the bifurcation of bundle of His and does not. have ECG morphology of atrial flutter SVT is the likely diagnosis in infants under 1 month if the ECG shows: *(i)* a heart rate above 200 bpm, *(ii)* origin of rhythm from the atrium, and *(iii)* a regular and narrow QRS-complex. A wide QRS (>0.07 sec) favors a diagnosis of ventricular tachycardia (4). P-waves are not always visible on the surface ECG during SVT. In such a situation, if the QRS complex is normal and the ventricular rate is abnormally rapid, the diagnosis is SVT(4). All our patients met these criteria. *The Clinical Presentation* in early infancy is nonspecific and often is the reason for delay in the diagnosis, as happened in our

cases. The usual early symptoms are poor feeding, lethargy, vomiting, irritability, tachypnea and dusky peripheries. Symptoms of congestive cardiac failure usually appear after 24 hours(4). Symptomatology, however, varies with the age at presentation, duration, heart rate, and associated structural heart disease(5).The majority of infants with SVT have an otherwise normal heart. The reported incidence of congenital heart disease as a predisposing factor is about 28%(3) and that of concealed accessory bypass tract between 20-50%(5,6). Ebsteins anomaly and L-transposition of great arteries are the commonest underlying congenital heart disease(3). Myocarditis, septicemia, hypoglycemia, high grade fever, neonatal hyperthyroidism and several drugs are other common predisposing factors(2).

*Management:* It is important to quickly terminate SVT in all infants especially in those with the first episode(7), as it is quite difficult to assess how close to decompensation a given baby may be. Short term treatment like vagal manoeuver, facial immersion in cold water and intravenous verapamil are not considered safe in neonatal period. At our center *direct current electrical cardioversion* is the treatment of choice for SVT presenting with CCF. We have found no morbidity related to the procedure. The initial energy dose used is 0.5 joule (w-sec)/kg which may be increased to 2 joules/kg(3,7). The dose is delivered with help of appropriate size paddles, in synchronization with the QRS complex, after sedation with ketamine (lmg/kg) or diazepam 0.2-0.3 mg/kg/IV (3,8).

In a hemodynamically stable infant intravenous (IV) *digitalis* may be used (10 Hg/kg slowly over 5 minutes stat and 5 mg/kg 6 and 12 hours after first dose). It has advantages of being a familiar drug devoid of negative inotropic effect. The reported success rate with IV digitalis in a series of 346 patients of which 18% were neonates, was about 90% in those who had Wolff-Parkinson-White (WPW) syndrome and about 66% in those without it(3,7). More recently, however digoxin has fallen from favor because of slow onset of action (up to 6 hours) and availability of safer drugs.

*Intravenous propranalol* in a dose of 0.1 mg/ kg may be used when digitals is not effective. It may however, cause severe bradycardia and reduce cardiac output. Hence it is advisable to administer one quarter of the total dose every 5 min with close observation of blood pressure and heart rate. If the blood pressure falls, an intravenous infusion of isoproterenol (0.05 to 0.5 mg/kg/ min) may be needed(3).

*Adenosine* is an endogenous purine metabolite which produces transient atrioventricular nodal block. The mean success rate with adenosine was 93% from over 600 reported cases in children of all age groups(9,10). It has the advantage of ultra-short onset of action (30 sec), and a short half-life. Adenosine is also a useful diagnostic tool in infants with broad or narrow QRS complex tachycardia, as it terminates arrhythmias dependent on the AV node only; it has no effect on ventricular tachycardias(ll). The safety profile of adenosine suggests that it should be the drug of first choice for acute termination of SVT in patients of all age groups with hemodynamic compromise or wide complex tachycardia(9-ll). However, the drug is not marketed in India, at present.

*Flecainida* a type IC antiarrhythamic agent, is a derivative of procainamide. It slows down atrial, AV nodal and infranodal conduction and has been used to control SVT caused by a variety of mechanisms(12). The effects of flecainide on neonatal Purkinje fibers and myocardial

tissue in experimental studies is similar to that noted in adults(13). A single oral dose (2.5 to 3.3 mg/kg) of flecainide was effective in terminating acute attack of SVT in 25 children(14). Maternal administration of flecainide also has been reported to be effective in the treatment of fetal tachycardia(13). There is no report of its use in neonates.

*The long term management* of infants with SVT depends on whether the infant has an underlying WPW syndrome. Oral therapy with digoxin should be started and continued for the first year of life in infants without WPW syndrome(8). After one year of age, with better skills of communication they are less likely to become hemodynamically compromised should they develop further episodes of SVT. In infants with WPW syndrome, digoxin should be avoided since there are occasional reports of sudden deaths in such patients(3). Flecainide in a dose of 100 mg/$m^2$/day has been reported to be a safe alternative for long term management of children with WPW syndrome(15).

After the diagnosis and successful treatment of the first episode, the prognosis for infants with AV re-entry tachycardia presenting in the first month of life is extremely good with the majority having no further recurrence(8). The cause of an apparent resolution of the condition is unclear. SVT in children with structural heart disease remains a difficult problem and treatment in this group remains far from satisfactory.

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