



A CASE REPORT OF SUPRAVENTRICULAR TACHYCARDIA

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ABSTRACT

Background: Supraventricular tachycardia (SVT) is the most common cardiac arrhythmia in children requiring therapy. An accessory conduction pathway that permits the atrioventricular impulses to re-enter the regular pathway, completing a circuit and rapidly activating the atrium and ventricle, is typically the cause of this arrhythmia. Diagnosis is frequently postponed in newborns and early children due to a lack of distinct clinical symptoms, which can cause serious issues for paediatric clinical practice. **Case Summary:** patient referred from sagbara CHC to NCHS with history of tachycardia, HR >240/min, respiratory distress and neonatal convulsion. Baby was delivered full term, NVD, DOB 03/06/2022, BW=3.7kg, CIAB, APGAR score at 1min= 10, 5min= 10, inj vit-k given at birth. Patient was admitted at sagbara CHC with convulsions on day of life 22, convulsion controlled with phenobarbitone. Later patient developed respiratory distress and tachycardia (HR>240/min) hence baby was transferred to NCHS. Baby was kept on O2 prong at 2L/min. ABGA, CBC, CRP, ECG, CXR done. ECG showed narrow complex tachyarrhythmia, P wave absent, fix R-R interval, systemic examination done. Patient was given adenosine 0.1mg/kg. adenosine mixed with 0.9% NaCl, 3ml given through rapid flush in antecubital IV line. After adenosine the heart rate reduced to 168/min. Cardiologist opinion was taken and it was normal heart study. Since no delta wave were present patient was started on digoxin and discharged home successfully. **Conclusion:** Treatment outcomes for SVT are favourable, and the condition is rarely life-threatening. Even while SVT frequently has a prenatal component, symptoms can appear at any time. The episodes appear to occur more frequently in the early years, then again in the 7–8 age range, and finally in the adolescent years. The episodes may or may not be connected to activity, and they can change in frequency and length over time.

KEYWORDS : Arrhythmia, Supraventricular Tachycardia, Pathophysiology, Prognosis, Pediatric.

INTRODUCTION:

Supraventricular tachycardia (SVT) is the most common cardiac arrhythmia in children requiring therapy. An accessory conduction pathway that permits the atrioventricular impulses to re-enter the regular pathway, completing a circuit and rapidly activating the atrium and ventricle, is typically the cause of this arrhythmia. While palpitations and chest discomfort are common symptoms in older children, poor feeding and tachypnea are typically the symptoms seen in infants and early children. The electrocardiogram (ECG), when combined with the clinical picture, aids in the identification of most patients by displaying a narrow complex tachycardia at a rate more than 220 beats per minute. The disorder's non-specific symptoms and frequently self-limiting nature make recognition challenging, and long-term management is still up for discussion.^[1] According to standard book definition the heart rate usually exceeds 180 beats/min in older children and adolescents, 220bpm in infants and younger children and 300 bpm in newborns.

Its incidence is 1 in 25000 and it represents 90% of all paediatric arrhythmias.^[2] It is distinguished by sudden beginning and ending. Prolonged episodes of PSVT can cause cardiac failure, hemodynamic instability, and other serious problems.^[3] Diagnosis is frequently postponed in newborns and early children due to a lack of distinct clinical symptoms, which can cause serious issues for paediatric clinical practice.

Early infancy and children, supraventricular tachycardia (SVT) is hazardous and can be deadly, however if it is identified. It has an outstanding prognosis if caught early and given the right care. Up until a few years ago, digoxin or cardioversion was typically advised as the first line of treatment. Nevertheless, digoxin was frequently unsuccessful, and cardioversion was difficult to perform if the tachycardia returned and required anaesthesia or sedation. There are now a lot of novel therapy options available, such as adenosine, verapamil, and flecanide. Some of these medications have been linked to risks in more recent times, most notably the use of verapamil in newborns.^[4] Therefore, here we presenting a case report of supraventricular tachycardia at our institute.

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convulsions on day of life 22, convulsion controlled with phenobarbitone. Later patient developed respiratory distress and tachycardia (HR>240/min) hence baby was transferred to NCHS. Baby was kept on O2 prong at 2L/min. ABGA, CBC, CRP, ECG, CXR done. ECG showed narrow complex tachyarrhythmia, P wave absent, fix R-R interval, on examination, HR >240/min, RR=60/min., BP 64/52mmHg, CRT<3sec, SpO2=90-92%, RS: AE=BS, Subcostal retractions present, CVS=S1S2 present, CNS tone normal, PA=soft, urine output=3ml/kg/hr.

Patient was given adenosine 0.1mg/kg. adenosine mixed with 0.9% NaCl, 3ml given through rapid flush in antecubital IV line. After adenosine the heart rate reduced to 168/min. Cardiologist opinion was taken and it was normal heart study. Since no delta wave were present patient was started on digoxin and discharged home successfully.

DISCUSSION:

Although SVT is the most common tachyarrhythmia in children, few reports of thrombosis result from SVT in children. The defining characteristic of SVT is its abrupt onset and termination, with varying durations. Clinical manifestations include anxiety, tachypnea, pallor, cyanosis, and poor appetite. An ECG examination is required for confirmation of SVT.^[5] SVT must be differentiated from sinus tachycardia, in sinus tachycardia P-wave is present and P-R interval is constant, whereas, in SVT there is no identifiable P-wave and QRS complex is narrow.^[6]

The clinical presentation of SVT is age and duration dependent. In infants with paroxysmal SVT, the heart rate is usually 220 to 320 beats/minute; in older children, it is 160 to 280 beats/minute. In infants, symptoms are usually nonspecific and include poor feeding, irritability, vomiting, cyanosis, and pallid spells. If the symptoms are unrecognized for hours to days, the infant can present with significant hemodynamic compromise or heart failure symptoms. It is rare for infants who have SVT for less than 24 hours to develop signs of congestive heart failure at the time of presentation; however, congestive heart failure is present in 19% of infants who have SVT for 24 to 36 hours and in 50% who have SVT for more than 48 hours. Approximately 20% of infants receive a diagnosis during routine office visits and during asymptomatic episodes. In verbal children with SVT, palpitations and fluttering in the chest are the usual presenting symptoms.^[7]

The treatment for SVT is divided into two categories: acute

termination and chronic prevention of recurrence. Acute conversion of the SVT begins with documentation of the arrhythmia either by a rhythm strip or preferably a 12-lead ECG. In life-threatening situations such as hypotension or severe congestive heart failure, immediate direct current (DC) cardioversion 0.5J/kg, if not effective 2J/kg is mandatory^[8]. If vagal maneuvers fail to terminate the SVT, adenosine is usually the first-line drug for acute pharmacological conversion. Adenosine at 0.1mg/kg (bolus) (maximum 6mg), second dose 0.2mg/kg (rapid bolus) (maximum 12mg)^[8]. With serial ECG monitoring during the process. Adenosine has a very short half-life, hence it is given through a large vein which is close to the heart (antecubital fossa) using a three-way tap, by rapid IV push followed immediately by a rapid push of 5ml normal saline through the other port. 25% of patients may experience some transient and minor side effects of bradycardia, premature ventricular ectopies, flushing, nausea, headache or respiratory disturbances. If the child does not revert back to sinus rhythm inspite of using the correct technique, one should review the diagnosis or consult a pediatric cardiologist. If adenosine fails to convert the SVT than amiodarone and procainamide is used. Other drugs such as verapamil which is a calcium channel blocker can be used in older children, however in children less than one year of age it has a tendency of causing hypotension and cardiac arrest^[6]. There are three management approaches for patients with SVT: "no treatment", continuous antiarrhythmic drug therapy, or radiofrequency catheter ablation. The decision for the individual patient is based on age, symptom severity, the frequency of attacks, the natural history of the specific disease, and the risks and benefits of each therapeutic approach.^[9]

Radiofrequency (RF) catheter ablation: RF catheter ablation has been utilized to treat SVT in children for a decade. The procedure is performed under conscious sedation or general anesthesia with electrode catheters inserted through femoral, subclavian and/or internal jugular veins for the electrophysiologic study of the SVT. With fluoroscopic guidance and intracardiac electrograms, an ablation catheter is manipulated to map the pathway location and RF energy is applied^[10,11]



Figure 1: Child with SVT



Figure 2: ECG of SVT [6]

CONCLUSION:

Treatment outcomes for SVT are favourable, and the condition is rarely life-threatening. In addition to causing irregular heartbeat, it can also result in lightheadedness, fainting, chest pain, and/or shortness of breath. Exercise may or may not be connected to the episodes. Even while SVT frequently has a prenatal component, symptoms can appear at any time. The episodes appear to occur more frequently in the early years, then again in the 7–8 age range, and finally in the adolescent years. The episodes may or may not be connected to activity, and they can change in frequency and length over time. If it occurs in infancy, it usually goes away by the time the child is 12 to 18 months old. There is then a 50% chance that the SVT will recur as an older child. If the SVT starts after the first year of life, it is likely to persist.

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