

Managing Newborn with Paroxysmal SupraVentricular Tachycardia

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Abstract

The incidence of paroxysmal supra ventricular tachycardia is approximately 1-3 cases per 1000 persons, with a prevalence of 0.2% [1]. Children may be asymptomatic or they may present with minor palpitations or more severe symptoms. Parents, mostly detect PSVT in children as tachycardias and heart failure signs, such as poor feeding, sweating and shortness of breath. The electrophysiological studies have helped to determine that the pathophysiology of SVT involves abnormalities in impulse formation and conduction pathways. The most common mechanism identified is reentry [2]. Infants require medical treatment because of the difficulty to recognize symptoms of tachycardia and a risk of heart failure leading to poor quality of life. Adenosine and beta blockers are drug of choice. Patients who present at <5 years old have a high likelihood of outgrowing their SVT and may not require chronic therapy.

Keywords: Paroxysmal Supraventricular Tachycardia; Heart Failure; Antiarrhythmic Drug.

Introduction

Paroxysmal supraventricular tachycardia (PSVT) is most frequent arrhythmia in newborns and infants. Prevalence [1] of SVT is approximately 2 per 1,000. Incidence of PSVT is about 36 per 100,000 people per year. A coordinated electrical signal is required for the heart muscle cells to contract in a coordinated way and generate a heartbeat. Normally, an electrical signal is generated in special pacemaker cells found in the sinoatrial (SA) node located in the right atrium or upper chamber of the heart. In PSVT, there is a "wiring" problem in the AV node and instead of having just one pathway for electricity to travel to the ventricle, there are two. This allows electricity to circle back and cause the atrium to beat more quickly than it should normally. Heart rate more than 220bpm in children below one year and higher than 180 bpm in children above one year is suspicious for PSVT. In 40% of cases, PSVT arises in first year of life [2] its incidence in the neonatal period has not been

estimated adequately (1 out 15,000-25,000 live births). A predisposing condition (congenital heart disease, medication, concomitant infection) is found in 15% of cases [3]. PSVT related tachycardia in newborn present as poor feeding, crying, irritability and sweating.

Case Report

Two month old single term, male, appropriate for date, non-consanguinity born in a Hindu family, at hospital through normal vaginal delivery to second gravida mother was admitted to our tertiary care facility. The baby cried immediately after birth. Initially the baby was admitted in a private hospital for first 8 days after birth. The neonate showed symptoms of rapid respiration, difficulty in feeding, cough and cold was intermittently present. No history of fever was present. Neonate heart rate was above 280/min. CXR was suggestive of cardiomegaly

with CT ratio 64%. ECHO showed LV dysfunction with LEF 20%.

The baby was administered Inj Dobutamine for cardiogenic shock, PSVT was aborted by Inj Adenosine and Amiodarone. Inj IVIG was administered suspecting myocarditis.

The baby was referred to our tertiary centre because of recurrent PSVT which was not subsiding. At the time of admission the baby was lethargic, had poor feeding. On examination heart rate 120bpm, respiratory rate 38/min. No radio-radial or radiofemoral delay was present. Investigation CRP - 0.32, LDH - 386, CPK - 85 (Range 40 - 226), Thyroid Profile T3 164.43 ng/dL (70-170), T4 13.5 mcg/dL (4.5-12.5), Vit D -19.43mg/ml. ECHO report suggested of mild LV dysfunction post myocarditis.

Treatment: Child received IVIG 2gm/kg over 2 days in view of suspicion of myocarditis. Developed cardiogenic shock for which Inj Dobutamine was administered. Child hemodynamically improved (EF improved from 20% to 40%) but continued to have irregularly irregular HR. Child was continued on propranolol and lasix. EF improved to 50%. During hospital stay child continued to have frequent atrial ectopic with intermittent episodes of SVT lasting 15 - 30 seconds. Holter study was done. Beta blocker doses were gradually increased with careful monitoring. The frequency of ectopic had decreased, and also the child did not develop SVT at least for 72 hrs before discharge.

Discussion

Paroxysmal supraventricular tachycardia (PSVT) is episodes of rapid heart rate that start in a part of the heart above the ventricles. According to their underlying electro-physiological mechanism, supraventricular tachycardia's are divided into 1) reentrant tachycardia and 2) automatic tachycardia [4].

Ectopic atrial tachycardia, which may originate at any point in the atria without involving in its mechanism the sinoatrial node. This is a well-organised tachycardia, whose normal sinus rhythm is replaced by high-frequency impulses (in the newborn up to 250-300/min) originating from a small area of the atrium. Atrial ectopic tachycardia is a relatively infrequent arrhythmia in newborns; it accounts for not more than 5-10% of all supraventricular tachycardias. In newborns PSVT is not recognised unless the episode continues for 12-24 hours causing decreased cardiac output. Baby

will show the signs of congestive heart failure such as vomiting, sweating, tachypnea and cold skin which may develop rapidly [5]. Other examinations like assessing intensity of first and second sound, presence of pathological murmurs, radial and femoral pulses. The first step in evaluation is to check the hemodynamic status.

A small proportion of children who have PSVT may present with shock. If the child is hypotensive or has poor capillary refill, one must start immediate measures to restore effective perfusion, including securing reliable intravenous (IV) access and supine positioning [6]. Adenosine is drug of choice, which causes a block at the level of the sinoatrial node and of the atrioventricular node. As it has a short half life, it must be administered in fast bolus followed by saline bolus. The initial recommended dose is 0.1mg/kg. If it's ineffective to subside it can be increased from 0.2mg/kg to 0.5mg/kg [77].

If any respiratory issues are present then adenosine should be administered only if good intravenous access is present. For infants born with a history of foetal PSVT, doctors choose to give maintenance antiarrhythmic therapy postnatal. Digoxin has long been the drug of choice in antiarrhythmic prophylaxis for PSVT. Success rates range between 42% and 75% [8]. Beta-blockers, in particular propranolol, are commonly used to prevent the recurrence of neonatal PSVT in cases where digoxin is contraindicated.

Conclusion

Most infants with ectopic atrial tachycardia at < 6 months of age will be free from atrial tachycardia after 12 months of antiarrhythmic therapy [9]. Parents of the children affected with PSVT should be educated to assess the pulse of the child and detect early signs of tachycardia like irritation, sweating, dyspnoea, chest pain, dizziness, fainting. Education regarding medicine dosage is required. Frequent follow up is necessary to maintain the dosage in therapeutic range. Management of PSVT should be individualized. Many children with paroxysmal supraventricular tachycardia do not require any therapy. The decision to proceed with treatment should be based on the frequency and severity of symptoms and on the effect of arrhythmia on the quality of life.

Contribution

Thomas Nisha has the prime responsibility of data acquisition and draft preparation and review of

literature. She is the first author for the paper. Thomas Nisha and Mathew Rejish did manuscript revision and editing.

Mathew Rejish will act as guarantor for the paper.

Compliance with ethical standards

Written informed permission was obtained from the parents before data collection. Since it is a clinical case report, no ethical approval was required for the article.

Conflict of Interest: None

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