

**An Unusual Case: Supraventricular Tachycardia Resistant to Adenosine with Comorbid Hypoxic-Ischemic Encephalopathy**

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**ABSTRACT**

This case report highlights the clinical course of a 33-week premature neonate (Twin 2) born to a non-consanguineous couple. The infant presented with various medical challenges, including supraventricular tachycardia (SVT), hypoxic-ischemic encephalopathy (HIE), and seizures. Despite intensive medical interventions, the SVT remained unresponsive to adenosine. This case emphasizes the need for a comprehensive diagnostic approach and the challenges faced in managing complex medical conditions in premature infants.

**Keywords:** Tachycardia, Newborn, Hyaline Membrane Disease, Hypoxic-Ischemic Encephalopathy.

**INTRODUCTION**

Tachycardia is characterized by a heart rate that surpasses the upper limit considered normal for the patient's age. In newborns, the most frequently encountered abnormal tachycardia is a narrow complex supraventricular tachycardia (SVT). SVT is described as any tachycardia necessitating the involvement of at least one supraventricular structure situated above the division of the His bundle (HB) for its perpetuation. These structures encompass the atrial myocardium, the atrioventricular node, the proximal HB, the coronary sinus, the pulmonary veins, the vena cavae, or abnormal atrioventricular connections distinct from the HB, such as bypass tracts. It is noteworthy that SVTs

are typically instigated by two distinct mechanisms: reentry and automaticity, with the majority attributed to reentrant atrioventricular tachycardia [1-3].

## **CASE REPORT**

A spontaneously conceived 33-week-old second twin, born to a 28-year-old mother with a gravidity of 4, parity of 2, and 2 living children from a non-consanguineous marriage, was delivered via lower segment cesarean section (LSCS) due to prematurity and very low birth weight (VLBW). The infant experienced respiratory distress and cried immediately after birth, with APGAR scores of 5/10 at 1 minute and 7/10 at 5 minutes.

Following birth, the baby was intubated, received surfactant therapy due to the presence of grade 3 hyaline membrane disease, and was initiated on inotropic support and antibiotics, showing steady progress until the sixth day of life.

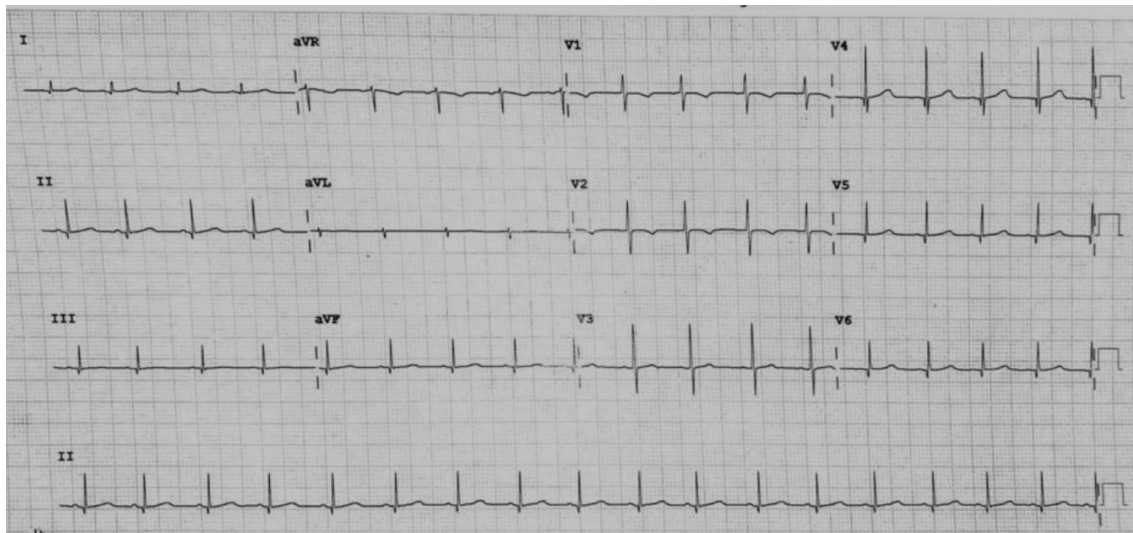
On the sixth day, the infant developed multiple episodes of seizures, necessitating treatment with intravenous phenobarbitone. Subsequently, intravenous levetiracetam and phenytoin were administered. On the seventh day, the patient's heart rate remained consistently elevated at approximately 200 beats per minute, unresponsive to three doses of intravenous adenosine (Figure 1). Thyroid function tests were conducted to rule out hyperthyroidism, and a sepsis screen yielded negative results. A 2D echocardiogram demonstrated normal findings, and the patient's temperature stabilized, allowing the discontinuation of inotropic support.

On the tenth day of life, the infant was started on oral propranolol, which was continued for 7 days and effectively resolved the tachycardia, as illustrated in Figure 2. On the fourteenth day, a plain MRI of the brain was conducted, revealing altered signal intensities exhibiting hyperintensity on T1/T2/FLAIR images, along with diffusion restriction and low ADC values observed in the bilateral gangliocapsular region, corticospinal tracts, midbrain, and pons.

Genetic testing was recommended to identify the cause of the tachycardia unresponsive to adenosine, but the patient chose to withdraw from treatment against medical advice. Twin 1, who also had grade 2 hyaline membrane disease, received surfactant therapy and did not exhibit similar issues. Subsequently, this infant was discharged from the Neonatal Intensive Care Unit (NICU) after the mother received instructions on infant care.



**Figure 1: No Effect of Adenosine on SVT (Above-Pre Adenosine, Below- Post Adenosine)**



**Figure 2: Normal sinus Rhythm after treatment with Propranolol**

**DISCUSSION**

Tachycardia in neonates can have various etiologies, including physiological factors such as anxiety, discomfort, or pain, as well as pathological causes. Among the pathological tachycardia observed in newborns, narrow complex SVT stands out as the most common [2]. While vagal stimulatory techniques are often effective in older children, their efficacy is notably limited in infants and neonates. Adenosine represents the preferred pharmacological intervention for the treatment of supraventricular tachycardia [4]. Adenosine exerts negative chronotropic, dromotropic, and inotropic effects, characterized by an extremely short duration of action (half-life of less than 10 seconds) and minimal impact on hemodynamics.

Adenosine proves effective in terminating nearly all reciprocating SVTs, wherein the atrioventricular (AV) node plays a role in the reentry circuit, regardless of whether the tachycardia is characterized by a narrow or wide complex regular rhythm. A 2014 investigation conducted by Gill BU and colleagues further reinforced adenosine's efficacy in terminating paroxysmal supraventricular tachycardia (PSVT) [5].

In a 2015 study by Andrea L. Barton et al., it was demonstrated that high-dose propranolol could be administered safely and with reasonable success for the treatment of SVT in infants [6]. Similarly, a 2020 study led by Eliana S. Nicastro and colleagues revealed that propranolol prevented recurrences in 70% of cases among a total of 107 patients [7].

An analogous case report hailing from Tamil Nadu described a situation in which one of a set of twins displayed SVT in a 23-year-old mother during the 25th week of gestation. The mother received oral digoxin treatment, and after 36 hours, the first twin developed pericardial effusion and cardiomegaly. In anticipation of impending fetal hydrops, oral sotalol was initiated and gradually increased. On the fourth day of admission, the first twin reverted to sinus rhythm,

while the second twin remained healthy. The patient was discharged on the seventh day of admission. Subsequent follow-up at two weeks revealed the well-being of both twins and the mother, with the resolution of pericardial effusion in the first twin [7].

## **CONCLUSION**

Typically, supraventricular tachycardia (SVTs) result from two distinct mechanisms, namely reentry and automaticity, with the majority being attributed to reentrant atrioventricular (AV) tachycardia. When vagal maneuvers prove ineffective, adenosine is regarded as the preferred pharmaceutical intervention. It is noteworthy that instances of supraventricular tachycardia that do not respond to adenosine therapy are infrequently encountered.

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