

A case report on supra ventricular tachycardia nonresponsive to adenosine with Hypoxic-ischemic encephalopathy

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Abstract

Introduction: SVT is defined as any tachycardia which requires participation of at least one supraventricular structure above the bifurcation of the His bundle (HB) for perpetuation, including the atrial myocardium, the atrioventricular node, the proximal HB, the coronary sinus, the pulmonary veins, the vena cavae, or abnormal atrioventricular connections other than the HB (i.e., bypass tracts)² Many infants tolerate SVT well. If the tachycardia is sustained for 6 to 12 hours, signs of CHF usually develop

Case report: A 34 Week twin 2 born spontaneously conceived out of a non consanguineous marriage to a 27 year G3P2L2 mother with no co morbidities via LSCS was admitted in NICU in view of prematurity and VLBW and respiratory distress Baby cried at birth APGAR at 1 min WAS 6/10 , 5 min 8/10 respectively Baby was intubated and surfactant given in view of grade 2 hyaline membrane disease and was started on inotropic support and antibiotics On day 7 of life patient had multiple episodes of seizures hence was Loaded with iv gardenal Baby was further loaded with iv levipil and epitoin On day 8 of life patient started having consistent heart rate of about 200bpm which did not respond to 3 doses of iv adenosine and thyroid function test were done to rule out hyperthyroidism and sepsis screen was done which turned out to be negative 2D echo was done which was normal On day 11 of life baby was started on Tab propranolol and continued for 6 days which resolved the tachycardia On day 14 MRI BRAIN plain was done which revealed Altered signal intensities appearing hyper intense on T1/T2/FLAIR images with diffusion restriction and low ADC values are seen in the bilateral gangliocapsular region, along corticospinal tracts, mid brain and pons.

Keywords: supra ventricular tachycardia adenosine Hypoxic-ischemic encephalopathy

Introduction

Tachycardia is defined as a heart rate beyond the upper limit of normal for the patient's age The most commonly presenting pathological tachycardia in the newborn is a narrow complex supraventricular

tachycardia¹.SVT is defined as any tachycardia which requires participation of at least one supraventricular structure above the bifurcation of the His bundle (HB) for perpetuation, including the atrial myocardium, the atrioventricular node, the proximal HB, the coronary sinus, the pulmonary veins, the vena cavae, or abnormal atrioventricular connections other than the HB (i.e., bypass tracts)²

.In general, SVTs are caused by two separate mechanisms reentry and automaticity of which majority are caused by reentrant AV tachycardia.

Case report

A 34 Week twin 2 born spontaneously conceived out of a non consanguineous marriage to a 27 year G3P2L2 mother with no co morbidities via LSCS was admitted in NICU in view of prematurity and VLBW and respiratory distress

Baby cried at birth APGAR at 1 min was 6/10 , 5 min 8/10 respectively

Baby was intubated and surfactant given in view of grade 2 hyaline membrane disease and was started on inotropic support and antibiotics and was doing good unit day 7

On day 7 of life patient had multiple episodes of seizures hence was Loaded with inj phenobarbitone Baby was further loaded with iv levatiracetam and phenytoin

On day 8 of life patient started having consistent heart rate of about 200bpm which did not respond to 3 doses of iv adenosine (figure1) and thyroid function test were done to rule out hyperthyroidism and sepsis screen was done which turned out to be negative 2D echo was normal patient had normal temperate was off inotropic support

Pre Adenosine therapy



Post adenosine therapy

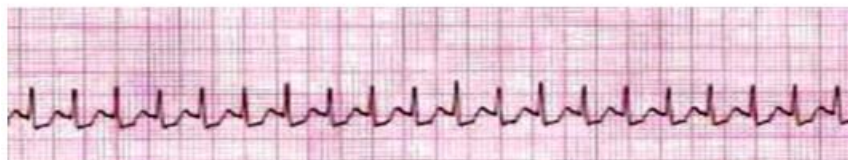


Figure 1 showing no effect of adenosine on the SVT

On day 11 of life baby was started on Tab propranolol and continued for 6 days which resolved the tachycardia as shown in figure 2

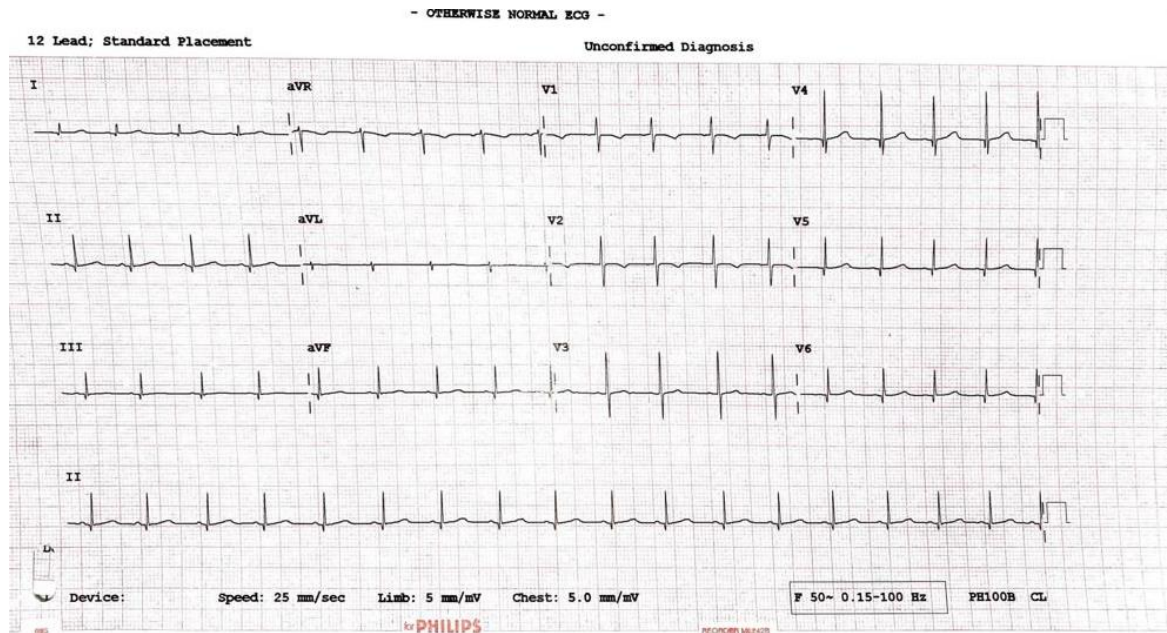


Figure 2 showing sinus rhythm after treatment with propranolol

On day 14 MRI BRAIN plain was done which revealed Altered signal intensities appearing hyper intense on T1/T2/FLAIR images with diffusion restriction and low ADC values are seen in the bilateral gangliocapsular region, along corticospinal tracts, mid brain and pons.

Patient was advised to get a genetic testing to find out the cause of tachycardia not responding to adenosine but have withdrawn from treatment against medical advice.

Twin 1 also had grade 2 Hyaline membrane disease was given surfactant and had no similar issues and was discharged from NICU after the the mother has been trained about baby care

Discussion

Tachycardia in neonate can be physiological due to causes like anxiety discomfort or pain and can be pathological

The most common pathological tachycardia in the newborn is a narrow complex supraventricular tachycardia¹ Vagal stimulatory maneuvers may be effective in older children but are rarely effective in infants and neonates Adenosine is considered the drug of choice for supraventricular tachycardia³. It has negative chronotropic, dromotropic, and inotropic actions with a very short duration of action (half-life <10 seconds) and minimal hemodynamic consequences.

Adenosine is effective for almost all reciprocating SVT (in which the AV node forms part of the reentry circuit) of both narrow- and wide-complex regular tachycardia.

In a 2014 study done by Gill BU et al adenosine is more effective for termination of PSVT⁴.

In a 2015 study by Andrea L Barton et al High-dose propranolol is safe and reasonably successful in the treatment of infant SVT⁵

In a 2020 study by Eliana S Nicastró et al Propranolol prevented recurrence in 70 % of cases in a total of 107 patients⁶

A similar case is reported from tamilnadu⁷ where one of the twins had SVT in a 23 year old mother at a gestation of 25 weeks where the mother was loaded with oral digoxin. After 36 h, twin 1 developed pericardial effusion and cardiomegaly. In view of impending fetal hydrops, oral sotalol was started and increased. Twin 1 reverted to sinus rhythm on day 4 of admission and twin 2 was healthy. The

patient was discharged on day 7 of admission. On follow-up after 2 weeks, both twins and mother were healthy, and pericardial effusion resolved in twin 1.

Conclusion In general, SVTs are caused by two separate mechanisms reentry and automaticity of which majority are caused by reentrant AV tachycardia. If the vagal maneuver is ineffective, adenosine is considered the drug of choice. Supraventricular tachycardia not responding to adenosine is rare occurrence

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