



REVIEW ARTICLE

Neonatal Supraventricular Tachycardia - A Review of Literature

Nargis Ara Begum¹, Suman Sharker²

¹Senior Consultant, Department of Neonatology, United Hospital Ltd. Dhaka, Bangladesh.

²Specialist, Department of Pediatrics and Neonatology United Hospital Ltd. Dhaka, Bangladesh.

Abstract

Supraventricular tachycardia (SVT) is the most common tachyarrhythmia requiring emergency cardiac care in newborn. SVT is defined as tachycardia resulting from an abnormal mechanism involving heart structures proximal to the bifurcation of the bundle of His. It requires participation of at least one supraventricular structures including atrial myocardium, atrioventricular node, proximal bundle of His, coronary sinus, pulmonary vein, vena cava and bypass tract. Atrioventricular reentrant tachycardia utilizing an atrioventricular by pass tract is the most common form of SVT presenting in the neonatal life. Precise diagnosis and risk stratification of SVT is needed before treatment is considered to reduce neonatal morbidity and mortality. SVT in neonatal life is very critical but if it is diagnosed early and treated appropriately it has an excellent prognosis.

Keywords: Newborn, supraventricular tachycardia.

Introduction

Supraventricular tachycardia (SVT) is the most challenging arrhythmia in newborn, requiring urgent medical attention. The incidence of SVT is estimated to be 1 in 200 to 250 for neonates [1, 2]. Most episodes of SVT (about 40% of all SVTs in pediatric age) occur during the 1st month of life, being their prevalence less in subsequent ages [3]. There is an inverse relationship between age of the first attack of SVT and the likelihood of recurrence. On the other hand, the younger the patient with SVT, the higher the incidence of arrhythmia induced heart failure [4]. Most premature supraventricular beats usually resolve over the first month of life, the remaining SVTs disappear during the ensuing six month. SVT is caused by one of three mechanisms such as reentry, increased automaticity or triggered activity. Most SVTs are caused by atrioventricular re-entry with the help of an accessory pathway. Majority of the SVT episodes occur in a structurally normal heart [5, 6]. Congenital heart diseases are reported in about 6.5% to 37% [7]. Most frequent cardiac morbidity of neonatal SVT is atrial septal defect, followed by Ebstein anomaly and L-transposition of great arteries, tricuspid atresia and double outlet right ventricle (DORV) [7, 8]. SVT in structural heart disease may be associated either with underlying abnormality or resulting from surgical intervention. The chronic hemodynamic stress of congenital heart disease (CHD) might create an electrophysiological and anatomic substrate highly favorable for reentrant tachycardia. Neonates who have SVT without associated cardiac diseases, adequate hemodynamic status is maintained for extended periods. The clinical presentation of SVT in the neonate is frequently subtle and may include

pallor, cyanosis, restlessness, feeding difficulty, tachypnea and grunting. If symptoms are unrecognized for several hours to days, newborn baby might present with significant hemodynamic compromise or heart failure. The diagnosis of SVT can be confirmed with ECG (electrocardiogram). The treatment of SVT is guided by baby's hemodynamic status. Vagal maneuvers and adenosine are treatment options in patient with hemodynamically stable SVT, although circulatory collapse with tachycardia needs synchronized direct-current cardioversion. Chronic therapy with digoxin, beta blockers, flecainide, sotalol and amiodarone has proved effective in controlling recurrent episodes of SVT [9-11]. Verapamil is considered an effective therapy for refractory SVT but require caution for its use in neonatal period [12]. Mechanisms of neonatal and fetal arrhythmias are similar and a proportion of neonatal SVT are a continuation of fetal SVT, extended to postnatal period [13]. The natural history and prevalence of neonatal SVT is in striking contrast to those seen in older children and adults, making it an important issue for the physician to be vigilant for diagnostic and therapeutic options. The prognosis of SVT is usually excellent but mortality rates of 2-6 % have been reported as a medical emergency. This review aims to focus updated spectrum of this very critical cardiac issue of newborn.

***Correspondence to:** Nargis Ara Begum, Senior Consultant, Department of Neonatology, United Hospital Ltd. Dhaka, Bangladesh. Email: nargisdr[at]yahoo[DOT]com

Accepted: July 20, 2020

Reviewed by: Stefan B, Germany

Pathophysiology:

The mechanisms underlying SVT are associated with reentry, automaticity and triggered activity. Reentrant and triggered forms are both induced and interrupted whereas automaticity is neither induced nor interrupted. The most common mechanism is reentry. Accessory pathway-related atrioventricular reentry tachycardia (AVRT), atrioventricular nodal reentrant tachycardia (AVNRT) and permanent junctional reciprocating tachycardia (PJRT) can be classified as reentry tachycardia. Another mechanism of tachycardia is increased automaticity which include ectopic atrial tachycardia (EAT) and junctional ectopic tachycardia (JET) [14, 15].

Atrioventricular Reentry Tachycardia (AVRT): The most common type of SVT in newborn, representing approximately 70% of SVT, results from a re-entry circuit between the atria and the ventricles and is called atrioventricular re-entry tachycardia (AVRT) (Figure 1). In this circuit, the AV node generally forms the antegrade pathway, and an accessory connection between the ventricle and the atria serves as the retrograde pathway for conduction of impulses. This is referred to as orthodromic AV reciprocating tachycardia. ECG shows a narrow QRS complex with a retrograde p wave immediately following QRS. In some cases, antidromic tachycardia is the other mechanism of AVRT. Antegrade AV conduction occurs over the accessory pathways and retrograde conduction occurs the AV node. In this case a wide QRS complex may be observed. WPW syndrome is SVT with evidence of pre-excitation. It is identified from a surface electrocardiogram (ECG) in sinus rhythm by a short P-R interval and a “delta wave.” WPW syndrome comprises 12% to 56% of AVRTs [16].

Atrioventricular Nodal Reentrant Tachycardia (AVNRT): The second type of SVT is AV nodal re-entry tachycardia (AVNRT) (Figure 1) which represents about 13% of SVTs. The tachycardia circuit also involves dual pathways, but both are situated within or near the AV node. Typical AVNRT conducts in an antegrade direction slowly down to the ventricles through one pathway and has a rapid retrograde conduction via the other pathway. It is, therefore, called “slow-fast” AVNRT.

On the ECG, this manifests as a QRS complex followed by a T wave, with the P wave often not visible (concealed in the QRS complex). Atypical AVNRT has a comparatively slower retrograde conduction, so the P wave is visible as an inverted complex after the QRS. This is called “fast-slow” AVNRT [17].

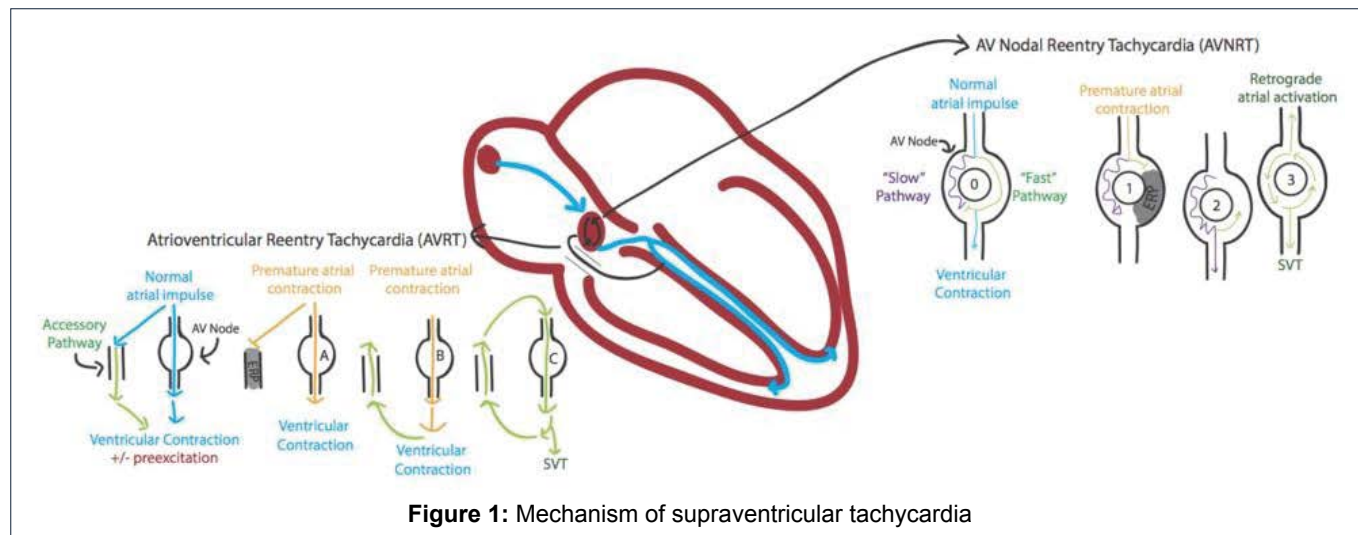
Atrial Ectopic Tachycardia (AET): The third most common type of SVT is atrial ectopic tachycardia, comprising approximately 14% of SVTs. This is due to ectopic atrial foci that display abnormal automaticity. These foci are reflected by an abnormal P-wave, narrow QRS and the unrecognized P wave because it is buried in previous T wave. EAT can lead to tachycardia-induced cardiomyopathy if not properly controlled [18, 19]. The type of heart disease most frequently associated with this SVT are congenital heart disease, myocarditis and cardiomyopathy [20].

Permanent form of Junctional Reciprocating Tachycardia (PJRT): PJRT is less common form of AVRT with a slow conducting retrograde accessory pathway. The majority of accessory pathways are located in the posteroseptal zone. The ECG hallmarks include an RP interval > PR with inverted P waves in leads II, III, a VF and V3-V6. PJRT is an infrequent form of reciprocating tachycardia usually drug refractory [21].

Junctional Atrial Tachycardia (JET): JET is characterized by single ectopic focus initiating at or near the AV node. ECG shows ratio of P wave to QRS complex <1.0 which indicates frequent AV dissociation (lead II). The mechanism of tachycardia is related to the enhanced automaticity of the AV junction. JET is usually observed in postoperative patients with CHD within the first several days after cardiopulmonary bypass. Congenital JET is highly associated with morbidity and mortality in neonates [22, 23].

Aetiology:

Fever, pain, anaemia, stress, hypoxia, hypovolemia, hypoglycemia, electrolyte disturbances (sodium, potassium, calcium, magnesium, phosphate) and acid-base disturbance (metabolic acidosis) are the aetiological factors. Infection,



familial, metabolic, toxic and inflammatory processes may be responsible although most cases remain idiopathic [24].

Important associations:

Congenital Heart Disease(CHD): Coexisting CHD are atrial septal defect, ventricular septal defect, patent ductus arteriosus, L-transposition of the great arteries, tricuspid atresia, double outlet right venticle (DORV), ebstein anomaly, dilated and hypertrophic cardiomyopathy, myocardial tumor. CHD not only affect the anatomical defect but also causes electrical changes that induce SVT. PPHN associated with SVT,might be the same aetiological factors involved for both the condition [25].

Gastroesophageal Reflux Disorder (GERD): Patients with SVT in the neonatal period frequently have GERD .The oesophagus is located immediately posterior to left atrium and the two structures share some innervations. The acid reflux in oesophagus could act as a trigger of SVT. Recent study reported GERD is associated with neonatal SVT in 50% cases and this association resulted in an increased difficulty in controlling tachycardia with pharmacologic treatment [26].

Infants of Diabetic Mother(IDM):The neonates born to mothers of gestational diabetes may develop diastolic dysfunction independent of ventricular hypertrophy, leads to neonatal SVT. The predominant variety in this case is atrial ectopic tachycardia [27].

Neonatal EnteroviralInfection: Newborn with tachycardia and a family history of febrile illness should be suspected to have enteroviral infection, highly contagious and potentially life- threatening infection. High catecholamine concentration in enteroviral sepsis may play role on in the pathogenesis of atrial tachycardia [28].

Catheterization In Newborn: Umbilical catheterization in newborn is very common procedure for performing exchange transfusion as well as maintenances of total parenteral nutrition. The development of SVT in catheterized newborn is not very common but poses serious complication. The primary cause is inappropriate position of the umbilical catheterwithin the heart leading to atrioventricular re-entry type of SVT, need immediate position change and abortive therapy [29].

Drugs: Use of some drugs like theophylline, salbutamol, digoxincauses SVT by their therapeutic overdoses [30].

Surgical Procedure: SVT is found in case of neonatal surgery, example; congenital diaphragmatic hernia surgery, probable explanation is heart manipulation and exacerbation of underlying PPHN. Post surgical SVT was found in case of cardiac operation like fontan’s operation, mustard and sennai operation [31].

Genetic and Metabolic: Autosomal dominant marfan’s syndrome , cardiac channelopathy related to mutation in genes encoding critical sodium ion channel (SCN5A) of heart termed Brugada syndrome, is a rare genetic disease associated with lethal rhythm abnormality [32]. Hyperthyroidism, inborn errors of metabolism (IEM) like congenital adrenal hyperplasia (CAH), storage disorders like mucopolysaccharidosis (sanfilipo), pompe’s disease, danon’s disease and mitochondrial disorders are rare evidence based association [33].

Clinical Feature:

The clinical presentation of SVT in the neonate is variable. Development of symptom depends on both the rate and duration of the arrhythmia. Heart rates are usually in the range of 240-300bpm. The presentation of SVT in the neonate is usually nonspecific and may include pallor, cyanosis, restlessness, irritability, increased sweating, feeding difficulty, tachypnea, diaphoresis and grunting. Some neonates do not become symptomatic, while others may develop signs of congestive heart failure and cardiogenic shock.If the symptoms are unrecognized for hours to days, the newborn baby can present with hemodynamic compromise or heart failure symptoms. It is rare for the newborn who have SVT for less than 24 hours to develop signs of heart failure at the time of presentation; however congestive heart failure is present in 19% cases who have SVT for 24-36 hours and in 50%, who have SVT for more than 48 hours [34, 35]. Duration of tachycardia and age of onset, especially younger age group seemed to be significant factor for production of heart failure in neonatal SVT.

Diagnosis:

ECG Features: A 12-lead ECG should be performed during tachycardia and after sinus rhythm is achieved, but ECG recording should not delay initiation of emergency treatment. ECG features (Figure 2) suggestive of neonatal SVT are:

- Rate: 240-300 beats/minute

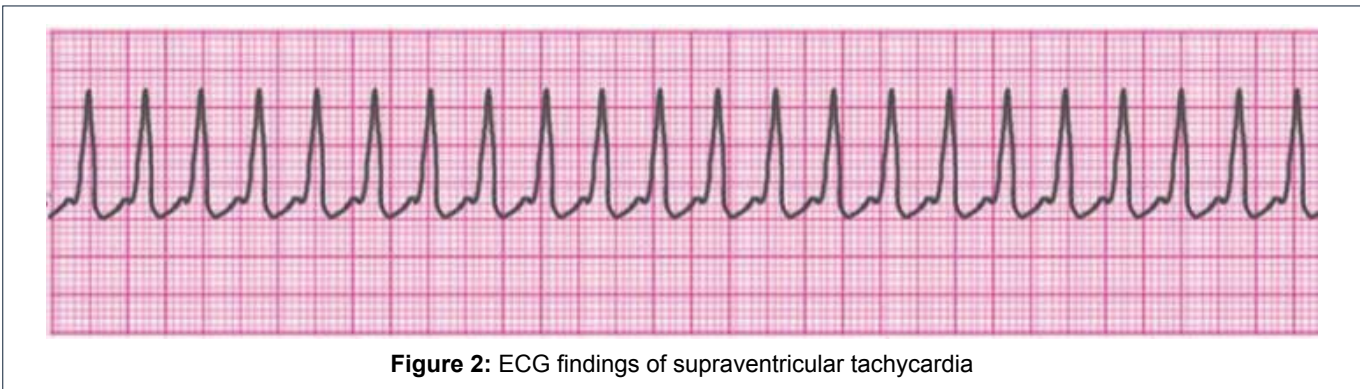


Figure 2: ECG findings of supraventricular tachycardia

- R-R interval: Absolute regularity, no beat to beat variation
- QRS complex: narrow, normal
- P wave: absent or abnormal (often inverted, seen just after QRS)

Echocardiography: echocardiogram should be performed to evaluate ventricular function and to identify the presence of structural heart disease.

Other supportive investigations: Bloods- CBC, CRP, Blood Culture (septic workup to exclude septicaemia), Creatinine, BUN (to assess renal status), electrolyte (Na, K, Ca, Mg, Po₄) to correct if any abnormality, blood sugar to detect hypoglycemia, blood gas (metabolic acidosis to correct if require), pro-BNP (to detect heart failure), serum lactate level, CPK-MB, troponin (to assess myocardial status), TSH and FT₄ (thyroid status).

Chestx-ray: to delineate cardiomegaly in case of heart failure and to find out any pulmonary congestion.

Treatment:

Acute/immediate treatment:

SVT management depends on many factors including age, the hemodynamic status of the patient, coexisting cardiac disease and onset of illness (acute or recurrence). When considering treatment for SVT, the most important diagnostic decision depends on hemodynamic stability. Stable SVT has many noninvasive treatment modalities. The hallmark of noninvasive treatment for stable SVT is increasing vagal

tone which can be done by vagal maneuvers. Unstable SVT is a medical emergency warranting immediate treatment due to hemodynamic compromise and synchronized cardioversion is sometime life saving. Options for acute management include use of the diving reflex, intravenous adenosine, transesophageal pacing (TEP) and cardioversion (Figure 3)

Vagal maneuvers: Vagal maneuvers are easy to perform, safe, often successful and cause less discomfort than chemical or electrical cardioversion. In neonates, vagal stimulation is commonly performed by eliciting the “diving reflex”, this is obtained by cooling the face with a bag (e.g. surgical glove) filled of iced water making temperature down to 10 degree centigrade for at least 30 seconds [36,37]. Second option is rectal stimulation of neonates by thermometer. The concept behind both the techniques is to elicit a strong vagal response. This parasympathetic stimulation causes interruption of a re-entrant SVT resulting in increased A-V nodal refractoriness and possible block of the reciprocating impulse; this may happen for AVRT, AVNRT, PJRT, thereby slowing heart rate. Cold water immersion to elicit diving reflex is effective in 90% of patients but SVT recurs in 69% of cases. Vagal maneuvers work best if can start early before adrenergic tone rises too high. If vagal maneuver is unsuccessful in terminating SVT, medical therapy is recommended.

Pharmacological therapy: There are several pharmacologic antiarrhythmic options but the mainstay of medical therapy of acute SVT is intravenous adenosine.

Adenosine:

Adenosine, when administered in the correct dose with the correct means is very effective with success rates of 85% to 100% [38]. Adenosine appears to be the most promising antiarrhythmic agent. It acts by slowing atrioventricular nodal conduction, thus disrupting a re- entry circuit. It has the advantage of ultra-short onset of action (30 seconds). It has also a shorthalf life of 10 to 15 seconds. So side effects which occur in one third of treated patients, are transient and rarely require intervention. Additionally adenosine is not negatively inotropic in this form and so may be given to a patient with low cardiac output without fear of hemodynamic worsening. Adenosine can be given in a starting dose of 0.1 mg/kg, increasing by 0.1mg/kg increment at 2 minutes interval till sinus rhythm is restored. Adenosine should be given very quickly and as proximally into a large vein. A three-way tap should be used so that the adenosine can be quickly flushed with normal saline. Nonetheless, because of high safety profile of adenosine, it will remain the drug of first choice for terminating SVT [39, 40]. Recurrence rate of 30% is its main disadvantage [41]. Its promising that it can be given in intraosseous route [42].

Verapamil:

Verapamil is the most effective anti-arrhythmic drug against refractory tachycardia in neonates with life threatening risk. Its advantage over digoxin is that it has a very rapid onset of action

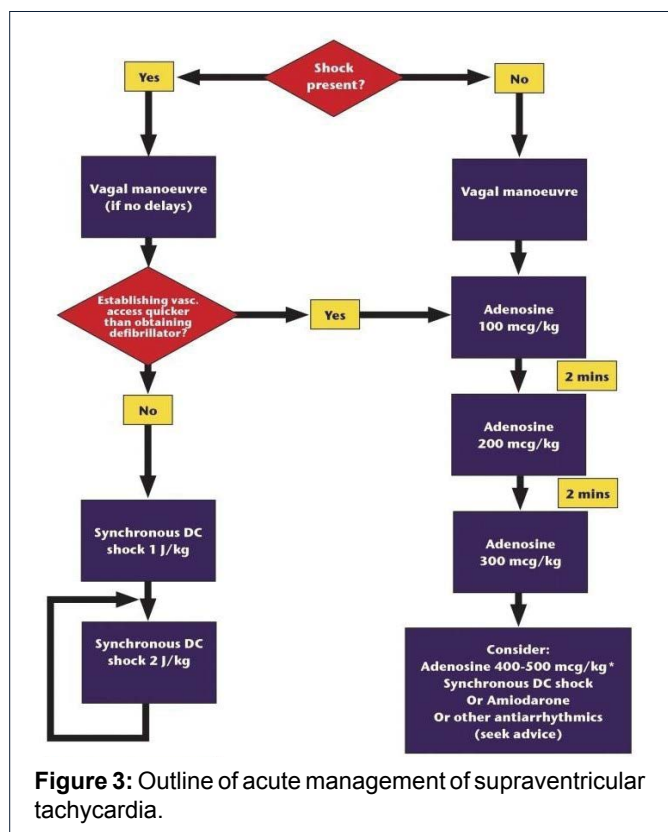


Figure 3: Outline of acute management of supraventricular tachycardia.

(3-5 minutes). The usual initial dose of verapamil is 0.075-0.15 mg/kg intravenously with a maximum dose of 5 mg. It can be repeated between 10 and 30 minutes following the initial dose. It has higher incidence of side effects, including AV block, extreme bradycardia, asystole, hypotension and congestive heart failure. The adverse cardiovascular effects can generally be overcome by treatment with isoproterenol and parenteral administration of calcium. Verapamil is contraindicated in patients with severe bradycardia, hypotension and collapse.

Amiodarone: This is the safest antiarrhythmic drug in the post-operative situation. Amiodarone is useful for atrial tachycardia which is not responsive to adenosine and resistant to reentrant circuits. Amiodarone slows the heart by depressing AV nodal conduction, modifying the automaticity and prolonging refractory period. The loading dose of amiodarone is 5 mg/kg over 20-60 minutes, may repeat twice up to a maximum of 15 mg/kg. Baseline thyroid function and liver enzymes need to be monitored during amiodarone administration.

Direct current cardioversion:

In the acute situation when vagal maneuvers fail and there is hemodynamic compromise, direct current cardioversion is the only choice. Appropriate resuscitative measures should be instituted in case of unstable SVT. Prerequisites are intravenous access and suitable sedation and analgesia. Selection of appropriate sized paddles is important. The preferred position for defibrillation is the sternum paddle at the base of the heart, apex paddle at the apex/axilla. SVTs usually require 0.5 -1 joule per kg. After defibrillation sinus rhythm is restored almost immediately in most of the cases but recurrence is common. Recurrent use causes myocardial damage.

Transesophageal overdrive pacing (TEP):

Normal sinus rhythm was successfully achieved on the first day of life by transesophageal atrial overdrive pacing. This is a noninvasive means to evaluate persistence of SVT substrate. Diagnosis and termination can also be achieved by this TEP method. TEP involves transnasal placement of a small pacing catheter in the esophagus posterior to the atrium of heart. A transesophageal stimulator is attached. Basic pacing maneuvers can be performed allowing evaluation of SVT, overdrive pacing to terminate SVT. TEP is successful in only 50% of the cases [43].

Prophylactic/ Long term therapy:

Indication of prophylactic therapy depends on age of onset, severity of symptoms and recurrence rate. This preventive treatment may consist of a beta blocker as first choice, with oral digoxin as an alternative. Anti-arrhythmic drugs flecainide and sotalol are widely used as next step therapy when digoxin and beta blocker fail to prevent recurrence [44].

Propranolol: This is a beta blocker agent, the most common first-line agent for maintenance therapy for SVT in neonate. The standard practice has been advocated to continue treatment for 6 months to a year, following which medications are

typically weaned off. The dose is 2-5 mg/kg/day, orally given in 3 divided doses. Important side effects are hypoglycemia, bronchospasm. Success rate of propranolol in the treatment of chronic therapy for SVT ranges from 50 to 90%.

Digoxin: This is a cardiac glycoside, given in a dose of 8-10 micro gram/kg/day, in 2 divided doses. Side effects are arrhythmia, digoxin toxicity. This drug is avoided in WPW syndrome.

Flecainide: Flecainide is a derivative of procainamide, slows down atrial as well as AV nodal conduction. It has been tried in chronic SVT caused by variety of mechanism. Dose is 2 mg/kg, slowly over 10-15 minutes. This medication should not be given to patients with organic heart disease or heart failure. Flecainide is negatively inotropic and can be proarrhythmic.

Catheter Radio Frequency Ablation (RFA) And Transcatheter cryoablation (TCA):

Catheter ablation using radiofrequency energy or cryoenergy is an effective therapy for ablation of additional pathway. Catheter tip uses hot energy in RFA and cold energy in TCA to destroy the target cardiac tissue. Advances in catheter design, energy delivery system, mapping as well as remote navigation system have rendered ablation of most SVT safe and effective. Indication of ablation therapy in neonate is drug resistant SVT accompanied by arrhythmogenic cardiomyopathy and heart failure. Also ablation therapy is effective in recurrent symptomatic atrial tachycardia in patient with congenital heart disease in early post-operative phase. Though RFA is a traditional technique, its short and long term success rate found to be 86% and 93% respectively [45]. Cryoablation is a safe and more effective newer technique with a very low procedure related risk and high success rate in the long term [46]. The procedure is quite invasive and need to postpone unless it's extreme necessity in neonatal life.

Prognosis: After the successful treatment of the neonatal SVT presenting in the first month of life, the prognosis is extremely good with the majority having no further recurrence [47,48]. Once the initial presentation is stabilized, most cases are controlled with oral medications and surprisingly many cases resolve spontaneously. The probability of complete resolution of SVT is dependent on age of onset. Most of the neonatal SVT resolve within the first month of life, compared to only 33%, if diagnosed after infancy. SVT with structural heart disease remains a difficult problem and treatment in this group remains guarded.

Follow-up plan:

SVT recurrence after hospital discharge is not uncommon. It is associated with low morbidity and mortality with a structurally normal heart. The follow-up plan, maintenance medication dose, duration should be made clear and issues to be kept in consideration:

- The first-line maintenance medication would likely be a beta-blocker, monitoring for hypoglycemia should be performed at home.
- The parents need to be taught how to take the heart

rate in sleeping newborn to identify SVT, might need the help of portable heart rate monitor or downloading heart rate apps in mobile phone.

- Vagal technique and CPR (cardio pulmonary resuscitation) should be taught to all family member.
- Therapeutic drug level monitoring, drug dose adjustment or ECG monitoring should be done at regular interval during hospital outpatient follow up.

Conclusion:

SVT can affect quality of life by leading morbidity and mortality of newborn. Prompt diagnosis and appropriate early management can potentially prevent life threatening events. Awareness about the various patterns of neonatal SVT is necessary for its early recognition. A careful search for preventive as well as associated factors of neonatal SVT should be undertaken to manage and prevent neonatal SVT in time.

References

1. Garson A, Gillette PC, McNamara DG (1981) Supraventricular tachycardia in children: clinical features, response to treatment, and long-term follow-up in 217 patients. *J Pediatr* 98:875–882. [View Article]
2. Sing HR, Garekar S, Epstein ML, Ecuyer TL (2005) Neonatal Supraventricular Tachycardia. *Neo Reviews* 6:339-349. [View Article]
3. Calabro MP (2008) Supraventricular Tachycardia in Infants: Epidemiology and Clinical Management. *Current Pharmaceutical Design* 14: 723-728. [View Article]
4. Konko J, Deal BJ, Strasburger JF, Benson DW (1992) Supraventricular tachycardia mechanisms and their age distribution in pediatric patients. *Am J Cardiol* 69: 1028-32. [View Article]
5. Allen HD, Shaddy RE, Penny DJ, Feltes TF, Cetta F (2016) *Moss And Adams' Heart disease in infants, children, and adolescents: including the fetus and young adult*. Volume 1. 9th ed. Philadelphia: *Wolters Kluwer* p. 623-54. [View Article]
6. Kantoch MJ (2005) Supraventricular tachycardia in children. *Indian J Pediatrics* 72: 609-619. [View Article]
7. Snyder CS, Fenrich AL, Friedman RA, Rosenthal G, Kertesz NJ et al. (2003) Usefulness of echocardiography in infants with supraventricular tachycardia. *Am J Cardiol* 91:1277–1279. [View Article]
8. Deal BJ, Keane JF, Gillette PC, Garson A (1985) Wolff-Parkinson-White syndrome and supraventricular tachycardia during infancy. *J Am Coll Cardiol*. 5: 130-135. [View Article]
9. Chua PY, Hill KD, Clark RH, Smith PB, Hornik CP (2015) Treatment of supraventricular tachycardia in infants: analysis of a large multicenter database. *Early Hum Dev* 91:345-350. [View Article]
10. William B, Selena R, Levine, Jyoti S, Kandlikar et al. (2011) Neonatal Supraventricular Tachycardia: recurrence in the first year of life. *JACC* 57. [View Article]
11. Sanatani S, Potts JE, Reed JH, Saul JP, Stephenson EA et al. (2012) The Study of Antiarrhythmic Medications in Infancy (SAMIS) A Multicenter, Randomized Controlled Trial Comparing the Efficacy and Safety of Digoxin Versus Propranolol for Prophylaxis of Supraventricular Tachycardia in Infants. *CircArrhythmElectrophysiol* 5: 984-991. [View Article]
12. LimYJ (2018) Management of intractable supraventricular tachycardia unresponsive to primary therapy with verapamil in a neonatal case. *J Pediatr Neonatal care* 8: 165-167. [View Article]
13. Hinkle KA, Peyvandi S, Stiver C, Killen SA, Weng HY et al. (2017) Postnatal outcomes of fetal supraventricular tachycardia: a multicenter study. *PediatrCardiol* 38:1317-1323. [View Article]
14. Chandra S, Seshadri B (2019) Neonatal supraventricular tachycardia. *Indian Pacing and Electrophysiology J* 19: 222-231. [View Article]
15. Ban JE (2017) Neonatal arrhythmias: diagnosis, treatment, and clinical outcome. *Korean J Pediatr* 60: 344-352. [View Article]
16. Goldman LE, Boramanand NK, Acevedo V, Gallagher P, Nehgme R et al. (2001) Preterm infants with paroxysmal supraventricular tachycardia: presentation, response to therapy, and outcome. *J Interv Card Electrophysiol* 5:293–297. [View Article]
17. Spearman AD, Williams P (2014) Supraventricular tachycardia in infancy and childhood. *Pediatr Ann* 43:456–460. [View Article]
18. Mehta AV, Sanchez GR, Sacks EJ, Casta A, Dunn JM et al. (1988) Ectopic automatic atrial tachycardia in children: clinical characteristics, management and follow-up. *J Am Coll Cardiol* 11:379–385. [View Article]
19. Poutiainen AM, Koistinen MJ, Airaksinen KE, Hartikainen EK, Kettunen RV, et al. (1999) Prevalence and natural course of ectopic atrial tachycardia. *Eur Heart J* 20:694–700. [View Article]
20. Neroni P (2014) Paroxysmal supraventricular tachycardia: pathophysiology and management. *J of Pediatric and Neonatal Individualized Medicine* 3:1-5. [View Article]
21. Dorostkar PC, Silka MJ, Morady F, Dick M (1999) 2nd Clinical course of persistent junctional reciprocating tachycardia. *J Am Coll Cardiol* 33:366–375. [View Article]
22. Sarubbi B, Vergara P, D'Alto M, Calabro R (2003) Congenital junctional ectopic tachycardia: presentation and outcome. *Indian Pacing Electrophysiol J* 3:143–147. [View Article]
23. Villain E, Vetter VL, Garcia JM, Herre J, Cifarelli A et al. (1990) Evolving concepts in the management of congenital junctional ectopic tachycardia. A multicenter study. *Circulation* 81:1544–1549. [View Article]
24. Keane JF, Lock JE, Fyler DC (2006) *Nadas Pediatric Cardiology*, Saunder Elsevier 2nd edition [View Article]
25. JK Reading, C Lin (2006) Supraventricular tachycardia during neonatal morgagni-larry hernia repair. *Pediatric anaesthesiology*. [View Article]
26. Fuertes A (2017) Supraventricular tachycardia in newborn and its association with gastroesophageal reflux disease. *AnPediatr(Barc)* 87:206-210. [View Article]
27. Sobaih BH, Hussain SA (2017) Fetal Tachycardia as Neonatal Atrial Flutter in Infants of Diabetic Mother (IDM): A Case Report. *Res J Clin Pediatr* 1: 2 [View Article]
28. Banjac L (2014) Tachycardia in a newborn with enterovirus infection. *Acta Clin Croat* 53:102- 106. [View Article]
29. Verheij G, Wintjens VS, Rozendaal L, Blom N, Walther F et al. (2009) Cardiac arrhythmia associated with umbilical venous catheterization in neonates. *BMJ Case Rep*. [View Article]

30. Say B (2015) Supraventricular tachycardia after nebulized salbutamol therapy in a neonate: case report. *Arch Argent Pediatr* 113:98-100. [[View Article](#)]
31. Yildirim SV, Tokel K, Saygili B, Varan B (2008) The incidence and risk factors of arrhythmias in the early period after cardiac surgery in pediatric patients. *Turk JPediatr* 50:549- 53[[View Article](#)]
32. Campuzano O, Brugada R, Iglesias A (2010) Genetics of Brugada syndrome. *Curr Opin Cardiol* 25:210-15[[View Article](#)]
33. JIE Hoffman, JH Mollar, Wiley-Blackwell, Chichester (2012) Pediatric Cardiovascular Medicine, UK, 2nd edition. [[View Article](#)]
34. Nadas AS, Daeschner CW, Roth A, Blumenthal SL (1952) Paroxysmal Tachycardia in infants and children: study of 41 cases. *Pediatrics* 9: 167-181. [[View Article](#)]
35. Hiranandani M, Kaur I, Kaur B, Singhi S C (1996) Neonatal supraventricular tachycardia. *Indian Pediatrics* 33: 678-683. [[View Article](#)]
36. Sreeram N, Wren C (1990) Supraventricular tachycardia in infants: Response to initial treatment. *Arch Dis Child* 65:127-129. [[View Article](#)]
37. Muller G, Deal BJ, Benson DW (1994) Vagal maneuvers and adenosine for termination of atrioventricular reentrant tachycardia. *Am J Cardiol* 74: 500-503. [[View Article](#)]
38. Overholt ED, Rheuban KS, Gutgesell HP, Lerman BB, Dimarco JP (1988) Usefulness of Adenosine for arrhythmia in infants and children. *Am J Cardiol* 61: 336-340. [[View Article](#)]
39. Munoz A, Leenhardt A, Saisine A (1984) Therapeutic use of adenosine for terminating spontaneous paroxysmal supraventricular tachycardia. *Eur Heart J*. 5: 735-738. [[View Article](#)]
40. Glatter K A, Cheng J, Dorostkar P (1999) Electrophysiologic effects of adenosine in patients with supraventricular tachycardia. *Circulation* 99: 1034-1040. [[View Article](#)]
41. Rhodes LA, Walsh EP, Saul JP (1994) Programmed atrial stimulation via the esophagus for management of supraventricular arrhythmia in infants and children. *Am J Cardiol*. 74: 353- 356. [[View Article](#)]
42. Till J, Shinebourne EA, Rigby ML, Clarke B, Ward DE et al. (1989) Efficacy and safety of adenosine in the treatment of supraventricular tachycardia in infants and children. *Br Heart J* 62:204-211. [[View Article](#)]
43. Gimovsky ML, Nazir M, Hashemi E, Polcaro J (2004) Fetal/neonatal supraventricular tachycardia. *J of perinatology* 24: 191-193. [[View Article](#)]
44. Malekin A, Khalilian MR, Dehdashtian M, Aramesh MR, Heydaripour K et al. (2016) Evaluation and Management of Neonatal Supraventricular Tachycardia. *J ComprPede* 35982 : 1- 5. [[View Article](#)]
45. Srintsova LI, Popov SV, Kovalev I A (2013) Radiofrequency ablation of drug refractory arrhythmia in small children younger than one year of age: single centre experience. *Pediatric Cardiology* 34: 1321-1329. [[View Article](#)]
46. Akdeniz C, Ergul Y, Kiplapinar N, Tuzcu V (2013) Catheter ablation of drug resistant supraventricular tachycardia in neonates and infants. *Cardiology Journal* 20:241-246. [[View Article](#)]
47. Chameides L, Brown GE, Raye JR (1977) Guideline for defibrillation in infants and children: Report of the American Heart Association target activity group. Cardio-pulmonary resuscitation in the young 56: 502 A. [[View Article](#)]
48. Salerno JC, Seslar SP (2009) Supraventricular Tachycardia. *Arch Pediatr Adolesc Med* 163:268-274[[View Article](#)]

Citation: Begum NA, Sharker S (2020) Neonatal Supraventricular Tachycardia - A Review of Literature. *Pediatr Res Child Health* 4(1): 001-007.

Copyright: © 2020 Begum NA, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.