Neonatal supraventricular tachycardia (SVT) – report of 2 cases

Vinko Vrdoljak[1], Lorita Mihovilović Prajz[1], Edi Paleka Bosak[1], Stella Radina Jurčić[1], Katarina Bojanić[1], Nikola Krmek[2]

[1] Division of Neonatology, Department of Obstetrics and Gynecology, „Sestre Milosrdnice“ University Hospital Center, Zagreb, Croatia
[2] Department of pediatrics, Division of Cardiology, Nephrology and Rheumatology with Immunology, “ Sestre Milosrdnice“ University Hospital Center, Zagreb, Croatia

Abstract:

Neonatal supraventricular tachycardia (SVT) is a significant medical emergency characterized by an abnormally rapid heart rhythm originating above the ventricles. It commonly manifests as atrioventricular reentrant tachycardia (AVRT) and presents diagnostic and therapeutic challenges in neonatology. Early recognition and appropriate management are critical to improving outcomes.

Case Reports

This paper details two cases of neonatal SVT. The first case involves a female neonate diagnosed with persistent fetal tachycardia at 31 weeks of gestation. Despite comprehensive maternal antiarrhythmic therapy, including sotalol, bisoprolol, digoxin, and flecainide, the tachycardia persisted, necessitating delivery at 37 weeks and subsequent neonatal interventions. The neonate required a combination of antiarrhythmic drugs, including propranolol, propafenone, and amiodarone, to achieve and maintain sinus rhythm. The clinical course was further complicated by early neonatal sepsis and human herpesvirus 6 (HHV-6) infection.

The second case describes a male neonate born with a history of suspected fetal tachyarrhythmia and congenital anomalies, including Wolff-Parkinson-White (WPW) syndrome and hereditary spherocytosis. Postnatal episodes of SVT were managed with adenosine and a regimen of antiarrhythmics, including propranolol, metoprolol, and sotalol. Despite recurrent SVT episodes and anemia requiring transfusions, the neonate achieved sustained remission of SVT before discharge.

Discussion

Management of neonatal SVT emphasizes stabilization, acute episode termination, and prevention of recurrence. Vagal maneuvers, adenosine, and synchronized cardioversion are first-line acute therapies. Long-term management often requires multiple antiarrhythmic medications tailored to the individual neonate. Spontaneous resolution of SVT is anticipated in most cases within the first year of life, reducing the long-term reliance on pharmacological therapy.

Conclusion

These cases highlight the complexity of neonatal SVT management, including the interplay of maternal and neonatal interventions, the risks of antiarrhythmic therapies, and the need for multidisciplinary collaboration. Early diagnosis and tailored treatment strategies are essential to improving neonatal outcomes, particularly in the presence of comorbid conditions like WPW syndrome and sepsis. Further research is needed to refine therapeutic protocols and optimize outcomes for this vulnerable population.

Keywords: Supraventricular Tachycardia; Neonatology; Arrhythmias, Cardiac; Anti-Arrhythmia Agents; Wolff-Parkinson-White Syndrome

OPEN ACCESS

Correspondence:
Vinko Vrdoljak
vinkov@gmail.com

This article was submitted to RAD CASA - Medical Sciences as the original article

Conflict of Interest Statement: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Received: 10 November 2024
Accepted: 2 December 2024
Published: 20 December 2024

Citation:
Vrdoljak V, Mihovilović Prajz L, Paleka Bosak E, Radina Jurčić S, Bojanić K, Krmek N. Neonatal supraventricular tachycardia – report of 2 cases
565=68-69 (2024): 96-102
DOI: 10.21857/mjrl3uopj9

Sažetak:

Neonatalna supraventrikularna tahikardija (SVT) – prikaz 2 slučaja

Neonatalna supraventrikularna tahikardija (SVT) značajno je hitno medicinsko stanje koje karakterizira abnormalno brzi srčani ritam koji nastaje iznad ventrikula. Obično se očituje kao atrioventrikularna reentrant tahikardija (AVRT) i predstavlja dijagnostički i terapijski izazov u neonatologiji. Rano prepoznavanje i odgovarajuće liječenje ključni su za poboljšanje ishoda.

Prikazi slučajeva

Ovaj rad opisuje dva slučaja neonatalne SVT. Prvi slučaj uključuje žensko novorođenče s dijagnozom perzistentne fetalne tahikardije u 31. tjednu trudnoće. Unatoč sveobuhvatnoj majčinoj antiaritmičkoj terapiji, uključujući sotalol, bisoprolol, digoksin i flekainid, tahikardija je trajala, što je zahtijevalo porod u 37. tjednu i naknadne neonatalne intervencije. Novorođenčetu je bila potrebna kombinacija antiaritmika, uključujući propranolol, propafenon i amiodaron, kako bi se postigao i održao sinusni ritam. Klinički tijek bio je dodatno kompliciran ranom neonatalnom sepsom i infekcijom humanim herpesvirusom 6 (HHV-6).

Drugi slučaj opisuje muško novorođenče rođeno sa poviješću sumnje na fetalnu tahiaritmiju i kongenitalne anomalije, uključujući Wolff-Parkinson-Whiteov (WPW) sindrom i nasljednu sferocitozu. Postnatalne epizode SVT-a liječene su adenozinom i režimom antiaritmika, uključujući propranolol, metoprolol i sotalol. Unatoč ponovljenim epizodama SVT i anemiji koja zahtijeva transfuziju, novorođenče je postiglo održivu remisiju SVT prije otpusta.

Rasprava

Liječenje neonatalne SVT naglašava stabilizaciju, završetak akutne epizode i prevenciju recidiva. Vagalni manevri, adenozin i sinkronizirana kardioverzija prva su linija akutne terapije. Dugoročno liječenje često zahtijeva višestruke antiaritmičke lijekove prilagođene pojedinom novorođenčadi. Spontano povlačenje SVT-a očekuje se u većini slučajeva unutar prve godine života, smanjujući dugoročno oslanjanje na farmakološku terapiju.

Zaključak

Ovi slučajevi naglašavaju složenost neonatalnog liječenja SVT-om, uključujući međudjelovanje majčinih i neonatalnih intervencija, rizike antiaritmijskih terapija i potrebu za multidisciplinarnom suradnjom. Rana dijagnoza i prilagođene strategije liječenja ključni su za poboljšanje neonatalnih ishoda, osobito u prisutnosti komorbidnih stanja poput WPW sindroma i sepse. Potrebna su daljnja istraživanja kako bi se poboljšali terapijski protokoli i optimizirali ishodi za ovu ranjivu populaciju.

Ključne riječi: supraventrikularna tahikardija; Neonatologija; Aritmije, srčane; Sredstva protiv aritmije; Wolff-Parkinson-Whiteov sindrom

Introduction

Supraventricular tachycardia (SVT) is defined as an abnormally accelerated heart rhythm that originates above the ventricles or the bifurcation of the Hiss bundle (atrial myocardium, AV node, proximal Hiss bundle, coronary sinus, pulmonary veins, vena cava or abnormal AV connections outside the Hiss bundle). It is often (not always) associated with a narrow QRS complex. Conventional SVT excludes atrial fibrillation and undulation as separate entities although they also originate from supraventricular structures (1). The incidence of SVT is estimated from 0.1 to 0.25 % (2-6.). In support of the occurrence of SVT in newborns, the following criteria are significant: the presence of new-onset tachycardia that is persistent and independent of activity, a heart rate above 220/min, and abnormal or absent P waves (7). In the therapeutic approach to SVT, it is essential to first terminate the existing episode using interventions such as vagal maneuvers, adenosine administration, electrocardioversion. Following the acute management, long-term strategies should be implemented to prevent the recurrence of SVT, which may include the use of antiarrhythmic medications, or catheter ablation.

Case report 1

A 39-year-old primiparous woman was admitted to the Department of Pregnancy Pathology at the KBC Sestre milosrdnice at 31 weeks of gestation due to fetal tachycardia. The maternal history includes a diagnosis of adult Still's syndrome (currently in remission) and gestational hypothyroidism, for which Euthyrox was prescribed during pregnancy. Upon admission, the mother underwent laboratory tests and an EKG, which were normal.

Due to persistent fetal tachycardia with frequency of approximately 200/min, consultations were sought from a rheumatologist, cardiologist, endocrinologist and pediatric cardiologist., Based on their recommendations antiarrhythmic therapy was initiated with Sotalol, which was subsequently swithed switched to Bisoprolol and then to Digoxin, with regular monitoring of therapeutic levels. However this treatment regimen did not lead to conversion of the fetal rhythm. Therefore, flecainide therapy was introduced alongside Digoxin,, with the dosage gradually increased (3 x 100 mg). Due to the development of nausea and dizziness, Digoxin therapy was discontinued. However, 3 days after discontinuation, SVT was again observed at a frequency of about 200/min. The dose of flecainide was subsequently (4x100 mg) increased, resulting in conversion to sinus rhythm, with a heart rate of about 165/min. During the subsequent course of treatment, the patient was monitored regularly by a pediatric cardiologist, who recommened a reduction in the dose of flecainide to 3x100 mg. Fetal echocardiography revealed no signs of decompensation or hydrops, with the fetal heart rate maintained at approximately 165/min. Due to an occurrence of bradycardia, and threatening asphyxia, delivery was performed via emergency caesarean section at 37 weeks and 3 days of gestation. . A healthy female neonate was delivered, weighing 3720 grams, and measuring 49 cm in length, with an Apgar score of 10 at 1 and 5 minutes. The newborn presented with tachycardia at birth and was subsequently transferred to the neonatal intensive care unit (NICU). Clinical examination revealed tachycardia with a rate ranging from 220 to230 beats per minute (Figure 1), no other significant abnormalities were noted. Upon admission to the NICU, an attempt was made to pharmacologically terminate the SVT, initially using adenosine and subsequently propafenone. These interventions resulted with a temporary cessation of the tachycardia; however the supraventricular tachycardia recurred shortly thereafter. The newborn remained hemodynamically stable, and propranolol was subsequently introduced into the treatment regimen. On the second day of life, in addition to the existing propranolol therapy, propafenone was introduced. From the ninth day of life, a third antiarrhythmic agent, Sotalol, was added. On the 20th day of life, Sotalol was replaced by Amiodarone, and on the 31st day of life, propranolol was substituted with metoprolol. A persistent sinus rhythm with a frequency of 130-160 beats per minute was achieved with this combination of antiarrhythmics, following careful titration of the doses. During the first 20days of hospitalization, the neonate required oxygen supplementation, provided either as ambient oxygen in the incubator or via a slow-flow nasal cannula. The clinical course was complicated by the development of early neonatal sepsis, necessitating the initiation of dual antimicrobial therapy with ampicillin and gentamicin on the second day of life. Due to the onset of fever on the 15th day of life, meropenem therapy was initiated and administered for a duration of eight days. Following the emergence of neurological symptoms, a lumbar puncture was performed, which resulted in the isolation of HHV-6.The infant was discharged home on the 52nd day of life in a hemodynamically and respiratory stable condition. Parents were educated on the recommended continuation of the three antiarrhythmic medications and the importance of follow-up with a pediatric cardiologist.

Case report 2

A male neonate was delivered vaginally from the mother's fourth pregnancy. The maternal history is significant for hereditary spherocytosis, for which a splenectomy was performed at the age of 12. Antenatal diagnostic tests ware not done. Ultrasound detected an anomaly of the right hand at 16 weeks of gestation Fetal echocardiography conducted at 30 weeks showed normal finding. The mother was subsequenly hospitalized at the pregnancy pathology department of another institution due to suspected fetal tachyarrhythmia. There were no signs of fetal hydrops observed. Due to the persistence of the tachyarrhythmia, digitalis was introduced, resulting in conversion to sinus rhythm. At 38 weeks and 6 days a healthy neonate with regular heart rhythm was born, weighing 3160 grams, 50 cm in length, and Apgar scores of 10 at 1. and 5. minute. Hypoplasia of the right forearm and hand was noted on clinical examination. An echocardiographic examination revealed a muscular VSD., and the ECG suggested Wolff-Parkinson-White (WPW) syndrome (Figure 2). Anemia and hyperbilirubinemia were observed from the first day of life, and a peripheral blood smear confirmed hereditary spherocytosis. The neonate underwent fototherapy until the sixth day of life. On the 10th day of live episodes of SVT were documented, which was successfully treated with adenosine on several occasions. Subsequenty, propafenone was introduced as part of the ongoing therapeutic regimen. On the 13th day of life, frequent paroxysms of SVT occured again and the neonate was transferred to our institution. Upon admission to the NICU, the neonate received a blood transfusion due to a significant drop in red blood count. On the same day, SVT reappeared, necessitating the repeated administration of adenosine. Consequently, propranolol was introduced on the 14th day of life in addition to thre previously administered propafenone. Despite titration of the antiarrhythmic therapy, recurrent episodes of SVT persisted. Propranolol was replaced by metoprolol on the 46th day of life, and for similar reasons, propafenone was replaced by sotalol on the 54th day of life. By the end of the hospitalization, a sustained remission of SVT was achieved with the combination of the previous two antiarrhythmics, SVT remission is achieved. During the hospitalization, the neonate remained clinically stable; however, due to a decrease in red blood count, he required blood transfusions on 3 additional occasions. The parents were educated regarding the care plan, and the neonate was discharged home on the 68th day of life with a recommended therapeutic regimen that included metoprolol and sotalol. Follow-up appointment with a pediatric cardiologist and hematologist were also advised.

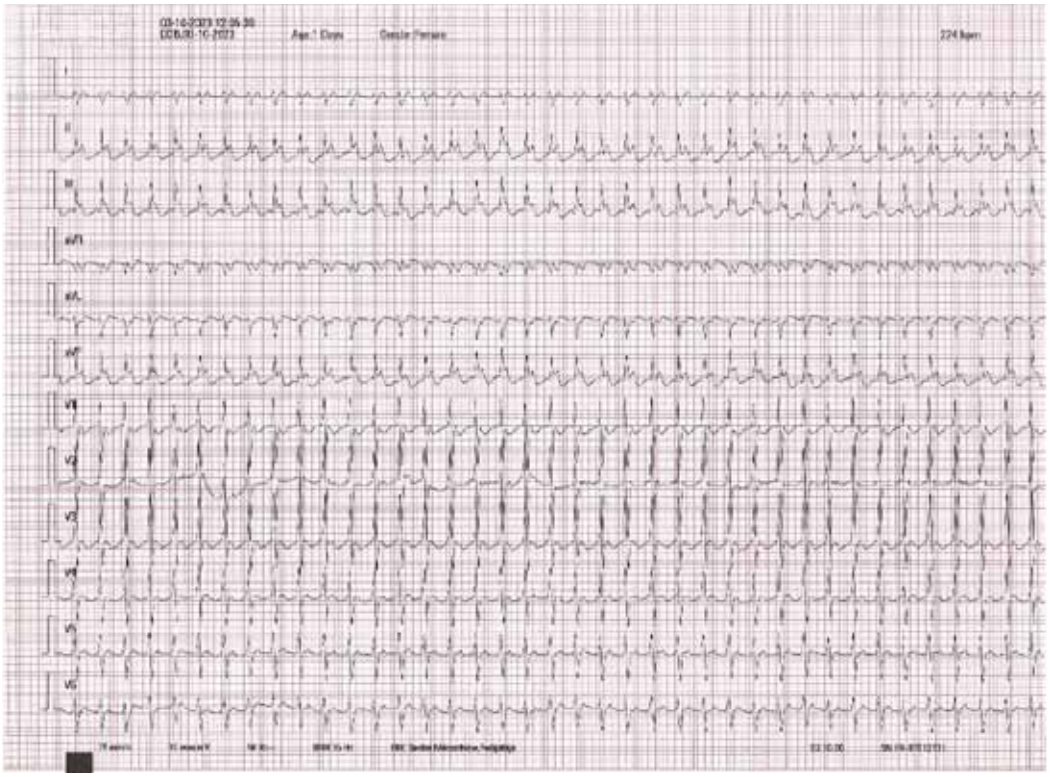

Figure 1 – SVT in the first patient

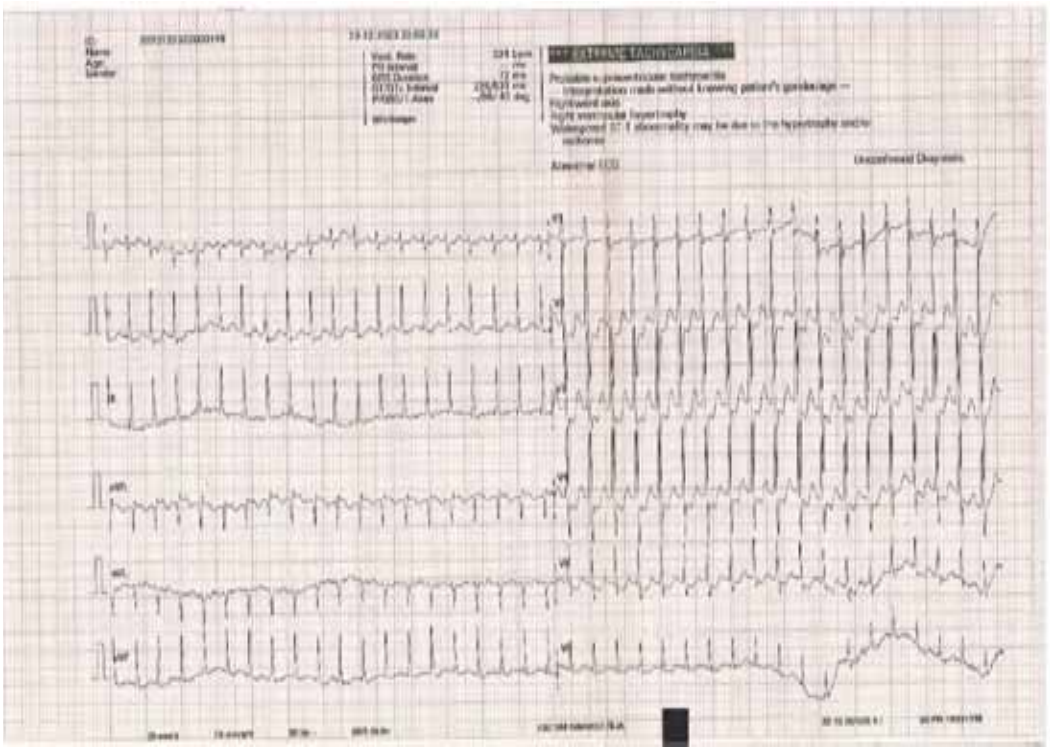

Figure 2 – SVT in another patient

Discussion

Supraventricular tachycardia (SVT) represents an emergency in neonatology, with the primary focus being the assessment of the patient's hemodynamic stability and the initiation of appropriate treatment. SVT encompasses several subtypes of arrhythmias, with atrioventricular reentrant tachycardia (AVRT) being the most common form observed in newborns (8). It is essential to evaluate the differential diagnosis of the mechanism of tachycardia based on the recording of a 12-lead ECG (width of the QRS complex, AV ratio, frequency regularity, PR interval and others). Ventricular tachycardia (VT) should always be ruled out. If P waves are visible, their axis can determine the origin of atrial activity. Sinus tachycardia is the most common type of "long PR, narrow QRS complex tachycardia" with a P wave directed between 0 and 90 degrees (positive in lead I and aVF). An A:V ratio of 1:1 is typical for AVRT and atrioventricular nodal reentrant tachycardia (AVNRT). More P waves than R waves imply an atrial tachyarrhythmia such as ectopic atrial tachycardia (Figure 4) (9).

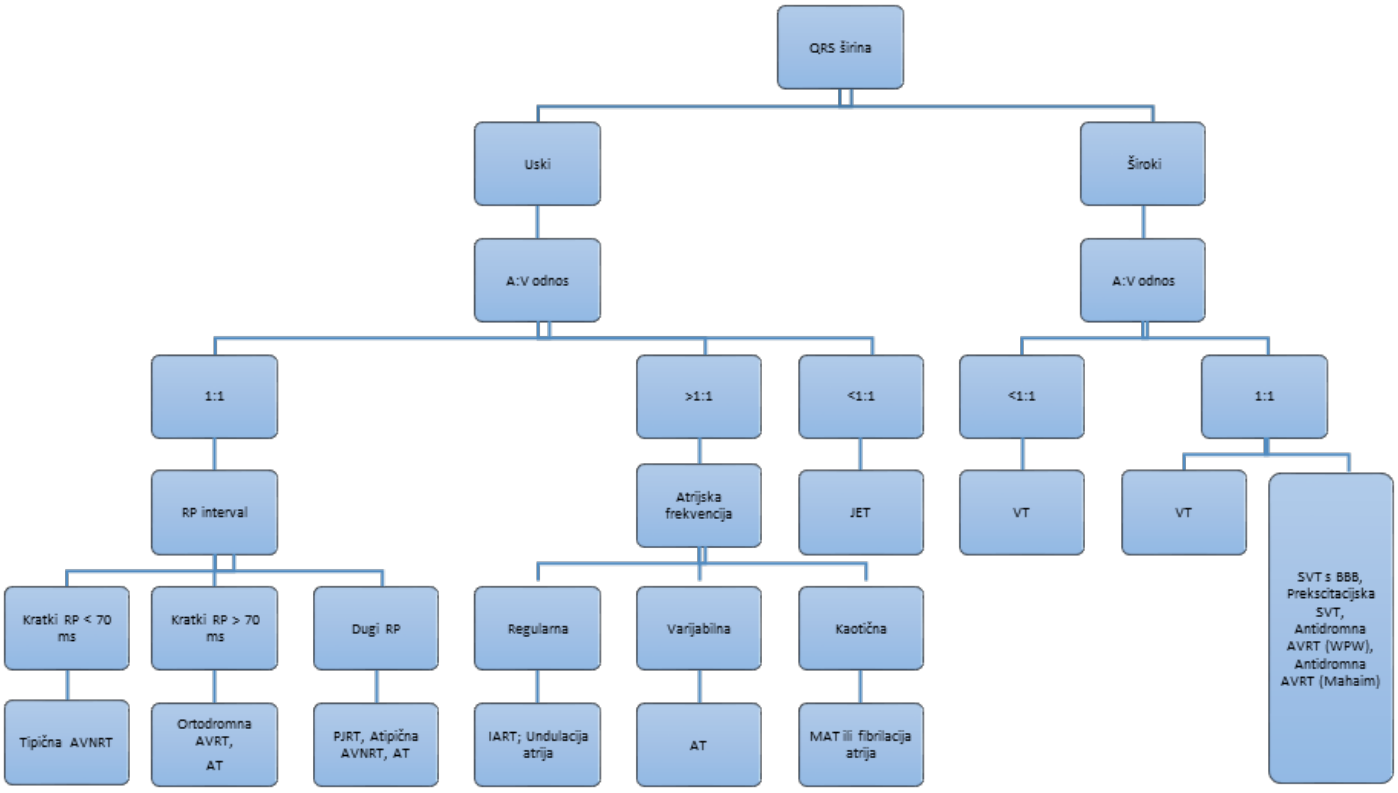

Figure 4. Tachyarrhythmia analysis algorithm based on ECG appearance (9); AT, Atrial tachycardia; BBB, bundle branch block; AVRT, Atrioventicular circular (reentrant) tachycardia; AVNRT, atrioventicular nodal circular (reentrant) tachycardia; IART, Intraatrial circular (reentrant) tachycardia; JET, Junctional ectopic tachycardia; MAT, Multifocal atrial tachycardia; PJRT, Permanent Junctional Reciprocal Tachycardia; SVT, Supraventricular Tachycardia; VT, Ventricular tachycardia; WPW, Wolff-Parkinson-White syndrome; RP interval, measured from the beginning of the QRS complex to the beginning of the next P wave on the ECG

The acute treatment of SVT begins with an assessment of the cardiovascular status of the neonate. In cases of cardiovascular and respiratory instability, immediate initiation of the cardiopulmonary resuscitation (CPR) protocol is warran ted. Most commonly, however, , the neonate is hemodynamically stable, allowing for treatment to focus on terminating the current episode of SVT. For this purpose, vagal maneuvers (ice on the face) and intravenous administration of adenosine (0.1 – 0.3 mg/kg i.v.) are employed, particularly for reentrant SVT involving the AV node. It is important to administer adenosin rapidly through the optimal venous access as close to the heart as possible. Following administration, the intravenous line should be flushed with a bolus of 0.9% NaCl to ensure effective delivery of the medication. Synchronized cardioversion is indicated for the conversion of unstable reentrant SVT (0.25 J/kg) and atrial undulation (0.5 J/kg)/fibrillation (1-2 J/kg). It is essential to assess whether SVT has been mechanically provoked by a presence of a central venous catheter (eg umbilical catheter) and to reposition or remove it if necessary. In case of recurrent SVT, the use of antiarrhythmic medications is indicated to prevent further episodes of SVT, while anticipation the spontaneous resolution of the underlying substrate responsible for SVT. Namely, 60-90% of newborns presenting with WPW syndrome or AVRT, as well as 20-50% of those with less frequent tachycardias, will not have a relapse after stopping antiarrhythmic therapy after 6-12 months, i.e. a spontaneous resolution is expected (10,11 ,12). The treatment regimen generally lasts 6-12 months (often multiple combinations of antiarrhythmic drugs), with gradual withdrawal of therapy (table 1) (9).

Table 1

First-line antiarrhythmics in the treatment of SVT in newborns (oral therapy)

Tretment (drug)	Class	Dosing	Most common side effects/Comment
Propranolol	Beta blockers	2-5 mg/kg/d in 3-4 doses	Hypoglycemia, bronchoconstriction
Digoxin	Cardiac Glycosides	8-10 ug/kg/d in 2 doses	Avoid with WPW, increases anterograde conduction, can cause serious arrhythmias
Second-line antiarrhythmics in the treatment of SVT in newborns (oral therapy)			
Sotalol	Class III	Initially 2 mg/kg/d in 3 doses, if necessary increase by 1-2 mg/kg/d over 3 days, upper dose 4 mg/kg/d in 3 doses (max 6 mg/kg/d in a 1-month-old infant	Monitor the QTc interval, deterioration of the systolic function of the ventricles
Flecainide	Class IC	Initially 2 mg/kg/d in 2 doses, titrate up to 3-6 mg/kg/d	Monitor QRS complex and QTc interval, monitor drug concentration in plasma, avoid simultaneous use with adapted milk products, in case of simultaneous administration with amiodarone, reduce the dose of flecainide by 50%
Propafenone	Class IC	Initially 7-10 mg/kg/d in 3 doses, increase by 20-30% to a maximum dose of 20 mg/kg/d	Monitor QRS complex and QTc
Amiodarone	Class III	Loading dose 10-20 mg/kg/d in 2 doses over 7-10 days, then 5-10 mg/kg/d once a day over 2-7 months	Monitor QTc, thyroid function tests, control GUK, electrolytes, transaminases, triglycerides, ophthalmological examination

Conclusion

SVT is an emergency condition in neonatology that requires prompt evaluation and intervention, initially aimed at restoring sinus rhythm, and if required prophylactic antiarrhythmic therapy. Atrioventricular circular ("re-entry") tachycardia due to atrioventricular bypass is the most common type of SVT in neonates, with a high rate of spontaneous resolution expected by the first year of life. The primary therapeutic approach includes the use of antiarrhythmic medications (often multiple combinations). Due to associated risks, catheter ablation is reserved for extreme cases where there is an inadequate response to prior therapies or a sighnificant risk of sudden cardiac death.

References

1. Josephson ME, Wellens HJ. Differential diagnosis of supraventricular tachycardia. Cardiol Clin 1990; 8:411.
2. Geggel RL. Conditions leading to pediatric cardiology consultation in a tertiary academic hospital. Pediatrics 2004;114:e409e17. https://doi.org/10.1542/peds.2003-0898-L.
3. Garson A, Gillette PC, McNamara DG. Supraventricular tachycardia in children: clinical features, response to treatment, and long-term follow-up in 217 patients. J Pediatr 1981;98:875e82. https://doi.org/10.1016/S0022-3476(81)80578-1.
4. Lundberg A. Paroxysmal tachycardia in infancy. A clinical and experimental study. Acta Paediatr 1963;52:192e5.
5. Nadas AS, Daeschner CW, Roth A, Blumenthal SL. Paroxysmal tachycardia in infants and children; study of 41 cases. Pediatrics 1952;9:167e81.
6. Weindling SN, Saul JP, Walsh EP. Efficacy and risks of medical therapy for supraventricular tachycardia in neonates and infants. Am Heart J 1996;131:66e72. https://doi.org/10.1016/S0002-8703(96)90052-6.
7. Kleinman ME, Chameides L, Schexnayder SM, et al. Part 14: pediatric advanced life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010; 122:S876.
8. Kothari, D & Skinner, Jonathan. (2006). Neonatal tachycardias: An update. Archives of disease in childhood. Fetal and neonatal edition. 91. F136-44. 10.1136/adc.2004.049049.
9. Srinivasan C, Balaji S. Neonatal supraventricular tachycardia. Indian Pacing Electrophysiol J. 2019 Nov-Dec;19(6):222-231. doi: 10.1016/j.ipej.2019.09.004. Epub 2019 Sep 18. PMID: 31541680; PMCID: PMC6904811.
10. Deal BJ, Keane JF, Gillette PC, Garson A. Wolff-Parkinson-White syndrome and supraventricular tachycardia during infancy: management and follow-up. J Am Coll Cardiol 1985;5:130e5. https://doi.org/10.1016/S0735-1097(85)80095-4.
11. Sanatani S, Potts JE, Reed JH, Saul JP, Stephenson EA, Gibbs KA, et al. 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. Circ Arrhythmia Electrophysiol 2012;5:984e91.
12. Perry JC, Garson A. Supraventricular tachycardia due to Wolff-Parkinson-White syndrome in children: early disappearance and late recurrence. J Am Coll Cardiol 1990;16:1215e20. https://doi.org/10.1016/0735-1097(90)90555-