Bibliografie

1. Alexander D. Hussey1, Robert G. Weintraub et. al. Drug Treatment of Heart Failure in Children: Focus on Recent Recommendations from the ISHLT Guidelines for the Management of Pediatric Heart Failure. Pediatr Drugs DOI 10.1007/s40272-016-0166-4.

2. Bressieux-Degueldre S., Sekarski N. Insuffisance cardiaque chez l'enfant: reconnaître et diagnostiquer. Paediatricia, 2015, vol. 26, nr. 1, p. 12-14.

3. Kirk R, Dipchand AI, Rosenthal DN, editors. ISHLT Guidelines for the Management of Pediatric Heart Failure. Birmingham: University of Alabama at Birmingham; 2014.

4. Kantor PF. ş.a. CCS Guidelines for HF in Children. Canadian journal of Cardiology, 2013, vol. 29, p. 1535-1552.

5. Masarone D., Valente F. et al. Pediatric Heart Failure: A Practical Guide to Diagnosis and Management. Pediatrics and Neonatology, 2017, vol. 58, p. 303-312.

6. Ponikowski P., ş.a. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC).Developed with the special contribution of the Heart Failure Association (HFA) of the ESC. European Heart Journal, 2016, vol. 37, nr. 27, p. 2129-2200.

7. Yancy et al. JACC Vol. 62, No. 16, 2013 2013 ACCF/AHA Heart Failure Guidelines: Executive Summary. JACC, 2013, vol. 62, nr. 16, p: 1495-539.

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SUPRAVENTRICULAR TACHYCARDIA IN CHILDREN

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Summary

TAHICARDIA SUPRAVENTRICULARĂ LA COPII

Cuvinte cheie: tahicardia supraventriculară, adenozina, copii

Tahicardia supraventriculară este cea mai frecventă aritmie pediatrică simptomatică. Incidența este estimată de la 1 la 250 până la 1 la 1000 copii. 50 la sută din copii cu tahicardie supraventriculară prezintă primul episod în primul an de viață. Manifestarea clinică a tahicardiei supraventriculare este dependentă de vârstă şi durata accesului. Frecvența cardiacă la sugari cu tahicardie paroxistică variază de la 220-320 b/min., la copiii de vârstă mare de la 160-280 b/min. Tahiaritmia netratată poate provocoa insuficiență cardiacă congestivă în 24-48 ore. Tratamentul acut începe cu documentarea aritmiei, aprecierea statutului hemodinamic şi recunoaşterea mecanismului aritmiei. Tratamentul cu medicație rămîne de prima linie la sugari şi copii de vârstă fragedă, pentru copiii de vârstă mare şi adolescenți ablația prin cateter este o metodă inofensivă şi efectivă.

Резюме

СУПРАВЕНТРИКУЛЯРНАЯ ТАХИКАРДИЯ У ДЕТЕЙ

Ключевые слова: суправентрикулярная тахикардия, аденозин, дети

Суправентрикулярная тахикардия является самой частой симптоматической аритмией в педиатрии. Частота суправентрикулярной тахикардии от 1 на 250 до 1 на 1000 детей, 50% детей имеют приступ тахикардии на первом году жизни. Клиническое проявление тахиаритмии зависит от возраста и продолжительности приступа. Пульс у новорожденных с суправентрикулярной тахикардией варьирует от 220-320 ударов в минуту у новорожденных и у детей старшего возраста от 160-280 ударов в минуту. Тахиаритмия без лечения может привести к сердечной недостаточности в течении 24-48 часов. Неотложная помощь начинается с диагностики аритмии, оценки гемодинамики и механизма аритмии. Консервативное лечение остается базовой у новорожденных и детей младшего возраста. Детям старшего возраста и подросткам рекомендуется катетерная аблация как малотравматичный и эффективный метод.

Introduction. Supraventricular tachyarrhythmia (SVT) is the most symptomatic pediatric arrhythmia resulting from an abnormal mechanism that requires structures in the heart above the bifurcation of the bundle of His for its continuation. Clinical symptoms of SVT are variable; ranging from asymptomatic or minor palpitations to more severe manifestations.

Supraventricular tachycardia (SVT) is defined as a narrow, complex tachycardia that requires atrial tissue or the atrioventricular node as an integral part of the arrhythmia substrate. The incidence of SVT in children has been estimated to be from 1 in 250 to 1 in 1000 children. Approximately 50% of children with SVT will present with their first episode in the first year of life. After infancy, the incidence peaks in early childhood (ages 6-9 years) and then again in adolescence. In infants, spontaneous resolution occurs in more than 90% by 1 year of age, up to one-third will have recurrence of SVT at a mean age of 8 years [1,9]. Although spontaneous resolution is the norm in infants, only a small minority (15%) of patients who receive the diagnosis after 1 year of age will have spontaneous resolution. Supraventricular tachycardia due to WPW accessory pathways predominates throughout childhood and adolescence, whereas the relative proportion of patients with atrioventricular nodal reentry tachycardia(AVNRT) tends to increase with age. Most individuals with SVT have a structurally normal heart. The prevalence of structural congenital heart disease in patients with SVT has been estimated at 9% to 32%, which is substantially higher than in the general population. The most common association is noted between WPW syndrome and the Ebstein anomaly of the tricuspid valve, but a number of defects have been found, including ventricular or atrial septal defects, among others [6,9].

Most cases of reentrant SVT are sporadic, with approximately 7% of patients having documented SVT in a first degree relative. Most cases of WPW syndrome are also sporadic, although patients with WPW syndrome have a 3-fold higher risk than the general population of having an affected first-degree relative with WPW [9].

Variable mechanisms of SVT exist; we concentrated on the three commonest forms occurring in children which are: atrioventricular re-entry tachyarrhythmia (AVRT), atrioventricular nodal re-entry tachyarrhythmia (AVNRT), and atrial tachyarrhythmia (AT) [4].

An accessory pathway (AP) is a microscopic bridge of muscle connecting atrium and ventricle that bypasses the normal electrical insulation of the AV ring. The impulse spreads down either via the atrioventricular (AV) node or the AP and then up the other. A re-entrant unconventional circuit is formed [7]. AVRT is the commonest type of SVT in the pediatric population forming two-thirds of the affected children, then comes the atrioventricular nodal reentry tachyarrhythmia (AVNRT) and the trial tachyarrhythmia (AT) [11]. AVRT is more common in males, but its incidence decreases with age. On the other hand, the incidence of AVNRT and atrial tachyarrhythmia is increased with age. The AV node is composed of ‘‘slow’’ pathway and ‘‘fast’’ pathway. AVNRT happens when an antegrade impulse is blocked at one pathway (usually the fast pathway), resulting in conduction over the other (usually the slow pathway). After the impulse is travelled down via the slow pathway, the fast pathway is now unblocked and the impulse can spread retrograde via the fast pathway [16]. AVNRT accounts for 15% of cases of pediatric SVT, mostly present over the age of 5 years and almost completely absent in infants. Females were more than males in cases diagnosed as AVNRT or AT. Atrial tachyarrhythmia (AT) is mostly due to abnormal automaticity, may be formed by remnant embryonic cells with automatic qualities, causing abnormal impulses that come away from normal sinus node but still within the atria. Although it is a rare condition, but still the third common mechanism of tachyarrhythmia occurring in children, if remains undetected can result in a dilated cardiomyopathy [16].

The clinical presentation of SVT is age and duration dependent. In infants with paroxysmal SVT, the heart rate is usually 220 to 320 beats/minute; in older children, it is 160 to 280 beats/minute. In infants, symptoms are usually nonspecific and include poor feeding, irritability, vomiting, cyanosis, and pallid spells. If the symptoms are unrecognized for hours to days, the infant can present with significant hemodynamic compromise or heart failure symptoms. It is rare for infants who have SVT for less than 24 hours to develop signs of congestive heart failure at the time of presentation; however, congestive heart failure is present in 19% of infants who have SVT for 24 to 36 hours and in 50% who have SVT for more than 48 hours. Approximately 20% of infants receive a diagnosis during routine office visits and during asymptomatic episodes. In verbal children with SVT, palpitations and fluttering in the chest are the usual presenting symptoms. Because reentrant arrhythmias are a circuit, they tend to be all or nothing, and the onset is frequently described as being abrupt, similar to a light switch being turned on. The offset may be less dramatic because the catecholamine level is typically elevated, with resultant sinus tachycardia at the termination of SVT and subsequent gradual slowing. Frequently, light headedness and dizziness due to transient hypotension can occur at the onset, but syncope is rare in SVT, and its presence should raise suspicion of something other than SVT. The frequency and duration of the episodes vary greatly

from a few minutes to a few hours and occur as often as daily or as infrequently as once or twice per year [6,9].

Most patients presenting with episodic palpitations have a structurally normal heart and will have normal findings on the physical examination, particularly older children. Infants are more likely to present with signs of heart failure because the tachycardia may have gone unrecognized for longer periods.

Auscultation should be done to reveal the presence of structural heart disease, heart rate and rhythm should be noted. Further evaluation will depend on the severity and frequency of symptoms, the age of the child, and the presence of structural heart disease. Untreated SVT can result in congestive heart failure (CHF) within 24 to 48 hours. Termination of the attack by vagal maneuvers may suggest a reentrant tachyarrhythmia involving AV node [11]. Recording a 12- Lead Electrocardiography (ECG) at rest should be done and examined for any abnormal rhythm, delta waves, abnormal QT interval, sinus tachyarrhythmia, or any sign of underlying structural heart disease [2]. ECG can clearly diagnose about 80% of AVNRT and AVRT, but incorrectly categorize approximately 20% of cases of SVT [15].

Holter monitor it can record continuous ECG tracing for 24 to 48 hours, having the same idea as classic ECG wires and electrodes but it is a portable device that provides information about symptoms that can occur during the day [16].

In selected patients with occasional complaints (less than two episodes of SVT per month) and associated with disabling symptoms (hemodynamic instability) can be diagnosed by implantable loop recorders. If the clinical history is not enough or other measures have failed to identify the SVT mechanism, then recordings and stimulation can be done via tranesophageal a trial loops for these patients for diagnosis or to provoke paroxysmal tachyarrhythmia [14].

The possibility of structural heart disease should be excluded by Echocardiography examination in children with identified attacks of SVT, which is not possible to be detected via physical examination or resting 12-lead ECG [5].

Other investigations can be done to exclude other causes included in the differential diagnosis of SVT. Chest x-ray with lateral and anteroposterior views is done to diagnose cardiomyopathy and CHF. Laboratory tests which include; serum electrolytes to diagnose any imbalance leading to abnormal cardiac rhythm, complete blood cell count (CBC) with differential to exclude infections or anemia, also, screening of toxic substances, thyroid function tests, and arterial blood gas can be helpful [11].

The most accurate diagnosis can be done with the electrophysiological study (EPS). It is used for clear classification of different mechanisms of SVT. Also, EPS combined with catheter ablation can be used as a definitive long-term therapy [16].

Management of supraventricular tachycardia

The dealing with SVT can be done in two ways: acute termination of the attack of tachyarrhythmia and prevention of recurrences. The acute treatment begins with documentation of the arrhythmia with a 12 lead ECG, assessment of hemodynamic status and recognition of the arrhythmia mechanism. Chronic therapy is based on the SVT mechanism, the patients age, and the frequency and patients complaint during the attack as well as access to medical care and sophistication of the patient caretaker [12]. For the aerodynamically tolerated episode, vagal physical maneuvers are to be used first. The patient may be taught to do Valsalva maneuver , induce vomiting, dive the face in the iced water for 10-20 seconds, or carotid sinus massage may be done, an ocular pressure is not recommended for children. These physical maneuvers are potent in terminating re-entry SVT; mostly AVRT and to less extent AVNRT [2].

An ultra-short-acting drug (Adenosine) is highly effective in terminating AV node-dependent SVT, but it is of limited value in the diagnosis of AT. Vagal maneuvers and adenosine produce transient AV node block and result in sudden termination of SVT in any tachyarrhythmia involving AV node but not in AT [13].

The use of Beta blockers or long-acting calcium channel blockers is of value especially for patients with frequent atrial or ventricular premature beats which may act as a cause of recurrence of SVT but they must be used with great caution as they may potentiate hypotension [2].

In an aerodynamically unstable child with severe hypotension, electrical cardio version by synchronized DC shocks is the treatment of choice. It is a safe and effective procedure in the majority of patients [3]. Long-term management is dependent on a number of factors, including the age of patient, duration and frequency of episodes and presence of ventricular dysfunction. In children with infrequent, mild and self-limiting episodes, usually no treatment is needed. In children in whom the episodes are frequent, prolonged, difficult to terminate or interfering with sports participation, treatment is indicated. Treatment options include medications or transcatheter ablation. The purpose of using anti-arrhythmic drugs is to slow conduction, preferentially within one limb of the reentrant circuit and therefore terminate the tachycardia. Treatment options include digoxin, beta-blockers, calcium-channel blockers and the sodium-channel blocker, flecainide. While there is signifcant variation in practice, the majority of European centres use flecainide or atenolol as the first choice of drug for the prevention of recurrent SVT [8]. There is little difference in the efficacy of various medications and

a randomised controlled trial comparing digoxin and propranolol found no difference in recurrence of SVT in the two groups. While any of the anti-arrhythmics can be used to initiate treatment, in cases of WPW syndrome, use of calcium-channel blockers or digoxin should be avoided. Management of SVT has been revolutionised with the development of transcatheter ablation which is now considered standard treatment for older children and adolescents. Radiofrequency (RF) is the preferred energy source for paediatric arrhythmias and catheter ablation is only chosen if two or more antiarrhythmic drugs have failed [8]. An alternative to RF is cryoablation, which is safer and minimises the risk of heart block during ablation. However, it is associated with a higher SVT recurrence rate and therefore most centres use cryoablation in cases where RF ablation is considered a higher risk [10].

Conclusions

SVT is a common condition in the pediatric population. In most cases, it represents a benign heart rhythm disorder, but infants, athletes, and patients with WPW syndrome deserve special consideration because of greater associated risks in these populations. Treatment with suppressive medications remains the first-line therapy for infants and small children. For symptomatic older children and adolescents, catheter ablation appears to be a safe, effective form of treatment.

Bibliography

1. Anand RG, Rosenthal GL, Van Hare GF, Snyder CS. Is the mechanism of Supraventricular tachyarrhythmia in pediatrics influenced by age, gender or ethnicity? In: Conge nit. Heart Dis, 2009,vol. 4(6),p. 464- 468.

2. Blomström-Lundqvist C, Scheinman MM, Aliot EM, Alpert JS, Calkins H, Camm AJ, et al. ACC/AHA/ESC guidelines for the management of patients with Supraventricular arrhythmias--executive summary. a report of the American college of cardiology/American heart association task force on practice guidelines and the European society of cardiology committee for practice guidelines (writing committee to develop guidelines for the management of patients with Supraventricular arrhythmias) developed in collaboration with NASPE-Heart Rhythm Society. In:J Am Coll Cardiol, 2003, vol. 42(8), p. 1493-1531.

3. Chun TU, Van Hare GF. Advances in the approach to treatment of Supraventricular tachyarrhythmia in the pediatric population. In: Carr Cardio Rep, 2004, vol. 6(5), p. 322-326.

4. Doniger SJ, Sheriff GQ. Pediatric dysrhythmias. In: Pediatr Clin North Am Pediatr, 2006, vol. 53 (1), p. 85-105.

5. Emmel M, Sreeram N, Schickendantz S, Brockmeier K. Experience with an ambulatory 12-lead Holter recording system for evaluation of pediatric dysrhythmias . In: J Electrocardiol , 2006, vol. 39(2), p. 188-193.

6. Hany MA Abo Hadeed. Diagnosis and Treatment of Supraventricular Tachyarrihythmia in Pediatric Population: a Review Article. In: Academic Journal of Pediatrics and Neonatology, 2017, vol. 2(4), p.555-595.

7. Hafez, MM. Abu-Elk heir, M. Shakier, HF. Al-Marsafawy, HM. Abo-Haded et al. Intermediate Term Follow Up Results. In: Clin Med Insigne Cordial, 2012, vol. 6, p. 7-16.

8. Hernandez-Madrid A, Hocini M, Chen J, et al; How are arrhythmias managed in the paediatric population in Europe? Results of the European Heart Rhythm survey. Europace, 2014, vol.16(12), p.1852-6.

9. Jack C. Salerno, MD; Stephen P. Sesler, MD, PhD. Supraventricular Tachycardia. In: Arch Pediatr Adolesc Med, 2009, vol. 163(3), p. 268-274.

10. Khairy P, Dubuc M; Transcatheter cryoablation part I: preclinical experience.In: Pacing Clin Electrophysiol, 2008, vol. 31(1), p.112-20.

11. Lad sans EJ, Diagnosis, evaluation and treatment of cardiac arrhythmias.In: Pediatr Child Health, 2009, vol. 1, p. 30-36.

12. Lee KW, Badhwar BS, Scheinman MM, Supraventricular tachyarrhythmia- Part II: History, Presentation, Mechanism, and treatment. In: Curr Probl Cardiol, 2008, vol. 33(10), p. 557-622

13. Lee PC, Hwang B, Chen SA, Tai CG, Chen YJ, et al. The results of radiofrequency catheter ablation of Supraventricular tachyarrhythmia in children. In: Pacing Clin Electrophysiology, 2007, vol. 30(5) p. 655-661.

14. Melton PE, Zlojutro M, Kimminau K, Crawford MH. Biological aging and Cox hazard analysis of mortality trends in a Mennonite community from south-central Kansas. Am J Hum Biol X, 2006, vol. 18(3), p. 387- 401.

15. Snyder CS, Fenrich AL, Friedman RA, Rosenthal G, Kertesz NJ. Usefulness of echocardiography in infants with Supraventricular tachyarrhythmia. In: Am J cardiol,2003, vol. 91(10), p. 1277-1279.

16. Schlechte EA, Boramanand N, Funk M. Supraventricular Tachyarrhythmia in the Pediatric Primary Care Setting: Age related Presentation, Diagnosis, and Management. In: J Ped Health Care, 2008, vol. 22(5), p. 289-299.