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EPIDEMIOLOGY OF RHYTHM DISORDERS IN CHILDREN

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Abstract:
Unlike the adult population, arrhythmias are less common in childhood. Only 5% of pediatric emergency hospitalizations are for symptomatic arrhythmias [3,6,9]. Most of these tend to be supraventricular tachyarrhythmias mediated by accessory pathways such as Wolff-Parkinson-White (WWS) syndrome, persistent junctional reentrant tachycardia (JRT) and Mahaheim tachycardia [1,2]. Supraventricular tachyarrhythmias mediated by non-accessory pathways, often observed in children, are nodal ectopic tachycardia (ET) and automatic ectopic atrial tachycardia (AET) [2] and occur mainly in the postoperative period after intracardiac repair for structural heart disease. Ventricular tachycardia (VT), although rare, occurs in the pediatric age group in association with hypertrophic cardiomyopathy (HCM), long QT syndrome (LQT), and Brugada syndrome. Occasionally, VT may also present symptomatically as incessant idiopathic infantile ventricular tachycardia, right ventricular outflow tract tachycardia, benign VT, catecholamine VT, idiopathic left ventricular tachycardia, and in patients after cardiac surgery.

Keywords: Ventricular tachycardia, hypertrophic cardiomyopathy, long QT syndrome, Brugada syndrome, atrioventricular re-entry

INTRODUCTION. In contrast to the adult population, arrhythmias are less common in pediatric patients. Only 5% of pediatric emergency hospitalizations are for symptomatic arrhythmias [3,6,9]. The majority of these tend to be supraventricular tachyarrhythmias mediated by additional pathways such as Wolff-Parkinson-White syndrome (WPWS), persistent nodal reciprocal tachycardia (PURT) and Mahaim tachycardia [1,2]. Supraventricular tachyarrhythmias mediated by a non-additional pathway, frequently observed in children, are nodal ectopic tachycardia (ET) and automatic ectopic atrial tachycardia (AET) [2] and occur mainly in the postoperative period after intracardiac plasty for structural heart disease. Ventricular tachycardia (VT), although rare, occurs in pediatric age group in association with hypertrophic cardiomyopathy (HCMP), prolonged QT interval (PQT) syndrome and Brugada syndrome. Occasionally, VT may also present symptomatically as unremitting idiopathic pediatric ventricular tachycardia, right ventricular outflow tract tachycardia, benign VT, catecholamine VT, idiopathic left ventricular tachycardia, and in patients after cardiac surgery.

Fetal tachyarrhythmias

Atrioventricular re-entry (AVRI) remains the most frequent mechanism of fetal tachyarrhythmias. Atrial reentry tachycardias are infrequently observed. In persistent tachycardia, there is a 50% risk of fetal congestive heart failure and a 50% risk of fetal death in untreated hydrocele [3,10,12]. Sustained tachycardia and earlier gestational age at admission are associated with a higher risk of congestive heart failure. Treatment options after the diagnosis of fetal tachycardia include observation, induction of labor if the gestational age is long enough, transplacental drug therapy, or direct fetal intrauterine therapy by intra-amniotic, intraperitoneal, intramuscular, or intravenous routes. Oral administration of digoxin, flecainide, or both to the mother has been a widely tried and successful treatment for fetal tachyarrhythmias.

Supraventricular arrhythmias

Supraventricular tachycardia (SVT) occurring at an earlier age is usually mediated by an additional pathway. Atrioventricular reciprocal tachycardia (AVRT) accounts for 85% of intrauterine arrhythmias and 82% of arrhythmias occurring in infancy. In most cases, the tachycardia resolves spontaneously by the end of infancy, although late recurrences may occur [4,5,12]. The incidence decreases to 65% in the 1-5 years age group, 56% in the 6-10 years age group, and 68% in the over 10 years age group. Atrioventricular nodal reciprocal tachycardia (AVURT) in infancy is rare and accounts for only 4% of arrhythmias. The incidence is about 23% in the 1 to 5 year age group, 34% in the 6 to 10 year age group, and 20% in children over 10 years of age. Most of them do not resolve spontaneously and

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require radiofrequency (RF) ablation [13,19]. Less common arrhythmias include atrial flutter (AF), chaotic atrial tachycardia, and atrial ectopic tachycardia. The incidence of atrial tachycardia in childhood is about 10-15%, and most cases resolve spontaneously. If persistent, radiofrequency ablation is curable [20,21].

Wolff-Parkinson-White (WPW) syndrome is a typical example of AVRT in children. The population prevalence of ventricular preexcitation is about 1.5 per 1000 in adolescence and probably lower in early childhood [16,22]. In a significant proportion of patients, the ECG does not detect ventricular preexcitation at the time of presentation. Nearly 50% remain asymptomatic at diagnosis, and 30% develop arrhythmia-related symptoms at a later time [16,21]. The overall risk of VSS in WPW syndrome is low and is estimated at 2 per 1000 patient-years [7]. VSS in WPW is probably due to ventricular fibrillation secondary to atrial fibrillation. The main predictor of the risk of VSS is symptoms. A patient with syncope or a patient previously resuscitated from cardiac arrest is at higher risk. Other risk factors are multiple additional conduction pathways, Ebstein's anomaly of the tricuspid valve, and familial preexcitation. High-risk patients usually show a short refractory period of less than 220 ms during electrophysiologic study. Noninvasively, the refractory period can only be measured in the presence of spontaneous atrial fibrillation. The shorter the RR interval, the higher the risk of syncope or sudden cardiac death. Right posterior septal pathway on resting ECG is also detected in WPW syndrome patients resuscitated due to VF and is considered a high risk for sudden cardiac death. In contrast, sudden loss of preexcitation on the stress ECG and intermittent or variable preexcitation on ambulatory ECG monitoring imply a prolonged refractory period and a lower risk of associated sudden cardiac death [18,23]. Radiofrequency ablation is the method of choice in symptomatic patients with WPW syndrome [11].

Permanent nodal reciprocal tachycardia is the most common form of incessant supraventricular tachycardia in children. It is an orthodromic AVRT with RP>PR that usually manifests at the age of 3-4 years. They are likely to persist for long periods of time and are known to cause tachycardiomyopathy. Although pharmacologic control is possible with amiodarone or verapamil or in combination with digoxin, radiofrequency ablation has been reported to be the best long-term treatment [10].

Atrial tachycardias in childhood are usually seen as a result of postoperative atrial scarring, distorted anatomy, changes in wall stress, and changes in atrial refractoriness associated with sinus node dysfunction. Atrial tachycardia with one-to-one conduction can cause VSS. In a collaborative study of 380 cases of atrial flutter by electrophysiologic study, significantly more young adults with atrial flutter died when medication control was not achieved compared with those whose atrial flutter was pharmacologically controlled. Surgical intervention to improve hemodynamic function of the heart has been shown to significantly reduce the incidence of atrial flutter and this should be considered when possible [2]. Atrial fibrillation in infants and children is very rare. Atrial frequency is usually increased, usually between 350-400 per minute. Most newborns with atrial fibrillation have a structurally normal heart. If there is an underlying heart pathology, it is usually a condition with right atrial enlargement, such as Ebstein's anomaly of the tricuspid valve or tricuspid valve atresia.

Ventricular arrhythmias

Ventricular extrasystole (VE) is the most common ventricular conduction abnormality seen in newborns. Up to 33% of newborns have EEs in the first week of life, and they usually resolve by two weeks of age. However, if they persist beyond this period, further evaluation is necessary to rule out an underlying structural heart defect. Ventricular arrhythmias in childhood are rare and may be benign or malignant. Benign VT is a condition diagnosed by exclusion, and these children usually have normal ECG, chest radiograph, and echocardiography and, in some cases, normal EP study findings when performed. The VT is suppressed by exercise and is usually unresponsive to drug treatment with a good long-term prognosis. Intractable idiopathic infantile VT presents in the first two years of life predominantly in males and is considered secondary to left ventricular hamartoma. Idiopathic LV tachycardia is rare and arises from the posterior bundle of the left bundle branch of Hiss and is thought to originate from the Purkinje network. It responds to verapamil treatment and is also amenable to radiofrequency ablation. Catecholaminergic VT, first described by Kumel, is induced by emotion or exertion and may lead to torsades associated with syncope. The resting ECG is normal. Administration of isoprenaline or stress tests may reproduce the GI. This is a dangerous form of VT and the prognosis is significantly improved with beta-blocker therapy [12,15].

Prolonged QT interval syndrome (PQT) is a familial disorder characterized by prolonged and abnormal ventricular repolarization and risk of life-threatening ventricular arrhythmias. Recent molecular genetic studies in families with SUIQT have identified aberrations in genes encoding cardiac ion channels. Dysfunction of these cardiac ion channels leads to action potential prolongation and inhomogeneity, resulting in arrhythmias based on abnormal impulse propagation, and increases the sensitivity of the heart to the arrhythmogenic action of catecholamines. At least 5 genes located on chromosomes 11, 7, 3, 4, and

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21 have been identified with aberrations of cardiac ion channel coding in families with SUIQT. Romano-Ward syndrome, an autosomal dominant SUIQT is subdivided into LQT 1, which is the most common form of SUIQT and in which the mutation is in the KVLQT1 gene on chromosome 11, LQT 2 in HERG on chromosome 7, LQT 3 in SCN5A on chromosome 3, LQT 4 on chromosome 4, and LQT 5 with a Min K mutation on chromosome 21. Jervell-Lange-Nielsen syndrome, an autosomal recessive LQTS, is caused by a KVLQT1 mutation on chromosome 11 and is associated with deafness. The main diagnostic criterion was QT interval prolongation on ECG, but it should be noted that patients with genetically confirmed LQTS may have a normal QT interval. Other diagnostic criteria include slow heart rate for age, alternation of the T plaque, and abnormal T plaque morphology. Patients commonly present with syncope or cardiac arrest caused by emotional or physical stress. Syncope is usually due to "pirouette tachycardia," often evolving into ventricular fibrillation. Newborns with SUIQT can be identified by partial AV blockade and heart rate slowing due to prolonged repolarization. A history of syncope, virulent family history, T-tooth alternation, QTc interval greater than 0.54 s, and associated AV blockade in newborns carry a higher risk. Mortality in untreated symptomatic patients is as high as 70% within 15 years of the first syncopal episode. Treatment options include beta-blockade, other antiarrhythmic drugs, stellate ganglionectomy, and automatic implantable cardiac defibrillator with marked improvement in survival [16].

Hypertrophic obstructive cardiomyopathy (HOCMP) is much less common in children than in adults. The Japanese screening program estimates the prevalence in children at 1 in 15,000 compared with an adult prevalence of 1 in 500. The overall risk of VSS in GOSM is low and is about one in a million in the population under 20 years of age. The mechanism of sudden cardiac death in HCMD is thought to be unrelated to arrhythmias, but ventricular fibrillation has been implicated in some cases [14].

Postoperative arrhythmias

Concomitant ectopic tachycardia (CET) is the most common (22%) arrhythmia observed in the immediate postoperative period after intracardiac plasty, mainly after correction of Fallot's tetrad. The etiology is thought to be due to resection of the right ventricular (RV) outflow tract muscle bundles, removal of RV obstruction through access to the right atrium, and increased bypass time. Risk factors predisposing to postoperative DET have been identified as lower age and body weight at the time of surgery, higher baseline Aristotle score, longer artificial circulation time and bypass time, use of deep hypothermia, and complete circulatory arrest [5,15]. The underlying mechanism is the increased automation of the HIS package. The ECG usually reveals tachycardia with narrow QRS complexes at 170-230 beats per minute, atrioventricular dissociation, with ventricular rate exceeding atrial rate. ETS usually responds to surface cooling to 34°C, atrial stimulation for AV synchrony, sedation and muscle relaxation to avoid stress, and intravenous amiodarone.

Late postoperative arrhythmias are the most common medical problem following correction of congenital heart disease. The incidence depends on the complexity of the underlying cardiac malformations, and the clinical significance depends on the interaction between arrhythmias and cardiac status. Arrhythmias occur for a variety of reasons.

Acquired due to preoperative natural history as in myocardial fibrosis, cyanotic congenital heart disease.

Up to 50% of adult patients undergoing surgery are found to have late unstable arrhythmias on ambulatory ECG monitoring. Occasionally, symptomatic sustained ventricular arrhythmias are recorded in these patients. EP studies have shown that these ventricular arrhythmias are due to reentry, which requires areas of slow conduction within the myocardium. Pulmonary valve regurgitation, often seen after recovery, which leads to chronic right ventricular overload and diastolic dysfunction, and has been shown to correlate with QRS prolongation on the ECG. Right ventricular QRS prolongation has also been shown to correlate with a higher incidence of symptomatic arrhythmias, and QRS duration greater than 180 ms on resting ECG is the most sensitive predictor of life-threatening ventricular arrhythmias [16].

Bradyarrhythmias

Congenital complete heart block (CPBB) is reported to occur at an incidence of 1 in 25,000 live births. If the condition occurs without an associated heart defect and is diagnosed at birth, the mortality rate is 15%. The risk of death is much lower, 3.5%, if the diagnosis is made after the first year of life. This study by Michaelsson and Engle reports that the risk of death is highest in early childhood, with half of all deaths occurring early in the first year of life. The overall survival rate in this group was 92%. However, the group with CCHD and associated structural heart disease had a higher mortality rate of 42.4% in those diagnosed at birth [17]. Another prospective study of 102 patients with CCHD aged 16 to 66 years by Michaelsson and colleagues showed that 36.2% either suffered symptoms of syncope or died, and of 40 patients followed for 30 years or more, only 4 (10%) remained symptom-free and without a pacemaker [18,20]. The risk of sudden death seems small in childhood but persists throughout adulthood. A low ventricular rate associated with a high atrial rate and a large heart on chest radiograph may be an indicator that children are at risk for death from heart

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failure. A prolonged QT interval has been consistently shown to be associated with death.

Conclusion. Dewey and colleagues suggested that an average daytime heart rate of less than 50 beats per minute, especially when combined with transient output block, is a risk factor for death. Concomitant tachycardia or flat response were indications of poor prognosis

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