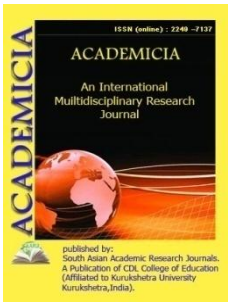
ACADEMICIA

ISSN: 2249-7137 Vol. 11, Issue 3, March 2021 Impact Factor: SJIF 2021 = 7.492

ACADEMICIA
An International Multidisciplinary Research Journal
(Double Blind Refereed & Peer Reviewed Journal)



DOI: 10.5958/2249-7137.2021.00802.8

CLINICAL SIGNIFICANCE OF ATRIOVENTRICULAR CONDUCTION DISORDERS IN CHILDREN

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ABSTRACT

Violation of atrioventricular conduction is a pathological condition characterized by changes in the frequency, regularity of the heart rhythm, with impaired communication and sequence between the activation of the atria and ventricles, which is benign or with pronounced changes in hemodynamics. Heart rhythm and conduction disorders in children are currently one of the most socially significant problems. So, according to statistics, a systole in patients with bradyarrhythmias is the cause of unexpected circulatory arrest in 15-20% of cases, and in patients with acquired high-grade autoventricular block, it is one of the main causes of death.

KEYWORDS: *Cardiac Arrhythmias, Cardiac Conduction Disturbances, Atrioventricular Block, Children, Cardiomyopathy, Myocarditis, Electrocardiography, Echocardiography.*

INTRODDUCTION

Atrioventricular block or atrioventricular block denotes a violation of the conduction of excitation from the atria to the ventricles. Depending on the degree of damage to the atrioventricular junction, incomplete and complete atrioventricular blocks are distinguished [1,2, 7, 10].

Cardiac arrhythmias occur in children of all ages. Often the causes of rhythm and conduction disturbances are not only cardiac (myocarditis, cardiomyopathies, pericarditis, primary pathology of the cardiac conduction system), but also extracardiac (diseases of the kidneys, endocrine system organs, impaired autonomic regulation of the heart rate, psychopathy), including electrolyte disturbances, exposure to physical and climatic factors [2, 6, 7, 9].

The largest number of cardiovascular diseases are polygenic. Monogenic diseases associated with heart damage include a number of syndromes and diseases accompanied by malignant

cardiac arrhythmias and a high risk of sudden cardiac death. The cause of hereditary heart rhythm disturbances is considered to be anomalies of the following main classes of proteins: contractile and cytoskeletal; ion channels and intercellular contacts; transmembrane carriers, as well as their modulators [3, 5, 10, 12].

Various etiological factors, such as ischemic heart disease, myocarditis and cardiomyopathies, the use of drugs, endocrine or electrolyte disturbances, surgical and endovascular interventions, lead to the occurrence of cardiac conduction disorders. Often, conduction disturbances arise "primarily", in the absence of obvious etiological factors, and in some cases are congenital[3, 4,8, 15].

The most common congenital conduction disorders include grade I atrioventricular block, complete and incomplete right bundle branch block, and blockade of the anterior and posterior branches of the left bundle branch (cases of congenital left bundle branch block have not been described). In most cases, conduction disturbances are isolated, do not have clinical manifestations and are detected when an electrocardiogram is recorded during routine medical examinations. These forms of conduction disorders are characterized by a favorable prognosis; they usually do not progress and do not require treatment [13, 21].

Violation of atrioventricular conduction can be permanent or transient. In some cases, manifestations of the disease can be detected already at the stages of intrauterine development with auscultation of fetal heart sounds or planned ultrasound examination, as a rule, starting from the 18th week of pregnancy [17, 18, 24].

The most common cause, accounting for 60-90% of cases of congenital complete AV block, is intrauterine systemic lupus erythematosus, which occurs with autoimmune diseases of the mother. Penetrating through the placenta, these antibodies bind to the antigens of the cells of the atrioventricular node of the fetus, disrupting the functioning of their ion channels, activate apoptosis and potentiate the development of the inflammatory process, followed by the replacement of dead cells with connective tissue[3, 13,15, 26].

In addition to the violation of atrioventricular and intraventricular conduction in carriers of these mutations, sinus node dysfunction, sinoatrial blockade of various degrees and the syndrome of "silent" atrium (atrial standstill - complete absence of electrical activity and, accordingly, contractions of the atrial myocardium) are described. In rare cases, congenital sinus node dysfunction is the only clinical manifestation of the disease; atrioventricular and intraventricular conduction disorders may be completely absent or occur later [15, 18, 23, 25].

Violation of conductivity at various levels of localization and severity is often found in congenital structural heart disease. They can be caused both by a primary disorder in the development of the cardiac conduction system, and be the result of morphological and hemodynamic changes accompanying the congenital defect. Transcription factors are genes expressed in the early stages of embryogenesis, whose protein products determine the direction of further cell proliferation and differentiation. Mutations in these genes can disrupt the formation of entire organs and its individual components. It has been established that the proliferation and correct differentiation of cells of the cardiac conduction system are regulated by genes belonging to the Homeodomain and T-box families [2, 16, 20].

Progressive cardiac conduction disorders are characteristic of a number of neuromuscular diseases, being the main cause of sudden death and one of the leading causes of overall mortality in these patients[3, 11,19, 22].

With a number of hereditary diseases (the so-called laminopathies), in which heart damage is manifested by dilated cardiomyopathy, ventricular and arrhythmias and progressive conduction disorders, which can occur before the development of violations of myocardial contractility and the appearance of arrhythmias. A morphological study of patients who died sudden death at a young age revealed replacement of the atrioventricular node and the His – Purkinje system with adipose and connective tissues [13, 15, 24, 27].

Conduction disorders (high-grade atrioventricular block and intraventricular conduction disorders) are found in myotonic dystrophy. This disease with an autosomal dominant type of inheritance is characterized by damage to the muscular system (muscle weakness is caused by impaired relaxation of muscle fibers with their intact contractility), eyes (development and rapid progression of cataracts) and the heart (progression of heart failure, cardiac conduction disorders, fascicular ventricular tachycardia, sudden death). A pathomorphological examination of the hearts of patients with myotonic dystrophy who died suddenly showed that the sinoatrial and atrioventricular nodes were replaced by fibrous and adipose tissues [5, 10, 15, 23].

Since congenital atrioventricular block may be caused by genetic disorders, in the absence of a diagnosed systemic disease in the patient's mother, it is advisable to examine close relatives of the patient for the timely detection of possible conduction disturbances in them, even in the absence of clinical manifestations characteristic of the disease [8, 14, 17].

It is impossible to understand the electrophysiological processes occurring in the heart without understanding the anatomical and physiological characteristics of the conducting system[3, 5, 10].

Literature data indicate that the presence of focal myocardial fibrosis is a prerequisite for the occurrence of ventricular arrhythmias. At the same time, the activation of autoimmune mechanisms can serve as an additional triggering factor that activates the arrhythmogenic zone. Left bundle branch block is an electrocardiographic phenomenon caused by disturbance or complete cessation of the conduction of excitation along the left bundle branch[1, 9,16, 22].

A fairly large number of neuromuscular diseases, most often myodystrophies, and mitochondrial diseases (cytopathies) are accompanied by damage to the cardiovascular system, the most characteristic of which are dilated cardiomyopathy, myocardial hypertrophy, various forms of ventricular and supraventricular arrhythmias, as well as progressive conduction disorders [17, 25].

The term "progressive lesion of the cardiac conduction system" unites a group of diseases that have significant etiological and pathogenetic differences, common to which are hereditary character, progressive course, poor prognosis and potential threat to the patient's life in the absence of timely implantation[5,11, 14].

In most cases, the blockade is due to the replacement of the atrioventricular node with fibrous tissue or impaired conduction of the impulse from the atrial myocardium to the atrioventricular node [12, 22].

The most typical development is dilated cardiomyopathy with the occurrence of life-threatening ventricular arrhythmias, less often there is a progressive lesion of various parts of the cardiac conduction system, which is manifested by sinus bradycardia, atrioventricular blockade of various degrees, as well as blockade of the legs and branches of the His bundle[13, 25].

Violation of the formation and conduction of excitation impulses from the atria to the ventricles of the heart leads to significant changes in hemodynamics. The most significant changes are due to complete atrioventricular block. As a result, such compensatory mechanisms are activated as: lengthening diastole, increasing filling of the ventricles, increasing the rate of circular filling of myofibrils. Remember that arrhythmias can cause the appearance of symptoms of heart failure, the constant existence of heart rhythm pathology can aggravate its course [5, 7].

The degree of conduction disturbances determines the severity of the course and the prognosis of patients. The most characteristic is transient or permanent complete atrioventricular block with periods of prolonged asystole and syncope, which is the most common cause of death in patients. A progressive course of generalized lesion of the vascular system has been described, which begins with a distal bundle branch block, then spreading to the atrioventricular and sinus nodes, which leads to the development of complete atrioventricular block and the onset of sick sinus syndrome [9, 11].

It has now been proven that in children, atrioventricular blockade can persist for a long time, progress, having a negative effect on hemodynamics, leading to a delay in the pace of physical, psychomotor and intellectual development of the child, and significantly reduce the quality of life indicators.[18, 23].

The clinical manifestations of congenital complete atrioventricular blockade and the prognosis in patients are determined by the level of blockade, which, in turn, determines the localization of the substitute pacemaker and, accordingly, the frequency of its automatic activity. If the driver of the replacement rhythm is localized in the atrioventricular junction (below the conduction block) or in the proximal parts of the His – Purkinje system (common trunk of the His bundle) and QRS complexes have normal morphology, clinical manifestations of the disease are often absent. The prognosis in such patients is significantly better than in patients with acquired complete atrioventricular block [19, 20].

Patients with grade II atrioventricular block may be asymptomatic and prone to the development of conditions such as dizziness and fainting, which are more common in patients with grade III atrioventricular block. Complete atrioventricular block can be congenital, acquired, or hereditary. The most common causes of acquired grade III atrioventricular block are cardiac surgery and viral infection. Children with complete atrioventricular block, regardless of the genesis of the block, are threatened by the development of syncope and, consequently, by the development of sudden cardiac death due to life-threatening brady- and tachyarrhythmias, leading to the development of inadequate cardiac output and impaired cerebral circulation [9, 11].

The change in atrioventricular conduction may be in direct proportion to the rhythmic activity of the main pacemaker. The appearance of atrioventricular block of the 1st degree against the background of bradycardia may be associated with an increase in the parasympathetic division of the autonomic nervous system and clinically may manifest as vegetative-vascular dystonia of the vagotonic type. Tachy-dependent atrioventricular blockade of the 1st degree, which occurs with

an increase in the heart rate, is obviously associated with a blockade of conduction along the fast (β) channel of the atrioventricular node. This blockage may persist in the tilt position of the patient, but may resolve after subcutaneous administration of atropine. Such a response gives reason to believe that with tachydependent atrioventricular blockade of the 1st degree, the nature of the influence of autonomic regulation of atrioventricular conduction is not the main one. The reasons for the development of I degree atrioventricular block are idiopathic degenerative diseases of the conducting system (Lev's disease, Lenegra's disease); systemic lesions of the connective tissue (juvenile rheumatoid arthritis, systemic lupus erythematosus, systemic scleroderma, etc.); infectious diseases (infective endocarditis, diphtheria, acute rheumatic fever, Lyme disease, tuberculosis, etc.); infiltrative diseases (amyloidosis, sarcoidosis, etc.). Grade I atrioventricular block can also occur after undergoing cardiac surgery or as a result of catheterization of the right heart. Atrioventricular block II degree is observed in children after heart surgery; sometimes atrioventricular block of both I and II degrees can be a consequence of congenital heart disease. It should be borne in mind that atrioventricular blockade of the I degree, II degree of the 2nd type and variants of high degree blockade often transform into a complete atrioventricular block and occur in children with severe cardiac pathology [5, 18].

Diagnosis of the disease is based on the detection of complete atrioventricular blockade during ECG recording and can present a certain difficulty with a sufficiently high frequency of the replacement rhythm and normal QRS morphology (with the localization of the source of automatism in the atrioventricular node). Since conduction disturbances can be transient, long-term ECG recording using stationary monitoring stations, Holter monitors, and in some cases implantable recorders plays an important role. Currently, there are no clinical guidelines for molecular genetic studies in patients with congenital complete atrioventricular block due to the relative rarity of detecting mutations in patients of this category [8, 20].

Conduction disturbances are usually localized distally at the level of the His-Purkinje system. When monitoring the ECG of patients in dynamics, it is characteristic to detect a gradual increase in the duration of the PQ and QRS intervals[15,27].

The use of continuous cardiac stimulation systems in patients with complete atrioventricular block can reduce the manifestations of heart failure and improve the quality of life [14, 26].

The lack of diagnostics of the nature of the lesion of the myocardial conduction system in infectious cardiomyopathy in children in the form of rhythm disturbances, myocardial conduction, repolarization of the ventricles of the heart leads to inadequate and untimely treatment, and as a consequence - the development of chronic forms of the disease, an increase in morbidity and mortality in older age groups [20, 25].

CONCLUSION

Thus, as can be seen from the presented literature data, the problems of rhythm and conduction disturbances in pediatric practice are very relevant and the study of the peculiarities of the course of rhythm and conduction disturbances in children is a poorly studied problem. Under these conditions, to develop prognostic criteria for the course of rhythm and conduction disturbances in children, research aimed at solving this problem is needed.

REFERENCES

1. Adeniran, Ismail. Modelling the Short QT Syndrome Gene Mutations: And Their Role in Cardiac Arrhythmogenesis. Springer, 2014.

2. Ebrahim, Mohammad A., et al. "Outcomes of pacemaker implantation in isolated congenital atrioventricular block." Progress in Pediatric Cardiology 60 (2021): 101231.

3. Eliasson, Håkan, et al. "Pacing therapy in children with isolated complete atrioventricular block: a retrospective study of pacing system survival and pacing-related complications in a national cohort." EP Europace 21.11 (2019): 1717-1724.

4. Escobar-Diaz, Maria C., et al. "Perinatal outcome in fetuses with heterotaxy syndrome and atrioventricular block or bradycardia." Pediatric cardiology 35.6 (2014): 906-913.

5. Gao, Lu, et al. "A case report of one vasovagal syncope patient with third-degree atrioventricular block caused by SCN5A gene mutation and literature review." BMC pediatrics 20 (2020): 1-5.

6. God, Bethany, Matthew Needleman, and Joseph May. "An Unusual Case of Reversible Heart Failure with Apparent Mobitz Ii Atrioventricular Block in a Teenage Male." (2018): 741-741.

7. Hansahiranwadee, Wirada. "Diagnosis and Management of Fetal Autoimmune Atrioventricular Block." International Journal of Women's Health 12 (2020): 633.

8. Kao, Ting-Wei, and Gi-Beom Kim. "A child with spontaneous recovery of normal sinus rhythm from idiopathic complete atrioventricular block." Cardiology in the Young (2020): 1-3.

9. Karnabi, Eddy, et al. "Congenital heart block: identification of autoantibody binding site on the extracellular loop (domain I, S5–S6) of α1D L-type Ca channel." Journal of autoimmunity 34.2 (2010): 80-86.

10. Khuraskina, NadezhdaVladislavovna, Lyudmila AntonovnaAleksandrova, and Lilia FakatievnaChemerova. "Age dynamics of heart rate regulation in students." Bulletin of the Chuvash State Pedagogical University. IYaYakovleva 4 (2010).

11. kiziYakubova, KamolaNigman, and AkidaValievnaMuratkhodjaeva. "Character of the current and risk factors of development of disorder of the heart rhythm in children." Chief Editor.

12. Kruchina, Tatiana Kimovna, et al. "Conductive system of the heart in children: structural features and role in the formation of cardiac arrhythmias." Russian Bulletin of Perinatology and Pediatrics 56.6 (2011).

13. Leshchinsky, Sergei Petrovich. "Genetically determined diseases associated with an increased risk of sudden cardiac death: issues of etiology, diagnosis and treatment." Sechenov Bulletin 4 (18) (2014).

14. Llanos, Carolina, et al. "Recurrence rates of cardiac manifestations associated with neonatal lupus and maternal/fetal risk factors." Arthritis & Rheumatism: Official Journal of the American College of Rheumatology 60.10 (2009): 3091-3097.

15. Mollerach, F. B., et al. "Causes of fetal third-degree atrioventricular block and use of hydroxychloroquine in pregnant women with Ro/La antibodies." Clinical rheumatology 38.8 (2019): 2211-2217.

16. Molokanova, YuliaPavlovna. "Factors determining the rhythm of heart contractions in adolescents." Bulletin of the Moscow State Regional University. Series: Natural Sciences 2 (2013): 63-69.

17. Salomonsson, Stina, et al. "A population- based investigation of the autoantibody profile in mothers of children with atrioventricular block." Scandinavian journal of immunology 74.5 (2011): 511-517.

18. Sauer, Andrew J., et al. "Long QT syndrome in adults." Journal of the American College of Cardiology 49.3 (2007): 329-337.

19. Senior, Roxy, et al. "Contrast echocardiography: evidence-based recommendations by European Association of Echocardiography." European Journal of Echocardiography 10.2 (2009): 194-212.

20. Shkolnikova, M. A., L. A. Kravtsova, and V. V. Bereznitskaya. "Tachycardia in children of the first year of life." Pediatrics. Journal named after GN Speransky 91.3 (2012).

21. Smits, Jeroen PP, Marieke W. Veldkamp, and Arthur AM Wilde. "Mechanisms of inherited cardiac conduction disease." EP Europace 7.2 (2005): 122-137.

22. Turner, Christian James, and Christopher Wren. "The epidemiology of arrhythmia in infants: A population- based study." *Journal of paediatrics and child health* 49.4 (2013): 278-281.

23. Velasquez Rodriguez, J., et al. "First-degree atrioventricular block in children and adolescent athletes." European Heart Journal 41.Supplement_2 (2020): ehaa946-3133.

24. Vrdoljak, Vinko, et al. "Third Degree Atrioventricular Block in Children." RadHrvatskeakademijeznanosti i umjetnosti. Medicinskeznanosti 540= 48-49 (2019): 67-70.

25. Yakubova, KamolaNigmanKizi, and AkidaValievnaMuratkhodjaeva. "Ethiopathogenetic Value Of Vitamin D During Heart Rhythm Disorders In Children." The American Journal of Medical Sciences and Pharmaceutical Research 2.09 (2020): 27-31.

26. Yakubova, KamolaNigmonKesey. "Medical and social factors in the development of atrioventricular blockade in children with cardiomyopathies." Science and Education Issues 25 (74) (2019).

27. Zhang, Lei, et al. "Dynamic electrocardiography is useful in the diagnosis of persistent atrial fibrillation accompanied with second-degree atrioventricular block." Acta Cardiologica Sinica 34.5 (2018): 409.