

Clinic of Acute Rheumatic Fever in Children Transmission Characteristics

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Abstract: In this article Correct information is given about the specifics of the clinical course of acute rheumatic fever in young children. Acute rheumatic fever (rheumatism) is a systemic inflammatory disease of the connective tissue, accompanied by damage to the cardiovascular system.

Keywords: Rheumatism, rheumatism, streptococcus, connective tissue, antistreptolysin-O, antistreptohyaluronidase, antistreptokinase.

Acute rheumatic fever (rheumatism) is a systemic inflammatory disease of the connective tissue, accompanied by damage to the cardiovascular system. The etiology is associated with B-hemolytic streptococcus of group A. Rheumatism occurs mainly (80%) in childhood (7-15 years), in 30% of cases it is familial, in 1% in school-age children, rarely in children under 2 years of age. The main cause of acquired heart defects in children is rheumatism. As a result of improved housing conditions, the organization of systematic specialized care and comprehensive prevention of rheumatism, the incidence of rheumatism decreased to 0.18% per 1000 children [1].

The incidence in Ukraine is 4.4 per 100,000 population, the primary incidence in Russia is 2.7 per 100,000 population, the detection rate of primary chronic heart disease is 9.7 per 100,000 population [1,2].

It is noted that the onset of the disease is associated with a period of exacerbation of streptococcal infection, chronic tonsillitis, nasopharyngitis, sinusitis, and otitis media. In places with high population density (city, among schoolchildren), transmission of infection from one child to another is also of great importance. Various diseases of streptococcal nature (scarlet fever, glomerulonephritis, rheumatism, catarrh of the upper respiratory tract) occur in school-age children in 10-20% of cases. The spreaders of the infection are mainly sick people, from whom it can be transmitted to the environment [3]. The contagiousness of this environmental infection persists for 24-48 hours. Carriers of virulent strains of group A streptococci are also important in the spread of infection. They are stable in the external environment and, under certain conditions, transform into the L-form (the non-gypsum form of streptococcus). Infected people may be asymptomatic (20-24%) or develop rheumatism [4].

Currently, a working classification and names of the rheumatic process have been developed that reflect the course of the rheumatic process, clinical and anatomical features, damage to organs and groups of organs, the nature of the course and performance of the cardiovascular system. part "Working classification and names of rheumatism" The classification adopted in 1964 based on the information of A.I. Nesterov was not used at the symposium held on [5].

In the 21st century, there was a need to revise the above-mentioned classification and nomenclature. Currently, the more appropriate term is “rheumatic fever” (not rheumatism!). Because it turned out to be important to establish a connection with hemolytic group A streptococcal infection and use antibacterial drugs to eliminate it and carry out preventive measures for prevention. Based on clinical experience, the term “primary rheumatic carditis” has led to the expansion of this concept. Because the causes of mitral valve prolapse have been confirmed from the appearance of hypermobility syndrome to rheumatic heart disease. Therefore, it was considered appropriate to use the classification recommended by the New York Heart Association in 2003[11].

Examples of clinical diagnosis

1	Acute rheumatic fever: carditis (mitral valvulitis), volatile (migratory) polyarthritis, CAB I (FC I)	101.1
2.	Acute rheumatic fever: chorea, QAB O, (FK O)	102.9
3.	Recurrent rheumatic fever: carditis, combined mitral heart disease. KAB II (FC II)	101.9
4.	Chronic rheumatic heart disease: post-inflammatory fibrosis of the mitral valve leaflets QAB O (FK O)	105.9
5.	Chronic rheumatic heart disease: mitral-aortic combined heart disease QAB II B (FK III)	108.0

“Recovery of the disease” is characterized by the complete return of clinical symptoms of acute rheumatic fever, normalization of laboratory parameters and the absence of traces of residual complications. Chronic rheumatic heart disease involves the formation of stenosis or platelet insufficiency caused by inflammation of the heart valve apparatus. The degree of heart failure is assessed based on the stages recommended by Straesko-Vasilenko (level) and the New York Heart Association (functional class) [6].

In the typical course of the disease, it appears 1-2-4 weeks after the disease with tonsillitis, scarlet fever, nasopharyngitis, mainly in schoolchildren and adolescents. On the one hand, there is evidence that anxiety and depressive disorders anticipate gastrointestinal complaints. Perhaps, in these individuals, psychological characteristics increase attention to gastrointestinal complaints and activate the pain mechanism [7]. Then the disease enters a “latent” period (lasting from 1 week to 3 weeks), characterized by asymptomatic or mild progression, weakness, arthralgia, and sometimes low-grade fever. Diffuse damage to connective tissue demonstrates a polymorphism of the clinical picture. Typical manifestations of the disease are carditis, polyarthritis, chorea. The disease can occur acutely, subacutely or latently; in such cases, the diagnosis is made on the basis of carditis and heart failure. The first sign of the disease in children is increased body temperature, weakness, and joint pain. When examining such patients, they are found to have anemia and a number of changes in the blood: leukocytosis, increased erythrocyte sedimentation rate (ESR) [6,8]. Along with this, the titers of ASLO (antistreptolysin O), antistreptokinase and antistreptohyaluronidase increase. In the second period of the disease, carditis, polyarthritis and other laboratory parameters change in the clinical picture. Sometimes rheumatism begins with chorea. Symptoms vary depending on the level of activity and course of rheumatic fever [8].

Primary rheumatic carditis is a heart disease that reflects the severity of the disease. One of the early manifestations in children is weakness and increased body temperature. There will not be many complaints about pain and discomfort in the heart area. Depending on the damage to the myocardium, endocardium and pericardium, objective symptoms appear. Cardiac pathology in rheumatism in children is unique and the most common is myocarditis (100%). Tachycardia or

bradycardia is objectively detected; in some cases, the number of heartbeats remains normal. In 85% of patients, expansion of the borders of the heart to the left is detected clinically and radiologically; in all patients, a systolic murmur is heard at the apex of the heart. With myocarditis, exudative inflammation with diffuse changes in the myocardial interstitium is observed. The general condition of the patient is serious, pale skin, shortness of breath, cyanosis, low pulse, decreased blood pressure, expansion of the borders of the heart, decreased heart sounds, signs of heart failure. The ECG reveals a disturbance of the monotopic rhythm, deterioration of ventricular conduction, changes in the bioelectrical process in the myocardium (decrease and change in the T wave, downward displacement of the ST segment, lengthening of the electrical systole) [9].

A decrease in the amplitude of the 1st tone on the ECG, its expansion and deformation: the appearance of pathological 3rd and 4th sounds, a systolic murmur of a muscular nature is added.

Endocarditis – often combined with myocarditis and occurs in 50-55% of children. Symptoms of mitral valve valvulitis can be detected from the first days of the disease. The main clinical sign is a “blowing” systolic murmur in the area of the mitral valve projection (5 points). The intensity of the noise increases with time or when lying on the left side, during physical activity. The noise is often best heard in the armpit area. The ECG records a pansystolic or protosystolic murmur of medium or low amplitude of medium or high frequency at the apex of the heart.

ECG: enlargement of the left border of the heart, mitral configuration of the heart. In 10% of patients, the aortic valve is damaged, and a diastolic murmur associated with diastolic pressure is heard along the left sternal border. At the same time, high-frequency protodiastolic noise is recorded in the FCG.

Echocardiography reveals dilatation of the mitral valve, “looseness” of echo signals due to changes in layers, signs of mitral and aortic regurgitation, signs of dilatation of the left chambers of the heart. Pericarditis occurs together with endomyocarditis and is considered part of rheumatic polyserositis. Clinical diagnosis in 1-1.5% of cases, radiological in 40% of cases, complex instrumental examination in combination with echocardiography, and this percentage is much higher [9, 10].

According to the nature of inflammation, pericarditis can be dry, fibrinous and exudative. With fibrinous pericarditis, pain is observed, and an intermittent friction noise of the pericardium is heard along the left half of the chest (the rustling of silk, similar to the rustling of snow).

Serial ECG shows exacerbation of the -R wave, a typical dynamic change of the T wave with the QT interval. Echocardiogram shows thickening and separation of the pericardium and epicardium [2,10]. Serous-fibrinous pericarditis worsens the general condition of patients. The general condition of the patient suddenly worsens, the color turns pale, and when a large amount of exudate accumulates, the patient has a swollen face, swollen neck veins, shortness of breath, he sits in a forced position (half-sitting) on the bed. . Pain may occur in the back of the chest, and symptoms of hemodynamic disorders rapidly increase. These symptoms intensify when the patient is in a horizontal position. The pulse is accelerated, less full, blood pressure decreases, in some cases the branch of the heart swells, the cardiac impulse is not detected. The boundaries of the heart are expanded, the sounds are reduced. The ECG shows that the electrical activity of the heart (myocardium) has decreased. On the radiograph, along with the expansion of the borders of the heart, the pulsation is of small amplitude and the contour of the heart is narrowed. On an, the shadow of the heart has a spherical or trapezoidal shape.

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