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# Acute Rheumatic Fever in Children

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**Abstract**

Acute rheumatic fever (ARF) is a non-purulent complication that appears 2-4 weeks after streptococcal tonsillopharyngitis. acute infectious disease. This article provides information about acute rheumatic fever in children.

**Key words:** Rheumatism, cardiovascular, streptococcus, viral etiology, valocordin, corvalol.

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## Introduction

Rheumatism (acute rheumatic fever) is a systemic inflammatory damage of connective tissue in the cardiovascular and musculoskeletal system. Rheumatism can affect any organs and systems: brain, kidney, liver, etc. The first attack of rheumatism mainly occurs in childhood (7-15 years) and adolescence. Rheumatism occupies an important place in the general structure of morbidity and mortality from cardiovascular diseases. In many developing countries, rheumatic heart disease accounts for about half of the cases of cardiovascular disease. The incidence of rheumatism in economically developed countries is at least 5 cases per 100,000 population, and in less developed countries - 27-100 cases per 100,000 population. Etiology and pathogenesis. The results of epidemiological, clinical observations, microbiological, immunological and experimental studies indicate the connection of streptococcal nasopharyngeal infection (group A P-hemolytic streptococci) with rheumatism. Streptococcal etiology of rheumatism is: streptococcal infection before an attack of rheumatism (angina, pharyngitis); β-hemolytic streptococcus group A exacerbation of rheumatism during ORK; increased titer of streptococcal antibodies (ASL, ASG, ASK); a sharp decrease in the incidence of rheumatism and its relapses after streptococcal prophylaxis and active treatment of streptococcal infection foci. The streptococcal etiology of rheumatism is typical for its "classic" forms, which are acute and involve the addition of ligaments to the process. There is almost no connection between relapsing and continuous forms of rheumatism and streptococcus. In the last forms of transient rheumatism, streptococcal antigens are not increased, bicillin prophylaxis of rheumatism relapses is ineffective. Therefore, the role of streptococcal infection in the latent, relapsing course of rheumatism is excluded. Allergic, infectious-toxic or viral etiology is assumed in these forms of rheumatism. An important role in the development of the disease is played by the individual sensitivity of the organism to streptococcal infection, which is associated with determined changes in humoral and cellular immunity. Hereditary predisposition to the disease is emphasized. There is a concept of "rheumatic families", in which the incidence is 2-3 times higher than in healthy ones. The genetic spread of this disease has been determined - polygenic. The pathogenesis of rheumatism is complex and often unclear. more orin is given to allergies in the development of li. This is confirmed by the similarity of the clinical and anatomical features of rheumatism with serum sickness, experimental allergy, the recurrence of the disease as a result of a number of non-specific effects, and the antirheumatic effect of anti-allergic drugs.

Later, autoallergic mechanisms are added to the process. Sensitizing agents (streptococci and their decomposition products, viruses, non-specific allergens) can cause allergic inflammation in the heart, then its antihistamine component causes a change in bee and their transition to autoantigens, and an autoimmune process occurs. Streptococcus produces many cells and extracellular substances, which have enzymatic and toxic properties (streptolysins, streptococcal proteinase, deoxyribonuclease, mucopeptides - "endotoxins", etc.). All of them have a pronounced cardiotoxic effect. Cell membrane M-protein, streptolysin S and O are of great importance in the pathogenesis of rheumatism; is the effect of mucopeptides. The autoimmune hypothesis of the pathogenesis of rheumatism is explained by the immunological connection of streptococcal group A antigens and myocardial tissue components. In most cases of rheumatism, antigens reacting with heart tissue are detected, including antibodies reacting with both myocardial antigens and streptococcal membrane antigens. As evidence of the development of autoimmune processes in rheumatism, the violation of humoral and cellular immunity against antigenic components of connective tissue - structural glycoproteins, proteoglycans, water-soluble components of connective tissue is considered. In rheumatism, there is an imbalance of blood immunocomponent cells; an increase in the

amount of total lymphocytes; shift in the level of circulating immune complexes, immunoglobulins. In rheumatism, periodic changes of the morphological connective tissue occur: mucoid swelling, fibrinoid changes, necrosis, cellular reactions (infiltration with lymphocytes and plasma cells, formation of Ashoffa-Talalaev granulomas), sclerosis. The rheumatic process usually ends with sclerosing. It can be said that the process will be limited or restored only during mucoid sputum. Changes in the connective tissue that are formed during the relapsing course of rheumatism are rarely localized in the place of sclerosis, new connective tissue areas are added to the process. The clinical picture of rheumatism is very diverse, first of all, it depends on the severity of the process, exudative, proliferative events, organ and system damage, the time of seeking medical help and previous treatment. In typical cases, the first attack of rheumatism begins 1-2 weeks after a previous acute or chronic streptococcal infection (angina, pharyngitis, ORK). Recurrences of rheumatism begin during or after intercurrent diseases, operative interventions, mental and physical stress can also cause the onset of rheumatism.

Differential diagnosis. It is often necessary to diagnose rheumatism with tonsillogenic heart damage, non-rheumatic carditis, infectious endocarditis, cardiac neurosis, RA, thyrotoxicosis, chronic tuberculosis intoxication, reactive arthritis. Tonsillogenic pathology of the myocardium manifests itself in the form of myocarditis or cardiomyopathy. Tonsillogenic myocarditis usually develops during or after angina. Patients often complain of asthenoneurotic nature. Disturbing pains in the area of the heart, insignificant displacement of the left border of the heart, soft systolic noise detected by auscultation and FKG are detected. These signs appear in conditions of relatively calm laboratory parameters (ECHT, proteinogram, level of neuraminic acids, etc.). If these indicators increase, they will quickly normalize as a result of treatment. There is always a pronounced pathology in the tonsils. Tonsillogenic (functional) cardiomyopathy is observed in chronic tonsillitis. Patients complain of throbbing, throbbing, stabbing pains in the heart area for a long time, which intensify during tonsillitis flare-ups, do not go away when taking coronary drugs, and only after taking sedative drugs (valocordin, corvalol, Zelenin drops, etc.) fades or disappears. Functional data are the same as in tonsillogenic myocarditis, but they are determined without any changes for several months or years. Polyarthralgic syndrome is often observed. In the absence of chronic tonsillitis, laboratory parameters are normal. Prolonged subfebrile can be noted for several months. Tonsillogenic cardiomyopathy is aggravated in the presence of chronic (often decompensated) tonsillitis. Non-rheumatic carditis usually develops during or after a few days of infection (influenza, SARS, typhus, etc.). It is often formed during the flu. Patients complain of throbbing pains in the heart area. Shifting of the left border of the heart, systolic noise that does not spread at the V point, I tone binding, are often detected. As a result of the violation of repolarization processes, the T wave is smoothed, the S-T interval decreases. Extra table alars (supraventricular, ventricular), partial ventricular conduction disorders, blockade of Gis bundles, etc. you can be The laboratory indicators reflecting the destructive-inflammatory process have changed insignificantly. Non-rheumatic carditis is characterized by continuous remission and relapse in the absence of endocardial damage and heart defect formation. Infectious endocarditis is usually accompanied by high fever, chills, profuse sweating, tachycardia, hemorrhagic syndrome, enlarged spleen, anemia, positive test name o I. in long-term high ECHT. Most often, it develops in rheumatic or congenital heart defects, in rare cases it damages previously intact valves - always the aortic valve. Echocardiographic examination of the heart is of great importance in the diagnosis.

Treatment. Currently, the most proven treatment is a three-stage treatment: the first stage is a long (4-6 weeks) stationary treatment in the active phase; the second stage - the last sanatorium or sanatorium-resort treatment from the hospital; the third stage - dispensary observation in polyclinic conditions with bicillino-drug treatment. Rheumatism should be treated as early as possible (in the first hours or days - up

to 3 days after the onset of the disease), because at this stage, changes in the connective tissue of the heart and other organs (mucoid secretions) are reversible; it should be complex, adequate and strictly individual. In the active phase of rheumatism, the patient should be hospitalized. if this is not possible, the patient should observe bed rest at home. The treatment complex includes treatment-restrictive and movement procedures, rational nutrition, medication and physiotherapeutic means. the order of action should be increased according to the reduction of the rheumatic process. Food should be rich in variety, proteins, vitamins, phospholipids.

Carbohydrate food is limited. In case of circulatory disorders, food should be mainly dairy-vegetable, salt and liquid should be limited, the diet should consist of dairy-vegetable products.

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