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Juvenile Idiopathic Arthritis Joint-Visceral Type (System Type)

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Abstract: In this articlecharacteristics of the clinical course of juvenile idiopathic arthritis in childrenThe identity of the correct information is given. Juvenile idiopathic arthritis is a systemic disease of the inflammatory nature of the connective tissue in the joint-visceral type, which is accompanied by damage to the cardiovascular system.

Keywords: juvenile idiopathic arthritis, rheumatism, T-lymphocyte, immunity, connective tissue, Bekhterev's disease.

Juvenile idiopathic (rheumatoid) arthritis (YUIA)- is a chronic, severe, progressive disease of children and adolescents, with unclear etiology and complex, autoimmune pathogenesis, which is manifested by the gradual destruction of joints, disrupts the growth and development of the child, and has a negative impact on the quality of life [1] .

According to the latest data, the prevalence of YUIA in children under 14 years of age in Russia is 49.57 per 100 thousand children, and YUIA is 121.53 per 100 thousand adolescents aged 15-17 years. Girls get sick 2 times more often than boys.

The exact causes of this disease are unknown. The etiology of YUIA is multifactorial, including genetic and environmental factors such as infections. According to statistics, the incidence of rheumatoid arthritis in first-degree consanguineous relatives is higher than in the general population. Associations of YUIA with histocompatibility Ag (HLA) — A2, B27, B35 and HLA DR-5, DR-8 were determined. The most common environmental factors are viral or bacterial-viral infection, trauma, inhalation or hypothermia, psychological stress and even preventive vaccinations[2].

The basis of the disease is the activation of cellular and humoral immunity. A foreign or modified antigen is taken up and processed by macrophages or other antigen-presenting cells, which present it to T-lymphocytes, leading to T-lymphocyte activation and proliferation. Macrophages, activated T-lymphocytes, fibroblasts, and synoviocytes produce anti-inflammatory cytokines that cause a cascade of pathological changes with the development of progressive inflammation in the joint space and the systemic manifestation of the disease. The production of large numbers of autoantibodies indicates the involvement of B-cell communication of the immune system. Thus, uncontrolled reactions of the immune system lead to the development of chronic inflammation, irreversible changes in the joints, and the development of extra-articular manifestations[1,3].

YUIA can be considered a diagnosis of exclusion. It is defined as arthritis of unknown etiology, present for 6 weeks, under the age of 16 years, excluding other diseases.

Systemic arthritisis a documented fever for at least 2 weeks and arthritis accompanied by or preceded by two or more of the following: transient, volatile erythematous rash, serositis, generalized lymphadenopathy, hepatomegaly, splenomegaly[4].

Polyarthritis-RF is defined as negative disease affecting 5 or more joints during the first 6 months. RF-positive is defined when 5 or more joints are affected during the first 6 months of the disease, the presence of positive RF in two tests during 3 months;

The basis of YUIA is an articular syndrome manifested by arthritis. Arthritis is characterized by the following symptoms: swelling, hyperemia, increased local temperature, pain and joint dysfunction. It can affect any joint that has a synovial membrane. Knee, ankle and wrist joints are more commonly affected in children. One of the characteristic symptoms of YUIA is damage to the joints of the cervical spine. Another characteristic symptom of YUIA is the symptom of morning stiffness - morning stiffness in the joints that lasts from 10-15 minutes to several hours[5].

In the systemic form of YUIA, in addition to the joints, other internal organs can be affected and develop: myopericarditis, pleurisy, serous peritonitis, hepatosplenomegaly, lymphadenopathy. Most often, YUIA affects the eyes and acuteiridocyclite/ uveitis and chronic anterior iridocyclitis can occur in the form of corneal dystrophy with cataract complications.

A systematic option.There are two options: the allergoseptic option and the Still option. The allergoseptic variant is characterized by a sudden onset with high prolonged fever up to 39-40°C. A rash usually appears with or after 2-3 weeks of fever. It is maculopapular and located over the affected joints, usually polymorphic. The disease is accompanied by hepatosplenomegaly, lymphadenopathy, pericarditis or myocarditis. Affects knee and ankle joints[7].

Still option its main feature is participation in the process in addition to the knee, hip, ankle, cervical spine and temporomandibular joints.

Differential diagnosis. The diagnosis of YUIA is a diagnosis of exclusion and is made at the end. First, pathologies such as sepsis, infections (yersiniosis, toxoplasmosis, etc.), oncohematology, solid tumors, diffuse connective tissue diseases (SLE, systemic vasculitis), inflammatory bowel diseases (UC, CD). The following methods should be used for differential diagnosis: inoculation of biological environments for flora with determination of sensitivity; serological methods to rule out infections; immunological studies; bone marrow biopsy; chest, abdomen, braincomputer tomographyand / or magnetic resonance imaging; endoscopic studies; procalcitonin test.

In recent years, the method of determining procalcitonin has gained special importance, which makes it possible to distinguish inflammatory reactions associated with YURA from inflammatory reactions caused by bacterial infection. It is known that an increase in the level of procalcitonin is noted with sepsis and even with a local infectious process, and with YURA, even with high clinical and laboratory activity of the systemic form of YURA, the level of procalcitonin remains[5,7].

Juvenile ankylosing spondylitis (Bekhterev's disease in children).Juvenile ankylosing spondylitis (JAS) is a disease with a genetic predisposition, characterized by a chronic inflammatory process in the joints, both peripheral and axial skeleton, often combined with enthesitis, RF seronegativity and ANF. Since damage to the spinal joints can be delayed for years, a mandatory criterion for diagnosis in childhood is the defeat of the sacroiliac joints.

In diagnosticsThere is no specific diagnosis for YUIA. There are diagnostic criteria recommended by the American Rheumatism Association:

1) The onset of the disease in children under 16 years of age;
2) Characterized by swelling of one or more joints or having at least two of the following symptoms: limitation of function, tenderness during palpation, increased local temperature.
3) Duration of articular changes is at least 6 weeks.

4) Exclusion of other rheumatic diseases. An increase in ESR, CRP, Ig M, Ig G was noted in the analysis. Antibodies to RF and cyclic citrulline peptide, symptoms of rheumatoid arthritis are detected in only 6% of cases. An important role is played by instrumental diagnostic methods - MRI, CT, X-ray examination, ultrasound[4,7,8].

Treatment includes drugs that slow the progression of the disease (especially methotrexate, tumor necrosis factor [TNF] inhibitors, and interleukin [IL]-1 inhibitors), intra-articular corticosteroid injections, and sometimes nonsteroidal anti-inflammatory drugs (NSAIDs) to relieve symptoms[8].

Similar to the treatment of rheumatoid arthritis in adults, the use of disease-modifying antirheumatic drugs (DMARDs), particularly methotrexate, and biologics (eg, etanercept, anakinra, canakinumab, tocilizumab, abatacept) have revolutionized the therapeutic approach.

Methotrexate is indicated for oligoarticular, psoriatic and polyarticular forms of JIA. Monitoring of side effects is carried out as in adults. Bone marrow depression and liver toxicity are monitored using a complete blood count, aspartate aminotransferase (AST), alanine aminotransferase (ALT), and blood albumin levels. Sulfasalazine is sometimes prescribed, especially if spondyloarthritis is suspected.

If methotrexate is ineffective, a TNF inhibitor is used. Etanercept is often administered subcutaneously once a week at a dose of 0.8 mg/kg (up to 50 mg). Other TNF inhibitors with proven efficacy are adalimumab and infliximab. The IL-1 inhibitors anakinra and canakinumab are particularly effective in systemic JIA. Tocilizumab, an IL-6 receptor antagonist, is also indicated for the treatment of systemic JIA and polyarticular JIA. The T-cell costimulatory inhibitor Abatacept and the Janus kinase inhibitor tofacitinib are also treatments for polyarticular JIA[4,6].

Systemic glucocorticoids are not usually used except in systemic diseases. When necessary, the minimum adequate dose should be used (for example, the dose range of oral prednisone is 0.0125-0.5 mg/kg 4 times a day; or this dose can be given once or twice a day). The main risks of long-term use of corticosteroids in children are growth retardation, osteoporosis and osteonecrosis. Corticosteroids can be given intra-articularly. The dose for children is set based on body weight. Some children may require sedation for intra-articular injections, especially if multiple joints are injected.

When taking NSAIDs, the intensity of the symptoms of idiopathic juvenile arthritis can be reduced, but the drugs do not affect the long-term course of the joint disease and do not prevent the development of complications. The use of NSAIDs is the most effective for enthesitis. Naproxen (5-10 mg/kg orally 2 times a day), ibuprofen (5-10 mg/kg orally 4 times a day), indomethacin (0.5-1.0 orally 4 times a day mg/kg) is used.

Iridocyclitis is treated with corticosteroid eye drops and mydriatics and may require systemic methotrexate and anti-TNF therapy, and sometimes surgery.

Physiotherapy, exercise therapy, orthoses and other supportive measures help prevent the development of flexion contractures. Various assistive devices can help improve function and reduce stress on affected joints.

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