ISSN NO: 2770-2936
March 2025

Clinical Pharmacological Approach To The Drug Treatment Of Arthritis, Arthralgia And Back Pain Syndromes

Akhmedov B.U.
Assistant of the Department of pharmacology,
clinical pharmacology and medical biotechnology
ASMI

Abstract. The article is devoted to one of the most common rheumatic diseases — juvenile arthritis (JA). The main issues of epidemiology, classification, pathogenesis are considered, in more detail — the clinical picture of different variants of the course of JA, diagnostics and differential diagnosis. The treatment section describes the drugs used in JA, and presents an algorithm for differentiated treatment of the disease. Approaches to biological therapy, conditions for prescribing and management of patients receiving genetically engineered biological drugs are described separately.
Keywords: children, etanercept, juvenile arthritis, adalimumab, treatment, infliximab, juvenile rheumatoid arthritis, rituximab.

INTRODUCTION

Juvenile arthritis (JA) is arthritis of unknown cause, lasting more than 6 weeks, developing in children under 16 years of age with the exclusion of other joint pathology. JA is one of the most common and most disabling rheumatic diseases occurring in children. The incidence of JA is from 2 to 16 per 100 thousand of the child population under 16 years of age. The prevalence of JA in different countries varies from 0.05 to 0.6%. In the territory of the Russian Federation, among children under 18 years of age, the incidence reaches 62.3, primary - 16.2 per 100 thousand, including among adolescents the corresponding figures are 116.4 and 28.3, and among children under 14 years - 45.8 and 12.6. Rheumatoid arthritis (RA) most often affects girls. Mortality is within 0.5-1%.

MATERIALS AND METHODS

The primary antigen is unknown. The foreign antigen is absorbed and processed by antigen-presenting cells (dendritic cells, macrophages, B lymphocytes, etc.), which in turn present it (or information about it) to T lymphocytes. The interaction of the antigen-presenting cell with CD4+ lymphocytes stimulates the synthesis of the corresponding cytokines by them. Interleukin (IL) 2, produced during Th1 activation, interacts with specific IL 2 receptors, which causes clonal expansion of T lymphocytes and stimulates the growth of B lymphocytes. The latter leads to massive synthesis of IgG by plasma cells, increases the activity of natural killers (NK) and activates macrophages. IL 4, synthesized by Th2 cells, causes activation of the humoral link of immunity (manifested by increased synthesis of antibodies), stimulation of eosinophils and mast cells, and development of allergic reactions. The pathological process in JA begins in the synovial membrane of the joint with impaired microcirculation and damage to the cells lining the synovial membrane. In response to this, altered IgG are formed in the patient's body, which are perceived by the patient's own immune system as autoantigens. Immunocompetent cells, including plasma cells of the synovial membrane of the joint, produce antibodies in response - anti-IgG. These antibodies, called rheumatoid factor (RF), in the presence of complement interact with the autoantigen, and immune complexes are formed. Circulating immune complexes have a damaging effect on both the vascular endothelium and surrounding tissues. The synovial membrane of the joint is primarily affected, resulting in arthritis. In the synovial fluid and joint tissues, an excessive amount of proinflammatory cytokines is formed. Activation of the synthesis of such cytokines (IL 1, IL 6, IL 8, IL 17, TNF α) underlies the systemic manifestations of rheumatoid arthritis [2].

RESULTS AND DISCUSSION

In addition, IL 17 stimulates the production of RANKL (a ligand of the TNF family), which can activate osteoclasts and enhance bone resorption. Thus, IL 17 as a whole makes a destructive contribution to the development of osteoporosis in RA. Disruption of osteogenesis processes of various origins is combined with pronounced changes in the production of cytokines such as IL 1, IL 6, IL 8, IL 17, TNF.

ISSN NO: 2770-2936
March 2025

Increased vascular neoplasms (angiogenesis), which occur as a result of the effect of cytokines on tissues, also increase cartilage destruction.

Thus, the pannus, or "cloak" covering the surface of articular cartilage, interferes with normal metabolic processes and enhances the destruction of osteochondral formations.

JA is characterized by chronic non-purulent inflammation of the synovial membranes. Microscopy reveals edema, hyperemia, and infiltration with lymphocytes and plasma cells in the affected synovial tissues.

Increased secretion of synovial fluid leads to the formation of intra-articular effusion. Protrusions of the thickened synovial membrane form villi that protrude into the joint cavity; the hyperplastic synovial membrane in rheumatoid arthritis spreads over the surface of the articular cartilage and fuses with it (formation of pannus). As synovitis progresses, erosion and gradual destruction of the articular cartilage occur [3].

Persistent damage to the articular cartilage in juvenile rheumatoid arthritis occurs later than in adult RA; moreover, in many children with JRA, persistent joint damage never develops, despite long-term synovitis. Destruction of joint structures most often develops in children with a positive form of JRA for RF or with a form that begins with systemic manifestations. Once initiated, the process of destruction of joint structures can lead to erosion of bone tissue at the subchondral level, narrowing of the "joint space" (due to loss of articular cartilage), destruction and fusion of bones, deformations, subluxations or ankylosis of joints. Tenosynovitis and myositis may occur. Osteoporosis, periostitis, accelerated growth of the epiphyses and premature disappearance of the epiphyseal cartilage may be observed in bone areas adjacent to the affected joints. Rheumatoid nodules are less common in children than in adults, and occur mainly in patients with RF; they are characterized by the presence of fibrinoid material surrounded by chronic inflammatory cells. On the part of the pleura, pericardium and peritoneum, phenomena of non-specific fibrinous serositis may be noted; chronic constrictive pericarditis occurs very rarely. Rheumatoid rash is histologically represented by moderately expressed vasculitis with a few inflammatory cells surrounding small vessels in subepithelial tissues.

There are several variants of the course of JA: Juvenile arthritis with systemic onset (10-20% of cases)

• Develops at any age.
• Boys and girls get sick with the same frequency.
• Onset - acute or subacute.
• Fever - febrile, hectic, temperature rises mainly in the morning, often accompanied by chills, temperature drop - profuse sweat.
• Rash - spotted and (or) maculopapular, linear, not accompanied by itching, unstable, appears and disappears within a short time, intensifies at the height of the fever, localized mainly in the area of the joints, on the face, on the lateral surfaces of the body, buttocks and limbs. In some cases, the rash can be urticarial or hemorrhagic [5].
• Damage to internal organs

Heart damage:
- myocarditis;
- pericarditis;
— endocarditis (rare);
— coronary artery disease (rare).

Microscopy reveals edema, hyperemia, and infiltration with lymphocytes and plasma cells in the affected synovial tissues.

Increased secretion of synovial fluid leads to the formation of intra-articular effusion. Protrusions of the thickened synovial membrane form villi that protrude into the joint cavity; the hyperplastic synovial membrane in rheumatoid arthritis spreads over the surface of the articular cartilage and fuses with it (formation of pannus). As synovitis progresses, erosion and gradual destruction of the articular cartilage occur.

Persistent damage to the articular cartilage in juvenile rheumatoid arthritis occurs later than in adult RA; Moreover, many children with JRA never develop persistent joint damage, despite long-term synovitis [6].

Destruction of joint structures most often develops in children with a positive form of JRA for RF or with a form that begins with systemic manifestations. Once started, the process of destruction of joint structures

ISSN NO: 2770-2936
March 2025

can lead to erosion of bone tissue at the subchondral level, narrowing of the "joint space" (due to loss of articular cartilage), destruction and fusion of bones, deformations, subluxations or ankylosis of joints. Tenosynovitis and myositis may occur. Osteoporosis, periostitis, accelerated growth of the epiphyses and premature disappearance of the epiphyseal cartilage may be observed in bone areas adjacent to the affected joints.

Rheumatoid nodules are less common in children than in adults and occur mainly in patients with RF; characterized by the presence of fibrinoid material surrounded by chronic inflammatory cells.

CONCLUSION

From the pleura, pericardium and peritoneum, nonspecific fibrinous serositis may be observed; chronic constrictive pericarditis occurs very rarely. Rheumatoid rash is histologically represented by moderate vasculitis with a few inflammatory cells surrounding small vessels in subepithelial tissues.

— pleuropneumonitis;
— fibrosing alveolitis.

Polyserositis:
— pericarditis;
— pleurisy;
— perihepatitis;
— perisplenitis;
— serous peritonitis.

Vasculitis:
— palmar capillaritis;
— plantar capillaritis;
— local angioedema, most often in the hand area;
— cyanotic coloration of the proximal parts of the upper and lower extremities (palms, feet);
— marbling of the skin.

REFERENCES

1. Bijlsma JWJ, Berenbaum F, Lafeber FPJG. Osteoarthritis: an update with relevance for clinical practice. Lancet. 2011;377(9783):2115-2126.
2. Firestein GS, McInnes IB. Immunopathogenesis of rheumatoid arthritis. Immunity. 2017;46(2):183-196.
3. McAlindon TE, Bannuru RR, Sullivan MC, et al. OARSI guidelines for the non-surgical management of knee osteoarthritis. Osteoarthritis Cartilage. 2014;22(3):363-388.
4. Hochberg MC, Altman RD, April KT, et al. American College of Rheumatology 2012 recommendations for the use of nonpharmacologic and pharmacologic therapies in osteoarthritis of the hand, hip, and knee. Arthritis Care Res (Hoboken). 2012;64(4):465-474.
5. Kolasinski SL, Neogi T, Hochberg MC, et al. 2019 American College of Rheumatology/Arthritis Foundation guideline for the management of osteoarthritis of the hand, hip, and knee. Arthritis Rheumatol. 2020;72(2):220-233.
6. Cohen SP, Vase L, Hooten WM. Chronic pain: an update on burden, best practices, and new advances. Lancet. 2021;397(10289):2082-2097.