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PATHOGENESIS OF RHEUMATOID ARTHRITIS

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Rheumatoid arthritis is an immune-inflammatory rheumatic disease characterized by progressive destruction of joints and damage to internal organs, the development of which is determined by a complex interaction of environmental factors and genetic predisposition, leading to global disturbances in the humoral and cellular immunity system [1, 2]. The heterogeneity of the pathogenetic mechanisms of rheumatoid arthritis is reflected in the existence of a wide range of phenotypes and endotypes of the disease, which allows us to consider it not as “one disease,” but as a clinical and immunological syndrome [3, 4].

In the absence of effective therapy, life expectancy in patients with rheumatoid arthritis is lower by 3 years in women and by 7 years in men, primarily due to the high risk of developing comorbid diseases - cardiovascular pathology, osteoporosis, severe infections, interstitial lung disease, and oncological diseases [5].

Many patients with rheumatoid arthritis have a life prognosis as poor as those with Hodgkin's disease, type 2 diabetes, three-vessel coronary artery disease, and stroke. Rheumatoid arthritis causes permanent disability in half of patients within the first 3-5 years of the onset of the disease, and after 20 years, a third of patients become completely disabled.

The pathogenesis of rheumatoid arthritis is determined by a complex interaction of environmental factors and genetic predisposition, leading to global disturbances in the innate and acquired immune system, which are detected long before the development of clinical symptoms of the disease [3].

The essence of the pathological process in rheumatoid arthritis is systemic autoimmune inflammation, which affects the synovial membrane of the joints with maximum intensity.

Rheumatoid arthritis is a common and one of the most severe immune-inflammatory diseases in humans, which determines the great medical and socio-economic significance of this pathology [4]. The prevalence of rheumatoid arthritis among the

adult population in different geographical zones of the world varies from 0.5 to 2% [5]. The ratio of women to men is 3:1. The disease occurs in all age groups, but the peak incidence occurs in the most working age - 40-55 years.

Rheumatoid arthritis causes persistent loss of ability to work in half of patients during the first 3-5 years from the onset of the disease and leads to a significant reduction in their life expectancy, both due to the high frequency of comorbid diseases, primarily infectious complications, and extra-articular (systemic) manifestations (EP) characteristic of rheumatoid arthritis and complications associated with the systemic immune-inflammatory process - rheumatoid vasculitis , AA amyloidosis, etc.

In about half of the cases, the disease begins with a gradual (over the course of months) increase in pain and stiffness, mainly in the small joints of the hands and feet. At the onset of the disease, clinical manifestations are moderately expressed and often subjective. Only in some patients with a very active course of the disease are classic signs of joint inflammation revealed, such as an increase in skin temperature over the joints and their swelling (usually knees, less often - proximal interphalangeal and wrist). Sometimes the disease debuts as acute monoarthritis of large joints, resembling septic or microcrystalline arthritis. The disease can begin with recurrent bursitis and tendosynovitis , especially often localized in the area of the wrist joints, leading to the development of carpal tunnel syndrome. In elderly people, the onset of the disease can manifest itself in the form of acute polyarthritis of small and large joints with generalized polyarthralgia or symptoms resembling rheumatic polymyalgia .

Joint lesions in rheumatoid arthritis can be divided into 2 categories: potentially reversible (usually early), associated with the development of synovitis , and irreversible structural, developing in the late stages of the disease. This division is important for assessing the stage of the disease, prognosis and treatment tactics. It should be borne in mind that structural damage can develop very quickly, already within the first 2 years from the onset of the disease.

- the onset of the disease is characterized by diversity, the following variants of the onset of rheumatoid arthritis are conventionally distinguished :

- symmetrical polyarthritis with a gradual (over several months) increase in pain and stiffness, mainly in the small joints of the hands (in half of the cases);
- acute polyarthritis with predominant damage to the joints of the hands and feet, pronounced morning stiffness (usually accompanied by the early appearance of RF in the blood);
- mono- oligoarthritis of the knee or shoulder joints with subsequent rapid involvement of the small joints of the hands and feet;
- acute monoarthritis of large joints, resembling septic or microcrystalline arthritis;
- acute oligo- or polyarthritis with systemic phenomena (febrile fever, lymphadenopathy , hepatosplenomegaly). More often observed in young patients (resembles Still's disease in adults);
- " palindromic rheumatism": multiple recurrent attacks of acute symmetrical polyarthritis of the joints of the hands, less often the knee and elbow joints, lasting several hours or days and ending in complete recovery;
- recurrent bursitis and tenosynovitis , especially frequent in the wrist joints;
- acute polyarthritis in the elderly: multiple lesions of small and large joints, severe pain, diffuse edema and limited joint mobility. It is called "RS3PE syndrome" (Remitting Seronegative symmetric synovitis with Pitting Edema - remitting seronegative symmetrical synovitis with "pillow-shaped" edema);
- generalized myalgia, stiffness, depression, bilateral carpal tunnel syndrome, weight loss.

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