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Spread of Rheumatoid Arthritis in Ariid Zones

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Abstract: The article presents the results the degree of spread of rheumatoid arthritis in the Bukhara regional department of rheumatology over the past three years. In most cases, the disease has a chronic course, leading (in the absence of timely adequate therapy) to an unfavorable prognosis (in case of untimely and inadequate therapy) as well as the need for long-term and often constant use of medications, usually in various combinations.

Keywords: Rheumatoid arthritis, old age, arid zone.

Introduction. Rheumatoid arthritis (RA) is a widespread autoimmune disease of unknown etiology, characterized by symmetrical erosive synovitis, destruction of cartilage and bone tissue, as well as the development of a wide range of systemic manifestations. In most cases, the disease has a chronic course, leading (in the absence of timely adequate therapy) to an unfavorable prognosis (in case of untimely and inadequate therapy) as well as the need for long-term and often constant use of medications, usually in various combinations [1,2]. In addition, some patients with RA require various orthopedic interventions. Modern approaches to the treatment of RA are associated with huge financial costs. The above explains the significant socio-economic losses associated with RA, which are comparable to those with coronary heart disease (CHD) [3]. RA is the most common systemic connective tissue disease. According to various research groups, RA affects 0.5-2.5% of the adult population. RA can occur in childhood (juvenile RA), but most often the age of onset is 52±15 years. Among people under 35 years of age, the prevalence of RA is 0.38%, and in people over 55 years of age - 1.4%. Women get sick more often than men - 2-3 : 1. A high incidence of RA is noted among first-degree relatives (3.5%), especially among females (5.1%). The etiology of RA remains unknown [4,5]. The role of a wide range of exogenous, including infectious (Epstein- Barr virus, parvovirus B19, retroviruses, antigens and stress proteins of bacteria) and non-infectious (smoking, coal dust, some components of mineral oils, various chemical compounds, medicinal substances), as well as endogenous (citrullinated proteins and peptides) factors. It is assumed that exogenous factors take an indirect part in the development of RA against the background of genetic predisposition.

In recent years, the role of genetic factors in the development of RA has been actively studied, the risk of which is associated with carriage of the major histocompatibility complex class II antigen HLA-DR4 and DR1, which includes more than 22 alleles. When studying individual alleles, two were identified that were most closely associated with RA: DRB1*0401 and DRB1*0404, carriage of which was detected in 50–61% and 27–37% of patients with RA, respectively. The characteristic amino acid sequence of these alleles is called the “ common” epitope " (shared epitips -SE). SE carriage is associated with the severity of RA. Thus, carriage of one or two DR4 alleles is associated with a twofold increase in the risk of developing joint erosions. Carriage of SE 0401, 0404 or 0408 increases the risk of extra-articular manifestations of RA (vasculitis, lung damage, Felty's syndrome). Carriage of two DR alleles with SE is associated with more severe RA than carriage of one. Of interest are data on the relationship between SE and autoimmune disorders in RA, in particular with the presence of rheumatoid factor (RF) and antibodies to cyclic citrullinated peptide (ACCP). The formation of citrulline is the result of deamination of the positively charged amino acid arginine, which is regulated by enzymes of the peptidylarginine deaminase family. The appearance of the neutral amino acid citrulline leads to a change in the structure and increase in the immunogenicity of the modified proteins, an increase in their affinity for DR4 and the ability to activate T lymphocytes. Citrullination of protein β is a universal process associated with inflammation, as well as with the influence of environmental factors, primarily smoking. As arthritogenic citrullated proteins are considered

fibrinogen, vimentin, fibronectin, a- enolase, antigens and nuclear proteins of the Epstein- Barr virus, autoepitopes of the antigen-binding sites of T and B-lymphocyte receptors. Recent studies have shown that the combination of SE carriage with smoking, caffeine abuse, and contraceptive use significantly increases the risk of developing RA, positive for RF and especially ACCP. At the same time, in the absence of SE carriage, such a relationship is not observed. The role of other genetic factors in the development of RA that are not directly related to HLA-DR is discussed. These include polymorphism of genes encoding protein synthesis and regulating the activation processes of T-leukocytes. The leading morphological sign of RA is the formation of an ectopic focus of the synovial membrane in the form of its villous growth (hyperplasia). Invasive growth of this structure (pannus) leads to the destruction of articular cartilage and subchondral bone. In this case, erosions, cracks, and crevices are detected in the articular cartilage; in the joint cavities there is an increased amount of viscous turbid synovial fluid. Thickening, sclerosis and fibrous layers of the joint capsule are noted.

Complete obliteration of the articular cavity leads to the development of fibrous ankylosis. Proliferation of osteoid tissue is accompanied by the development of osteophytes and bone ankylosis. Histologically, the synovial membrane shows an increase in the number of synoviocytes, thickening of the intima, infiltration by immune inflammatory cells (macrophages, T and B lymphocytes, plasma and dendritic cells), formation of follicles from inflammatory cells, proliferation of granulation tissue, sclerosis, lipomatosis of the collagen and elastic layers of the synovial shells. The permeability of the walls of microvasculature vessels increases, accompanied by the release of fibrinous exudate and the formation of fibrinoid foci in the synovial lamina propria and fibrin-like deposits on the surface of the synovial membrane. At an early stage of the disease, neoangiogenesis is observed. In the deep layers of the synovial membrane, rheumatoid nodules are occasionally detected - small areas of fibrinoid necrosis, surrounded by macrophages and lymphocytes. In 80% of cases, hyperplasia and hypertrophy of synoviocytes occur with their characteristic palisade-like arrangement. Immunomorphologically, fibrin, immunoglobulins (G, A, M) and the C3 fraction of complement are detected in fibrinoid foci and fibrin-like overlays. The development of RA is associated with a T-cell immune response, which is characterized by hyperproduction pro-inflammatory cytokines, such as interleukins (IL): IL-1, -12, -7, -17, -6, -18, -2, tumor necrosis factor a (TNF- a), interferon gamma. The vast majority of T lymphocytes involved in the development of RA belong to the CD4+ subpopulation with a phenotype characteristic of memory cells. They exhibit helper cell activity that stimulates the synthesis of autoantibodies by B cells. In addition, in synovial tissue in RA, accumulation of CD8+ and CD28-T cells, which have the activity of autoreactive natural killer cells, is noted. An increase in their number correlates with the development of joint erosions. In RA, suppression of suppressor mechanisms (subpopulations of T-lymphocytes CD 25+) is also observed with increased production of IL-15 and IL-17, which supports autoimmune processes. B cells are involved in the activation of CD4 + lymphocytes, serving as specific antigen-presenting cells. B lymphocytes that synthesize RF interact with immune complexes and present a wide range of autoantigens and costimulatory molecules necessary for the activation of T cells. Mast cells activated with the participation of complement components, autoantibodies, and cytokines take part in the development of synovitis in RA. Mast cells synthesize a wide range of inflammatory mediators (histamine, TNF- a, tryptase, chymase, etc.), stimulating chondrocytes, synovial fibroblasts and macrophages, which, in turn, synthesize inflammatory mediators that cause swelling and destruction of joint tissue. Under the influence of proinflammatory cytokines, integumentary synoviocytes acquire the so-called transformation phenotype (characteristic of tumor cells). Although, unlike tumor cells, synovial cells do not metastasize, they acquire the ability to invade cartilage and ligament tissue and stimulate the activation and differentiation of osteoclasts, causing bone resorption. Synovial cells, as well as macrophages, synthesize matrix metalloproteinases, which play an important role in the destruction of cartilage and bone tissue. In this case, the processes of bone destruction significantly prevail over the processes of repair. Other stimulators of inflammation and destruction include complement activation products, the formation of which is associated with RF-containing immune complexes, ACCP antibodies, as well as a wide range of non-immune mediators, including nitric oxide, neuropeptides, arachidonic acid

metabolites, coagulation factors and fibrinolysis. Thus, the pathogenesis of RA appears to be multifaceted, and many of its components require further study and clarification.

Materials and methods research. In the area of the prevalence of disease and disability, the prevention of disability remains incompletely studied. Therefore, in order to study the extent of the spread of the disease in the Bukhara Regional Multidisciplinary Hospital in the Department of Rheumatology over the past three years, the extent of the spread of rheumatoid arthritis was studied.

In 2021, the department treated 1082 patients, of which 900 were patients with rheumatoid arthritis. This amounts to 0.8% per 100,000 population. Women make up 0.45%, men 0.35%, disabled people make up 0.19%.

In 2020, the total number of cured patients was 985, of which 800 patients had rheumatoid arthritis, which is 0.75% per 100,000 population. Women 0.54%, men 0.30%, disability is 0.35%.

In 2019, the number of patients was 1056, of which 692 were patients with rheumatoid arthritis, which is 0.5%, women 0.7%, men 0.3%, disability is 0.18%. Additionally, when comparing conditions among rural areas, the incidence rate is 4:1.

Results and discussions. Based on the above, we can say that the disease takes on a rejuvenating character year after year and leads to disability in people of childbearing age and able-bodied people.

Conclusion. Environmental factors, economic problems in everyday life, improper treatment, and late visits to a rheumatologist lead to various complications. And therefore, timely registration of patients with rheumatoid arthritis, study of disability, the degree of spread of the disease among the population, and the development of preventive measures is one of the global problems of timely arthrology.

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