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PATHOGENESIS AND IMMUNOLOGICAL MECHANISMS OF RHEUMATOID ARTHRITIS

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Annotation. Rheumatoid arthritis (rheumatoid arthritis) is a chronic, progressive autoimmune disease that mainly affects the joints and leads to systemic inflammation. The pathogenesis of rheumatoid arthritis is multifactorial and is associated with the interaction of genetic predisposition, environmental factors and immune system dysfunction. In the onset of the disease, impaired antigen recognition, activation of T-lymphocytes and β-lymphocytes play an important role. Cytokines, particularly tumor necrosis factor alpha (TNF-α), interleukin-1, and interleukin-6, promote inflammation and synovial tissue destruction in rheumatoid arthritis. Autoantibodies, including rheumatoid factor (RF) and anticyclic citrullinated peptide antibodies (ACPA), are key markers in the diagnosis and prognosis of the disease. Pathologically, rheumatoid arthritis is characterized by hypertrophy of the synovial membrane, pannus formation, and erosion of the joint surface. Studying the immunological mechanisms of rheumatoid arthritis may help develop therapeutic strategies, particularly targeted biologics. These studies are also important in developing therapeutic strategies to prevent chronic inflammation and joint deformity in rheumatoid arthritis.

Keywords: rheumatoid arthritis, autoimmune disease, pathogenesis, t-lymphocyte, b-lymphocyte, cytokines, tnf-α, interleukins, rheumatoid factor, acpa, inflammation, synovial membrane, pannus, joint destruction.

Rheumatoid arthritis (rheumatoid arthritis) is a chronic, systemic autoimmune disease that primarily causes joint damage and functional impairment. The worldwide prevalence of rheumatoid arthritis is approximately 0.5–1% of the adult population and is three times more common in women than in men. The disease usually begins between the ages of 30 and 50 and has a significant impact on the patient's quality of life. The clinical signs of rheumatoid arthritis include joint pain, swelling, stiffness, and deformity over time. However, rheumatoid arthritis is not just a joint disease, but also an immunological disease in which systemic inflammation causes rheumatoid arthritis. The pathogenesis of rheumatoid arthritis is multifactorial and multistep. Genetic predisposition plays an important role in the development of the disease; in particular, “damage haplotypes” associated with the HLA-DRB1 alleles significantly increase the risk of rheumatoid arthritis. At the same time, environmental factors, such as chronic infections, smoking, and microbiota imbalance, contribute to the pathological activation of the immune system. In rheumatoid arthritis, the immune system begins to misrecognize its own antigens, resulting in the activation of T-lymphocytes and β-lymphocytes. B lymphocytes produce autoantibodies, including rheumatoid factor (RF) and anticyclic citrullinated peptide antibodies (ACPA), which contribute to the chronic inflammation and joint destruction of rheumatoid arthritis. T lymphocytes, in turn, increase the secretion of cytokines, further intensifying the inflammation of rheumatoid arthritis. Cytokines are central to the pathogenesis of rheumatoid arthritis. Proinflammatory cytokines such as tumor necrosis factor alpha (TNF-α), interleukin-1 (IL-1), and interleukin-6 (IL-6) increase inflammation in synovial tissues, leading to hypertrophy and pannus formation, which ultimately leads to erosion of the joint surface. Also, in rheumatoid arthritis, immune system elements such as macrophages, dendritic cells, and neutrophils participate in synovial interactions. A thorough understanding of the pathogenesis and immunological mechanisms not only provides clarity in the diagnosis of

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rheumatoid arthritis, but is also important in developing targeted treatment strategies for the disease. Currently, biological drugs, in particular TNF-α, IL-6 antagonists, and preparations that affect β-lymphocytes, are effective in reducing inflammation and preserving joint function in rheumatoid arthritis patients. Therefore, identifying the molecular mechanisms of rheumatoid arthritis and studying the immunological aspects of rheumatoid arthritis are of urgent importance for clinical practice and scientific research. Rheumatoid arthritis is a multi-step autoimmune disease, the pathogenesis of which is the result of the interaction between genetic, immunological and environmental factors. This situation determines the main directions of modern scientific research to improve the treatment of the disease and improve the quality of life of patients.

Rheumatoid arthritis is a chronic autoimmune disease that causes destruction of joints, bones and connective tissues. The pathogenesis of the disease is multi-step and develops as a result of the interaction of genetic, immunological and environmental factors.[2]

Genetic predisposition plays an important role in the development of rheumatoid arthritis. In particular, “damage haplotypes” associated with the hla-drb1 alleles significantly increase the risk of developing rheumatoid arthritis. However, other genes, such as ptpn22, stat4, and traumatoid arthritis f1/c5, are involved in immune regulation and are considered additional factors in the pathogenesis of rheumatoid arthritis. In addition to genetic factors, in rheumatoid arthritis, environmental factors, such as chronic infections, smoking, microbiota imbalance, and stress, contribute to the inappropriate activation of the immune system.[1]

In the early stages of rheumatoid arthritis, the immune system begins to misrecognize its own antigens. In rheumatoid arthritis, T-lymphocytes and β-lymphocytes are activated, which increase the production of cytokines, as well as the production of autoantibodies - rheumatoid factor (RF) and anticyclic citrullinated peptide antibodies (ACPA). These immune reactions lead to inflammation in the synovial tissues and destruction of the joint surface.[4]

The immunological mechanisms of rheumatoid arthritis depend on the interaction of many cells and cytokines. The synovial membrane of rheumatoid arthritis contains T-lymphocytes, β-lymphocytes, macrophages and dendritic cells, the activation of which increases inflammation. T-lymphocytes, especially CD4+ helper cells, play a central role in the pathogenesis of rheumatoid arthritis. They produce cytokines such as interferon-gamma (IFN-γ), TNF-α, and IL-17, which promote synovial cell proliferation and inflammation.

B lymphocytes produce autoantibodies, including RF and ACPA, which lead to the formation of immune complexes. These immune complexes accumulate in synovial tissues, activating the complement system and increasing inflammation. The activity of B lymphocytes is enhanced by cytokines such as TNF-α and IL-6, which support the chronic inflammation of rheumatoid arthritis.

Cytokines play a central role in the pathogenesis of rheumatoid arthritis. TNF-α is the most important proinflammatory cytokine, which increases inflammation in synovial cells, inhibits angiogenesis, and activates osteoclasts, resulting in erosion of the joint surface and bone destruction.[3]

Interleukin-1 (IL-1) and interleukin-6 (IL-6) also play important roles in inflammation in rheumatoid arthritis. IL-1 activates fibroblasts and macrophages, increases the production of proteases, and causes joint erosion. IL-6, on the other hand, activates T lymphocytes and inhibits the production of autoantibodies by β lymphocytes. IL-6 is also involved in the development of systemic inflammation in rheumatoid arthritis, such as fatigue, anemia, and fever.

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The cytokine IL-17, specifically Th17, produced by rheumatoid arthritis cells, recruits neutrophils and promotes inflammation in the synovial tissues. This cytokine network is a major mechanism for the chronic inflammation and joint deformity of rheumatoid arthritis.[4]

In rheumatoid arthritis, hypertrophy of the synovial membrane occurs. Synovial cells proliferate and pannus, an invasive fibroblastic tissue, forms. Pannus covers the joint surface and has an erosive effect on bone and cartilage. This process is mediated by osteoclasts and proteolytic enzymes, which destroy bone and cartilage, resulting in joint deformity and functional impairment.

Rheumatoid factor (RF) and ACPA play an important role in the diagnosis and prognosis of rheumatoid arthritis. Rf IgM si It is an autoantibody in the NF1 and has the property of forming immune complexes. ACPA recognizes citrullinated peptides and enhances the inflammatory process in rheumatoid arthritis. These autoantibodies accumulate in synovial tissues and damage cells through the complement system.

Rheumatoid arthritis damages not only the joints, but also systemic organs. Dysfunction of the immune system in rheumatoid arthritis can develop pathologies related to the cardiovascular system, lungs, and blood system. Cytokines such as TNF-α and IL-6 cause systemic inflammation, causing fatigue, anemia, anorexia, and metabolic disorders in the patient.[5]

In conclusion, a deep understanding of the immunological mechanisms of rheumatoid arthritis and the development of targeted treatments for rheumatoid arthritis are the basis for the development of targeted therapies. TNF-α, IL-6 antagonists, and biological preparations that affect β-lymphocytes have been shown to be effective in reducing inflammation and preserving joint function in patients with rheumatoid arthritis. At the same time, immune modulation and cytokine activity in the early stages of rheumatoid arthritis may slow the progression of the disease.

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