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RHEUMATOID ARTHRITIS: ETIOLOGY, SYMPTOMS, DIAGNOSIS, TREATMENT

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Abstract
This article discusses the etiology, pathogenesis, clinical manifestations, diagnostic approaches and modern treatment strategies of rheumatoid arthritis based on the most recent data.

Keywords: Rheumatoid arthritis; pathogenesis; genetic susceptibility; HLA-DRB1; anti-CCP antibodies; immune dysregulation; DMARDs; biological therapy.

Introduction

Rheumatoid arthritis is a chronic inflammatory disease of connective tissue, which is characterized by joint damage and systemic damage to internal organs. The disease most often affects the joints, but can also affect other systems (lungs, heart, and nervous system). Joint damage occurs due to chronic inflammation of the synovial membrane - the inner layer lining the joint capsule. As the disease progresses, bone erosion and joint deformity occur. The disease is autoimmune, meaning the body produces antibodies against its own tissues. Prolonged exposure to these antibodies leads to tissue destruction and inflammation [1,11,12].

The prevalence in the adult population is 0,5-2% (in women < 65 years ~ 5%). The incidence of rheumatoid arthritis is approximately 50-100 new cases per 100,000 population per year. The prevalence ratio among women and men is 2-3:1. All age groups are affected, including children and the elderly. The peak onset of the disease is between 30 and 55 years [11,12,14].

The causes of rheumatoid arthritis are unknown.

Predisposing factors:

- Infections (Epstein-Barr virus, parvovirus B19, etc.).
- Genetic factors, carriage of the histocompatibility antigen HLA-DR4.
- Smoking, excessive coffee consumption, high body mass index, stress.
- Contact with mineral oils (motor or hydraulic). In an experiment, it was shown that these oils have arthritic (i.e., causing joint inflammation) properties.
- Sex hormones and reproductive factors (pregnancy, childbirth, etc.) [1,3,11].

Genetic Factors in Rheumatoid Arthritis

Rheumatoid arthritis is characterized by a significant genetic component, with studies indicating a heritability rate of approximately 60%. The genetic predisposition to RA has been linked primarily to specific alleles within the HLA region, particularly HLA-DRB1*01 and HLA-DRB1*04. These alleles are associated with an increased risk of developing RA, but other HLA alleles, such as HLA-DRB1*10 and DPB*1, have also been implicated in susceptibility. In addition to HLA genes,

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non-HLA genes such as PTPN22, TRAF1, and STAT4 play a crucial role in the disease's genetic landscape.

Genome-wide association studies (GWAS) have identified over 150 single nucleotide polymorphisms (SNPs) associated with rheumatoid arthritis susceptibility. These genetic variations interact with environmental factors, such as smoking and infections, to initiate the autoimmune response that characterizes RA. Importantly, genetic heterogeneity and epigenetic modifications, including DNA methylation and histone modifications, contribute to the complex pathogenesis of the disease. Understanding these genetic and epigenetic interactions is vital for developing targeted therapeutic approaches and improving disease management [1,4,15].

Immune Dysregulation in Rheumatoid Arthritis

The pathogenesis of rheumatoid arthritis is heavily influenced by immune dysregulation, involving various immune cells such as T cells, B cells, and macrophages. In RA, the immune system mistakenly attacks the synovial tissue, leading to persistent inflammation and synovial hyperplasia. B cells play a crucial role by producing autoantibodies, including rheumatoid factors (RFs) and anti-citrullinated protein antibodies (ACPAs), which are associated with disease severity. These autoantibodies not only serve as biomarkers but also contribute to the inflammatory process by activating complement pathways and recruiting additional immune cells to the inflamed joints. T cells, particularly CD4+ T helper cells, are pivotal in orchestrating the immune response in RA, promoting the activation of macrophages and synovial fibroblasts, which further exacerbates tissue damage. This immune-mediated destruction is a hallmark of rheumatoid arthritis and underscores the need for therapies that target these immune pathways to alleviate symptoms and prevent joint damage [1,4,10].

Pathogenesis of rheumatoid arthritis

Normally, the synovial membrane is only a few cells thick and produces synovial fluid, which lubricates and nourishes the joint. The mechanism of rheumatoid arthritis development can be described as follows.

1. Rheumatoid arthritis causes immune cells to attack the healthy synovial membrane. Subsynovial edema develops, and lymphocytes, polymorphonuclear leukocytes, monocytes, and plasma cells accumulate in the synovial membrane.
2. At the same time, immune cells release protective proteins – cytokines, which provoke the proliferation of blood vessels in the synovial membrane.
3. Increased blood flow leads to excessive tissue growth. Synovial cells proliferate rapidly, leading to thickening of the synovial tissue. This abnormally thickened tissue is called "pannus".
4. Pannus cells secrete proteolytic enzymes that destroy cartilage.
5. At the same time, the overproduction of proinflammatory cytokines (TNF-alpha, etc.) activates osteoclasts (bone cells that destroy old bone tissue), leading to bone damage. Subsequently, bone tissue is destroyed, resulting in erosions.
6. Bone erosive changes also occur as a result of the activation of fibroblasts (the main cells of loose connective tissue), which begin to produce enzymes that can destroy articular cartilage.
7. As the disease progresses, the pannus transforms into mature fibrous tissue, leading to fusion of the articular surfaces [1,11,12].

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Classification and stages of development of rheumatoid arthritis

International Classification of Diseases, 10th revision (ICD-10):

- M05 - Seropositive rheumatoid arthritis.
- M06 - Other rheumatoid arthritis.
- M05.0 - Felty's syndrome.
- M06.1 - Adult Still's disease.
- M06.9 - Rheumatoid arthritis, unspecified.

Working classification of rheumatoid arthritis (draft 2002):

Seropositive rheumatoid arthritis (M 05).

- Polyarthritis (M05).
- Rheumatoid vasculitis (M 05.2) (digital arteritis, chronic skin ulcers, Raynaud's syndrome, etc.).
- Rheumatoid nodules (M 05.3).
- Polyneuropathy (M 05.3).
- Rheumatoid pulmonary disease (M 05.1) (alveolitis, rheumatoid lungs).
- Felty's syndrome (M 05.1).

Seronegative rheumatoid arthritis (M 06.0).

- Polyarthritis (M 06.0).
- Adult Still's syndrome (M 06.1)

For a long time, the 1987 criteria were used to establish a reliable diagnosis of RA.

Revised diagnostic criteria for RA (ARA 1987):

- Morning stiffness (at least 1 hour).
- Arthritis of three or more joint areas.
- Arthritis of the joints of the hands.
- Symmetrical arthritis.
- Rheumatoid nodules.
- Rheumatoid factor.
- Radiographic changes.

A reliable diagnosis of RA is established when four of the seven above criteria are present, with the first four having to exist for at least six weeks.

It has now been proven that the negative prognosis of RA can be reversed with early initiation of pathogenetic disease-modifying therapy. Therefore, methods for the early diagnosis of RA have been developed for a long time. In 2010, the Association of American and European Rheumatologists adopted criteria for the early diagnosis of RA, in which clinical and laboratory parameters are expressed in points. A total score of more than 6 points allows for a reliable diagnosis of early-stage RA, suggesting the possibility of initiating pathogenetic therapy with disease-modifying drugs [1,2,6].

A. Joint damage	Points
1 large joint	0
2-10 large joints	1
1-3 small joints	2
4-10 small joints	3
More than 10 joints	4

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B. Serological test	
Negative RF ADC	0
Weak + RF and ADC tests	1
High + tests for RF and ADC	2
C. Markers of the acute phase of inflammation	
Normal CRP and ESR	0
Abnormal ESR and CRP	1
D. Duration of symptoms	
Less than 6 weeks	0
More than 6 weeks	1

A score of 6 out of 10 indicates definite RA.

In 2007, the Russian Association of Rheumatologists adopted a new classification of RA, which included several sections.

Main diagnosis:

1. Rheumatoid arthritis seropositive (M05.8).
2. Rheumatoid arthritis seronegative (M06.0).
3. Special clinical forms of rheumatoid arthritis:

4. Felty syndrome (M05.0);
5. Adult-onset Still's disease (M06.1).
6. Rheumatoid arthritis probable (M05.9, M06.4, M06.9).

Clinical stage:

1. Very early stage: lasts less than 6 months.
2. Early stage: lasts from 6 months to a year.
3. Advanced stage: lasts more than a year with typical symptoms of rheumatoid arthritis.
4. Late stage: lasts 2 years or more, occurs with severe damage to small (III-IV radiographic stage) and large joints, and the presence of complications. (Clinical stages of rheumatoid arthritis)

Disease activity:

- 0 = remission: DAS28 < 2.6 (DAS28 is the disease activity index).
- 1 = low: 2.6 < DAS28 < 3.2.
- 2 = average: DAS28 = 3.2-5.1.
- 3 = high (DAS28 > 5.1).

Extra-articular (systemic) manifestations:

1. Rheumatoid nodules.
2. Cutaneous vasculitis (ulcerative necrotic vasculitis, nail bed infarctions, digital arteritis, livedoangiitis).
3. Vasculitis of other organs.
4. Neuropathy (mononeuritis, polyneuropathy).
5. Pleurisy (dry, exudative), pericarditis (dry, exudative).
6. Sjögren's syndrome.
7. Eye lesions (scleritis, episcleritis, retinal vasculitis).

Instrumental characteristics:

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1. Presence of erosions (using radiography, possibly MRI, ultrasound):
2. nonerosive;
3. erosive.
4. Radiographic stage (according to Steinbrocker, modification):

I - periarticular osteoporosis.
II - osteoporosis + narrowing of the joint space, isolated erosions are possible.
III - signs of the previous stage + multiple erosions + subluxations in the joints.
IV – signs of the previous stage + bone ankylosis.

An additional immunological characteristic is antibodies to cyclic citrullinated peptide (CCP). Citrullinated peptide is a protein produced as a result of metabolic processes in the body. Normally, citrulline is completely eliminated from the body. As RA develops, CCP levels increase, and the immune system perceives it as foreign and begins to produce antibodies against it.

• ACCP - present (+);
• ACCP - absent (-).

Functional class:

• I — the patient performs all three vital functions: self-care, professional and non-professional duties.
• II — the patient is limited only in non-professional activities (all elements of leisure, recreation, entertainment, sports).
• III — non-professional and professional activities (work and study, housekeeping) are limited, but the ability to self-care is preserved.
• V - all three vital functions are lost, even the ability to care for oneself [1,2,6].

Diagnosis of rheumatoid arthritis

Laboratory tests recommended for examination of patients with suspected rheumatoid arthritis:

1. Complete blood count with platelet count.
2. Biochemical blood test (total protein, albumin, globulin fractions, total bilirubin, urea, creatinine, electrolytes, calcium, cholesterol, blood lipid profile, C-reactive protein, rheumatoid factor).
3. Study of the level of antibodies to cyclic citrullinated peptide (CCP).
4. General urine analysis, determination of protein in urine.
5. Study of serum immunoglobulins

Early diagnosis involves testing the level of anti-CCP:

- Anti-CCP testing is essential for the early diagnosis of rheumatoid arthritis, as it is a more sensitive and specific serological marker for early rheumatoid arthritis than rheumatoid factor (RF). (RF is a protein produced by the human immune system that attacks its own cells, mistaking them for foreign substances.)
- ACPA is detected in 40-50% of patients with rheumatoid arthritis with negative RF.
- ACPA "+" is an indication for the initiation of antirheumatic therapy at an early stage (less than six months), which allows for the effective inhibition of progressive joint damage.

• ACPA "+" is significant for differential diagnosis and prognosis of the course of rheumatoid arthritis.

Instrumental diagnostic methods recommended for examining patients with suspected rheumatoid arthritis:

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- joint radiography;
- Ultrasound of joints;
- MRI of joints;
- arthroscopy;
- diagnostic joint puncture: immunological, cytological examination, bacterial culture of synovial fluid [1,6,11].

Rheumatoid Arthritis Differential Diagnosis and Discriminating Features [2]

CLINICAL FEATURES

Articular Manifestations

With its primary manifestation involving synovial tissues, the signs and symptoms of RA are primarily driven by active synovitis that results in joint swelling, stiffness, palpable warmth, and pain. The classic joint distribution in early disease includes the small joints of the hands and feet (MCP, PIP, and MTPs)

Hands and Wrists

The hands and wrists are often considered together because they form a single functional unit. For example, wrist involvement is strongly associated with the presence of ulnar deviation of the MCP joints. This may occur via weakening of the extensor carpi ulnaris muscle or tendon, either as a direct or indirect result of inflammation, which leads to radial deviation of the wrist as the carpal bones rotate (the proximal row in an ulnar direction and the distal row in a radial direction). Subsequently, ulnar deviation of the MCP joints and the associated palmar subluxation may serve as compensatory mechanisms to maintain digital tendons

in a normal line with the radius. Together, changes of the wrist and MCP joints result in a highly characteristic “zigzag” deformity (Fig.B and C). Although classically described as irreversible, reducible ulnar deviation (Fig.A) of the MCP joints can result from bulky synovitis, tendon laxity occurring after the reduction of synovitis, tendon rupture, or muscle weakness in the hands. Nevertheless, reversible MCP deviation is more typical of nonerosive processes such as SLE or non-inflammatory neurologic conditions such as Parkinson’s disease. Proliferative synovium contains both the inflammatory and enzymatic machinery sufficient to destroy ligaments, tendons, and the articular regions distal to the ulnar head, resulting in joint subluxation, dislocation, and bony erosions. This inflammatory synovium may also invade and impinge normal tissue spaces, and lead to nerve entrapment syndromes, such as carpal tunnel syndrome (median nerve). The inflammatory and enzymatic degradation of tissue, coupled with naturally occurring forces, act together to impair the integrity of the radiocarpal, radioulnar, and midcarpal joints. The ulnar collateral ligament can become stretched or even destroyed by synovitis of the radioulnar joint. As a result, the ulnar head may “spring up” into dorsal prominence where it

“floats” and can be easily depressed (the so-called piano key styloid) (Fig. E). Severe disease progression in the wrist can lead to marked loss of joint space, bone erosions, and ankyloses; the latter is more common with wrist immobilization resulting from

unremitting disease. Perhaps the best recognized of the articular deformities observed in RA are swan neck and boutonnиre deformities of the fingers (Fig. F and G). The swan neck deformity involves DIP flexion with simultaneous PIP hyperextension. This condition is thought to result from direct

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involvement of the PIP and/or MCP joints. Boutonniиre deformities, PIP flexion, and DIP hyperextension are related to relaxation of the central slip, with buttonholing of the
PIP joint between its lateral bands. Involvement of the thumb in RA also has unique characteristics. Several corresponding deformities are described and result from involvement of the first MCP, interphalangeal (IP), or carpal metacarpal (CMC) joints, either alone or in combination.
(A) Polyarticular arthritis with fusiform swelling of the proximal
interphalangeal joints along with wrist deformity
(B) ulnar deviatio (C) "Zig-zag" defor (D) Piano key styloid. (F) Swan neck deformity (G) Boutonniere deformity
(A) Shoulder findings in rheumatoid arthritis include glenohumeral joint space narrowing with marginal erosions and cystic changes of the humeral head (lower arrow), a high-riding humeral head indicative of a rotator cuff tear, and tapering of the distal end of the clavicle with widening of the acromioclavicular joint (upper arrow). (B) Ruptured Baker's cyst of the left calf with an associated pseudothrombophlebitis. (C) Bilateral protrusio acetabuli. Note the symmetric joint space narrowing with femoral head remodeling and medial migration toward the pelvis [1,2,14].

Extra-articular Manifestations

Recognizing that RA is a systemic inflammatory disease is requisite for optimal management. Inflammation in RA involves not only articular structures but also potentially extra-articular tissues, leading to the development of subcutaneous nodules and other mucocutaneous findings, as well as serositis and cardiopulmonary, neurologic, ocular, and hematologic complications. Extra-articular manifestations are observed in up to 50% of patients with RA and generally portend a poor prognosis, including increased morbidity and mortality. On examination, rheumatoid nodules are typically subcutaneous and firm and may be either mobile or adherent to underlying structures. Because the physical findings are nonspecific and subcutaneous, nodules can be seen with other forms of inflammatory arthritis; biopsy may be needed to confirm the diagnosis in select
circumstances. Other conditions that can result in inflammatory arthritis and nodules include rheumatic fever, tophaceous gout, SLE, and multicentric reticulohistiocytosis Histologically, rheumatoid nodules are characterized by a central area of necrosis rimmed by a corona of palisading fibroblasts, which are
surrounded by a collagenous capsule with perivascular collections of inflammatory cells. These nodules grow by accumulating cells that expand centrifugally, leaving behind central necrosis of the connective tissue matrix. Inspection of early lesions suggests that the nodule is composed of small, inflamed arterioles, suggesting
that rheumatoid nodules are a form of small vessel vasculitis. This fact may explain the relationship of this finding to other forms of extra-articular disease, such as CVD. Although rheumatoid nodules may regress either spontaneously or with disease-remitting therapy, increased nodulosis is an uncommon but well-recognized complication of methotrexate treatment that often paradoxically occurs in patients with well-controlled disease. Amyloidosis may also complicate long-standing inflammatory disease. The purpura of amyloidosis may be confused with the
skin fragility and "senile purpura" seen in patients who have been on long-standing glucocorticoid therapy. Renal disease, especially proteinuria, or diarrhea with malabsorption in a patient with long-

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standing RA should prompt investigation for secondary amyloidosis. Vasculitis in RA may present with multiple skin findings, ranging from relatively benign nail-fold infarcts to a deep, erosive, scarring pyoderma gangrenosum.

The presence of livedo reticularis, with its characteristic lacey appearance (most prominent on the lower extremities), should raise a concern for a medium-vessel vasculitis or an associated connective tissue disease, such as SLE or antiphospholipid antibody syndrome (APS).The prevalence of secondary Sjцgren's syndrome (autoimmune exocrinopathy) in RA has been estimated at 17%, resulting in symptoms of keratoconjunctivitis sicca (dry eyes) or xerostomia (dry mouth). These symptoms are accompanied by objective evidence of lacrimal or salivary gland dysfunction [1,5,11].

Treatment of rheumatoid arthritis

Objectives: reduction or elimination of arthritis symptoms and extra-articular manifestations, control of inflammatory activity, prevention of progression of osteoarticular destruction, preservation and significant improvement of quality of life, increase of life expectancy to the average level in the population [3, 5,10] .

Treat to Target (T2T) – treatment until the target is reached (EULAR recommendations)

General recommendations:

1. Elimination of stress.
2. Avoid excessive solar radiation exposure. Intense sun exposure also triggers the development of rheumatic diseases.
3. Active treatment of concomitant infections, vaccination if necessary.
4. Prevention of atherosclerosis: eating a diet low in fat, cholesterol and high in polyunsaturated fatty acids, quitting smoking, controlling body weight, exercising, taking folic acid.
5. Osteoporosis prevention: foods high in calcium, vitamin D intake, and possibly bisphosphonates.

Drug treatment

Basic therapy for rheumatoid arthritis

DMARDs (modifying antirheumatic drugs, disease-modifying antirheumatic drugs, slow-acting drugs) are a key component of RA treatment. In the absence of contraindications, they should be prescribed to every patient with this diagnosis. It is especially important to prescribe DMARDs immediately after diagnosis at an early stage, when there is a limited period (several months) to achieve the best long-term results—the so-called "therapeutic window" [9,10].

Methotrexate is one of the mainstays of RA therapy. It is a cytotoxic drug belonging to the antimetabolite group. It is structurally similar to folic acid. Every patient with RA should be prescribed methotrexate therapy unless there are clear contraindications. Methotrexate is the drug of choice for active rheumatoid arthritis, in both the early and advanced stages. This drug has a significant therapeutic range and allows for individualized dosage adjustments. Effective doses range from 15-30 mg/week, with an average of 20 mg/week. Methotrexate is quite safe and requires minimal laboratory monitoring (complete blood count and transaminase analysis). It is advisable to prescribe folic acid 3-5 mg/day on days without drug administration [5,11]. It is administered orally, subcutaneously and intramuscularly.

Main adverse events: M-erosive stomatitis, erosive gastritis, dyspepsia, hemorrhagic enterocolitis, hepatocellular syndrome, nephrotoxicity, obstructive uric acid nephropathy.

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Leflunomide (Arava) has anti-inflammatory, immunomodulatory, and antiproliferative properties. It was specifically developed for the treatment of RA. Adverse events: gastrointestinal toxicity, liver toxicity, skin rash, alopecia, cytopenias, fibrosing alveolitis, weight loss, fever, renal toxicity - if these develop, it is necessary to take cholestyramine 8 g three times a day for 11 days [5,10,11].

Biological therapy for RA

The introduction of genetically engineered biological agents (GEBIs) has been a major breakthrough in the treatment of rheumatic diseases. GEBIs are artificial antibodies (proteins from the immunoglobulin group). With the advent of biological agents, it has become possible to achieve long-term and stable remission in patients with rheumatoid arthritis. Currently, GEBIs are divided into groups based on their mechanism of action:

• drugs aimed at suppressing the production of TNF-alpha (tumor necrosis factor): infliximab, etanercept, adalimumab, certolizumab pegol, golimumab [1,5,8].

• immunoglobulin inhibitors: IL-1, IL-6.

• abatacept is a soluble protein that targets T cell costimulation,

• Drugs that block the action of B cells (rituximab and belimumab) are used for rheumatoid arthritis and systemic lupus erythematosus.

Recently, ustekinumab (IL-12/23 blocker) and secukinumab (IL-17 inhibitor) have been widely used in the treatment of seronegative spondyloarthritis, including psoriatic arthritis [8,9].

Side effects. Biological drugs, in addition to their positive properties, also have negative effects: Decreased anti-infective and (potentially) anti-tumor immunity. Adverse reactions (ARs) common to all TNF-alpha antagonists include increased susceptibility to infections, including the potential for exacerbation of histoplasmosis (a fungal disease) and hepatitis B. There is a risk of developing demyelinating diseases, lupus-like syndrome (drug-induced lupus, which occurs as a result of taking medications), malignant neoplasms, thromboembolism (blockage of a blood vessel by a clot), and hypersensitivity reactions. The most common ADRs include nasopharyngitis (inflammation of the mucous membrane of the nasopharynx), urinary and upper respiratory tract infections, abdominal pain, diarrhea, the development of ANF (antinuclear factor, which indicates the presence or absence of an autoimmune disease) and antibodies to double-stranded DNA. Susceptibility to tuberculosis is particularly increased. When using TNF-alpha antagonists, tuberculosis can produce an atypical clinical picture. Given this, before initiating therapy with TNF-alpha antagonists, all patients should be examined for both active and latent tuberculosis infection: Mantoux test, Diaskin test [3,6].

The risk of developing allergic reactions and immunogenicity, i.e., the reaction of the body's immune system to biological therapy (BITs) that contain a foreign murine protein. The resulting neutralizing antibodies to BITs reduce the effectiveness of the therapy and contribute to the development of infusion/post-injection reactions. Most often, these antibodies are formed to infliximab, which contains a higher murine component than humanized anti-TNF-alpha drugs. The frequency of their occurrence in RA ranges from 7 to 53%, depending on the dose. Prescribing infliximab in combination with methotrexate reduces the likelihood of antibody formation. With treatment with etanercept and adalimumab, antibodies are detected less frequently (5-17%). When using adalimumab in combination with methotrexate, the frequency of antibody formation is minimal and amounts to approximately 1% [1,4,6].

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TNF-alpha antagonists cause a small but long-lasting increase in HDL cholesterol (the so-called "good" cholesterol). This increase, in turn, may have a beneficial effect on cardiovascular risk in patients with chronic arthritis. A reduction in the incidence of cardiovascular events and mortality has been demonstrated in a group of RA patients treated with anti-TNF-alpha drugs, compared with patients who did not receive this group of drugs in their combination therapy. However, it should be borne in mind that TNF-alpha antagonists should be used with extreme caution in patients with RA and congestive heart failure, as they can lead to circulatory decompensation and increased mortality. Severe chronic heart failure is a contraindication for the use of biological therapy [4,9]. Class of small molecules: Tofacitinib (Yaquinus) is a new class of drug for the treatment of RA. It can be used either as monotherapy or in combination with methotrexate or other DMARDs, regardless of food intake [4,6]. Symptomatic therapy NSAIDs (nonsteroidal anti-inflammatory drugs) are characterized by a rapid onset of therapeutic effect and active suppression of pain and inflammation (diclofenac, Nimesil, Nise, Arcoxia, etc.).

GCS (glucocorticosteroids) are used in the following cases:

- at maximum activity of the inflammatory process;
- for severe pain that is not relieved by NSAIDs;
- in generalized arthritis with exudative manifestations;
- in systemic manifestations of RA;
- as a component of bridge therapy(Metipred, prednisolone, Polcortolone)

Non-drug treatment

Although drug therapy is the primary component of RA treatment, non-drug approaches play a vital role in achieving a complete therapeutic effect. The goal of rehabilitation measures is to restore the patient's impaired or lost abilities and their adaptation to the chronic disease [3].

Physical therapy and occupational therapy (special exercises that simulate movements during self-care, aimed at restoring motor skills) are useful for patients with RA.

Physiotherapy and balneotherapy are beneficial for patients with low inflammatory activity and can significantly reduce symptoms and improve mobility [3].

Herbal Interventions in the Management of Rheumatoid Arthritis

Rheumatoid arthritis (RA) is characterized by persistent inflammation and severe joint destruction, leading to significant morbidity. In recent years, there has been growing interest in the use of herbal interventions as complementary treatments for RA. A comprehensive review evaluated the efficacy, safety, and mechanisms of action of various herbal remedies, including turmeric, ginger, Boswellia serrata (frankincense), green tea, and Ashwagandha, in managing RA symptoms [13,14].

Early diagnosis and timely initiation of disease-modifying antirheumatic drugs remain the cornerstone of effective rheumatoid arthritis management. The introduction of biologic agents and targeted synthetic therapies, along with the treat-to-target approach, has made it possible to achieve sustained remission or low disease activity in a substantial proportion of patients. Comprehensive management, combining pharmacological treatment with non-pharmacological interventions, is essential for preventing irreversible joint damage, reducing systemic complications, and improving quality of life. Continued research into personalized therapeutic strategies and novel targets is crucial for further optimizing outcomes in patients with rheumatoid arthritis.

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