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THE ROLE OF IMMUNOGENETIC FACTORS IN THE DEVELOPMENT OF RHEUMATOID ARTHRITIS

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Annotation: Epidemiology, etiology, pathogenesis, clinic, clinical variants, classification, diagnosis, symptoms, treatment of rheumatoid arthritis, modern therapy: This article talks in detail about rheumatoid arthritis!
Key words: Epidemiology, etiology, pathogenesis, clinic, clinical variants, classification, diagnosis, symptoms, treatment of rheumatoid arthritis.

Rheumatoid arthritis (Russian: Rheumatoid arthritis) is a systemic connective tissue disease of complex autoimmune pathogenesis of an erosive-destructive type of polyarthritis of unclear etiology, characterized mainly by lesions of small joints.

The cause of the disease is currently unknown. Indirect data: An increase in the number of leukocytes and an erythrocyte sedimentation rate (ESR) indicates an infectious process. It is believed that the disease develops as a result of an infection that leads to disruption of the immune system in individuals with a genetic predisposition; forms immune complexes (consisting of antibodies, viruses, etc.), which accumulate in tissues and cause damage to the joints. However, the ineffectiveness of antibiotic therapy for rheumatoid arthritis probably indicates the incorrectness of this assumption.

The disease is characterized by a high frequency of early disability (70%). The main causes of death from the disease are infectious complications and renal failure.

Treatment of rheumatoid arthritis is mainly aimed at relieving pain, slowing down the progression of the disease, and repairing damaged tissues through surgery. With the early detection of the disease with the help of modern means, it is possible to significantly reduce its damage to the joints and other tissues.

The disease first manifests itself during periods of heavy physical exertion, emotional upheaval, overwork, hormonal adaptation, as a result of adverse factors or infection.

EPIDEMIOLOGY

Rheumatoid arthritis is widespread throughout the world and affects all ethnic groups. The prevalence is 0.5-1% (up to 5% in the elderly). Every year, between 5 and 50 out of 100,000 people fall ill. In 2010, about 49,000 people died from rheumatoid arthritis.

The median age of onset is 40-50 years in young women and somewhat higher in men. It is 3-5 times more common in women than in men.

ETIOLOGY

As with most autoimmune diseases, there are three main factors in the development of rheumatoid arthritis (rheumatic triad):

1. Genetic (hereditary) predisposition

Hereditary predisposition to autoimmune reactions.

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Class II MHC specific antigen: more common in carriers of HLA-DR1, DR4.
2. Infectious factor: hypothetical triggers of rheumatic diseases.

Paramyxoviruses - mumps, measles, respiratory syncytial infections, viruses;
Hepatoviruses - hepatitis B virus;
Herpes viruses - herpes simplex viruses, herpes zoster, cytomegalovirus;
Epstein-Barr virus;
Retroviruses are T-lymphotropic viruses.
3. Factors of initiative

Hypothermia, hyperisolation, poisoning, mutagenic drugs, endocrinopathy, stress and more.

In women, the duration of breastfeeding reduces the likelihood of developing rheumatoid arthritis. Breastfeeding for 24 months or more cuts the risk of developing RA by half.

PATHOGENESIS

Rheumatoid arthritis belongs to the group of autoimmune diseases. This group of diseases is characterized by the behavior of protective cells - lymphocytes. Instead of actively detecting and destroying foreign bacteria, fungi and viruses, they begin to attack their healthy cells. This pathological process of violation of the interaction of cells of the immune system in the immune response consists of the following stages:

Synoviocytes acquire the properties of macrophages, secrete pro-inflammatory cytokines (primarily tumor necrosis factor alpha, interleukin 1), become antigen-presenting cells, and activate type 1 T-helpers.

The synovial membrane and cells of the synovial fluid of the joint produce a large amount of T-helper type 1, which activates interferon gamma and macrophages.

CLINICS

Rheumatoid arthritis occurs in three stages. In the first stage, there is periarticular edema of the bursae, which causes pain, local fever, and swelling around the joints. The second stage is the rapid division of cells, which leads to the formation of a synovial membrane. In the third stage, the inflamed cells produce an enzyme that damages the bones and joints, which often leads to deformation of the affected joints, increased pain and loss of motor function.

Possible prodromal clinical manifestations (minor transient pain, pain associated with weather conditions, vegetative disorders). Distinguish between "damaged joints" and "exceptional joints". To the first (according to the frequency of observation): II and III palmo-finger phalanges, proximal interphalangeal and metatarsophalangeal, knee and radiocarpal-palmar, elbow and ankle joints. The “exclusion joints” include: distal interphalangeal, I palmar-phalangeal (thumb).

Rheumatoid arthritis is often accompanied by other joint diseases - osteoarthritis, rheumatism, systemic diseases of the connective tissue.

Extra-articular manifestations:

From the side of the cardiovascular system: pericarditis, vasculitis, granulomatous valvular lesions, atherosclerosis.
Respiratory system: pleurisy, interstitial disease.
Skin: rheumatoid nodules, thickening and malnutrition, vasculitis, reticular levicide.

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Nervous system: compression neuropathy, sensorimotor neuropathy, mononeuritis multiplex, cervical myelitis.

Organs of vision: dry keratoconjunctivitis, episcleritis, scleritis, peripheral wound keratopathy.

Kidneys: amyloid vasculitis, nephritis, NYQP nephropathy;

Blood: anemia, thrombocytosis, neutropenia.

CLINICAL TRANSITION OPTIONS

There are the following variants of the clinical course of rheumatoid arthritis:

The classic variant (symmetrical damage to small and large joints, slow progression of the disease).

Mono- or oligoarthritis, which is characterized by damage to large joints (usually knees). The exact onset of the disease and the possibility of stopping all manifestations after 1-1.5 months (migratory arthralgia, radiological changes are not observed, anti-inflammatory drugs have a relatively positive effect; later, rheumatoid arthritis of all signs).

Rheumatoid arthritis with pseudoseptic syndrome (hectic-type fever, chills, hyperhidrosis, weight loss with the development of amyotrophy, anemia, vasculitis of internal organs, in some cases reversible clinical signs of arthritis).

Felty's syndrome (a combination of polyarthritis and splenomegaly, may be a non-splenomegalic variant, but with leukocytopenia, neutropenia of the internal organs).

Still syndrome.

Juvenile rheumatoid arthritis (onset before age 16):

allergic septic syndrome;

articular-visceral form with limited organs.

Articular-visceral form:

rheumatoid vasculitis;

damage to the heart, lungs, kidneys, gastrointestinal tract;

damage to the nervous system.

CLASSIFICATION

Stages of clinical manifestations I.

- very early: up to 6 months;
- early: 6 - 12 months;
- common: more than a year;
- late: more than two years.

Disease activity II (DAS28)

0 (remission): DAS28 less than 2.6;
1 (past): DAS28 2.6- 3.2;
2 (medium): DAS28 3.2-5.1;
3 (high): DAS28 above 5.1.

III Instrumental Features

Presence of erosion

X-ray stage (1-4)

IV Immunological features

Rheumatoid factor: seropositive/seronegative;

Anti-CCP: seropositive / seronegative.

V Functional class

I Self-service, non-professional and professional succession

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II Preservation of non-professional abilities and abilities for self-service, violation of professional activities

III Preservation of self-service, violation of professional and non-professional activities

IV Disruption of all activities

DIAGNOSIS

In a blood test, ECT, rheumatoid factor (rheumatoid factor), platelet count, etc. are checked. The most advanced analyzes are antibody titers to citrulline-containing cyclic peptide, anti-CCP. The specificity of this indicator is about 90%, although it is present in 79% of the blood serum of patients with rheumatoid arthritis.

Diagnostically important clinical signs are the absence of skin discoloration on the surface of inflamed joints, the development of tendon synovitis of the fingers or fingers, amyotrophy, the formation of typical palmar deformities (“rheumatoid wrist”).

The criteria for a negative forecast are:

Involvement of new joints with subsequent strengthening;

Systemic features of the disease;

Continuous disease activity in the absence of remission for more than a year;

Steady growth of ECHT;

Early radiological changes in the affected joints (up to four months) - the rapid development of destructive changes;

be a carrier of HLA-DR4 antigens;

Intolerance to essential drugs.

SYMPTOMS

Rheumatoid arthritis can start in any joint, but most often it starts in the small joints of the fingers, hands, and wrists. Usually the damage to the joint is symmetrical, which means that if the joint of the right hand hurts, then the joint of the left hand should also hurt. The more affected the joints, the more advanced the stage of the disease.

Other general symptoms:

Fatigue, asthenovegetative syndrome;

Morning traffic restriction. As a rule, the longer the restriction lasts, the more active the disease.

Weakness;

flu-like symptoms, including a slight fever;

Pain with prolonged sitting;

Outbreaks increase during remission;

Muscle pain;

Loss of appetite, depression, weight loss, anemia, sweating and/or swelling of the palms and soles;

Dysfunction of the salivary and lacrimal glands, leading to their deficiency.

TREATMENT OF RHEUMATOID ARTHRITIS

In the presence or suspicion of an infection (tuberculosis, yersiniosis, etc.), therapy with an appropriate antibacterial drug is required. In the absence of striking manifestations outside the joints (for example, high fever, Felty's syndrome, or polyneuropathy), treatment of articular syndrome begins with the selection of non-steroidal anti-inflammatory drugs (NSAIDs). At the same time, corticosteroids are prescribed for most inflamed joints. The immunocomplex nature of the disease dictates courses of plasmapheresis, which in most cases have a significant effect. The instability of the results of this therapy is an indication for the addition of basic funds. These

drugs have a slow action, so they should be used for at least 6 months, and with a positive effect of therapy, their treatment will continue (for years).

In the treatment of rheumatoid arthritis, the prevention of osteoporosis plays an important role in restoring the disturbed calcium balance by increasing absorption in the intestine and reducing its excretion from the body. An important component of measures to combat osteoporosis is a diet high in calcium. Sources of calcium include dairy products (especially hard cheeses containing 600-1000mg of calcium per 100g, as well as processed cheeses; less calcium is found in cottage cheese, milk and sour cream), almonds, walnuts and walnuts, and Vitamins. Calcium supplements are listed in combinations with D or its active metabolites.

Therapeutic exercise, aimed at ensuring maximum joint mobility and maintaining muscle mass, is essential in treatment.

Physiotherapeutic procedures and spa treatment are of secondary importance and are used only with a low severity of arthritis.

With stable mono- and oligorarthritis, synovectomy is performed by introducing an isotope of gold, yttrium, etc. into the joint or by surgery. Reconstructive surgeries are performed with stable deformities of the joints.

MODERN THERAPY

Systemic drug therapy involves the use of four groups of drugs:

Symptomatic treatment includes non-steroidal anti-inflammatory drugs (NSAIDs) and glucocorticosteroids (GCS),

Major antirheumatic drugs

Genetically engineered biological (to fight diseases) drugs with cytostatic immunosuppressants.

NOSTEROID ANTI-INFLAMMATORY

Modern NYQPs have significant anti-inflammatory effects by inhibiting the activity of cyclooxygenase (SOG), a key enzyme in arachidonic acid metabolism. Of particular interest is the discovery of two isoforms of SOG, described as SOG-1 and SOG-2, which play different roles in the regulation of prostaglandin (PG) synthesis. NYQPs have been shown to inhibit the activity of SOG isoforms, but their anti-inflammatory activity is due to inhibition of SOG-2.

Many known NYQPs primarily inhibit SOG-1 activity, which explains complications such as NYQP-induced gastropathy (especially erosions and ulcerations), renal dysfunction, encephalopathy, and hepatotoxicity.

Thus, depending on the nature of the blocking of SOG, NYQP is divided into SOG-2 inhibitors ("coccyx") and non-selective.

Relatively selective SOG-2 inhibitors include meloxicam, lornoxicam, and other representatives of oxicam and nimesulide. Representative highly selective SOG-2 inhibitors are celecoxib and etoricoxib. This drug has a minimal side effect on the gastrointestinal tract, while maintaining high anti-inflammatory and analgesic properties. SOG-2 inhibitors can be used in all rheumatoid arthritis treatments, requiring the use of NYQP in all programs. At the beginning of therapy with an active inflammatory process, meloxicam is prescribed at a dose of 15 mg per day, and then, as maintenance therapy, the dose is reduced to 7.5 mg per day. Nimesulide is prescribed at a dose of 100 mg 2 times a day.

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Celecoxib, a special COG-2 inhibitor, is prescribed at a dose of 100-200 mg 2 times a day. There is no need to select the dosage of the drug for the elderly. However, in patients with body weight below average (50 kg), it is recommended to start treatment with the lowest recommended dose.

The simultaneous use of two or more NYQPs should be avoided, as their effectiveness will remain unchanged and side effects will increase.

GLUCOCORTICOSTEROIDS

The use of a combination of high-dose corticosteroids (“pulse therapy”) and slow-acting drugs (increasing the effectiveness of corticosteroids) is a new approach: the combination of methotrexate with aminoquinoline drugs, gold salts, sulfasalazine, and the selective immunosuppressant cyclosporine.

Corticosteroids are used with a high activity of the inflammatory process, with systemic manifestations of rheumatoid arthritis - pulse therapy (only corticosteroids or cytostatic - in combination with cyclophosphamide), and in the absence of systemic manifestations - in the form of a course of treatment. Corticosteroids are also used as maintenance anti-inflammatory therapy when other medications have failed.

In some cases, GCS is used as a local therapy. Instructions for their use:

Predominantly mono- or oligoarthritis of large joints;
Prolonged exudative process in the joint;
the superiority of "local status" over systemic status;
There are recommendations against the systematic use of corticosteroids.

The drug also has a systemic effect when corticosteroids are injected into the depot joint. The drug of choice is betamethasone dipropionate ("diprospan"), which has a long-term effect.

MAIN ANTI-RHEUMATIC DRUGS

Essential drugs still play a primary role in the treatment of rheumatoid arthritis, but there is now a new approach to prescribing them. In contrast to the known therapy for rheumatoid arthritis (“pyramid principle”), aggressive treatment with basic drugs in the early stages of the disease from the moment of diagnosis is being promoted to change the course of rheumatoid arthritis and improve the quality of remission. horseback riding The reason for this is the absence of severe complications caused by pannus, deformities, osteopenia, autoimmune mechanisms in the early stages of rheumatoid arthritis, and a high probability of remission.

The main drugs for the basic therapy of rheumatoid arthritis are: methotrexate, leflunomide, sulfasalazine, gold preparations, penicillamine, aminoquinoline preparations (for example, hydroxychloroxine). Substitutes include cyclophosphamide, azathioprine, cyclosporine.

Basal drugs that are ineffective for 1.5-3 months should be replaced by others or used in combination with small doses of corticosteroids, the latter of which reduce the activity of rheumatoid arthritis before the onset of action of the basic drugs. The critical period is six months, during which an effective basic therapy should be selected.

When treating a disease with basic drugs, the activity of the disease, as well as the appearance and development of side effects, are carefully monitored.

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