

EXTRA-ARTICULAR MANIFESTATIONS OF RHEUMATOID ARTHRITIS

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Abstract. Rheumatoid arthritis (RA) is a chronic systemic autoimmune disease of connective tissue with predominant joint involvement in the form of erosive-destructive progressive polyarthritis. Felty's syndrome is a variant of RA that includes chronic polyarthritis, splenomegaly, and leukopenia.

Keywords: Rheumatoid arthritis (RA), polyarthritis, Joint damage, inflammation.

Introduction: Epidemiology: incidence 0.5-1% of the population, peak incidence in the 5th decade of life; the most disabling lesion of the joints - after 10 years from the onset of the disease 50% of patients are disabled, after 20 years - all 100%.

The etiology is unknown, the involvement of the following factors is discussed:

- a) genetic predisposition (correlates with HLA DR4, Dw4 and other HLA antigens)
- b) infectious agents (Epstein-Barr virus (increased titres to it are detected in 80% of patients), retroviruses, rubella, herpes viruses, mycoplasmas, mycobacteria are considered to be the etiological factor).

Pathogenesis of RA:

1. It is based on genetically determined autoimmune processes arising due to deficiency of T-suppressor function of lymphocytes under the influence of an unknown etiological factor. In the debut of the process is characterised by AG-specific activation of T-helper cells with hyperproduction of pro-inflammatory (Φ HO $\square$ - the most important factor, IL-1, IL-8, etc.) and relative deficiency of anti-inflammatory (IL-4, etc.) mediators, stimulation of synovial B-lymphocytes with synthesis of rheumatoid factor - altered IgM, sometimes IgG or IgA to IgG fragment of own tissues (is an autoantigen).

2. Joint damage starts with inflammation of the synovial membrane (synovitis) producing synovial fluid, then due to autoimmune inflammation panus is formed - granulation tissue originating from the inflamed synovial membrane, consisting of actively proliferating fibroblasts, macrophages, lymphocytes and very rich in blood vessels; panus grows intensively, sprouts into cartilage and destroys it with the help of a number of enzymes

3. cartilage is gradually destroyed and disappears, it is replaced by granulation tissue with the development of ankylosis, deformity of the joint, formation of bone erosions (usuras), inflammation of periarticular tissues.

Classification of RA:

- a) according to the degree of seropositivity: seropositive and seronegative
- b) according to the degree of activity: minimal, moderate, high
- c) according to joint X-ray data: I - periarticular osteoporosis, II - osteoporosis + slight narrowing of the articular gap, there may be single nodules, III - osteoporosis + sharp narrowing of the articular gap + multiple nodules, IV - osteoporosis + narrowing of the articular gap + multiple nodules + there may be bone ankyloses.

d) depending on the functional insufficiency of the joint: 0 - no functional insufficiency, I - restriction of professional ability to work, II - loss of professional ability to work, III - loss of ability to self-care.

RA clinic:

- several weeks to months of RA may be preceded by a prodromal period (malaise, fatigue, depression, etc.)

(a) Joint syndrome

- the disease begins subacutely with a gradual increase in pain and stiffness in small peripheral joints (wrist, metacarpophalangeal, proximal interphalangeal, ankle, metatarsophalangeal); almost never affect the joints of the spine, distal interphalangeal joints and joints of the big toe, proximal interphalangeal joint of the little finger (joints-exceptions).

- bilateral, symmetrical joint involvement (inflammatory synovitis)

- pain in the area of the joints for a long time, increasing with exertion and decreasing at night, stronger in the morning than in the evening

- morning stiffness of joints for more than 1 hour,

b) extra-articular manifestations of RA:

- constitutional: weakness, malaise, weight loss, subfebrile temperature

- rheumatoid nodules - accumulations of immune complexes over the affected joints or in the area of the extensor surface of the elbow bone; nodule size from 2-3 mm to 4-5 cm, painless, can be both mobile (located in the subcutaneous tissue) and immobile (located under the subcutaneous tissue).

- Cardiovascular: pericarditis, 'early atherosclerosis', digitalis arteritis (up to Raynaud's syndrome).

- pulmonary: dry pleurisy, interstitial pulmonary fibrosis

- CN lesions: compression neuropathy (compression of nerve trunks due to joint deformity), symmetrical neuropathy, multiple mononeuritis (vasa nervorum), cervical myelitis.

- Renal: amyloidosis, renal tubular acidosis, interstitial nephritis (more often due to LS)

- Haematological: anaemia, moderate leucocytosis, thrombocytosis; neutropenia in Felty's syndrome

Diagnostic criteria for RA:

At least 4 of the following criteria must be present to make a diagnosis of RA:

1. morning stiffness for more than 1 hour

2. arthritis of ≥ 3 joints (polyarthritis)

3. arthritis of the joints of the hands

4. symmetrical arthritis

5. rheumatoid nodules

6. rheumatoid factor

7. characteristic radiological changes.

These signs should last for at least 6 weeks as sometimes other arthritis can manifest in the same way.

Treatment of RA.

1. treatment should be permanent (lifelong), complex (medication + physiotherapy + sanatorium-resort + surgery as indicated), individual, stage-by-stage.

2. Drug therapy:

A. Basis therapy (slow acting drugs):

- Arava (leflunamide) (ranked 1st in efficacy, used > 5 years)

- methotrexate (2nd most effective, used > 20 years)

- gold preparations (turedone) (3rd most effective, used > 60 years)

- sulfasalazine (4th most effective, used > 50 years)
- D-penicillamine (5th most effective, used > 40 years)
- Azathioprine (6th most effective, used > 30 years)
- aminoquinoline drugs (Plaquenil) (7th most effective, used > 40 years);

All these drugs are effective in 40% of cases, 30% have side effects, and another 30% have no effect; on average, the effect of baseline therapy occurs after 2 months (Arava - 1 month). It is possible to combine baseline drugs (methotrexate + sulfasalazine + plaquenil, etc.), but only if they are pathogenetically combined.

B. Anti-inflammatory therapy:

1) NSAIDs: traditional, classical (indomethacin, diclofenac 75-150 mg/day in 2-3 doses, ibuprofen 1.2-3.2 g/day in 3-4 doses, etc.) and selective COX-2 inhibitors (less side effects: meloxicam / movalis 7.5-15 mg/day, nimesulide / nimesil / nimesil / nayz 100 mg/day in 2 doses, celecoxib / celebrex).

CONCLUSION:

Extra-articular manifestations of rheumatoid arthritis (RA) represent a significant aspect of the disease's systemic nature, affecting multiple organs and contributing to increased morbidity and mortality. These manifestations, including rheumatoid nodules, pulmonary involvement, cardiovascular complications, vasculitis, and ocular or hematologic disorders, often indicate a more severe disease course. Early recognition and comprehensive management of extra-articular features are essential to improve patient outcomes, prevent irreversible damage, and tailor individualized treatment strategies. Understanding the full spectrum of RA beyond joint involvement underscores the importance of multidisciplinary care in achieving optimal disease control and quality of life for patients.

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