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БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ
КАФЕДРА ПРОПЕДЕВТИКИ ВНУТРЕННИХ БОЛЕЗНЕЙ

ЗАБОЛЕВАНИЯ КОСТНО-МЫШЕЧНОЙ СИСТЕМЫ

DISEASES OF THE MUSCULOSKELETAL SYSTEM

Учебно-методическое пособие

Минск БГМУ 2026

УДК 616.7(075.8)=111
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З-12

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Освещены вопросы этиологии, патогенеза, классификации и клинических проявлений, изложены основные методы обследования пациентов с заболеваниями суставов, принципы лечения и профилактики.

Предназначено для студентов 3-го курса медицинского факультета иностранных учащихся, обучающихся по специальности «Лечебное дело» на английском языке.

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LIST OF ABBREVIATIONS

anti-CCP — anti-cyclic citrullinated peptide (antibodies)
CRP — C-reactive protein
DMARDs — disease-modifying antirheumatic drugs
ESR — erythrocyte sedimentation rate
GCs — glucocorticoids
HLA — human leukocyte antigen
HLA-B27 — human leukocyte antigen B27
HLA-DR4 — human leukocyte antigen DR4
MRI — magnetic resonance imaging
MTP — metatarsophalangeal (joint)
NSAIDs — nonsteroidal anti-inflammatory drugs
OA — osteoarthritis
PIP — proximal interphalangeal (joint)
RA — rheumatoid arthritis
ReA — reactive arthritis
RF — rheumatoid factor
SYSADOA — symptomatic slow-acting drugs for osteoarthritis
WBC — white blood cells

EXPLANATORY NOTE

Total duration of classes is 3,0 hours.

The examination of patients with musculoskeletal system diseases is based on the general principles of clinical examination and includes: studying of the patient's complaints; present and past medical history (*anamnesis morbi and vitae*); objective examination data (inspection, palpation,); the results of laboratory and instrumental investigations. Often, diseases of internal organs lead to the appearance of arthralgias; on the other hand, in many joint diseases (RA, infectious arthritis, OA, gouty arthritis), internal organs are affected. This educational manual is devoted to the symptomatology, diagnosis, treatment principles, and prevention of RA, infectious arthritis, OA and gouty arthritis.

The purpose of the class to teach students definitions, the most common clinical manifestations, diagnostic criteria, principles of treatment and prevention of RA, infectious arthritis, OA, and gout.

Control questions:

1. RA: definition, etiology and pathogenesis, clinical manifestations, laboratory and instrumental investigations, principles of treatment and prevention.

2. OA: definition, etiology and pathogenesis, clinical manifestations, laboratory and instrumental investigations, principles of treatment and prevention.

3. Infectious arthritis (reactive and infectious (septic) arthritis): definition, etiology and pathogenesis, clinical manifestations, laboratory and instrumental investigations, principles of treatment and prevention.

4. Gouty arthritis: definition, etiology and pathogenesis, clinical manifestations, laboratory and instrumental investigations, principles of treatment and prevention.

TERMINOLOGY AND SYMPTOMS OF JOINT INVOLVEMENT

Arthralgia — pain arising in a joint.

Arthritis — inflammation of a joint, defined by the five cardinal signs of inflammation: swelling, increased local temperature, redness, pain, and loss of function.

Monoarthritis — arthritis of only one joint.

Oligoarthritis — arthritis of two or three joints.

Polyarthritis — arthritis of more than three joints.

Synovitis — clinically apparent inflammation of a joint that has a synovial membrane in its structure.

Tendinitis — inflammation of a tendon.

Tenosynovitis — inflammation of the tendon sheath.

Chondropathy — a process leading to the loss of cartilage.

Enthesopathy — inflammation/damage of the enthesis (the site where tendons and ligaments attach to the bone).

Pain — the location, character, radiation, and factors that aggravate or relieve the pain are determined.

Stiffness — a subjective feeling of impediment to movement. It is most pronounced immediately after waking up or after a period of rest. The duration and severity of morning stiffness reflect the degree of local inflammation.

Swelling — can be caused by an accumulation of fluid in the joint cavity or by involvement of the periarticular soft tissues.

Joint deformity is an abnormal change in a joint's shape, size and position. It can be caused by changes in the soft tissues (synovial membrane, muscles, ligaments, and periarticular tissues) or by changes in bons and cartilages.

Joint position — most often, the position in which the joint pain is minimal. For example, in synovitis of the knee joint, it is a semi-flexed position.

Tenderness — pain elicited by palpation. Local tenderness is an important symptom when examining joints. Tenderness over the entire joint area indicates

its inflammation, while tenderness along the joint line, for example in the knee, is characteristic of meniscopathy or OA.

Increased temperature — one of the cardinal signs of inflammation. It can be assessed by the dorsum of the physician's hand in comparison with the skin temperature above and below the affected area.

Muscle atrophy — a frequently observed symptom in joint involvement. A quantitative measure can be a comparative assessment of muscle strength using a dynamometer.

Ankylosis — complete immobility in the affected joint. Ankylosis is classified as bony (true) or fibrous (false).

Limitation of joint mobility (joint restriction) can be a consequence of joint pain or a result of scar contracture of muscles and their tendons due to myositis, inflammation of tendons and their sheaths, or injuries.

Crepitus — an audible or palpable crunching sensation that is present throughout the movement of the affected structure. Coarse crepitus can be heard at a distance or through the bone. It usually reflects damage to the cartilage or the bone itself.

Joint instability is determined by demonstrating excessive mobility during joint loading. The findings should be compared with the joint on the opposite side.

RHEUMATOID ARTHRITIS

Definition. Rheumatoid arthritis (RA) is a chronic systemic autoimmune disease of the connective tissue, characterized primarily by damage to the peripheral joints with the development of erosive-destructive changes and ankylosis.

Etiology of RA remains unknown. Predisposing factors are the following:

1. Infections (Epstein-Barr virus, parvovirus B19, etc.).

2. Genetic factors, carriage of the histocompatibility antigen HLA-DR4. There is a certain association between the HLA-DR4 locus and the severity of RA, as well as with the overproduction of RF and the rapid development of erosive changes in the joints.

3. Contact with mineral oils (motor or hydraulic). Experiments have shown that these oils possess arthritogenic (i. e., joint inflammation-inducing) properties.

4. Smoking, excessive coffee consumption, high body mass index, stress.

Pathogenesis. Mechanism of RA development includes:

1. At the onset of the process, immune cells attack the healthy synovial membrane of the joint, resulting in subsynovial edema (accumulation of lymphocytes, polymorphonuclear leukocytes, monocytes, and plasma cells in the synovial membrane).

2. Immune cells produce protective proteins — cytokines — which promote the proliferation of blood vessels in the synovial membrane.

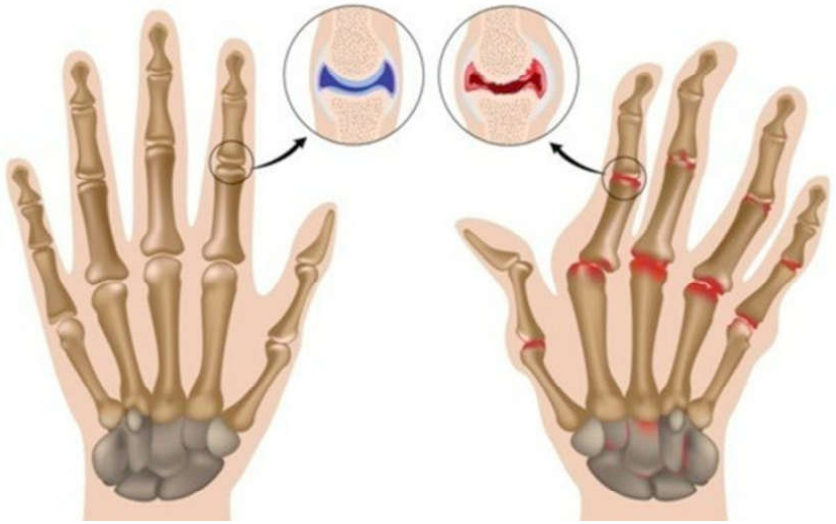
3. Excessive blood supply leads to excessive tissue growth and thickening of the synovial membrane (the thickened tissue is called «pannus»).

4. Pannus cells release proteolytic enzymes that destroy cartilage (fig. 1).

5. Due to the overproduction of pro-inflammatory cytokines (TNF-alpha), osteoclasts (bone cells that break down old structure) are activated, leading to bone damage and destruction of bone tissue with the formation of erosions.

6. Bone erosions also form due to the activation of fibroblasts (cells of loose connective tissue), which, by producing enzymes, destroy articular cartilage.

7. As the disease progresses, the pannus transforms into mature fibrous tissue, leading to the fusion of joint surfaces.



a ***b***

Fig. 1. Joint changes in RA:
a — healthy joints; *b* — RA

Clinical manifestations. Main symptoms (complaints) are the following:

– pain in the small joints of the hands and feet;
– morning stiffness;
– limited joint mobility;
– deformation.

RA is characterized by symmetrical involvement of the metacarpophalangeal (MCP), proximal interphalangeal (PIP), and metatarsophalangeal (MTP) joints. Subsequently, pathological changes are observed in the wrists, knees, shoulders, hips, elbows, ankles, tarsal joints, cervical spine, and temporomandibular joints.

In the early stage, **joint pain** occurs with movement, but as the disease progresses, it is also present at rest. This pain typically bothers patients in the

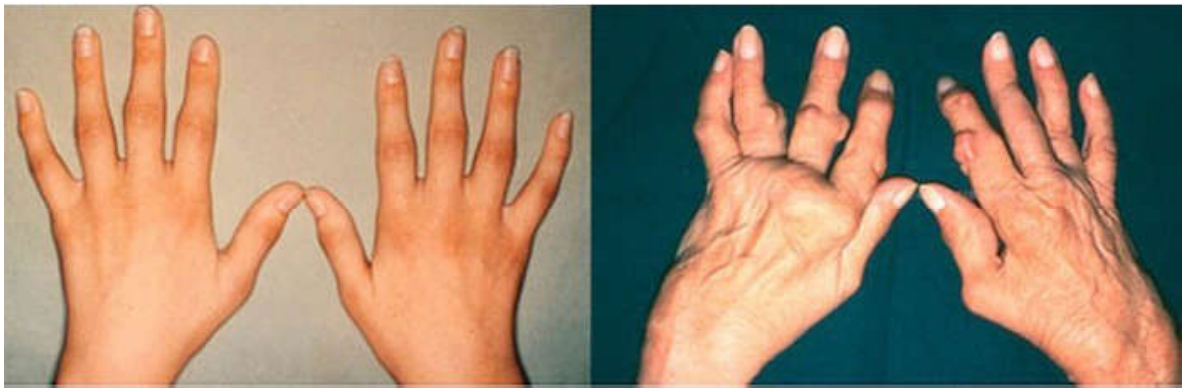
morning and decreases towards evening. Swelling and redness of the skin over the joints appear, followed by stiffness during movement and, later, impaired joint function. Later in the process, muscles and bones, tendons, and joint capsules become involved. Muscle atrophy develops on the dorsal surface of the hands.

Myalgias or muscle pain occur when muscle tissue is affected. Patients may also experience pain in the tendons and ligaments, particularly at the sites where tendons attach to bones (entheses).

Patients report **morning stiffness** in the joints. This is explained by a disruption in the circadian rhythm of hormone production by the adrenal glands, with its peak shifting to a later time of day, and by the accumulation of cytokines in the edema fluid of inflamed joints overnight.

The duration of morning stiffness can range from a few minutes (referred to as joint «tightness») to several hours. It manifests as an inability to flex and extend the joints fully («like wearing tight gloves»). The severity of stiffness is clearly correlated with the level of inflammation. Morning stiffness may decrease after movement.

On *physical examination*, one can observe changes in joint shape (fig. 2), swelling, discoloration of the skin over the joint, and limited range of motion.



a ***b***

Fig. 2. Changes in the joints of the hand in RA:
a — initial stage RA; *b* — end-stage RA

Joint deformities appear as a result of the inflammatory process spreading to the articular cartilage and bone segments, as well as the development of contractures in nearby muscles. Due to the stretching of the joint capsule and ligaments, joint subluxations develop.

Ankyloses form as a result of cartilage destruction and the formation of connective tissue (fibrous ankylosis) and later bony elements between the subchondral bone structures of both epiphyses, ultimately fixing the joint. The developing ankyloses significantly limit the patient's mobility and lead to severe functional joint impairment.

Joint deviations occur as a result of an angle forming between two adjacent bones. This happens due to the development of subluxations and prolonged contracture of individual muscle groups, which over time become irreversible. In patients with RA, ulnar deviation — also known as «**Ulnar drift**» — is often detected. This condition involves the deviation of the fingers of the hands toward the ulna (fig. 3).

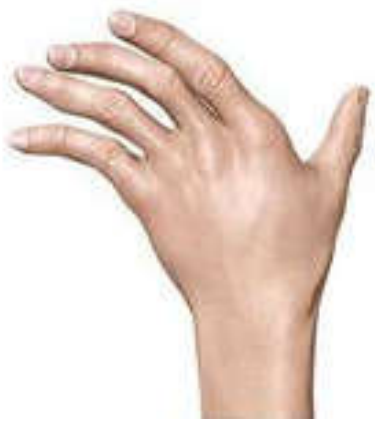


Fig. 3. Ulnar drift

Other deformities are in the advanced stage of RA.

«Spider claw» or spider-like hands — the patient cannot place the palm flat on a table surface due to an inability to fully extend the fingers.

«Swan-neck» deformity — the base of the finger is bent, the middle joint is straightened (hyperextended), and the fingertip is bent (fig. 4).

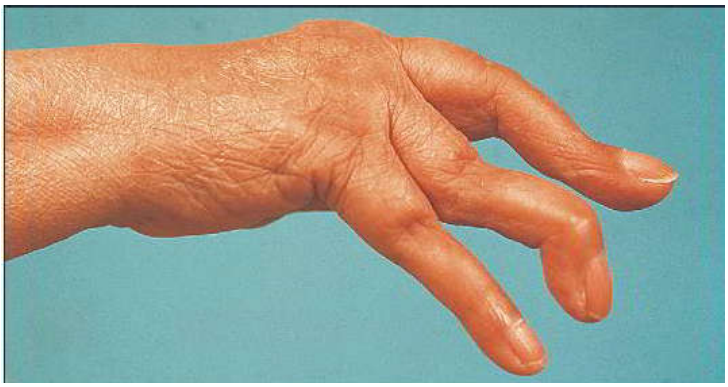


Fig. 4. «Swan-neck» deformity

«Boutonnière» deformity — the middle finger joint is bent, and the end joint is hyperextended (fig. 5).

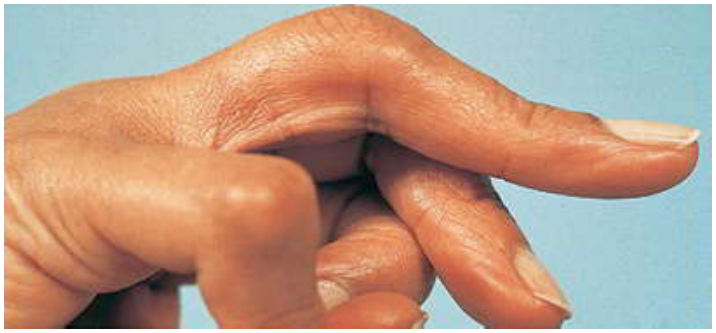


Fig. 5. «Boutonnière» deformity

Changes in the hand joints lead to significant impairment of its function. Patients are unable to perform routine actions such as lifting a kettle, holding a cup, turning a key in a door, dressing independently, etc.

In RA patients, rheumatoid nodules form on tendons, causing severe pain during finger flexion. The developing contractures further disrupt hand function.

Extraarticular manifestations of rheumatoid arthritis

Cardiovascular System. Symptoms can be caused both by the inflammatory process in the heart itself and by the complication of RA — early development of atherosclerosis. Cardiovascular disease is the leading cause of mortality in RA, with a mortality rate 1.5- to 3.0-fold higher than that in the general population. Cardiac manifestations may include pericarditis, coronary artery vasculitis, myocarditis, Coronary Heart Disease.

Lung Involvement, which is common in RA, manifests as pleuritis (inflammation of the pleural membranes), interstitial lung disease, obliterative bronchiolitis, rheumatoid nodules in the lungs (Caplan's syndrome).

Ocular Involvement in RA presents as dry keratoconjunctivitis (Sjögren's syndrome), episcleritis, scleritis, peripheral ulcerative keratopathy (fig. 6).

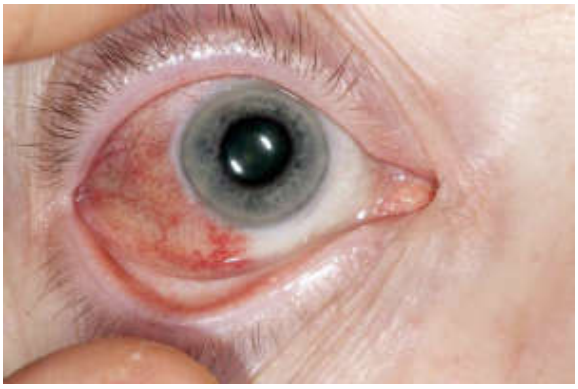


Fig. 6. Ocular involvement in RA

Nervous System. Most complications are a consequence of articular inflammation that compresses or invades the adjacent spinal cord, peripheral nerve, or neural tissues. Neurological manifestation may be as follows: cervical myelopathy (it results from arthritis of the atlantoaxial joint), compression and non-compressive neuropathy (e. g., carpal tunnel syndrome), stroke.

Other extraarticular manifestation include *kidney* (nephritis), *skin* (rheumatoid nodules, livedo reticularis (a mottled, net-like purplish discoloration of skin), *Sjögren's syndrome* (immune-mediated damage to moisture-secreting glands, it leads to dryness and inflammation on mucous membranes).

Functional Impairment Classes (degree of impairment of joint function) see Appendix 1.

Criteria for the diagnosis of RA see Appendix 2.

Principles of treatment

Goals. Reduction or elimination of arthritis symptoms and extra-articular manifestations, control of inflammatory activity, prevention of progression of bone and joint destruction, preservation and significant improvement of quality of life, increasing life expectancy to the average population level.

Disease-modifying antirheumatic drugs (DMARDs) are the main component of RA treatment (e. g., Methotrexate, Leflunomide, Sulfasalazine). They modify the disease course by suppressing the underlying immune dysfunction and should be initiated as early as possible.

Corticosteroids (Prednisone, Methylprednisolone) have both anti-inflammatory and immunoregulatory activity. They can be given orally, intravenously, intramuscularly or can be injected directly into the joint. Corticosteroids are useful in early disease as temporary adjunctive therapy while waiting for DMARDs to exert their anti-inflammatory effects. Corticosteroids are also useful as chronic adjunctive therapy in patients with severe disease that is not well controlled by other medicines. Intra-articular corticosteroids are effective for controlling a local flare in a joint without changing the overall drug regimen.

Biological therapy for RA are genetically engineered, targeted medicines that inhibit specific immune system pathways, such as TNF-alpha, IL-6, or B-cells, to reduce inflammation and joint damage.

Symptomatic therapy includes nonsteroidal anti-inflammatory drugs (NSAIDs, e. g. Ibuprofen, Paracetamol, Naproxen, Meloxicam, etc.). Their characteristic feature is the rapid onset of therapeutic effect, actively suppressing pain and inflammation. It is important to note however that these drugs alone do not change the course of the disease of RA or prevent joint destruction. Surgical approaches can be useful at late stage of RA (joint arthroplasty).

Prevention. Life-style modification: regular physical activity, dietary adjustments, smoking cessation, alcohol limitation, joint protection (using assistive devices and learning joint-friendly techniques).

OSTEOARTHRITIS

Definition. Osteoarthritis (OA) is a type of degenerative joint disease that results from breakdown of joint cartilage and underlying bone. The condition is based on the involvement of all joint components, primarily cartilage, as well as the subchondral bone, synovial membrane, ligaments, capsule, and periarticular muscles.

Etiology. According to current understanding, OA is a multifactorial disease. Several factors contribute to the development of degenerative changes in articular cartilage, among which two main ones can be distinguished:

– excessive mechanical and functional overload of the cartilage;
– decrease in its resistance to normal physiological load.

As a result, degeneration and destruction of cartilage develops.

Risk factors for the OA:

1. Genetic predisposition.
2. Obesity.
3. Professional, athletic, or domestic overload.
4. Trauma.
5. Patient age over 50 years.
6. Reduced levels of sex hormones.
7. Other joint diseases.

OA is classified into primary (idiopathic) and secondary (resulting from dysplasia, arthritis, trauma, hypermobility, etc.).

Pathogenesis. Due to the influence of various etiological factors, disturbances in the metabolism and synthetic activity of chondrocytes occur, as well as physicochemical damage to the matrix of the articular combination of biomechanical stress, low-grade chronic inflammation, and metabolic changes lead to the degradation of articular cartilage, subchondral bone remodeling (osteophytes), and synovial inflammation (table 1, fig. 7).

Table 1

Radiographic grade of cartilage loss

Grade 1	Mild	Minor cartilage wear
Grade 2	Moderate	Joint space narrowing. Formation of bone spurs (osteophytes)
Grade 3	Severe	Significant cartilage loss. Pronounced joint inflammation
Grade 4	End-stage	Bone-on-bone contact due to near-complete cartilage loss

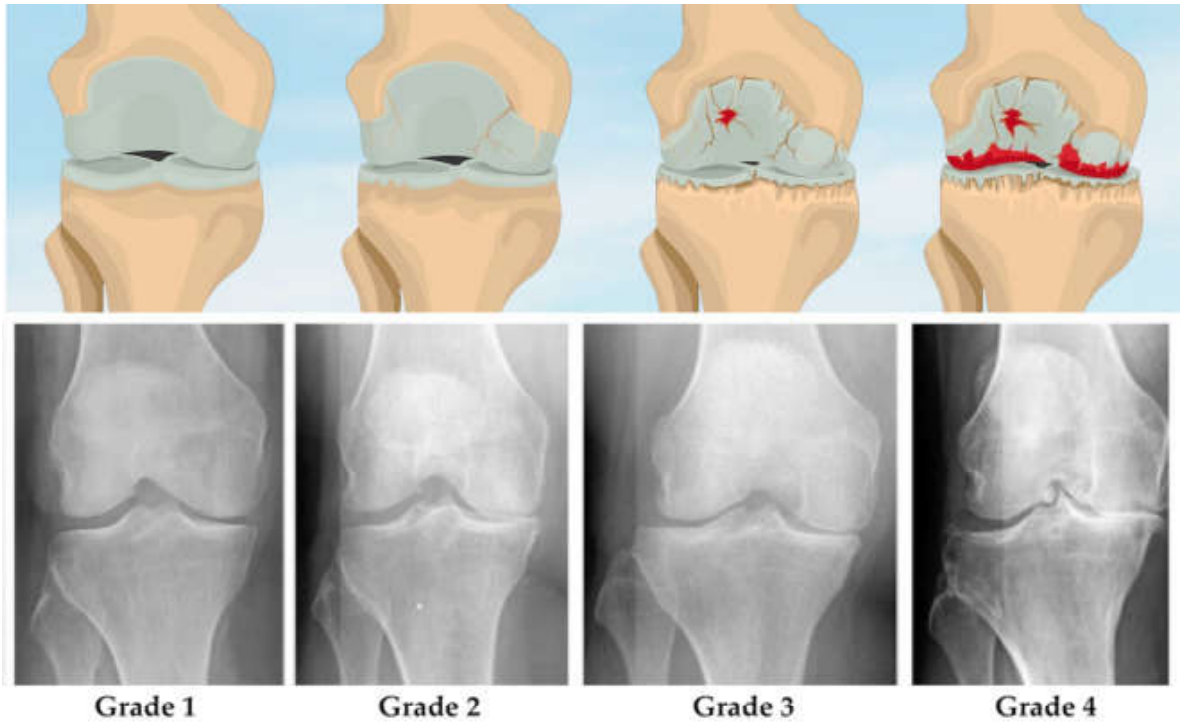


Fig. 7. Stages of OA development

Clinical manifestation. The most commonly affected joints in OA are the knees, hips, hands (especially the distal and PIP joints and the base of the thumb), spine (especially the cervical and lumbar spine), and feet (especially the first MTP joint). Main symptoms (complaints) are the following:

- pain in joint;
- crunching (crepitus);
- swelling;
- stiffness;
- limited joint mobility;
- joint deformation.

The primary clinical symptom of OA is pain in the affected joints of a mechanical nature, which occurs during loading and is usually absent at rest. Initially, the pain occurs with significant stress (prolonged walking, lifting heavy objects, maintaining a forced body position for a long time) and subsides quickly with rest or after eliminating the cause of joint overload.

However, as OA progresses, the pain becomes more intense, longer-lasting, occurs with any movement, does not disappear completely at rest, and can even bother the patient at night. Since articular cartilage is not innervated and therefore not sensitive to pain, its occurrence is associated with the development of pathological changes in the non-cartilaginous structures of the joint. Most often, the causes of pain in OA are reactive synovitis, periarthritis, and spasm of nearby muscles.

Even in the early stages of the disease, **joint deformity** may be noted, caused by synovial or periarticular edema, as well as the presence of intra-articular effusion. As the disease progresses, articular **crepitus** turns into a coarse crunch, accompanied by increasing pain and joint stiffness, although the pain does not reach the intensity seen in RA.

Periodically, **swelling** occurs in the joint area, accompanied by an increase in skin temperature, intensification of the pain syndrome, and prolonged morning **stiffness** (reactive synovitis). Synovitis is often accompanied by signs of tendobursitis, manifesting as a small, localized swelling and tender points at the site of tendon attachment to the joint, as well as pain during specific movements associated with the contraction of the affected tendon. Upon palpation of the affected joint, tenderness is detected, especially along the joint line, usually moderate in severity. In the presence of synovitis, swelling in the joint area and increased skin temperature are noted. Sometimes a small amount of fluid can be detected within the joint cavity.

Patients often exhibit atrophy of regional muscles. With joint deformity, trophic changes of the skin, such as dryness and thinning, are sometimes observed.

Gradually, **joint deformity** arises and increases due to thickening of the synovial membrane and joint capsule, formation of osteophytes, destruction of cartilage and bone with remodeling (alteration of shape) of the articular surfaces, and the occurrence of subluxations.

Significant changes in the bone-cartilage structures and soft tissues of the joint, along with severe pain syndrome, can be accompanied by substantial limitation of joint mobility and a forced position of the limb, even in the absence of bony ankylosis.

The progression of the disease is accompanied by **limitation of movement in the joints**, which can be caused by the presence of pain syndrome, muscle spasm, the formation of tendon-muscle contractures, the development of osteophytes, the presence of joint «mice» (loose bodies), and the occurrence of subluxations.

Upon *palpation* of the affected joint, tenderness is detected, especially along the joint line, usually moderate in severity. Initially, skin color and temperature are normal, but in the presence of synovitis, swelling in the joint area and increased skin temperature are noted. Sometimes a small amount of fluid can be detected within the joint cavity.

Patients often exhibit atrophy of regional muscles. With joint deformity, trophic changes of the skin, such as dryness and thinning, are sometimes observed.

A characteristic feature of OA is limited joint mobility and a forced position of the limb; however, complete absence of movement is not observed. With prolonged disease, distinct joint deformity becomes evident.

Principles of treatment

Non-pharmacological treatment:

1. Exercise: physical exercise for OA helps reduce pain and maintain joint functional activity.

2. Diet: weight loss slows down the progression of OA. Patients should be guided toward maintaining a normal body weight from the standpoint of mechanical joint unloading.

3. Knee orthoses (braces): used in the early stages of the disease, and specifically for deformities — varus and valgus variants — providing biomechanical correction of up to 20°.

Pharmacological treatment (main medications):

1. NSAIDs short courses should be used intermittently.

2. Symptomatic slow-acting drugs (SYSADOA, chondroprotectors) are therapeutic agents — commonly chondroitin sulfate, glucosamine, and hyaluronic acid — designed to slow the progression of OA, reduce joint pain, and protect cartilage by stimulating synthesis and inhibiting degradation.

3. Glucocorticoids (locally, long-acting in case of severe inflammation).

4. Intra-articular injections of sodium hyaluronate (for both large and small joint involvement).

5. Antidepressants (in case of chronic severe pain).

Surgery: arthroscopy (minimally invasive removal of damaged tissue and osteophytes from joint), joint replacement (fig. 8).

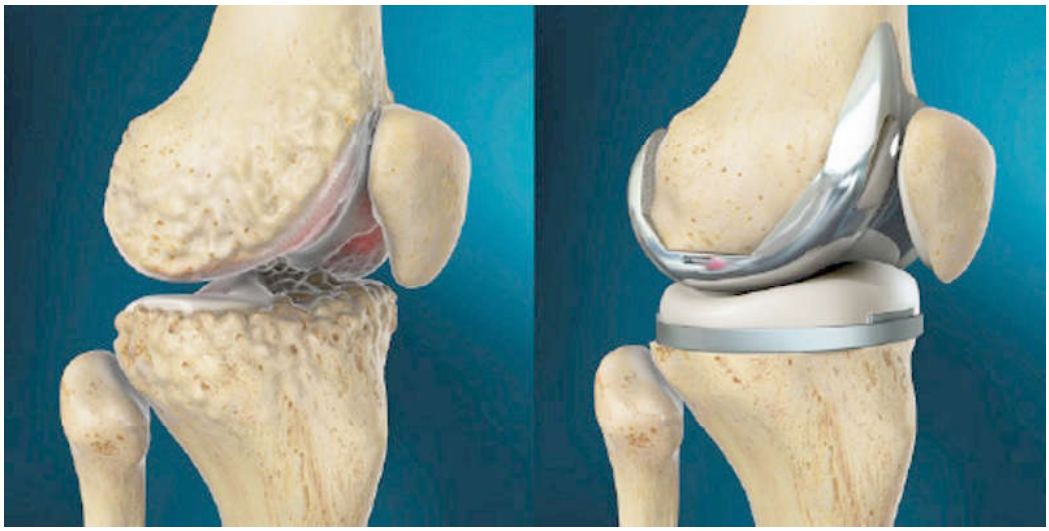


Fig. 8. Joint replacement

Prevention. Life-style modification: maintaining a healthy body weight is a cornerstone of prevention, as it significantly reduces the chronic mechanical stress on weight-bearing joints like the knees and hips. This is complemented

by regular, sensible physical activity such as swimming, cycling, and walking. Equally important is the mindful protection of joints from injury by using proper techniques during sports and ensuring adequate rehabilitation after any joint trauma to prevent future instability.

INFECTIOUS ARTHRITIS

Definition. Infectious (septic) arthritis is an acute inflammatory arthritis caused by direct invasion of the joint by microorganisms, most commonly bacteria, leading to purulent synovitis, rapid cartilage destruction, and systemic inflammatory response.

Etiology. The most frequent pathogen in adults is Staphylococcus aureus, but streptococci, Gram-negative bacilli, Neisseria gonorrhoeae, mycobacteria, fungi, and viruses may also be involved.

Pathogenesis. Microorganisms reach the joint via hematogenous spread, direct inoculation (trauma, intra-articular injection, surgery), or contiguous spread from adjacent infected tissues.

The pathogenesis involves rapid bacterial proliferation in synovial fluid, activation of the innate immune system, and massive neutrophilic infiltration with release of proteolytic enzymes and cytokines, causing increased intra-articular pressure and destruction of cartilage and subchondral bone.

Risk factors: age (> 80 years), diabetes mellitus, chronic kidney disease, immunosuppression, pre-existing RA, prosthetic joints, recent joint surgery or intra-articular injections, intravenous drug use, alcoholism, skin infections, and joint trauma.

Clinical manifestation. Infectious arthritis usually presents with an abrupt onset over hours to days of intense joint pain, marked swelling, erythema, and inability to move or bear weight on the affected joint.

Systemic symptoms such as high fever, chills, and profound malaise are common, and sepsis may develop in severe cases.

A large joint (most often the knee, but also hip, shoulder, or ankle) is typically involved as an acute monoarthritis, although oligo- or polyarticular involvement can occur in high-risk patients.

On examination, the joint is markedly swollen, hot, and erythematous, with extreme pain on both active and passive movement and an antalgic position; systemic signs of infection (fever, tachycardia) are often present (fig. 9).

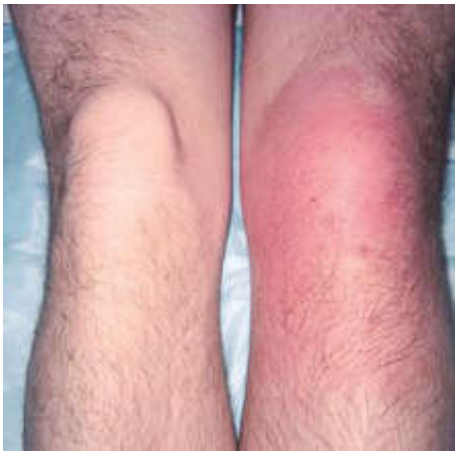


Fig. 9. Infectious arthritis of the left knee

Laboratory tests. Blood test: elevated ESR and CRP, leukocytosis with neutrophilia, and sometimes increased procalcitonin. Blood cultures are important and frequently positive if obtained before antibiotics.

Synovial fluid analysis is crucial: the fluid is typically turbid or frankly purulent, with very high leukocyte counts (often > 50 · 10^9/L) and neutrophil predominance. Gram stain and synovial fluid culture usually identify the causative organism and guide targeted antimicrobial therapy.

Instrumental tests. X-ray of joints: may initially show only soft tissue swelling but, if diagnosis or treatment is delayed, can later demonstrate joint space narrowing, periarticular osteopenia, marginal erosions, and frank joint destruction (fig. 10).

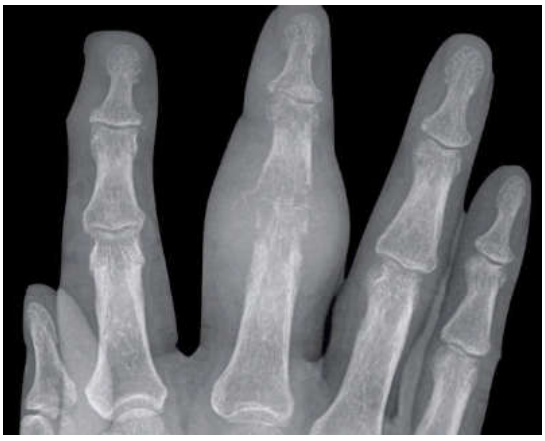


Fig. 10. X-ray of the 3rd finger

Ultra sound: joint effusion, synovial thickening, guidance for aspiration.

MRI helps to evaluate the extent of infection in bone (osteomyelitis) and surrounding soft tissues, which is especially important for deep joints such as the hip.

Principles of treatment

Non-pharmacological treatment:

1. Immediate diagnostic and therapeutic joint aspiration with adequate drainage (repeated needle aspiration, arthroscopic lavage, or open surgical drainage) to remove purulent material and reduce intra-articular pressure.

2. Short-term immobilization of the affected joint in the acute phase to relieve pain, followed by early mobilization and physiotherapy to prevent stiffness and contractures.

3. Comprehensive management of sepsis and optimization of comorbidities (e. g. diabetes, chronic kidney disease).

Pharmacological treatment:

1. Prompt initiation of empiric intravenous antibiotic therapy targeting the most likely pathogens, started immediately after synovial fluid sampling.

2. Subsequent adjustment of the antibiotic regimen according to culture and sensitivity results, with adequate duration of therapy based on clinical response and pathogen.

REACTIVE ARTHRITIS

Reactive arthritis (ReA) is the post-infective type of arthritis, triggered by an infection elsewhere in the body, such as the gut or genitals. Differences between reactive and infectious (septic) arthritis see at Appendix 3.

Definition. ReA is a sterile inflammatory arthritis that usually develops 1–4 weeks after an extra-articular urogenital or intestinal infection, without viable pathogens present in the joint cavity.

Etiology. It is typically triggered by infections such as Chlamydia trachomatis (urogenital), enteric pathogens including Salmonella, Shigella, Yersinia, and Campylobacter. Nasopharyngeal: less common, but can be caused by streptococci, staphylococci, and viruses (HIV).

Pathogenesis. The pathogenesis is based on molecular mimicry and a cross-reactive immune response in which T-cells and antibodies directed against microbial antigens also target joint and entheseal tissues, leading to aseptic synovitis and enthesitis. A key role in this process is played by genetic predisposition, particularly the HLA-B27 gene, and cytokine imbalance.

Risk factors: a recent urogenital or enteric infection, young age, male, unprotected sexual activity.

Clinical manifestation. The onset is generally subacute, with arthritis appearing 1–4 weeks after the triggering infection.

Articular: ReA usually presents as an asymmetric oligoarthritis of the lower limb joints (knees, ankles, joints of the feet), often with dactylitis («sausage digit»),

enthesitis (e. g. Achilles tendon, plantar fascia), and possible involvement of the sacroiliac joints and spine (fig. 11).

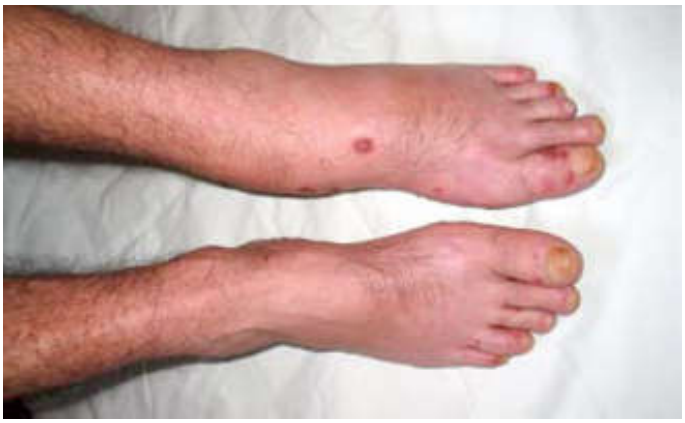
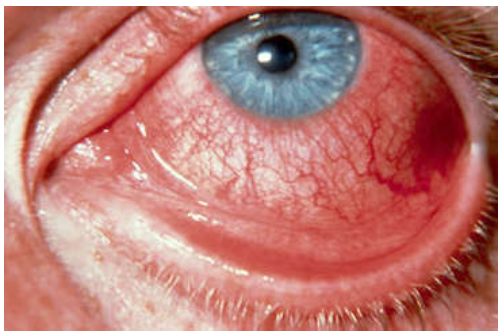


Fig. 11. ReA. Articular involvement — swelling of the left ankle

On physical examination, there are asymmetric swollen and tender joints of the lower limbs, dactylitis, and enthesitis, with limited spinal motion if sacroiliitis is present.

Extra-articular manifestations such as urethritis or cervicitis, conjunctivitis or uveitis, mucocutaneous lesions, and low-grade fever (37,1 to 38,0 °C) are common (fig. 12).

a



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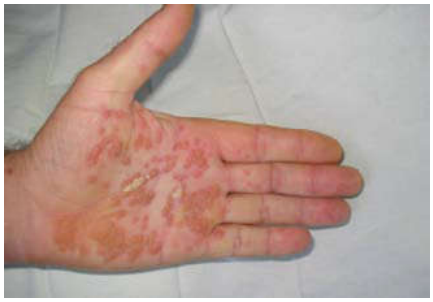


c

Fig. 12. Extra-articular manifestations:
a — urethritis; *b* — uveitis; *c* — keratoderma

Laboratory tests. Blood test: moderately elevated inflammatory markers (ESR, CRP). HLA-B27 is often positive, and laboratory evidence of the triggering infection can be obtained using PCR or serology for Chlamydia and enteric pathogens.

Synovial fluid analysis reveals an inflammatory profile with elevated WBC count and neutrophil predominance, but cultures remain negative, confirming the sterile nature of the process.

Instrumental tests. X-ray of joints — may be normal in early disease; in later disease ill-defined erosions, bone proliferation but also juxta-articular osteoporosis and enthesopathy/enthesitis, uniform joint space narrowing.

Ultrasound can demonstrate synovitis, joint effusion, enthesitis, and dactylitis.

MRI is useful for early detection of sacroiliitis by visualizing bone marrow edema and inflammatory changes in the sacroiliac joints.

Principles of treatment

Non-pharmacological treatment: physiotherapy and patient education are integral components of management.

Pharmacological treatment:

1. Treatment of the triggering infection, especially Chlamydia trachomatis (appropriate antibiotic therapy when indicated).
2. NSAIDs as first-line drugs to relieve pain and reduce inflammation.
3. Intra-articular glucocorticoid injections for severe local synovitis.
4. Conventional synthetic DMARDs (e. g. sulfasalazine, methotrexate) in persistent or chronic ReA.

GOUTY ARTHRITIS

Definition. Gouty arthritis (gout, GA) is a disease associated with disordered purine metabolism, characterized by increased uric acid levels in the blood (hyperuricemia) and deposition of monosodium urate crystals in articular and/or periarticular tissues, kidneys, and other organs.

Etiology. Hyperuricemia may result from increased uric acid production, decreased renal excretion, or a combination of these mechanisms. In most patients (more than 90 %), insufficient uric acid excretion in response to purine load is explained by a congenital, isolated defect associated with decreased tubular excretory capacity. In some patients (so-called hyperproducers), uric acid formation is increased due to metabolic disorders of unknown origin; the main sources are the purine bases adenine and guanine.

Primary hyperuricemia is usually idiopathic. Secondary hyperuricemia and gout develop in the setting of:

– Chronic Kidney Disease with reduced glomerular filtration;

– long-term use of diuretics and other drugs (e. g., low doses of acetylsalicylic acid, cytostatics);

– chronic lead intoxication (saturnine gout, lead-induced gout);

– oncohematological diseases and conditions with high cell turnover.

Primary hyperproduction of uric acid is rarely (less than 1 % of cases) associated with inherited defects of enzymes involved in purine metabolism, such as hypoxanthine-guanine phosphoribosyltransferase deficiency and increased activity of ribose phosphate pyrophosphokinase. These enzymes are encoded by genes located on the X chromosome, so primary hyperproduction is observed almost exclusively in males.

Pathogenesis. When the serum uric acid concentration exceeds approximately 0,42 mmol/L, blood plasma becomes supersaturated with urates. For a long time, crystallization may not occur, probably due to the presence of unidentified plasma factors that increase urate solubility. With decreased temperature, crystallization is facilitated, particularly in tissues with relatively poor blood supply (cartilage, ligaments). As a result, clusters of uric acid crystals (microtophi) are formed in the synovial membrane and cartilage.

Under the influence of various triggers (joint microtrauma, local overheating or hypothermia, acute changes in serum or synovial uric acid concentration), microtophi are disrupted and crystals are released into the joint cavity. Synovial cells produce cytokines (IL-1, IL-6, IL-8, TNF-α), which act as chemoattractants for neutrophils. Immunoglobulins and complement components opsonize urate crystals, stimulating phagocytosis by neutrophils. The ingestion of crystals by neutrophils leads to further release of inflammatory mediators and development of an intense acute inflammatory reaction in the joint.

Clinical manifestations. Three main stages are distinguished: acute gouty arthritis, the intercritical period, and chronic gouty arthritis.

Acute gouty arthritis. The classic manifestation is an acute attack of arthritis of the first MTP joint (fig. 13).

Provoking factors include:

1. Local trauma or excessive physical exertion.

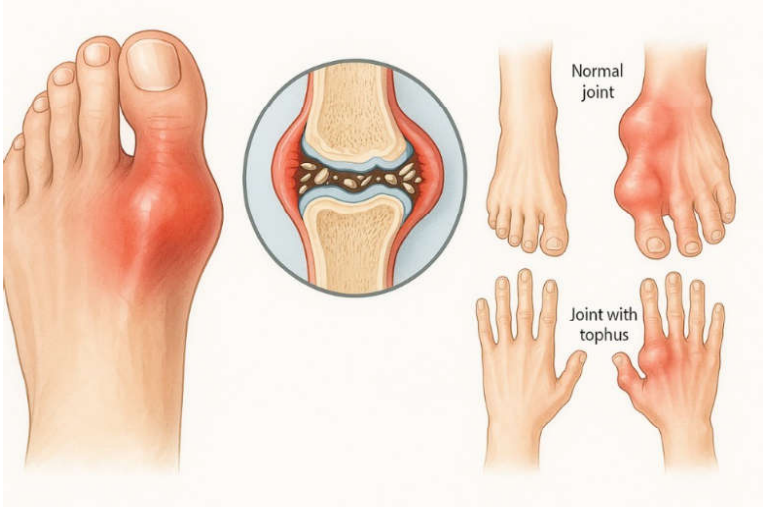
2. Sauna or bath visits, overheating or hypothermia.

3. Emotional stress.

4. Diet (overeating, fasting, alcohol intake, consumption of purine-rich foods).

5. Bleeding, surgery, acute illness or infection.

6. Use of certain medications (most often thiazide diuretics, cytotoxic agents, low-dose aspirin).



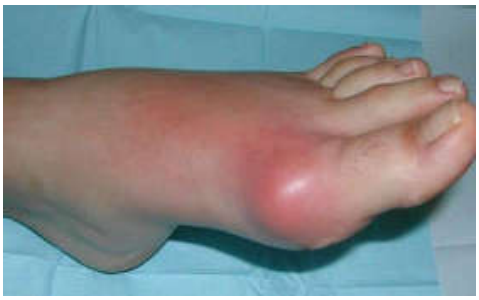


Fig. 13. Acute gouty arthritis

Typical features of an acute attack:

1. Very rapid onset, often at night, with maximal symptoms within 2–6 hours.
2. Intense pain in the affected joint, marked swelling and hyperemia of the skin.
3. Reversibility of all signs of arthritis within 5–14 days with complete restoration of joint function.
4. Possible systemic manifestations: malaise and fever.

Although the first MTP joint is most commonly affected, other joints can also be involved, in decreasing order of frequency: ankle, knee, wrist, elbow, and small joints of the feet and hands. Axial joints (shoulder, hip, spine) are rarely affected. A characteristic feature of acute gout is complete spontaneous resolution of symptoms and the absence of complaints between attacks in the early stages of the disease.

Intercritical period. The intercritical period is the asymptomatic interval between acute gout attacks. In the absence of adequate urate-lowering therapy and lifestyle modification, recurrent attacks occur. Each subsequent attack tends to be more severe, of longer duration, and the intercritical periods gradually become shorter. Over time, subclinical inflammation and progressive crystal deposition can lead to chronic gouty arthritis even if attacks are relatively infrequent.

Chronic gouty arthritis (tophaceous gout). Chronic gout is characterized by the formation of tophi — aggregates of urate crystals surrounded by inflammatory cells and fibrous tissue. Clinically, tophi present as dense, firm, often mobile nodules of whitish-yellow color; after ulceration, a chalk-like material may be discharged. Tophi may develop in many locations, most often subcutaneously and intradermally on the fingers, toes, feet, knees, elbows, and auricles, as well as in internal organs (fig. 14).

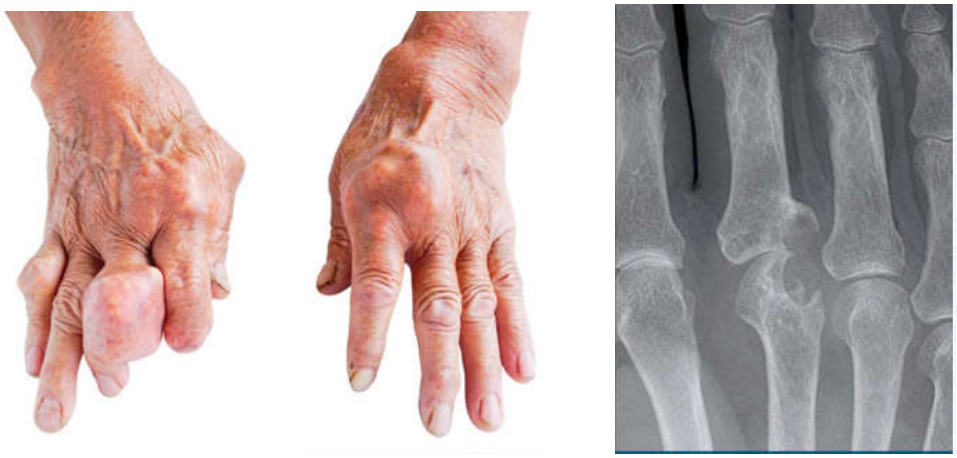


Fig. 14. Chronic gouty arthritis

Kidney damage may occur at any stage of gout. It manifests as nephrolithiasis (uric acid stones) and tubulointerstitial nephritis. Terminal chronic kidney disease develops in about 10 % of patients. Acute obstructive uric acid nephropathy due to tubular blockage with urate crystals can cause acute renal failure. At urine pH values below 5,0, uric acid remains largely non-dissociated, which promotes stone formation. With daily urinary excretion of more than 1100 mg of uric acid, urolithiasis develops in up to 50 % of patients. In addition, uric acid crystals may be deposited in the renal tissue, leading to interstitial nephritis and secondary arterial hypertension.

Gout is strongly associated with components of the metabolic syndrome, including obesity, hyperlipidemia, impaired glucose metabolism, arterial hypertension, and coronary heart disease, which significantly increases cardiovascular risk.

Serum uric acid: often elevated (hyperuricemia approximately 7,0 mg/dL [≈ 416 µmol/L] in men and above 6,0 mg/dL [≈ 357 µmol/L] in women), although levels may be normal during an acute attack; interpretation must take the clinical context into account.

Principles of treatment

The main goals of gout management are:

– rapid and safe relief of acute gouty arthritis;

– prevention of recurrent attacks and complications of hyperuricemia;

– prevention and treatment of comorbidities and drug-related adverse effects.

The key component of long-term management is lifestyle modification and sustained reduction of serum uric acid to target levels (generally < 360 μmol/L, and < 300 μmol/L in severe tophaceous gout).

Non-pharmacological measures:

1. Normalization of body weight; weight loss should be gradual, achieved by reducing caloric intake and engaging in moderate physical activity. Excessive physical exertion is contraindicated during acute attacks.

2. Adequate hydration: patients are encouraged to drink about 2–3 L of fluids per day in the absence of cardiac or renal contraindications.

3. Dietary recommendations:

– avoid fatty foods (fatty meats, excessive butter);

– limit total protein intake and prefer protein sources with low purine content;

– recommend a predominantly dairy vegetable diet with low fat milk and dairy products, and vegetables (cabbage, potatoes, cucumbers, carrots, onions, etc.);

– strictly limit foods rich in purines: meat of wild animals, processed meats and sausages, offal (brain, liver, kidneys, lungs), certain seafood (shrimp, mussels, scallops), and oily fish (mackerel, sardines, anchovies);

– prefer boiled meat and fish 2–3 times per week, as about half of the purines are released into the broth; broths and aspic/jellied dishes should be avoided;

– use simple spices such as bay leaf and vinegar;

– limit fructose-containing foods and drinks (fruit juices, sweet berries, citrus fruits, honey, sugar sweetened beverages), as fructose elevates serum uric acid.

Pharmacological treatment. Modern gout therapy is based on two complementary approaches — rapid control of acute attacks and long-term urate lowering therapy to maintain target serum uric acid levels:

1. Relief of an acute attack:

– NSAIDs (diclofenac, nimesulide, naproxen, meloxicam, celecoxib) are effective when started early and are considered first-line agents for rapid anti-inflammatory effect, provided there are no contraindications;

– Colchicine is a classic («gold standard») drug for acute attacks and can be used in low-dose regimens to relieve pain and inflammation, especially if started within the first 24 hours of symptom onset;

– Glucocorticoids can be administered orally or as intra-articular injections when NSAIDs and colchicine are ineffective, poorly tolerated, or contraindicated.

2. Urate-lowering therapy. This approach addresses the underlying cause — persistent hyperuricemia — and is indicated in patients with recurrent attacks, tophi, chronic gouty arthritis, or gout with renal or cardiovascular complications. Two main strategies are used:

– inhibition of uric acid production using xanthine oxidase inhibitors (allopurinol and febuxostat);

– enhancement of renal uric acid excretion with uricosuric agents (e. g. benzbromarone, probenecid), particularly when xanthine oxidase inhibitors alone are insufficient and renal function allows their use.

Initiation and titration of urate-lowering therapy should follow a «treat to target» strategy, with regular monitoring of serum uric acid and appropriate prophylaxis against flares (e. g., low-dose colchicine or NSAIDs during the first months).

INTERPRETATION OF LABORATORY AND INSTRUMENTAL INVESTIGATIONS

Summarizes the laboratory data obtained from patients with various joint diseases in table 2.

Table 2

Laboratory tests

Laboratory test	Rheumatoid arthritis	Osteoarthritis	Infectious arthritis	Gouty arthritis
Complete blood count	↑ESR anemia	Are typically normal; in cases of synovitis ↑ESR	↑WBC ↑ESR	↑WBC ↑ESR during an acute attack
Biochemical tests of blood	RF positive; ↑CRP	Are typically normal; in cases of synovitis ↑CRP, α_2-globulin, and fibrinogen	↑CRP	↑CRP hyperuricemia (men — 7,0 mg/dL [≈ 416 μmol/L], women — 6,0 mg/dL [≈ 357 μmol/L])
Urinalysis	Proteinuria	Are typically normal	Proteinuria	↑Uric acid
Immuno-logical methods	↑ Anti-Streptolysin-O (ASL-O); anti-cyclic citrullinated peptide (anti-CCP) positive	Are typically normal	↑ IgM, IgG; antibodies to Chlamydia trachomatis; markers of the hepatitis B virus	Are typically normal

End of the table 2

Laboratory test	Rheumatoid arthritis	Osteoarthritis	Infectious arthritis	Gouty arthritis
Synovial fluid analysis	Yellowish; turbid; decreased viscosity; an ↑WBC (typically 2–75 · 10^9/L) with neutrophil predominance culture: negative; crystals: absent	Viscosity is normal; cell count is normal (< 0,2 · 10^9/L) or slightly increased (0,2–2,0 · 10^9/L) with mononuclear predominance	↑WBC (typically 2–10 · 10^9/L) with neutrophil predominance	↑WBC (typically 10–60 · 10^9/L) with neutrophil predominance; monosodium urate crystals under polarized light

Data from instrumental investigations for various joint diseases are presented in table 3.

Table 3

Instrumental investigations

Instrumental investigations	Rheumatoid arthritis	Osteoarthritis	Infectious arthritis	Gouty arthritis
X-ray examination	Periarticular osteopenia; uniform joint space narrowing; marginal erosions; in advanced disease — subluxations, deformities, possible ankylosis	Non-uniform joint space narrowing; marginal osteophytes; subchondral sclerosis; subchondral cysts; bone remodeling and subluxations	Soft tissue swelling; joint space narrowing; periarticular osteopenia; in advanced cases — subchondral erosions and bone destruction	Well-defined «punched-out» juxta-articular erosions with overhanging edges; soft tissue tophi; relative preservation of joint space until late disease
Joint ultrasound	Synovial hypertrophy, joint effusion, power Doppler signal indicating active synovitis, bone erosions	Joint effusion, mild synovial hypertrophy, osteophytes; assessment of periarticular bursae and tendons	Joint effusion, marked synovial thickening, periarticular soft tissue swelling; guidance for diagnostic aspiration	«Double contour» sign (urate crystal deposition on the surface of articular cartilage)

End of the table 3

Instrumental investigations	Rheumatoid arthritis	Osteoarthritis	Infectious arthritis	Gouty arthritis
MRI	More early inflammatory changes	Soft tissue swelling; ligament rupture; traumatic injuries of cartilage and intra-articular ligaments	–	–
Radionuclide imaging	More early inflammatory changes	Inflammatory changes	Inflammatory changes	–
Arthroscopy	–	Traumatic injuries of cartilage and intra-articular ligaments	–	–

TEST CONTROL

1. The main symptoms case of musculoskeletal diseases include all of the following except:

a) joint pain;
b) joint deformity;
c) dyspnea;
d) morning stiffness;
e) joint crepitus.

2. Inflammation of two or three joints is called:

a) polyarthritis;
b) oligoarthritis;
c) synovitis;
d) monoarthritis;
e) tendinitis.

3. Non-invasive methods for joint examination:

a) ultrasound;
b) arthroscopy;
c) MRI;
d) X-ray.

4. RA is characterized by all of the following except:

a) symmetry of involvement;
b) morning stiffness;

c) genetic predisposition: HLA-B27 Gene;
d) «Swan-neck» or «boutonniere» deformities in the hands;
e) Ulnar deviation.

5. Which of the listed extra-articular manifestations of RA is considered the most common:

a) rheumatoid nodules;
b) pericarditis;
c) lung involvement;
d) skin itching?

6. The laboratory tests used to diagnose RA include all of the following, except:

a) RF;
b) low-density lipoprotein;
c) anti-CCP;
d) synovial fluid test.

7. Which of the following is considered the cornerstone of RA treatment and should be initiated as early as possible:

a) NSAIDs;
b) GCs;
c) DMARDs;
d) biologic agents?

8. Factor(s) contributing to the gout arthritis include:

a) genetic predisposition;
b) impaired joint vascularization due to pathology of circulation;
c) mechanical and functional overload of the joint;
d) disturbance of purine metabolism.

9. Which of the following imaging modalities is considered the gold standard for the diagnosis and staging of OA:

a) Computed Tomography;
b) MRI;
c) Conventional Radiography;
d) Ultrasonography?

10. The cardinal symptom of OA that typically worsens with activity and improves with rest is:

a) morning stiffness lasting > 60 minutes;
b) inflammatory joint swelling with erythema;
c) deep, aching joint pain;
d) systemic fever and fatigue.

11. A common objective finding on physical examination of an osteoarthritic joint is:

a) Bouchard's nodes;
b) «Swan-neck» deformity;
c) «Sausage» digit (dactylitis);
d) Ulnar deviation of the fingers.

12. According to major guidelines, what is the first-line non-pharmacological treatment for knee and hip OA:

a) intra-articular hyaluronic acid injections;
b) oral NSAIDs;
c) patient education and structured exercise programs;
d) arthroscopic debridement?

13. What is the pathognomonic finding for acute gout:

a) elevated serum urea level;
b) a rapid and significant response to colchicine therapy;
c) visualization of needle-shaped, negatively birefringent crystals in synovial fluid;
d) inflammation of the first metacarpophalangeal joint?

14. What is the recommended first-line pharmacological treatment for an acute gout attack:

a) Allopurinol (to lower uric acid);
b) systemic corticosteroids or NSAIDs;
c) intra-articular hyaluronic acid injections;
d) long-term low-dose aspirin?

15. Reactive Arthritis is best defined as an asymmetric oligoarthritis that typically develops following:

a) a distant genitourinary or gastrointestinal infection;
b) an episode of gout;
c) a traumatic injury to the joint;
d) a purulent joint inflammation.

Correct answers: 1 — c; **2** — b; **3** — a, c, d; **4** — c; **5** — a; **6** — b; **7** — c; **8** — d; **9** — c, **10** — c; **11** — a; **12** — c; **13**– c; **14** — b, **15** — a.

CLINICAL CASE

Clinical case 1

A 42-year-old woman complains of pain and morning stiffness in the hands and feet for 8 months. Stiffness lasts about 1,5 hours and improves after movement. She notes swelling of the wrists and «thickening» of the finger joints, difficulty turning door handles and holding a cup. Over the last 3 months, fatigue and low-grade fever up to 37,5 °C have appeared. On examination: symmetrical swelling and tenderness of the metacarpophalangeal and PIP joints of both hands, limited flexion and extension, mild ulnar deviation of the fingers. Small, firm, painless nodules are palpated over the extensor surface of the forearms.

Questions:

1. Formulate the preliminary diagnosis.
2. Which additional laboratory tests would you order to confirm the diagnosis?
3. Which basic (disease-modifying) drugs are indicated as first-line therapy in this patient?

Clinical case 2

A 69-year-old man complains of chronic pain in both knees for 5 years, worsening over the last year. The pain appears when walking and climbing stairs, intensifies in the evening, and decreases after rest. Morning stiffness lasts no more than 10–15 minutes. He reports intermittent swelling of the knees after increased physical activity. History: obesity, arterial hypertension. On examination: varus deformity of both knees, slight swelling, palpable crepitus during movement, pain on palpation along the joint line. Range of motion is moderately reduced (especially flexion), but complete immobility is absent. Skin temperature over the joints is normal.

Questions:

1. Formulate the preliminary diagnosis.
2. Which instrumental method is considered the basic method for confirming this diagnosis, and what typical radiographic signs do you expect to see?
3. Name two key non-pharmacological and two pharmacological approaches to treatment for this patient.

Clinical case 3

A 35-year-old man presents with acute onset of severe pain and swelling in the right knee for 2 days. The pain increases with the slightest movement; he cannot bear weight on the leg. He reports fever up to 38,9 °C, chills, and weakness. History: 1 week ago he had a skin abrasion on the leg while playing football; he did not seek medical care. On examination: severe swelling of the right knee, skin over the joint is hyperemic and hot to the touch, any passive movement is sharply

painful. Temperature 38,5 °C, pulse rate 104/min, Blood pressure 120/75 mmHg. Laboratory tests: WBC 15 · 10^9/L with a left shift, ESR 60 mm/h, CRP markedly elevated.

Questions:

1. Formulate the preliminary diagnosis.

2. Which immediate diagnostic procedure on the joint is necessary, and what key findings do you expect?

3. What are the main principles of initial treatment in this situation (mention at least two components)?

Clinical case 4

A 58-year-old man complains of sudden severe pain in the right first MTP joint that began at night 24 hours ago. The pain is so intense that he cannot touch the sheet with his foot. He notes swelling and redness of the joint, fever up to 38,0 °C. History: arterial hypertension, obesity, frequent consumption of red meat and beer; thiazide diuretics have been taken for several years. On examination: marked swelling and hyperemia of the first MTP joint on the right, local hyperthermia, sharp tenderness, the patient avoids any movement. On the extensor surface of the left elbow, a dense, painless, whitish nodule is palpated.

Questions:

1. Formulate the preliminary diagnosis.

2. Which study of synovial fluid would confirm this diagnosis, and what characteristic finding do you expect?

3. Name one drug class (or specific drug) for relief of the acute attack and one drug class for long-term urate-lowering therapy.

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Appendix 1

FUNCTIONAL IMPAIRMENT CLASSES
(THE DEGREE OF IMPAIRMENT OF JOINT FUNCTION)

Class	Characteristics
Class I	No limitations; can perform all usual activities
Class II	Moderate limitation; adequate performance of normal activities despite discomfort or limited motion in one or more joints
Class III	Significant limitation; limited performance of usual self-care and daily activities
Class IV	Incapacitated; largely or wholly bedridden or wheelchair-bound, with little or no self-care

Appendix 2

CRITERIA FOR THE DIAGNOSIS OF RHEUMATOID ARTHRITIS (2010 RHEUMATOID ARTHRITIS CLASSIFICATION CRITERIA: AN AMERICAN COLLEGE OF RHEUMATOLOGY/EUROPEAN LEAGUE AGAINST RHEUMATISM COLLABORATIVE INITIATIVE)

Joint involvement	Points
1 large joint (shoulder, elbow, hip, knee and ankle)	0
2–10 large joints	1
1–3 small joints (MCP, PIP, MTP, carpal; with or without large joint involvement)	2
4–10 small joints (with or without large joint involvement)	3
10 joints (at least 1 small joint)	5
Serology	
Negative RF and anti-CCP	0
Low positive RF and/or anti-CCP	2
High positive RF and/or anti-CCP	3
Acute-phase reactants	
Normal CRP and/or ESR	0
Abnormal CRP and/or ESR	1
Duration of symptoms	
< 6 weeks	0
> 6 weeks	1

Scoring: a diagnosis of definite RA is supported by at least one swollen joint not otherwise explained and score of 6/10 from the above criteria.

Appendix 3

DIFFERENCES BETWEEN REACTIVE AND INFECTIOUS (SEPTIC) ARTHRITIS

Feature	Reactive arthritis	Infectious (septic) arthritis
Definition	Sterile inflammatory arthritis developing 1–4 weeks after an extra-articular urogenital or intestinal infection	Acute inflammatory arthritis caused by direct invasion of the joint by microorganisms (usually bacteria), with purulent synovitis and rapid joint destruction
Etiology	No viable pathogen in the joint! Triggered by infections: Chlamydia trachomatis (urogenital), Salmonella, Shigella, Yersinia, Campylobacter and others (enteric)	Staphylococcus aureus most common; also streptococci, Gram-negative bacteria, Neisseria gonorrhoeae, mycobacteria, fungi, viruses
Pathogenesis	Molecular mimicry (cross-reactive immune response against joints antigens) → aseptic synovitis and enthesitis	Microbial proliferation in synovial fluid → intense neutrophilic inflammation, high intra-articular pressure, protease-mediated cartilage and bone destruction
Risk factors	HLAB27 positivity; recent urogenital or enteric infection; young age, male, unprotected sexual activity	Older age, diabetes, CKD, immunosuppression, RA, prosthetic joints, recent joint surgery or injection, IV drug use, skin infections, trauma
Onset	Subacute; arthritis usually appears 1–4 weeks after infection	Acute, rapid onset over hours–days, often with severe pain and inability to move the joint
Physical examination	Asymmetric swollen and tender joints of the lower limbs (knees, ankles, feet) with oligoarthritis; dactylitis and enthesitis (e. g. Achilles tendon, plantar fascia); limited spinal motion when sacroiliac joints are involved	Acute monoarthritis of a large joint (usually knee, hip, shoulder, or ankle) with marked swelling, warmth, erythema, severe pain on passive movement, antalgic joint position, and clinical signs of systemic infection (fever, chills, malaise)
Extra-articular features	Urethritis, cervicitis, conjunctivitis/uveitis, skin and mucosal lesions (keratoderma, balanitis), low-grade fever (37,1–38,0 °C)	Systemic signs of infection: high fever (39,1–39,4 °C), chills, malaise; possible sepsis and hemodynamic instability
Laboratory blood date	↑ESR, ↑CRP, HLAB27 often positive; evidence of triggering infection (PCR/serology for Chlamydia, enteric pathogens)	↑ESR, ↑WBC, ↑CRP, ↑procalcitonin, positive blood cultures in many cases; identification of causative microorganism from synovial fluid culture and Gram stain

End of appendix 3

Feature	Reactive arthritis	Infectious (septic) arthritis
Synovial fluid*	Inflammatory: ↑WBC (usually 5–50 · 10^9/L), neutrophil predominance; culture negative (sterile)	Purulent: ↑↑↑WBC (often > 50 · 10^9/L) neutrophil predominance; Gram-positive bacteria culture
Instrumental date	X-ray: normal early; later — erosions, bone proliferation, juxta-articular osteoporosis, uniform joint space narrowing, enthesopathy/ enthesitis, US: synovitis, enthesitis, dactylitis. MRI: bone marrow edema in sacroiliitis	X-ray: soft tissue swelling early; later — joint space loss, erosions. US: joint effusion, synovial thickening, guidance for aspiration. MRI: extent of bone and soft tissue infection (osteomyelitis, abscess)
Principles of therapy	Treat triggering infection (especially Chlamydia); NSAIDs first-line; intra-articular GCs for severe synovitis; csDMARDs (sulfasalazine, methotrexate) in persistent/chronic ReA	Emergency joint aspiration; prompt empiric IV antibiotics with later adjustment to culture results; repeated aspiration or surgical/arthroscopic drainage; short-term immobilization followed by early mobilization; management of sepsis and comorbidities

*Normal synovial fluid contains up to ~ 0,2–0,3 · 10^9/L WBC, predominantly mononuclear cells, neutrophils accounting for less than 25 %.

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