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MANAGEMENT OF A PATIENT WITH JOINT SYNDROME (OSTEOARTHROSIS, GOUTY ARTHRITIS)

Methodical recommendations
for the preparation of students of higher education
in the 6th year of study in the discipline «Internal Medicine»

Electronic resource

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M 24 **Management** of a patient with joint syndrome (osteoarthrosis, gouty arthritis) : methodical recommendations for the preparation of students of higher education in the 6th year of study in the discipline «Internal Medicine» [Electronic resource] / comp. T. M. Tykhonova, M. Yu. Gorshunska, N. V. Lysenko, O. Yu. Bychkova, L. O. Martymianova, I. V. Soldatenko, N. Ye. Barabash. – Kharkiv : V. N. Karazin KhNU, 2025. – (PDF 41 p.)

The methodical recommendations specify the definition of joint syndrome and its diagnosis in rheumatic patients based on complaints, anamnesis, objective examination data and laboratory-instrumental diagnostics. Management of a rheumatic patient with joint syndrome is shown on the example of osteoarthrosis and gouty arthritis.

For students of the 6th year to prepare for practical classes in the discipline «Internal Medicine».

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CONTENT

LIST OF CONVENTIONAL ABBREVIATIONS

ANF – antinuclear factor

AS – ankylosing spondylitis

ASL-O – anti-streptolysin-O

PAN – polyarteritis nodosa

GCs – glucocorticosteroids

ARF – acute rheumatic fever

DNA – deoxyribonucleic acid

CPK – creatinine phosphokinase

MRI – magnetic resonance imaging

NUC – non-specific ulcerative colitis

NSAIDs – non-steroidal anti-inflammatory drugs

OA – osteoarthrosis / osteoarthritis

OP – osteoporosis

GA – gouty arthritis/gout

PsA – psoriatic arthritis

RA – rheumatoid arthritis

RF – rheumatoid factor

CRP – C reactive protein

SSD – systemic scleroderma

SLE – systemic lupus erythematosus

US – ultrasound examination

CD – Crohn's disease

ReA – reactive arthritis (formerly termed Reiter syndrome)

ESR – erythrocyte sedimentation rate

JRA – juvenile rheumatoid arthritis

Anti-CCP – Anti-cyclic citrullinated peptide antibodies

C3, C4 – serum complement components

Anti-La – antibodies to Laen’s nuclear antigen

pANCA, cANCA – antinuclear cytoplasmic antibodies

Anti-Ro – antibodies to Robert’s nuclear antigen

Anti-Sm – antibodies to Smith’s nuclear antigen

1. BASIC KNOWLEDGE, SKILLS, ATTAINMENTS NECESSARY FOR THE TOPIC STUDYING

Names of previous disciplines	Acquired skills
Foreign language	To be able to work with foreign literature to obtain data on modern methods of diagnosis and treatment of rheumatological patients
Medical informatics	Apply modern computer programs and be able to work with them, have statistical methods for processing the results of clinical studies, analyze the results of studies, be able to evaluate and interpret the results of clinical research from information sources, have the ability to work with electronic databases
Human anatomy. Normal physiology. Histology, cytology and embryology	Know human anatomy, the normal structure and functions of joints. Understand and determine the relationship between the structure and function of the musculoskeletal system and other organs and systems of the human body.
Microbiology, virology and immunology	To be able to analyze the peculiarities of the microorganisms vital activity, to know the basics of antibiotic therapy, the conditions for the development of microorganisms resistance, to evaluate the effectiveness and safety of antimicrobial agents. To understand the pathogenesis of infectious diseases of the body and their influence on the autoimmune processes initiation with the subsequent occurence of rheumatic diseases.
Pathomorphology Pathophysiology	Know typical pathological processes in the human body: the mechanisms of the joints destruction development, and separately of cartilage and bone tissue, the impact of these changes on human organs and systems, the development of «cause-effect» relationships in the pathology of the whole organism. Describe and schematically represent the mechanism of development of typical pathological syndromes in rheumatological diseases, justify pathogenetic approaches to drug therapy

Pharmacology	Be able to navigate in the nomenclature of medicines. Know the mechanism of action of medicines, their pharmacodynamics, indications and contraindications for their use Know the features of the clinical pharmacology of drugs used in the treatment of rheumatological diseases, the features of the pharmacological action of these drugs in different categories of patients Make a reasonable choice of individual drugs and therapy schemes in accordance with the principles of evidence-based medicine, optimization of treatment schemes, evaluate the effectiveness and safety of pharmacotherapy taking into account the individual characteristics of the patient, the presence of concomitant diseases
Propaedeutics of internal medicine	Conduct a physical examination of rheumatological patients according to the recommended scheme with a targeted examination of the musculoskeletal system. To determine the leading syndromes and symptoms in rheumatological patients. Analyze the results of basic laboratory and instrumental research methods. To be able to make a differential diagnosis based on the physical examination of rheumatological patients and the data of additional research methods, justify and formulate the diagnosis.

1.1. The student should know:

– anatomy and functions of joints;

– mechanisms of joints damage, and separately cartilage and bone tissue, including – autoimmune;

– the main risk factors and etiological factors, pathogenesis, clinic, differential diagnosis of rheumatic diseases in which joint syndrome is the leading factor;

– classification of rheumatic diseases, according to the modern international recommendations;

– basic standards of instrumental and data of laboratory research methods used for the examination of rheumatic patients, in whom joint syndrome is the leading factor;
– diagnostic standards (diagnostic criteria) of rheumatic diseases, in which joint syndrome is the leading factor, according to the modern international recommendations;
– modern international standards and protocols for the treatment of rheumatic patients in whom joint syndrome is the main cause;
– medical and social examination and prevention of rheumatic diseases, in which joint syndrome is the main factor;
– rehabilitation methods of preserving and improving the quality of life of rheumatic patients, in whom joint syndrome is the leading factor.

1.2. The student must be able to:

– collect complaints, anamnesis;
– to carry out the method of objective examination of a rheumatic patient with joint syndrome as the main factor, as recommended by the scheme with a targeted examination of the musculoskeletal system;
– interpret the data of instrumental and laboratory research methods used for the examination of rheumatic patients, in whom joint syndrome is the main factor;
– diagnose the disease in a rheumatic patient with joint syndrome, using diagnostic criteria;
– carry out a differential diagnosis of a rheumatic disease in which joint syndrome is the main factor;
– on the basis of these complaints, anamnesis, objective examination of the patient, instrumental and laboratory diagnostics, using diagnostic criteria, establish a clinical diagnosis of a rheumatic patient with joint syndrome as the main factor;

- prescribe treatment to the patient in accordance with modern international standards and protocols for the treatment of rheumatic patients in whom joint syndrome is the main cause;
- to conduct a medical and social examination of a rheumatic patient with joint syndrome as the leading cause;
- to recommend rehabilitation methods to maintain and improve the quality of life to a patient with joint syndrome.

2. TOPIC CONTENT

2.1. Definition of joint syndrome

Pain in the joints (arthralgia), deformation, swelling or disfigurement of the joints, as well as a violation of their function is called joint syndrome.

Joint syndrome is found in about 80 rheumatic and several dozen non-rheumatic diseases, although the vast majority of all its causes are about 20 common rheumatic pathological conditions. In order to avoid diagnostic errors in determining the nosological affiliation of the joint syndrome, it is necessary to use all the classic stages of the patient's examination, starting with passport data, complaints, medical history and life data, objective examination of the patient, and ending with the analysis of the results of special examination methods – laboratory and instrumental.

In the early stages of the rheumatological disease development, conventional clinical examination methods are of great importance in establishing a diagnosis, because for many common causes of joint syndrome there are no specific laboratory markers, or they become positive later (for example, rheumatoid factor), and radiological changes in the initial stages of diseases joints are absent or non-specific.

General information about the patient, analysis of complaints and a carefully collected medical history make it possible to establish a diagnosis in 70 % of cases. Further results of the musculoskeletal system investigation, assessment of other organs and systems damage symptoms, routine laboratory data (clinical blood analysis, CRP, etc.) allow the doctor to determine the diagnosis in 90 % of cases of pain syndrome in the joints.

Special laboratory, X-ray and other instrumental methods of examination (including biopsy) in the early stages of most rheumatic diseases, which debut with joint syndrome, are able to change the diagnosis established on the basis of traditional clinical and laboratory information in only 10 % of cases.

Differential diagnosis of joint syndrome is extremely important for early establishment of an accurate diagnosis, which is the key to timely initiation of pathogenetically determined treatment, and, accordingly, improvement of the prognosis, work capacity preservation, duration and quality of life of rheumatological patients.

2.2. Patient's general information in the diagnosis of joint syndrome

General information about the patient (gender, age, general appearance of the patient) can in most cases guide the diagnostic process in the right direction. So, for example, a hypersthenic physique and excess weight are typical for patients with osteoarthrosis (OA), gouty arthritis/gout (GA). Significant weight loss, characteristic of polyarteritis nodosa (PAN), can occur in rheumatoid arthritis (RA), systemic autoimmune diseases, primarily systemic lupus erythematosus (SLE), and paraneoplastic arthritis. In the late stages, the diagnosis of ankylosing spondylitis (AS) is established ad oculus (at first sight) – a typical pose of a «beggar».

In some rheumatic diseases manifested by joint syndrome, the predominance of individuals of a certain gender is so important that it has a significant diagnostic value: the share of men among patients with gout, AS, reactive arthritis (ReA) exceeds 90 %, with PAN – about 75 %. Among patients with Sjögren's disease, SLE, systemic scleroderma (SSD), RA, women make up 75 % or more.

Some rheumatic diseases have a rather pronounced tendency to debut in a certain age period (Table 1). For example, acute rheumatic fever (ARF), ReA, polymyalgia rheumatica, Takayasu's arteritis, temporal arteritis, practically do not occur outside the specified age ranges.

Table 1.

Age peaks of the some rheumatic diseases debuts

Age range, years	Illness
1–15	Juvenile rheumatoid arthritis (JRA)
8–25	Acute rheumatic fever
≥ 50	Temporal arteritis
≤ 40	Takayasu vasculitis
≥ 60	Polymyalgia rheumatica
25–40	Reactive arthrities
≥ 30	Gouty arthritis/gout

2.3. Patient's complaints in the diagnosis of joint syndrome

The patient's complaints are the most important component of the examination for joint syndrome. Their careful analysis is usually the key to establishing the correct diagnosis. In the process of detailing the patient's complaints, they analyze, first of all, the nature of the pain syndrome in the joints – arthralgia, which occurs in 90 % of patients with rheumatic diseases.

Find out:

a) duration, daily frequency and nature of pain.

Pain caused by inflammatory diseases of the joints (RA, SLE, AS), is usually long or constant, increases with any movement, its maximum traditionally occurs in the second half of the night and in the morning (inflammatory nature of the pain). Pain associated with degenerative changes in the joint apparatus (OA, GA) occurs periodically, during or after loads (regardless of the time of day), when performing certain movements, for example, when squatting, getting up from a chair, bending the body in some directions. As a rule, this pain passes overnight or significantly weakens (mechanical nature of pain) (Table 2).

Table 2.

Differential diagnosis of inflammatory and mechanical nature of joint pain based on patient complaints

Sign	Inflammatory	Mechanical
Pain	Constant, decreases or passes after the start of movement	Periodic, during or after loading, with certain movements, may be accompanied by crepitation or crunching
Daily frequency	The second half of the night and early morning	It intensifies until the end of the day, passes overnight
A change in the skin over the joint	Maybe - hyperemia	There is none
An increase in the temperature of the skin over the joint	Maybe	There is none

b) intensity. The most intense, unbearable pain, which increases with the slightest movement or touching the joint, is characteristic of acute gouty arthritis. Pronounced are arthralgias in ARF (up to the complete impossibility of moving the limbs), rheumatic polymyalgia, sometimes – in reactive arthritis. Severe localized pain in the spine, may be due to discogenic radicular syndrome, vertebral fracture in osteoporosis (OP) and non-rheumatic pathological processes – osteomyelitis, tumors or metastases

c) localization. The number of joints is important, in which joints pain sensations and the symmetry of the process are determined.

Table 3.

Localization of joint syndrome

	Symmetrically	Asymmetrically	Large joints	Small joints	Upper limbs	Lower limbs
RA	+		+	+	+	+
OA	+		+			+
AC	+		+			+
GA		+	+	+	+	+
ReA		+	+			+

In addition to the pain syndrome analysis, the patient's complaints about morning stiffness (its presence and duration), swelling, deformation, disfigurement, restriction of movement, crunching or crepitation in the joints, joint dysfunction are clarified. If the patient applied after the exacerbation of the process has passed, it is found out whether there were changes in the color of the skin over the painful joints (redness, purplish-bluish tint) and an increase in the temperature of the skin over them.

Morning stiffness accompanies the vast majority of inflammatory arthritis, although it is most characteristic of RA (it has diagnostic value if it lasts more than 30 minutes). It can periodically occur in non-inflammatory arthritis - OA with the development of concomitant reactive synovitis, but in these cases it is usually short-lived (no more than 30 minutes) and has no diagnostic value.

Swelling (swelling) of the joint is a change in the shape of the joint due to swelling of periarticular tissues and / or an increase in the amount of synovial fluid. Deformation of the joint is a change in the shape of the joint as a result of displacement of the bones that make it up (dislocations and subluxations), pathological growths of tissues in the joint or periarticular, ankylosing of the joints. Joint deformity is a combination of joint swelling and deformation.

As we can see from the above definitions, joint syndrome can be caused by pathological changes both in the joint itself and in its surrounding tissues.

The main options for impaired joint function are stiffness, limitation (less often – increase) of range of motion, crunching, crepitation or clicking during movement.

Thus, analyzing the patient's complaints, the doctor receives three blocks of information that are directly related to establishing a diagnosis:

1) articular syndrome has an inflammatory or mechanical nature;

2) which and how many joints are affected;

3) damage to the joints is symmetrical or asymmetrical.

The second block of information (about the localization of the process) makes it possible to distinguish two groups of possible diseases: with predominant damage to the joints of the limbs or with the presence of symptoms of damage to the spine, each of which includes two subgroups, depending on the inflammatory / mechanical nature of the joint syndrome.

For the category of patients with dominant damage to the joints of the limbs, the second and third blocks of information are used to distinguish groups of monoarthritis (1 joint), oligoarthritis (2–3 joints) or polyarthritis (≥ 4 joints), among which it is already possible to search for a presumptive diagnosis.

In 30 % of cases, RA debuts as symmetric oligoarthritis-polyarthritis, which is characterized by symmetric damage, first of all, to the small joints of the upper and lower limbs. OA often proceeds like symmetrical polyarthritis. 80 % of patients with a peripheral form of AS develop symmetric arthritis of 2–4 joints of the lower limbs, most often – knees. ReA in 1/3 of cases manifests as mono-oligoarthritis with asymmetrical involvement of large or small joints of the lower extremities. For ReA and reactive urogenic arthritis, if treatment is not started in time, the increasing dynamics of joint involvement (for example, the joint of the toe of the left foot, then the right ankle, left knee, right hip or sacroiliac, so-called «ladder lesion») with possible transformation into polyarthritis, which retains the same localization; the joints of the upper limbs (almost exclusively – large, asymmetric) are involved very rarely and at late stages. GA in the initial stages of the disease almost always proceeds as a transient (3–5 days) monoarthritis, in more than half of the cases, 1 metatarsal-phalangeal joint is affected, less often – ankle-foot or knee. In hemorrhagic vasculitis, the arthritis is always symmetrical, with predominant damage to the large joints of the lower extremities, knee or ankle.

In some arthritis involving the small joints of the hands, the topography of the lesions is so typical that such hands can be considered business cards of the corresponding diseases. For RA, these are the proximal interphalangeal, metacarpal-phalangeal joints, and wrist joints; at the onset of the disease, the proximal interphalangeal and metacarpal-phalangeal joints of II–IV fingers are more often affected. For psoriatic arthritis (PsA), pathognomonic lesions of the distal interphalangeal joints (which distinguishes it from RA, for which they are «joints of exclusion»), as well as «axial» lesions of all three joints of one finger, more often – II and / or III («axial » lesions of finger joints are called dactylitis). In case of OA of the hand joints, bony growths (osteophytes) are formed on the lateral surfaces of the distal interphalangeal (Heberden's nodules) and proximal interphalangeal joints (Bouchard's nodules), and early damage to the carpo-carpal joints is also typical, especially in women.

When detailing complaints, the localization of the pain syndrome should be analyzed for the category of patients with symptoms of spinal cord injury. With AS, PsA, arthritis on the background of inflammatory bowel diseases, all parts of the spine without exception are involved in the inflammatory process, in the early stages, as a rule, the lumbosacral part is symptomatic. The typical localization of pain in ReA and reactive urogenic arthritis is the mid-thoracic and lumbar regions of the spine. Cervical spondyloarthritis is characteristic of RA, usually it does not occur at the onset of the disease. Other parts of the spine are not damaged in RA.

Pain and stiffness in the lumbosacral region in seronegative spondyloarthritis is usually caused by inflammation of the sacroiliac joint - sacroiliitis, which is a clinical criterion in patients carrying the HLA B27 antigen. Sacroiliitis, more often unilateral or asymmetric in PsA, ReA, reactive urogenic and postenterocolitic arthritis, arthritis in non-specific ulcerative colitis (NUC) and Crohn's disease (CD). With AS, it is always bilateral and, as a rule, symmetrical. This is evidenced by the results of instrumental research methods (x-rays, magnetic resonance imaging, computer tomography), although clinically one-sided symptoms may dominate in some patients at certain stages of the disease.

The most frequent complaint of patients with seronegative spondyloarthritis (AS, ReA and PsA) is pain in various places of the skeleton, caused by inflammation of the places of attachment to bones of ligaments, tendons, fibrous part of intervertebral discs, joint capsules – so-called enthesitis. One of the most frequent localizations of enthesitis is the place of attachment of the Achilles tendon and plantar fascia, which causes pain in the heel bone – talalgia. Pain on the back surface of the calcaneus can also be caused by achilobursitis or (rarely) achilleitis, which is characteristic of seronegative spondyloarthritis. Over time, new bone tissue is formed and spurs are formed in places of heel enthesitis (more often on the plantar surface, less often on the back).

Damage to the joints, which are usually rarely involved in the pathological process, can have a certain diagnostic value. Inflammation of the temporomandibular joint (pain during movement and palpation, limitation of mobility of the lower jaw) develops mainly in JRA. Symptoms from the side of the sterno-arginal and costo-sternal joints (sickness and swelling) are most common in seronegative spondyloarthritis. Tietz syndrome is a disease of unknown etiology, which is manifested by pain and thickening of the front ends of the II, III or IV ribs, usually on one side. It is important to remember the possible non-rheumatic causes of localized pain in the ribs and sternum, among which myeloma and tumor metastases are the most common. This is all the more relevant because in all these non-rheumatic diseases, an increase in ESR and CRP is possible.

It is necessary to take into account the presence of fever in a patient with joint syndrome. If it accompanies mono- or oligoarthritis, differential diagnosis should be made between infectious bacterial (septic, gonococcal, brucellosis) arthritis, microcrystalline arthritis (GA, pyrophosphate arthropathy), reactive arthritis (including ReA), Steele's disease, RA, ARF, PAN, paraneoplastic arthritis. Most patients with persistent oligoarthritis (> 4 weeks) without fever will be diagnosed with one of the seronegative spondyloarthritis or RA. In the presence of fever in a patient with polyarthritis, first of all, the presence of such diseases as SLE, mixed connective tissue disease, ARF, systemic vasculitis (primarily, PAN, microscopic polyangiitis, less likely – Wegener's granulomatosis, nonspecific aortoarteritis, temporal arteritis with polymyalgia rheumatica), recurrent polychondritis is considered.

2.4. Patient's anamnesis data in the diagnosis of joint syndrome

Medical history. The next stage of examination of a patient with joint syndrome is the analysis of anamnestic data. At the same time, they find out:

a) possible provoking factors: physical activity – for OA; alcoholic and food provocations – for GA; insolation, childbirth – for SLE;

b) chronological relationship with transferred infections (usually within 3–4 weeks):

1. streptococcal (angina, pharyngitis) – for ARF,
2. urogenital (primarily, chlamydia) – for reactive urogenital arthritis,
3. acute intestinal (salmonellosis, dysentery, yersiniosis) – for reactive postenterocolitic arthritis,
4. viral – for viral arthritis and many systemic rheumatic diseases (as etiological risk factors),
5. stay in regions unfavorable for certain arthritogenic infections;

c) the nature of the joint syndrome at the onset of the disease, in particular, the sequence of involvement of the joints, the persistence of their damage, the presence of local and systemic signs of inflammation, weight loss;

d) accompanying joint syndrome involvement of other organs and systems, including skin and mucous membranes (psoriasis, all variants of infectious and non-infectious exanthema and enanthema);

e) the course of joint syndrome is of great diagnostic value for mono- and oligoarthritis;

f) effectiveness of nonsteroidal anti-inflammatory drugs (NSAIDs), especially in chronic arthritis.

Anamnesis vitae. Important information can be obtained from family history. A disease with a joint syndrome that has a family tendency is AS, PsA, GA, seropositive RA, nodular form of OA. It is often possible to observe a cross susceptibility to rheumatic diseases, especially in the female line, for example, a mother has RA, a daughter has SLE, etc. The basis of a genetically determined susceptibility to diseases from the group of seronegative spondyloarthritis is the carrier of the HLA B27 histocompatibility antigen. Among patients with AS, HLA B27 is positive in approximately 95 % of patients, with ReA – 60 %, with enteropathic arthritis – 60–70 %, with PsA – 55 %, while in healthy individuals this antigen is detected in 6–8 % of cases.

2.5. Data of objective examination of joints and adjacent tissues (Status localis) in the diagnosis of joint syndrome

Objective examination of the joints and adjacent tissues (Status localis) – the final stage of the clinical examination of a patient with a joint syndrome, in the process of which the nature and localization of the joint syndrome are specified based on complaints and history, changes that could have remained unnoticed by the patient are revealed – limitation of the mobility of the joints and spine, symptoms of sacroiliitis, changes in skin color, etc. First of all, pay attention to the color of the skin over the affected joint. Bright hyperemia of the skin over the joint is typical for acute gouty or infectious arthritis, it is less pronounced in acute rheumatic arthritis. With all the mentioned diseases, this is combined with significant pain in the joints. A red-bluish shade of the skin can be observed in PsA, ReA, reactive urogenic and postenterocolitic arthritis, first of all, over the small joints of the hands and feet. Such coloration of the skin in combination with dactylitis is the basis for determinations with pronounced arthritis, such as a sausage-shaped finger (with «axial» damage to the joints of one finger).

Diffuse hyperpigmentation of the skin is often found in arthritis caused by SSD and hemochromatosis. Slight local hyperpigmentation over chronically inflamed small joints can be observed in several chronic polyarthritis (RA, reactive arthritis, PsA). The presence of local hyperthermia is determined by palpation as a sign of inflammation.

The cause of joint deformation can be osteophytes (in OA), destructive changes with the congruence violation of joint surfaces, subluxations / dislocations as a result of tendovaginitis, stretching of the fixing joint-capsular apparatus in chronic synovitis («rheumatoid brush», Jaccoud's arthropathy in SLE), massive nodules, which are sodium uric acid deposits (tophus in gout). Attention is paid to typical deformations of fingers and hands: ulnar deviation of the hand, «swan neck» and «button loop» fingers in RA, «telescopic» fingers in PsA, dactylitis (diffuse swelling of the finger, tenosynovitis of the tendon and reaction of surrounding tissues) in seronegative spondyloarthritis and sarcoidosis.

An objective examination also includes conducting functional tests to detect sacroiliitis (Kushelevsky), which is of great diagnostic value, as well as determining the amount of active and passive movements in the joints and various parts of the spine.

When examining the patient's limbs, they note the presence of amyotrophies that develop in the corresponding area of the affected joints in many chronic arthritis, especially if they are accompanied by persistent pronounced pain (atrophy from inactivity) and signs of systemic inflammation. The most typical is the development of muscle atrophy for RA (hands, hips, shoulders), coxarthrosis (hips and buttocks), less often it occurs in ReA, severe chronic PsA (mainly hip muscles). Pronounced atrophy of the limbs muscles, as a rule, is asymmetric, can develop with PAN as a result of peripheral neuritis typical for this vasculitis.

Rashes on the skin, symptoms of vascular damage, nodule formations, most of which are characteristic of a limited range of nosologies, deserve close attention during the examination of the patient. Ring-shaped erythema, as a rule, multiple – a major diagnostic criterion of ARF. Her favorite localization is the trunk and proximal parts of the limbs. An erythematous rash on the bridge of the nose and cheeks («butterfly»), in the décolletage area, in the interarticular areas of the skin of the hands and forearms is a classic sign of SLE. In dermatomyositis, erythematous spots or papules are persistent, have a purple color, are located over the extensor surfaces of joints (Gottron's symptom), around the eyes (symptom of «glasses»), in the «shawl» area. Polymorphic rash (erythema, papules, vesicles, purpura) occurs in patients with SLE and PAN. A small palpable hemorrhagic rash (purpura) which is not associated with thrombocytopenia – a classic clinical sign of hemorrhagic vasculitis. Her favorite localization is in the area of the ankle joints and lower legs, which can then spread upwards. The development of purpura is possible in systemic vasculitis with damage to small vessels (cryoglobulinemic vasculitis, microscopic polyangiitis, Wegener's granulomatosis, Churg-Strauss syndrome), and in these vasculitis and sometimes in PAN more often than in hemorrhagic vasculitis, confluent hemorrhagic-necrotic elements appear. For example, with Wegener's granulomatosis, one of the frequent localizations of draining hemorrhagic foci is the extensor surface of the elbow joints. To detect psoriatic plaques, it is necessary to examine not only the «traditional» areas of the skin (extensor surfaces of the elbows, knee joints, fingers, behind the ears and inner ear areas), but also hidden localization – the hairy part of the head, navel, inguinal areas, perineum and gluteal folds. Psoriatic nail lesions are usually found next to inflamed finger joints. If a patient with oligoarthritis of the limbs has a papulo-pustular rash on the soles of the feet, the diagnosis of ReA is highly likely. Keratoderma of the soles and/or palms has the same diagnostic value.

No less than a skin rash, the detection of signs of vascular damage in patients with joint syndrome is of diagnostic value. Raynaud's syndrome most naturally occurs in SSD (90 % of patients), much less often in Sjögren's disease, SLE and RA, and it is precisely in SSD that it has a poor prognostic value and often leads to necrosis of the skin of the fingertips or gangrene, osteolysis of nail phalanges. Painless point, sometimes linear necrosis of the skin of black or brown color around the nail bed (digital arteritis) can be detected in patients with SLE, less often – with RA and dermatomyositis. Ulcerative-necrotic lesions of the skin are found in PAN, SLE, sometimes – in RA, antiphospholipid syndrome, SSD. Their frequent localization is the lower legs and the area around the ankle joints. In some cases of SLE, ulcers occur against the background of livedo and secondary antiphospholipid syndrome. In patients with PAN and SSD, necrosis is multifocal, including the skin of the upper limbs and trunk, but never occurs on the scalp and neck. Ulcerative lesions of the skin can also be observed in undifferentiated systemic necrotizing vasculitis, necrotizing skin vasculitis, including those associated with AIDS and hepatitis C. Gangrene of the fingers of the upper extremities - a consequence of obliterating arteriopathy - is more common in SSD, much less often – in RA and PAN, as casuistry – with SLE. If gangrene of fingers in a patient with joint syndrome occurs on the lower extremities, first of all, it is necessary to rule out obliterative thromboangiitis. Another important visual pathological vascular phenomenon that can be detected in patients with joint syndrome is reticular or tree-shaped livedo. The diagnostic value of this type of vascular damage is quite high, since it is combined with joint syndrome in only two diseases – SLE and PAN. Without arthritis, livedo can be evidence of the presence of antiphospholipid syndrome (primary or secondary – with SLE) or be identified as an independent pathology, including livedo-vasculitis.

Quite often, nodular formations can be detected in patients with joint syndrome. In clinical practice, their most common variant is erythema nodosum – painful inflammatory infiltrates in the skin and subcutaneous tissue with hyperemia and hyperthermia of the skin above them, several centimeters in diameter, without clear borders with the surrounding tissues. They are located first in the lower third of the lower leg, usually on both sides, then they can spread in the cranial direction, sometimes including the anterior abdominal wall and upper extremities. The formation of nodes in a typical place – on the front surface of the lower third of the lower leg, where the lymph outflow paths are located – is often accompanied by swelling of the distal part of the limb. In approximately half of the cases, erythema nodosum occurs in connection with infections, usually nasopharyngeal, or allergic reactions, and is considered an independent nosological form (allergic, infectious-allergic), in other cases it is a manifestation of other diseases: sarcoidosis, tuberculosis of the throat, yersiniosis, Crohn’s disease, Behcet's disease (listed in order of decreasing frequency). Heberden’s and Bouchard’s nodules are also among the most common nodular formations. They are usually painless or mildly painful, very dense and immovable. Rheumatoid nodules are one of the systemic manifestations of seropositive RA. During the period of their formation, as manifestations of nodular vasculitis, they can be painful, elastic, later, when fibro-sclerotic changes prevail, the nodules become painless. Depending on whether they are located subcutaneously or subperiosteally, nodules are removable or not. The size is from a few millimeters to 2 cm. Their favorite places of localization are the proximal third of the extensor surface of the forearm (very rarely the top of the elbow), the fingers of the hands, the wrist, less often - the front surface of the lower leg and knee joint, small joints of the feet, the area of the buttock humps, as a casuistry, the occipital region, the Achilles tendon. Tophus (surrounded by a fibrous capsule of conglomerates of sodium uric acid crystals with a size of 0.5 to 5 cm) are found in patients with gout, which usually lasts at least 5 years. They are always painless, stony density, are localized subcutaneously (therefore they are displaced), can be either single or in the form of draining conglomerates. They are located on the top of the elbow, on the auricles, above the small joints of the hands and feet. Through the thinned skin, they shine through as a whitish crusty mass, sometimes open and partially empty. Unlike rheumatoid nodules, their size and number are not affected by the use of anti-inflammatory drugs, but under the influence of pathogenetic (hypouricemic) therapy, they can decrease and even disappear completely. Externally, calcifications of soft tissues are practically indistinguishable from tophi – an attribute of SSD. They are very dense, absolutely painless, localized in the accessible parts of the limbs, very typically – on the pads of the fingers of the hands. They can ulcerate with the release of a curd-like mass.The easiest way to differentiate sclerodermic calcifications and tophi is radiography (the former are radiopaque, the latter are not) or examination of the contents of nodules under a polarizing microscope. Subcutaneous and intradermal nodules – small, usually single, moderately painful on palpation, without inflammatory reaction of the surrounding tissues or with hyperemia of the skin above them – occur in PAN, SLE, ARF and isolated cutaneous vasculitis. They are found mainly on the lower extremities and the front abdominal wall.

Detection of mucosal lesions in a patient with joint syndrome will also help with establishing a diagnosis. Thus, aphthous stomatitis occurs in SLE (painless or mild), Behçet's disease (morbid, erythematous asymptomatic enanthema of the hard palate in SLE; painless erosions of the oral mucosa and ring-shaped balanitis – in ReA; painful recurrent ulcers on the genitals, scrotum – in the disease Behchet.

Certain diagnostic information can be obtained by palpation of the lymph nodes. Generalized lymphadenopathy occurs in SLE, RA and its individual cases – Steele’s syndrome and Felty’s syndrome, less often – in Sjögren’s disease. With the exception of lymphoproliferative diseases, lymph nodes are usually small in size, little or painless, mobile. Regional lymphadenopathy can be detected in RA, ReA, infectious arthritis. In infectious bacterial arthritis, sometimes in ReA, nodes can be painless.

2.6. Instrumental diagnosis of joint syndrome

Among the instrumental research methods, radiographic is rather common. In the early stages of arthritis, radiography is not very informative, as it allows to visualize only an increase in the volume of soft tissues due to edema, rarely – an expansion of the joint space with accompanying synovitis. An exception is infectious bacterial (purulent) arthritis, in which already in the first 1–2 weeks, signs of osteolysis of one of the epiphyses can be detected against the background of peri-articular osteoporosis and narrowing of the joint space. Thus, at the stage of the clinical debut of joint syndrome, the main purpose of this method is to rule out traumatic damage (bone cracks, intra-articular fractures, compression fractures of vertebrae, etc.), or other, as long as subclinical, bone pathology (aseptic necrosis, osteomalacia, Paget’s disease, primary bone tumors or metastases, etc.) or cartilage pathology (calcification in pyrophosphate arthropathies). In case of non-inflammatory arthritis, the diagnostic value of radiography can be higher: for example, the detection of osteophytes, subchondral sclerosis (less significant – subchondral cyst, narrowing of the joint space) in a patient with the corresponding clinical picture is a sufficient argument in favor of the diagnosis of OA of the peripheral joints and spine. At the same time, it is necessary to remember the well-known dissociation between clinical symptoms and radiological changes in OA, so the absence of characteristic radiographic symptoms is not an obstacle to establishing a clinically justified diagnosis of OA, just as their presence should not be considered as a decisive diagnostic argument without appropriate clinical support.

With a long course of arthritis (minimum – several months, usually – years), in most cases, radiography allows to detect bone changes, some of which are characteristic or even specific for certain nosologies.

In the vast majority of other causes of inflammatory joint syndrome (systemic connective tissue diseases, systemic vasculitis, rheumatic fever, infectious viral arthritis, Löfgren’s syndrome), significant changes on radiographs are absent or nonspecific (moderate osteoporosis of the epiphyses, slight narrowing of the joint space, etc.) and not have diagnostic value. Undoubtedly, the value of this method remains for the detection of other bone pathology specific to some systemic diseases (aseptic bone necrosis in SLE and SSD, osteolysis of nail phalanges in SSD). The main disadvantage of radiography is the late diagnosis of arthritis, since only structural changes in bones and calcified soft tissues are detected. The method does not allow obtaining information about inflammation of bones (osteitis), intra-articular and peri-articular tissues, which is of fundamental importance for the timely diagnosis of RA, seronegative spondyloarthritis.

The same disadvantages, as well as a high dose of radiation, are inherent in computer tomography. For this reason, it is mainly used for the study of complex joints and bone structures, which are overlapped by massive soft tissues (shoulder, hip joints, vertebrae, pelvic bones, other flat bones) in conventional radiography. Much more informative methods in the early stages of joint syndrome are ultrasound (ultrasound) and magnetic resonance imaging (MRI). The main advantages of these methods are the possibility of visualization of inflammation in bones (first of all, MRI) and non-bone structures, including detection of synovitis, tendovaginitis, pannus, rheumatoid nodules, enthesitis; diagnosis of bone erosions and cartilage degradation at earlier stages, absence of radiation exposure.

The US method is affordable, economical (compared to all methods except radiography), can be used multiple times, including for dynamic visual control of intra-articular diagnostic and therapeutic manipulations. However, it is not sensitive enough to detect structural changes in bone tissue, its results strongly depend on the technical capabilities of the device and the doctor's qualifications.

One of the main limitations of MRI is the impossibility of using it in people with implanted metal objects, an electric pacemaker. In such cases, in addition to ultrasound, X-ray computed tomography is used.

Thus, for the correct diagnosis of joint syndrome and the establishment of a rheumatological diagnosis, a decisive role is played by: interviewing the patient, clinical examination of joints, skin and mucous membranes, the diagnostic value of which in most cases is leading in relation to laboratory and instrumental research methods. If you focus only on the results of the latter, you can come to an incorrect diagnosis.

2.7. Laboratory diagnosis of joint syndrome

In order to maintain a rational ratio of price/diagnostic significance, all laboratory studies in patients with joint syndrome should be divided into first-line and second-line (special) tests. The first include clinical tests of blood and urine; blood tests to determine C-reactive protein (CRP), rheumatoid factor (RF), antinuclear factor (ANF), anti-streptolysin-O (ASL-O), protein fraction, uric acid level, aminotransferases, less often (according to clinical indications) other biochemical indicators, as well as synovial fluid analysis. If, according to the results of the first-line tests, the diagnosis does not become clear or there is a suspicion of less common diseases, second-line studies are prescribed: immunological (antibodies to cyclic citrullinated peptide (anti-CCP), native DNA, Sm, Ro, La, centromere, cardiolipin, pathogens intestinal and genitourinary infections, pANCA, cANCA, SZ, C4, etc.), microbiological (culture of synovial fluid, PCR analysis, scrapings from the urethra, etc.), biochemical (CPK, serum iron, ferritin, etc.), determination of HLA B27, examination of synovial fluid for crystals, etc. Of course, if the clinical picture is quite unambiguous, the majority of screening tests and, selectively, some special tests aimed at a certain diagnosis and control of the safety of possible treatment are immediately prescribed.

In non-inflammatory joint diseases (OA of peripheral joints and spine, periarthritis, Forestier's disease, Tietze syndrome, etc.), laboratory changes are not detected. In inflammatory arthropathies, in the laboratory results, you can find both non-specific changes that occur in many inflammatory diseases, including joints, and characteristic / specific for certain nosologies.

With chronic or subacute inflammatory diseases of the joints, normochromic anemia is usually recorded, with the exception of gout, in which, if there is no kidney damage or bleeding, the hemoglobin level tends to the upper limit of normal. RF is a characteristic but non-specific immunological marker for RA. It manifests itself in many systemic rheumatic diseases (albeit in low titers), in a number of non-rheumatic diseases, including infectious endocarditis, chronic active hepatitis, tuberculosis, syphilis, lymphogranulomatosis, sarcoidosis, and in 1–4 % of healthy individuals under the age of 60 and 5–1,200 people over 60 years of age. Therefore, a more specific indicator – anti-CCP – is currently used to confirm the diagnosis of RA.

It should be remembered that antibodies of the lgG class to ChIamidia trachomatis are only evidence of a previous chlamydial infection and can be determined for many months even after successful treatment. A recent (several weeks) infectious process and, accordingly, its possible connection with the joint syndrome can only be indicated by antibodies of the lgM class and/or detection of the DNA of the pathogen by the PCR method in the cylindrical epithelium of the genitourinary tract, scrapings from the urethra and cervix, or urine analysis.

In the study of synovial fluid, two of its analyzes have the greatest diagnostic value:

1) bacterioscopy after Gram staining (and/or Ziel-Nielsen if mycobacterial infections are suspected) and culture to detect bacterial flora (infectious bacterial arthritis);

2) research in a polarizing microscope to identify crystals of sodium uric acid or calcium pyrophosphate (gout and chondrocalcinosis). Distinguishing between inflammatory and non-inflammatory arthritis based on a general analysis of synovial fluid is quite unreliable due to significant fluctuations in its color, sediment, mucin clot density and viscosity, cytosis, protein and glucose content,

due to the duration of synovitis, treatment, the almost always present inflammatory component of non-inflammatory arthritis (first of all, in those cases where it is possible to obtain synovial fluid for analysis).

Certain diagnostically significant information can be obtained from a clinical urinalysis.

In order to systematize the use of the above information on the diagnosis of joint syndrome in rheumatic patients, and the successful curation of patients in practical classes, familiarization with two nosologies is recommended: 1. Osteoarthritis and 2. Gouty arthritis (gout).

2.8. Management of patients with joint syndrome on the example of osteoarthrosis.

Osteoarthrosis / Osteoarthritis (OA) is a heterogeneous group of diseases of different etiology, but with similar biological, morphological and clinical manifestations, the basis of which is progressive degenerative damage to all components of the joint, primarily cartilage, subcartilaginous bone, synovial membrane, capsule ligaments and periarticular muscles, with the formation of marginal osteophytes, as well as overt or hidden moderate synovitis.

Clinical classification of osteoarthrosis

I. Primary (idiopathic):

A. Localized (less than 3 joints):

1. Joints of the hands;
2. Foot joints;
3. Knee joints;
4. Hip joints;
5. Spine;
6. Other joints.

B. Generalized (3 joints or more):

1. With damage to the proximal and distal interphalangeal joints (Heberden’s nodules, Bouchard's nodules);

2. With damage to large joints;

3. Erosive.

II. Secondary:

A. Congenital, acquired or endemic diseases (Perthes disease, hypermobility syndrome, etc.);

B. Metabolic diseases:

1. Ochronosis;

2. Hemochromatosis;

3. Wilson's disease;

4. Gaucher disease.

C. Endocrinopathies:

1. Acromegaly;

2. Hyperparathyroidism;

3. Diabetes mellitus;

4. Hypothyroidism.

D. Calcium pyrophosphate deposition disease.

E. Neuropathies (Charcot's disease).

F. Other diseases.

III. X-ray grades (for J.H. Kellgren and J.S. Lawrence):

0 – no radiographic changes;

I – doubtful X-ray signs;

II – minimal changes (slight narrowing of the joint space, subchondral osteosclerosis, isolated osteophytes);

III – moderate manifestations (moderate narrowing of the joint space, multiple osteophytes);

IV – pronounced changes (joint gap is almost not visualized, coarse osteophytes).

IV. Synovitis:

- with synovitis,
- without synovitis.

The main components of OA therapy

1. Non-drug treatment:

– patient education,

– diet therapy and reduction of excess body weight,

– orthopedic mode,

– increased physical activity and physical therapy,

– physiotherapy treatment,

– rehabilitation.

2. Pharmacotherapy:

– drugs of symptomatic action:

– fast-acting (NSAIDs, glucocorticosteroids – GC_S),

– slow action (chondroitin sulfate, glucosamine sulfate, hyaluronic acid, diacerein).

– drugs of general action:

– medicines that improve microcirculation,

– inhibitors of proteolytic enzymes,

– metabolic drugs.

3. Intra-articular treatment:

– chondroprotectors for intra-articular use,

– anti-inflammatory and antihomotoxic therapy,

– glucocorticosteroid therapy,

– lavage

4. Surgical treatment:

– arthroscopic intervention,

– corrective osteotomies,

– endoprosthesis of joints and arthroplasty,

– arthrodesis

Examples of formulation of OA diagnoses

1. Osteoarthrosis: oligoosteoarthrosis with damage to the left hip joint (left-sided coxarthrosis), Rö III grade; knee joints (bilateral gonarthrosis), Rö II grade; synovitis of the right knee joint.
2. Osteoarthrosis: polyosteoarthrosis (nodular form) with damage to the proximal and distal interphalangeal joints of the hands, Rö III grade; carpocarpal joint of the first finger of the left hand with synovitis; metatarsal-phalangeal joint of the first toe of the left foot (hallus valgus) with synovitis; right hip joint (right-sided coxarthrosis), Rö IV grade; and cervical spine.

2.9. Management of patients with joint syndrome on the example of gouty arthritis / gout

Gouty arthritis/gout (GA) – a chronic progressive disease that is associated with purine metabolism disorders, occurs against the background of hereditary predisposition and non-compliance with the nutritional regime, is accompanied by a significant increase in the uric acid salts content in the blood with subsequent deposition of their crystals in various tissues, primarily near-scapular and articular, and clinically manifested by paroxysmal arthritis and frequent damage to internal organs.

Clinical classification

I. Clinical stages:

1. acute gouty arthritis
2. intercritical (interval) gout
3. chronic gouty arthritis:
   - aggravation,
   - remission.

4. chronic tophous arthritis

II. X-ray grades of joint damage:

I – large cysts (tophus) in the subchondral bone (symptom of the «puncher») and in deeper layers, sometimes compaction of the articular surfaces of soft tissues

II – large cysts near the joint and small erosions of the joint surfaces, constant compaction of peri-articular soft tissues, sometimes with calcifications

III – large erosions of at least 1/3 of the articular surface, osteolysis of the epiphysis, significant compaction of soft tissues with calcium deposits

III. Nephrolithiasis

– Gouty nephropathy.

The main components of gouty arthritis/gout therapy

1. Non-pharmacological means:

- Diet:

– restriction of meat and fish products, legumes, strong coffee and tea;

– stop drinking alcoholic beverages (especially beer);

– increasing the amount of liquid drunk (up to 2–3 liters per day).

- Reduction of excessive body weight;
- Smoking cessation
- Regular exercise;
- Correction of concomitant diseases.

2. Relieving an acute attack:

– NSAIDs (colchicine, etc.)

– GC_S – intra-articular, oral or intramuscular

3. Treatment in the inter-attack period (basic treatment – hypouricemic agents):

- uricodepressants,
- uricosuric drugs,
- drugs that increase the solubility of uric acid in urine.

4. Physiotherapy procedures.

5. Sanatorium-resort treatment.

Examples of formulation of diagnoses of gouty arthritis/gout

1. Gout, gouty arthritis, acute course with damage to the first metatarsophalangeal joint of the right foot; Rö 0 grade. Nephrolithiasis.

2. Gout, gouty arthritis, chronic course, stage of exacerbation with damage to the joints of the feet, knee joints; Rö II grade. Peripheral tophi in the area of the auricles. Gouty nephropathy (chronic gouty interstitial nephritis, nephrolithiasis, CKD I stage). Symptomatic arterial hypertension of renoparenchymal origin.

3. Gout, gouty arthritis, chronic course, stage of exacerbation with damage to ankle and elbow joints; Rö II grade. Secondary osteoarthritis. Peripheral tophi in the area of the auricles and elbow joints. Gouty nephropathy (urolithiasis, chronic pyelonephritis, CKD II stage).

TEST TASKS FOR SELF-CONTROL

1. To stop a gout attack use:
   A. Allopurinol;
   B. Colchicine;
   C. Indomethacin;
   D. Naproxen;
   E. Fenbuxostat.

2. What is the nature of the pain syndrome inherent in osteoarthritis in contrast to idiopathic arthritis:
   A. Mixed;
   B. Acute;
   C. Inflammatory;
   D. Mechanical;
   E. Nocturnal.

3. Choose hypouricemic agent for the basic treatment of gouty arthritis:
   A. Diclofenac;
   B. Methylprednisolone;
   C. Ketoprofen;
   D. Celecoxib;
   E. Allopurinol.

4. To confirm the diagnosis of gouty arthritis, it is necessary to determine the following laboratory indicators:
   A. C-reactive protein;
   B. Antinuclear cytoplasmic antibodies (ANCA);
   C. Rheumatoid factor;
   D. Uric acid;
   E. Urea and creatinine.

5. The leading mechanism in the pathogenesis of osteoarthritis is:
   A. Violation of purine metabolism;
   B. Cartilage degeneration;
   C. Autoimmune inflammation in the joints;
   D. Chlamydial infection;
   E. Previous trauma to the limb.

6. The leading mechanism in the pathogenesis of gouty arthritis is:
   A. Violation of purine metabolism;
   B. Cartilage degeneration;
   C. Autoimmune inflammation in the joints;
   D. Chlamydial infection;
   E. Previous joint injury;

7. What is the nature of the pain syndrome inherent in an attack of gouty arthritis:
   A. Mixed;
   B. Acute;
   C. Inflammatory;
   D. Mechanical;
   E. Nocturnal.

8. What X-ray changes are characteristic of osteoarthritis:
   A. Osteosclerosis, osteophytes;
   B. Osteoporosis, narrowing of the joint space;
   C. Symptom of «puncher»;
   D. Expansion of the joint gap;
   E. Osteoporosis, patterns/erosions of joint surfaces.

9. An X-ray of the patient's knee joints revealed: narrowing of the joint space, subchondral osteosclerosis, single osteophytes. What X-ray grade of osteoarthritis is characterized by the above changes:
   A. 0;
   B. I;
   C. II;
   D. III;
   E. IV.

10. What nepharmacological agents are used to treat a patient with gouty arthritis?
   A. Diet;
   B. Reduction of excessive body weight;
   C. Smoking cessation;
   D. Regular exercise;
   E. All of the above.

11. Heberden's nodules in the nodular form of osteoarthritis are:
   A. Granuloma formation;
   B. Tofus;
   C. Bone growths (osteophytes) on the lateral surfaces of the distal interphalangeal joints;
   D. Bone growths (osteophytes) on the lateral surfaces of the proximal interphalangeal joints;
   E. None of the above.

KEYS

1. – B; 2. – D; 3. – E; 4. – D; 5. – B; 6. – A; 7. – B; 8. – A; 9. – C; 10. – E; 11. – C.

SITUATION TASKS

1. The female is 56 years old, complains of pain in the carpal and knee joints. She has been ill for 8 years. Body temperature is normal. The interphalangeal joints of the hands are thickened and painful during palpation, crunch in the knee joints when bending. X-ray of the knee joints: osteoporosis, narrowing of the joint space, minor bony growths on the edges of the joints. Blood analysis: leuk. 6.2×109/l, ESR 13 mm/h. What is the diagnosis?

A. Osteoarthritis with predominant damage to the knee and carpal joints;
B. Acute rheumatic fever;
C. Gouty arthropathy;
D. Rheumatoid arthritis;
E. Reactive polyarthritis.

2. A 64-year-old female patient complains of pain in the knee joints that occurs 2–3 hours after walking. The examination revealed deformation of the knee joints, limitation of mobility in them due to pain, crunching during movements, Heberden' nodules on both hands. In the anamnesis – peptic ulcer disease. Which of the listed drugs should be prescribed to the patient?

A. Indomethacin;
B. Diclofenac;
C. Meloxicam;
D. Piroxicam;
E. Acetylsalicylic acid.

3. A 56-year-old woman complains of pain in the knee joints. The joints are enlarged, the movements are limited and painful, there is a crunch in the joints during movements. An X-ray examination is prescribed. What radiological changes would you expect to see?

A. Osteoporosis, narrowing of the joint space;
B. Osteosclerosis, osteophytosis;
C. Osteosclerosis, a symptom of a «puncher»;
D. Expansion of the joint gap;
E. Osteoporosis, erosions of joint surfaces.

4. A man, 30 years old, complains on severe pain, «redness» of the skin, swelling in the area of the ankle joint, temperature rise to 39° C. He fell ill suddenly. In the past, there were similar attacks lasting up to 5–6 days without final changes from the side of the joint. The skin over the joint is hyperemic without clear contours and an infiltrative shaft on the periphery. What is the most likely diagnosis?

A. Infectious arthritis;
B. Rheumatoid arthritis;
C. Erysipelas inflammation;
D. Gout;

E. Reactive arthritis.

5. The patient is 36 years old suffers from gout. After consuming alcohol and fatty food the night before, an attack of acute arthritis of the 1st metatarsophalangeal joint of the right foot occurred at night. The joint is bluish, sharply painful, swollen. Blood analysis: leuk. 17×109/l, ESR 42 mm/h. Choose a therapy option:

A. Allopurinol 300 mg/day, diclofenac 75 mg IM 2 twice/day;
B. Allopurinol 100 mg, Diprospan intra-articularly;
C. Allopurinol 300 mg;
D. Colchicine 0.5 mg 4 times a day;
E. Diclofenac 100 mg/day.

KEYS

1. – A; 2. – C; 3. – B; 4. – D; 5. – D.

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Електронне навчальне видання комбінованого використання
Можна використовувати в локальному та мережному режимі

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ВЕДЕННЯ ПАЦІЄНТА З СУГЛОБОВИМ СИНДРОМОМ (ОСТЕОАРТРОЗ, ПОДАГРИЧНИЙ АРТРИТ)

Методичні рекомендації
для підготовки здобувачів вищої освіти 6-го року навчання
з дисципліни «Внутрішня медицина»

(Англ. мовою)

В авторській редакції

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