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ARTICULAR SYNDROME IN THE PRACTICE OF A RHEUMATOLOGIST

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ABSTRACT

Articular syndrome is a clinical set of symptoms, expressed by joint pain, morning stiffness, joint deformity, mobility restriction, and pathological changes in the ligaments. The most important diagnosis components for the disease underlying the patient's existing articular syndrome are: a properly collected anamnesis, a medical examination, a complex of laboratory tests and radiation imaging methods.

The article aims at helping the doctor in the early diagnosis of joint disease underlying the patient's existing articular syndrome. The article also discusses the main elements of the articular syndrome, highlights the characteristic symptoms and key manifestations of common rheumatic diseases, based on the articular syndrome, and presents modern methods for the diagnosis of this syndrome. Timely assessment of the articular syndrome contributes to the formation of an appropriate program comprising laboratory and instrumental research methods for patients, allows to suspect rheumatic disease at an earlier stage and promptly refer patients to a rheumatologist. In turn, timely diagnosis of rheumatic disease and early indication of basic therapy by a rheumatologist to patients with articular syndrome will improve the life quality and prognosis of such patients.

Keywords: rheumatic diseases, articular syndrome, diagnostics, adiography, ultrasound, magnetic resonance imaging.

Joint syndrome is a clinical symptom complex manifested by pain in the joints, morning stiffness, deconfiguration and deformation of the joints, limitation of their mobility, pathological changes in the tissues surrounding the joint [1, 2]. The most important components of the diagnosis of the disease underlying the patient's joint syndrome is a properly collected anamnesis, general examination, examination of the musculoskeletal system, organs of cardiovascular, respiratory, digestive and genitourinary system, as well as conducting a complex of laboratory tests and radiation diagnostic methods [3, 4].

The clinical manifestations of joint disease are non-specific and include pain, morning stiffness, local signs of inflammation, such as hyperemia, hyperthermia, swelling, as well as soreness, determined during the examination of the joints, a change in the shape of the joints and a limitation of the amplitude of movement in the joints [1, 3, 4]. One of the main parameters of the joint syndrome is pain. The cause of pain can be damage to muscles or periarticular tissues (ligaments, tendons, bursa), damage to the skin, blood vessels, peripheral nerves, joint damage. An important stage in the differential diagnosis of pain is the determination of its nature. The pain can be inflammatory or degenerative (mechanical) in nature [1, 3, 5, 6].

Pain of an inflammatory nature is characteristic of arthritis, for example, rheumatoid arthritis (RA), spondyloarthritis (SpA). It is constant, often symmetrical, more pronounced at rest, more disturbing to patients in the morning; combined with morning stiffness, lasting more than half an hour; decreases or passes in the evening or after physical exertion [6, 9].

SpA is a group of inflammatory RH, divided into peripheral (psoriatic arthritis (PsA), arthritis in ulcerative colitis and Crohn's disease, undifferentiated SpA, reactive arthritis (ReA)) and axial SpA (ankylosing spondylitis (AS) and axial SpA without radiographic signs of AS) [8, 13]. This group of diseases is characterized by back pain of an inflammatory nature lasting more than 3 months. and the presence of at least four of the five signs, such as gradual onset, debut at a young age (before age 40), pain disturbing in the early morning and at night, pain at rest and its decrease after physical exertion [6, 9, 10, 14,16].

Pain of a degenerative (mechanical) nature is characteristic of osteoarthritis (OA). It occurs under the influence of physical exertion. At rest, it decreases or does not bother. Subsides during the night. OA is also characterized by short-term "starting" pain that occurs at the beginning of the patient's movement, after a period of rest, passing 15–20 minutes from the beginning of the movement [6, 17]. The severity of joint pain is assessed using a 10-point visual analog scale (VAS). To do this, ask the patient to note the severity of pain on YOUR, where a score of 0 means no pain, 10 - unbearable pain [3, 4].

Another important parameter of joint syndrome is soreness , a typical sign of inflammation. It occurs in response to palpation of the joint with a force of about 4 kg / cm^2 (until the phalange of the finger turns white). Even in the absence of obvious swelling, the soreness of the joint on palpation indicates its defeat [3].

A symptom valuable for the differential diagnosis of joint syndrome is the syndrome of morning stiffness - a feeling of stiffness in the affected joints, requiring their "development", especially in the morning. So, the symptom of morning stiffness in the hands lasting more than 30 minutes is characteristic of RA. Usually it bothers at rest, increases in the second half of the night, by the morning, can be observed in various joints. The symptom of morning stiffness in the spine in the form of a feeling of "stiffness" is characteristic of SpA [3, 15, 16]. In contrast, patients with OA rarely complain of morning stiffness, the duration of which can be no more than 30 minutes, its presence may indicate PeA in these patients [1, 5, 17].

Of great importance for the diagnosis is the localization of the pathological process and the distribution of pathological changes - symmetry. Symmetrical simultaneous lesion of the same articular zones on the right and left is characteristic of RA. Thus, a patient with RA has a symmetrical lesion of the proximal interphalangeal, metacarpophalangeal and wrist joints [3, 5, 18]. Asymmetric arthritis of the mainly lower extremities, sacroiliitis, combined with spondylitis, arthritis of all three joints of one finger with a "sausage-like" change in the shape of the joint is characteristic of the SpA group [1, 3, 10, 12, 15]. With acute arthritis of the I metatarsophalangeal joint, manifested by a bright pain syndrome and a change in skin color over the painful joint, the exclusion of gout is required [19]. It is important to determine the number of affected joints: monoarthritis - damage to one joint, oligoarthritis - two or three joints, polyarthritis - more than three joints [4].

The early stage of arthritis is characterized by deconfiguration of the joint - a reversible change in its shape. It occurs due to effusion into the joint cavity, thickening of the synovial membrane,

swelling of periarticular tissues. The late stage of arthritis is characterized by deformation of the joints, manifested by an irreversible change in their shape due to changes in bone tissue, the formation of subluxations and ankylosis [4, 6]. The most common deformities in RA are ulnar deviation of the fingers of the hand ("walrus fin"), deformation of the fingers by the type of "swan neck" and "button loop" [1, 6, 18]. The deformities detected in OA include Heberden and Bouchard's nodules. Another phenomenon typical for OA is the defeat of the I metatarsophalangeal joint, accompanied by the formation of a dense thickening [1, 6, 17].

Most patients with systemic lupus erythematosus (SLE) suffer from joint pain. More often, small joints of the hands and wrists are affected. In 5–40% of patients with SLE, changes in the joints of the hand are similar to those characteristic of RA. This phenomenon is called "Jacquet's syndrome". In patients with SLE, a rheumatoid-like hand is formed, not accompanied by bone destruction, the cause of the changes is the pathology of the ligamentous apparatus [1, 6, 20].

PsA as a representative of the SpA group is characterized by asymmetrical involvement of joints in the pathological process, damage to the distal interphalangeal joints of the feet and hands, three joints of one toe or hand. This is the so-called sausage- or radish-like deformation of the fingers, which occurs in 30–40% of patients and is a prognostically unfavorable sign of erosion [1, 6, 13].

It is important to carefully examine the skin of patients with joint syndrome, since skin lesions are one of the important manifestations of some RH accompanied by joint syndrome [5, 6]. So, it is not difficult to establish a clinical diagnosis of PsA if the patient has skin psoriatic plaques, nail lesions with indentations resembling a thimble (a symptom of a thimble), longitudinal and transverse delineation of the nails. Also, PsA is characterized by a violation of the connection between the nail bed and the nail - onycholysis. It is edge and central - in the form of oil stains. Sometimes psoriatic plaques are localized in places where the examination is difficult (for example, in the scalp, gluteal folds, in the navel). Both the fact of psoriasis in the patient and the existence of psoriasis in its close relatives are included in the diagnostic criteria for PsA [1, 6, 13].

Skin lesions are one of the important manifestations of RH, accompanied by joint syndrome. Evaluation of skin lesions helps in the differential diagnosis of joint syndrome. Skin lesions are one of the main diagnostic signs of systemic scleroderma (SSD). In patients with SSD, there is a thickening of the skin of the hands associated with dense edema and induration; progressive limitation of mobility and the formation of contractures - sclerodactyly; thickening of the skin - scleroderma, as a result of which the face becomes mask-like due to the development of dense edema, induration and further partial atrophy of tissues, the appearance of radial folds around the mouth, a decrease in the oral aperture - a symptom of "kiset"; the formation of digital scars on the skin of the distal phalanges of the fingers of the hands is the so-called symptom of a "rat bite". Sclerodactyly and scleroderma make it possible to diagnose SSD at the first meeting with the patient [1, 6, 21]. Skin syndrome in SLE is expressed by photosensitivity, erythema on the face, resembling a "butterfly", discoid rash [20].

On the extensor surfaces of such joints as elbow, knee, ankle, in the Achilles tendon, on the extensor surfaces of the metatarsophalangeal and metacarpophalangeal joints, in the projection of the spinous processes of the vertebrae, rheumatic nodules located in small groups can be seen. They are rounded, dense, sedentary, painless, with a diameter of 1 mm to 1 cm [22, 23].

On the extensor surfaces of the elbow joints, forearms, less often - in the area of small joints of the hands and feet, rheumatoid nodules can be localized subcutaneously. They are rounded, dense, subcutaneously located, mobile, painless, with a diameter of 2–3 mm to 2–3 cm [18, 23]. Involvement in the pathological process of the mucous membranes (conjunctivitis, anterior uveitis, stomatitis, cervicitis, urethritis, balanitis), skin (keratoderma of the plantar part of the palms and feet), enthesies is characteristic of SpA, in particular for ReA, which is associated with urogenital or intestinal infection [11, 12, 23]. In chronic gout, crystals of sodium monourate or uric acid ranging in size from a few millimeters to 1-2 cm (tophi) are deposited in various tissues of the body. Their most frequent localization is the auricles, elbows, fingers of the hands and feet. In postmenopausal women, tophi can be located in the region of Heberden's nodules [9, 18].

The next important parameter of the joint syndrome is a decrease in the volume of motion in the joints due to pain and changes in the structure of the musculoskeletal system. Arthritis is characterized by the restriction of active and passive movements. For periarthritis, neurological pathology, it is characterized by the restriction of only active movements. The absence of a difference in the volume of active and passive movements may indicate a bone blockade. With movements, the patient may hear a "crunch". This symptom is characteristic of OA and advanced inflammatory arthritis or joint hypermobility syndrome [3–6].

Diagnosis of the causes of joint syndrome requires a careful study of the patient's history. In 70% of cases, it allows you to reliably establish a diagnosis. It is necessary to establish the connection of the disease with trauma, infections or other provoking factors [1, 3, 5]. Injuries and chronic microtraumas in the anamnesis may testify in favor of OA [1, 17]. Transferred streptococcal infection (sore throat, pharyngitis, scarlet fever) can be the cause of the development of acute rheumatic fever, in favor of which the presence of articular syndrome, rheumatic carditis, small chorea and an increase in the titer of streptococcal antibodies will testify [22]. Acute joint pain, the cause of which could be the intake of alcohol or abundant meat and fatty foods the day before, prolonged use of diuretics, indicate an acute gouty attack. A gouty attack can also be triggered by prolonged walking, trauma, and surgical procedures [19]. In the presence of a clear chronological connection of asymmetric arthritis, mainly the joints of the lower extremities, sometimes even sacroiliitis, with an intestinal or urogenital infection, it is necessary first of all to exclude ReA [1, 12]. With arthritis, a thorough examination of not only the musculoskeletal system, but also other organs and systems is necessary, since arthritis can be an early manifestation of not only RH, but also infectious and oncological diseases [3, 5, 23].

Modern laboratory diagnostics is an important part of the examination of a patient with a possible RH. However, there is no single laboratory indicator sufficient to make a diagnosis, determine the prognosis of the disease, its stage and activity. All results should be interpreted in accordance with the clinical picture [5, 23]. The erythrocyte sedimentation rate (ESR) and the content of C-reactive protein (CRP) allow us to assess the activity of inflammation, the severity of RH [23, 24]. Serum detection of rheumatoid factor (RF) and antibodies to cyclic citrullinated peptide (ACCP) are diagnostic criteria for RA. RF is a sensitive, but not specific enough marker. For early diagnosis of RA, including differential diagnosis with other RHs, it is

of great importance to determine ACPD as a more highly specific diagnostic marker [6, 18, 23, 24]. Antinuclear antibodies (AHA) are detected in many patients with RH, in particular in all patients with SLE and SSD. Antibodies to double-stranded DNA are a serological marker of SLE. SpA is characterized by an association with the histocompatibility antigen HLA B27, negative RF and AHA in the blood serum [6, 23, 24]. With OA, a moderate increase in ESR and CRP is possible, which is characteristic of secondary synovitis, their pronounced increase requires the exclusion of another pathology [6, 17, 23, 24].

Of the instrumental methods for diagnosing the diseases underlying the articular syndrome, the most commonly performed are radiography, ultrasound examination (ultrasound) and magnetic resonance imaging (MRI) of the joints [2, 11, 25, 26].

The "gold standard" for diagnosing RH is an X-ray examination. However, in the debut of RZ, radiography does not allow to see early pathological changes in the joints [24], so currently the advanced methods of early diagnosis of RH are ultrasound and MRI [26].

Ultrasound examination of the joints of the joints helps to identify subclinical synovitis, bone erosion, which is not yet visible during X-ray examination, to evaluate the ligamentous apparatus. Ultrasound allows you to visualize the decrease in the thickness and unevenness of the contours of hyaline cartilage, characteristic of the degenerative-dystrophic process; verify synovial proliferation (significant thickening of the synovial membrane due to granulation growths with the formation of large villi) and hypervascularization of the synovial membrane, characteristic of inflammatory arthritis [27–29]. Ultrasound of the joints also allows you to identify syndromes associated with arthritis of inflammatory genesis: tenosynovitis, tendonitis, fasciitis, polymyositis [27].

The use of MRI in rheumatology allows you to visualize signs of active inflammation of the peripheral joints (effusion, synovial changes, bone marrow edema), subsequent structural changes (damage to the articular surface, cortical bone erosion), signs of active inflammation in the sacroiliac joints (bone marrow edema, capsulitis, synovitis, enthesitis) and structural changes (subchondral sclerosis, bone erosion, periarticular fat deposits, bone bridges, ankylosis) in them, inflammatory and post-inflammatory changes in the intervertebral joints (signs of active inflammation, aseptic spondylodiscitis, atlanto-axial / atlanto-occipital structural changes), tenosynovitis and enthesitis [30-32]. Ultrasound and MRI have comparable indicators of sensitivity in determining the activity of RH. The sensitivity of ultrasound in detecting erosion is low, since it is technically impossible to examine the entire joint [30, 31]. Also, with diseases of the joints, it is necessary to examine the organs of the gastrointestinal tract, since patients constantly take NSAIDs and, in particular, to conduct studies of the microbiocenosis of the stomach. [33].

CONCLUSION

Thus, the joint syndrome is a clinical symptom complex that includes any possible deviations from the joints. Competent assessment of the joint syndrome (the nature and localization of pain, morning stiffness, deformation, restriction of movement and other parameters) contributes to the correct formation of a program of laboratory and instrumental research methods for patients, allows you to suspect RH at an earlier stage of its development and timely refer patients to consultation with a rheumatologist. Timely diagnosis of RH and early

appointment of basic therapy by a rheumatologist to patients with articular syndrome can improve the quality of life and prognosis of RH in these patients.

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