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DISTRIBUTION REASONS, DEVELOPMENT MECHANIZM, FEATURES OF THE COURSE OF OSTEOARTHOSIS

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ABSTRACT

Osteoarthrosis is the most common joint disease that affects at least 20% of the world's population. The disease usually begins at the age of over 40. Osteoarthrosis of the knee joint (gonarthrosis) often develops in women, and hip joint (coxarthrosis) - in men.

OA is a multifactorial chronic progressive joint disease that develops as a result of mechanical and biological causes that destabilize the articular cartilage and subchondral bone normal relationships between the degradation and synthesis of the components of the matrix with chondrocytes. Osteoarthrosis is an urgent problem of modern medicine, which is confirmed by the steady increase in the cost of treatment of patients with morbidity.

Key words: osteoarthrosis, tendon-ligamentous apparatus, synovial membrane, subchondral bone, chondrocytes.

Joint pathology occurs in millions of patients, causing pain and disability and having a major impact on a person's lifestyle. Osteoarthritis is the most common of the joint pathologies, ranking second among women with disabilities and fourth among men. A recent report by the World Health Organization on the global prevalence of the disease showed that osteoarthritis of the knee is the 4th leading cause of disability in women and the 8th in men. Osteoarthritis occurs mainly among the elderly. According to the World Health Organization, this number is expected to double in the age group over 50 between 2000 and 2020.

The problem is exacerbated by the fact that the incidence of osteoarthritis has increased significantly in recent years: almost 1 percent of those seeking medical care among adults have osteoarthritis, so timely and effective treatment is of great medical, social, and economic importance. The prevalence of osteoarthritis in the population is age-related and peaks in people over 45 years of age. Some major studies of osteoarthritis were conducted in the 1960-1962 National Health Examination Survey (based on X-ray signs of osteoarthritis in the fingers and lower limbs) and the 1971-1975 NHANES - National Health and Nutrition Examination Survey (knee and pelvic joints).) The study found that one in three people between the ages of 25 and 74 had radiological signs of at least one localized osteoarthritis.

According to modern concepts, osteoarthritis is a group of heterogeneous diseases of various etiologies, characterized by a chronic course of biological, morphological and clinical changes, and the pathological process affects all components of the joint, mainly the uncle, subchondral bone, synovial membrane (shell), connective tissue, joint capsule, accompanied by degenerative-dystrophic processes in the pre-articular muscles. In addition, as a result of chronic stress is manifested by hemodynamic, neurotrophic changes in the musculoskeletal system. [2.8].

The development of osteoarthritis includes genetic, constitutional, acquired, environmental risk factors. Genetic defects, in particular Stickler syndrome, and type II collagen mutations, ethnic origin, hereditary diseases of the

musculoskeletal system, increased mineral density of bone tissue; constitutional-gender, the second period of puberty, old age, overweight; acquired - age-related decrease in estrogen, developmental disorders or anomalies in the bone and joint structure, acquired pathology, joint surgery, hormone therapy, smoking; environment - occupational characteristics and physical stress of the joints, a history of any trauma to the joint [8.6..11].

The progressive course of osteoarthritis is determined by mechanical, biochemical and genetic factors, as well as the manifestation of the inflammatory process in the subchondral bone, hyaline uncle, synovial membrane and periarticular soft tissues [5.7].

Primary pathogenetic factors of osteoarthritis include deficiency of proteoglycan synthesis in the joint, decreased concentration and degradation of proteoglycan aggregates, followed by dehydration of the joint, imbalance of anabolic and catabolic processes, A2 phospholipase and collagenosis, synaptic cleavage, inflammatory synthesis, activation of protease processes deficiency of necrofactor, prostaglandin (PG E2) and anti-inflammatory cytokines predominates, for example, transforming growth factor-b vaplasminogen-1 inhibitor. Chondritis, osteitis vasinovitis is a common result of inflammation. [1.20].

Intensive pathomorphological changes in osteoarthritis occur in the connective matrix, where their firm and elastic surface dries out. Changes in connective tissue are manifested by disruption of proteoglycan complexes, decreased biosynthetic activity of chondrocytes, resulting in decreased synthesis of major macromolecules - proteoglycans and type II collagen. The synthesis of normal connective tissue types I, III, X is enhanced. The uncle matrix, synthesized by uncle cells, loses chondroitin sulfate and hyaluronic acid. [10.11]. In addition, increased production of nitric oxide (NO) stimulates the process of apoptosis of chondrocytes. Deficiency of the proteoglycan matrix develops, the connective tissue loses glycosaminoglycans. Disorders of uncle products have antigenic properties. The final product enters the synovial fluid and causes synovitis, resulting in disruption of metabolic processes in synoviocytes and decreased formation of endogenous hyaluronate and synovial fluid (Lippielo L.etal., 2000). Disruption and calcification of the joint integrity leads to subchondral bone rupture and fragmentation of wound defects as well as the formation of detritus within the joint [7.12].

Many researchers speculate that “Osteoarthritis and its subsequent development may be a consequence of atheromatous vascular disease of the subchondral bone”. Metabolic diseases play an important role in the development of osteoarthritis. Regardless of body weight index, metabolic syndrome has been found to increase the risk of developing severe gonarthrosis, a direct correlation of adipokines, and the risk of developing osteoarthritis of the hands [5.6].

Thus, the chain of pathological changes is degeneration and decrease in the size of the joint, which leads to local strains, the development of osteosclerosis, subchondral cystic changes and the appearance of osteophytes. Progressive and reactive changes in the bones lead to longitudinal, joint and muscle pathology, resulting in movement stereotypes and biomechanical disorders in the joints. The main clinical manifestations of osteoarthritis are pain, deformity and limited joint movement. The main symptom is pain, intensity and duration of joint pain. this leads to a limitation of functional activity and the full development of the disability [13.14.15].

The main role in the development of pain syndrome in OA is chronic inflammation of the joint structure, manifested by the development of synovitis, entesitis, ostitis, chondritis. Mechanisms of pain in patients with osteoarthritis include pathological processes in all joint structures and intracranial hypertension due to subchondral bone injury. microcracks, venous hyperemia, and the development of stasis (stagnation). Pain syndrome is exacerbated by spasm of nearby muscles and damage to the connective tissue. [9.16].

A combination of different pain syndromes may be observed in patients with OA, so this should be taken into account when recommending syndromic therapy. Nociceptive pain syndrome occurs due to activation of nociceptors as a result of trauma, inflammation, ischemia; neuropathic - associated with damage to the peripheral nervous system or central nervous system; psychogenic is determined by social and psychological factors, regardless of esasomatic, visceral, or neuronal pathologies [15]. The prevalence of neuropathic pain in such patients varies widely. Thus, studies using the Pain Detect questionnaire found neuropathic pain in 5–50% of patients with osteoarthritis [17.19].

Chichasova N.V. it is estimated that approximately 20% of patients with osteoarthritis are not adequately treated with chronic pain syndrome; Chronic pain in OA leads to a decrease in life expectancy in women by an average of 10–12 years, and it has been found that life expectancy in elderly patients with OA is more dependent on pain intensity than in the presence of life-threatening diseases (Chichasova N.V., 2012).

The combination of nociceptive and neurogenic mechanisms leads to a chronic course of pain and the compatibility of the two types of pain. It should be noted that, according to G. Hawker et al., It may also support the chronic state of bipsychological and cognitive factors in depression or catastrophic pain that exacerbate pain in OA [16]. Risk factors for chronic pain include decreased social or physical activity and mood swings [17].

The course of osteoarthritis is chronic and the symptoms are constantly increasing. Joint deformity is exacerbated by the development of osteophytes, capsule fibrosis, disruption of joint and joint surfaces, and muscle malnutrition (Luchixina L.V., 2001). Impairment of joint surface compatibility, development of joint-joint apparatus weakness, and muscle atrophy can lead to joint injury. On palpation of the affected joint, pain is observed, especially along the joint fracture pathway. The reason for this is osteophytes, and in the knee joint is a damaged meniscus (Tsurko V.V. i dr., 1999).

When there is synovitis, it is manifested by pain syndrome, morning numbness, paraarticular edema and superficial skin hyperemia. Synovitis is common during the development of the disease. Deformation and mobility of joints are reduced as a result of the formation of fibrous-sclerotic and hypotrophic processes in the paraarticular tissues, which are altered by the above processes. The development of the disease is influenced by osteophytes, longitudinal muscle contractures, as well as impairment of congruence in the joints in the reduction of range of motion. Thus, the clinical presentation of OA consists of the following syndromes: pain, configuration, and limited joint movement. [1.2.3.]

Clinical signs of osteoarthritis include pain, decreased range of motion, morning numbness, joint instability, crepitation, and swelling. Pain syndrome is one of the most obvious and common symptoms of osteoarthritis, involving heterogeneous conditions and several factors. The clinician should combine orthopedic and rehabilitation measures in the selection of analgesic drugs, especially in the treatment of elderly patients, taking into account the effectiveness of drugs and the degree of development of osteoarthritis. [19.20].The development of pain should be approached taking into account the vital activity of the whole organism and pain should be considered not as a local pathological process but as a general disorder in which connective tissue weakness plays a role in the connective tissue system. The joint is the main target organ of injury in osteoarthritis, and pain in the joint often determines the clinic of the disease [4.6.9].

Pain in OA is characterized by intraarticular, paraarticular, and extraarticular, and is associated with damage to other organs and systems, i.e., pain can be broadly accompanied by diseases of the internal and lateral organs. [7.13].

Intraarticular mechanical rhythm pain is caused by physical exertion and decreases during rest, which is associated with a decrease in the ability of the clavicle and subclavian bone structures to absorb tension. Pain of a mechanical nature is caused by adverse meteorological conditions - high atmospheric pressure, low temperature, increased humidity, affecting the intraarticular baroreceptors, leading to increased pain. The pain appears in the first movements after the resting state and then passes as a result of the activation of the movement. The product of the uncle tissue disorder - detritus settles on the surface of the joint and there is an initial pain as a result of friction. During movement, the detritus in the joint is pushed, and the pain disappears. [7.9].

Paraarticular pain is associated with degenerative changes in paraarticular soft tissues: ischemic, neurotrophic, fibrous-sclerotic disorders lead to inflammatory and dystrophic changes in paraarticular structures along with pain. Along with changes in the stereotype of movements in the arthritic joint, increasing the compensatory load on the paraarticular apparatus, local ligamentitis, myositis, bursitis, tenosynovitis, entesopathy also develop. [4.7.11].

Extraarticular pain is associated with comorbidities, especially in older patients. Knee pain in such patients is significantly affected by paresthesias of the legs as a result of MNS atherosclerotic distirculatory damage, signs of stagnation in the large circulatory system, vascular atherosclerosis of the legs. [19.20].

Pain syndrome is the basis of the OA clinic, which identifies other symptoms. Joint pain in osteoarthritis alters the lymph and blood circulation in the joint, disrupts its trophism, exacerbates the processes of joint degeneration, subchondral bone and uncle destructive, degradation. Increased pain leads to reactive spasm of the muscles that move the joint, which in turn leads to joint contracture. The change in the nature of the pain is due to an increase in synovitis. Pain syndrome in osteoarthritis determines the diagnosis and reflects the quality of movement regimen and therapy. [16.17].

The prevalence of osteoarthritis, the various pathogenetic mechanisms of damage to joint structures, and the subsequent disability, the need for surgical intervention, determine the social importance of developing optimal methods of treatment of patients with OA. The multi-stage pathogenesis of OA is focused on the correction of the main pathogenetic link of this disease, and the development of complex therapy determines the actions. But treating OA remains a daunting task and consists of several areas: training programs, exercise; drug therapy; manual of orthopedic surgery. [4.20].

Thus, osteoarthritis is one of the leading diseases of the musculoskeletal system and is very common, leading to a high degree of disability. It requires a huge amount of money in the treatment of osteoarthritis. Prevention of osteoarthritis in Uzbekistan and the need for new physiotherapeutic treatment of the disease on a scientific basis and its application in medical practice.

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