EARLY DIAGNOSIS OF MONOGONARTHROSIS (literature review)

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ABSTRACT

Background. OsteoArthritis of the Knee Joint (OAKJ) is a progressive incurable disease, which in severe cases necessitates total joint arthroplasty.

Aim. To conduct a systematic analysis of the scientific literature on the prospects and possibilities of early diagnosis of monogonarthrosis.

Materials and Methods. A literature search was conducted in the PubMed and Medline databases for the period 2010–2024, using the following keywords: "osteoarthrosis of the knee joint", "monogonarthrosis" (42 sources).

Results. The mainstream of modern literature on the problem of clinical diagnosis of osteoarthritis, including with monoarticular lesions, includes the results of studies of biomarkers of hyaluronic cartilage degradation both in the case of visualization on magnetic resonance imaging scans and according to the data of biochemical and immunological analyses of blood serum. The importance of radiological signs of mainly early stages of osteoarthritis of the knee joint is also separately studied. There are isolated contradictory professional works on the correlation of clinical and radiological manifestations of gonarthrosis. Reports on the results of a comprehensive clinical and radiological examination of patients with monogonarthrosis are almost not presented in the literature.

Conclusions. It was established on the basis of information and analytical studies of modern scientific literature that osteoarthritis of the knee joint is accompanied by persistent pain, significant limitation of lower limb function, decreased working capacity, which often leads to joint replacement. Diagnosis of osteoarthritis in the early stages is difficult due to the lack of pathognomonic clinical, radiological and laboratory indicators, and in the case of monogonarthrosis with synovitis it is complicated by differentiation with specific arthritises of the knee joint. The above data indicate the feasibility of further research to find opportunities for improving methods for early diagnosis of monogonarthrosis.

Keywords: *osteoarthritis of the knee joint, pathogenesis, clinical and laboratory studies.*

Introduction

OsteoArthritis of the Knee Joint (OAKJ) is a progressive incurable disease, which in severe cases necessitates total joint arthroplasty, which requires significant economic costs [1; 2] and medical and social adaptation [3; 4] and has a significant number of postoperative complications and adverse outcomes [5; 6].

OsteoArthritis (OA) of the knee joint is accompanied by persistent pain, significant limitation of lower limb function, decreased performance, which often leads to joint arthroplasty. Diagnosis of the disease in the early stages is complicated by the lack of pathognomonic clinical, radiological and laboratory indicators, and in the case of monogonarthrosis with synovitis it is complicated by differentiation with specific arthritis of the knee joint. Clinical symptoms and structural changes of the knee joint elements in patients with monogonarthrosis are covered in isolated studies, a significant part of which was published more than 10 years ago.

It should be noted that there is still no generally accepted definition of knee osteoarthritis, which leads to certain discrepancies in determining epidemiology indicators, factors of etiology and pathogenesis, and protocols for examination and tre-

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atment of this category of patients. According to clinical guidelines developed by the Associations of Rheumatologists and Orthopedic Traumatologists of Ukraine, osteoarthritis is a metabolically active, dynamic process that involves all tissues of the joint (cartilage, bone, synovial membrane/capsule, ligaments and muscles). The main pathological changes include localized loss of articular (hyaline) cartilage and alteration of the adjacent bone with the formation of new bone (osteophyte) at the edges of the joint [3; 7; 8]. According to the International Statistical Classification of Diseases and Related Health Problems, 10th revision, osteoarthritis is included in sections M15–M19. In this block, the term "osteoarthritis" is used synonymously with the terms "arthrosis" and "osteoarthrosis".

Osteoarthritis is the most common form of joint disease due to increased life expectancy and body mass index [9–11]. Clinical symptoms of osteoarthritis of the hip and knee joints are observed in 242 million people worldwide [10; 12; 13].

In Ukraine, the average long-term prevalence of osteoarthritis was [1836.23±229.19] per 100,000 population, for the period 2014–2017 – [1770.96± ±32.56], respectively, the upward trend continued during 1993–2013. The average long-term incidence rate of deforming arthrosis during 1993–2014 was [266.15±11.73] cases per 100,000 population, for the period 2014-2017 – [229.84± ±5.60] cases per 100,000 population. After 2015, a slight decrease in incidence and prevalence rates was recorded. The prevalence of deforming arthrosis in 2010 exceeded the incidence by 5.66 times, and in 2017 – by 7.80 times, while the gap between the indicators was constantly increasing, there was an accumulation of chronic disorder in the population [3; 8; 14].

The prevalence and incidence ratio in women and men is 1:69 (p<001) and 1:39 (p<0001), respectively [15; 16]. 62% of people with OA are women. Among people younger than 45 years of age, OA is more common in men; over 45 years of age, OA is more common in women [13; 17]. The high prevalence of OA is reflected in enormous social and personal costs. The total economic burden associated with OA in the United States is estimated at $136.8 billion per year. This figure has more than doubled in the last decade. In the long term, the annual economic cost of OA exceeds the consequences of diseases related to smoking, cancer, and diabetes. The direct medical costs of treating patients with knee OA reach $65 billion per year [18]. OA was the second most expensive disease in US hospitals in 2020 [16]. Approximately 1 million knee and hip replacement surgeries are performed annually due to OA [16].

There is now considerable evidence linking knee osteoarthritis to genetic predisposition, obesity, aging [4; 19; 20], mechanical stress [21], and inflammation [22]. However, the pathogenesis and causes of OA are not fully understood.

Diagnosis of knee osteoarthritis in the early stages of the disease presents significant difficulties, which are associated with the absence of typical clinical manifestations, minor radiological signs and uncharacteristic changes in laboratory parameters of biological fluids of the body [22–24]. The diagnosis of the early stages of gonarthrosis is additionally complicated by the lack of correlation between structural changes and their functional consequences, as well as the visualization of the main radiological (including tomographic) signs inherent in knee osteoarthritis in asymptomatic subjects [2; 9; 25; 26].

The clinical diagnosis of knee OA is based on the clinical guidelines of the Ukrainian Association of Rheumatologists and the Ukrainian Association of Orthopedic Traumatologists, as well as the criteria of the American College of Rheumatologists (ACR) and the European League Against Rheumatism (EULAR) [7; 18; 27].

Available literature either discusses differential diagnosis with monoarthritis [9; 22; 28] or provides results of instrumental diagnosis of early stages of knee osteoarthritis [29; 30]. Therefore, the clinical symptoms of monogonarthrosis are almost not presented in the available literature.

In unilateral gonarthrosis in the initial stages, especially in the case of the development of monosynovitis, diagnosis and differential diagnosis are also difficult, due to the variability of the clinical course and the lack of typical data of radiological and laboratory examination [18; 28; 31].

Aim. To conduct a systematic analysis of the scientific literature on the prospects and possibilities of early diagnosis of monogonoarthrosis.

Materials and Methods.

A literature search was conducted in the PubMed and Medline databases for the period 2010–2024, using the following medical subject headings and keywords: "deforming osteoarthritis of the knee joint", "monogonarthrosis". If necessary, in some cases, literature sources that go beyond the search period were used.

The general search revealed 78 sources on diagnostic and treatment methods in the early stages of monogonarthrosis. The primary exclusion con-

cerned literary sources that reflected the diagnosis and treatment of late stages of osteoarthritis of the knee joint (n=22). The secondary exclusion included literary sources that contained only reference information (abstract, figures, references) (n=14). As a result, only relevant full-text articles in professional journals remained (n=42).

Results and Discussion

Osteoarthritis of the knee joint is a chronic progressive pathological process that involves all elements of the joint: articular cartilage, subchondral plate, underlying subchondral bone, synovial membrane, menisci, ligamentous apparatus, periarticular muscles, etc. [19; 23; 32–34]. Osteoarthritis is a multifactorial disease, among the risk factors for its development are age, gender, obesity [2; 26; 35; 36], previous injuries, hereditary predisposition, repeated loads accompanied by cumulative microdamage to the joint tissues (frequent kneeling, prolonged squatting), discongruence of the articular surfaces, joint instability, etc. [4; 23; 30]. An important factor in the pathogenesis of OA is considered to be a decrease in the stabilizing effect of the periarticular muscles due to hypodynamia [30; 34] or sarcopenia [20; 33], which is accompanied by a decrease in the resistance of the knee joint elements to the action of daily loads. However, OA is not a purely mechanical problem. In the pathogenesis and progression of osteoarthritis, especially in the early phase, inflammatory and metabolic processes play a significant role, in which all tissues of the joint are involved. Among the numerous predictors of osteoarthritis, the main independent risk factor is age [4; 29; 35], however, aging and OA are interrelated, not interdependent [33; 35]. The involutive process is accompanied by the cumulative effect of many years of mechanical loads, which leads to "wear and tear" and pathological destruction of articular cartilage [36; 37]. Based on this, some authors consider osteoarthritis as a natural irreversible disease, and not as a degenerative, potentially curable disease. Not only cartilage, but also subchondral bone, menisci, muscles, as well as adipose and synovial tissues play an important role [17; 35; 20].

In the intact joint, hyaline cartilage is an avascular, aneural tissue with chondrocytes as the only cell type in the cartilage tissue [33; 38; 39]. In addition to chondrocytes, articular cartilage is formed by an extracellular matrix.

Since cell division or death is practically absent in adults, chondrocytes of articular cartilage are considered long-lived cells and, therefore, can accumulate age-related changes over time [36; 37]. As a result, aging profoundly alters chondrocyte function, matrix structure, and function. There is increased apoptosis and decreased cell regeneration [38], at the molecular level, the stiffness of the collagen network increases, and glycation increases, which potentiates dysfunction of articular cartilage and, consequently, the joint [11; 39].

OA develops due to the inability of chondrocytes to maintain homeostasis between the synthesis and degradation of extracellular matrix components [4; 5; 34; 35]. Disruption of homeostasis leads to an increase in water content and a decrease in proteoglycans in the extracellular matrix; weakening of the collagen network due to a decrease in the synthesis of type II collagen and an increased breakdown of pre-existing collagen [6; 36]. On the other hand, there is increased apoptosis of chondrocytes. Initially, compensatory mechanisms, such as increased synthesis of matrix molecules and proliferation of chondrocytes in the deeper layers of the cartilage, are able to maintain the integrity of the articular cartilage, but over time, chondrocyte loss and changes in the extracellular matrix prevail, and osteoarthritic changes develop [18; 34]. Early superficial changes in articular cartilage in osteoarthritis manifest as thinning and fibrillation, which spread distally, forming deep cracks with cartilage delamination. Subsequently, the underlying calcified cartilage and subchondral bone are exposed [23; 28; 37], and the calcified cartilage proliferates, which further increases the mechanical load with subsequent production of catabolic factors [28]. In addition, the increased layer of calcified cartilage advances into the underlying articular cartilage. These changes are due to the penetration of vascular channels, sympathetic and sensory nerves from the bone marrow through the subchondral bone and calcified cartilage to the articular cartilage [39].

Subchondral sclerosis develops with an increase in the volume and thickness of the subchondral plate. These changes are accompanied by thinning of the subchondral trabecular bone in the early stage and its sclerosis in the late stage of OA [28].

Altered osteoblast and osteoclast activity leads to bone remodeling with the formation of subchondral cysts and osteophytes [39]. These changes are usually accompanied by erosion and fissures of the overlying cartilage with exposure of the subchondral bone [33; 34]. The appearance of altered bone marrow areas and exposed subchondral bone correlates with clinical symptoms, especially pain [9; 18].

Synovial fluid plays a crucial role in the trophism of avascular articular cartilage as a source of nutrients, as well as a reservoir for its degradation products [24; 28].

Synovitis is an important feature in patients with OA and is associated with both clinical symptoms and structural progression of the disease. Inflammation in OA causes synovial proliferation and infiltration of T and B lymphocytes and mast cells [29], followed by synovial hypertrophy. The latter is defined as synovial thickening ≤4 mm and a depth of effusion ≤4 mm or ≤4 mm in the suprapatellar fossa [24; 38].

Synovial hypertrophy is associated with radiographic and clinical progression of OA with the development of knee pain and dysfunction. Areas of altered synovial membrane are usually associated with sites of articular cartilage degradation [36; 37]. However, in later stages of osteoarthritis, synovitis becomes diffuse [24].

Patients with radiologically confirmed OA almost always have degenerative damage to the meniscus, leading to loss of load-bearing capacity and cushioning in the corresponding (medial or lateral) area of the joint with the development of local instability. This situation can result in static changes in the tibiofemoral joint with pathological changes in the articular cartilage, subchondral bone, and subchondral bone marrow [18; 35; 39].

The cause of joint pain in osteoarthritis is not fully understood. It is recognized that pain has a complex biopsychosocial model; in osteoarthritis, pain reflects a state of altered pain processing, in which every day stimuli are perceived as painful. These changes occur in response to critical interactions with specific joint, bone, and periarticular factors that may vary from person to person. The resulting sensitization of nociceptive pathways at both the peripheral and central levels depends on many independent and unique factors for the individual. These include individual characteristics of the patient (gender, age, previous medical history, (effectiveness in pain perception and management, pain catastrophizing, environmental factors, lifestyle, and social (social support, working conditions) [9; 11; 23; 26].

The relationship between joint pain and the progression of osteoarthritis may be that pain with concomitant aseptic inflammation and decreased joint mobility produces structural disorders in all intra- and periarticular tissues [19], which will lead to increased functional disorders [2; 9; 40] with a corresponding increase in pain intensity and movement limitation and, thus, contributes to the further development of the pathological process.

One of the important clinical manifestations of gonarthrosis is periarticular muscle dysfunction due to reduced strength of the muscles that stabilize the knee joint and altered proprioception indicators, mainly such muscle sensations as the feeling of force and movement of the knee joint [18; 28; 39].

Pain in the affected joint, together with associated functional limitations, is the main cause of disability in patients with gonarthrosis [9; 15] and is the main reason that prompts this category of patients to undergo arthroplasty [41; 42].

The prevalence of clinical symptoms of knee OA is negatively associated with education level [11; 43]. This situation may be explained by the fact that people with a lower level of education are often engaged in heavy physical labor or have little knowledge about the prevention of knee OA [33]. The same authors [33] did not find a statistically significant difference in the prevalence of clinical manifestations of knee OA between rural and urban areas.

Knee OA negatively affects the quality of life and physical function in both sexes, but in women this disease is more pronounced compared to men [15]. In almost 44% of cases, patients with OA experience activity limitations due to pain. By 2040, the number of patients with OA is expected to grow, with an increase in the frequency of observations with activity limitations by [11–14] %. About 30% of patients with knee OA report significant limitation of kneeling and bending; 20% are unable to walk 3 blocks without pain. Activity limitations associated with clinical manifestations of OA can lead to loss of work [11]. The presence of effusion in the suprapatellar bursa increases the risk of arthralgia [28; 38]. At the same time, other studies using the WOMAC scale have shown that in the presence of joint effusion in only 3.6% of cases (when examining 409 participants and 775 knee joints), pain and stiffness in the joint were reported in 31% and 34% of observations, respectively. Almost 42% of the examined had limited movement in the affected joint, and 52% of cases had at least one of the WOMAC indicators [19]. Such data may indicate that one of the reasons for increased joint pain in the presence of effusion may be purely mechanical, due to the activation of proprioceptors of intra-articular tissues that are in a stretched state and subjected to increased pressure by an excessive amount of synovial fluid.

Structural changes in knee joint tissues in osteoarthritis are not visualized by modern radiological methods at all stages of the clinical course. The initial changes that define the early stage of OA are characterized by a long period of molecular changes in extracellular matrix macromolecules, which are detected by biochemical analyses of biological fluids of the body [23; 24; 27; 28]. The stage of molecular changes is followed by changes in the articular tissue. Early damage to the articular tissue, detected by sensitive imaging methods such as magnetic resonance imaging [29], is also considered to be early stages of OA. Radiographic diagnostics is informative, usually when arthralgia occurs with the development of structural damage in the subchondral plate and underlying subchondral bone. In the terminal stages, with the development of deformation of the joint elements, the diagnosis of osteoarthritis is usually beyond doubt.

OA is a disease that affects joint tissues, and therefore the search for biomarkers of degenerative and inflammatory processes characteristic of osteoarthritis has focused on biopolymer molecules associated mainly with cartilage tissue, bone metabolism and inflammation. These biomarkers are soluble in serum, synovial fluid and urine.

C-reactive protein, type 2 collagen and myeloperoxidase correlate with the severity of erosive OA. In addition, hyaluronic acid is considered a marker of synovitis [17]. Some authors [1] considered the concentration of C-reactive protein and oligomeric cartilage matrix protein as biomarkers of the development of knee OA.

C-Reactive Protein (CRP) is a central component of the innate immune inflammatory response; by binding to the cell surface of dead or dying cells and some bacteria, it leads to activation of the complement system. The synthesis of CRP is mediated by factors secreted by macrophages and adipocytes. CRP also contributes to the stimulation of pro-inflammatory cytokines, which enhances the inflammatory response [26]. Several studies have also attempted to establish a relationship between serum CRP levels and osteoarthritis [23]. A recent meta-analysis of 32 studies found statistically significant differences in serum CRP levels between patients with osteoarthritis and healthy controls. The study also found that CRP was significantly associated with pain and decreased physical function, but not with radiographic OA [28]. In studies that adjusted for body weight, CRP has been shown to be independently associated with osteoarthritis, whereas others have found no such association.

Hunter T.M. et al. [18] reported that serum C-reactive protein and Erythrocyte Sedimentation Rate (ESR) in patients with knee OA significantly correlated with clinical and radiographic features of the disease. ESR was significantly higher in patients with arthralgia and/or patellar bulging compared with patients without these symptoms. High-sensitivity CRP levels were significantly higher in patients with pain, swelling, and patellar bulging compared with patients without swelling, pain, or patellar bulging. ESR and CRP levels were not significantly different in patients without flexion contracture (>5º) and reduced flexion (<120º).

Osteoarthritis with an inflammatory component is a debilitating and very common disease, but it often occurs subclinically. There is increasing evidence that inflammatory and destructive synovial reactions play an important role in OA. In addition, the role of inflammation in OA has been recognized through the association of joint effusion with joint pain [9]. It is still unclear to what extent inflammation is the initiator or the result of the destructive process of the joint [33]. Of particular interest is the emerging evidence that the extent to which immune and wound-healing responses can be activated partly controls an individual's susceptibility to chronic diseases, including OA. Despite the global burden of OA, diagnostic tests and treatments at molecular or early subclinical stages are still not available for clinical use.

Inflammatory and metabolic processes, which involve all tissues of the joint, play a significant role in the pathogenesis and progression of osteoarthritis, especially in the early phase. Macrostructural changes occur in the form of early superficial changes in the articular cartilage, namely thinning and fibrillation, which spread distally, forming deep cracks with cartilage delamination. Subsequently, the underlying calcified cartilage and subchondral bone are exposed [22; 36; 39], subchondral sclerosis develops with an increase in the volume and thickness of the subchondral plate. These changes are accompanied by thinning of the subchondral trabecular bone in the early stage and its sclerosis in the late stage of OA [39].

Subchondral bone marrow changes occur in the form of microtrauma of bone tissue with localized fibrosis, fat necrosis, and local increased bone remodeling with a predominance of bone re-

sorption, leading to microfractures of trabecular bone [20]. These changes are usually accompanied by erosion and fissures of the overlying cartilage with exposure of subchondral bone [34]. The appearance of altered bone marrow areas and exposed subchondral bone correlates with clinical symptoms, especially pain [9; 18]. Most people with knee OA suffer from pain, joint stiffness, and limitations in daily activities. Orthopedic examination may reveal clinical signs such as joint crepitus, smoothing of the contours, or, less commonly, swelling of the affected joint, deformity, or a slight increase in joint temperature. In addition, this category of patients often has impaired indicators such as lower limb muscle strength and knee proprioceptive accuracy, which are considered important factors for knee joint stabilization [9; 19; 31].

Thus, the mainstream of modern literature on the problem of clinical diagnosis of osteoarthritis, including with monoarticular lesions, includes the results of studies of biomarkers of hyaluronic cartilage degradation both in the case of visualization on magnetic resonance imaging scans [28] and according to biochemical and immunological analyses of blood serum [24; 26]. The importance of radiographic signs of mainly early stages of knee osteoarthritis is also studied separately [22; 28]. There are isolated contradictory professional works on the correlation of clinical and radiographic manifestations of gonarthrosis [9]. Reports on the results of a comprehensive clinical and radiological examination of patients with monogonarthrosis are almost absent in the literature.

Conclusions

1. It was established on the basis of information and analytical studies of modern scientific literature that osteoarthritis of the knee joint is accompanied by persistent pain, significant limitation of lower limb function, decreased working capacity, which often leads to joint replacement.

2. Diagnosis of OA in the early stages is difficult due to the lack of pathognomonic clinical, radiological and laboratory indicators, and in the case of monogonarthrosis with synovitis it is complicated by differentiation with specific arthritises of the knee joint. The above data indicate the feasibility of further research to find opportunities for improving methods for early diagnosis of monogonarthrosis.

DECLARATIONS:

Disclosure Statement

The authors have no potential conflicts of interest to disclosure, including specific financial interests, relationships, and/or affiliations relevant to the subject matter or materials included.

Statement of Ethics

The authors have no ethical conflicts to disclosure.

Data Transparency

The data can be requested from the authors.

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All authors give their consent to publication.

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