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EARLY KNEE OSTEOARTHRITIS AND COMORBID CONDITIONS

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Introduction

Osteoarthritis (OA) is one of the most common chronic joint diseases, representing an important medical and social problem due to its progressive course, pain, and reduced quality of life. Osteoarthritis of the knee (gonarthrosis) is the most prevalent form of OA and a frequent cause of loss of work capacity and disability. According to the Global Burden of Disease 2019, around 7% of the world's population (more than 500 million people) suffers from osteoarthritis [1]. Prevalence increases with population aging and the obesity epidemic: OA accounts for up to 65–70% of all rheumatic diseases in the elderly, and radiological signs are found in ~80% of people over 60 years. Meanwhile, incidence before age 50 is low (only 3–5%) but rises sharply in older age groups [2]. In real-world conditions, the figures may be even higher, considering that many patients do not seek help in a timely manner. In Uzbekistan, as in the rest of the world, OA is becoming an increasingly pressing issue. Precise epidemiological data on early knee OA in the region are scarce; however, it is known that musculoskeletal diseases rank third among the overall morbidity of adults [3, 4]. The high prevalence of risk factors – in particular, excess body weight in the population (per WHO, 50% of adults 18–64 in Uzbekistan are overweight and 20% have obesity) – contributes to a significant burden of knee OA in the country. Thus, the problem of early osteoarthritis of the knee joints (early knee OA, EKOA) is extremely urgent, requiring attention to early diagnosis and multidisciplinary patient management.

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Definition and Clinical and Functional Features of Early Knee Osteoarthritis

Early osteoarthritis of the knee is understood as the initial stage of degenerative-dystrophic involvement of the knee joint, when the pathological process is already underway but pronounced radiographic changes are still absent or minimal. The classic definition of OA is a chronically progressive joint disease of various etiologies, accompanied by degradation of articular cartilage, remodeling of subchondral bone, and reactive inflammation of the synovial membrane. In early OA, the damage is pre-radiographic in nature, i.e. there are no obvious osteophytes or joint space narrowing on X-ray; however, clinical symptoms are already present (pain, brief morning stiffness <30 minutes, crepitus, and reduced joint function). Patients often report start-up pain – discomfort at the beginning of movement after rest – and pain on load that disappears at rest. MRI is utilized to detect structural changes at early stages, allowing identification of cartilage thinning, subchondral bone edema and synovitis long before radiographic signs appear. Clinically, early knee OA is usually observed in middle-aged individuals (around 40–50 years), often still of working age. Such patients experience knee pain during prolonged walking, climbing stairs or squatting; in the early stages the pain is episodic and decreases significantly at rest. Localized swelling may occur due to a small effusion in the joint, but without marked inflammation. The early stage corresponds to Kellgren–Lawrence grade I–II or the pre-radiographic period, when X-rays can be normal despite the presence of symptoms. Delineating the concept of “early OA” is important because it is precisely at this stage that there is a window of opportunity to slow disease progression with modern treatments and lifestyle modification.

Functional Features

Even with minimal structural changes, early OA leads to moderate functional impairment: patients report reduced endurance during prolonged walking,

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difficulty with squatting or rising from a chair, and occasionally knee instability. In contrast to late stages, early OA usually lacks pronounced joint deformity (varus or valgus), and range of motion is preserved or only slightly reduced. Nevertheless, quality of life can already suffer – chronic pain and activity limitations affect daily activities and the psychoemotional state.

Comorbid Conditions in Early Knee Osteoarthritis

Comorbidity (the presence of multiple diseases in one patient) is a characteristic feature of osteoarthritis, especially in middle-aged and older patients. In individuals over 50 with OA, typically at least 2–3 concomitant chronic diseases are identified, and cases of “isolated” osteoarthritis are extremely rare. Early knee OA is most often associated with metabolic disorders (obesity, metabolic syndrome, type 2 diabetes, dyslipidemia), cardiovascular diseases (primarily arterial hypertension), as well as mental disorders (depression, anxiety). Below is an analysis of each of these conditions and their relationship with OA (see **Fig. 1**).

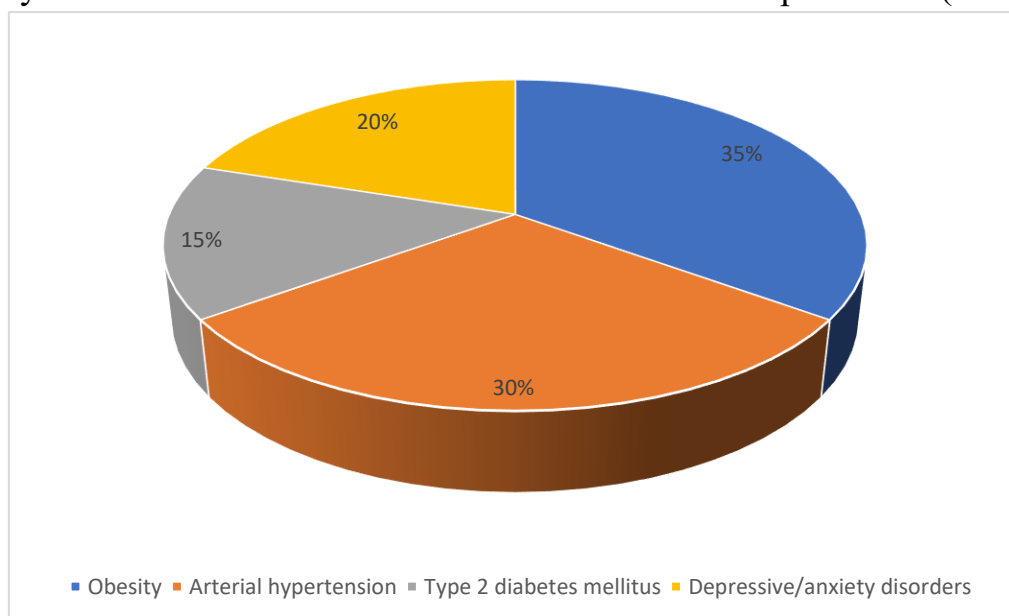


Figure 1: Major comorbid conditions in early knee osteoarthritis

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Obesity and Metabolic Syndrome

Obesity is recognized as one of the key modifiable risk factors for the development and progression of osteoarthritis. Excess body weight increases the load on the knees, contributing to mechanical stress on cartilage and its premature wear. In addition, adipose tissue functions as an endocrine organ, secreting pro-inflammatory adipokines (e.g., leptin, resistin, IL-6, TNF- α) that cause chronic low-grade inflammation and exacerbate damage to joint tissues. Epidemiological data indicate that 76–80% of patients with OA are overweight or obese [2]. In particular, in a sample of relatively young OA patients (mean age ~42 years), excess weight was noted in 78%, and obesity (BMI ≥ 30) in 26% [5, 6]. Obesity often precedes the development of knee OA and significantly increases its risk: for example, a cohort study of over 1.7 million people showed that the risk of knee osteoarthritis in individuals with obesity is more than 3 times higher than in people of normal weight [2]. Not surprisingly, weight loss of even 5–10% can substantially reduce pain and improve joint function in OA patients.

Metabolic syndrome (MetS) – a complex of metabolic disturbances (abdominal obesity, arterial hypertension, dyslipidemia, insulin resistance/hyperglycemia) – is also an important risk factor for OA. In recent years, a “metabolic osteoarthritis” phenotype has been highlighted – OA associated with MetS (so-called MetOA) in which obesity, visceral fat and low-grade systemic inflammation play a decisive role in pathogenesis. The cumulative effect of MetS components is combined with the independent influence of each of them (diabetes, hypertension, hypertriglyceridemia, etc.). According to studies, metabolic syndrome is present in nearly 59% of patients with osteoarthritis (versus ~23% in people without OA) [6, 7]. In other words, more than half of OA patients have MetS, much higher than among people without joint pathology. Thus, obesity and MetS underlie the so-called metabolic phenotype of OA, where joint involvement is accompanied by endocrine-metabolic abnormalities. The presence of MetS worsens the course of OA: more rapid progression of

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radiographic stages of gonarthrosis has been noted in patients with metabolic factors compared to “non-metabolic” counterparts. Patients with OA and MetS also have an elevated risk of cardiovascular mortality compared to patients without MetS [6]. Therefore, combating obesity and correcting components of metabolic syndrome are regarded as part of the strategy for OA prevention and treatment.

Arterial Hypertension and Cardiovascular Diseases

Arterial hypertension (HTN) is one of the most common comorbid conditions in OA, especially in elderly patients. In one study, concomitant cardiovascular diseases (including hypertension and atherosclerosis) were identified in 48% of young patients with OA, and among older individuals this proportion was even higher [8, 9]. OA and cardiovascular pathology often occur in parallel and aggravate each other. This is partly explained by common risk factors – age, obesity, and physical inactivity predispose to both HTN/ischemic heart disease and osteoarthritis. However, there are also specific mechanisms linking these conditions. For example, systemic inflammation in obesity and MetS contributes to endothelial dysfunction and accelerated atherosclerosis, which impairs blood supply to subchondral bone and cartilage. Impaired microcirculation in the subchondral plate can exacerbate joint degeneration – a phenomenon of intersecting pathogenetic pathways in atherosclerosis and OA. Conversely, chronic pain and reduced physical activity in OA adversely affect the course of cardiovascular diseases, creating a vicious circle. Studies have shown that OA patients have a higher risk of cardiac events (myocardial infarctions, strokes) compared to peers without osteoarthritis. Thus, hypertension and OA frequently coexist: according to various data, 40–60% of OA patients suffer from HTN, which requires special physician attention to blood pressure control in this category of patients. Comprehensive management should include cardioprotection, optimization of antihypertensive therapy taking into account

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NSAID use (NSAIDs can raise blood pressure), as well as control of the lipid profile. It should be noted that NSAID treatment for OA in comorbid patients must be done with consideration of cardiovascular risk and gastropathy – gastroprotectors are often required and selective COX-2 inhibitors should be chosen according to guidelines.

Type 2 Diabetes Mellitus

Type 2 diabetes (T2D) is another common comorbid condition in osteoarthritis. T2D is part of the MetS cluster, but by itself it is an independent risk factor for OA. Epidemiological studies show that the prevalence of osteoarthritis among diabetics is higher than in the general population. For instance, some data indicate that the presence of T2D increases the risk of developing severe osteoarthritis by approximately 20–50%, independent of body weight. In cohorts of OA patients, the frequency of T2D ranges from ~8–10% up to 15–20%, exceeding the averages in an age-matched population [9]. T2D affects the joints via several pathways. Chronic hyperglycemia leads to accumulation in cartilage and ligaments of advanced glycation end products (AGEs), which reduce tissue elasticity and stimulate inflammation. At the same time, diabetes is accompanied by oxidative stress and elevated levels of cytokines (IL-6, TNF- α , etc.), which exacerbates cartilage degradation. Insulin resistance and hyperinsulinemia can directly impair the functions of the synovial membrane and chondrocytes, and are also linked to visceral obesity and a systemic proinflammatory background. As a result, patients with diabetes more often exhibit generalized forms of OA and more pronounced pain and stiffness. In addition, T2D limits OA therapy options: corticosteroids must be used cautiously (risk of hyperglycemia), NSAIDs (risk of nephropathy) and even some chondroprotective agents require caution. Thus, timely detection and control of diabetes is an important part of managing an OA patient. In Uzbekistan, according to national registries, the prevalence of T2D is rising

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(especially in the context of obesity), making the issue of “diabetic osteoarthritis” highly relevant [10].

Depressive and Anxiety Disorders

Chronic pain and restricted mobility in osteoarthritis have a significant impact on patients’ mental health. Depression and anxiety disorders are widespread among people with OA, both as a consequence of chronic pain and due to reduced quality of life and social activity. According to meta-analyses, about 27–30% of patients with knee osteoarthritis suffer from clinically significant depression, and roughly the same proportion experience anxiety. In other words, every third patient with gonarthrosis has some symptoms of depression and/or anxiety. These rates are substantially higher than in people without chronic pain conditions. For example, in a U.S. population over 45 years old with arthritis, about 33% report anxiety and/or depression, whereas among those without arthritis – less than 20%.

The pathogenetic mechanisms of psychological disturbances in OA are related to unremitting pain, inflammatory changes, and functional limitations. Chronic pain leads to activation of stress axes (the hypothalamic–pituitary–adrenal system), causing neurohumoral shifts that contribute to depressive symptoms. In addition, constant stiffness and inability to maintain a normal active lifestyle often lead to social isolation, loss of hobbies, and a feeling of helplessness in patients. A vicious cycle ensues: depression amplifies pain perception and reduces patient adherence to rehabilitation, ultimately worsening OA treatment outcomes. It has also been shown that OA patients with depression have a higher risk of escalating to stronger analgesics (up to opioids) and developing chronic pain. Anxiety disorders frequently accompany depression, manifesting as excessive worry about one’s health and fear of pain exacerbation and movement (kinesiophobia). This can impede performing the exercises recommended for OA, thereby adversely affecting the joint.

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Given the high comorbidity of osteoarthritis with depression and anxiety, modern protocols recommend screening the mental state of patients with chronic joint pain [10]. Involving psychologists and psychiatrists in the multidisciplinary treatment team, implementing cognitive-behavioral therapy, and when necessary using antidepressants (for example, duloxetine, which is indicated for chronic pain management in OA) can improve not only mood but also somatic outcomes. It is also important to explain to patients with early OA the need to continue physical activity—overcoming the fear of pain with adequate analgesia—since this improves functional outcomes and reduces the severity of depression.

Other Comorbid Conditions

In addition to the above, patients with osteoarthritis often have other chronic diseases: osteoporosis, chronic lung diseases (COPD), kidney disease, non-alcoholic fatty liver disease (NAFLD), gout, etc. For example, one study found that 17.5% of OA patients had concomitant diseases of other organ systems: COPD in 3.8%, chronic pyelonephritis in 2.5%, chronic hepatitis in 2.5%. Osteoarthritis is often one component of overall multimorbidity that includes cardiovascular and metabolic disorders, as noted above. The combination of OA with osteoporosis deserves special attention: prolonged physical inactivity in joint disease leads to loss of bone mass; furthermore, some factors (age, postmenopause, vitamin D deficiency) are common to both conditions. According to some data, osteoporosis is detected in 20% of patients 60–74 years old with osteoarthritis and in 38% of those over 75 years [11, 12]. Thus, the clinical picture of a patient with OA often comprises numerous components requiring an integrated approach.

Pathogenetic Mechanisms Linking OA and Comorbid Conditions

Mechanical load and cartilage damage: Excess body weight in obesity increases the load on the knee joints by about 4 N for each extra kilogram of

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weight. This leads to microtrauma of cartilage and subchondral bone, triggering a degenerative cascade. Micro-injuries induce chondrocytes to release metalloproteinases and pro-inflammatory mediators, initiating chronic “low-level” inflammation in the joint. Repeated mechanical stress is considered a key mechanism explaining the association of OA with obesity and certain occupations (prolonged standing, heavy lifting, working in a kneeling position, etc.).

Adipokines and systemic inflammation: Adipose tissue in obesity and MetS secretes cytokines (TNF- α , IL-1 β , IL-6) and adipokines (leptin, visfatin, resistin) that act on the joint both locally and systemically. Elevated leptin levels are found in the synovial fluid of OA patients and correlate with the degree of inflammation. Leptin and other adipokines enhance degradation of the cartilage matrix by stimulating the synthesis of metalloproteinases and nitric oxide, and also contribute to oxidative stress. Systemic subclinical inflammation (elevated CRP, IL-6) is characteristic of patients with obesity, MetS, and T2D, and is considered a common background that damages blood vessels (leading to atherosclerosis) and joints (leading to OA). Accordingly, inflammatory pathways underlie the comorbidity of “OA + obesity + HTN + T2D”. This is supported by studies showing that OA patients have significantly higher CRP and IL-6 levels compared to healthy controls, even after adjusting for body weight.

Insulin resistance and hyperglycemia: In T2D, chronically high blood glucose causes non-enzymatic glycation of cartilage matrix proteins and collagen, leading to the formation of AGEs. These products accumulate in cartilage, making it more rigid and brittle, and they interact with RAGE receptors on cells, amplifying inflammatory responses. The synovial membrane is also adversely affected by hyperglycemia – hyaluronic acid production is disrupted and oxidative stress is increased. Insulin resistance, the core of MetS, is associated with a local “resistance” of joint tissues to the anabolic effects of insulin, which impairs

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regenerative processes in cartilage. Taken together, these mechanisms explain why T2D is associated with more severe arthritis: diabetic patients more often require joint replacement and respond worse to analgesic therapy, according to a number of clinical observations.

Hormonal and metabolic factors: Hypoestrogenism in postmenopausal women is a risk factor for both OA and osteoporosis; vitamin D deficiency disrupts bone-cartilage homeostasis; hyperuricemia (in gout or MetS) may contribute to joint inflammation. These factors frequently overlap in the comorbid OA picture. For example, the combination of OA with gout occurs more often than would be expected by chance, suggesting common metabolic pathways (obesity, diet, inflammation).

Biomechanical changes with comorbidity: The presence of other diseases can indirectly affect the joint by altering physical activity and biomechanics. For instance, in severe coronary disease a patient is forced to walk less, which on one hand reduces load on the joint (the “obesity paradox” — obese patients with heart disease may less often subject their joints to high loads), but on the other hand physical inactivity leads to sarcopenia, quadriceps weakness and knee instability, accelerating the progression of gonarthrosis. In another scenario, with depression a patient becomes less active, eats improperly, and gains weight — all of which worsens OA. Thus, the complex interplay between diseases also involves the patient’s lifestyle.

In general, the pathogenesis of osteoarthritis in the presence of comorbidity is multifactorial. There are common pathways (inflammatory, metabolic, vascular) through which different diseases mutually exacerbate each other. Modern research confirms that OA is not merely a localized joint pathology, but a systemic disorder of the musculoskeletal system against the background of overall multimorbidity. This dictates the need for a holistic treatment approach directed

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not only at the joint symptoms, but also at correcting accompanying conditions [12].

Diagnosis of Early Knee Osteoarthritis and Associated Diseases

Diagnosis of early knee osteoarthritis is based primarily on clinical criteria, since radiographic changes may be absent in the early stages. In practice, the criteria of the American College of Rheumatology (ACR) adapted for the knee joint are used: the presence of knee pain on most days of the month **plus** at least three of the following (age >38–50 years, morning stiffness <30 min, crepitus, bony enlargements at the joint margins, no marked inflammation, etc.). The sensitivity and specificity of this combination of criteria reach about 90%. The national Clinical Protocol for Osteoarthritis (Uzbekistan, 2024) likewise states that the diagnosis is made on the basis of the 1991 ACR criteria [10]. Importantly, an early form of OA should be suspected as soon as persistent knee pain is present in a middle-aged patient with risk factors, even if radiographs are normal.

It should be noted that Uzbekistan has developed and implemented national clinical guidelines for the diagnosis and treatment of osteoarthritis, taking into account local epidemiology and healthcare resources. The 2024 protocol approved by the Ministry of Health is based on advanced international recommendations (EULAR 2018, ACR 2019, ESCEO 2019, AAOS 2021) adapted to the conditions of the republic. It emphasizes the need for early detection of OA at the primary care (pre-hospital) stage and timely referral of patients to rheumatologists and orthopedists to slow disease progression. The national standard also focuses on screening for comorbidity: in every case of OA, the physician should conduct comprehensive screening for non-communicable diseases – from hypertension to depression. This proactive approach will improve outcomes, since treating accompanying conditions (weight loss, blood pressure control, blood sugar control, treating depression) has been shown to alleviate OA symptoms and improve patients' quality of life.

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Osteoporosis: If the patient is over 50 (especially a postmenopausal woman), it is advisable to perform bone densitometry or at least a FRAX screening for osteoporotic fracture risk. This is important because when OA and osteoporosis are combined, the approach to exercise and treatment must be modified (emphasis on resistance exercises to strengthen bones, and possibly the use of calcium and vitamin D).

Depression and anxiety: Use screening questionnaires – the Hospital Anxiety and Depression Scale (HADS) or PHQ-9 for depression. If signs of a depressive disorder are detected, referral to a specialist is necessary. According to studies, about 30% of OA patients require psychological or psychiatric care [11].

Visualization of Data: Prevalence and Age Trends

The epidemiology of osteoarthritis shows a clear dependence on age. Below is a chart illustrating the increase in knee OA prevalence with age (Fig. 2).

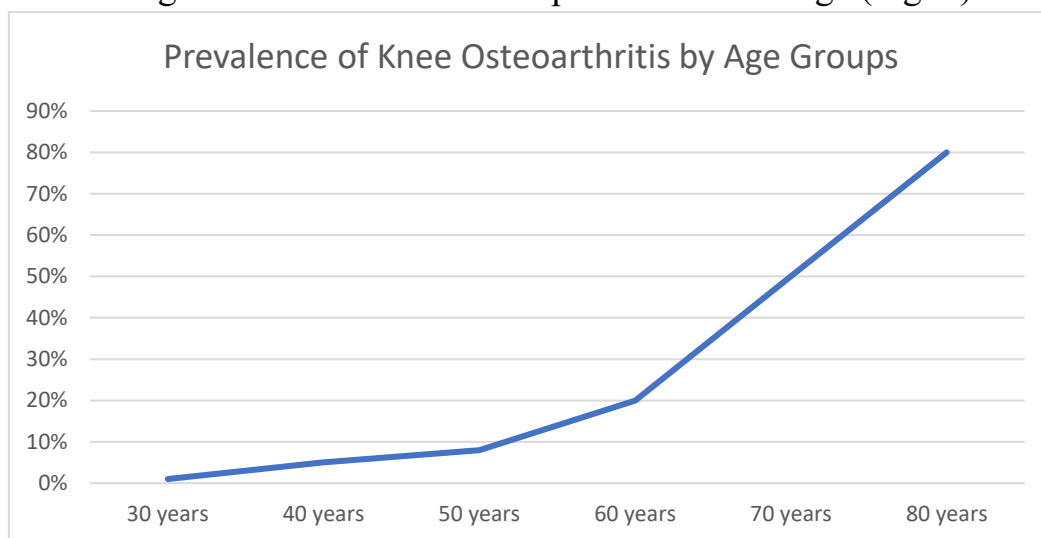


Figure 2: Increasing prevalence of knee osteoarthritis with age. By age 60, clinical and radiographic signs of OA are observed in the majority of patients, whereas in young age the disease is rare.

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As seen in Fig. 2, prevalence rises sharply after 50–60 years: if at age 30–40 knee OA is recorded in only a few percent of the population, then after 60 it is present in more than half, and by 70–80 years radiographic signs of knee OA are noted in 70–80% of people. At the same time, the “rejuvenation” of the cohort is notable: osteoarthritis is increasingly being identified in patients under 50, largely due to obesity and a sedentary lifestyle in the population. The same trend is observed in Uzbekistan: over the past decade there has been an increase in clinic visits for joint pain among individuals 35–45 years old, whereas previously the majority of patients were retirees. Thus, the concept of “early OA” reflects the real situation: the onset of the degenerative process is increasingly occurring at a relatively young age, which requires shifting the focus to prevention and early treatment.

Aside from age, the structure of comorbidity is also of interest, as visualized in Fig. 1. From this we see that the greatest “contribution” to comorbid pathology in OA is made by obesity and hypertension – together accounting for roughly half of all comorbidity cases. These are followed by mental disorders (~20%) and diabetes (~15%). Metabolic syndrome, effectively combining obesity, HTN, dyslipidemia and prediabetes, is present in approximately 3 out of 5 patients with OA, although it is not shown as a separate category in the diagram due to overlap of components. This visual evidence underscores that treating a patient with osteoarthritis is impossible without taking into account and managing the accompanying diseases.

Treatment and Prevention with Consideration of Comorbidity (Discussion)

Management of patients with early knee osteoarthritis should be carried out within a multidisciplinary framework that accounts for comorbid conditions. Numerous studies have shown that treating only the joint (for example, with painkillers) does not provide optimal results if risk factors and concomitant pathology are not addressed. Therefore, the modern strategy is a coordinated

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effort by a rheumatologist (or orthopedist), internist, cardiologist, endocrinologist, nutritionist, psychotherapist and physiotherapist, all working together on the patient's condition.

Control of risk factors and lifestyle modification. The foundation of early OA therapy is non-pharmacological measures: weight reduction, graded physical exercise, and exercises to strengthen the thigh muscles (particularly the quadriceps, whose weakness contributes to knee instability progression). Low-intensity aerobic activity (walking, Nordic walking, swimming) for at least 150 min/week is recommended, along with stretching and range-of-motion exercises. In cases of obesity, a goal of gradual weight loss of 5–10% over 6–12 months is set – it has been proven that this leads to pain reduction and improved joint function. A calorie-restricted diet rich in omega-3 fatty acids, antioxidants (vitamins C, E) and adequate calcium and vitamin D is beneficial for the joints and for preventing osteoporosis, which often accompanies OA. Educating the patient in self-management principles is a crucial part of the program: patients should understand the nature of their disease, know how to dose physical activity, and be aware of pain-relief techniques (for example, applying ice locally after exercise).

Pharmacological treatment of early OA is aimed at symptom relief and chondroprotection. The main drug classes are nonsteroidal anti-inflammatory drugs (NSAIDs) for pain control (taking comorbidity into account: in patients with T2D and HTN it is preferable to use topical NSAIDs or selective COX-2 inhibitors at the minimum effective dose to avoid systemic side effects). Many comorbid patients have contraindications to NSAIDs (e.g., peptic ulcer disease or severe hypertension), in which case other modalities are employed: topical ointments, physiotherapy (ultrasound, laser therapy), and if necessary intra-articular injections of hyaluronic acid or corticosteroids. The latter are effective in the presence of synovitis, but should be used with caution in diabetics (corticosteroids can temporarily raise blood glucose). It is important to account

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for polypharmacy: comorbid patients often take numerous medications (for blood pressure, blood sugar, heart conditions), so the risk of drug interactions is high. For example, NSAIDs can reduce the efficacy of certain antihypertensives; some antidepressants can increase bleeding risk when combined with NSAIDs, and so on. Accordingly, the treatment regimen must be personalized with the involvement of all relevant specialists.

Promising therapies for comorbid osteoarthritis are those that influence both the joint and metabolic processes. For example, studies are underway on the use of statins (lipid-lowering agents) in patients with OA and MetS, since statins have an anti-inflammatory effect in the joint as well. The potential of metformin (a diabetes drug) to slow cartilage degeneration is also being investigated, via activation of AMPK. In cases of pronounced depression and chronic pain, duloxetine is used – an antidepressant approved for the treatment of chronic (neuropathic) pain in OA. It improves mood and modestly reduces pain, which is beneficial for patients with comorbid conditions. Thus, treatment of EKOA should be comprehensive and take into account the entire spectrum of the patient's diseases – only then can success be achieved.

Conclusions

Early osteoarthritis of the knee is a stage of the disease at which effective intervention is still possible to prevent the severe disabling form of gonarthrosis. However, the clinical picture of early OA is not confined to the joint: it is almost always complicated by comorbid conditions – obesity, metabolic syndrome, hypertension, type 2 diabetes, depressive-anxiety disorders, and others. These comorbidities share common pathogenetic roots with OA (chronic inflammation, mechanical damage, metabolic imbalances), as a result of which osteoarthritis and its accompanying diseases reinforce each other.

A multidisciplinary approach is necessary for successful management of such patients. Treatment of the joint (with non-pharmacological and pharmacological

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methods) should be undertaken in parallel with correction of all identified risk factors and comorbid diseases. The optimal model is patient management by a team of specialists: rheumatologist (or orthopedist), endocrinologist, cardiologist, nutritionist, physiotherapist, and psychotherapist. Only by eliminating or mitigating the impact of comorbid conditions – reducing weight, controlling blood pressure and blood glucose, stabilizing the psychoemotional state – can one hope to slow the progression of OA and improve the patient's quality of life.

For public health practice, it is important to implement screening programs to detect early OA (for example, examining people with risk factors such as obesity or heavy physical labor for joint pain), as well as early identification of risk factors in those who already have OA. National protocols of the Republic of Uzbekistan emphasize that osteoarthritis is “not an inevitable consequence of aging” to be accepted, but a disease that must and can be treated from the earliest stages. The implementation of these principles – early diagnosis, multidisciplinary treatment, patient education – can significantly reduce the burden of osteoarthritis at both global and regional levels. In conclusion, one can say that the fight against early knee osteoarthritis is at the same time a fight against the epidemics of obesity, diabetes, physical inactivity, and other diseases of civilization. Only a synergy of efforts by various specialists and the patient themselves will lead to success in this multifaceted clinical challenge.

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