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Osteoarthritis and geriatric syndromes: features of the relationship and management opportunities (literature review)

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Osteoarthritis (OA) is one of the leading age-associated musculoskeletal disorders, the prevalence of which is increasing due to population aging. The aim of this study is to analyze current literature data regarding the relation and management possibilities of OA and common geriatric syndromes. Methods. A systematic literature review was conducted using analytical methods across scientific databases such as PubMed, Web of Science, Scopus, and Google Scholar for the period 2019–2024. The search was performed using the keywords: "osteoarthritis," "sarcopenia," "sarcopenic obesity," "dysmobility," "malnutrition," and "undernutrition." Results. Typical geriatric syndromes (sarcopenia, sarcopenic obesity, dysmobility syndrome, and malnutrition) are the common phenomenons among OA patients. These conditions share common pathophysiological mechanisms that mutually aggravate each other's course. The analysis of current literature revealed a lack of comprehensive studies on the combination of OA with geriatric syndromes, especially in the Ukrainian scientific space. This article provides an overview and analysis of current scientific data regarding prevalence, risk factors, pathophysiological mechanisms, diagnostic features, clinical manifestations, as well as potential approaches to treatment, prevention, and rehabilitation of patients with OA in combination with the most common geriatric syndromes. Conclusions. The literature analysis demonstrated bidirectional interaction mechanisms between OA and other geriatric syndromes, highlighting the importance of developing effective strategies for early detection, prevention, and management of such patients within a multidisciplinary approach.

Остеоартрит (ОА) є одним із провідних вік-асоційованих захворювань опорно-рухової системи, поширеність якого зростає на фоні старіння населення. Мета. Проаналізувати сучасні літературні дані стосовно взаємозв'язку та можливостей менеджменту ОА та загальновідомих геріатричних синдромів. Методи. Систематичний огляд літературних джерел проведено з використанням інформаційного аналізу наукометричних баз даних PubMed, Web of Science, Scopus та Google Scholar за період 2019–2024 р. Пошук виконували за ключовими словами «остеоартрит», «саркопенія», «саркопенічне ожиріння», «дисмобільність», «недостатнє харчування», «мальнутриція». Результати. Типові геріатричні синдроми (саркопенія, саркопенічне ожиріння, синдром дисмобільності та мальнутриції) є поширеним явищем у хворих з ОА. Вони мають спільні патофізіологічні механізми, які взаємно посилюють перебіг одне одного. Аналіз сучасної літератури виявив недостатню кількість робіт щодо комплексного вивчення поєднання ОА з геріатричними синдромами, особливо в україномовному науковому просторі. У статті наведено огляд та аналіз актуальних наукових даних щодо поширеності, факторів ризику, патогенетичних механізмів виникнення, особливостей діагностики, клінічних проявів, а також потенційних підходів до лікування, профілактики та реабілітації хворих з ОА у поєднанні з розповсюдженими геріатричними синдромами. Висновки. Проведений аналіз літератури продемонстрував двонаправлені механізми взаємодії між ОА й геріатричними синдромами та підкреслив важливість розробки ефективних стратегій раннього виявлення, профілактики та ведення таких пацієнтів у межах мультидисциплінарного підходу. Ключові слова. Остеоартрит, саркопенія, саркопенічне ожиріння, синдром дисмобільності, синдром мальнутриції.

Keywords. Osteoarthritis, sarcopenia, sarcopenic obesity, dysmobility syndrome, malnutrition syndrome

Introduction

Osteoarthritis (OA) is one of the most common age-associated musculoskeletal diseases (MSD), the prevalence of which is constantly increasing, taking into account current trends in global population aging [1]. It affects every third person aged 65 years and older, with a higher incidence among women than men [2]. In European countries, the prevalence of symptomatic OA of the knee joints is from 5.4 to 29.8 %, OA of the hip joints from 0.9 to 9.7 % [3, 4].

Comorbidity is a widespread phenomenon in patients with OA, from 59 to 87 % of patients have at least one concomitant chronic disease, in particular, on average, a patient with OA has 2.6 concomitant diseases of moderate or severe compensation, and 31 % have 5 or more chronic diseases [2].

In older people, OA is often associated with a number of geriatric diseases and conditions, including sarcopenia (SP), sarcopenic obesity (SPO), dysmobility syndrome (DS), and malnutrition. They mutually worsen the overall health of a person, creating a vicious circle: OA causes a decrease or restriction of mobility, which contributes to the development of SP, SPO, and other syndromes, and they, in turn, aggravate OA symptoms, negatively affecting all aspects of daily life. For effective management of such patients, it is necessary to develop diagnostic algorithms that will allow detecting the combination of OA and concomitant geriatric conditions at an early stage. Understanding the relationships between them is key to developing a comprehensive approach to their diagnosis, treatment, and prevention. Unfortunately, the number of works on this issue, including in the Ukrainian-language literature, is limited.

Purpose: to analyze modern literary sources on the relationship and management options of osteoarthritis and common geriatric syndromes.

Material and Methods

An information analysis of literary data (meta-analyses, systematic reviews, experimental and clinical, including randomized controlled and cohort studies) was conducted using the scientific-metric databases PubMed, Web of Science, Scopus and Google Scholar for the period 2019–2024. Key works on the search topic published earlier were not excluded during the review. The search was conducted using the keywords “osteoarthritis”, “sarcopenia”, “sarcopenic obesity”, “dysmobility”, “inadequate nutrition”, “malnutrition”.

Osteoarthritis and sarcopenia

SP is a progressive generalized skeletal muscle disease associated with an increased risk of falls, fractures, impaired motor activity and a high mortality rate. SP leads to a decrease in muscle mass, in particular lean skeletal muscle mass, and muscle function (physical performance) [5].

Recently, the relationship between SP and OA has been actively studied, since both diseases have common development mechanisms and risk factors associated with aging. The frequency of SP among patients with OA according to various studies varies from 4.5 to 45.2 % and, accordingly, is twice as high compared to individuals without OA, and the presence of SP increases the risk of developing OA by 91 % [6–9].

The authors emphasize that SP develops more often in elderly people, especially among those with a history of falls or low physical activity. However, the question remains whether these are two separate and independent diseases that occur in individuals of the same age group or whether, on the contrary, they contribute to and potentiate the progression of each other [9, 12–16].

The main risk factors for the development of OA in individuals with SP are, in particular, age, reduced estrogen levels, and altered body mass index (BMI). On the other hand, decreased muscle strength is a major characteristic of SP and is considered one of the key risk factors for the development of OA. Muscle weakness reduces the stability of the knee joint and can accelerate the degeneration of articular cartilage. Studies in animal models have confirmed this relationship: experimental atrophy of muscles around the knee joint caused degenerative changes in articular cartilage [11, 17].

Irisin protein (IP) may play an important role in the development of SP and OA. It is a sensitive marker of muscle weakness and atrophy and may help predict the development of SP [13]. In 2016, it was first demonstrated that IP levels in synovial fluid and serum of patients with gonarthrosis negatively correlated with disease severity according to the radiological Kellgren-Lawrence criteria, indicating a possible relationship between IP concentration and OA progression. Further experimental studies confirmed that IP expression is reduced in the cartilage tissue of mice with knee OA after anterior cruciate ligament transection, while intra-articular injection of IP in mice resulted in a slowdown of destructive changes in the knee joint [15].

In addition, there are observations that demonstrate a link between IP and the development of SP. A Korean study of 715 individuals aged 18–90 years found that low IP levels were correlated with the presence of SP (males $r = 0.28$; females $r = 0.32$) and with

hand muscle strength measured by dynamometry (males r = 0.22; females r = 0.31) ($p < 0.01$ for all measures). The mean circulating IP level was significantly lower in the SP group compared with the control group, with no changes in body composition. In logistic regression models, the association between serum IP concentration and the presence of SP remained statistically significant even after adjustment for age, sex, and fat mass (FM) (odds ratio (OR) = 0.20; 95 % confidence interval (CI): 0.07–0.60; $p < 0.01$). The predictive values of IP levels for the diagnosis of SP were < 1.0 μg/mL for men and < 1.16 μg/mL for women (area under the operating characteristic curve, AUC) of 0.87 (95 % CI: 0.77–0.99; $p < 0.01$) for men and 0.68 (95 % CI: 0.55–0.81; $p < 0.01$) for women [16].

The mechanisms of action of IP in knee OA are realized through stimulation of chondrogenesis and inhibition of pro-inflammatory signaling pathways. In particular, IP promotes increased expression of cartilage anabolic genes (COL2A1, Aggrecan and SOX9), while simultaneously inhibiting the activity of JNK, p38 MAPK, NF-κB and AKT pathways, which reduces the production of matrix metalloproteinases (MMP-1, MMP-13), pro-inflammatory cytokines (IL-1, IL-6) and iNOS. In addition, IP is able to enhance chondrogenic differentiation by activating the Rap1/PI3K/AKT signaling cascade through the miR-125b-5p microRNA, which indicates its promising application in regenerative therapy of OA [17].

To date, other factors have been studied that significantly increase the risk of OA in patients with SP. In particular, these are smoking (OR = 1.54; 95 % CI: 1.21–1.95; $p < 0.05$), advanced age (> 75 years, OR = 10.6; 95 % CI: 3.7–30.2; $p < 0.05$), low income (OR = 5.4; 95 % CI: 1.4–21.4; $p < 0.05$), reduced Barthel index (< 90 points, OR = 11.0; 95 % CI: 3.5–34.5; $p < 0.05$), falls during the last 12 months (OR = 3.1; 95 % CI: 1.4–6.6; $p < 0.05$), malnutrition (OR = 3.5; 95 % CI: 1.3–9.3; $p < 0.05$), history of acute cerebrovascular accident (OR = 7.5; 95 % CI: 1.3–41.4; $p < 0.05$), vitamin D deficiency (OR = 9.4; 95 % CI: 1.1–82.5; $p < 0.05$) and presence of malignant neoplasms (OR = 4.8; 95 % CI: 1.2–19.5; $p < 0.05$). In contrast, higher education was associated with a reduced risk of SP (OR = 0.85; 95 % CI: 0.74–0.98; $p < 0.05$). Therefore, the analysis of these factors is important for the development of preventive strategies and timely diagnosis of OA in patients with SP [16, 21].

A causal relationship between SP and OA was confirmed by the Mendelian randomization (MR) method. It was proven that a decrease in appendicular fat-free mass (AFM) is associated with an increased risk of developing knee OA (OR = 1.32; 95 % CI: 1.22–1.43; p = 2.07×10–12) and hip OA (OR = 1.18; 95 % CI: 1.10–1.27; p = 2.05×10–6) in patients with SP. In a two-stage MR analysis, it was found that obesity (OB) plays an indirect role in reducing AFM and the development of knee OA (the proportion of the indirect effect is 5.9 %). It has also been found that walking pace is inversely correlated with the risk of developing OA, confirming the role of muscle mass and strength in preventing degenerative changes in the joints [12].

Another two-sample MR analysis confirmed that SP may have a causal effect on the development of OA through changes in muscle structure rather than through decreased muscle strength. However, the evidence for an effect of OA on the development of SP was not statistically significant (OR = 1.08; 95 % CI: 0.89–1.31; p = 0.46) [18]. At the same time, the results of another similar MR analysis established not only a reliable causal effect of SP on the development of OA (AM: HR = 1.10; 95 % CI: 1.05–1.16; handgrip dynamometry: HR = 0.82; 95 % CI: 0.71–0.95; walking speed: HR = 0.34; 95 % CI: 0.20–0.56), but also the effect of OA on the development of SP (AM β = –0.26; 95 % CI: –0.37–0.15; handgrip dynamometry β = –0.06; 95 % CI: –0.10–0.02; walking speed: β = –0.10; 95 % CI: –0.15–0.06) ($p < 0.05$ for all factors) [20].

A study based on data from the US National Health and Nutrition Examination Survey (NHANES) examined the causal relationship between OA and OA. Subgroup analysis showed that the positive association between OA and OA persisted among men and women (46–59 years) with normal BMI (18.5–24.9 kg/m²). MRI findings revealed a causal relationship between right and left hand grip strength and knee OA (OR = 0.67; 95% CI: 0.51–0.88; $p < 0.01$) and (OR = 0.79; 95% CI: 0.61–0.92; p = 0.04) [21].

In a retrospective study of patients undergoing elective knee osteotomy or total knee arthroplasty (TKA), the association between muscle mass (MM) loss and progression of knee OA was investigated. The authors found that the serum creatinine to cystatin C ratio, also known as the CP index, was significantly correlated with skeletal muscle mass. In addition, a positive association was found between the CP index and the functional activity scores of the Knee Society Score (Knee Society Score β = 0.37; p = 0.02), Knee injury and Osteoarthritis Outcome Score (KOOS), Activity of Daily Living Index (β = 0.42; $p < 0.01$), and Oxford Knee Score

(Oxford Knee Score, β = 0.42; p = 0.01). This observation emphasizes the role of reduced body MM for the functional activity of patients with knee OA, and the authors suggest using the above-mentioned index as a marker for determining MM loss in patients with gonarthrosis [10].

Some studies emphasize the role of chronic low-intensity inflammation as a common pathophysiological mechanism of the development of SP and OA. Increased levels of pro-inflammatory cytokines lead to an imbalance between protein synthesis and degradation in muscles and cartilage, which ultimately causes MM loss and cartilage destruction [15, 16].

In addition, a relationship has been established between a decrease in muscle strength and pain in the presence of OA. In a population-based cohort study involving 947 subjects (10.7 years of follow-up), it was found that higher muscle strength is associated with a decrease in pain intensity. Three different pain trajectories were identified: "minimal pain" (53 %), "mild pain" (34 %), and "moderate pain" (13 %). Higher lower limb and knee extensor muscle strength, as well as better muscle quality as measured by two-photon X-ray absorptiometry (DXA), were associated with a reduced risk of pain in the "mild pain" (relative risk (RR) = 0.95; 95 % CI: 0.92–0.98) and "moderate pain" (RR = 0.92; 95 % CI: 0.87–0.96; p < 0.01 for both groups) groups [22].

In another study examining body composition in patients with bilateral knee OA, skeletal muscle mass index (SMMI) was negatively correlated with the functional activity subscale of the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC-F) questionnaire (β = –0.16; 95 % CI: –0.66–0.03), and daily moderate-to-low intensity physical activity was negatively associated with bilateral knee pain (β = –0.80; 95 % CI: –0.10–0.01) [23]. Patients with OA had more severe knee pain and worse functional activity (p < 0.05). In the general group, FM was positively correlated with bilateral knee pain (β = 1.21; 95 % CI: 0.03–0.15), the pain subscale of the questionnaire (β = 0.25; 95 % CI: 0.23–1.22), and WOMAC functional activity (β = 0.28; 95 % CI: 0.35–1.29), as well as the 5-time sit-to-stand test (β = 0.19; 95 % CI: 0.03–0.42) [23]. Hip and knee OA are important predictors of high risk of falls [24–26]. The question arises whether the risk of falls increases in patients with SP and knee OA. The study by Iijima H. et al. [25] assessed the risk of falls in four groups: patients with isolated SP, isolated knee OA, a combination of SP and knee OA, and a control group (no SP or OA) in 291 individuals aged 60–90 years (78.7 % women). Patients with a combination of SP and knee OA had a 4.17-fold higher chance (95 % CI: 0.84–20.6) of recurrent falls (≥ 2 falls) than controls, while no statistically significant differences in the frequency of recurrent falls were found between the groups of patients with isolated SP and isolated knee OA [25]. According to existing studies, the presence of knee OA also affects balance and gait speed, with the results of the 6-minute walk test, sit-stand and stand-and-go tests showing lower performance compared to individuals without OA [24, 27, 28].

SP and OA are often found in patients with other chronic diseases, in particular, type 2 diabetes mellitus (T2DM). The study by S. Basat et al. [29] first revealed the relationship between SP and OA in elderly individuals with concomitant T2DM. Examination of patients included assessment of muscle function, body composition, inflammatory markers and the degree of joint damage. The group of patients with OA and T2DM demonstrated a significant decrease in MM, strength and functional capabilities compared to patients with T2DM without OA disorder. In particular, they observed a significant decrease in albumin and hemoglobin levels, as well as a decrease in shoulder and lower leg circumference.

Correlation analysis confirmed a negative relationship between MM and the severity of OA in patients with type 2 diabetes (r = –0.41; p < 0.05).

Unfortunately, therapeutic options for both OA and SP are limited. A randomized, double-blind, placebo-controlled trial in 124 subjects aged 50–70 years (38.7 % men) with knee OA and SP examined the effects of a high-protein plant-based dietary supplement (32 g) twice daily for 12 weeks. The results demonstrated that plant-based protein supplementation may improve symptoms of stage 1–2 knee OA in subjects with concomitant SP. After 12 weeks, patients receiving the aforementioned supplement showed a significant increase in MM, strength and physical performance, namely SMMI by 10 % (0.66 kg/m^2; 95 % CI: 0.45–0.86; p < 0.0001), muscle strength by handgrip dynamometry by 13.2% (2.83 kg; 95 % CI: 2.13–3.53; p < 0.0001) and physical performance by the Short Physical Performance Battery (SPPB) by 13.2 % (1.03; 95 % CI: 0.69–1.38, p < 0.0001). In addition, there was a 12 % improvement in the overall WOMAC score (3.95 points; 95 % CI: –5.02–2.89, p < 0.0001), as well as in the subscales of pain (by 20.6 %), stiffness (by 21.3 %), and daily activities (by 7.4 %). Quality of life in patients with OA and SP improved by 7.9 % as measured by the World Health Organization Quality of Life Brief Scale [30].

Nowadays, the search for potentially modifiable risk factors that may worsen symptoms or accelerate the progression of these two diseases is important. For example, the knowledge that muscle weakness is associated with knee pain and that exercises aimed at strengthening the quadriceps muscle improve the patient's overall condition by reducing pain intensity is valuable and useful [31].

Summarizing the results of current studies on the relationship between SP and OA, it should be noted that these two conditions share common risk factors and mechanisms for the development of SP, as a disease characterized by a decrease in MM and strength can contribute to the progression of OA by reducing the stability of joint structures and changing the biomechanics of movement. At the same time, pain, stiffness and mobility limitations, which are common symptoms of OA, can accelerate the development of SP by reducing physical activity. Such a bidirectional relationship progressively increases the level of disability among the elderly.

Osteoarthritis and sarcopenic obesity

SPO is a syndrome characterized by a simultaneous decrease in muscle strength and function, i. e. SPO combined with OA [32, 33]. In recent years, more and more studies have appeared in the scientific literature on this syndrome, as it is gaining relevance due to changes in nutrition and a decrease in the level of physical activity of the population. Although SPO and OA are separate diseases, they share common pathophysiological mechanisms and risk factors, including lifestyle characteristics, age-related and hormonal changes, and increased synthesis of pro-inflammatory cytokines and reactive oxygen species. In addition, these two conditions exacerbate the clinical manifestations and consequences of each other, which creates a vicious circle [34].

In the Ukrainian population aged 20–90 years, the frequency of SPO in women reaches 9.8 % and in men 9.6 % [32]. These figures are consistent with global data, where the prevalence varies from 0.8 to 22.3 % in women and from 1.3 to 15.4 % in men [34]. However, in patients with OA, the frequency of SPO is significantly higher and reaches 49.6 %, which is important to consider in the treatment and rehabilitation of patients [35].

A recent meta-analysis, which included 12 studies, found that low SMMI and SPO increase the risk of developing gonarthrosis by 1.36 and 1.78 times, respectively [36]. In addition, SPO is a factor that increases the risk of falls and fractures in elderly people with OA [37, 38].

A review by S. Balogun et al. [39] demonstrates that SPO is more frequently diagnosed in patients with bilateral knee OA compared with unilateral knee OA. In addition, patients with SPO complained of pain more often than patients with SP without OA, but the authors did not record a significant difference in pain intensity between patients with SPO and OA without SP.

The NHANES study [40] found that patients with SPO had a higher incidence of OA (23.4 %), as well as comorbidities such as hypertension (47.8 %) and type 2 diabetes (12.0 %) compared with patients regardless of the presence of SP and OA. In addition, they had increased serum levels of triglycerides (TG), cholesterol, glucose, urea, creatinine and uric acid. Multivariate analysis showed that the TG index can be used to predict the risk of OA in patients with SPO [40].

Recently, the association between OA, SPO and OA without SP in 4,362 postmenopausal women was analyzed. The authors found that SPO is associated with an increased risk of OA (61.49 vs. 41.54 % in individuals without SP and OB, $p < 0.001$) and more severe pain syndrome (39.11 vs. 27.55 %; $p < 0.001$). Patients with SPO had a 20% stronger association with OA and 11% more often complained of knee pain compared to individuals without musculoskeletal diseases. In addition, patients with SPO required joint replacement more often [41].

The increased need for TKA in individuals with SPO and OA is also demonstrated by the results of other studies [42]. In addition, unbalanced nutrition and insufficient nutrient intake are factors that contribute to the development of SP, physical weakness and can negatively affect the results of TKA. A retrospective cohort study of 587 subjects aged 60 years and older examined the effect of SPO and associated factors on the recovery of range of motion after total knee arthroplasty. Patients were divided into three groups: with SPO, OB and without it. Knee flexion range of motion was measured before and after SPO. Patients with OB and SPO had a higher probability of poor recovery compared with the group without OB (for both groups, $p < 0.001$). The SPO group had the highest risk of postoperative complications in the form of low range of motion (adjusted hazard ratio HR = 1.63; $p = 0.03$) [43].

There are currently several mechanisms linking OA and SPO. The first involves the interaction of inflammatory, biomechanical, and metabolic factors. The accumulation of adipose tissue around muscle leads to an increase in the concentration of pro-inflammatory cytokines, which, in turn, accelerate the loss of MM, which causes the development

of joint instability, increased pain, and progression of OA symptoms. Univariate logistic regression analysis revealed that SPO (OR = 6.68; 95 % CI: 4.70–9.49; p < 0.001) and TG index (OR = 1.46; 95 % CI: 1.34–1.59; p < 0.001) are significant independent factors for the development of OA. In addition, the analysis showed that older age (OR = 3.12; 95 % CI: 2.65–3.68, p < 0.001), female gender (OR = 1.87; 95 % CI: 1.58–2.20; p < 0.001), high BMI (OR = 1.04; 95 % CI: 1.02–1.06; p < 0.001), arterial hypertension (OR = 2.16; 95 % CI: 1.85–2.52; p < 0.001), type 2 diabetes (OR = 1.92; 95 % CI: 1.58–2.33; p < 0.001) and smoking at the time of examination (OR = 1.37; 95 % CI: 1.16–1.62; p < 0.001) were also significant risk factors for OA [40].

Literature data indicate that a decrease in AFM in combination with an increase in FM contributes to increased joint load, the development of chronic inflammation and degenerative-dystrophic changes in cartilage tissue. In a study by J. N. Chopp-Hurley et al. [44] analyzed the characteristics of nutrition and physical activity in individuals with different forms of OA and found that low physical activity and insufficient fiber intake were associated with an increased risk of OA, although they were not related to total body weight. Given that dietary interventions demonstrated a simultaneous decrease in both AFM and FM, the results emphasize the importance of analyzing body composition, not just body weight, to determine the effectiveness of diet and physical activity in OA. At the same time, intensive water exercises with resistance for OA reduced FM and contributed to the improvement of walking speed after a 4-month intervention period.

In summary, current literature data indicate that a decrease in MM combined with an increase in FM leads to increased load on the joints, the development of chronic inflammatory processes and acceleration of degenerative-dystrophic changes in cartilage tissue. The results obtained demonstrate that patients with SPO have more pronounced symptoms of OA, more often require TKA and recover worse after surgical intervention.

Osteoarthritis and Dysmobility Syndrome

DS is a condition that combines several factors that cause functional disorders and increase the risk of developing musculoskeletal complications, including: osteoporosis (OP), SP and SPO. This term was first proposed by Professor N. Binkley and colleagues in 2013, who defined DS as the presence of three or more of the following six criteria in a person: 1) bone mineral density (BMD) of the lumbar spine, femoral neck, or proximal femur ≤ –2.5 standard deviations; 2) BMI ≤ 7.26 kg/m² in men and 5.45 in women; 3) body fat content > 30% in men and > 40 in women; 4) walking speed < 1 m/s; 5) hand muscle strength (dynamometry) < 30 kg in men and < 20 in women; 6) the presence of one or more falls in the last 12 months. These criteria allow for a comprehensive assessment of the patient's condition and identify risks associated with DS. This approach is not new, the use of a combination of factors associated with adverse health outcomes is widely accepted in clinical practice, for example in the case of metabolic syndrome [38, 45, 46].

The prevalence of DS according to various authors ranges from 3.9 to 54.1 % [37, 46–48]. At the same time, it was found that this indicator is higher in urban populations compared to rural residents (31.6 vs. 27.9 %, respectively). A similar trend was recorded for comorbid pathology in the form of a combination of SP and DS (32.6 in the urban cohort vs. 28.4 % in the rural cohort) [49]. Risk factors for DS development include female gender, advanced age, history of falls and fractures, including osteoporotic fractures, SP, osteopenia and OP, presence of chronic diseases, OA, metabolic syndrome or its individual components (e. g., obesity, hypertension, dyslipidemia), low physical activity and excessive alcohol consumption. These factors may interact with each other, increasing the risk of developing DS and its associated complications [46, 48].

Insufficient physical activity is one of the pathogenic factors for the development of DS. Thus, a study involving 375 people aged 60–97 years found that insufficiently active people were approximately 2 times more likely to develop DS (95 % CI: 1.14–3.79; p < 0.05), regardless of BMI, smoking, and ethnicity [47].

In their study, N. Hong et al. [49] assessed jump power in residents of the USA and South Korea as a predictor of SP, DS, or their combination. The jump with the maximum height was selected for analysis with subsequent registration of its peak power (product of force and jump velocity) adjusted for the mass of the subjects (jump power to body weight ratio, W/kg). US participants were more likely to have elevated FM compared to South Korean participants (46.3 % vs. 19.5 %, p < 0.001), while the latter had worse gait speed (43.7 % vs. 11.9 %, p < 0.001) and PD (26.3 % vs. 42.1 %, p < 0.001). This analysis of age- and sex-matched individuals from the two cohorts suggests that jump power values of less than 19.0 W/kg in women and less than 23.8 in men can be used as international cutoffs for identifying individuals with SP and DS. Low power was associated

with an increased risk of developing SP (OR = 4.07), DS (OR = 4.32), or a combination of SP and DS (OR = 4.67, $p < 0.01$ for all measures) regardless of age, sex, height, and ethnicity.

In a study by M. M. Khaleghi et al. [48], which examined the relationship between the distribution of adipose tissue in different parts of the body and the likelihood of developing DS in 2,426 individuals over 60 years of age, it was found that FM and FM to AFM ratio were significantly associated with DS (OR = 1.04; 95 % CI: 1.02–1.05) and (OR = 3.42; 95 % CI: 1.95–5.99, respectively). The accumulation of FM within the trunk was also associated with the development of DS, but had a weaker relationship (OR = 1.02; 95 % CI: 1.00–1.03) and OR = 2.45; (95 % CI: 1.36–4.39, respectively). No statistically significant effect of smoking, excessive alcohol consumption, and the prevalence of chronic diseases on the development of DS was found, but the prevalence of DS was lower among married individuals ($p < 0.001$). In addition, it was proven that height, body weight, BMI, neck, waist, and hip circumferences were significantly lower in individuals with DS compared to individuals without DS. These patients also had a significant decrease in body MM, AFM, and BMD at the level of the lumbar spine and femur. In addition, individuals with DS demonstrated a low level of physical activity (82.6 % were sedentary compared to 69.5 % without DS), had lower physical performance (8.84 % vs. 9.97 %), lower walking speed (0.71 % vs. 1.0 m/s), and lower muscle strength measured by handgrip dynamometry (17.64 % vs. 27.62 kg). The results suggest that screening for body fat distribution in the elderly may be a valuable strategy for early diagnosis of DS and management aimed at preventing disability and improving their quality of life [48].

In their study, W. Sun et al. [37] found that the characteristics of the frequency body mass index (FBMI), which is calculated based on the calculation of the frequency of human body oscillations according to the three-dimensional gait analysis system and BMI of patients aged 60–90 years with DS, which is associated with bone quality, fracture risk, percentage of body fat, SMMI, hand muscle strength and walking speed, HR and 95 % CI for FBMI in the groups without DS and DS were 0.82 (0.74–0.90), respectively. According to the results of ROC analysis, FBMI had a prognostic value for differentiating individuals without DS from patients with DS (AUC = 0.67; $p < 0.05$), with an optimal threshold for prediction of 16.04 (sensitivity = 0.48, specificity = 0.77).

To date, research results demonstrate that the accumulation of adipose tissue (especially in the lower extremities and trunk) and low physical activity are associated with an increased risk of DS in elderly people with OA, thereby increasing pain and mobility limitations. On the other hand, OA promotes the development of muscle atrophy, which in turn accelerates the progression of DS.

Given this, the diagnosis and treatment of DS are important for the prevention of falls and fractures, especially among older people. Preventive measures may include muscle-strengthening exercises, weight control, treatment of OP and other strategies aimed at improving mobility and reducing the risk of injury.

Osteoarthritis and Malnutrition Syndrome

Malnutrition syndrome (MS) is a broad concept that combines both nutrient deficiencies and excesses or imbalances in the intake of proteins, vitamins and trace elements. The prevalence of MS among patients with musculoskeletal diseases varies between 9–39 %, while the average rate among hospitalized patients is 20–50 %. In patients with OA, the frequency of MS is up to 69.5 % [50], while one in five patients does not receive enough nutrients to meet their energy and protein needs. The prevalence of MS increases with age, as well as with an increase in the frequency of comorbid conditions, including OA and SP [51, 52]. MS is correlated with an increased risk of SP, increasing its chances by almost 3 times (OR = 2.99; 95 % CI: 2.40–3.72) [6].

MS is an independent risk factor that negatively affects the clinical outcomes of treatment of somatic and surgical pathologies, reduces the quality of life, disrupts the functional state of the body and limits the autonomy of patients. It is also a reliable and modifiable predictor of postoperative complications and adverse outcomes in orthopedic practice. MS is a well-known cause of increased length of hospital stay and mortality [50–52].

A review of the literature demonstrates a variety of approaches to the diagnosis and confirmation of MS, including anthropometric (mid-upper arm and lower leg circumference, BMI), laboratory (white blood cell count, hemoglobin, total protein, albumin, transferrin) parameters, and standardized nutritional status assessment scales [50, 53, 54]. Each of the above items is scored on a scale from 0 to 3, with age > 70 years considered an additional risk factor (+1 point). A total score ≥ 3 indicates an increased risk of MS. An albumin concentration < 3.5 g/dL is often considered a key marker of MS and has been defined as the “gold standard” in some studies. [50, 53].

In elderly patients, protein deficiency causes a decrease in the level of insulin-like growth factor 1 (IGF-1), the main anabolic regulator of cartilage

homeostasis, which contributes to the development of OA. In an experimental study in rats, the effect of an isocaloric low-protein diet (ILPD) on cartilage and subchondral bone was studied. Morphometric parameters of trabecular and cortical subchondral bone, thickness of hyaline and calcified cartilage, and proteoglycan content were analyzed using micro-computed tomography. After 2 months of the study, a decrease in IGF-1 levels by 18 % ($p < 0.001$) was observed in the ILPD group. The mass of trabecular subchondral bone decreased by 10 % ($p < 0.01$), and the thickness of the cortical plate of the subchondral bone of the medial condyle was reduced by 12 % ($p < 0.05$). A deterioration in the biomechanical properties of hyaline cartilage (strength, elasticity, and work energy) was found, which decreased by 47, 58, and 41 %, respectively ($p < 0.01$). No changes in the content of proteoglycans were recorded. This pattern of cartilage degradation is similar to that observed in the early stages of OA [55].

It is known that MS worsens the course of OA and slows down recovery after surgical interventions for TKA. In particular, insufficient protein intake (less than 1.2–1.5 g/kg body weight) and low energy value of the diet (less than 27–30 kcal/kg body weight) correlate with lower indicators of motor activity of patients according to the results of the Hip disability and osteoarthritis outcome score (HOOS) questionnaire and the short-form health survey (SF-12) after hip arthroplasty, and adherence to the Mediterranean diet is associated with better functional status of joints and higher hemoglobin values ($p < 0.05$) [56].

According to the results of systematic reviews and meta-analyses, in patients with MS who underwent TKA, including for knee or hip OA, an overall increase in the number of complications by 93 % was observed, including: an increase in the duration of hospital stay (according to different authors from 0.3 to 1.7 days), repeated hospitalizations, an increased risk of postoperative complications, in particular sepsis (OR = 2.13; 95 % CI: 1.29–3.51; $p = 0.03$) and acute postoperative infections (OR = 5.9; 95 % CI: 1.32–26.06; $p = 0.02$) [50, 57, 58] The analyzed sources indicate the presence of a close relationship between MS and OA. Insufficient protein in the diet, as well as its reduced calorie content, increases the risk of SP by more than 30 %, which, in turn, accelerates the progression of OA by reducing MM. Patients with a combination of OA and MS have been shown to have slower recovery after surgical interventions and a higher risk of complications. Given these common pathophysiological mechanisms, the need for an integrated approach to the diagnosis, treatment and prevention of MS and OA is important.

Conclusions

OA is a serious medical and social problem that worsens the quality of life of patients, affecting not only the condition of the joints, but is also closely related to other geriatric syndromes, such as SP, SPO, DS and MS. A decrease in muscle mass together with an increase in adipose tissue contributes to the acceleration of degenerative changes in cartilage tissue, the progression of inflammation and impaired biomechanics of movement. Insufficient physical activity and unhealthy diet increase the risk of developing SP, DS and MS, which, in turn, complicate the course of OA. The literature analysis demonstrated bidirectional mechanisms of interaction between these conditions, which emphasizes the importance of early diagnosis and a multidisciplinary approach to the combination of geriatric syndromes.

Conflict of interest. The authors declare no conflict of interest.

Prospects for further research. Conducting prospective cohort studies to determine the frequency of sarcopenia, sarcopenic obesity, dysmobility syndrome and malnutrition in the Ukrainian population both in isolation and in combination with osteoarthritis of various localizations. In addition, the task of developing and validating comprehensive screening algorithms that combine clinical assessment of OA symptoms with the detection of signs of the above-mentioned geriatric syndromes is relevant.

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OSTEOARTHRITIS AND GERIATRIC SYNDROMES: FEATURES OF THE RELATIONSHIP AND MANAGEMENT OPPORTUNITIES
(LITERATURE REVIEW)

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