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ABSTRACT

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SARCOPENIA AND INFLAMMATORY BOWEL DISEASE: DEVELOPMENTAL MECHANISMS AND PRACTICAL IMPLICATIONS

Introduction. Sarcopenia is a generalised and progressive decline in skeletal muscle mass, strength and performance, leading to a reduction in quality of life, increased risk of falls and fractures, hospitalisation and mortality. While primary sarcopenia is a natural manifestation of ageing, secondary loss of muscle strength and mass can occur in many acute or chronic pathological conditions and is associated with a poor prognosis. Currently, there is growing interest in premature sarcopenia, which occurs in the setting of acute and chronic inflammatory processes, including inflammatory bowel disease.

Materials and methods. The literature review was based on scientific studies published by Science Direct using the information retrieval systems PubMed, Web of Science, Scopus, Google Scholar, The Cochrane Library, Medknow. The research was mainly reviewed for the last 5 years. A total of 65 scientific papers were selected for this article.

Results. Published studies suggest an association between sarcopenia and disease activity, the need for surgical treatment and adverse postoperative outcomes. Sarcopenia occurs in 20–70% of patients with inflammatory bowel disease. In recent years, the diagnosis of nutritional status disorders in patients with inflammatory bowel disease has changed due to the prevalence of obesity. Sarcopenia in such patients may occur in the setting of obesity and remain undiagnosed for a long time.

Conclusions. Sarcopenia is associated with high activity and higher incidence of postoperative complications in patients with IBD and is an independent predictor of surgical intervention. Sarcopenic obesity is one of the features of the modern phenotype of a patient with inflammation bowel disease. Early diagnosis, prevention and treatment of sarcopenia and malnutrition in patients with inflammation bowel disease are likely to become one of the therapeutic goals in the future, in addition to clinical and endoscopic remission of the disease.

Keywords: sarcopenia, inflammatory bowel disease, nutritional status, sarcopenic obesity.

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САРКОПЕНІЯ ТА ЗАПАЛЬНІ ЗАХВОРЮВАННЯ КИШЕЧНИКУ: МЕХАНІЗМ РОЗВИТКУ ТА ПРАКТИЧНЕ ЗНАЧЕННЯ

Вступ. Саркопенія – генералізоване і прогресуюче зниження маси скелетних м'язів, їхньої сили та працездатності, що призводить до зниження якості життя, збільшення ризику падіння та переломів, госпіталізації та смертності. У той час як первинна саркопенія є закономірним проявом старіння, вторинна втрата м'язової сили і маси може виникати на тлі багатьох гострих або хронічних патологічних станів та асоціюється з несприятливим прогнозом. Нині зростає інтерес до саркопенії, що передчасно розвинулася і спостерігається на тлі гострих і хронічних запальних процесів, у тому числі на тлі запальних захворювань кишечника.

Матеріали і методи. Літературний огляд проведено на основі опублікованих наукових досліджень Science Direct за допомогою інформаційно-пошукових систем PubMed, Web of Science, Scopus, Google Scholar, The Cochrane Librar, Medknow. Проведений огляд досліджень переважно за останні 5 років. Для написання статті відібрано 65 наукових праць.

Результати. Опубліковані дослідження вказують на взаємозв'язок саркопенії з активністю захворювання, потребою в хірургічному лікуванні та несприятливими післяопераційними наслідками. Саркопенія зустрічається у 20–70% пацієнтів із запальними захворюваннями кишечника. Останніми роками діагностика характеру порушень нутритивного статусу в пацієнтів із запальними захворюваннями кишечника зазнає змін у зв'язку з поширенням ожиріння. Саркопенія у таких пацієнтів може протікати на тлі ожиріння, довго залишаючись недіагностованою.

Висновки. Саркопенія асоційована з високою активністю та більшою частотою післяопераційних ускладнень у хворих із запальними захворюваннями кишечника і виступає в ролі незалежного предиктора хірургічного втручання. Саркопенічне ожиріння – одна з особливостей сучасного фенотипу хворого на запальні захворювання кишечника. Своєчасна діагностика, профілактика і лікування саркопенії та мальнутриції у хворих на запальні захворювання кишечника, імовірно, стане однією з терапевтичних цілей на додачу до клінічної та ендоскопічної ремісії захворювання в майбутньому.

Ключові слова: саркопенія, запальні захворювання кишечнику, нутритивний статус, саркопенічне ожиріння.

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ABBREVIATIONS

UC – ulcerative colitis
IBD – inflammatory bowel disease
BMI – body mass index
SCFA – short chain fatty acids
CD – Crohn's disease
GI – gastrointestinal

INTRODUCTION

The term “sarcopenia” was coined by Professor Irwin Rosenberg in 1989 (from the Greek *sarx* – body, flesh and *penia* – lack). In 2010, the European Working Group on Sarcopenia in Older People (EWGSOP) defined the condition as a progressive and generalised loss of muscle mass and strength in older people, with an increased risk of adverse outcomes such as reduced quality of life, disability and death. In 2016, sarcopenia was recognised as a disease and included in the International Classification of Diseases. In 2019, the updated EWGSOP-2 guidelines [1] expanded and clarified the diagnostic criteria for sarcopenia and the methods for assessing muscle mass and strength. Since the first EWGSOP consensus, scientists and clinicians have studied many aspects of the disease and concluded that sarcopenia can occur not only in older people, but also in younger people as a complication of certain chronic and acute diseases (secondary sarcopenia). Studies have shown that muscle strength is an important marker for predicting adverse outcomes [2]. When comparing the importance of muscle strength and muscle mass, early diagnosis of muscle strength deficiency (dyspnoea) is of paramount importance [3], as it is more convenient to measure, whereas measuring muscle mass is technically difficult. However, the researchers point out that as tools and methods for assessing the quantitative characteristics of muscle mass improve, the practical importance of the latter will increase.

Search strategy. Data from the scientometric databases PubMed, Web of Science, Scopus, Google Scholar, The Cochrane Library, Medknow were used for the scientific review using a combination of the terms 'sarcopenia', 'inflammatory bowel disease', 'sarcopenic obesity', 'nutritional status', 'malnutrition', 'muscle mass'. The search was conducted from September 2024 to January 2025. The literature data on the relationship between sarcopenia and inflammatory bowel disease activity, its impact on the development of postoperative complications and the course of inflammatory bowel disease in the setting of sarcopenic obesity were reviewed and summarised.

The 65 papers selected for the article are either the most recent publications (within the last 5 years) or the most recent publications on the topic under review (regardless of age).

RESULTS AND DISCUSSION

Factors such as malnutrition, physical inactivity and concomitant polymorbidity in the elderly are involved in the development of primary (age-related) sarcopenia. Drug treatment, malabsorption syndrome, systemic inflammatory reactions, endocrine disorders, obesity and malnutrition play a key role in the development of secondary sarcopenia. The decline in muscle mass begins in the third or fourth decade of life and progresses at a rate of 0.5–1% per year, with a sharp decline after the eighth decade [4]. Muscle strength also decreases, but not in direct proportion to the loss of muscle mass. Age-related loss of skeletal muscle mass is due to atrophy of muscle fibres, deposition of lipids and their derivatives both directly in and between myocytes (myosteatosis), which not only causes mitochondrial dysfunction but also disrupts β-oxidation of fatty acids, increasing the production of reactive oxygen species. These processes lead to lipotoxicity, insulin resistance and increased secretion of a number of pro-inflammatory cytokines. Thanks to modern research methods, we now know that muscles, which account for almost 50% of body weight, produce myokines [5] – substances that counteract pro-inflammatory signalling molecules – in addition to their main function, which is locomotion.

Myokines include myostatin, leukaemia suppressor factor, interleukin (IL) 6, IL-7, brain-derived neurotrophic factor, insulin-like growth factor 1 (IGF-1), somatomedin C, fibroblast growth factor and irisin [6]. The role of tumour necrosis factor alpha (TNF-alpha) and IGF-1 in the development of sarcopenia has been well studied. Expression of TNF-α promotes inflammation through activation of the nuclear factor NF-kB, leading to muscle loss. Another pathway that regulates mitochondrial metabolism involves IGF-1. The age-related decline in IGF-1 levels is thought to impair mitochondrial activity by reducing ATP citrate lyase, contributing to the development of sarcopenia [7]. Sarcopenia is also associated with changes in motor neuron function, leading to both muscle wasting and reduced muscle function [8]. A genetic predisposition to sarcopenia has been discussed. For example, the genes ACE (angiotensin converting enzyme), MSTN (myostatin), IGF1 (IGF-1), IL-6 (IL-6) and VDR (vitamin D receptor) have been shown to be associated with muscle strength and/or mass. Double-blind studies have shown that the heritability of some muscle mass and strength traits is up to 80% [9, 10].

Sarcopenia and ageing

The progressive loss of muscle mass and strength is considered a hallmark of ageing. Numerous studies have

shown that sarcopenia in older people is associated with a number of adverse outcomes, including an increased risk of disability and mortality, and the need for more frequent and longer hospitalisations. The following factors play a role in the development of sarcopenia in older and senile people:

- Chronic systemic inflammation of low severity, characterised by the expression of pro-inflammatory cytokines [11];
- Vitamin D deficiency, which leads to a decrease in the expression of 1,25-dihydroxyvitamin D receptors in skeletal muscle, with a subsequent decrease in protein synthesis and functional responses of myocytes [12,13];
- Low physical activity with a decrease in myokine production [14];
- Unbalanced diet and reduced food intake [15].

Mechanism of sarcopenia in inflammatory bowel disease

According to various studies, sarcopenia occurs in 20–70% of patients with inflammatory bowel disease (IBD) [16,]. Malnutrition is the leading link in the pathogenesis of its development [17]. Malnutrition syndrome is observed in 20–85% of patients with IBD due to chronic non-specific inflammation, dietary restrictions caused by poor tolerance to certain foods, malabsorption syndrome and intestinal epithelial damage [18, 19].

The development of the malnutrition syndrome is accompanied by vitamin D deficiency, the role of which has been actively studied in recent years. In addition to maintaining bone mineral density, vitamin D is involved in muscle contraction by regulating intracellular calcium concentration, myocyte proliferation and differentiation. In the gastrointestinal (GI) mucosa, it is involved in modulating the immune response by exerting anti-inflammatory effects [20] and also ensures the integrity of the mucosal epithelial barrier by regulating tight junction proteins [21]. Vitamin D deficiency occurs in 30–47% of patients with IBD [22].

There is an association between vitamin D deficiency and the risk of developing sarcopenia, cardiovascular disease, obesity and osteoporosis [23]. Vitamin D deficiency contributes to a decrease in the expression of receptors for this vitamin [24], inhibition of oxidative phosphorylation, development of mitochondrial dysfunction, and increased formation of reactive oxygen species [25, 26], which directly or indirectly leads to the breakdown of muscle fibres and, as a result, muscle wasting.

The resection of part of the small or large bowel also contributes to the development of malnutrition, which, in combination with chronic inflammation, leads to a reduction in the contact time between the food lump and the absorptive surface. Crohn's disease (CD) is characterised by the involvement of all parts of the gastrointestinal tract, including the small intestine, where nutrient absorption is predominant, which is naturally associated with a more frequent development of malnutrition [27]. The mechanism of sarcopenia development in IBD also includes the uncontrolled release of pro-inflammatory cytokines such as TNF-α and IL-6 into the circulation [28], which are synthesised in particular by adipocytes of mesenteric adipose tissue [29], drug effects and physical activity restriction [30].

A dynamic balance between the synthesis and breakdown of skeletal muscle proteins is maintained by a constant supply of amino acids from the diet. The intestinal microflora plays a direct role in their absorption, and changes in the composition and diversity of this flora can affect the bioavailability of amino acids [31]. Substances produced by intestinal bacteria as part of their vital activity, of which short-chain fatty acids (SCFA) are the most studied, can also influence muscle fibre synthesis [32]. Once they enter the systemic circulation and are taken up by skeletal muscles, SCFA act on the mitochondria to stimulate muscle protein synthesis [33]. The pathways by which the gut microbiota affect the muscle system are collectively referred to as the 'skeletal muscle-gut axis'. Patients with IBD are characterised by a decrease in the number and species diversity of gut bacteria, an imbalance between bacteria that produce pro- and anti-inflammatory cytokines, which naturally leads to changes in signalling pathways [34]. Thus, intestinal dysbiosis in IBD patients is considered to be one of the mechanisms inducing the development of sarcopenia.

People with IBD are at high risk of developing nutritional deficiencies. For this reason, appropriate screening tests are recommended at the time of IBD diagnosis and at least once a year thereafter. This attention is justified by the fact that the development of malnutrition in patients with IBD worsens prognosis and quality of life, and increases the incidence of complications and mortality [35]. There are currently no validated screening tools to assess the nutritional status of patients with IBD [36]. However, tools that have been used include the Nutritional Risk Screening (NRS-2002), the Multiple Undernutrition Screening Tool (MUST), the Malnutrition Screening Tool (MST), the Malnutrition in Inflammation Risk Taking Tool (MIRT), and the SaskIBD Nutritional Risk Screening for IBD (SaskIBD-NR) [37].

To assess skeletal muscle mass, clinical studies use dual-energy x-ray absorptiometry, bioimpedance analysis and measurement of the cross-sectional area of

the lumbar muscle at the level of the third lumbar vertebra on images obtained during computed tomography or magnetic resonance imaging. The advantage of radiological methods is the possibility of simultaneous diagnosis of the abdominal cavity and assessment of the muscular system. In general, there is a need to develop diagnostic criteria for sarcopenia in patients with IBD, as the currently available criteria were developed to diagnose primary age-related sarcopenia [38].

Practical importance of early diagnosis of sarcopenia in IBD

Scales developed to date for perioperative risk stratification do not always adequately reflect the body's physiological reserves. The main task in the preoperative phase is to assess the body's ability to withstand surgical stress. Patients with IBD are characterised by a predominantly young age, active inflammation and the need for immunosuppressive therapy and surgical treatment. In order to improve the outcome of surgery, it is recommended that an appropriate pre-operative risk assessment be carried out, taking into account the nutritional status of the patient.

In the preoperative period, a decrease in body mass index (BMI) and albumin levels, as well as high levels of C-reactive protein, have been observed in patients with IBD and sarcopenia [39]. According to other publications, 40% of these patients had a normal body mass index (BMI) and 20% were overweight – so-called sarcopenic obesity [40]. The authors emphasise the need to assess these parameters when it is not possible to use more accurate methods to diagnose sarcopenia [41].

Studies including only patients with ulcerative colitis (UC) have shown that sarcopenia is a marker for the need to extend medical therapy and colectomy in patients with severe exacerbations [42]. A high incidence of colectomy in patients with sarcopenia was found in the study by T. Zhang et al [43]. In addition, sarcopenia is considered an independent factor in determining the need for surgery [44].

Retrospective cohort studies have shown that sarcopenia is an independent predictor of adverse postoperative outcomes in IBD patients under 40 years of age [45, 26]. Postoperative complications of Clavien-Dindo class III and higher were more common in patients with sarcopenia [47]. Malnutrition and hypoalbuminemia were also found to play a leading role in the development of postoperative complications [48].

In a study conducted in China, patients with UC were divided into 2 groups according to the degree of disease activity: the first group consisted of patients with a Mayo index <6 and the second group >6. Sarcopenia was significantly more common in patients with a high disease activity index and was an independent predictor of a high Mayo index. After colectomy, albumin, haemoglobin concentrations and muscle mass increased significantly and the severity of sarcopenia decreased, confirming the role of active inflammation [49]. These data are consistent with those obtained in another study that observed the reversal of sarcopenia during infliximab treatment in patients with CD [50].

Patients with IBD often exclude certain foods (e.g. fruit and vegetables) from their diet, which is associated with changes in body composition. The BMI index is used in clinical practice for routine assessment of nutritional status. Anthropometric data do not allow determination of the state of the muscular system, the volume of visceral, subcutaneous and intermuscular adipose tissue, and therefore the calculation of BMI as the main method in IBD is not informative [51]. This is because patients with IBD have a decrease in muscle mass, while body fat and visceral fat indices increase [52]. At the same time, an increase in visceral fat does not necessarily correlate with BMI [53].

The proliferation of mesenteric adipose tissue that is most characteristic of CD is associated with a 4-fold increase in the number of adipocytes per unit area [54]. A number of investigators have associated an increase in visceral fat mass with a more aggressive course of the disease, poor response to medical therapy and, consequently, the need for surgical treatment [55]. Together with sarcopenia, visceral obesity is an independent risk factor for the development of postoperative complications [57]. An increase in visceral fat is also associated with postoperative recurrence of CD [58]. However, no such associations have been found in patients with CD, probably due to the localisation of the pathological process exclusively in the mucosa and the absence of transmural lesions [33].

Sarcopenic obesity in IBD patients

Obesity is associated with hyperplasia and hypertrophy of adipocytes, infiltration of adipose tissue with immunocompetent cells and, most importantly, the production of adipokines. The ability of adipose tissue to produce signalling molecules and to influence the metabolism of internal organs, i.e. to act as an organ of the endocrine system, has only recently been discovered [59]. One of the effector organs is muscle tissue, whose paracrine regulation is mediated by inter- and intramuscular adipocytes. Dysregulation of adipose tissue endocrine function is accompanied by the development of local inflammation due to the production of free fatty acids, TNF-α and IL-6 [60, 61]. Adipose tissue proinflammatory cytokines induce mitochondrial dysfunction associated with impaired lipid β-oxidation and reactive oxygen species generation, thereby initiating and maintaining skeletal muscle dysfunction. These effects of adipokines on muscle tissue raise the

question of whether the term 'sarcopenic obesity is appropriate. The ability of adipose tissue cells to induce skeletal muscle dysfunction and myocyte apoptosis shifts the balance towards the primary development of obesity and then, as a consequence, sarcopenia. To highlight the mechanism and sequence of pathogenesis, the term 'sarcopenia in obesity has been proposed [62].

The risk of developing sarcopenia in patients with IBD is increased due to the prolonged course of the inflammatory process, concomitant malnutrition and medication. Its presence is not always obvious due to the prevalence of obesity both in the general population and in patients with IBD [63]. As the diagnostic criteria for sarcopenia were originally developed to assess age-related sarcopenia, researchers emphasise the need to develop specific criteria for patients with secondary sarcopenia, including those with IBD. In the available publications, the diagnosis of sarcopenia was usually based on the assessment of muscle mass without considering strength and performance. Due to the lack of diagnostic criteria, the lower limit of normal varies in most studies, making it impossible to meta-analyse the available data. There is no doubt about the negative effects associated with sarcopenia, but no multicentre prospective studies have been conducted on the impact of sarcopenia on the course, activity and outcomes of surgical treatment in patients with IBD [64, 65].

CONCLUSIONS

Sarcopenia is associated with high IBD activity, a higher incidence of postoperative complications in IBD patients and is an independent predictor of surgical intervention. Sarcopenic obesity is one of the features of the modern IBD patient phenotype. Early diagnosis, prevention and treatment of sarcopenia and malnutrition in patients with IBD, in addition to clinical and endoscopic remission of the disease, will be one of the therapeutic goals in the future.

PROSPECTS FOR FUTURE RESEARCH

Prospects for further research include the investigation and development of diagnostic criteria for assessing sarcopenia in IBD patients, the inclusion of sarcopenia in existing models for predicting surgical risk, and multicentre prospective studies to confirm the impact of sarcopenia on the course and activity of IBD.

AUTHOR CONTRIBUTIONS

All authors substantively contributed to the drafting of the initial and revised versions of this paper. They take full responsibility for the integrity of all aspects of the work.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

ARTIFICIAL INTELLIGENCE DISCLOSURE

The authors confirm that no artificial intelligence-based technologies were used in the writing or editing of the manuscript.

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