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SARCOPENIC OBESITY IN NON-ALCOHOLIC FATTY LIVER DISEASE

Multivariate analysis has shown that osteosarcopenic obesity, which included obesity, sarcopenia, and osteopenia, has a significant association with NAFLD in women over 50 years of age, even with a normal body mass index [1].

Recent studies have shown that skeletal muscle acts as an endocrine organ [2]. Sarcopenia is based on several pathological processes: reduction in the number of muscle fibers, reduction in their size, violation of the innervation of myofibrils, as well as fatty infiltration of muscles (myosteatosis). A number of studies have shown that fatty infiltration of muscles not only leads to loss of muscle mass and strength, but also contributes to insulin resistance, NAFLD and type 2 diabetes [3].

At a sarcopenia first of all quantity of cells-satellites and fibers of the II type decreases. Mature muscle fibers consist of multinucleated cells that are unable to divide, so muscle growth and regeneration occur due to the proliferation of satellite cells. These processes lead to the inability of the patient to make rapid movements.

Clinical studies describe a strong association between NAFLD and sarcopenia, although a causal relationship remains to be determined [4].

Skeletal muscle plays a crucial role in the transmission of insulin signals as the primary tissue responsible for insulin-mediated glucose utilization. Decreased skeletal muscle mass can cause insulin resistance and dysglycemia, which will eventually lead to NAFLD and its characteristic features. Oxidative stress and chronic inflammation cause muscle atrophy and lead to stress responses in hepatocytes, leading to the progression of liver fibrosis associated with NASH and NAFLD [5].

Interestingly, in the state of chronic inflammation, proinflammatory cytokines, especially

IL-6, IL-1 and TNF-α, promote osteoclast activation and subsequent bone resorption. IL-6 and IL-1 directly modulate osteoclastogenesis by enhancing osteoclast function. Because IL-6 increases during liver damage to stimulate liver regeneration, existing elevated levels of IL-6 may affect bone remodeling in various types of liver disease [6].

In particular, TNF-α enhances CSF-1 receptor gene expression in the early stages of osteoclastogenesis and subsequently stimulates osteoblast precursors, leading to increased osteoclast formation regardless of the kappa nuclear factor receptor (RANKL) pathway. These proinflammatory cytokines affect the development of osteoporosis in viral hepatitis and nonalcoholic steatohepatitis (NASH).

According to the mechanism of sarcopenia is divided into primary (due only to the aging process) and secondary, which occurs in the presence of any pathology that can affect the condition of muscle tissue (eg, systemic inflammatory diseases). To date, many studies have been conducted to study this problem in the elderly and in patients with a number of chronic diseases. However, scientific work on the study of the composition of the body in the elderly and long-lived is not so much, and their results are quite contradictory.

Our surveys of men and women with NAFLD revealed the most informative anthropometric indicators for the screening diagnosis of NAFLD [7]. According to Mateiko's formulas, body fat in patients with NAFLD was statistically significantly higher, and muscle and bone mass were statistically significantly lower than in healthy men and women of the same sex. Inverse correlations of medium strength ($r = 0.52$, $p < 0.001$) have been established between Matejko's bone mass and the body mass index in men and women with NAFLD [8].

Numerous studies convincingly suggest that abdominal obesity, hypertension, dyslipidemia, and dysglycemia are considered components of the metabolic syndrome and are closely associated with sarcopenia and osteoporosis. Patients with NAFLD with liver fibrosis were found to be significantly more likely to have severe osteoporosis and hip fractures in Korean men over the age of 50, and this positive association was more pronounced in patients with sarcopenia [9].

The primary task of treating patients with NAFLD with overweight and obesity is to reduce the body weight of patients. There are currently no tools for the pathogenetic therapy of NAFLD that have a convincing evidence base. The pathogenetic treatment of NAFLD is to reduce insulin resistance, systemic inflammation of oxidative and cytokine-mediated stress, reduce the level of free fatty acids and stop fibrogenesis [10].

Despite the high prevalence and clinical significance of osteoporosis and sarcopenia in patients with liver disease, the treatment of these musculoskeletal disorders is often not considered in the clinical practice of patients with liver disease. Currently, there is no specific treatment for sarcopenia, the primary prevention is. Exercise, especially those where strength is gradually increased, is a stimulus for the synthesis of muscle protein [11].

Adipokines (mainly leptin), sex hormones, glucocorticoids and impaired glucose metabolism also play a role in the development of myosteatosis [12].

It is known that exercise with exercise significantly reduces the loss of bone and muscle mass associated with age, as well as benefit the whole body. In particular, it has been suggested that dynamic resistance supplements, supported by adequate dietary supplements, may be the most promising strategy for improving the clinical condition of elderly patients with osteosarcopenia with beneficial metabolic effects and positive effects on the nervous and cardiovascular systems.

Thus, the analysis of recent studies has shown that a number of metabolic factors [13,14] in NAFLD (insulin resistance, lipolysis, oxidative stress, chronic inflammation, decreased insulin-like growth factor, decreased sex hormone levels, hyperammonemia) are the basis of the pathogenetic mechanism sarcopenia.

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