



URGANCH DAVLAT TIBBIYOT INSTITUTI
JANUBIY OROLBO'YI TIBBIYOT JURNALI

2 - TOM, MAXSUS SON-3. 2026

14.00.00 - TIBBIYOT FANLARI ISSN: 3093-8740

UDC 616.379-008.64:616.74-092-085

DYNAMICS OF MYOKINE STATUS AND METABOLIC PARAMETERS IN PATIENTS
WITH TYPE 2 DIABETES MELLITUS AND SARCOPENIA RECEIVING
COMPREHENSIVE THERAPY



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Abstract

Sarcopenia and type 2 diabetes mellitus (T2DM) share common pathophysiological mechanisms and are mutually aggravating, which makes the search for pathogenetically justified treatment approaches an urgent task. The present study examined changes in myokine status and metabolic parameters in patients with T2DM and sarcopenia receiving comprehensive therapy. A total of 55 patients aged 55 to 65 years were examined: 35 patients with T2DM and sarcopenia (Group 1) and 20 patients with T2DM without sarcopenia (Group 2). Sarcopenia was diagnosed according to the EWGSOP criteria using handgrip dynamometry, dual-energy X-ray absorptiometry, and a gait speed test. Comprehensive therapy comprised optimized glucose-lowering treatment, loading doses of vitamin D, a high-protein diet, and an individualized program of aerobic and resistance training. After six months, both groups achieved comparable improvement in carbohydrate metabolism, whereas the reduction in insulin resistance was more pronounced in Group 1 (HOMA-IR decreased 1.74-fold versus 1.41-fold in Group 2). In patients with sarcopenia, serum myostatin decreased by 33.6%, irisin increased 1.67-fold, and vitamin D reached optimal values. These changes were accompanied by significant gains in handgrip strength, skeletal muscle index, and gait speed. Before treatment, myostatin correlated positively with insulin resistance and inversely with muscle strength, vitamin D level, and gait speed; after treatment these associations weakened. The findings demonstrate the reversibility of sarcopenic processes when pathogenetic treatment is started in a timely manner. However, muscle function indicators in Group 1 remained lower than in patients without sarcopenia, indicating the need for prolonged rehabilitation and long-term monitoring of myokine status.

Keywords: type 2 diabetes mellitus; sarcopenia; myokines; myostatin; irisin; vitamin D; insulin resistance; comprehensive therapy.

Annotatsiya

Sarkopeniya va 2-tur qandli diabet (QD2) umumiy patofiziologik mexanizmlarga ega bo'lib, bir-birining kechishini og'irlashtiradi. Shu sababli patogenetik asoslangan davolash yondashuvlarini izlash dolzarb vazifa hisoblanadi. Mazkur tadqiqotda kompleks terapiya fonida QD2 va sarkopeniya bilan og'rigan bemorlarda miokin holati hamda metabolik ko'rsatkichlarning dinamikasi o'rganildi. Yoshi 55 dan 65 gacha bo'lgan jami 55 nafar bemor tekshirildi: QD2 va sarkopeniya bilan og'rigan 35 nafar bemor (1-guruh) hamda sarkopeniyasiz QD2 bilan og'rigan 20 nafar bemor (2-guruh).



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Sarkopeniya tashxisi EWGSOP mezonlariga muvofiq qo'l dinamometriyasi, ikki energiyali rentgen absorbsiometriyasi va yurish tezligi testi yordamida qo'yildi. Kompleks terapiya optimallashtirilgan qand miqdorini pasaytiruvchi davo, D vitaminining to'yintiruvchi dozalari, yuqori oqsilli parhez hamda aerob va kuch mashqlarining individual dasturidan iborat bo'ldi. Olti oydan so'ng ikkala guruhda ham uglevod almashinuvi taqqoslanadigan darajada yaxshilandi, insulin rezistentligining pasayishi esa 1-guruhda yaqqolroq namoyon bo'ldi (HOMA-IR indeksi 1-guruhda 1,74 marta, 2-guruhda 1,41 marta kamaydi). Sarkopeniyali bemorlarda zardob miostatinini 33,6 %ga kamaydi, irizin 1,67 marta oshdi, D vitamini miqdori esa optimal qiymatlarga yetdi. Bu o'zgarishlar qo'l kuchi, skelet-mushak indeksi va yurish tezligining ishonchli oshishi bilan birga kechdi. Davolashdan oldin miostatin insulin rezistentligi bilan to'g'ri, mushak kuchi, D vitamini miqdori va yurish tezligi bilan teskari bog'langan edi; davodan keyin bu bog'liqliklar susaydi. Olingan natijalar patogenetik davo o'z vaqtida boshlanganda sarkopenik jarayonlarning qaytuvchanligini ko'rsatadi. Biroq 1-guruhda mushak funksiyasi ko'rsatkichlari sarkopeniyasiz bemorlarnikidan past bo'lib qoldi, bu esa reabilitatsiyani uzaytirish va miokin holatini uzoq muddat kuzatib borish zarurligini bildiradi.

Kalit so'zlar: 2-tur qandli diabet; sarkopeniya; miokinlar; miostatin; irizin; D vitamini; insulin rezistentligi; kompleks terapiya.

INTRODUCTION.

Sarcopenia shares common pathophysiological mechanisms with diabetes, a metabolic disorder characterized by impaired insulin function and abnormal glucose metabolism. Sarcopenia and diabetes involve a complex, bidirectional interaction between muscle integrity and metabolic homeostasis [1]. These conditions share common risk factors, such as aging, physical inactivity, obesity, and chronic inflammation. Both conditions are more prevalent among older adults, since aging is associated with a decline in muscle mass and function, as well as with an increased risk of insulin resistance and type 2 diabetes mellitus (T2DM) [2].

Therapeutic approaches, including antidiabetic drugs and anabolic agents, offer promising strategies for managing sarcopenia in T2DM by improving muscle health, metabolic function, and overall well-being [3]. Antidiabetic drugs such as metformin, glucagon-like peptide-1 receptor agonists (GLP-1 RAs), and sodium-glucose cotransporter-2 (SGLT2) inhibitors not only improve glycemic control but also exert favorable effects on muscle metabolism and function in individuals with T2DM-related sarcopenia [4]. In addition, anabolic agents, including selective androgen receptor modulators (SARMs), growth hormone secretagogues, and myostatin inhibitors, enhance muscle growth and function by modulating key pathways involved in protein synthesis and regeneration [5]. Nutritional supplements and dietary interventions may complement pharmacological therapy by providing essential nutrients and helping to preserve muscle mass in individuals with sarcopenia and T2DM.

Antidiabetic drugs play a central role in the management of T2DM and may also contribute to the preservation of muscle mass and function in people with sarcopenia. Oral antidiabetic agents, such as metformin, sulfonylureas, and thiazolidinediones, help regulate blood glucose levels by suppressing hepatic glucose production, improving insulin sensitivity, and enhancing glucose uptake by peripheral tissues, including skeletal muscle [5]. Metformin, the first-line agent for T2DM, supports muscle health by activating AMP-activated protein kinase, a key regulator of energy metabolism and mitochondrial function in skeletal muscle [6]. This activation promotes mitochondrial biogenesis, oxidative metabolism, and muscle glucose uptake, thereby improving muscle function and insulin sensitivity in T2DM and sarcopenia.

Newer classes of antidiabetic drugs, such as GLP-1 receptor agonists and SGLT2 inhibitors, represent promising therapeutic strategies for sarcopenia in individuals with T2DM [7]. GLP-1 receptor agonists, including liraglutide and semaglutide, exert pleiotropic effects on muscle health by stimulating muscle protein synthesis, inhibiting muscle protein degradation, and improving



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mitochondrial function in skeletal muscle. In addition, they improve glycemic control, reduce body weight, and lower cardiovascular risk factors, which makes them attractive agents for the management of sarcopenia in T2DM [7]. Similarly, SGLT2 inhibitors, such as empagliflozin and dapagliflozin, promote muscle glucose uptake, reduce intramuscular fat deposition, and improve muscle function [7]. Beyond their metabolic effects, SGLT2 inhibitors have cardioprotective properties, reduce the risk of heart failure, and improve exercise tolerance, which further enhances their value in the management of sarcopenia associated with T2DM.

Anabolic agents represent a promising pharmacological approach to stimulating muscle growth and function in sarcopenia [8]. These agents, including selective SARMs, growth hormone secretagogues, and myostatin inhibitors, enhance muscle hypertrophy by acting on key pathways involved in muscle protein synthesis, satellite cell proliferation, and muscle regeneration. SARMs, such as enobosarm and RAD140, selectively bind to androgen receptors in skeletal muscle and bone, leading to increased muscle mass, improved muscle strength, and enhanced physical performance [9].

Dipeptidyl peptidase-4 (DPP-4) inhibitors, which increase the levels of incretin hormones, have favorable effects on muscle metabolism and may help preserve muscle tissue. Insulin therapy remains a cornerstone of diabetes management owing to its potent effect on glycemic control. In addition to regulating blood glucose, insulin markedly stimulates muscle protein synthesis. In a study of Japanese patients, insulin therapy attenuated the decline in lower-limb muscle strength, although no effect was observed in the upper limbs, supporting its clinical use to reduce the risk of sarcopenia in patients with T2DM [10].

The complex interplay between sarcopenia and T2DM underscores the need for comprehensive treatment strategies that address both conditions simultaneously.

The above served as the rationale for the present study.

AIM OF THE STUDY.

To assess the levels of tumor necrosis factor- α (TNF- α) and interleukin-6 (IL-6) in patients with sarcopenia and type 2 diabetes mellitus.

MATERIALS AND METHODS

The study included 35 patients aged 55 to 65 years, of whom 17 were men (mean age 58.3 ± 3.5 years) and 18 were women (mean age 59.6 ± 3.8 years). The control group consisted of 20 individuals with T2DM.

The patients were allocated to two groups: Group 1 comprised patients with T2DM and sarcopenia ($n = 35$), and Group 2 comprised patients with T2DM without sarcopenia ($n = 20$).

The study was conducted at the Department of Diabetology of the Andijan Branch of the Republican Specialized Scientific and Practical Medical Center of Endocrinology, Ministry of Health of the Republic of Uzbekistan.

Inclusion criteria: T2DM, sarcopenia, male and female patients.

Exclusion criteria: patients with severe comorbid diseases were not included in the study.

The study methods comprised general clinical, biochemical (total, direct, and indirect bilirubin, alanine aminotransferase [ALT], aspartate aminotransferase [AST], prothrombin index [PTI], coagulogram, C-reactive protein [CRP], glucose, glycated hemoglobin, and oral glucose tolerance test), hormonal (insulin, C-peptide, tumor necrosis factor- α [TNF- α], and interleukin-6 [IL-6]), anthropometric (height [cm], weight [kg], body mass index [BMI, kg/m^2], waist circumference, and hip circumference), and instrumental examinations (electrocardiography, chest radiography, handgrip dynamometry, dual-energy X-ray absorptiometry [DXA], and a gait speed test).

The diagnosis of sarcopenia was established in three consecutive steps in accordance with the European guidelines (EWGSOP criteria):

1. **Handgrip dynamometry** (squeezing a dynamometer by hand) or the chair stand test (five repetitions without using the arms).
2. **Bioelectrical impedance analysis (BIA)** or dual-energy X-ray absorptiometry (DXA).



3. **Gait speed test** (the time taken to walk 4 meters).

Tests for assessing physical performance (the Short Physical Performance Battery, the gait speed test, the six-minute walk test, and the stair climb test) are used only in clinical research owing to the complexity of their administration and interpretation [8]. In routine practice, the 4-meter gait speed test is used to assess physical performance.

The European consensus on the diagnosis of sarcopenia proposes an algorithm for identifying patients with this condition. It is recommended that patients older than 65 years (or those with risk factors for sarcopenia) be examined. First, gait speed is assessed (the time taken to walk 4 m). If gait speed exceeds 0.8 m/s, handgrip strength is measured with a dynamometer. If the values are normal, assessment of muscle mass is not required. If gait speed is below 0.8 m/s or handgrip strength is inadequate, muscle mass must be assessed (using DXA). The diagnosis is established only after this step.

Comprehensive therapy for patients with T2DM and sarcopenia included optimized glucose-lowering therapy (metformin, SGLT2 inhibitors/GLP-1 receptor agonists), loading doses of vitamin D, a high-protein diet, and an individualized program of aerobic and resistance training aimed at a synergistic reduction of myostatin and restoration of muscle mass.

Microsoft Excel and STATISTICA 6 software were used for statistical analysis, and $p < 0.05$ was considered to indicate a statistically significant difference. Quantitative data with a normal distribution were expressed as the mean and standard deviation ($M \pm SD$).

RESULTS AND DISCUSSION.

The clinical and demographic characteristics of the study groups are presented in Table 1.

Table 1. Clinical and demographic characteristics of the study groups (baseline)

Parameter	Group 1: T2DM + sarcopenia (n = 35)	Group 2: T2DM without sarcopenia (n = 20)	p value
Men, n (%)	17 (48.6%)	10 (50.0%)	> 0.05
Women, n (%)	18 (51.4%)	10 (50.0%)	> 0.05
Age of men (years), $M \pm SD$	58.3 ± 3.5	57.9 ± 3.2	> 0.05
Age of women (years), $M \pm SD$	59.6 ± 3.8	59.1 ± 3.4	> 0.05
T2DM duration (years), Me [Q1; Q3]	6.4 [4.2; 8.8]	5.8 [3.9; 7.9]	> 0.05
BMI (kg/m^2), $M \pm SD$	27.4 ± 2.1	31.2 ± 2.6	< 0.01
Vitamin D (ng/mL), Me [Q1; Q3]	16.2 [12.4; 19.8]	24.5 [21.1; 28.4]	< 0.01

As shown in Table 1, the comparative analysis of baseline clinical and demographic parameters demonstrated that the groups were comparable in terms of sex, age, and duration of T2DM ($p > 0.05$), which confirms the methodological homogeneity of the sample. However, in patients with concomitant sarcopenia (Group 1), BMI values were significantly lower ($27.4 \pm 2.1 \text{ kg}/\text{m}^2$ versus $31.2 \pm 2.6 \text{ kg}/\text{m}^2$ in Group 2; $p < 0.01$), which is explained by a deficit of muscle mass against a background of excess adipose tissue deposition. Furthermore, Group 1 showed a statistically significant, profound vitamin D deficiency (median 16.2 ng/mL), whereas the control group without sarcopenia exhibited only vitamin D insufficiency (24.5 ng/mL ; $p < 0.01$).

Next, we examined the dynamics of metabolic status indicators after 6 months of therapy (Table 2).



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Table 2. Dynamics of metabolic status indicators after 6 months of therapy

Parameter	Group 1 (before)	Group 1 (after)	Group 2 (before)	Group 2 (after)	p ₁ (within Group 1)	p ₂ (between groups, after)
HbA1c (%), M ± SD	8.2 ± 0.7	6.9 ± 0.4	7.9 ± 0.6	7.1 ± 0.5	< 0.001	> 0.05
Fasting glucose (mmol/L)	8.6 ± 0.9	6.4 ± 0.5	8.1 ± 0.8	6.6 ± 0.6	< 0.01	> 0.05
HOMA-IR index, Me [Q1; Q3]	5.4 [4.2; 6.8]	3.1 [2.4; 3.9]	4.8 [3.9; 5.9]	3.4 [2.7; 4.1]	< 0.01	> 0.05
Triglycerides (mmol/L)	2.3 [1.8; 2.9]	1.7 [1.4; 2.1]	2.1 [1.6; 2.6]	1.8 [1.5; 2.2]	< 0.05	> 0.05

As shown in Table 2, after 6 months of comprehensive treatment, a significant and comparable improvement in carbohydrate metabolism was achieved in both groups. The glycated hemoglobin (HbA1c) level in Group 1 decreased from $8.2 \pm 0.7\%$ to the target value of $6.9 \pm 0.4\%$, and fasting glucose fell to 6.4 ± 0.5 mmol/L ($p_1 < 0.001$). The dynamics of the HOMA-IR index deserve particular attention: in Group 1, which received, in addition to glucose-lowering drugs, correction of vitamin D deficiency and personalized physical exercise, insulin resistance decreased more markedly (1.74-fold) than in Group 2 (1.41-fold). This indicates a synergistic effect of physical training and the correction of vitamin D deficiency on the restoration of insulin sensitivity in peripheral tissues. No between-group differences in the lipid profile (triglycerides) were found at the end of the study ($p_2 > 0.05$).

The changes in sarcopenia parameters and muscle function after 6 months of treatment are shown in Table 3.

Table 3. Changes in sarcopenia parameters and muscle function in the study groups after 6 months of treatment

Sarcopenia parameter	Group 1 (before)	Group 1 (after)	Group 2 (before)	Group 2 (after)	p ₁ (within Group 1)	p ₂ (between groups, after)
Handgrip strength (kg) – men	24.2 ± 2.1	29.4 ± 2.5	34.5 ± 3.1	35.1 ± 2.8	< 0.01	< 0.01
Handgrip strength (kg) – women	14.1 ± 1.6	17.8 ± 1.9	21.2 ± 2.3	21.5 ± 2.1	< 0.01	< 0.01
Skeletal muscle index (kg/m ²)	6.2 ± 0.4	6.8 ± 0.5	7.6 ± 0.6	7.5 ± 0.5	< 0.05	< 0.01
Gait speed (m/s), M ± SD	0.72 ± 0.08	0.94 ± 0.11	1.12 ± 0.14	1.14 ± 0.12	< 0.01	< 0.05
Chair stand test (s)	16.4 ± 1.8	12.1 ± 1.4	10.8 ± 1.1	10.5 ± 1.2	< 0.01	< 0.05

As shown in Table 3, the assessment of the functional status of muscle tissue demonstrated the high efficacy of the proposed rehabilitation program in patients with sarcopenia (Group 1). Handgrip strength increased significantly in both men (from 24.2 ± 2.1 to 29.4 ± 2.5 kg) and women (from 14.1



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± 1.6 to 17.8 ± 1.9 kg; $p_1 < 0.01$). The skeletal muscle index (SMI), which reflects muscle mass, increased significantly in Group 1 to 6.8 ± 0.5 kg/m² ($p_1 < 0.05$), whereas in the control group it remained unchanged. Gait speed increased by 30.5%, and the time to complete the chair stand test decreased from 16.4 ± 1.8 to 12.1 ± 1.4 s ($p_1 < 0.01$). Despite the marked improvement in Group 1, the final values remained inferior to those of patients without sarcopenia ($p_2 < 0.05$ and $p_2 < 0.01$), which substantiates the need for longer courses of therapy.

Table 4 presents the dynamics of myokine status and vitamin D levels in the study groups after 6 months of treatment.

Table 4. Dynamics of myokine status and vitamin D levels in the study groups after 6 months of treatment

Parameter, Me [Q1; Q3]	Group 1 (before)	Group 1 (after)	Group 2 (before)	Group 2 (after)	p ₁ (within Group 1)	p ₂ (between groups, after)
Myostatin (ng/mL)	42.8 [36.1; 49.4]	28.4 [22.1; 33.5]	25.1 [20.4; 31.2]	24.8 [19.9; 30.5]	< 0.001	> 0.05
Irisin (μg/mL)	3.1 [2.4; 3.8]	5.2 [4.1; 6.4]	4.8 [3.9; 5.7]	4.9 [4.0; 5.8]	< 0.01	> 0.05
Vitamin D (ng/mL)	16.2 [12.4; 19.8]	34.6 [30.2; 41.5]	24.5 [21.1; 28.4]	25.2 [22.0; 29.1]	< 0.001	< 0.01

As shown in Table 4, the study revealed a pronounced remodeling of the myokine profile in Group 1 patients under the influence of comprehensive treatment. The initially high level of myostatin (a muscle growth inhibitor) in Group 1 (42.8 ng/mL) decreased significantly to 28.4 ng/mL ($p_1 < 0.001$), becoming virtually equal to that of the control group (24.8 ng/mL; $p_2 > 0.05$). At the same time, a 1.67-fold increase in the concentration of the anabolic myokine irisin was recorded, from 3.1 [2.4; 3.8] to 5.2 [4.1; 6.4] μg/mL ($p_1 < 0.01$), reflecting an adequate response of muscle tissue to regular physical activity. The concentration of 25(OH)D in Group 1 rose, as expected, to optimal values (34.6 ng/mL; $p_1 < 0.001$), whereas in Group 2 neither myokine status nor vitamin D level changed over the 6 months of follow-up.

Table 5. Correlation analysis of myostatin level with metabolic and functional parameters in Group 1

Parameter	Correlation coefficient (r) before treatment	p value	Correlation coefficient (r) after treatment	p value
Myostatin and HOMA-IR index	+0.48	< 0.01	+0.31	< 0.05
Myostatin and handgrip strength	-0.54	< 0.01	-0.36	< 0.05
Myostatin and vitamin D	-0.42	< 0.01	-0.24	> 0.05
Myostatin and gait speed	-0.39	< 0.05	-0.21	> 0.05



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As shown in Table 5, the correlation analysis confirmed the pathogenetic involvement of myostatin in the development of metabolic and muscular disorders in the combined pathology. Before treatment, a high myostatin level showed a moderate positive correlation with the severity of insulin resistance ($r = +0.48$; $p < 0.01$) and was inversely related to muscle strength ($r = -0.54$; $p < 0.01$), vitamin D level ($r = -0.42$; $p < 0.01$), and gait speed ($r = -0.39$; $p < 0.05$). After comprehensive therapy and normalization of serum levels of this myokine, a statistical weakening of these pathological associations was observed. The correlations with muscle strength and the HOMA-IR index decreased, while the associations with gait speed and vitamin D level lost statistical significance ($p > 0.05$). The findings indicate that a reduction in the myostatin pool is an important link in the restoration of skeletal muscle function and the improvement of metabolic control in patients with T2DM.

The results obtained demonstrate a close pathogenetic relationship between metabolic disturbances, vitamin D deficiency, and an imbalance of myokine status in patients with type 2 diabetes mellitus (T2DM) and sarcopenia.

The role of myostatin as a metabolic regulator

The key finding of this work was the identification of an initially high myostatin level in patients with sarcopenia and T2DM, which correlated directly with the severity of insulin resistance (HOMA-IR index). Myostatin is traditionally regarded as a negative regulator of skeletal muscle mass. However, current evidence indicates that its excess not only suppresses myogenesis but also blocks insulin signaling in myocytes, thereby aggravating systemic metabolic disturbances. The inverse correlation of myostatin with vitamin D level and with indicators of muscle strength (dynamometry) confirms that vitamin D deficiency is a potent trigger of muscle atrophy, potentiating the expression of catabolic myokines.

Mechanisms of action of comprehensive therapy

The inclusion of personalized physical exercise and targeted correction of vitamin D deficiency in the treatment regimen led to a substantial remodeling of the myokine profile. The 33.6% reduction in myostatin level, together with the parallel 1.67-fold increase in the anabolic myokine irisin, indicates a partial restoration of the regenerative potential of muscle tissue. Irisin, which is secreted by muscle in response to contraction, activates glucose uptake and improves lipid metabolism. It is precisely this synergy between physical activity and the correction of vitamin D deficiency that explains the more pronounced reduction in the HOMA-IR index in the main group compared with glucose-lowering therapy alone.

Clinical significance and prospects

The improvement in functional tests (chair stand test and gait speed) and the increase in the skeletal muscle index confirm the reversibility of sarcopenic processes when pathogenetic treatment is initiated in a timely manner. Nevertheless, the persistent lag of muscle function parameters in the main group behind those of the control group at month 6 indicates that structural and functional recovery of muscle tissue in T2DM proceeds more slowly than the normalization of carbohydrate metabolism. This finding substantiates the need for long-term (more than 6 months) monitoring of myokine status and for the prolongation of rehabilitation programs in this category of patients.

CONCLUSIONS.

1. During 6 months of comprehensive treatment, patients with sarcopenia showed a statistically significant remodeling of the myokine profile: a 33.6% decrease in myostatin, a suppressor of muscle mass, and a compensatory 1.67-fold increase in the anabolic myokine irisin. These changes were accompanied by a significant increase in the skeletal muscle index and improvement in functional tests (dynamometry and gait speed).

2. Despite the highly significant positive changes in all parameters in the comprehensive therapy group, after 6 months of treatment the indicators of muscle strength and mass in patients with initial sarcopenia did not reach the values of the control group without sarcopenia ($p_2 < 0.05$), which



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pathogenetically substantiates the need to prolong physical rehabilitation programs and nutritional and metabolic support in this category of patients.

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