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Original Article

Morphometric and structural changes in the rectus abdominis muscle of rats under conditions of experimental obesity and diabetes mellitus

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Abstract

The modern metabolic paradigm considers skeletal muscles not only as an organ of locomotion but as a key metabolic unit responsible for the regulation of glucose homeostasis in the body. The aim of the work is to establish the morphofunctional features of changes in the rectus abdominis muscle of rats under conditions of 8-month modeling of alimentary obesity caused by a high-fat diet and subsequent induction of diabetes mellitus. The study was conducted on 30 sexually mature Wistar rats, divided into three groups of 10 individuals: a control group, a group with a high-fat diet, and a group with a diet and induced diabetes mellitus. Histological examination of the muscles of rats in the high-fat diet and diet with diabetes mellitus groups revealed pronounced signs of diabetic myopathy. In the alimentary obesity group, initial changes were observed in the form of unevenness in the caliber of muscle fibers and signs of intracellular lipid accumulation. In the diet and diabetes group, structural disorders became degenerative in nature, areas with pronounced myocyte atrophy, areas of fiber architectonics disturbance, sarcolemma fragmentation, and focal infiltration were visualized. The nuclear apparatus of myocytes in these groups showed signs of chromatin condensation and pyknosis, which indicates the activation of destructive processes. Additionally, a significant decrease in the density of the capillary network in the endomysium was recorded, which was accompanied by signs of endothelial dysfunction, creating conditions for chronic local energy deficiency of the tissue. The study allowed us to establish the complex nature of structural and functional changes in the rectus abdominis muscle under conditions of metabolic stress induced by a combination of a high-fat diet and type 2 diabetes.

Keywords: diabetic myopathies, obesity, muscle, skeletal, insulin resistance, muscular atrophy

Introduction

Today, the problem of metabolic disorders, in particular the combination of obesity and type 2 diabetes mellitus (T2DM), remains one of the most discussed topics in world endocrinology and pathophysiology [1, 2]. According to current data, the prevalence of these pathologies has become a pandemic, which causes increased interest among researchers in the mechanisms of the formation of the so-called diabetic heart and other organ complications against the background of insulin resistance [3, 4].

A fundamental feature of modern pathogenesis is the long latent period of disease development, caused

by dietary factors. The transition from normal metabolism to the stage of decompensation during the consumption of a high-fat diet (HFD) occurs gradually, which often complicates timely diagnosis [5]. In contrast to short-term experimental models, long-term observation allows us to recreate the chronic course of metabolic stress, which is as close as possible to pathophysiological processes in the human body.

A critical point in the study of this pathology is the synergism of obesity and induced hyperglycemia [6]. Obesity, which develops due to excessive lipid consumption, forms the basis on which further induction of diabetes with streptozotocin (or its analogues) leads to a more aggressive clinical course. In recent years, the scientific literature has been actively discussing the question of whether structural changes in tissues at the stage of obesity are still reversible or not [7].

The use of an experimental model allows us to clearly distinguish the effects of pure obesity from obesity complicated by severe hyperglycemia. This is fundamentally important for understanding which molecular mechanisms play a leading role at each stage. Most existing works focus either on metabolic parameters or on morphological changes [8, 9], while an integrated approach over a long time period allows us to obtain a more holistic picture.

The modern metabolic paradigm considers skeletal muscle not only as an organ of locomotion, but also as a key metabolic unit responsible for the regulation of glucose homeostasis in the body [10]. Under metabolic stress, HFD and diabetes, muscle tissue undergoes profound structural and functional transformations, which becomes a critical factor in the progression of insulin resistance [11, 12].

Skeletal muscle consumes up to 80% of insulin-dependent glucose in the postprandial period. Chronic lipid overload leads to the accumulation of intracellular lipid metabolites in myocytes. This process, known as ectopic fat deposition, directly blocks the insulin signaling pathway, which gradually reduces the translocation of GLUT4 transporters to the cell membrane [13, 14].

Researchers indicate that prolonged hyperglycemia on the background of HFD provokes not only a decrease in insulin sensitivity, but also atrophy of muscle fibers, especially type II [12, 14]. Changes in protein metabolism and activation of proteolytic pathways lead to a decrease in muscle mass and a decrease in muscle strength, which significantly worsens the quality of life of patients and complicates glycemic control.

Thus, the study of pathophysiological changes in muscle tissue under chronic exposure to HFD and diabetes has not only theoretical significance for basic science, but also practical value for clinical medicine, as it allows to substantiate new approaches to the correction of metabolic disorders through the preservation of the functional state of skeletal muscles. The choice of the rectus abdominis muscle as an object of study is due to its high metabolic activity and sensitivity to systemic disorders of carbohydrate and lipid metabolism. In addition, the analysis of this particular muscle allows us to assess the features of the course of diabetic myopathy in tissues prone to the accumulation of visceral fat.

The aim of the work is to establish the morphofunctional features of changes in the rectus abdominis muscle of rats under conditions of 8-month modeling of alimentary obesity caused by a high-fat diet and subsequent induction of diabetes mellitus.

Material and methods

Research on laboratory animals was conducted in accordance with the provisions of the European Convention for the Protection of Vertebrate Animals used for Experimental and other Scientific Purposes of 18.03.1986, the Council of Europe Directive 2010/63/EU, and the Law of Ukraine "On the Protection of Animals from Cruelty to Animals". A total of 30 sexually mature Wistar rats were kept in an accredited vivarium at the I. Horbachevsky Ternopil National Medical University.

The experimental animals were divided into 3 groups of 10 individuals. The age of the rats was 8 months. The control group (CG) included rats with normal weight (220.00±15.00) g, which did not model any pathology. In the second, obese group (HFD) and the third, obese + diabetes group (HFD+DM), starting from the first month after birth, they were fed a high-calorie diet. In the total amount of calories, from 30% to 60% were from animal fats (lard), bread was also added to the daily diet – 6.0 g, pearl barley – 4 g, barley – 15 g, carrots – 10 g per individual. The lard was crushed, and sunflower seeds, peanuts, and cake were added to improve palatability and attract animals (Figure 1). The diet was prepared in accordance with the author's certificate for the work [15]. The weight of animals in these groups did not differ statistically between themselves and was (427.30±25.16) g and (429.62±23.45) g, but was statistically significantly different from the CG ($p<0.001$). The general scheme of the experiment is shown in Figure 2.

At the 7th month of the experiment, type 2 diabetes mellitus was induced in the animals of the third group,

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Figure 1: Body habitus differences between control and HFD-fed rats.

which had been on a high-fat diet for 6 months. Modeling was performed by a single intraperitoneal injection of a streptozotocin solution at a dose of 35 mg/kg of body weight. Before the drug administration, the animals were fasted for 12 hours with free access to water. The development of diabetes mellitus was confirmed by measuring blood glucose levels from the tail vein using a glucometer and verifying the presence of persistent hyperglycemia. The animals continued to receive a high-fat diet for another month (until the end of the 8th month of the study).

At the end of the experiment, the rectus abdominis muscle was harvested from the right side and fixed in a 10% neutral formalin solution. After standard dehydration in ascending alcohol concentrations, the tissue was embedded in paraffin. Histological sections (5–7 µm thick) were prepared using a microtome. Following deparaffinization, the sections were stained with hematoxylin and eosin, as well as according to the Masson and Heidenhain protocols, for further light-optical analysis. For electron microscopic studies, small pieces of the rectus muscle were fixed in a 2.5% glutaraldehyde solution (SPI Supplies, USA) prepared in Millonig's phosphate buffer (pH 7.2–7.4) and processed according to standard protocols.

A morphometric study of the muscle tissue was performed on histological sections using image analysis software. To characterize trophic processes, the following parameters were evaluated: average muscle fiber diameter (µm) and nuclear area (µm^2). The degree of degenerative changes was determined by the relative volume of damaged muscle fibers (%). The state of the microcirculatory bed was assessed by calculating capillary density (the number of vessels per unit area of muscle), which allowed for an analysis of tissue vascularization levels under the experimental conditions.

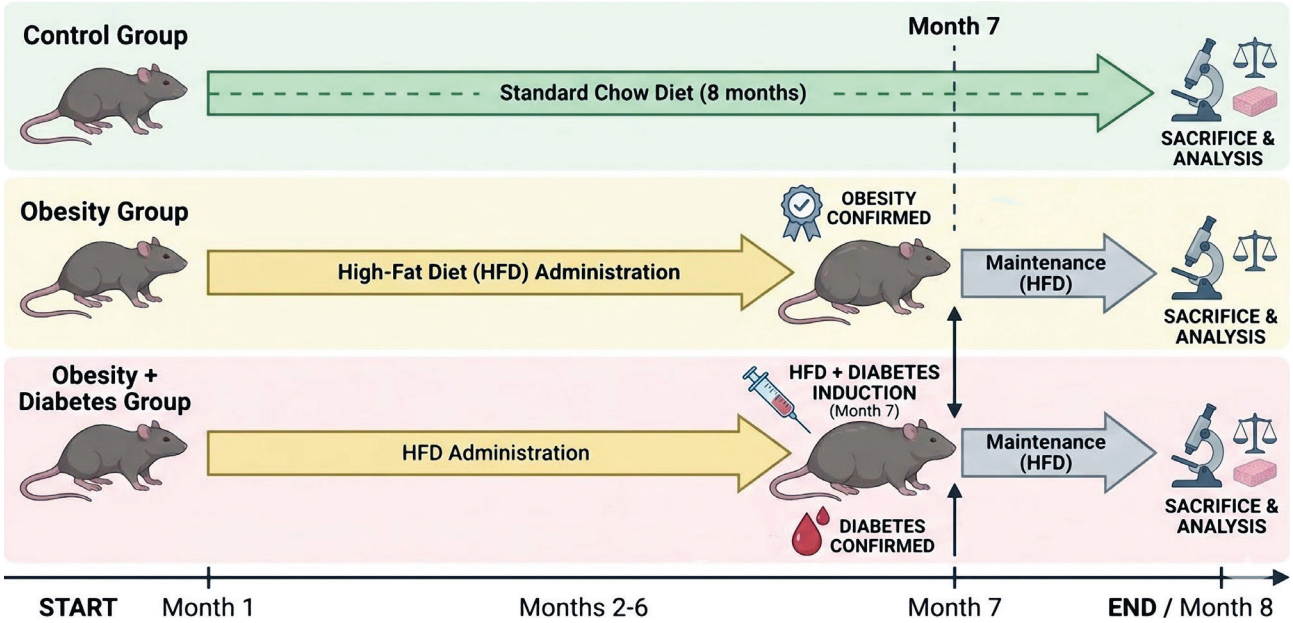

Figure 2: Schematic representation of the experimental study design. The study included three groups of rats: the Control Group (standard chow diet), the Obesity Group (high-fat diet, HFD), and the Obesity + Diabetes Group (HFD combined with diabetes induction at month 7).

Statistical analysis was performed using STATISTICA 10.0 software (StatSoft, USA). The normality of the sample distribution was verified using the Shapiro-Wilk test. Intergroup differences were assessed using one-way analysis of variance (ANOVA), followed by Tukey's post hoc test. For data confirming a normal distribution, intergroup differences were evaluated using the parametric Student's t-test for independent samples. The nonparametric Mann-Whitney U test was used for data that did not conform to a normal distribution. Results are presented as the arithmetic mean±standard deviation (M±SD). Correlations between morphometric indicators and glycemia levels were assessed using the Pearson correlation coefficient (r) for normally distributed data. Differences were considered statistically significant at $p<0.05$.

Results

Analysis of the obtained glycemia data indicates pronounced metabolic disorders in animals of the experimental groups compared to the control group. A long-term high-fat diet was shown to lead to a significant increase in blood glucose levels, even at the stage of obesity modeling. The blood glucose concentration in the control group was (4.45±0.36) mmol/l. In the HFD group, this indicator was significantly higher (7.60±0.59) mmol/l, whereas in the HFD+DM group, the glycemia level reached (11.80±1.19) mmol/l. The results of the statistical analysis, presented in Table 1, confirm the significance of these differences between all groups ($p<0.001$).

As the data indicate, the most pronounced hyperglycemia was observed in the HFD+DM group. Glycemia levels in this group significantly exceeded those in both the control group 7.36 mmol/l and the isolated obesity group by 4.21 mmol/l. The administration of streptozotocin at the 7th month of the experiment led to the decompensation of carbohydrate metabolism, confirming the efficacy of this type 2 diabetes mellitus model against a background of alimentary obesity.

Histological examination of the rectus abdominis muscle revealed a clear progression of pathomorphological changes. In the control group, the architectonics of the muscle tissue were preserved, the fibers exhibited a regular parallel orientation and were tightly packed, forming the characteristic structure of skeletal muscle (Figure 3). The muscle fiber nuclei were oval, with distinct contours, and located primarily beneath the sarcolemma, consistent with healthy myocytes. Interfibrous spaces were minimal, with no signs of edema, fatty infiltration, or inflammatory cell infiltration.

In the HFD group, against the background of induced obesity, the initial signs of metabolic dystrophy were observed. Morphologically, this manifested as focal myosteatosis – the accumulation of lipid droplets in the interfiber spaces – which indicates an early stage of lipotoxic myocyte damage (Figure 4). The space between fibers appeared expanded, suggesting the presence of interstitial edema, a characteristic feature of muscle tissue injury. While there were no pronounced foci of complete fiber necrosis with nuclear disintegration, the observed patterns are indicative of early degenerative changes.

The most pronounced destructive changes were observed in the HFD+DM group. This group exhibited massive interstitial myosteatosis, accompanied by significant muscle fiber deformation and severe microcirculatory disorders, particularly vascular congestion with erythrocytes (Figure 5). Some muscle fibers appeared fragmented or exhibited a loss of their regular parallel orientation, likely resulting from the mechanical pressure exerted by accumulated adipose tissue.

In the HFD+DM group, signs of progressive dystrophic-destructive changes in muscle fibers were also detected. These were characterized by pronounced myocyte atrophy, manifested as significant heterogeneity in fiber diameter and the appearance of centrally located nuclei, indicating deep degenerative processes within the muscle tissue architecture (Figure 6). In several fibers, the migration of nuclei from the periphery toward the center was observed – a classic histological marker of regenerative-degenerative processes

Table 1: Blood glucose levels and post-hoc comparison between experimental groups.

Comparison groups	Mean difference (mmol/L)	t-value	p-value
Control *vs.* HFD	- 3.15	- 8.86	<0.001
Control *vs.* HFD+DM	- 7.36	- 20.7	<0.001
HFD *vs.* HFD+DM	- 4.21	- 11.8	<0.001

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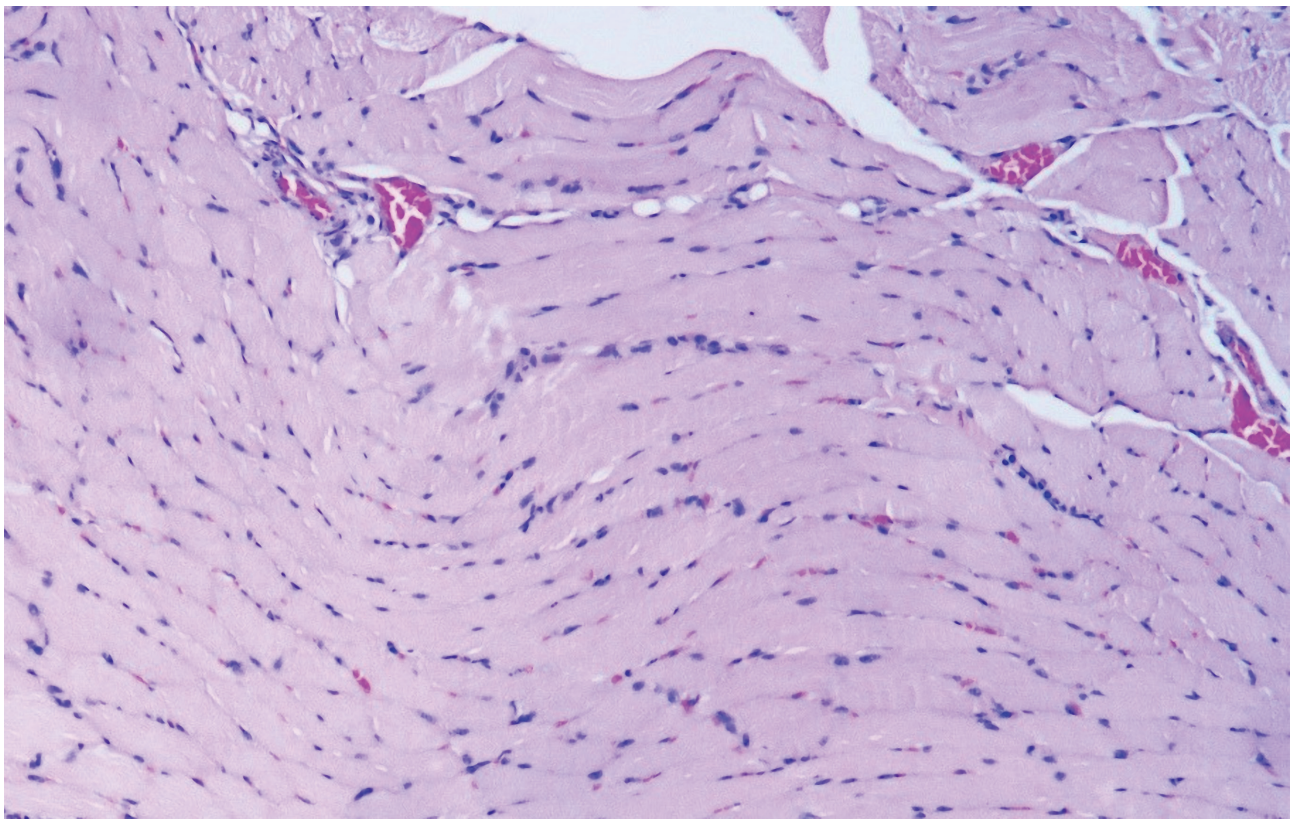

Figure 3: Histological structure of the rectus abdominis muscle of the control group. Hematoxylin and eosin staining. Magnification: × 100.

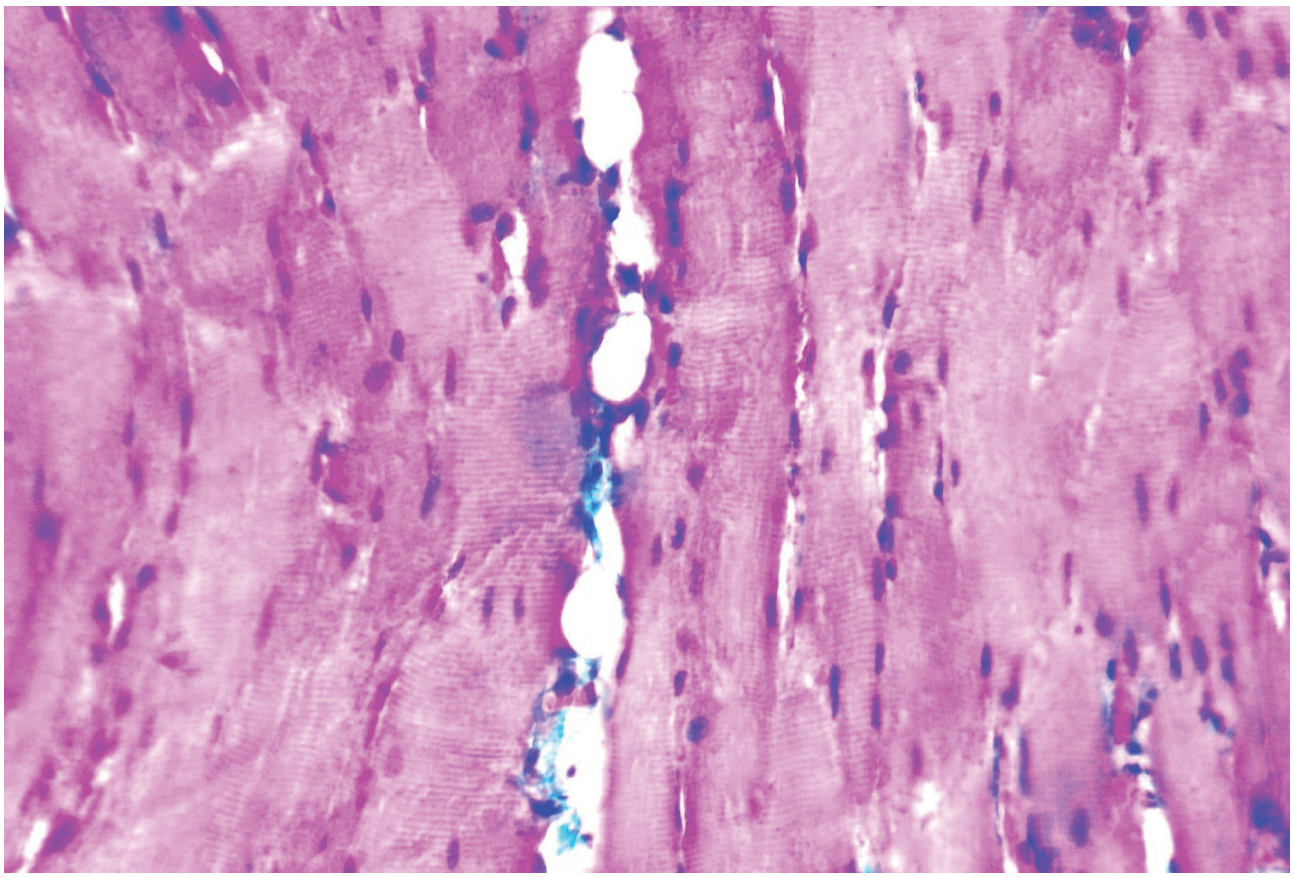

Figure 4: Myosteatosis in the rectus abdominis muscle of HFD group rats. Masson's trichrome stain. Magnification: × 400.

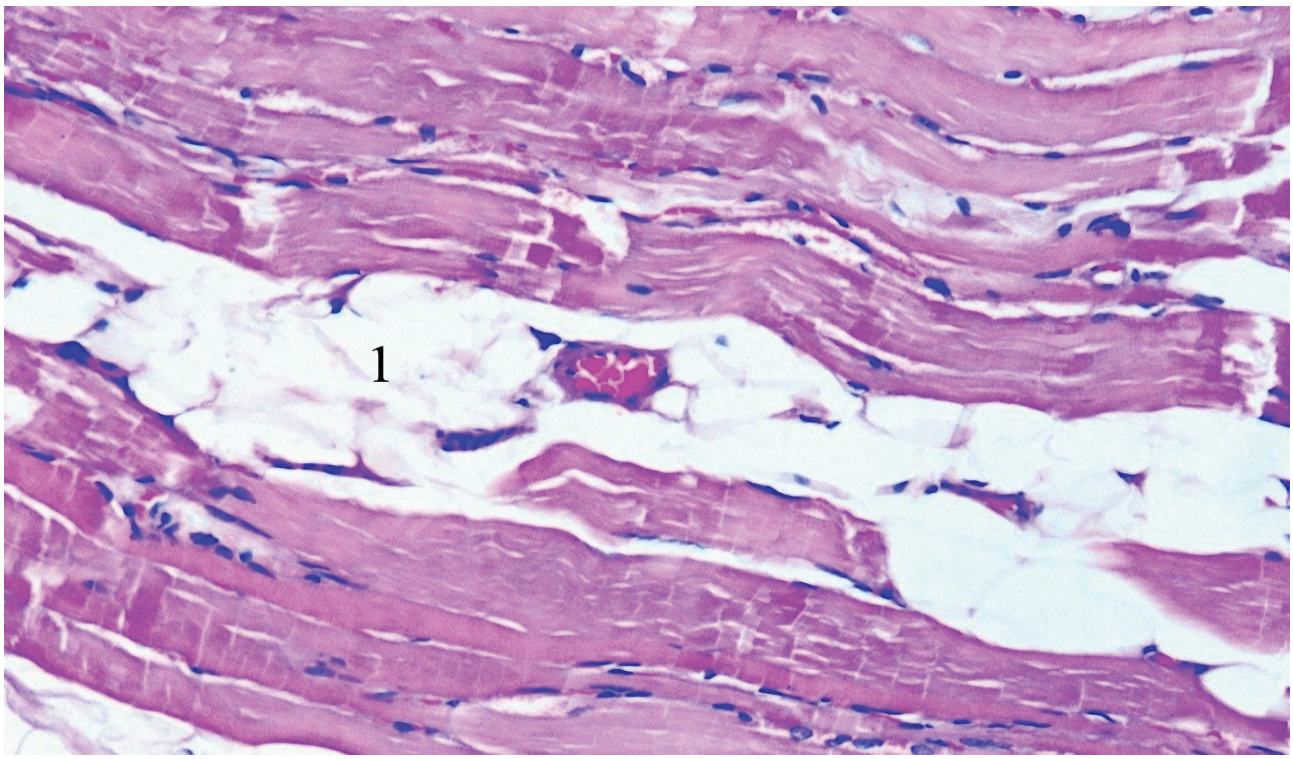

Figure 5: Pronounced interstitial myosteatosis and microcirculatory disorders in the HFD+DM group. Note the accumulation of adipose tissue (1). Hematoxylin and eosin staining. Magnification: × 200.

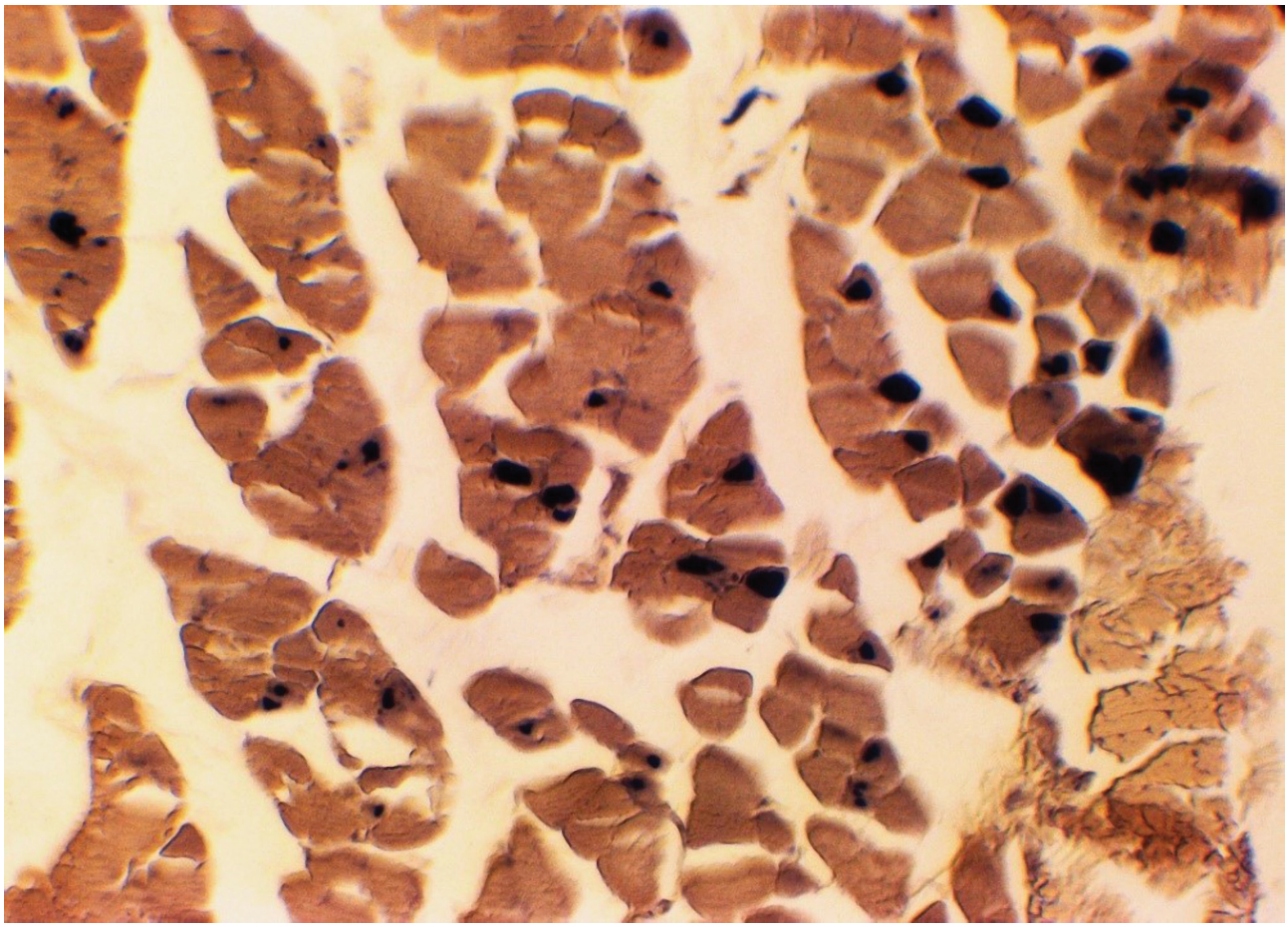

Figure 6: Signs of atrophy and degeneration of muscle fibers in the HFD+DM group. Heidenhain's hematoxylin stain. Magnification: ×200.

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occurring during chronic muscle damage. Additionally, dark areas within the fibers indicated the presence of dystrophic changes, which are frequently observed under conditions of metabolic stress.

The cumulative effect of glucose and lipotoxicity in the HFD+DM group leads to significantly more profound structural damage in the rectus abdominis muscle compared to the isolated obesity group. These morphological data, combined with indicators of severe hyperglycemia, suggest the development of decompensated carbohydrate and lipid metabolism, which serves as a key pathophysiological mechanism for muscle damage under experimental diabetes mellitus.

Electron microscopic examination of the rectus abdominis muscle in the HFD+DM group revealed profound ultrastructural changes (Figure 7). Evidence of severe mitochondrial dysfunction was established, characterized by the destruction of internal cristae and uneven density of the mitochondrial matrix. Concurrently, significant intracellular accumulation of lipid droplets was observed, physically disorganizing the structure of sarcomeres and myofibrils. These findings confirm the structural disorganization of myocytes at the organelle level, which correlates with the severity of hyperglycemia and indicates the progression of irreversible degenerative processes in the muscle tissue.

Morphometric analysis of the rectus abdominis muscle revealed clear dynamics in the average diameter of muscle fibers (Figure 8). In the control group, the average fiber diameter was (55.8±2.24) μm. In the HFD group, a statistically significant decrease in fiber diameter to (53.0±1.70) μm was observed ($p=0.004$ *vs.* CG), indicating the onset of muscle atrophy at the stage of metabolic disorders induced by the high-fat diet. The most pronounced degenerative changes were recorded in the HFD+DM group, where the average fiber diameter decreased significantly to (41.8±1.31) μm ($p<0.001$ *vs.* both CG and HFD).

The diagram clearly demonstrates the high homogeneity of indicators within each group, as well as a pronounced shift in the distribution of values toward smaller diameters in animals with diabetes mellitus.

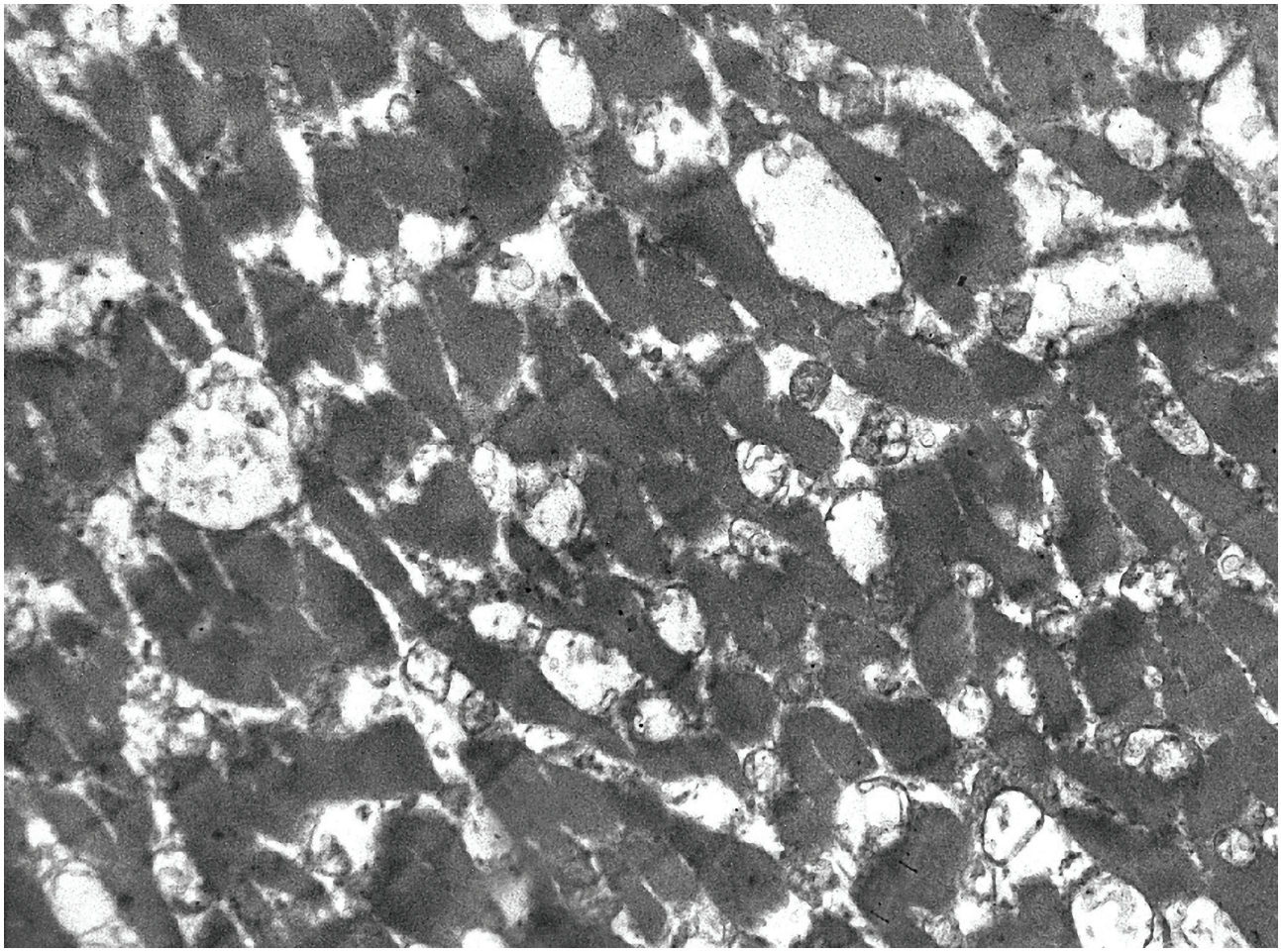

Figure 7: Ultrastructural changes in the rectus abdominis muscle of the HFD+DM group. Note the marked mitochondrial dysfunction (cristalolysis, matrix swelling) and massive intrasarcoplasmic accumulation of lipid inclusions, leading to myofibril fragmentation. Transmission electron microscopy, magnification: × 12 000

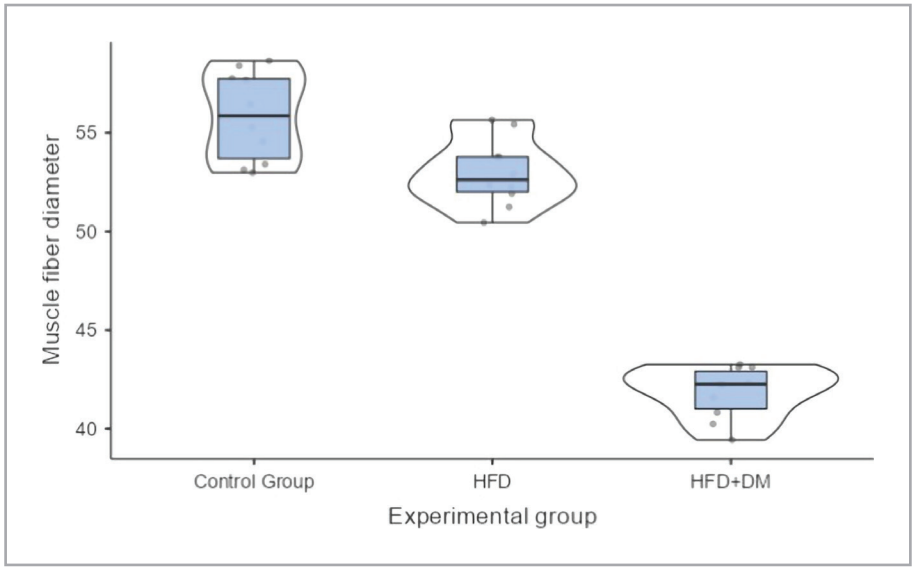

Figure 8: Dynamics of the average rectus abdominis muscle fiber diameter across the experimental groups.

These results confirm the progressive nature of the pathological process, wherein the synergy of insulin resistance and hyperglycemia leads to profound atrophy of muscle fibers.

Histological analysis of the rectus abdominis muscle revealed a progressive increase in the proportion of damaged fibers in response to metabolic disorders. In the control group, the relative volume of damaged fibers was minimal at (1.38±0.33)%, corresponding to the baseline level of physiological tissue turnover. In the HFD group, a statistically significant increase in this indicator was observed (6.34±0.69)%, whereas in the HFD+DM group, the level of damage reached critical values of (17.7±1.71)% ($p<0.001$ *vs*. control).

Regression analysis demonstrated a strong positive correlation between glycemia levels and the percentage of damaged muscle fibers ($r=0.978$; $p<0.001$; $df=28$) (Figure 9). These data confirm that hyperglycemia is a critical factor driving destructive changes in myofibrils. The visualization of the Pearson correlation analysis clearly demonstrates a close relationship between blood glucose levels and the extent of morphological tissue damage, highlighting the pathogenic role of metabolic stress in the development of atrophic processes in skeletal muscles.

Morphometric analysis of the nuclear area revealed minor variations in mean values across the experimental groups (63.1±0.60) μm² in the CG, (63.9±0.78) μm² in the HFD group, and (60.1±1.19) μm² in the HFD+DM group. Despite the relative stability of this parameter, Pearson correlation analysis revealed a moderate negative relationship between blood glucose levels and nuclear area ($r=-0.646$; $p<0.001$; $df=28$). This indicates that increasing blood glucose levels are associated with a tendency toward a decrease in the nuclear area of muscle fibers. These data support the hypothesis that chronic hyperglycemia is linked to degenerative changes in the cellular nuclear apparatus, which may be attributed to chromatin condensation or other pathological mechanisms (Figure 10).

Analysis of capillary density revealed a progressive decline in microcirculatory intensity under conditions of metabolic disorders. In the control group, the average capillary density was (1732±93.4) cap/mm². In the HFD group, a decrease in this parameter to (1680±60.0) cap/mm² was observed a 3.1% reduction compared to control ($p>0.05$). The most pronounced changes were recorded in the HFD+DM group, where capillary density decreased to (1490±70.0) cap/mm², representing a 14.1% reduction compared to the control group ($p<0.001$). A graphic representation of these changes is shown in Figure 11. These data indicate that chronic hyperglycemia and obesity-related metabolic stress significantly impair muscle tissue vascularization, likely contributing to the observed myofibril atrophy and degenerative changes.

Discussion

Our results indicate that the combination of a high-fat diet and streptozotocin-induced diabetes leads to the development of pronounced morphofunctional changes in the rectus abdominis muscle. These changes

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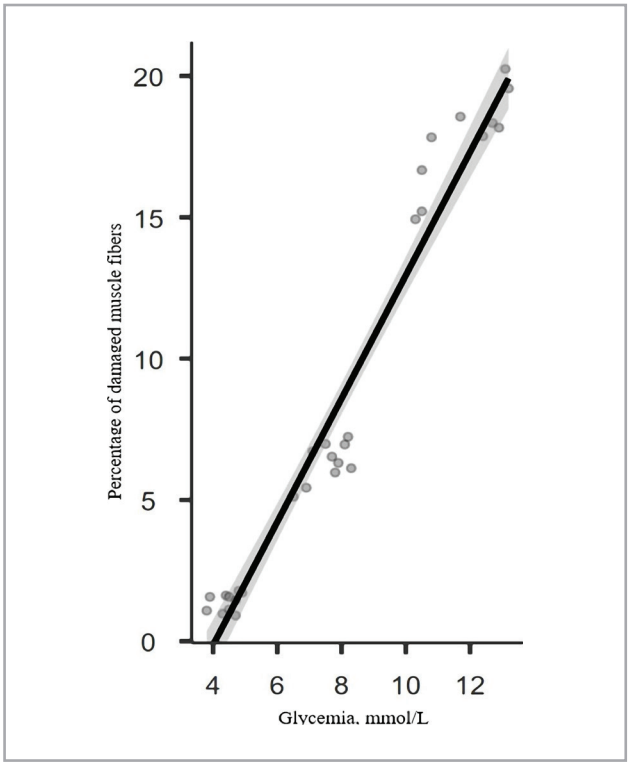

Figure 9: Correlation between blood glycemia levels and the percentage of damaged muscle fibers.

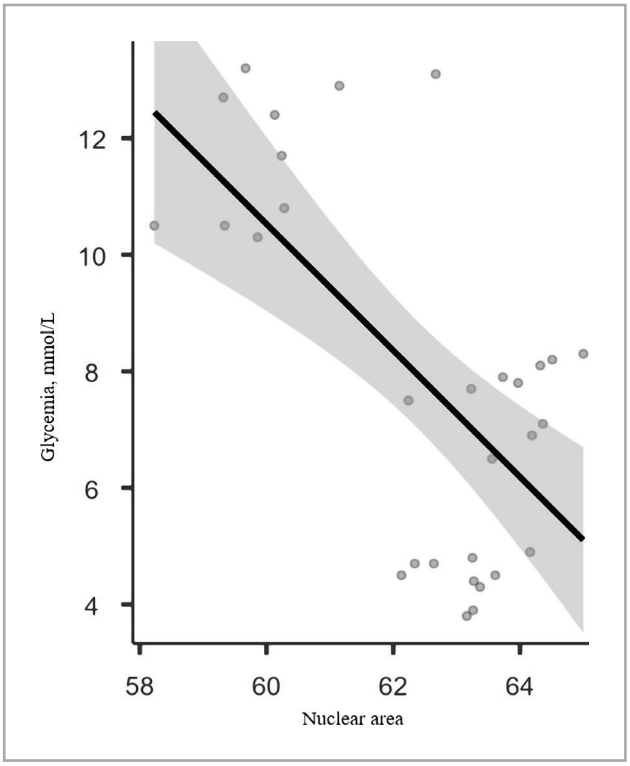

Figure 10: Correlation between blood glycemia levels and the nuclear area

are manifested by fiber atrophy, a decrease in capillary bed density, and degenerative alterations in the nuclear apparatus of the cells.

Our data on the reduction in muscle fiber diameter in the HFD+DM group (41.8±1.31) μm compared to the control group (55.8±2.24) μm are consistent with the findings of other authors, who link insulin resistance with impaired protein synthesis in myocytes [15, 16]. In particular, it is known that chronic hyperglycemia activates the ubiquitin-proteasome degradation system, which is a key mechanism of muscle atrophy. It is worth noting that even isolated exposure to a high-fat diet causes a significant reduction in fiber diameter (53.0±1.70) μm, indicating that lipotoxicity acts as an independent factor of damage even prior to the onset of overt hyperglycemia.

The significant destruction of muscle tissue observed in the HFD+DM group, as evidenced by the high relative volume of damaged muscle fibers, is primarily attributed to the absence of pharmacological intervention or insulin therapy, which is characteristic of this experimental model. In a state of persistent, uncompensated hyperglycemia, degenerative processes within myocytes proceed unchecked, leading to a critical accumulation of damaged fibers.

The morphometric analysis is strongly supported by the identified pathomorphological changes. The transition from focal myosteatosis, characteristic of isolated obesity, to massive interstitial lipid accumulation and pronounced myocyte atrophy signifies deep metabolic decompensation. The detected ultrastructural disorganization warrants special attention, mitochondrial cristalolysis and myofibril fragmentation indicate that under conditions of untreated diabetes mellitus, energy deficiency and lipotoxicity trigger irreversible degenerative processes.

Furthermore, the presence of centrally located nuclei and significant fiber diameter heterogeneity serve as classic markers of chronic damage, which, in the absence of insulin therapy and dietary intervention, leads to the depletion of the regenerative potential of skeletal muscle. Collectively, these morphological findings confirm the development of diabetic myopathy in the HFD+DM group.

The results of the capillary density analysis warrant special attention. We observed a progressive decline in this parameter from (1732±93.4) cap/mm^2 in the control group to (1496±73.2) cap/mm^2 in the HFD+DM group. This reduction in vascular density creates conditions conducive to chronic muscle tissue hypoxia. Similar studies have demonstrated that decreased capillary density correlates with lower levels of vascular endothelial growth factor (VEGF), which accounts for impaired tissue perfusion [17]. In our study, we observed

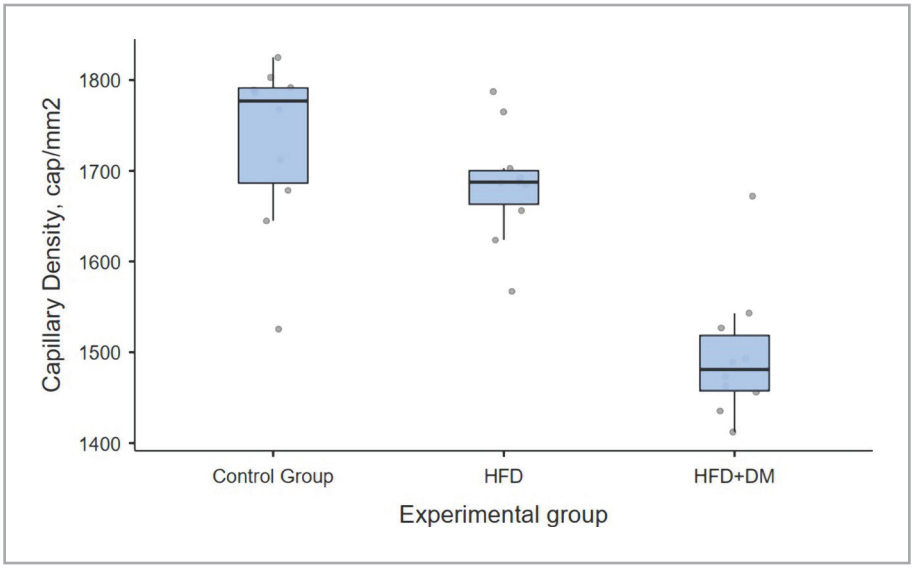

Figure 11: Dynamics of changes in capillary density in the rectus abdominis muscle across experimental groups.

that microcirculatory alterations are most pronounced under conditions of combined metabolic stress, confirming the synergistic impact of obesity and diabetes mellitus on vascular integrity.

A compelling aspect of our research is the negative correlation identified between blood glucose levels and nuclear area ($r=-0.646$; $p<0.001$). We hypothesize that the reduction in nuclear area observed amidst hyperglycemia reflects chromatin condensation, a morphological marker of impaired transcriptional activity. Similar alterations in the nuclear apparatus have been previously documented in studies of diabetic cardiomyopathy, however, this mechanism remains under-investigated in the skeletal muscles of the anterior abdominal wall. Consequently, further clarification via immunohistochemical methods, particularly focusing on the assessment of apoptosis markers, is warranted to elucidate the precise nature of these degenerative changes.

When comparing our data with the findings of other researchers, it becomes evident that the pathogenesis of diabetic myopathy is multifaceted. The combination of muscle fiber atrophy and a decline in capillary density creates a self-perpetuating cycle of tissue hypoxia and metabolic dysfunction, which further accelerates the loss of muscle mass [18]. The strong correlation between glycemia levels and the percentage of damaged fibers ($r=0.978$; $p<0.001$) convincingly suggests that chronic hyperglycemia acts as the primary trigger for the destructive changes observed in this experimental model.

A limitation of our study is the exclusive focus on morphometric indices. We identify further research prospects in evaluating the state of the muscle mitochondrial apparatus, as mitochondrial dysfunction serves as the cornerstone for the development of insulin resistance in skeletal muscle. Consequently, our findings support the hypothesis that metabolic stress, induced by a high-fat diet and diabetes mellitus, triggers profound structural remodeling that significantly diminishes the adaptive potential of skeletal muscle.

Conclusion

The combined effect of a high-fat diet (HFD) and streptozotocin-induced diabetes mellitus leads to critical structural destruction of the rectus abdominis muscle. This is manifested by an increase in the relative volume of damaged fibers to (17.7±1.71)%, which is approximately 12.8 times higher than that of the control group. The morphological substrate of muscle tissue damage in this combined pathology is characterized by massive interstitial myosteatosis and pronounced myocyte atrophy, accompanied by significant fiber diameter heterogeneity and the displacement of nuclei to the center of the sarcoplasm. At the ultrastructural level, uncompensated diabetes induces profound mitochondrial dysfunction specifically, cristalolysis and matrix edema alongside intrasarcoplasmic lipid accumulation. These changes culminate in myofibril fragmentation and the physical disorganization of sarcomeres.The identified pathomorphological alterations indicate a synergistic effect of glucotoxicity and lipotoxicity. In the absence of pharmacological intervention, this metabolic synergy leads to the depletion of the regenerative

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potential of the rectus abdominis muscle and the progression of irreversible degenerative processes.

Conflict of interest

The authors declare no conflict of interest.

References

1. Nakamura K, Miyoshi T, Yoshida M, Akagi S, Saito Y, Ejiri K, Matsuo N, Ichikawa K, Iwasaki K, Naito T, Namba Y, Yoshida M, Sugiyama H, Ito H. Pathophysiology and Treatment of Diabetic Cardiomyopathy and Heart Failure in Patients with Diabetes Mellitus. Int J Mol Sci. 2022 Mar 25;23(7):3587. doi: 10.3390/ijms23073587.
2. Kritsak M, Konovalenko S, Stechyshyn I, Pavliuk B, Gargula T, Shatskyi V. Assessment of Adipose Tissue Hormone Levels After Sleeve Gastrectomy in Rats With Experimental Metabolic Syndrome. RJDNMD 2023, 30(3), 330-335.
3. Ritchie RH, Abel ED. Basic Mechanisms of Diabetic Heart Disease. Circ Res. 2020 May 22;126(11):1501-1525. doi: 10.1161/CIRCRESAHA.120.315913.
4. Xie SY, Liu SQ, Zhang T, Shi WK, Xing Y, Fang WX, Zhang M, Chen MY, Xu SC, Fan MQ, Li LL, Zhang H, Zhao N, Zeng ZX, Chen S, Zeng XF, Deng W, Tang QZ. USP28 Serves as a Key Suppressor of Mitochondrial Morphofunctional Defects and Cardiac Dysfunction in the Diabetic Heart. Circulation. 2024 Feb 27;149(9):684-706. doi: 10.1161/CIRCULATIONAHA.123.065603.
5. Tilg H, Petta S, Stefan N, Targher G. Metabolic Dysfunction-Associated Steatotic Liver Disease in Adults: A Review. JAMA. 2026 Jan 13;335(2):163-174. doi: 10.1001/jama.2025.19615. PMID: 41212550.
6. Rabbani N, Thornalley PJ. Glyoxalase 1 Modulation in Obesity and Diabetes. Antioxid Redox Signal. 2019 Jan 20;30(3):354-374. doi: 10.1089/ars.2017.7424.
7. Khajuria DK, Soliman M, Elfar JC, Lewis GS, Abraham T, Kamal F, Elbarbary RA. Aberrant structure of fibrillar collagen and elevated levels of advanced glycation end products typify delayed fracture healing in the diet-induced obesity mouse model. Bone. 2020 Aug; 137:115436. doi: 10.1016/j.bone.2020.115436.
8. Dutta S, Singhal AK, Suryan V, Chandra NC. Obesity: An Impact with Cardiovascular and Cerebrovascular Diseases. Indian J Clin Biochem. 2024 Apr;39(2):168-178. doi: 10.1007/s12291-023-01157-w
9. Tukhovskaya EA, Shaykhutdinova ER, Pakhomova IA, Slashcheva GA, Goryacheva NA, Sadovnikova ES, Rasskazova EA, Kazakov VA, Dyachenko IA, Frolova AA, Brovkin AN, Kaluzhsky VE, Beburov MY, Murashev AN. AICAR Improves Outcomes of Metabolic Syndrome and Type 2 Diabetes Induced by High-Fat Diet in C57Bl/6 Male Mice. Int J Mol Sci. 2022 Dec 11;23(24):15719. doi: 10.3390/ijms232415719.
10. Guo A, Li K, Tian HC, Fan Z, Chen QN, Yang YF, Yu J, Wu YX, Xiao Q. FGF19 protects skeletal muscle against obesity-induced muscle atrophy, metabolic derangement and abnormal irisin levels via the AMPK/SIRT-1/PGC-α pathway. J Cell Mol Med. 2021 Apr;25(7):3585-3600. doi: 10.1111/jcmm.16448.
11. Izzo A, Massimino E, Riccardi G, Della Pepa G. A Narrative Review on Sarcopenia in Type 2 Diabetes Mellitus: Prevalence and Associated Factors. Nutrients. 2021 Jan 9;13(1):183. doi: 10.3390/nu13010183.
12. Espino-Gonzalez E, Dalbram E, Mounier R, Gondin J, Farup J, Jessen N, Treebak JT. Impaired skeletal muscle regeneration in diabetes: From cellular and molecular mechanisms to novel treatments. Cell Metab. 2024 Jun 4;36(6):1204-1236. doi: 10.1016/j.cmet.2024.02.014.
13. Lopez-Pedrosa JM, Camprubi-Robles M, Guzman-Rolo G, Lopez-Gonzalez A, Garcia-Almeida JM, Sanz-Paris A, Rueda R. The Vicious Cycle of Type 2 Diabetes Mellitus and Skeletal Muscle Atrophy: Clinical, Biochemical, and Nutritional Bases. Nutrients. 2024 Jan 4;16(1):172. doi: 10.3390/nu16010172.
14. Mesinovic J, Fyfe JJ, Talevski J, Wheeler MJ, Leung GKW, George ES, Hunegnaw MT, Glavas C, Jansons P, Daly RM, Scott D. Type 2 Diabetes Mellitus and Sarcopenia as Comorbid Chronic Diseases in Older Adults: Established and Emerging Treatments and Therapies. Diabetes Metab J. 2023 Nov;47(6):719-742. doi: 10.4093/dmj.2023.0112.
15. Ida S, Kaneko R, Imataka K, Okubo K, Shirakura Y, Azuma K, Fujiwara R, Murata K. Effects of Antidiabetic Drugs on Muscle Mass in Type 2 Diabetes Mellitus. Curr Diabetes Rev. 2021;17(3):293-303. doi: 10.2174/1573399816666200705210006.
16. Lopez-Pedrosa JM, Camprubi-Robles M, Guzman-Rolo G, Lopez-Gonzalez A, Garcia-Almeida JM, Sanz-Paris A, Rueda R. The Vicious Cycle of Type 2 Diabetes Mellitus and Skeletal Muscle Atrophy: Clinical, Biochemical, and Nutritional Bases. Nutrients. 2024 Jan 4;16(1):172. doi: 10.3390/nu16010172.
17. Acosta FM, Pacelli S, Rathbone CR. Diabetes diminishes muscle precursor cell-mediated microvascular angiogenesis. PLoS One. 2023 Aug 4;18(8):e0289477. doi: 10.1371/journal.pone.0289477.
18. Chen C, Lin LY, Chen JW, Chang TT. CXCL5 suppression recovers neovascularization and accelerates wound healing in diabetes mellitus. Cardiovasc Diabetol. 2023 Jul 7;22(1):172. doi: 10.1186/s12933-023-01900-w. Erratum in: Cardiovasc Diabetol. 2023 Aug 1;22(1):196. doi: 10.1186/s12933-023-01929-x.