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CARDIAC REMODELING IN EXPERIMENTAL METABOLIC OBESITY: HISTOLOGICAL AND MORPHOMETRIC EVIDENCE OF EARLY MYOCARDIAL ALTERATIONS

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Abstract. Metabolic obesity is a complex systemic condition leading to structural and functional alterations of the heart known as metabolic cardiomyopathy. Despite extensive studies on metabolic disorders, the morphological mechanisms of myocardial remodeling under chronic metabolic overload remain insufficiently characterized. Objective: This study aimed to evaluate the morphological and morphometric features of myocardial remodeling in a rat model of diet-induced metabolic obesity and to determine correlations between metabolic and structural parameters of the heart. Methods: an experimental model of metabolic obesity was reproduced in 20 male Wistar rats fed a high-fat and high-carbohydrate diet (HFD) for 12 weeks. Myocardial tissue was examined histologically using hematoxylin and eosin (H&E) staining, followed by morphometric assessment of cardiomyocyte cross-sectional area, left ventricular wall thickness, and the extent of interstitial fibrosis. Body weight, heart mass index, and serum lipid profile were measured. Statistical analysis included Student's t-test and Pearson correlation. Results: chronic exposure to HFD caused significant increases in body weight (+38.6%), heart mass index (+18.4%), left ventricular wall thickness (+19.8%), and cardiomyocyte cross-sectional area (+33.4%) compared with controls ($p < 0.01$). The relative area of interstitial fibrosis increased 2.6-fold ($p < 0.001$). Strong positive correlations were found between body weight and cardiomyocyte size ($r = 0.78$; $p < 0.001$) and between total cholesterol and fibrosis area ($r = 0.65$; $p < 0.01$). Histologically, the myocardium showed hypertrophy, cytoplasmic vacuolization, nuclear hyperchromasia, and focal interstitial edema, indicating early structural disorganization and fibrosis. Conclusion: prolonged consumption of a high-fat, high-carbohydrate diet induces adaptive–pathological myocardial remodeling manifested by cardiomyocyte hypertrophy, interstitial fibrosis, and vascular wall thickening. These morphological changes correlate closely with body mass and lipid metabolism disorders, reflecting the early stages of metabolic cardiomyopathy.

Keywords: metabolic obesity; myocardium; cardiomyocytes; morphometry; interstitial fibrosis; myocardial remodeling; high-fat diet; experimental model.

Introduction

Over the past few decades, metabolic obesity has become one of the most pressing global health challenges, affecting not only metabolic and endocrine regulation but also the structural and functional integrity of the cardiovascular system. According to the World Health Organization (WHO, 2024), more than 1.3 billion adults worldwide are classified as obese, and the prevalence of metabolic syndrome continues to rise steadily [1]. Among the numerous

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complications of obesity, cardiac remodeling and the development of so-called *metabolic cardiomyopathy* represent a major cause of morbidity and mortality [1, 2, 4, 8].

The pathogenesis of obesity-associated myocardial remodeling is complex and multifactorial. It involves chronic low-grade inflammation, lipotoxicity, oxidative stress, and endothelial dysfunction. Excessive accumulation of triglycerides and free fatty acids in adipose tissue and cardiomyocytes leads to mitochondrial dysfunction, activation of macrophages, and the release of pro-inflammatory cytokines such as tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6). These processes impair energy metabolism, increase reactive oxygen species (ROS) production, and promote apoptosis and fibrosis in cardiac tissue. Eventually, structural disorganization of the myocardium develops, accompanied by thickening of the left ventricular wall, interstitial collagen deposition, and diastolic dysfunction [3, 5-7, 9, 11].

Experimental and clinical evidence indicates that morphological alterations of the myocardium may precede the clinical manifestations of heart failure in obesity. Even in the absence of hypertension or ischemic heart disease, individuals with obesity frequently exhibit myocardial hypertrophy, impaired relaxation, and extracellular matrix expansion—findings that support the concept of obesity as an independent inducer of myocardial remodeling. Animal models based on a high-fat, high-carbohydrate diet (HFD) closely reproduce the human metabolic phenotype, including hyperlipidemia, insulin resistance, and visceral fat accumulation. Such models enable detailed investigation of the structural, cellular, and metabolic mechanisms underlying obesity-related cardiomyopathy [8, 10, 12, 13].

Histological analysis using hematoxylin and eosin (H&E) staining remains the cornerstone for identifying the morphological basis of myocardial injury, while morphometric assessment provides quantitative evaluation of hypertrophy and fibrosis. Together, these methods yield a comprehensive picture of cardiac remodeling and allow correlation of tissue-level changes with systemic metabolic parameters [10,13].

Despite considerable research progress, the detailed morphological and morphometric features of myocardial remodeling under chronic metabolic overload remain insufficiently characterized. Understanding these changes is essential for early diagnosis and development of targeted interventions aimed at preventing metabolic cardiomyopathy [5-7, 9, 11-14].

The present study aimed to investigate the histological and morphometric characteristics of cardiac remodeling in a rat model of diet-induced metabolic obesity and to identify correlations between myocardial structure and systemic metabolic alterations.

Materials and Methods

Study design and ethical approval. The experimental study was conducted at the Laboratory of Experimental Surgery and Pathomorphology, Republican Specialized Scientific and Practical Medical Center of Surgery named after Academician V. Vakhidov (Tashkent, Uzbekistan). All procedures were performed in compliance with national and international ethical standards, including the European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes (ETS No. 123). The experimental protocol was approved by the Institutional Ethics Committee. All efforts were made to minimize animal suffering and reduce the number of animals used.

Animals and experimental model. A total of 20 adult male albino rats (6–8 weeks old, 180–220 g) were included in the study. The animals were housed under standard vivarium conditions (22 ± 2 °C, relative humidity 50–60%, 12-hour light/dark cycle) with free access to food and water. After a one-week acclimatization period, the rats were randomly divided into two groups (n = 10 per group):

- Control group: received a standard laboratory chow diet.

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- Metabolic obesity group: received a high-fat and high-carbohydrate diet (HFD) for 12 weeks.

This dietary protocol has been previously shown to induce stable features of metabolic syndrome and early myocardial remodeling in rodents within a 10–12-week period.

Diet composition and experimental procedure. The experimental HFD provided 45–60% of total calories from fat and 20–30% from carbohydrates. The fat source included a mixture of butter and vegetable oil, while carbohydrates consisted of sucrose and starch. Feeding was *ad libitum*. The control group received a standard balanced diet meeting the physiological requirements for rats. Body weight was recorded weekly throughout the 12 weeks of the experiment. At the end of the study, animals were anesthetized with diethyl ether and euthanized for collection of biological material for histological, morphometric, and biochemical analyses.

Organ Weighing and Cardiac Mass Index. Immediately after euthanasia, the heart, liver, and kidneys were excised and weighed. The cardiac mass index (CMI) was calculated as the ratio of heart weight to total body weight and expressed as a percentage. This index served as a quantitative marker of myocardial hypertrophy associated with metabolic overload.

Histological Processing and Staining. For histological examination, myocardial samples (approximately 5 × 5 mm) were collected from the middle portion of the left ventricular wall. Tissue fragments were fixed in 10% neutral buffered formalin at +4 °C for 24 hours, dehydrated in graded ethanol series, cleared in xylene, and embedded in paraffin. Sections 4–5 μm thick were cut using a rotary microtome and stained with hematoxylin and eosin (H&E) according to standard procedures.

Microscopic analysis was performed using a DN-300 M light microscope (China) equipped with a digital camera. Photomicrographs were captured for documentation and subsequent morphometric evaluation using ImageJ 1.53u software (NIH, USA).

Morphometric evaluation. Quantitative morphometric analysis was carried out in ten non-overlapping microscopic fields per section at ×400 magnification. The following parameters were assessed:

- Cross-sectional area of cardiomyocytes (μm²): indicator of hypertrophy.
- Left ventricular wall thickness (mm): macromorphometric marker of concentric remodeling.
- Relative area of interstitial fibrosis (%): reflects the degree of connective tissue proliferation.

All measurements were averaged per animal and expressed as mean ± standard deviation (M ± SD).

Biochemical assays. To characterize lipid metabolism, serum concentrations of total cholesterol (TC), triglycerides (TG), low-density lipoproteins (LDL-C), and high-density lipoproteins (HDL-C) were determined. Blood samples were obtained via cardiac puncture immediately after euthanasia. Serum was separated by centrifugation at 3000 rpm for 10 minutes. Analyses were performed using an automated biochemical analyzer Cyan Smart (Cypress Diagnostics, Belgium) with certified reagent kits according to the manufacturer's instructions.

Statistical analysis. All quantitative data were analyzed using Statistica 10.0 (StatSoft, USA). Data distribution was assessed using the Shapiro–Wilk test. Group comparisons were performed using the Student's t-test for normally distributed variables or the Mann–Whitney U-test for nonparametric data. Correlations between body weight, lipid parameters, and morphometric indices were analyzed using the Pearson correlation coefficient (r). Differences were considered statistically significant at $p < 0.05$.

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Results

General findings and body weight dynamics. After 12 weeks of dietary intervention, rats fed a high-fat and high-carbohydrate diet (HFD) developed a stable phenotype of metabolic obesity. The mean body weight of the HFD group was significantly higher compared to controls (455.2 ± 18.3 g vs. 328.3 ± 15.7 g; $p < 0.001$), corresponding to a 38.6% increase. This was accompanied by an elevation in absolute heart mass and the cardiac mass index (CMI) (0.45 ± 0.03% vs. 0.38 ± 0.02%; $p < 0.01$), reflecting the development of moderate myocardial hypertrophy (Fig. 4).

Histological characteristics of the myocardium. Hematoxylin and eosin staining revealed distinct morphological differences between groups.

In the control group, myocardial tissue exhibited normal architecture: cardiomyocytes were arranged in orderly bundles, cytoplasm was evenly stained, and nuclei were centrally located and oval-shaped. Capillaries of the microcirculatory bed showed moderate fullness, with no evidence of dystrophic alterations (Fig. 2).

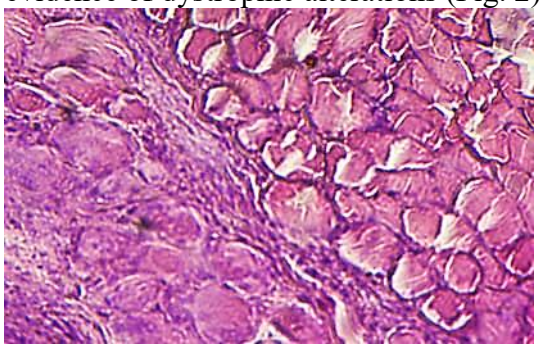

Figure 2. *Heart, control group. Normal myocardial architecture with organized cardiomyocyte bundles, evenly stained cytoplasm, and centrally located oval nuclei. H&E. ×10×40. Week 12.*

In contrast, the HFD-fed rats demonstrated evident morphological remodeling. Cardiomyocytes were enlarged in cross-section, often exhibiting cytoplasmic vacuolization, nuclear hyperchromia, and focal myofibrillar disorganization. Interstitial tissue displayed moderate edema and accumulation of connective tissue elements, particularly in perivascular areas. Thickening of vascular walls and narrowing of arteriolar lumina were also observed (Fig. 3).

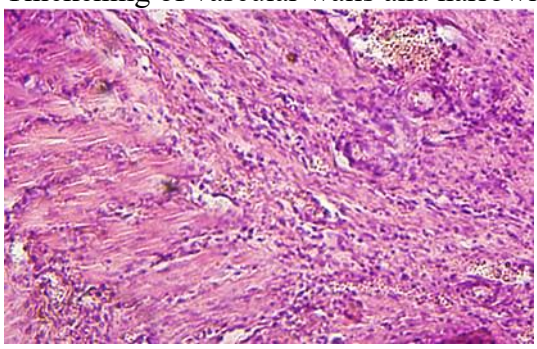

Figure 3. *Heart, metabolic obesity. Cardiomyocyte hypertrophy with cytoplasmic vacuolization and nuclear hyperchromia; mild interstitial edema. H&E. ×10×10. Week 12.*

These findings indicate the formation of concentric myocardial remodeling, combining hypertrophic and early fibrotic changes associated with metabolic overload.

Morphometric analysis. Quantitative morphometric evaluation supported the histological observations (Table 1, Fig. 4).

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The cross-sectional area of cardiomyocytes in the HFD group increased by 33.4% relative to controls (303.3 ± 14.5 μm² vs. 227.4 ± 11.3 μm²; $p < 0.001$). The left ventricular wall thickness was 19.8% greater (2.18 ± 0.09 mm vs. 1.82 ± 0.06 mm; $p < 0.01$). The relative area of interstitial fibrosis rose 2.6-fold (10.9 ± 1.6% vs. 4.2 ± 0.8%; $p < 0.001$), indicating activation of collagen deposition and extracellular matrix remodeling.

Table 1.

Changes in morphometric and functional parameters of the heart in diet-induced metabolic obesity (mean ± SD)

Parameter	Control	Obesity	Change (%)	p value
Body weight (g)	328.3 ± 15.7	455.2 ± 18.3	+38.6	< 0.001
Cardiac mass index (%)	0.38 ± 0.02	0.45 ± 0.03	+18.4	< 0.01
LV wall thickness (mm)	1.82 ± 0.06	2.18 ± 0.09	+19.8	< 0.01
Cardiomyocyte area (μm²)	227.4 ± 11.3	303.3 ± 14.5	+33.4	< 0.001
Fibrosis area (%)	4.2 ± 0.8	10.9 ± 1.6	× 2.6	< 0.001

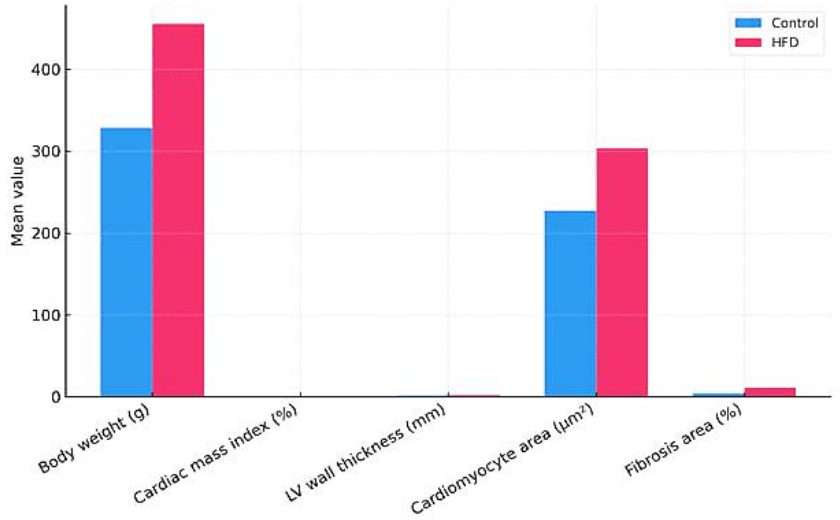

Figure 4. *Morphometric characteristics of myocardial remodeling in control and high-fat diet (HFD) groups. Body weight and cardiac mass index in control and HFD rats (mean ± SD).*

Correlation analysis. Correlation analysis revealed significant associations between morphometric and metabolic parameters. A strong positive correlation was found between body weight and cardiomyocyte cross-sectional area ($r = 0.78$; $p < 0.001$), indicating that the degree of myocardial hypertrophy was directly linked to obesity severity (Fig. 5). Furthermore, a moderate positive correlation was identified between serum total cholesterol and fibrosis area ($r = 0.65$; $p < 0.01$), confirming the involvement of lipid metabolism disorders in myocardial connective-tissue remodeling (Fig. 6).

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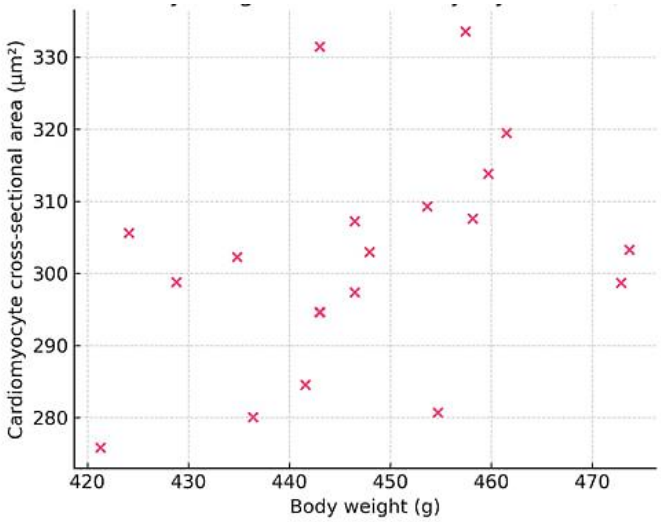

Figure 5. *Correlation between body weight and cardiomyocyte cross-sectional area (r = 0.78; p < 0.001). Week 12.*

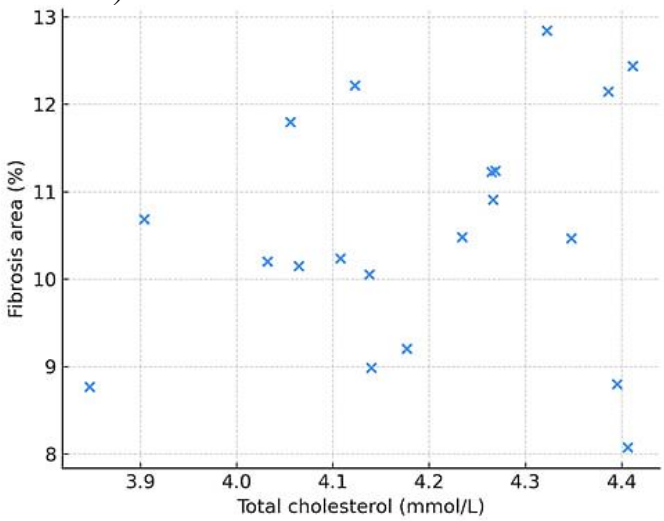

Figure 6. *Correlation between total cholesterol level and interstitial fibrosis area in the myocardium (r = 0.65; p < 0.01). Week 12.*

Overall, the obtained data demonstrate the development of adaptive-pathological cardiac remodeling in metabolic obesity, characterized by hypertrophy of cardiomyocytes, increased interstitial fibrosis, and vascular restructuring.

Discussion

The present study demonstrates that prolonged consumption of a high-fat and high-carbohydrate diet induces significant morphological remodeling of the myocardium consistent with features of metabolic cardiomyopathy. The observed increases in body weight, cardiac mass index, and cardiomyocyte size reflect a hypertrophic response of the myocardium to chronic metabolic overload rather than to mechanical hemodynamic stress. These findings align with previous experimental studies showing that obesity-related cardiac hypertrophy arises primarily due to lipid-induced cellular injury, mitochondrial dysfunction, and energy imbalance rather than from pressure or volume overload (Birilina, 2022; Ivanova & Petrov, 2021; Kruszewska et al., 2022).

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Histological analysis revealed cytoplasmic vacuolization, nuclear hyperchromia, and myofibrillar disorganization in cardiomyocytes of obese rats, indicating structural disintegration and metabolic distress. Such features have been associated with mitochondrial lipid accumulation and oxidative stress in high-fat diet models (Martins et al., 2015; Gao et al., 2023). The impairment of mitochondrial β-oxidation and the shift toward inefficient glycolytic metabolism contribute to ATP depletion and the initiation of apoptotic cascades, further promoting cardiomyocyte remodeling (Pellieux et al., 2012).

The significant increase in left ventricular wall thickness and cardiomyocyte area (by 19.8% and 33.4%, respectively) indicates a mixed compensatory–pathological pattern of hypertrophy. This concentric remodeling pattern is characteristic of the early stages of metabolic cardiomyopathy, where increased sarcomere synthesis occurs without adequate angiogenesis, resulting in local hypoxia and fibroblast activation (Schiattarella & Hill, 2021; Shiou et al., 2018). Our data are in line with these observations, confirming that metabolic stress alone, independent of hypertension, can trigger structural myocardial remodeling.

The relative area of interstitial fibrosis increased more than 2.5-fold in HFD-fed rats, which is a hallmark of excessive extracellular matrix deposition. Current evidence suggests that obesity-associated cardiac fibrosis arises from activation of fibroblasts and myofibroblasts under the influence of proinflammatory cytokines (IL-6, TNF-α, TGF-β1) and reactive oxygen species (Wang et al., 2022; Zou et al., 2018). The resulting overproduction of collagen types I and III disrupts myocardial elasticity and impairs diastolic relaxation, thereby predisposing to heart failure with preserved ejection fraction (HFpEF).

An important aspect of this study is the correlation analysis linking metabolic and structural parameters. The strong positive correlation between body weight and cardiomyocyte cross-sectional area ($r = 0.78;\ p < 0.001$) indicates that myocardial hypertrophy is directly proportional to the degree of obesity. Similarly, the positive association between serum total cholesterol and the extent of fibrosis ($r = 0.65;\ p < 0.01$) underscores the role of dyslipidemia in fibrogenic remodeling through lipotoxic and inflammatory mechanisms (Santos et al., 2023; Portes et al., 2024). These relationships are consistent with clinical data showing that hypertriglyceridemia and elevated LDL levels in obese patients are correlated with increased myocardial mass and impaired diastolic function (Yusuf et al., 2020).

Vascular changes observed in this study—arteriolar wall thickening, luminal narrowing, and perivascular edema—suggest microcirculatory dysfunction and endothelial impairment, which are recognized as central components of obesity-related cardiac damage (Pankova et al., 2022; Tian et al., 2022). Endothelial dysfunction promotes oxidative stress, inflammation, and reduced nitric oxide bioavailability, further exacerbating myocardial hypoxia and collagen deposition.

Taken together, our findings provide experimental confirmation that long-term high-fat diet feeding leads to adaptive–pathological myocardial remodeling, characterized by cardiomyocyte hypertrophy, interstitial fibrosis, and vascular restructuring. These structural alterations occur in parallel with metabolic dysregulation and represent the morphological substrate of early metabolic cardiomyopathy. Understanding these processes is crucial for developing preventive and therapeutic strategies aimed at reversing or attenuating structural cardiac damage in metabolic obesity.

Conclusion

The findings of this experimental study provide compelling evidence that prolonged high-fat and high-carbohydrate feeding induces significant structural and morphometric remodeling of the myocardium in rats. The cardiac response to metabolic overload was characterized by

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cardiomyocyte hypertrophy, cytoplasmic vacuolization, nuclear hyperchromia, and the development of interstitial fibrosis. Quantitative morphometric analysis revealed a 33.4% increase in cardiomyocyte cross-sectional area, a 19.8% thickening of the left ventricular wall, and a more than 2.5-fold increase in fibrosis compared with controls ($p < 0.001$).

Strong correlations between body weight and cardiomyocyte area ($r = 0.78$; $p < 0.001$), and between serum cholesterol levels and fibrosis extent ($r = 0.65$; $p < 0.01$), confirm the close relationship between metabolic dysregulation and structural myocardial remodeling.

Collectively, these results indicate the development of adaptive–pathological myocardial remodeling in metabolic obesity, combining hypertrophic and fibrotic processes with vascular alterations. This pattern can be regarded as an early morphological manifestation of metabolic cardiomyopathy, emphasizing the importance of early metabolic correction and lifestyle intervention to prevent irreversible cardiac damage.

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