MYOCARDIAL REMODELING AND FIBROTIC CHANGES IN EXPERIMENTAL METABOLIC OBESITY

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Introduction. Metabolic obesity is a complex systemic disorder characterized by chronic low-grade inflammation, lipid dysmetabolism, and oxidative stress, leading to structural and functional remodeling of the myocardium known as metabolic cardiomyopathy. Fibrosis is a key pathological mechanism contributing to diastolic dysfunction and impaired cardiac performance. However, the morphological patterns of interstitial and perivascular fibrosis under sustained metabolic overload remain insufficiently characterized. The present study aimed to assess myocardial remodeling and the degree of fibrosis in experimental metabolic obesity and to correlate morphological changes with biochemical markers of oxidative stress and inflammation.

Materials and Methods. The experimental study was conducted at the Laboratory of Experimental Surgery and Pathomorphology, RSSPMCS named after acad. V. Vakhidov. All procedures complied with the principles of bioethics and were approved by the local ethics committee.

Thirty-six male albino rats (180–220 g, 6–8 weeks old) were divided into three groups (n=12 each):

• Control — standard diet;

• Obesity — high-fat/high-carbohydrate diet for 12 weeks;

• Recovery — after 12 weeks of diet, rats were returned to a standard diet for 4 weeks.

Body weight, lipid profile, myocardial morphology (H&E), immunohistochemistry ($CD68^+$ macrophages), and biochemical markers (MDA, SOD, catalase) were analyzed.

Results. By week 4, obese rats showed a significant increase in body weight (270 ± 8 g vs. 245 ± 8 g in control, $p<0.05$), reaching 410 ± 12 g by week 12 (+37%, $p<0.001$). Dyslipidemia was evident, with total cholesterol and triglycerides increasing twofold and HDL decreasing by 36% ($p<0.01$). Histologically, pronounced cardiomyocyte hypertrophy, cytoplasmic vacuolization, and marked interstitial and perivascular fibrosis were observed. The mean cardiomyocyte area rose from 220 ± 8

μm² (control) to 295 ± 10 μm² ($p<0.01$). $CD68^+$ macrophage count doubled (18.2 ± 1.4 vs. 7.9 ± 0.8 cells/field, $p<0.001$). Biochemically, MDA increased by 2.3-fold, while SOD and catalase activities decreased by 35–40% versus control. After dietary withdrawal, partial improvement occurred, but parameters did not return to baseline.

Discussion and Conclusion. Metabolic obesity induces significant myocardial remodeling characterized by cardiomyocyte hypertrophy, interstitial and perivascular fibrosis, macrophage infiltration ($CD68^+$), and enhanced oxidative stress. Withdrawal of the high-fat diet led to only partial regression of structural and biochemical abnormalities, suggesting the persistence of residual pathological remodeling even after metabolic correction.