DEVELOPMENT OF HYPERCHOLESTERINEMIA IN METABOLIC SYNDROME AND ITS CORRECTION

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Introduction. Currently, metabolic syndrome is manifested by symptoms such as obesity, weight gain and excess fat accumulation among the population, which, as a result, is manifested by diseases such as diabetes mellitus and insulin resistance among the population. According to the World Health Organization, the number of diabetes mellitus patients currently exceeds 400 million worldwide. In 2019 alone, diabetes ranked ninth in terms of causes of death, leading to approximately 1.5 million deaths. The prevalence of obesity and diabetes has reached pandemic levels over the past 50 years, which in turn leads to the development of metabolic syndrome in the body and dysfunction of all organs and tissues of the body, including lung tissue. This disease, as a result of the accumulation of hypoxic cells around organs and tissues, causes respiratory diseases such as pneumonia, bronchial asthma, pulmonary hypertension, acute respiratory distress syndrome (ARDS) and obstructive sleep apnea syndrome, disruption of lung homeostasis and the immune system, and changes in the population of inflammatory cells in the lung in obesity have been studied by a number of foreign researchers. Scientists from the CIS countries have elucidated the complex pathophysiological mechanisms of obesity and diabetes, the presence of mitochondrial dysfunction, endoplasmic reticulum stress in this process, and the mechanisms of changes in cellular and intracellular redox balance. In our country, few studies have been conducted on non-respiratory lung function disorders in metabolic syndrome, their early diagnosis, and correction of identified changes. For this reason, the implementation of this scientific research is of scientific and practical importance.
Research objective: To observe the effect of a traditional and modified ketogenic diet on non-respiratory lung functions in metabolic syndrome and analyze the results obtained.

Material and research methods: To achieve the goal of the study, rats were given a high-fat, low-carbohydrate diet and a glucose-fructose mixture instead of water for 2 months, and metabolic syndrome was modeled. Starting from 3 months, they were

divided into groups and a traditional and modified ketogenic diet was used for 1 month. Biochemical tests were performed on the blood.

Results: It is important to study the changes in the levels of alpha-1 antitrypsin, myeloperoxidase, and surfactant protein in the blood serum to monitor changes in the lungs during the diet used in metabolic syndrome. It was found that body weight and alpha-1 antitrypsin levels increased in rats modeled with metabolic syndrome compared to intact rats. In particular, it was found that alpha-1 antitrypsin levels in the blood plasma of rats modeled with metabolic syndrome increased by 1.28; 1.38 and 1.68 times compared to the intact group at 1; 2 and 3 months, respectively. The reason for such changes in the blood is that alpha-1 antitrypsin increases in the amount of metabolism in the lungs, when inflammatory processes increase.

In such disorders, an imbalance is observed in the non-respiratory functions of the lungs and the amount of substances produced in it. In the groups of rats treated with a modified ketogenic diet, it was found that the amount of alpha-1 antitrypsin decreased compared to the conventional and diseased groups, and this indicator was close to that of healthy rats. In particular, in the group treated with a modified ketogenic diet, it decreased by 25.8%, and in the group treated with a conventional ketogenic diet, it decreased by 18.2%. This indicates that the modified diet used has a more effective effect compared to the conventional ketogenic diet.

Conclusion. The results obtained indicate that the amount of alpha-1 antitrypsin increases as a result of structural and functional impairment of the lungs in metabolic syndrome. The use of a modified ketogenic diet effectively affected the morphology and non-respiratory functions of the lungs.