DISORDERS OF THE LIPIDS METABOLISM AND CHANGES OF MAO ACTIVITIES AT THE DYLATATION CARDIOMYOPATHY

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Introduction: According to WHO data, among cardiovascular diseases, cardiomyopathies account for a significant cause of mortality every year, taking many lives. In primary cardiomyopathy, dilation of the heart ventricles and chronic heart failure are observed. In many cases, its development may be due to the combined effect of coronary heart disease (CHD) and risk factors, among which is an increase in the level of low-density lipoproteins (LDL) in the blood serum. The term "cardiomyopathy" was first proposed by Bridgen in 1957 to describe myocardial disease of unknown origin, characterized by ECG changes, progressive circulatory failure, and potentially fatal outcomes. Godwin also supported this explanation of cardiomyopathy and conducted fundamental research from 1961 to 1982, classifying cardiomyopathy into three groups: dilated, hypertrophic, and restrictive. However, the definition of "cardiomyopathy" by the WHO is somewhat controversial, as the term is used in a very broad sense. For example, so-called "specific cardiomyopathies" include myocardial dysfunction due to CHD ("ischemic cardiomyopathy"), and due to heart valve disease ("valvular cardiomyopathy"), among others. Dilated cardiomyopathy is characterized by the dilation of the left or both ventricles and impaired myocardial contractility. Depending on the cause, it may be familial/hereditary, viral or immune, alcoholic (toxic), idiopathic (of unknown origin), or related to cardiovascular diseases where the degree of myocardial dysfunction is not clearly determined. In modern medicine, diagnostic criteria for cardiomyopathies are still insufficient. The interrelationship between monoamine oxidase (MAO) activity and lipid metabolism alterations sparked our interest.

The purpose of the scientific work: to study lipid metabolism and Mao activity in patients with dilated cardiomyopathy.

Materials and Methods: The diagnosis was made according to the recommendations of the American College of Cardiology (ACC), American Heart Association (AHA), and European Society of Cardiology (ESC). Clinical data, central and intracardiac hemodynamics, ECG, and type and number of rhythm disorders were analyzed.

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To quantify pain in the heart, a 5-point verbal rating scale (VRS) was used: 1 point – discomfort behind the sternum; 2 – mild pain; 3 – moderate pain; 4 – severe pain; 5 – extremely severe (compressive) pain.

Total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG) were determined using the 'Reflotron Plus' biochemical analyzer (Roche, Germany). Blood samples were collected after 12 hours of fasting. Low-density lipoprotein cholesterol (LDL-C) and very-low-density lipoproteins (VLDL-C) were calculated using Friedwald's formula: LDL-C = TC – HDL-C – VLDL-C, VLDL-C = 0.45 x TG. The atherogenic index (AI) was calculated using the formula: AI = (TC – HDL-C)/HDL-C, as recommended by A. Klimov.

MAO activity in serum was determined using the method by A.I. Balakleevsky. Clinical lab tests, ECG, and radiological exams were conducted. A total of 26 patients were examined and divided into four groups:

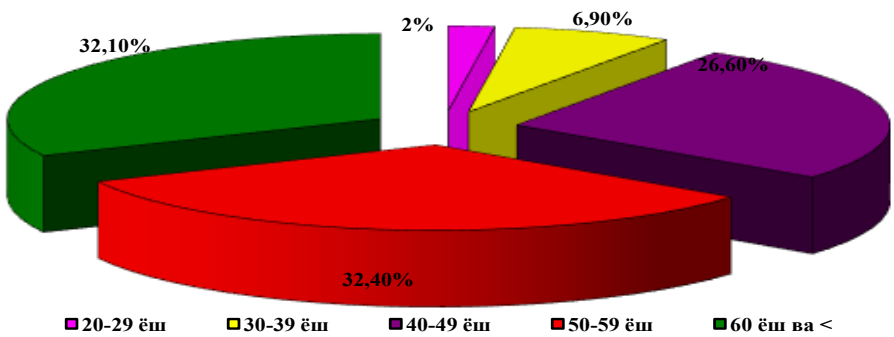
I – control group (15 healthy individuals),

II – DCM with CHF stage I, FC II (NYHA) (8 patients),

III – DCM with CHF stage II, FC II (NYHA) (8 patients),

IV – DCM with CHF stage III, FC III (NYHA) (10 patients).

An increased incidence of sari was observed in patients aged 20 to 69 years (1st figure)



Results: When comparing the measured parameters among the patients, it was revealed that the average functional class (FC) of chronic heart failure (CHF) according to NYHA classification was 16% higher in Group IV patients.

Regarding the ECG features of the patients, 74% showed negative T waves and ST segment inversion (in both standard and chest leads), while 12% of the patients exhibited atypical Q waves (including QS complexes). According to data by Kelly B.S. (2007), such signs may be present in up to 50% of cases.

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Among cardiac rhythm disturbances, ventricular extrasystole was observed in 48% of patients, and atrial fibrillation (AF) was detected in 3.5% of cases.

It is well known that dyslipidemia is one of the primary risk factors for ischemic heart disease (IHD) [2,5]. Clinical and biochemical examinations show that the development and progression of diseases such as IHD, hypertension (HT), and diabetes mellitus (DM) are strongly associated with disorders of lipid metabolism.

Indicator	Group I (Healthy, n=15)	Group II (DCM, CHF Stage I FC II, n=8)	Group III (CHF Stage II FC II, n=8)	Group IV (CHF Stage III FC III, n=10)
Total Cholesterol (TC), mmol/L	4.4 ± 0.18	5.88 ± 0.1	6.23 ± 0.13	6.47 ± 0.11 *
Triglycerides (TG), mmol/L	1.37 ± 0.2	1.69 ± 0.06	2.2 ± 0.06 *	2.9 ± 0.07 *
HDL-C, mmol/L	1.4 ± 0.07	1.21 ± 0.06	0.82 ± 0.04 *	0.78 ± 0.03 *
LDL-C, mmol/L	2.08 ± 0.13	3.1 ± 0.07	3.5 ± 0.11 *	3.8 ± 0.11 *
VLDL-C, mmol/L	0.38 ± 0.04	0.43 ± 0.01 *	0.78 ± 0.02 *	0.96 ± 0.04 *
Atherogenic Index (AI)	2.14 ± 0.15	3.03 ± 0.07 *	3.84 ± 0.11 *	4.04 ± 0.22 *
MAO activity (unit/extract)	0.08 ± 0.007	0.068 ± 0.006	0.052 ± 0.0014 *	0.042 ± 0.004 *

Note: * - statistically significant difference compared to control group ($p < 0.05$)

Explanation of Results: During our research, the following findings were observed:

In Group II, total cholesterol was 5.88 ± 0.1 mmol/L, increased by 34.4% compared to the control group.

Triglyceride levels were 11.8% higher than in Group I.

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HDL-C was 1.21 ± 0.06 mmol/L in Group II, decreased by 18.1% compared to controls ($p < 0.05$).

LDL-C increased significantly to 3.1 ± 0.07 mmol/L – a 46.3% rise compared to Group I.

Atherogenic index (AI) reached 3.03 ± 0.07 units, 38% higher than the control ($p < 0.05$).

In Group III:

TC was 6.23 ± 0.13 mmol/L, which was 40.2% higher than control and 7.3% higher than Group II.

HDL-C levels dropped by 43.1% compared to control, and by 26.5% compared to Group II.

AI was 63.7% higher than control, and 25.7% higher than Group II.

In Group IV (CHF Stage III FC III):

Compared to Groups II and III, TC increased by 66%, TG by 58.3%, LDL-C by 69.2%, and AI by 82%. HDL-C decreased by 15.9%.; the atherogenic index (AI) was found to have increased by 82% in Group IV compared to Groups II and III.

Currently, the increased activity of free radical peroxidation (FRP) processes is known to play an important role in the development of internal organ diseases, particularly atherosclerosis and ischemic heart disease (IHD) [5]. According to numerous sources, in comorbid pathological conditions, the activity of monoamine oxidase (MAO) can undergo significant qualitative modifications, primarily due to excessive intensification of lipid peroxidation (LPO) processes. In certain pathological states, both quantitative and qualitative repetitive changes in the catalytic properties of MAO activity have been observed [5,10,12]. From the early days of this study, a significant reduction in MAO activity was observed in the examined patients. In patients with DCM and CHF stage II, FC II, the average MAO activity was 0.042 ± 0.0013 units/extract, which was 35.7% lower than in the control group.

A sharp decrease in MAO activity was detected in patients with DCM and CHF stage III, FC III, with an average value of 0.036 ± 0.002 units/extract — 57.8% (2.1 times) lower than the control group and 18% lower than the values in patients from Groups II and III ($p < 0.05$, see Table 1).

In healthy individuals, the same indicator was 0.08 ± 0.007 units/extract.

In contrast to the increased LPO, a simultaneous decline in the body's antioxidant defense system was also identified.

Analysis: It is well known that dyslipidemia is one of the main contributing factors in the development of ischemic heart disease (IHD). Our investigations revealed that lipid spectrum disorders are also present in patients with dilated cardiomyopathy (DCM). An

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increase in cholesterol and low-density lipoproteins (LDL), along with a decrease in high-density lipoproteins (HDL), was observed.

Furthermore, our study found that in certain pathological processes, the catalytic function of monoamine oxidase (MAO) can undergo both quantitative and qualitative repetitive changes. A significant increase in free radical lipid peroxidation (LPO) was detected. According to the literature, LPO intensification greatly influences MAO activity.

Our results showed that MAO activity was notably reduced. A significant decline in MAO activity was found in the studied patients. In contrast to increased LPO, the antioxidant defense system of the body was found to be suppressed.

The conducted investigations demonstrated that significant changes in both lipid profiles and MAO activity were particularly evident in Groups III and IV. This includes the reduction in MAO quantity and catalytic properties, the increase in malondialdehyde (MDA) levels, and the presence of dyslipidemia—all clearly confirm the pathological process in DCM.

It was also determined that MAO is responsible for the deamination of catecholamines (CAs).

In general, alterations in MAO activity and lipid spectrum in DCM reflect increased cholesterol levels and enhanced lipoperoxidation processes. These conditions may contribute to the development of oxidative stress, which complicates the progression of atherosclerosis, IHD, arterial hypertension (AH), and diabetes mellitus (DM).

Conclusion: In summary, significant alterations in lipid spectrum and MAO activity were identified in patients with dilated cardiomyopathy (DCM). Our study confirmed a marked decrease in MAO activity among the examined patients. Noticeable dyslipidemia was observed in this pathology. The obtained results demonstrate that lipid metabolism disorders are characterized by increased levels of total cholesterol (TC), triglycerides (TG), low-density lipoproteins (LDL), and atherogenic index (AI), along with a significant decrease in high-density lipoproteins (HDL).Correlation analysis indicated a strong relationship between lipid spectrum changes and MAO activity.

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