ASSESSMENT OF LIPID PEROXIDATION PROCESSES AND LIPID METABOLISM DISORDERS IN PATIENTS WITH ISCHEMIC HEART DISEASE UNDERGOING COMBINED HYPOLIPIDEMIC THERAPY

Juraboyev X.O., Tashtemirova I.M.
Andijan State Medical Institute

Introduction: In the 20th and 21st centuries, cardiovascular diseases (CVD), particularly complications associated with atherosclerosis, remain among the leading causes of death and disability. In many cases, ischemic heart disease (IHD) develops due to the combined influence of several risk factors, including increased levels of low-density lipoproteins (LDL) in blood serum. Genetic factors account for approximately 50% of LDL variability. The onset of IHD is influenced by a complex interaction of hemodynamic, neurohumoral, metabolic, and other factors [4,8,11].

In recent years, the growing incidence of IHD, increasing mortality rates, and the loss of work capacity among people of different ages and professions have made this issue not only a medical but also a social concern [1,4]. It is known that ischemia is accompanied by a marked increase in lipid peroxidation (LPO), the intensity of which indicates the depth of metabolic changes in the body (Titeeva G.R., Korovina N.N., 2016). The activation of lipid peroxidation plays a major role in the pathogenesis of CVD. Lipid peroxidation produces important intermediates in the biosynthesis of lipoperoxides, prostaglandin E, and progesterone. The cholesterol nucleus is involved in sterol hydroxylation. Over the past decade, experimental and clinical studies have confirmed that the formation of IHD is largely driven by endothelial dysfunction and immune inflammation, which are early indicators of cardiovascular disease and major contributors to atherothrombosis [3,8,12].

Aim of the Study: To investigate lipid metabolism and lipid peroxidation activity in patients with IHD undergoing combined hypolipidemic therapy.

Materials and Methods:Diagnosis was made according to the guidelines of the American College of Cardiology (ACC), American Heart Association (AHA), and the European Society of Cardiology (ESC). To evaluate disease progression, central and intracardiac hemodynamics, ECG data, and types and frequencies of arrhythmias were analyzed.

Chest pain was assessed using a 5-point verbal rating scale (VRS) completed by the physician based on the patient's description: 1 point — Discomfort behind the sternum
2 points — Mild pain
3 points — Moderate pain
4 points — Severe pain
5 points — Very severe (crushing) pain

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Total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG) were measured using the "Reflotron Plus" biochemical express analyzer (Roche, Germany). Blood samples were collected after 12 hours of fasting.

LDL-C and very low-density lipoprotein cholesterol (VLDL-C) were calculated using the W. Friedewald formula:

LDL-C = TC – HDL-C – VLDL-C

VLDL-C = 0.45 × TG

The LDL/HDL ratio and atherogenic index (AI) were also calculated using the formula proposed by A. Klimov: AI = (TC – HDL-C) / HDL-C Monoamine oxidase (MAO) activity in blood serum was assessed by A.I. Balakleevsky's method. Patients underwent standard clinical, ECG, and radiographic investigations.

A total of 26 patients were examined and divided into 4 groups:

Group I: Control group (15 healthy individuals)

Group II: Stable angina, functional class (FC) I (8 patients)

Group III: Stable angina FC II (10 patients)

Group IV: Stable angina FC III (10 patients)

Results: ECG changes included negative T waves and ST segment inversion in 74% of patients, and atypical Q waves (with QS complexes) in 12%. According to Kelly B.S., such signs can occur in up to 50% of cases. Among arrhythmias, ventricular extrasystoles were found in 48% of patients, and atrial fibrillation in 3.5%. Since dyslipidemia is a major factor in IHD development [2,8,10,12], clinical and biochemical tests showed that abnormalities in lipid metabolism are closely associated with the progression of IHD, hypertension (HTN), and diabetes mellitus (DM).

Table 1. Lipid Spectrum Parameters in the Blood Serum of Examined Patients

	I group (healthy) n=15	II group n=8	III group, n=10	group iv, n=10
TC	4,4+0,18	5,88+0.1	6,23+0,13	6,47+0,11*
TG	1,37+0,2	1,69+0,06	2,2+0,06*	2,9+0,07*
HDL-C	1,4+0,07	1,21+0,06	0,82+0,04*	0,78+0,03*
LDL-C	2,08+0,13	3.1+0,07	3,5+0,11*	3,8+0,11*
VLDL-C	0.38+0,04	0,43+0,01*	0,78+0,02*	0,96+0,04*
AI	2,14+0,15	3,03+0,07*	3,84+0,11*	4,04+0,22*

Note: *P < 0.05 indicates statistically significant differences compared to the control group.

During our investigation, the following results were obtained:

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In Group II, the level of total cholesterol (TC) was 5.88±0.1 mmol/L, which represents a 34.4% increase compared to the control group. The concentration of triglycerides (TG) in Group II increased by 11.8% compared to Group I.

The level of high-density lipoprotein cholesterol (HDL-C) in Group II was 1.21±0.06 mmol/L, which is 18.1% lower than in the control group ($P < 0.05$).

Low-density lipoprotein cholesterol (LDL-C) increased significantly to 3.1±0.07 mmol/L, which is a 46.3% rise compared to Group I.

The atherogenic index (AI) in Group II reached 3.03±0.07 units, which is 38% higher than in the control group ($P < 0.05$).

In Group III, serum lipid spectrum analysis yielded the following results:

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

HDL-C levels were 43.1% lower compared to the control group and 26.5% lower than in Group II.

The AI in Group III increased by 63.7% compared to Group I and by 25.7% compared to Group II (see Table 1).

In patients with silent myocardial ischemia, our study found the following changes in lipid parameters compared to Groups II and III:

TC increased by 66%, TG by 58.3%, LDL-C by 69.2%, HDL-C decreased by 15.9%, The atherogenic index increased by 82%

Table 2: Average MDA and Catalase Levels in Serum

The group	MDA (nm/l)	Ca (earth/land by 10.6)
I-Control	of 3.3±0,11	19,5±0,66
II,	4,8±0,16	14,6±0,26
III	6,14±0,18	12,9±0,33
IV	7,41±0.2*	10,5±0,51*

Note: *$P < 0.05$ indicates statistically significant differences compared to the control group.

MDA levels were 59.8% higher in Groups III and IV than in controls, reaching 7.41±0.2 nmol/mL in Group IV (78.2% higher). Catalase activity, representing antioxidant defense, was significantly reduced—by 45.7% compared to controls.

Analysis: Dyslipidemia remains a key contributor to IHD. Changes in lipid profile were observed even in early-stage angina. Increased cholesterol, LDL-C, decreased HDL-C, and intensified lipid peroxidation with suppressed antioxidant defense were evident.

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The most pronounced changes in lipid metabolism and MDA levels were observed in Groups III and IV, confirming the link between dyslipidemia and disease severity. In patients with FC III stable angina, both lipid peroxidation and cholesterol levels were significantly elevated, indicating progressive oxidative stress, potentially worsening the course of atherosclerosis, IHD, HTN, and DM.

Conclusion: Patients with IHD exhibited significant changes in lipid spectrum and increased lipid peroxidation. The pathology was marked by clear dyslipidemia: elevated TC, TG, LDL-C, AI, and decreased HDL-C. Correlation analysis confirmed a direct relationship between LPO activity and lipid spectrum abnormalities.

References:

1. Almazov V.A., Ermilov L.P., Kuleshova E.V. Unstable Angina: Issues of Diagnosis, Pathogenesis and Treatment Tactics. Cardiology. 2024; (10): 5–11.
2. Aronov D.M., Lupanov V.P. Diagnosis and Treatment of Chronic Ischemic Heart Disease. Quality of Life. Medicine. 2017; (3): 16–24.
3. Bochkarev E.V., Kokurina E.V. IHD at Various Levels of Blood Pressure in Patients with Exertional Angina (U-shaped dependence). Cardiology. 2019; (5): 34–37.
4. Belozerov S.M., Borovkov N.N. Oxidative Stress in Patients with Silent Myocardial Ischemia Syndrome. Nizhny Novgorod Medical Journal. 2020; (1): 35–39.
5. Olesin A.I., Pavlova R.N. Clinical Evaluation of Changes in Lipid Peroxidation in Patients with Ischemic Heart Disease and Cardiac Arrhythmias. Therapeutic Archive. 2022; (4): 82–86.
6. Tashtemirova I.M., Qosimov I.S. Features of Purine Metabolism and Microalbuminuria in Ischemic Heart Disease Patients with Metabolic Syndrome.
7. Tashtemirova I.M., Ravzatov Zh.B., Uzbekova N.R., Kodirova G.I. Evaluation of Lipid Spectrum and Fatty Acid Changes in Patients with Genetic Predisposition to IHD. Uzbekistan Therapeutic Bulletin. 2020; (1): 23–27.
8. Allaberganov O.Kh. The Features of Cardiac Function and ECG Parameters in Patients with Hypertrophic Cardiomyopathy. Proceedings of the International Congress of the Society for Holter and Noninvasive Electrophysiology (ISHNE-2011), Moscow, 2023: p. 52.
9. Loddenkemper R., Gibson G.J., Sibille Y., et al. European Respiratory Journal. 2021; 64 p.
10. Marshall R.P. The Pulmonary Renin-Angiotensin System. Current Pharmaceutical Design. 2018; 9(9): 715–722.
11. Randall W.C., Armour J.A. Regional Vagosympathetic Control of the Heart. American Journal of Physiology. 2017; 227: 444–452.
12. Loyola University Chicago Medical School Resources: http://www.meddean.luc.edu

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