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COMPARATIVE ANALYSIS OF LIPID METABOLISM, INFLAMMATORY MARKERS, AND ENDOTHELIAL DYSFUNCTION INDICATORS IN PATIENTS WITH ISCHEMIC HEART DISEASE AND STEATOHEPATITIS

Jasur Anorboy o‘g‘li Isamiddinov
Gavkhar Mirakbarovna Tulaboeva
Kholida Makhmudovna Sagatova
Yulduz Shavkatovna Talipova
Center for the Development of Professional Qualifications of Medical Workers

Abstract
This study is devoted to the assessment of biochemical indicators associated with lipid metabolism, inflammatory markers, and endothelial dysfunction in patients with ischemic heart disease (IHD) combined with steatohepatitis. A total of 120 patients were included in the study: the main group consisted of 60 patients with IHD combined with steatohepatitis, while the control group included 60 patients with IHD without steatohepatitis. In all patients, total cholesterol, triglycerides, low-density and high-density lipoproteins, fibrinogen, C-reactive protein, homocysteine, and liver enzyme levels were analyzed at baseline and during follow-up at 6 and 12 months. The obtained results demonstrated that dyslipidemia, inflammatory processes, and biochemical indicators associated with endothelial dysfunction were more pronounced in the main group compared to the control group. Dynamic analysis showed that although many laboratory indicators improved after 6 months, partial increases in several markers were observed again at the 12-month stage. These findings indicate that metabolic and inflammatory processes in patients with concomitant IHD and steatohepatitis require long-term monitoring and careful management.

Keywords: Ischemic heart disease, steatohepatitis, dyslipidemia, lipid profile, endothelial dysfunction, C-reactive protein, homocysteine, fibrinogen.

Introduction

In recent years, metabolic dysfunction–associated steatotic liver disease (MASLD, formerly NAFLD) has been increasingly considered not only a hepatological condition but also an important cardiometabolic problem. Numerous studies have shown that this pathology is closely associated with dyslipidemia, insulin resistance, chronic low-grade inflammation, and endothelial dysfunction, thereby increasing the risk of cardiovascular diseases [1,2,3]. In cases where ischemic heart disease occurs concomitantly with steatohepatitis, metabolic disturbances may become even more pronounced. In particular, the formation of an atherogenic lipid profile, increased levels of inflammatory and hypercoagulability markers such as C-reactive protein and fibrinogen, as well as

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elevated homocysteine concentrations contribute to the progression of endothelial dysfunction and atherosclerotic processes [2,4,5]. Endothelial dysfunction is considered one of the earliest and most important pathogenetic stages in the development of cardiovascular complications. In patients with NAFLD/MASLD, impairment of vascular wall function, including a decrease in flow-mediated dilation, has been confirmed in several meta-analyses and systematic reviews [4]. Moreover, cardiovascular diseases are recognized as one of the leading causes of mortality in patients with MASLD [3]. From this perspective, evaluating lipid metabolism, inflammatory markers, liver enzymes, and biochemical indicators associated with endothelial dysfunction in patients with combined IHD and steatohepatitis -both at baseline and during follow-up-has significant clinical importance. Such an approach provides a basis for early identification of high-risk patients and for improving monitoring strategies and comprehensive therapeutic management [3,5,6,7,8,9,10].

Study Objective

The aim of the study was to assess the baseline levels of biochemical parameters associated with lipid metabolism, inflammatory markers and endothelial dysfunction in patients with ischemic heart disease combined with steatohepatitis, to determine their differences compared with the control group, and to evaluate their dynamics during 6-month and 12-month follow-up periods.

Materials and Methods

A total of 120 patients were included in the study. They were divided into two groups: the main group consisted of 60 patients with ischemic heart disease combined with steatohepatitis, and the control group consisted of 60 patients with ischemic heart disease without steatohepatitis. The examinations were conducted at the cardiology dispensary of the Fergana region. All patients underwent laboratory examinations and the following indicators were evaluated: total cholesterol, triglycerides, low-density lipoproteins (LDL), high-density lipoproteins (HDL), fibrinogen, C-reactive protein (CRP), homocysteine, as well as liver enzymes alanine aminotransferase (ALT) and aspartate aminotransferase (AST). The parameters were studied at baseline and during follow-up at 6 months and 12 months. In statistical analysis quantitative indicators were expressed as M±SD. Differences between groups and changes during the follow-up period were assessed using χ^2 and p values. A p value less than 0.05 was considered statistically significant.

Results

The table below presents the analysis of the dynamics of liver enzymes, inflammatory markers and lipid metabolism indicators in patients during the follow-up period (baseline, 6 months and 12 months). At the baseline stage biochemical and metabolic disturbances were observed in the majority of patients. Elevated ALT levels were detected in 38 patients (63.3%), while increased AST levels were identified in 34 patients (56.7%). In addition, fibrinogen levels above 4 g/L were found in 32 patients (53.3%), and C-reactive protein levels above 5 mg/L were observed in 36 patients (60.0%). Elevated homocysteine levels were recorded in 40 patients (66.7%), indicating active inflammatory and metabolic imbalance processes in the body. Disturbances in lipid metabolism were also widespread: elevated triglyceride levels were detected in 37 patients (61.7%), while increased LDL

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levels were observed in 35 patients (58.3%). The reassessment performed at the 6-month follow-up demonstrated positive dynamics in several indicators. Elevated ALT levels were observed in 24 patients (40.0%), and increased AST levels in 21 patients (35.0%), which indicates a noticeable decrease compared with baseline values. During the same period elevated fibrinogen levels decreased to 19 patients (31.7%), while elevated CRP levels remained in 22 patients (36.7%). Increased homocysteine levels were observed in 26 patients (43.3%), elevated triglycerides in 25 patients (41.7%), and increased LDL levels in 23 patients (38.3%). In general, during the 6-month observation period a tendency toward normalization of inflammatory and lipid metabolism indicators was observed. The results of the 12-month follow-up showed that positive dynamics persisted for some indicators, although a partial increase was noted in several cases. Elevated ALT levels were recorded in 28 patients (46.7%), while increased AST levels were detected in 25 patients (41.7%). Elevated fibrinogen levels were observed in 23 patients (38.3%), and increased CRP levels were recorded in 27 patients (45.0%). Elevated homocysteine levels persisted in 30 patients (50.0%), while increased triglyceride levels were observed in 29 patients (48.3%) and elevated LDL levels in 27 patients (45.0%). These findings indicate that certain metabolic and inflammatory processes require long-term monitoring. According to the statistical analysis significant differences in the dynamics of the indicators were observed during the follow-up period (χ^2=5.44–7.01; $p<0.05$). This confirms that the changes in liver enzymes, inflammatory markers and lipid metabolism indicators during the observation period were statistically significant. Overall, the study results show that biochemical and inflammatory indicators in patients with ischemic heart disease may partially improve during treatment and follow-up, although some markers may remain elevated over a long period (Table 1).

Table 1 Dynamics of Laboratory Parameters (χ^2 analysis)

Indicator	Baseline n (%)	6 months n (%)	12 months n (%)	χ^2	p
Elevated ALT (>40 U/L)	38 (63.3%)	24 (40.0%)	28 (46.7%)	6.18	<0.05
Elevated AST (>40 U/L)	34 (56.7%)	21 (35.0%)	25 (41.7%)	5.72	<0.05
Elevated fibrinogen (>4 g/L)	32 (53.3%)	19 (31.7%)	23 (38.3%)	6.44	<0.05
Elevated CRP (>5 mg/L)	36 (60.0%)	22 (36.7%)	27 (45.0%)	7.01	<0.05
Elevated homocysteine (>15 µmol/L)	40 (66.7%)	26 (43.3%)	30 (50.0%)	6.92	<0.05
Elevated triglycerides (>1.7 mmol/L)	37 (61.7%)	25 (41.7%)	29 (48.3%)	5.86	<0.05
Elevated LDL-C (>3.4 mmol/L)	35 (58.3%)	23 (38.3%)	27 (45.0%)	5.44	<0.05

The results of the analysis showed that biochemical indicators associated with lipid metabolism, inflammatory processes, and endothelial dysfunction changed in a more unfavorable direction in the main group compared with the control group. In particular, the mean level of total cholesterol in the main group was 6.18 ± 0.86 mmol/L, which was significantly higher than the value recorded in the control group (5.42 ± 0.74 mmol/L) ($\chi^2 = 4.26$; $p < 0.05$). At the same time, triglyceride levels were also higher in the main group and amounted to 2.34 ± 0.51 mmol/L. In the control group, this indicator was recorded at 1.72 ± 0.39 mmol/L, and the difference between the groups was statistically significant ($\chi^2 = 7.11$; $p < 0.01$). The analysis of low-density lipoproteins (LDL-C) also demonstrated that the atherogenic lipid profile was more pronounced in the main group. Specifically, the LDL-C level in the main group was 3.98 ± 0.64 mmol/L, whereas in the control group it was observed at 3.21 ± 0.55 mmol/L ($\chi^2 = 4.08$; $p < 0.05$). In contrast, high-density lipoproteins (HDL-C), which have

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cardioprotective properties, showed an opposite tendency. In the main group, HDL-C levels were 0.94 ± 0.17 mmol/L, which was significantly lower than the 1.18 ± 0.22 mmol/L observed in the control group ($\chi^2 = 5.63$; $p < 0.05$).

Evaluation of the coagulation system and inflammatory processes also revealed significant differences between the groups. In particular, the fibrinogen level in the main group was 4.71 ± 0.79 g/L, which was significantly higher than the value in the control group (3.76 ± 0.63 g/L) ($\chi^2 = 6.48$; $p < 0.01$). The level of C-reactive protein (CRP), one of the markers of inflammation, was also higher in the main group, averaging 7.38 ± 2.11 mg/L. In the control group, this indicator was recorded at 4.65 ± 1.73 mg/L ($\chi^2 = 6.77$; $p < 0.01$). A similar tendency was observed in the analysis of homocysteine, an important biochemical marker associated with endothelial dysfunction. In the main group, the homocysteine level was 17.6 ± 3.7 µmol/L, whereas in the control group it was 12.9 ± 2.6 µmol/L, and this difference was statistically significant ($\chi^2 = 6.59$; $p < 0.01$).

Thus, the obtained results indicate that in patients with ischemic heart disease combined with steatohepatitis, metabolic changes associated with dyslipidemia, inflammatory processes, and endothelial dysfunction are more pronounced. This condition may represent an important pathogenetic factor contributing to the progression of atherosclerosis and the increased risk of cardiovascular complications in this group of patients.

Table 2 Comparative analysis of lipid profile, fibrinogen, C-reactive protein, and homocysteine between the groups

Indicator	Main group n = 60	Control group n = 60	χ^2	p
Total cholesterol, mmol/L	6.18 ± 0.86	5.42 ± 0.74	4.26	<0.05
Triglycerides, mmol/L	2.34 ± 0.51	1.72 ± 0.39	7.11	<0.01
LDL-C, mmol/L	3.98 ± 0.64	3.21 ± 0.55	4.08	<0.05
HDL-C, mmol/L	0.94 ± 0.17	1.18 ± 0.22	5.63	<0.05
Fibrinogen, g/L	4.71 ± 0.79	3.76 ± 0.63	6.48	<0.01
C-reactive protein, mg/L	7.38 ± 2.11	4.65 ± 1.73	6.77	<0.01
Homocysteine, µmol/L	17.6 ± 3.7	12.9 ± 2.6	6.59	<0.01

During the study, the condition of the vascular endothelium in patients with ischemic heart disease combined with steatohepatitis was assessed using biochemical markers. Factors reflecting endothelial dysfunction were evaluated through the analysis of inflammatory markers, hemostasis indicators, and lipid metabolism parameters. The obtained results demonstrated that biochemical changes associated with vascular wall function were more pronounced in the main group compared with the control group. In particular, the level of C-reactive protein, one of the key inflammatory markers, averaged 7.38 ± 2.11 mg/L in the main group, whereas in the control group this indicator was recorded at 4.65 ± 1.73 mg/L ($\chi^2 = 6.77$; $p < 0.01$). This finding indicates that inflammatory processes in the vascular wall were significantly more active in the main group. To evaluate the condition of the coagulation system, fibrinogen levels were also analyzed. In the main group, the fibrinogen concentration was 4.71 ± 0.79 g/L, while in the control group it was observed at 3.76 ± 0.63 g/L ($\chi^2 = 6.48$; $p < 0.01$). An increase in fibrinogen levels may indicate activation of inflammatory and hypercoagulable processes in the vascular wall. A similar trend was observed in the analysis of homocysteine, an

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important biochemical factor associated with endothelial dysfunction. In the main group, the homocysteine level was 17.6 ± 3.7 μmol/L, whereas in the control group it was 12.9 ± 2.6 μmol/L (χ^2 = 6.59; $p < 0.01$). It is well known that elevated homocysteine levels may exert a negative effect on vascular endothelium by reducing nitric oxide synthesis and accelerating atherosclerotic processes. Analysis of the lipid profile also confirmed changes related to endothelial function. In the main group, higher levels of triglycerides and low-density lipoproteins, together with lower levels of high-density lipoproteins, indicated the formation of an atherogenic lipid profile. This condition creates favorable circumstances for endothelial dysfunction and the progression of atherosclerosis. Overall, the study results indicate that signs of endothelial dysfunction are more pronounced in patients with ischemic heart disease combined with steatohepatitis. Increased levels of inflammatory markers, the presence of hypercoagulability, and elevated homocysteine concentrations are associated with impaired vascular endothelial function, which may contribute to the progression of atherosclerosis and increase the risk of cardiovascular complications.

Table 3 Dynamics of lipid and inflammatory markers at 6 and 12 months

Indicator	Main group 6 months (n=60)	Main group 12 months (n=60)	p	Control group 6 months (n=60)	Control group 12 months (n=60)	p
Total cholesterol, mmol/L	5.42 ± 0.74	5.08 ± 0.63	<0.05	4.96 ± 0.63	4.72 ± 0.55	<0.05
Triglycerides, mmol/L	1.92 ± 0.42	1.74 ± 0.39	<0.05	1.54 ± 0.34	1.42 ± 0.31	<0.05
LDL-C, mmol/L	3.41 ± 0.53	3.18 ± 0.47	<0.05	2.96 ± 0.49	2.82 ± 0.44	>0.05
HDL-C, mmol/L	1.02 ± 0.18	1.07 ± 0.19	>0.05	1.27 ± 0.21	1.32 ± 0.23	>0.05
Fibrinogen, g/L	4.08 ± 0.67	3.86 ± 0.58	>0.05	3.38 ± 0.52	3.19 ± 0.49	<0.05
C-reactive protein, mg/L	5.12 ± 1.74	4.21 ± 1.52	<0.01	3.62 ± 1.41	3.08 ± 1.24	<0.05
Homocysteine, μmol/L	15.1 ± 3.2	13.8 ± 2.9	<0.05	11.6 ± 2.1	10.9 ± 1.9	>0.05

In the following table, a comparative analysis of the dynamics of lipid metabolism and inflammatory markers in the main and control groups during the 6- and 12-month follow-up periods was performed. The obtained results demonstrated that, over time, positive changes were observed in several biochemical parameters in both groups. However, the manifestation and intensity of these dynamics differed between the groups, reflecting variations in metabolic status and inflammatory processes. First, when analyzing lipid metabolism indicators, it was found that in the main group the mean total cholesterol level at 6 months was 5.42 ± 0.74 mmol/L, which decreased to 5.08 ± 0.63 mmol/L after 12 months of follow-up ($p < 0.05$). A similar positive trend was observed in the control group, where cholesterol levels decreased from 4.96 ± 0.63 mmol/L to 4.72 ± 0.55 mmol/L ($p < 0.05$). Thus, although both groups demonstrated improvement in cholesterol metabolism during treatment and follow-up, the level remained relatively higher in the main group compared with the control group. The dynamics of triglyceride levels showed a similar pattern. In the main group, triglyceride levels decreased from 1.92 ± 0.42 mmol/L at 6 months to 1.74 ± 0.39 mmol/L after 12 months ($p < 0.05$). In the control group, triglycerides decreased from 1.54 ± 0.34 mmol/L to 1.42 ± 0.31 mmol/L ($p <$

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0.05). These findings indicate that the atherogenic components of lipid metabolism demonstrated a decreasing trend in both groups.

Analysis of low-density lipoproteins (LDL-C) also provided important information. In the main group, LDL-C levels decreased from 3.41 ± 0.53 mmol/L at 6 months to 3.18 ± 0.47 mmol/L at 12 months ($p < 0.05$). In the control group, LDL-C decreased from 2.96 ± 0.49 mmol/L to 2.82 ± 0.44 mmol/L; however, this difference did not reach statistical significance ($p > 0.05$). This suggests that the reduction of atherogenic lipid fractions was more pronounced in the main group. High-density lipoproteins (HDL-C), which have anti-atherogenic properties, demonstrated a slightly different trend. In the main group, HDL-C increased from 1.02 ± 0.18 mmol/L at 6 months to 1.07 ± 0.19 mmol/L at 12 months, although this change was not statistically significant ($p > 0.05$). In the control group, HDL-C increased from 1.27 ± 0.21 mmol/L to 1.32 ± 0.23 mmol/L, but here as well the difference was not statistically significant ($p > 0.05$). At the same time, the relatively higher HDL-C levels observed in the control group may indicate a better preservation of the anti-atherogenic lipid profile.

The analysis of biomarkers reflecting inflammatory and coagulation processes also revealed important findings. In the main group, the fibrinogen level at 6 months was 4.08 ± 0.67 g/L and decreased to 3.86 ± 0.58 g/L at 12 months. In the control group, fibrinogen levels decreased from 3.38 ± 0.52 g/L to 3.19 ± 0.49 g/L, and this difference was statistically significant ($p < 0.05$). These results indicate a partial reduction in coagulation activity during treatment.C-reactive protein (CRP), an important laboratory marker of inflammation, also demonstrated a decrease over time. In the main group, CRP levels decreased from 5.12 ± 1.74 mg/L at 6 months to 4.21 ± 1.52 mg/L at 12 months ($p < 0.01$). In the control group, CRP decreased from 3.62 ± 1.41 mg/L to 3.08 ± 1.24 mg/L ($p < 0.05$). This suggests that inflammatory activity decreased in both groups, although the inflammatory background remained more pronounced in the main group. Analysis of homocysteine levels also provided important information. In the main group, homocysteine decreased from 15.1 ± 3.2 µmol/L at 6 months to 13.8 ± 2.9 µmol/L at 12 months ($p < 0.05$). In the control group, homocysteine levels decreased from 11.6 ± 2.1 µmol/L to 10.9 ± 1.9 µmol/L; however, this change did not reach statistical significance ($p > 0.05$). It is well known that elevated homocysteine levels are an important biomarker associated with endothelial dysfunction and the progression of atherosclerotic processes.

Thus, a comparative analysis of the 6-month and 12-month follow-up results indicates that a positive trend in lipid profile and inflammatory markers was observed in both groups. However, in the main group, atherogenic lipid fractions, inflammatory markers, and homocysteine levels remained higher compared with the control group. This finding suggests that metabolic and inflammatory processes play an important role in the development of cardiovascular pathology and that such patients require long-term monitoring and comprehensive therapeutic management.

Conclusion:

In patients with ischemic heart disease combined with steatohepatitis, biochemical changes associated with dyslipidemia, inflammatory activity, hypercoagulation, and endothelial dysfunction are more pronounced compared with the control group. Although positive dynamics in most laboratory indicators were observed during the 6-month follow-up period, a partial increase in some markers at the 12-month stage indicates that metabolic and inflammatory processes remain unstable. The

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persistence of elevated levels of total cholesterol, triglycerides, LDL-C, fibrinogen, C-reactive protein, and homocysteine in the main group indicates a higher risk of atherosclerosis and cardiovascular complications in these patients. Therefore, in patients with ischemic heart disease combined with steatohepatitis, it is advisable to complement the standard cardiological approach with metabolic and hepatological monitoring, strengthen long-term follow-up, and implement individualized preventive and therapeutic strategies.

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