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LIVER FIBROSIS AND DISORDERS OF LIPID METABOLISM IN NON-ALCOHOLIC FATTY LIVER DISEASE

Raisa Sharobiddinovna Sayfutdinova
PhD (Medicine), Associate Professor, Department of Endocrinology,
Andijan State Medical Institute, Andijan, Republic of Uzbekistan

Zarnigor Azamatovna Nazirova
Assistant, Department of Endocrinology,
Andijan State Medical Institute, Andijan, Republic of Uzbekistan

Abstract: Objective. To evaluate the relationship between liver fibrosis stage and lipid metabolism parameters in patients with non-alcoholic fatty liver disease (NAFLD).

Materials and Methods. The study included 83 patients (33 men and 50 women) aged 29–72 years with confirmed NAFLD. Fibrosis stage was assessed by elastography (F0–F4). Anthropometric and biochemical parameters were measured, including body mass index, ALT, AST, total bilirubin, lipid profile (total cholesterol, triglycerides, HDL-C, LDL-C), and atherogenic index. Statistical analysis was performed using Pearson correlation.

Results. The mean age of patients was 45.8 ± 10.6 years, mean BMI — 31.2 ± 5.4 kg/m². Most patients (≈65%) had fibrosis stages F2–F3, while advanced fibrosis (F4) was found in 8%. Increasing fibrosis stage was associated with significant elevation of ALT and AST ($p < 0.05$), higher total cholesterol and triglycerides, lower HDL-C, and increased atherogenic index. Correlation analysis revealed positive associations between fibrosis stage and triglycerides ($r = 0.42$), LDL-C ($r = 0.37$), and a negative association with HDL-C ($r = –0.40$).

Conclusion. Progression of liver fibrosis in NAFLD is accompanied by pronounced disturbances in lipid metabolism. The most significant markers are elevated triglycerides and atherogenic index, along with reduced HDL-C. Comprehensive lipid profile assessment should be considered an essential component of diagnosis and monitoring in NAFLD patients.

Keywords: non-alcoholic fatty liver disease, liver fibrosis, lipid metabolism, triglycerides, atherogenic index.

Introduction. Non-alcoholic fatty liver disease (NAFLD) is one of the most prevalent disorders of the hepatobiliary system, closely associated with obesity, metabolic syndrome, and insulin resistance [2,3,4]. According to epidemiological studies, the global prevalence of NAFLD reaches 25–30% of the adult population, and in countries with high rates of obesity it exceeds 40% [4,5]. In Russia and other CIS countries, the incidence of NAFLD continues to rise, as confirmed by national guidelines and clinical studies [1,2,3].

Progression of the disease from simple steatosis to steatohepatitis and fibrosis is accompanied by alterations in lipid metabolism, which not only reflect the severity of hepatic injury but also contribute to the development of cardiovascular complications [7,10,19]. Dyslipidemia—manifested by elevated triglycerides, increased low-density lipoprotein cholesterol (LDL-C), and reduced high-density lipoprotein cholesterol (HDL-C)—is considered a key risk factor for fibrosis progression [8,13,14,20].

Current clinical guidelines emphasize the importance of comprehensive assessment of the lipid profile in patients with NAFLD for early identification of the risk of fibrotic changes in the liver [2,3,6]. Non-invasive diagnostic methods such as elastography and FibroScan allow simultaneous evaluation of fibrosis stage and metabolic parameters, thereby improving prognostic accuracy [3,9,11].

International studies also confirm the close relationship between fibrosis severity and lipid metabolism disturbances. Adams et al. demonstrated that dyslipidemia is an independent predictor of fibrosis progression [8], while Targher and Byrne highlighted the role of NAFLD as a multisystem disease strongly linked to increased cardiovascular risk [5,10]. A meta-analysis by Musso and colleagues showed that dynamic changes in the lipid profile may serve as a reliable indicator of unfavorable NAFLD outcomes [7].

Thus, investigation of the relationship between liver fibrosis stage and lipid metabolism parameters is of great importance for understanding the pathogenesis of NAFLD, predicting its course, and optimizing therapeutic strategies [10,13,14,19,20].

The objective of the study was to evaluate the relationship between the degree of liver fibrosis and lipid metabolism parameters in patients with non-alcoholic fatty liver disease (NAFLD).

Materials and Methods. A total of 83 patients with a confirmed diagnosis of NAFLD were included, comprising 33 men and 50 women aged between 29 and 72 years. The diagnosis was established on the basis of clinical data and ultrasound examination and was further confirmed by elastography (FibroScan), in accordance with current recommendations for non-invasive assessment of fibrosis. The stage of fibrosis was determined using the F0–F4 scale. Patients were enrolled if they had evidence of hepatic steatosis on ultrasound, a confirmed diagnosis of NAFLD, and the ability to undergo FibroScan examination. Exclusion criteria included viral hepatitis (HBV, HCV), alcoholic liver disease, autoimmune liver disorders, and the use of hepatotoxic medications, which is consistent with international standards for patient selection.

All participants underwent a comprehensive assessment of anthropometric and biochemical parameters, including body mass index, alanine aminotransferase, aspartate aminotransferase, total bilirubin, and a full lipid profile consisting of total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), as well as calculation of the atherogenic index. These parameters are recognized in the literature as key markers of metabolic disturbances in NAFLD and predictors of fibrosis progression.

Statistical analysis was performed using SPSS Statistics v.26.0 (IBM, USA). Quantitative variables were expressed as mean ± standard deviation. Correlation between fibrosis stage and lipid metabolism parameters was assessed using Pearson's correlation coefficient, with statistical significance set at $p < 0.05$, in line with accepted standards in clinical hepatology research. Thus, the study methodology was designed in accordance with contemporary national and international guidelines, ensuring the reliability of the obtained data and their comparability with results from other studies.

Results. The mean age of the patients was 45.8 ± 10.6 years, with a minimum age of 29 years and a maximum of 72 years. The age distribution showed a predominance of middle-aged individuals (35–55 years), which corresponds to published data indicating the highest prevalence of NAFLD in this age group [4,5]. Gender distribution revealed that women accounted for 60.2% of the cohort and men for 39.8%; however, no statistically significant differences in fibrosis stage were observed between sexes.

The mean body mass index (BMI) was 31.2 ± 5.4 kg/m², consistent with class I obesity. Among the study population, 22 patients (26.5%) had class II obesity (BMI ≥ 35 kg/m²), and 6 patients (7.2%) had class III obesity. Higher BMI values were associated with an increased likelihood of advanced fibrosis (F3–F4), which is in agreement with international studies [5,10].

FibroScan assessment demonstrated that the majority of patients (approximately 65%) had fibrosis stages F2–F3, indicating progressive disease. Early stages (F0–F1) were identified in 27% of patients, while advanced fibrosis (F4) was present in 8%.

Biochemical analysis revealed that serum ALT and AST levels increased significantly with advancing fibrosis stage ($p < 0.05$). In patients with F0–F1, mean ALT was 42 ± 8 U/L, compared with 68 ± 12 U/L in F2–F3 and 95 ± 15 U/L in F4. A similar trend was observed for AST, with values of 38 ± 7 U/L in F0–F1, 61 ± 10 U/L in F2–F3, and 88 ± 14 U/L in F4.

Progression of fibrosis was also accompanied by significant alterations in lipid metabolism. Total cholesterol increased from 4.3 ± 0.8 to 5.6 ± 0.9 mmol/L, triglycerides rose from 1.6 ± 0.4 to 2.4 ± 0.6 mmol/L, HDL-C decreased from 1.5 ± 0.2 to 1.1 ± 0.2 mmol/L, and the atherogenic index increased from 2.1 ± 0.4 to 3.6 ± 0.5. These findings confirm the role of lipid imbalance as a key factor in NAFLD progression [7,8,19].

Correlation analysis demonstrated a positive association between fibrosis stage and triglyceride levels ($r = 0.42$) as well as LDL-C ($r = 0.37$), and a negative correlation with HDL-C ($r = –0.40$). These results suggest that dyslipidemia may serve as an independent predictor of liver fibrosis progression (Tables 1, 2, 3).

Table 1.
Demographic characteristics of patients (n = 83)

Parameter	**Value**
Mean age, years	45.8 ± 10.6
Age range, years	29–72
Male	33 (39.8%)
Female	50 (60.2%)
Mean BMI, kg/m²	31.2 ± 5.4
Class I obesity	55 (66.3%)
Class II obesity	22 (26.5%)
Class III obesity	6 (7.2%)

Table 2.
Distribution of patients by fibrosis stage

Fibrosis stage	**Number of patients**	**%**
F0–F1	22	27%
F2–F3	54	65%

F4	7	8%

Table 3.
Biochemical parameters according to fibrosis stage

Parameter	**F0–F1**	**F2–F3**	**F4**
ALT, U/L	42 ± 8	68 ± 12	95 ± 15
AST, U/L	38 ± 7	61 ± 10	88 ± 14
Total cholesterol, mmol/L	4.3 ± 0.8	5.1 ± 0.7	5.6 ± 0.9
Triglycerides, mmol/L	1.6 ± 0.4	2.0 ± 0.5	2.4 ± 0.6
HDL-C, mmol/L	1.5 ± 0.2	1.3 ± 0.2	1.1 ± 0.2
Atherogenic index	2.1 ± 0.4	2.9 ± 0.5	3.6 ± 0.5

Thus, the findings of this study demonstrate that the progression of liver fibrosis in patients with non-alcoholic fatty liver disease is accompanied by adverse alterations in lipid metabolism, which further increase the risk of cardiovascular complications. The obtained results are consistent with both national and international studies [1–20] and confirm the necessity of comprehensive lipid profile assessment in the management of patients with NAFLD.

Discussion. The results obtained in this study demonstrate that the progression of liver fibrosis in patients with non-alcoholic fatty liver disease (NAFLD) is accompanied by adverse alterations in lipid metabolism. The mean age of the cohort was 45.8 years, which corresponds to national and international data indicating the highest prevalence of NAFLD among individuals aged 35–55 years [1,4,5]. The predominance of women in the sample did not exert a significant influence on fibrosis stage, which is consistent with clinical observations reported in the literature [2,3].

The observed association between increased body mass index and fibrosis severity confirms the role of obesity as a major risk factor for NAFLD progression [5,10]. Previous studies emphasize that visceral obesity and the resulting insulin resistance are key mechanisms underlying the development of steatohepatitis and fibrosis [4,6,7].

Biochemical markers of liver injury (ALT and AST) increased significantly with advancing fibrosis stage, in agreement with clinical guidelines and numerous studies [2,3,8,9]. More importantly, dynamic changes in the lipid profile were identified, including elevated total cholesterol and triglycerides, reduced HDL-C, and increased atherogenic index. These alterations

reflect not only fibrosis progression but also the emergence of heightened cardiovascular risk [10,13,14,19,20].

Correlation analysis confirmed a positive relationship between fibrosis stage and triglyceride as well as LDL-C levels, and a negative relationship with HDL-C. Similar findings have been reported in international studies, where dyslipidemia was considered an independent predictor of unfavorable NAFLD outcomes [7,8,10,20]. In particular, Adams et al. demonstrated that lipid imbalance accelerates fibrosis progression independently of transaminase levels [8], while Targher and Byrne highlighted the role of NAFLD as a multisystem disease strongly associated with increased cardiovascular risk [5,10].

Taken together, our findings underscore the necessity of comprehensive lipid profile assessment in patients with NAFLD. Incorporating lipid metabolism parameters into risk stratification algorithms may improve the accuracy of fibrosis progression prediction and enable timely therapeutic interventions [2,3,6,9,11,15].

Conclusion. The present study demonstrated that the progression of liver fibrosis in patients with non-alcoholic fatty liver disease (NAFLD) is accompanied by pronounced disturbances in lipid metabolism. The mean age of the examined cohort was 45.8 years, and the mean body mass index corresponded to class I obesity, confirming the close association of NAFLD with metabolic syndrome and obesity.

The majority of patients were found to have fibrosis stages F2–F3, while advanced fibrosis (F4) was observed in 8% of cases. Increasing fibrosis stage was associated with a statistically significant elevation of ALT and AST levels, consistent with both national and international studies.

The most notable changes in the lipid profile included increased total cholesterol and triglycerides, reduced HDL-C, and a higher atherogenic index. Correlation analysis confirmed a positive association between fibrosis stage and triglyceride as well as LDL-C levels, and a negative association with HDL-C. These findings suggest that dyslipidemia may serve as an independent predictor of liver fibrosis progression.

Therefore, comprehensive assessment of the lipid profile in patients with NAFLD should be considered an essential component of clinical practice. Incorporating lipid metabolism parameters into diagnostic and monitoring algorithms will allow more accurate prediction of disease progression, timely identification of patients at high risk of cardiovascular complications, and optimization of therapeutic strategies.

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