



## Frequency of Dyslipidemia in Non-Alcoholic Fatty Liver Disease Patients

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### Declaration

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### ABSTRACT

**Background and Objective:** Non-alcoholic fatty liver disease (NAFLD) is frequently associated with dyslipidemia, contributing to increased cardiovascular risk. This study aimed to assess the prevalence and patterns of dyslipidemia in NAFLD patients and its association with hepatic steatosis severity and clinical risk factors. **Materials and Methods:** A descriptive cross-sectional study was conducted over six months (March 2024 to August 2024) at the Department of Medicine, National Hospital and Medical Center, Lahore. A total of 116 adults with ultrasonographically confirmed NAFLD were enrolled using consecutive sampling. Baseline clinical and demographic data were collected. Fasting lipid profiles were obtained, and dyslipidemia was defined according to established guidelines. Hepatic steatosis was graded as mild, moderate, or severe. Data analysis was performed using SPSS version 25. **Results:** Dyslipidemia was identified in 86 (74.1%) patients. Elevated triglycerides (57.8%) and low HDL cholesterol (55.2%) were the most frequent abnormalities, followed by elevated LDL cholesterol (50.0%) and total cholesterol (44.8%). Multiple lipid abnormalities were observed in 48 (41.4%) patients. The prevalence of dyslipidemia increased with steatosis severity: 58.8% in mild, 76.9% in moderate, and 86.7% in severe cases ( $p = 0.008$ ). Elevated triglycerides and low HDL cholesterol were also significantly associated with steatosis grade ( $p = 0.003$  and  $p = 0.019$ , respectively). Dyslipidemia was more common in males (80.9% vs 64.6%,  $p = 0.041$ ), patients aged  $\geq 45$  years (81.4% vs 68.5%,  $p = 0.032$ ), obese individuals (88.2%,  $p = 0.004$ ), and those with a family history of dyslipidemia (87.8%,  $p = 0.014$ ). **Conclusion:** Dyslipidemia is highly prevalent in NAFLD and correlates with steatosis severity and key metabolic risk factors. Regular lipid screening and comprehensive management are essential in this population.

### INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) has become one of the most widespread liver disorders across the world, now estimated to affect roughly one in four adults globally [1]. The condition covers a range of liver changes, beginning with simple fat accumulation and sometimes progressing to more severe stages such as non-alcoholic steatohepatitis, fibrosis, cirrhosis, and even liver cancer [2,3]. The causes of NAFLD are complex and involve a mix of genetic, metabolic, and environmental influences that together contribute to the onset and advancement of the disease [4].

Obesity is recognized as a key factor that raises the risk of NAFLD, largely due to its strong relationship with metabolic syndrome, which includes problems like insulin resistance, altered blood lipids, and high blood pressure [3,5]. Abnormalities in blood lipids, including higher levels of total cholesterol, LDL cholesterol, and triglycerides as well as lower HDL cholesterol, are well known to increase the risk of cardiovascular disease and are also closely linked to the development and worsening of NAFLD [6].

Emerging evidence suggests that dyslipidemia, particularly hypertriglyceridemia and low HDL-C levels, are frequent in patients with NAFLD, and may contribute to hepatic steatosis, inflammation, and fibrosis [7]. Dyslipidemia is associated with an increased risk of NAFLD, and in turn, NAFLD can exacerbate dyslipidemia, creating a vicious cycle that may accelerate liver disease progression [6,8]. Furthermore, recent studies have identified genetic variants and molecular mechanisms that may link dyslipidemia and NAFLD, highlighting the importance of lipid metabolism in the pathogenesis of liver disease [9,10]. Liver biopsy (LB) is considered the gold standard for the diagnosis and staging of liver diseases, although it is associated with several drawbacks, including pain, cost, and a high rate of complications, leading to patient discomfort and distress [11]. Non-invasive diagnostic methods, such as ultrasonography combined with a significantly elevated fasting lipid profile, have emerged as safe, affordable, convenient, and accurate alternatives for the diagnosis of non-alcoholic fatty liver disease (NAFLD) [12].

Understanding this association could have significant consequences for public health, as it could inform strategies to lessen the impact of NAFLD, identify at-risk populations, and improve patient management by enabling healthcare professionals to develop personalized treatment plans. The findings from this study can serve as a basis for future longitudinal and interventional studies.

## MATERIALS AND METHODS

This study was descriptive cross-sectional study conducted at the Department of Medicine at National Hospital and Medical Center, Lahore. The study was conducted over a duration of 6 months from March 2024 to August 2024. The sample size was calculated to be 116, assuming a dyslipidemia prevalence of 26%, with a 95% confidence level and an 8% margin of error [13]. A non-probability consecutive sampling technique was employed to recruit eligible patients.

Patients aged 18 to 60 years of either gender were included if they had a diagnosis of non-alcoholic fatty liver disease (NAFLD) confirmed by ultrasonography or other imaging modalities. Patients were excluded if they had a history of significant alcohol consumption (more than 14 drinks per week for men, or more than 7 drinks per week for women), were using medications known to affect lipid metabolism (such as amiodarone, corticosteroids, tamoxifen, methotrexate, oral contraceptives, statins, fibrates, or bile acid sequestrants within the previous three months), or had a history of viral hepatitis (HBV, HCV), autoimmune hepatitis, drug-induced liver injury, chronic kidney disease, malignancy, or thyroid abnormalities. Pregnant and lactating women were also excluded.

After obtaining informed consent, baseline demographic and clinical data including age, gender, height, body weight, smoking habits, and family history of dyslipidemia were recorded. The diagnosis of NAFLD was established using ultrasonography or other imaging, and the grading of hepatic steatosis was performed according to standard sonographic features. Ultrasonography was performed by a single experienced radiologist using a Toshiba Xario 100 system, and hepatic steatosis was classified as mild, moderate, or severe based on established echogenicity criteria. Blood samples were collected following an overnight fast for lipid profile analysis, including triglycerides, HDL cholesterol, LDL cholesterol, and total cholesterol. Serum was separated by centrifugation at 3000 rpm for 15 minutes and stored at -80°C until analysis. Lipid parameters were measured using a Micro-lab 300 analyzer with colorimetric enzymatic assays. Dyslipidemia was defined according to AACE 2017 guidelines: triglycerides  $\geq 150$  mg/dL, HDL cholesterol  $< 40$  mg/dL for men or  $< 50$  mg/dL for women, LDL cholesterol  $\geq 130$  mg/dL, or total cholesterol  $> 200$  mg/dL.

Statistical analysis was performed using SPSS version 25.0. Baseline characteristics were summarized using descriptive statistical methods. For continuous variables, results were reported as mean with standard deviation, while categorical variables were expressed as frequencies and percentages. Associations between dyslipidemia and the severity of hepatic steatosis, along with other factors such as age, gender, body mass index, and smoking status,

were evaluated through subgroup analyses. Statistical significance was determined at a p-value threshold of 0.05.

## RESULTS

A total of 116 patients with NAFLD were enrolled in this cross-sectional study. The mean age of participants was  $42.8 \pm 11.7$  years, with 68 (58.6%) being male and 48 (41.4%) being female. The majority of patients were overweight or obese, with a mean BMI of  $28.4 \pm 4.2$  kg/m<sup>2</sup>. Regarding hepatic steatosis severity, 34 (29.3%) patients had mild steatosis, 52 (44.8%) had moderate steatosis, and 30 (25.9%) had severe steatosis. A family history of dyslipidemia was present in 41 (35.3%) patients, while 29 (25.0%) patients were current smokers.

**Table 1**

*Baseline Characteristics of Study Participants (n=116)*

| Variable                       | Frequency       | Percentage (%) |
|--------------------------------|-----------------|----------------|
| Age (years)                    | 42.8 $\pm$ 11.7 | -              |
| Gender                         |                 |                |
| Male                           | 68              | 58.6           |
| Female                         | 48              | 41.4           |
| BMI (kg/m <sup>2</sup> )       | 28.4 $\pm$ 4.2  | -              |
| BMI Categories                 |                 |                |
| Normal (18.5-24.9)             | 18              | 15.5           |
| Overweight (25.0-29.9)         | 64              | 55.2           |
| Obese ( $\geq 30.0$ )          | 34              | 29.3           |
| Hepatic Steatosis Severity     |                 |                |
| Mild                           | 34              | 29.3           |
| Moderate                       | 52              | 44.8           |
| Severe                         | 30              | 25.9           |
| Family History of Dyslipidemia | 41              | 35.3           |
| Smoking Status                 | 29              | 25.0           |

The mean lipid profile parameters revealed significant abnormalities in the study population. Total cholesterol levels averaged  $201.6 \pm 38.4$  mg/dL, with triglycerides showing a mean of  $168.7 \pm 52.3$  mg/dL. HDL cholesterol was notably low at  $38.2 \pm 9.6$  mg/dL, while LDL cholesterol averaged  $135.8 \pm 32.1$  mg/dL. Dyslipidemia was present in 86 (74.1%) patients. Elevated triglycerides were observed in 67 (57.8%), low HDL cholesterol in 64 (55.2%), elevated LDL cholesterol in 58 (50.0%), and elevated total cholesterol in 52 (44.8%) patients. Multiple lipid abnormalities were identified in 48 (41.4%) patients (Table 2).

**Table 2**

*Frequency of Dyslipidemia Components*

| Dyslipidemia Component                        | Frequency | Percentage (%) |
|---|-----------|----------------|
| Overall Dyslipidemia                          | 86        | 74.1           |
| Individual Components:                        |           |                |
| Elevated Triglycerides ( $\geq 150$ mg/dL)    | 67        | 57.8           |
| Low HDL Cholesterol                           | 64        | 55.2           |
| - Men ( $< 40$ mg/dL)                         | 42        | 61.8           |
| - Women ( $< 50$ mg/dL)                       | 22        | 45.8           |
| Elevated LDL Cholesterol ( $\geq 130$ mg/dL)  | 58        | 50.0           |
| Elevated Total Cholesterol ( $> 200$ mg/dL)   | 52        | 44.8           |
| Multiple Abnormalities ( $\geq 2$ components) | 48        | 41.4           |

A significant association was found between dyslipidemia and severity of hepatic steatosis ( $p = 0.008$ ). Dyslipidemia was present in 20 (58.8%) patients with mild steatosis, 40 (76.9%) with moderate steatosis, and 26 (86.7%) with severe steatosis. Elevated triglycerides were detected in 14 (41.2%) cases of mild, 32 (61.5%) moderate, and 23

(76.7%) severe steatosis ( $p = 0.003$ ). Low HDL cholesterol was observed in 14 (41.2%) mild, 30 (57.7%) moderate, and 21 (70.0%) severe steatosis cases ( $p = 0.019$ ).

**Table 3**

*Association Between Dyslipidemia and Hepatic Steatosis Severity*

| Dyslipidemia Component     | Mild Steatosis (n=34) | Moderate Steatosis (n=52) | Severe Steatosis (n=30) | p-value |
|----------------------------|-----------------------|---------------------------|-------------------------|---------|
| Overall Dyslipidemia       | 20 (58.8%)            | 40 (76.9%)                | 26 (86.7%)              | 0.008   |
| Elevated Triglycerides     | 14 (41.2%)            | 32 (61.5%)                | 23 (76.7%)              | 0.003   |
| Low HDL Cholesterol        | 14 (41.2%)            | 30 (57.7%)                | 21 (70.0%)              | 0.019   |
| Elevated LDL Cholesterol   | 14 (41.2%)            | 26 (50.0%)                | 18 (60.0%)              | 0.248   |
| Elevated Total Cholesterol | 12 (35.3%)            | 24 (46.2%)                | 16 (53.3%)              | 0.312   |

Male patients exhibited a higher prevalence of dyslipidemia than females (80.9% vs 64.6%,  $p = 0.041$ ), mainly due to increased rates of elevated triglycerides (66.2% vs 45.8%,  $p = 0.026$ ) and low HDL cholesterol (61.8% vs 45.8%,  $p = 0.089$ ). Dyslipidemia was more common in patients aged  $\geq 45$  years compared to those  $< 45$  years (81.4% vs 68.5%,  $p = 0.032$ ). Prevalence was highest in obese individuals (88.2%), followed by overweight (73.4%) and normal weight (50.0%) patients ( $p = 0.004$ ). A family history of dyslipidemia was associated with increased prevalence (87.8% vs 66.7%,  $p = 0.014$ ). Smokers showed a non-significant trend toward higher dyslipidemia (82.8% vs 71.3%,  $p = 0.218$ ) (Table 4).

**Table 4**

*Subgroup Analysis of Dyslipidemia among demographic characteristics.*

| Variable               | Dyslipidemia Present | Dyslipidemia Absent | p-value |
|------------------------|----------------------|---------------------|---------|
| Gender                 |                      |                     | 0.041   |
| Male (n=68)            | 55 (80.9%)           | 13 (19.1%)          |         |
| Female (n=48)          | 31 (64.6%)           | 17 (35.4%)          |         |
| Age Groups             |                      |                     | 0.032   |
| $< 45$ years (n=65)    | 44 (68.5%)           | 21 (31.5%)          |         |
| $\geq 45$ years (n=51) | 42 (81.4%)           | 9 (18.6%)           |         |
| BMI Categories         |                      |                     | 0.004   |
| Normal (n=18)          | 9 (50.0%)            | 9 (50.0%)           |         |
| Overweight (n=64)      | 47 (73.4%)           | 17 (26.6%)          |         |
| Obese (n=34)           | 30 (88.2%)           | 4 (11.8%)           |         |
| Smoking Status         |                      |                     | 0.218   |
| Smoker (n=29)          | 24 (82.8%)           | 5 (17.2%)           |         |
| Non-smoker (n=87)      | 62 (71.3%)           | 25 (28.7%)          |         |

## DISCUSSION

The findings of this study demonstrate a high prevalence of dyslipidemia among patients with non-alcoholic fatty liver disease (NAFLD), with 74.1% of individuals exhibiting abnormal lipid profiles. The most frequent lipid abnormalities observed were elevated triglycerides (57.8%) and low HDL cholesterol (55.2%), while elevated LDL cholesterol and total cholesterol were noted in 50.0% and 44.8% of patients, respectively. Notably, multiple lipid derangements were present in over two-fifths of the

population, highlighting the complex metabolic profile frequently encountered in NAFLD.

These results are broadly consistent with the patterns reported in both global and regional literature. Internationally, studies have shown that NAFLD is commonly associated with atherogenic dyslipidemia, characterized by high triglycerides, low HDL cholesterol, and increased levels of small dense LDL particles. A systematic review found that the prevalence of low HDL cholesterol in NAFLD ranged from 61–72%, while high triglycerides were reported in 65–66% of cases and elevated total cholesterol in 64% of patients [14–16]. The present study's prevalence rates align closely with these observations, especially regarding low HDL and high triglyceride levels. Furthermore, research has highlighted that the presence of multiple lipid abnormalities is frequent among NAFLD patients, emphasizing the need for comprehensive metabolic assessment in this population [17–20].

The regional data from Pakistan, however, show certain differences in reported frequencies. One recent study identified low HDL cholesterol in 61.3% of NAFLD patients, but the rates of general hyperlipidemia, hypertriglyceridemia, and combined lipid abnormalities were comparatively lower than those observed in the current study, with combined hyperlipidemia reported in only 3.7% and hypertriglyceridemia in 2% of cases [15]. These discrepancies may be attributed to variations in patient selection, diagnostic criteria, and underlying population risk factors. Nonetheless, the consistent finding across both international and Pakistani cohorts is the predominance of low HDL and elevated triglycerides as the principal lipid abnormalities in NAFLD [21,22].

An association between the severity of hepatic steatosis and dyslipidemia was apparent in this study. The frequency of dyslipidemia increased with the degree of steatosis: 58.8% in mild, 76.9% in moderate, and 86.7% in severe cases. These results align with large-scale studies reporting a positive correlation with dyslipidemic and increased hepatic fat accumulation [17,20]. Moreover, the present results indicate that the progression of hepatic steatosis is associated not only with higher overall dyslipidemia rates but also with more pronounced elevations in triglycerides and further reductions in HDL cholesterol. These trends have been corroborated by recent literature, which suggests that liver fat content itself is a strong determinant of atherogenic lipid changes, independent of obesity or glycemic control [22,23]. Mechanistically, these associations may be explained by increased hepatic triglyceride synthesis and augmented very low-density lipoprotein (VLDL) secretion, which are hallmarks of NAFLD pathogenesis [15,18].

Dyslipidemia was found more often among men than women, with a noticeably higher occurrence in male patients. Much of this difference was linked to a greater frequency of raised triglycerides and lower HDL cholesterol in men. These findings are similar to previous studies, which have also reported that men having NAFLD generally display less favorable cardiometabolic profiles compared to their female counterparts [24,25]. Age and body mass index (BMI) also demonstrated strong associations, with dyslipidemia occurring more frequently



in individuals aged 45 years or older and in those with obesity. These results reflect the documented role of age-related hormonal and metabolic changes, as well as adiposity, in increasing both NAFLD and its associated dyslipidemia [15,18].

The clinical implications of these findings are important. Dyslipidemia is not only a common comorbidity in NAFLD but also a driver of adverse hepatic and cardiovascular outcomes. Elevated triglycerides and low HDL cholesterol, as shown in this and other studies, are associated with increased risk of diabetes, hypertension, and, most critically, cardiovascular disease the primary cause of morbidity and mortality in NAFLD

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