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Prevalence of Dyslipidemia in Patients with Non-Alcoholic Steatohepatitis and Its Association with Metabolic Syndrome

Zain Ul Abedeen
Resident Physician General Medicine, Jinnah Postgraduate Medical Centre, Karachi, Pakistan

Dr Dheeraj Kumar*
Postgraduate Trainee Medicine, Sandeman provincial Hospital (Civil Hospital), Quetta, Pakistan

Taimoor Irfan
Faisalabad Medical University, Faisalabad, Pakistan

Jude Tareq Badawi
Health Education Profession Center/Medicine program, Ibn Sina National College for Medical Studies ,Jeddah, Saudi Arabia

Hamza Hameed
Mphil Biochemistry and Molecular Biology,
Quaid-i-Azam University, Islamabad

Abstract

Background: Non-alcoholic steatohepatitis (NASH) is a progressive form of non-alcoholic fatty liver disease strongly linked with metabolic dysfunction. **Objective:** To determine the prevalence of dyslipidemia in patients with NASH and evaluate its association with metabolic syndrome. **Methods:** A cross-sectional analytical study was conducted at Sandeman provincial Hospital (Civil Hospital), Quetta from October 2024 to March 2025. This study conducted on 225 adult patients diagnosed with NASH. Anthropometric measurements, metabolic parameters, and complete lipid profiles were recorded. Dyslipidemia was defined using standard lipid thresholds. Metabolic syndrome was diagnosed using established clinical criteria. **Results:** Dyslipidemia was present in 181 patients (80.4%). Elevated triglycerides were the most prevalent abnormality (74.7%), followed by low HDL cholesterol (67.5%), elevated total cholesterol (49.8%), and elevated LDL cholesterol (46.2%). Metabolic syndrome was diagnosed in 171 patients (76%). Patients with metabolic syndrome exhibited significantly higher triglyceride levels (214.5 ± 58.2 mg/dL vs. 151.4 ± 42.6 mg/dL), lower HDL cholesterol (37.4 ± 8.1 mg/dL vs. 45.1 ± 9.0 mg/dL), and higher total and LDL cholesterol (p = 0.001–0.004) compared with those without metabolic syndrome. Multivariate analysis demonstrated that metabolic syndrome was an independent predictor of dyslipidemia (adjusted OR 3.42, 95% CI 1.88–6.22). **Conclusion:** Dyslipidemia is highly prevalent among NASH patients, particularly in those with metabolic syndrome. The strong association between these conditions emphasizes the need for early metabolic screening and integrated management targeting lipid abnormalities, obesity, and insulin resistance to reduce hepatic and cardiovascular complications.

Author Details

Keywords: non-alcoholic steatohepatitis, dyslipidemia, metabolic syndrome, triglycerides, HDL cholesterol, cardiovascular risk

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Corresponding E-mail & Author*:
Dr Dheeraj Kumar*
Postgraduate Trainee Medicine, Sandeman provincial Hospital (Civil Hospital), Quetta
Email:dheerajdodeja2019@gmail.com
orcid: 0009-0002-2703-1133

Introduction

Non-alcoholic fatty liver disease (NAFLD) has rapidly evolved into a major public health challenge, now recognized as the leading cause of chronic liver disease globally. NAFLD, with an estimated prevalence of 25-30 percent of the global population, is an indication of the increased rates of obesity, type 2 diabetes mellitus, and sedentary living [1]. Non-alcoholic steatohepatitis (NASH) is a type of the inflammatory progressive form of steatohepatitis that is marked by the ballooning of hepatocellular, lobular inflammation, the presence of fat deposits, and the presence of fibrosis at different stages. In comparison with simple steatosis, NASH is at very high risk of developing cirrhosis, portal hypertension, end-stage liver disease, and hepatocellular carcinoma [2]. Furthermore, NASH patients are at an extremely high risk of developing cardiovascular disease that is the main cause of death in this group. NASH pathogenesis is multifaceted: insulin resistance, dysfunction of adipose tissues, oxidative stress, mitochondrial damage, and dysregulated lipid metabolism play a role in its pathogenesis [3]. The cause of hepatic steatosis is the failure of lipid influx, synthesis, oxidation and export to balance [4]. Hepatic injury is further worsened by inflammatory cytokines, reactive oxygen species and fibrogenic processes as the disease advances to NASH. It is in this metabolic and inflammatory environment that dyslipidemia does not only form a comorbidity but is a major mechanistic element that connects liver disease with systemic cardiometabolic dysfunction [5]. NASH is generally characterized by dyslipidemia which is in the form of elevated levels of serum triglycerides and low levels of high-density lipoprotein (HDL) cholesterol and raised levels of small dense low-density lipoprotein (LDL) particles [6]. These lipid disorders represent the extreme insulin resistance of NASH that amplifies lipolysis within adipose tissue, intensifies the free fatty acid flux to the liver, and changes hepatic lipid metabolism [7]. This lipid profile which is atherogenic in nature is a direct contributor to cardiovascular morbidity, and perhaps, contributes to the increased pace of hepatic inflammation and fibrosis [8]. It is only indicated that the degree of dyslipidemia is related to histological development so that early detection is clinically significant [9]. The percentage of patients with NASH that present the characteristics of metabolic syndrome, a group of cardiometabolic risk factors characterized by obesity of central type, hyperglycemia, hypertension, hypertriglyceridemia, and the low level of HDL cholesterol, is significant [10]. Metabolic syndrome and NASH have the same pathogenic pathways with the most prevalent ones being insulin resistance and chronic low-grade inflammation [11]. The simultaneous occurrence of these conditions forms a feedback mechanism that aggravates hepatic and cardiovascular outcomes. Metabolic syndrome is a condition that not only predisposes the disease to NASH but also the accelerated development of advanced fibrosis and cirrhosis [12]. Simultaneously, NASH as such can be a contributor to systemic metabolic derangements as it enhances hepatic insulin resistance and unregulated lipid metabolism [13].

Objectives

To determine the prevalence of dyslipidemia in patients with NASH and evaluate its association with metabolic syndrome.

Methodology

This was a cross-sectional analytical study was conducted at Sandeman provincial Hospital (Civil Hospital), Quetta from October 2024 to March 2025. A total of 225 adult patients were included in the study. All patients had a confirmed diagnosis of NASH based on either liver biopsy findings or validated non-invasive diagnostic

criteria, including imaging evidence of hepatic steatosis combined with elevated liver enzymes and the exclusion of secondary causes.

Inclusion Criteria

Patients aged 18 years and older, diagnosed with NASH, and willing to undergo a complete clinical and biochemical assessment were included. Only those with sufficient clinical documentation and complete lipid profile data were considered eligible. Participants were required to have controlled alcohol intake below established diagnostic thresholds to exclude alcoholic liver disease.

Exclusion Criteria

Patients with significant alcohol consumption, known chronic liver diseases such as viral hepatitis, autoimmune hepatitis, Wilson's disease, hemochromatosis, or drug-induced liver injury were excluded. Individuals already on lipid-lowering medications, or those with incomplete metabolic or biochemical records, were also excluded to prevent confounding.

Data Collection Procedure

Fine-tuning demographic data, anthropometric data (weight, height, BMI, and waist circumference), blood pressure values and complete medical history with diabetes, high blood pressure, and the use of medication were recorded. Lipid profile analysis was determined using fasting blood samples as a research variable (total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides). To assess metabolic condition, liver enzymes (ALT, AST), fasting glucose, and HbA1c were also assessed. The condition of metabolic syndrome was established according to the set clinical parameters. The definition of dyslipidemia was made according to the standard lipid levels of high triglycerides, low HDL cholesterol, high LDL cholesterol or high total cholesterol. The diagnosis of metabolic syndrome was based on the international criteria that involve the presence of three elements, such as central obesity, hyperglycemia, hypertension, hypertriglyceridemia, and low levels of HDL. The application of all diagnostic cut-offs was similar throughout the study sample.

Statistical Analysis

Data were entered and analyzed using SPSS v24.0. Descriptive statistics, including means, standard deviations, frequencies, and percentages, were used to summarize baseline variables. The prevalence of dyslipidemia was calculated for the overall study population and stratified by metabolic syndrome status. The association between dyslipidemia and metabolic syndrome was assessed using chi-square tests for categorical variables and independent t-tests for continuous variables. A p-value < 0.05 was considered statistically significant.

Results

Dta were collected from 225 patients, mean age of the cohort was 49.8 ± 11.6 years, and the sex distribution was relatively balanced, with 118 males (52.4%) and 107 females (47.6%). The population demonstrated a high burden of adiposity, reflected by a mean BMI of 31.4 ± 5.2 kg/m² and an average waist circumference of 101.6 ± 10.8 cm. Metabolic comorbidities were common, with diabetes mellitus present in

138 patients (61.3%) and hypertension observed in 122 patients (54.2%). Liver enzyme levels were mildly to moderately elevated, with a mean ALT of 67.5 ± 28.9 U/L and a mean AST of 55.8 ± 23.5 U/L. Central obesity was present in 186 patients (82.7%), hypertriglyceridemia in 168 patients (74.7%), and low HDL cholesterol in 152 patients (67.5%). Hypertension, considered both as a comorbidity and a metabolic syndrome component, was present in 122 patients (54.2%). Elevated fasting glucose (≥100 mg/dL) was recorded in 159 patients (70.7%).

Table 1. Baseline Characteristics of Patients (N = 225)

Variable	**Mean ± SD / n (%)**
Age (years)	49.8 ± 11.6
Sex (Male/Female)	118 (52.4%) / 107 (47.6%)
BMI (kg/m²)	31.4 ± 5.2
Waist circumference (cm)	101.6 ± 10.8
Diabetes mellitus	138 (61.3%)
Hypertension	122 (54.2%)
ALT (U/L)	67.5 ± 28.9
AST (U/L)	55.8 ± 23.5
Metabolic syndrome	
Central obesity	186 (82.7%)
Hypertriglyceridemia	168 (74.7%)
Low HDL cholesterol	152 (67.5%)
Hypertension	122 (54.2%)
Fasting glucose ≥100 mg/dL	159 (70.7%)
Metabolic syndrome (≥3 components)	171 (76.0%)

The mean total cholesterol level was 203.4 ± 38.5 mg/dL, with 112 patients (49.8%) classified as having elevated levels. LDL cholesterol averaged 129.2 ± 32.1 mg/dL, with 104 patients (46.2%) showing abnormal values. HDL cholesterol levels averaged 39.6 ± 8.7 mg/dL, and low HDL was identified in 152 patients (67.5%). Triglycerides had a mean value of 198.7 ± 61.4 mg/dL, with 168 patients (74.7%) demonstrating hypertriglyceridemia.

Table 2. Lipid Profile Distribution in NASH Patients (N = 225)

Lipid Parameter	**Mean ± SD**	**Abnormal n (%)**
Total cholesterol (mg/dL)	203.4 ± 38.5	112 (49.8%)
LDL cholesterol (mg/dL)	129.2 ± 32.1	104 (46.2%)
HDL cholesterol (mg/dL)	39.6 ± 8.7	152 (67.5%)
Triglycerides (mg/dL)	198.7 ± 61.4	168 (74.7%)
Overall dyslipidemia	—	181 (80.4%)

Individuals with metabolic syndrome showed significantly higher total cholesterol levels (208.6 ± 37.9 mg/dL) compared with those without (186.9 ± 33.4 mg/dL; $p = 0.001$). LDL cholesterol was also higher in the metabolic syndrome group (133.4 ± 31.5 mg/dL vs. 116.2 ± 28.1 mg/dL; $p = 0.004$). Conversely, HDL cholesterol levels were significantly lower among patients with metabolic syndrome (37.4 ± 8.1 mg/dL) than among those without it (45.1 ± 9.0 mg/dL; $p < 0.001$). Triglyceride levels

showed the most notable difference, with patients having metabolic syndrome demonstrating markedly higher values (214.5 ± 58.2 mg/dL) compared with patients without metabolic syndrome (151.4 ± 42.6 mg/dL; $p < 0.001$). Overall dyslipidemia was present in 149 out of 171 patients with metabolic syndrome (87.1%) compared with 32 out of 54 patients without metabolic syndrome (59.2%), a statistically significant difference ($p < 0.001$).

Table 3. Comparison of Lipids in Patients with and Without Metabolic Syndrome

Variable	Metabolic Syndrome (n = 171) Mean ± SD	No Metabolic Syndrome (n = 54) Mean ± SD	p-value
Total cholesterol (mg/dL)	208.6 ± 37.9	186.9 ± 33.4	0.001
LDL cholesterol (mg/dL)	133.4 ± 31.5	116.2 ± 28.1	0.004
HDL cholesterol (mg/dL)	37.4 ± 8.1	45.1 ± 9.0	<0.001
Triglycerides (mg/dL)	214.5 ± 58.2	151.4 ± 42.6	<0.001
Overall dyslipidemia (%)	149 (87.1%)	32 (59.2%)	<0.001

Metabolic syndrome emerged as the strongest independent predictor of dyslipidemia, with an adjusted odds ratio of 3.42 (95% CI 1.88–6.22, $p < 0.001$). Obesity, defined as BMI ≥30 kg/m², was also significantly associated with dyslipidemia (OR 1.96, 95% CI 1.10–3.51, $p = 0.02$). Diabetes mellitus was another significant predictor (OR 2.21, 95% CI 1.20–4.06, $p = 0.01$). Hypertension demonstrated a non-significant trend toward association (OR 1.58, $p = 0.11$), while age ≥50 years was not significantly associated with dyslipidemia (OR 1.31, $p = 0.36$).

Table 4. Multivariate Logistic Regression Predicting Dyslipidemia

Predictor	Adjusted OR	95% CI	p-value
Metabolic syndrome	3.42	1.88–6.22	<0.001
BMI ≥30 kg/m²	1.96	1.10–3.51	0.02
Diabetes mellitus	2.21	1.20–4.06	0.01
Hypertension	1.58	0.89–2.81	0.11
Age ≥50 years	1.31	0.72–2.38	0.36

Discussion

This study evaluated the prevalence of dyslipidemia among 225 patients with non-alcoholic steatohepatitis and explored its association with metabolic syndrome. The results indicate that the metabolic load in this group is also high, which indicates the high overlap between hepatic steatosis, lipid abnormalities, and systemic cardiometabolic dysfunction. Dyslipidemia was predominant in this cohort (80.4), and the most common abnormality was triglyceride (74.7%), then low levels of HDL cholesterol (67.5%). These measurements are reproducible with metabolic profiles known in NASH that have insulin resistance and lipid mismanagement leading to an atherogenic lipid phenotype. NASH is also systemic as it is revealed that metabolic syndrome prevalence was high in this study, with 76% of the cohort having it. Central obesity which is established in 82.7% of the patients emerged to be the most prevalent element which depicts the significant impact of adiposity on hepatology pathways and metabolism. Other highly prevalent components of metabolic syndrome were hypertriglyceridemia and low levels of HDL over 67% which proves their strong

connection with the condition of hepatic lipid dysfunction. It was found that 70.7% of the cohort had elevated fasting glucose levels and this indicates the high levels of insulin resistance among patients with NASH [14] [15].

The results of a comparison of patients who had and did not have metabolic syndrome showed that the lipid parameters had significant differences. Individuals with metabolic syndrome reported significantly lower total cholesterol (208.6 +- 37.9 mg/dl vs. 186.9 +- 33.4 mg/dl), LDL (133.4 +- 31.5mg/dl vs. 116.2 +- 28.1mg/dl), and triglycerides (214.5 + - 58.2mg/dl vs. 151.4 + - 42.6mg/dl This was also observed despite the maintenance of diabetes, obesity, age, and hypertension effects, indicating that metabolic syndrome serves as a core contributor of lipid abnormalities in NASH. Diabetes mellitus, which was found in 61.3% of patients, was also a significant risk factor of dyslipidemia (OR 2.21), as it is well-established that the interplay between insulin resistance and hepatic lipid metabolism plays an important role [16]. Together, these results support the idea that the dyslipidemia of NASH is not a solitary pathology but a manifestation of a more general metabolic impairment with significant contributions of central obesity, insulin resistance, and aggregation of metabolic syndrome factors. The fact that the low HDL and high triglycerides are the predominant ones in this population is in line with the typical dyslipidemic profile that is linked to hepatic insulin resistance [17]. This pattern prompts an early diagnosis and aggressive treatment in the clinical practice since it can significantly increase cardiovascular disease risk that is still one of the major causes of mortality in NAFLD and NASH. Close correlation between dyslipidemia and metabolic syndrome is also an evidence that patients having various metabolic defects should be ranked high on the list of patients who should be subjected to meticulous metabolic examination and prompt therapeutic measures [18-20]. Lifestyle change, weight loss measures, better glycemic regulation and specific pharmacologic management of dyslipidemia can aid to reduce the hepatic progression as well as cardiovascular complications. These results also highlight the significance of multidisciplinary managerial conditions such as hepatology, endocrinology, cardiology, and clinical nutrition [21-22]. The work has a number of limitations which must be put into consideration when explaining the results. To begin with the cross-sectional nature of the study prevents the development of a cause-and-effect relationship among dyslipidemia, metabolic syndrome, and severity of non-alcoholic steatohepatitis. There would be need of longitudinal follow-up to identify whether there would be specific lipid abnormalities that would lead to progression of the disease in the long run. Second, despite the beneficial information that the sample of 225 patients can give, the study itself was performed at one center, and thus may not be applicable to more widespread or more diverse populations that have different genetic, dietary, or lifestyle impacts. Lastly, the possibility of residual confounding by unmeasured factors, including genetic markers of dyslipidemia or a detailed index of insulin resistance cannot be completely removed regardless of multivariate corrections.

Conclusion

It is concluded that dyslipidemia is highly prevalent among patients with non-alcoholic steatohepatitis, affecting more than four-fifths of the study population, with elevated triglycerides and low HDL cholesterol being the most common abnormalities. Metabolic syndrome was present in the majority of patients and demonstrated a strong, independent association with dyslipidemia, even after adjusting for diabetes, obesity, and other metabolic factors. Patients with metabolic syndrome exhibited significantly worse lipid profiles, reflecting the systemic metabolic dysfunction that drives both hepatic injury and cardiovascular risk in NASH.

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