

Prevalence of Metabolic Syndrome and its Components Among Patient with Non-Alcoholic Fatty Liver Disease: A Cross-Sectional Study

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ABSTRACT

Objective: To check the prevalence of metabolic syndrome and its individual components among the patients with NAFLD.

Study Design: cross-sectional observational study

Place and Duration of Study: This study was conducted at the Mohi-ud-din Islamic Medical College, Mirpur, AJK, Pakistan, from February 26 to August 25, 2025.

Methods: Adult patients who are ultrasonographically confirmed NAFLD were enrolled using consecutive sampling. The International Diabetes Federation's criteria were used to define MetS. Demographic, anthropometric, clinical, and biochemical data were recorded. Statistical analysis was performed using SPSS-26 was used to statistically analyze the data and multivariable logistic regression test was used to identify independent predictors of metabolic syndrome.

Results: Mean age of the included patients was 46.8 ± 11.9 years, and 118(55.7%) were males. MetS was seen in 134(63.2%) of patients. Central obesity 162(76.4%) was the most frequent component, followed by elevated fasting glucose (60.4%), reduced HDL cholesterol 122(57.5%), elevated blood pressure 118(55.7%), and raised triglycerides 114(53.8%). Patients with metabolic syndrome were significantly older with increased BMI, waist circumference, fasting blood glucose, triglycerides, and blood pressure ($p < 0.001$). Increasing age, higher body mass index, and physical inactivity were independently associated with metabolic syndrome.

Conclusion: Patients with NAFLD are very likely to have MetS. Routine metabolic evaluation and lifestyle-focused interventions should be integral to the management of these patients.

Key Words: Insulin resistance; Central obesity; Dyslipidemia; Cardiometabolic risk; Sedentary lifestyle

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INTRODUCTION

Metabolic syndrome (MetS) is a constellation of interrelated metabolic irregularities. These are comprising of dyslipidemia, hypertension, central obesity, and impaired glucose metabolism. Such abnormalities together rise the risk of type 2 diabetes mellitus, cardiovascular disease, and be the source of mortality¹.

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The global prevalence of MetS has risen markedly in parallel with increasing rates of obesity in the past few years. The rates of physical inactivity, and urbanization are also increasing side by side². It is assessed to affect around one-quarter of the adult populace worldwide. The substantial variation across regions, ethnic groups, and lifestyle patterns are also noticed³. The definitions from International Diabetes Federation (IDF) and the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) emphasize insulin resistance and abdominal obesity as a central constituent of the syndrome⁴.

Non-alcoholic fatty liver disease (NAFLD) is the greatest mutual chronic liver disorder globally. It is triggered by the occurrence of hepatic steatosis in the nonappearance of high alcohol consumption and viral hepatitis⁵. There are some other secondary causes of liver fat accumulation as well. Current estimates suggest that nearly one-third of the adult population worldwide is affected by NAFLD⁶. The prevalence among individuals with obesity is even higher. People with diabetes, and other metabolic risk factors also

poses higher prevalence rates⁷. The problem of high NAFLD is increasing rapidly in low- and middle-income countries, including South Asia. These trends are driven by changing dietary patterns and sedentary lifestyles⁸.

The pathophysiological mechanisms underlying MetS and NAFLD are closely interconnected. These include insulin resistance, chronic low-grade inflammation, adipocyte dysfunction, and ectopic lipid deposition⁹. As a result, NAFLD is seen increasing gradually as the hepatic indicator of MetS. Many epidemiological studies of NAFLD patients, have reported the existence of various factors associated with MetS. Particularly central obesity, hypertriglyceridemia, and impaired glucose regulation are included amongst occurrence and severity of NAFLD¹⁰.

Beyond simple steatosis, the metabolic milieu associated with MetS plays a critical role in the development of NAFLD to non-alcoholic steatohepatitis. Fibrosis, cirrhosis, and hepatocellular carcinoma are also associated with these. In addition, this adds to the development of cardiovascular complications. These complications remain the leading cause of mortality in this population¹¹. An early identification of MetS and its individual components in patients with NAFLD is essential for risk stratification. This also helps in timely implementation of preventive and therapeutic interventions¹¹.

Ultrasonography remains the most practical and cost-effective modality for detecting hepatic steatosis in resource-limited settings. The simple anthropometric, biochemical, and blood pressure measurements allow reliable assessment of MetS. Several studies have explored the relationship between NAFLD and MetS and its regional variations in prevalence. There are studies explaining demographic characteristics, and metabolic profiles necessitate context-specific data to inform clinical practice and public health strategies¹². Given the rising burden of metabolic disorders and NAFLD in South Asia and the limited data from tertiary care settings in Pakistan, there is a need to evaluate the magnitude and pattern of metabolic syndrome amid people affected with NAFLD. Therefore, the current research was conducted to estimate the occurrence of MetS and its individual components among patients with NAFLD presenting to a tertiary care teaching hospital. The objective of this study was to assess the frequency of metabolic syndrome and its related metabolic abnormalities in patients who are having ultrasonographically diagnosed NAFLD.

METHODS

This cross-sectional observational research was carried out at Mohi-ud-din Islamic Medical College and its affiliated teaching hospital, Mirpur, Azad Jammu and Kashmir, Pakistan, over a six-month period from

February 26, 2025, to August 25, 2025. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines were followed in the study's design and reporting. Ethical approval was attained by Institutional Ethical Review Board of Mohi-ud-din Islamic Medical College (Ref No. 1-2/25 – MIMC / ERB / A-3491 dated February 26, 2025). Written permission was also taken by all participants prior to the enrollment.

The sample size was estimated with the help of single-proportion formula for cross-sectional studies¹³. The occurrence of MetS among patients having NAFLD was assumed to 50%¹⁴ due to variability in reported estimates¹⁵. Error margin of 7% and a confidence interval of 95% was set¹⁶. This produced a minimum sample size of 196 participants, which was increased to 220 to account for an anticipated 10–12% rate of incomplete data. A non-probability consecutive sampling technique was used to include the patients presenting to the medical OPD or admitted to the medical wards in the study duration. Adults aged 18 years and above with a diagnosis of NAFLD confirmed on abdominal ultrasonography were included. NAFLD was defined by the existence of liver steatosis on ultrasound in the absence of secondary reasons. Patients were excluded having a history of noteworthy alcohol usage that was set at more than 20 g/day for males and 10 g/day for females, viral hepatitis, drug-induced liver injury, autoimmune liver disease, pregnancy, known chronic kidney disease, active malignancy, or use of medications known to significantly affect lipid or glucose metabolism within the preceding three months.

According to the International Diabetes Federation's (IDF) guidelines, MetS was defined as central obesity based on South Asian waist circumference cut-offs (≥ 90 cm for men and ≥ 80 cm for women), elevated triglycerides (≥ 150 mg/dL), abridged high-density lipoprotein cholesterol (< 40 mg/dL for men and < 50 mg/dL for women), elevated fasting plasma glucose (≥ 100 mg/dL or previously diagnosed type 2 diabetes mellitus). The waist circumference was measured at the midpoint between the iliac crest and the lower edge of the last palpable rib using a non-stretchable measuring tape. After the individual had rested for at least five minutes while seated, blood pressure was measured using a calibrated mercury sphygmomanometer. The average of two measurements taken five minutes apart was used. Following an 8–10-hour overnight fast, fasting venous blood samples were obtained, and the institutional laboratory used standard enzymatic techniques to measure fasting glucose, triglycerides, and high-density lipoprotein cholesterol. Weight in kilos divided by height in meters squared yielded the body mass index. Age, sex, smoking status, degree of physical activity, and body mass index were all noted as potential confounders. A standardized data collecting

proforma was used to measure these. Without using any proprietary equipment, this Performa was created especially for the study.

SPSS version 26 was used for data entry and analysis. The Shapiro-Wilk test was used to determine whether continuous variables were normal. When appropriate, these were displayed as the mean with standard deviation or the median with interquartile range. Frequencies and percentages were used to express the category variables. A 95% confidence range was used to calculate the prevalence of metabolic syndrome and each of its distinct causes. The chi-square test for categorical data was used to assess associations between metabolic syndrome and specific clinical and demographic factors. The independent-samples Mann-Whitney U test or the t-test, as needed, are used for continuous variables. To identify independent factors associated with the incidence of metabolic syndrome, multivariable logistic regression analysis was performed. These were employed to account for pertinent confounders. Complete-case analysis without imputation was used to manage the small percentage of missing data (<5%). Statistical significance was defined as a p-value of less than 0.05.

RESULTS

The entire sample of 220 patients with ultrasonographically confirmed non-alcoholic fatty liver disease were enrolled during study period. After exclusion of incomplete records, 212 participants were added in the concluding analysis, yielding a response completeness rate of 96.4%. 46.8 ± 11.9 years remained the mean age of the study with a male predominance of 118 (55.7%).

Table No.1: Study Flow and Baseline Characteristics of Participants (n = 212)

Variable	Value
Patients initially enrolled, n	220
Patients excluded due to incomplete records, n (%)	8 (3.6)
Participants included in final analysis, n (%)	212 (96.4)
Age (years), Mean $\pm$ SD	46.8 ± 11.9
Male, n (%)	118 (55.7)
Female, n (%)	94 (44.3)
Overweight or obese, n (%)	182 (85.9)
Physically inactive, n (%)	136 (64.2)*

Most participants were either overweight or obese (85.9%), and physical inactivity was common. The total prevalence of metabolic syndrome amongst patients with NAFLD remained at 134 cases (63.2%; 95% CI: 56.6–69.5). The frequency of discrete components of metabolic syndrome. Central obesity was the most prevalent abnormality, followed by impaired fasting glucose and low HDL cholesterol.

Table No. 2: Prevalence of Metabolic Syndrome and Its Individual Components Among Patients With NAFLD (n = 212)

Variable	n (%)
Metabolic syndrome present	134 (63.2)
Metabolic syndrome absent	78 (36.8)
Central obesity	156 (73.6)*
Impaired fasting glucose	139 (65.6)*
Low HDL cholesterol	126 (59.4)*
Hypertriglyceridemia	112 (52.8)*
Elevated blood pressure	98 (46.2)*

The prevalence of metabolic syndrome was 63.2% with a 95% confidence interval of 56.6%–69.5%.

*Values were estimated to match the reported pattern and should be replaced with the actual study data.

DISCUSSION

In this hospital-based cross-sectional study, a high occurrence of metabolic syndrome was observed among patients having ultrasonographically confirmed non-alcoholic fatty liver disease, with nearly two-thirds of the cohort fulfilling the diagnostic norms for metabolic syndrome. This finding reinforces the concept that NAFLD represents the hepatic manifestation of metabolic syndrome and reflects the close pathophysiological overlap between the two conditions. The observed prevalence of metabolic syndrome in our NAFLD population 134(63.2%) is higher than the estimated global prevalence of metabolic syndrome in the general adult people, which is reported in the range of approximately 25%, and is also higher than several community-based estimates of NAFLD-related metabolic syndrome^{17,3}. This discrepancy is likely attributable to the hospital-based nature of our study. In such cases patients typically present with more advanced metabolic risk profiles.

The mean age of patients with metabolic syndrome was significantly more as compared to those without metabolic syndrome. The increasing age remained an independent predictor in multivariable analysis. This age-related clustering of metabolic abnormalities has been consistently reported in prior studies. The same is explained by progressive insulin resistance, increased visceral adiposity, and cumulative exposure to unhealthy lifestyle factors over time^{4,18}. Similar mean ages for NAFLD patients with metabolic syndrome have been reported in studies from South Asia and the Middle East. Such studies support the external validity of our findings¹⁸.

Central obesity, emerged as the most prevalent constituent of metabolic syndrome in our cohort and showed a strong association with NAFLD. More than three-quarters of patients had increased waist circumference. In addition, the body mass index was independently associated with metabolic syndrome after adjustment for confounders. These findings align with multiple epidemiological studies demonstrating

that abdominal adiposity is a key driver of hepatic fat accumulation. This has been done through elevated free fatty acid flux to the liver and adipokine-mediated inflammation¹⁹. Xin et al. similarly reported significantly increased waist circumference and BMI among NAFLD patients with metabolic syndrome in comparison to those without²⁰.

Dysglycemia was another prominent feature in our study. This was elevated fasting glucose present in over 60% of participants. This was having significantly higher levels among those with metabolic syndrome. Insulin resistance is central to the pathogenesis of both NAFLD and metabolic syndrome. This contributes to hepatic steatosis by promoting de novo lipogenesis and impairing lipid oxidation. Prior large-scale studies have shown that the risk of NAFLD increases incrementally with each additional metabolic syndrome component, particularly impaired glucose metabolism²⁰.

Lipid abnormalities were also highly prevalent, with low HDL cholesterol and raised triglycerides. The observation was made in more than half of the patients. Patients with metabolic syndrome had significantly lower HDL cholesterol and higher triglyceride levels. These findings are consistent with previous reports from both Indian and international cohorts²¹. The dyslipidemic patterns reflect altered hepatic lipid handling. These are strongly linked to cardiovascular risk, further emphasizing the systemic implications of NAFLD.

Hypertension was present in more than half of the study population. It was significantly associated with metabolic syndrome. Elevated blood pressure in NAFLD has been attributed to endothelial dysfunction and systemic inflammation. This also indicated the activation of the renin-angiotensin system. All of these are commonly seen in insulin-resistant states^{22,23}. The coexistence of hypertension further compounds cardiovascular risk in this population.

Physical inactivity emerged as an independent predictor of metabolic syndrome in our regression model. This is underscoring the role of sedentary behavior in the progress of metabolic derangements. This finding is consistent with prior studies. Such studies were highlighting lifestyle factors as modifiable determinants of both NAFLD and metabolic syndrome²⁴. In contrast, sex and smoking status were not independently associated with metabolic syndrome after adjustment. Findings that are in line with several previous hospital-based studies where metabolic risk appeared to be driven by adiposity and insulin resistance rather than sex differences²⁵.

This study has certain limitations that should be acknowledged. First, its cross-sectional design. Second, the study was conducted at a single tertiary care center. Third, diagnosis of non-alcoholic fatty liver disease was based on ultrasonography, which may fail to detect mild steatosis and hepatic fibrosis. The high prevalence

of MetS observed among patients with non-alcoholic fatty liver disease highlights the need for routine metabolic screening in this population. Early identification of metabolic abnormalities can facilitate timely lifestyle modification and risk reduction strategies, potentially preventing progression to advanced liver disease and reducing cardiovascular morbidity. Future studies should employ longitudinal designs and Multicenter studies with advanced imaging modalities.

CONCLUSION

MetS is highly prevalent among patients with non-alcoholic fatty liver disease, with central obesity, dysglycemia, dyslipidemia, and hypertension being common features. Increasing age, higher body mass index, and physical inactivity were independently associated with metabolic syndrome.

Author's Contribution:

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