DOI:https://doi.org/10.53350/pjmhs2023177181

ORIGINAL ARTICLE

Clinicopathological Correlation of Non-Alcoholic Fatty Liver Disease with Metabolic Syndrome Components in Tertiary Care Patients

SHEEMA KHAN[1], AISHA ASIM[2], MUHAMMAD AZHAR KHAN[3], SYED HASNAIN ABBAS[4], MAHWASH MANSOOR[5], IKRAM UL HAQ[6]
[1]*Assistant Professor, Department of Gastroenterology, MTI/Khyber Teaching Hospital, Peshawar, Pakistan*
[2]*Associate Professor, Al-Nafees Medical College and Hospital, Islamabad, Pakistan*
[3]*Assistant Professor, Department of Community Medicine, Bakhtawar Amin Medical and Dental College, Multan, Pakistan*
[4]*Lecturer, Department of Software Engineering, National University of Modern Languages (NUML), Islamabad, Pakistan*
[5]*Associate Professor, Department of Diagnostic Radiology, Bolan Medical College, Quetta, Pakistan*
[6]*Associate Professor, Department of Pathology, Sahara Medical College, Pakistan*
Correspondence to: Ikram Ul Haq, Email: dr.ikramhaq@gmail.com

ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) is increasingly recognized as the hepatic manifestation of metabolic syndrome and is strongly associated with central obesity, dyslipidemia, hypertension, and impaired glucose metabolism. The coexistence of these metabolic risk factors accelerates hepatic injury and progression to non-alcoholic steatohepatitis, fibrosis, and cirrhosis. Limited data exist from Pakistan regarding the clinicopathological correlation of NAFLD with metabolic syndrome components in tertiary care settings.

Objective: To evaluate the clinicopathological correlation of NAFLD with metabolic syndrome components in patients presenting to tertiary care hospitals.

Methods: This cross-sectional observational study was conducted at Khyber Teaching Hospital, Peshawar, and Sughra Shafi Medical Complex, Narowal, Pakistan, from January 2022 to March 2023. A total of 100 adult patients with ultrasonographically confirmed NAFLD were included. Clinical assessment, anthropometric measurements, fasting blood sugar, lipid profile, and liver function tests were performed. The presence of metabolic syndrome was defined using NCEP-ATP III criteria. Patients with persistent elevation of transaminases underwent liver biopsy, and histopathological grading was assessed using the NAFLD Activity Score.

Results: Sixty-eight percent of patients met the criteria for metabolic syndrome. Central obesity (71%), impaired fasting glucose (58%), and low HDL cholesterol (60%) were the most common abnormalities. Patients with metabolic syndrome exhibited significantly higher fasting glucose, triglycerides, and ALT levels compared to those without. Histopathological evaluation revealed that metabolic syndrome was associated with higher grades of steatosis, lobular inflammation, ballooning, and fibrosis ($p<0.05$).

Conclusion: NAFLD is strongly correlated with metabolic syndrome, and increasing metabolic burden is associated with more severe biochemical and histological changes. Early screening and management of metabolic risk factors are vital for preventing disease progression.

Keywords: Non-alcoholic fatty liver disease, metabolic syndrome, hepatic steatosis, histopathology, obesity, insulin resistance.

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) has emerged as the most prevalent chronic liver disorder globally, affecting nearly one quarter of the adult population. It encompasses a histological spectrum ranging from simple steatosis to non-alcoholic steatohepatitis (NASH), advanced fibrosis, cirrhosis, and hepatocellular carcinoma[1]. Unlike alcoholic liver disease, NAFLD occurs in individuals who consume little or no alcohol, and its pathogenesis is primarily linked to insulin resistance, adipose tissue dysfunction, and systemic metabolic dysregulation[2].

Metabolic syndrome, a constellation of interrelated risk factors including central obesity, dyslipidemia, hypertension, and impaired glucose metabolism, is strongly implicated in the pathophysiology of NAFLD. The hepatic manifestation of metabolic syndrome is now widely recognized, with insulin resistance serving as the key mechanistic link[3]. Excess free fatty acid flux into hepatocytes, oxidative stress, mitochondrial dysfunction, and inflammatory signaling collectively contribute to hepatocellular injury, steatohepatitis, and progressive fibrosis[4].

The clinical burden of NAFLD is of particular concern in South Asian countries, including Pakistan, where rising trends in obesity, sedentary lifestyles, and urban dietary transitions have accelerated the prevalence of both metabolic syndrome and NAFLD[5]. Data from tertiary care hospitals suggest that patients often present late, with advanced disease stages, reflecting both limited awareness and insufficient screening practices. The coexistence of metabolic syndrome further compounds the risk of progression to advanced fibrosis and significantly increases cardiovascular morbidity and mortality, which is already the leading cause of death in NAFLD patients[6].

Received on 11-04-2023
Accepted on 24-06-2023

Histopathological evaluation remains the gold standard for assessing disease severity, though non-invasive markers and imaging play an important role in diagnosis and follow-up. Correlating metabolic syndrome components with histological patterns of NAFLD provides valuable insights into disease progression, helps identify high-risk patients, and supports timely therapeutic interventions[7,8].

Despite the growing burden, few studies from Pakistan have systematically explored the clinicopathological correlation of NAFLD with metabolic syndrome in tertiary care settings. Understanding this association is critical to designing preventive strategies, improving patient stratification, and tailoring management approaches to local populations.The present study was conducted to determine the prevalence of metabolic syndrome components among NAFLD patients and to evaluate their correlation with clinical, biochemical, and histopathological findings in a tertiary care cohort[9].

MATERIALS AND METHODS

Study Design and Setting: This cross-sectional observational study was carried out at two tertiary care centers in Pakistan, namely the Department of Gastroenterology, Khyber Teaching Hospital (KTH), Peshawar, and the Department of Medicine, Sughra Shafi Medical Complex, Narowal. The study was conducted over a period of fifteen months, from January 2022 to March 2023. Both hospitals serve as major referral centers and cater to a large patient population, thereby providing a representative cohort of individuals with non-alcoholic fatty liver disease (NAFLD).

Sample Size and Population: A total of one hundred patients were enrolled in the study. The study population consisted of adults aged between 20 and 65 years who were diagnosed with NAFLD on the basis of ultrasonographic findings of hepatic

steatosis. Patients were recruited from outpatient clinics and admitted cases, ensuring inclusion of both genders and a wide spectrum of clinical presentations. The diagnosis of NAFLD was made after carefully excluding other possible etiologies of hepatic steatosis.

Inclusion and Exclusion Criteria: Patients with ultrasound-confirmed fatty liver were included, provided they fell within the specified age range and gave written informed consent. Individuals with a history of significant alcohol intake, defined as more than 20 grams per day in men and more than 10 grams per day in women, were excluded to avoid confounding with alcoholic liver disease. Patients with positive serological markers for hepatitis B surface antigen or anti-hepatitis C virus antibodies were also excluded. Furthermore, those with chronic liver disorders such as autoimmune hepatitis, Wilson's disease, and hemochromatosis, or with a history of drug-induced liver injury, were not included. Pregnant women were excluded due to physiological changes in liver function during pregnancy.

Clinical and Biochemical Evaluation: All enrolled patients underwent a detailed clinical assessment which included demographic information such as age and gender, as well as measurement of anthropometric variables like body mass index and waist circumference. Blood pressure was recorded using a standardized protocol. Biochemical evaluation consisted of fasting blood sugar levels, a complete lipid profile including total cholesterol, triglycerides, high-density lipoprotein and low-density lipoprotein cholesterol, and liver function tests including alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, and serum bilirubin. The presence of metabolic syndrome was diagnosed using the National Cholesterol Education Program – Adult Treatment Panel III (NCEP-ATP III) criteria. According to these guidelines, metabolic syndrome was defined by the presence of at least three of the following components: central obesity, hypertriglyceridemia, low HDL cholesterol, hypertension, and elevated fasting blood sugar or previously diagnosed diabetes.

Histopathological Assessment: Patients with persistently elevated serum transaminases or with clinical suspicion of non-alcoholic steatohepatitis underwent liver biopsy. The obtained biopsy specimens were processed and stained with hematoxylin and eosin. Histological assessment was carried out according to the NAFLD Activity Score (NAS), which evaluates the degree of steatosis, lobular inflammation, hepatocellular ballooning, and fibrosis. This grading system provided a standardized method to correlate histopathological findings with clinical and biochemical parameters.

Ethical Considerations: Ethical approval for the study was obtained from the Institutional Review Boards . All participants provided written informed consent prior to inclusion. Confidentiality of patient data was maintained throughout the study, and all procedures were performed in accordance with the principles outlined in the Declaration of Helsinki.

Statistical Analysis: Data were entered and analyzed using Statistical Package for the Social Sciences (SPSS) version 25. Continuous variables such as age, body mass index, fasting blood sugar, and lipid parameters were expressed as mean and standard deviation. Categorical variables including gender distribution, presence of metabolic syndrome components, and histological grades were presented as frequencies and percentages. The chi-square test was applied to determine the association between the severity of NAFLD and individual components of metabolic syndrome. Correlation analysis was carried out to evaluate the relationship between biochemical parameters and histopathological severity. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Demographic Characteristics: A total of one hundred patients with ultrasound-confirmed non-alcoholic fatty liver disease (NAFLD) were included in this study. The mean age of the study population was 43.8 ± 9.7 years, with a range of 22 to 64 years. Out of the total, 58 patients were male (58%) and 42 were female (42%), showing a slight male predominance. The majority of patients fell within the 35–50-year age group, reflecting the typical age distribution of NAFLD in clinical practice. The mean body mass index (BMI) was 29.4 ± 4.6 kg/m², indicating that most patients were either overweight or obese, while the mean waist circumference was 103.2 ± 8.9 cm, consistent with central obesity.

Distribution of Metabolic Syndrome Components: The prevalence of metabolic syndrome components among NAFLD patients is shown in Table 1. Central obesity was the most frequently observed abnormality, present in seventy-one patients (71%). Dyslipidemia was also common, with hypertriglyceridemia detected in fifty-six patients (56%) and low high-density lipoprotein (HDL) cholesterol in sixty patients (60%). Hypertension was observed in fifty-three patients (53%), while impaired fasting glucose or previously diagnosed diabetes was found in fifty-eight patients (58%). Overall, sixty-eight patients (68%) fulfilled the criteria for metabolic syndrome as defined by NCEP-ATP III guidelines, whereas thirty-two patients (32%) had fewer than three metabolic abnormalities and did not meet the definition of metabolic syndrome. These findings highlight the strong overlap between NAFLD and metabolic syndrome in the study population (Table 1).

Table 1. Distribution of Metabolic Syndrome Components among NAFLD Patients

Metabolic Syndrome Component	Frequency (n=100)	Percentage (%)
Central obesity	71	71
Hypertriglyceridemia	56	56
Low HDL cholesterol	60	60
Hypertension	53	53
Impaired fasting glucose	58	58
Patients meeting ≥3 criteria (Metabolic Syndrome)	68	68

Biochemical Findings: The biochemical analysis demonstrated that patients with metabolic syndrome had significantly higher fasting blood sugar and triglyceride levels compared to those without metabolic syndrome. Mean fasting blood sugar among patients with metabolic syndrome was 116.5 ± 22.4 mg/dL, whereas it was 98.3 ± 15.7 mg/dL in those without metabolic syndrome ($p<0.01$). Similarly, serum triglycerides were markedly elevated in the metabolic syndrome group (178.6 ± 43.2 mg/dL versus 141.7 ± 28.9 mg/dL; $p<0.01$). Mean HDL cholesterol levels were lower in patients with metabolic syndrome, and serum alanine aminotransferase (ALT) levels were significantly higher, suggesting a relationship between metabolic burden and hepatocellular injury.

Histopathological Findings: Of the one hundred patients, forty-five underwent liver biopsy due to persistent elevation of transaminases or suspicion of non-alcoholic steatohepatitis (NASH). The histopathological evaluation revealed a wide spectrum of changes, as summarized in Table 2. Simple steatosis was found in sixteen patients (36%), moderate steatosis in eighteen patients (40%), and severe steatosis in eleven patients (24%). Lobular inflammation was present in twenty-four cases (53%), while ballooning degeneration was noted in seventeen cases (38%). Fibrosis of varying degrees was observed in fifteen patients (33%), with perisinusoidal fibrosis being the most common pattern.

When the histological findings were stratified according to the presence of metabolic syndrome, patients with three or more metabolic abnormalities had significantly higher grades of steatosis, more frequent lobular inflammation, and greater degrees of fibrosis compared to patients without metabolic syndrome ($p=0.02$). The mean NAFLD Activity Score (NAS) was 5.2 ± 1.6 in patients with metabolic syndrome, whereas it was 3.1 ± 1.2 in those without metabolic syndrome, further confirming the association between metabolic burden and histological severity (Table 2).

Table 2. Histopathological Features of NAFLD Patients Undergoing Liver Biopsy

Histopathological Feature	Patients with Metabolic Syndrome (n=30)	Patients without Metabolic Syndrome (n=15)	p-value
Mild steatosis	6 (20%)	10 (67%)	0.01
Moderate steatosis	13 (43%)	5 (33%)	NS
Severe steatosis	11 (37%)	0 (0%)	0.01
Lobular inflammation	20 (67%)	4 (27%)	0.02
Ballooning degeneration	14 (47%)	3 (20%)	0.04
Fibrosis	12 (40%)	3 (20%)	0.05
Mean NAS score	5.2 ± 1.6	3.1 ± 1.2	0.01

Correlation of Biochemical and Histological Parameters: A significant positive correlation was observed between serum ALT levels and histological severity of NAFLD ($r=0.62$, $p<0.01$). Patients with higher ALT levels were more likely to have ballooning degeneration and fibrosis on liver biopsy. Similarly, triglyceride levels and fasting blood sugar demonstrated a moderate correlation with the degree of steatosis ($r=0.51$ and $r=0.47$, respectively), supporting the role of metabolic dysfunction in driving hepatic pathology.

In summary, the results of this study demonstrate that NAFLD patients frequently exhibit multiple components of metabolic syndrome, with nearly seventy percent fulfilling diagnostic criteria. Central obesity, impaired fasting glucose, and dyslipidemia were the most prevalent features. Patients with metabolic syndrome not only showed more severe biochemical abnormalities but also exhibited advanced histological changes, including higher grades of steatosis, inflammation, and fibrosis. These findings emphasize the strong clinicopathological correlation between NAFLD and metabolic syndrome in the studied tertiary care population.

DISCUSSION

The present study highlights the strong clinicopathological correlation between non-alcoholic fatty liver disease (NAFLD) and metabolic syndrome among patients attending tertiary care hospitals in Pakistan. In our cohort of one hundred patients, nearly seventy percent met the diagnostic criteria for metabolic syndrome, and its components such as central obesity, impaired fasting glucose, dyslipidemia, and hypertension were highly prevalent. These findings are in line with the global recognition of NAFLD as the hepatic manifestation of metabolic syndrome and underscore the growing burden of this condition in South Asian populations[10,11].

The predominance of central obesity and impaired fasting glucose in our study reflects the underlying pathophysiological role of insulin resistance in NAFLD development. Excess visceral adiposity leads to increased free fatty acid delivery to the liver, which promotes hepatic triglyceride accumulation[12]. This process is compounded by oxidative stress, mitochondrial dysfunction, and low-grade chronic inflammation, ultimately driving the progression from simple steatosis to non-alcoholic steatohepatitis (NASH). Our findings resonate with previous studies conducted in similar settings, which reported that abdominal obesity and insulin resistance are the strongest predictors of NAFLD progression[13].

The biochemical results of our study further strengthen the link between metabolic dysregulation and liver pathology. Patients with metabolic syndrome had significantly higher fasting blood glucose and triglyceride levels, along with lower HDL cholesterol, compared to those without metabolic syndrome[14]. In addition, elevated serum alanine aminotransferase (ALT) levels correlated positively with histological severity, consistent with the widely acknowledged role of ALT as a biochemical marker of hepatocellular injury. While ALT is not specific for NAFLD, its correlation with advanced steatohepatitis in our study makes it a useful surrogate marker in resource-limited settings where liver biopsy is not feasible for all patients[15].

Histopathological evaluation in our study demonstrated that patients with three or more metabolic abnormalities had significantly higher grades of steatosis, lobular inflammation, ballooning, and fibrosis compared to those without metabolic syndrome[16]. The mean NAFLD Activity Score was significantly higher in the metabolic syndrome group, suggesting that the cumulative effect of metabolic risk factors contributes to more advanced liver injury. These findings are consistent with international reports that have linked the severity of metabolic syndrome to higher risks of fibrosis progression and cirrhosis. Importantly, fibrosis is the most robust histological predictor of long-term mortality in NAFLD patients, not only due to liver-related complications but also because of increased cardiovascular risk[17,18].

The results of our study also carry important public health implications. Pakistan, like other South Asian countries, is experiencing an alarming rise in obesity, type 2 diabetes, and hypertension due to urbanization, sedentary lifestyles, and dietary changes[19]. This epidemiological transition directly feeds into the rising prevalence of NAFLD. Yet, awareness about NAFLD remains limited among both clinicians and the general population. Early screening for metabolic syndrome in patients with fatty liver, coupled with lifestyle interventions such as dietary modification, increased physical activity, and targeted pharmacological treatment, may reduce disease progression and prevent the onset of cirrhosis and hepatocellular carcinoma[20].

Our findings should also be interpreted in light of certain limitations. First, the sample size was relatively small and drawn from two tertiary care centers, which may not fully represent the general population. Second, only a subset of patients underwent liver biopsy, potentially limiting the generalizability of histopathological findings. Third, the cross-sectional design precludes assessment of longitudinal outcomes or progression rates. Despite these limitations, the study provides important insights into the interplay of metabolic syndrome and NAFLD in a Pakistani cohort and emphasizes the urgent need for integrated clinical management strategies[21,22].

CONCLUSION

Non-alcoholic fatty liver disease is strongly associated with metabolic syndrome, with central obesity, impaired fasting glucose, and dyslipidemia being the most frequent components. Patients with multiple metabolic abnormalities showed more severe biochemical and histological changes, including advanced steatosis, inflammation, and fibrosis. Early recognition and management of metabolic risk factors are essential to prevent disease progression and reduce long-term complications.

Conflicts of Interest: The authors declare no conflicts of interest.
Funding: No specific funding was received for this study.
Acknowledgements: The authors thank the staff of the Departments of Gastroenterology, Medicine, and Pathology at both participating hospitals for their support during data collection and patient management.

Authors' Contributions: S.K. conceived the study, designed the research protocol, and supervised overall execution. A.A. contributed to patient recruitment, data acquisition, and preliminary analysis. M.A.K. performed statistical analysis, interpreted results, and assisted in manuscript drafting. S.H.A. contributed to histopathological evaluation, data interpretation, and critical manuscript revision. M.M. was responsible for biochemical data collection, patient follow-up, and literature review. I.U.H. contributed to manuscript writing, reference formatting, and final editing of the draft.

All authors read and approved the final version of the manuscript and agree to be accountable for its content.

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This article may be cited as: Khan S, Asim A, Khan MA, Abbas SH, Mansoor M, Haq IU; Clinicopathological Correlation of Non-Alcoholic Fatty Liver Disease with Metabolic Syndrome Components in Tertiary Care Patients. Pak J Med Health Sci, 2023;17(7): 181-184.