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A COMPARATIVE ASSESSMENT OF CLINICAL AND BIOCHEMICAL MARKERS OF METABOLIC SYNDROME IN PATIENTS WITH AND WITHOUT NON-ALCOHOLIC FATTY LIVER DISEASE

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ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) is an escalating public health issue and is commonly recognized as the hepatic aspect of metabolic syndrome (MetS). Metabolic syndrome is defined by central obesity, insulin resistance, dyslipidemia, and hypertension. NAFLD and MetS frequently coexist, sharing analogous pathophysiological pathways, notably insulin resistance. Comprehending the correlation between NAFLD and MetS via clinical and biochemical indicators is essential for early diagnosis, risk assessment, and the avoidance of cardiovascular and hepatic consequences.

Aims and Objective: To assess and compare clinical and biochemical markers of metabolic syndrome in patients with and without NAFLD.

Materials and Methods: A cross-sectional study was conducted on 56 patients diagnosed with metabolic syndrome, divided into two groups based on the presence (n = 28) or absence (n = 28) of NAFLD as confirmed by abdominal ultrasonography. Clinical parameters including body mass index (BMI), waist circumference, and blood pressure were recorded. Biochemical markers assessed included fasting blood glucose (FBG), fasting insulin, HOMA-IR, lipid profile (triglycerides, HDL-C), and liver enzymes (ALT, AST).

Results: Patients with NAFLD had significantly higher BMI, waist circumference, and systolic blood pressure compared to those without NAFLD ($p < 0.05$). Biochemically, the NAFLD group showed elevated fasting insulin, HOMA-IR, triglyceride levels, and liver enzymes (ALT and AST), alongside reduced HDL-C levels ($p < 0.05$). The prevalence of insulin resistance and dyslipidemia was notably greater in the NAFLD group.

Conclusion: In patients with metabolic syndrome, the presence of NAFLD is associated with more pronounced clinical and biochemical derangements, particularly related to insulin resistance and hepatic dysfunction. Routine evaluation for NAFLD in MetS patients may aid in early detection and targeted therapeutic strategies.

Keywords: Dyslipidemia, Insulin resistance, Liver enzymes, Metabolic syndrome, Non-alcoholic fatty liver disease

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) has emerged as a major public health issue, being the most prevalent chronic liver condition worldwide. It is marked by an excessive accumulation of lipids in hepatocytes in persons who consume minimal or no alcohol.[1] Non-alcoholic fatty liver disease (NAFLD) includes a range from simple steatosis to non-alcoholic steatohepatitis (NASH), which may advance to fibrosis, cirrhosis, and hepatocellular cancer.[2]

The global prevalence of NASH in the adult population is estimated to be between 1.5% and 6.45%, with recent meta-analyses indicating a mean prevalence of approximately 5.3%. Regionally, South Asia, including India, has found a comparable prevalence of roughly 5.4%, highlighting the escalating impact of this illness in developing countries. These findings underscore the progressive characteristics of NAFLD and the significant percentage of patients susceptible to liver inflammation and fibrosis.[3]

Metabolic syndrome is characterized by a combination of connected metabolic disorders, including central obesity, dyslipidemia, hypertension, and poor glucose tolerance or insulin resistance. These factors substantially elevate the risk of cardiovascular illnesses and type 2 diabetes mellitus.[4]

Multiple diagnostic criteria, including those from the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) and the International Diabetes Federation (IDF), necessitate the identification of three or more metabolic abnormalities to diagnose Metabolic Syndrome (MetS). The clinical and biochemical criteria for diagnosing MetS coincide with those involved in the etiology and advancement of NAFLD, hence establishing a robust correlation between the two disorders.[5,6]

Multiple studies have indicated that persons with NAFLD are more prone to display components of MetS compared to those without hepatic steatosis. NAFLD indicates underlying metabolic dysfunction and may exacerbate systemic insulin resistance and inflammation, hence deteriorating metabolic profiles.[7]

In contrast, persons with MetS face a heightened risk of developing NAFLD as a result of the persistent impacts of insulin resistance and adipose tissue dysfunction. Notwithstanding this bidirectional relationship, further comparison studies are necessary to investigate the differences in clinical and biochemical markers of MetS between patients with and without NAFLD.[8]

A comprehensive comparison analysis can provide insights into the degree and characteristics of metabolic abnormalities in NAFLD patients, thereby facilitating early diagnosis and enhanced risk stratification. Clinical parameters including body mass index

(BMI), waist circumference, and blood pressure, alongside biochemical markers such as fasting blood glucose, lipid profile (triglycerides, HDL cholesterol), liver enzymes (ALT, AST), and indices of insulin resistance (e.g., HOMA-IR), can be valuable instruments in this assessment.[9,10] Recognizing substantial disparities in these markers between NAFLD and non-NAFLD patients may assist doctors in comprehending disease development and customizing therapies accordingly.

The research aims to elucidate the degree of metabolic dysfunction in NAFLD and its correlation with MetS by examining critical metabolic indicators. The results are anticipated to improve comprehension of the pathophysiological connections between these illnesses and aid in the formulation of more effective preventative and management methods.

AIMS

- To assess and compare clinical and biochemical markers of metabolic syndrome in patients with and without NAFLD.

MATERIALS AND METHODS

A cross-sectional, observational study was carried out over a defined period of 10 months from March 2024 to December 2024, at Sree Mookambika Institute of Medical Sciences. Written informed consent was obtained from all participants prior to enrollment. A total of 56 adult patients diagnosed with metabolic syndrome according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criteria were included in the study. Participants were divided into two groups**:**

- **Group 1 (NAFLD group):** 28 patients with metabolic syndrome and NAFLD.
- **Group 2 (non-NAFLD group):** 28 patients with metabolic syndrome but without NAFLD.

The diagnosis of NAFLD was established using abdominal ultrasonography, performed by an experienced radiologist blinded to the clinical and laboratory data. Ultrasound criteria included increased hepatic echogenicity compared to renal cortex, blurring of vascular margins, and deep attenuation, in the absence of significant alcohol intake, viral hepatitis, or other secondary causes of fatty liver.

Inclusion Criteria

- Adults aged 18–65 years.
- Diagnosed with metabolic syndrome based on NCEP ATP III criteria (presence of ≥3 of the following: waist circumference >102 cm in men or >88 cm in women,

triglycerides ≥150 mg/dL, HDL-C <40 mg/dL in men or <50 mg/dL in women, blood pressure ≥130/85 mmHg, fasting glucose ≥100 mg/dL).

Exclusion Criteria

- History of alcohol consumption >20 g/day.
- Known liver diseases (e.g., hepatitis B/C, autoimmune hepatitis, hemochromatosis).
- Use of hepatotoxic drugs or lipid-lowering agents.
- Pregnancy or lactation.
- Known malignancy or endocrine disorders affecting metabolism.

All participants underwent a detailed clinical evaluation. BMI was calculated as weight in kilograms divided by the square of height in meters (kg/m^2), with measurements taken using a standardized scale and stadiometer. Waist circumference (WC) was measured in centimeters at the midpoint between the lower margin of the last palpable rib and the top of the iliac crest using a non-elastic measuring tape, with the patient in a standing position.

Blood pressure was recorded in the right arm using a calibrated sphygmomanometer after the participant had rested for at least five minutes in a seated position. Two readings were taken five minutes apart, and the average of the two was used for analysis. These clinical parameters were selected as core markers of metabolic syndrome, in line with established diagnostic criteria.

Venous blood samples were collected from all participants after an overnight fast of 8 to 12 hours. Fasting blood glucose (FBG) levels were measured using the glucose oxidase-peroxidase method. Fasting serum insulin concentrations were determined by a commercially available enzyme-linked immunosorbent assay (ELISA).

Insulin resistance was estimated using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), calculated with the formula: HOMA-IR = (Fasting Insulin [μU/mL] × Fasting Glucose [mg/dL]) / 405. The lipid profile included serum triglycerides (TG), measured by enzymatic colorimetric methods, and high-density lipoprotein cholesterol (HDL-C), assessed using a precipitation method.

Liver function was evaluated by measuring alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels, both quantified using the International Federation of Clinical Chemistry (IFCC) kinetic method without pyridoxal phosphate activation.

Data were entered and analyzed using SPSS v25. Continuous variables were expressed as mean ± standard deviation (SD). Differences between the two groups were analyzed using

the Student's t-test**.** Categorical variables were compared using the Chi-square test. A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 56 patients were included in the study, with 28 patients in the NAFLD group and 28 in the non-NAFLD group. The two groups were compared based on demographic, clinical, and biochemical parameters associated with metabolic syndrome. There was no statistically significant difference in age and gender between the groups, indicating comparability. However, BMI and waist circumference were significantly higher in patients with NAFLD, suggesting a stronger association of central obesity with NAFLD. (Table 1)

Parameter	**NAFLD Group (n=28)**	**Non-NAFLD Group (n=28)**	**p-value**
Age (years)	45.7 ± 10.3	43.9 ± 9.8	0.412
Gender (Male/Female)	18 / 10	17 / 11	0.793
BMI (kg/m²)	28.9 ± 2.5	26.1 ± 2.3	<0.001
Waist Circumference (cm)	101.2 ± 6.9	93.8 ± 6.2	<0.001

Table 1: Comparison of demographic and Anthropometric Profile of patients

Patients with NAFLD exhibited significantly higher systolic and diastolic blood pressures, fulfilling one of the key clinical markers of metabolic syndrome. (Table 2)

Blood pressure	**NAFLD Group (n=28)**	**Non-NAFLD Group (n=28)**	**p-value**
Systolic BP (mmHg)	137.5 ± 11.2	126.8 ± 10.5	<0.001
Diastolic BP (mmHg)	85.1 ± 7.3	78.4 ± 6.9	<0.001

Table 2: Comparison of mean blood pressure in both groups

Fasting glucose, insulin levels, and HOMA-IR scores were significantly higher in the NAFLD group, indicating greater degrees of insulin resistance which with the pathophysiological overlap between NAFLD and metabolic syndrome.

Parameters for Glucose metabolism	**NAFLD Group (n=28)**	**Non-NAFLD Group (n=28)**	**p-value**
Fasting Blood Glucose (mg/dL)	115.2 ± 17.8	98.6 ± 13.7	<0.001
Fasting Insulin (μU/mL)	18.4 ± 4.2	12.3 ± 3.6	<0.001

HOMA-IR	5.23 ± 1.58	2.99 ± 1.12	<0.001

Table 3: Comparison of Glucose metabolism between the groups

Serum triglyceride levels were significantly elevated and HDL-C levels were significantly reduced in NAFLD patients, fulfilling lipid criteria of metabolic syndrome. (Table 4)

Lipid profile	**NAFLD Group (n=28)**	**Non-NAFLD Group (n=28)**	**p-value**
Triglycerides (mg/dL)	183.1 ± 36.4	148.7 ± 26.9	<0.001
HDL-C (mg/dL)	38.6 ± 5.8	45.2 ± 6.3	<0.001

Table 4: Comparison of lipid profile between the groups

ALT and AST levels were significantly elevated in the NAFLD group, indicating ongoing hepatic inflammation and supporting imaging-based diagnosis of fatty liver. (Table 5)

Liver Enzymes	**NAFLD Group (n=28)**	**Non-NAFLD Group (n=28)**	**p-value**
ALT (IU/L)	75.3 ± 21.6	34.8 ± 12.4	<0.001
AST (IU/L)	58.2 ± 19.4	29.3 ± 10.6	<0.001

Table 5: Comparison of liver enzymes between the groups

DISCUSSION

The present study evaluated a group of patients with and without NAFLD, emphasizing factors linked with metabolic syndrome. The demographic resemblance between the two groups regarding age and gender bolsters the validity of the identified disparities in clinical and biochemical indicators by reducing any confounding influences from these variables.

A key finding was the markedly elevated BMI and waist circumference in the NAFLD cohort. The results highlight central obesity as a significant risk factor for NAFLD, aligning with current literature that identifies visceral fat accumulation as a contributor to hepatic steatosis and systemic metabolic dysfunction.

A NHANES investigation conducted by Yari Z et al.[11] in the United States revealed that abdominal obesity, particularly increased waist circumference, had the most significant correlation with the risk of NAFLD; moreover, combination obesity heightened this risk more than general obesity alone.

Sun J et al.[12] reported that, in comparison to the normal weight cohort, the odds ratios (ORs) [95% confidence interval] for non-NAFLD in persons with overweight, general obesity, abdominal obesity, and combination obesity were 6.90 [3.74–12.70], 2.84 [2.38–3.39], 3.02 [2.02–4.51], and 9.53 [7.79–11.64], respectively. Subgroup analysis indicated that the influence of various obesity patterns on NAFLD risk was consistent across persons with differing clinical conditions.

Additionally, blood pressure readings were significantly heightened in the NAFLD cohort, hence reinforcing the correlation between NAFLD and metabolic syndrome. Increased systolic and diastolic pressures are established elements of the condition, and their occurrence in NAFLD patients highlights the systemic vascular strain associated with hepatic steatosis.

Ciardullo S et al.[13] noted that NAFLD was linked to approximately a 1.6-fold increased risk of developing hypertension, which in turn was strongly correlated with a higher incidence of NAFLD. Song Q et al.[14] indicated that in persons with advanced NAFLD, the five-year incidence of hypertension increased from 14.1% (absence of NAFLD) to 30.1% (moderate to severe NAFLD).

The study revealed substantial disturbances in glucose metabolism, with NAFLD patients showing elevated fasting glucose, insulin levels, and HOMA-IR scores. The findings indicate that insulin resistance, a characteristic of metabolic syndrome, is more severe in persons with NAFLD, reinforcing the pathophysiological connection between hepatic fat storage and disrupted insulin signaling pathways.

Fatahi S et al.[15] found that patients with NAFLD exhibited a strong correlation with elevated BMI, liver enzyme levels, triglycerides, low-density lipoprotein cholesterol (LDL), total cholesterol, and fasting blood sugar (FBS) in comparison to healthy individuals ($p < 0.05$). The upper tertiles of the Food Insulin Index were linked to increased risks of NAFLD (OR=1.4, 95% CI: 0.88-2.48, p for trend <0.001) and obesity (OR=2.33, 95% CI: 0.97-5.75) in comparison to the lower tertiles.

NAFLD patients had elevated blood triglyceride levels and diminished HDL-C levels relative to non-NAFLD controls. These modifications meet the dyslipidemia criteria of metabolic syndrome and additionally indicate that problems in lipid metabolism are closely associated with liver fat levels.

Bril F et al.[16] reported that patients with NAFLD exhibited a more detrimental atherogenic lipoprotein profile, characterized by an elevated apolipoprotein B/A1 ratio and reduced LDL particle size, irrespective of BMI and the severity of insulin resistance.

Finally, liver enzyme levels (ALT and AST) were markedly higher in the NAFLD cohort, indicating persistent hepatic damage and inflammation. This biochemical signature facilitates the imaging-based diagnosis of NAFLD and underscores the necessity for early detection and intervention to avert progression to non-alcoholic steatohepatitis (NASH) or cirrhosis.

Watt J et al.[17] observed a mean ALT of approximately 56 U/L and AST of around 38 U/L at baseline in NAFLD patients, with approximately 39% exhibiting normal ALT levels, indicating that increased enzymes possess considerable sensitivity but are not universally present. A study in 31,718 Chinese individuals conducted by Wang G et al.[18] indicated that ALT is independently related with NAFLD, alongside age, BMI, and triglycerides, obtaining a diagnostic area under the curve (AUC) of 0.88 when paired with TG and HDL.

The findings together emphasized that NAFLD is not solely a liver disorder but a hepatic presentation of systemic metabolic dysfunction. The significant correlations with obesity, insulin resistance, dyslipidemia, and hypertension underscore the necessity for a comprehensive, multidisciplinary strategy in addressing at-risk individuals. Timely lifestyle management and metabolic regulation may avert or alleviate the advancement of NAFLD and its related cardiovascular and hepatic consequences.

CONCLUSION

The study demonstrated a strong association between non-alcoholic fatty liver disease (NAFLD) and key clinical and biochemical markers of metabolic syndrome. Patients with NAFLD exhibited significantly higher BMI, waist circumference, blood pressure, fasting glucose, insulin resistance (HOMA-IR), triglycerides, and liver enzyme levels, along with lower HDL-C values, compared to those without NAFLD. The prevalence of metabolic syndrome was also markedly higher in the NAFLD group

These findings reinforce the role of NAFLD as a hepatic manifestation of metabolic syndrome and highlight its utility as a surrogate marker for early identification of individuals at cardiometabolic risk. Early screening and intervention in patients with NAFLD may therefore be pivotal in preventing long-term complications such as cardiovascular disease and type 2 diabetes mellitus.

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Nil.

CONFLICTS OF INTEREST

There are no conflicts of interest

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