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Original Research

Metabolic Syndrome and Its Association with Non-Alcoholic Fatty Liver Disease: A Clinical Study

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Abstract

Introduction: Metabolic syndrome (MetS) represents a cluster of interrelated metabolic abnormalities—central obesity, insulin resistance, hypertension, and dyslipidemia—that markedly increase cardiovascular and hepatic risk. Non-alcoholic fatty liver disease (NAFLD), the hepatic manifestation of MetS, is now the most common chronic liver disease worldwide. **Materials and Methods:** A cross-sectional observational study was conducted in 250 patients aged 18-65 years attending a tertiary-care centre. MetS was defined using the International Diabetes Federation (IDF) criteria. Abdominal ultrasonography was employed to diagnose NAFLD, and biochemical tests (LFTs, fasting glucose, lipid profile) were obtained. **Results:** Among 250 participants, MetS prevalence was 48%. NAFLD was present in 58.8% overall, with a significantly higher prevalence among MetS subjects (77%) compared with non-MetS (42%; $p < 0.001$). Obesity, insulin resistance, hypertriglyceridemia, and hypertension showed strong associations with NAFLD. **Conclusion:** NAFLD prevalence is significantly higher in patients with MetS, reinforcing the bidirectional interplay of metabolic and hepatic dysfunction. Routine screening for NAFLD in MetS patients, and aggressive lifestyle and pharmacological interventions, may reduce long-term morbidity.

Keywords: Metabolic syndrome; NAFLD; obesity; insulin resistance; dyslipidemia; ultrasonography.

Introduction

Metabolic syndrome (MetS) is a constellation of risk factors, including central obesity, insulin resistance, dyslipidemia, and hypertension, that synergistically elevate the risk for cardiovascular disease, type 2 diabetes, and hepatic complications.^{1,2} The International Diabetes Federation (IDF) and National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) define MetS based on a combination of these criteria, with prevalence rates in adults worldwide ranging from 20-35% depending on the population.³⁻⁵

Non-alcoholic fatty liver disease (NAFLD) represents the hepatic component of MetS and encompasses a spectrum from simple steatosis to non-alcoholic steatohepatitis (NASH), advanced fibrosis, cirrhosis, and hepatocellular carcinoma.⁶⁻⁸ NAFLD has become the most prevalent cause of chronic liver disease globally, affecting up to 25-30% of the adult population.^{9,10} Its pathogenesis is closely tied to insulin resistance, adipose tissue dysfunction, and systemic inflammation, processes central to MetS.¹¹

Recent data demonstrate that the coexistence of MetS markedly accelerates NAFLD progression. Obesity and central adiposity lead to increased free fatty acid flux to the liver, while hyperinsulinemia promotes hepatic de novo lipogenesis. Hypertriglyceridemia and low HDL cholesterol contribute to lipotoxicity, oxidative stress, and hepatocellular injury.^{12,13} The “two-hit” and, more recently, “multiple-hit” hypotheses explain the sequential development of steatosis and inflammation-fibrosis in NAFLD, where MetS components act as key “hits.”¹⁴

In South Asia, NAFLD prevalence is rising sharply, paralleling increases in obesity and type 2 diabetes. Indian cohort studies have reported prevalence rates exceeding 30% in urban adults, with higher rates in those with MetS.^{15,16} The National Institute for Health and Care Excellence (NICE) and American Association for the Study of Liver Diseases (AASLD) guidelines recommend NAFLD screening in high-risk individuals, particularly those with MetS.^{17,18}

Despite this, routine screening is often underutilized. Ultrasonography, while operator-dependent, remains a cost-effective first-line tool for detection, supported by serum biomarkers (ALT, AST, FIB-4, NAFLD fibrosis score) and elastography in advanced cases.^{19,20} Early detection in MetS patients provides a critical opportunity for lifestyle interventions (diet, exercise, weight reduction) and, where necessary, pharmacotherapy targeting insulin resistance and dyslipidemia.^{21,22}

Given the escalating burden, studying the clinico-biochemical association between MetS and NAFLD in tertiary-care populations can provide insight into screening priorities and prevention strategies. This study evaluates the prevalence of NAFLD among patients with MetS and identifies the individual metabolic components most strongly linked to NAFLD risk.

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Materials and Methods

A cross-sectional observational study was conducted in the Departments of Medicine and Gastroenterology at a tertiary-care teaching hospital over 12 months.

Participants: Adults aged 18-65 years attending outpatient clinics with features suggestive of MetS or undergoing evaluation for metabolic/abdominal complaints.

Inclusion criteria:

- Age 18-65 years
- Patients fulfilling IDF criteria for MetS (central obesity + ≥ 2 of the following: raised triglycerides, low HDL-C, raised blood pressure, raised fasting plasma glucose)
- Willingness to participate and provide written informed consent

Exclusion criteria:

- Significant alcohol intake (>20 g/day women; >30 g/day men)
- Known chronic liver diseases (viral hepatitis, autoimmune, Wilson's disease, hemochromatosis)
- Prior liver surgery or malignancy
- Pregnant women
- Patients on hepatotoxic drugs (e.g., methotrexate, amiodarone)

Data collection: Detailed clinical history (demographics, anthropometry, dietary and physical activity history, alcohol intake, comorbidities). Anthropometric measurements included BMI, waist circumference, blood pressure.

Laboratory investigations:

- Fasting plasma glucose, HbA1c
- Lipid profile (triglycerides, HDL, LDL, total cholesterol)
- Liver function tests (ALT, AST, ALP, bilirubin, albumin)
- Viral hepatitis serologies (HBsAg, anti-HCV)
- Ultrasonography of the abdomen performed by experienced radiologists to detect hepatic steatosis (graded 1-3 based on echogenicity).

Outcome measures: Primary outcome was prevalence of NAFLD in MetS versus non-MetS patients. Secondary outcomes included associations between individual MetS components and NAFLD.

Statistical analysis: Continuous variables were expressed as mean $\pm$ SD; categorical as proportions. χ^2 test compared categorical variables; Student's t-test compared means. Logistic regression identified independent predictors of NAFLD. A p-value <0.05 was considered statistically significant.

Results

Table 1. Baseline Demographics (n=250)

Characteristic	Value
Mean age (years)	44.8 $\pm$ 10.2
Male : Female	142 (56.8%) : 108 (43.2%)
BMI (kg/m ²)	28.7 $\pm$ 4.1
Waist circumference (cm)	98.3 $\pm$ 10.7

Table 2. Prevalence of MetS and NAFLD

Parameter	n (%)
Metabolic Syndrome	120 (48.0%)
NAFLD overall	147 (58.8%)
NAFLD with MetS	92 (77.0%)
NAFLD without MetS	55 (42.3%)

Table 3. Distribution of MetS Components in NAFLD Patients

Component	Prevalence in NAFLD (%)
Central obesity	82%
Hypertriglyceridemia	71%
Low HDL-C	64%
Hypertension	60%
Hyperglycemia	68%

Table 4. Liver Function Tests in NAFLD vs non-NAFLD

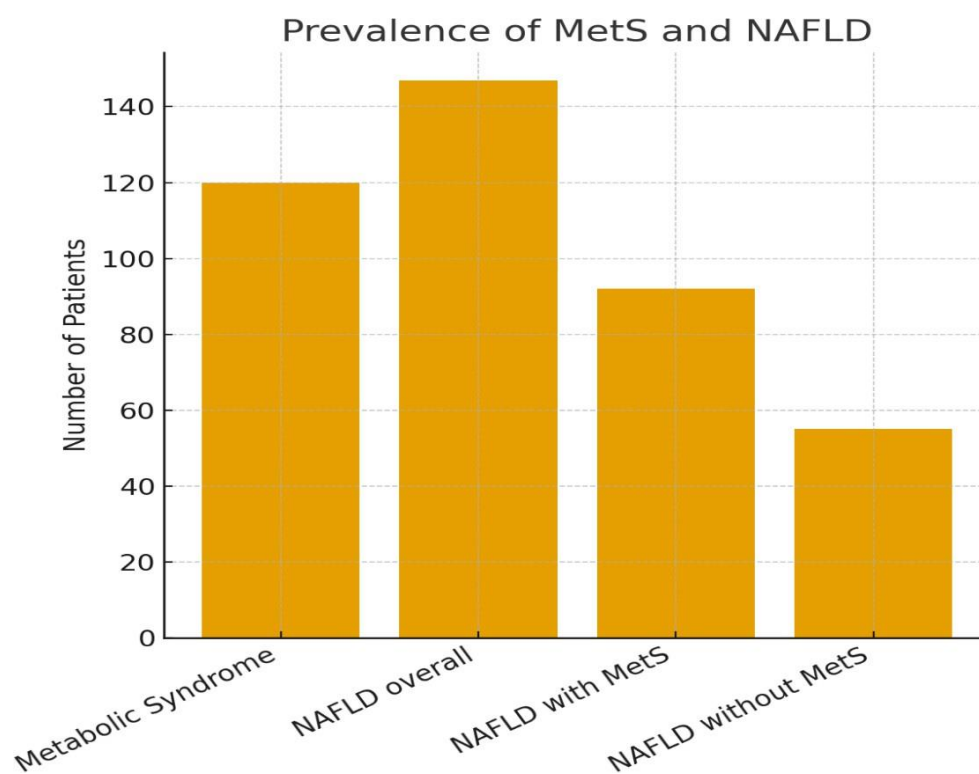
Parameter	NAFLD (mean $\pm$ SD)	Non-NAFLD	p-value
ALT (U/L)	52.4 $\pm$ 15.7	29.8 $\pm$ 12.3	<0.001
AST (U/L)	45.6 $\pm$ 14.2	27.3 $\pm$ 9.6	<0.001
ALT/AST ratio >1	68%	29%	<0.001

Table 5. Logistic Regression Predictors of NAFLD

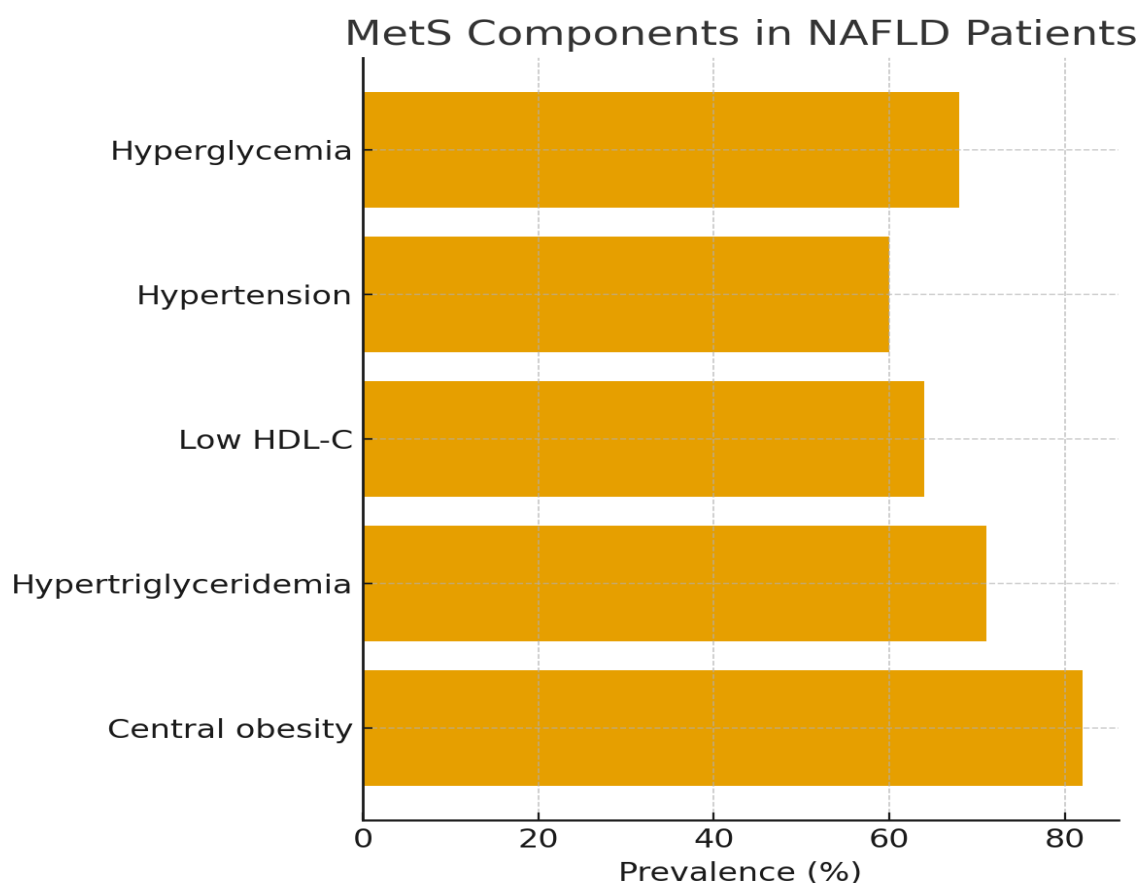
Factor	aOR (95% CI)	p-value
Central obesity	2.8 (1.7-4.6)	<0.001
Hypertriglyceridemia	2.3 (1.4-3.8)	0.001
Hyperglycemia	1.9 (1.1-3.3)	0.015
Hypertension	1.6 (0.9-2.7)	0.07

Table 6. NAFLD Severity (Ultrasound grading) in MetS vs Non-MetS

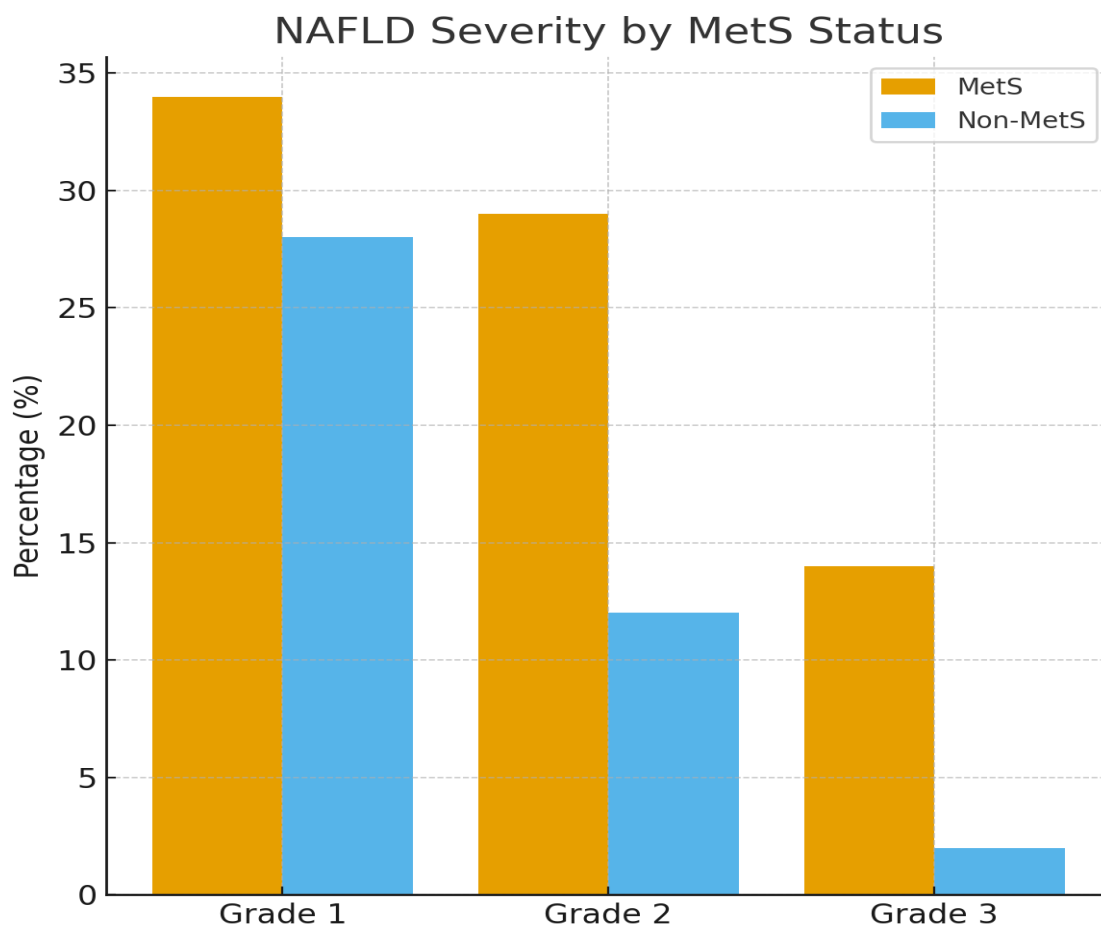
Grade	MetS (%)	Non-MetS (%)
Grade 1	34	28
Grade 2	29	12
Grade 3	14	2



Graph 1: Prevalence of MetS and NAFLD



Graph 2. Logistic Regression Predictors of NAFLD



Graph 3. NAFLD Severity (Ultrasound grading) in MetS vs Non-MetS

Discussion

This study demonstrates a strong association between metabolic syndrome and NAFLD, consistent with global evidence that NAFLD represents the hepatic manifestation of MetS.^{6,7,12} Nearly 77% of patients with MetS had NAFLD, significantly higher than non-MetS individuals, reflecting shared pathophysiological pathways of insulin resistance, obesity, and dyslipidemia.

Our findings align with Younossi et al., who reported NAFLD prevalence of ~70-80% in patients with MetS.⁹ Central obesity emerged as the strongest predictor, supporting evidence that visceral adiposity drives hepatic fat accumulation via increased lipolysis and free fatty acid flux.¹¹ Hypertriglyceridemia was the second strongest predictor, consistent with previous Indian and Asian studies where dyslipidemia was a key risk factor.^{16,19}

We observed elevated ALT and AST in NAFLD patients, with ALT predominance, a hallmark of hepatic steatosis. Similar patterns have been reported in population-based cohorts.²⁰ However, normal transaminases do not exclude NAFLD, highlighting the role of imaging in diagnosis.²¹ The distribution of ultrasound severity showed higher grades of steatosis in MetS, reinforcing the cumulative effect of multiple metabolic abnormalities on disease progression. This supports the “multiple-hit” hypothesis, where obesity, insulin resistance, and inflammatory cytokines act synergistically.^{14,23}

International guidelines recommend screening for NAFLD in high-risk groups, including MetS and type 2 diabetes.^{17,18} Our study reinforces the rationale for early ultrasound evaluation in such patients, especially in resource-limited settings. Lifestyle modification remains the cornerstone of management, with weight loss >7-10% reducing steatosis and improving fibrosis.^{22,24}

Strengths of this study include robust clinical and biochemical characterization and use of standardized IDF criteria. Limitations include reliance on ultrasonography rather than liver biopsy or elastography, single-centre design, and cross-sectional nature precluding causal inference. In conclusion, our study highlights the significant burden of NAFLD in patients with MetS, emphasizing the need for integrated cardiometabolic-hepatology approaches in prevention and management.

Conclusion

NAFLD prevalence is significantly higher in patients with MetS, with central obesity and hypertriglyceridemia as the strongest predictors. Routine screening for NAFLD should be considered in all MetS patients. Lifestyle interventions targeting weight reduction, diet, and exercise remain the primary strategy, supported by pharmacological therapy in selected cases.

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