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A Study to Assess the Association between Nonalcoholic Fatty Liver Disease (NAFLD) and Microalbuminuria in Non-Diabetic Adult Subjects

[1]Nishant Singh, Department of General Medicine, GSVM Medical College, Kanpur, U.P
[1]AC Gupta, Department of General Medicine, GSVM Medical College, Kanpur, U.P
[2]Tanu Midha, Department of Community Medicine, GSVM Medical College, Kanpur, U.P
[3]Vinay Kumar, Department of Gastroenterology, PMSSY, GSVM Medical College, Kanpur, U.P
Corresponding Author: Nishant Singh, Department of General Medicine, GSVM Medical College, Kanpur, U.P

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Abstract

Introduction: Nonalcoholic fatty liver disease (NAFLD) is a leading cause of chronic liver disease worldwide and is increasingly recognized as a multisystem disorder. Microalbuminuria, an early marker of renal dysfunction, has been associated with metabolic disorders, including NAFLD. While the relationship between NAFLD and microalbuminuria is well established in diabetic individuals, its prevalence and association in non-diabetic adults remain unclear. Understanding this link may provide insights into early renal impairment in NAFLD patients without diabetes.

Objectives:

- To evaluate the association between NAFLD and microalbuminuria in non-diabetic adults.
- To assess the prevalence of microalbuminuria in NAFLD patients without diabetes.
- To analyze the correlation between NAFLD severity (measured by FIB-4 scoring and Fibroscan parameters) and microalbuminuria.

Methods: This cross-sectional study included 132 non-diabetic adult NAFLD patients (F0–F2 grade on Fibroscan) with no history of alcohol consumption, kidney disease, or secondary liver disease. Biochemical and radiological investigations included liver function tests, urine albumin-to-creatinine ratio (ACR), and Fibroscan analysis. The presence of microalbuminuria was defined as ACR between 30–300 mg/g. Statistical analysis included correlation tests and logistic regression models.

Results: Microalbuminuria was present in 39.39% of NAFLD patients. The prevalence of microalbuminuria increased with NAFLD severity, with 19.23% of patients in the low-risk fibrosis category (FIB-4 < 1.3) having microalbuminuria, compared to 51.92% in the high-risk fibrosis group (FIB-4 > 2.67). A statistically significant correlation was observed between microalbuminuria and increasing Fibroscan scores ($p < 0.001$).

Conclusion: This study highlights a significant association between NAFLD and microalbuminuria in non-diabetic adults, with increasing liver fibrosis severity

correlating with higher rates of microalbuminuria. These findings suggest that NAFLD may contribute to early renal dysfunction, even in the absence of diabetes. Routine screening for microalbuminuria in NAFLD patients could aid in the early detection and prevention of kidney-related complications.

Keywords: Nonalcoholic fatty liver disease (NAFLD), microalbuminuria, non-diabetic adults, liver fibrosis, kidney dysfunction, Fibroscan, FIB-4 score.

Introduction

Nonalcoholic fatty liver disease (NAFLD) is the most common cause of chronic liver disease globally, affecting around one-quarter of the population. It ranges from simple steatosis to nonalcoholic steatohepatitis (NASH), which may progress to fibrosis, cirrhosis, and hepatocellular carcinoma. Increasingly, NAFLD is recognized as a multisystem disease, not limited to the liver but also associated with obesity, insulin resistance, dyslipidemia, metabolic syndrome, cardiovascular disease, and chronic kidney disease (CKD).

Microalbuminuria, defined as a urine albumin-to-creatinine ratio (ACR) between 30–300 mg/g, is an early marker of renal dysfunction and a predictor of CKD progression and cardiovascular events. The mechanisms driving microalbuminuria, such as endothelial dysfunction, oxidative stress, insulin resistance, and chronic inflammation, overlap with those underlying NAFLD, suggesting a potential link. While this association is well documented in diabetic populations, evidence is scarce in non-diabetic adults. Given that NAFLD can independently contribute to systemic complications, exploring its association with microalbuminuria in non-diabetic individuals is clinically important.

Aims and Objectives

The primary aim of this study was to evaluate the association between NAFLD and microalbuminuria in non-diabetic adults. Secondary objectives included estimating the prevalence of microalbuminuria in this group, analyzing its correlation with NAFLD severity as measured by FIB-4 scoring and Fibroscan parameters, and contributing to the understanding of NAFLD as an early marker of renal dysfunction in non-diabetic adults.

Materials and Methods

This was a cross-sectional analytical study carried out from February 2024 to February 2025 at GSVM Medical College, Kanpur. A total of 132 patients aged ≥18 years with NAFLD confirmed on Fibroscan (F0–F2 fibrosis) were included. All were strictly non-diabetic, afebrile, and without history of alcohol misuse, kidney disease, or secondary liver disease. Exclusion criteria comprised diabetes, prediabetes, CKD, viral hepatitis, HIV, cirrhosis, recent infection, or nephrotoxic drug use.

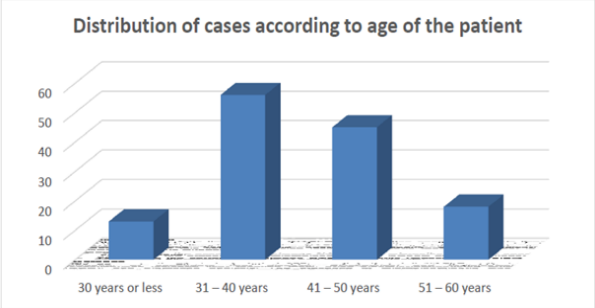
Data collection included demographic details, BMI, biochemical tests (CBC, LFTs, lipid profile, renal profile, fasting and postprandial glucose, HbA1c), and urine ACR to detect microalbuminuria. Imaging comprised ultrasound, Fibroscan (CAP and E-Median scores), and echocardiography to exclude cardiac dysfunction. Microalbuminuria was defined as ACR 30–300 mg/g. Statistical analysis was performed using SPSS v20, employing chi-square, ANOVA, Pearson correlation, and ROC curve analysis. A p-value <0.05 was considered significant. Ethical clearance and informed consent were obtained.

Results

The mean age of participants was 40.93 years (range 28–60), with males comprising 56.06% and females 43.93%. Most patients were overweight, with a mean BMI of 26.10 kg/m² and 70.45% classified as overweight.

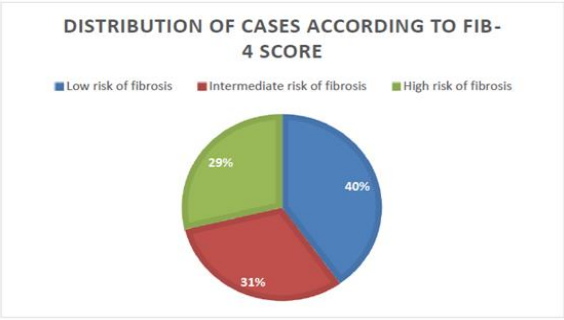
Biochemical analysis revealed mean triglycerides of 215.57 mg/dL, HDL of 37.62 mg/dL, AST 39.09 U/L, ALT 47.83 U/L, and platelet count 123.31 $\times 10^9$/L. Fibroscan showed a mean CAP score of 271.21 and E-Median score of 4.90.

Graph 1:

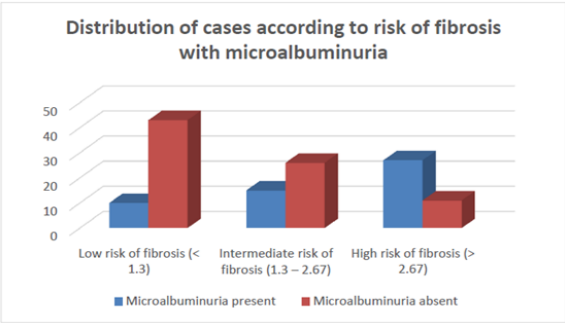


According to FIB-4 scoring, 40.15% had low fibrosis risk (<1.3), 31.06% intermediate (1.3–2.67), and 28.78% high risk (>2.67). Microalbuminuria was detected in 39.39% of patients (52 of 132). Stratification showed that 19.23% of low-risk fibrosis patients had microalbuminuria, rising to 28.84% in intermediate-risk and 51.92% in high-risk fibrosis groups. This demonstrated a strong positive correlation (r=0.428, p<0.001).

Graph 2:



Graph 3:

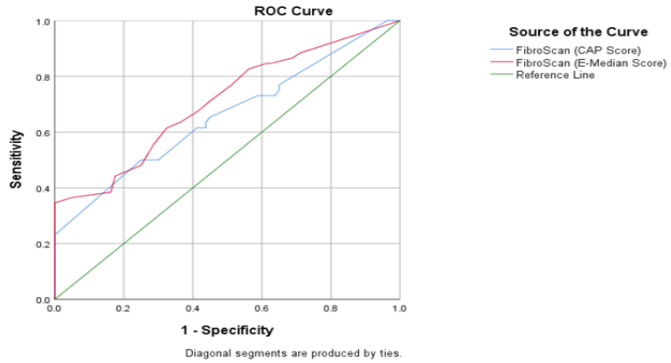


Comparison of Fibroscan parameters showed significantly higher CAP scores (274.52 vs 269.06; p=0.001) and E-Median scores (5.32 vs 4.62; p<0.001) in patients with microalbuminuria. ROC analysis confirmed both CAP and E-Median as predictors of microalbuminuria, with AUC values of 65.8% and 70.8%, respectively. The optimal cutoffs were 274.5 for CAP and 4.85 for E-Median.

Table 1:

	CAP Score	E-Median Score
The area under the curve	65.8%	70.8%
The ideal cutoff cutoff value	274.50	4.85
p-value	0.001	<0.001
Sensitivity	61.5%	63.5%
Specificity	58.7%	63.7%

Graph 4:



Discussion

The study demonstrated that microalbuminuria is highly prevalent in non-diabetic NAFLD patients, affecting nearly 40% of cases, suggesting early renal involvement. This finding is consistent with Yilmaz et al. (2009) and Musso et al. (2014), who reported a similar prevalence and concluded that NAFLD is an independent risk factor for kidney dysfunction. The positive correlation between fibrosis severity and microalbuminuria mirrors findings from Targher et al. (2010) and Sun et al. (2017), both of which established liver fibrosis as a predictor of renal impairment.

The underlying mechanisms likely include systemic endothelial dysfunction, chronic low-grade inflammation (mediated by CRP, IL-6, TNF-α), insulin resistance leading to glomerular hyperfiltration, and dyslipidemia-induced lipotoxicity. These factors act synergistically to promote renal damage in NAFLD patients.

From a clinical standpoint, these results emphasize that NAFLD is not only a hepatic disorder but a multisystem condition with renal implications. Routine microalbuminuria screening should be integrated into NAFLD management, even for non-diabetic individuals. Early identification allows timely lifestyle modifications such as weight reduction, diet changes, and increased physical activity. Pharmacological agents such as statins, GLP-1 receptor agonists, and SGLT2 inhibitors may hold promise in protecting both liver and kidney function.

The strengths of the study include its focus on a non-diabetic population and use of non-invasive fibrosis assessments. Limitations include its single-center, cross-sectional design, which restricts generalizability and prevents causal inference. The absence of liver biopsy is another limitation, as it remains the gold standard for fibrosis assessment.

Conclusion

This study established that NAFLD is significantly associated with microalbuminuria in non-diabetic adults, with a prevalence of 39.39%. The risk of microalbuminuria increased progressively with advancing fibrosis severity, from 19.23% in low-risk patients to 51.92% in high-risk groups. Fibroscan parameters, particularly E-Median score, also correlated with microalbuminuria, making them useful non-invasive predictors of early renal dysfunction.

These findings confirm that NAFLD is a multisystem disorder, with implications for renal health even in the absence of diabetes. Screening for microalbuminuria in NAFLD patients could allow earlier identification of those at risk for CKD and cardiovascular disease, leading to better outcomes through timely intervention. Future multicenter and longitudinal studies are needed to clarify causal pathways and assess therapeutic strategies. Ultimately, a multidisciplinary approach to NAFLD management, addressing both hepatic and systemic complications, is essential for improving patient care.

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