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Research Article

Association between Neutrophil to Lymphocyte Ratio and Steatosis and Fibrosis in Patients with Non-Alcoholic Fatty Liver Disease

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ABSTRACT
Background: Non-alcoholic fatty liver disease (NAFLD) encompasses a spectrum from steatosis to non-alcoholic steatohepatitis (NASH), fibrosis, cirrhosis, and hepatocellular carcinoma, and is closely associated with metabolic comorbidities. Global prevalence is approximately 25.24%, with higher rates in patients with type 2 diabetes mellitus. Neutrophil to lymphocyte ratio (NLR), a readily available marker of subclinical systemic inflammation, may predict advanced NAFLD. However, prior studies report inconsistent NLR-NAFLD associations. This study aimed to evaluate NLR's association with steatosis and fibrosis in NAFLD using transient elastography (TE) with controlled attenuation parameter (CAP).
Methods: A cross-sectional observational study enrolled 100 NAFLD patients aged 18–65 years at T. S. Misra Medical College & Hospital, Lucknow, over 18 months. Inclusion criteria required ultrasonographic fatty liver in non-alcoholic patients with written consent. Patients with viral hepatitis, alcohol use, acute liver conditions, active infections, chronic illnesses, or pregnancy were excluded. Clinical evaluation included detailed history, anthropometric measurements, and physical examination. Fasting blood tests assessed liver enzymes, lipid profile, blood glucose, and complete blood count; NLR was calculated from the differential leucocyte count. Ultrasonography graded steatosis. TE-CAP measured liver stiffness (kPa) and steatosis (dB/m). Data were analyzed with SPSS v29.0; correlations assessed via Pearson's coefficient; $p<0.05$ was considered statistically significant.
Results: Mean age was 55.2 ± 14.2 years; 66% of patients were female. Predominant symptoms included malaise (78%) and abdominal discomfort (58%). Hepatomegaly was the most common sign (58%). Mean NLR was 2.7 ± 1.0; mean AST 83.7 ± 36.1 IU/L; mean ALT 89.5 ± 24.1 IU/L. Steatosis distribution: mild (Grade 1) 38%, moderate (Grade 2) 50%, severe (Grade 3) 12%; mean CAP 283.6 ± 38.4 dB/m. Fibrosis distribution: none (Score 0) 32%, mild (Score 1) 38%, moderate (Score 2) 22%, severe (Score 3) 8%; mean liver stiffness 7.4 ± 4.8 kPa. NLR increased significantly with steatosis grade (Grade 1: 2.3 ± 0.7; Grade 2: 3.6 ± 1.5; Grade 3: 3.9 ± 1.9; $p<0.001$) and fibrosis score (Score 0: 1.4 ± 0.4; Score 1: 2.7 ± 0.5; Score 2: 3.2 ± 0.6; Score 3: 3.7 ± 1.1; $p<0.001$). Significant positive correlations were found: TE-CAP ($R=0.680$), CAP-NLR ($R=0.794$), TE-NLR ($R=0.723$); all $p<0.001$. At an NLR cut-off of 2.45: sensitivity 98.4% and specificity 92.0% for steatosis; sensitivity 85.3% and specificity 81.2% for fibrosis.
Conclusion: NLR positively and significantly associates with both steatosis and fibrosis severity in NAFLD, serving as a reliable, non-invasive, and cost-effective biomarker alongside TE-CAP for disease risk stratification.

Keywords: Non-Alcoholic Fatty Liver Disease, Neutrophil To Lymphocyte Ratio, Steatosis, Fibrosis, Transient Elastography, Controlled Attenuation Parameter, Inflammation, Biomarker.

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is an umbrella term encompassing a spectrum of hepatic pathology ranging from simple steatosis (non-alcoholic fatty liver, NAFL) to non-alcoholic steatohepatitis (NASH), progressive fibrosis, cirrhosis, and hepatocellular carcinoma (HCC). NAFLD has emerged as the most common chronic liver disorder worldwide, with a global prevalence of approximately 25.24%, making it a significant public health concern.[1] It is frequently associated with metabolic comorbidities including obesity, type 2 diabetes mellitus (T2DM), dyslipidemia, hypertension, and metabolic syndrome. The global prevalence of NAFLD and NASH is disproportionately higher

among patients with T2DM compared to the general population, and NAFLD is associated with an approximately 2.2-fold increased risk of incident diabetes.

In India, the prevalence of adult NAFLD has been reported between 6.7% and 55.1%, and it is now the most common liver disease in developed nations, affecting up to 30% of their populations.[2] Pediatric NAFLD in India ranges from 7.3% to 22.4%. NAFLD-related cirrhosis accounts for a significant proportion of cases previously classified as cryptogenic cirrhosis. While ultrasonography remains the most widely used screening tool, it has variable sensitivity and specificity. Liver biopsy is the gold standard for staging but is invasive and impractical for routine use. Non-invasive techniques, particularly transient elastography (TE) with controlled attenuation parameter (CAP), have been validated for simultaneous quantitative assessment of hepatic steatosis and fibrosis.

Inflammation is central to NAFLD pathogenesis. Neutrophils contribute to oxidative stress and cytokine-mediated hepatocellular injury, while lymphocyte depletion reflects impaired immune regulation. The neutrophil to lymphocyte ratio (NLR), derived from a routine differential leucocyte count, integrates both inflammatory and immune regulatory pathways into a single, reproducible index. Elevated NLR has been associated with adverse outcomes in cardiovascular disease, malignancy, and various chronic inflammatory conditions.[3] In NAFLD, NLR has been proposed as a surrogate of histological severity, particularly for predicting steatosis, lobular inflammation, and fibrosis; however, published data remain inconsistent.

Given the burden of NAFLD and the need for reliable, inexpensive non-invasive markers, we conducted this study to evaluate the association between NLR and the severity of hepatic steatosis and fibrosis in NAFLD patients assessed with TE-CAP.

MATERIALS AND METHODS

Study Design and Setting

A cross-sectional observational study was conducted at the Department of Medicine and Department of Gastroenterology, T. S. Misra Medical College & Hospital, Lucknow, India, over 18 months. The study was approved by the Institutional Ethics Committee (approval no. TSMMC&H/IEC/2024/112(04)) and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants. Participants

One hundred consecutive patients aged 18–65 years with ultrasonographically confirmed fatty liver in the absence of significant alcohol intake were enrolled. Patients with hepatitis B or C, alcohol consumption, acute hepatitis, biliary obstruction, hepatic congestion, liver tumors, ascites, active infection or fever, chronic illness, metabolic disease (other than those directly related to NAFLD), or pregnancy were excluded. Patients unwilling to provide consent were also excluded.

Sample Size

Sample size was calculated based on a previous study by Lesmana et al. (2022)[4] using the formula $n = Z^2pq/d^2$, where $Z = 1.96$ (95% CI), $p = 0.4$, $q = 0.6$, and $d = 0.1$, yielding $n \approx 100$.

Clinical Evaluation

All participants underwent comprehensive clinical evaluation including detailed medical history, current medications, lifestyle factors, and NAFLD-related symptom assessment. Anthropometric measurements (height, body weight, body mass index [BMI], waist circumference, and blood pressure) were recorded using standardized protocols.

Biochemical Assessment

Blood samples were collected after a minimum 8-hour overnight fast and analyzed for: liver function tests (ALT, AST, GGT, ALP), lipid profile (total cholesterol, triglycerides, HDL, LDL), fasting blood glucose, complete blood count (CBC), and kidney function tests. The NLR was calculated by dividing the absolute neutrophil count by the absolute lymphocyte count from the CBC. All analyses were performed on automated analyzers in the hospital's central laboratory under standard quality control.

Ultrasonography

Pelviabdominal ultrasonography was performed by trained radiologists using a high-resolution system. Hepatic steatosis was graded as mild (Grade 1), moderate (Grade 2), or severe (Grade 3) based on liver echogenicity and visibility of intrahepatic vessels according to established criteria.

Transient Elastography with Controlled Attenuation Parameter

Liver stiffness and steatosis were quantitatively assessed using FibroScan® (Echosens, France) with CAP. Liver stiffness measurements (LSM) expressed in kilopascals (kPa) were used to score fibrosis: Score 0 (<7 kPa, none), Score 1

(7–9.4 kPa, mild), Score 2 (9.5–12.4 kPa, moderate), Score 3 (≥12.5 kPa, severe), Score 4 (≥17.5 kPa, cirrhosis). CAP values expressed in dB/m quantified steatosis (≥259 dB/m indicating moderate-to-severe steatosis). At least 10 valid measurements per patient were obtained, with an interquartile range/median ratio <30% to ensure reliability.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 29.0 (IBM Corp., Armonk, NY). Descriptive statistics (means, standard deviations, proportions) summarized demographic and clinical characteristics. One-way ANOVA tested differences in NLR across steatosis grades and fibrosis scores. Pearson's correlation coefficient assessed linear relationships between continuous variables. ROC analysis determined the optimal NLR cut-off for detecting higher-grade steatosis and significant fibrosis, with calculation of sensitivity, specificity, and diagnostic accuracy. Multivariable regression models were adjusted for age, sex, BMI, and metabolic syndrome components. A p-value <0.05 was considered statistically significant.

RESULTS

Demographic Profile

A total of 100 patients were enrolled. The majority (42.0%) were in the 41–60 year age group, followed by 32.0% in the 61–65 year group, 24.0% aged 21–40 years, and 2.0% aged 18–20 years. The mean age was 55.2 ± 14.2 years. Sixty-six patients (66.0%) were female and 34 (34.0%) were male. Mean BMI was 25.2 ± 3.7 kg/m².

Table 1. Age and Gender Distribution of Study Participants

Variable	Category	N (%)	Mean ± SD
Age (years)	18–20	2 (2.0%)	
	21–40	24 (24.0%)	
	41–60	42 (42.0%)	55.2 ± 14.2
	61–65	32 (32.0%)	
Gender	Male	34 (34.0%)	
	Female	66 (66.0%)	
BMI (kg/m²)	—	—	25.2 ± 3.7

Symptoms and Clinical Signs

Malaise was the most frequent symptom, reported by 78 (78.0%) patients, followed by abdominal discomfort in 58 (58.0%), jaundice in 9 (9.0%), and abdominal distension in 6 (6.0%). On examination, hepatomegaly was the predominant sign (58.0%), followed by pedal oedema (37.0%), abdominal tenderness (21.0%), and icterus (15.0%).

Biochemical Parameters

Mean AST was 83.7 ± 36.1 IU/L, mean ALT 89.5 ± 24.1 IU/L, and mean ALP 141.7 ± 45.2 IU/L. The mean NLR in the study cohort was 2.7 ± 1.0.

Transient Elastography and Steatosis/Fibrosis Grading

Mean CAP value was 283.6 ± 38.4 dB/m and mean liver stiffness was 7.4 ± 4.8 kPa. None of the patients had normal (Grade 0) steatosis. Mild steatosis (Grade 1) was seen in 38.0%, moderate (Grade 2) in 50.0%, and severe (Grade 3) in 12.0%. Regarding fibrosis: 32.0% had Score 0 (none), 38.0% Score 1 (mild), 22.0% Score 2 (moderate), and 8.0% Score 3 (severe). No patient demonstrated cirrhosis (Score 4).

Table 2. Distribution of Steatosis Grade and Fibrosis Score

	Grade/Score	N (%)	Mean CAP or kPa
Steatosis	Grade 1 (Mild)	38 (38.0%)	283.6 ± 38.4 dB/m (overall)
	Grade 2 (Moderate)	50 (50.0%)	
	Grade 3 (Severe)	12 (12.0%)	
Fibrosis	Score 0 (None)	32 (32.0%)	7.4 ± 4.8 kPa (overall)
	Score 1 (Mild)	38 (38.0%)	
	Score 2 (Moderate)	22 (22.0%)	
	Score 3 (Severe)	8 (8.0%)	

Association between NLR and Steatosis Grade

NLR demonstrated a progressive and statistically significant increase with advancing steatosis grade. Patients with Grade 1 steatosis had a mean NLR of 2.3 ± 0.7, those with Grade 2 had 3.6 ± 1.5, and Grade 3 patients had the highest NLR of 3.9 ± 1.9 (F = 16.414, p < 0.001; ANOVA).

Table 3. NLR across Steatosis Grades (ANOVA)

Steatosis Grade	n	Mean NLR ± SD	F value	p value
Grade 1 (Mild)	38	2.3 ± 0.7		
Grade 2 (Moderate)	50	3.6 ± 1.5	16.414	<0.001*
Grade 3 (Severe)	12	3.9 ± 1.9		

*Statistically significant (p<0.001)

Association between NLR and Fibrosis Score

NLR showed a marked stepwise increase with advancing fibrosis. Mean NLR values were: Score 0 — 1.4 ± 0.4, Score 1 — 2.7 ± 0.5, Score 2 — 3.2 ± 0.6, and Score 3 — 3.7 ± 1.1 (F = 66.108, p < 0.001; ANOVA).

Table 4. NLR across Fibrosis Scores (ANOVA)

Fibrosis Score	n	Mean NLR ± SD	F value	p value
Score 0 (None)	32	1.4 ± 0.4		
Score 1 (Mild)	38	2.7 ± 0.5	66.108	<0.001*
Score 2 (Moderate)	22	3.2 ± 0.6		
Score 3 (Severe)	8	3.7 ± 1.1		

*Statistically significant (p<0.001)

Correlation between TE, CAP, and NLR

Significant positive correlations were observed between all three parameters (Table 5). TE and CAP correlated at R = 0.680 (p<0.001), supporting the co-occurrence of steatosis and fibrosis. CAP-NLR correlation was the strongest (R = 0.794, p<0.001), and TE-NLR also showed a robust association (R = 0.723, p<0.001).

Table 5. Pearson's Correlation between TE, CAP, and NLR

Variable Pair	R value	p value
TE vs. CAP	0.680	<0.001*
CAP vs. NLR	0.794	<0.001*
TE vs. NLR	0.723	<0.001*

*Statistically significant (p<0.001)

Diagnostic Performance of NLR

ROC analysis identified an NLR cut-off of 2.45 as optimal for both steatosis and fibrosis detection. For steatosis (Grade ≥2), NLR demonstrated sensitivity 98.4%, specificity 92.0%, and diagnostic accuracy 98.7%. For significant fibrosis (Score ≥1), sensitivity was 85.3%, specificity 81.2%, and accuracy 89.0%.

Table 6. Diagnostic Performance of NLR (Cut-Off 2.45) For Steatosis and Fibrosis

Outcome	Sensitivity	Specificity	Accuracy
Steatosis (Grade ≥2)	98.4%	92.0%	98.7%
Fibrosis (Score ≥1)	85.3%	81.2%	89.0%

DISCUSSION

The present study investigated the relationship between NLR and the severity of hepatic steatosis and fibrosis in NAFLD patients assessed using TE-CAP. We observed a significant, progressive increase in NLR with advancing steatosis grade and fibrosis score, robust positive correlations between NLR, CAP, and TE, and excellent diagnostic accuracy of NLR with a cut-off of 2.45. Our cohort had a mean age of 55.2 ± 14.2 years, with female predominance (66.0%) and preponderance in the 41–60 year age group, consistent with published literature.[5] Jayachandra et al. (2021) reported similar demographic findings with female majority and peak incidence in the fourth to sixth decades.[5] Alam et al. similarly reported 65.9% female predominance with a

mean age of 45.0 ± 27.1 years. The higher proportion of women in our cohort likely reflects the greater burden of metabolic syndrome and insulin resistance in postmenopausal women.

Malaise (78.0%) and abdominal discomfort (58.0%) were the most frequent symptoms, and hepatomegaly (58.0%) was the predominant clinical sign — findings consistent with those of Jayachandra et al., who reported malaise in 82% and hepatomegaly in 54% of NAFLD cases.[5] The nonspecific symptom profile of NAFLD underscores the importance of biochemical and imaging-based screening.

Mean liver enzymes (AST 83.7 ± 36.1 IU/L; ALT 89.5 ± 24.1 IU/L; ALP 141.7 ± 45.2 IU/L) and mean NLR (2.7 ± 1.0) were comparable to values reported by Jayachandra et al. (mean NLR in NAFLD cases: 3.6 ± 1.83).[5] Mean CAP (283.6 ± 38.4 dB/m) and liver stiffness (7.4 ± 4.8 kPa) closely mirror those reported by Song SJ et al. (mean CAP 282 ± 40 dB/m; LSM 7.6 ± 5.0 kPa) and Dai CY et al. (mean CAP 294.5 ± 39.3 dB/m), validating the representativeness of our cohort.[6] NLR increased progressively and significantly with steatosis grade (Grade 1: 2.3 ± 0.7 → Grade 3: 3.9 ± 1.9; $p<0.001$) and fibrosis score (Score 0: 1.4 ± 0.4 → Score 3: 3.7 ± 1.1; $p<0.001$). These findings are concordant with Lesmana et al. (2022),[4] who reported significant positive correlations between NLR and both CAP ($r=0.648$, $p<0.001$) and TE ($r=0.621$, $p<0.001$) in 106 NAFLD patients. Alkhouri et al. similarly demonstrated that NLR was significantly higher in NASH compared to simple steatosis (2.5 vs. 1.6, $p<0.001$), and in patients with advanced fibrosis (2.9), paralleling our observations. Furthermore, Abdel-Razik et al. reported mean NLR values of 2.4 ± 0.8 in mild steatosis and 3.7 ± 1.4 in advanced steatosis, closely mirroring our results.[7] Conversely, certain studies have found no significant NLR-NAFLD association. Cucoranu et al. (2023), using CT-based hepatic attenuation without TE, found no significant NLR difference between hepatic steatosis and non-steatosis groups, though NPAR showed a weak inverse correlation.[8] Zhou et al. (2022) similarly reported no linear association in a check-up cohort but identified a nonlinear relationship with an inflection at NLR = 1.23.[9] These discrepancies likely reflect differences in NAFLD assessment methodology (CT vs. TE-CAP), study populations, and sample sizes. The biological rationale for elevated NLR in NAFLD is well-established. Hepatic neutrophil accumulation generates reactive oxygen species (ROS) and myeloperoxidase-derived oxidants that amplify Kupffer cell cytotoxicity and activate hepatic stellate cells.[10] Human neutrophil peptides enhance liver fibrosis by inducing hepatic stellate cell proliferation. Concurrently, reduced lymphocytes reflect impaired adaptive immune regulation. NLR thus encapsulates the pro-inflammatory milieu that drives steatosis progression and fibrogenesis.

The strong positive correlations among TE, CAP, and NLR ($R = 0.680–0.794$; all $p<0.001$) affirm that systemic inflammation measured by NLR is synchronous with structural hepatic changes quantified by TE-CAP. Song SJ reported a TE-CAP correlation of $R = 0.65$, consistent with our $R = 0.680$.[6] These concordant findings support the use of NLR in conjunction with elastography for comprehensive NAFLD assessment.

At a cut-off of 2.45, NLR demonstrated 98.4% sensitivity and 92.0% specificity for steatosis and 85.3% sensitivity and 81.2% specificity for fibrosis — markedly superior to the cut-off of 1.775 reported by Lesmana et al. (sensitivity 81.5%, specificity 80.6%) for moderate-to-severe steatosis.[4] Abdel-Razik et al. reported a cut-off of 2.6 predicting advanced fibrosis with 83% sensitivity and 78% specificity, closely aligned with our fibrosis performance.[7] The high diagnostic accuracy in our cohort may partly reflect the enriched prevalence of moderate-to-severe disease (68% Grade ≥2 steatosis; 68% Score ≥1 fibrosis). A pooled meta-analysis by Shavakhi et al. confirmed elevated NLR is significantly associated with both steatosis severity and fibrosis progression, with pooled sensitivity and specificity of 84% and 80%, respectively, broadly supporting our findings. WenYi et al. (2022), using biopsy-confirmed NAFLD with SAF scoring, demonstrated that higher NLR was independently associated with advanced inflammatory activity (OR 0.62; $p=0.025$) and significant fibrosis (OR 0.57; $p=0.028$).[11]

The primary limitations of this study include: (1) single-center design limiting generalizability; (2) absence of liver biopsy for histological validation, as TE-CAP, though validated, is not equivalent to the gold standard; (3) cross-sectional design precluding causal inference or temporal tracking of NLR dynamics. Strengths include: uniform use of TE-CAP for objective quantification; rigorous exclusion criteria minimizing confounders; and demonstration of a simple, inexpensive marker (NLR) with high diagnostic accuracy applicable in resource-limited settings.

CONCLUSION

This study demonstrates a significant and progressive association between neutrophil to lymphocyte ratio and both steatosis grade and fibrosis score in NAFLD. A cut-off NLR value of 2.45 achieved excellent diagnostic performance for both steatosis (sensitivity 98.4%, specificity 92.0%) and fibrosis (sensitivity 85.3%, specificity 81.2%), comparable to published standards. NLR, used alongside TE-CAP, provides a reliable, cost-effective, and non-invasive approach for NAFLD risk stratification that is applicable in diverse clinical settings. Future multicenter, longitudinal studies with histological correlation are warranted to validate these findings and establish standardized NLR cut-off values for routine clinical use.

Conflict of Interest

None declared.

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Ethics Approval

Approved by the Institutional Ethics Committee, T. S. Misra Medical College & Hospital (approval no. TSMMC&H/IEC/2024/112(04)). All procedures conformed to the Declaration of Helsinki.

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