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Correlation of serum gamma-glutamyl transferase, lipid profile and inflammatory biomarkers with non-alcoholic fatty liver disease: A prospective observational study in India

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Abstract:
Non-alcoholic fatty liver disease (NAFLD) requires accessible non-invasive biomarkers for early severity assessment. Therefore, it is of interest evaluate the serum GGT, lipid profile and inflammatory biomarkers in 180 adults with ultrasonographically confirmed NAFLD. Serum GGT, total cholesterol, triglycerides, LDL-C, hs-CRP, NLR and PLR increased significantly with advancing NAFLD severity, while HDL-C decreased (all $p<0.001$). NAFLD severity showed positive correlations with GGT, hs-CRP, triglycerides, LDL-C, NLR, PLR and total cholesterol, while HDL-C showed an inverse correlation (all $p<0.001$). Thus, data support serum GGT, lipid abnormalities and inflammatory biomarkers as simple, non-invasive indicators for NAFLD severity assessment and early risk stratification.

Keywords: Non-alcoholic fatty liver disease (NAFLD), gamma-glutamyl transferase, lipid profile, inflammatory biomarkers, hs-CRP, metabolic dysfunction

Background:
Non-alcoholic fatty liver disease (NAFLD), now termed metabolic dysfunction-associated steatotic liver disease (MASLD), is the most common chronic liver disease worldwide. It is strongly associated with obesity, insulin resistance, diabetes and metabolic syndrome and may progress to cirrhosis and hepatocellular carcinoma **[1, 2]**. Insulin resistance, lipid dysregulation, oxidative stress and chronic inflammation contribute to hepatic fat accumulation and disease progression **[3]**. Serum gamma-glutamyl transferase (GGT) reflects hepatocellular injury and oxidative stress and may serve as an accessible biomarker of NAFLD **[4]**. Dyslipidemia, characterized by increased triglycerides, total cholesterol and LDL-C with reduced HDL-C, is commonly associated with NAFLD **[5]**. Inflammatory biomarkers may also reflect disease activity, while liver biopsy remains invasive; therefore, this study evaluated GGT, lipid profile and inflammatory biomarkers as non-invasive indicators of NAFLD severity **[6].** Therefore, it is of interest to evaluate the correlation of serum gamma-glutamyl transferase, lipid profile and inflammatory biomarkers with non-alcoholic fatty liver disease and to determine their utility as non-invasive indicators of disease severity.

Materials and Methods:
Study design and setting:
A prospective observational study was conducted in the Department of General Medicine in collaboration with the Department of Biochemistry at a tertiary care teaching hospital over a period of 12 months, from January 2025 to December 2025. As calculated by the appropriate sample size formula, a total of 180 adult patients attending the outpatient or inpatient departments with ultrasonographically confirmed NAFLD were consecutively recruited during the study period.

Inclusion criteria:
[1] Adults aged 18 years or older
[2] Ultrasonographic evidence of non-alcoholic fatty liver disease
[3] Individuals willing to provide written informed consent

Exclusion criteria:
[1] Significant alcohol consumption (>20 g/day in women or >30 g/day in men)
[2] Viral hepatitis (HBV or HCV infection)
[3] Autoimmune liver disease, Wilson's disease, hemochromatosis, or other chronic liver disorders.
[4] Drug-induced liver injury or use of hepatotoxic medications
[5] Malignancy, acute infection, or chronic inflammatory disorders

Clinical assessment:
A detailed clinical history, including demographic characteristics, lifestyle habits, medical history and medication use, was obtained using a structured proforma. Anthropometric measurements, including height, weight, body mass index (BMI) and waist circumference, were recorded. Blood pressure was measured using standard techniques.

Laboratory investigations:
Serum gamma-glutamyl transferase (GGT), liver function tests, fasting blood glucose and fasting lipid profile, including total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C), were analyzed using an automated biochemistry analyzer following the manufacturer's recommendations. Inflammatory biomarkers, including high-sensitivity C-reactive protein (hs-CRP), were measured using standardized immunoturbidimetric assays.

Complete blood count was performed using an automated hematology analyzer and inflammatory indices such as the neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) were calculated where applicable.

Diagnosis and grading of NAFLD:
The diagnosis of NAFLD was established by abdominal ultrasonography performed by an experienced radiologist who was blinded to laboratory findings. Fatty liver was graded according to standard ultrasonographic criteria (Grade I–III) based on hepatic echogenicity, visualization of intrahepatic vessels and posterior beam attenuation.

Outcome measures:
The primary outcome was to evaluate the correlation between serum gamma-glutamyl transferase levels and the severity of NAFLD. Secondary outcomes included assessment of the association between lipid profile parameters, inflammatory biomarkers and ultrasonographic grades of NAFLD, as well as determination of their diagnostic utility in predicting disease severity.

Statistical analysis:
Data were analyzed using IBM SPSS Statistics version 26.0. Continuous variables were expressed as mean ± standard deviation (SD) or median, while categorical variables were presented as frequencies and percentages. Student's t-test or Mann–Whitney U test for continuous variables and the Chi-square or Fisher's exact test for categorical variables. A two-tailed p value <0.05 was considered statistically significant.

Ethical approval:
The Ethical approval was obtained from the Institutional Ethics Committee and study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Written informed consent was obtained from all participants before enrollment and confidentiality of patient information was maintained throughout the study.

Results:
The study included 180 patients with ultrasonographically confirmed NAFLD, with a mean age of 46.8 ± 11.2 years **(Table 1)**. Males accounted for 104 (57.8%) participants and females for 76 (42.2%), consistent with the higher prevalence of NAFLD reported among men, possibly because of greater visceral fat deposition and the protective effect of oestrogen in premenopausal women. The study population showed a marked metabolic risk profile. The mean BMI was 29.3 ± 3.8 kg/m², which falls in the obese range by Asian-Indian criteria, and the mean waist circumference was 96.8 ± 9.4 cm, indicating central obesity. Type 2 diabetes mellitus was present in 74 (41.1%) patients and hypertension in 68 (37.8%), reflecting the close association of NAFLD with the components of metabolic syndrome **(Table 1)**. Grade I NAFLD was the most common presentation, observed in 74 (41.1%) patients, followed by Grade II in 68 (37.8%) and Grade III in 38 (21.1%) **(Figure 1)**. Although most patients had mild-to-moderate disease, more than one-fifth already had severe steatosis at presentation, which highlights the often silent progression of NAFLD. Mean serum GGT increased progressively with disease severity **(Table 2)**. It rose from 48.9 ± 13.8 U/L in Grade I to 71.6 ± 18.9 U/L in Grade II and 102.4 ± 24.7 U/L in Grade III ($p < 0.001$). Patients with Grade III disease therefore had more than twice the GGT level of those with Grade I disease. GGT is involved in glutathione metabolism and is a marker of hepatocellular injury, so this stepwise rise suggests increasing oxidative stress and hepatocyte damage as hepatic fat accumulation worsens. Lipid abnormalities also worsened progressively with increasing NAFLD grade **(Table 3)**. Total cholesterol increased from 184.5 ± 24.3 mg/dL in Grade I to 221.9 ± 30.6 mg/dL in Grade III. Triglycerides rose from 151.2 ± 39.4 to 219.8 ± 49.5 mg/dL, and LDL-C from 108.7 ± 20.8 to 144.9 ± 28.6 mg/dL (all $p < 0.001$). Over the same range, HDL-C declined significantly from 45.8 ± 6.2 to 37.2 ± 5.1 mg/dL ($p < 0.001$). Triglycerides showed the largest relative increase, of approximately 45%, reflecting the central role of triglyceride accumulation in hepatic steatosis. Elevated triglycerides and LDL-C with reduced HDL-C is the typical pattern of atherogenic dyslipidaemia driven by insulin resistance, and it indicates increasing cardiovascular risk as NAFLD severity advances **(Table 3)**. Inflammatory biomarkers showed a significant stepwise increase with worsening NAFLD grade **(Table 4)**. hs-CRP increased from 2.6 ± 1.1 mg/L in Grade I to 4.4 ± 1.6 mg/L in Grade II and 7.3 ± 2.2 mg/L in Grade III ($p < 0.001$), a nearly threefold rise. NLR increased from 2.1 ± 0.6 to 3.7 ± 1.0, and PLR increased from 118.6 ± 21.4 to 149.5 ± 28.3 between Grade I and Grade III (both $p < 0.001$). These findings indicate greater systemic low-grade inflammation in advanced disease, likely mediated by pro-inflammatory cytokines such as IL-6 and TNF-α released from fat-laden hepatic and adipose tissue. NLR and PLR are derived from a routine complete blood count, which makes them particularly cost-effective markers of this inflammatory burden. On correlation analysis, NAFLD severity showed significant positive correlations with all biochemical and inflammatory parameters except HDL-C **(Table 5)**. Serum GGT showed the strongest correlation ($r = 0.69$, $p < 0.001$), followed by hs-CRP ($r = 0.63$, $p < 0.001$). Triglycerides ($r = 0.58$), LDL-C ($r = 0.55$), NLR ($r = 0.52$) and total cholesterol ($r = 0.49$) were moderately correlated with disease severity, while PLR showed a weaker positive correlation ($r = 0.39$) (all $p < 0.001$). HDL-C showed a significant inverse correlation with NAFLD severity ($r = −0.47$, $p < 0.001$), consistent with its protective and anti-inflammatory role **(Table 5)**. Overall, these findings suggest that markers of hepatocellular injury and systemic inflammation reflect NAFLD severity more closely than lipid parameters. They also support the use of a combined panel of GGT, hs-CRP and lipid markers for non-invasive assessment of disease severity.

Table 1: Baseline demographic and clinical characteristics of the study population (n=180)

Variable	Value
Age (years), Mean ± SD	46.8 ± 11.2
Male	104 (57.8%)

Female	76 (42.2%)
BMI (kg/m²), Mean ± SD	29.3 ± 3.8
Waist circumference (cm), Mean ± SD	96.8 ± 9.4
Hypertension	68 (37.8%)
Type 2 Diabetes Mellitus	74 (41.1%)

Table 2: Serum GGT levels according to NAFLD grade

NAFLD Grade	N	GGT (U/L), Mean ± SD	p-value
Grade I	74	48.9 ± 13.8	<0.001
Grade II	68	71.6 ± 18.9	
Grade III	38	102.4 ± 24.7	

Table 3: Lipid profile according to NAFLD grade

Parameter	Grade I	Grade II	Grade III	p-value
Total cholesterol (mg/dL)	184.5±24.3	201.8±28.5	221.9±30.6	<0.001
Triglycerides (mg/dL)	151.2±39.4	183.5±44.1	219.8±49.5	<0.001
LDL-C (mg/dL)	108.7±20.8	126.4±24.2	144.9±28.6	<0.001
HDL-C (mg/dL)	45.8±6.2	41.3±5.8	37.2±5.1	<0.001

Table 4: Inflammatory biomarkers according to NAFLD grade

Biomarker	Grade I	Grade II	Grade III	p-value
hs-CRP (mg/L)	2.6±1.1	4.4±1.6	7.3±2.2	<0.001
NLR	2.1±0.6	2.8±0.8	3.7±1.0	<0.001
PLR	118.6±21.4	131.8±24.6	149.5±28.3	<0.001

Table 5: Correlation of biochemical parameters with NAFLD severity

Variable	Correlation coefficient (r)	p-value
GGT	0.69	<0.001
Triglycerides	0.58	<0.001
Total cholesterol	0.49	<0.001
LDL-C	0.55	<0.001
HDL-C	-0.47	<0.001
hs-CRP	0.63	<0.001
NLR	0.52	<0.001
PLR	0.39	<0.001

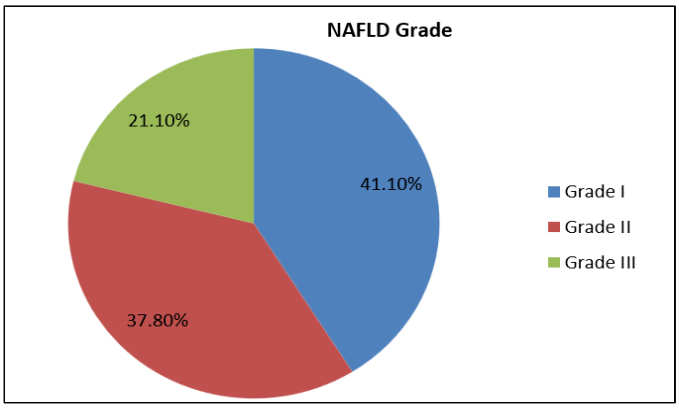


Figure 1: Distribution of patients according to NAFLD grade

Discussion:

A major finding of the present study was the strong positive correlation between serum GGT and NAFLD severity. Similar observations have been reported by Singhai *et al* **[6],** who demonstrated that elevated GGT independently predicted steatosis hepatic and fibrosis in individuals with metabolic dysfunction. Likewise, Panikar *et al* **[7],** identified GGT as an early biochemical indicator reflecting oxidative stress and hepatocellular injury in fatty liver disease. The progressive increase in GGT with worsening disease may be explained by increased oxidative stress, lipid peroxidation and hepatocyte damage accompanying hepatic fat accumulation. As GGT estimation is inexpensive and routinely available, it may serve as a practical biomarker for identifying patients at greater risk of disease progression **[8].** The present study also demonstrated a significant deterioration in lipid profile with advancing NAFLD grade, characterized by increasing triglycerides, total cholesterol and LDL-C together with declining HDL-C. These findings are consistent with those reported by Asadullah *et al* **[8],** who described dyslipidemia as a hallmark of metabolic dysfunction-associated fatty liver disease. Similarly, Pyati *et al* **[9],** reported that elevated triglycerides and LDL-C are strongly associated with hepatic steatosis and cardiovascular risk. Excess hepatic lipid synthesis, impaired fatty acid oxidation and increased insulin resistance are likely mechanisms responsible for these abnormalities. These observations emphasize that routine lipid assessment has value not only for cardiovascular risk stratification but also for identifying patients with progressive NAFLD. Inflammatory biomarkers increased significantly with worsening disease severity in the present study. Patients with Grade III NAFLD demonstrated the highest hs-CRP, NLR and PLR values, suggesting amplification of systemic inflammation during disease progression. Comparable findings were reported by Harshini *et al* **[10]** and Saran *et al* **[11],** observed that inflammatory indices correlate closely with histological severity of fatty liver disease. Similarly, Ray and Kumar showed that chronic low-grade inflammation plays a central role in the transition from simple steatosis to steatohepatitis and fibrosis. Activation of inflammatory pathways by excess lipid accumulation promotes cytokine release, oxidative injury and immune-cell infiltration, thereby accelerating hepatic damage. These inexpensive hematological markers may therefore complement conventional biochemical tests during clinical evaluation. Correlation analysis further demonstrated that serum GGT, triglycerides, LDL-C, hs-CRP, NLR and PLR were positively associated with NAFLD severity, whereas HDL-C showed a significant inverse relationship. Similar correlations have been described by Kannan *et al* **[12]** and Ray and Kumar **[13],** reported that metabolic and inflammatory biomarkers provide useful non-invasive information regarding disease severity. These findings support the concept that no single biomarker adequately reflects NAFLD progression; rather, combining biochemical and inflammatory parameters may improve risk stratification and reduce dependence on invasive investigations such as liver biopsy **[14, 15]**. The clinical implications of the present study are important, particularly in resource-limited healthcare settings where advanced imaging or liver biopsy may not be readily available. Routine estimation of serum GGT together with lipid profile and inflammatory biomarkers may facilitate early identification of patients with advanced NAFLD, enabling timely lifestyle modification, metabolic risk reduction and closer follow-up **[16]**. Incorporating these readily available laboratory parameters into routine clinical practice could improve patient management while minimizing healthcare costs.

Conclusion:

Rising GGT, triglycerides, LDL-C, hs-CRP, NLR and PLR, with declining HDL-C, correlated with worsening ultrasonographic grades of NAFLD. These inexpensive, readily available biomarkers may help with non-invasive risk stratification and severity assessment in resource-limited settings. Larger multicentre longitudinal studies are needed to validate their prognostic value.

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