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Association Between Gamma-Glutamyl Transferase Levels and Framingham-Estimated Cardiovascular Risk in Patients with Non-Alcoholic Fatty Liver Disease: A Cross-Sectional Study

M. C. Lalith Raja Deepak[1], S. Ashok Kumar[2], R. Elanchezian[3]

[1]Assistant Professor, Department of Medicine, Government Kilpauk Medical College, Chennai - 600010, Tamil Nadu, India.
[2]Assistant Professor, Department of Medicine, Government Kilpauk Medical College, Chennai - 600010, Tamil Nadu, India.
[3]Assistant Professor, Department of Medicine, Government Chengalpattu Medical College, Chengalpattu - 603001, Tamil Nadu, India.

Corresponding Author: Dr. R. Elanchezian

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ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) is a common cause of chronic liver test derangement and is closely linked to metabolic syndrome. Patients with NAFLD show a higher burden of cardiovascular risk factors than the general population. Gamma-glutamyl transferase (GGT), routinely measured as part of the liver panel, is a marker of oxidative stress and has been associated with cardiovascular morbidity in several population-based cohorts. Its association with calculated cardiovascular risk specifically in patients with NAFLD warrants further characterisation.
Objective: To categorise patients with ultrasonographic evidence of fatty liver by grade of hepatic steatosis, estimate their serum GGT levels, and examine the association between GGT levels and 10-year cardiovascular risk estimated using the Framingham General Cardiovascular Disease Risk Score.
Materials and Methods: This cross-sectional, single-centre study was conducted over six months (May–October 2017) among 100 consecutive patients with ultrasonographically confirmed fatty liver attending the Departments of General Medicine and Medical Gastroenterology, Government Stanley Medical College Hospital, Chennai. Fatty liver was graded (Grade 1–3) by abdominal ultrasonography, serum GGT was estimated using a standardised laboratory protocol (cut-off 45 IU/L, the institutional upper limit of normal), and 10-year cardiovascular risk was estimated using the Framingham General Cardiovascular Disease Risk Score, categorised as low (<10%) or moderate-to-high (≥10%) risk.
Results: The mean age of the study population was 45.5 years, with 69% men and 31% women. Serum GGT was elevated (>45 IU/L) in 73% of patients, with a mean value of 58.7 IU/L. Seventy-one percent of patients fell into the moderate-to-high Framingham risk category. Elevated GGT was significantly associated with moderate-to-high Framingham risk (84.9% vs 33.3% in the normal-GGT group; $\chi^2 = 25.49$, $p < 0.0001$), with an odds ratio of 11.27 (95% CI 4.04–31.43) and a relative risk of 2.55 (95% CI 1.48–4.38). Neither smoking

status (χ^2 = 0.15, p = 0.93) nor Framingham risk category (χ^2 = 0.86, p = 0.65) varied significantly by grade of hepatic steatosis.
Conclusion: In this cross-sectional study, serum GGT was significantly associated with a higher calculated Framingham cardiovascular risk category in patients with NAFLD, independent of the grade of hepatic steatosis. As a single-centre, cross-sectional study without multivariable adjustment or prospective cardiovascular outcome data, these findings demonstrate an association rather than independent prediction of future cardiovascular events. GGT, being inexpensive and widely available, may nonetheless be a useful adjunct for cardiovascular risk assessment in NAFLD, pending validation in larger prospective studies.

Keywords: Non-alcoholic fatty liver disease; Gamma-glutamyl transferase; Framingham risk score; Cardiovascular risk; Fatty liver; Cross-sectional study

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is among the most common causes of chronic liver disease worldwide. It represents a broad clinical spectrum ranging from simple hepatic steatosis to non-alcoholic steatohepatitis (NASH), which can progress to cirrhosis, hepatocellular failure and, in some cases, hepatocellular carcinoma **[1,2]**. NAFLD is diagnosed in the absence of significant alcohol intake (less than 30 g/day in men and 20 g/day in women) after exclusion of other causes of hepatic steatosis, and is now widely recognised as the hepatic manifestation of metabolic syndrome **[3,4]**.

Patients with NAFLD show a higher burden of cardiovascular risk factors and cardiovascular morbidity than the general population, an association that appears to extend beyond that explained by traditional risk factors such as age, smoking, hypertension and dyslipidaemia **[5,6]**. Among liver function parameters, gamma-glutamyl transferase (GGT) has attracted particular interest: elevated GGT reflects not only hepatobiliary stress but also systemic oxidative stress, and has been associated with cardiovascular morbidity and mortality in several large population-based cohorts **[7,8]**. Because GGT is inexpensive, widely available and already included in routine liver function testing, it is a practical candidate marker for cardiovascular risk assessment in patients with NAFLD.

The Framingham General Cardiovascular Disease Risk Score is a validated tool that estimates 10-year cardiovascular risk using age, sex, smoking status, total cholesterol, HDL cholesterol and blood pressure/ treatment status, and has been used in several studies to characterise cardiovascular risk in NAFLD [9]. However, the association between GGT levels and Framingham-estimated cardiovascular risk specifically in patients with ultrasonographically graded NAFLD is not well characterised in the Indian population.

This study was undertaken to grade patients with ultrasonographic evidence of fatty liver, estimate their serum GGT levels, and examine the association between GGT levels and cardiovascular risk estimated using the Framingham General Cardiovascular Disease Risk Score.

MATERIALS & METHODS

Study design and setting: This was a cross-sectional, observational, single-centre study conducted in the Departments of General Medicine and Medical Gastroenterology, Government Stanley Medical College Hospital, Chennai, over a period of six months (May 2017 to October 2017).

Study population and sampling: A total of 100 consecutive patients with ultrasonographic evidence of fatty liver, presenting to the above departments during

the study period, were enrolled using a convenience (consecutive-sampling) approach; a formal a priori sample-size calculation was not performed, and the final sample size was determined pragmatically by the number of eligible patients identified within the six-month study window. This is acknowledged as a limitation in the discussion.

Inclusion criteria: (i) age ≥18 years; (ii) ultrasonographic evidence of hepatic steatosis (fatty liver), whether detected on evaluation for hepatic symptoms or as an incidental finding during abdominal ultrasonography performed for unrelated indications (e.g. fever, renal calculi, routine health check-up); (iii) willingness to provide written informed consent.

Exclusion criteria: (i) significant alcohol intake (≥30 g/day in men, ≥20 g/day in women); (ii) other identifiable causes of hepatic steatosis or chronic liver disease (e.g. viral hepatitis, autoimmune liver disease, drug-induced liver injury, Wilson's disease); (iii) pre-existing diagnosed cardiovascular disease or prior cardiovascular event, where this would preclude meaningful use of a 10-year primary-risk estimate; (iv) incomplete clinical, biochemical or ultrasonographic data required for grading or risk-score calculation.

Ultrasonographic Diagnosis and Grading of Fatty Liver: Fatty liver was diagnosed and graded on abdominal ultrasonography using standard qualitative sonographic criteria based on hepatic echogenicity relative to the renal cortex and the clarity of intrahepatic vascular and diaphragmatic margins **[16]**. Hepatic steatosis was graded ultrasonographically according to the degree of increased hepatic echogenicity and the visibility of intrahepatic anatomical structures. Grade 1 (mild) steatosis was defined by a mild, diffuse increase in hepatic echogenicity, with preserved visualisation of the diaphragm and intrahepatic vessel borders. Grade 2 (moderate) steatosis was characterised by a moderate, diffuse increase in hepatic echogenicity accompanied by slight impairment in the visualisation of the intrahepatic vessels and diaphragm. Grade 3 (severe) steatosis was defined by a marked increase in hepatic echogenicity with poor or complete absence of visualisation of the intrahepatic vessel borders, diaphragm, and posterior aspect of the right hepatic lobe. All ultrasonographic examinations were performed by qualified radiologists in the institutional radiology department.

Biochemical Estimation of GGT: Venous blood samples were drawn from all patients after an overnight fast, using a standardised phlebotomy protocol, and serum GGT was estimated in the institutional biochemistry laboratory by a standard kinetic photometric assay. A cut-off value of 45 IU/L, corresponding to the upper limit of the institutional laboratory's normal reference range for adults, was used to classify patients as having normal (≤45 IU/L) or elevated (>45 IU/L) GGT.

Framingham Cardiovascular Risk Estimation: Ten-year cardiovascular risk was estimated for each patient using the Framingham General Cardiovascular Disease Risk Score (D'Agostino RB Sr, et al., Circulation 2008) **[20]**, a validated, sex-specific, point-based algorithm incorporating age, total cholesterol, HDL cholesterol, systolic blood pressure (scored separately according to whether the patient was on antihypertensive treatment) and current smoking status. For each patient, points were assigned for each variable according to the published sex-specific point tables, summed, and converted into a 10-year probability (%) of a first cardiovascular event (coronary heart disease, cerebrovascular disease, peripheral artery disease or heart failure) using the corresponding published risk tables. Based on this calculated probability, patients were classified into three conventional risk strata: low risk (10-year risk <10%), moderate risk (10-year risk 10–20%) and high risk (10-

year risk >20%). As the moderate- and high-risk strata individually contained small numbers of patients, these two categories were combined into a single 'moderate-to-high risk' group (10-year risk ≥10%) for statistical comparison against the low-risk group (10-year risk <10%). It is emphasised that this score represents a calculated estimate of future cardiovascular risk based on validated population algorithms, and not an observed or measured cardiovascular outcome in this cross-sectional cohort.

Ethical considerations: The study was conducted after obtaining approval from the Institutional Ethical Committee (IEC), Stanley Medical College, Chennai, granted at the IEC meeting held on 21.04.2017. Written informed consent was obtained from all participants, and patient anonymity and confidentiality were maintained throughout the study.

Statistical Analysis

Data were entered and analysed using SPSS version 20. Categorical variables were expressed as frequencies and percentages, and continuous variables as mean ± standard deviation. The association between GGT category (≤45 IU/L vs >45 IU/L) and Framingham risk category (low risk, <10% 10-year risk, vs moderate-to-high risk, ≥10% 10-year risk) was assessed using the chi-square test (uncorrected, Yates-corrected and Mantel-Haenszel), with Fisher's exact test used to confirm significance for the primary 2×2 comparison. Odds ratio (OR), relative risk (RR) and risk difference (RD), each with 95% confidence intervals (calculated using the standard log-transformation method), were computed to quantify the strength of this association, with moderate-to-high risk as the outcome of interest and elevated GGT as the exposure of interest. Associations between grade of hepatic steatosis (Grade 1/2/3) and smoking status, and between grade of hepatic steatosis and Framingham risk category, were each assessed using the chi-square test for 2×3 contingency tables (2 degrees of freedom). No multivariable or adjusted analysis was performed; all reported associations are unadjusted (univariate). A two-tailed p value of <0.05 was considered statistically significant.

RESULT

A total of 100 patients with ultrasonographic evidence of fatty liver were included in the study.

Age and Sex Distribution

The mean age of the study population was 45.5 ± 10.4 years (range 25–69 years). The largest proportion of patients (37%) belonged to the 31–40-year age group, followed by 29% in the 51–60-year group (Table 1). Of the 100 patients, 69% were men (mean age 44.4 ± 9.4 years) and 31% were women (mean age 47.7 ± 12.4 years).

Table 1: Age distribution of study population (n=100)

Age group (years)	Frequency	Percentage
21–30	2	2.0%
31–40	37	37.0%
41–50	24	24.0%
51–60	29	29.0%
61–70	8	8.0%
Total	100	100.0%

Serum GGT Levels

The mean serum GGT level in the study population was 58.7 ± 16.3 IU/L. GGT was elevated (>45 IU/L) in 73% of patients, while 27% had GGT within the normal range (Table 2). Mean GGT was marginally higher in men (59.6 IU/L) than in women (56.9 IU/L).

Table 2: Distribution of serum GGT levels (n=100)

GGT category	Frequency	Percentage
≤45 IU/L (normal)	27	27.0%
>45 IU/L (elevated)	73	73.0%
Total	100	100.0%

Smoking Status and Grade of Fatty Liver

Fifty-three percent of patients reported a history of smoking (53/100); smoking was markedly more common among men (51/69, 73.9%) than among women (2/31, 6.5%). Smoking status did not differ significantly by grade of hepatic steatosis (Grade 1: 21/38, Grade 2: 23/44, Grade 3: 9/18 were smokers; $\chi^2 = 0.15$, df = 2, $p = 0.93$) (Table 3).

Table 3: Smoking status by grade of fatty liver

Smoking	Grade 1	Grade 2	Grade 3	Total
No	17	21	9	47
Yes	21	23	9	53
Total	38	44	18	100

Framingham Risk Score Distribution

Based on the Framingham General Cardiovascular Disease Risk Score, 71% of patients were categorised as moderate-to-high risk (10-year risk ≥10%) and 29% as low risk (10-year risk <10%) (Table 4). Moderate-to-high risk was more frequent among men (56/69, 81.2%) than women (15/31, 48.4%), and increased with advancing age.

Table 4: Distribution of Framingham risk category (n=100)

Framingham risk category	Frequency	Percentage
Low risk (<10%)	29	29.0%
Moderate-to-high risk (≥10%)	71	71.0%
Total	100	100.0%

Association of GGT Levels with Framingham Risk Score

Among patients with normal GGT (≤45 IU/L), 18/27 (66.7%) were in the low-risk category and 9/27 (33.3%) in the moderate-to-high-risk category. Among patients with elevated GGT (>45 IU/L), 11/73 (15.1%) were low risk while 62/73 (84.9%) were moderate-to-high risk (Table 5). This association was statistically significant (χ^2 uncorrected = 25.49, $p < 0.0001$; χ^2 Yates-corrected = 23.04, $p < 0.0001$; Fisher's exact $p < 0.0001$).

Table 5: Association of GGT level with Framingham risk category

GGT category	Low risk	Moderate-to-high risk	Total
≤45 IU/L	18	9	27
>45 IU/L	11	62	73
Total	29	71	100

Table 6: Measures of association between elevated GGT and moderate-to-high Framingham risk

Statistic	Value	95% Confidence Interval
Odds ratio	11.27	4.04 – 31.43
Relative risk	2.55	1.48 – 4.38
Risk difference (%)	51.60	32.01 – 71.18
Chi-square (uncorrected)	25.49	$p < 0.0001$
Chi-square (Yates-corrected)	23.04	$p < 0.0001$
Fisher's exact test	–	$p < 0.0001$

Association of GGT Levels and Framingham Risk Category with Grade of Fatty Liver

Elevated GGT (>45 IU/L) was observed across all grades of hepatic steatosis — 26/38 (68.4%) in Grade 1, 32/44 (72.7%) in Grade 2 and 15/18 (83.3%) in Grade 3 — with no statistically significant association between GGT category and grade of fatty liver ($\chi^2 = 1.38$, df = 2, p = 0.50) (Table 7). Similarly, Framingham risk category did not differ significantly by grade of fatty liver ($\chi^2 = 0.86$, df = 2, p = 0.65) (Table 8).

Table 7: Distribution of GGT category across grades of fatty liver ($\chi^2 = 1.38$, df = 2, p = 0.50)

GGT category	Grade 1	Grade 2	Grade 3	Total
≤45 IU/L	12	12	3	27
>45 IU/L	26	32	15	73
Total	38	44	18	100

Table 8: Distribution of Framingham risk category across grades of fatty liver ($\chi^2 = 0.86$, df = 2, p = 0.65)

Framingham risk category	Grade 1	Grade 2	Grade 3	Total
Low risk	13	11	5	29
Moderate-to-high risk	25	33	13	71
Total	38	44	18	100

DISCUSSION

This cross-sectional study evaluated 100 patients with ultrasonographically confirmed fatty liver to examine the association between serum GGT levels and 10-year cardiovascular risk estimated by the Framingham General Cardiovascular Disease Risk Score.

The mean age of the study population (45.5 years) and the male predominance (69%) are consistent with previously reported NAFLD cohorts, in which middle-aged men are disproportionately represented, likely reflecting patterns of metabolic risk factor clustering and health-seeking behaviour rather than a true sex-specific predisposition to NAFLD [10,11].

Serum GGT was elevated in 73% of patients, with a mean value of 58.7 IU/L, closely comparable between men and women. This finding aligns with previous work showing that GGT is frequently deranged in NAFLD independent of overt alcohol use, and that its elevation reflects oxidative stress and altered glutathione metabolism rather than cholestasis or hepatocellular injury alone [12,13]. Importantly, elevated GGT showed a statistically significant association with moderate-to-high Framingham cardiovascular risk (OR 11.27, 95% CI 4.04–31.43; RR 2.55, 95% CI 1.48–4.38), consistent with the hypothesis that GGT elevation in NAFLD reflects broader cardiometabolic risk rather than being an isolated hepatic finding [14,15]. This is in keeping with large population-based cohorts, such as the Vorarlberg Health Monitoring and Promotion Programme and the Framingham Offspring cohort, which have linked elevated GGT with cardiovascular morbidity and mortality even after adjustment for traditional risk factors [7,8]. As this was a univariate, unadjusted analysis in a cross-sectional design, this association cannot be interpreted as evidence that GGT independently predicts cardiovascular risk; multivariable analysis adjusting for the components already used to derive the Framingham score, and prospective follow-up for actual cardiovascular events, would be required to support any claim of independent predictive value.

In contrast, neither the grade of hepatic steatosis on ultrasonography nor smoking status showed a statistically significant association with GGT category or Framingham risk category in this cohort (Tables 3, 7 and 8). This suggests that the severity of steatosis as assessed radiologically may not, by itself, reliably reflect the degree of biochemical or cardiometabolic derangement captured by

GGT and the Framingham score, and that these measures may provide complementary rather than redundant information **[16]**.

These findings are consistent with a growing body of evidence associating elevated GGT in patients with NAFLD with a higher burden of calculated cardiovascular risk, independent of the severity of liver steatosis alone **[17,18]**. However, caution is warranted in extrapolating cross-sectional, score-based associations to statements of prediction; systematic evaluations of risk-marker studies have repeatedly cautioned against overstating incremental predictive value without appropriate prospective validation and reclassification analysis **[19]**.

Limitations

This study has several limitations that should be considered when interpreting these findings. First, the sample size (n=100) was determined pragmatically based on patients presenting during a fixed six-month period at a single centre, without a prior formal sample-size calculation, and the study was not powered a priori for any specific effect size; this may limit precision, as reflected in the wide confidence intervals around some estimates. Second, this was a single-centre, cross-sectional study, which limits generalisability to other populations and precludes assessment of temporal or causal relationships. Third, no multivariable or adjusted analysis was performed; the reported associations between GGT and Framingham risk category are unadjusted and may be confounded by shared underlying determinants (e.g. age, adiposity, glycaemic status) that were not modelled jointly. Fourth, the Framingham score used here is itself a calculated 10-year risk estimate derived from validated population algorithms, not an observed clinical cardiovascular outcome; this study did not include prospective follow-up for actual cardiovascular events, and therefore cannot demonstrate that GGT predicts future cardiovascular events, only that it is associated with a higher calculated risk category at a single time point. Future prospective, multivariable, and ideally multicentric studies with actual cardiovascular event follow-up are needed to determine whether GGT provides independent predictive or incremental value over the Framingham score.

CONCLUSION

In this cross-sectional study of patients with ultrasonographically confirmed NAFLD, serum GGT was significantly associated with a higher Framingham-estimated 10-year cardiovascular risk category, independent of the radiological grade of hepatic steatosis. As GGT is an inexpensive, widely available component of routine liver function testing, it may serve as a practical adjunct marker to flag patients with NAFLD who may benefit from closer cardiovascular risk-factor assessment. Given the cross-sectional, single-centre design and the absence of multivariable adjustment and prospective cardiovascular outcome data, these findings should be interpreted as demonstrating an association rather than independent prediction of future cardiovascular events. Larger, multicentric, prospective studies with multivariable adjustment and actual cardiovascular outcome follow-up are warranted to validate these findings and to define the incremental value, if any, of GGT-based risk stratification in NAFLD.

Declaration by Authors

Ethical Approval: Approved

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Conflict of Interest: The authors declare no conflict of interest.

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