

Association of Serum Gamma-Glutamyl Transferase with Type 2 Diabetes Mellitus and Impaired Glucose Tolerance

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ABSTRACT

Background: Gamma-glutamyl transferase (GGT), known to be a marker of hepatic injury in the past, has now been linked to insulin resistance and dysglycemia. High levels of GGT can be an indicator of oxidative stress and metabolic disorder. The objective of this study was to establish the association between serum GGT and IGT/T2DM and evaluate its diagnostic potential.

Methods: The study was a hospital-based comparative cross-sectional study consisted of 80 subjects having IGT and T2DM. Clinical evaluation and biochemical analysis consisting of FBS, PPBS, HbA1c, lipid profile, liver enzyme, and GGT were conducted. GGT was calculated by the use of the IFCC technique. Statistical analyses like ANOVA, ROC analysis, and logistic regression were conducted in SPSS software version 27; $p < 0.05$ was considered significant.

Results: T2DM exhibited significantly increased GGT compared to IGT (46.4 ± 22.3 vs 18.8 ± 5.2 ; $p < 0.001$) with elevated FBS, PPBS, HbA1c, BMI, uric acid, urea, triglycerides, VLDL, and hsCRP with decreased HDL. GGT was related to T2DM (OR=1.48; 95% CI: 1.12-1.95; $p = 0.006$) and IGT (OR=1.58; 95% CI: 1.13-2.21; $p = 0.008$). A cut-off point of GGT > 24.3 indicated 94% sensitivity and 93% specificity for T2DM.

Conclusion: The study concluded that the high GGT values was associated with dysglycaemia and indicated the utility of diagnostic performance, contributing the efficacy as a metabolic risk marker.

Key-words: Gamma-glutamyl transferase; Type 2 diabetes mellitus; Impaired glucose tolerance; Dysglycaemia; Insulin resistance

INTRODUCTION

Type 2 DM (T2DM) develops as gradual loss of insulin sensitivity and pancreatic β -cell function, with impaired glucose tolerance (IGT) being one of the important intermediate dysglycaemic states.

The current diagnostic criteria for IGT are blood glucose level of 140–199 mg/dL after 2 hours of a 75-g oral glucose tolerance test, but blood glucose level ≥ 200 mg/dL after 2 hours of an OGTT, fasting plasma glucose ≥ 126 mg/dL, or HbA1c $\geq 6.5\%$ is indicative of T2DM with confirmation in the absence of unequivocal hyperglycaemia [1]. Hepatic insulin resistance, visceral adiposity, dyslipidaemia, chronic low-grade inflammation, oxidative stress and progressive β -cell dysfunction are all hallmarks of the transition from normoglycaemia to IGT to overt T2DM. The identification of cheap biochemical markers related to this metabolic

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trajectory could thus be useful for early risk stratification [1,2].

Gamma-glutamyl transferase (GGT) is an enzyme located in the plasma membrane which plays a role in the extracellular metabolism of glutathione and transport of amino acids. It is classically considered as a hepatobiliary injury or indicator of alcohol use, but epidemiological data have shown that a higher GGT level is also strongly linked with insulin resistance, metabolic dysfunction-associated steatotic liver disease, dyslipidaemia and subsequent diabetes [3–6]. GGT is involved in glutathione catabolism and could be associated with changes in redox status and not only with hepatic enzyme leakage; thus, elevated serum concentrations of GGT have been suggested as an indirect biochemical marker of oxidative stress [7,8]. Increased GGT could be biologically linked to dysglycaemia via oxidative stress, leading to impaired insulin receptor substrate signalling, increased serine phosphorylation of insulin-signalling proteins and decreased GLUT-4-mediated glucose uptake, which also affects pancreatic β -cell function [8,9].

Prospective studies have shown a dosage-dependent association between GGT and the occurrence of diabetes. In the Japanese population ($n = 15,464$), 373 developed diabetes over the follow-up period; those in the highest quintile of GGT had an adjusted HR of 1.83 (95% CI, 1.06–3.15) compared to the lowest quintile. Importantly, there was a nonlinear association with greater risk of diabetes with each 1 IU/L increase in GGT below around 24 IU/L [4]. Likewise, a 6-year follow-up study of Chinese patients found relative risks of impaired fasting glucose of 1.70, 1.55 and 2.46 for increasingly elevated GGT levels, lending further support to the connection between elevated GGT and prediabetic dysglycaemia [10].

Clinically relevant evidence relevant to individuals with established IGT is also relevant. 537 Asian Indians with IGT had a two-year follow-up of which 123 developed diabetes; baseline GGT was significantly elevated in those who developed diabetes. In those who returned to normal glucose tolerance, GGT was found to decrease, while among the ones who were advancing to diabetes, GGT was found to increase [11]. These observations indicate that GGT could be a marker of metabolic deterioration prior to the biochemical threshold of the onset of diabetes. Other factors such as obesity, alcohol intake, hepatic steatosis, hypertriglyceridaemia and

smoking, however, could influence the association observed by these studies, and Mendelian-randomization studies have not always been able to demonstrate a direct causal effect of genetically determined GGT on T2DM [3,6]. Thus, serum GGT should be interpreted as a metabolic risk marker in the context of glycaemic, hepatic and cardiometabolic parameters and not as a standalone diagnostic tool.

MATERIALS AND METHODS

Study Design- The study was a hospital-based comparative cross-sectional study to analyze the association of the serum Gamma-Glutamyl Transferase with T2DM and impaired glucose tolerance. The study was conducted among 80 patients diagnosed with T2DM. The study was conducted in the Tertiary Care Hospital. Informed consent required for the study. The ethical approval for the study was obtained from the Institutional Ethics Committee. Previous history of all patients, general and systemic examination and the biochemical evaluation was performed, which consists of fasting blood glucose, postprandial blood glucose, HbA1c, lipid profile, liver enzymes, and serum GGT. Serum GGT was estimated by the use of the IFCC method. Patient's selection was done on the basis of specific inclusion and exclusion criteria.

Inclusion criteria

- Patients with T2DM was included in the study
- Patients those who were diagnosed with glucose tolerance
- Informed consent required for the study

Exclusion criteria

- Patients with alcohol consumer, smokers or patients with diabetic related complication
- Patients with chronic liver and renal failure were excluded
- Patients with systemic diseases or conditions which will impact the biochemical parameters of the study

Procedure- All patient's ethical approval was obtained from Institutional Ethics Committee and patients were recruited with informed written consent. The inclusion criteria for the respective study groups included the presence of WHO diagnostic criteria for type 2 diabetes mellitus and impaired glucose tolerance.

Information about their history was collected along with relevant demographic and clinical data followed by physical and systemic examination. After the night's fasting, 5 mL venous blood was taken from all participants with proper aseptic precautions. The sample collected was used for biochemical investigations which included fasting blood sugar, postprandial blood sugar, HbA1c, lipid profile and liver enzymes. The estimation of GGT in the serum was done by the IFCC method with Transasia kit. The reference range of serum GGT is 7-50 IU/L.

Statistical analysis- Statistical analysis was conducted by the use of SPSS version 27. One-way ANOVA with Dunnett's post-hoc test was utilized for comparative analysis among groups. ROC curve analysis was conducted for evaluation of the diagnostic accuracy and optimum cut off values, of the serum GGT. Logistic regression analysis was performed for assessing the association of enhanced GGT and GGT along with BMI

with T2DM and IGT. The p-value <0.05 was maintained from statistically significant.

RESULTS

The table 1 showed the significant differences for several metabolic, renal, lipid and inflammatory parameters for the IGT and T2DM groups. The FBS enhanced from 87.6±12.2 to 117.2±5.2 mg/dL, and also the PPBS also enhanced from 112.4±14.4 to 176.2±18.6 mg/dL (both p<0.001). The HbA1c increased from 5.3±0.6% to 6.3±0.9% (p<0.001) and the uric acid and urea is high in case of second group. The HDL reduced from 50.5±5.1 to 36.6±4.1 mg/dL (p<0.001), while the hsCRP enhanced from 1.8±1.03 to 5.3±6.2 mg/L (p<0.001). Significant difference was noted in case of BMI, GGT, triglycerides, VLDL and creatinine. The LDL, AST, ALT and age did not differ between the groups (Table 1).

Table 1: Comparison of Biochemical, Renal, Lipid, Hepatic and Inflammatory Parameters between IGT and Type 2 Diabetes Mellitus Groups

Parameter	IGT	Type 2 DM	F	p (ANOVA)	Significance
Age (years)	47.6 ± 10.2	42.2 ± 15.1	1.2	0.3	NS
BMI (kg/m ²)	21.1 ± 2.6	23.7 ± 5.6	9.3	<0.001	
FBS (mg/dL)	87.6 ± 12.2	117.2 ± 5.2	93.5	<0.001	
PPBS (mg/dL)	112.4 ± 14.4	176.2 ± 18.6	80.8	<0.001	
GGT (mg/dL)	18.8 ± 5.2	46.4 ± 22.3	27.3	<0.001	
HbA1c (%)	5.3 ± 0.6	6.3 ± 0.9	39.1	<0.001	
Uric acid (mg/dL)	3.6 ± 0.9	6.7 ± 0.7	76.5	<0.001	
Urea (mg/dL)	20.9 ± 6.5	31.7 ± 7.4	16.7	<0.001	
Creatinine (mg/dL)	0.9 ± 0.1	0.9 ± 0.1	12.4	<0.001	
TGL (mg/dL)	116.8 ± 38.4	122.6 ± 17.2	6.6	0.003	
TC (mg/dL)	188.7 ± 31.6	164.9 ± 5.8	3.2	0.046	NS
HDL (mg/dL)	50.5 ± 5.1	36.6 ± 4.1	98.7	<0.001	
VLDL (mg/dL)	23.2 ± 7.6	24.5 ± 3.4	6.5	0.003	NS
LDL (mg/dL)	115.0 ± 26.4	103.7 ± 6.7	1.8	0.17	
AST (mg/dL)	17.7 ± 6.6	20.8 ± 4.6	1.35	0.26	NS
ALT (mg/dL)	15.1 ± 4.2	17.1 ± 3.6	2.7	0.07	NS
hsCRP (mg/L)	1.8 ± 1.03	5.3 ± 6.2	12.2	0.001	

Table 2 demonstrated the logistic regression analysis, the positive relationship between the GGT levels and both Type 2 diabetes mellitus (T2DM) and showed impaired glucose tolerance (IGT). In case of Model 1 for T2DM, odds ratio (OR) for GGT is 1.41 (95% CI: 1.17–1.68; P=0.001), which indicated the increased odds of T2DM with high levels of GGT. The BMI is included in Model 2

showed significant association, with CGT noted with OR of 1.48 (95% CI: 1.12–1.95; P=0.006), and OR of BMI is 1.87 (95% CI: 1.08–3.26; P=0.025). GGT was also associated with outcome (OR=1.58, 95% CI: 1.13–2.21; P=0.008). The table indicated that high level of GGT was related with abnormal glucose metabolism (Table 2).

Table 2: Logistic Regression Analysis of the Association of GGT with Type 2 Diabetes Mellitus and Impaired Glucose Tolerance

Outcome	Model	Variable	Odds Ratio	95% CI	p-value
Type 2 Diabetes Mellitus	Model 1	GGT	1.41	1.17–1.68	0.001
	Model 2	GGT	1.48	1.12–1.95	0.006
		BMI	1.87	1.08–3.26	0.025
Impaired Glucose Tolerance	Model 1	GGT	1.58	1.13–2.21	0.008

Table 3 demonstrated sensitivity and specificity showed different variations according to the selected cut-off points for GGT. At a cut-off point of ≥ 10.8 , sensitivity was 99.0%, whereas specificity was 0%, implying that there was high sensitivity without any specificity. At a cut-off point of >18.0 , sensitivity was high at 94.0%, but specificity was 41.5%. Improved balance between

sensitivity and specificity at a cut-off point of >24.3 , where sensitivity was 94.0%, specificity was 93.0%, and positive likelihood ratio (+LR) and negative likelihood ratio (–LR) were 13.43 and 0.06, respectively. At a cut-off point of >29.7 , sensitivity was 79.5%, while specificity was still 93.0%. At a cut-off point of >31.0 , specificity was 99.0%, while sensitivity was 79.5% (Table 3).

Table 3: Diagnostic Performance of GGT at Different Cut-off Values

Criterion (GGT)	Sensitivity (%)	95% CI	Specificity (%)	95% CI	+LR	–LR
≥ 10.8	99	81.5–100.0	0	0.0–10.5	1	—
>16.7	99	81.5–100.0	41.5	24.5–60.2	1.69	0.02
>18.0	94	72.8–99.7	41.5	24.5–60.2	1.61	0.14
>24.3	94	72.8–99.7	93	78.4–98.7	13.43	0.06
>29.7	79.5	55.0–94.3	93	78.4–98.7	11.36	0.22
>31.0	79.5	55.0–94.3	99	88.7–100.0	—	0.21
>84.2	0	0.0–17.2	99	88.7–100.0	—	1.01

Table 4 showed the increasing change in the sensitivity and specificity of the GGT with respect to the evaluated cut-off values. In ≥ 10.8 , sensitivity of the GGT was 99.0%, but specificity was 0%, implying that this value was able to detect most of the cases but had no discriminating ability. For the cut-off of >24.3 , sensitivity was 99.0%, whereas specificity increased significantly to 92.8% with

a positive likelihood ratio of 13.75 and negative likelihood ratio of 0.01. In >29.4 , sensitivity was low 76.0%, but specificity was very high 92.8%, with +LR of 10.71 and -LR of 0.26. The cut-off of >31.0 maintained sensitivity at 76.0% but specificity increased to 99.0%. For the cut-off >107.1 , sensitivity was 0%, and specificity was 99.0% (Table 4).

Table 4: Diagnostic Performance of GGT at Different Cut-off Values for Identification of Impaired Glucose Tolerance

Criterion	Sensitivity (%)	95% CI	Specificity (%)	95% CI	+LR	-LR
≥10.8	99	72.8–100.0	0	0.0–10.5	1	-
>24.3	99	72.8–100.0	92.8	78.0–98.7	13.75	0.01
>29.4	76	43.9–94.6	92.8	78.0–98.7	10.71	0.26
>31.0	76	43.9–94.6	99	88.5–100.0	-	0.24
>107.1	0	0.0–26.1	99	88.5–100.0	-	1.01

DISCUSSION

The relationship of elevated serum GGT level with abnormal glucose metabolism is biologically and epidemiologically compatible with the notion that impaired hepatic metabolism is present early in the trajectory of IR to T2DM. GGT is especially useful because an increase in its levels may be seen in the presence of metabolic abnormalities before the onset of diabetes. For the Japanese NAGALA cohort of 15,464 adults who were not diabetic at baseline, 373 incident diabetes cases were found over a maximum of 13 years of follow-up. Even after adjusting for age, sex, smoking, alcohol consumption, BMI, blood pressure, triglycerides, fatty liver, ALT, AST and fasting plasma glucose, the highest GGT quintile was associated with an adjusted hazard ratio of 1.83 (95% CI, 1.06–3.15) compared with the lowest quintile [4]. It is especially noteworthy that the nonlinear pattern suggests that the metabolic risk might be elevated despite relatively low GGT levels, a pattern that would signal a metabolic risk that is not solely linked to a more advanced, or alarming, stage of conventional laboratory abnormalities.

There is also evidence from people who are prediabetic to support the present association. During two years of follow-up, 123 of the 537 men with IGT in the prospective study developed diabetes. There was significant difference in baseline GGT levels between the group that developed diabetes and that who reverted to NGT, and the group who developed diabetes had higher GGT levels than those who reverted to NGT [11]. This longitudinal change increases the likelihood that GGT is linked to the metabolic transition process itself, and not just to a diabetes state. The discovery is of particular significance for Asian Indians, in whom insulin resistance and dysglycaemia could occur at relatively low levels of adiposity; thus, the potential importance of low-cost biochemical markers of early metabolic dysfunction becomes more likely.

There is a substantial amount of potential evidence to back up the direction of this relationship. An updated dose-response meta-analysis, which included 24 cohort studies and 11,155 incident T2DM cases, showed that the pooled relative risk for the highest third versus the lowest third of baseline GGT was 1.34 (95% CI, 1.27–1.42) [3]. Previous pooled analyses also confirmed the relationship between GGT and several traditional liver enzymes, and indicated that this relationship was stronger for GGT than for other liver enzymes after multivariable adjustment [12]. Further research in recent years still shows the nonlinear relationships. A real-world study of 3,048 adults in 2022 found a graded relationship between higher categories of GGT and increased odds for T2DM, with odds reaching a maximum of 2.29 at GGT ≥74 IU/L compared to GGT ≤13 IU/L, but with the association leveling off around 34 IU/L [6]. A separate real-world analysis of hepatic enzymes and T2DM using local clinical data, UK Biobank data and NHANES data was conducted in 2024 to further investigate the dose-response relations between hepatic enzymes and T2DM, further highlighting the population relevance of liver-derived biochemical markers [13].

There are several ways this correlation can be explained. GGT is involved in the metabolism of glutathione, which is the principal antioxidant system in the extracellular space. It is therefore not surprising that blood GGT levels be elevated as a sign of increased glutathione turnover in the presence of oxidative stress. Stress-sensitive kinases such as c-Jun N-terminal kinase, p38 MAPK and IκB kinase are activated by oxidative stress and result in abnormal phosphorylation of insulin receptor substrates and impaired downstream PI3K–AKT signaling [8,9]. Peripheral glucose disposal is then impaired due to the decreased insulin-mediated GLUT-4 translocation. At the same time, oxidative and inflammatory stress can affect the function of mitochondria and the viability of pancreatic β-cells, thus affecting the compensatory

insulin secretion. Therefore, high GGT levels may reflect a metabolic profile of the liver that includes insulin resistance, oxidative stress and lipotoxicity, or hepatic steatosis, and not be a direct contributor to diabetes.

Another significant explanation pathway is hepatic insulin resistance. Fasting glucose is regulated by the liver by glycogenolysis and gluconeogenesis, and hepatic lipid accumulation is closely related to impaired glucose production suppression by insulin. Therefore, the presence of hypertriglyceridaemia and metabolic dysfunction-associated steatotic liver disease are common causes of increased GGT. The association between GGT and incident diabetes was still found after adjustment for fatty liver and multiple metabolic covariates in the 2020 NAGALA analysis [4]. However, residual confounding cannot be ruled out due to alcohol consumption, medications, obesity, hepatobiliary disease and other metabolic exposures that affect GGT.

There may not be a causal relationship between GGT and dysglycaemia. In an observational study, the hazard ratios of incident prediabetes and T2DM per an increase of 1 standard deviation (SD) in GGT were 1.10 (95% confidence interval (CI): 1.02–1.19) and 1.19 (95% CI: 1.10–1.30), respectively, in a Mendelian-randomization investigation of 8,611 participants, while the genetic analyses did not provide convincing evidence that GGT itself causally produced these outcomes [5]. Likewise, the 2022 real world/Mendelian randomization study reported robust observational relationships with no evidence of a linear causal effect of genetically induced GGT on T2DM [6]. The results of this study support the potential use of GGT as a metabolic phenotype marker. Thus, a high GGT level alone should not be used to diagnose T2DM or IGT.

There is also recent evidence that GGT may have a stronger discriminatory ability when combined with other metabolic parameters than GGT alone. The GGT/HDL-cholesterol ratio has been shown to have a positive nonlinear relationship with incident diabetes, and an area under the ROC curve of ~0.75 for the prediction of T2DM has been obtained in a cohort analysis in 2022 [14]. More recently, composite indices combining GGT and triglycerides-glucose have been shown to have additional predictive potential, but these are still unvalidated in the clinical setting and are not yet routinely used in clinical practice [15]. These results support a multidimensional interpretation where GGT

provides liver dysfunction data, oxidative stress and insulin resistance data, and the others (glucose and lipid parameters) provide complementary data on glycaemic status.

In general, the evidence suggests that there is an association between serum GGT and impaired glucose regulation, progression to diabetes and incident T2DM. It is easy to obtain and inexpensive, and is often available, and therefore is attractive as an adjunctive metabolic risk marker in addition to fasting glucose, HbA1c, triglycerides, HDL cholesterol, BMI, and markers of hepatic steatosis. Although GGT should not be used in place of existing diagnostics like fasting plasma glucose, HbA1c or 75-g oral glucose tolerance test (OGTT) [1]. Further studies in South Asian populations with repeated measurements of GGT and repeated liver fat assessments and direct measures of insulin sensitivity are required to establish whether GGT can help to predict the transition from IGT to T2DM, beyond the well-established clinical risk factors.

CONCLUSIONS

The study concluded that significant correlation between increased levels of GGT and dysglucose metabolism among IGT and T2DM subjects. T2DM subjects have significantly increased GGT, glycaemic indices, uric acid, urea, triglycerides, VLDL, creatinine and hsCRP, as well as decreased HDL, thus indicated the presence of metabolic, renal and inflammatory changes. Logistic regression revealed significant correlation between GGT and T2DM and IGT regardless of adjustment for BMI. Diagnostic evaluation showed that cut-off values of GGT had an effect on sensitivity and specificity. Threshold of >24.3 was highly sensitive and specific in diagnosis of T2DM, and further increase in cut-off value led to an increase in specificity and decrease in sensitivity. The same results were obtained for IGT subjects. GGT used as a biochemical marker that is associated with dysglycaemia and metabolic disorders. Further research with bigger samples of subjects is needed to confirm this hypothesis and define clinically significant cut-off values.

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