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Association Between Serum Gamma-Glutamyl Transferase and Glycemic Control in Newly Diagnosed Type 2 Diabetes Mellitus: A Cross-Sectional Study.

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Abstract

Background: Type 2 Diabetes Mellitus (T2DM) poses a growing global burden, with mounting evidence linking liver dysfunction to worsening blood glucose regulation. Gamma-glutamyl transferase (GGT), an enzyme central to cellular antioxidant defense via the glutathione pathway, is gaining attention as a potential indicator of early metabolic derangement. ***Objective:*** To evaluate how serum GGT levels relate to glycemic control (HbA1c) in patients newly diagnosed with T2DM, and to assess its relationship with lipid levels and other metabolic markers. ***Materials and Methods:*** This prospective cross-sectional study included 50 newly diagnosed, drug-untreated T2DM patients evaluated at a tertiary care facility between November 2024 and June 2026. Blood samples were analyzed for serum GGT, HbA1c, fasting and postprandial blood glucose, and lipid profiles. Statistical analysis was performed using SPSS (version 29.0), employing Pearson correlation to test associations. ***Results:*** Serum GGT showed a highly significant positive correlation with HbA1c ($r = 0.884$, $p < 0.001$). The cohort had a mean GGT of 38.82 ± 14.03 U/L, with elevated levels (>40 U/L) present in 46% (n = 23) of subjects. Higher HbA1c also correlated strongly with increased total cholesterol ($r = 0.731$), triglycerides ($r = 0.843$), LDL-C ($r = 0.641$), and VLDL-C ($r = 0.844$), alongside an inverse correlation with HDL-C ($r = -0.337$, $p = 0.017$). Overall parameter differences were statistically significant ($p < 0.001$). ***Conclusions:*** Serum GGT levels closely track the severity of glycemic control and dyslipidemia in newly diagnosed T2DM. Measuring GGT alongside HbA1c provides a simple, accessible way to detect early hepatic involvement, underlying insulin resistance, and heightened cardiovascular risk.

Keywords: Type 2 Diabetes Mellitus, Gamma-Glutamyl Transferase, HbA1c, Glycemic Control, Metabolic Syndrome, Biomarker.

INTRODUCTION

Diabetes mellitus (DM) stands as one of the most critical public health threats of the modern era, defined by persistent hyperglycemia caused by defects in insulin secretion, insulin action, or both. The global burden of Type 2 Diabetes Mellitus (T2DM) has escalated dramatically over recent decades, climbing from an estimated 30 million individuals in 1985 to 463 million in 2019 [1]. Projections from the International Diabetes Federation (IDF) estimate that this burden will climb to 642 million by 2040.

India carries a remarkably heavy share of this global health crisis. With roughly 77 million adults affected, India accounts for the second-largest diabetic population in the world, trailing only China [1]. The 2019 National Diabetic Retinopathy Survey reported an overall national prevalence of 11.8%, with urban centers reporting even higher rates ranging between 10.9% and 14.2% [2]. This upward trend is closely linked to shifting demographics across the country, including rapid urbanization, increasingly sedentary habits, and rising rates of central obesity.

Although rates of both Type 1 and Type 2 diabetes are increasing globally, T2DM represents the vast majority of cases and is growing at a far faster pace [3]. Detecting metabolic derangements early is vital for delaying or preventing long-term microvascular and macrovascular damage. Because T2DM is driven by underlying systemic low-grade inflammation and cellular stress, identifying reliable circulating biomarkers has become a primary goal for early risk stratification [4].

Gamma-glutamyl transferase (GGT)—a membrane-bound enzyme found predominantly in liver tissue—has attracted significant interest as a potential marker for metabolic syndrome and cardiovascular disease [5]. Biochemically, GGT is

key to maintaining intracellular antioxidant reserves through its role in glutathione (GSH) breakdown and recycling [6]. While historically viewed strictly as an indicator of liver injury or heavy alcohol intake, emerging evidence links elevated serum GGT to insulin resistance, non-alcoholic fatty liver disease (NAFLD), metabolic syndrome, and higher rates of incident diabetes [7,8].

Although previous reports have observed elevated GGT concentrations in diabetic cohorts compared to healthy populations and noted links with atherogenic lipid profiles [9,10], its specific relationship with glycemic severity (HbA1c) in patients at the precise point of initial T2DM diagnosis remains less explored. To address this gap, this study investigated the correlation between serum GGT levels and glycemic control (HbA1c) in newly diagnosed, drug-untreated T2DM patients, while evaluating its potential value as an additional marker for cardiometabolic screening.

MATERIALS AND METHODS

Study Design and Setting:

This prospective, cross-sectional observational study was carried out at the Department of General Medicine, Chalmeda Anand Rao Institute of Medical Sciences, Karimnagar, Telangana, India, over an 18-month period from November 2024 to June 2026. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Protocol approval was granted by the Institutional Ethics Committee of Chalmeda Anand Rao Institute of Medical Sciences, Karimnagar, Telangana, India (Reference No.: CAIMS/IEC/PG/016/2025), and written informed consent was obtained from all participants prior to enrollment.

Study Population and Eligibility Criteria:

A cohort of 50 newly diagnosed, drug-untreated individuals with T2DM was selected according to the following eligibility criteria.

Inclusion Criteria:

1. Individuals newly diagnosed with T2DM (HbA1c ≥ 6.5% or fasting plasma glucose ≥ 126 mg/dL).
2. Adults aged between 35 and 75 years.
3. Patients who had not taken any antidiabetic drugs or insulin prior to the study.

Exclusion Criteria:

1. Individuals with a history of regular tobacco smoking or habitual alcohol intake.
2. Known cases of underlying liver, kidney, heart, or chronic lung disease.
3. Patients presenting with thyroid disorders.
4. Pregnant or lactating women.
5. Patients currently receiving anticoagulant therapy (specifically warfarin).
6. Individuals with hemolytic anemia or a past history of splenectomy.

Clinical and Biochemical Assessments

Each participant underwent a detailed medical evaluation, including clinical history taking and physical examination. Physical parameters such as height, weight, Body Mass Index (BMI), and baseline vital signs (blood pressure, pulse, and respiratory rate) were documented.

Fasting venous blood samples were collected after a minimum 8-hour overnight fast to run the following laboratory assays:

- Fasting Plasma Glucose (FPG): Analyzed after an 8-hour fast.
- Postprandial Plasma Glucose (PPBG): Evaluated 2 hours after a standardized meal.
- Glycated Hemoglobin (HbA1c): Quantified using High-Performance Liquid Chromatography (HPLC).
- Serum Gamma-Glutamyl Transferase (GGT): Measured via a standard colorimetric assay.
- Serum Lipid Panel: Total cholesterol, triglycerides, High-Density Lipoprotein Cholesterol (HDL-C), Low-Density Lipoprotein Cholesterol (LDL-C), and Very Low-Density Lipoprotein Cholesterol (VLDL-C).

All blood tests were performed following standardized clinical laboratory procedures under strict quality-control protocols.

Statistical Analysis:

Statistical computations were carried out using IBM SPSS version 29.0. Continuous data were summarized as mean ± standard deviation (SD), and categorical variables were described using counts and percentages.

The Shapiro-Wilk test was applied to check data normality. One-sample t-tests were used for normally distributed variables, whereas non-parametric quantitative variables were analyzed using the Mann-Whitney U test. Bivariate associations between serum GGT, HbA1c, and lipid fractions were measured using Pearson correlation coefficients (r). A two-tailed p-value < 0.05 was set as the threshold for statistical significance, with 95% confidence intervals reported where appropriate.

RESULTS

1. Demographic Profile

A total of 50 newly diagnosed, drug-untreated T2DM patients completed the study. The mean age of the participants was 51.94 ± 9.11 years (range: 35–75 years).

The age distribution showed a distinct peak in middle-aged adults: 34% (n = 17) were between 46 and 55 years, 30% (n = 15) were between 56 and 65 years, and 28% (n = 14) were aged 35 to 45 years. Only 8% (n = 4) belonged to the 66–75-year bracket. Men constituted four-fifths of the cohort (80%, n = 40), while women made up 20% (n = 10).

Characteristic	Subgroup	Count (n)	Percentage (%)
Age Group	35–45 years	14	28%
	46–55 years	17	34%
	56–65 years	15	30%
	66–75 years	4	8%
Sex	Male	40	80%
	Female	10	20%

2. Glycemic and Liver Enzyme Parameters

Marked hyperglycemia was present across the entire cohort at initial diagnosis.

Fasting Plasma Glucose (FPG): The mean FPG was 242.80 ± 74.78 mg/dL (range: 128–310 mg/dL). No patient had normal fasting glucose (<100 mg/dL). Overall, 16% (n = 8) presented with mild elevation (126–180 mg/dL), 40% (n = 20) had moderate elevation (181–250 mg/dL), and 44% (n = 22) showed severe fasting hyperglycemia (>250 mg/dL).

Postprandial Plasma Glucose (PPBG): Mean PPBG reached 346.04 ± 106.61 mg/dL (range: 220–400 mg/dL). All 50 subjects (100%) exceeded the 200 mg/dL threshold, confirming significant postprandial glucose intolerance.

Glycated Hemoglobin (HbA1c): The cohort exhibited a mean HbA1c of 8.10 ± 1.52% (range: 6.2%–10.2%). Ninety percent (n = 45) presented with poorly controlled diabetes (HbA1c > 6.4%), whereas only 10% (n = 5) had an HbA1c ≤ 6.4%.

Serum GGT: The mean serum GGT concentration was 38.82 ± 14.03 U/L (range: 8–80 U/L; $t = 19.565$, $df = 49$, $p < 0.001$). Normal GGT values (<40 U/L) were recorded in 54% (n = 27) of subjects, while 46% (n = 23) had elevated levels (>40 U/L).

3. Lipid Profile Analysis

Disturbances across the lipid panel were widely prevalent, most notably marked by widespread reductions in HDL-C and high rates of hypertriglyceridemia.

Parameter	Mean ± SD (mg/dL)	Classification Category	n	%
Total Cholesterol	210.28 ± 38.67	Near-optimal (150–200 mg/dL)	21	42%
		Borderline high (200–239 mg/dL)	18	36%
		High (≥240 mg/dL)	11	22%
Triglycerides	171.84 ± 42.77	Normal (<150 mg/dL)	17	34%
		Mildly elevated (150–199 mg/dL)	23	46%
		High (200–499 mg/dL)	10	20%
HDL-C	36.08 ± 2.28	Low (<40 mg/dL)	47	94%
		Borderline (40–45 mg/dL)	3	6%
		Optimal (>45 mg/dL)	0	0%
LDL-C	139.58 ± 34.13	Optimal (<100 mg/dL)	6	12%
		Near-optimal (100–129 mg/dL)	15	30%
		Borderline high (130–159 mg/dL)	15	30%
		High (160–189 mg/dL)	12	24%
		Very high (≥190 mg/dL)	2	4%
VLDL-C	34.62 ± 8.48	Normal (<30 mg/dL)	19	38%
		Elevated (>30 mg/dL)	31	62%

4. Correlation Testing

Primary Association: Serum GGT vs HbA1c

Pearson correlation testing revealed a robust, positive association between serum GGT levels and HbA1c ($r = 0.884$, $p < 0.001$). This confirms that higher liver enzyme activity closely tracks worsening long-term glycemic control.

Relationships with Lipid Fractions:
Both HbA1c and serum GGT demonstrated statistically significant correlations across all measured lipid parameters:

Metabolic Variable	Correlation with HbA1c (r)	p-value	Correlation with Serum GGT (r)	p-value
Total Cholesterol	0.731	<0.001	0.739	<0.001
Triglycerides	0.843	<0.001	0.710	<0.001
LDL-C	0.641	<0.001	0.683	<0.001
VLDL-C	0.844	<0.001	0.711	<0.001
HDL-C	-0.337	0.017	-0.323	0.022

DISCUSSION

The primary objective of this study was to evaluate the relationship between serum Gamma-Glutamyl Transferase (GGT) levels and glycemic control, as measured by glycated hemoglobin (HbA1c), in newly diagnosed, drug-untreated patients with Type 2 Diabetes Mellitus (T2DM). Our findings demonstrate a strong, statistically significant positive correlation between serum GGT activity and HbA1c ($r = 0.884$, $p < 0.001$). Furthermore, serum GGT exhibited significant positive correlations with all atherogenic lipid fractions—including total cholesterol, triglycerides, Low-Density Lipoprotein Cholesterol (LDL-C), and Very Low-Density Lipoprotein Cholesterol (VLDL-C)—while demonstrating an inverse association with High-Density Lipoprotein Cholesterol (HDL-C). Collectively, these observations highlight the clinical utility of serum GGT as an integrative, low-cost biomarker reflecting systemic oxidative stress, hepatic steatosis, and atherogenic dyslipidemia at the initial diagnosis of T2DM.

Mechanistic Links Between GGT and Glycemic Dysregulation

The robust association observed between serum GGT and HbA1c aligns with a growing body of pathophysiological evidence linking hepatic enzyme activity to glucose intolerance and metabolic dysfunction [11,12]. Three main biological mechanisms account for this relationship.

First, cellular oxidative stress plays a pivotal role. Serum GGT is an essential cell-membrane enzyme involved in the gamma-glutamyl cycle, which regulates the breakdown and resynthesis of intracellular glutathione (GSH)—the body's principal endogenous antioxidant [6]. In chronic hyperglycemia, excess glucose flux through oxidative pathways generates high levels of reactive oxygen species (ROS) [13]. In response to rising oxidative damage, GGT expression is adaptively up-regulated to facilitate extracellular GSH transport back into cells to preserve cellular redox balance [14]. Pancreatic beta-cells possess low intrinsic expression of protective antioxidant enzymes, such as superoxide dismutase, catalase, and glutathione peroxidase, rendering them particularly susceptible to ROS-mediated apoptosis and functional decline [15]. Sustained oxidative injury blunts beta-cell sensitivity and impairs insulin secretion, thereby perpetuating a vicious cycle of glycemic deterioration.

Second, elevated serum GGT serves as a surrogate marker for hepatic steatosis (non-alcoholic fatty liver disease, NAFLD), which is a key driver of insulin resistance [16,17]. Intrahepatic lipid accumulation directly disrupts the insulin signaling cascade in hepatocytes, impairing the liver's ability to suppress fasting gluconeogenesis and glycogenolysis [18]. Consequently, hepatic fat accumulation increases basal hepatic glucose output, contributing to elevated Fasting Plasma Glucose (FPG) levels. Furthermore, steatotic hepatocytes secrete pro-inflammatory hepatokines and adipokines that worsen peripheral insulin resistance in skeletal muscle and adipose tissue [19].

Third, serum GGT elevation reflects subclinical low-grade systemic inflammation, as evidenced by its established correlation with high-sensitivity C-reactive protein (hs-CRP) in metabolic cohorts [20]. Chronic low-grade inflammation disrupts normal insulin receptor substrate (IRS) phosphorylation, providing a direct molecular bridge between hepatic enzyme elevation and systemic glycemic dyscontrol [21].

GGT and Atherogenic Dyslipidemia:

The significant correlations observed between GGT activity and total cholesterol ($r = 0.739$, $p < 0.001$), triglycerides ($r = 0.710$, $p < 0.001$), LDL-C ($r = 0.683$, $p < 0.001$), VLDL-C ($r = 0.711$, $p < 0.001$), and HDL-C ($r = -0.323$, $p = 0.022$) confirm that hepatic enzyme elevation occurs in tandem with generalized lipid dysregulation [22,23].

The characteristic dyslipidemic triad—elevated triglycerides, elevated VLDL-C, and profoundly suppressed HDL-C (present in 94% of our cohort)—is a hallmark of insulin-resistant states [24]. The near-perfect positive correlation observed between triglycerides and VLDL-C ($r = 0.999$, $p < 0.001$) reflects both mathematical consistency and the biological reality of hepatic lipoprotein kinetics. Specifically, concurrent elevations in GGT and VLDL-C point to heightened hepatic secretion of apolipoprotein B-containing, triglyceride-rich lipoproteins driven by hepatic insulin resistance [25]. Because this lipid pattern is strongly linked to atherosclerotic cardiovascular disease (ASCVD), measuring serum GGT alongside standard lipid panels may enhance cardiovascular risk stratification in newly diagnosed diabetic individuals [26].

Comparison with Previous Studies:
The results of this study both corroborate and build upon previous epidemiological and clinical investigations into liver enzymes and diabetes.

Study (Author, Year)	Design & Cohort	Primary Finding	Comparison with Present Study
Present Study (2026)	N = 50, newly diagnosed, drug-untreated T2DM	GGT vs. HbA1c: r = 0.884 ($p < 0.001$); GGT vs. Triglycerides: r = 0.710 ($p < 0.001$)	Demonstrates a stronger correlation (r = 0.884) by strictly isolating unmedicated, drug-untreated subjects.
Gohel & Chacko (2013) [20]	N = 150, T2DM with good vs. poor control vs. controls	GGT vs. HbA1c: positive correlation ($p < 0.001$)	Corroborates our findings that serum GGT activity increases alongside worsening glycemic control and oxidative stress.
Rajarajeswari & Jayaseelan (2014) [9]	Cross-sectional T2DM cohort	GGT vs. Triglycerides: r = 0.112; GGT vs. Total Cholesterol: r = 0.027	Aligns with our observed lipid trends, though our cohort showed stronger correlations (r = 0.710), reflecting higher baseline insulin resistance.
Al-Jameil et al. (2014) [10]	N = 157, established T2DM vs. non-diabetic controls	GGT vs. HbA1c & lipids: positive correlation with FPG, PPBG, TC, TG, and LDL-C ($p < 0.05$)	Validates the global consistency of concurrent liver enzyme elevation and dyslipidemia in T2DM.
Kashinakunti et al. (2016) [7]	Cross-sectional T2DM study	GGT elevation: significantly higher GGT in T2DM vs. controls ($p < 0.001$)	Corroborates our mean GGT elevation (38.82 ± 14.03 U/L), supporting GGT as an accessible marker of metabolic derangement.

Notably, the correlation coefficient between serum GGT and HbA1c in our cohort (r = 0.884) is higher than that reported in several general diabetic populations. This difference is largely explained by our strict enrollment criteria: by limiting the study exclusively to drug-untreated, newly diagnosed patients, we eliminated the confounding, glucose-lowering, and hepatoprotective effects of chronic antidiabetic medications (e.g., metformin, SGLT-2 inhibitors) and lipid-lowering therapies (e.g., statins). Thus, our data capture the unmitigated pathophysiological relationship between acute metabolic stress and hepatic enzyme elevation.

Furthermore, 46% (n = 23) of our newly diagnosed patients exhibited elevated serum GGT levels (>40 U/L). This prevalence rate aligns closely with large-scale prospective studies by Lim et al. [5] and Meisinger et al. [28], which demonstrated that subclinical GGT elevation is present in 40% to 50% of individuals with early-stage metabolic syndrome and NAFLD, confirming that hepatic involvement is established early in the course of clinical diabetes.

Clinical Implications:
These findings carry direct clinical implications for diabetes screening and management:

1. Complementary Biomarker Value: Serum GGT should be recognized as an accessible, cost-effective adjunctive biomarker that provides insights into underlying subclinical hepatic steatosis, oxidative stress, and insulin resistance that standard HbA1c testing alone does not capture.
2. Targeted Therapeutic Selection: Identifying elevated GGT levels at initial diabetes diagnosis can help clinicians tailor pharmacotherapy. Selecting glucose-lowering agents with proven hepatoprotective and cardiometabolic benefits—such as GLP-1 receptor agonists, SGLT-2 inhibitors, or pioglitazone—may yield optimal long-term clinical outcomes in diabetic patients presenting with evidence of hepatic metabolic dysfunction [29,30].

Strengths and Limitations:
A primary strength of this study is its enrollment of a homogeneous, drug-untreated cohort, which eliminated medication-induced confounding on liver enzyme and lipid profiles. Additionally, the simultaneous measurement of fasting/postprandial glucose indices, HbA1c, GGT, and full lipid panels provided a comprehensive metabolic assessment for each participant.

However, several limitations must be acknowledged. First, the cross-sectional design prevents establishing causal relationships or a temporal sequence between GGT elevation and glycemic deterioration. Second, the absence of a non-diabetic control group limits our ability to determine specific diagnostic threshold cutoffs. Third, the sample size (N = 50)

from a single tertiary care center in South India limits broad generalizability to other ethnic and geographic populations. Fourth, unmeasured lifestyle factors, such as exact dietary composition and physical activity levels, could have influenced baseline GGT activity. Finally, single-timepoint blood sampling does not capture intra-individual physiological variability over time. Future prospective, multi-center longitudinal studies with larger sample sizes are warranted to confirm the predictive utility of serum GGT in diabetes progression.

CONCLUSION

This study establishes a strong, statistically significant positive correlation between serum Gamma-Glutamyl Transferase (GGT) activity and glycated hemoglobin (HbA1c) levels in newly diagnosed, drug-untreated Type 2 Diabetes Mellitus (T2DM) patients ($r = 0.884$, $p < 0.001$). In addition to tracking glycemic severity, elevated serum GGT levels are tightly coupled with generalized metabolic dysregulation, characterized by significant hypertriglyceridemia, elevated LDL-C and VLDL-C, and profoundly suppressed HDL-C.

These findings support the clinical role of serum GGT as an accessible, cost-effective surrogate biomarker for underlying oxidative stress, intrahepatic lipid accumulation, and systemic insulin resistance—the core pathophysiological drivers of T2DM and its vascular complications. Incorporating routine serum GGT testing alongside conventional glycemic parameters (HbA1c and FPG) during initial diagnostic workups can help clinicians better characterize individual metabolic phenotypes, identify early subclinical hepatic dysfunction, and select targeted cardiometabolic therapies.

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Source of Funding: Nil.

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