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RESEARCH ARTICLE

Serum Gamma-Glutamyl Transferase as a Predictive Biomarker for Metabolic Syndrome and Insulin Resistance: Implications for Early Risk Assessment and Preventive Healthcare

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ABSTRACT

Background: Metabolic syndrome (MetS) is a significant global public health concern. Each component of MetS is linked to various non-communicable chronic diseases, including type 2 diabetes mellitus (T2DM), coronary artery disease (CAD), cerebrovascular disease, and non-alcoholic fatty liver disease (NAFLD), which collectively are major causes of mortality worldwide. **Aims:** This study aimed to investigate the association between serum gamma-glutamyl transferase (GGT) levels and the risk of MetS and insulin resistance (IR) in a sample of adults, exploring how GGT levels correlate with MetS components and IR markers. Additionally, we examined whether these associations vary by gender. **Methods:** A total of 440 participants were selected for this study, including 196 individuals diagnosed with MetS and 244 without. Participants were selected based on predefined criteria from those attending the outpatient department of a biochemistry clinic in Dhaka, Bangladesh. Logistic regression analysis was conducted, adjusting for potential confounders, including age, sex, body mass index (BMI), alanine aminotransferase (ALT), uric acid, and low-density lipoprotein cholesterol (LDL-C), to assess the odds ratios (95% CI) for MetS across GGT tertiles. **Results:** Logistic

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regression analysis indicated that, after adjusting for confounders, the odds ratios for MetS increased significantly across GGT tertiles (1, 1.22 (0.36-4.12), p=0.738; 5.09 (2.06-12.58), p<0.001). Participants with higher GGT levels showed elevated levels of insulin, HOMA-IR, and all MetS components except HDL-C, which was inversely associated with GGT. Specifically, BMI, waist circumference, blood pressure, fasting plasma glucose (FPG), insulin, triglycerides (TG), and GGT were higher, while HDL-C was lower in individuals with MetS and IR compared to those without these conditions. Elevated GGT levels were associated with a higher risk of MetS, independently of other confounding factors, and demonstrated a clear relationship with insulin resistance. Notably, this association persisted across genders without variation. **Conclusion:** This study highlights a strong positive correlation between serum GGT levels and most MetS components, excluding HDL-C, which was inversely correlated with GGT. The relationship between GGT and MetS components, including HOMA-IR, remained substantial across genders, reinforcing the role of GGT as a potential biomarker for MetS and insulin resistance.

Keywords: Metabolic Syndrome (MetS); Gamma-Glutamyl Transferase; HOMO-IR; Insulin resistance

1. Introduction

Metabolic syndrome (MetS), characterized by a combination of metabolic risk factors such as central obesity, elevated blood sugar, high blood pressure, increased serum triglycerides[1,2], and low HDL-C, significantly increases the risk of cardiovascular diseases (CVD)[3], type 2 diabetes mellitus (T2DM), and other leading causes of death[4]. It primarily results from abnormal adipose deposition and function, coupled with insulin resistance (IR), and has become a major global public health issue. According to the International Diabetes Federation, approximately 20-25% of adults worldwide suffer from MetS[5]. Park observed an increasing prevalence of MetS with age, particularly from 20 years to the sixth decade of life in the U.S. population[6]. To reduce the incidence of MetS and related cardiovascular diseases, the National Cholesterol Education Program-Adult Treatment Panel III (NCEP; ATP III) recommends lipid profile screening starting at age 20, with subsequent screenings every five years[7]. An increasing trend of MetS has also been reported in Asian populations, including a rising rate among Bangladeshi adults in both urban and rural areas[8]. Several expert organizations have proposed different criteria for diagnosing MetS, incorporating parameters from the American Heart Association/National Heart, Lung, and Blood Institute (AHA/NHLBI), the World Health Organization (WHO), and others, all of which emphasize the crucial role of insulin resistance (IR) in the development of MetS[9].

Gamma-glutamyl transferase (GGT) is an enzyme found in various tissues, with its highest concentration in the kidney, though the hepatobiliary system is the primary source of serum GGT[10]. Traditionally used to detect alcoholism and hepatobiliary diseases, GGT is now recognized as a sensitive biomarker for oxidative stress and cardiovascular risk[11]. GGT catalyzes the breakdown of glutathione, leading to the formation of superoxide and hydrogen peroxide, which contribute to oxidative stress, inflammation, and impaired insulin signaling in the liver, muscle, and adipose tissue[12,13]. This disruption results in glucose intolerance and dyslipidemia. As such, elevated GGT levels have been associated with oxidative stress, inflammation, and insulin resistance—all of which are key factors in the development of MetS[14,15]. Several prospective studies have suggested that elevated serum GGT may serve as an important predictor for the development of MetS and its associated complications, such as T2DM and CVD[16]. One research highlighted a link between elevated GGT and hypertriglyceridemia, raised blood glucose and IR[17]. Other studies have shown significant associations between high GGT levels and the development of hyperglycemia, dyslipidemia, hypertension, and obesity. Therefore, GGT appears to be associated with all major components of MetS, making it a potentially valuable biomarker for identifying individuals at risk[18]. The aim of this study is to explore the

relationship between serum GGT levels and MetS in a cohort of individuals, analyzing how GGT may serve as a reliable biomarker for MetS diagnosis and as a potential predictor of its development.

2. Materials and methods

2.1. Study settings and study population

This was an analytical cross-sectional study conducted in the biochemistry department between February 2021 and March 2022. Participants were selected using purposive sampling, totaling 440 individuals, with 196 diagnosed with metabolic syndrome (MetS) and 244 without. The study included adults aged 20 to 60 years of both genders who were apparently healthy. Exclusion criteria included pregnant and lactating women; individuals with acute severe infections, cardiovascular disease, liver disease, kidney disease, pulmonary disease, chronic debilitating conditions (such as cancer or HIV), alcoholism, smoking habits; patients on medications that impact liver enzyme levels; insulin users; and those taking oral hypoglycemic agents.

2.2. Anthropometric data collection

A metal tape was used to measure height. The study subject was instructed to stand with their feet flat, together, and against a level surface—such as a wall—and to softly note the location where the head piece's bottom touched the wall. In order to determine height, metal tape was finally utilized to measure from the base on the floor to the designated measurement on the wall. With both feet in the center of the scale, light clothing, and no shoes on, the weight was measured using a digital scale. To determine BMI, divide weight (in kilograms) by square[16]. The midpoint between the top edge of the iliac crest and the lower ribs was identified as the waist circumference (WC), which was measured using a measuring tape that had an insertion buckle at one end. WC was measured with a centimeter accuracy. After five minutes of rest, the BP was measured with a manual sphygmomanometer and the appropriate cuff size. Two sitting-position blood pressure readings on the left upper arm were taken, separated by five minutes. Two readings were averaged and then analyzed.

2.3. Study procedure

This cross-sectional analytical study was conducted from February 2021 to March 2022. Participants were purposively selected from those visiting the outpatient department (OPD) based on specific inclusion criteria, and only individuals who appeared to be in good health were enrolled. All participants provided informed written consent after being fully informed about the study's purpose. Each participant received a thorough explanation of the study's objectives, purpose, and methodology following counseling, and only those who willingly agreed were included. Sociodemographic and other relevant data were collected and recorded on data collection sheets. Comprehensive physical and relevant clinical assessments were conducted and documented for each participant.

2.4. Blood sample collection and Laboratory Analysis

Fasting blood samples were collected from all participants. They were allowed to fast overnight (10–12 hours). Blood was collected from the antecubital vein after all aseptic precautions, 5 ml venous blood was taken by sterile disposable syringe. 2 ml of collected blood was taken in a test tube coated with dried sodium fluoride-potassium oxalate mixture and plasma was separated after centrifugation at 3000 rpm for five minutes for fasting glucose and insulin. The remaining 3 ml blood was collected in a plain tube. This tube was allowed to stand for 20 to 30 minutes so that blood was clotted properly. Then serum was separated after centrifuging at 3000 rpm for 10 minutes and was collected into microcentrifuge tubes, labelled properly to measure serum

GGT, lipid profile, ALT, uric acid. Samples were preserved in a deep freezer at-37^{0}C and were analyzed later. All the Biochemical tests were done in the biochemistry laboratory[19].

2.5. Ethical Statement

Ethical permission for this study was obtained from the relevant institutional review board (IRB) to ensure compliance with ethical standards. Participants were informed about the study's purpose, procedures and potential risks and written informed consent was obtained from all participants. The study adhered to ethical principles in research, including confidentiality, voluntary participation and the right to withdraw at any time without consequence.

2.6. Data analysis

Data were analyzed using SPSS version 23. Results are presented as mean ± SD. Unpaired Student's t-test was used to compare MetS vs. non-MetS subjects and IR vs. non-IR subjects, as well as gender differences. Serum GGT levels were divided into tertiles to observe trends. ANOVA was applied to compare means of quantitative variables. A p-value <0.05 was considered statistically significant.

3. Results

This study included 440 participants, with 196 having MetS and 244 without. **Table 1** presents the mean ± SD of various parameters in relation to MetS. Compared to non-MetS subjects, those with MetS had significantly higher BMI, WC, BP, GGT, FPG, insulin, and HOMA-IR (p<0.001). There were no significant differences in age, uric acid, ALT, or LDL-C between the two groups. Additionally, HDL-C was lower, and TC and TG were higher in MetS subjects compared to non-MetS subjects (p<0.001).

Table 1. Basic characteristics and Biochemical parameters of study subjects according to metabolic syndrome (n=440).

Variables	Subjects with MetS (n=196)	Subjects without MetS (n=244)	p-value
Age (years)	40.04±12.77	38.84±12.60	0.621
BMI (kg/m^2)	28.03±4.07	22.00±3.74	**<0.001**
WC (cm)	102.08±9.69	80.18±7.37	**<0.001**
SBP (mmHg)	127.76±13.23	110.00±9.13	**<0.001**
DBP (mmHg)	88.37±10.07	73.52±7.82	**<0.001**
FPG (mmol/L)	6.51±1.41	4.78±0.63	**<0.001**
FPI (µU/ml)	13.47±±5.80	7.39±3.41	**<0.001**
HOMA-IR	1.85±0.83	0.98±0.47	**<0.001**
GGT (U/L)	33.18±15.69	17.90±7.15	**<0.001**
ALT (U/L)	24.78±6.86	23.20±6.00	0.201
Uric acid (mg/dl)	3.96±2.60	3.86±1.75	0.814
TC (mg/dl)	189.47±29.59	168.11±21.11	**<0.001**
TG (mg/dl)	208.71±55.06	137.41±18.81	**<0.001**
HDL-C (mg/dl)	32.53±4.91	37.54±5.66	**<0.001**
LDL-C (mg/dl)	108.65±29.02	104.39±21.19	0.376

The features of both male and female study participants are displayed in **Table 2**. All parameters showed no significant difference based on gender, with the exception of BMI, which was greater (p<0.05) in females.

Table 2. Characteristics of all study population, male subjects and female subjects (n=440).

Variables	Total (n=440)	Male (n=232)	Female (n=208)	p-value
Age (years)	39.37±12.63	41.07±12.94	37.48±12.13	0.138
BMI (kg/m^2)	24.68±4.91	23.63±4.27	25.86±5.33	**<0.05**
WC (cm)	89.94±13.81	92.62±11.85	88.69±16.20	0.147
SBP (mmHg)	117.91±14.20	118.10±12.31	117.50±16.19	0.825
DBP (mmHg)	80.14±11.54	80.52±10.33	79.52±12.77	0.652
FPG (mmol/L)	5.55±1.35	5.53±1.30	5.60±1.43	0.795
FPI (μU/ml)	10.10±5.51	10.63±5.90	9.51±5.04	0.287
HOMA-IR	1.37±0.79	1.44±0.77	1.29±0.80	0.341
GGT (U/L)	24.71±13.95	25.86±14.58	23.42±13.24	0.363
ALT (U/L)	23.90±6.42	23.98±6.67	23.81±6.19	0.887
Uric acid (mg/dl)	3.90±2.16	4.12±2.39	3.66±1.86	0.266
TC (mg/dl)	177.63±27.29	177.98±27.86	177.23±26.90	0.886
TG (mg/dl)	169.17±52.89	173.12±55.34	166.96±51.14	0.547
HDL-C (mg/dl)	35.31±5.88	34.46±6.82	36.07±4.81	0.153
LDL-C (mg/dl)	106.29±24.95	105.45±28.08	107.23±21.16	0.710

Data were expressed as mean±SD

Unpaired student t-test was done to compare between males and females.

Table 3 presents the characteristics of male and female subjects across serum GGT tertiles. For both genders, individuals in higher GGT tertiles displayed significantly higher BMI, waist circumference, blood pressure, fasting plasma glucose, fasting insulin, HOMA-IR, and triglycerides, along with lower HDL-C levels. Age, uric acid, and LDL-C levels did not significantly vary across tertiles. In males, ALT was slightly elevated with higher GGT, whereas in females, ALT and uric acid levels showed no significant difference across tertiles.

Table 3. Characteristics of Male and Female Participants Across Serum GGT Tertiles.

Variables	Male	Female	p-value
Serum GGT (U/L)	**Tertile 1 (<18)**	**Tertile 2 (18-29)**	**Tertile 3 (>29)**
Participants (n)	**76**	**76**	**80**
Age (years)	38.89 ± 13.13	40.95 ± 14.12	43.70 ± 11.65
BMI (kg/m²)	20.74 ± 3.03	23.97 ± 3.65	26.05 ± 4.33
Waist Circumference (cm)	82.37 ± 7.02	88.05 ± 8.51	100.90 ± 10.94
Systolic BP (mmHg)	109.47 ± 9.11	116.32 ± 4.96	128.00 ± 13.12
Diastolic BP (mmHg)	74.74 ± 9.64	77.89 ± 7.87	88.50 ± 8.13
Fasting Plasma Glucose (mmol/L)	4.57 ± 0.59	5.13 ± 0.51	6.81 ± 1.30
Fasting Insulin (μU/ml)	6.81 ± 3.35	9.66 ± 4.49	15.19 ± 6.10
HOMA-IR	0.87 ± 0.44	1.33 ± 0.49	2.08 ± 0.79

Table 3. (*Continued*).

Variables	Male	Female	p-value
Serum GGT (U/L)	**Tertile 1 (<18)**	**Tertile 2 (18-29)**	**Tertile 3 (>29)**
Participants (n)	**76**	**76**	**80**
ALT (U/L)	20.95 ± 6.10	25.84 ± 7.13	25.10 ± 6.00
Uric Acid (mg/dL)	3.87 ± 1.58	4.28 ± 2.41	4.21 ± 3.03
Total Cholesterol (mg/dL)	170.63 ± 27.14	174.63 ± 22.60	188.15 ± 31.20
Triglycerides (mg/dL)	128.63 ± 22.58	159.68 ± 19.54	228.15 ± 54.74
HDL-C (mg/dL)	39.37 ± 3.34	37.79 ± 3.19	31.30 ± 3.39
LDL-C (mg/dL)	103.89 ± 26.60	106.74 ± 22.80	105.70 ± 34.62

In **table 4**, both males and females, those in Tertile 3 (highest GGT levels) had significantly higher waist circumference (WC), systolic and diastolic blood pressure (SBP, DBP), fasting plasma glucose (FPG), HOMA-IR, and triglycerides (TG), and significantly lower HDL-C compared to Tertiles 1 and 2. Tertiles 1 and 2 showed few differences, except in TG and HDL-C for both sexes.

Table 4. Post-hoc Analysis (Bonferroni Test) of Differences Between Serum GGT Tertiles in Male and Female Subjects.

Variables	Male: Tertile 1 vs Tertile 2 (p)	Male: Tertile 1 vs Tertile 3 (p)	Male: Tertile 2 vs Tertile 3 (p)	Female: Tertile 1 vs Tertile 2 (p)	Female: Tertile 1 vs Tertile 3 (p)	Female: Tertile 2 vs Tertile 3 (p)
WC (cm)	0.171	<0.001	<0.001	1	<0.001	<0.001
SBP (mmHg)	0.104	<0.001	<0.01	0.188	<0.001	<0.01
DBP (mmHg)	0.784	<0.001	<0.01	0.101	<0.001	<0.001
FPG (mmol/L)	0.17	<0.001	<0.001	0.355	<0.001	<0.001
HOMA-IR	0.064	<0.001	<0.01	0.532	<0.001	<0.01
ALT (U/L)	0.067	0.145	1	—	—	—
TG (mg/dl)	<0.05	<0.001	<0.001	0.948	<0.001	<0.001
HDL-C (mg/dl)	0.441	<0.001	<0.001	<0.001	<0.001	<0.05

Table 5 shows correlation of GGT with components of MetS and HOMA-IR. Significant correlations of GGT with components of MetS and HOMA-IR were observed in all subjects. These correlations were also significant among male and female subjects.

Table 5. Correlation of serum GGT with components of MetS and HOMO-IR.

Variables	All subjects		Male		Female	
	r-value	p-value	r-value	p-value	r-value	p-value
WC (cm)	+.670	**<0.001**	+.731	**<0.001**	+.614	**<0.001**
SBP (mmHg)	+.735	**<0.001**	+.764	**<0.001**	+.729	**<0.001**
DBP (mmHg)	+.628	**<0.001**	+.557	**<0.05**	+.707	**<0.001**
FPG (mmol/L)	+.804	**<0.001**	+.820	**<0.001**	+.806	**<0.001**
TG (mg/dl)	+.823	**<0.001**	+.842	**<0.001**	+.793	**<0.001**
HDL-C (mg/dl)	-.619	**<0.001**	-.663	**<0.001**	-.656	**<0.001**
HOMA-IR	+.567	**<0.001**	+.511	**<0.05**	+.652	**<0.001**

Correlations were determined by Pearson's correlation coefficient test

Table 6 presents the odds ratios (OR) for MetS across serum GGT tertiles, with the lowest tertile as the reference (OR=1). In both the non-adjusted model (Model 1) and the age- and gender-adjusted model (Model 2), the ORs for tertiles 2 and 3 were significantly higher than tertile 1. In Model 3, after adjusting for BMI, ALT, uric acid, and LDL-C along with age and gender, the ORs were reduced, and the OR for tertile 2 became insignificant, while the OR for tertile 3 remained significant. In Model 4, when only HOMA-IR was adjusted along with age and gender, the ORs for both tertiles were further attenuated and became non-significant.

Table 6. Odds ratio (95% CI) for MetS according to tertiles of serum GGT.

		Tertile 1 (n=148)	**Tertile 2 (n=148)**	**Tertile 3 (n=144)**
	Cases, N(%)	7 (18.9%)	17 (45.9%)	25 (69.4%)
Model 1	OR (95% CI)	1	3.64 (1.29-10.37)	9.74 (3.28-28.6)
	p-value		**<0.05**	**<0.001**
Model 2	OR (95% CI)	1	2.99 (1.11-8.13)	8.12(2.74-16.52)
	p-value		**<0.05**	**<0.001**
Model 3	OR (95% CI)	1	1.22 (0.36-4.12)	5.09 (2.06-12.58)
	p-value		0.738	**<0.001**
Model 4	OR (95% CI)	1	1.08 (0.16-7.29)	2.20 (0.55-8.87)
	p-value		0.935	0.266

Logistic regression analysis was done for adjusted odds ratio; Model 1: Non-adjusted; Model 2: Adjusted for age and sex; Model 3: Adjusted for age, sex, BMI, ALT, uric acid and LDL-C; Model 4: Adjusted for age, sex and HOMA-IR.

4. Discussion

The study found that, with the exception of BMI—which was higher in women—there were no significant gender differences in any of the measured parameters. Participants with metabolic syndrome (MetS) exhibited significantly elevated levels of GGT, BMI, waist circumference (WC), and blood pressure (BP) compared to those without MetS (**Table 1**). Additionally, BMI, WC, and BP were significantly elevated among those in the higher tertile of GGT (Tables 2, 3, and 4). Notably, positive associations between GGT and both WC and BP were observed (**Table 5**), suggesting a link between increased abdominal fat, hypertension, and higher GGT levels. These findings align with research by Lawlor et al.[18,20], which indicates that serum GGT may contribute to oxidative stress and insulin resistance (IR), both of which are connected to obesity and high blood pressure.

Subjects with MetS, in comparison to those without MetS, displayed significantly lower levels of HDL-C and significantly higher levels of triglycerides (TG) and total cholesterol (TC) (**Table 1**). As demonstrated in **Table 3**, individuals in the upper GGT tertiles had lower HDL-C levels alongside higher TG and TC. Additionally, a strong positive correlation was found between GGT and TG, while an inverse relationship was noted with HDL-C (**Table 5**), consistent with Masilamani's study[21]. This investigation also found a linear association between serum GGT and IR. Increased lipolysis due to IR in adipose tissue[22] may elevate free fatty acid (FFA) flow from peripheral tissues to the liver, enhancing hepatic TG synthesis and VLDL secretion into the bloodstream[23]. Elevated TG levels lead to secondary reductions in HDL-C, with cholesterol ester transfer protein (CETP) facilitating TG and cholesteryl ester exchange between lipoproteins, producing TG-enriched HDL that is rapidly catabolized[24]. Furthermore, subjects with IR had significantly higher GGT levels and all MetS components, excluding HDL-C (**Table 2**). Fasting plasma glucose (FPG), insulin, and HOMA-IR were significantly higher in the upper GGT tertile (**Table 3**). Positive correlations between GGT and both

HOMA-IR and FPG observed in this study (**Table 5**) further suggest a relationship between GGT and IR. These findings align with those of Marchesini et al.[17] and Kang[25], who reported a closer link between serum GGT and hepatic IR, independent of nonalcoholic fatty liver disease (NAFLD) presence.

This study also demonstrated significant associations between serum GGT and all MetS components, as well as HOMA-IR (**Table 5**). Odds ratios for MetS increased with higher GGT tertiles (**Table 6**). The association remained significant after adjusting for age, gender, BMI, ALT, uric acid, and LDL-C. However, when adjusted solely for age, gender, and HOMA-IR, the association was diminished, suggesting IR as the primary confounding factor in this relationship. This underscores the impact of HOMA-IR on MetS, as confirmed by multiple linear regression analysis, which indicated a significant independent association between serum GGT and HOMA-IR. Similar findings were reported in a Japanese cross-sectional study[18], and Kim et al[16]. identified serum GGT as a potential predictor of MetS in a study of 2,579 MetS-free Korean adults, even after adjusting for age, sex, alcohol use, smoking, and family diabetes history. Serum GGT serves as a sensitive marker for alcohol consumption and biliary obstruction, and elevated levels are also noted in fatty liver disease, viral hepatitis, primary biliary cirrhosis (PBC), and drug-induced liver injury. Given the potential presence of undiagnosed liver disorders within community-dwelling individuals, some study participants may have had subclinical liver conditions[26]. However, since PBC is rare and viral hepatitis was accounted for and excluded, the effect of liver diseases on the results is likely minimal. Furthermore, the GGT-MetS association persisted even after adjusting for ALT.

There is substantial research regarding how GGT reflects MetS and IR risk. Elevated GGT activity initiates glutathione breakdown[27,28], releasing reducing agents that convert ferric to ferrous ions, generating hydrogen peroxide and superoxide in sequence[29]. Consequently, higher GGT levels increase oxidative stress, which fosters inflammation and impairs insulin signaling in the liver, muscle, and adipose tissue, leading to MetS[30]. Individuals with elevated GGT, even within the normal range, frequently exhibit hepatic steatosis, strongly linked to visceral fat accumulation and enhanced lipolysis[31]. Hepatic steatosis contributes to hepatic IR, and prolonged IR in the liver can precipitate metabolic irregularities[32]. Moreover, the inflammatory responses triggered by elevated GGT levels disrupt insulin signaling in the liver and other organs[22,33]. This study concludes that serum GGT is positively associated with IR and MetS, with IR as the main factor linking GGT to MetS. Thus, serum GGT may serve as an independent predictor for MetS.

5. Conclusion

This study emphasizes the role of serum gamma-glutamyl transferase (GGT) as a significant biomarker for predicting metabolic syndrome (MetS) and its components. We found strong associations between elevated GGT levels and key MetS indicators, including BMI, waist circumference, blood pressure, triglycerides, and total cholesterol, alongside an inverse relationship with HDL-C. Higher GGT levels were also linked to insulin resistance (IR), as indicated by increased fasting plasma glucose and HOMA-IR, and this remained significant even after adjusting for confounding factors. GGT contributes to oxidative stress, which disrupts insulin signaling and exacerbates metabolic abnormalities, such as elevated blood pressure and triglycerides, and reduced HDL-C. Given these findings, GGT could be a valuable marker for identifying individuals at risk of MetS, even within normal reference ranges. Early GGT monitoring may enable timely interventions to prevent MetS-related complications. In conclusion, GGT is a robust independent predictor of MetS, highlighting its potential in risk assessment and preventive healthcare strategies.

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Conflict of interest

The authors have no conflict of interest to declare.

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