Diabetes, Metabolic Syndrome and Obesity

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ORIGINAL RESEARCH

Serum Gamma Glutamyltransferase: A Biomarker for Identifying Postprandial Hypertriglyceridemia

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Purpose: Elevated serum gamma-glutamyltranspeptidase (GGT) is an independent marker of the activation of systemic inflammation, while conditions associated with elevated triglyceride (TG) levels, such as type 2 diabetes, non-alcoholic fatty liver disease, obesity, and metabolic syndrome, are associated with an increased inflammatory burden. Moreover, serum liver enzymes (GGT, alanine aminotransferase [ALT], aspartate aminotransferase [AST], and alkaline phosphatase [ALP]) are associated with metabolic syndrome and its components, including hypertriglyceridemia. However, the relationship between liver enzymes and postprandial hypertriglyceridemia (PHTG) remains unclear. Therefore, in this study we conducted oral fat tolerance tests (OFTTs) to understand the differences in serum liver enzyme levels among individuals with different lipid tolerance levels and their correlation with PHTG.

Patients and Methods: For the OFTT, we enrolled 202 non-diabetic volunteers whose fasting triglyceride (TG) levels were less than 1.7 mmol/L in this case-control study. The participants were categorized into two groups according to the TG levels at the 0- and 4-h OFTT: a postprandial normal TG (PNTG) group and a PHTG group. Routine fasting serum biochemical indices, liver enzyme (GGT, ALT, AST, and ALP) levels, and 0- and 4-h OFTT lipid levels were assessed.

Results: The PHTG group had significantly higher serum GGT and ALT levels and a lower AST/ALT ratio than those in the PNTG group. However, no significant difference was observed in AST and ALP levels compared with the PNTG group. After adjusting for major confounders, logistic regression analysis indicated a significant correlation between serum GGT and PHTG (odds ratio = 1.168, $P < 0.001$), but not with ALT level, AST level, AST/ALT ratio, and ALP level. The receiver operating characteristic curve analysis demonstrated that the serum GGT level was an effective predictor of PHTG.

Conclusion: Serum GGT levels are significantly associated with PHTG risk and serve as an effective biomarker for early identification.

Keywords: gamma-glutamyl transferase, alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, inflammation, triglyceride, oral fat tolerance test

Introduction

With rapid economic development and lifestyle changes, the prevalence of hypertriglyceridemia (HTG) in our country has gradually increased. Dyslipidemia poses a serious threat to human health. Elevated levels of triglyceride (TG)-rich lipoprotein cholesterol have been linked to an increased risk of cardiovascular disease, according to large clinical trials.[1,2] In individuals with HTG, fibrate therapy may drastically reduce the incidence of heart attack and death.[3,4] Postprandial hypertriglyceridemia (PHTG) has emerged as a risk factor for atherosclerotic cardiovascular disease (ASCVD).[5–7] Non-fasting lipid profiles are convenient for predicting ASCVD risk and comparable to or even more meaningful than fasting samples.[8,9] Of note, most people only fast for a few hours daily (in the early morning). The postprandial retention of TG-rich residual lipoproteins in the

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arterial walls may result in the development of atherosclerosis and ASCVD.[6] Therefore, early detection of abnormal PHTG significantly reduces ASCVD occurrence.

Serum gamma-glutamyl transferase (GGT) has recently garnered extensive attention because of its role in cardiovascular and metabolic diseases.[10,11] GGT, a glycoprotein present on the surface of the cell membrane, is widely distributed in various organs and cells. It is an enzyme that determines glutathione hydrolysis inside and outside of cells, playing a biological role in antioxidative stress.[12] Elevated serum GGT is an independent marker of the activation of systemic inflammation.[13] Diseases associated with elevated TG levels, such as type 2 diabetes, non-alcoholic fatty liver disease, obesity, and metabolic syndrome, are associated with an increased inflammatory burden.[14–17] Studies have found that serum GGT is linked to HTG and metabolic syndrome (MetS).[18–20] Individuals with elevated serum GGT levels face a significantly increased risk of developing MetS and its components, including overweight or obesity, HTG, hyperglycemia, and hypertension.[18,19] Additionally, research indicates that other liver enzymes, such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP), are linked to MetS and its components.[21–24] Therefore, we can hypothesize that serum liver enzyme levels may be increased in individuals with normal fasting TG levels who exhibit only PHTG and that this elevation could relate to PHTG. To test this hypothesis, we measured the serum liver enzyme levels by performing a high-fat meal test and evaluated the correlation between liver enzyme levels and PHTG, providing a diagnostic basis for the early detection of lipid disorders.

Materials and Methods

Participants

The Hebei General Hospital Ethics Committee approved this investigation, which was registered in the Chinese Clinical Trial Registry (registration number: ChiCTR1800019514) and conducted according to the principles of the Declaration of Helsinki. In the endocrinology outpatient department, 202 volunteers were enlisted between May 2018 and December 2019.[25] All Participants signed informed consent forms and completed the necessary questionnaires. The ages of volunteers ranged from 23 to 70 years, and all were Han Chinese from the Hebei Province.

Oral glucose tolerance tests (OGTTs) were conducted for all volunteers. In addition to the previously reported exclusion criteria,[26] volunteers with fasting TG level ≥1.7 mmol/L, smokers, alcohol drinkers (alcohol intake >30 g per day for men and >20 g per day for women), and volunteers with viral hepatitis or a history of liver disease, including cirrhosis, chronic hepatitis, and autoimmune hepatitis, were also excluded.

Oral Fat Tolerance Test

All volunteers who met the inclusion criteria were given a normal diet for 1 week before the trial, which was not high in fat or protein. The volunteers underwent water fasting after 22:00 on the night before the research and then ingested a standard high-fat meal at 8:00 the following day. Professional nutritionists prepared the high-fat meals that each contained 1500 calories and comprised 60%, 20%, and 20% of fat, carbohydrates, and protein, respectively. All volunteers consumed their meals within 10 min, refrained from eating for 4 h, and had free access to plain water. Simultaneously, both smoking and physical activity were prohibited. Blood samples were drawn on an empty stomach and 4 h after the high-fat meal; the serum was stored at −80 °C for later use.

Detection of Clinical and Biochemical Indicators and Definition of Fatty Liver

Data on sex, age, height, weight, waist circumference (WC), systolic blood pressure (SBP), and diastolic blood pressure were collected by professional physicians from all participants. Body mass index (BMI) was calculated as: weight (kg)/height (m^2). Fasting blood glucose (FBG), 2-h OGTT blood glucose, serum uric acid (SUA), GGT, ALT, AST, ALP, albumin, total/direct/indirect bilirubin, apolipoprotein A1, apolipoprotein B (ApoB), oral fat tolerance test (OFTT) at 0 and 4 h, total cholesterol (TC), TG, high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) levels were measured using a 7600 automatic biochemical analyzer (Hitachi, Tokyo, Japan). Fasting insulin (FINS) and 2-h OGTT insulin were measured by electrochemical luminescence. The homeostasis model was used to evaluate insulin resistance and the islet β cell function index: homeostasis model assessment-estimated insulin

resistance (HOMA-IR) = FBG (mmol/L) × FINS (μIU/mL) /22.5; homeostasis model assessment of β-cell function = 20×FINS (μIU/mL)/(FBG [mmol/L] −3.5). Non-HDL-C and TG-rich lipoprotein residues (TRLRs) were calculated using the following formula: Non-HDL-C = TC-HDL-C; TRLRs = TC−(HDL-C)−(LDL-C). A fatty liver was defined as having at least two ultrasonic abnormalities, as follows:[27] 1) increased near-field echo and decreased far-field echo; 2) dense and stronger echo of the liver parenchyma than that of the kidney parenchyma; and 3) an unclear structure of the hepatic vessels and biliary tract.

Definition and Grouping of PHTG

According to the 2019 expert panel statement,[28] a TG concentration at 4 h after an OFTT meal of 2.5 mmol/L was used as the criterion for PHTG. Based on the results of the OFTT, the volunteers were classified into two groups: 1) a group with normal TG levels after the high-fat meal (postprandial normal triglyceride [PNTG], 0-h TG level < 1.7 mmol/L and OFTT 4-h TG level < 2.5 mmol/L); and 2) a PHTG group (0-h TG level < 1.7 mmol/L and OFTT 4-h TG level > 2.5 mmol/L).

Statistical Analysis

Statistical analyses were performed using SPSS software (version 21.0; IBM Corp., Armonk, NY, USA). Normality was determined using the Kolmogorov–Smirnov test. Normally distributed measurement data are expressed as the mean ± standard deviation (x±s), and non-normally distributed measurement data are expressed as the median (interquartile range). An independent sample *t*-test was used for comparisons between two groups if the data were normally distributed and the variance was equal; otherwise, the Mann–Whitney *U*-test was used. Blood lipid levels before and after a high-fat diet were compared using a paired sample *t*-test. For linear correlation analysis between two variables, Pearson's correlation analysis was used when the distribution of the data variables was normal; otherwise, Spearman's rank correlation was used. A binary logistic regression analysis was conducted to assess the factors influencing PHTG. Receiver operating characteristic (ROC) curves were used to analyze the diagnostic value of GGT for PHTG. A P-value < 0.05 was considered statistically significant.

Results

Baseline data comparison between the two groups. This study involved 202 participants in total: 108 in the PNTG group and 94 in the PHTG group. BMI, WC, SBP, SUA level, FINS level, HOMA-IR, ApoB level, non-HDL-C level, TRLRs, and the prevalence of fatty liver were higher in the PHTG group than those in the PNTG group (P<0.05; Table 1). GGT and ALT levels in the PHTG group were significantly higher than those in the PNTG group, and the AST/ALT ratio was lower than that in the PNTG group (P<0.05).

Table 1 Baseline Data Comparison Between the Two Groups

Group	PNTG (108)	PHTG (94)	P
Age (years)	44.61±12.39	46.98±13.02	0.187
Male, n (%)	40 (37.0)	45 (47.9)	0.120
Body mass index (kg/m^2)	24.42±3.32	25.99±3.3	0.001
Waist circumference (cm)	83.47±10.13	87.78±9.54	0.002
Systolic blood pressure (mmHg)	122.33±13.78	127.79±15.58	0.009
Diastolic blood pressure (mmHg)	76.08±9.05	78.27±9.33	0.093
Serum uric acid (μmol/L)	284.03±76.92	321.09±85.34	0.001
Fasting blood glucose (mmol/L)	5.0 (4.75, 5.38)	5.09 (4.92, 5.38)	0.186
2-h blood glucose (mmol/L)	5.89 (5.04,7.02)	5.72 (4.95,6.82)	0.614
Fasting insulin (μIU/mL)	8.34 (6.14, 10.55)	9.42 (6.75, 13.00)	0.016
2-h insulin (μIU/mL)	48.27 (26.99, 68.21)	43.52 (27.50, 84.24)	0.602
HOMA-IR	1.89 (1.36, 2.34)	2.18 (1.57, 3.00)	0.009
HOMA-β	109.15 (73.91, 143.20)	113.08 (81.28, 171.93)	0.247
Apolipoprotein A1 (g/L)	1.43 (1.28, 1.61)	1.40 (1.25, 1.55)	0.192

(*Continued*)

Table 1 (Continued).

Group	PNTG (108)	PHTG (94)	P
Apolipoprotein B (g/L)	0.67 (0.56, 0.82)	0.81 (0.69, 0.95)	<0.001
Apolipoprotein A1/Apolipoprotein B	2.05 (1.75, 2.59)	1.75 (1.45, 2.05)	<0.001
Non-HDL-C (mmol/L)	3.12±0.84	3.54±0.76	<0.001
TRLRs (mmol/L)	0.35±0.2	0.47±0.18	<0.001
Fatty liver, n (%)	12 (11.11)	21 (22.34)	0.031
ALB (g/L)	44.75±2.68	44.35±2.27	0.261
Total bilirubin	12.15 (10.15, 15.7)	12.75 (9.78, 16.08)	0.909
Direct bilirubin	2.10 (1.68, 2.60)	2.10 (1.50, 2.50)	0.554
Indirect bilirubin	10.20 (8.58, 13.7)	10.70 (8.25, 13.45)	0.933
GGT (U/L)	14.00 (11.00, 17.00)	19.00 (15.00, 26.00)	<0.001
ALT (U/L)	13.00 (10.25, 17.00)	16.00 (12.00, 22.00)	0.002
AST (U/L)	19.50 (16.25, 22.00)	19.00 (17.00, 22.00)	0.798
AST/ALT	1.33 (1.11, 1.7)	1.13 (0.86,1.54)	0.001
ALP (U/L)	66.15±18.02	70.35±17.83	0.098

Abbreviations: ALB, albumin; ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GGT, gamma glutamyltransferase; HOMA-β, homeostasis model assessment of β-cell function; HOMA-IR, homeostasis model assessment-estimated insulin resistance; non-HDL-C, non-high-density lipoprotein-cholesterol; PHTG, postprandial hypertriglyceridemia; PNTG, postprandial normal triglyceride; TRLRs, triglyceride-rich lipoprotein remnants.

Comparison of Fasting and 4-h Postprandial Lipid Levels Between the Two Groups

At the 0- and 4-h OFTT, the TG, TC, LDL-C, and HDL-C levels were significantly different between the two groups (P<0.05, Figure 1). The fasting TG, HDL-C, and LDL-C levels in both groups were significantly different from those at 4 h after the high-fat diet (P<0.05).

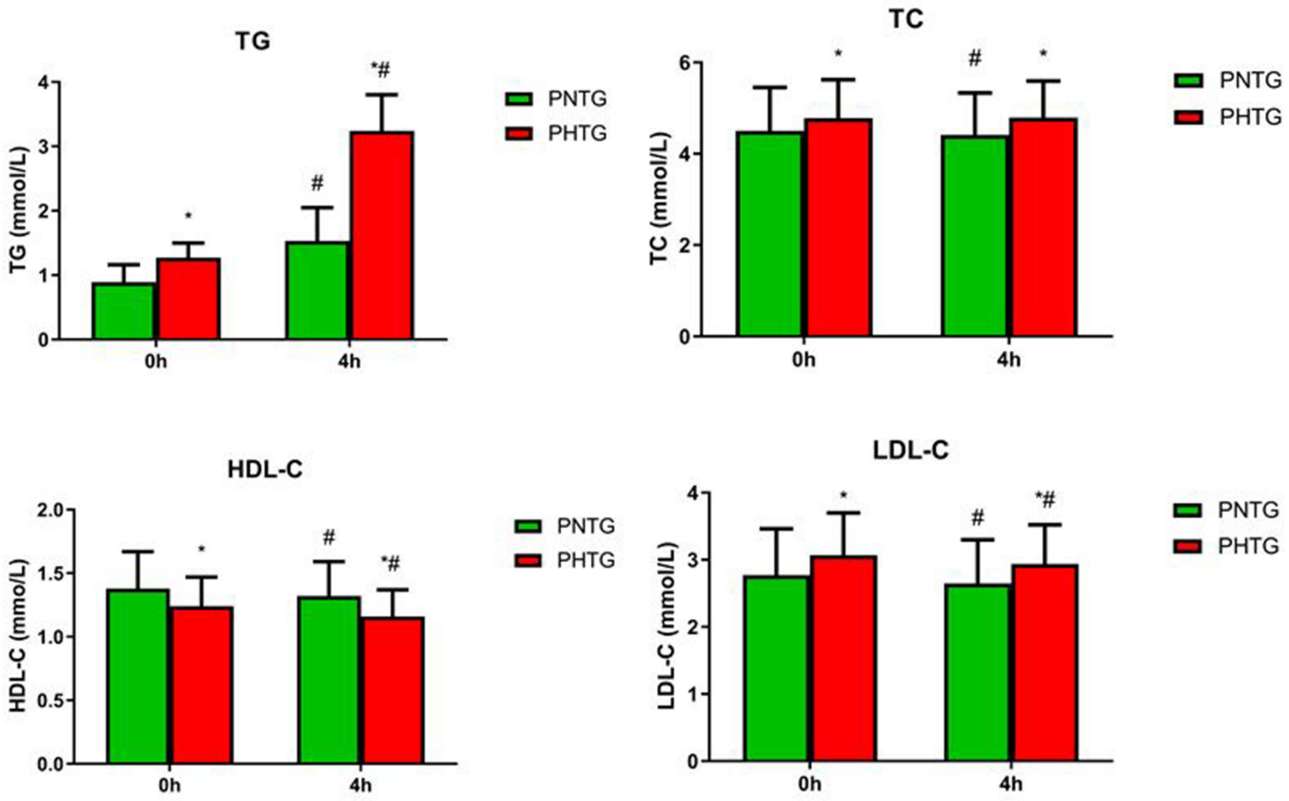

Figure 1 Changes in serum lipid levels after fasting and 4 h after a high-fat meal. *P < 0.05 versus the PNTG group; #P < 0.05 versus 0 h in the same group.
Abbreviations: PHTG, postprandial hypertriglyceridemia; PNTG, postprandial normal triglyceride.

Correlation Analysis of Serum Liver Enzymes and Metabolic Indexes

Among the four liver enzymes, serum GGT and ALT levels significantly correlated with glycolipid metabolism indices, with GGT having a stronger correlation than that of ALT. Serum ALP had a certain correlation with serum lipids, whereas serum AST did not significantly correlate with glycolipid metabolism indices (Table 2).

The Influencing Factors of PHTG Analyzed by Logistic Regression

The occurrence of PHTG was the dependent variable, and sex, age, BMI, WC, SUA level, HOMA-IR, fatty liver, lipid levels, and liver enzyme levels were the independent variables for univariate and multivariate binary logistic regression analyses. Univariate regression analysis revealed that BMI, WC, SUA level, HOMA-IR, fatty liver, TC level, TG level, HDL-C level, LDL-C level, GGT level, ALT level, and the AST/ALT ratio significantly correlated with PHTG (Table 3). Multivariate regression analysis indicated that GGT and TG levels were significantly correlated with PHTG levels (odds ratio [OR] =1.168, P<0.001; OR = 238.169, P<0.001, respectively).

The Predictive Value of Serum GGT for PHTG

We applied the ROC curve to evaluate whether the serum GGT level was a predictor of PHTG.

The Results indicated that the serum GGT level significantly predicted PHTG, with an area under the curve of 0.731 (95% confidence interval: 0.663–0.8000), a sensitivity of 61.70%, specificity of 75.90%, and cutoff value of 17.5 U/L (Figure 2).

Table 2 Correlation Analysis of Serum Liver Enzymes and Metabolic Indexes

	GGT		ALT		AST		ALP	
	r	P	r	P	r	P	r	P
Body mass index	0.279	<0.001	0.295	<0.001	−0.017	0.806	0.030	0.669
Waist circumference	0.475	<0.001	0.408	<0.001	0.087	0.220	0.136	0.054
Systolic blood pressure	0.223	0.001	0.213	0.002	0.090	0.203	0.167	0.017
Diastolic blood pressure	0.180	0.01	0.187	0.008	0.055	0.436	0.084	0.234
Serum uric acid	0.399	<0.001	0.312	<0.001	0.041	0.562	0.017	0.812
Fasting blood glucose	0.240	0.001	0.213	0.002	−0.012	0.869	0.104	0.142
Fasting insulin	0.106	0.132	0.186	0.008	−0.037	0.603	−0.122	0.084
HOMA-IR	0.152	0.031	0.398	<0.001	−0.036	0.608	−0.088	0.214
HOMA-β	−0.041	0.559	0.053	0.453	−0.034	0.630	−0.178	0.011
0-h total cholesterol	0.175	0.013	0.164	0.020	0.052	0.465	0.243	0.001
4-h total cholesterol	0.215	0.002	0.191	0.006	0.035	0.626	0.232	0.001
0-h triglyceride	0.353	<0.001	0.301	<0.001	0.055	0.433	0.194	0.006
4-h triglyceride	0.481	<0.001	0.315	<0.001	0.074	0.294	0.139	0.049
0-h HDL-C	−0.211	0.003	−0.165	0.019	0.125	0.076	−0.032	0.652
4-h HDL-C	−0.217	0.002	−0.179	0.011	0.072	0.312	−0.035	0.624
0-h LDL-C	0.254	<0.001	0.233	0.001	0.036	0.611	0.280	<0.001
4-h LDL-C	0.260	<0.001	0.224	0.001	0.013	0.855	0.260	<0.001
Non-HDL-C	0.274	<0.001	0.235	0.001	0.030	0.677	0.240	0.001
TRLRs	0.246	<0.001	0.164	0.020	−0.043	0.544	0.183	0.009
GGT	–	–	0.602	<0.001	0.254	<0.001	0.116	0.100
ALT	0.602	<0.001	–	–	0.53	<0.001	0.187	0.008
AST	0.254	<0.001	0.53	<0.001	–	–	0.203	0.004
ALP	0.116	0.100	0.187	0.008	0.203	0.004	–	–

Abbreviations: ALT, alanine aminotransferase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; GGT, gamma glutamyltransferase; HDL-C, high-density lipoprotein cholesterol; HOMA-β, homeostasis model assessment of β-cell function; HOMA-IR, homeostasis model assessment-estimated insulin resistance; LDL-C, low-density lipoprotein cholesterol; non-HDL-C, non-high-density lipoprotein-cholesterol; TRLRs, triglyceride-rich lipoprotein remnants.

Table 3 The Influencing Factors of PHTG Analyzed by Logistic Regression

Variable	Univariate Analysis		Multivariate Analysis	
	OR (95% CI)	P value	OR (95% CI)	P value
Sex	———	0.121	———	0.158
Age (years)	———	0.187	———	0.465
Body mass index (kg/m^2)	1.156 (1.057, 1.263)	0.001	———	0.375
Waist circumference (cm)	1.046 (1.015, 1.077)	0.003	———	0.377
Serum uric acid (μmol/L)	1.006 (1.002,1.009)	0.002	———	0.243
HOMA-IR	1.450 (1.110,1.894)	0.006	———	0.835
Fatty liver	2.301 (1.064, 4.979)	0.034	———	0.740
Total cholesterol (mmol/L)	1.425 (1.041, 1.952)	0.027	———	0.865
Triglyceride (mmol/L)	233.572 (52.493,1039.300)	<0. 001	238.169 (34.926,1624.141)	<0. 001
HDL-C (mmol/L)	0.128 (0.040, 0.404)	<0. 001	———	0.229
LDL-C (mmol/L)	2.006 (1.291, 3.117)	0.002	———	0.937
GGT (U/L)	1.126 (1.073,1.182)	<0. 001	1.168 (1.074, 1.271)	<0. 001
ALT (U/L)	1.056 (1.018, 1.097)	0.004	———	0.678
AST (U/L)	———	0.476	———	0.283
AST/ALT	0.389 (0.202, 0.751)	0.005	———	0.224
ALP (U/L)	———	0.100	———	0.592

Abbreviations: ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CI, confidence interval; GGT, gamma glutamyltransferase; HDL-C, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment-estimated insulin resistance; LDL-C, low-density lipoprotein cholesterol; OR, odds ratio; PHTG, postprandial hypertriglyceridemia.

Discussion

In this study, 202 volunteers with normal fasting TG levels were recruited for the OFTT. Our study found that GGT levels in the PHTG group were significantly higher than those in the PNTG group, and further analysis revealed that GGT correlated significantly with PHTG, serving as an independent predictor of PHTG. Our study indicates that even when fasting TG levels are normal, serum GGT levels increase in individuals with elevated TG levels after meals. Monitoring GGT levels can detect PHTG early, providing a basis for early diagnosis and treatment of ASCVD.

We found the fasting TG and 4-h TG levels after the OFTT in the PHTG group to be higher than those in the PNTG group, indicating that screening for PHTG is also necessary when the fasting TG level is in the normal reference range. Increased TG levels were found to be positively associated with increased all-cause mortality in patients with coronary heart disease (CHD) in a clinical study including 22 years of follow-up and analysis of mortality data from 15,355 patients with CHD. The study also found that, even among patients with TG levels of 1.2–1.7 mmol/L, there was a discernible increased risk of death in comparison to patients with lower TG levels.[29] As demonstrated by an earlier

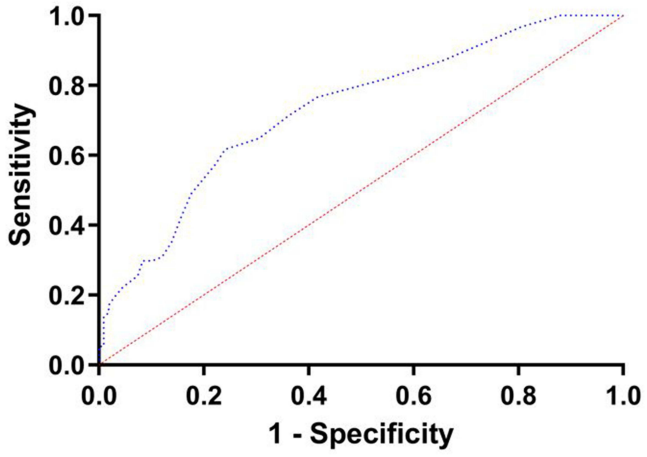

Figure 2 ROC analysis of GGT prediction of PHTG.
Abbreviations: GGT, gamma glutamyltransferase; PHTG, postprandial hypertriglyceridemia; ROC, receiver operating characteristic.

study conducted by our research group, individuals with PHTG have significantly poorer insulin sensitivity index and islet beta cell function than individuals with normal lipid tolerance. Additionally, the postprandial 4-h TG level is a distinct risk factor for insulin resistance and decreased islet beta cell function.[30] Since most people only fast for a few hours daily, detecting lipids in a non-fasting state relative to a fasting state may better reflect an individual's comprehensive metabolic status.[8,31] As indicated in a 2019 statement on PHTG,[28] when the fasting TG concentration ranges from 1–2 mmol/L, OFTT detection of PHTG should be considered for assessing PHTG and predicting metabolic risk.

Our study found that, after adjusting for major confounders, only GGT level was independently correlated with PHTG, whereas ALT, AST, and ALP levels were not significantly correlated with PHTG. Similarly, Wei et al[18] found that GGT and ferritin levels correlated positively with the risk of MetS and its components, such as overweight or obesity, HTG, hyperglycemia, and hypertension, in the Yi female population in China, indicating that GGT and ferritin levels may serve as predictive biomarkers for MetS. One study showed that serum GGT was significantly and positively correlated with MetS and its component, HTG, in Korean children and adolescents.[32] Contrary to our results, several studies have indicated that liver enzymes other than GGT are associated with HTG. A cross-sectional study involving adults in Beijing revealed that serum GGT, ALT, AST, and ALT levels were all predictors of MetS and its component, HTG, with GGT having the highest predictive value.[21] Zhao et al[22] found that MetS and its component, HTG, were closely associated with serum ALT levels in Beijing adolescents. Studies have shown that elevated levels of ALT, GGT, and ALP correlate positively with the prevalence of MetS and its component, HTG, and reduced HDL-C levels in the older population, while AST does not significantly correlate with MetS or lipid disorders.[23] A study involving Korean adults found that the risk of MetS and its component, HTG, increased with increasing ALT and AST levels, even when within the normal reference range.[24] The reasons for these differing results may be as follows: First, our study focused on PHTG (the early stage of HTG), while the aforementioned studies examined MetS and their component, HTG; second, the results may vary depending on the population studied. Our study further indicated that serum GGT was an independent predictor of PHTG diagnosis, with a cutoff value of 17.5 U/L, indicating that even when within the normal range, an increase in GGT level corresponded to an increased risk of PHTG.

However, the mechanism underlying the association between PHTG risk and serum GGT levels remains unknown. Possible reasons are as follows. First, serum GGT is significantly correlated with liver fat content[33] and is an independent predictor of nonalcoholic fatty liver disease (NAFLD).[34] Studies have indicated that liver fat content of less than 10% correlates positively with very low-density lipoprotein (VLDL)-TG secretion in the liver; specifically, the liver VLDL-TG secretion of individuals with high serum GGT increases, elevating serum TG levels.[35] In addition, NAFLD promotes liver fat accumulation and VLDL-TG synthesis and secretion by increasing liver TG synthesis and decreasing fatty acid decomposition, resulting in elevated serum TG levels.[36] GGT, a redox-related enzyme, is considered a significant indicator of oxidative stress because it helps to mitigate the adverse effects of oxidative stress by preserving the metabolism and homeostasis of cellular glutathione.[12] Therefore, oxidative stress upregulates intracellular GGT levels. The onset of hyperlipidemia is accompanied by oxidative stress. Studies have found that serum markers of oxidative stress increase in patients with hyperlipidemia.[37] Several animal experiments have shown that oxidative stress levels in hyperlipidemic rats are increased, and administering antioxidant therapy can improve lipid levels in rats.[38,39] Therefore, GGT may induce PHTG production via an oxidative stress mechanism.

To our knowledge, this is the first study to analyze serum liver enzyme expression levels and their correlation with PHTG in individuals with normal fasting TG levels and only postprandial TG elevation. However, this study had certain limitations. First, it used cross-sectional data, which could not infer a causal relationship between GGT and triglyceride levels. Second, owing to the small sample size, the grouping was not further classified by sex. Additionally, the recruited volunteers were of Han ethnicity from Hebei Province, with certain regional limitations, and their physical activity was not recorded, potentially affecting the accuracy of the statistical results. Further expansion of the sample size and prospective cohort studies are needed to analyze the effects of GGT on serum TG levels.

Conclusion

In Conclusion, by comparing the differences in serum liver enzyme expression levels between the PHTG and PNTG populations, we found that serum GGT was an independent predictor of PHTG. As GGT increases, the prevalence of PHTG gradually increases. Monitoring serum GGT levels can aid in the early detection of abnormal lipid metabolism and provide a new direction for the prevention, diagnosis, and treatment of ASCVD.

Abbreviations

ALP, alkaline phosphatase; ALT, alanine aminotransferase; ApoB, apolipoprotein B; ASCVD, atherosclerotic cardiovascular disease; AST, aspartate aminotransferase; BMI, body mass index; CHD, coronary heart disease; FBG, fasting blood glucose; FINS, fasting insulin; GGT, gamma glutamyltransferase; HDL-C, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment-estimated insulin resistance; HTG, hypertriglyceridemia; LDL-C, low-density lipoprotein cholesterol; MetS, metabolic syndrome; NAFLD, nonalcoholic fatty liver disease; OFTT, oral fat tolerance test; OGTT, oral glucose tolerance test; OR, odds ratio; PHTG, postprandial hypertriglyceridemia; PNTG, postprandial normal triglycerides; ROC, receiver operating characteristic; SBP, systolic blood pressure; SUA, serum uric acid; TC, total cholesterol; TG, triglyceride; TRLRs, triglyceride-rich lipoprotein residues; VLDL, very low-density lipoprotein; WC, waist circumference.

Data Sharing Statement

The data are available from the corresponding author upon reasonable requests.

Ethical Approval and Informed Consent

This study was approved by the Ethics Committee of Hebei General Hospital (approval number: 2018, No. 2). Informed consent forms were signed by each participant prior to their enrollment.

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Disclosure

The authors report no conflicts of interest in this work.

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