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ORIGINAL PAPER

Correlation between serum gamma glutamyl transferase with atherogenic index of plasma with angiographic severity in patients with coronary artery disease

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ABSTRACT

Introduction and aim. Coronary artery disease (CAD) is a leading cause of morbidity and mortality worldwide. Gamma-glutamyl transferase (GGT) has been found to be involved in the pathogenesis of CAD. The aim of the study was to study the correlation between serum GGT and the atherogenic index of plasma (AIP) with angiographic severity in patients with CAD.

Material and methods. This was an analytical cross-sectional study performed in 150 CAD patients in a tertiary-care teaching hospital in Puducherry, India. The patients were categorized as ST-elevated myocardial infarction (STEMI), non-ST-elevated MI (NSTEMI) and unstable angina. Routine biomarkers including troponin-I, AIP, GGT, and angiographic severity were calculated by applying a Gensini score (GS).

Results. The mean age of the study participants was 55.7±10.2 years, predominantly males. The GGT and GS was higher in STEMI group followed by NSTEMI and unstable angina groups ($p<0.001$ and 0.016, respectively). This indicates that GGT could be a potential biomarker for CAD, specifically in STEMI. AIP was shown to be statistically significant in unstable angina patients ($p=0.029$). GGT and GS showed a positive correlation with each other, and were statistically significant ($r=0.1685$, $p=0.0387$).

Conclusion. Elevated serum GGT levels were positively correlated with angiographic severity of CAD with stronger associations in patients who had STEMI.

Keywords. angiographic severity, atherogenic index of plasma, coronary artery disease, gensini score, serum gamma-glutamyl transferase

Introduction

Cardiovascular disease (CVD), such as ischemic heart disease and stroke are the major cause of death, accounting for 17.7 million fatalities.[1] According to the World Health Organization (WHO), India is responsible for one-fifth of these deaths globally, particularly

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among the younger population.[1] CVDs affected Indians a decade earlier than the rest of the world,[2] with highest rate of coronary artery disease (CAD) and considered as a primary cause of death and morbidity in India.[3] According to the Global Burden of Disease (GBD) report, India has a mortality from CVD of 272 per 100,000, higher than the global average of 235 per 100,00.[2] India has the greatest prevalence of acute coronary syndrome (ACS) and ST-elevation myocardial infarction at the moment (STEMI).[1,4]

Patients with CAD are at an increased risk of developing subsequent cardiac events and mortality. Traditional risk variables and prognostic risk models, on the other hand, are insufficient to account for the development of CAD. Thus, identifying novel prognostic indicators is critical for more aggressive secondary prevention in individuals with CAD.[5]

Inflammation is a major contributor to the development of atherosclerosis. Changes in the blood levels of specific inflammatory indicators can influence the development and progression of atherosclerosis, as well as on the risk of thrombotic consequences. Gamma-glutamyl transferase (GGT) has been linked to the development of CAD and its mortality.[6,7] It is the membrane-bound enzyme involved in glutathione catabolism (G-SH) and promotes the oxidation of low-density lipoprotein (LDL) and the generation of reactive oxygen species (ROS), that contribute to the atherosclerotic process.[8] Various literature implies that elevated serum GGT can be used as a predictive biomarker for variety of illnesses including conditions such as liver function, excessive alcohol intake, and oxidative stress.[6,9–11] Similarly, the atherogenic index of plasma (AIP) is a novel index composed of triglycerides and high-density lipoprotein (HDL) cholesterol that is used to quantify blood lipid levels and commonly used as optimal indicator of dyslipidemia and associated diseases especially CVD.[5]

Currently, serum GGT level is considered as the risk factor for CVD.[12] Yet, the prognostic usefulness of GGT levels in predicting CV and all-cause mortality in individuals with CAD is controversial.[9,11,13–15] Pooled studies, established an association between an elevated GGT level and an increased risk of cardiovascular and all-cause mortality in the general population.[16–18] Also, it can be considered as an early marker for atherosclerosis, and also as an independent biomarker for coronary artery calcification.[19-23] Even with the Framingham offspring study, was one of the first epidemiological studies to examine the relationship between GGT levels and CVD risk, resulted that GGT may be used to forecast metabolic and CV risks associated with the beginning of metabolic syndrome and acute CVD, as well as to determine mortality.[24]

Aim

With this background, in this study we want to determine the correlation between serum GGT and AIP with angiographic severity in patients with CAD.

Material and methods

Study setting and design

The study was conducted in the General Medicine and Cardiology outpatient departments (OPD) in a tertiary care teaching hospital in Puducherry, India. State-of-the-art equipment at this tertiary care facility empowers specialists to perform a wide range of cardiac management and treatments, including surgery. The present study was a hospital-based analytical cross-sectional study. Data collection was done for the period of January 2020 to April 2021.

Study population

The inclusion criteria for the study considered were adult patients over 18 years of age, admitted to the Department of Medicine and Cardiology, who had ACS, including STEMI, NSTEMI, and unstable angina diagnosed on a clinical basis involving relevant history, biochemical test, and electrocardiogram (ECG) recording and patients with chronic stable angina. Patients with a history of myocardial infarction (MI), coronary intervention, congestive heart failure (CHF), history of alcoholic liver disease, chronic obstructive pulmonary disease (COPD), recent alcohol intake (<3 weeks), respiratory failure, renal failure, on medications such as oral contraceptives, statin therapy, and antiepileptic drugs (phenytoin, phenobarbital, carbamazepine) were excluded from this study.

Sample size and sampling technique

Considering the prevalence of CAD to be 9.7%,[25] as the leading cause of disability in 2016, with 5% absolute precision and 95% as the confidence interval, the calculated sample size was 135. With an attrition rate, the final sample size was 148.5, rounded to the highest figure of 150 patients. A consecutive sampling technique was used to include all patients eligible for the study according to inclusion criteria until the desired sample size was achieved.

Data collection procedure

After obtaining informed consent, data was collected using a patient proforma. It includes demographic details, risk factors, comorbidities, anthropometry, and previous medical and clinical history. A trained postgraduate paid visit to the and collected data by a face-to-face interview. The confidentiality, anonymity, and privacy of the participants were guaranteed throughout the study.

Operational definitions

STEMI is defined as ST-elevation of ≥0.1m V in >1 limb leads or ≥0.2 mV in contiguous chest leads or left bundle branch block (LBBB) on presentation to the hospital. Those without ST elevations were diagnosed with UA or NSTEMI differentiated by the presence of cardiac enzymes.

NSTEMI is defined as those who have persistent or transient ST segment depression or T wave inversion, flat T waves, pseudo-normalization of T waves or no ECG changes at presentation with elevated cardiac enzymes however those without elevated cardiac enzymes will be defined as unstable angina.

Laboratory procedure

Peripheral blood was drawn in an EDTA containing tube and stored for biochemical experiments within 24 hours after admission. The glucose oxidase method was used to detect fasting blood glucose and post-prandial glucose. Blood lipid indexes, including triglycerides, total cholesterol, HDL, LDL, and serum GGT levels, were measured by the 902 automatic nano auto analyzer (Hitachi, Tokyo, Japan). AIP was calculated by using the formula,

$$AIP = log\ log\ (triglycerides\ /\ HDL)$$

Where AIP less than 0.11 is associated with a low risk of CVD; the values between 0.11 and 0.21 and higher than 0.21 are associated with intermediate and increased risks, respectively.[26]

Angiographic severity is calculated by applying the Gensini severity score (GS).[27] It is a measure of the severity of coronary stenosis (luminal narrowing) and its location. A severity coefficient was given for each segment as follows: 1-point for <25% obstruction, 2-points for 26–50% obstruction, 4-points for 51–75% obstruction, 8-points for 76–90% obstruction, 16-points for 91–99% obstruction, 32-points for complete occlusion (100%). The score is multiplied by the factor which depends on the functional significance of the area supplied by that segment (5 for the left main coronary artery (LMCA), 2.5 for the proximal segment of the left anterior descending artery (LAD) or circumflex artery, 1.5 for the middle segment of the LAD artery, 1 for the apical segment of the LAD artery or the middle or distal segment of the circumflex artery or the entire segment of the right coronary artery, 0.5 for other small branches of the coronary artery. Consequently, total digital GS were obtained that indicated the severity of CAD.

Ethical approval

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committees and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for the publication of this case report and accompanying images. This study was approved by the Institutional Ethics Committee (MGMCRI/Res/01/2019/33/IHEC/104).

Data analysis

Data were entered in MS EXCEL (Ver_2007) software and analysis was performed using Statistical Package for the Social Sciences (SPSS) software (version 24.0, IBM, Armonk, New York, USA). Categorical variables were measured in terms of frequencies and percentages. Continuous variables were expressed as mean and standard deviation (SD) or median with interquartile range (IQR). Data were analyzed according to the type of variables and the normal distribution between two groups. ANOVA was performed between three groups. Correlation was made between the GGT, and GS score. Statistical significance was considered as a $p<0.05$ for the analyzed data.

Results

Among 150 study participants, the mean age was 55.7±10.2 years and ranged from 29–76 years. The demographic and baseline laboratory parameters are presented in Table 1. Cardiac enzymes were taken and presented in Table 2. The mean AIP was 0.41±0.19 (95% confidence interval (CI): 0.380, 0.442) and the GS score were 47.75±44.68 (95% CI: 37.008, 52.365).

Table 1. Demographic and baseline laboratory parameters among study participants (n=150)*

Variables	n (%) or mean±SD	95% CI
Age (in years)	55.69±10.13	54.060–57.317
Gender		
Male	102 (68)	
Female	48 (32)	
Anthropometric indices		
WC (cm)	38.05±3.53	37.485–38.621
BMI (kg/m^2)	31.97±3.96	31.328–32.604
Blood glucose and lipid profile		
FBG (mg/dL)	109.95±7.50	108.747–111.16
PPBG (mg/dL)	165.56±21.55	162.097–169.029
Total cholesterol (mg/dL)	147.23±34.98	141.613–152.864
Triglycerides (mg/dL)	176.44±27.39	172.039–180.847
HDL-c (mg/dL)	28.70±7.21	27.542–29.861
LDL-c (mg/dL)	165.89±17.96	162.993–168.768
VLDL-c (mg/dL)	31.61±23.63	27.793–35.42

* WC – waist circumference, BMI – body mass index, FBG – fasting blood glucose, PPBG – post-prandial blood glucose, HDL-c – high density lipoprotein cholesterol, LDL-c – low-density lipoprotein cholesterol, VLDL-c – very low-density lipoprotein cholesterol, CI – Confidence interval, SD – standard deviation

Table 2. Cardiac biomarkers among the study participants (n=150)*

Variables	mean±SD	95% CI
CPK NAC (U/l)	430.32±257.6	188.402–326.790
CK MB (IU/l)	33±9.96	31.398–34.602
Troponin I (ng/mL)	258.58±243.48	201.909–285.066
GGT (U/L)	80.73±39.96	74.302–87.155
AIP	0.41±0.19	0.380–0.442
GS score	47.75±44.68	37.008–52.365

* AIP – atherogenic index of plasma, CPK NAC – creatine phosphokinase-N acetyl cysteine, CK MB – creatine kinase myocardial band, GGT – gamma glutamyl transferase, GS – Gensini severity score, CI – confidence interval, SD – standard deviation

Among study participants, 82 (54.7%) patients had STEMI, 36 patients (24%) had NSTEMI, and 32 (21.3%) had unstable angina. The gender-wise distribution of CAD was assessed and found a male preponderance, where 36.7% of the male patients had STEMI, 18% and 13.3% of the patients had NSTEMI and unstable angina, respectively. CADs were compared with the baseline laboratory parameters and are presented in Table 3. BMI, triglycerides, HDL-c, and LDL-c were statistically significant for the development of CAD. The association between CAD types with cardiac biomarkers was done and found that CK-NAC, CK-MB, troponin I and GGT were statistically significant for the development of CAD. Furthermore, the AIP and GS score were also found to be statistically significant for the development of CAD (Table 4).

Table 3. Association of CAD types with anthropometric and laboratory parameters among study participants (n=150)*

Variables	STEMI Mean±SD	NSTEMI Mean±SD	Unstable angina Mean±SD	F ratio, p
Age	54.83±10.73	54.97±8.33	58.72±10.12	1.841, 0.162
WC (cm)	38.18±3.67	37.89±3.14	37.90±3.70	0.120, 0.887
BMI (kg/m^2)	32±3.93	30.72±4.13	33.21±3.47	3.858, 0.023
Laboratory parameters				
FBG (mg/dL)	110.24±7.99	109.73±7.67	109.47±6.06	0.143, 0.867
PPBG (mg/dL)	166.04±22.44	166.24±22.59	163.53±18.25	0.179, 0.836
CHO (mg/dL)	148.72±38	150.18±29.68	140.03±32.47	0.883, 0.416
TGL (mg/dL)	176.89±27.58	168.38±21.55	184.62±30.9	**3.129, 0.047**
HDL-c (mg/dL)	27.31±6.83	34.32±6.81	25.75±4.87	**19.203, <0.001**
LDL-c (mg/dL)	167.89±16.22	159.45±22.14	168.15±15.43	**3.229, 0.042**
VLDL-c (mg/dL)	30.93±25.17	35.76±21.38	30.81±22.61	0.201, 0.818

* STEMI – ST elevated myocardial infarction, NSTEMI – non-ST elevated myocardial infarction, WC – waist circumference, BMI – body mass index, FBG – fasting blood glucose, PPBG – post-prandial blood glucose, CHO – total cholesterol, TGL – triglycerides, HDL-c – high density lipoprotein cholesterol, LDL-c – low-density lipoprotein cholesterol, VLDL-c – very low-density lipoprotein cholesterol, SD – standard deviation, ANOVA was performed

Table 4. Association of types of CAD with cardiac biomarkers among the study participants (n=150)*

Variables	STEMI Mean±SD	NSTEMI Mean±SD	Unstable angina Mean±SD	F ratio, p
CPK NAC (U/l)	402.70±325.13	607.65±253.51	89.25±45.94	3.57, 0.030
CK MB (IU/l)	53.69±48.04	82.08±76.31	92.19±61.07	4.41, 0.014
Troponin I (ng/mL)	181.68±79.17	63.48±36.90	8.49±4.87	109.52, <0.001
GGT (U/L)	139.98±26.85	64.40±12.31	37.09±13.36	290.45, <0.001
AIP	0.38±0.18	0.411±0.18	0.49±0.28	3.610, 0.029
GS score	51.92±45.98	51.31±46.75	43.07±23.75	4.225, 0.016

* AIP – atherogenic index of plasma, GS – Gensini severity score, STEMI – ST elevated myocardial infarction, NSTEMI – non-ST elevated myocardial infarction, CPK NAC – creatine phosphokinase-N acetyl cysteine, CK MB – creatine kinase myocardial band, GGT – gamma glutamyl transferase, SD – standard deviation, ANOVA was performed

The mean GS score was performed for patients with CAD types and resulted that in patients with STEMI. The mean score was 51.92±45.98, for patients with NSTEMI and unstable angina was 51.31±46.75 and 43.07±23.75, respectively. They were statistically significant (F=4.225, p= 0.016) among the three groups, implying that the GS score was varied between the groups. In STEMI group, it was high and followed by in NSTEMI and unstable angina groups (Fig. 1).

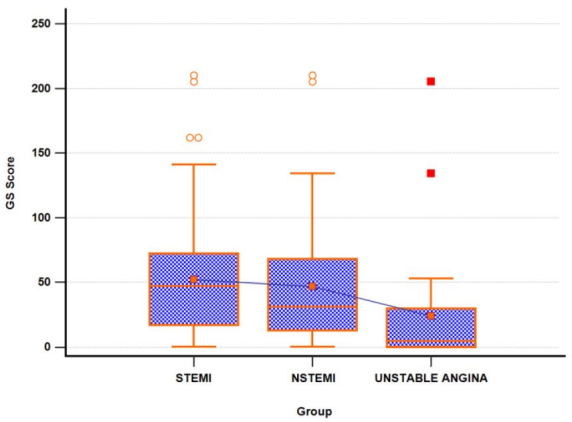

Fig. 1. Comparison of angiographic severity using GS score between three groups (in STEMI group, the GS score was high, followed by NSTEMI and unstable angina groups)

The GGT was compared with the GS Score to determine the correlation between the GGT and the severity of CAD with GS score and scatterplot was made (Fig. 2). The present study observed that GGT and GS score had positive correlation with each other. Therefore, when the GGT increases, the GS Score also increased. This correlation was statistically significant (r=0.168, p=0.0387).

Discussion

In this present study, the correlation between serum GGT and angiographic severity in patients with CAD

was performed in 150 patients and found that GGT was positively correlated with the GS score, implying that increasing GGT will subsequently increase the angiographic severity among the patients with CAD.

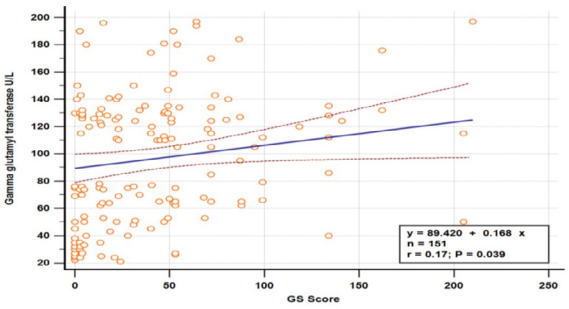

Fig. 2. Scatter plot for comparison of the GGT and GS score (GGT and GS score were positively correlated with each other)

The mean age of patients with STEMI at the time of presentation was 54.83±10.73 years in this present study, which is consistent with the findings of the CREATE registry, and another study from the southern Indian state of Tamil Nadu.[28,29] Male preponderance was detected in all age groups of patients with STEMI, and the sex ratios observed in both the younger and older age groups in our study were comparable to the study conducted by Holay et al.[30]

GGT is a serum and cell surface enzyme that contributes to G-SH equilibrium, a vital component of the body's defense against free radicals. Once GGT hydrolyzes G-SH extracellularly, glycine and cysteine are generated.[31,32] The synthesis of G-SH, the main antioxidant involved in the defense against oxidative stressors, is facilitated by the amino acids that are subsequently transported into the cell.[21] On the other hand, the extracellular cysteine-glycine combination stops Fe^{3+} from being reduced to redox active Fe^{2+}.[31–33] This process results in the formation of peroxide, oxidized LDL, and free oxygen radicals. Thus, more oxidized LDL receptors are present on the cell surface as a result of increased oxidative stress, which facilitates the entry of LDL/GGT complexes into the plaque.[24,31,33] The development and progression of atheromatous plaque in the arteries are caused by these processes.[11,21,24,31,32] The idea that GGT directly contributes to the development of atherosclerosis is supported by research results that show GGT activity within the atherosclerotic plaque.[31]

According to the findings of Kittleson et al., increased GGT activity, even when it is within the normal range, is related to increased oxidative stress.[34] In our study, we found that patients with CAD presented with higher GGT. Many studies established that GGT is associated with cardiovascular disease in the form of atherosclerosis development and its degree of CAD and consistent with our study findings.[35,36] Among them, STEMI patients had higher levels of GGT, compared to NSTEMI and unstable angina, which was similar to the study by Breitling et al.,[14] Kunutsor et al.[37] Various studies demonstrated that patients with STEMI have elevated GGT and considered as an independent predictor of premature mortality.[13,17,35,36,38] Thus, from all these studies, it appears that GGT levels are associated with not only the development of atherosclerosis, but also with the development of CAD where the results of the current investigation were also consistent with the findings of the previous study.

In our study, we found that serum GGT, HDL-c, LDL-c and triglycerides were statistically significant and associated with CAD. Our findings were consistent with the study by Aksakal et al., where the serum GGT, diabetes, HDL-c, eGFR, and ejection fraction were all independent predictors of a high SYNTAX score.[15] Also, study done by Mao et al., among the Chinese population, showed that serum GGT and CAD had a favorable relationship indicating that GGT is a novel biomarker for CAD.[39]

From our study, we found a statistically significant correlation between the serum GGT and atherogenic severity in CAD patients. When comparing patients with <50% blockage in their coronary arteries with healthy controls and patients with <50% obstruction, the level of GGT in patients with obstruction was higher.35 Similarly, study done by Sheikh et al. showed that association was present between serum GGT and CAD.[12] Also, the study showed that every 10 unit rise in serum GGT was found to be robust predictor of existence of the early CAD.[12]

In a prospective cohort study, Hartopo et al., evaluated the link between AIP value and significant adverse cardiovascular events in patients with acute MI who were admitted to critical care throughout their stay, which was in line with the present study.[40] In the study it has found that a low AIP value, as opposed to a high AIP value, was an independent predictor of all-cause death in patients with acute MI who were receiving intensive hospitalization.[40]

The major strength of this study is that we assessed serum GGT as the novel biomarker along with the evidence of AIP and GS score. Additionally, the detailed categorization of cardiovascular conditions including STEMI, NSTEMI, and unstable angina enables a nuanced understanding of each subgroup. Furthermore, the study uses a broad range of clinical and biochemical parameters, including CPKMB, CPKNAC, troponin I, and GGT, facilitating a detailed investigation of cardiac health.

Every study always presents with the limitations. The main limitation of the study includes the smaller sample size. Being a cross-sectional study, the temporality of the association could not be assessed. Also, this was a single-centric hospital study, in which the results cannot be extrapolated to the general population. Lastly, potential confounding factors, such as medication use

and lifestyle factors, were not extensively controlled or discussed, which could affect the study's results.

Conclusion

Therefore, in our study, an elevated serum GGT is correlated with the angiographic severity of CAD assessed by the GS score. Stronger associations were observed in patients with STEMI. Similarly, we found a positive correlation between the serum GGT and GS score and significant statistically.

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Declarations

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Author contributions

Conceptualization, V.R. and A.P.; Methodology, V.R. and A.P.; Software, J.J.F.M.; Validation, A.P. and S.M.; Formal Analysis, J.J.F.M.; Investigation, V.R.; Resources, V.R.; Data Curation, J.J.F.M.; Writing – Original Draft Preparation, V.R.; Writing – Review & Editing, A.R. and S.M.; Visualization, S.M.; Supervision, A.P.; Project Administration, V.R.

Conflicts of interest

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Data availability

All data generated during or analysed during this study are included in this published article.

Ethics approval

This study was approved by the Institutional Ethical Committee (MGMCRI/Res/01/2019/33/IHEC/104).

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