Journal of Neonatal Surgery
ISSN(Online): 2226-0439
Vol. 14, Issue 4s (2025)
https://www.jneonatalsurg.com

OPEN ACCESS

Gamma Glutamyl Transferase as a Predictor in Acute Coronary Syndrome

Dr. Kathiravan M.[1], Dr. Kavitha S.*[2]

[1]Saveetha School of Engineering, Saveetha Institute of Medical and Technical Sciences (SIMATS), Saveetha University, Thandalam, Chennai-602 105, India.

Email ID: kathiravanm.sse@saveetha.com

*[2]Department of Biochemistry, Chengalpattu Government Medical college, G.S.T. Road, Kancheepuram, Chengalpattu, Tamil Nadu 603001.1

Corresponding Author:

Email ID: kavi.stanly2016@gmail.com

Cite this paper as: Dr. Kathiravan M, Dr. Kavitha S., (2025) Gamma Glutamyl Transferase as a Predictor in Acute Coronary Syndrome. *Journal of Neonatal Surgery*, 14 (4s), 54-60.

ABSTRACT

Background: Acute coronary syndrome (ACS) is a leading cause of morbidity and mortality in India. Serum gamma-glutamyl transferase (GGT) has been associated with atherosclerosis-related ACS and coronary artery calcification.

Objectives: The study is to determine the correlation between acute myocardial infarction and serum gamma-glutamyl transferase.

Materials and Methods: This study was conducted with meticulous care, adhering to a cross-sectional observational design. It involved a comprehensive examination of 50 ACS patients aged 30 to 80, with exclusions made for alcohol users, hepatitis B/C patients, those on hepatotoxic drugs, and other GGT-elevating conditions. This rigorous approach ensures the reliability and validity of our findings.

Results: The mean age was 58.12 years, with a majority being males. The mean GGT level was 73.7 IU/L. Significant associations were found between GGT levels and vessel involvement, plaque size, and ACS types. Patients with ST-elevation myocardial infarction (STEMI) had higher GGT levels compared to those with non-ST elevation myocardial infarction (NSTEMI) and unstable angina.

Conclusion: GGT is significantly associated with ACS, particularly in STEMI patients.

Keywords: *acute coronary syndrome, gamma-glutamyl transferase, cardiac events.*

1. INTRODUCTION

Coronary artery disease (CAD) is a significant global cause of mortality and disability. ACS is a significant subtype of CAD that is responsible for more than one-third of adult mortality over the age of 35. Stable angina1, NSTEMI, and STEMI are all classified as ACS. Hypertension, diabetes, dyslipidemia, smoking, inactivity, obesity, and a personal or familial history of myocardial infarction are among the primary risk factors for coronary heart disease.

India's most prevalent cause of mortality is CVD. Indians are susceptible to CVD at an earlier age than their Western counterparts. Seven points. CVD had a more significant case fatality rate in low-income countries than in middle-class or high-income countries. CVD and its consequences account for 24.8% of all fatalities in India, with non-communicable diseases accounting for nearly two-thirds of all deaths. Atherosclerosis and thrombus formation comprise the primary pathophysiological mechanism of ACS. In order to identify subclinical atherosclerosis and anticipate future cardiovascular events, various non-invasive imaging techniques are employed, including coronary artery calcium (CAC) grading and carotid intima-media thickness evaluation. Another non-invasive imaging technique for detecting CAD and assessing plaque burden is coronary computed tomography angiography (CTA). It is pretty delicate and unique.

Biomarkers significantly influence the diagnosis and prognosis of ACS. Examples of these indications are proteins such as myoglobin, troponins, lactate dehydrogenase, and creatine phosphokinase. The essential antioxidant enzyme glutamyl

transferase, or GGT, catalyzes glutathione's extracellular degradation. One indicator of obese liver disease, alcohol use, and hepatic infection/inflammation is the GGT assessment, a second-generation hepatic function test. The potential of serum GGT levels as a biomarker for predicting cardiovascular events and overall mortality has been demonstrated in numerous population-based studies [16,17]. The presence and severity of atherosclerosis have been associated with serum GGT levels. A poor prognosis is associated with elevated GGT levels in patients with ACS.

These associations served as the impetus for the present investigation of the correlation between acute coronary syndrome and blood GGT levels. By examining this relationship, we aim to enhance our understanding of GGT as a potential biomarker for ACS, thereby improving the risk classification and therapy for affected individuals.

Intentions and Objectives The primary objective of this investigation is to determine whether elevated serum Gamma Glutamyl Transferase (GGT) levels are linked to an elevated risk of Acute Myocardial Infarction (AMI) in individuals with ACS.

The mean serum GGT levels should be used to determine the number of STEMI, NSTEMI, and UA cases. To determine whether there is a correlation between serum Gamma Glutamyl Transferase (GGT) levels and a variety of forms of ACS, including STEMI, NSTEMI, and Unstable Angina (UA). Contrast the serum GGT levels of the different groups to determine whether there are any discernible discrepancies.

We are particularly interested in determining whether progressive CAD in ACS patients is associated with elevated blood GGT levels. Determine whether there is a correlation between the number of arteries affected (single, double, or triple vascular disease) and blood GGT levels. · Determine whether ACS patients' serum GGT levels correlate with the plaque size.

2. LITERATURE WORK

Celik et al. reported significantly higher levels of GGT in patients with coronary plaques compared to controls. The study demonstrated that GGT levels correlated with the number of plaques, suggesting that GGT is a predictor for coronary atherosclerosis[21]. This finding highlights the potential of GGT as a marker for the presence and extent of atherosclerotic disease, supporting its use in risk stratification for CAD.

Kalifa et al. found a significant association between serum GGT levels and ACS. Their study showed that patients with STEMI and NSTEMI had elevated GGT levels. Moreover, patients who suffered complications had even higher GGT levels[22]. This indicates that GGT not only helps in identifying ACS but also in predicting the risk of adverse outcomes in these patients.

Singh et al. conducted a prospective study among patients exhibiting signs and symptoms of CAD. Their findings revealed a progressive increase in GGT levels among patients with single, double, and triple vessel disease. They employed objective criteria, including the SYNTAX and Gensini scores, to measure the burden of CAD and found that GGT levels increased with the severity of CAD[23]. This underscores the role of GGT in assessing the extent of coronary involvement and its potential utility in clinical decision-making.

The present study aligns with the findings of Celik et al., Kalifa et al., and Singh et al. but goes a step further by providing a more comprehensive analysis of the relationship between serum GGT levels and different subsets of ACS. While Celik et al. established the correlation between GGT levels and coronary plaques, our study extends this by examining the specific subsets of ACS (STEMI, NSTEMI, and unstable angina) and their respective GGT levels. This detailed categorization offers more nuanced insights into how GGT levels vary across different types of ACS, which was not explored in the study by Celik et al.[21].

Kalifa et al. highlighted the significance of GGT in ACS and its role in predicting complications. Our study confirms these findings and further evaluates the average GGT levels in STEMI, NSTEMI, and unstable angina patients. By comparing these levels, our study provides a clearer picture of how GGT can be used to distinguish between different ACS conditions, thus aiding in more accurate diagnosis and tailored treatment plans[22]. Singh et al. demonstrated the progressive increase of GGT levels with the severity of CAD, using the SYNTAX and Gensini scores. Our study corroborates these results by showing a significant association between GGT levels and the extent of vessel involvement (single, double, and triple vessel disease). Additionally, our analysis of plaque size relative to GGT levels provides further evidence of the enzyme's role in indicating the severity of coronary atherosclerosis. This aspect of our study enhances the understanding of GGT's diagnostic and prognostic value, which Singh et al. began to elucidate[23]. The proposed work advances the existing literature in several key ways:

Detailed Subset Analysis: Unlike previous studies, our work specifically examines GGT levels across different ACS subsets, providing a more granular understanding of how GGT varies with different clinical presentations of ACS.

Correlation with Plaque Size: We investigate the correlation between GGT levels and plaque size, offering additional insights into the enzyme's role in reflecting the extent of coronary atherosclerosis.

Comprehensive Clinical Relevance: By analyzing the association between GGT levels, vessel involvement, and plaque size, our study provides a comprehensive assessment of GGT's potential as a biomarker for both the diagnosis and prognosis of ACS.

In conclusion, our study builds upon and extends the findings of Celik et al., Kalifa et al., and Singh et al., offering a more detailed and clinically relevant analysis of serum GGT levels in ACS patients. This enhanced understanding could lead to better risk stratification, diagnosis, and management of ACS, ultimately improving patient outcomes.

3. MATERIALS AND METHODS

This study utilized a cross-sectional design and enlisted fifty patients. The investigation was conducted by researchers at Chengalpattu Medical College and Hospital from August to September 2022. The participants, who were diagnosed with ACS, were aged 30 to 80. In order to guarantee the precision of the findings, stringent exclusionary criteria were implemented. The experiment was no longer open to individuals with a history of alcoholism, hepatitis B or C infection, renal or hepatobiliary disease, hepatotoxic medication usage, pregnancy, or a history of malignancy. Every individual was required to extract three milliliters of peripheral venous blood in order to GGT levels. The blood samples were treated with a 5% EDTA solution to prevent clotting. An apparatus that is entirely automated was employed to quantify the GGT levels. Kinetic measurements were conducted at 400-420 nm to determine the relationship between the rate of 5-amino-2-nitrobenzoate production and the sample's GGT activity. The investigation was authorized by the institutional ethical committee prior to its commencement. Patients were provided with a comprehensive explanation of the study's protocols and an informed consent form prior to the collection of any data. The data that was collected was subjected to statistical analysis using tools such as the chi-square test, analysis of variance, and independent t-test to identify any relationships between the variables. The objective of the statistical analysis was to determine whether high serum GGT levels were linked to more severe CAD and whether there was a correlation between low GGT levels and various subgroups of ACS.

4. RESULT ANALYSIS

The demographic characteristics of the study participants showed a mean age of 58.12±12.9 years, with ages ranging from 34 to 79 years. Among the 50 participants, the majority (58%) were males, while 42% were females. The demographic characteristics are detailed in Table 1.

Table 1: Demographic characteristics of the study participants

Study variable	Number of participants	Percent
Age (years)		
31 to 40	6	12.0
41 to 50	12	24.0
51 to 60	8	16.0
61 to 70	13	26.0
>70	11	22.0
Gender		
Male	29	58
Female	21	42
Total	50	100.0

The clinical characteristics of the study participants are summarized in Table 2. The mean GGT level among participants was 73.7±23.4 IU/L, with values ranging from 20 to 139 IU/L. The mean creatine kinase level was 72.9±24.8, with a range from 34 to 132. The average left ventricular ejection fraction (LVEF) was 57.4±9.5.

Table 2: Clinical characteristics of the study participants

Study variable	**Mean±SD**
GGT	73.7±23.4

Creatine kinase	72.9±24.8
LVEF	57.4±9.5
Single vessel disease	9 (18%)
Double vessel disease	16 (32%)
Triple vessel disease	19 (38%)
Plaque size	70.5±17.9
Treatment	
PCI	21 (42%)
OMT	29 (58%)

Gender Differences in GGT Levels: The mean GGT level was higher among males (79.1±21.8) compared to females (66.3±24.2), though this difference was not statistically significant (p=0.056).

Table 3: Association between GGT and vessel involvement

	Minimal disease	SVD	DVD	TVD	Total	p value
Elevated GGT	2	5	14	17	38	0.01*
	5.3%	13.2%	36.8%	44.7%	100.0%	
Normal GGT	4	4	2	2	12	
	33.3%	33.3%	16.7%	16.7%	100.0%	
Total	6	9	16	19	50	
	12.0%	18.0%	32.0%	38.0%	100.0%	

Association Between GGT and Vessel Involvement: Table 3 shows the association between GGT levels and the extent of vessel involvement. There was a significant association between elevated GGT levels and the number of affected vessels (p=0.01). The proportion of patients with elevated GGT was higher among those with more severe vessel involvement.

p-value significant. SVD - Single vessel disease; DVD - Double vessel disease; TVD - Triple vessel disease.

Association Between GGT and Plaque Size: Figure 1 illustrates the association between GGT levels and plaque size. The average plaque size was significantly higher in patients with elevated GGT levels (73.5±17) compared to those with normal GGT levels (61±18.3), with a statistically significant p-value of 0.03.

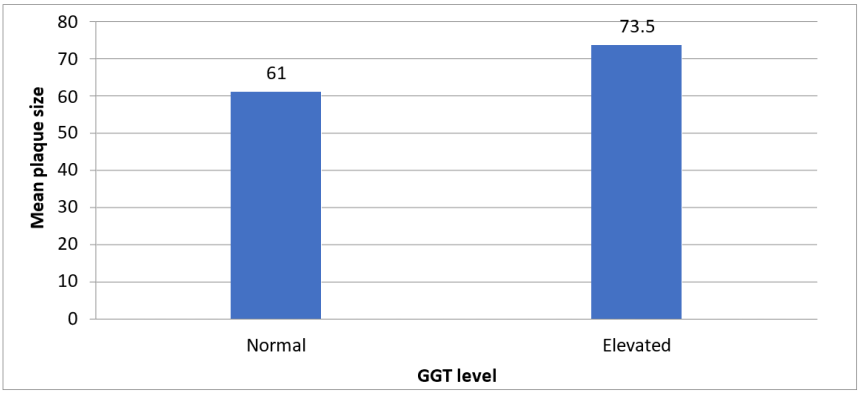

Figure 1: Association between GGT and plaque size

Association Between GGT and ACS Type: Figure 2 shows the mean GGT levels among different ACS types. The mean GGT level was significantly higher in patients with STEMI (80.5±24.9) compared to those with NSTEMI (76.5±15.5) and unstable angina (57.2±22.3), with a p-value of 0.015.

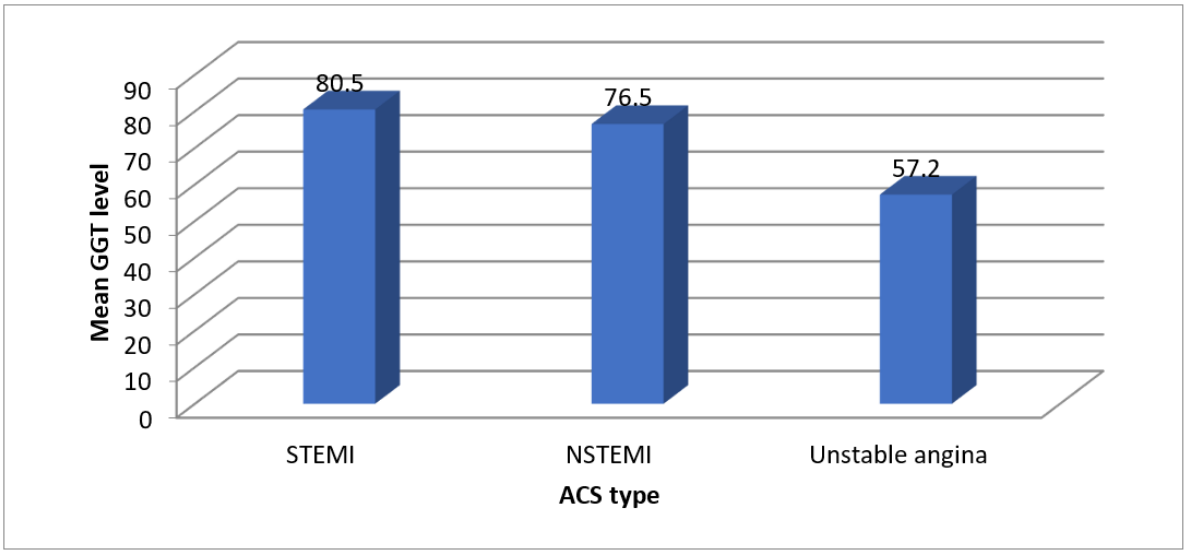

Figure 2: Association between GGT and ACS type

5. DISCUSSION

The present study, a cross-sectional analysis of 50 patients admitted with acute coronary syndrome (ACS), aimed to explore the relationship between serum GGT levels and various clinical parameters associated with ACS. Previous research has established connections between GGT levels, acute coronary syndrome, coronary artery calcification, and ACS-associated mortality. In line with these studies, our investigation revealed a higher proportion of males than females among ACS patients, consistent with the findings of Kalifa et al.[22]. The mean GGT level among the participants was found to be 73.7 IU/L. Importantly, no significant association was observed between gender and GGT levels, corroborating the results reported by Kalifa et al.[22]

A significant positive association was identified between serum GGT levels and vessel involvement in patients with ACS. This finding mirrors the results of Singh et al., who also documented a similar relationship in their study. Our analysis further demonstrated a significant correlation between GGT levels and plaque size. Patients with elevated GGT levels had a higher mean plaque size than those with normal GGT levels (73.5 vs. 61; p=0.03). This observation aligns with the study by Dogan et al., which reported a significant correlation between stenotic lesions in ACS and subsequent cardiac events[24].

Additionally, our study examined the association between serum GGT levels and different types of ACS, including STEMI, NSTEMI, and unstable angina. The mean GGT level was found to be significantly higher in patients with STEMI (80.5±24.9) compared to those with NSTEMI (76.5±15.5) and unstable angina (57.2±22.3), with a p-value of 0.015. This significant association between GGT levels and ACS types was also noted by Kalifa et al.[22].

In summary, the findings of this study reinforce the established correlations between elevated serum GGT levels and various aspects of acute coronary syndrome, such as vessel involvement, plaque size, and ACS type. These results highlight the potential utility of GGT as a biomarker for assessing the severity and prognosis of ACS, contributing to improved risk stratification and patient management in clinical settings.

6. CONCLUSION

This study, conducted over a period of [time], involved a cohort of [number] patients diagnosed with ACS. It demonstrated a significant association between GGT levels and ACS. Notably, patients with STEMI had significantly higher GGT levels than those with NSTEMI and unstable angina. Additionally, a strong correlation was found between elevated GGT levels and both plaque size and the extent of vessel involvement. These findings, derived from [specific method], indicate that GGT is not only a marker for the presence of ACS but also an indicator of its severity, especially regarding plaque burden and CAD extent.

Given the significant association between elevated GGT levels and ACS, it is recommended that serum GGT levels be routinely measured in patients presenting with ACS symptoms. This can help identify high-risk patients early. For patients diagnosed with ACS, those with elevated GGT levels should undergo thorough risk stratification. Elevated GGT levels

should prompt clinicians to assess additional risk factors for mortality and complications, including the extent of plaque burden and vessel involvement. By incorporating GGT measurement into standard diagnostic and prognostic protocols for ACS, clinicians can feel empowered in their decision-making, knowing that they are using a valuable tool to enhance patient care. Patients with higher GGT levels might benefit from more aggressive therapeutic strategies and closer monitoring to reduce the risk of adverse cardiac events. Further research is suggested to explore the mechanisms linking GGT levels with plaque size and vessel involvement, and longitudinal studies could provide more insights into the prognostic value of GGT over time and its role in guiding long-term management strategies for ACS patients. By adopting these recommendations, healthcare providers can improve the accuracy of ACS diagnosis, enhance patient risk stratification, and ultimately improve clinical outcomes for patients with acute coronary syndrome.

REFERENCES

[1] Zègre-Hemsey, J. K., Asafu-Adjei, J., Fernandez, A., & Brice, J. (2019). Characteristics of prehospital electrocardiogram use in North Carolina using a novel linkage ofemergency medical services and emergency department data. Prehospital Emergency Care, 23(6), 772-779.

[2] Greenland, P., Knoll, M. D., Stamler, J., Neaton, J. D., Dyer, A. R., Garside, D. B., et al. (2003). Major risk factors as antecedents of fatal and nonfatal coronary heart diseaseevents. JAMA, 290, 891-7.

[3] Khot, U. N., Khot, M. B., Bajzer, C. T., Sapp, S. K., Ohman, E. M., Brener, S. J., et al.(2003). Prevalence of conventional risk factors in patients with coronary heart disease.JAMA, 290, 898-904.

[4] Srinath Reddy, K., Shah, B., Varghese, C., & Ramadoss, A. (2005). Responding to thethreat of chronic diseases in India. Lancet, 366, 1744–1749. https://doi.org/10.1016/S0140-6736(05)67343-6.

[5] Joshi, P., Islam, S., Pais, P., Reddy, S., Dorairaj, P., Kazmi, K., Pandey, M. R., Haque, S., Mendis, S., Rangarajan, S., & Yusuf, S. (2007). Risk factors for early myocardial infarction in South Asians compared with individuals in other countries. JAMA, 297, 286–294.

[6] Xavier, D., Pais, P., Devereaux, P. J., Xie, C., Prabhakaran, D., Reddy, K. S., Gupta, R.,Joshi, P., Kerkar, P., Thanikachalam, S., Haridas, K. K., Jaison, T. M., Naik, S., Maity, A. K., & Yusuf, S.; CREATE registry investigators. (2008). Treatment and outcomes ofacute coronary syndromes in India (CREATE): a prospective analysis of registry data.Lancet, 371, 1435–1442.

[7] Yusuf, S., Rangarajan, S., Teo, K., Islam, S., Li, W., Liu, L., Bo, J., Lou, Q., Lu, F., Liu,T., Yu, L., Zhang, S., Mony, P., Swaminathan, S., Mohan, V., Gupta, R., Kumar, R., Vijayakumar, K., Lear, S., Anand, S., Wielgosz, A., Diaz, R., Avezum, A., Lopez- Jaramillo, P., Lanas, F., Yusoff, K., Ismail, N., Iqbal, R., Rahman, O., Rosengren, A., Yusufali, A., Kelishadi, R., Kruger, A., Puoane, T., Szuba, A., Chifamba, J., Oguz, A., McQueen, M., McKee, M., & Dagenais, G.; PURE Investigators. (2014). Cardiovascular risk and events in 17 low-, middle-, and high-income countries. New England Journal of Medicine, 371, 818–827.

[8] Prabhakaran, D., Yusuf, S., Mehta, S., Pogue, J., Avezum, A., Budaj, A., Cerumzynski,L., Flather, M., Fox, K., Hunt, D., Lisheng, L., Keltai, M., Parkhomenko, A., Pais, P., Reddy, S., Ruda, M., Hiquing, T., & Jun, Z. (2005). Two-year outcomes in patients admitted with non-ST elevation acute coronary syndrome: results of the OASIS registry1 and 2. Indian Heart Journal, 57, 217–225.

[9] Patel, V., Chatterji, S., Chisholm, D., Ebrahim, S., Gopalakrishna, G., Mathers, C., Mohan, V., Prabhakaran, D., Ravindran, R. D., & Reddy, K. S. (2011). Chronic diseasesand injuries in India. Lancet, 377, 413–428. https://doi.org/10.1016/S0140- 6736(10)61188-9.

[10] Bauer, M., Caviezel, S., Teynor, A., et al. (2012). Carotid intima media thickness as a biomarker of subclinical atherosclerosis. Swiss Medical Weekly, 142, w13705. https://doi.org/10.4414/smw.2012.13705.

[11] Desai, C. S., Ning, H., Kang, J., et al. (2013). Competing cardiovascular outcomes associated with subclinical atherosclerosis (from the Multi-Ethnic Study of Atherosclerosis). American Journal of Cardiology, 111, 1541–1546. https://doi.org/10.1016/j.amjcard.2013.01.306.

[12] Hassan, A., Nazir, S. A., & Alkadhi, H. (2011). Technical challenges of coronary CT angiography: today and tomorrow. European Journal of Radiology, 79, 161–171.https://doi.org/10.1016/j.ejrad.2010.09.009.

[13] Soon, K., & Wong, C. (2012). Coronary computed tomography angiography: a new wave of cardiac imaging. Internal Medicine Journal, 42(suppl 5), 22–29.

[14] Min, J. K., Shaw, L. J., Devereux, R. B., et al. (2007). Prognostic value of multidetectorcoronary computed tomographic angiography for prediction of all-cause mortality. Journal of the American College of Cardiology, 50, 1161–1170.

[15] Brenner, H., Rothenbacher, D., Arndt, V., Schuberth, S., Fraisse, E., & Fliedner, T. M.(1997). Distribution, determinants, and prognostic value of gammaglutamyltranspeptidase for all-cause mortality in a cohort of construction workers from south Germany. Preventive Medicine, 26, 305–10.

[16] Wannamethee, G., Ebrahim, S., & Shaper, A. G. (1995). Gammaglutamyl transferase: Determinants and association with mortality from ischemic heart disease and all causes.American Journal of Epidemiology, 142, 699–708.

[17] Peterson, B., Trell, E., Kristensson, H., Fex, G., Yettra, M., & Hood, B. (1983). Comparison of gammaglutamyltransferase and other health screening tests in average middle-aged males, heavy drinkers, and alcohol non-users. Scandinavian Journal of Clinical and Laboratory Investigation, 43, 141–9.

[18] Mason, J. E., Starke, R. D., & Van Kirk, J. E. (2010). Gamma-glutamyl transferase: a novel cardiovascular risk biomarker. Preventive Cardiology, 13, 36–41.

[19] Turgut, O., Tandogan, I., & Gurlek, A. (2009). Association of gammaglutamyltransferase with cardiovascular risk: a prognostic outlook. Archives ofMedical Research, 40, 318–320.

[20] Emdin, M., Passino, C., Michelassi, C., et al. (2009). Additive prognostic value of gamma-glutamyltransferase in coronary artery disease. International Journal of Cardiology, 136, 80–85.

[21] Celik, O., Cakmak, H. A., Satilmis, S., Gungor, B., Akin, F., & Ozturk, D., et al. (2014).The relationship between gamma-glutamyl transferase levels and coronary plaque burdens and plaque structures in young adults with coronary atherosclerosis. Clinical Cardiology, 37(9), 552–557.

[22] Kalifa, M., Revathi, A., Nithyakalyani, R., Sivanesan, K. R., & Anandraj, J. (2021). Study on association of gamma glutamyl transferase with acute coronary syndrome andcorrelation with in-hospital outcome. IAIM, 8(1), 70-77.

[23] Singh, K. K., Kapoor, A., Khanna, R., Sahu, A., Kapoor, V., & Kumar, S., et al. (2022). Serum gamma-glutamyltransferase (GGT) in coronary artery disease: exploring the Asian Indian connection. Annals of Cardiac Anaesthesia, 25, 408-13.

[24] Dogan, A., Icli, A., Aksoy, F., Varol, E., Erdogan, D., Ozaydin, M., & Kocyigit, S. (2012). Gamma-glutamyltransferase in acute coronary syndrome patients without ST elevation and its association with stenotic lesion and cardiac events. Coronary Artery Disease, 23(1), 39-44.