Rwanda Medical Journal RMJ

ORIGINAL ARTICLE Open Access

The clinical significance of the TG/HDL ratio in acute coronary syndrome subtype presentation

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ABSTRACT

INTRODUCTION: Acute Coronary Syndrome (ACS) rates vary globally, and new lipid biomarkers like the TG/HDL and LDL/HDL ratios are gaining attention for potential risk stratification. However, their relationship with ACS subtype presentation has not been thoroughly investigated. This study aimed to determine the relationship between Acute Coronary Syndrome (ACS) subtypes (STEMI vs. NSTEMI) and the TG/HDL and LDL/HDL ratios.

METHODS: A retrospective cross-sectional study using hospital medical records was conducted on 136 ACS patients from August 2024 to September 2025 at Buleleng General Hospital. Comprehensive data were extracted, and the primary outcome measure was the ACS subtype. The main independent variables were the TG/HDL ratio and the LDL/HDL ratio. Statistical analyses included ROC curve analysis, chi-squared tests, and logistic regression.

RESULTS: The majority of patients were under 65 years old (58.8%). ROC curve analysis determined cut-off values of 2.40 for TG/HDL ratio and 2.38 for LDL/HDL ratio. Univariate analysis revealed a statistically significant relationship between the TG/HDL ratio (OR = 0.462; p = 0.047) and ACS subtype presentation, whereas no significant relationship was observed for the LDL/HDL ratio (OR = 1.122; p = 0.766). However, multivariate analysis showed a borderline significant relationship between a high TG/HDL ratio and NSTEMI presentation (AOR=2.163; p=0.049), warranting a cautious clinical interpretation.

CONCLUSION: A significant relationship exists between a high TG/HDL ratio and the presentation of ACS subtypes. These findings suggest that the TG/HDL ratio may be a useful adjunct marker for ACS phenotype stratification when integrated into multivariate models, though its standalone predictive capacity remains weak. Caution is required due to its low discriminatory performance, and further investigation in larger, prospective studies is warranted.

INTRODUCTION

Acute Coronary Syndrome (ACS) encompassing conditions like myocardial infarction and unstable angina, remains a primary cause of morbidity and mortality worldwide. Cardiovascular disease is the top cause of death globally, with ACS accounting for a significant proportion—estimates suggest

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Received: 24th December 2025; **Initial decision given:** 19th March 2026; **Revised manuscript received:** 28th March 2026; **Accepted:** 16th June 2026.
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Citation for this article: Putu Gupta Arya Gumilang; Ketut Susila; Putu Kiki Wulandari et al. The clinical significance of the TG/HDL ratio in acute coronary syndrome subtype presentation. Rwanda Medical Journal, Vol. 83, no. 2, p. 28-35, 2026. https://dx.doi.org/10.4314/rmj.v83i2.3

around 8–9 million deaths annually and one-third of all cardiovascular-related mortality, especially in those over 35 years old [1,2]. While high-income countries have seen a >50% reduction in age-standardized mortality rates (ASMRs) for ACS over the past two decades, low- and middle-income countries (LMICs) have experienced less progress, with some regions even seeing increases due to rising risk factors and limited healthcare resources [3,4].

The pathophysiology of ACS is complex, involving various risk factors and biomarkers. Traditional cardiovascular risk factors are well-established, but recent attention has focused on novel lipid biomarkers, particularly the triglyceride to high-density lipoprotein (TG/HDL) ratio and the low-density lipoprotein to high-density lipoprotein (LDL/HDL) ratio [5,6]. These ratios have shown promise in risk stratification and disease prediction for ACS [7,8]. The TG/HDL ratio, a simple yet effective indicator of insulin resistance and cardiovascular risk, is derived by dividing triglyceride levels by HDL cholesterol levels [9,10]. Higher ratios are associated with increased cardiovascular risk and atherogenic dyslipidemia, common in ACS patients. This ratio may help identify individuals at higher risk of adverse cardiac events and guide treatment interventions [7,8,11].

Similarly, the LDL/HDL ratio has emerged as a potential unique marker for atherosclerotic cardiovascular disease risk. By simultaneously considering both LDL-C and HDL-C levels, this ratio provides a comprehensive view of an individual's lipid profile and its associated cardiovascular risk [12,13]. Despite the growing interest in these lipid ratios, research findings have been inconsistent, and their predictive value in ACS subtype presentation remains unclear. The variations in reported results highlight the need for further investigation to elucidate the relationship between these ratios and ACS occurrence.

This study aimed to address this knowledge gap by examining the relationship between ACS subtype presentation and both the TG/HDL and LDL/HDL ratios. By focusing on these specific lipid biomarkers, we hope to contribute to the development of more severity and phenotype stratification tools for ACS, potentially leading to improved prevention strategies and patient outcomes in the future.

METHODS

Study Design

This single-center retrospective cross-sectional study using hospital medical records was conducted at Buleleng General Hospital from August 2024 to September 2025. The study aimed to investigate the relationship between TG/HDL and LDL/HDL ratios and ACS subtype presentation (STEMI vs. NSTEMI). The study protocol was approved by the Health Research Ethics Committee (reference number: 001/EC/KEPK-RSB/III/2024), and the need for informed consent was waived.

Study Participants

Patients diagnosed with ACS were identified from the hospital's medical records using relevant diagnostic codes. The diagnosis of ACS and its subtypes was based on established clinical guidelines integrating clinical presentation, 12-lead electrocardiogram (ECG) findings, and cardiac biomarkers. Specifically, ACS was diagnosed in patients presenting with ischemic chest pain or anginal equivalents accompanied by diagnostic ECG changes and/or elevated cardiac troponin levels. STEMI was defined by the presence of persistent chest pain alongside ST-segment elevation (≥1 mm in two or more contiguous leads, or ≥2 mm in leads V2–V3) on the initial ECG, subsequently confirmed by elevated cardiac troponin levels. NSTEMI was diagnosed in patients presenting with clinical symptoms of acute ischemia and a verified rise and/or fall of cardiac troponin levels above the 99th percentile upper reference limit, in the absence of persistent ST-segment elevation. Inclusion criteria were: (1) age 18 years or older, (2) diagnosis of ACS confirmed by medical records between 2024 and 2025, and (3) complete medical record data available. Exclusion criteria included: (1) patients who declined to participate in the study, (2) patients with incomplete medical records, (3) patients with atypical non-anginal symptoms, (4) patients with renal failure, sepsis, or non-cardiac systemic illness that could affect lipid metabolism, and (5) patients with hypovolemic shock. Due to the retrospective nature of this registry-based review, a formal a priori sample size calculation was not performed. Instead, a consecutive sampling approach was utilized, including all eligible patients (n = 136) who met the strict inclusion criteria during the defined study period at Buleleng General Hospital.

Data Collection Procedures

Comprehensive data were extracted from patients' medical records, including demographic information (age, education level, marital status, occupation), clinical data (comorbidities, body mass index, clinical symptoms), and laboratory results (total cholesterol, LDL, HDL, and triglyceride levels). The primary outcome measure was the ACS subtype (STEMI vs. NSTEMI), while the main independent variables were the TG/HDL ratio and the LDL/HDL ratio. Trained medical professionals experienced in chart review compiled the study data. The data were entered into a standardized, password-protected Microsoft Excel workbook and securely stored. To ensure objectivity, a statistician blinded to the study hypothesis received a coded data sheet for analysis.

Statistical Analysis

Statistical analyses were performed using SPSS software. Descriptive statistics were summarized using means, percentages, and standard deviations. Comparisons of means and proportions were performed using t-tests and chi-square tests, respectively. Chi-squared tests were employed to assess the relationship between these ratios and ACS subtypes. The Receiver Operating Characteristic (ROC) curve analysis was used to determine optimal cut-off values for TG/HDL and LDL/HDL ratios. Logistic regression analysis was conducted to adjust for potential confounding factors and to determine the independent association of TG/HDL and LDL/HDL ratios with NSTEMI vs. STEMI presentation. A two-sided significance level of 5% ($p<0.05$) was used throughout the analysis.

RESULTS

A total of 136 ACS patients were included in the study. The majority were younger than 65 years of age (58.8%) and male (66.2%). Most patients had an elementary school education (29.4%), were married (97.8%), and unemployed (65.4%). Regarding ACS classification, 72.8% were diagnosed with non-ST-segment elevation myocardial infarction (NSTEMI) and 27.2% with ST-segment elevation myocardial infarction (STEMI). The most common comorbidity was hypertension (39.7%), and most patients had a normal body mass index (52.9%). The predominant clinical symptom was chest pain (55.1%). The majority of patients did not undergo revascularization (76.5%), and the in-hospital mortality rate was 0.7%. Lipid profile analysis revealed a wide range of values, with total cholesterol levels ranging from 2.76 to 303 mg/dL and triglyceride levels from 2.4 to 510 mg/dL. The

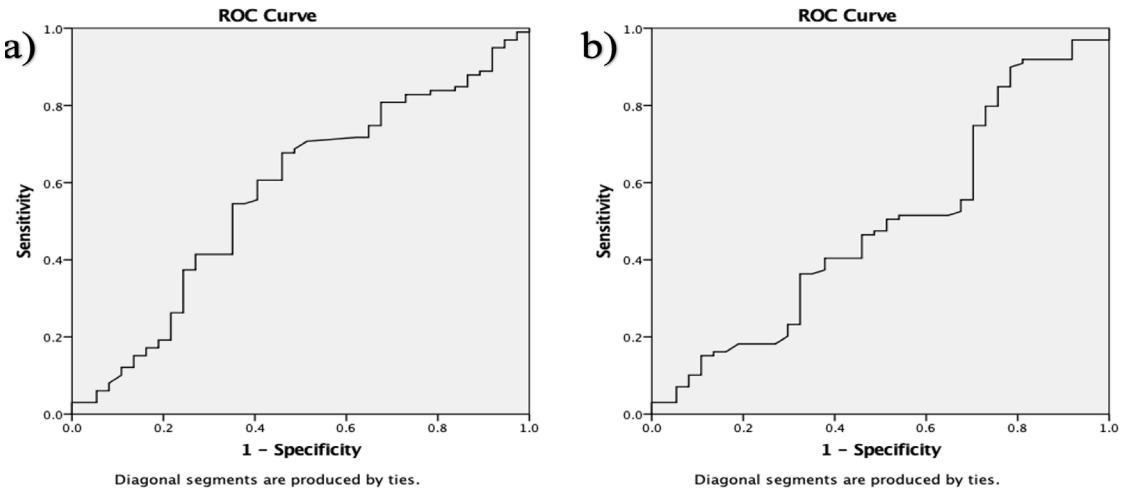

Figure 1: Receiver Operating Characteristic (ROC) curves evaluating standalone discriminatory performance for acute coronary syndrome subtype presentation.

Panel (a) displays the ROC curve for the TG/HDL ratio (AUC = 0.573; 95% CI: 0.462–0.684; p = 0.192), indicating a non-significant, weak standalone performance with an optimal cut-off value of 2.40. Panel (b) displays the ROC curve for the LDL/HDL ratio (AUC = 0.489; 95% CI: 0.376–0.602; p = 0.779), demonstrating a lack of predictive discrimination with an optimal cut-off value of 2.38. The Y-axis represents Sensitivity (True Positive Rate) and the X-axis represents 1 – Specificity (False Positive Rate).

Table 1: Association between the TG/HDL ratio and ACS subtype presentation

Variable	NSTEMI	STEMI	p-value	OR/aOR	95%CI
Univariate analysis					
TG/HDL ratio					
Low (≤2.40)	40 (64.5%)	22 (35.5%)	0.047	0.462	0.214-0.998
High (>2.40)	59 (79.7%)	15 (20.3%)			
LDL/HDL ratio					
Low (≤2.38)	51 (73.9%)	18 (26.1%)	0.766	1.122	0.527-2.388
High (>2.38)	48 (71.6%)	19 (28.4%)			
Multivariate analysis					
	Coefficient	**S.E.**	**p-value**	**AOR**	**95%CI**
TG/HDL ratio	0.772	0.393	0.049	2.163	1.002-4.669

The multivariate logistic regression model adjusted for potential confounding baseline patient characteristics, which included: age, gender, and the presence of primary comorbidities (hypertension and diabetes mellitus).
Abbreviations: S.E., standard error; OR, odds ratio; AOR, adjusted odds ratio; CI, confidence interval; TG/HDL, triglyceride to high-density lipoprotein; LDL/HDL, low-density lipoprotein to high-density lipoprotein.

mean LDL level was 106.31 mg/dL, and the median HDL level was 44 mg/dL. The median TG/HDL ratio was 2.78, and the mean LDL/HDL ratio was 2.48 (Appendix Table 1).

Receiver operating characteristic (ROC) curve analysis showed that the optimal cut-off value for the TG/HDL ratio was 2.40 (area under the curve [AUC] = 0.573; 95% CI: 0.462-0.684; p=0.192), and for the LDL/HDL ratio, it was 2.38 (AUC = 0.489; 95% CI: 0.376-0.602; p<0.779) (Figure 1). These low AUC values and non-significant p-values indicate that neither ratio possesses sufficient standalone discriminatory performance for predicting ACS subtype presentation.

In the univariate analysis, there was a statistically significant association between the TG/HDL ratio (OR = 0.462; 95% CI: 0.214–0.998; p = 0.047) and ACS subtype presentation. However, no significant association was found for the LDL/HDL ratio (OR = 1.122; 95% CI: 0.527–2.388; p = 0.766).. After adjusting for patient characteristics, a higher TG/HDL ratio was found to be borderline significantly associated with an increased likelihood of NSTEMI presentation over STEMI (adjusted OR = 2.163; 95% CI: 1.002-4.669; p=0.049) (Table 1). Because the p-value approaches the 0.05 threshold and the lower bound of the confidence interval is very close to 1.000, this independent association should be interpreted with caution.

DISCUSSION

This study found an independent association between a high triglyceride-to-high-density lipoprotein (TG/HDL) ratio and ACS subtype presentation, specifically a higher proportion of NSTEMI. However, it is critical to note that this multivariate association was borderline significant. The proximity of the lower confidence limit to 1.00 suggests that while the TG/HDL ratio shows promise for clinical phenotype stratification upon admission, its true predictive stability requires validation in larger sample sizes to rule out marginal effects. Moreover, there was no significant relationship was observed between LDL/HDL ratio and ACS presentation. The TG/HDL ratio has emerged as a useful marker for cardiovascular risk assessment. An elevated TG/HDL ratio reflects an atherogenic lipid profile characterized by high triglycerides and low HDL cholesterol levels [9]. This lipid imbalance shifts the vascular environment toward endothelial dysfunction, oxidative stress, and chronic inflammation, promoting unstable plaque architecture and high-risk coronary plaque characteristics [7,9,11]. Furthermore, this metabolic milieu drives the formation of small, dense LDL particles that readily penetrate the arterial wall and oxidize, a hallmark often observed in metabolic syndrome and diabetes where NSTEMI phenotypes frequently predominate over transmural STEMI presentations [14,15]. The clinical implication of this finding is

that the TG/HDL ratio could be used as a simple, readily available tool to help stratify ACS clinical phenotypes upon admission. However, this clinical utility must be interpreted with significant caution given its poor standalone discriminatory power in the ROC analysis; its significance becomes apparent only when controlling for other clinical confounders in multivariate models.

Patients with a high TG/HDL ratio often have insulin resistance, a hallmark of metabolic syndrome. Insulin resistance increases the hepatic synthesis of very-low-density lipoprotein (VLDL) and reduces the clearance of triglyceride-rich lipoproteins, resulting in elevated triglyceride levels [16,17]. Simultaneously, HDL cholesterol levels decline due to increased catabolism and compositional changes. This lipid imbalance also leads to the formation of small, dense LDL particles, which are highly atherogenic and more susceptible to oxidation [16].

The lack of a significant association between the LDL/HDL ratio and ACS subtype suggests that this ratio may not be a sensitive or independent predictor of specific acute coronary phenotypes in the study population. While LDL cholesterol and HDL cholesterol are well-established risk factors for atherosclerotic cardiovascular disease, their simple ratio may not fully capture the complex interactions of lipid metabolism and other non-lipid risk factors that contribute to the development of ACS [13,18]. One possible explanation for the absence of a correlation is that LDL cholesterol levels do not always accurately reflect the atherogenic capacity of circulating lipoproteins.

Multiple studies show that individuals with the same LDL cholesterol can have very different numbers and types of LDL particles, especially in metabolic syndrome or diabetes, where small, dense LDL particles predominate [19–21]. These small, dense LDL particles are more atherogenic due to their greater susceptibility to oxidation, longer circulation time, and increased ability to penetrate the arterial wall [15,22]. Additionally, the functionality of HDL, such as its capacity to support reverse cholesterol transport and exhibit antioxidant effects, may vary among individuals [23,24], rendering the ratio an imprecise marker of lipid-related phenotype differentiation.

A notable finding in our analysis is the apparent discrepancy between the poor standalone discriminatory performance of the TG/HDL ratio in the ROC analysis and its significant association within the multivariate logistic regression model. This variation highlights an important epidemiological reality: in a clinical vacuum, an unadjusted, isolated lipid ratio lacks sufficient predictive power due to the complex, multifactorial pathophysiology of coronary artery disease. However, when confounding noise from baseline clinical covariates—specifically age, gender, hypertension, and diabetes mellitus—is mathematically controlled within a multivariate framework, the true independent signal of the TG/HDL ratio emerges. This indicates that the ratio should not be used as an isolated diagnostic test, but rather interpreted as an adjunct stratification tool integrated alongside traditional cardiovascular risk factors.

These findings carry important clinical implications across both local and global healthcare landscapes. Globally, while high-income regions have achieved substantial reductions in age-standardized ACS mortality rates, low- and middle-income countries (LMICs) continue to struggle with an escalating burden of cardiovascular disease due to surging metabolic risk factors and constrained healthcare infrastructure [25]. Locally, in regional settings such as Buleleng, Bali, accessing advanced cardiac biomarker assays, routine coronary CT angiography, or immediate percutaneous revascularization can be restricted by resource availability. In these environments, the TG/HDL ratio offers immense clinical utility. Because it is easily calculated from a standard, highly affordable, and universally accessible baseline fasting lipid panel, it provides clinicians with an immediate, cost-effective tool to improve objective risk stratification and phenotype evaluation upon admission [26], ultimately facilitating more timely targeted interventions.

The limitations of this study include its retrospective cross-sectional design, which precludes causal conclusions, and its single-center setting, which limits immediate generalizability to broader, more diverse populations. Epidemiologically, the applicability of these findings may vary in high-income nations or advanced tertiary centers where baseline metabolic profiles differ and resources like immediate revascularization are universally available. However, our results remain highly relevant and applicable to similar resource-constrained regional settings in low- and middle-income countries (LMICs) facing an increasing cardiovascular burden. Crucially, due to retrospective medical record constraints, we lacked

data on key confounding variables such as pre-admission statin use, smoking status, diet, physical activity, and a family history of cardiovascular disease, all of which heavily influence lipid metabolism and ACS phenotype presentation. Furthermore, a formal sample size calculation was not conducted, and the relatively small sample size restricted the statistical power of the study, which likely contributed to the borderline significance observed in our multivariate model. Finally, the low, non-significant AUC values underscore that these lipid ratios possess weak standalone predictive power, indicating they should strictly be utilized as adjunct markers within a broader clinical assessment rather than isolated diagnostic tools.

CONCLUSION

This study demonstrated a significant relationship between a high TG/HDL ratio and ACS subtype presentation, while no significant association was found between the LDL/HDL ratio and ACS subtypes. These findings suggest that the TG/HDL ratio may be a more useful marker for cardiovascular phenotype stratification compared to the LDL/HDL ratio in the study population. However, further research with larger sample sizes and prospective study designs is needed to confirm these results and elucidate the underlying mechanisms.

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Appendix Table 1: Characteristic of patients included

Variable	n	%		n	%
Age Group (Years)			Body Mass Index (BMI)		
< 65	80	58.8	Underweight	4	2.9
65–75	38	27.9	Normal	72	52.9
> 75	18	13.2	Overweight	54	39.7
Gender			Obesity	6	4.4
Female	46	33.8	Primary Clinical Symptoms		
Male	90	66.2	Chest Pain	75	55.1
Educational Status			Shortness of Breath	58	42.6
No formal education	16	11.8	Fatigue	2	1.5
Elementary School	40	29.4	Decreased Consciousness	1	0.7
Junior High School	24	17.6	Nausea and Vomiting	0	0.0
Senior High School	38	27.9	Revascularization Therapy		
College	18	13.2	Fibrinolytic	24	17.6
Marital Status			PCI	8	5.9
Married	133	97.8	None	104	76.5
Unmarried	3	2.2	In-Hospital Mortality		
Occupation			Survived	135	99.3
Employed	22	16.2	Deceased	1	0.7
Unemployed	89	65.4	Clinical and Lipid Profiles	Value	Statistical Measure
Housewife	25	18.4	Total Cholesterol (mg/dL)	166.0 (2.76–303)	Median (Range)
ACS Subtype Presentation			Triglycerides (mg/dL)	108.5 (2.4–510)	Median (Range)
NSTEMI	99	72.8	HDL-C (mg/dL)	44 (12–107)	Median (Range)
STEMI	37	27.2	TG/HDL ratio	2.68 (0.09–22.50)	Median (Range)
Comorbidities			LDL-C (mg/dL)	106.31 ± 36.57	Mean ± SD
Hypertension	54	39.7	LDL/HDL ratio	2.48 ± 0.97	Mean ± SD
Diabetes Mellitus	34	25.0			
Chronic Kidney Disease	12	8.8			
Dyslipidemia	4	2.9			
Pneumonia	1	0.7			
Heart Disease	1	0.7			
Hyperthyroidism	0	0.0			
No comorbidities	6	4.4			

Abbreviations: ACS, acute coronary syndrome; NSTEMI, non-ST-segment elevation myocardial infarction; STEMI, ST-segment elevation myocardial infarction; PCI, percutaneous coronary intervention; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; TG, triglycerides; SD, standard deviation.