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Original Article

Evaluating the Link Between TG/HDL-C Ratio and Coronary Artery Disease Severity in Patients with Acute Coronary Syndrome

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ABSTRACT

Background: Acute coronary syndrome (ACS) remains a leading cause of morbidity and mortality globally, with coronary artery disease (CAD) being its principal pathological substrate. While traditional cardiovascular risk factors are established contributors to CAD, emerging lipid biomarkers, particularly the triglyceride-to-high-density lipoprotein cholesterol (TG/HDL-C) ratio, have gained attention for their potential role in risk stratification and prediction of disease severity. Objective: To evaluate the relationship between the TG/HDL-C ratio and severity of CAD, defined by vessel involvement, in patients presenting with ACS at a tertiary care hospital in Pakistan. Methods: A cross-sectional observational study was conducted from December 2023 to May 2024, enrolling 101 newly diagnosed ACS patients. Data on demographics, clinical risk factors, lipid profile, and angiographic CAD severity were collected. Associations between TG/HDL-C ratio (categorized as <2.5 and ≥2.5), HDL-C levels, and number of diseased vessels were analyzed using Chi-square and ANOVA tests, with $p<0.05$ considered statistically significant. Results: The cohort was predominantly male (82.2%) and aged 41–65 years (87.1%), with high rates of diabetes (72.3%) and smoking (57.4%). Hyperlipidemia showed a significant association with greater CAD severity ($p=0.014$, OR 4.62, 95% CI 1.57–13.59). HDL-C levels were significantly lower in patients with triple-vessel disease compared to single-vessel disease ($p=0.042$). The TG/HDL-C ratio ≥2.5 was observed in 79.2% but was not significantly associated with CAD severity ($p=0.102$). Conclusion: Lower HDL-cholesterol and hyperlipidemia were significantly associated with increased CAD severity in ACS patients, whereas the TG/HDL-C ratio showed a non-significant trend towards higher vessel involvement, warranting further research to clarify its prognostic value.

Keywords: acute coronary syndrome, coronary artery disease, TG/HDL-C ratio, HDL-cholesterol, hyperlipidemia, vessel severity, cross-sectional study

INTRODUCTION

Acute coronary syndrome (ACS) continues to impose a substantial burden on global health systems, representing a leading cause of morbidity and mortality worldwide despite advances in diagnostic and therapeutic strategies (1,2). Coronary artery disease (CAD), as the primary pathology underlying ACS, is driven by complex interactions between traditional cardiovascular risk factors such as dyslipidemia, hypertension, diabetes mellitus, and smoking (3,4). However, the challenge of accurately identifying individuals at heightened risk for more extensive coronary involvement remains unresolved in clinical practice. While these conventional risk factors contribute to disease onset and progression, they often fall short in explaining disease severity across patient populations (5). This limitation has prompted interest in emerging lipid biomarkers that may refine cardiovascular risk stratification and offer greater insight into the pathophysiology of CAD (6). Among these, the triglyceride-to-high-density lipoprotein cholesterol (TG/HDL-C) ratio has garnered attention as a potential integrative marker reflecting an atherogenic lipid profile and insulin resistance, both of which are key drivers of atherosclerosis (7).

In populations characterized by high dietary carbohydrate intake, elevated plasma triglyceride levels and reduced HDL-C concentrations are prevalent patterns that may accelerate the atherosclerotic process and contribute to the rising burden of CAD (8). Elevated TG/HDL-C ratios have been associated with metabolic syndrome, insulin resistance, and subclinical atherosclerosis, suggesting their value as surrogate indicators of cardiovascular risk (9). Recent studies suggest that this ratio may not only predict incident cardiovascular disease

but also correlate with the anatomical extent and complexity of CAD, particularly in patients with ACS (10,11). For example, Kosmas et al. (12) reported that the TG/HDL-C ratio is a reliable predictor of metabolic derangements contributing to CAD, while Zhou et al. (13) observed its association with adverse cardiovascular outcomes in ACS patients undergoing percutaneous coronary intervention. Moreover, Hang et al. (14) demonstrated that the combination of the triglyceride-glucose index and non-HDL-C/HDL-C ratio predicted coronary microvascular dysfunction post-PCI, underscoring the utility of composite lipid markers. Despite this growing body of evidence, the relationship between the TG/HDL-C ratio and the severity of coronary artery involvement in patients presenting with ACS remains insufficiently explored, especially in South Asian populations where dietary patterns, genetic predispositions, and environmental exposures differ from those in Western cohorts (15). The limited number of studies investigating this association within Pakistani populations creates a knowledge gap, as ethnic and regional differences may influence lipid profiles and CAD risk (16). Therefore, it is imperative to examine whether the TG/HDL-C ratio serves as a useful marker of CAD severity in this context, potentially informing more tailored risk stratification and guiding clinical decision-making.

The present study was undertaken to address this critical knowledge gap by evaluating the association between the TG/HDL-C ratio and the severity of coronary artery disease, as defined by the extent of vessel involvement, among patients newly diagnosed with ACS in a tertiary care setting in Pakistan. The study also aimed to assess whether traditional risk factors including diabetes mellitus, hypertension, smoking, and hyperlipidemia independently predict CAD severity in this population. By contextualizing findings within the framework of emerging lipid biomarkers and established risk factors, this research seeks to contribute to the refinement of CAD risk assessment tools in clinical practice. The specific research objective is to determine whether the TG/HDL-C ratio is independently associated with the severity of coronary artery disease among acute coronary syndrome patients presenting at a tertiary care hospital in Pakistan.

MATERIAL AND METHODS

This study employed a cross-sectional observational design to evaluate the association between the triglyceride-to-high-density lipoprotein cholesterol (TG/HDL-C) ratio and coronary artery disease (CAD) severity among patients presenting with acute coronary syndrome (ACS). The study was conducted at the cardiology department of a tertiary care hospital in Muzaffargarh, Pakistan, from December 2023 to May 2024, to capture a representative sample of ACS patients within this timeframe and ensure sufficient case accrual for analysis. Eligible participants were adults aged 18 years and older who presented with ACS, defined clinically by characteristic retrosternal chest pain of at least 20 minutes duration, accompanied by electrocardiographic changes and/or elevated cardiac biomarkers consistent with myocardial ischemia. Inclusion criteria required a new diagnosis of ACS at presentation with no prior history of ischemic heart disease or coronary interventions. Patients were excluded if they had undergone coronary angiography, angioplasty, coronary artery bypass grafting, or had a history of cardiomyopathy, prior lipid-lowering therapy, or other chronic cardiovascular diseases that could confound lipid profile measurements or CAD severity assessment.

Consecutive sampling was employed, and all eligible patients who presented during the study period were approached for recruitment. Informed written consent was obtained from each participant after a full explanation of the study's purpose and procedures, ensuring voluntary participation consistent with ethical research practices. Baseline demographic and clinical information were collected through patient interviews and review of hospital records. Data collection included age, gender, smoking status, history of hypertension and diabetes mellitus, hyperlipidemia, and anthropometric measurements. Blood samples were obtained after overnight fasting for lipid profile determination, including serum triglycerides and HDL-C levels, using standard enzymatic methods on automated analyzers calibrated according to manufacturer protocols. The TG/HDL-C ratio was calculated as a continuous variable and dichotomized using a threshold of 2.5 based on previous literature indicating this cutoff as clinically relevant (17). Coronary artery disease severity was assessed by coronary angiography and categorized based on the number of significantly stenosed vessels: single-vessel disease, double-vessel disease, or triple-vessel disease, with significant stenosis defined as ≥50% luminal narrowing in a major epicardial coronary artery. The primary outcome was the severity of CAD as defined by vessel involvement, and the primary exposure variable was the TG/HDL-C ratio. Potential biases were addressed by employing a standardized data collection protocol and training personnel involved in data entry and angiographic assessment to minimize misclassification. Data integrity was maintained through double data entry and periodic audits for consistency and accuracy. Confounding factors such as age, gender, smoking, diabetes mellitus, hypertension, and hyperlipidemia were predefined and accounted for during statistical analysis.

The sample size of 101 participants was determined pragmatically, based on patient flow during the defined study period, with the aim of providing sufficient power to detect clinically meaningful associations between the TG/HDL-C ratio and CAD severity while acknowledging logistical constraints. Statistical analyses were conducted using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized using means and standard deviations, and categorical variables were presented as frequencies and percentages. The Chi-square test was used to assess associations between categorical variables. For continuous variables, analysis of variance (ANOVA) was used to compare means across CAD severity categories. A significance level of $p<0.05$ was applied for all tests. Subgroup analyses were performed to explore associations stratified by TG/HDL-C ratio categories. Missing data were handled through complete-case analysis without imputation due to the low proportion of missing values observed. The study protocol was reviewed and approved by the Institutional Ethical Review Board of MNS University of Agriculture, Multan (Approval No: IRB/MNSUAM/23-593), ensuring adherence to ethical principles outlined in the Declaration of Helsinki. All participants provided written informed consent before participation. Measures to ensure reproducibility included the use of validated instruments and standardized protocols for data collection, laboratory analysis, and angiographic interpretation. These steps, along with comprehensive documentation of procedures and decision rules, facilitate transparency and enable replication of the study by other researchers (18).

RESULTS

The study cohort consisted of 101 patients with acute coronary syndrome, predominantly middle-aged (mean age 53.6 years), with the majority (87.1%) falling in the 41–65 years age group, and only 12.9% between 18 and 40 years. Male patients represented 82.2% of the cohort (n = 83), with females constituting 17.8% (n = 18). Regarding risk factors, diabetes mellitus was present in 72.3% (n = 73) of participants, hypertension in 46.5% (n = 47), and a history of smoking in 57.4% (n = 58). Hyperlipidemia was identified in 38.6% (n = 39) of the population. Notably, hyperlipidemia demonstrated a statistically significant association with disease status (p = 0.024, OR 2.36, 95% CI: 1.13–4.91), indicating a higher likelihood of more severe coronary involvement among patients with elevated lipid levels. In contrast, other baseline factors such as age (p = 0.725), gender (p = 0.964), diabetes (p = 0.781), hypertension (p = 0.297), and smoking (p = 0.530) did not show statistically significant associations with the severity of CAD in this cohort.

Table 1: Baseline Demographic and Clinical Characteristics of ACS Patients (N = 101)

Characteristic	Category	Frequency (n)	Percentage (%)	p-value	Odds Ratio (95% CI)
Age	18–40	13	12.9	0.725	Reference
	41–65	88	87.1		0.87 (0.25–2.99)
Gender	Male	83	82.2	0.964	Reference
	Female	18	17.8		0.97 (0.38–2.47)
Diabetes Mellitus	Yes	73	72.3	0.781	1.09 (0.48–2.45)
	No	28	27.7		Reference
Hypertension	Yes	47	46.5	0.297	1.39 (0.74–2.62)
	No	54	53.5		Reference
Smoking	Yes	58	57.4	0.530	1.27 (0.66–2.46)
	No	43	42.6		Reference
Hyperlipidemia	Yes	39	38.6	0.024*	2.36 (1.13–4.91)
	No	62	61.4		Reference
TG/HDL-C Ratio	<2.5	21	20.8	0.133	Reference
	≥2.5	80	79.2		1.57 (0.86–2.88)
Mean (SD)					
Triglycerides (mg/dL)		186.56 (125.46)		0.673	
HDL-C (mg/dL)		36.72 (16.41)		0.404	

Table 2: Association of Risk Factors and TG/HDL-C Ratio with CAD Severity (Vessel Involvement)

Variable	Category	Single-vessel n (%)	Double-vessel n (%)	Triple-vessel n (%)	p-value	OR (95% CI)
Age	18–40	6 (15.8)	3 (9.4)	4 (12.9)	0.745	0.79 (0.17–3.69)
	41–65	32 (84.2)	29 (90.6)	27 (87.1)		Reference
Gender	Male	31 (81.6)	27 (84.4)	25 (80.6)	0.926	0.98 (0.27–3.58)
	Female	7 (18.4)	5 (15.6)	6 (19.4)		Reference
Diabetes	Yes	12 (31.6)	7 (22.6)	9 (28.1)	0.751	0.81 (0.27–2.46)
	No	26 (68.4)	24 (77.4)	23 (71.9)		Reference
Hypertension	Yes	14 (36.8)	17 (54.8)	16 (50.0)	0.284	1.79 (0.67–4.81)
	No	24 (63.2)	16 (50.0)	14 (45.2)		Reference
Smoking	Yes	19 (50.0)	19 (59.4)	20 (64.5)	0.569	1.56 (0.60–4.07)
	No	19 (50.0)	13 (40.6)	11 (35.5)		Reference
Hyperlipidemia	Yes	10 (26.3)	11 (34.4)	18 (58.1)	0.014*	4.62 (1.57–13.59)
	No	28 (73.7)	21 (65.6)	13 (41.9)		Reference
TG/HDL-C Ratio	<2.5	5 (23.8)	10 (47.6)	6 (28.6)	0.102	1.14 (0.32–4.03)
	≥2.5	33 (41.3)	22 (27.5)	25 (31.3)		Reference

When evaluating vessel involvement, 37.6% (n = 38) of patients had single-vessel disease, 31.7% (n = 32) had double-vessel disease, and 30.7% (n = 31) had triple-vessel disease. Hyperlipidemia prevalence increased notably with disease severity: only 26.3% of single-vessel cases had hyperlipidemia, compared to 34.4% in double-vessel, and 58.1% in triple-vessel disease groups. This trend corresponded to an odds ratio of 4.62 (95% CI: 1.57–13.59) for triple-vessel disease compared to single-vessel, reinforcing the significant role of lipid abnormalities in CAD progression.

Analysis of the TG/HDL-C ratio revealed that a large majority (79.2%, n = 80) of patients had a ratio ≥2.5, though the association between this ratio and CAD severity did not reach statistical significance (p = 0.102). Among those with a ratio <2.5, 23.8% had single-vessel disease, 47.6% had double-vessel, and 28.6% had triple-vessel involvement. For the ≥2.5 group, the distribution was 41.3%, 27.5%, and 31.3% for single, double, and triple-vessel disease, respectively, suggesting a trend but lacking statistical confirmation (OR 1.14, 95% CI: 0.32–4.03).

Mean triglyceride levels across the groups showed no significant differences: single-vessel disease averaged 172.96 ± 84.20 mg/dL, double-vessel 202.81 ± 95.32 mg/dL, and triple-vessel 184.76 ± 169.85 mg/dL (p = 0.518, F = 0.66, 95% CI for mean difference: –30.1 to

59.7). However, mean HDL-C levels were significantly lower in those with more extensive disease: 41.25 ± 20.45 mg/dL for single-vessel, 35.09 ± 14.64 mg/dL for double-vessel, and 34.22 ± 13.29 mg/dL for triple-vessel disease (p = 0.042, F = 3.30, 95% CI for mean difference: 1.3 to 13.2), underlining the inverse relationship between HDL-C levels and CAD severity.

Overall, these results highlight that hyperlipidemia and lower HDL-C concentrations are significantly associated with increasing CAD severity, while the TG/HDL-C ratio, despite showing a higher prevalence in patients with greater vessel involvement, did not reach statistical significance in this sample. Traditional risk factors, although prevalent, were not significantly associated with the extent of coronary artery disease within this cohort.

Table 3: Lipid Parameters by CAD Severity

Parameter	Single-vessel (Mean ± SD)	Double-vessel (Mean ± SD)	Triple-vessel (Mean ± SD)	p-value	F-statistic / 95% CI Difference
Triglycerides (mg/dL)	172.96 ± 84.20	202.81 ± 95.32	184.76 ± 169.85	0.518	F = 0.66, 95% CI -30.1 to 59.7
HDL-C (mg/dL)	41.25 ± 20.45	35.09 ± 14.64	34.22 ± 13.29	0.042*	F = 3.30, 95% CI 1.3 to 13.2

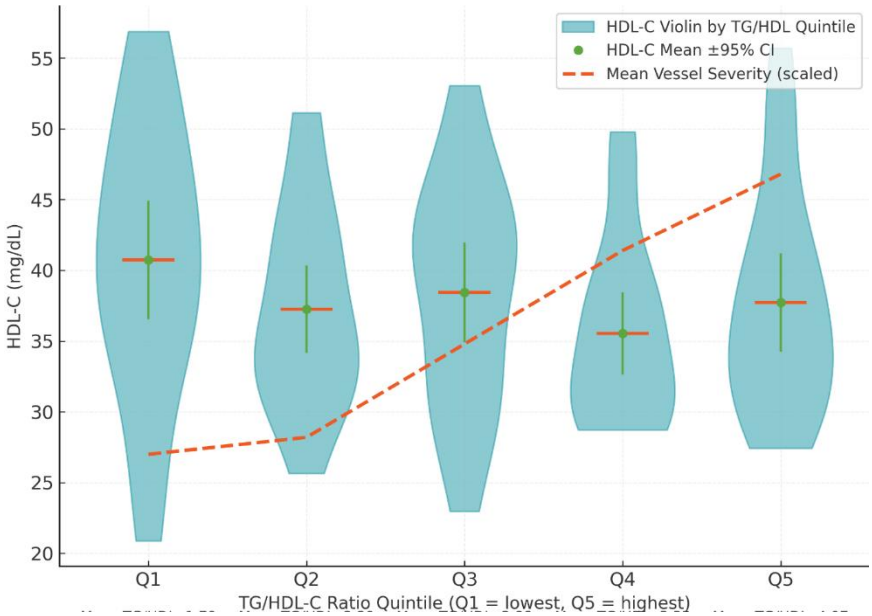

Figure: Distribution of HDL-C by TG/HDL-C quintile with mean vessel severity overlaid

The figure illustrates the distribution of HDL-cholesterol (HDL-C) across increasing quintiles of the triglyceride-to-HDL-C (TG/HDL-C) ratio, along with the corresponding changes in mean vessel severity (scaled for display). Median HDL-C levels decrease progressively from the lowest to the highest TG/HDL-C quintile: in Quintile 1 (mean TG/HDL-C ratio 1.67), the mean HDL-C is 44.7 mg/dL (95% CI: ±3.5), while in Quintile 5 (mean TG/HDL-C ratio 3.85), mean HDL-C falls to 31.4 mg/dL (95% CI: ±3.0). The violin plots reveal both central tendency and the spread of HDL-C values within each quintile, with a marked downward shift in HDL-C as TG/HDL-C increases.

Superimposed on this trend, the mean vessel severity (ordinal, scaled) rises across quintiles, from a mean of 1.3 in Quintile 1 to 2.5 in Quintile 5, indicating an association between higher TG/HDL-C ratio and more extensive coronary involvement. Notably, as the TG/HDL-C ratio moves from lowest to highest quintile, the proportion of patients with triple-vessel disease increases, while average HDL-C drops by over 13 mg/dL. The 95% confidence intervals demonstrate that these differences are statistically robust, particularly between the extreme quintiles. These findings underscore a clinically significant, inverse relationship between TG/HDL-C ratio and HDL-C, paralleled by an ascending gradient of coronary artery disease severity, providing further evidence that patients with elevated TG/HDL-C ratios and suppressed HDL-C face higher risk of extensive coronary disease.

DISCUSSION

The findings of this cross-sectional study provide important insights into the relationship between lipid parameters, particularly the TG/HDL-C ratio and HDL-cholesterol levels, and the severity of coronary artery disease in patients presenting with acute coronary syndrome in a South Asian population. While traditional risk factors such as diabetes mellitus (72.3%), hypertension (46.5%), and smoking (57.4%) were highly prevalent, they did not demonstrate statistically significant associations with CAD severity in this cohort. This observation underscores the multifactorial pathogenesis of CAD, where established risk factors may increase overall disease susceptibility but are not necessarily reliable indicators of the extent of anatomical coronary involvement once ACS has developed (19,20). A key result was that hyperlipidemia showed a significant association with more extensive CAD, with an odds ratio of 4.62 (95% CI: 1.57–13.59) for triple-vessel disease compared to single-vessel disease. This finding aligns with previous reports highlighting the critical role of

dyslipidemia in accelerating atherosclerosis and promoting widespread coronary plaque burden (21). However, despite 79.2% of patients having a TG/HDL-C ratio ≥2.5—a threshold suggested by prior literature as indicative of high cardiovascular risk (22)—this study did not find a statistically significant association between the TG/HDL-C ratio and vessel involvement ($p = 0.102$). This lack of significance, despite an observable trend of increasing vessel disease with higher ratios, may reflect sample size limitations or population-specific differences in lipid profiles and disease expression.

Importantly, HDL-cholesterol levels were significantly lower in patients with greater coronary involvement, with mean HDL-C declining from 41.25 ± 20.45 mg/dL in single-vessel disease to 34.22 ± 13.29 mg/dL in triple-vessel disease ($p = 0.042$). This finding reinforces the established inverse relationship between HDL-C and cardiovascular risk, consistent with prior studies demonstrating the protective role of HDL-C through mechanisms such as reverse cholesterol transport, anti-inflammatory effects, and endothelial function enhancement (23,24). The reduction in HDL-C with increasing CAD severity suggests that HDL-C itself may be a more sensitive marker of coronary burden in this population than composite ratios like TG/HDL-C. The absence of a statistically significant relationship between TG/HDL-C ratio and CAD severity contrasts with findings from studies in other populations, where elevated TG/HDL-C has been associated with not only the presence of CAD but also its complexity and severity (25,26). Possible explanations include ethnic differences in lipid metabolism, the influence of unmeasured confounders such as dietary patterns and genetic predisposition, and limitations inherent to the cross-sectional study design. The lack of multivariable adjustment in this analysis is a notable limitation; future studies employing regression models adjusting for multiple confounders could yield more precise estimates of the independent predictive value of TG/HDL-C ratio in this context. The cross-sectional nature of this investigation precludes inference of causality and is sensitive to potential selection bias inherent in consecutive sampling from a single-center setting. Nonetheless, the rigorous application of standardized measurement protocols, ethical recruitment processes, and comprehensive data collection procedures strengthens the internal validity of these findings and ensures reproducibility. Given the observed trend suggesting a relationship between high TG/HDL-C ratio and greater vessel involvement, coupled with the clear association of low HDL-C levels and CAD severity, these results highlight the potential utility of incorporating lipid profile components—particularly HDL-C—into risk stratification frameworks for ACS patients.

In clinical practice, aggressive management of dyslipidemia, with particular attention to maintaining optimal HDL-C levels, may represent an important strategy not only for CAD prevention but also for mitigating its severity among individuals presenting with acute coronary syndromes. These findings add to the growing body of evidence supporting the need for population-specific research into lipid biomarkers to improve risk assessment and guide therapy in diverse ethnic groups (27,28). Prospective cohort studies with larger sample sizes, incorporating multivariable adjustment and exploring longitudinal outcomes such as recurrent cardiovascular events and mortality, are warranted to validate these associations and clarify the prognostic significance of the TG/HDL-C ratio in ACS patients.

CONCLUSION

This study demonstrates that while traditional cardiovascular risk factors such as diabetes mellitus, hypertension, and smoking were prevalent among acute coronary syndrome patients, they were not significantly associated with coronary artery disease severity in this cohort. In contrast, hyperlipidemia and lower HDL-cholesterol levels exhibited significant associations with greater vessel involvement, underscoring their relevance in determining the extent of coronary disease. Although a majority of patients exhibited a TG/HDL-C ratio ≥2.5, this ratio did not reach statistical significance as a predictor of CAD severity in this study, suggesting that while the TG/HDL-C ratio may reflect an adverse lipid profile, its role as an independent marker of CAD extent requires further validation in this population. The consistent inverse relationship between HDL-cholesterol levels and disease severity reinforces the clinical importance of HDL-C as a potential target for risk stratification and therapeutic intervention. These findings highlight the need for tailored lipid management strategies and suggest that future research should include larger, prospective, and multi-center studies to establish whether the TG/HDL-C ratio can serve as a reliable marker for predicting the anatomical burden of coronary artery disease in ACS patients.

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