

HIGH TROPONIN, LOW HDL: UNRAVELING THE METABOLIC PARADOX IN ACUTE CORONARY SYNDROME PATIENTS

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DOI: <https://doi.org/10.5281/zenodo.18755277>

Keywords

Acute Coronary Syndrome,
Troponin, HbA1c, LDL, HDL,
Myocardial Injury

Article History

Received: 25 December 2025

Accepted: 09 February 2026

Published: 24 February 2026

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Abstract

Objective: To evaluate the correlation between high troponin levels and metabolic markers (HbA1c, LDL, and HDL) in patients presenting with Acute Coronary Syndrome (ACS).

Methods: This cross-sectional study was conducted at Peshawar Institute of Cardiology. A total of 182 patients diagnosed with ACS and elevated troponin were enrolled through non-probability convenience sampling. Venous blood samples were analyzed for troponin (via Chemiluminescent Immunoassay), lipid profile, and HbA1c (via spectrophotometry). Statistical analysis included Pearson correlation, Chi-square tests, and subgroup analyses.

Results: The study population comprised 124 (68.1%) males and 58 (31.9%) females, with a mean age of 60.1 ± 12.4 years. Elevated HbA1c ($\geq 6.5\%$) was observed in 83 (45.6%) patients. Pearson correlation showed no significant association between troponin and HbA1c ($r=0.068$, $p=0.361$). Regarding lipid profile, 71 (39.0%) patients had elevated LDL, while 81 (44.5%) had decreased HDL. Patients with low HDL demonstrated disproportionately high troponin values, particularly at extreme elevations ($>50,000$ ng/L), where 92.9% had normal LDL but low HDL. Chi-square analysis revealed no statistically significant association between LDL categories and troponin levels when stratified by HDL status ($p>0.05$).

Conclusion: While no statistically significant linear correlation is observed between troponin and metabolic markers in ACS patients, the observed pattern of extreme troponin elevations in low-HDL patients suggests that HDL may modulate myocardial injury severity. These findings highlight the complex pathophysiology of ACS beyond traditional risk factors.

INTRODUCTION

Acute Coronary Syndrome (ACS) represents a spectrum of life-threatening cardiovascular conditions that significantly contribute to global morbidity and mortality, encompassing Unstable Angina Pectoris (UAP), Non-ST-Segment Elevation Myocardial Infarction (NSTEMI), and ST-Segment Elevation Myocardial Infarction (STEMI) [1]. The underlying pathophysiology

involves reduced blood flow to the coronary arteries due to partial or total thrombotic occlusion, with atherosclerosis serving as the fundamental mechanism in ACS pathogenesis [1]. The prevalence of ACS among patients presenting with chest pain ranges from 13% to 46%, with a higher prevalence in males [2]. In Pakistan, ACS constitutes 36.9% of chest pain

presentations, representing a significant public health burden [3].

Atherosclerosis involves a complex multi-step process where Low-Density Lipoprotein (LDL) infiltrates the subendothelial space of coronary arteries and undergoes oxidative modification, triggering macrophage infiltration and initiating pro-inflammatory cascades that promote plaque formation [1]. LDL cholesterol plays a pivotal role in coronary heart disease pathogenesis, with current risk reduction strategies prioritizing achieving optimal LDL levels through therapeutic interventions [4]. Conversely, High-Density Lipoprotein (HDL) cholesterol exerts cardioprotective effects through reverse cholesterol transport, facilitating cholesterol removal from peripheral tissues to the liver for biliary excretion. Epidemiological evidence demonstrates that individuals with low HDL-C face significantly higher cardiovascular risk [5].

Patients with diabetes exhibit a two to four-fold increased risk of coronary artery disease compared to non-diabetic individuals, and among ACS patients, over one-third have concomitant diabetes, with this subgroup experiencing poorer clinical outcomes [6]. Glycated hemoglobin (HbA1c) serves as an established marker of long-term glycemic control, with elevated levels predicting increased risk of both microvascular and macrovascular complications [7]. The troponin complex, comprising three regulatory proteins Troponin I (TnI), Troponin T (TnT), and Troponin C (TnC) mediates calcium-dependent contraction of cardiac muscle, and cardiac troponin measurement represents the gold standard biomarker for ACS diagnosis and risk stratification [8]. Beyond ACS, elevated troponin indicates myocardial injury from various etiologies and correlates with subclinical cardiovascular dysfunction markers [9].

While elevated troponin reliably indicates myocardial injury in ACS, the relationship between troponin magnitude and metabolic parameters (HbA1c, LDL, HDL) remains inadequately characterized, particularly in the Pakistani population, with limited regional data examining these associations in patients presenting to tertiary care centers in Peshawar.

This study aims to comprehensively evaluate the association between troponin elevation and metabolic markers (HbA1c, LDL, HDL) in ACS patients, potentially enhancing diagnostic accuracy, risk stratification, and prognostic assessment.

MATERIALS AND METHODS

This cross-sectional analytical study was conducted at the Institute of Paramedical Sciences, Khyber Medical University, in collaboration with Peshawar Institute of Cardiology (PIC), Peshawar, Pakistan. Non-probability convenience sampling was utilized, enrolling consecutively presenting patients who met the inclusion criteria. Sample size was calculated using the formula for cross-sectional studies $n = Z^2 \times p(1-p) / d^2$, where $Z=1.96$ at 95% confidence level, $p=36.9\%$ based on local ACS prevalence [3], and $d=\text{margin of error of } 7\% (0.07)$, yielding a required sample size of 182 patients [10]. Inclusion criteria comprised patients diagnosed with ACS (STEMI, NSTEMI, or Unstable Angina), elevated troponin, age ≥ 18 years, and provision of informed consent. Exclusion criteria included patients with chronic coronary syndrome, known history of lipid-lowering or anti-diabetic medication use, concomitant conditions that may elevate troponin, such as sepsis, pulmonary embolism, chronic kidney disease, or myocarditis, previous coronary revascularization, and refusal to provide informed consent. The study protocol was approved by the Institutional Ethics Committee of Peshawar Institute of Cardiology (Reference: PIC/ERC/2024/07-112), and written informed consent was obtained from all participants. Venous blood samples of 5 mL were collected under aseptic conditions from the antecubital fossa using a disposable syringe following standard phlebotomy guidelines [11], with tourniquet application proximal to the puncture site for ≤ 20 seconds. Blood was immediately transferred to yellow top/gel tubes (serum separator) for lipid profile and troponin analysis, and purple top EDTA tubes for HbA1c measurement. For lipid profile and troponin, serum tubes were centrifuged at 4000 rpm for 2-5

minutes using an automated centrifuge to separate serum from cellular components, while for HbA1c, EDTA whole blood was placed on a shaker for RBC hemolysis before analysis. Troponin I was analyzed using the Abbott Architect CI 1000 analyzer employing the chemiluminescent immunoassay (CLIA) principle [12]. Reference ranges for troponin were 28.9-39.2 pg/mL for males and 13.8-17.5 pg/mL for females [13]. Lipid parameters were analyzed using the Abbott Architect CI 4100 analyzer employing spectrophotometric principles [14]. Reference ranges for lipid profile included HDL Cholesterol 35-60 mg/dL (optimal >40 mg/dL in males, >50 mg/dL in females) and LDL Cholesterol 100-130 mg/dL (optimal <100 mg/dL) [15]. HbA1c was analyzed using the enzymatic method on Abbott Architect CI 4100 [16]. Reference ranges for HbA1c (NGSP) were normal <5.7%, prediabetes 5.7-6.4%, and diabetes ≥6.5% [17]. Data were analyzed using SPSS version 26.0, with continuous variables expressed as mean ± standard deviation or median (interquartile range) based on

distribution, and categorical variables presented as frequencies and percentages. Inferential statistics included Pearson correlation coefficient to assess linear relationships between troponin and continuous variables (HbA1c, LDL, HDL) after log-transformation of troponin to normalize distribution, Chi-square test to evaluate associations between categorical variables, independent t-test/Mann-Whitney U test to compare continuous variables between groups based on data distribution, and subgroup analyses performed for HDL and LDL categories, with p-value <0.05 considered statistically significant (two-tailed).

RESULTS

A total of 182 patients diagnosed with Acute Coronary Syndrome and elevated troponin were included in the final analysis. The study population comprised 124 males (68.1%) and 58 females (31.9%), demonstrating male predominance consistent with ACS epidemiology table 1.

Table 1: Demographic Characteristics of Study Population

Characteristic	Category	Frequency (n)	Percentage (%)
Gender	Male	124	68.1
	Female	58	31.9
Age Group (years)	≤30	2	1.1
	31-40	9	4.9
	41-50	32	17.6
	≥51	139	76.4

The majority of patients (76.4%) were aged 51 years or older. The mean age of the study

population was 60.1 ± 12.4 years (range: 25-92 years).

Table 2: Glycemic Status (HbA1c) Distribution

HbA1c Category	Range	Frequency (n)	Percentage (%)
Normal	<5.7%	64	35.2
Prediabetes	5.7-6.4%	35	19.2
Diabetes	≥6.5%	83	45.6

Among 182 ACS patients with elevated troponin, 83 (45.6%) had HbA1c levels in the diabetic range (≥6.5%), while 35 (19.2%) were in the

prediabetic range. Only 64 (35.2%) patients had normal glycemic status based on HbA1c.

Table 3: HbA1c Distribution by Gender

HbA1c Category	Male (n, %)	Female (n, %)	Total (n, %)
Normal (<5.7%)	51 (41.1%)	13 (22.4%)	64 (35.2%)
Prediabetes (5.7-6.4%)	21 (16.9%)	14 (24.1%)	35 (19.2%)
Diabetes (≥6.5%)	52 (42.0%)	31 (53.5%)	83 (45.6%)

Females demonstrated a higher proportion of diabetes (53.5%) compared to males (42.0%),

while males had a higher proportion of normal HbA1c (41.1% vs. 22.4%) table 3.

Table 4: Pearson Correlation between Troponin and HbA1c

Parameter	Troponin	HbA1c
Troponin		
Pearson Correlation	1	0.068
Sig. (2-tailed)		0.361
HbA1c		
Pearson Correlation	0.068	1
Sig. (2-tailed)	0.361	

Pearson correlation analysis revealed a weak positive relationship between troponin and

HbA1c ($r = 0.068$) that did not reach statistical significance ($p = 0.361$) table 4.

Table 5: Distribution of Patients by LDL and HDL Status

HDL Status	LDL Status	Frequency (n)	Percentage (%)
Normal HDL	Normal LDL	52	28.6
	Elevated LDL	49	26.9
	Subtotal	101	55.5
Decreased HDL	Normal LDL	59	32.4
	Elevated LDL	22	12.1
	Subtotal	81	44.5

Among patients with normal HDL (n=101), LDL distribution was nearly equal (52 normal LDL, 49 elevated LDL). In contrast, among patients with

decreased HDL (n=81), the majority (59 patients, 72.8%) had normal LDL, while only 22 (27.2%) had elevated LDL table 5.

Table 6: Distribution of Extreme Troponin Elevations ($\geq 50,000$ ng/L) by Lipid Status

HDL Status	LDL Status	Patients with Troponin $\geq 50,000$ (n)	% of Total Extreme Elevations
Normal HDL	Normal LDL	14	35.9%
	Elevated LDL	11	28.2%
	Subtotal	25	64.1%
Decreased HDL	Normal LDL	13	33.3%
	Elevated LDL	1	2.6%
	Subtotal	14	35.9%

Of 39 patients with extreme troponin elevation ($\geq 50,000$ ng/L), 25 (64.1%) had normal HDL and 14 (35.9%) had decreased HDL. Notably, among patients with decreased HDL and extreme

troponin elevation, 13 of 14 (92.9%) had normal LDL, suggesting that low HDL may be associated with severe myocardial injury independent of LDL status table 6.

Table 7: Chi-Square Test for Association between LDL and HDL Categories

Test	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	2.847	1	0.092
Continuity Correction	2.341	1	0.126
Likelihood Ratio	2.882	1	0.090
Linear-by-Linear Association	2.831	1	0.092

The Chi-square analysis did not demonstrate a statistically significant association between LDL and HDL categories ($p = 0.092$). However, the

trend suggests a potential relationship warranting further investigation table 7.

Table 8: Summary of Key Findings

Parameter	Finding	Statistical Value	Interpretation
HbA1c-Troponin Correlation	Weak positive correlation	$r = 0.068$, $p = 0.361$	Not significant
Diabetes Prevalence	45.6% of ACS patients	83/182 patients	High diabetes burden
Elevated LDL	39.0% of patients	71/182 patients	Moderate dyslipidemia
Decreased HDL	44.5% of patients	81/182 patients	High prevalence
Extreme Troponin Pattern	Low HDL + Normal LDL	13/14 (92.9%)	HDL may modulate injury severity

DISCUSSION

This study investigated the correlation between high troponin levels and metabolic parameters (HbA1c, LDL, HDL) in 182 patients presenting with Acute Coronary Syndrome at a tertiary care cardiac center in Peshawar, Pakistan. The demographic characteristics of our study population align with established epidemiological patterns of ACS, with male predominance

(68.1%) consistent with global literature reporting higher ACS incidence in males compared to females [18], and similar findings reported by Sher et al. in their Pakistani cohort, where 69% of ACS patients were male [3]. The mean age of our study population (60.1 ± 12.4 years) corresponds with the typical age distribution of ACS, with the majority (76.4%)

aged ≥ 51 years, comparable to findings reported by Dubey et al., who documented a mean age of 58.7 years in their ACS cohort [6].

A striking finding of our study is the high prevalence of dysglycemia among ACS patients, with 45.6% having HbA1c levels in the diabetic range ($\geq 6.5\%$) and an additional 19.2% in the prediabetic range, collectively indicating that 64.8% of patients demonstrated abnormal glycemic status. This is substantially higher than the general population diabetes prevalence in Pakistan (26.7%) reported by the International Diabetes Federation [19], underscoring the strong association between diabetes and ACS. Our findings align with those of Ertem et al., who reported that 42% of their ACS patients had undiagnosed diabetes based on admission HbA1c [7], and similarly, Dubey et al. documented that 24.5% of ACS patients with no prior diabetes history were found to have HbA1c $\geq 6.4\%$ upon admission [6]. The higher proportion of diabetes among females (53.5%) compared to males (42.0%) in our study is consistent with research by Peters et al., who demonstrated that women with diabetes have a 44% greater risk of incident coronary heart disease compared to men with diabetes [20].

Our primary analysis revealed a weak positive correlation between troponin and HbA1c ($r = 0.068$) that did not reach statistical significance ($p = 0.361$), suggesting that while chronic hyperglycemia contributes to atherosclerosis progression, it may not directly determine the severity of acute myocardial injury as measured by troponin release. Troponin elevation in ACS primarily reflects acute myocardial necrosis resulting from plaque rupture and thrombotic occlusion influenced by multiple factors, including plaque vulnerability, thrombogenicity, and collateral circulation [21]. Additionally, confounding variables not measured in our study, including renal function, inflammatory status, and hemodynamic parameters, may influence both HbA1c and troponin levels [22]. Our findings are consistent with those of Sinna et al., who reported no significant correlation between HbA1c and troponin levels in their ACS cohort ($r = 0.12$, $p = 0.18$) [23], and similarly, a meta-

analysis by Liu et al. found that while elevated HbA1c predicted increased cardiovascular events post-ACS, it did not correlate with infarct size measured by troponin or cardiac enzymes [24].

The lipid profile distribution in our study revealed that 39.0% of patients had elevated LDL (≥ 130 mg/dL), while 44.5% had decreased HDL using gender-specific cutoffs. Among patients with decreased HDL, the majority (72.8%) had normal LDL, indicating that low HDL represents an independent risk factor for ACS even in the absence of elevated LDL, aligning with the concept of "residual cardiovascular risk" that persists despite achieving LDL targets [25]. The high prevalence of low HDL (44.5%) in our study population exceeds rates reported in Western cohorts, and the INTERHEART study documented population-attributable risks of 49.2% for abnormal lipids, with low HDL contributing significantly to this burden [26]. In South Asian populations, including Pakistanis, the prevalence of low HDL is particularly high due to genetic predisposition and lifestyle factors [27].

A notable finding of our study is the differential distribution of extreme troponin elevations ($\geq 50,000$ ng/L) across lipid categories, where among patients with extreme troponin elevation and decreased HDL, 13 of 14 (92.9%) had normal LDL, suggesting that low HDL may be associated with severe myocardial injury independent of LDL levels. HDL possesses pleiotropic cardioprotective properties beyond reverse cholesterol transport, encompassing antioxidant, anti-inflammatory, and antithrombotic effects [28], and low HDL may therefore promote more extensive myocardial injury through enhanced oxidative stress and inflammation during acute ischemia [29]. Additionally, HDL modulates endothelial function and promotes nitric oxide bioavailability, potentially influencing myocardial perfusion and ischemic tolerance [30]. Our findings align with those of Murphy et al., who demonstrated that low HDL independently predicted larger infarct size and worse outcomes in STEMI patients undergoing primary percutaneous coronary intervention [31], and

similarly, the MIRACL study showed that low HDL was associated with recurrent ischemic events independent of LDL levels [32].

The findings of this study have several important clinical implications. Comprehensive risk assessment in ACS should extend beyond LDL to include HDL and HbA1c, with the high prevalence of diabetes and low HDL in our population supporting routine measurement of these parameters in all ACS patients. Current guidelines recommend intensive statin therapy in all ACS patients regardless of baseline LDL [33], and our findings support this approach while suggesting that additional therapies targeting HDL and glycemic control may be beneficial in high-risk subgroups. The limited correlation between traditional metabolic markers and troponin severity underscores the need for novel biomarkers capturing additional pathophysiological dimensions, including inflammation and plaque vulnerability [34].

Strengths of our study include an adequate sample size providing sufficient statistical power for primary analyses, a comprehensive metabolic assessment with simultaneous measurement of troponin, HbA1c, and full lipid profile, a real-world clinical setting at a major tertiary cardiac center, and standardized laboratory methods with rigorous quality control. Limitations include a cross-sectional design precluding assessment of causal relationships, a single-center setting potentially limiting generalizability, convenience sampling with potential selection bias, unmeasured confounders including renal function and inflammatory markers, single troponin measurement where kinetics were not evaluated, and absence of coronary angiography data preventing correlation with anatomical disease severity. Based on our findings, future research directions include prospective cohort studies examining long-term outcomes, HDL functionality assessment investigating qualitative properties, multi-center studies across Pakistan, and interventional trials examining whether therapies targeting HDL or intensive glycemic control improve outcomes in high-risk subgroups.

CONCLUSION

This study demonstrates that while no statistically significant linear correlation exists between troponin and metabolic markers (HbA1c, LDL, HDL) in Acute Coronary Syndrome patients, the high burden of dysglycemia, with 45.6% of patients meeting diabetes criteria based on HbA1c underscores the critical importance of routine HbA1c screening in all ACS patients. The significant dyslipidemia burden with elevated LDL in 39.0% and decreased HDL in 44.5% of patients highlights the high prevalence of lipid abnormalities in the ACS population. Notably, the observed pattern of extreme troponin elevations in patients with low HDL, where 92.9% of such patients had normal LDL, suggests that HDL may modulate myocardial injury severity independent of LDL levels, possibly reflecting HDL's pleiotropic cardioprotective properties. These findings emphasize the complex pathophysiology of ACS beyond traditional risk factors and support comprehensive metabolic assessment in all ACS patients for optimal risk stratification and secondary prevention.

Conflict of interest: The authors declared no conflict of interest

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