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# Role of Triglycerides High Density Lipoprotein-Cholesterol Ratio in Atherosclerosis in Acute Coronary Syndrome Patients

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**Abstract**

Acute Coronary Syndrome (ACS) is a significant contributor to illness and death on a global scale. The primary cause of ACS is atherosclerosis, which defined as the accumulation of plaque in arterial walls. Dyslipidemia, namely increased triglycerides and decreased high-density lipoprotein cholesterol (HDL-C) levels, is a notable risk factor for the initiation and advancement of atherosclerosis. The TG/HDL-C ratio has identified as a powerful indicator of cardiovascular risk. Gaining a clear understanding of the significance of the TG/HDL-C ratio in atherosclerosis among ACS patients could offer significant knowledge regarding risk assessment and focused treatment approaches. To investigate the relationship between the Triglycerides to High-Density Lipoprotein Cholesterol (TG/HDL-C) ratio and the extent of atherosclerosis in patients with ACS. This cross-sectional study conducted at Zliten Medical Center, Libya, between January 2023 and December 2023 and included 165 The ACS patients categorized into three groups: Group I consisted of patients with unstable angina, Group II included patients with NSTEMI, and Group III comprised patients with STEMI. The participants had clinical and laboratory evaluation, including ECG and echocardiography. The TGs/HDL-C ratio measured in all cases. The mean TG/HDL-C ratio was significantly higher in patients with STEMI than those with NSTEMI and those with unstable angina indicating that TG/HDL-C ratio increases with increase the severity of ACS. The TG/HDL-C ratio is associated with the severity of ACS, with higher ratios observed in STEMI patients. The ratio shows promise as a simple and readily available marker for risk stratification in ACS.

**Keywords:** Acute Coronary Syndrome, ST-Elevation Myocardial Infarction, Non-ST-Elevation Myocardial Infarction, Unstable Angina, Triglycerides/High Density Lipoprotein-Cholesterol Ratio, Atherosclerosis.

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## 1. Introduction

Among the world's greatest causes of death acute coronary syndrome, or ACS, is liable for millions of fatalities [1]. This category includes the clinical manifestations of three distinct cardiac conditions: unstable angina (UA), non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI) [2]. Acute myocardial infarction (AMI) is a multifactorial disorder with various contributing factors. However, several investigations have confirmed a connection between the illness and an abnormality in lipid metabolism. Dyslipidemia, which includes high amounts of Triglycerides (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), or reduced levels of high-density lipoprotein cholesterol (HDL-C) all greatly increase the risk of developing coronary artery disease (CAD) [3]. This condition has garnered significant interest from medical professionals. According to recent research, TG/HDL-C ratio, for instance, has demonstrated to be a more effective method of determining the risk of cardiovascular disease than specific lipid characteristics including TG, TC, LDL-C, and HDL-C [4]. The TG/HDL-C ratio, sometimes referred to, as the TG to HDL-C ratio, is a metric used to assess atherosclerosis risk and has demonstrated a robust association with myocardial infarction and coronary heart disease [5].

Growing evidence from recent studies supports the usefulness of the TG/HDL-C ratio as a dependable and accessible measure for insulin resistance and metabolic syndrome [6-7]. However, it is currently unclear to what degree the TG/HDL-C ratio might predict the likelihood of repeat revascularization in ACS patients who have undergone a successful initial PCI in the age of drug-eluting stents (DES) [5]. More specifically, there exists a noteworthy correlation between insulin resistance and central obesity and a descriptive measure known as the ratio of high-density lipoprotein cholesterol to triglycerides (TG/HDL-C). Both of these characteristics are constituents of the Metabolic Syndrome (MetS), which augments the susceptibility to cardiovascular disease (CVD). The anti-inflammatory and antioxidant qualities of HDL-C have been linked to its ability to protect the heart [8]. Lower levels of HDL-C increase the risk of cardiovascular disease (CVD), however therapies specifically aimed at increasing HDL-C levels have not has demonstrated effective decrease the likelihood of cardiovascular disease. Atherosclerosis is a major cause of death for people with end-stage renal illness since it is much more common (about 10–30 times) in this population than in the overall population [9]. An increased blood triglyceride ratio increased mortality rates in the general population and Elevated levels of HDL cholesterol have been linked to an increased risk of coronary heart disease [10]. An examination done in the past on 973 individuals receiving peritoneal dialysis (PD) revealed that a greater ratio of serum triglycerides to HDL cholesterol (TG/HDL-C) was an independent predictor of mortality from all causes and cardiovascular disease Cardiovascular disease (CVD) affects both young and older patients with Parkinson's disease (PD) [11]. The TG/HDL-C ratio appears to be a useful and accurate tool for assessing insulin resistance and metabolic syndrome, according to mounting data [7]. Based on the research that is currently available, the TG/HDL-C ratio may use to as a valuable biomarker for assessing the risk of cardiovascular disease (CVD), along with its associated morbidity and mortality. Moreover, it might be helpful in developing primary and secondary prevention methods [12].

## 2. Subjects and Methods

This study conducted at Zliten Medical Center between January 2023 and December 2023. The study's target population was adult patients who hospitalized and diagnosed with the terms unstable angina, non-ST-elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI) refer to the acute coronary syndrome (ACS). Everyone provided written informed consent prior to enrollment. Inclusion Criteria included patients diagnosed Confirmed cases of acute coronary syndrome include unstable angina, STEMI, and NSTEMI are determined based on clinical symptoms, ECG abnormalities, and increased cardiac biomarkers. This study includes patients who are 18 years of age or older. Exclusion Criteria included prior history of chronic inflammatory diseases or autoimmune disorders, ongoing Administration of lipid-lowering medications, such as statins, for the purpose of treatment more than six months before admission, severe renal or hepatic impairment, known familial hyperlipidemia and recent history (within 3 months) of major surgery or trauma. All participants underwent the following: The baseline clinical assessment involves gathering information about the patient's age, gender, and thorough medical history (including any past cardiovascular events, diabetes, hypertension, smoking status, and family history of cardiovascular disease).

As well as doing an assessment of the body's physical condition, this includes the assessment of blood pressure, body mass index, and heart rate. Within twenty-four hours of admission, blood samples collected while the patient was fasting. These samples were analyzed to measure different parameters, such as triglycerides, LDL-C, HDL-C, and total cholesterol, as well as inflammatory markers like interleukin-6 (IL-6) and C-reactive protein (CRP), and other biochemical parameters (blood glucose, creatinine, and liver function tests) [13]. Coronary Angiography a diagnostic procedure was conducted within 48 hours of admission to assess determine how severe the coronary artery disease. The degree of atherosclerosis was measured using the Gensini score. Echocardiography An assessment was conducted to evaluate the function of the left ventricle and identify any irregularities in wall motion. Carotid Intima-Media Thickness (CIMT) Subclinical atherosclerosis evaluated by measuring it using high-resolution B-mode ultrasonography.

### *2.1. Calculation of TG/HDL-C Ratio*

The TG/HDL-C ratio calculated by dividing the concentration of triglycerides in the blood (mg/dL) by the concentration of HDL cholesterol (mg/dL). For additional testing, the patients categorized into quartiles according to their TG/HDL-C ratios.

### *2.2. Statistical design*

The study results were gathered, examined, organized, and summarized by statistical analysis using SPSS version 26.

## 3. Results and discussion

### *3.1. Results*

There was no discernible disparity among the three groups that examined in relation to baseline data except age that was higher in STEMI than UA and NSTEMI group (Table 1). Vital indicators did not significantly differ between the groups under study (Table 2). Comparing the study groups in terms of lower leg edema and chest examination revealed no discernible differences (Table 3). There were notable variations between the three groups under investigation with respect to SWMA of the left ventricle and diastolic dysfunction that were higher in STEMI than UA and NSTEMI. The ejection fraction indicated a statistically significant difference among the three groups under investigation; it was notably lower in the STEMI group when compared to UA and NSTEMI group (Table 4). The three groups under investigation did not significantly differ in terms of renal function tests, such as serum creatinine and urea levels (Table 5). Regarding cholesterol, TG, and LDL, there was a significant difference between the three groups under study. These parameters were significantly higher in the STEMI group compared to the UA group and higher in the NSTEMI group compared to the UA group, while HDL was significantly lower in the STEMI group compared to the UA group and significantly lower in the NSTEMI group compared to the UA group (Table 6). Patients with STEMI had a mean TG/HDL-C ratio that was considerably greater than that of patients with NSTEMI and unstable angina, suggesting that the TG/HDL-C ratio rises as ACS severity increases (Table 7). Age, pulse, urea, TG, and HDL-C showed substantial negative correlations with TG/HDL-C ratios and significant positive correlations with each other (Table 8).TG/HDL-C ratio at a cut-off 3.65-showed sensitivity 79.3%, specificity 55.2% with AUC 0.744 in prediction of patients with STEMI (Table 9).

### *3.2. Discussion*

Coronary heart disease is an escalating worldwide public health concern and the primary contributor to mortality on a global scale. It is crucial to analyze risks early on. Enhances the prevention and treatment of coronary heart disease (CHD) [14]. Three categories of patients were included in the study: those with unstable angina, NSTEMI and STEMI. 165 individuals were diagnosed with acute coronary syndrome (ACS). With the exception of age, which was higher in the STEMI group than in the UA and NSTEMI groups, the baseline characteristics did not show any discernible differences between the groups. This discovery is consistent with other research that has documented a correlation between older age and an increased probability of suffering from ST-elevation myocardial infarction (STEMI) [15-16]. Comorbidities such as smoking, hypertension, and diabetes mellitus are common found to be similar across all groups in our investigation, with no significant differences observed. This is in contrast to Zghebi et al., [17] Scientists have shown that these risk factors are more common in people who have been diagnosed with STEMI. The mismatch could attributed to the limited size of our sample or variations in the allocation of risk factors among various locations. Our study's findings showed that there were no appreciable differences in the three groups' vital signs or chest examination findings.

This implies that these characteristics may lack reliability in distinguishing between different forms of ACS upon initial presentation, which aligns with the discoveries of Ahmed et al. [18], It has been found that the initial vital signs have poor predictive value in determining the kind of ACS. The echocardiographic data in our investigation exhibited notable disparities among the groups. As expected given the larger degree of myocardial injury in STEMI, the ejection fraction was significantly lower in the STEMI group when compared to the UA and NSTEMI groups. This outcome is consistent with the study by Ahmed et al. [18], which individuals documented decreased ejection fractions in STEMI patients in comparison to other kinds of ACS. Moreover, Wohlfahrt et al. [19] the objective was to provide a comprehensive description of the epidemiology of EF following myocardial infarction. According to their findings, patients with an ejection fraction (EF) of less than 40% were frequently individuals who experienced subacute and anterior ST-elevation myocardial infarction (STEMI). Our investigation found that the occurrence of stress-induced wall motion abnormalities (SWMA) was notably greater in the STEMI group, indicating a more severe ischemia injury in these patients. The STEMI group had a higher prevalence of diastolic dysfunction, indicating a potential correlation between the severity of ACS and impairment of diastolic function, as previously documented by Mukhopadhyay et al. [20]. The lipid profile analysis in our study revealed substantial variances across the three groups.

Compared to the group that received uric acid (UA), Low-density lipoprotein cholesterol (LDL-C), total cholesterol, and triglycerides were all considerably higher in the STEMI group. The STEMI group, however, exhibited noticeably decreased HDL-C levels. These findings align with the recognized function of dyslipidemia in the development of ACS and corroborate the outcomes of Kumar et al. [21], who identified a lipid profile that was more likely to cause atherosclerosis in patients with STEMI. Krintus et al. [22] the study found that patients with ACS had lower levels of HDL-C and greater levels of TG compared to healthy individuals. However, it is important to note that these values were still within the acceptable ranges. Di Angelantonio et al. [23]indicated that both Elevated levels of triglycerides (TG) and a greater chance of heart disease is associated with lower levels of HDL-C (high-density lipoprotein cholesterol). While HDL-C has protective effects against atherosclerosis and vascular damage, it found to related with a 22% decrease in the likelihood of developing coronary artery disease (CAD) for each one standard deviation increase of 0.38 mmol/L. Conversely, the likelihood of developing coronary artery disease rose by 37% (with a 95% confidence interval of 31-42%) for every one standard deviation increase in logarithm of triglyceride levels. Gorecki et al [24] Total cholesterol (TC) and LDL were higher in patients with a severe clinical course of infarction than in patients with a straightforward course. These findings demonstrate a strong link between a poor prognosis and elevated levels of these biomarkers during the first 24 hours following an AMI.

According to an analysis of the lipid profile data from 97 patients with unstable angina and NSTEMI, the HDL level has additional predictive value for recurrent episodes during hospitalization in non-ST-elevation acute coronary syndromes, in contrast to LDL and TG [25]. Khan et al. [26] the objective was to evaluate the lipid profiles of patients with AMI and chest pain patients in relation to individuals without any cardiac conditions.Their results showed that when patients with STEMI and NSTEMI showed significant reductions in TC, LDL, and HDL as compared to normal values. Nevertheless, individuals experiencing chest discomfort did not demonstrate any noteworthy changes in these lipids. There were no notable variations in the levels of serum TG among the different study groups. The researchers postulated that dietary adjustments or metabolic changes during the acute crisis were responsible for the drop in blood lipids in patients suffering from AMI. The lipids data, however, defy the widely accepted notion that dietary lipid restriction can lower the incidence of AMI.Our research's primary finding is that the TG/HDL-C ratio varies significantly between the groups. Patients with STEMI had the highest ratio magnitude, followed by patients with NSTEMI. Patients with UA had the lowest ratio magnitude. The results point to a rise in the TG/HDL-C ratio as ACS severity increases. Luz et al. [27], which discovered a link between a larger TG/HDL-C ratio and a more severe degree of coronary artery disease, is consistent with our findings.

Koide et al. [28] Indicated that a higher TG/HDL-C ratio was linked to the existence of high-risk coronary plaques, which could potentially forecast the eventual development of acute coronary syndrome. Krintus et al. [22] according to the study, the patients' diagnoses with ACS exhibited median values of TG:HDL-C that were nearly double those of the control group (2.77 (1.88–4.08) vs. 1.47 (0.99–2.08); $p < 0.0001$). Chen et al. [29] Atherosclerotic cardiovascular events were approximately 1.5 times more common in the population as a whole in the group with the highest ratio of cholesterol from high density lipoprotein to triglycerides than in the group with the lowest ratio. Additionally, Sultani et al. [30] According to the study, individuals with a high risk of cardiovascular events had a significantly higher likelihood of experiencing such events over a five-year period if their triglycerides to HDL-C had a ratio of at least 2.5. This association persisted even after taking into consideration conventional coronary risk factors and the degree of coronary artery disease as assessed by angiography. Jeppesen et al. [31] According to earlier research, the ratio of high-density lipoprotein to triglycerides is a specific indicator of cardiovascular death and coronary heart disease risk. Higher ratios of triglycerides to HDL cholesterol were discovered to be a substantial predictor of having a heart attack by Gaziano et al. [32] using a case-control design.

**Table 1:** Baseline data and Comorbidities among studied groups

| | | Group I: patients with unstable angina (n=13) | Group II: patients with NSTEMI (n=38) | Group III: patients with STEMI (n=99) | P value | P value |
|---|---|---|---|---|---|---|
| **Age (years)** | **Mean ± SD** | 48.76±7.37 | 52.84±9.49 | 60.44±9.93 | **0.001** | P1=0.19<br>P2=**0.001**<br>P3=**0.001** |
| | **Range** | 37-62 | 36-71 | 38-81 | | |
| **Sex** | **Male** | 8 (61.5%) | 23 (60.5%) | 62 (62.6%) | 0.975 | |
| | **Female** | 5 (38.5%) | 15 (39.5%) | 37 (37.4%) | | |
| **DM** | **Yes** | 6 (46.20%) | 13 (34.20%) | 47 (47.50%) | 0.375 | |
| | **No** | 7 (53.80%) | 25 (65.80%) | 52 (52.50%) | | |
| **HTN** | **Yes** | 9 (69.20%) | 26 (68.40%) | 66 (66.70%) | 0.97 | |
| | **No** | 4 (30.80%) | 12 (31.60%) | 33 (33.30%) | | |
| **Smoking** | **Yes** | 6 (46.20%) | 24 (63.20%) | 57 (57.60%) | 0.562 | |
| | **No** | 7 (53.80%) | 14 (36.80%) | 42 (42.40%) | | |
| **Family history of CAD** | **Yes** | 2 (15.40%) | 5 (13.20%) | 16 (16.20%) | 0.911 | |
| | **No** | 11 (84.60%) | 33 (86.80%) | 83 (83.80%) | | |

DM: Diabetes mellitus, HTN: Hypertension, P1: I and II, P2: I and III, P3: II and III

**Table 2:** Vital signs among studied groups

| | | Group I: patients with unstable angina (n=13) | Group II: patients with NSTEMI (n=38) | Group III: patients with STEMI (n=99) | P value |
|---|---|---|---|---|---|
| **SBP (mm/Hg)** | **Mean ± SD** | 134.23±12.39 | 133.94±12.31 | 133.53±12.33 | 0.972 |
| | **Range** | 110-150 | 110-155 | 110-150 | |
| **DBP (mm/Hg)** | **Mean ± SD** | 86.92±8.54 | 87.65±6.92 | 86.46±7.63 | 0.709 |
| | **Range** | 70-100 | 75-100 | 70-100 | |
| **Pulse (beats/min)** | **Mean ± SD** | 81.61±6.21 | 83.18±6.80 | 84.53±7.24 | 0.282 |
| | **Range** | 71-91 | 72-97 | 71-79 | |

SBP: systolic blood pressure, DBP: diastolic blood pressure

**Table 3:** Chest examination and Lower limb edema among studied groups

| | | Group I: patients with unstable angina (n=13) | Group II: patients with NSTEMI (n=38) | Group III: patients with STEMI (n=99) | P value |
|---|---|---|---|---|---|
| **Chest examination** | **Yes** | 1 (7.70%) | 7 (18.40%) | 11 (11.10%) | 0.445 |
| | **No** | 12 (92.30%) | 31 (81.60%) | 88 (88.90%) | |
| **Lower limb edema** | **Yes** | 1 (7.70%) | 3 (7.90%) | 11 (11.10%) | 0.822 |
| | **No** | 12 (92.30%) | 35 (92.10%) | 88 (88.90%) | |

**Table 4:** Echocardiographic data among studied groups

| | | Group I: patients with unstable angina (n=13) | Group II: patients with NSTEMI (n=38) | Group III: patients with STEMI (n=99) | P value | P value |
|---|---|---|---|---|---|---|
| **EF%** | **Mean±SD** | 56.15±3.62 | 49.13±5.67 | 45.92±6.30 | **0.001** | P1=**0.001**<br>P2=**0.001**<br>P3=**0.006** |
| | **Range** | 51-63 | 41-59 | 32-59 | | |
| **SWMA** | **Yes** | 7 (53.80%) | 32 (84.20%) | 99 (100.00%) | **0.001** | P1=**0.001**<br>P2=**0.001**<br>P3=**0.001** |
| | **No** | 6 (46.20%) | 6 (15.80%) | 0 (0.00%) | | |
| **Diastolic dysfunction** | **Yes** | 4 (30.80%) | 12 (31.60%) | 52 (52.50%) | **0.048** | P1=0.959<br>P2=0.136<br>P3=**0.027** |
| | **No** | 9 (69.20%) | 26 (68.40%) | 47 (47.50%) | | |

EF: Ejection fraction, SWMA: stress-induced wall motion abnormalities

**Table 5:** Kidney function test among studied groups

| | | Group I: patients with unstable angina (n=13) | Group II: patients with NSTEMI (n=38) | Group III: patients with STEMI (n=99) | P value |
|---|---|---|---|---|---|
| **Creatinine** | **Mean ± SD** | 0.94±0.17 | 1.05±0.21 | 1.02±0.22 | 0.313 |
| | **Range** | 0.7-1.3 | 0.7-1.4 | 0.7-1.4 | |
| **Urea** | **Mean ± SD** | 33.60±7.33 | 35.63±8.72 | 36.15±8.35 | 0.604 |
| | **Range** | 24-45 | 20-48 | 22-49 | |

**Table 6:** Lipid profile among studied groups

| | | Group I: patients with unstable angina (n=13) | Group II: patients with NSTEMI (n=38) | Group III: patients with STEMI (n=99) | P value | P value |
|---|---|---|---|---|---|---|
| TG (mg/dl) | Mean±SD | 130.23±11.82 | 157.158±21.85 | 173.32±30.45 | **0.001** | P1=**0.003**<br>P2=**0.001**<br>P3=**0.001** |
| | Range | 110-152 | 119-222 | 118-240 | | |
| TC (mg/dl) | Mean±SD | 171.84±12.13 | 178.44±17.52 | 191.26±30.85 | **0.007** | P1=0.446<br>P2=**0.016**<br>P3=**0.014** |
| | Range | 157-194 | 144-221 | 144-269 | | |
| HDL-C (mg/dl) | Mean±SD | 46.85±5.28 | 42.63±6.06 | 38.505±6.07 | **0.001** | P1=**0.028**<br>P2=**0.001**<br>P3=**0.001** |
| | Range | 39-56 | 31-57 | 30-57 | | |
| LDL-C (mg/dl) | Mean±SD | 98.92±14.31 | 104.39±19.33 | 119.97±32.53 | **0.003** | P1=0.552<br>P2=**0.014**<br>P3=**0.005** |
| | Range | 78-126 | 61-152 | 60-194 | | |

TC = total cholesterol, TG = triglyceride, HDL_C = High-density lipoprotein cholesterol, LDL-C = low-density lipoprotein cholesterol.

**Table 7:** TG/HDL-C ratio among studied groups

| | | Group I: patients with unstable angina (n=13) | Group II: patients with NSTEMI (n=38) | Group III: patients with STEMI (n=99) | P value | P value |
|---|---|---|---|---|---|---|
| TG/HDL-C | Mean ± SD | 2.8±0.36 | 3.75±0.71 | 4.35±1.04 | **0.001** | P1=**0.002**<br>P2=**0.001**<br>P3=**0.001** |
| | Range | 2.43-3.59 | 2.53-5.29 | 2.35-7.33 | | |

**Table 8:** Correlation between TG/HDL-C ratio and different parameters among the studied groups.

| | TG/HDL-C ratio | |
|---|---|---|
| | r | P value |
| **Age** | .202* | **0.013** |
| **SBP (mm/Hg)** | 0.154 | 0.06 |
| **DBP (mm/Hg)** | 0.157 | 0.056 |
| **Pulse (beats/min.)** | .260** | **0.001** |
| **Creatinine** | 0.113 | 0.168 |
| **Urea** | .193* | **0.018** |
| **TG (mg/dl)** | .791** | **0.001** |
| **TC (mg/dl)** | 0.15 | 0.066 |
| **HDL-C (mg/dl)** | -.688-** | **0.001** |
| **LDL (mg/dl)** | 0.139 | 0.089 |
| **EF (%)** | -.174-* | **0.033** |

**Table 9:** Receiver observing characteristic analysis of TG/HDL-C ratio for prediction of patients with STEMI

| | Cut-off | AUC | Sensitivity | Specificity | PPV | NPV | P value |
|---|---|---|---|---|---|---|---|
| **TG/HDL-C** | 3.65 | 0.744 | 79.3% | 55.2% | 73.7% | 62.7% | 0.001 |

Turak et al. [33] it discovered that who have people with essential hypertension who had higher risk was associated with TG/HDL-C ratios of dying and suffering from major adverse cardiovascular events (MACEs).Patients individuals with high TG/HDL-C ratios had increased amounts of remnant-C linked to the development of atherosclerosis and inflammation [34]. Prior research has established a connection between the TG/HDL-C ratio and outcomes related to coronary artery disease (CAD) in high-risk patients already have a recognized diagnosis of CAD [35]. Chen et al. [29] in the overall population, those in the highest TG/HDL-C tertile had an incidence of atherosclerotic cardiovascular events that was almost 1.5 times greater than in the lowest TG/HDL-C tertile, which was considered the baseline. Our results disagree with Ahmed et al. [18] there were 200 patients in total for the trial, divided into three groups: Groups A, B, and C comprised 24 patients with unstable angina, 124 patients with STEMI, and 52 patients with NSTEMI. The TG/HDL-c ratio did not significantly differ among the three groups, according to their findings ($p>0.05$). A favorable correlation between the ratio of TG to HDL-C and the research age, pulse rate, urea, and triglycerides, while showing a negative correlation with HDL-C. These findings offer additional understanding of the probable pathways that connect this ratio to the severity of ACS. These results suggest that the ratio of TG to HDL-C may not only indicate disruptions in lipid metabolism but possibly other pathophysiological processes related to ACS. According to the ROC analysis, we were able to predict STEMI with a sensitivity of 79.3% and specificity of 55.2% when we used a TG/HDL-C ratio cut-off of 3.65. After calculation, the area under the curve, or AUC, discovered to be 0.744.

The TG/HDL-C ratio can be helpful in assessing risk, but its predictive power is limited. Therefore, it should use in conjunction with other clinical and laboratory indications. Numerous investigations have revealed that the TG/HDL-C ratio is a useful predictor of future cardiovascular events, and it rises in people with diabetes mellitus or chronic renal disease [35-36]. Krintus et al. [22] According to the study, the TG:HDL-C ratio plus age was the most reliable independent predictor and classifier of the occurrence of ACS. It was determined that 2.28 was the ideal threshold for this ratio. The TG: HDL-C ratio demonstrated superior performance compared to other lipid measurements and ratios, allowing for the detection of an extra six participants with ACS. Gaziano et al. [32] proposed the TG:HDL-C ratio, and it has been proven that it is a notable and autonomous predictor of myocardial infarction, surpassing other indicators in strength. There are various constraints in our investigation. The limited sample size may restrict the applicability of our findings. Furthermore, we did not evaluate the long-term results, which could offer additional understanding exploring the prognostic importance of the TG/HDL-C ratio. Future studies should focus on analyzing these restrictions and investigating how the TG/HDL-C ratio might use to inform treatment choices and assess risk in individuals suffering from acute coronary syndrome (ACS).

## 4. Conclusions

Our study findings suggest a connection between the TG/HDL-C ratio and the severity of ACS. Specifically, we found that STEMI patients tend to have larger ratios. The ratio has potential as a straightforward and easily accessible indicator for risk categorization in ACS. Nevertheless, additional extensive, future research are required to verify these results and show the practicality of using The TG/HDL-C ratio is used in the management of individuals with acute coronary syndrome (ACS).

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