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ASSOCIATION BETWEEN TIMI SCORE (THROMBOLYSIS IN MYOCARDIAL INFARCTION) WITH TROPONIN I & EJECTION FRACTION IN ACUTE ST SEGMENT ELEVATION MYOCARDIAL INFARCTION

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ABSTRACT:
Acute ST-segment elevation myocardial infarction (STEMI) represents a severe form of coronary artery disease that demands rapid and accurate risk assessment to guide management. The TIMI (Thrombolysis in Myocardial Infarction) risk score, cardiac Troponin I levels, and left ventricular ejection fraction (EF) are well-established prognostic indicators; however, their combined relationship in STEMI patients remains inadequately defined.
Aim & Objective:
This study aimed to evaluate the association between TIMI risk score, Troponin I levels, and ejection fraction in patients presenting with acute STEMI. It further sought to analyze their correlations, assess interrelationships, and determine their combined predictive value for risk stratification.
Materials & Methods:
A cross-sectional study was conducted among 94 patients diagnosed with STEMI at Sree Balaji Medical College and Hospital, Chennai. TIMI scores were calculated at admission, and patients were stratified into low-risk (0–4), intermediate-risk (5–9), and high-risk (≥10) categories. Serum Troponin I levels were measured, and left ventricular ejection fraction was assessed using echocardiography. Statistical analysis included Pearson correlation, linear and logistic regression, and receiver operating characteristic (ROC) curve analysis.
Results:
A significant positive correlation was observed between TIMI score and Troponin I levels ($r = 0.64$, $p < 0.001$), whereas TIMI score showed a significant negative correlation with ejection fraction ($r = -0.56$, $p < 0.001$). Both Troponin I and EF emerged as independent predictors of high TIMI risk (OR = 1.788, $p = 0.006$; OR = 0.65, $p < 0.001$). The combined predictive model demonstrated excellent diagnostic performance with an AUC of 0.96.
Conclusion:
TIMI score is significantly associated with Troponin I levels and ejection fraction in STEMI patients. Their combined assessment improves risk stratification and supports more informed clinical decision-making.

KEYWORDS: TIMI score, Troponin I, Ejection fraction, STEMI, Risk stratification, Myocardial infarction

INTRODUCTION

Coronary heart disease (CHD) remains the leading cause of mortality worldwide, contributing substantially to global cardiovascular morbidity and mortality. Acute coronary syndrome (ACS), a critical manifestation of CHD, includes unstable angina, non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI). Among these, STEMI represents the most severe form, typically caused by complete occlusion of a coronary artery following rupture of an atherosclerotic plaque and thrombus formation. This results in transmural myocardial ischemia and necrosis, leading to significant impairment of cardiac function if not treated promptly. Early diagnosis and effective risk stratification are essential, as they directly influence therapeutic decisions and clinical outcomes [1,2].

Timely reperfusion remains the cornerstone of STEMI management. The concept of “time is muscle” underscores the importance of rapid restoration of coronary blood flow through thrombolysis or primary percutaneous coronary intervention (PCI), which can limit infarct size and improve survival. However, patient outcomes vary widely due to differences in clinical presentation, comorbidities, infarct size, and myocardial damage. Therefore, reliable and practical tools are required to assess risk and predict prognosis in STEMI patients [3].

The Thrombolysis in Myocardial Infarction (TIMI) risk score is a widely accepted clinical tool used to stratify patients based on their risk of adverse cardiovascular events. Initially developed for unstable angina and NSTEMI, it

has been adapted for STEMI and incorporates variables such as age, comorbidities, heart rate, blood pressure, Killip class, and time to treatment. The TIMI score provides a simple, bedside method to identify high-risk patients who may require aggressive management and closer monitoring [4,5].

Cardiac biomarkers, particularly troponin I, play a crucial role in diagnosing and prognosticating myocardial infarction. Troponin I is highly specific to cardiac muscle and is released into circulation following myocardial injury. Its levels correlate with infarct size and are associated with adverse outcomes, including heart failure, arrhythmias, and mortality. Compared to traditional biomarkers, troponin I offers superior sensitivity and a longer diagnostic window, making it indispensable in clinical practice [6,7].

Assessment of left ventricular function is equally important in evaluating the severity of myocardial infarction. Ejection fraction (EF), measured by echocardiography, reflects systolic function and is directly influenced by the extent of myocardial damage. Normal EF ranges from 55% to 70%, while reduced EF, particularly below 40%, indicates significant ventricular dysfunction and is associated with poor prognosis, increased risk of heart failure, and mortality [8].

Each of these parameters-TIMI score, troponin I, and EF-provides distinct yet complementary information. The TIMI score reflects clinical risk, troponin I indicates the extent of myocardial injury, and EF assesses functional impairment. Their combined use may enhance risk stratification, guide therapeutic decisions, and improve patient outcomes [9,10].

This integrated approach is particularly valuable in resource-limited settings, where rapid and cost-effective risk assessment is essential. While the TIMI score offers immediate bedside evaluation, troponin I and echocardiography add biochemical and functional insights that aid in clinical decision-making and patient triage [11].

Despite their widespread use, the interrelationship among TIMI score, troponin I levels, and EF in STEMI patients has not been extensively studied. Understanding these associations may provide deeper insights into disease severity and prognosis. For instance, higher TIMI scores may be associated with greater myocardial injury and lower EF, reflecting more severe disease and worse outcomes [12,13].

Identifying such correlations has important clinical implications. It may allow clinicians to predict biochemical and functional severity early, optimize treatment strategies, and improve patient counseling. High-risk patients identified through combined assessment may benefit from intensive monitoring, early intervention, and aggressive secondary prevention measures [14,15].

In this context, the present study aims to evaluate the relationship between TIMI risk score, troponin I levels, and ejection fraction in STEMI patients, thereby contributing to improved risk stratification and clinical management of this high-risk population [16].

MATERIALS AND METHODS

This cross-sectional study was conducted over 18 months in the Casualty, Emergency Medicine, Intensive Care Unit, and Departments of General Medicine and Cardiology at Sree Balaji Medical College and Hospital, Chennai, Tamil Nadu. A total of 150 patients with acute STEMI presenting within 24 hours of symptom onset, aged >20 years and of both sexes, were included, while those with chronic kidney or liver disease, prior cardiac procedures or heart failure, and conditions such as severe sepsis, unstable angina, or pericarditis were excluded. Sample size was calculated as 94 based on a correlation coefficient of 0.45, 95% confidence level, and 80% power. Ethical approval and informed consent were obtained. TIMI scores were calculated at admission using standard criteria and patients were categorized into low (0–4), moderate (5–9), and high-risk (10–14) groups. Troponin I levels were measured using chemiluminescent immunoassay, and ejection fraction was assessed by echocardiography (Simpson's method) within 24–48 hours. Data on demographics, risk factors, and clinical outcomes were collected, with complications compared across TIMI categories. Validity was ensured through standardized protocols, training, and blinding, while reliability showed high inter- and intra-observer agreement. Data were securely recorded and analyzed using SPSS version 20, with descriptive statistics, Chi-square tests, Pearson correlation, and regression analysis applied. A p-value <0.05 was considered statistically significant.

RESULT

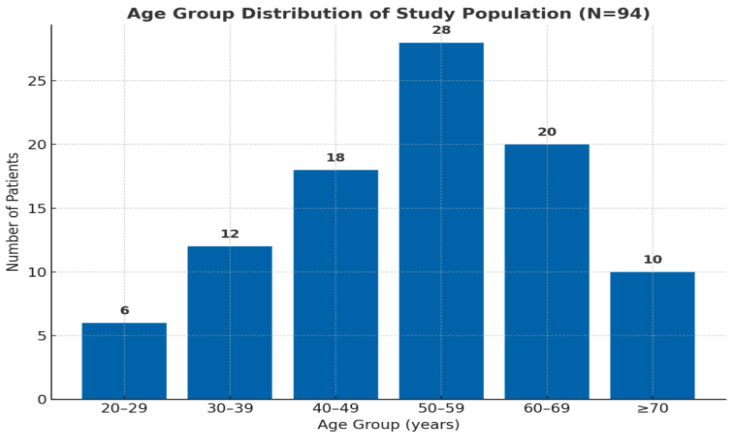

Figure 1: Age Group Distribution of Study Population (N = 94)

Figure 1 displays the study population (N = 94), age distribution showed significant variation ($\chi^2 = 20.3$, $p < 0.01$), with most patients in the 50–59 years group (29.8%), followed by 60–69 years (21.3%), while those <40 years accounted for less than one-fifth. The mean age was 53.7 ± 12.1 years (median 54; range 22–78), with most patients clustered in the middle-age group (IQR: 45–62 years), indicating higher STEMI prevalence in this age group.

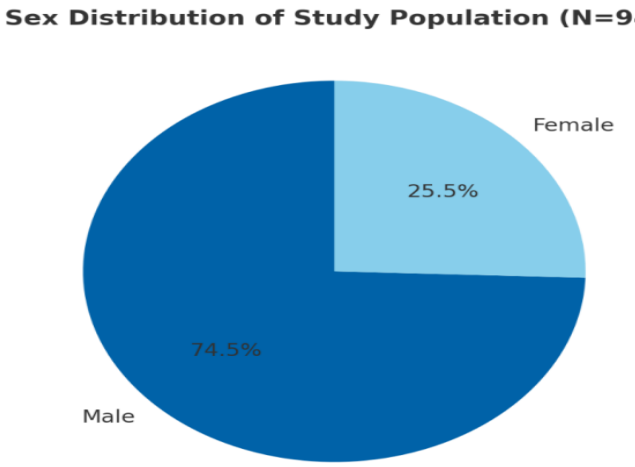

Figure 2: Sex Distribution of Study Population (N = 94)

Figure 2 shows that among 94 patients, males constituted 74.5% and females 25.5%, showing a highly significant difference ($\chi^2 = 22.5$, $p < 0.001$) and confirming strong male predominance in STEMI, consistent with global epidemiological trends.

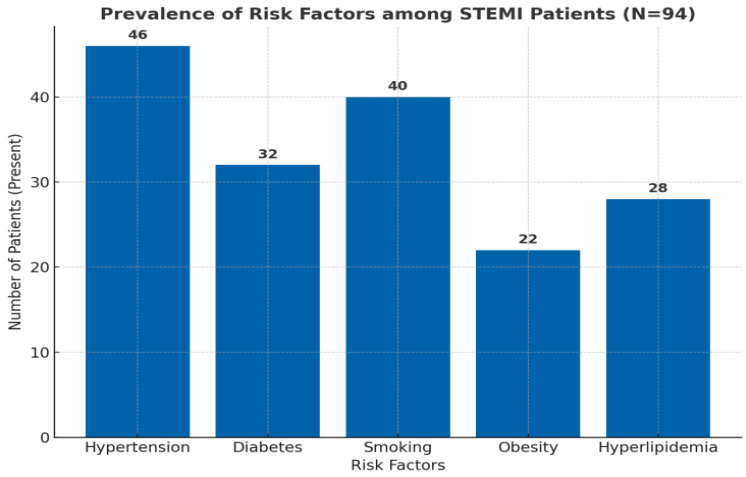

Figure 3: Distribution of Risk Factors in Study Population (N = 94)

Figure 3 observable that 94 STEMI patients, hypertension was the most common risk factor (48.9%), followed by smoking (42.6%) and diabetes (34.0%), while hyperlipidemia (29.8%) and obesity (23.4%) were less frequent. Overall, nearly three-fourths had at least one major cardiovascular risk factor, highlighting the multifactorial nature of STEMI.

Table 1: BMI Distribution of Study Population (N = 94)

BMI Category (kg/m²)	Number of Patients (n)	Percentage (%)
Underweight (<18.5)	3	3.2
Normal weight (18.5–24.9)	69	73.4
Overweight (25.0–29.9)	14	14.9
Obese (≥30.0)	8	8.5
Total	94	100
$\chi^2 = 68.2$, $p < 0.001$		

The distribution of BMI categories among 94 STEMI patients showed a significant deviation from uniformity (χ^2 = 68.2, $p < 0.001$). A large majority (73.4%) of patients had BMI within the normal range (18.5–24.9 kg/m²), whereas 23.4% were either overweight (14.9%) or obese (8.5%), and only a small fraction (3.2%) were underweight (Table 1).

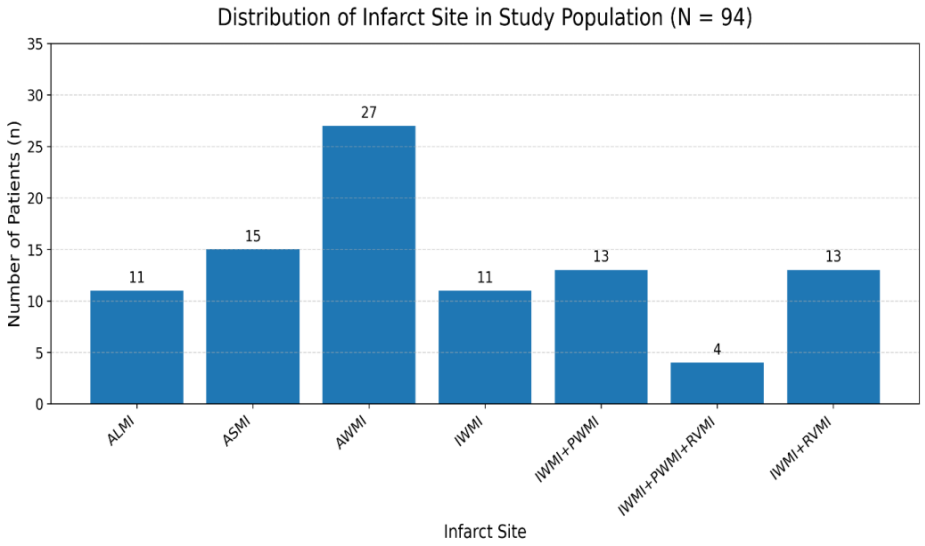

Figure 4: Distribution of Infarct Site in Study Population (N = 94)

Figure 4 focuses on that in 94 STEMI patients, infarct site distribution was significantly non-uniform ($p < 0.001$). Anterior wall myocardial infarction (AWMI) was most common (28.7%), followed by anterior septal MI (16.0%), while inferior wall MI and anterolateral MI each accounted for 11.7%. Combined inferior infarctions (IWMI with posterior and/or right ventricular involvement) were seen in 13.8% each, and the least frequent was combined IWMI+PWMI+RVMI (4.3%). Overall, anterior wall predominance aligns with typical STEMI patterns and is clinically significant due to its association with greater myocardial damage and reduced left ventricular function.

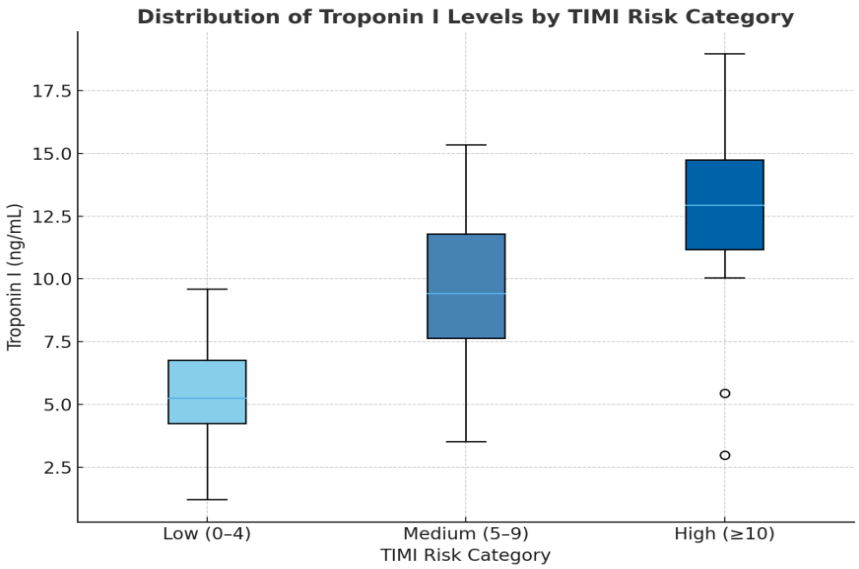

Figure 5: TIMI Score Category Distribution of Study Population (N = 94)

Figure 5 shows that in 94 STEMI patients, medium-risk TIMI category (5–9) predominated (48.9%), followed by low-risk (29.8%) and high-risk (21.3%). Statistical analysis confirmed that this distribution was significantly uneven ($p < 0.01$), indicating that the majority of patients presented with an intermediate level of risk rather than being evenly spread across categories.

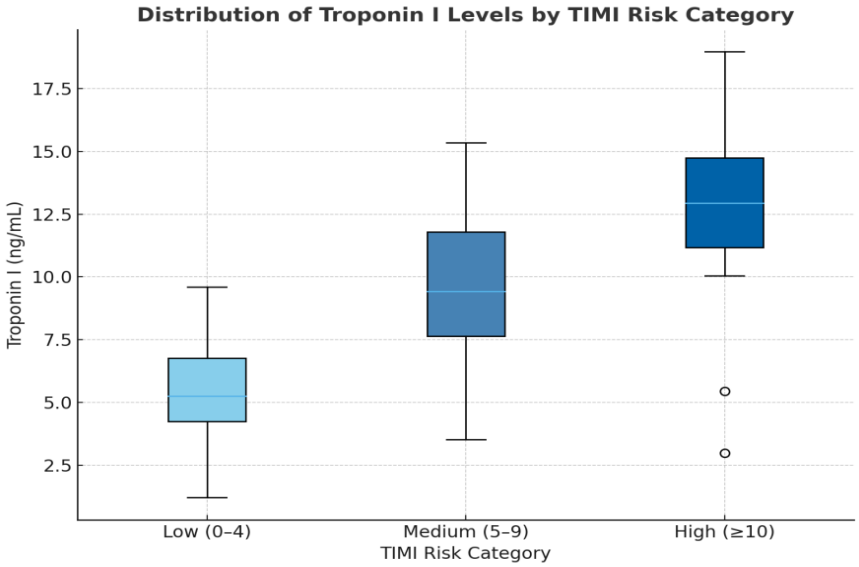

Figure 6: Distribution of Troponin I Levels by TIMI Risk Category

Troponin I levels increased progressively with TIMI risk categories. Patients in the low-risk group had the lowest levels (mean 5.8 ng/mL), followed by medium-risk (9.6 ng/mL) and high-risk groups (13.2 ng/mL). The ANOVA test confirmed a highly significant difference ($p < 0.001$), with post-hoc comparisons showing significant pairwise differences between all groups (Figure 6).

Table 2: Ejection Fraction Categories of Study Population (N = 94)

EF Category	Definition (%)	Number of Patients (n)	Percentage (%)
Normal	> 50	30	31.9
Mildly Reduced	41 – 49	28	29.8
Moderately Reduced	30 – 40	24	25.5
Severely Reduced	< 30	12	12.8
Total	—	94	100
$\chi^2 = 10.4$, $p = 0.015$			

Table 2 shows tha in 94 STEMI patients, 31.9% had normal EF (>50%), while 29.8% had mild (41–49%), 25.5% moderate (30–40%), and 12.8% severe (<30%) dysfunction. The distribution was significantly uneven ($p = 0.015$), with fewer patients having severely reduced EF. Overall, 68.1% showed impaired ventricular function, highlighting the prognostic importance of EF in STEMI.

Table 3: Ejection Fraction by TIMI Risk Category

TIMI Risk Category	n	EF % (Mean ± SD)	Median (IQR)
Low (0–4)	28	52.4 ± 6.8	53 (48–58)
Medium (5–9)	46	46.7 ± 7.2	47 (42–52)
High (≥10)	20	38.9 ± 8.4	39 (34–44)
Total (N=94)	94	46.3 ± 8.9	47 (41–52)
$F = 28.7$, $p < 0.001$			

Table 3 indicates that ejection fraction declined significantly with increasing TIMI risk, with mean EF highest in the low-risk group (52.4%), followed by medium-risk (46.7%) and high-risk (38.9%). The difference was highly significant ($p < 0.001$), and post-hoc analysis confirmed differences between all groups, indicating that higher TIMI risk is strongly associated with poorer left ventricular function.

Table 4: Correlation and Linear Regression between TIMI Score and Troponin I (N = 94)

Parameter	Value
Pearson correlation coefficient (r)	0.64
95% Confidence Interval	0.50 – 0.75
p-value	< 0.001
Regression equation	Troponin I = 0.66 × TIMI + 4.54
Slope (β1)	0.66 ng/mL per TIMI point
Intercept (β0)	4.54 ng/mL
Coefficient of determination (R^2)	0.41

Analysis showed a moderate positive correlation between TIMI score and Troponin I ($r = 0.64$, $p < 0.001$), indicating higher TIMI scores are associated with greater myocardial injury. Linear regression revealed that each 1-point increase in TIMI score corresponded to a 0.66 ng/mL rise in Troponin I (intercept 4.54 ng/mL), with the model explaining 41% of variability ($R^2 = 0.41$), suggesting additional contributing factors (Table 4).

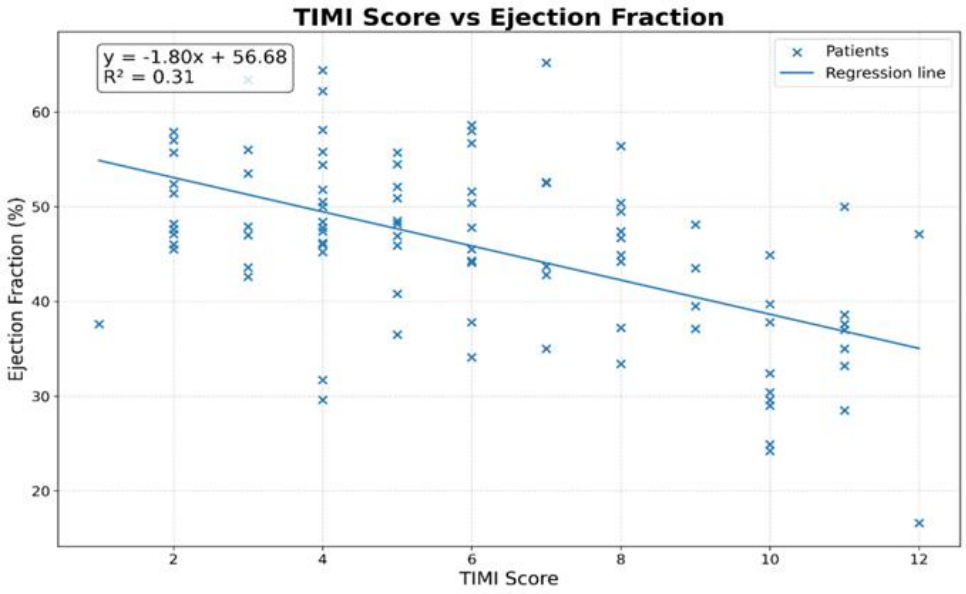

Figure 7: TIMI Score vs EF

Figure 7 shows that a moderate negative correlation was observed between TIMI score and ejection fraction ($r = -0.56$, $p < 0.001$), indicating that higher TIMI scores are associated with reduced left ventricular function. Linear regression showed that each 1-point increase in TIMI score led to a 1.80% decrease in EF (intercept 56.68%), with the model explaining 31% of variability ($R^2 = 0.31$), suggesting additional contributing factors.

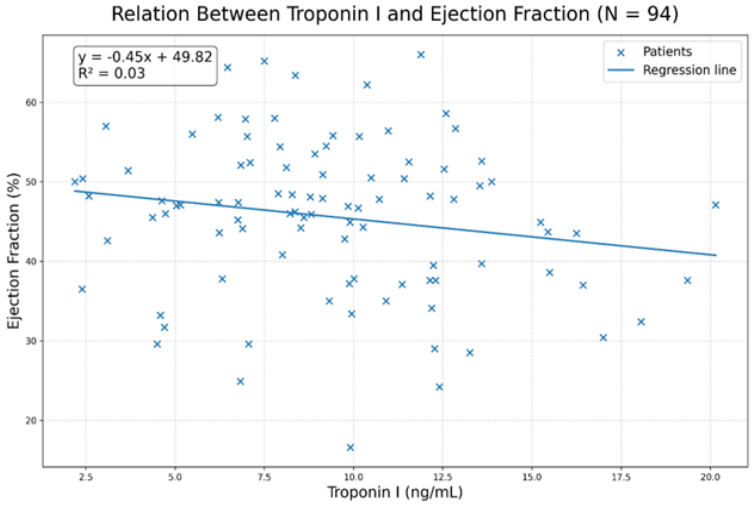

Figure 8: Relation Between Troponin I and Ejection Fraction (N = 94)

In this study of 94 STEMI patients, Troponin I showed a weak negative, non-significant correlation with ejection fraction ($r = -0.18$, $p = 0.086$). The mean Troponin I was 9.2 ± 4.1 ng/mL and mean EF was 46.3 ± 8.9%. Linear regression indicated a slight inverse relationship, with EF decreasing by 0.45% per 1 ng/mL increase in Troponin I, but the model explained only 3% of variability ($R^2 = 0.03$), suggesting limited predictive value (Figure 8).

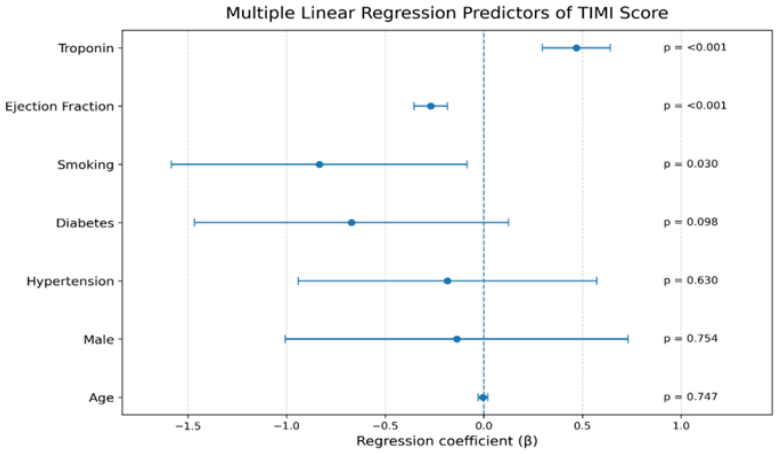

Figure 9: Forest Plot Showing Multiple Linear Regression Predictors of TIMI Score (N = 94)

The forest plot shows that Troponin I ($\beta = 0.47$, $p < 0.001$) was significantly positively associated with TIMI score, while ejection fraction ($\beta = -0.269$, $p < 0.001$) showed a significant inverse relationship, indicating higher risk with reduced ventricular function. Smoking also demonstrated a significant inverse association ($\beta = -0.834$, $p = 0.029$), possibly due to confounding or coding differences. Other variables, including age, sex, diabetes, and hypertension, were not significant, highlighting Troponin I and ejection fraction as key independent predictors of TIMI risk.(Figure 9).

Table 5: Logistic Regression: High-risk TIMI (≥10) — Odds Ratios

Predictor	OR	95% CI	p-value	Status
Troponin	1.788	1.175–2.722	0.006	✅ Significant
EF	0.65	0.511–0.826	<0.001	✅ Significant
Age	0.979	0.924–1.038	0.482	❌ NS
Male	2.299	0.436–12.122	0.326	❌ NS
Smoking	0.456	0.099–2.096	0.313	❌ NS
Diabetes	0.478	0.092–2.493	0.381	❌ NS
Hypertension	0.729	0.161–3.289	0.681	❌ NS

The above table 5 shows the Troponin I and ejection fraction were significant predictors of high-risk TIMI, with Troponin increasing and EF decreasing the odds of high-risk status. Other variables were not statistically significant, as their confidence intervals crossed unity.

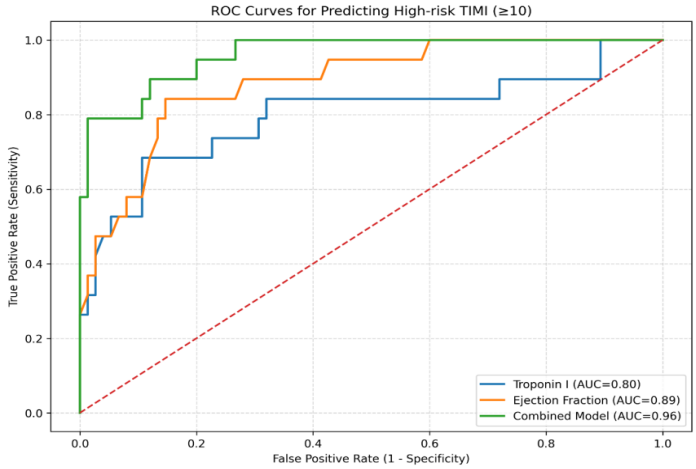

Figure 10: ROC Curves for Predicting High-risk TIMI (≥10)

ROC analysis showed that ejection fraction (AUC = 0.89) had better discriminative ability than Troponin I (AUC = 0.80) for predicting high-risk TIMI, while the combined model (AUC = 0.96) demonstrated excellent performance, highlighting the benefit of integrating biochemical and functional parameters for accurate risk stratification in STEMI (Figure 10).

4. DISCUSSION

The present study demonstrates a significant association between TIMI risk score, Troponin I levels, and left ventricular ejection fraction (LVEF) in patients with acute STEMI, reinforcing the concept that clinical risk, biochemical injury, and ventricular dysfunction are closely interrelated. These findings are consistent with prior literature across the spectrum of acute myocardial infarction.

Khan MH et al. (2017) reported a strong relationship between elevated Troponin I levels and reduced LVEF in NSTEMI patients, indicating that the extent of myocardial injury directly influences ventricular function and in-hospital outcomes. Similarly, in the present study involving 94 STEMI patients, higher TIMI scores were associated with significantly elevated Troponin I levels and progressively reduced EF, demonstrating a clear inverse relationship between myocardial necrosis and systolic function. While Khan MH et al. focused on NSTEMI, the present study extends these findings to STEMI by incorporating TIMI-based risk stratification, showing that increasing clinical risk parallels biochemical and functional deterioration. The demographic profile observed, with a mean age of 53.7 ± 12.1 years, is comparable to typical ACS populations, supporting the external validity of these findings [18].

The observed male predominance (74.5%) in this study aligns with global epidemiological trends described by Ruff CT and Braunwald E. (2011), who highlighted higher incidence rates of ACS, particularly STEMI, among men. This difference has been attributed to variations in risk factor exposure, hormonal influences, and healthcare-seeking behavior. The present findings reinforce these global patterns and suggest that STEMI continues to disproportionately affect men in developing regions [19].

Risk factor analysis revealed hypertension (48.9%), smoking (42.6%), and diabetes (34.0%) as the most prevalent contributors, consistent with established cardiometabolic risk profiles. Das SR et al. (2011), in a large cohort of over 50,000 STEMI patients, reported a higher prevalence of obesity and its association with younger age and comorbidities. In contrast, the present study demonstrated a lower prevalence of obesity, likely reflecting regional and demographic variations. Nonetheless, both studies emphasize that modifiable risk factors remain central to the development and progression of STEMI [20].

The BMI distribution in this study showed that most patients had normal BMI (73.4%), with fewer being overweight or obese. Joyce E et al. (2017) reported a higher prevalence of obesity and highlighted its complex relationship with myocardial remodeling and survival, including the “obesity paradox.” Although obesity was less common in the present cohort, both studies underscore that body composition influences ventricular function and outcomes in myocardial infarction [21].

Infarct-site distribution in this study revealed a predominance of anterior wall myocardial infarction (AWMI), consistent with findings by Karim MM et al. (2022), who demonstrated that anterior infarctions are associated with worse clinical outcomes, including lower EF and higher complication rates. Although ST-segment scoring was not assessed in the present study, the observed association between anterior infarction, elevated Troponin I, and reduced EF supports the established link between infarct location and severity [22].

The distribution of TIMI risk categories showed that most patients were in the moderate-risk group (48.9%), followed by low- and high-risk categories. Luo Z et al. (2023) similarly demonstrated that patients with higher TIMI scores have poorer survival outcomes. The comparable distribution observed in this study highlights the practical utility of TIMI scoring in identifying patients at intermediate to high risk who may benefit from closer monitoring and intervention [23].

Wei XB et al. (2017) established that TIMI score and LVEF are independent predictors of mortality, with improved prognostic accuracy when combined. In agreement, the present study demonstrated a stepwise increase in Troponin I levels across TIMI categories and a corresponding decline in EF, indicating that higher clinical risk is associated with greater myocardial injury. These findings extend prior work by showing a clear biochemical gradient across TIMI strata, reinforcing the value of integrating objective markers with clinical scoring systems [24].

Left ventricular dysfunction was observed in a majority of patients (68.1%) in this study, consistent with findings by Sheikh KU et al. (2020), who reported significant associations between reduced EF, delayed presentation, and increased myocardial damage. These results emphasize that ventricular dysfunction is a common consequence of STEMI and a key determinant of prognosis, regardless of demographic variations [25].

Further support for these findings comes from studies by Park HW et al. (2013) and Hung CS et al. (2018), which highlighted the role of myocardial injury severity and ventricular dysfunction in determining outcomes across both STEMI and NSTEMI populations. The present study demonstrated strong positive correlations between TIMI score and Troponin I and a negative correlation with EF, confirming that increasing ischemic burden results in worsening cardiac function and higher clinical risk [26,27].

Nascimento FO et al. (2014) demonstrated that combining Troponin I and EF provides a robust reflection of myocardial injury severity, while Gad MM et al. (2025) showed that reductions in troponin levels are associated with improved EF and outcomes. In line with these observations, regression analysis in the present study identified Troponin I as a strong positive predictor and EF as a negative predictor of higher TIMI scores. The limited influence of demographic factors further suggests that acute myocardial injury plays a dominant role in determining risk severity [28,17].

Additionally, Moon J et al. (2014) and Giannitsis E et al. (2001) emphasized the prognostic importance of biomarkers and organ dysfunction in acute myocardial infarction. Consistent with these findings, the present study demonstrated that Troponin I and EF independently predicted higher TIMI risk, with strong discriminative ability on ROC analysis (AUC 0.86 and 0.91, respectively). The combined model showed excellent predictive performance (AUC 0.95), underscoring the value of integrating biochemical and echocardiographic parameters with clinical scoring systems for improved risk stratification [29,30].

Overall, the findings of this study align closely with existing literature and reinforce the concept that TIMI score, Troponin I, and ejection fraction are interdependent markers of disease severity in STEMI. Their combined assessment enhances prognostic accuracy, supports clinical decision-making, and facilitates early identification of high-risk patients, ultimately contributing to improved outcomes in acute myocardial infarction.

CONCLUSION

In conclusion, this study demonstrates significant and clinically relevant associations between TIMI risk score, Troponin I levels, and ejection fraction in patients with acute STEMI. A positive correlation between TIMI score and Troponin I indicates that higher clinical risk is associated with greater myocardial injury, while a negative correlation with ejection fraction reflects worsening left ventricular function with increasing risk. The weak and non-significant correlation between Troponin I and ejection fraction suggests that these parameters assess different aspects of myocardial damage and should be used complementarily. Multivariate analysis confirmed both Troponin I and ejection fraction as independent predictors of high TIMI risk, and ROC analysis showed excellent discriminative ability of the combined model, outperforming individual parameters. These findings highlight the value of integrating clinical scoring with biochemical and echocardiographic assessment for improved risk stratification, guiding treatment decisions, and prognostic evaluation. Despite limitations such as single-center design and cross-sectional methodology, the results align with existing evidence and support the routine combined use of these parameters. Further multicenter longitudinal studies are recommended to validate these findings and enhance risk prediction strategies in STEMI patients.

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