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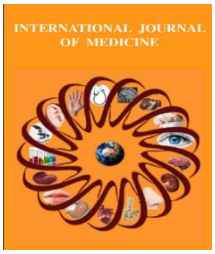

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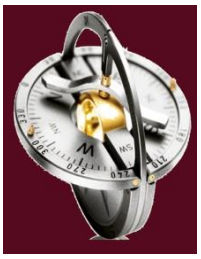

Original Research

The Study of Correlation between Troponin I, N Terminal Pro B Type Natriuretic Peptide and Left Ventricular Ejection Fraction in Stemi Patients with Mortality

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Abstract

Background: ST-segment elevation myocardial infarction (STEMI) remains a leading cause of mortality worldwide, including in India, where outcomes are worsened by late presentations and high prevalence of diabetes and other risk factors. Early risk stratification is essential to improve survival and guide therapy. Cardiac biomarkers such as Troponin I and N-terminal pro B-type Natriuretic Peptide (NT-proBNP), along with echocardiographic measurement of left ventricular ejection fraction (LVEF), are established prognostic tools. However, integrated data on their combined predictive value for short-term (1-month) mortality in Indian STEMI patients remain limited. Aim: To evaluate the correlation between admission Troponin I, NT-proBNP, and LVEF and their association with 1-month mortality in STEMI patients presenting to a tertiary care center in India. ***Methods:*** A prospective observational study enrolled 300 STEMI patients admitted within 24 hours of symptom onset. Admission blood samples measured Troponin I and NT-proBNP levels, while transthoracic echocardiography assessed LVEF within 24 hours. Patients were followed for 1 month to record mortality outcomes. Statistical analyses included correlation tests, logistic regression, and receiver operating characteristic curve analysis to assess predictive performance. ***Results:*** The 1-month mortality rate was 8.3%. Mortality increased significantly with higher Troponin I levels and NT-proBNP concentrations and with lower LVEF ($p < 0.001$). NT-proBNP demonstrated the strongest predictive ability (AUC 0.91), followed by LVEF (AUC 0.84) and Troponin I (AUC 0.71). In multivariate analysis, NT-proBNP was the most significant independent predictor of mortality. The combination of biomarkers and LVEF improved prognostic accuracy compared to any single parameter. Traditional risk factors such as HbA1c and LDL cholesterol showed no significant association with short-term mortality. ***Conclusions:*** Admission NT-proBNP, Troponin I, and LVEF provide complementary prognostic information in Indian STEMI patients, with NT-proBNP being the most powerful predictor of 1-month mortality. A multifaceted approach incorporating these markers can enhance early risk stratification and guide clinical management, underscoring the need for wider adoption of NT-proBNP testing in Indian cardiac care settings.

Keywords:

Introduction

ST-segment elevation myocardial infarction (STEMI) remains one of the most acute manifestations of ischemic heart disease and is associated with significant morbidity and mortality, despite advanced therapeutic strategies such as timely reperfusion and evidence- based adjunct therapies. Globally, ischemic heart disease is responsible for an estimated 9 million deaths annually, with STEMI being a major contributor to this burden[1]. Even in tertiary- care settings, in-hospital mortality rates for STEMI continue to range between 4-12% depending on the population structure, comorbidities, and access to prompt care[2]. In the wake of such statistics, accurate and early risk stratification assumes paramount importance, guiding both therapeutic intensification and post-discharge surveillance. Cardiac biomarkers and echocardiography, which reflect respectively biochemical and mechanical derangements at the myocardial level, are central to this endeavor[3].

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Troponin I, N-terminal pro-B-type natriuretic peptide (NT-proBNP), and left ventricular ejection fraction (LVEF) are well-established parameters in the context of acute myocardial infarction for diagnosis, prognosis, and ongoing management2-4. However, most primary studies and existing guidelines have investigated their individual prognostic values, with scarce focus on their direct correlation with each other and their composite impact on short-term (1- month) mortality in STEMI patients[5]. The necessity of this research is further substantiated by a consistent under-reporting of Indian subcontinent data that integrate biochemical and echocardiographic markers for survival prediction following STEMI4-6. Consequently, our research was directed to fill this gap by evaluatively correlating Troponin I, NT-proBNP, and LVEF at presentation with 1-month mortality in STEMI patients, thereby seeking a multifaceted risk model relevant to clinical practice in Indian settings2-4.

Cardiac troponins–specific to cardiac myocytes and released in response to ischemic injury– are the gold standard for detection of myocardial necrosis[6]. Among these, Troponin I is highly sensitive and specific, now routinely included in the universal definition of myocardial infarction[36]. Numerous observational studies have confirmed the association of higher admission troponin levels with not only larger infarct size but also increased risk of adverse outcomes, both in-hospital and over the longer term[7]. For example, Sabatine et al. demonstrated that patients with cTnI levels above median had a 2.3-fold increase in 30-day mortality post- STEMI[7]. However, beyond diagnostic confirmation, the incremental prognostic value of troponin I–when integrated with other clinical or laboratory risk factors–remains a subject of further exploration7,8.

NT-proBNP originates from cardiac ventricles in response to increased wall stress and myocardial stretch, reflecting neurohormonal activation and degree of hemodynamic compromise[89]. It has been integrated into the multi-marker risk stratification model in both acute and chronic heart failure, as well as in acute coronary syndromes (ACS)[10]. The Val-HeFT study, for instance, reported that each doubling of baseline NT-proBNP was linked to an approximately 40% higher risk of cardiovascular death in patients post-myocardial infarction[10].

Materials and Methods

This study was conducted as a hospital-based, analytic, observational cohort study, employing a prospective approach to evaluate the correlation between Troponin I, N-terminal pro B-type natriuretic peptide (NT-proBNP), and left ventricular ejection fraction (LVEF) at admission with 1-month mortality in patients presenting with ST-segment elevation myocardial infarction (STEMI). The methodology adhered to rigorous clinical research standards and was designed to ensure robust and reproducible results, minimizing bias and maximizing validity.

The research was carried out at the Department of Cardiology, Jawaharlal Nehru Medical College (JNMC), Belagavi, a tertiary care teaching hospital well-equipped with advanced cardiac diagnostic and treatment facilities. The source population comprised all patients presenting with a clinical diagnosis of STEMI to the emergency or intensive cardiac care unit.

Inclusion Criteria

- Adults aged ≥18 years.
- Confirmed diagnosis of STEMI, defined as new ST elevation at the J point in two contiguous leads ≥2mm in men or ≥1.5mm in women in leads V2-V3 and/or ≥1mm in other contiguous chest or limb leads, with positive cardiac biomarkers as per the fourth universal definition.
- Presentation within 24 hours of onset of chest pain or equivalent symptoms.

Exclusion Criteria

- Previous history of myocardial infarction within the last 6 months.
- Patients with severe valvular heart disease or known congenital heart disease.
- Patients with end-stage renal disease (eGFR <15ml/min/1.73m² or on dialysis).
- Patients unwilling or unable to provide informed consent.
- Those with concurrent acute illnesses known to independently elevate NT-proBNP or troponins (e.g., acute stroke, sepsis).
- Pregnant or lactating women.

Enrollment Procedure

Patients with suspected STEMI were initially screened in the emergency department or cardiac care unit. Diagnosis was confirmed based on history, clinical examination, 12-lead ECG, and admission cardiac enzyme profiles. Eligibility was ascertained according to the inclusion and exclusion criteria. Eligible patients or their surrogates were provided with detailed information about the study, and written informed consent was obtained before any study-specific procedures were initiated. Enrollment continued consecutively until the required sample size of 300 was reached.

Electrocardiography

A 12-lead ECG was performed for all patients at presentation, confirming the diagnosis of STEMI and documenting any initial arrhythmias or conduction defects.

Laboratory Investigations

Venous blood samples were drawn on admission for the following:

- Troponin I: Quantitative estimation using a standardized chemiluminescence immunoassay.
- NT-proBNP: Measured using sandwich immunoassay techniques, as per manufacturer instructions.
- Renal and liver function tests, hemogram, fasting blood glucose, and lipid profile.

The blood for Troponin I and NT-proBNP was collected within the first hour of admission, processed in the institution's accredited central laboratory using quality-assured kits with internal and external controls.

Echocardiography

All patients underwent 2D transthoracic echocardiography (TTE) within 24 hours of admission, performed by an experienced cardiologist who was blinded to the biomarker values. LVEF was measured using the Simpson's biplane method of discs, in accordance with ASE/EACVI guidelines, and recorded as a continuous variable.

Reperfusion Strategies

Details regarding the initial treatment strategy (primary percutaneous coronary intervention [PCI], thrombolysis, or conservative management) were documented for each case, as this could potentially affect outcomes.

In-Hospital Monitoring

Patients were monitored throughout their hospital stay for the development of complications such as recurrent ischemia, heart failure (Killip class), arrhythmias, shock, and requirement for mechanical ventilation or advanced interventions. These events were systematically recorded.

Statistical Analysis:
Continuous variables such as age, HbA1c, LDL, Troponin I, NT-pro BNP, and left ventricular ejection fraction (LVEF) were summarized as mean ± standard deviation (SD), while categorical variables such as gender, risk factors, biomarker categories, and mortality were presented as frequencies and percentages.

Associations between categorical variables and mortality were assessed using the Chi-square test (χ^2 test). Correlation between continuous and ordinal variables (such as biomarker levels and LVEF) was analyzed using Kendall's tau-b and Spearman's rho correlation coefficients, as appropriate. Variables showing significance on univariate analysis ($p < 0.05$) were further entered into a binary logistic regression model to identify independent predictors of 1-month mortality. Results were expressed as odds ratios (OR) with 95% confidence intervals (CI).
The predictive performance of Troponin I, NT-pro BNP, and LVEF was evaluated using Receiver Operating Characteristic (ROC) curve analysis. The area under the curve (AUC) with 95% CI was calculated, and optimal cut off values were determined to assess sensitivity and specificity for predicting 1-month mortality.

Additionally, improvement in predictive performance of the combined model over individual biomarkers was quantified using Net Reclassification Improvement (NRI) and Integrated Discrimination Improvement (IDI). A p-value < 0.05 was considered statistically significant for all analyses. All data were entered into Microsoft Excel and analyzed using SPSS software v 25.0.

Results

Table - 1: Age distribution

Age	Frequency	Percentage
30-39	8	2.7
40-49	52	17.3
50-59	72	24.0
60-69	109	36.3
70-79	50	16.7
80-89	9	3.0
Total	300	100.0

In the present study, the majority of patients with STEMI were in the 60-69 year age group (36.3%), followed by the 50-59 year group (24.0%). Together, these two groups comprised nearly 60% of the study population, indicating that middle-aged and early elderly individuals formed the largest proportion of cases. Patients in the younger age group (<40 years) were relatively uncommon (2.7%), while those aged 70 years and above constituted 19.7% of the study population.

Table - 2: Sex distribution

Sex	Frequency	Percentage
Female	89	29.7
Male	211	70.3
Total	300	100.0

In the present study, the majority of STEMI patients were male (70.3%), while female patients accounted for 29.7% of the study population. This finding reflects the well-established male predominance in the incidence of STEMI, particularly in middle-aged groups.

Table - 3: Trop I distribution

Trop I	Frequency	Percentage
Normal	36	12.0
Mildly elevated	137	45.7
Moderately elevated	32	10.7
Highly elevated	95	31.7
Total	300	100.0

In the present study, 45.7% of patients had mildly elevated Troponin I levels (0.04-0.5 ng/mL), while 31.7% showed highly elevated levels (>1.0 ng/mL). Moderate elevation (0.51-1.0 ng/mL) was observed in 10.7%, and 12% had normal values at the time of admission. The substantial proportion (31.7%) with highly elevated levels is of particular prognostic significance, as marked Troponin elevation has been associated with larger infarct size, reduced left ventricular ejection fraction, and higher short-term mortality.

Table -4: NT-PROBNP distribution

NT PROBNP	Frequency	Percentage
Low	162	54.0
Moderate	99	33.0
High	21	7.0
Very high	18	6.0
Total	300	100.0

In the present study, the majority of patients (54%) had low NT-proBNP levels (<1,000 pg/mL), while 33% demonstrated moderate elevation (1,000-5,000 pg/mL). A smaller subset showed high (7%) and very high (6%) levels (>10,000 pg/mL).

Table -5: Hba1c distribution

Hba1c	Frequency	Percentage
Normal	109	36.3
Prediabetes	67	22.3
Diabetes (controlled)	42	14.0
Poor control	82	27.3
Total	300	100.0

In the present study, 36.3% of patients had normal HbA1c levels (<5.7%), while 22.3% were in the prediabetes range (5.7-6.4%). Among those with diabetes, 14% had controlled diabetes (6.5-7.9%), whereas a notable 27.3% exhibited poor glycemic control (≥8%).

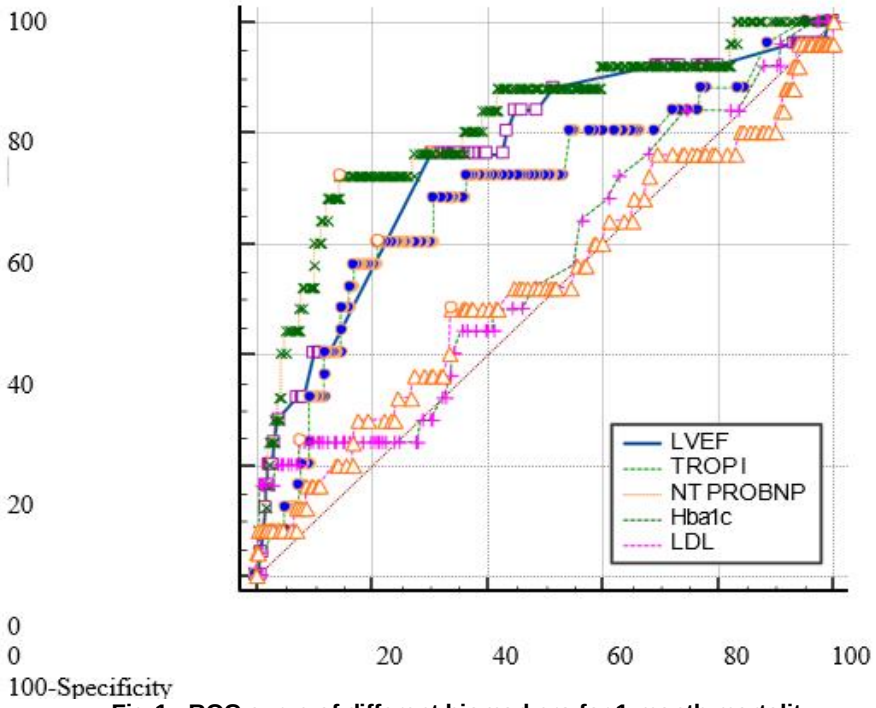

Fig-1 : ROC curve of different biomarkers for 1-month mortality.

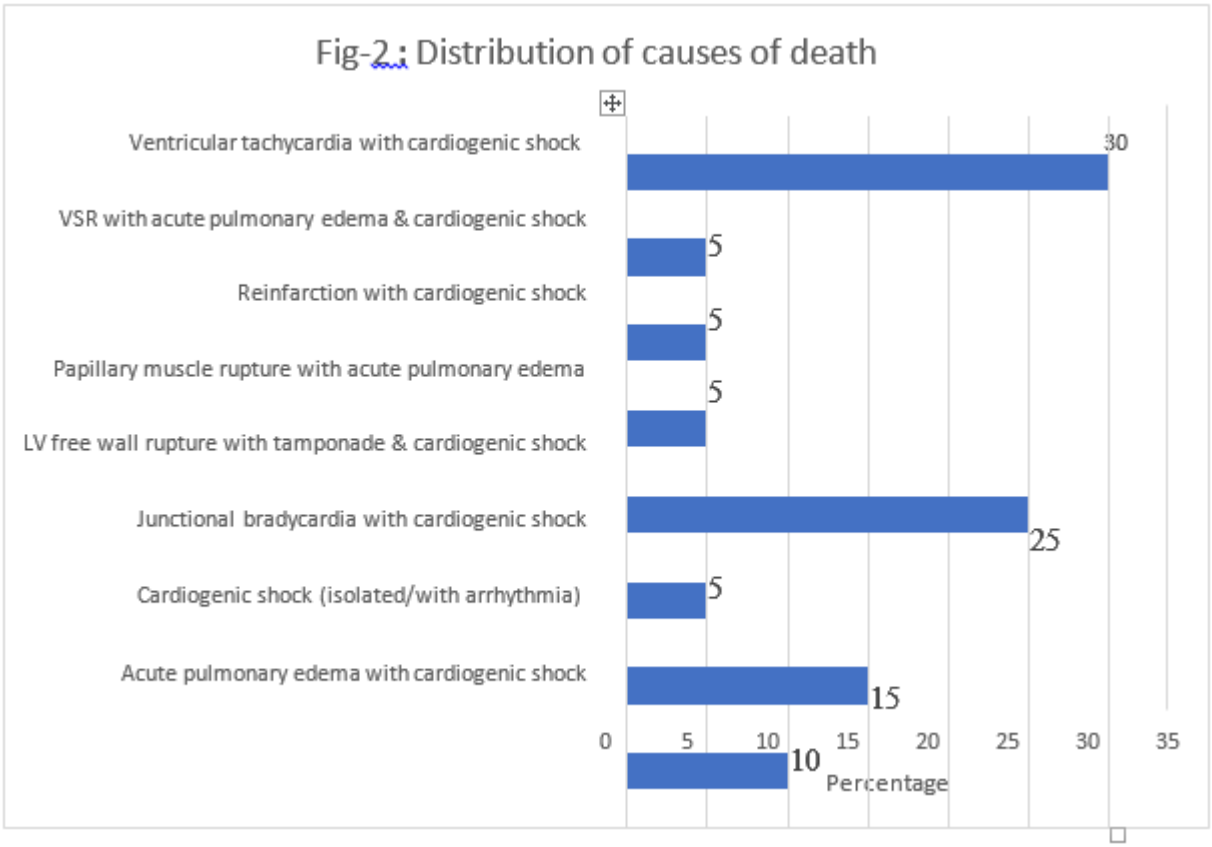

Table - 6: ROC curve analysis and pairwise comparison of predictive models for 1-month mortality

Predictor / Model	AUC	SE	95% CI	*p*-value
Troponin I	0.312	0.061	0.192 - 0.432	0.002
NT-proBNP	0.189	0.051	0.090 - 0.289	<0.001
LVEF	0.247	0.054	0.141 - 0.353	<0.001
Troponin I + NT-proBNP	0.205	0.059	0.090 - 0.320	<0.001
Troponin I + LVEF	0.329	0.061	0.210 - 0.448	0.005
NT-proBNP + LVEF	0.207	0.051	0.107 - 0.307	<0.001
Troponin I + NT-proBNP + LVEF	0.213	0.058	0.098 - 0.327	<0.001

The ROC analysis revealed relatively low discriminative power for individual biomarkers when predicting 1-month mortality (AUCs: Troponin I = 0.312, NT-proBNP = 0.189, LVEF = 0.247). Among them, Troponin I showed the highest, though still modest, predictive ability. Pairwise comparisons demonstrated that Troponin I significantly outperformed NT-proBNP ($p = 0.004$) and NT-proBNP + LVEF ($p = 0.018$). Interestingly, the combination of Troponin I and LVEF yielded better performance than Troponin I alone ($p = 0.005$). The triple combination (Troponin I + NT-proBNP + LVEF) did not significantly improve prediction compared to Troponin I alone ($p < 0.001$, favoring Trop I). Overall, combinations involving LVEF + Troponin I tended to perform better than NT-proBNP-based models.

Table - 7: Pairwise comparisons of biomarkers vs. combined model (1-month mortality)

Comparison	z	*p*-value	ce (95% CI)	Interpretation
Trop I vs NT-proBNP	2.86	0.004	0.123 (0.039-0.206)	Trop I > NT-proBNP
Trop I vs LVEF	0.95	0.340	0.065 (-0.069-0.198)	No significant difference
NT-proBNP vs LVEF	-0.93	0.351	-0.058 (-0.179-0.063)	No difference
Trop I vs (Trop I + NT- proBNP)	3.97	<0.001	0.107 (0.054-0.160)	Trop I better
Trop I vs (Trop I + LVEF)	-2.78	0.005	-0.017 (-0.029- -0.005)	Combination better
Trop I vs (NT-proBNP + LVEF)	2.37	0.018	0.105 (0.018-0.192)	Trop I better
NT-proBNP vs (Trop I + LVEF)	-3.22	0.001	-0.140 (-0.225- -0.055)	Combo better
NT-proBNP vs (NT-proBNP + LVEF)	-3.08	0.002	-0.017 (-0.029- -0.006)	Combo slightly better
Troponin I vs. Combined	3.75	<0.001	0.099 (0.048 - 0.151)	Troponin I performed better than the combined model
NT-proBNP vs. Combined	-1.04	0.297	-0.023 (-0.067 - 0.020)	No significant difference
LVEF vs. Combined	0.52	0.606	0.034 (-0.096 - 0.165)	No significant difference
Troponin I + NT-proBNP vs. Combined	-2.33	0.020	-0.008 (-0.014 - -0.001)	Combined model performed slightly better
Troponin I + LVEF vs. Combined	4.25	<0.001	0.117 (0.063 - 0.170)	Troponin I + LVEF performed better

When compared to the full combined model (Troponin I + NT-proBNP + LVEF), Troponin I alone ($p < .001$) and the two-marker combination of Troponin I + LVEF ($p < .001$) showed significantly higher discriminatory ability for predicting 1-month mortality. In contrast, NT- proBNP alone, LVEF alone, and NT-proBNP + LVEF did not differ significantly from the combined model ($p > .05$). The model combining Troponin I + NT-proBNP performed marginally worse than the triple model ($p = .020$). Overall, Troponin I (alone or with LVEF) demonstrated the most useful predictive value, whereas NT-proBNP added little incremental benefit.

Discussion

Troponin I:

Globally, Troponin I is well established as a diagnostic and prognostic marker. Sabatine et al.
[7] reported that patients with troponin above median had a 2.3-fold higher 30-day mortality. Omland et al. [15] further showed that troponin measured at admission and after 24 hours independently predicted outcomes. Our findings echo this: mortality was significantly higher in patients with markedly elevated troponin, reflecting larger infarct size and greater myocyte necrosis. However, in multivariate analysis, troponin was not an independent predictor once NT-proBNP and EF were accounted for. This suggests that troponin's prognostic effect is mediated through its downstream impact on LV dysfunction and hemodynamic compromise, aligning with observations from multi-marker models [25].

NT-proBNP:

Several international studies have shown NT-proBNP's robust prognostic value in acute coronary syndromes. A meta-analysis of >8,000 ACS patients demonstrated that elevated NT- proBNP was associated with a 6-fold higher risk of 30-day mortality (pooled OR: 6.0, 95% CI 3.8-10.1), with an AUC of 0.77-0.86 [11]. Zdravkovic et al. [17] reported NT-proBNP cutoff of 4,390 pg/ml predicted in-hospital mortality with 88.2% sensitivity and specificity. Our study supports these findings, with NT-proBNP emerging as the strongest independent predictor even after adjusting for troponin and EF. The progressive mortality gradient observed in our cohort highlights its dose-response nature, consistent with both Val-HeFT [10] and international ACS studies [8].

Prognostic Role:

Cardiac troponins are the gold-standard biomarkers for detecting myocardial necrosis. Among them, Troponin I is highly specific to cardiac myocytes and is now mandatory in the universal
definition of myocardial infarction [3]. Troponin release follows ischemic injury, with levels rising within 3-6 hours, peaking at 12-24 hours, and remaining elevated for up to 7-10 days [5]. The magnitude of elevation correlates with infarct size, left ventricular dysfunction, and subsequent prognosis [6].

In our study, 31.7% of patients had highly elevated Troponin I (>1.0 ng/mL) and these patients demonstrated the highest 1-month mortality (15.8%) compared to only 2.8% among those with normal troponin ($p = 0.011$). This gradient mirrors findings from Sabatine et al., who reported that ACS patients with troponin above the population median had a 2.3-fold higher 30-day mortality [7]. Similarly, Omland et al. demonstrated that troponin measured at admission and after 24 hours provided strong independent prognostic information [15].

However, in multivariate regression analysis in our study, troponin did not retain independent predictive value once NT-proBNP and LVEF were included. This suggests troponin's prognostic impact is indirect, mediated through its effect on ventricular systolic dysfunction and neurohormonal activation. This is consistent with Richards et al., who showed that troponin alone had modest prognostic accuracy, but when integrated with NT-proBNP and echocardiographic parameters, predictive ability improved significantly [25].

Another Indian study by Ahmad et al. found a negative correlation between Troponin I and LVEF, confirming that higher troponin levels reflect more severe systolic dysfunction [27]. While they did not evaluate mortality outcomes, their findings support the biological plausibility observed in our results—patients with very high troponin are at high risk largely because of the downstream effect on ventricular contractility and remodeling.

NT-proBNP is secreted from ventricular myocytes in response to stretch and pressure overload, reflecting hemodynamic stress and neurohormonal activation [8, 9]. Its prognostic value in acute coronary syndromes has been consistently demonstrated. Li et al. conducted a meta- analysis of >8,000 ACS patients, reporting that elevated NT-proBNP conferred a 6-fold increase in 30-day mortality risk (pooled OR: 6.0, 95% CI: 3.8-10.1) and had an AUC of 0.77-
0.86 for short-term prediction [11].

In our study, NT-proBNP showed the strongest association with mortality ($\chi^2 = 42.24$, $p < 0.001$). Mortality increased stepwise with higher categories: Low (<1,000 pg/mL): 1.9%, Moderate (1,000-5,000 pg/mL): 9.1%, High (5,001-10,000 pg/mL): 28.6%, Very high
(>10,000 pg/mL): 38.9%.

Conclusion

This study demonstrated that Troponin I, NT-proBNP, and LVEF are important predictors of short-term outcomes in STEMI, with NT-proBNP emerging as the strongest independent marker of 1-month mortality. Mortality increased progressively with higher NT-proBNP levels, markedly reduced LVEF, and markedly elevated troponin, establishing a clear correlation between the severity of biochemical and functional derangements and adverse prognosis.

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