



Original Research Article

Role of High-Sensitivity Cardiac Troponin and NT-proBNP in Predicting Short-Term Outcomes Following Acute Myocardial Infarction

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Abstract

Background: Early risk stratification following acute myocardial infarction (AMI) is essential for identifying patients at increased risk of adverse clinical outcomes. High-sensitivity cardiac troponin (hs-cTn) and N-terminal pro-B-type natriuretic peptide (NT-proBNP) are established cardiac biomarkers reflecting myocardial injury and ventricular stress, respectively.

Methods: This prospective observational study included 80 patients with AMI admitted to a tertiary care hospital. Baseline hs-cTn and NT-proBNP levels were measured at admission. Patients were followed for 30 days to assess composite adverse outcomes, including acute heart failure, cardiogenic shock, recurrent myocardial infarction, significant arrhythmias, prolonged hospital stay, and all-cause mortality. Receiver operating characteristic (ROC) analysis and multivariable logistic regression were performed to evaluate the prognostic performance of the biomarkers.

Results: Composite adverse outcomes occurred in 29 (36.3%) patients. Patients with adverse outcomes had significantly higher hs-cTn (4826 ± 1928 vs. 2633 ± 1397 ng/L; $p < 0.001$) and NT-proBNP levels (4212 ± 1836 vs. 1504 ± 864 pg/mL; $p < 0.001$). NT-proBNP demonstrated superior predictive ability (AUC 0.901) compared with hs-cTn (AUC 0.842), while the combined biomarker model achieved the highest accuracy (AUC 0.936). Multivariable analysis identified NT-proBNP > 2500 pg/mL, hs-cTn > 3650 ng/L, and LVEF $< 40\%$ as independent predictors of adverse outcomes.

Conclusion: Both hs-cTn and NT-proBNP were valuable prognostic biomarkers in AMI, with NT-proBNP showing superior predictive performance. Combined assessment of both biomarkers improved early risk stratification and may facilitate timely therapeutic interventions in high-risk patients.

Keywords: Acute myocardial infarction; High-sensitivity cardiac troponin; NT-proBNP; Prognosis; Risk stratification; Biomarkers; Short-term outcomes; Receiver operating characteristic.

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Introduction

Acute myocardial infarction (AMI) remains a major cause of morbidity and mortality worldwide despite substantial advances in early diagnosis, prompt reperfusion therapy, and evidence-based pharmacological management[1]. Cardiovascular diseases account for nearly one-third of global deaths, with ischaemic heart disease representing the leading cause. Although contemporary treatment strategies, including primary percutaneous coronary intervention, have significantly reduced in-hospital mortality, many patients continue to experience

adverse short-term outcomes such as heart failure, recurrent myocardial infarction, malignant arrhythmias, cardiogenic shock, prolonged hospitalisation, and death. Early identification of high-risk patients is therefore essential to optimise management and improve clinical outcomes [2,3]. Risk stratification following AMI traditionally relies on clinical assessment, electrocardiography, echocardiography, and validated scoring systems such as the TIMI and GRACE scores. While these tools provide important prognostic information, they

may not adequately reflect the extent of myocardial injury or ventricular dysfunction during the acute phase of infarction. Consequently, circulating cardiac biomarkers have emerged as valuable adjuncts for objective and early risk assessment [4,5]. High-sensitivity cardiac troponin (hs-cTn) has revolutionised the diagnosis of AMI by enabling the detection of minimal myocardial injury with excellent analytical sensitivity. Beyond its diagnostic role, hs-cTn has important prognostic significance, as elevated concentrations correlate with infarct size, impaired left ventricular function, and an increased risk of adverse cardiovascular events. Persistently elevated hs-cTn levels have been associated with heart failure, recurrent ischaemic events, cardiogenic shock, and short-term mortality after AMI [6–8]. N-terminal pro-B-type natriuretic peptide (NT-proBNP) is released predominantly from ventricular myocardium in response to increased wall stress and pressure overload. During AMI, myocardial ischaemia and ventricular dysfunction stimulate NT-proBNP secretion even before overt heart failure develops. Elevated NT-proBNP levels are strongly associated with left ventricular systolic dysfunction, adverse ventricular remodelling, cardiogenic shock, and increased mortality. Unlike hs-cTn, which reflects myocardial necrosis, NT-proBNP primarily indicates ventricular stress and haemodynamic compromise, providing complementary prognostic information [9–11]. Cardiogenic shock remains one of the most serious complications of AMI and is associated with extremely high short-term mortality despite advances in revascularisation and intensive care. Since extensive myocardial injury and ventricular dysfunction are major determinants of shock, biomarkers reflecting these processes may facilitate early identification of patients at greatest risk before clinical deterioration becomes apparent [2,12,13]. Recent studies suggest that combining hs-cTn and NT-proBNP improves prognostic accuracy compared with either biomarker alone by simultaneously assessing myocardial injury and ventricular dysfunction. However, evidence regarding their combined utility in predicting short-term outcomes after AMI remains limited, particularly in developing countries [11,14]. Therefore, the present study was undertaken to evaluate the prognostic role of hs-cTn and NT-proBNP in predicting short-term adverse outcomes following AMI and to assess their usefulness as reliable biomarkers for early risk stratification.

Methodology:

The present study was conducted as a prospective observational hospital-based study to evaluate the prognostic role of high-sensitivity cardiac troponin (hs-cTn) and N-terminal pro-B-type natriuretic peptide (NT-proBNP) in predicting short-term clinical outcomes among patients presenting with

acute myocardial infarction (AMI). The study was carried out in the Department of General Medicine at a tertiary care teaching hospital.

Study Population

The study population consisted of adult patients admitted with a diagnosis of acute myocardial infarction who fulfilled the predefined eligibility criteria. A total of 80 consecutive patients diagnosed with acute myocardial infarction were included in the study.

Sampling Technique

Consecutive sampling was employed, and all eligible patients admitted during the study period were enrolled until the required sample size of 80 participants was achieved.

Eligibility Criteria

Inclusion Criteria

Patients fulfilling all of the following criteria were included:

- Age ≥ 18 years.
- Diagnosis of acute myocardial infarction (ST-elevation myocardial infarction or non-ST-elevation myocardial infarction) based on the Fourth Universal Definition of Myocardial Infarction.
- Presentation within 24 hours of onset of symptoms.
- Willingness to provide written informed consent.

Exclusion Criteria

Patients were excluded if they had any of the following:

- Previous myocardial infarction within the preceding three months.
- Chronic heart failure with known reduced or preserved ejection fraction.
- Significant valvular heart disease.
- Cardiomyopathy of any aetiology.
- End-stage renal disease or estimated glomerular filtration rate < 30 mL/min/1.73 m².
- Severe hepatic dysfunction.
- Active infection, sepsis, or systemic inflammatory disease.
- Malignancy with expected survival less than six months.
- Pregnancy.
- Patients unwilling to participate in the study.

Study Procedure

After obtaining written informed consent, eligible patients were enrolled consecutively. A detailed

clinical history was obtained, and a comprehensive physical examination was performed at admission. Demographic characteristics, cardiovascular risk factors, presenting symptoms, duration of symptoms, Killip class, blood pressure, heart rate, and relevant comorbidities were recorded using a predesigned case record form. All patients underwent a standard 12-lead electrocardiogram at presentation. The diagnosis of STEMI or NSTEMI was established according to contemporary international guidelines. Patients received standard medical management and coronary revascularization whenever indicated, as per institutional treatment protocols.

Laboratory Investigations

Venous blood samples were collected at the time of admission before or immediately after initiation of treatment according to standard hospital protocols.

The following investigations were performed:

- High-sensitivity cardiac troponin (hs-cTn)
- NT-proBNP
- Complete blood count
- Random blood glucose
- Renal function tests
- Liver function tests
- Serum electrolytes
- Lipid profile
- HbA1c (where applicable)

Measurement of High-Sensitivity Cardiac Troponin

Serum high-sensitivity cardiac troponin levels were measured using a commercially available high-sensitivity immunoassay according to the manufacturer's recommendations. Values were expressed in ng/L.

Measurement of NT-proBNP

Serum NT-proBNP concentrations were determined using a standardized electrochemiluminescence immunoassay. Values were expressed in pg/mL.

Echocardiographic Assessment

Two-dimensional transthoracic echocardiography was performed within 24–48 hours of admission by an experienced cardiologist. Left ventricular ejection fraction (LVEF), regional wall motion abnormalities, and other relevant structural cardiac abnormalities were documented.

Outcome Assessment

Patients were followed throughout their hospital stay and for 30 days after the index admission.

Primary Outcome

The primary outcome was the occurrence of any major adverse short-term clinical outcome during the follow-up period.

Secondary Outcomes

The following outcomes were recorded:

- Acute heart failure
- Cardiogenic shock
- Recurrent myocardial infarction
- Significant ventricular or supraventricular arrhythmias
- Requirement for intensive care or mechanical circulatory support
- Prolonged hospital stay (>7 days)
- All-cause mortality within 30 days

Patients developing one or more of the above events were considered to have experienced an adverse short-term outcome.

Data Collection

All demographic, clinical, laboratory, echocardiographic, treatment, and outcome variables were entered into a structured data collection proforma. Data accuracy was verified before statistical analysis.

Statistical Analysis

The collected data were entered into Microsoft Excel and analysed using Statistical Package for the Social Sciences (SPSS) software version 26. Continuous variables were expressed as mean $\pm$ standard deviation (SD) for normally distributed data or median with interquartile range (IQR) for skewed data. Categorical variables were expressed as frequency and percentage. Normality of continuous variables was assessed using the Shapiro–Wilk test. Comparisons between groups were performed using the independent Student's *t*-test for normally distributed variables and the Mann–Whitney *U* test for non-normally distributed variables. Categorical variables were compared using the Chi-square test or Fisher's exact test whenever appropriate. Receiver operating characteristic (ROC) curve analysis was performed to evaluate the predictive performance of hs-cTn and NT-proBNP for adverse short-term outcomes. The area under the curve (AUC), optimal cut-off values, sensitivity, specificity, positive predictive value, and negative predictive value were calculated. Univariable logistic regression analysis was initially performed to identify variables associated with adverse outcomes. Variables with a *p*-value <0.10 in univariable analysis were entered into multivariable logistic regression to identify independent predictors. Results were expressed as odds ratios (ORs) with 95% confidence intervals (CIs). A two-tailed *p*-value <0.05 was considered statistically significant.

Results:

A total of 80 patients with acute myocardial infarction were included in the study. The mean age of the study population was 59.8 ± 11.6 years. The largest proportion of patients belonged to the 60–69 years age group (35.0%), followed by 50–59 years (27.5%), while patients aged <50 years and ≥ 70 years each constituted 18.8% of the cohort. There was a marked male predominance, with 56 (70.0%) males and 24 (30.0%) females. Hypertension was the most common comorbidity (57.5%), followed by diabetes mellitus (40.0%), current smoking (42.5%), dyslipidaemia (36.3%), and previous coronary artery disease (22.5%). ST-elevation myocardial infarction (STEMI) was observed in 65.0% of patients, whereas 35.0% had non-ST-elevation myocardial infarction (NSTEMI). The mean symptom-to-door time was 5.4 ± 2.3 hours, and the mean left ventricular ejection fraction (LVEF) was $46.8 \pm 8.5\%$ (Table 1). The baseline laboratory characteristics of the study population are presented in Table 2. The mean high-sensitivity cardiac troponin (hs-cTn) level was 3428 ± 1964 ng/L, while the mean NT-proBNP concentration was 2485 ± 1718 pg/mL. The mean haemoglobin level was 12.9 ± 1.7 g/dL, total leucocyte count was $10.8 \pm 3.1 \times 10^3/\mu\text{L}$, serum creatinine was 1.12 ± 0.38 mg/dL, and random blood glucose was 184 ± 62 mg/dL. The mean LDL cholesterol level was 118 ± 34 mg/dL, whereas the mean estimated glomerular filtration rate (eGFR) was 78.6 ± 18.2 mL/min/1.73 m². The distribution of these laboratory parameters is illustrated in Figure 1. During the 30-day follow-up period, 29 patients (36.3%) experienced at least one composite adverse outcome. Acute heart failure was the most frequent complication, occurring in 18 (22.5%) patients, followed by prolonged hospital stay (>7 days) in 24 (30.0%), ICU admission in 15 (18.8%), significant arrhythmias in 12 (15.0%), 30-day mortality in 9 (11.3%), cardiogenic shock in 8 (10.0%), mechanical ventilation in 7 (8.8%), and recurrent myocardial infarction in 6 (7.5%) patients (Table 3). Patients who developed composite adverse outcomes had significantly higher baseline hs-cTn and NT-proBNP levels compared with those who remained free of adverse events. The mean hs-cTn concentration was 4826 ± 1928 ng/L in the adverse outcome group compared with 2633 ± 1397 ng/L in patients without adverse outcomes ($p < 0.001$). Similarly, the mean NT-proBNP level was significantly higher among patients with adverse outcomes (4212 ± 1836 pg/mL) than among those without adverse outcomes (1504 ± 864 pg/mL, $p < 0.001$). Patients experiencing adverse outcomes also had significantly lower LVEF ($39.2 \pm 6.4\%$ vs. $51.1 \pm 6.8\%$, $p < 0.001$) and higher serum creatinine levels (1.36 ± 0.44 mg/dL vs. 0.98 ± 0.26 mg/dL, $p = 0.002$) (Table 4). These differences are graphically represented in Figure 2.

Receiver operating characteristic (ROC) analysis demonstrated that both hs-cTn and NT-proBNP showed excellent predictive ability for composite adverse outcomes. NT-proBNP exhibited superior discriminatory performance with an AUC of 0.901 (95% CI: 0.833–0.970) compared with hs-cTn (AUC 0.842; 95% CI: 0.749–0.935). The combined biomarker model demonstrated the highest predictive accuracy, with an AUC of 0.936 (95% CI: 0.883–0.989). The optimal cut-off values were >3650 ng/L for hs-cTn and >2500 pg/mL for NT-proBNP, providing high sensitivity and specificity for predicting adverse outcomes (Table 5). Multivariable logistic regression analysis identified elevated hs-cTn (>3650 ng/L), elevated NT-proBNP (>2500 pg/mL), and reduced LVEF (<40%) as independent predictors of composite adverse outcomes. Elevated NT-proBNP demonstrated the strongest independent association with adverse outcomes (adjusted OR 4.82; 95% CI: 1.92–12.08; $p = 0.001$), followed by elevated hs-cTn (adjusted OR 3.46; 95% CI: 1.41–8.47; $p = 0.007$) and reduced LVEF (adjusted OR 3.18; 95% CI: 1.24–8.18; $p = 0.016$). Although age ≥ 65 years, diabetes mellitus, and STEMI were associated with increased odds of adverse outcomes, these variables did not reach statistical significance in the multivariable model (Table 6). The adjusted odds ratios with corresponding 95% confidence intervals are illustrated in Figure 3.

Table 1. Baseline Demographic and Clinical Characteristics of the Study Population (N = 80)

Variable	Value
Age (years), mean $\pm$ SD	59.8 ± 11.6
Age group, n (%)	
<50 years	15 (18.8)
50–59 years	22 (27.5)
60–69 years	28 (35.0)
≥ 70 years	15 (18.8)
Male, n (%)	56 (70.0)
Female, n (%)	24 (30.0)
Hypertension, n (%)	46 (57.5)
Diabetes mellitus, n (%)	32 (40.0)
Current smoker, n (%)	34 (42.5)
Dyslipidaemia, n (%)	29 (36.3)
Previous CAD, n (%)	18 (22.5)
STEMI, n (%)	52 (65.0)

NSTEMI, n (%)	28 (35.0)
Mean symptom-to-door time (hours), mean $\pm$ SD	5.4 $\pm$ 2.3
Left ventricular ejection fraction (%), mean $\pm$ SD	46.8 $\pm$ 8.5

Table 2. Laboratory Profile of the Study Population

Variable	Mean $\pm$ SD
High-sensitivity Troponin (ng/L)	3428 $\pm$ 1964
NT-proBNP (pg/mL)	2485 $\pm$ 1718
Haemoglobin (g/dL)	12.9 $\pm$ 1.7
Total leucocyte count ($\times 10^3/\mu\text{L}$)	10.8 $\pm$ 3.1
Serum creatinine (mg/dL)	1.12 $\pm$ 0.38
Random blood glucose (mg/dL)	184 $\pm$ 62
LDL cholesterol (mg/dL)	118 $\pm$ 34
eGFR (mL/min/1.73 m ²)	78.6 $\pm$ 18.2

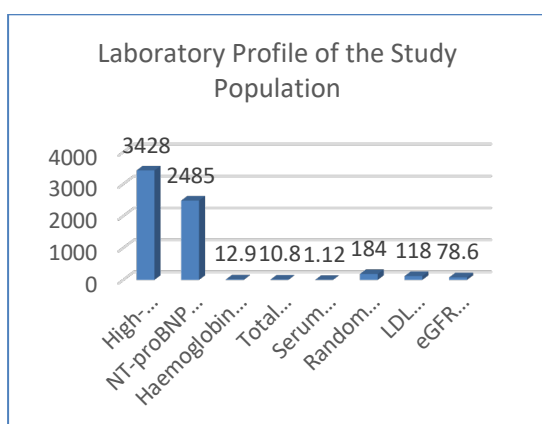


Figure 1 Laboratory Profile of the Study Population

Table 3. Short-Term Clinical Outcomes Following Acute Myocardial Infarction

Outcome	n (%)
Acute heart failure	18 (22.5)
Cardiogenic shock	8 (10.0)
Significant arrhythmias	12 (15.0)

Recurrent myocardial infarction	6 (7.5)
Prolonged hospital stay (>7 days)	24 (30.0)
ICU admission	15 (18.8)
Mechanical ventilation	7 (8.8)
30-day mortality	9 (11.3)
Composite adverse outcome	29 (36.3)

Table 4. Comparison of Biomarker Levels Between Patients With and Without Composite Adverse Outcome

Variable	Adverse Outcome (n=29)	No Adverse Outcome (n=51)	p-value
hs-cTn (ng/L), mean $\pm$ SD	4826 $\pm$ 1928	2633 $\pm$ 1397	<0.001
NT-proBNP (pg/mL), mean $\pm$ SD	4212 $\pm$ 1836	1504 $\pm$ 864	<0.001
LVEF (%), mean $\pm$ SD	39.2 $\pm$ 6.4	51.1 $\pm$ 6.8	<0.001
Serum creatinine (mg/dL), mean $\pm$ SD	1.36 $\pm$ 0.44	0.98 $\pm$ 0.26	0.002

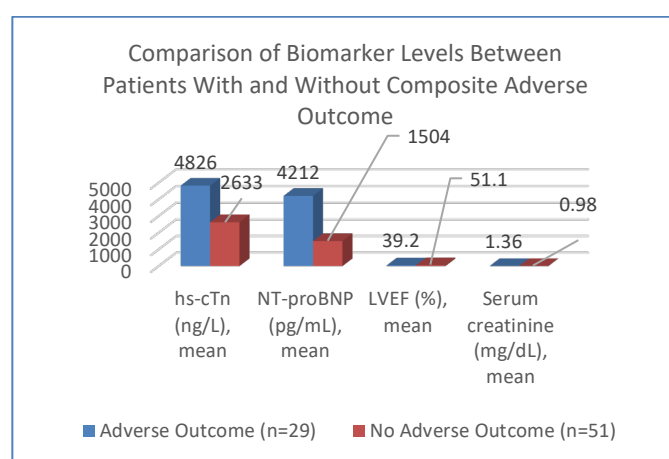


Figure 2 Comparison of Biomarker Levels Between Patients With and Without Composite Adverse Outcome

Table 5. Receiver Operating Characteristic (ROC) Analysis for Prediction of Composite Adverse Outcome

Biomarker	AUC (95% CI)	Optimal Cut-off	Sensitivity (%)	Specificity (%)	p-value
hs-cTn	0.842 (0.749–0.935)	>3650 ng/L	82.8	78.4	<0.001
NT-proBNP	0.901 (0.833–0.970)	>2500 pg/mL	86.2	84.3	<0.001
Combined model (hs-cTn + NT-proBNP)	0.936 (0.883–0.989)	—	89.7	88.2	<0.001

Table 6. Multivariable Logistic Regression Analysis for Predictors of Composite Adverse Outcome

Variable	Adjusted OR	95% CI	p-value
hs-cTn >3650 ng/L	3.46	1.41–8.47	0.007
NT-proBNP >2500 pg/mL	4.82	1.92–12.08	0.001
LVEF <40%	3.18	1.24–8.18	0.016
Age ≥65 years	2.14	0.92–4.99	0.077
Diabetes mellitus	1.76	0.74–4.16	0.200
STEMI	1.38	0.57–3.34	0.475

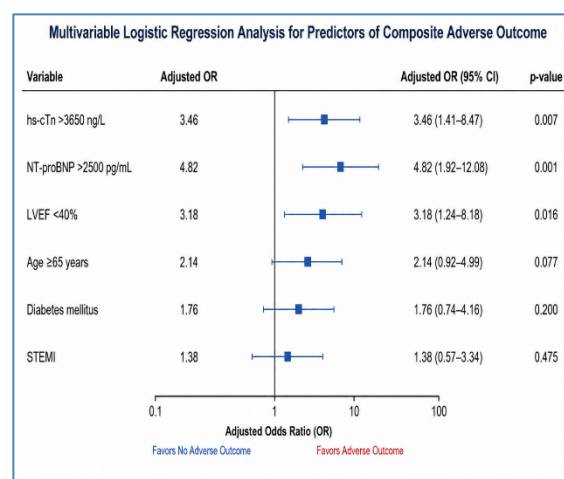


Figure 3 Multivariable Logistic Regression Analysis for Predictors of Composite Adverse Outcome

Discussion

The present prospective study evaluated the prognostic utility of high-sensitivity cardiac troponin (hs-cTn) and N-terminal pro-B-type natriuretic peptide (NT-proBNP) in predicting short-term adverse outcomes among patients with acute myocardial infarction (AMI). Both biomarkers were significantly associated with adverse outcomes, although NT-proBNP demonstrated superior prognostic performance. The combination of hs-cTn and NT-proBNP provided the highest predictive accuracy, highlighting the complementary roles of myocardial injury and ventricular stress in early risk stratification. The demographic characteristics of our cohort were comparable with previous AMI studies. The mean age was 59.8 ± 11.6 years, with 35.0% of patients aged 60–69 years, and males constituted 70.0% of the study population. Hypertension (57.5%), diabetes mellitus (40.0%), and smoking (42.5%) were the most common cardiovascular risk factors, consistent with the established epidemiology of coronary artery disease. 36.3% of patients developed the composite adverse outcome, including acute heart failure, cardiogenic shock, malignant arrhythmias, recurrent myocardial infarction, prolonged hospitalisation, or death. Acute heart failure (22.5%), ICU admission (18.8%), and 30-day mortality (11.3%) were the most frequent individual events, comparable to rates reported in contemporary AMI cohorts. Patients with adverse outcomes had significantly higher hs-cTn levels than those without complications (4826 ± 1928 vs. 2633 ± 1397 ng/L; $p < 0.001$). High-

sensitivity troponin reflects the extent of myocardial necrosis, and elevated concentrations have consistently been associated with increased mortality and heart failure after MI. Omland et al.[15] demonstrated that hs-cTn independently predicted cardiovascular mortality and heart failure, while Eggers et al.[16] confirmed its strong prognostic value across acute coronary syndrome populations. NT-proBNP showed even greater prognostic discrimination. Patients with adverse outcomes had markedly higher NT-proBNP levels (4212 ± 1836 vs. 1504 ± 864 pg/mL; $p < 0.001$), reflecting greater ventricular dysfunction and haemodynamic stress. Similar findings were reported by Omland et al.[15] and Eggers et al.[16], who demonstrated that NT-proBNP independently predicted mortality and heart failure and outperformed hs-cTn for long-term cardiovascular prognosis. Left ventricular systolic dysfunction was also strongly associated with adverse outcomes, with significantly lower LVEF among affected patients ($39.2 \pm 6.4\%$ vs. $51.1 \pm 6.8\%$; $p < 0.001$), consistent with previous evidence identifying impaired ventricular function as a major predictor of post-MI mortality.

ROC analysis demonstrated excellent diagnostic performance, with an AUC of 0.901 for NT-proBNP compared with 0.842 for hs-cTn. The combined biomarker model achieved the highest predictive accuracy (AUC 0.936), with 89.7% sensitivity and 88.2% specificity, corroborating previous findings that combined assessment of myocardial injury and ventricular dysfunction enhances prognostic discrimination.[15,16] Multivariable logistic regression identified NT-proBNP >2500 pg/mL (adjusted OR 4.82), hs-cTn >3650 ng/L (adjusted OR 3.46), and LVEF $<40\%$ (adjusted OR 3.18) as independent predictors of adverse outcomes, whereas age, diabetes, and STEMI were not independently significant. These findings are consistent with previous studies demonstrating the incremental prognostic value of NT-proBNP beyond conventional clinical variables.[15,16]

Conclusion

The present study demonstrated that both high-sensitivity cardiac troponin (hs-cTn) and NT-proBNP were significant predictors of short-term adverse outcomes in patients with acute myocardial infarction. NT-proBNP showed superior prognostic performance compared with hs-cTn, while the combined assessment of both biomarkers provided the highest predictive accuracy for early risk stratification. Elevated hs-cTn and NT-proBNP levels, along with reduced left ventricular ejection fraction, were independent predictors of adverse clinical outcomes. Routine measurement of these biomarkers at admission may facilitate early

identification of high-risk patients and guide timely therapeutic interventions, thereby improving short-term clinical outcomes following AMI.

Limitations

The present study was conducted at a single tertiary care centre with a relatively small sample size, which may limit the generalisability of the findings. Biomarker levels were measured only at baseline, and serial measurements were not performed to assess their dynamic changes over time. In addition, the follow-up period was limited to 30 days; therefore, the long-term prognostic value of hs-cTn and NT-proBNP could not be evaluated.

Ethical approval

Ethical approval for this study was obtained from the Institutional Ethics Committee prior to the commencement of the study. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki and applicable institutional guidelines.

Informed Consent

Written informed consent was obtained from all participants prior to their enrolment in the study.

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Conflict of Interest Declaration

The authors declare no conflict of interest.

Data Availability Statement

All data generated or analysed during this study are available from the corresponding author upon reasonable request.

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