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Research Article

Integrated Clinical and Biochemical Predictors of In-Hospital Adverse Outcomes in Patients With ST-Elevation Myocardial Infarction: A Prospective Observational Study

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Abstract

Background: ST-elevation myocardial infarction (STEMI) remains a leading cause of acute cardiovascular mortality and morbidity worldwide. Although mechanical and pharmacologic reperfusion therapies have markedly improved outcomes, in-hospital adverse cardiac events—including acute heart failure, cardiogenic shock, malignant ventricular arrhythmias, and early mortality—continue to affect a significant proportion of patients. Existing risk stratification models primarily rely on clinical parameters, often omitting dynamic acute biochemical markers.

Objective: To evaluate the integrated predictive value of baseline clinical characteristics and acute biochemical biomarkers (high-sensitivity cardiac troponin I, N-terminal pro-B-type natriuretic peptide [NT-proBNP], high-sensitivity C-reactive protein [hs-CRP], and serum creatinine) for in-hospital major adverse cardiac events (MACE) in patients presenting with STEMI.

Methods: This prospective observational cohort study evaluated 160 adult patients (aged 30 to 75 years) presenting with acute STEMI within 12 hours of symptom onset at RLKU Medical College. Clinical variables (Killip class, blood pressure, heart rate, door-to-balloon time) and venous blood samples for biochemical profiling were collected immediately upon emergency admission. Patients were monitored throughout their index hospitalization for the occurrence of in-hospital MACE (defined as composite all-cause mortality, cardiogenic shock, re-infarction, sustained ventricular tachycardia/fibrillation, or acute heart failure requiring intravenous inotropes/diuretics).

Multivariable logistic regression and Receiver Operating Characteristic (ROC) curve analyses were performed to identify independent predictors and establish a combined prognostic model.

Results: Among 160 patients (mean age 56.4 +/- 10.2 years; 114 males [71.3 percent]), in-hospital MACE occurred in 28.8 percent (n = 46). Patients experiencing MACE presented with significantly higher admission NT-proBNP levels (3240 +/- 860 pg/mL vs. 980 +/- 340 pg/mL in non-MACE patients, $p < 0.001$), elevated hs-CRP (18.4 +/- 5.2 mg/L vs. 6.2 +/- 2.4 mg/L, $p < 0.001$), higher peak hs-cTnI (48.6 +/- 14.2 ng/mL vs. 22.4 +/- 8.6 ng/mL, $p < 0.001$), and lower left ventricular ejection fraction (LVEF: 36.4 +/- 6.8 percent vs. 48.2 +/- 7.2 percent, $p < 0.001$). Admission NT-proBNP > 1800 pg/mL predicted in-hospital MACE with an Area Under the Curve (AUC) of 0.864 (sensitivity 82.6 percent, specificity 80.7 percent, $p < 0.001$). Multivariable binary logistic regression revealed that admission Killip class >= II (adjusted OR = 4.12, 95 percent CI: 1.86 to 9.12, $p = 0.001$), NT-proBNP > 1800 pg/mL (adjusted OR = 3.86, 95 percent CI: 1.72 to 8.66, $p = 0.001$), LVEF < 40 percent (adjusted OR = 3.24, 95 percent CI: 1.44 to 7.28, $p = 0.004$), hs-CRP > 10 mg/L (adjusted OR = 2.64, 95 percent CI: 1.16 to 6.02, $p = 0.021$), and serum creatinine > 1.3 mg/dL (adjusted OR = 2.42, 95 percent CI: 1.06 to 5.52, $p = 0.036$) were independent predictors of in-hospital adverse outcomes. The combined clinical-biochemical model demonstrated superior predictive power (AUC = 0.912) compared to clinical parameters alone (AUC = 0.814, $p = 0.008$).

Conclusion: Integrating acute biochemical markers (NT-proBNP, hs-CRP, peak hs-cTnI, and serum creatinine) with established clinical parameters (Killip class and LVEF) significantly enhances early risk stratification for in-hospital MACE in STEMI patients. Routine dual clinical-biochemical profiling enables rapid identification of high-risk patients, facilitating timely therapeutic escalation and intensive monitoring.

Keywords: ST-elevation myocardial infarction; STEMI; Major adverse cardiac events; In-hospital outcomes; NT-proBNP; High-sensitivity C-reactive protein; Risk stratification.

1. Introduction

ST-elevation myocardial infarction (STEMI) is one of the most severe manifestations of acute coronary syndrome and remains a major cause of cardiovascular morbidity and mortality worldwide. It typically occurs following acute thrombotic occlusion of a coronary artery due to rupture or erosion of an atherosclerotic plaque, resulting in myocardial ischemia and subsequent cardiomyocyte necrosis. Despite significant advances in reperfusion strategies, particularly primary percutaneous coronary intervention (PCI), antithrombotic therapy, and intensive cardiovascular care, STEMI continues to be associated with considerable in-hospital mortality and complications. The clinical course of patients varies considerably, ranging from uncomplicated recovery to life-threatening complications, including acute heart failure, cardiogenic shock, malignant ventricular arrhythmias, recurrent myocardial ischemia, and mechanical complications. Therefore, early identification of patients at increased risk of adverse in-hospital outcomes is essential for improving clinical management and optimizing patient prognosis [1,2].

The prognosis of patients with STEMI is influenced by several demographic, clinical, and cardiovascular risk factors. Advanced age, diabetes mellitus, hypertension,

dyslipidemia, chronic kidney disease, previous myocardial infarction, and pre-existing heart failure have consistently been associated with unfavorable cardiovascular outcomes. Furthermore, clinical parameters at hospital admission, including heart rate, systolic blood pressure, oxygen saturation, Killip classification, and evidence of hemodynamic instability, provide important prognostic information. In particular, hypotension, tachycardia, pulmonary congestion, and impaired peripheral perfusion may indicate extensive myocardial injury and an increased likelihood of cardiogenic shock or in-hospital mortality. Established risk stratification tools, including the Global Registry of Acute Coronary Events (GRACE) and Thrombolysis in Myocardial Infarction (TIMI) scores, incorporate several of these variables to estimate cardiovascular risk and guide clinical decision-making [1,3,4].

In addition to conventional clinical characteristics, biochemical parameters provide valuable information regarding myocardial injury, systemic inflammation, metabolic disturbances, and organ dysfunction. Cardiac troponins, particularly high-sensitivity cardiac troponin I and T, are established biomarkers for diagnosing myocardial injury and assessing the severity of acute myocardial infarction. Elevated troponin concentrations may reflect greater myocardial damage and provide additional prognostic information regarding adverse cardiovascular outcomes. Creatine kinase-MB (CK-MB) may also contribute to the assessment of myocardial injury, particularly when interpreted alongside clinical findings and other biochemical markers. However, individual cardiac biomarkers may not fully represent the complex pathophysiological mechanisms responsible for adverse outcomes following STEMI [1,2].

Inflammatory and hematological markers have increasingly attracted attention as potential predictors of cardiovascular complications. White blood cell count, neutrophil count, lymphocyte count, neutrophil-to-lymphocyte ratio (NLR), and C-reactive protein (CRP) reflect the systemic inflammatory response associated with atherosclerotic plaque instability and myocardial necrosis. Following coronary artery occlusion, inflammatory activation contributes to myocardial tissue injury, infarct healing, and ventricular remodeling. An elevated NLR may indicate an exaggerated inflammatory response and has been investigated as a potential predictor of adverse cardiovascular outcomes. These readily available hematological parameters may therefore provide additional prognostic information when combined with conventional clinical assessment.

Renal function and metabolic parameters also play important roles in determining short-term outcomes in patients with STEMI. Serum creatinine, blood urea nitrogen, and estimated glomerular filtration rate (eGFR) are particularly relevant because impaired renal function is associated with increased cardiovascular risk, fluid and electrolyte disturbances, endothelial dysfunction, and altered drug metabolism. Similarly, admission hyperglycemia has been associated with unfavorable outcomes in patients with and without previously diagnosed diabetes mellitus. Elevated blood glucose may reflect increased sympathetic activation and physiological stress during acute myocardial injury. Furthermore, electrolyte abnormalities, particularly disturbances in serum sodium and potassium concentrations, may increase the risk of cardiac arrhythmias and hemodynamic instability.

Cardiac function represents another important determinant of in-hospital prognosis. Echocardiographic assessment, particularly left ventricular ejection fraction (LVEF), provides valuable information regarding myocardial contractility and the extent of ventricular dysfunction. Reduced LVEF following STEMI is associated with an increased risk of acute heart failure, ventricular arrhythmias, and adverse cardiovascular outcomes. Natriuretic peptides, including B-type natriuretic peptide (BNP) and N-terminal pro-B-type natriuretic peptide (NT-proBNP), may provide additional prognostic information regarding ventricular dysfunction and the development of heart failure [5].

Although established clinical risk scores provide useful prognostic information, predicting individual outcomes remains challenging because adverse events frequently result from interactions between myocardial injury, systemic inflammation, metabolic abnormalities, renal dysfunction, and hemodynamic compromise. Evaluating clinical and biochemical parameters independently may overlook important interactions between these variables. Consequently, integrating routinely available clinical characteristics with biochemical markers may provide a more comprehensive approach to risk stratification and potentially improve the identification of patients at increased risk of in-hospital complications.

Accordingly, the present study aims to investigate the association between clinical characteristics and biochemical parameters and the occurrence of adverse in-hospital outcomes among patients presenting with STEMI. Specifically, it seeks to identify independent clinical and biochemical predictors of unfavorable outcomes, evaluate the prognostic contribution of routinely available laboratory markers, and assess whether an integrated clinical-biochemical approach can improve risk stratification. Early identification of high-risk patients may facilitate timely therapeutic interventions, appropriate allocation of intensive care resources, closer clinical monitoring, and individualized patient management, ultimately contributing to improved short-term outcomes in patients with STEMI.

ST-elevation myocardial infarction (STEMI) represents the most acute and life-threatening presentation of coronary artery disease, caused by sudden atherothrombotic occlusion of an epicardial coronary artery (5). Despite widespread implementation of primary percutaneous coronary intervention (pPCI), modern pharmacotherapy, and standardized acute coronary syndrome (ACS) care pathways (2), in-hospital adverse events remain a primary determinant of short-term mortality and healthcare resource utilization (6).

In-hospital major adverse cardiac events (MACE)—including acute heart failure, cardiogenic shock, malignant tachyarrhythmias, re-infarction, and early mortality—typically manifest within the first 48 to 72 hours of admission (5). Early identification of patients at heightened risk for post-infarction failure is essential for guiding aggressive hemodynamic support, mechanical circulatory assistance, and intensive coronary care unit (ICCU) monitoring (5).

Traditional clinical risk scoring systems, such as the TIMI (Thrombolysis in Myocardial Infarction) and GRACE (Global Registry of Acute Coronary Events) risk scores, rely heavily on baseline clinical parameters (age, systolic blood pressure, heart rate, Killip class) (6). However, these clinical scores often fail to capture real-time subclinical pathophysiology, including systemic

neurohormonal activation, acute microvascular injury, dynamic myocardial wall stress, and intense systemic inflammatory cascades (7, 8).

Biochemical markers reflect distinct biological pathways involved in acute ischemic injury:

- Cardiac Troponins (hs-cTnI): Quantify the absolute magnitude of acute cardiomyocyte necrosis (9).
- Natriuretic Peptides (NT-proBNP): Reflect acute ventricular wall stretch, end-diastolic pressure elevation, and hemodynamically significant LV dysfunction (10).
- Inflammatory Biomarkers (hs-CRP): Measure the intensity of systemic and localized vascular inflammation, which drives post-infarction microvascular obstruction and adverse ventricular remodeling.
- Renal Function Markers (Serum Creatinine / eGFR): Indicate cardiorenal syndrome and impaired systemic organ perfusion.

Although individual biomarkers have been evaluated separately, a comprehensive model integrating acute biochemical profiling with established clinical predictors at emergency presentation has not been fully characterized.

Therefore, this prospective observational cohort study was conducted to evaluate the combined predictive value of clinical parameters and acute biochemical markers for in-hospital MACE in patients presenting with STEMI.

2. Rationale of the Study

Early risk stratification in STEMI is vital to prevent sudden in-hospital clinical deterioration. Relying solely on physical examination or baseline electrocardiography can underestimate the severity of ongoing myocardial injury or neurohormonal stress. Integrating quantitative acute biochemical markers (NT-proBNP, hs-CRP, serum creatinine) with clinical status provides clinicians with an objective diagnostic panel to identify patients at elevated risk for cardiogenic shock, acute heart failure, and early in-hospital death.

3. Objectives

Primary Objective: To evaluate the combined predictive value of clinical features and baseline biochemical markers (NT-proBNP, hs-CRP, peak hs-cTnI, serum creatinine) for in-hospital major adverse cardiac events (MACE) in STEMI patients.

Secondary Objectives:

1. To compare baseline clinical and biochemical profiles between STEMI patients who experience in-hospital MACE and those with an uncomplicated hospital course.
2. To determine the diagnostic accuracy, optimal cutoff thresholds, sensitivity, and specificity of admission NT-proBNP and hs-CRP for predicting in-hospital adverse events.
3. To assess the relationship between left ventricular ejection fraction (LVEF), Killip class, and biomarker elevation during the acute post-infarction period.

4. To develop a multivariable logistic regression model establishing independent predictors of in-hospital MACE in STEMI.

4. Materials and Methods

4.1 Study Design and Setting A hospital-based prospective observational cohort study was conducted at RLKU Medical College.

4.2 Study Duration The study was conducted over a 12-month period, encompassing emergency patient presentation, primary percutaneous or pharmacologic reperfusion management, serial blood biomarker collection, daily in-hospital monitoring, and discharge disposition.

4.3 Study Population and Selection A total of 160 adult patients (aged 30 to 75 years) presenting to the emergency department with acute STEMI within 12 hours of symptom onset were enrolled. STEMI was defined according to Fourth Universal Definition of Myocardial Infarction criteria: characteristic ischemic chest pain lasting >= 20 minutes accompanied by ST-segment elevation >= 1 mm in two anatomically contiguous leads (or >= 2 mm in leads V2-V3) or new left bundle branch block (LBBB).

4.4 Sample Size A sample size of 160 patients was calculated using G-Power software based on an estimated expected in-hospital MACE incidence of 25 to 30 percent, an alpha error level of 0.05, a statistical power of 85 percent, and accommodating up to 8 multivariable logistic regression covariates.

4.5 Inclusion Criteria

1. Adult patients aged 30 to 75 years presenting with confirmed acute STEMI within 12 hours of symptom onset.

2. Undergoing reperfusion therapy (primary PCI or pharmacologic thrombolysis).

3. Availability of complete baseline clinical assessment and blood sampling at admission.

4. Provision of written informed consent by the patient or legal guardian.

4.6 Exclusion Criteria

1. Presentation > 12 hours after symptom onset.

2. Known end-stage renal disease (eGFR < 15 mL/min/1.73m squared or chronic maintenance dialysis).

3. Active malignant neoplasm, severe chronic obstructive pulmonary disease (COPD), or systemic autoimmune disease.

4. Prior history of severe valvular heart disease or pre-existing cardiomyopathy.

5. In-hospital death occurring within 2 hours of presentation prior to baseline lab acquisition.

4.7 Clinical Management and Biomarker Assays All patients received standard acute STEMI care per international guidelines:

- Initial Clinical Evaluation: Heart rate, blood pressure, Killip class (Class I: no heart failure; Class II: rales/S3 gallop; Class III: overt pulmonary

edema; Class IV: cardiogenic shock), and door-to-balloon / door-to-needle times were recorded at admission. Echocardiography was performed within 24 hours to measure Left Ventricular Ejection Fraction (LVEF %).

- Reperfusion Strategy: Primary PCI was performed when feasible; thrombolysis with recombinant tissue plasminogen activator (rt-PA) or streptokinase was administered when primary PCI was delayed, followed by pharmaco-invasive angiography.

- Biochemical Quantification: Venous blood samples were collected upon arrival prior to heparinization:

    - High-sensitivity cardiac troponin I (hs-cTnI, ng/mL): Measured at arrival and 6, 12, and 24 hours to determine peak value (chemiluminescent immunoassay).

    - NT-proBNP (pg/mL): Quantified on arrival using electrochemiluminescence immunoassay (Roche Cobas).

    - hs-CRP (mg/L): Quantified using immunoturbidimetric assay.

    - Serum Creatinine (mg/dL) and Fasting Blood Glucose (mg/dL): Measured using standard clinical chemistry automated analyzers.

4.8 Primary Endpoint In-hospital Major Adverse Cardiac Events (MACE), defined as a composite of:

1. All-cause in-hospital mortality.

2. Cardiogenic shock (sustained hypotension with systolic blood pressure < 90 mmHg requiring vasopressors).

3. Acute pulmonary edema / acute heart failure requiring intravenous inotropes or invasive mechanical ventilation.

4. Sustained ventricular tachycardia / ventricular fibrillation (VT/VF) requiring electrical cardioversion/defibrillation.

5. Re-infarction during index admission.

4.9 Statistical Analysis Data were analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY). Continuous variables were tested for normality using the Kolmogorov-Smirnov test and expressed as mean +/- standard deviation (SD). Categorical variables were presented as numbers and percentages (n, percent).

Comparisons between MACE and non-MACE cohorts were conducted using Student's independent t-test for continuous parameters, and Pearson's Chi-square test for categorical parameters. Receiver Operating Characteristic (ROC) curve analysis evaluated diagnostic performance (AUC, sensitivity, specificity) for biomarker cutoff points. Multivariable binary logistic regression models identified independent predictors of in-hospital MACE. DeLong's test was used to compare AUCs between

model iterations. A p-value < 0.05 was considered statistically significant.

5. Results

5.1 Baseline Clinical and Demographic Characteristics The study cohort comprised 160 STEMI patients (mean age 56.4 +/- 10.2 years; 114 males [71.3 percent], 46 females [28.7 percent]). Primary PCI was performed in 65.0 percent (n = 104) and thrombolysis in 35.0 percent (n = 56). In-hospital MACE occurred in 28.8 percent (n = 46) of patients (including 12 in-hospital deaths [7.5 percent], 16 cases of cardiogenic shock [10.0 percent], 10 cases of acute severe heart failure [6.3 percent], 6 cases of sustained VT/VF [3.8 percent], and 2 re-infarctions [1.3 percent]). Baseline clinical features across groups are presented in Table 1

.

Clinical Parameter	Non-MACE Group (n = 114)	MACE Group (n = 46)	Total Cohort (n = 160)	p-value
Age (years)	54.8 +/- 9.6	60.4 +/- 10.8	56.4 +/- 10.2	0.002
Male Sex, n (%)	84 (73.7)	30 (65.2)	114 (71.3)	0.284
Female Sex, n (%)	30 (26.3)	16 (34.8)	46 (28.7)	0.284
Diabetes Mellitus, n (%)	38 (33.3)	26 (56.5)	64 (40.0)	0.007
Hypertension, n (%)	52 (45.6)	30 (65.2)	82 (51.3)	0.024
Anterior STEMI Location, n (%)	54 (47.4)	34 (73.9)	88 (55.0)	0.002
Admission Heart Rate (bpm)	78.4 +/- 12.6	96.8 +/- 16.4	83.6 +/- 15.8	<0.001
Admission Systolic BP (mmHg)	134.2 +/- 18.4	112.6 +/- 24.8	128.0 +/- 22.4	<0.001

Clinical Parameter	Non-MACE Group (n = 114)	MACE Group (n = 46)	Total Cohort (n = 160)	p-value
Admission Killip Class >= II, n (%)	18 (15.8)	28 (60.9)	46 (28.8)	<0.001
LVEF at 24 Hours (%)	48.2 +/- 7.2	36.4 +/- 6.8	44.8 +/- 8.6	<0.001

Table 1: Baseline clinical and hemodynamic characteristics of STEMI patients stratified by in-hospital MACE occurrence.

5.2 Laboratory Biochemical Profile Comparison Patients experiencing in-hospital MACE presented with marked elevations in cardiac necrosis, hemodynamic stress, inflammatory, and renal dysfunction biomarkers compared to those with an uncomplicated course ($p < 0.001$). Admission NT-proBNP was markedly higher in the MACE group (3240 +/- 860 pg/mL vs. 980 +/- 340 pg/mL, $p < 0.001$). Laboratory findings are detailed in Table 2.

Biochemical Biomarker	Non-MACE Group (n = 114)	MACE Group (n = 46)	p-value
Admission NT-proBNP (pg/mL)	980 +/- 340	3240 +/- 860	<0.001
Peak hs-cTnI (ng/mL)	22.4 +/- 8.6	48.6 +/- 14.2	<0.001
High-Sensitivity CRP (mg/L)	6.2 +/- 2.4	18.4 +/- 5.2	<0.001
Serum Creatinine (mg/dL)	0.98 +/- 0.22	1.48 +/- 0.38	<0.001

Biochemical Biomarker	Non-MACE Group (n = 114)	MACE Group (n = 46)	p-value
Fasting Blood Glucose (mg/dL)	138.4 +/- 32.6	188.6 +/- 48.2	<0.001
White Blood Cell Count (x10^9/L)	9.2 +/- 2.4	13.8 +/- 3.6	<0.001

Table 2: Comparison of acute biochemical laboratory parameters between groups.

5.3 Diagnostic Performance of Key Biomarkers (ROC Analysis) Receiver Operating Characteristic (ROC) curve analysis demonstrated that admission NT-proBNP and hs-CRP had strong prognostic accuracy for in-hospital MACE. Admission NT-proBNP achieved an AUC of 0.864 (sensitivity 82.6 percent, specificity 80.7 percent at a cutoff > 1800 pg/mL). Combining clinical predictors with all four biomarkers yielded the highest discrimination (AUC = 0.912).

Biomarker / Risk Model	AUC	95% CI	Cutoff Value	Sensitivity (%)	Specificity (%)	p-value
Admission NT-proBNP	0.864	0.804–0.924	> 1800 pg/mL	82.6 percent	80.7 percent	<0.001
High-Sensitivity CRP	0.828	0.760–0.896	> 10.0 mg/L	78.3 percent	77.2 percent	<0.001
Peak hs-cTnI	0.812	0.742–0.882	> 35.0 ng/mL	76.1 percent	75.4 percent	<0.001

Biomarker / Risk Model	AUC	95% CI	Cutoff Value	Sensitivity (%)	Specificity (%)	p-value
Clinical Model Alone (Age + Killip + LVEF)	0.814	0.744–0.884	N/A	73.9 percent	74.6 percent	<0.001
Combined Clinical + Biochemical Model	**0.912**	**0.864–0.960**	**N/A**	**87.0 percent**	**85.1 percent**	**<0.001**

Table 3: ROC curve performance of individual biomarkers and combined clinical-biochemical risk model for in-hospital MACE.

5.4 Multivariable Logistic Regression Analysis In multivariable binary logistic regression modeling for in-hospital MACE, admission Killip class >= II (adjusted OR = 4.12, 95 percent CI: 1.86 to 9.12, p = 0.001), admission NT-proBNP > 1800 pg/mL (adjusted OR = 3.86, 95 percent CI: 1.72 to 8.66, p = 0.001), LVEF < 40 percent (adjusted OR = 3.24, 95 percent CI: 1.44 to 7.28, p = 0.004), hs-CRP > 10 mg/L (adjusted OR = 2.64, 95 percent CI: 1.16 to 6.02, p = 0.021), and serum creatinine > 1.3 mg/dL (adjusted OR = 2.42, 95 percent CI: 1.06 to 5.52, p = 0.036) were confirmed as independent predictors.

Predictor Variable	Unadjusted OR (95% CI)	p-value	Adjusted OR (95% CI)	p-value
Killip Class >= II at Presentation	8.24 (3.92–17.32)	<0.001	**4.12 (1.86–9.12)**	**0.001**
Admission NT-proBNP > 1800 pg/mL	7.64 (3.62–16.14)	<0.001	**3.86 (1.72–8.66)**	**0.001**

Predictor Variable	Unadjusted OR (95% CI)	p-value	Adjusted OR (95% CI)	p-value
LVEF < 40 Percent	5.86 (2.82–12.18)	<0.001	**3.24 (1.44–7.28)**	**0.004**
High-Sensitivity CRP > 10 mg/L	4.92 (2.36–10.24)	<0.001	**2.64 (1.16–6.02)**	**0.021**
Serum Creatinine > 1.3 mg/dL	3.84 (1.82–8.12)	<0.001	**2.42 (1.06–5.52)**	**0.036**
Age > 65 Years	2.68 (1.28–5.62)	0.009	1.58 (0.68–3.68)	0.286
Anterior STEMI Location	3.14 (1.48–6.66)	0.003	1.82 (0.78–4.24)	0.164

Table 4: Multivariable logistic regression modeling independent predictors of in-hospital MACE in STEMI.

6. Discussion

This prospective observational study evaluated the combined predictive value of clinical parameters and acute biochemical markers for in-hospital MACE in STEMI patients. The primary finding demonstrates that integrating admission biochemical profiling (specifically NT-proBNP, hs-CRP, peak hs-cTnI, and serum creatinine) with established clinical parameters (Killip class and LVEF) significantly enhances early risk discrimination (AUC = 0.912) compared to clinical evaluation alone (AUC = 0.814) (11).

6.1 Neurohormonal Activation and NT-proBNP Dynamics

In the acute phase of STEMI, sudden ventricular ischemia induces regional wall motion abnormalities, elevating left ventricular end-diastolic pressure (LVEDP) and triggering immediate synthesis and release of NT-proBNP from ventricular cardiomyocytes (12). Patients presenting with elevated NT-proBNP (> 1800 pg/mL) experienced a nearly four-fold increased risk of in-hospital MACE (aOR = 3.86). Elevated

NT-proBNP acts as a sensitive surrogate for acute subclinical pump failure, microvascular hypoperfusion, and impending cardiogenic shock before frank clinical pulmonary edema manifests (13, 14).

6.2 Inflammatory Exudation via hs-CRP

In parallel with neurohormonal activation, acute myocardial infarction triggers a robust local and systemic inflammatory response (15). High-sensitivity C-reactive protein (hs-CRP) reflects hepatic acute-phase reactant synthesis driven by circulating IL-6 and TNF-alpha (16). Patients presenting with hs-CRP > 10 mg/L demonstrated higher rates of in-hospital adverse events (aOR = 2.64). Intense systemic inflammation contributes to microvascular obstruction, no-reflow phenomenon post-PCI, and impaired myocardial healing, predisposing patients to early malignant arrhythmias and heart failure (17, 18).

6.3 Cardiorenal Interactions and Necrosis Burden

Peak hs-cTnI levels reflect total infarcted myocardial mass (19). Elevated serum creatinine (> 1.3 mg/dL) acts as an independent predictor of adverse outcomes (aOR = 2.42), highlighting the critical prognostic impact of acute cardiorenal syndrome. Reduced renal perfusion combined with contrast-induced acute kidney injury exacerbates fluid overload and systemic toxicity in acute STEMI (20).

6.4 Clinical and Diagnostic Implications

These results support integrating an emergency dual clinical-biochemical risk score into acute chest pain triage protocols. Identifying patients with high NT-proBNP (> 1800 pg/mL) and elevated inflammatory markers upon admission alerts clinicians to assign high-risk status, ensuring placement in intensive cardiac care beds, early hemodynamic monitoring, targeted vasopressor support, and consideration of early mechanical circulatory support (10, 11).

6.5 Limitations

Limitations of this study include its single-center prospective design and a 12-month recruitment period. Furthermore, long-term post-discharge cardiovascular outcomes (such as 1-year mortality) were not included in this primary report on in-hospital events. Multi-center studies evaluating extended post-discharge outcomes are recommended.

7. Conclusion

Integrating acute biochemical profiling (NT-proBNP, hs-CRP, peak hs-cTnI, serum creatinine) with baseline clinical parameters (Killip class and LVEF) significantly improves early risk prediction for in-hospital MACE in STEMI patients. Admission NT-proBNP > 1800 pg/mL and hs-CRP > 10 mg/L serve as strong independent predictors of acute in-hospital heart failure, cardiogenic shock, and early mortality. Dual clinical-biochemical risk assessment should be integrated into acute coronary care protocols to optimize early management strategies.

8. Recommendations

1. Implement routine rapid testing of NT-proBNP, hs-CRP, and serum creatinine upon emergency arrival for all patients presenting with acute STEMI.
2. Utilize an admission NT-proBNP threshold of > 1800 pg/mL to identify patients at elevated risk for acute in-hospital heart failure and cardiogenic shock.
3. Combine quantitative biomarker measurements with traditional clinical risk scores (TIMI/GRACE) to improve early triage decision-making.
4. Assign patients flagged with dual clinical and biochemical risk to high-dependency or intensive cardiac care unit beds for continuous monitoring.
5. Conduct multi-center prospective trials evaluating whether biomarker-guided therapeutic escalation improves in-hospital survival rates in STEMI.

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