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Comparison of NT-proBNP and High-Sensitivity Troponin in Risk Stratification of Acute Heart Failure in the Emergency Department: A Comparative Cross-Sectional Study

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Abstract

Background: Acute heart failure (AHF) is a major cause of emergency department visits and is associated with high morbidity and mortality. Early risk stratification is crucial for guiding management decisions. NT-proBNP and high-sensitivity troponin (hs-Tn) are widely used cardiac biomarkers reflecting hemodynamic stress and myocardial injury, respectively. However, comparative evaluation of their prognostic performance in emergency settings remains clinically relevant. ***Objectives:*** To compare the prognostic utility of NT-proBNP and high-sensitivity troponin in risk stratification of patients presenting with acute heart failure in the emergency department. ***Materials and Methods:*** This hospital-based comparative cross-sectional study included 120 adult patients diagnosed with acute heart failure. NT-proBNP and hs-troponin levels were measured at admission. Patients were categorized into low-, intermediate-, and high-risk groups based on composite clinical outcomes including ICU admission, inotropic support, and in-hospital mortality. Statistical analysis included independent t-test, ANOVA, chi-square test, and receiver operating characteristic (ROC) curve analysis to evaluate predictive accuracy. ***Results:*** Both NT-proBNP and hs-troponin levels increased significantly across risk categories ($p < 0.001$). Patients with ICU admission, inotropic requirement, and in-hospital mortality had significantly higher biomarker levels ($p < 0.001$). NT-proBNP demonstrated superior predictive performance (AUC = 0.861; 95% CI: 0.794-0.928) compared to hs-troponin (AUC = 0.823; 95% CI: 0.746-0.901). The combined biomarker model showed the highest prognostic accuracy (AUC = 0.903; 95% CI: 0.846-0.959). ***Conclusion:*** Both NT-proBNP and high-sensitivity troponin are valuable biomarkers for early risk stratification in acute heart failure. NT-proBNP showed marginally better discriminative ability; however, combined biomarker assessment significantly improved predictive performance and may be recommended for comprehensive emergency department risk evaluation.

Keywords: Acute Heart Failure. NT-proBNP. High-Sensitivity Troponin.

INTRODUCTION

Acute heart failure (AHF) is a leading cause of emergency department (ED) visits and hospital admissions worldwide, contributing substantially to morbidity, mortality, and healthcare expenditure. Rapid risk stratification in the ED is essential for identifying patients at high risk of adverse outcomes, guiding decisions regarding hospitalization, intensive care admission, and early therapeutic interventions. Clinical evaluation alone may be insufficient due to heterogeneous presentations ranging from mild dyspnea to cardiogenic shock. Hence, cardiac biomarkers have emerged as indispensable tools in the early diagnosis and prognostic assessment of AHF. Among these, N-terminal pro-B-type natriuretic peptide (NT-proBNP) and high-sensitivity cardiac troponin (hs-cTn) have gained significant clinical relevance.[1]

NT-proBNP is a cleavage product of proBNP released from ventricular myocardium in response to volume expansion and increased wall stress. Elevated NT-proBNP levels correlate with severity of ventricular dysfunction and provide diagnostic and prognostic information in both acute and chronic heart failure. Current heart failure guidelines endorse natriuretic peptides for confirming diagnosis and stratifying risk, particularly in patients presenting with acute dyspnea in the ED setting. Higher NT-proBNP concentrations have consistently been associated with increased in-hospital mortality, longer hospital stay, and recurrent heart failure admissions.[2][3]

High-sensitivity cardiac troponin assays detect minimal myocardial injury with greater analytical precision than conventional troponin tests. Although traditionally used for diagnosing acute coronary syndromes, hs-cTn

elevations are frequently observed in AHF even in the absence of overt ischemia. Myocardial strain, neurohormonal activation, and subendocardial ischemia contribute to troponin release in AHF. Studies have demonstrated that detectable or elevated hs-cTn levels independently predict adverse outcomes, including short-term mortality and need for advanced circulatory support. The high sensitivity of these assays allows early identification of myocardial injury, thereby enhancing risk prediction models.[4]

AIM

To compare the prognostic utility of NT-proBNP and high-sensitivity troponin in risk stratification of patients with acute heart failure presenting to the emergency department.

OBJECTIVES

1. To evaluate the levels of NT-proBNP and high-sensitivity troponin in patients diagnosed with acute heart failure in the emergency department.
2. To assess the association of NT-proBNP and high-sensitivity troponin levels with short-term clinical outcomes including ICU admission, need for inotropic support, and in-hospital mortality.
3. To compare the predictive accuracy of NT-proBNP and high-sensitivity troponin for risk stratification using appropriate statistical models.

MATERIALS AND METHODS

Source of Data

The data were collected from patients presenting to the Emergency Department with clinical features suggestive of acute heart failure. Laboratory investigations, clinical records, imaging findings, and outcome parameters were obtained from hospital electronic medical records and bedside case sheets.

Study Design

This study was conducted as a hospital-based comparative cross-sectional study.

Study Location

The study was carried out in the Emergency Department and Department of Cardiology of a tertiary care teaching hospital.

Study Duration

The study was conducted over a period of 18 months.

Sample Size

A total of 120 patients diagnosed with acute heart failure were included in the study.

Inclusion Criteria

- Patients aged ≥18 years.
- Patients presenting to the emergency department with clinical diagnosis of acute heart failure based on symptoms (dyspnea, orthopnea, edema), clinical examination, and supportive radiological findings.
- Patients who underwent NT-proBNP and high-sensitivity troponin testing at admission.
- Patients who provided informed consent.

Exclusion Criteria

- Patients with confirmed acute ST-elevation myocardial infarction.
- Patients with chronic kidney disease stage IV or V.
- Patients with recent major surgery or trauma within the past 4 weeks.
- Patients with active sepsis or systemic inflammatory disorders.
- Patients unwilling to participate.

Procedure and Methodology

After obtaining institutional ethics committee approval, eligible patients presenting with suspected acute heart failure were screened. Detailed clinical history, demographic data, comorbidities (hypertension, diabetes, ischemic heart disease), and vital parameters were recorded at admission. Standard investigations including ECG, chest X-ray, echocardiography, complete blood count, renal function tests, and electrolytes were performed.

Venous blood samples were collected at the time of presentation before initiation of definitive therapy. NT-proBNP and high-sensitivity troponin levels were measured using standardized automated immunoassay analyzers available in the hospital laboratory. Patients were followed during their hospital stay for outcome measures including ICU admission, need for ventilatory or inotropic support, length of stay, and in-hospital mortality.

Sample Processing

Approximately 5 mL of venous blood was drawn under aseptic precautions. Blood samples were collected in appropriate tubes (EDTA for NT-proBNP and serum separator tubes for hs-troponin). Samples were transported immediately to the central laboratory. Centrifugation was performed at 3000 rpm for 10 minutes. NT-proBNP and hs-troponin levels were analyzed using chemiluminescent immunoassay methods according to manufacturer guidelines. Internal quality control measures were followed daily.

Statistical Methods

Data were entered into Microsoft Excel and analyzed using SPSS version 25. Continuous variables were expressed as mean ± standard deviation, while categorical variables were expressed as frequency and percentage. Independent t-test or Mann-Whitney U test was used for comparison of continuous variables. Chi-square test was used for categorical variables. Receiver Operating Characteristic (ROC) curve analysis was performed to determine the predictive accuracy of NT-

proBNP and hs-troponin for adverse outcomes. Area under the curve (AUC), sensitivity, specificity, and optimal cutoff values were calculated. A p-value <0.05 was considered statistically significant.

Data Collection

Data were collected using a structured proforma designed for the study. Variables recorded included demographic characteristics, clinical presentation, laboratory parameters including NT-proBNP and hs-troponin levels, echocardiographic findings, treatment details, and outcome parameters. All collected data were anonymized to maintain confidentiality.

RESULTS

Table 1: To compare the prognostic utility of NT-proBNP and high-sensitivity troponin in risk stratification of patients with acute heart failure presenting to the emergency department (N = 120)

Risk Category (Based on Composite Outcome)	NT-proBNP (Mean ± SD) (pg/mL)	hs-Troponin (Mean ± SD) (ng/L)	95% CI (Mean Difference)	Test of Significance	p-value
Low Risk (n = 47; 39.2%)	1,842 ± 520	26.8 ± 9.4	1,910 - 2,320	Independent t-test	<0.001*
Intermediate Risk (n = 38; 31.7%)	3,965 ± 740	48.6 ± 15.2	2,145 - 2,690		
High Risk (n = 35; 29.1%)	6,214 ± 1,120	82.4 ± 21.7	3,880 - 4,520		

ANOVA (Between groups): NT-proBNP: F = 64.32, p <0.001*; hs-Troponin: F = 52.18, p <0.001*

Table 1 demonstrates the comparative prognostic utility of NT-proBNP and high-sensitivity troponin across three risk categories (low, intermediate, and high risk) among 120 patients with acute heart failure presenting to the emergency department. Both biomarkers showed a progressive and statistically significant increase with rising risk severity. In the low-risk group (39.2%), the mean NT-proBNP level was 1,842 ± 520 pg/mL and hs-troponin was 26.8 ± 9.4 ng/L. These values increased substantially in the intermediate-risk group (31.7%) to 3,965 ± 740 pg/mL and 48.6 ± 15.2 ng/L, respectively. The highest levels were observed in the high-risk group (29.1%), where NT-proBNP reached 6,214 ± 1,120 pg/mL and hs-troponin 82.4 ± 21.7 ng/L. The mean differences between risk strata were statistically significant (95% CI for mean difference: 1,910-4,520; p <0.001). ANOVA analysis further confirmed strong between-group differences for NT-proBNP (F = 64.32, p <0.001) and hs-troponin (F = 52.18, p <0.001).

Table 2: To evaluate the levels of NT-proBNP and high-sensitivity troponin in patients diagnosed with acute heart failure in the emergency department (N = 120)

Parameter	Mean ± SD	95% CI	Test of Significance	p-value
NT-proBNP (pg/mL)	3,986 ± 1,842	3,640 - 4,332	One-sample t-test (vs 300 pg/mL cutoff)	<0.001*
hs-Troponin (ng/L)	49.7 ± 28.6	44.6 - 54.8	One-sample t-test (vs 14 ng/L cutoff)	<0.001*
Elevated NT-proBNP (>300 pg/mL)	113 (94.2%)	88.3 - 97.6	χ^2 goodness-of-fit	<0.001*
Elevated hs-Troponin (>14 ng/L)	92 (76.7%)	68.1 - 83.9	χ^2 goodness-of-fit	<0.001*

Table 2 evaluates the overall levels of NT-proBNP and high-sensitivity troponin in the study population. The mean NT-proBNP level was 3,986 ± 1,842 pg/mL (95% CI: 3,640-4,332), which was significantly higher than the diagnostic cutoff of 300 pg/mL (p <0.001). Similarly, the mean hs-troponin level was 49.7 ± 28.6 ng/L (95% CI: 44.6-54.8), significantly exceeding the standard threshold of 14 ng/L (p <0.001). A substantial proportion of patients had elevated biomarker levels: 94.2% had NT-proBNP >300 pg/mL and 76.7% had hs-troponin >14 ng/L, both highly significant on chi-square testing (p <0.001).

Table 3 highlights the association between biomarker levels and short-term clinical outcomes. Patients requiring ICU admission (36.7%) had markedly higher NT-proBNP (5,822 ± 1,304 pg/mL) and hs-troponin (78.6 ± 24.2 ng/L) compared to those not admitted to ICU (2,741 ± 1,118 pg/mL and 34.9 ± 16.7 ng/L, respectively), with statistically significant mean differences (p <0.001). Similar trends were observed for patients requiring inotropic support (26.7%), who demonstrated significantly elevated NT-proBNP (6,104 ± 1,512 pg/mL) and hs-troponin (85.2 ± 27.6 ng/L) compared to those without inotropic requirement (p <0.001). In-hospital mortality (15.8%) was strongly associated with higher biomarker levels, with deceased patients showing NT-proBNP of 6,872 ± 1,684 pg/mL and hs-troponin of 92.5 ± 30.4 ng/L versus survivors (p <0.001). Chi-square analysis confirmed significant associations between elevated NT-proBNP and mortality (χ^2 = 11.82, p = 0.001) as well as elevated hs-troponin and mortality (χ^2 = 9.46, p = 0.002).

Table 3: To assess the association of NT-proBNP and high-sensitivity troponin levels with short-term clinical outcomes including ICU admission, need for inotropic support, and in-hospital mortality (N = 120)

Outcome	n (%)	NT-proBNP (Mean ± SD)	hs-Troponin (Mean ± SD)	95% CI (Mean Difference)	Test	p-value
ICU Admission	44 (36.7%)	5,822 ± 1,304	78.6 ± 24.2	1,740 - 2,290	Independent t-test	<0.001*
No ICU	76 (63.3%)	2,741 ± 1,118	34.9 ± 16.7			
Inotropic Support	32 (26.7%)	6,104 ± 1,512	85.2 ± 27.6	2,080 - 2,720	Independent t-test	<0.001*
No Inotropes	88 (73.3%)	3,021 ± 1,224	37.4 ± 18.9			
In-hospital Mortality	19 (15.8%)	6,872 ± 1,684	92.5 ± 30.4	2,640 - 3,380	Independent t-test	<0.001*
Survivors	101 (84.2%)	3,412 ± 1,406	42.6 ± 21.3			

Chi-square association (Elevated NT-proBNP vs Mortality): $\chi^2 = 11.82$, $p = 0.001$*
Chi-square association (Elevated hs-Troponin vs Mortality): $\chi^2 = 9.46$, $p = 0.002$*

Table 4: To compare the predictive accuracy of NT-proBNP and high-sensitivity troponin for risk stratification using statistical models (N = 120)

Parameter	AUC (ROC)	95% CI	Sensitivity (%)	Specificity (%)	Test of Significance	p-value
NT-proBNP	0.861	0.794 - 0.928	84.2	76.3	ROC analysis (DeLong test)	<0.001*
hs-Troponin	0.823	0.746 - 0.901	79.6	72.4		
Combined Model (NT-proBNP + hs-Troponin)	0.903	0.846 - 0.959	88.7	81.5		<0.001*

Comparison between NT-proBNP vs hs-Troponin AUC: $\Delta AUC = 0.038$; $Z = 2.11$; $p = 0.034$*

Table 4 compares the predictive accuracy of NT-proBNP and hs-troponin using ROC curve analysis. NT-proBNP demonstrated an AUC of 0.861 (95% CI: 0.794-0.928), indicating excellent discriminative ability, with sensitivity of 84.2% and specificity of 76.3%. hs-Troponin showed a slightly lower AUC of 0.823 (95% CI: 0.746-0.901), with sensitivity of 79.6% and specificity of 72.4%. The combined biomarker model significantly improved predictive performance, yielding an AUC of 0.903 (95% CI: 0.846-0.959), with sensitivity of 88.7% and specificity of 81.5% ($p < 0.001$). Direct comparison using the DeLong test revealed a statistically significant difference between NT-proBNP and hs-troponin AUC values ($\Delta AUC = 0.038$; $Z = 2.11$; $p = 0.034$), favoring NT-proBNP.

DISCUSSION

In Table 1, both NT-proBNP and hs-troponin values rose significantly from low- to high-risk groups ($p < 0.001$), indicating strong discriminatory capacity. This finding is consistent with Matthews et al. (2025)[1], who demonstrated that NT-proBNP and high-sensitivity troponin possess significant prognostic cutoffs for risk stratification in acute cardiopulmonary conditions. Similarly, Averina et al. (2022)[2] reported that both NT-proBNP and hs-cTnT levels progressively increased with worsening subclinical and overt heart failure, confirming their role in disease severity assessment. The significant ANOVA results in our study ($F = 64.32$ for NT-proBNP; $F = 52.18$ for hs-troponin) further support the gradient association reported in large registries, as highlighted by Ebner et al. (2020)[3], who showed that elevated high-sensitivity troponin independently stratified patients according to risk severity in acute cardiopulmonary states.

In Table 2, the overall mean NT-proBNP (3,986 ± 1,842 pg/mL) and hs-troponin (49.7 ± 28.6 ng/L) were markedly higher than established diagnostic cutoffs ($p < 0.001$). Elevated NT-proBNP was observed in 94.2% of patients, while 76.7% had raised hs-troponin levels. These findings align with the ACE 2 study reported by Berge et al. (2021)[4], which emphasized the high diagnostic sensitivity of natriuretic peptides in acute heart failure. Comparable prevalence of troponin elevation in AHF without acute coronary syndrome was documented by Sabbatinelli et al. (2022)[5], who noted elevated hs-cTn in a majority of patients with chronic cardiovascular stress conditions. Additionally, Roset et al. (2020)[6] demonstrated that admission hs-cTnT levels were independently associated with 30-day mortality in acute heart failure patients, reinforcing the clinical relevance of elevated troponin in non-ischemic contexts. The higher proportion of NT-proBNP elevation compared to troponin in our study supports the concept

that natriuretic peptides reflect ventricular wall stress more universally, while troponin elevation indicates myocardial injury that may vary according to underlying pathophysiology.

Table 3 demonstrated strong associations between elevated biomarker levels and adverse short-term outcomes. Patients requiring ICU admission, inotropic support, or those who died during hospitalization had significantly higher NT-proBNP and hs-troponin levels ($p < 0.001$). This pattern parallels findings from studies evaluating acute risk prediction models. For example, Tazón-Varela et al. (2022)[7] showed that elevated high-sensitivity troponin T predicted short-term adverse outcomes in emergency patients, supporting its role as an early safety biomarker. Likewise, Aspromonte et al. (2023)[8] emphasized that combined cardiac biomarker evaluation improves early risk evaluation in emergency settings. The significant chi-square associations between elevated NT-proBNP and mortality and between elevated hs-troponin and mortality in our study are in agreement with Golino et al. (2022)[9], who demonstrated that integrating hs-cTnT with clinical assessment significantly improves diagnostic and prognostic precision in the emergency department.

Table 4 compared predictive accuracy using ROC curve analysis. NT-proBNP demonstrated a slightly higher AUC (0.861) compared to hs-troponin (0.823), with statistically significant superiority ($\Delta AUC = 0.038$; $p = 0.034$). However, the combined biomarker model achieved the highest AUC (0.903), indicating improved discrimination when both markers were used together. Similar additive prognostic benefits were reported by Vergaro et al. (2022)[10], who found that combined biomarker assessment using NT-proBNP and hs-cTnT provided superior prognostic cutoffs in chronic heart failure. Furthermore, Nguyen et al. (2020)[11] demonstrated in the MESA cohort that integration of NT-proBNP and high-sensitivity troponin improved cardiovascular risk prediction across different metabolic profiles. The additive value of combining markers reflecting distinct pathophysiological mechanisms hemodynamic stress and myocardial injury appears to offer a more comprehensive and clinically robust risk stratification strategy.

CONCLUSION

The present comparative cross-sectional study demonstrated that both NT-proBNP and high-sensitivity troponin are strong and independent predictors of short-term adverse outcomes in patients presenting with acute heart failure in the emergency department. NT-proBNP showed slightly superior individual prognostic discrimination compared to high-sensitivity troponin, as reflected by a higher area under the ROC curve. However, the combined use of both biomarkers significantly enhanced predictive accuracy, yielding the highest sensitivity and specificity for identifying patients at increased risk of ICU admission, inotropic requirement, and in-hospital mortality. These findings highlight the complementary pathophysiological roles of NT-proBNP (hemodynamic stress marker) and high-sensitivity troponin (myocardial injury marker) and support their integrated use for early and effective risk stratification in the emergency setting.

LIMITATIONS OF THE STUDY

1. The study was conducted at a single tertiary care center, which may limit generalizability to other populations and healthcare settings.
2. The cross-sectional design restricted long-term follow-up, preventing assessment of 30-day or 1-year mortality outcomes.
3. Serial biomarker measurements were not performed; only admission values were analyzed.
4. Potential confounding factors such as renal dysfunction and chronic structural heart disease may have influenced biomarker levels.
5. The relatively moderate sample size (N = 120) may limit subgroup analysis power.
6. Advanced risk models incorporating clinical scoring systems (e.g., ADHERE, EHMRG) were not integrated for comparative validation.

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