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Admission NT-proBNP-to-Albumin Ratio, Systemic Immune-Inflammation Index, and Early Severe Clinical Deterioration in Acute Decompensated Heart Failure

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Abstract

Background

Early risk stratification in acute decompensated heart failure (ADHF) remains challenging in the emergency department. This study aimed to evaluate the association between admission-based laboratory biomarkers, composite inflammatory indices, and early severe clinical deterioration in patients hospitalized with ADHF.

Methods

This retrospective single-center cohort study included 1,450 adult patients hospitalized from the emergency department with ADHF between January 2023 and January 2025. Admission laboratory biomarkers and composite indices, including the systemic immune-inflammation index (SII), C-reactive protein-to-albumin ratio (CAR), prognostic nutritional index (PNI), and log10 NT-proBNP-to-albumin ratio, were evaluated. The primary outcome was a composite severe clinical course defined as intensive care unit admission, non-invasive ventilation (NIV), or endotracheal intubation during index hospitalization.

Results

Severe clinical course occurred in 338 patients (23.3%). In multivariable analysis, the log10 NT-proBNP-to-albumin ratio remained independently associated with severe clinical course in both tested models (Model 1: OR 2.008, 95% CI 1.493–2.701; Model 2: OR 1.988, 95% CI 1.498–2.640; both $p < 0.001$). In the extended model, SII was also independently associated with severe clinical course (OR 1.179 per doubling, 95% CI 1.075–1.293, $p < 0.001$). ROC curve analysis demonstrated statistically significant but limited discriminatory performance for both the log10 NT-proBNP-to-albumin ratio (AUC 0.608, 95% CI 0.570–0.646, $p < 0.001$) and SII (AUC 0.595, 95% CI 0.559–0.631, $p < 0.001$).

Conclusion

In patients hospitalized with ADHF, early severe clinical deterioration was associated with higher inflammatory, cardiorenal, and myocardial stress markers. The log10 NT-proBNP-to-albumin ratio showed the most consistent independent association, while SII provided an additional inflammatory risk signal. These routinely available admission-based indices may provide complementary information for early risk assessment but demonstrated only modest discriminatory performance.

Introduction

Heart failure is a major global health problem associated with substantial morbidity, mortality, and healthcare utilization. Its prevalence increases with age, and acute decompensated heart failure (ADHF)

remains a common cause of emergency department visits and unplanned hospitalization. ADHF represents a clinically vulnerable phase during which patients may rapidly develop respiratory failure, hemodynamic instability, or require intensive care (1).

Respiratory compromise is an important determinant of early severity in ADHF. Patients with pulmonary congestion or cardiogenic pulmonary edema may require non-invasive ventilation (NIV) or invasive mechanical ventilation. In a large National Inpatient Sample analysis of 6,534,675 acute heart failure hospitalizations, NIV was required in 4.16% and invasive mechanical ventilation in 0.79% of patients within the first 24 hours. Corresponding in-hospital mortality rates were 5.0% and 27%, respectively, compared with 2.1% among patients without respiratory support (2). These findings highlight the clinical importance of early recognition of patients requiring respiratory support or intensive care in the emergency department (3).

Early risk stratification in ADHF remains challenging because clinical deterioration involves multiple mechanisms, including congestion, myocardial stress, systemic inflammation, renal dysfunction, and impaired physiological reserve. Previous studies have demonstrated the value of readily available clinical variables for predicting short-term outcomes in acute heart failure (4). Admission-based laboratory indices may provide additional information for early risk assessment; however, evidence regarding their association with severe in-hospital deterioration requiring respiratory support or intensive care remains limited.

This study aimed to evaluate the association of admission laboratory biomarkers and derived composite indices with early severe clinical deterioration among patients hospitalized from the emergency department with ADHF. The composite outcome included endotracheal intubation, NIV, or intensive care unit admission during the index hospitalization. We sought to assess whether routinely available laboratory indices could provide complementary information for early risk stratification.

Methods

Study Design and Participants

This retrospective, observational, single-center cohort study was conducted at the Emergency Department of Muğla Training and Research Hospital. Consecutive adult patients (≥ 18 years) hospitalized with acute decompensated heart failure (ADHF) between January 1, 2023, and January 1, 2025, were included. ADHF was diagnosed in the emergency department based on new-onset or worsening heart failure symptoms and signs, including dyspnea, orthopnea, pulmonary crackles, peripheral edema, and clinical or radiographic evidence of congestion. NT-proBNP levels and chest imaging findings were considered supportive diagnostic evidence, consistent with the 2021 European Society of Cardiology (ESC) heart failure guidelines and their 2023 focused update. Patients with known heart failure were evaluated for acute decompensation, whereas cardiology consultation was obtained for diagnostic confirmation in patients with suspected newly diagnosed heart failure.

Eligible patients were those hospitalized in a general ward or intensive care unit following an emergency department diagnosis of ADHF, with complete admission laboratory data, including complete blood count, routine biochemical parameters, and NT-proBNP. Patients discharged directly from the emergency department, those younger than 18 years, and those with missing clinical, laboratory, or outcome data were excluded. For patients with multiple eligible admissions, only the first admission was included. The screening and selection process is presented in Fig. 1.

Patient records were retrospectively reviewed using the hospital information management system. The study was conducted in accordance with the Declaration of Helsinki and approved by the Muğla Sıtkı Koçman University Medical Ethics Committee (Approval No: 250175-181). Informed consent was waived due to the retrospective design, and all data were anonymized before analysis.

Data Collection

All data were retrospectively collected from the hospital information management system. Demographic characteristics, including age and sex were recorded. Laboratory parameters obtained at the time of emergency department admission were analyzed. Hematological variables included hemoglobin, white blood cell (WBC) count, platelet count, neutrophil count, lymphocyte count, and red cell distribution width (RDW). Biochemical parameters included urea, creatinine, sodium, potassium, chloride, albumin, high-sensitivity cardiac troponin T (hs-cTnT), C-reactive protein (CRP), and N-terminal pro–B-type natriuretic peptide (NT-proBNP).

In addition to individual laboratory parameters, composite indices reflecting systemic inflammation, nutritional status, and cardiorenal interaction were calculated based on admission values. The primary indices of interest were defined a priori and included the CRP-to-albumin ratio (CAR), systemic immune–inflammatory index (SII; calculated as platelet count × neutrophil count / lymphocyte count), prognostic nutritional index (PNI; calculated as 10 × albumin [g/dL] + 0.005 × lymphocyte count [/mm³]), and a log10 NT-proBNP-to-albumin ratio (defined as log10 [NT-proBNP/albumin]).

Additional exploratory indices were also evaluated. These included the red cell distribution width–to-albumin ratio (RAR), lymphocyte-to-C-reactive protein ratio (LCR), and BUN-related variables. BUN was estimated from serum urea levels using the standard conversion formula (BUN = urea/2.14), and both estimated BUN and a BUN-to-albumin ratio–like index (BAR-like index) were included in the analysis. All variables were defined using the first laboratory measurements obtained at the time of emergency department admission, prior to the initiation of in-hospital treatment.

Study Outcomes

The primary outcome was a composite of early severe clinical deterioration, defined as the occurrence of any of the following events during the index hospitalization: endotracheal intubation, non-invasive ventilation (NIV), or intensive care unit admission. NIV included continuous positive airway pressure

(CPAP) and bilevel positive airway pressure (BiPAP), whereas endotracheal intubation was recorded separately as an invasive airway intervention. These endpoints were selected to reflect the need for advanced respiratory support or escalation of care.

Statistical Analysis

Statistical analyses were performed using SPSS version 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables were presented as mean ± standard deviation or median (interquartile range [IQR]), according to their distribution, and categorical variables as frequencies and percentages. Normality was assessed using the Kolmogorov–Smirnov test with Lilliefors correction and visual inspection of histograms. Comparisons between patients with and without severe clinical outcomes were performed using the independent-samples t-test, Mann–Whitney U test, or chi-square test, as appropriate. Univariable logistic regression analyses were performed to evaluate associations between laboratory parameters, derived indices, and severe clinical outcomes. Variables selected on the basis of clinical relevance and/or univariable associations were entered into multivariable logistic regression models. Results were expressed as odds ratios (ORs) with 95% confidence intervals (CIs).

Because SII was markedly right-skewed (skewness = 5.38) and violated the linearity assumption on the logit scale (Box–Tidwell test, p = 0.002), it was log2-transformed before inclusion in regression models (Box–Cox $\lambda \approx 0$). Accordingly, the ORs for log2-SII represent the change in odds associated with each doubling of SII. The linearity assumption for log2-SII was evaluated using restricted cubic splines (p for nonlinearity = 0.67). Sensitivity analyses were also performed using SII quartiles, with the lowest quartile as the reference category. Five patients had physiologically implausible leukocyte differential counts, with the sum of neutrophil and lymphocyte counts exceeding the total white blood cell count. These values were considered potential data-entry errors, and the affected differential counts and derived indices were treated as missing.

Receiver operating characteristic (ROC) curve analysis was used to assess the discriminatory performance of selected indices. Areas under the curve (AUCs) with 95% CIs were calculated, and optimal cut-off values were determined using the Youden index. Corresponding sensitivity and specificity values were reported. A two-sided p-value < 0.05 was considered statistically significant.

Results

A total of 1,450 patients were included in the study, of whom 338 (23.3%) experienced a severe clinical outcome. Age and sex did not differ significantly between groups. Patients with severe outcomes had higher inflammatory markers, renal function parameters, high-sensitivity cardiac troponin T, and NT-proBNP levels, together with lower lymphocyte counts. Among the derived indices, SII (1605 vs. 1177, p < 0.001), CAR (0.59 vs. 0.41, p = 0.003), and the log10 NT-proBNP-to-albumin ratio (2.37 vs. 2.15, p < 0.001) were higher in the severe outcome group, whereas PNI was lower (42.0 vs. 43.6, p = 0.002). Detailed baseline characteristics and between-group comparisons are presented in Table 1.

In univariable logistic regression analysis, the log10 NT-proBNP-to-albumin ratio (OR 2.091, 95% CI 1.597–2.737, p < 0.001) and log2-transformed SII (OR 1.240 per doubling, 95% CI 1.144–1.345, p < 0.001) were significantly associated with severe clinical outcomes. Estimated BUN, the BUN-to-albumin ratio–like index, and creatinine also showed significant positive associations, whereas LCR was inversely associated with severe outcomes. In contrast, CAR, PNI, and RAR were not significantly associated with the outcome. Detailed univariable regression results are presented in Table 2.

In multivariable logistic regression analysis, the log10 NT-proBNP-to-albumin ratio remained independently associated with severe clinical outcomes in both models (Model 1: OR 2.008, 95% CI 1.493–2.701, p < 0.001; Model 2: OR 1.988, 95% CI 1.498–2.640, p < 0.001). In Model 2, log2-transformed SII was also independently associated with severe outcomes (OR 1.179 per doubling, 95% CI 1.075–1.293, p < 0.001). Age and estimated BUN were not independently associated with severe outcomes after adjustment. In the quartile-based sensitivity analysis, only the highest SII quartile was independently associated with severe outcomes compared with the lowest quartile (OR 2.128, 95% CI 1.413–3.205, p < 0.001; p for trend < 0.001), whereas the second and third quartiles were not significantly associated with the outcome. Detailed multivariable regression results are presented in Table 3.

ROC curve analysis demonstrated modest discriminatory performance for both indices. The log10 NT-proBNP-to-albumin ratio had an AUC of 0.608 (95% CI 0.570–0.646, p < 0.001), with an optimal cut-off of 2.2511, sensitivity of 60.2%, and specificity of 58.6%. SII had an AUC of 0.595 (95% CI 0.559–0.631, p < 0.001), with an optimal cut-off of 2876.9, sensitivity of 36.1%, and specificity of 79.5%. The corresponding Youden indices were 0.189 and 0.156, respectively. Despite statistically significant associations with severe clinical outcomes, both indices demonstrated limited standalone discriminatory performance (Table 4).

Discussion

In this retrospective cohort of patients hospitalized with acute decompensated heart failure (ADHF), severe clinical deterioration was associated with higher admission levels of inflammatory markers, NT-proBNP, hs-cTnT, and renal function parameters, whereas age and sex were comparable between outcome groups. After multivariable adjustment, the log10 NT-proBNP-to-albumin ratio remained independently associated with severe clinical outcomes across both models, while SII provided an additional independent association. These findings suggest that admission-based indices reflecting cardiac stress and systemic inflammation may provide complementary information regarding the risk of early clinical deterioration in hospitalized patients with ADHF.

The differences in laboratory profiles between outcome groups are consistent with previous studies demonstrating associations between renal-metabolic markers, systemic inflammation, and adverse outcomes in heart failure. Blood urea nitrogen, albumin, and lactate have been associated with short-term mortality risk in ADHF (5), while renal dysfunction and azotemia have been linked to adverse clinical outcomes (6). Previous investigations have also identified associations between SII and mortality in

heart failure (7). Furthermore, elevated hs-cTnT and NT-proBNP levels have been associated with impaired cardiac function, cardiorenal involvement, and adverse prognosis in patients with heart failure (8, 9).Together, these findings suggest that the laboratory differences observed in our cohort may reflect overlapping processes involving myocardial stress, systemic inflammation, and renal dysfunction.

Among the evaluated indices, the log10 NT-proBNP-to-albumin ratio showed the most consistent independent association with severe clinical outcomes. NT-proBNP reflects myocardial wall stress and hemodynamic congestion, whereas albumin may be influenced by nutritional status, systemic inflammation, hepatic congestion, and physiological reserve. Previous studies have demonstrated the prognostic relevance of NT-proBNP in heart failure. Carubelli et al. reported that changes in NT-proBNP during hospitalization were associated with subsequent cardiovascular death or rehospitalization, although NT-proBNP-guided treatment intensification did not improve outcomes (10). Dai et al. also reported associations between admission natriuretic peptide-based measurements and both in-hospital and long-term outcomes (11). Regarding albumin, Peng et al. identified an association between hypoalbuminemia and increased mortality in acute heart failure, while Rehman et al. reported hypoalbuminemia as an independent predictor of in-hospital mortality in ADHF (12, 13). Combining NT-proBNP and albumin may therefore capture complementary aspects of cardiac stress and systemic illness. However, the modest discriminatory performance observed in our ROC analysis indicates that this ratio should not be considered a stand-alone prognostic tool.

SII was also independently associated with severe clinical outcomes, supporting the potential relevance of systemic inflammation in ADHF. By integrating platelet, neutrophil, and lymphocyte counts, SII reflects several components of the inflammatory and immune response. Previous studies have associated SII with adverse outcomes in heart failure populations (7, 14), while other inflammatory markers and indices, including CRP, NLR, and CRP–NLR-based parameters, have been linked to mortality and rehospitalization (15, 16). Population-based evidence has also demonstrated associations between inflammatory indices and the presence of heart failure (17). In our study, each doubling of SII was associated with approximately 18% higher adjusted odds of severe clinical deterioration. The quartile-based sensitivity analysis further indicated that the association was most evident in the highest SII quartile. Nevertheless, the limited ROC discrimination suggests that SII provides complementary rather than definitive prognostic information.

Renal dysfunction was another characteristic of patients experiencing severe clinical deterioration. Admission urea and creatinine levels were higher in the severe outcome group, while estimated BUN, the BAR-like index, and creatinine were associated with severe outcomes in univariable analyses. Previous studies have demonstrated associations between BUN and adverse outcomes in ADHF (5), as well as between cardiorenal dysfunction and mortality, rehospitalization, and renal disease progression (18, 19). Higher BUN-to-albumin ratios have also been associated with increased mortality risk in heart failure (20). However, estimated BUN did not retain an independent association in our multivariable models. This finding may reflect overlapping information captured by renal parameters and other markers of systemic illness and cardiac stress, although residual confounding cannot be excluded.

Although CAR, RAR, PNI, and LCR reflect different aspects of inflammation, nutritional status, and hematological alterations, their associations with severe clinical outcomes were less consistent in our cohort. Previous studies have reported associations of CAR and PNI with adverse outcomes in heart failure and other high-risk populations (21, 22). However, these exploratory indices did not emerge as prominent independent predictors in our analysis, whereas the log10 NT-proBNP-to-albumin ratio and SII showed more consistent associations with early severe clinical deterioration.

These findings may have implications for early risk assessment in the emergency department. As routinely available admission-based indices, the log10 NT-proBNP-to-albumin ratio and SII may provide complementary information for identifying hospitalized patients at increased risk of deterioration. However, given their modest discriminatory performance, these indices should be considered adjuncts to clinical assessment rather than stand-alone decision-making tools.

This study has several limitations. First, its retrospective, single-center design limits causal inference and generalizability. Second, all indices were calculated using laboratory values obtained at emergency department admission, without evaluating dynamic biomarker changes during hospitalization. Third, potentially relevant clinical variables, including left ventricular ejection fraction, echocardiographic phenotype, hemodynamic parameters, treatment strategies, diuretic response, and concomitant infections, were not comprehensively evaluated. Major comorbidities, including diabetes mellitus, hypertension, and chronic kidney disease, were not collected and therefore could not be included in multivariable analyses, potentially introducing residual confounding. Information on heart failure etiology, previous heart failure diagnosis, and prior heart failure-related hospitalizations was also unavailable, limiting clinical characterization of the study population. Furthermore, the composite outcome comprised heterogeneous endpoints, including intensive care unit admission, non-invasive ventilation (NIV), and endotracheal intubation, which may differ in clinical severity and determinants. Post-discharge mortality and readmission data were unavailable, preventing assessment of long-term prognostic value. Finally, restricting the cohort to hospitalized patients limits generalizability to those discharged from the emergency department or managed in outpatient settings.

In conclusion, among patients hospitalized from the emergency department with ADHF, admission-based laboratory indices were associated with severe clinical deterioration. The log10-transformed NT-proBNP-to-albumin ratio showed a consistent independent association with severe clinical outcomes, while SII provided an additional inflammatory risk signal. However, both indices demonstrated modest discriminatory performance and should not be used as stand-alone risk assessment tools. Their potential role as complementary markers in early clinical risk assessment warrants further prospective validation.

Declarations

Conflict of Interest:

The authors declare no conflict of interest.

Ethical Approval:

Ethical approval was obtained from the Muğla Sıtkı Koçman University Medical Ethics Committee.

Informed Consent:

Due to the retrospective design of the study, the requirement for informed consent was waived.

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Conflict of Interest

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Author Contribution

MGK conceived and designed the study, collected the data, and contributed to data interpretation and manuscript drafting. MK contributed to the study design, interpretation of the findings, critical revision of the manuscript for important intellectual content, and final approval of the manuscript. Both authors read and approved the final version of the manuscript.

Data Availability:

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Tables

Tables 1-4 are available in the Supplementary Files section.

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Figures

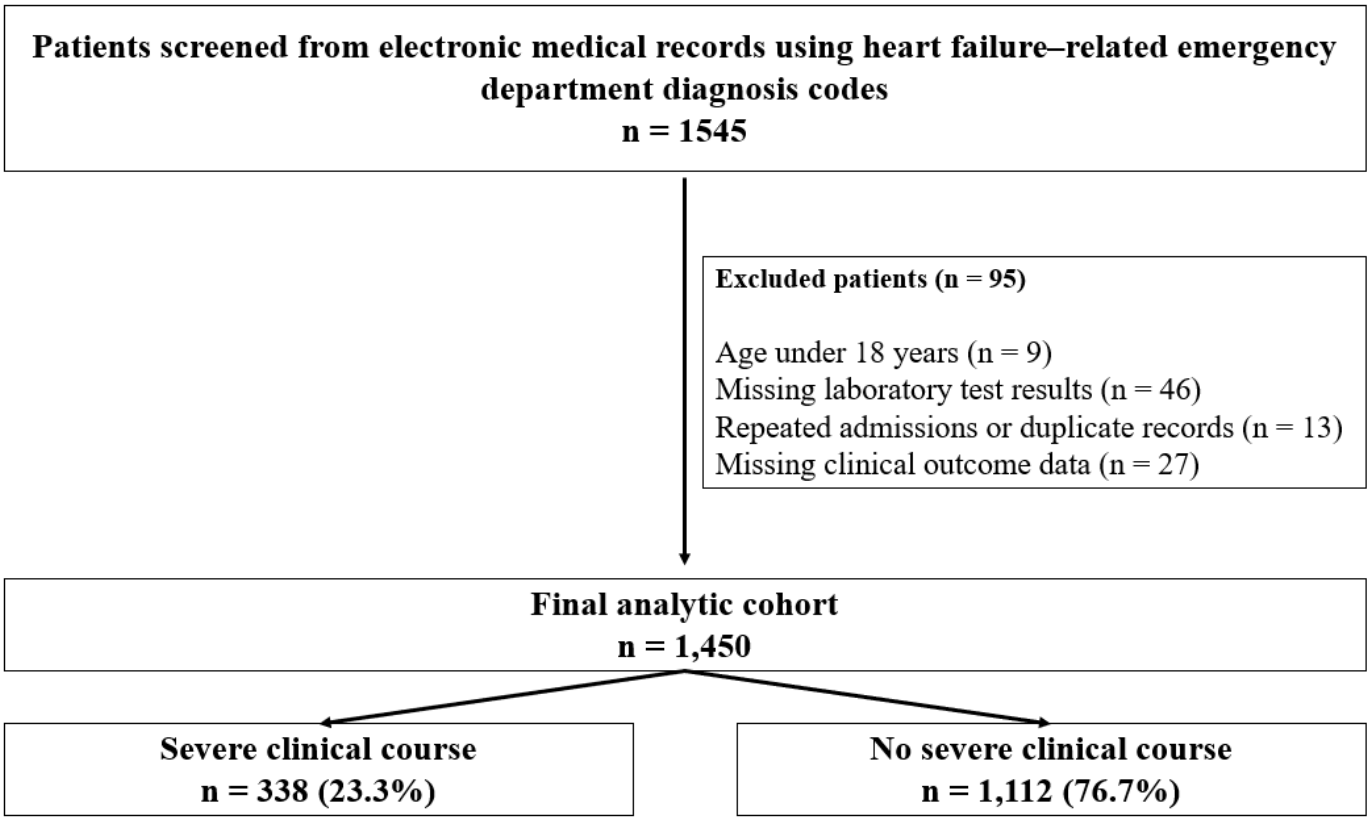


Figure 1

Flow diagram of patient screening, eligibility assessment, and final analytic cohort selection.

Supplementary Files

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- Tables.docx