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Research Article

Prognostic Value of Eosinophil-Derived Hematological Indices in Acute Decompensated Heart Failure

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ABSTRACT

Objective: Eosinophil-derived hematological indices have been proposed as potential prognostic markers for cardiovascular disease; however, their clinical value in acute decompensated heart failure (ADHF) remains unclear. This study aimed to comprehensively evaluate the prognostic significance of eosinophil-based hematological indices, particularly the neutrophil-to-eosinophil ratio (NER), in hospitalized patients with ADHF.

Methods: This single-center, retrospective observational study included 245 consecutive adult patients hospitalized for ADHF between January 2023 and January 2024. Patients with conditions known to influence eosinophil counts, including allergic diseases, recent systemic corticosteroid use, active infection, malignancy, chronic inflammatory disorders, end-stage renal failure, and severe liver disease, were excluded. Hematological indices calculated from blood samples obtained within the first 24 h of admission included NER, eosinophil-to-lymphocyte ratio (ELR), eosinophil-to-monocyte ratio (EMR), eosinophil percentage, neutrophil-to-lymphocyte ratio (NLR), and systemic immune-inflammation index (SII). The primary outcome was all-cause mortality during the one-year follow-up period.

Results: During the follow-up period, 93 (38.0%) patients died. Non-survivors were significantly older and exhibited a higher inflammatory burden, with increased C-reactive protein levels, NLR, and SII values. Eosinophil counts, eosinophil percentages, and EMR values were significantly lower in non-survivors, whereas the NER and ELR did not differ between the groups. Receiver operating characteristic analysis demonstrated poor discriminative performance for eosinophil-derived indices, with area under the curve values of ≤ 0.55. In the multivariate logistic regression analysis, only age was an independent predictor of mortality.

Conclusion: Eosinophil-based hematological indices have limited prognostic value for mortality in patients hospitalized with ADHF. Although eosinophil suppression is associated with adverse outcomes, these indices do not provide independent prognostic information beyond age.

Keywords: Acute decompensated heart failure, Eosinophils, Eosinophil-derived hematological indices, inflammation, mortality, prognosis

INTRODUCTION

Acute decompensated heart failure (ADHF) continues to be one of the leading causes of hospitalization among adults worldwide.[1,2] It is also associated with significant morbidity, prolonged hospitalization, and increased healthcare utilization. Inflammation plays an important role in both the development and progression of heart failure.[3,4]. Numerous hematological indices derived from hemogram parameters have emerged as practical and cost-effective biomarkers for risk stratification in cardiovascular disease and are currently used as auxiliary techniques in clinical practice.[5,6]

Among these markers, eosinophils are valuable because of their role in immune regulation, cytokine response formation, and tissue-level inflammatory responses.[7,8] Although traditionally associated with parasitic infections and allergic diseases, eosinophils also participate in cardiovascular inflammation and stress responses through mechanisms involving interleukin-5 (IL-5), IL-4, and IL-13.[3,9] Importantly, eosinophil levels may

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be significantly affected by allergic disorders such as asthma, atopic dermatitis, and allergic rhinitis, which promote type-2 immune activation and eosinophil proliferation.[10,11]

Various eosinophil-derived hematological indices, including the neutrophil-to-eosinophil ratio (NER), eosinophil-to-monocyte ratio (EMR), and eosinophil-to-lymphocyte ratio (ELR), have been investigated as potential prognostic markers for cardiovascular disease.[12-14] Previous studies have suggested that excessive eosinopenia or high ratios may reflect increased physiological stress or systemic inflammation and may potentially be associated with adverse outcomes, such as death or rehospitalization.[15,16] However, the prognostic value of these indices has been inconsistent across studies, and their independent contributions beyond traditional clinical predictors remain uncertain.

Further studies are needed to clarify the prognostic importance of eosinophil-derived indices in heart failure. In this context, the study aimed to investigate the relationship between eosinophil-based hematological indices, specifically the NER, and mortality in patients hospitalized with ADHF.

MATERIAL AND METHODS

This single-center, retrospective, observational study included consecutive adult patients hospitalized with a diagnosis of ADHF between January 2023 and January 2024. This study was approved by the Ankara Bilkent City Hospital Medical Research Scientific and Ethical Evaluation Board (decision number: TABED 2-26-1823, date: 07.01.2026). The diagnosis of ADHF was established according to the current European Society of Cardiology guidelines based on compatible clinical symptoms, physical examination findings, laboratory results, and echocardiographic assessments.[17] Only the first hospitalization of each patient during the study period was considered for the analysis. A one-year period after hospital admission was used as the basis for mortality assessment.

Patients aged 18 years or older who had a complete blood count obtained within the first 24 h of hospitalization were considered eligible for the study. To minimize factors affecting eosinophil levels, patients with active infection at admission; hematological malignancies or active solid tumors; chronic inflammatory or autoimmune diseases; allergic diseases known to alter eosinophil counts, including asthma, atopic dermatitis, and allergic rhinitis; systemic corticosteroid use within the last 30 days; end-stage renal failure requiring dialysis; severe liver failure; or missing or incomplete laboratory or clinical data required for analysis were excluded from the study. After applying these criteria, 245 patients were included in the final study.

MAIN POINTS

- Among patients hospitalized with acute decompensated heart failure, eosinophil counts and some eosinophil-derived indices were lower in non-survivors; however, their overall prognostic performance was poor.
- Classical inflammatory markers such as C-reactive protein, neutrophil-to-lymphocyte ratio, and systemic immune-inflammation index showed stronger univariate associations with mortality; however, none remained independent predictors after multivariable adjustment.
- Age emerged as the only independent determinant of all-cause mortality, indicating that eosinophil-based hematological indices provide limited incremental prognostic value beyond established clinical predictors.

Demographic characteristics, comorbidities, clinical findings, medication history, laboratory parameters, echocardiographic measurements, and follow-up outcomes were obtained from electronic medical records. Cardiac function was assessed using transthoracic echocardiography, and the left ventricular ejection fraction (LVEF) was recorded.

Blood parameters were used to calculate the following ratios: neutrophil-to-lymphocyte ratio (NLR) = neutrophil/lymphocyte, NER = neutrophil/eosinophil, ELR = eosinophil/lymphocyte, EMR = eosinophil/monocyte, eosinophil percentage (EOS percent) = (eosinophil/white blood cell) × 100, and systemic immune-inflammation index (SII) = (platelet × neutrophil)/lymphocyte.[18] All hematological indices were calculated using the earliest laboratory values obtained within the first 24 hours of hospitalization. NER could not be calculated in 38 patients because the eosinophil count, used as the denominator, was zero. These patients were excluded only from NER-related analyses; other hematological indices were calculated when the required parameters were available.

The primary outcome of the study was all-cause mortality, defined as death from any cause during the follow-up. Mortality status was available for all patients included in the study. Mortality data were obtained from hospital records and national death registry data, and no patient was lost to follow-up for the primary endpoint. Secondary descriptive outcomes included hematological and inflammatory differences between survivors and non-survivors.

Statistical Analysis

Statistical analyses were performed using SPSS (version 26). Continuous variables were evaluated for normality using the Kolmogorov-Smirnov test and presented as medians and interquartile ranges. Categorical variables are expressed as numbers and percentages. Differences between survivors and non-survivors were compared using the Mann-Whitney U test for continuous variables or the chi-square test for categorical variables.

The diagnostic performance of hematological indices in predicting mortality was evaluated using receiver operating characteristic (ROC) curve analysis, and the area under the curve (AUC) was calculated (Figure 1). Optimal cut-off values were determined using the Youden index. Variables deemed clinically relevant or demonstrating significance in the univariate analysis were included in a multivariate logistic regression model to identify independent determinants of all-cause mortality. Because exact time-to-event data were not consistently available, all-cause mortality was analyzed as a binary outcome, and multivariable logistic regression was used instead of Cox proportional hazards regression. In addition,

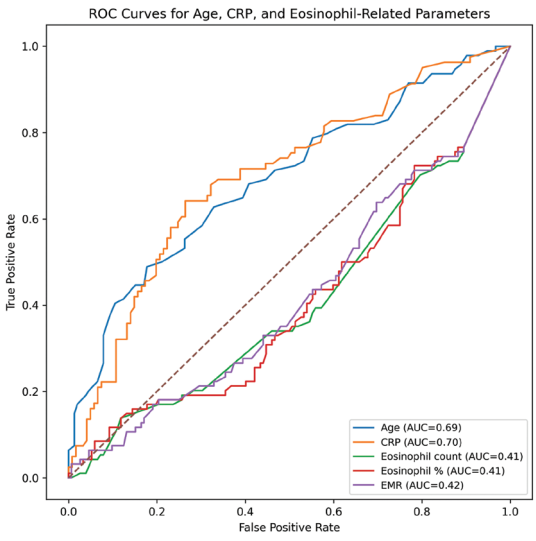


Figure 1. ROC curves for age, CRP, and Eosinophil-Related Parameters

ROC, receiver operating characteristic; AUC, area under curve; CRP, c-reactive protein; EMR, eosinophil-to-monocyte ratio.

renal function and major comorbidities, including hypertension, chronic obstructive pulmonary disease, and atrial fibrillation, were incorporated into the model when sufficient data were available. Odds ratios with 95 percent confidence intervals are reported. A two-sided *P* value of less than 0.05 was considered statistically significant.

RESULTS

In total, 245 patients were included in this study. In the study population, 152 patients (62.0 percent) survived, while 93 patients (38.0 percent) died during the one-year follow-up. The baseline clinical, laboratory, and inflammatory characteristics of survivors and non-survivors are summarized in Table 1. Patients who died were significantly older than survivors ($P < 0.001$). The mean LVEF was slightly higher in non-survivors than in survivors ($P = 0.009$).

Inflammatory burden differed significantly between the groups. C-reactive protein (CRP) levels were markedly higher in non-survivors than in survivors ($P < 0.001$). Lymphocyte counts were significantly lower in the mortality group ($P < 0.001$), whereas neutrophil counts were similar between groups. Eosinophil counts ($P = 0.016$) and eosinophil percentages ($P = 0.015$) were significantly lower in patients who died. The EMR was also reduced in non-survivors ($P = 0.029$). In contrast, the NER did not differ significantly between survivors and non-survivors ($P = 0.28$). Similarly, the ELR did not differ significantly between groups ($P = 0.15$).

Classical inflammation-based markers were more strongly associated with mortality than eosinophil-derived markers. The NLR was significantly higher in the mortality group ($P = 0.0015$). The SII was also higher among the non-survivors ($P = 0.013$).

Variables with clinical significance in the univariate analysis were included in the multivariable logistic regression model: age, LVEF, CRP, NLR, NER, eosinophil percentage, renal function,

Table 1. Baseline Demographic, Clinical, and Laboratory Characteristics of Survivors and Non-Survivors

Variable	Survivors (n=152)	Non-survivors (n=93)	*P* value
Age (years)	58 (48.8-67)	67 (56-78)	**< 0.001**
Male sex, n (%)	96 (62.7)	60 (64.5)	0.78
LVEF (%)	25 (20-30)	28 (21-35)	**0.009**
CRP (mg/L)	3.85 (0.58-19.0)	20.9 (3.2-65.0)	**< 0.001**
WBC (10^3/μL)	8.30 (6.65-10.3)	8.50 (6.6-11.5)	0.69
Neutrophils (10^3/μL)	5.45 (4.1-7.3)	5.50 (3.8-7.5)	0.31
Lymphocytes (10^3/μL)	1.90 (1.3-2.6)	1.40 (0.90-2.20)	**< 0.001**
Eosinophils (10^3/μL)	0.165 (0.10-0.26)	0.10 (0.04-0.20)	**0.016**
NLR	2.86 (1.91-4.53)	4.22 (2.40-6.46)	**0.001**
NER	28.4 (15-60)	36.9 (15-89)	0.28
EMR	0.20 (0.11-0.35)	0.17 (0.07-0.29)	**0.029**
Eosinophil %	1.94 (1.09-3.04)	1.23 (0.55-2.38)	**0.015**
SII	619 (392-1081)	874 (493-1399)	**0.013**
DM, n(%)	56 (36.8)	35 (37.2)	1.000
HT, n(%)	86 (56.6)	70 (74.5)	**0.007**
HL, n(%)	33 (21.7)	29 (30.9)	0.146
CAD, n(%)	71 (46.7)	53 (56.4)	0.179
COPD, n(%)	26 (17.1)	29 (30.9)	**0.018**
AF, n(%)	35 (23.0)	42 (44.7)	**0.001**

LVEF, left ventricular ejection fraction; CRP, C-reactive protein; WBC, white blood count; NLR, neutrophil-to-lymphocyte ratio; NER, neutrophil-to-eosinophil Ratio, EMR, eosinophil-to-monocyte ratio; SII, systemic immune-inflammation index; DM, diabetes mellitus; HT, hypertension, HL, hyperlipidemia; CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease; AF: atrial fibrillation.

hypertension, chronic obstructive pulmonary disease (COPD), and atrial fibrillation (Table 2). In this model, only age remained an independent predictor of all-cause mortality. Other variables, including LVEF, CRP, NLR, NER, eosinophil percentage, renal function, hypertension, COPD, and atrial fibrillation, were not independently associated with mortality. SII was evaluated in the ROC analysis and was presented in Table 3, but it was not included in the final multivariable logistic regression model.

The discriminative performance of the hematological indices in predicting all-cause mortality is summarized in Table 3. Overall, age and CRP levels exhibited the highest discriminative ability, whereas eosinophil-derived indices showed poor predictive performance, with AUC values approximately 0.55 or lower.

DISCUSSION

In our study, we evaluated the prognostic value of eosinophil-derived hematological indices, particularly the NER, for predicting all-cause mortality among patients hospitalized for heart failure. Non-survivors had significantly lower eosinophil counts, eosinophil percentages, and EMR values than survivors. Classical inflammatory markers such as CRP, NLR, and SII were more strongly associated with mortality. The NER did not differ significantly between survivors and non-survivors, and was a poor predictor in the ROC analysis. In the multivariate regression analysis, only age remained an independent predictor of all-cause mortality, whereas eosinophil-based indices failed to maintain statistical significance. By systematically assessing all commonly used eosinophil-derived hematological indices, this study provides a comprehensive overview of their prognostic relevance in ADHF.

Table 2. Multivariable Logistic Regression for All-Cause Mortality

Variable	OR	95% CI	*P* value
Age (per 1 year increase)	1.05	1.01-1.09	**0.007**
LVEF (%)	1.00	0.95-1.05	0.98
Creatinine	1.18	0.87-1.60	0.28
Hypertension	1.42	0.74-2.73	0.29
COPD	1.51	0.75-3.06	0.24
Atrial fibrillation	1.93	0.94-3.98	0.071
CRP	1.10	0.90-1.35	0.31
NLR	1.02	0.91-1.14	0.68
NER	1.00	0.98-1.02	0.89
Eosinophil %	1.04	0.80-1.36	0.77

LVEF, left ventricular ejection fraction; COPD, chronic obstructive pulmonary disease; CRP, C-reactive protein; NLR, neutrophil-to-lymphocyte ratio; NER, neutrophil-to-eosinophil ratio; OR, odds ratio; CI, confidence interval.

Table 3. ROC Curve Analysis for Predicting All-Cause Mortality

Variables	AUC	95% CI	Optimal cut-off values
Age	0.686	0.619-0.761	≥ 69 years
CRP	0.683	0.623-0.771	≥ 10.7 mg/L
NLR	0.621	0.550-0.696	≥ 4.06
SII	0.595	0.514-0.661	≥ 749.9
EF	0.599	0.525-0.669	≥ 25%
NER	0.545	0.469-0.633	≥ 23.48
Eosinophil count	0.409	0.334-0.476	≤ 0.13
Eosinophil %	0.407	0.332-0.478	≤ 2.40
EMR	0.417	0.344-0.490	≤ 0.176

CI, confidence interval; AUC, area under curve; CRP, c-reactive protein; NLR, neutrophil-to-lymphocyte ratio; SII, systemic immune-inflammation index; LVEF, left ventricular ejection fraction; NER, neutrophil-to-eosinophil ratio, EMR, eosinophil-to-monocyte ratio; ROC, receiver operating characteristic.

In our study, the LVEF was slightly higher in non-survivors than in survivors. This unexpected finding may be related to older age and a higher comorbidity burden in these patients, particularly atrial fibrillation, hypertension, and chronic obstructive pulmonary disease. Therefore, mortality may have been influenced more by age, rhythm disorders, and systemic comorbidities than by systolic dysfunction alone.

These findings partially overlap with previous studies demonstrating an association between eosinophil-related indices and adverse outcomes in heart failure.[12,19] Some reports have suggested that high NER or low eosinophil counts may be linked to an increased risk of death or major adverse cardiovascular events, possibly reflecting heightened systemic stress responses and dysregulated inflammation.[20,21] However, the prognostic power of eosinophil-derived indices has varied significantly between studies, and many did not adjust for important clinical confounders, particularly age.[20,22] Our results show that although eosinophil parameters differed between survivors and non-survivors in univariate analysis, their predictive value diminished after adjustment, suggesting that eosinophil dynamics may reflect overall disease severity rather than provide incremental prognostic information.

The biological rationale for eosinopenia in critically ill or decompensated patients is well recognized.[23,24] Acute physiological stress, adrenergic activation, and elevated endogenous corticosteroids suppress eosinophil function and accelerate apoptosis, leading to low circulating eosinophil levels.[25,26] Similarly, allergic diseases, type-2 immune activation, and exogenous corticosteroid use may significantly alter eosinophil kinetics. These mechanisms highlight the complexity of interpreting eosinophil-related indices in heterogeneous clinical populations such as patients with heart failure.[10,27] In our study, eosinophil counts and EMR were significantly lower in non-survivors; however, these markers did not provide prognostic information beyond age and broader inflammatory markers. This reinforces the concept that eosinophil suppression may be a non-specific indicator of physiological stress, rather than a heart failure-specific predictor.[28]

In contrast, CRP, NLR, and SII were moderately associated with mortality consistent with substantial evidence linking systemic inflammation and adverse outcomes in heart failure. However, these indices did not remain independent predictors after accounting for age and LVEF. This underscores the dominant prognostic role of age, which has consistently emerged as a strong predictor of mortality in heart failure cohorts. The lack of independent prognostic value for NER and related indices in our

study suggests that their utility may be limited in populations with wide age variability or in settings where stronger clinical predictors overshadow the inflammatory indices.

Study Limitations

This study had several strengths, including detailed hematological profiling and comprehensive adjustment for multiple inflammatory variables. However, this study has some limitations. This retrospective single-center design may limit the generalizability of our results. Eosinophil counts may be affected by unmeasured confounding factors, including subclinical infections, undocumented allergic conditions, or undocumented corticosteroid exposure. Although our dataset included a substantial number of patients, certain eosinophil-derived indices (particularly NER) could not be calculated for patients with zero eosinophil counts, reducing statistical power. Another limitation was that exact time-to-event data were not uniformly available; therefore, Cox proportional hazards regression could not be performed, and mortality was analyzed as a binary outcome at one-year. Because of the retrospective design, BNP/NT-proBNP levels and admission blood pressure values were not uniformly available; therefore, they could not be included in the multivariable model. We did not perform subgroup analyses by heart failure phenotype (e.g., HFrEF vs. HFpEF), which may have provided additional insights. Finally, we did not include long-term cardiovascular outcomes beyond all-cause mortality, which may have provided additional insights.

CONCLUSION

This study evaluated the relationship between eosinophil-based hematological indices and all-cause mortality in patients with acute heart failure and demonstrated that these indices had limited value in predicting all-cause mortality. Although low eosinophil levels were observed in patients who died, ROC analysis and multivariate statistical models revealed that eosinophil-derived indices were not independent prognostic markers. In contrast, age was identified as the strongest and only independent determinant of mortality. These findings indicate that eosinophil-based indices may assist in risk stratification for acute heart failure, but they are not appropriate for stand-alone use in clinical decision-making.

Ethics

Ethics Committee Approval: Ankara Bilkent City Hospital Medical Research Scientific and Ethical Evaluation Board (decision number: TABED 2-26-1823, date: 07.01.2026).

Informed Consent: Retrospective study. Permission was obtained from hospital management to collect study data from the Information Management System.

Footnotes

Author Contributions

Concept Design – B.K. F.K.; Data Collection or Processing – B.K. F.K.; Analysis or Interpretation – B.K. F.K.; Literature Review – B.K. F.K.; Writing, Reviewing and Editing – B.K. F.K.

Declaration of Interests: The authors have no conflict of interest to declare.

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