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Association of Pan-Immune-Inflammation Value with In-Hospital Mortality in Acute Decompensated Heart Failure with Reduced Ejection Fraction

Düşük Ejeksiyon Fraksiyonlu Akut Dekompanse Kalp Yetersizliğinde Pan-Immün-İnflamasyon Değerinin Hastane İçi Mortalitedeki Rolü

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ABSTRACT

Aim: Systemic inflammation contributes to the pathogenesis and prognosis of heart failure. The pan-immune-inflammation value (PIV) is a novel biomarker reflecting the balance between pro- and anti-inflammatory immune components. This study investigated the relationship between PIV and in-hospital mortality in patients with acute decompensated heart failure (ADHF) with reduced ejection fraction (HFrEF).

Materials and Methods: This retrospective study included 346 patients hospitalized with ADHF and HFrEF at a tertiary center between January 2022 and May 2025. Clinical, laboratory, and echocardiographic data were retrieved. PIV was calculated as (neutrophils × platelets × monocytes) / lymphocytes. The primary endpoint was in-hospital mortality. Prognostic performance was evaluated using ROC curve analysis and multivariable logistic regression.

Results: In-hospital mortality occurred in 65 patients (18.8%). The mortality group had lower systolic blood pressure, glomerular filtration rate, sodium, albumin, and LVEF, while CRP, monocyte count, and PIV were higher (all $p < 0.05$). PIV predicted mortality with an AUC of 0.735 (95% CI: 0.667–0.796, $p < 0.001$). A cut-off of 883.07 provided 76.8% sensitivity and 70.4% specificity. In multivariable analysis, PIV independently predicted mortality both as a continuous variable (OR: 1.002, 95% CI: 1.001–1.003, $p < 0.001$) and as a categorical variable above the cut-off (OR: 3.893, 95% CI: 2.057–7.369, $p < 0.001$).

Conclusion: PIV is an independent predictor of in-hospital mortality in patients with ADHF and reduced EF. As a simple, inexpensive, and widely available marker, PIV may facilitate early risk stratification in clinical practice.

Keywords: heart failure, inflammation, pan-immune-inflammation value, in-hospital mortality.

ÖZ

Amaç: Sistemik inflamasyon, kalp yetersizliğinin patogenezinde ve prognozunda rol oynamaktadır. Pan-immün-inflamasyon değeri (PIV), bağışıklık sistemindeki proinflamatuvar ve antiinflamatuvar bileşenleri yansıtan yeni bir biyobelirteçtir. Bu çalışmada, ejeksiyon fraksiyonu düşük akut dekompanse kalp yetersizliği (ADKY) hastalarında PIV ile hastane içi mortalite arasındaki ilişki araştırıldı.

Gereç ve Yöntem: Bu retrospektif çalışmaya Ocak 2022–Mayıs 2025 arasında üçüncü basamak bir merkezde ADKY ve düşük ejeksiyon fraksiyonu (EF) (≤%40) tanısıyla yatırılan 346 hasta dahil edildi. Klinik, laboratuvar ve ekokardiyografik veriler kaydedildi. PIV; (nötrofil × trombosit × monosit) / lenfosit formülüyle hesaplandı. Birincil sonlanım noktası hastane içi mortaliteydi. ROC eğrisi analizi ve çok değişkenli lojistik regresyon kullanıldı.

Bulgular: Hastane içi mortalite 65 hastada (%18,8) izlendi. Mortalite grubunda sistolik kan basıncı, glomerüler filtrasyon hızı, sodyum, albümin ve EF daha düşük; CRP, monosit sayısı ve PIV ise daha yüksek bulundu (tümü için $p < 0,05$). PIV, ROC analizinde mortaliteyi AUC 0,735 (GA: 0,667–0,796, $p < 0,001$) ile öngördü. 883,07 kesim değerinde %76,8 duyarlılık ve %70,4 özgüllük saptandı. Çok değişkenli analizde PIV, hem sürekli (OR: 1,002; GA: 1,001–1,003; $p < 0,001$) hem de kategorik değişken olarak (OR: 3.893, 95% CI: 2.057–7.369; $p < 0,001$) bağımsız prediktör olarak bulundu.

Sonuç: PIV, düşük EF'li ADKY hastalarında hastane içi mortalitenin bağımsız belirtecidir. Rutin hemogramdan kolay hesaplanabilen, ucuz ve uygulanabilir bir parametre olarak PIV, klinik uygulamada erken risk sınıflamasına katkı sağlayabilir.

Anahtar Kelimeler: kalp yetersizliği, inflamasyon, pan-immün-inflamasyon değeri, hastane içi mortalite.

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Introduction

Heart failure (HF) remains a global public health crisis, affecting over 23 million individuals and accounting for high morbidity, mortality, and economic burden [1]. Acute decompensated heart failure (ADHF) represents a particularly severe stage of the disease and is associated with short-term mortality rates of 4% to 10% and high early readmission rates despite contemporary management strategies [2, 3]. Accurate and early risk stratification at admission is therefore critical for guiding treatment decisions, optimizing resource allocation, and improving outcomes.

The pathophysiology of ADHF involves a complex interplay between neurohormonal activation, oxidative stress, endothelial dysfunction, and systemic inflammation [4]. Pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) have been shown to exert direct negative inotropic effects and contribute to adverse cardiac remodeling [5]. Clinical and experimental studies have demonstrated that inflammation-related markers, including C-reactive protein (CRP), neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and systemic immune-inflammation index (SII), are associated with adverse outcomes in HF [6-9]. However, these indices provide only a partial assessment of the complex immune-inflammatory response.

The pan-immune-inflammation value (PIV)—calculated as (neutrophil count × platelet count × monocyte count) / lymphocyte count—was first proposed in oncology research, where it demonstrated superior prognostic accuracy compared with traditional inflammatory markers [10]. In cardiovascular diseases, PIV has emerged as a promising biomarker in several contexts. Studies in ST-segment elevation myocardial infarction (STEMI) have shown that higher PIV is associated with increased risk of major adverse cardiovascular events after primary percutaneous coronary intervention [11]. Similarly, in non–ST-segment elevation myocardial infarction (NSTEMI), elevated PIV has been linked to higher angiographic complexity and worse clinical outcomes [12]. In patients with coronary artery disease (CAD) undergoing percutaneous coronary intervention, higher PIV has been associated with more severe coronary atherosclerosis and adverse prognoses [13].

In the context of HF, recent studies have shown that elevated admission PIV independently predicts short-term and long-term mortality in ADHF. Furthermore, PIV appears to outperform traditional inflammatory markers such as NLR, PLR, and CRP in prognostic performance [14, 15]. Additional evidence from patients with HF with reduced ejection fraction (HFrEF) also supports the prognostic value of PIV [16]. Given that ADHF represents a state of heightened systemic inflammation, PIV—a low-cost, readily available, and comprehensive biomarker—may provide incremental value for early risk stratification.

Therefore, this study aimed to investigate the association between admission PIV and in-hospital mortality in patients hospitalized with ADHF, building upon prior evidence from both HF and other cardiovascular disease populations.

Methods

This retrospective, observational study was conducted at Karaman Training and Research Hospital between January 2022 and May 2025. Consecutive patients who were admitted with a diagnosis of ADHF through the emergency department or cardiology outpatient clinics and subsequently hospitalized were evaluated for inclusion. The study was designed to investigate the prognostic value of the pan-immune-inflammation value (PIV) in predicting in-hospital mortality among patients with ADHF. For this purpose, patients were categorized into two groups according to in-hospital outcomes: those who died during hospitalization (mortality group) and those who survived to discharge (survival group).

Only patients with HFrEF, defined as a left ventricular ejection fraction (LVEF) <40% according to the current European Society of Cardiology (ESC) guidelines [2], were included. Patients with active malignancy and pneumonia, acute or chronic inflammatory diseases, severe hepatic dysfunction, end-stage renal disease requiring dialysis, autoimmune or hematological disorders, or those with incomplete laboratory or

echocardiographic data were excluded. Patients with acute coronary syndrome at admission or who had undergone recent cardiac surgery or invasive procedures within the last three months were also excluded (Figure 1). The study protocol was reviewed and approved by the Ethics Committee of Karamanoğlu Mehmetbey University, Faculty of Medicine (Approval date: 26/06/2025, Approval number: 22–2025/11). All procedures were conducted in accordance with the ethical principles stated in the Declaration of Helsinki.

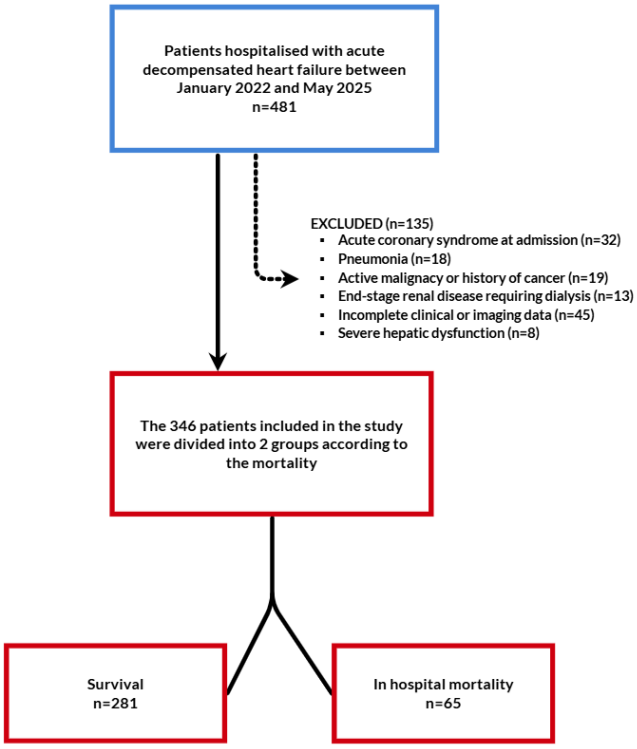

Figure 1. Flow chart of the study

Data Collection:

Demographic data (age and sex), cardiovascular risk factors (hypertension, diabetes mellitus, hyperlipidemia, smoking status), and comorbid conditions (atrial fibrillation, coronary artery disease, chronic kidney disease, chronic obstructive pulmonary disease) were obtained from the hospital electronic records.

Venous blood samples collected at the time of admission were retrospectively retrieved from the institutional laboratory database. Standard laboratory measurements included complete blood count, renal function tests, liver enzymes, electrolytes, lipid profile, and inflammatory markers such as C-reactive protein (CRP, mg/dL). Hematological indices, including neutrophil, lymphocyte, monocyte, and platelet counts, were determined using an automated hematology analyzer (Mindray BC-6000, Mindray Bio-Medical Electronics Co., Shenzhen, China). Serum chemistry analyses, including creatinine and other biochemical parameters, were performed with a Beckman Coulter AU5800 modular chemistry analyzer (Beckman Coulter Inc., Brea, CA, USA).

The pan-immune-inflammation value (PIV) was calculated using the following formula: PIV = (Neutrophil count (10^3/μL) × Platelet count (10^3/μL) × Monocyte count (10^3/μL)) / Lymphocyte count (10^3/μL).

All patients underwent transthoracic echocardiography within 24 hours of hospital admission using commercially available ultrasound systems. Standard measurements were obtained according to current guidelines, and LVEF was calculated using the biplane Simpson's method in the apical 4-chamber and 2-chamber view. Comprehensive two-dimensional, M-mode, and Doppler studies were carried out with a Philips iE33 xMatrix echocardiography system (Philips Healthcare, Andover, MA, USA) using 2.5- and 3.5-MHz transducers. All echocardiographic assessments were conducted by experienced cardiologists using standardized imaging protocols, and measurements were performed objectively to minimize inter-observer variability and measurement bias.

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics version 25.0 (IBM Corporation, Armonk, NY, USA). Since the sample size exceeded 50 participants, data distribution was evaluated by examining skewness and kurtosis values, with values between –2 and +2 considered indicative of normality [17]. Categorical variables were expressed as numbers and percentages, while continuous variables were summarized as mean ± standard deviation (SD) for normally distributed data or as median with interquartile range (IQR, 25th–75th percentile) for non-normally distributed data. Comparisons between survivor and non-survivor groups were conducted using

the independent samples t-test or the Mann–Whitney U test, depending on the underlying data distribution. Categorical variables were compared using Pearson's chi-square test (Fisher's exact tests where appropriate). Receiver operating characteristic (ROC) curve analysis was performed to assess the predictive ability of the pan-immune-inflammation value (PIV) for in-hospital mortality. The optimal cut-off value was determined using Youden's index, which incorporates both sensitivity and specificity. Univariate logistic regression analysis was first performed to identify variables associated with in-hospital mortality. Variables found to be statistically significant in univariate analysis, along with age as a clinically relevant factor, were entered into the multivariable logistic regression model using the enter method. Serum sodium was excluded from the multivariable model due to collinearity with renal function parameters, which could confound its independent effect on mortality. The independent prognostic value of PIV was assessed in two separate models: model 1 as a continuous variable and model 2 as a categorical variable based on the cut-off point determined by the Youden index. For all statistical tests, a two-sided p value <0.05 was considered statistically significant.

Results

A total of 346 patients hospitalized with ADHF were included in the study, and in-hospital mortality was observed in 65 patients (18.8%), while 281 patients (81.2%) survived to discharge. Patients in the mortality group tended to be older and more frequently had hypertension compared with survivors; however, these differences did not reach statistical significance (age: 70.8 ± 10.9 vs. 67.8 ± 12.1 years, p = 0.073; hypertension: 60% vs. 47.3%, p = 0.066). Other comorbidities, including diabetes mellitus, atrial fibrillation, coronary artery disease, and chronic kidney disease, were similarly distributed between the groups. With respect to guideline-directed medical therapy, the prescription rates of ACE inhibitors/ARBs, angiotensin receptor–neprilysin inhibitors (ARNI), beta-blockers, and spironolactone were comparable between the survivor and mortality groups (all p > 0.05).

Patients who experienced in-hospital mortality presented with significantly lower systolic blood pressure (107.9 ± 22.6 vs. 116.6 ± 23.4 mmHg, p = 0.007), reduced glomerular filtration rate (51.8 ± 13.9 vs. 57.0 ± 15.9 mL/min/1.73 m², p = 0.015), lower serum sodium (133 vs. 136 mmol/L, p = 0.031), and decreased albumin levels (3.38 ± 0.56 vs. 3.56 ± 0.56 g/dL, p = 0.021). Inflammatory activity was more pronounced in the mortality group, with higher CRP concentrations (4.77 [2.3–10] vs. 3.05 [0.9–7] mg/L, p = 0.002) and elevated monocyte counts (0.71 ± 0.18 vs. 0.65 ± 0.19 ×10³/μL, p = 0.031). Importantly, the pan-immune-inflammation value (PIV) was markedly higher among patients who experienced mortality compared to survivors (1151.3 [770.2–2042.8] vs. 718.1 [452.8–1150.7], p < 0.001). Left ventricular ejection fraction (LVEF) was also significantly lower in the mortality group (29.9 ± 5.6% vs. 31.8 ± 6.8%, p = 0.040).

Receiver operating characteristic (ROC) curve analysis demonstrated that PIV had a significant discriminatory capacity for predicting in-hospital mortality, with an AUC of 0.735 (95% CI: 0.667–0.796, p < 0.001) (Figure 2). Based on Youden's index, the optimal cut-off value was determined to be 883.07, yielding 76.8% sensitivity and 70.4% specificity. When stratified according to this threshold, patients with PIV >883.07 experienced significantly higher mortality rates compared with those below the cut-off (p < 0.001). The distribution of PIV values between groups are illustrated in Figure 3.

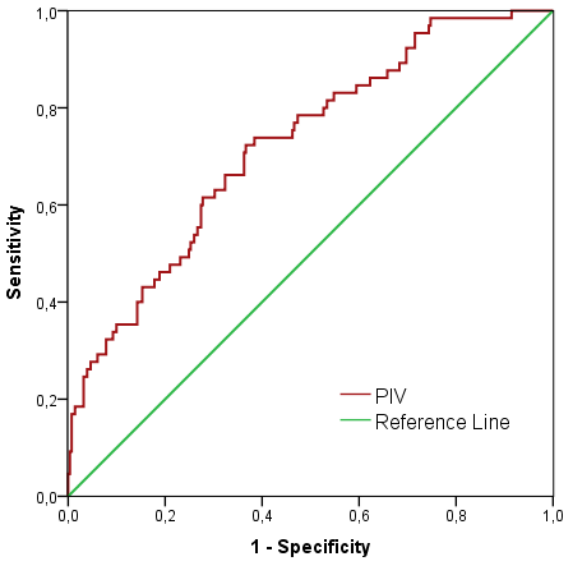

Figure 2. The ROC curve of Pan-immune-inflammation value (PIV)

Table 1. Demographic, clinical characteristics and laboratory measurements of the patients according to the groups survivals and mortality

	Overall (n=346)	Survival (n=281)	Mortality (n=65)	p-value
Age (years)	68.4±11.9	67.8±12.1	70.8±10.9	0.073[A]
Sex, (Male)	193 (55.7%)	156 (55.5%)	37 (56.9%)	0.837[B]
BMI (kg/m2)	24.5±2.2	24.4±2.2	24.6±2.1	0.250[A]
NYHA class, n(%)				
II	100 (28.9%)	90 (32%)	10 (15.3%)	0.001[B]
III	151 (43.6%)	125 (44.4%)	26 (40%)	
IV	95 (27.4%)	66 (23.4%)	29 (44.6%)	
Hypertension, n(%)	172 (49.7%)	133 (47.3%)	39 (60%)	0.066[B]
Diabetes mellitus, n(%)	155 (44.7%)	131 (46.6%)	24 (36.9%)	0.157[B]
Hyperlipidemia, n(%)	45 (13%)	32 (11.3%)	13 (20%)	0.098[B]
Smoking, n(%)	87 (25.1%)	70 (24.9%)	17 (26.1%)	0.835[B]
CAD, n(%)	195 (56.3%)	157 (55.8%)	38 (58.4%)	0.704[B]
COPD, n(%)	53 (15.3%)	42 (14.9%)	11 (16.9%)	0.836[B]
Atrial fibrillation, n(%)	162 (46.8%)	130 (46.2%)	32 (49.2%)	0.666[B]
Beta-blocker, n(%)	245 (70.8%)	201 (71.5%)	44 (67.6%)	0.540[B]
ACE/ARB, n(%)	131 (37.8%)	108 (38.4%)	23 (35.3%)	0.648[B]
Spironolactone, n(%)	114 (32.9%)	94 (33.4%)	20 (30.7%)	0.678[B]
ARNI, n(%)	46 (13.2%)	34 (12%)	12 (18.4%)	0.173[B]
SGLT-2 inhibitor, n(%)	176 (50.8%)	148 (52.6%)	28 (43%)	0.163[B]
Furosemide, n(%)	273 (78.9%)	218 (77.5%)	55 (84.6%)	0.210[B]
Heart rate, BPM	92.7±25.4	92.2±25.6	95.1±24.4	0.408[A]
Sistolic BP, (mmHg)	115±23.5	116.6±23.4	107.9±22.6	0.007[A]
Diastolic BP, (mmHg)	67.8±11.7	68.2±11.1	65.8±11.1	0.109[A]
Hemoglobin, (g/dL)	11.9±2	12±2	11.5±1.9	0.097[A]
Glucose, (mg/dL)	129.5 (101 – 182.5)	130 (102 – 182)	128 (99.5 – 192)	0.806[C]
GFR, (ml/dk/1.73 m2)	56.1±15.7	57±15.9	51.8±13.9	0.015[A]
Sodium, (mmol/l)	136 (130 – 139)	136 (131 – 139)	133 (128.5 – 138)	0.031[C]
Potassium, (mmol/l)	4.4 (4.03 – 4.86)	4.39 (4.03 – 4.84)	4.5 (3.96 – 5.07)	0.380[C]
Total cholesterol, (mg/dL)	144.7±41.3	143±41.8	152.2±38.3	0.105[A]
CRP, (mg/l)	3.2 (1.07 – 7.55)	3.05 (0.9 – 7)	4.77 (2.3 – 10)	0.002[C]
Albumin, (g/dL)	3.52±0.56	3.56±0.56	3.38±0.56	0.021[A]
WBC, (103/µL)	10.87 (8.57 – 13.74)	10.61 (8.44 – 13.23)	11.28 (9.56 – 14.43)	0.072[C]
Platelet, (103/µL)	231.7±78.7	228.2±77.8	246.9±81.1	0.095[A]
Neutrophile, (103/µL)	7.18 (5.62 – 9.5)	7 (5.49 – 9.21)	8.3 (5.72 – 10.65)	0.091[C]
Lymphocyte, (103/µL)	1.38±0.62	1.4±0.58	1.27±0.76	0.116[A]
Monocyte, (103/µL)	0.66±0.19	0.65±0.19	0.71±0.18	0.031[A]
PIV*	792.9 (510.3 – 1264)	718.1 (452.8 – 1150.7)	1151.3 (770.2 – 2042.8)	<0.001[C]
PIV** (>883.07), n(%)	149 (43%)	102 (36.2%)	47 (72.3%)	<0.001[B]
LVEF,(%)	31.4±6.66	31.8±6.83	29.9±5.6	0.04[A]
LA diameter, mm	41.29±5.51	41±5.7	42.2±4.5	0.113[A]
End-diastolic diameter, mm	54.32±6.6	54±6.65	55.3±6.34	0.153[A]
End-sistolic diameter,mm	42 (39 – 46.25)	42 (39 – 46)	43 (39 – 49)	0.299[C]

ACE: angiotensin converting enzyme, ARB: angiotensin receptor blocker, ARNI: angiotensin receptor-neprilysin inhibitor, BMI: body mass index, BP: Blood pressure, BPM: beats per minute, CAD: coronary artery disease, COPD: chronic obstructive pulmonary disease, CRP: C-reactive protein, GFR: glomerular filtration rate, LA: left atrium, LVEF: left ventricular ejection fraction, NYHA: New york heart association, PIV: pan-immune-inflammation value, SGLT: sodium glucose cotransporter, WBC: White blood cell. Continuous variables were shown as mean ± SD or median (25th – 75th) percentiles; where appropriate. A Student's t test, B Pearson's χ^2 test, C Mann Whitney U test.

Table 2: Independent predictors of mortality by logistic regression analysis

Variables	Univariate Analysis		Multivariate Analysis–Model 1*		Multivariate Analysis–Model 2**	
	OR (95%CI)	p-Value	OR (95%CI)	p-Value	OR (95%CI)	p-Value
Age	1.022 (0.998–1.046)	0.074	1.016 (0.989–1.044)	0.249	1.014 (0.987–1.042)	0.301
Sistolic BP	0.982 (0.969–0.995)	0.007	0.988 (0.973–1.004)	0.133	0.986 (0.971–1.001)	0.067
GFR	0.978 (0.961–0.996)	0.016	0.980 (0.961–0.999)	0.049	0.979 (0.961–0.998)	0.032
CRP	1.037 (1.001–1.076)	0.047	1.023 (0.979–1.069)	0.306	1.021 (0.980–1.064)	0.317
Albumin	0.577 (0.362–0.918)	0.02	0.973 (0.583–1.712)	0.924	0.886 (0.520–1,512)	0.658
Monocyte	1.756 (1.326–2.256)	0.037	0.877 (0.316–3.216)	0.875	1.636 (0.311–4.601)	0.561
*PIV	1.002 (1.001–1.004)	<0.001	1.002 (1.001–1.003)	<0.001	–	–
**PIV >883.07	4.582 (2.527–8.309)	<0.001	–	–	3.893 (2.057–7.369)	<0.001
LVEF (%)	0.959 (0.921–0.998)	0.041	0.966 (0.922–1.011)	0.138	0.970 (0.926–1.015)	0.184

BP: Blood pressure, CI: Confidence interval, CRP: C–reactive protein, GFR: Glomerular filtration rate, LVEF: Left ventricle ejection fraction, OR: Odds ratio, PIV: pan-immune-inflammation value. *PIV presented as a continuous variable (R2: 0.284), **PIV presented as a categorical variable (R2: 0.271).

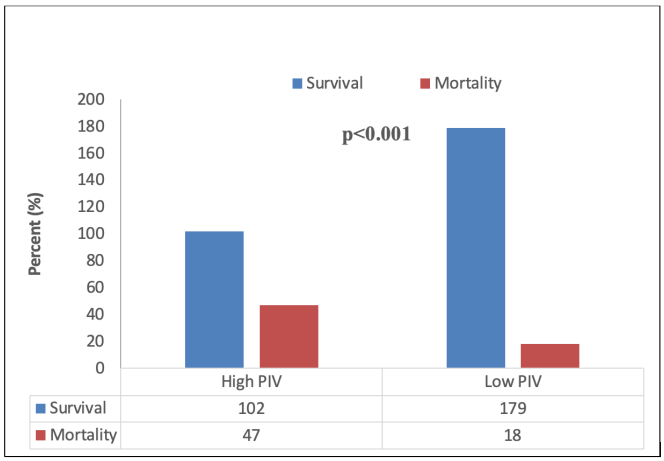

Figure 3. The distribution low and high (based on cut-off point 883.07) PIV in survival and mortality groups. (Pearson's χ^2 test).

In univariate logistic regression analyses, lower systolic blood pressure, reduced GFR, lower albumin, higher CRP, elevated monocyte count, reduced LVEF, and increased PIV were all significantly associated with in-hospital mortality.

In the multivariable models, PIV remained an independent predictor of in hospital mortality. When assessed as a continuous variable (Model 1), higher PIV was independently associated with increased mortality risk (OR: 1.002, 95% CI: 1.001–1.003, p < 0.001). When analyzed as a categorical variable according to the 883.07 cut-off (Model 2), patients with elevated PIV demonstrated a more than fourfold increased risk of in-hospital mortality (OR: 3.893, 95% CI: 2.057–7.369, p < 0.001). Impaired renal function also emerged as an independent predictor in the final models (Model 1: GFR: OR: 0.980, 95% CI: 0.961–0.999, p = 0.049, Model 2: GFR: OR: 0.979, 95% CI: 0.961–0.998, p = 0.032).

Discussion

In this single-center retrospective study, we demonstrated that the PIV, a novel hematological marker of systemic inflammation, is independently associated with in-hospital mortality in patients with ADHF in HFrEF. Our study is particularly relevant because it focuses exclusively on patients with LVEF <40%, a population at inherently higher risk, whereas many prior reports included mixed HF phenotypes [14, 15].

We observed an in-hospital mortality rate of 18.8%, which is higher than that reported in several international registries but in line with outcomes from other Turkish cohorts involving high-risk patients. For instance, the Journey HF-TR registry reported an overall in-hospital mortality of 7.3% in ADHF, but this population included mid-range and preserved EF patients, who typically have better short-term outcomes [18]. Thus, our findings emphasize the prognostic value of PIV in a more severe and homogeneous HFrEF population.

Systemic inflammation is increasingly recognized as a key driver of HF progression, contributing to adverse remodeling, neurohormonal activation, and multi-organ dysfunction [4]. Several studies have shown that traditional inflammatory markers, such as CRP, NLR, PLR, and SII predict outcomes in HF [19-21]. However, these indices represent only a fraction of the inflammatory response. By integrating neutrophil, monocyte, platelet, and lymphocyte counts, PIV provides a more comprehensive assessment of systemic immune-inflammation. This may explain why, in our study, PIV remained independently associated with mortality, whereas CRP, serum albumin, and LVEF

lost significance after multivariable adjustment.

Although hypoalbuminemia, reduced LVEF, and elevated CRP have been linked to worse prognosis in HF in prior studies [21-23], their lack of independent significance in our analysis may be attributable to the characteristics of our cohort. Both groups had globally depressed systolic function (mean LVEF ~31% vs. 29%), minimizing intergroup variability. Similarly, baseline serum albumin levels were already low in both survivors and non-survivors, reflecting chronic malnutrition and systemic inflammation common in advanced HF [4, 24]. Moreover, persistently elevated CRP across the entire cohort may have masked its discriminative capacity.

The ROC analysis in our study confirmed the strong predictive ability of PIV, with an AUC of 0.735 and an optimal cut-off of 883.07. Patients with PIV values above this threshold had more than a fourfold increased risk of in-hospital mortality, even after adjustment for confounders. These findings are consistent with previous reports linking PIV with both short- and long-term outcomes in ADHF [14, 16].

From a clinical standpoint, natriuretic peptides such as N-terminal pro-B-type natriuretic peptide (NT-proBNP) are well-established prognostic biomarkers in HF [25-27]. However, similar to many centers in Türkiye, including ours, NT-proBNP measurement is not universally available due to financial and logistic limitations. In contrast, PIV can be calculated from routine complete blood counts, making it an inexpensive, universally accessible, and rapid tool for risk stratification. Our findings highlight its potential role in guiding early clinical decisions, especially in resource-limited healthcare systems.

Limitations: This study has several limitations. First, it was a retrospective, single-center analysis, which may limit the generalizability of the findings. Second, the sample size was relatively modest. Third, NT-proBNP levels were not available for comparison with PIV. Finally, our study specifically focused on patients with reduced ejection fraction. While this design choice allowed us to investigate a high-risk and clinically important subgroup, further studies evaluating PIV across the full spectrum of HF phenotypes, including HFmrEF and HFpEF, would be valuable to establish its broader applicability.

Conclusions: In this retrospective single-center study of patients hospitalized with acute decompensated heart failure and reduced ejection fraction, the pan-immune-inflammation value (PIV) was identified as an independent predictor of in-hospital mortality. Our findings highlight that PIV, derived from routine complete blood counts, is a simple, inexpensive, and easily applicable biomarker that provides incremental prognostic information beyond traditional clinical and laboratory parameters. Given its accessibility and cost-effectiveness, PIV may serve as a valuable tool for early risk stratification, particularly in healthcare settings where advanced biomarkers such as NT-proBNP are not routinely available. Future large-scale, multicenter studies across the full spectrum of heart failure phenotypes are warranted to validate these results and to further clarify the clinical role of PIV in guiding management strategies.

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