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Platelet–Albumin–Bilirubin Score Predicts In-Hospital Mortality in Patients with HFrEF Presenting with Acute Heart Failure

ABSTRACT

Background: Patients with heart failure and reduced ejection fraction presenting with acute heart failure (HFrEF-AHF) carry a high in-hospital mortality. Conventional predictors do not fully capture the interplay between hepatic dysfunction, systemic inflammation, and nutritional decline. The platelet–albumin–bilirubin (PALBI) score, originally developed in hepatology, may offer incremental prognostic information in this setting.

Methods: A total of 401 patients hospitalized with HFrEF-AHF were retrospectively studied. Baseline demographic, clinical, and laboratory data were collected. Platelet–albumin–bilirubin and albumin–bilirubin (ALBI) scores were calculated. The primary endpoint was all-cause in-hospital mortality.

Results: In-hospital mortality occurred in 47 patients (11.7%). Non-survivors were older, more frequently had chronic kidney disease (CKD), and showed lower albumin but higher bilirubin and N-terminal pro-B-type natriuretic peptide (NT-proBNP). Both PALBI and ALBI were significantly higher in non-survivors. In multivariable Cox analysis, PALBI independently predicted mortality (hazard ratio = 2.638, 95% CI: 1.177-4.099, $P < .001$), along with age, CKD, and NT-proBNP. Receiver operating characteristic analysis showed that PALBI demonstrated slightly better performance than conventional predictors and, compared with ALBI (area under the curve = 0.737 vs. 0.708), yielded a modest but significant net reclassification improvement (~10%). Kaplan–Meier curves demonstrated clear separation in survival across PALBI strata (log-rank, $P < .001$), and restricted cubic spline analysis confirmed a graded, continuous association with risk.

Conclusion: The PALBI score independently predicts in-hospital mortality in patients with HFrEF-AHF and may represent a simple and clinically useful tool for early risk stratification. External validation in larger, multicenter cohorts is warranted.

Keywords: Acute heart failure, in-hospital mortality, PALBI score, prognosis, risk assessment

INTRODUCTION

Heart failure with reduced ejection fraction (HFrEF) is a major global health problem, accounting for substantial morbidity, frequent hospitalizations, and high rates of in-hospital mortality.[1,2] Patients with HFrEF presenting with acute heart failure (HFrEF-AHF) represent one of the most severe clinical presentations, often requiring urgent hospitalization and intensive management.[3,4] Despite advances in guideline-directed therapies, the short-term prognosis of these patients remains unfavorable,[5] underscoring the importance of accurate, early risk stratification. Identifying reliable prognostic markers at admission is crucial for guiding therapeutic decision-making, allocating healthcare resources efficiently, and improving clinical outcomes.

Traditional risk predictors, such as age, comorbidities, renal function, and serum sodium, have long been recognized as important prognostic factors.[6] However, recent studies have emphasized the role of systemic inflammation, hepatic dysfunction, and nutritional status in the pathophysiology of HFrEF-AHF.[7-9] Congestion and impaired forward flow frequently result in hepatic venous hypertension and hypoperfusion, leading to cholestasis, hyperbilirubinemia, impaired

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albumin synthesis, and thrombocytopenia.[10] At the same time, neurohormonal activation and systemic inflammation drive endothelial dysfunction and oxidative stress, further aggravating multi-organ impairment.[11] These complex pathophysiological processes link cardiac dysfunction with hepatic and hematologic abnormalities, suggesting that biomarkers reflecting this interplay may provide incremental prognostic value.

Among liver-derived indices, the albumin–bilirubin (ALBI) score has been increasingly studied in AHF and shown to carry prognostic significance.[12] Building on this concept, the platelet–albumin–bilirubin (PALBI) score incorporates platelet count, in addition to albumin and bilirubin, thereby embedding a hematologic dimension into the assessment of cardihepatic interactions. Although originally applied in hepatology,[13] recent evidence suggests that the PALBI score may also be beneficial in cardiovascular disorders. To date, only 1 study has evaluated the prognostic impact of the PALBI score in cardiovascular disease, demonstrating that a higher PALBI score was independently associated with all-cause mortality following transcatheter aortic valve replacement.[14] However, data regarding its role in HFrEF-AHF remain scarce, and its predictive utility for in-hospital mortality has not been clearly established.

Given this background, the present study was designed to evaluate the prognostic significance of the PALBI score in patients hospitalized with HFrEF-AHF. Specifically, this study aimed to determine whether the PALBI score could serve as an independent predictor of in-hospital mortality, thereby providing clinicians with an additional, easily obtainable tool for early risk stratification in this high-risk population.

METHODS

Study Population

This single-center, retrospective cohort study included patients diagnosed with HFrEF (left ventricular ejection fraction [LVEF] of 40% or less) according to the European Society of Cardiology guidelines for heart failure[15] who were hospitalized with a primary diagnosis of HFrEF-AHF, encompassing the full clinical spectrum of AHF, including acutely decompensated heart failure, acute pulmonary edema, and cardiogenic shock, in order to reflect routine clinical practice and derive real-world data. All consecutive patients admitted to the tertiary cardiology clinic between January 2024 and December 2024 were screened for eligibility. Patients were eligible for inclusion if they were 18 years of age or older, had a confirmed diagnosis of HFrEF prior to or during admission, and had complete laboratory data necessary for calculating the PALBI score at the time of admission. Patients admitted with a diagnosis of acute coronary syndrome, with a history of chronic liver disease or cirrhosis, chronic inflammatory disease, active malignancy or hematological disease, prior liver transplantation, or those receiving immunosuppressive therapy were excluded. Additionally, patients with missing data for any parameter required for PALBI calculation, as well as those who were transferred to another institution or were lost to follow-up during their hospitalization, were excluded from the final analysis. After applying the inclusion and exclusion criteria, 401 patients with HFrEF-AHF were included in the final study cohort. All clinical and laboratory data of the patients were obtained retrospectively from electronic medical records and institutional databases. The study was approved by the Local Ethics Committee of the Ankara Etlik City Hospital, approval date and number: 22 July 2025 – AEŞH-BADEK2-2025-278 and was conducted in accordance with the Declaration of Helsinki. The Local Ethics Committee waived the requirement for informed consent because the research was retrospective.

HIGHLIGHTS

- Patients with HFrEF presenting with acute heart failure (HFrEF-AHF) still carry a high in-hospital mortality.
- Platelet–albumin–bilirubin combines platelet count, serum albumin, and bilirubin into one index.
- Platelet–albumin–bilirubin independently predicted mortality.
- Kaplan–Meier and spline analyses confirmed acceptable prognostic performance.
- Simple lab-based indices may guide early risk stratification in HFrEF-AHF.

Data Collection

Baseline demographic and clinical characteristics, including age, sex, active smoking status, history of hypertension, diabetes mellitus, peripheral artery disease, atrial fibrillation, coronary artery disease, chronic kidney disease (CKD), and chronic obstructive pulmonary disease (COPD), were recorded for each patient. In addition, baseline medications at the time of index hospitalization, including angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, beta-blockers, mineralocorticoid receptor antagonists, sodium-glucose cotransporter 2 inhibitors, antiplatelet agents, and anticoagulants, were documented.

Laboratory data were collected from venous blood samples obtained within the first 24 hours of hospital admission. These included complete blood count (platelet count, white blood cells, neutrophils, lymphocytes, hemoglobin), liver function parameters (total bilirubin, serum albumin), renal function tests (urea, creatinine), electrolytes (sodium, potassium, chloride), lipid profile, C-reactive protein (CRP), and N-terminal pro-brain natriuretic peptide (NT-proBNP) levels. Chronic kidney disease was defined as an estimated glomerular filtration rate <60 mL/min/1.73 m^2, in accordance with the Kidney Disease: Improving Global Outcomes guidelines.[16]

Transthoracic echocardiography was performed on all patients during hospitalization by experienced cardiologists to evaluate left ventricular function. Left ventricular ejection fraction was measured according to the recommendations of the American Society of Echocardiography[17] using the biplane Simpson's method.

Platelet–albumin–bilirubin score was calculated using the following formula:[13]

PALBI score = 2.02 × $\log_{10}$ [bilirubin (μmol/L)] − 0.37 × ($\log_{10}$ bilirubin)^2 − 0.04 × albumin (g/L) − 3.48 × $\log_{10}$ [platelet count (×10^9/L)] + 1.01 × ($\log_{10}$ platelet count)^2.

Albumin–bilirubin score was calculated as follows:[13]

ALBI score = (0.66 × $\log_{10}$ bilirubin [μmol/L]) + (−0.085 × albumin [g/L]).

Study Endpoint

The outcome of the study was all-cause in-hospital mortality, defined as death occurring during the index hospitalization. Survival status was determined from hospital medical records and, when necessary, verified through the national death registry.

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics, version 26.0 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was evaluated with the Kolmogorov–Smirnov test. Normally distributed data are summarized as mean ± SD, and non-normally distributed data were expressed as median (interquartile range). Categorical variables are presented as frequencies and percentages. For comparisons between 2 groups (survivors vs. non-survivors), the independent-samples *t*-test or Mann–Whitney *U*-test was applied for continuous variables. In contrast, the chi-square test or Fisher's exact test was used for categorical variables. For comparisons across 3 groups, a 1-way ANOVA was used for normally distributed variables, with post hoc Tukey's test where appropriate. When assumptions of normality were violated, the Kruskal–Wallis test with Bonferroni-adjusted pairwise comparisons was used. To investigate prognostic factors for in-hospital mortality, univariable Cox proportional hazards regression analyses were first performed. Variables associated with the outcome in univariable analysis and considered clinically relevant were subsequently evaluated for inclusion in a multivariable Cox proportional hazards regression model. Given the limited number of mortality events (n = 47), a parsimonious multivariable model was constructed to minimize the risk of overfitting. The final model included age, CKD, CRP, NT-proBNP, and PALBI score. Variables exhibiting substantial clinical overlap or multicollinearity were not entered simultaneously into the model. Specifically, serum creatinine was not included alongside CKD because both variables reflect renal dysfunction, whereas serum albumin, total bilirubin, and ALBI score were excluded because they are components of or closely related to the PALBI score. Hazard ratios (HRs) and 95% CIs were reported. Assumptions of proportional hazards were assessed using log–log survival plots, and multicollinearity was evaluated using variance inflation factors. The discriminatory ability of the predictors was further assessed using receiver operating characteristic (ROC) curve analyses; area under the curve (AUC) values and optimal cutoffs (based on the Youden index) were calculated. Comparative ROC analyses were performed for both PALBI and ALBI scores against traditional predictors. In addition, the incremental prognostic value was evaluated using net reclassification improvement (NRI) and integrated discrimination improvement (IDI) metrics, which quantify the added reclassification and discrimination capacity of PALBI relative to ALBI. For survival analysis, patients were stratified by PALBI score, and Kaplan–Meier curves were generated; comparisons were made using the log-rank test. Two complementary stratification approaches were applied: (i) Median-based dichotomization (PALBI < −2.047 vs. ≥ −2.047); (ii) interquartile-based stratification (≤25th percentile, 25th-75th percentile, and ≥75th percentile). Finally, a restricted cubic spline model was fitted to illustrate the continuous relationship between the PALBI score and the risk of in-hospital mortality. All statistical tests were 2-sided, and $P < .05$ was considered statistically significant.

RESULTS

A total of 401 patients hospitalized for HFrEF-AHF were included in the study. The cohort's mean age was 62.9 ± 14.4 years, and 72.3% (n = 290) of participants were male. Baseline demographic, clinical, laboratory, and echocardiographic characteristics of survivors (n = 354) and patients who died during hospitalization (n = 47) are summarized in Table 1. Compared to survivors, non-survivors were significantly older (72.6 ± 11.1 vs. 61.5 ± 14.6 years, $P < .001$) and had a markedly higher prevalence of CKD (68.1 vs. 26.6%, $P < .001$). Laboratory analyses showed that non-survivors had lower serum albumin levels and higher CRP, NT-proBNP, and total bilirubin levels. The mean PALBI score was also significantly higher among non-survivors (−1.80 ± 0.45 vs. −2.19 ± 0.39, $P < .001$). Similarly, the mean ALBI score was significantly higher (less negative) among non-survivors compared to survivors (−1.93 ± 0.66 vs. −2.38 ± 0.51, $P < .001$). No significant differences were observed between the 2 groups with respect to other baseline comorbidities, medication use, or echocardiographic parameters.

In univariable regression analysis, age, CKD, CRP, serum creatinine, NT-proBNP, serum albumin, total bilirubin, PALBI score, and ALBI score were significantly associated with in-hospital mortality (Table 2). In the final parsimonious multivariable model, age (HR = 1.067, 95% CI: 1.031-1.105, $P < .001$), CKD (HR = 1.266, 95% CI: 1.120-1.591, $P = .001$), NT-proBNP (per 500-unit increase) (HR = 1.245, 95% CI: 1.106-1.567, $P = .001$), and PALBI score (HR = 2.638, 95% CI: 1.177-4.099, $P < .001$) remained as independent predictors of in-hospital mortality.

The ROC curve analysis demonstrated that both hepatic scores carried significant discriminatory power for in-hospital mortality. The AUC for PALBI was 0.737 (95% CI: 0.653-0.816), which was higher than that of ALBI (AUC = 0.708, 95% CI: 0.615-0.788), age, CKD, and NT-proBNP (Table 3). The optimal cutoff value for PALBI was −2.047 (sensitivity, 70%; specificity, 68%), whereas for ALBI, it was −2.010 (sensitivity, 60%; specificity, 76%). Comparative ROC curves are shown in Figure 1A. To further characterize patients according to the optimal PALBI cutoff identified by ROC analysis, baseline demographic, clinical, laboratory, and echocardiographic characteristics were compared between patients with PALBI < −2.047 and those with PALBI ≥ −2.047 (Table 4). The 2 groups were comparable with respect to age, sex, cardiovascular comorbidities, baseline medications, and LVEF. However, patients with

Table 1. Baseline Demographic, Clinical, and Laboratory Characteristics of the Study Population, Stratified by Study Outcome

Variables	Survivors (n = 354)	Deceased (n = 47)	*P*
Demographics			
Age (years)	61.5 ± 14.6	72.6 ± 11.1	<.001
Male, n (%)	254 (71.8)	36 (76.6)	.486
Comorbidities, n (%)			
Smoking, n (%)	157 (44.4)	18 (38.3)	.293
Hypertension	215 (60.7)	32 (68.1)	.330
Diabetes mellitus	119 (33.6)	14 (29.8)	.600
Atrial fibrillation	136 (38.4)	20 (42.6)	.699
Coronary artery disease	138 (39.0)	20 (42.6)	.637
Peripheral artery disease	25 (7.1)	6 (12.8)	.169
Chronic kidney disease	94 (26.6)	32 (68.1)	<.001
Chronic obstructive pulmonary disease	100 (28.2)	18 (38.3)	.155
Medications, n (%)			
Angiotensin-converting enzyme inhibitors	259 (73.2)	29 (61.7)	.101
Angiotensin II receptor blockers	50 (14.1)	10 (21.3)	.197
Beta-blockers	325 (91.8)	43 (91.5)	1.000
Mineralocorticoid receptor antagonists	258 (72.9)	29 (61.7)	.110
SGLT-2 inhibitors	114 (32.2)	14 (29.8)	.738
Asetylsalicylic acid	217 (61.3)	26 (55.3)	.430
Oral anticoagulant	115 (32.5)	21 (44.7)	.100
Statins	162 (45.8)	22 (46.8)	.892
Laboratory findings			
Hemoglobin (g/dL)	13.2 ± 2.2	12.6 ± 2.7	.398
White blood cells (×10^9/L)	7.7 (6.5-9.2)	7.6 (6.4-8.6)	.494
Neutrophil (×10^9/L)	6.1 (4.8-9.1)	6.4 (5.0-9.4)	.204
Lymphocyte (×10^9/L)	1.6 (1.1-2.1)	1.6 (1.0-2.0)	.123
Platelets (×10^9/L)	241 (190-297)	251 (177-301)	.182
C-reactive protein (mg/dL)	18.6 (8.0-42.0)	41.0 (21.0-78.4)	<.001
Serum creatinine (mg/dL)	1.2 (0.9-2.0)	2.7 (1.9-3.2)	<.001
Serum sodium (mmol/L)	138 (135-140)	134 (132-137)	.104
Serum potassium (mmol/L)	4.3 (4.0-4.6)	4.5 (4.1-4.7)	.142
Serum chloride (mmol/L)	102 (98-104)	102 (98-103)	.902
AST (U/L)	16 (13-23)	17 (13-23)	.881
ALT (U/L)	21 (10-31)	24 (12-32)	.462
Total cholesterol (mg/dL)	162 (131-200)	163 (124-222)	.887
LDL-cholesterol (mg/dL)	114 (88-161)	124 (86-200)	.283
HDL-cholesterol (mg/dL)	36 (29-44)	33 (26-42)	.163
Triglyceride (mg/dL)	114 (86-156)	123 (81-228)	.211
NT-proBNP (pg/mL)	1892 (712-5378)	2024 (589-10 276)	<.001
Serum albumin (g/L)	37.1 ± 5.9	33.9 ± 7.1	.001
Total bilirubin (μmol/L)	15 (11-19)	33 (16-43)	<.001
PALBI score	−2.19 ± 0.39	−1.80 ± 0.45	<.001
ALBI score	−2.38 ± 0.51	−1.93 ± 0.66	<.001
LVEF (%)	26.3 ± 6.3	27.8 ± 5.2	.103
Length of in-hospital stay (days)	8 (6-13)	6 (4-10)	.021

ALBI, albumin–bilirubin; ALT, alanine aminotransferase; AST, aspartate aminotransferase; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro-B-type natriuretic peptide; PALBI, platelet–albumin–bilirubin; SGLT-2, sodium-glucose cotransporter-2.

PALBI ≥ −2.047 had a higher prevalence of COPD (37.4% vs. 24.8%, $P = .011$). They also exhibited lower hemoglobin and serum albumin levels, and higher neutrophil counts, platelet counts, CRP, creatinine, alanine aminotransferase, NT-proBNP, and total bilirubin levels (all $P < .05$). Importantly, in-hospital mortality was markedly higher in

Table 2. Univariable and Multivariable Cox Proportional Hazards Regression Analyses for Predictors of In-hospital Mortality in Patients with Acute Decompensated Heart Failure with Reduced Ejection Fraction

Variables	Univariable Regression Analysis		Multivariable Regression Analysis	
	HR (95% CI)	*P*	HR (95% CI)	*P*
Age (years)	1.067 (1.038-1.096)	<.001	1.067 (1.031-1.105)	<.001
Chronic kidney disease	1.169 (1.088-1.327)	<.001	1.266 (1.120-1.591)	.001
C-reactive protein	1.011 (1.004-1.017)	.001	1.009 (1.000-1.019)	.054
Serum creatinine†	1.685 (1.456-1.950)	<.001	–	–
NT-proBNP, per 500-unit increase	1.169 (1.081-1.350)	<.001	1.245 (1.106-1.567)	.001
Serum albumin‡	0.933 (0.892-0.976)	.003	–	–
Total bilirubin‡	1.080 (1.058-1.102)	<.001	–	–
PALBI score	2.523 (1.285-3.761)	<.001	2.638 (1.177-4.099)	<.001
ALBI score‡	3.153 (1.955-5.084)	<.001	–	–

ALBI, albumin–bilirubin; HR, hazard ratio; NT-proBNP, N-terminal pro-B-type natriuretic peptide; PALBI, platelet–albumin–bilirubin; VIF, variance inflation factor.
†Serum creatinine was not included simultaneously with chronic kidney disease due to clinical overlap and multicollinearity between these renal function markers (pairwise VIF = 6.391).
‡Serum albumin, total bilirubin, and ALBI score were not included in the multivariable model because they are components of, or closely related to, the PALBI score. When PALBI, ALBI, albumin, and bilirubin were entered together, marked multicollinearity was observed, particularly for ALBI (VIF = 200.042), albumin (VIF = 164.011), and bilirubin (VIF = 12.197). Therefore, PALBI was retained as the representative composite index in the final parsimonious model.

patients with PALBI ≥ −2.047 compared with those with PALBI < −2.047 (22.4% vs. 5.5%, $P < .001$).

Beyond AUC, PALBI provided incremental prognostic information over ALBI as reflected by an NRI (9.9%) and a small IDI (0.5%). The reclassification scatter plot (Figure 1B) shows that PALBI shifted a subset of patients—particularly those who died in the hospital—toward higher predicted-risk categories than ALBI, thereby improving bedside risk stratification.

Table 3. Receiver Operating Characteristic Curve Analysis of Age, Chronic Kidney Disease, NT-proBNP, PALBI, and ALBI Scores for Predicting In-hospital Mortality in Patients with Acute Decompensated Heart Failure with Reduced Ejection Fraction

Variables	AUC (95% CI)	Cutoff Point	*P*	Sensitivity (%)	Specificity (%)
Age (years)	0.722 (0.657-0.790)	68	<.001	68	67
Chronic kidney disease	0.708 (0.637-0.775)	–	<.001	–	–
NT-proBNP	0.702 (0.633-0.765)	982	<.001	69	68
PALBI score	0.737 (0.653-0.816)	−2.047	<.001	70	68
ALBI score	0.708 (0.615-0.788)	−2.010	<.001	60	76

ALBI, albumin–bilirubin; AUC, area under the curve; NT-proBNP, N-terminal pro-B-type natriuretic peptide; PALBI, platelet–albumin–bilirubin.

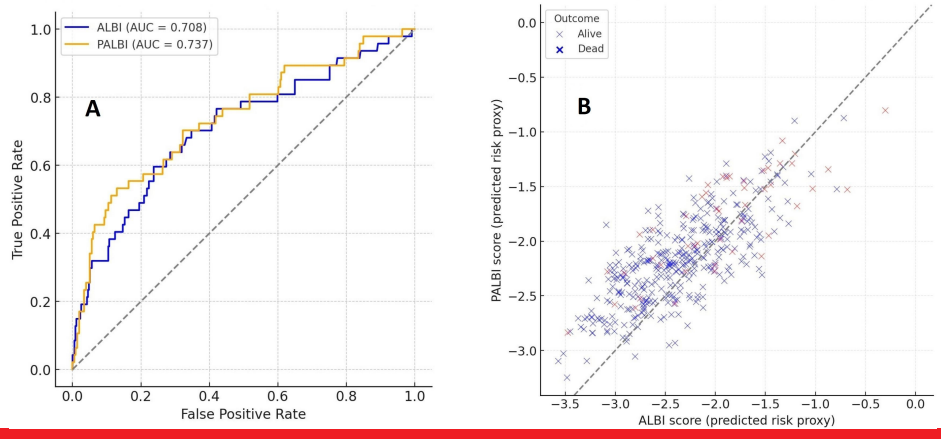

Figure 1. Prognostic performance of PALBI and ALBI in patients with heart failure and reduced ejection fraction presenting with acute heart failure: (A) Receiver operating characteristic curves comparing the discriminative ability of PALBI and ALBI for in-hospital mortality. (B) Reclassification plot illustrating the relationship between PALBI and ALBI scores, stratified by survival status. ALBI, albumin–bilirubin; PALBI, platelet–albumin–bilirubin.

Table 4. Baseline Demographic, Clinical, Laboratory, and Echocardiographic Characteristics of Patients Stratified According to the Optimal PALBI Cutoff Value (PALBI < −2.047 vs. PALBI ≥ −2.047)

Variables	PALBI < −2.047 (n = 254)	PALBI ≥ −2.047 (n = 147)	*P*
Demographics			
Age (years)	62.2 ± 14.5	63.7 ± 14.8	.317
Male, n (%)	188 (74.0)	102 (69.4)	.378
Comorbidities, n (%)			
Smoking, n (%)	112 (44.1)	63 (42.9)	.820
Hypertension	151 (59.4)	96 (65.3)	.291
Diabetes mellitus	86 (33.9)	47 (32.0)	.782
Atrial fibrillation	92 (36.2)	64 (43.4)	.146
Coronary artery disease	99 (39.0)	59 (40.1)	.902
Peripheral artery disease	20 (7.9)	11 (7.5)	1.000
Chronic kidney disease	77 (30.3)	49 (33.3)	.606
Chronic obstructive pulmonary disease	63 (24.8)	55 (37.4)	.011
Medications, n (%)			
Angiotensin-converting enzyme inhibitors	183 (72.0)	105 (71.4)	.986
Angiotensin II receptor blockers	37 (14.6)	23 (15.6)	.883
Beta-blockers	233 (91.7)	135 (91.8)	1.000
Mineralocorticoid receptor antagonists	183 (72.0)	104 (70.7)	.871
SGLT-2 inhibitors	87 (34.3)	41 (27.9)	.228
Asetylsalicylic acid	155 (61.0)	88 (59.9)	.902
Oral anticoagulant	80 (31.5)	56 (38.1)	.180
Statins	112 (44.1)	72 (49.0)	.400
Laboratory findings			
Hemoglobin (g/dL)	13.5 ± 2.2	12.5 ± 2.3	<.001
White blood cells (×10^9/L)	7.6 (6.4-8.9)	7.9 (6.6-9.2)	.186
Neutrophil (×10^9/L)	5.1 (3.9-6.8)	6.0 (4.8-8.1)	<.001
Lymphocyte (×10^9/L)	1.9 (1.3-2.4)	1.8 (1.1-2.4)	.996
Platelets (×10^9/L)	216 (174.2-268.5)	283 (241.0-334.8)	<.001
C-reactive protein (mg/dL)	18.4 (8.0-41.8)	22.0 (10.7-57.5)	.025
Serum creatinine (mg/dL)	1.2 (0.9-2.0)	1.3 (1.0-2.3)	.043
Serum sodium (mmol/L)	138 (135-140)	137 (132.5-139)	.030
Serum potassium (mmol/L)	4.3 (4.0-4.6)	4.4 (4.0-4.7)	.875
Serum chloride (mmol/L)	101.5 (98-104)	102 (98-104)	.916
AST (U/L)	16 (13-23)	16 (12-23)	.640
ALT (U/L)	20 (9-29.5)	24 (12-35)	.018
Total cholesterol (mg/dL)	167 (131-201.5)	158.5 (130-200)	.236
LDL-cholesterol (mg/dL)	116 (88-167.5)	117.5 (88-171.5)	.893
HDL-cholesterol (mg/dL)	36 (29.8-44)	34 (27.6-43)	.665
Triglyceride (mg/dL)	118 (89.2-165.8)	110 (79.5-150.5)	.088
NT-proBNP (pg/mL)	1676 (638-4899)	2736 (931-7797)	<.001
Serum albumin (g/L)	39.1 ± 5.1	32.6 ± 5.6	<.001
Total bilirubin (μmol/L)	13 (10-16)	23 (15-33)	<.001
PALBI score	−2.4 ± 0.3	−1.7 ± 0.3	<.001
LVEF (%)	26.4 ± 6.3	26.5 ± 6.0	.830
Length of in-hospital stay (days)	8 (5-12)	8 (5-15)	.587
In-hospital mortality, n (%)	14 (5.5)	33 (22.4)	<.001

ALT, alanine aminotransferase; AST, aspartate aminotransferase; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro-B-type natriuretic peptide; PALBI, platelet–albumin–bilirubin; SGLT-2, sodium-glucose cotransporter-2.

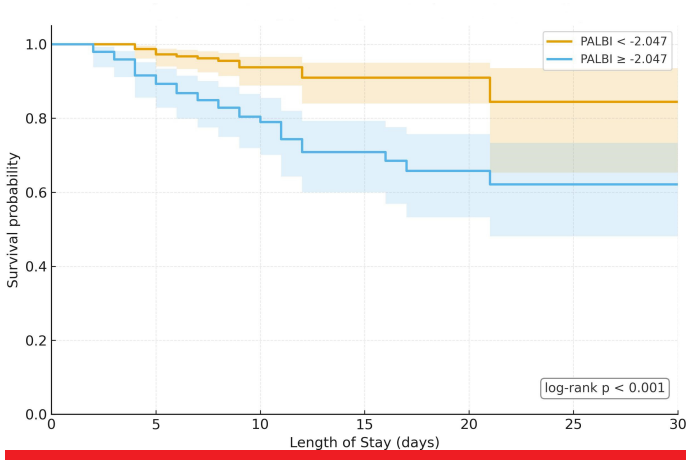

Figure 2. Kaplan–Meier survival curves stratified by PALBI score. Patients with higher PALBI values (≥ −2.047) had significantly lower in-hospital survival compared with those with lower scores (< −2.047) (log-rank *P* < .001). PALBI, platelet–albumin–bilirubin.

Kaplan–Meier survival curves showed that patients in the high-risk group (PALBI ≥ −2.047) had significantly higher in-hospital mortality than those in the low-risk group (PALBI < −2.047) (log-rank *P* < .001; Figure 2). Similarly, when patients were divided into tertiles based on PALBI scores [tertile 1 (< −2.458), tertile 2 (−2.458 to −1.896), tertile 3 (> −1.896)], survival rates declined gradually from tertile 1 to tertile 3 (log-rank *P* < .001; Figure 3).

Further stratified analyses are presented in Table 5, in which patients were categorized into 3 groups based on the PALBI score distribution (< −2.458, −2.458 to −1.896, and > −1.896). Comparisons revealed that higher PALBI scores were associated with more adverse clinical profiles, including a greater prevalence of COPD, as well as significant laboratory differences, including lower hemoglobin levels, higher neutrophil counts, and higher NT-proBNP levels. Platelet counts, serum albumin, and total bilirubin levels also differed significantly across the groups in line with the PALBI score components. Importantly, in-hospital mortality increased markedly across the groups, from 4% in the lowest PALBI group to 27% in the highest group (*P* < .001).

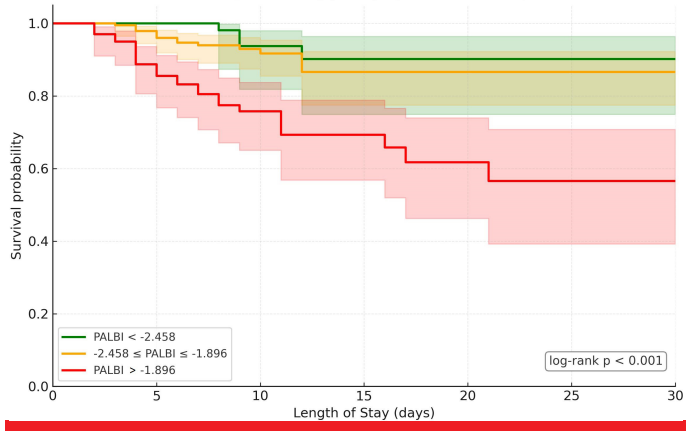

Figure 3. Kaplan–Meier survival curves stratified by PALBI tertiles. Patients with higher PALBI scores (> −1.896, red line) had significantly lower in-hospital survival compared with intermediate (−2.458 ≤ PALBI ≤ −1.896, yellow line) and lower PALBI scores (< −2.458, green line) (log-rank *P* < .001). PALBI, platelet–albumin–bilirubin.

Restricted cubic spline analysis was performed to further evaluate the relationship between PALBI score and in-hospital mortality. This analysis demonstrated a significant overall association, indicating that higher PALBI scores were associated with a higher risk of in-hospital mortality (*P* < .001). However, the test for nonlinearity was not statistically significant (*P* = .360), suggesting that the association was primarily linear rather than curvilinear. The predicted probability of in-hospital mortality increased progressively across the PALBI score spectrum, as illustrated in Figure 4.

DISCUSSION

The present study provides novel evidence that the PALBI score, a composite index traditionally used to assess hepatic functional reserve, has prognostic value in HFrEF-AHF. By systematically examining its association with short-term outcomes, this study identified PALBI as an independent predictor of in-hospital mortality. To the authors' knowledge, this is the first report to establish a direct link between PALBI and adverse prognosis in HFrEF-AHF, thereby expanding the clinical applicability of this biomarker beyond hepatology and opening a new perspective for integrated risk assessment in cardiovascular care.

The pathophysiology of HFrEF-AHF is inherently complex, involving the interplay between cardiac dysfunction, systemic inflammation, venous congestion, and multi-organ impairment.[18] Hepatic involvement is well recognized; elevated right atrial pressures and impaired forward flow lead to hepatic venous congestion, hypoperfusion, and subsequent biochemical abnormalities, including cholestasis, hyperbilirubinemia, and reduced albumin synthesis.[19] Concomitantly, systemic inflammation and neurohormonal activation promote oxidative stress, endothelial dysfunction, and bone marrow suppression, leading to thrombocytopenia and contributing to adverse outcomes.[20,21] The PALBI score integrates 3 readily available laboratory markers that mirror these pathophysiological processes. Thrombocytopenia may reflect systemic inflammation, increased platelet destruction, or impaired bone marrow function.[22] Hypoalbuminemia is a well-established surrogate of both malnutrition and impaired hepatic synthetic capacity.[23] Hyperbilirubinemia reflects cholestasis and impaired clearance due to hepatic congestion.[19] By combining these parameters into a single index, PALBI captures multiple dimensions of HFrEF-AHF pathophysiology, potentially providing a more robust assessment of overall disease burden than individual markers. The association between higher PALBI scores and mortality in HFrEF-AHF underscores the importance of systemic inflammation, nutritional decline, and hepatic dysfunction in driving adverse outcomes. These mechanisms are not merely epiphenomena but central features of the pathophysiology of AHF. Inflammatory cytokines contribute to

Table 5. Comparison of Baseline Characteristics, Comorbidities, Laboratory Results, Echocardiographic Parameters, and In-hospital Outcomes Among Patients Divided into 3 Groups Based on the Platelet–Albumin–Bilirubin Score

Variables	Group 1 (PALBI Score < −2.458, Below 25th Percentile) (n = 100)	Group 2 (PALBI Score Between −2.458 and −1.896, Inclusive, 25th-75th Percentile) (n = 201)	Group 3 (PALBI Score > −1.896, Above 75th Percentile) (n = 100)	*P*
Demographics				
Age (years)	61.4 ± 14.4	63.1 ± 14.4	63.5 ± 15.3	.524
Male, n (%)	75 (75.0)	146 (72.6)	69 (69.0)	.631
Comorbidities, n (%)				
Smoking, n (%)	44 (44.0)	91 (45.3)	40 (40.0)	.249
Hypertension	61 (61.0)	120 (59.7)	66 (66.0)	.566
Diabetes mellitus	34 (34.0)	66 (32.8)	33 (33.0)	.979
Atrial fibrillation	37 (37.0)	80 (39.8)	39 (39.0)	.986
Coronary artery disease	40 (40.0)	75 (37.3)	43 (43.0)	.684
Peripheral artery disease	11 (11.0)	11 (5.5)	9 (9.0)	.206
Chronic kidney disease	25 (25.0)	65 (32.3)	36 (36.0)	.227
Chronic obstructive pulmonary disease	21 (21.0) [a]	54 (26.9) [a]	43 (43.0) [b]	.002
Medications, n (%)				
Angiotensin-converting enzyme inhibitors	74 (74.0)	144 (71.6)	70 (70.0)	.818
Angiotensin II receptor blockers	16 (16.0)	24 (11.9)	20 (20.0)	.172
Beta-blockers	90 (90.0)	186 (92.5)	92 (92.0)	.749
Mineralocorticoid receptor antagonists	65 (65.0)	152 (75.6)	70 (70.0)	.145
SGLT-2 inhibitors	33 (33.0)	63 (32.2)	32 (32.0)	.907
Asetylsalicylic acid	60 (60.0)	124 (61.7)	59 (59.0)	.895
Oral anticoagulant	32 (32.0)	68 (33.4)	36 (36.0)	.740
Statins	47 (47.0)	87 (43.3)	50 (50.0)	.527
Laboratory findings				
Hemoglobin (g/dL)	13.8 ± 2.2 [a]	13.1 ± 2.2 [b]	12.4 ± 2.3 [b]	<.001
White blood cells ($\times 10^9$/L)	7.7 (6.6-8.8)	7.6 (6.4-9.1)	8.0 (6.6-9.2)	.305
Neutrophil ($\times 10^9$/L)	4.7 (3.8-7.0) [a]	5.4 (4.1-6.7) [b]	6.5 (4.9-8.9) [c]	<.001
Lymphocyte ($\times 10^9$/L)	1.9 (1.2-2.4)	1.9 (1.3-2.5)	1.8 (1.1-2.4)	.257
Platelets ($\times 10^9$/L)	195 (156-227) [a]	245 (198-290) [b]	303 (251-353) [c]	<.001
C-reactive protein (mg/dL)	18.1 (8.0-46.8)	20.0 (8.6-39.8)	22.0 (12.0-55.0)	.125
Serum creatinine (mg/dL)	1.1 (0.9-2.0)	1.3 (1.0-2.0)	1.3 (1.1-2.3)	.115
Serum sodium (mmol/L)	138 (136-140)	138 (135-139)	137 (132-139)	.102
Serum potassium (mmol/L)	4.3 (4.0-4.6)	4.3 (3.9-4.6)	4.4 (4.1-4.7)	.532
Serum chloride (mmol/L)	102 (98-105)	102 (98-104)	101 (98-103)	.664
Total cholesterol (mg/dL)	168 (131-201)	160 (130-196)	165 (128-209)	.730
LDL-cholesterol (mg/dL)	115 (87-160)	112 (88-153)	124 (92-190)	.248
HDL-cholesterol (mg/dL)	37 (31-46)	34 (28-43)	34 (28-43)	.071
Triglyceride (mg/dL)	128 (86-180)	110 (88-153)	118 (85-166)	.452
NT-proBNP (pg/mL)	1447 (515-4488) [a]	2001 (760-5491) [b]	2940 (915-5970) [c]	<.001
Serum albumin (g/L)	41 (38-44) [a]	37 (33-41) [b]	32 (28-35) [c]	<.001
Total bilirubin (μmol/L)	10 (8-14) [a]	15 (12-19) [b]	26 (17.8-36.5) [c]	<.001
LVEF (%)	26.5 ± 6.4	26.2 ± 6.5	26.9 ± 5.4	.697
In-hospital mortality, n (%)	4 (4.0) [a]	16 (8.0) [a]	27 (27.0) [b]	<.001

Different superscripts indicate the statistical difference between groups.
HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVEF, left ventricular ejection fraction; N-terminal pro-B-type natriuretic peptide; SGLT-2, sodium-glucose cotransporter-2.

endothelial dysfunction, renal impairment, and myocardial injury.[24] Nutritional depletion reduces physiological reserve and impairs recovery.[25] Hepatic congestion and dysfunction perpetuate multi-organ failure.[26] Platelet–albumin–bilirubin captures these interrelated processes in a single index, offering mechanistic plausibility for its prognostic value. Moreover, the linear relationship observed in spline analysis suggests that even modest worsening of these parameters is associated with increased risk, reinforcing the need for comprehensive early assessment.

This study's results align with and extend prior evidence that liver-derived composite indices convey prognostic information in AHF. Multiple independent studies have now evaluated the ALBI score in AHF and consistently demonstrated prognostic utility. In the multicenter Registry Focused on Very Early Presentation and Treatment in Emergency Department of Acute Heart Failure (REALITY-AHF) registry (n = 1190), higher ALBI at emergency department presentation was associated with clinical congestion (peripheral edema, jugular venous distension, pleural effusions), added discriminative value beyond established risk factors, and independently predicted 1-year mortality.[12] These data supported ALBI as an integrated marker of liver dysfunction related to fluid overload with a genuine prognostic signal in AHF. Complementing those findings, a separate single-center study of 262 AHF admissions reported that ALBI measured at admission was independently associated with in-hospital all-cause mortality even after adjustment for the The Get With the Guidelines-Heart Failure (GWTG-HF) risk score and natriuretic peptides, and that adding ALBI improved risk classification over a clinical model.[27] Subsequent investigations have expanded the ALBI evidence base. In intensive care unit (ICU) heart failure cohorts from the Medical Information Mart for Intensive Care III (MIMIC-III) and MIMIC-IV databases, ALBI independently predicted both short-term and long-term mortality.[28] Luo et al[28] showed that combining ALBI with the GWTG-HF score (GWTG-HF-ALBI) improved discrimination compared with GWTG-HF alone. At the same time, Wang et al[29] demonstrated that each increment in ALBI tertile was associated with a ~10% increase in 1-year mortality and that a 1-unit increase conferred a 36% higher risk, reinforcing ALBI as a robust cardio-hepatic risk marker. More recently, in elderly patients hospitalized for AHF, higher ALBI identified markedly worse long-term survival, underscoring its generalizability across vulnerable age groups.[30] Collectively, these analyses indicate that a composite built from albumin and bilirubin—capturing both nutritional/inflammatory states and cholestasis from venous congestion[10]—stratifies risk across diverse AHF populations, including unselected, ICU, and elderly cohorts.

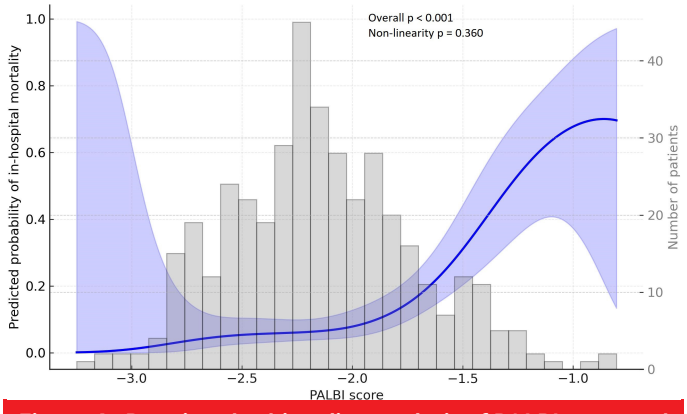

Figure 4. Restricted cubic spline analysis of PALBI score and in-hospital mortality. The plot demonstrates a graded, approximately linear association between higher PALBI scores and the probability of in-hospital mortality (overall $P < .001$; nonlinearity $P = .360$). Histogram bars represent the distribution of PALBI scores within the study population. PALBI, platelet–albumin–bilirubin.

The present work advances this literature in 3 important ways. First, whereas prior ALBI studies enrolled broad AHF cohorts with mixed ejection fraction phenotypes.[12,27-30] The present analysis focused specifically on patients with HFrEF presenting with acute decompensation—an especially high-risk subgroup in whom organ cross-talk (hepatic congestion, systemic inflammation, and catabolic/nutritional decline)[26] is pronounced. Within this population, this study confirmed the prognostic relevance of ALBI, consistent with prior reports, and found that PALBI showed modestly improved discriminatory performance. Second, by explicitly incorporating thrombocytopenia into the composite, PALBI embeds a hematologic/inflammatory dimension that ALBI does not capture. Although platelet counts alone did not differ significantly between survivors and non-survivors in this cohort, their integration into the composite with albumin and bilirubin conferred incremental discriminatory value. This suggests that PALBI reflects a broader cardio-hepatic-inflammatory axis that extends beyond individual components, and its prognostic utility derives from the synergy among these parameters rather than from isolated platelet variation. This is biologically relevant in HFrEF-AHF, where systemic inflammation and bone marrow suppression may reduce circulating platelets, and where endothelial dysfunction and oxidative stress can exacerbate multi-organ injury.[31] Taken together, the expanding external evidence on ALBI[12,27-30] and the current study's HFrEF-specific results with PALBI are convergent: simple, routinely available laboratory metrics that reflect hepatic synthetic function/cholestasis (albumin, bilirubin) as well as systemic inflammation/hematologic perturbation (platelets) carry actionable prognostic information in the acute setting.

From a practical perspective, PALBI offers several advantages for clinicians at the front line of HFrEF-AHF care, where in-hospital mortality remains substantial and early therapeutic decisions must be made amid uncertainty. Because it is derived from universally available and inexpensive laboratory tests and captures multiple pathophysiologic domains, PALBI can aid in identifying high-risk patients at admission, support decisions regarding monitoring and therapeutic escalation, and potentially enhance generalizability across diverse patient subgroups. The supportive evidence from ALBI across all-comer AHF, ICU, and elderly cohorts further strengthens the plausibility that the PALBI findings in HFrEF reflect a common cardio-hepatic-inflammatory axis, while

underscoring the need for external validation and direct head-to-head comparisons to establish whether the addition of platelets confers incremental clinical utility.

In addition to PALBI, the current analysis confirmed several well-established prognostic determinants in HFrEF-AHF, including advanced age, CKD, and NT-proBNP.[32-34] These variables have consistently been shown in prior studies to predict adverse outcomes in HFrEF-AHF,[32-34] and their emergence as significant predictors in this cohort strengthens the validity and representativeness of the study's findings. Importantly, the prognostic value of PALBI was demonstrated after adjustment for these conventional risk factors, indicating that it provides incremental information beyond these known predictors. This suggests that PALBI, when combined with established clinical and biochemical markers, could contribute to a more refined and accurate risk-stratification strategy in HFrEF-AHF. Such a multidimensional assessment may enable clinicians to better identify high-risk patients early and tailor therapeutic intensity accordingly.

Study Limitations

This study has several limitations that should be acknowledged. First, its retrospective and single-center design may limit generalizability, and residual confounding cannot be fully excluded despite comprehensive multivariable adjustment. Second, although adequate for primary analyses, the sample size was limited to 47 in-hospital deaths. This inevitably raises concern about the potential for overfitting in multivariable modeling. To mitigate this, this study pre-specified a parsimonious model, applied penalization techniques with bootstrap-based internal validation, and confirmed the robustness of the results through sensitivity analyses; inferences were directionally stable across specifications. Nevertheless, external validation in larger, multicenter cohorts is essential to confirm these findings. Third, although patients with chronic liver disease, active malignancy, or hematologic disorders were excluded, patients with concomitant active infection were not systematically excluded from the study. This may be considered a limitation, as underlying infections could have contributed not only to elevated inflammatory markers such as CRP and neutrophil counts, but also to changes in platelet and albumin levels, potentially influencing PALBI values. Furthermore, subclinical hepatic or inflammatory conditions cannot be entirely excluded and may have influenced PALBI values; moreover, although median aminotransferase levels were not markedly elevated at the cohort level, transient hepatocellular injury related to hemodynamic compromise may have been present in a small subset of patients, reflecting the heterogeneity of clinical presentations within the AHF spectrum. Fourth, the study focused exclusively on patients with HFrEF, thereby strengthening internal homogeneity but limiting extrapolation to other heart failure populations. Fifth, PALBI was assessed only at admission, and dynamic changes during hospitalization—which may provide additional prognostic information—were not evaluated. Additionally, laboratory measurements were obtained from routine clinical testing, and potential interassay or interlaboratory variability may affect reproducibility in other settings. Moreover, PALBI was not directly compared with established biomarkers such as natriuretic peptides or high-sensitivity troponin, which could have further clarified its incremental value. In addition, information on angiotensin receptor—neprilysin inhibitor use could not be reliably assessed. In Türkiye, Angiotensin Receptor-Neprilysin Inhibitors (ARNI) therapy is not reimbursed by the national health insurance system, and its use largely depends on out-of-pocket payments, resulting in inconsistent prescribing and documentation in retrospective hospital records. Therefore, ARNI use was not included in the analysis to avoid potential misclassification and reporting bias. Finally, whether PALBI-guided risk stratification influences therapeutic decision-making or improves outcomes remains to be determined in prospective interventional studies.

CONCLUSION

This study showed for the first time that the PALBI score independently predicts in-hospital mortality in patients with HFrEF-AHF. Derived from simple, widely available laboratory parameters, PALBI captures systemic inflammation, nutritional status, and hepatic dysfunction and provides prognostic information beyond conventional risk factors. By helping identify high-risk patients early, PALBI may support timely, targeted management strategies. External validation in larger and more diverse cohorts is needed to confirm its generalizability and clinical utility.

Ethics Committee Approval: This study was approved by the Ethics Committee of Ankara Etlik City Hospital (Approval No.: AEŞH-BADEK2-2025-278; Date: July 22, 2025).

Artificial Intelligence Usage Statement: The authors confirm that no artificial intelligence (AI)-assisted technologies (such as large language models, chatbots, or image generators) were used in the preparation, writing, analysis, or methodology of this manuscript. While the study itself explores an AI-related hypothesis, all aspects of the research design, data collection, analysis, and manuscript preparation were conducted entirely by the authors, without the assistance of AI tools.

Informed Consent: The Local Ethics Committee waived the requirement for informed consent because the research was retrospective.Peer-review: Externally peer-reviewed.

Author Contributions: Concept – VH, VOT, BÖ; Design – VH VOT, KA; Supervision – VH, VOT, KA, BÖ; Resources – VH, VOT, KA, AS, ÇT, YBŞ, YT; Materials – VH, VOT, KA, AS, ÇT, YBŞ, YT; Data Collection and/or Processing – VH, VOT, KA, AS, ÇT, YBŞ, YT; Analysis and/or Interpretation – AT, BÖ; Literature Search – All authors; Writing – All authors; Critical Review – VH, VOT, BÖ.

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

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REFERENCES

1. Murphy SP, Ibrahim NE, Januzzi JL Jr. Heart failure with reduced ejection fraction: a review. *JAMA*. 2020;324(5):488-504. [CrossRef]

2. Lavalle C, Di Lullo L, Jabbour JP, et al. New challenges in heart failure with reduced ejection fraction: managing worsening events. *J Clin Med*. 2023;12(22):6956. [CrossRef]
3. Lala A, Hamo CE, Bozkurt B, et al. Standardized definitions for evaluation of acute decompensated heart failure therapies: HF-ARC expert panel paper. *JACC Heart Fail*. 2024;12(1):1-15. [CrossRef]
4. Akyıldız Akçay F, Sinan ÜY, Gürbüz DÇ,et al. Gender-related differences in patients with acute heart failure: observation from the journey heart failure-Turkish population study. *Anatol J Cardiol*. 2023;27(11):639-649. [CrossRef]
5. İlhan B, Bozdereli Berikol G, Doğan H, et al. The prognostic accuracy of get with the guidelines-heart failure score alone and with lactate among acute symptomatic heart failure patients: a retrospective cohort study. *Anatol J Cardiol*. 2024;28(6):305-311. [CrossRef]
6. Cohen-Solal A, Laribi S, Ishihara S, et al. Prognostic markers of acute decompensated heart failure: the emerging roles of cardiac biomarkers and prognostic scores. *Arch Cardiovasc Dis*. 2015;108(1):64-74. [CrossRef]
7. Qiu J, Huang X, Kuang M, et al. Evaluating the prognostic value of systemic immune-inflammatory index in patients with acute decompensated heart failure. *ESC Heart Fail*. 2024;11(5):3133-3145. [CrossRef]
8. Pena DL, Ilieșiu AM, Aurelian J, et al. Assessment of decongestion status before discharge in acute decompensated heart failure: a review of clinical, biochemical, and imaging tools and their impact on management decisions. *Medicina (Kaunas)*. 2025;61(5):816. [CrossRef]
9. Huang X, Qiu J, Kuang M, et al. Assessing the predictive value of the controlling nutritional status score on all-cause mortality during hospitalization in patients with acute decompensated heart failure: a retrospective cohort study from Jiangxi, China. *Front Nutr*. 2024;11:1392268. [CrossRef]
10. Park AC, Schilling JD. The cardiohepatic axis in heart failure. *JACC Basic Transl Sci*. 2025;10(7):101312. [CrossRef]
11. Roman-Pepine D, Serban AM, Capras RD, et al. A comprehensive review: unraveling the role of inflammation in the etiology of heart failure. *Heart Fail Rev*. 2025;30(5):931-954. [CrossRef]
12. Matsue Y, Kagiyama N, Yamaguchi T, et al. Clinical and prognostic values of ALBI score in patients with acute heart failure. *Heart Lung Circ*. 2020;29(9):1328-1337. [CrossRef]
13. Yang H, Shen S, Yang Y, et al. Platelet-albumin-bilirubin versus albumin-bilirubin as a predictor of long-term survival for hepatitis B-Induced hepatocellular carcinoma after hepatic resection. *BMC Cancer*. 2025;25(1):855. [CrossRef]
14. Duan X, Qu L, Chen H. The prognostic value of platelet-albumin-bilirubin score in patients undergoing transcatheter aortic valve replacement. *Clin Cardiol*. 2025;48(3):e70100. [CrossRef]
15. McDonagh TA, Metra M, Adamo M, et al. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. *Eur Heart J*. 2021;42(36):3599-3726. [CrossRef]
16. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2024 clinical practice guideline for the evaluation and management of chronic kidney disease. *Kidney Int*. 2024;105(4S):S117-S314. [CrossRef]
17. Mitchell C, Rahko PS, Blauwet LA, et al. Guidelines for performing a comprehensive transthoracic echocardiographic examination in adults: recommendations from the American Society of Echocardiography. *J Am Soc Echocardiogr*. 2019;32(1):1-64. [CrossRef]
18. Njoroge JN, Teerlink JR. Pathophysiology and therapeutic approaches to acute decompensated heart failure. *Circ Res*. 2021;128(10):1468-1486. [CrossRef]
19. Haddadin R, Aboujaoude C, Trad G. Congestive hepatopathy: a review of the literature. *Cureus*. 2024;16(4):e58766. [CrossRef]
20. Mongirdienė A, Laukaitienė J, Skipskis V, et al. Platelet activity and its correlation with inflammation and cell Count Readings in chronic heart failure patients with reduced ejection fraction. *Medicina (Kaunas)*. 2021;57(2):176. [CrossRef]
21. Amara M, Stoler O, Birati EY. The role of inflammation in the pathophysiology of heart failure. *Cells*. 2025;14(14):1117. [CrossRef]
22. Getawa S, Bayleyegn B. Platelet, neutrophil and lymphocyte quantitative abnormalities in patients with heart failure: a retrospective study. *Vasc Health Risk Manag*. 2023;19:69-78. [CrossRef]
23. Biancucci M, Barbiero R, Pennella B, et al. Hypoalbuminaemia and heart failure: a practical review of current evidence. *Eur J Heart Fail*. 2025;27(2):293-306. [CrossRef]
24. Zheng H, Mao X, Fu Z, et al. The role of circulating cytokines in heart failure: a bidirectional, two-sample Mendelian randomization study. *Front Cardiovasc Med*. 2024;11:1332015. [CrossRef]
25. Macrì R, Mollace R, Serra M, et al. Nutritional and nutraceutical support to the failing myocardium: A possible way of potentiating the current treatment of heart failure. *Int J Mol Sci*. 2024;25(22):12232. [CrossRef]
26. Mocan D, Jipa R, Jipa DA, et al. Unveiling the systemic impact of congestion in heart failure: A narrative review of multisystem pathophysiology and clinical implications. *J Cardiovasc Dev Dis*. 2025;12(4):124. [CrossRef]
27. Kawata T, Ikeda A, Masuda H, et al. Association between albumin-bilirubin score at admission and in-hospital mortality in patients with acute heart failure. *Int Heart J*. 2021;62(4):829-836. [CrossRef]
28. Luo Y, Li Z, Liu J, et al. Prognostic value of the albumin-bilirubin score in critically ill patients with heart failure. *Ann Palliat Med*. 2021;10(12):12727-12741. [CrossRef]
29. Wang J, Wang K, Feng G, et al. Association between the albumin-bilirubin (ALBI) score and all-cause mortality risk in Intensive Care Unit patients with heart failure. *Glob Heart*. 2024;19(1):97. [CrossRef]
30. Jurkiewicz M, Szczurek-Wasilewicz W, Skrzypek M, et al. Albumin-bilirubin (ALBI) score predicts long-term survival in elderly patients with decompensated heart failure. *J Clin Med*. 2025;14(3):808. [CrossRef]
31. Theofilis P, Sagris M, Oikonomou E, et al. Inflammatory mechanisms contributing to endothelial dysfunction. *Biomedicines*. 2021;9(7):781. [CrossRef]
32. Baydemir C, Ural D, Karaüzüm K, et al. Predictors of long-term mortality and frequent re-hospitalization in patients with acute decompensated heart failure and kidney dysfunction treated with renin-angiotensin system blockers. *Med Sci Monit*. 2017;23:3335-3344. [CrossRef]
33. Coles AH, Tisminetzky M, Yarzebski J, et al. Magnitude of and prognostic factors associated with 1-year mortality after hospital discharge for acute decompensated heart failure based on ejection fraction findings. *J Am Heart Assoc*. 2015;4(12):e002303. [CrossRef]
34. Nguyen DV, Nguyen SV, Pham AL, et al. Prognostic value of NT-proBNP in the new era of heart failure treatment. *PLoS One*. 2024;19(9):e0309948. [CrossRef]