Journal of Geriatric Cardiology | JGC
RESEARCH ARTICLE
J Geriatr Cardiol 2025; 22(6): 587–595

The prognostic significance of the fibrosis-5 index in patients with acute decompensated heart failure

Sefa Tatar[1,✉], Yunus Emre Yavuz[2], Hilal Nur Gedik[1], Abdullah Içli[1], Hakan Akilli[1]

1. Department of Cardiology, Meram Faculty of Medicine, Necmettin Erbakan University, Konya, Turkey; 2. Department of Cardiology, T.C. Ministry of Health Siirt Training and Research Hospital, Siirt, Turkey
✉ Correspondence to: ssefa_tatar@hotmail.com
https://doi.org/10.26599/1671-5411.2025.06.002

ABSTRACT

BACKGROUND Acute decompensated heart failure (ADHF) is one of the leading causes of mortality, highlighting the importance of early identification of high-risk patients. The fibrosis-5 (FIB-5) index, traditionally used to evaluate hepatic fibrosis, may hold prognostic value in ADHF patients by reflecting systemic congestion, inflammation, and organ dysfunction. The hypothesis of this study is that the FIB-5 index is an independent predictor of 1-month mortality in patients with ADHF.

METHODS This retrospective study included 155 patients diagnosed with ADHF between 2020 and 2024. Patients were divided into two groups based on their left ventricular ejection fraction (LVEF ≤ 40% or LVEF > 50%). Survival was monitored for one month, and clinical, biochemical, and echocardiographic parameters were compared between survivors and death. Logistic regression and receiver operating characteristic curve analyses were performed to assess the prognostic value of the FIB-5 index.

RESULTS During the 1-month follow-up, 66 patients (42.6%) died. The mean FIB-5 index was significantly lower in non-survivors (−10.46 ± 6.93) compared to survivors (−8.10 ± 6.67) (P = 0.03). Multivariate regression analysis identified the FIB-5 index as an independent predictor of 1-month mortality (OR = 1.089, 95% CI: 1.022–1.160, P = 0.009). The receiver operating characteristic curve analysis demonstrated an area under the curve of 0.609 (95% CI: 0.51–0.699) with sensitivity of 59.6% and specificity of 63.4%. Kaplan-Meier survival analysis revealed significantly higher mortality rates among patients with lower FIB-5 values (log-rank: 7.887, P = 0.005).

CONCLUSIONS The FIB-5 index is an independent predictor of 1-month mortality in ADHF patients. Its low cost, non-invasive nature, and ability to reflect systemic inflammation and congestion make it a promising tool for risk stratification. Prospective studies are needed to validate its utility in clinical practice and evaluate its role in guiding therapeutic decisions.

Heart failure (HF) is a complex and progressive clinical syndrome characterized by the inability of the heart to pump sufficient blood to meet metabolic demands of the body. HF is associated with high morbidity and mortality rates worldwide, with a particularly elevated risk of mortality within the first month following acute decompensation.[1] This risk increases with aging, highlighting the critical need for reliable biomarkers to predict prognosis and optimize treatment strategies.

Fibrosis plays a central role in the pathophysiology of HF. Myocardial fibrosis exacerbates ventricular dysfunction, predisposes patients to arrhythmias, and worsens patient outcomes.[2] In patients with HF, systemic circulation is affected, often leading to long-term complications such as renal and hepatic failure. Hepatic congestion due to proximity to the heart impairs liver function and may eventually result in hepatic fibrosis. Recent studies have suggested that indices traditionally used to evaluate systemic fibrosis in liver diseases may also provide prognostic insights into cardiovascular conditions.[3–6] The fibrosis-5 (FIB-5) index, originally developed to assess liver fibrosis, has emerged as a potential marker of systemic fibrosis and inflammation in patients with HF.[7] Despite its promise, the association between the FIB-5 index and short-term (1-month) mortality in patients with HF remains underexplored.

This study aimed to investigate the relationship between the FIB-5 index and 1-month mortality in patients with HF. Establishing this association could provide an

accessible and non-invasive tool to improve risk stratification and guide early intervention strategies.

METHODS

Study Population

This retrospective study was conducted between 2020 and 2024. Initially, 200 patients with HF were included. Patients were divided into two groups: those with heart failure with reduced ejection fraction (HFrEF) (HF with an LVEF ≤ 40%) and those with heart failure with preserved ejection fraction (HFpEF) (HF with an LVEF > 50%). Patients with HFrEF were classified as New York Heart Association (NYHA) class III–IV based on functional capacity or presented with pulmonary or systemic congestion requiring intravenous diuretics. Additional markers of disease progression included more than two hospitalizations or recurrent emergency department visits within the past year, progressive renal function decline, unexplained cachexia, or contraindications to angiotensin-converting enzyme inhibitors due to renal impairment or hypotension. Other critical clinical markers included worsening HF symptoms, intolerance to beta-blockers due to hypotension, diuretic resistance, escalation of furosemide dosage to ≥ 160 mg/dL, and the onset of hyponatremia.[8,9]

The HFpEF group was diagnosed using the current guidelines and diagnostic algorithms.[1] Patients with LVEF > 50%, evidence of cardiac dysfunction as the cause of symptoms, and current or previous HF symptoms, were included. HFpEF diagnosis was further validated using an HFA-PEFF score of ≥ 5.[10] All patients had elevated N-terminal pro-B-type natriuretic peptide (NT-proBNP) levels. Both HFrEF and HFpEF patients were included in the study within 24 h of admission for acute decompensation.

Patients were excluded from the study for the following reasons: advanced aortic insufficiency (n = 10), advanced aortic stenosis (n = 7), prosthetic valve disease (n = 5), advanced mitral insufficiency (n = 8), presence of a cardiac pacemaker (n = 1), non-cardiac causes of death (n = 10), or incomplete medical records (n = 4). After excluding 45 patients, 155 patients were included in the final analysis. A flowchart of the study is shown in Figure 1.

Patients' survival status was assessed within one month after discharge via hospital medical records and follow-up phone calls. The cause of death for deceased patients was determined from medical records, and for unclear cases, the attending physician was consulted to verify whether the cause was cardiac-related. By the end of the 1-month follow-up period, 66 patients were classified as dead, and 89 patients as survivors.

Data Collection

Clinical, echocardiographic, and laboratory findings were evaluated in all patients. Routine hemogram parameters and, biochemical markers such as albumin, aspa-

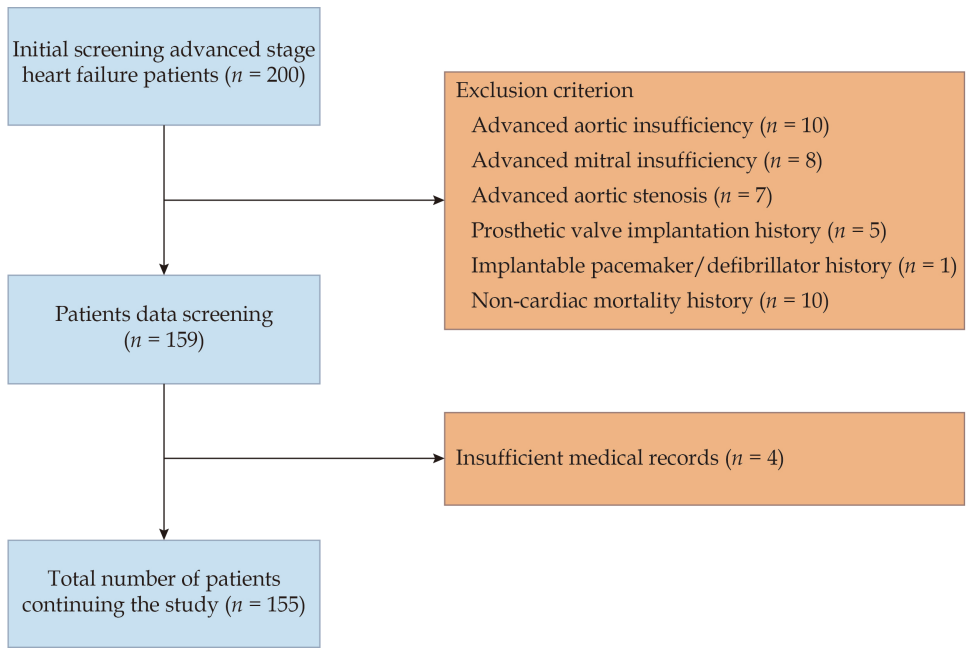

Figure 1 Study flowchart.

rtate aminotransferase (AST), alanine aminotransferase (ALT), and alkaline phosphatase (ALP), were analyzed. The FIB-5 index was calculated using the following formula: FIB-5 = [albumin (g/L) × 0.3 + PLT (10^9/L) × 0.05] − [ALP (U/L) × 0.014 + AST to ALT ratio × 6 + 14].[10] Patients were then stratified into the low and high FIB-5 index groups for comparison. This study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of Necmettin Erbakan University (No.2024/4928).

Statistical Analysis

SPSS Statistics 20.0 software (SPSS Inc., IBM Corp., Armonk, NY, USA) was used for statistical analyses. Normal distribution of the continuous variables was assessed using the Kolmogorov-Smirnov test. Data with normal distribution were expressed as mean ± SD, whereas data without normal distribution were expressed as medians (interquartile range). Categorical variables were expressed as counts (percentages) and the difference between categorical variables was evaluated using the Pearson's chi-squared test. The independent Student's *t*-test was used to compare normally distributed parameters. Skewed parameters were compared using the Mann-Whitney *U* test. Binary logistic regression analyses were used to assess the prognostic value of FIB-5 parameters for predicting mortality. Any *P*-value < 0.05 was accepted as statistically significant.

RESULTS

Patients were categorized into two groups based on their survival status during the 1-month follow-up period: death and survivors. The mean age of the dead was 71.97 ± 10.82 years, compared to 64.35 ± 12.34 years for the survivors ($P < 0.001$). Clinical and demographic characteristics such as gender, hypertension, diabetes mellitus, hyperlipidemia, stroke, and chronic obstructive pulmonary disease were similar between the groups ($P > 0.05$). However, the prevalence of coronary artery disease was significantly higher in the death group ($P = 0.008$), as was the rate of chronic renal failure ($P = 0.004$) (Table 1).

The majority of deaths had HFrEF compared to survivors, which was statistically significant ($P = 0.0001$). The mean FIB-5 index was significantly lower in the death group (−10.46 ± 6.93) compared to the survivor group (−8.10 ± 6.67) ($P = 0.03$). Albumin levels were also significantly lower in the death (35.9 ± 5.9) compared to the survivors (39.9 ± 3.8) ($P = 0.001$). Additionally, NT-proBNP levels were significantly higher in the death group, with a *P*-value of 0.002 (Table 1).

When patients were stratified based on low and high FIB-5 index values, those with a low FIB-5 index had a higher mean age (71 ± 11.5 years) compared to those (64.1 ± 12 years) with a high FIB-5 index, which was statistically significant ($P = 0.001$). A significant difference in LVEF was also observed between these groups ($P = 0.007$). A low FIB-5 index was identified as a predictor of mortality ($P = 0.004$) (Table 2).

Univariate Cox regression analysis was conducted to examine the potential confounders of FIB-5 index, such as age, ALP, chronic renal failure, left ventricular end-diastolic diameter, left ventricular end-systolic diameter, LVEF, C-reactive protein (CRP), and NT-proBNP, as shown in Tables 1 & 2. Both the FIB-5 index and CRP level were found to be statistically significant predictors of mortality ($P = 0.01$ and $P = 0.03$, respectively). Multivariate Cox regression analysis further confirmed that the FIB-5 index and CRP were independent predictors of 1-month mortality (FIB-5 index: OR = 1.089, 95% CI: 1.022–1.160, $P = 0.009$; CRP: OR = 0.957, 95% CI: 0.934–0.981, $P = 0.001$) (Table 3). In order to enhance the clinical interpretability of our findings, we performed a multivariate logistic regression analysis including CRP and NT-proBNP alongside the FIB-5 index. The analysis revealed that both the FIB-5 index and CRP were statistically significant and independent predictor of 1-month mortality. Specifically, lower FIB-5 scores and elevated CRP levels were associated with higher short-term mortality, supporting the hypothesis that FIB-5 index reflects systemic inflammation and disease severity in acute decompensated HF (ADHF). Although NT-proBNP is a well-established biomarker in HF, it did not demonstrate independent prognostic value in the adjusted model.

Receiver operating characteristic curve analysis yielded an area under the curve of 0.609 (95% CI: 0.51–0.699) with a sensitivity of 59.6%, specificity of 63.4%, positive predictive value of 53.8%, negative predictive value of 68.8%, and a *P*-value of 0.021. Receiver operating characteristic curve analysis demonstrated that the FIB-5 index demonstrated a 60% accuracy in predicting mortality (Figure 2).

Kaplan-Meier survival analysis revealed significantly higher mortality rates among patients with low FIB-5 index values (log-rank: 7.887, $P = 0.005$) (Figure 3). Based on these findings, the FIB-5 index can be considered a reliable predictor of 1-month mortality in HF patients.

Table 1 Demographic, clinical and laboratory findings of surviving and death patients.

Variables	Death (n = 66)	Surviving (n = 89)	P-value
Age, yrs	72 ± 10.8	64.3 ± 12.3	0.001
Sex			0.84
Female	30 (45.5%)	39 (43.8%)	
Male	36 (54.5%)	50 (56.2%)	
Coronary artery disease	46 (69.7%)	43 (48.3%)	0.008
Hypertension	50 (75.8%)	59 (66.3%)	0.20
Diabetes mellitus	35 (53%)	42 (47.2%)	0.47
Smoking	27 (33.3%)	35 (34.8%)	0.7
Hyperlipidemia	33 (52%)	47 (52.8%)	0.72
Cerebrovascular disease	12 (18.2%)	7 (7.9%)	0.11
Chronic renal failure	30 (45.5%)	21 (23.6%)	0.004
Left ventricular ejection fraction			0.0001
Heart failure with reduced ejection fraction	55 (35.5%)	42 (27.1%)	
Heart failure with preserved ejection fraction	11 (7.1%)	47 (30.3%)	
Chronic obstructive pulmonary disease	11 (16.7%)	14 (15.7%)	0.11
Atrial fibrillation	26 (39.4%)	18 (20.2%)	0.001
Diuretic	42 (63.6%)	58 (65.2%)	0.22
Beta-blockers	47 (71.2%)	64 (71.9%)	0.20
Angiotensin-converting enzyme inhibitors	37 (56.1%)	49 (55.1%)	0.20
Mineralocorticoid receptor antagonist	17 (25.8%)	14 (15.7%)	0.001
FIB-5 index	−10.46 ± 6.93	−8.10 ± 6.67	0.03
Systolic blood pressure, mmHg	126 ± 18	127 ± 14	0.61
Diastolic blood pressure, mmHg	76 ± 14	79 ± 12	0.13
Heart rate, beat/min	83 ± 22	81 ± 16	0.59
Left ventricular end-diastolic diameter, mm	47.1 ± 2.5	28.3 ± 2.6	0.001
Left ventricular end-systolic diameter, mm	36.7 ± 2.1	21.1 ± 2	0.001
Alkaline phosphatase, U/μL	110 ± 9.6	90.5 ± 3.8	0.044
Pulmonary artery pressure, mmHg	39.1 ± 12.5	37.5 ± 10.0	0.38
Septal E	5.41 ± 1.3	6.25 ± 1.3	0.001
Lateral E	7.9 ± 2.4	8.7 ± 2.0	0.044
Right ventricular size, mm	10.3 ± 2.8	11.5 ± 2.5	0.012
Hemoglobin, g/L	11.87 ± 2.1	12.78 ± 1.9	0.006
White blood cell, × 10^9/L	10.14 ± 5.7	9.67 ± 5.6	0.61
Platelet count, × 10^9/L	244.2 ± 104.2	272.8 ± 93.6	0.7
Glucose, mmol/L	156.2 ± 71.8	162.9 ± 84.4	0.6
Glomerular filtration rate, mL/min per 1.73 m^2	48.3 ± 20.2	62.3 ± 24.2	0.001
Urea, mg/dL	62.9 ± 8.5	64.6 ± 10.3	0.28
Creatinine, mg/dL	1.59 ± 0.8	1.29 ± 0.6	0.014
Sodium	137.5 ± 4.2	138.4 ± 3.3	0.11
Potassium	4.57 ± 0.6	4.44 ± 0.6	0.24
Calcium	8.63 ± 0.6	8.96 ± 0.6	0.003
Albumin, g/L	35.9 ± 5.9	39.9 ± 3.8	0.001
Aspartate aminotransferase, IU/L	30.6 ± 3.4	29.4 ± 4.9	0.85
Alanine aminotransferase, U/L	28.3 ± 4.0	24.5 ± 3.2	0.49
C-reactive protein, mg/L	34.6 ± 5.9	10.2 ± 1.6	0.001
N-terminal pro-B-type natriuretic peptide, pg/mL	15,721 ± 2578	6874 ± 1445	0.002

Data are presented as means ± SD or n (%). FIB-5: fibrosis-5.

Table 2 Demographic, clinical and laboratory findings of patients according to FIB-5 index values.

Variables	Low FIB-5 index	High FIB-5 index	*P*-value
Age, yrs	71 ± 11.5	64.1 ± 12	0.001
Sex			0.28
Female	38 (48.7%)	31 (59.7%)	
Male	40 (51.3%)	46 (40.3%)	
Coronary artery disease	50 (64.1%)	39 (50.6%)	0.09
Hypertension	56 (71.8%)	53 (68.8%)	0.68
Diabetes mellitus	42 (53.8%)	35 (45.5%)	0.29
Smoking	34 (38.5%)	28 (29.9%)	0.52
Hyperlipidemia	42 (53.8%)	38 (49.4%)	0.57
Cerebrovascular disease	9 (11.5%)	10 (13%)	0.57
Chronic renal failure	34 (43.6%)	17 (22.1%)	0.004
Left ventricular ejection fraction			0.007
Heart failure with reduced ejection fraction	57 (36.8%)	40 (25.8%)	
Heart failure with preserved ejection fraction	21 (13.5%)	37 (23.9%)	
Chronic obstructive pulmonary disease	13 (16.7%)	12 (15.6%)	0.69
Atrial fibrillation	26 (33.3%)	18 (23.4%)	0.20
Diuretic	52 (66.7%)	48 (62.3%)	0.26
Beta-blockers	58 (74.4%)	53 (68.8%)	0.19
Angiotensin-converting enzyme inhibitors	44 (56.4%)	42 (54.5%)	0.35
Mineralocorticoid receptor antagonist	21 (26.9%)	10 (13%)	0.035
Mortality	42 (53.8%)	24 (31.2%)	0.004
Systolic blood pressure, mmHg	125 ± 20	128 ± 18	0.3
Diastolic blood pressure, mmHg	76 ± 13	80 ± 12	0.059
Heart rate, beat/min	80 ± 18	84 ± 19	0.2
Left ventricular end-diastolic diameter, mm	42 ± 2.6	30.6 ± 2.8	0.004
Left ventricular end-systolic diameter, mm	32.1 ± 2.1	23.2 ± 2.3	0.005
Alkaline phosphatase, U/μL	106.9 ± 8.4	91.2 ± 4.4	0.1
Pulmonary artery pressure, mmHg	39.2 ± 12.8	77.1 ± 9.1	0.2
Septal E	5.8 ± 1.4	6 ± 1.3	0.4
Lateral E	7.8 ± 2.2	8.9 ± 2.1	0.004
Right ventricular size, mm	10.6 ± 2.9	11.5 ± 2.4	0.06
Hemoglobin, g/L	11.9 ± 2	12.8 ± 1.9	0.012
White blood cell, × 10^9/L	9.5 ± 6.6	10.1 ± 4.4	0.5
Platelet count, × 10^9/L	198.3 ± 64.7	323.8 ± 87.1	0.001
Glucose, mmol/L	149.8 ± 75	170.4 ± 82.3	0.1
Glomerular filtration rate, mL/min per 1.73 m^2	53.9 ± 24.4	58.9 ± 22.6	0.19
Urea, mg/dL	61.9 ± 11	65.7 ± 7.5	0.015
Creatinine, mg/dL	1.46 ± 0.7	1.37 ± 0.8	0.48
Sodium	138 ± 3.6	138 ± 3.9	0.99
Potassium	4.5 ± 0.7	4.49 ± 0.5	0.9
Calcium	8.6 ± 0.6	8.9 ± 0.6	0.014
Albumin, g/L	37.9 ± 5.3	38.4 ± 5.1	0.5
Aspartate aminotransferase, IU/L	29.9 ± 4.7	29.9 ± 4.2	0.99
Alanine aminotransferase, U/L	19.6 ± 2.4	32.7 ± 4.3	0.009
C-reactive protein, mg/L	32.5 ± 5	21.2 ± 3.6	0.8
N-terminal pro-B-type natriuretic peptide, pg/mL	11,788 ± 2127	9068 ± 1783	0.33

Data are presented as means ± SD or *n* (%). FIB-5: fibrosis-5.

Table 3 Cox regression analysis results for the predictors of mortality.

Variables	HR (95% CI)	*P*-value
Univariate Cox regression analysis		
Age	0.942 (0.882–1.007)	0.07
Alkaline phosphatase	1.000 (0.987–1.013)	0.97
Chronic renal failure	1.246 (0.159–9.761)	0.83
Left ventricular end-diastolic diameter	1.175 (0.931–1.483)	0.17
Left ventricular end-systolic diameter	0.914 (0.747–1.119)	0.38
Left ventricular ejection fraction	1.127 (0.996–1.276)	0.057
Septal E	1.539 (0.884–2.679)	0.12
Coronary artery disease	1.816 (0.366–9.013)	0.46
Lateral E	0.825 (0.544–1.253)	0.36
Right ventricular size	1.008 (0.725–1.401)	0.96
Hemoglobin	1.071 (0.714–1.607)	0.74
Glomerular filtration rate	1.047 (0.979–1.121)	0.18
Creatinine	1.431 (0.194–10.546)	0.72
Calcium	0.309 (0.057–1.674)	0.17
Albumin	1.185 (0.971–1.446)	0.09
C-reactive protein	0.967 (0.937–0.998)	0.03
N-terminal pro-B-type natriuretic peptide	1.000 (1.000–1.000)	0.70
Atrial fibrillation	3.766 (0.714–19.870)	0.11
Mineralocorticoid receptor antagonist	3.014 (0.385–23.712)	0.29
FIB-5 index	1.241 (1.048–1.470)	0.01
Multivariate Cox regression analysis		
C-reactive protein	0.957 (0.934–0.981)	0.001
FIB-5 index	1.089 (1.022–1.160)	0.009

FIB-5: fibrosis-5.

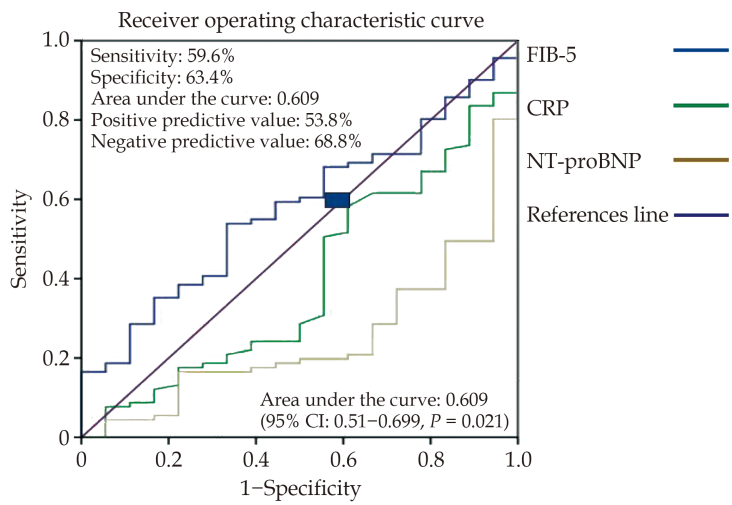

Figure 2 Receiver operating characteristic curves of FIB-5 index prediction of mortality in acute decompensated heart failure. For CRP, the area under the curve of 0.420 (95% CI: 0.288–0.552, $P = 0.255$); for NT-proBNP, the area under the curve of 0.423 (95% CI: 0.313–0.532, $P = 0.169$). CRP: C-reactive protein; FIB-5: fibrosis-5; NT-proBNP: N-terminal pro-B-type natriuretic peptide.

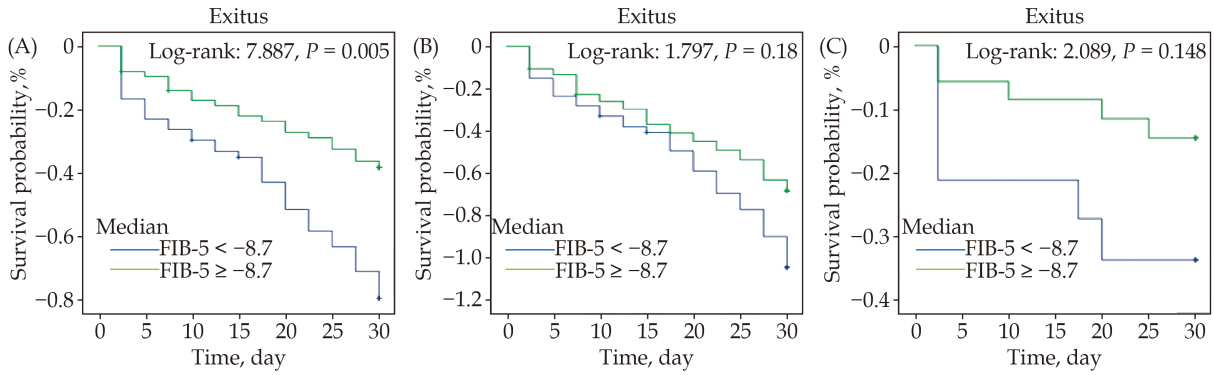

Figure 3 Kaplan-Meier survival analysis estimates for FIB-5 index in acute decompensated heart failure and a comparison of survival for FIB-5 index according to the median FIB-5 index value with long-rank test. (A): Both patients with HFrEF and those with HFpEF are included; (B): the survival curve for patients with HFrEF; and (C): the survival curve for patients with HFpEF. FIB-5: fibrosis-5; HFpEF: heart failure with preserved ejection fraction; HFrEF: heart failure with reduced ejection fraction.

DISCUSSION

This study demonstrates that the FIB-5 index can serve as a low-cost and simple prognostic marker in patients with ADHF. Lower FIB-5 values are strongly associated with increased mortality rates.[11] Although initially developed to evaluate hepatic fibrosis, the FIB-5 index encompasses parameters reflecting systemic inflammation, congestion, and organ dysfunction, making it applicable beyond hepatic conditions to scenarios involving multiorgan dysfunction.[12] Previous studies have highlighted the prognostic value of FIB-5 index in various clinical settings, supporting its potential use in ADHF for early risk stratification and optimization of personalized treatment strategies.

Inflammation is a key component of the pathophysiology of HF. The association of inflammatory markers such as CRP and pro-inflammatory cytokines (e.g., IL-6, TNF-α) with the FIB-5 index suggests that it may serve as a holistic reflection of systemic inflammation.[3,13,14] In this context, our findings that both the FIB-5 index and CRP level are independent predictors of 1-month mortality provide critical evidence supporting this relationship.

HF is one of the leading causes of chronic liver and kidney dysfunction. The role of liver dysfunction in HF pathophysiology is well-documented. Prolonged congestion in patients with HF leads to hepatocellular injury, elevated AST and ALT levels, portal hypertension, and hypersplenism, which can result in decreased platelet counts and consequently lower FIB-5 values.[15,16] Additionally, reduced serum albumin levels may reflect malnutrition and inflammatory processes associated with HF. van Deursen, *et al.*[13] in their Pre-RELAX AHF study, reported that elevated ALP levels were independently associated with 60-day mortality and rehospitalization in 234 acute HF patients. Our study shows that lower FIB-5 values reflect the severity of these pathological processes, thereby correlating with poor prognosis. Similarly, Shibata, *et al.*[17] reported that the FIB-4 index is associated with mortality in acute HF patients, although the TOPCAT trial found no association between the FIB-4 index and adverse cardiac events in HFpEF patients.[17–21] This discrepancy suggests that the FIB-5 index, as a broader marker of systemic fibrosis and inflammation, may be a superior prognostic tool compared to the FIB-4 index.[22,23]

There is a significant relationship between hepatorenal syndrome and HF. Systemic congestion due to HF may increase hepatic venous pressure and lead to liver dysfunction. This situation becomes especially evident in patients with right HF or advanced HF. FIB-5 index is considered as an indicator of systemic fibrosis and inflammation and may be related to liver dysfunction. Since the clinical picture of hepatorenal syndrome is usually characterized by hepatic congestion and renal dysfunction, low FIB-5 values may be a reflection of these systemic effects. Therefore, it should be considered that the low FIB-5 index in our study may reflect not only liver fibrosis but also hepatorenal syndrome.[14]

Moreover, Maeda, *et al.*[6] demonstrated that lower FIB-5 values were associated with rehospitalization and cardiac death in patients with acute HF, although their study was limited to patients with HFpEF. One of the key strengths of our study was its inclusion of both patients with HFrEF and HFpEF, providing a more comprehensive analysis compared to the existing literature. The association between lower FIB-5 values and poor prognosis underscores the need for further investigation of novel biomark-

ers in ADHF. While biomarkers such as CRP, NT-proBNP, and growth differentiation factor-15 have established prognostic value in HF, the low cost and widespread availability of the FIB-5 index make it a practical alternative.[6,13,17] In our study, patients with lower FIB-5 values exhibited significantly higher mortality rates. Kaplan-Meier survival analysis revealed shorter survival times in patients with low FIB-5 values (log-rank: 7.887, $P = 0.005$). Receiver operating characteristic curve analysis demonstrated that the FIB-5 index predicted mortality with an accuracy of 60%. This level of accuracy suggests that while the FIB-5 index may not be a standalone prognostic tool, its integration with other biomarkers and clinical parameters could enhance risk stratification and guide management strategies in patients with ADHF.

LIMITATIONS

This study has several limitations. Firstly, its retrospective design prevents the establishment of definitive cause-and-effect relationships. Secondly, being a single-center study limits the generalizability of the findings. Studies involving data from diverse geographical regions and larger patient populations are needed to better understand the prognostic value of the FIB-5 index. The sample size of 155 patients restricted our ability to perform the subgroup analyses. Thirdly, this study focused only on the baseline values of the FIB-5 index, without evaluating its temporal changes or their association with mortality or treatment response. Dynamic follow-up data could provide a clearer understanding of the role of FIB-5 index in clinical decision-making. Fourthly, the FIB-5 index was not compared with other liver fibrosis markers (e.g., FIB-4, aspartate aminotransferase-to-platelet ratio index) or established HF biomarkers (e.g., NT-proBNP, growth differentiation factor-15), limiting the opportunity to assess its advantages and limitations relative to these markers. The patients' initial FIB-5 values were examined. However, the FIB-5 values could not be examined during follow-up to monitor changes over time and response to treatment and to evaluate the index's potential as a dynamic prognostic tool. This situation is among our limitations. One of the limitations of this study is the lack of data on the duration of HF in patients. This lack of information may limit the interpretation of the relationship between the duration of HF and FIB-5 index value. Inclusion of this parameter in future studies will contribute to a better understanding of its impact on prognostic outcomes. Last but not least, this study focused solely on the 1-month mortality. Future studies with longer follow-up periods are required to evaluate the role of the FIB-5 index in long-term prognosis.

CONCLUSIONS

This study demonstrates that the FIB-5 index is an independent prognostic marker for predicting 1-month mortality in patients with ADHF. The FIB-5 index stands out as a tool not only for evaluating hepatic fibrosis but also for reflecting systemic inflammation, congestion, and organ dysfunction associated with HF. The association of low FIB-5 values with liver dysfunction, malnutrition, and increased inflammation highlights its significance in assessing systemic effects. In this context, the FIB-5 index emerges as a promising risk assessment tool that can be easily integrated into clinical practice due to its low cost, widespread availability, and simplicity of calculation. Our study contributes significantly to the limited body of research evaluating the applicability of the FIB-5 index in both HFrEF and HFpEF patients. Furthermore, this study focuses solely on the baseline values of the FIB-5 index. Future research should explore the potential of this parameter to reflect temporal changes and treatment responses, which could enhance its utility as a dynamic prognostic tool in the management of HF.

ACKNOWLEDGMENTS

All authors had no conflicts of interest to disclose. The authors thank Necmettin Erbakan University Faculty of Medicine Cardiology Clinics for their support.

REFERENCES

[1] 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: 3599–3726.
[2] González A, Ravassa S, López B, *et al*. Myocardial remodeling in hypertension. *Hypertension* 2018; 72: 549–558.
[3] Paulus WJ, Zile MR. From systemic inflammation to myocardial fibrosis: the heart failure with preserved ejection fraction paradigm revisited. *Circ Res* 2021; 128: 1451–1467.
[4] Yoshihisa A, Sato Y, Yokokawa T, *et al*. Liver fibrosis score predicts mortality in heart failure patients with preserved ejection fraction. *ESC Heart Fail* 2018; 5: 262–270.
[5] Matsue Y, Kagiyama N, Yamaguchi T, *et al*. Clinical and prognostic values of ALBI score in patient with acute heart failure. *Heart Lung Circ* 2020; 29: 1328–1337.
[6] Maeda D, Kagiyama N, Jujo K, *et al*. Aspartate aminotransferase to alanine aminotransferase ratio is associated with frailty and mortality in older patients with heart failure. *Sci Rep* 2021; 11: 11957.

[7] Yan Z, Liu Y, Li W, *et al*. Liver fibrosis scores and prognosis in patients with cardiovascular diseases: a systematic review and meta-analysis. *Eur J Clin Invest* 2022; 52: e13855.
[8] Crespo-Leiro MG, Metra M, Lund LH, *et al*. Advanced heart failure: a position statement of the Heart Failure Association of the European Society of Cardiology. *Eur J Heart Fail* 2018; 20: 1505–1535.
[9] Tatar S, İcli A, Arıbaş A, *et al*. Diastolic strain parameters are associated with short term mortality and rehospitalization in patients with advanced heart failure. *Arq Bras Cardiol* 2024; 121: e20230670.
[10] Yavuz YE, Soylu A, Gürbüz AS. The relationship of systemic and pulmonary arterial parameters with HFpEF scores (H_2FPEF, HFA-PEFF) and diastolic dysfunction parameters in heart failure patients with preserved ejection fraction. *J Clin Ultrasound* 2024; 52: 39–50.
[11] Chen S, Zhu B, Luo Z, *et al*. The fibrosis-5 index predicts major adverse cardiovascular events in patients with ST-segment elevation myocardial infarction undergoing percutaneous coronary intervention. *Angiology* 2024; 31: 331972-41231051.
[12] He HM, He C, You ZB, *et al*. Non-invasive liver fibrosis scores are associated with contrast-associated acute kidney injury in patients undergoing elective percutaneous coronary intervention. *Angiology* 2023; 74: 333–343.
[13] van Deursen VM, Edwards C, Cotter G, *et al*. Liver function, in-hospital, and post-discharge clinical outcome in patients with acute heart failure: results from the relaxin for the treatment of patients with acute heart failure study. *J Card Fail* 2014; 20: 407–413.
[14] Wang Z, Li G, Huang R, *et al*. Prognostic value of fibrosis-5 index combined with C-reactive protein in patient with acute decompensated heart failure. *BMC Cardiovasc Disord* 2023; 23: 492.
[15] Nakashima M, Sakuragi S, Miyoshi T, *et al*. Fibrosis-4 index reflects right ventricular function and prognosis in heart failure with preserved ejection fraction. *ESC Heart Fail* 2021; 8: 2240–2247.
[16] Attallah AM, Shiha GE, Omran MM, *et al*. A discriminant score based on four routine laboratory blood tests for accurate diagnosis of severe fibrosis and/or liver cirrhosis in Egyptian patients with chronic hepatitis C. *Hepatol Res* 2006; 34: 163–169.
[17] Shibata N, Kondo T, Kazama S, *et al*. Impact of predictive value of fibrosis-4 indexs in patients hospitalized for acute heart failure. *Int J Cardiol* 2021; 324: 90–95.
[18] Maeda D, Sakane K, Ito T, *et al*. Fibrosis-4 index reflects right-sided filling pressure in patients with heart failure. *Heart Vessels* 2020; 35: 376–383.
[19] Peters AE, Pandey A, Ayers C, *et al*. Association of liver fibrosis risk scores with clinical outcomes in patients with heart failure with preserved ejection fraction: findings from TOPCAT. *ESC Heart Fail* 2021; 8: 842–848.
[20] Maeda D, Kanzaki Y, Sakane K, *et al*. Prognostic impact of a reduction in the fibrosis-4 index among patient hospitalized with acute heart failure. *Heart Vessels* 2023; 38: 1235–1243.
[21] Kawahira M, Tamaki S, Yamada T, *et al*. Prognostic value of impaired hepato-renal function and liver fibrosis in patients admitted for acute heart failure. *ESC Heart Fail* 2021; 8: 1274–1283.
[22] Maeda D, Kanzaki Y, Sakane K, *et al*. Prognostic value of the liver fibrosis marker fibrosis-5 index in patients with acute heart failure. *ESC Heart Fail* 2022; 9: 1380–1387.
[23] Nakashima M, Miyoshi T, Tanakaya M, *et al*. Prognostic value of the liver fibrosis marker fibrosis-5 index in patients with severe isolated tricuspid regurgitation: comparison with fibrosis-4 index. *Heart Vessels* 2023; 38: 1181–1189.

Please cite this article as: Tatar S, Yavuz YE, Gedik HN, Içli A, Akilli H. The prognostic significance of the fibrosis-5 index in patients with acute decompensated heart failure. *J Geriatr Cardiol* 2025; 22(6): 587–595. DOI: 10.26599/1671-5411.2025.06.002