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ORIGINAL RESEARCH

Prognostic Significance of C-Reactive Protein to Albumin Ratio in Predicting Long-Term Mortality Among Patients with Acute Heart Failure

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Background and Aims: Acute heart failure (AHF) is a prevalent cardiovascular condition. C-reactive protein (CRP) and low albumin levels are established significant prognostic markers in AHF. This study aimed to evaluate the prognostic value of the C-reactive protein to albumin ratio (CAR) for predicting long-term all-cause mortality in patients with AHF. In this study, AHF was defined as either newly diagnosed decompensated heart failure (HF) or an exacerbation of chronic compensated HF requiring hospitalization. The enrolled AHF patients encompassed the entire spectrum of ejection fractions.

Methods: This single-center, retrospective study enrolled a total of 227 patients. Participants were categorized into two groups: a high CAR group (n = 96) and a low CAR group (n = 131). The study endpoints were all-cause mortality, cardiac mortality, and non-cardiac mortality after admission for AHF.

Results: The follow-up period was 46 months. Patients with an elevated CAR exhibited significantly higher rates of all-cause mortality and cardiac mortality compared to those with lower CAR levels (49% vs 13%, $P < 0.001$ and 39.6% vs 7.6%, $P < 0.001$, respectively). The areas under the curve (AUC) for predicting all-cause mortality were 0.78 (95% CI: 0.713–0.841; $p < 0.001$) for CAR and 0.76 (95% CI: 0.695–0.826; $p < 0.001$) for NT-proBNP. Kaplan–Meier survival analysis demonstrated a significantly increased risk of all-cause mortality in patients with elevated CAR or NT-proBNP levels (Log rank test, $p < 0.001$ for both). Multivariate Cox regression analysis identified CAR as an independent predictor of all-cause mortality (hazard ratio [HR]: 1.043; 95% CI: 1.011–1.047; $p = 0.008$).

Conclusion: The CAR is an independent predictor of long-term all-cause mortality in patients with AHF.

Keywords: acute heart failure, albumin, C-reactive protein, C-reactive protein to albumin ratio, mortality

Introduction

Acute heart failure (AHF) is a prevalent cardiovascular condition associated with high morbidity and mortality.[1] Various clinical indicators are utilized to predict outcomes in patients with AHF. C-reactive protein (CRP) and hypoalbuminemia have been established as significant prognostic markers in this population.[2,3] The C-reactive protein to albumin ratio (CAR) has been demonstrated to reflect inflammatory status more accurately than either CRP or albumin alone, thereby offering superior prognostic value in patients with acute medical conditions and malignancies.[4–6] Emerging evidence indicates an association between CAR and cardiovascular diseases, including its role in predicting the severity and outcomes of coronary artery disease (CAD).[7,8] Notably, CAR has been shown to predict all-cause in-hospital mortality in AHF,[9] and is independently associated with long-term mortality in chronic heart failure (HF).[10–12] Therefore, this study aimed to evaluate the prognostic significance of CAR for predicting long-term all-cause mortality in patients with AHF. CAR is not only readily obtainable, practical, and cost-effective but may also provide novel insights into HF treatment strategies.

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Methods

Patients

This retrospective study initially enrolled 348 adult patients (aged ≥18 years) who were diagnosed with AHF and admitted to the Department of Cardiology at the Eastern Medical Branch of the PLA General Hospital, Beijing, China, between January 2018 and December 2019. The diagnosis of AHF was based on modified Framingham criteria and was defined as either newly diagnosed decompensated HF or an exacerbation of chronic compensated HF requiring hospitalization. The included AHF patients represented the entire spectrum of ejection fractions. We excluded 105 patients due to missing CRP or albumin data, acute coronary syndrome, acute or chronic infection, autoimmune diseases, or cancer. An additional 6 patients were excluded due to loss to follow-up, resulting in a final cohort of 227 patients for analysis. These patients were stratified into a high CAR group (n = 96) and a low CAR group (n = 131) based on the optimal cutoff value (Figure 1). The study was conducted in accordance with the Declaration of Helsinki. The study protocol was reviewed and approved by the Institutional Review Board of the Seventh Medical Center of Chinese PLA General Hospital (approval number: S2025-077-01).

Data Collection

Clinical and laboratory parameters were retrieved from the hospital's electronic medical records. Data collected within the first 24 hours following admission to either the emergency department or the cardiology ward were included in the analysis. Serum CRP and albumin levels were measured using an automated biochemical analyzer (Mindray BS-2800M, China). The CAR was calculated using the following formula: CAR = [CRP (mg/L)/albumin (g/L)] × 100.

Follow-Up and Study Endpoints

The follow-up period for this study was 46 months. The primary study endpoints were all-cause mortality, cardiac mortality, and non-cardiac mortality following admission for AHF. Cardiac mortality was defined as death attributable to fatal myocardial infarction, sudden cardiac death, mortality following cardiovascular intervention, or other direct cardiac causes. All other deaths were classified as non-cardiac mortality. Follow-up data were collected via structured telephone interviews with participants or their family members. For deceased patients who did not die in our hospital, death certificates were reviewed to ascertain and verify the cause of death (cardiac vs non-cardiac).

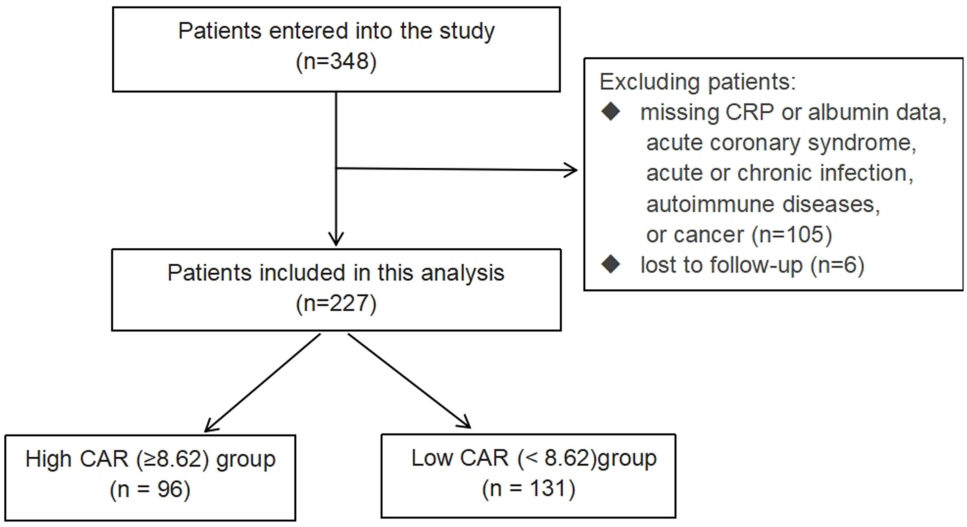

Figure 1 Flowchart of the study patients. CRP, C-reactive protein; CAR, C-reactive protein to albumin ratio.

Statistical Analysis

Patients were stratified into two groups based on their CAR levels: a low CAR group (<8.62) and a high CAR group (≥8.62). This cut-off value was determined by receiver operating characteristic (ROC) curve analysis for predicting all-cause mortality. The optimal threshold was identified by maximizing the Youden's index (Youden's index = sensitivity +specificity-1). Data normality was assessed using the Kolmogorov–Smirnov test. Continuous variables with a normal distribution are expressed as mean ± standard deviation (SD), while skewed variables are presented as median (interquartile range, IQR). Categorical variables are expressed as numbers and percentages (n, %). Comparisons between the two groups were performed using Student's *t*-test for normally distributed continuous variables, the Mann–Whitney-*U* test for non-normally distributed continuous variables, and the chi-square (χ^2) test for categorical variables. Survival curves were constructed using the Kaplan–Meier method, and differences between groups were compared with the Log rank test. Univariate Cox proportional hazards regression analysis was performed to identify potential risk factors associated with all-cause mortality in AHF. Variables with a p-value <0.05 in the univariate analysis were subsequently included in a multivariate Cox proportional hazards regression model to identify independent predictors of all-cause mortality. All statistical analyses were performed using SPSS software (version 26.0; IBM Corp., Armonk, NY, USA). A two-sided p-value <0.05 was considered statistically significant. Assess the robustness of the optimal CAR cut-off value through bootstrap sensitivity analysis.

Results

Baseline Characteristics

The baseline clinical characteristics of the study participants, stratified by CAR levels, are summarized in Table 1. Compared to patients in the low CAR group, those in the high CAR group were significantly older (median age: 77 vs 72 years, p = 0.006) and had a higher proportion of males (62.9% vs 48.9%, p = 0.041). CAD was also significantly more prevalent in the high CAR group (66.7% vs 48.1%, p = 0.005). No significant intergroup differences were observed in the prevalence of the following comorbidities: dilated cardiomyopathy, hypertension, hyperlipidemia, diabetes mellitus, atrial fibrillation, valvular heart disease, chronic pulmonary disease, stroke or transient ischemic attack (TIA), and smoking history. Patients in the high CAR group exhibited significantly higher values for body mass index (BMI), heart rate, systolic blood pressure, white blood cell (WBC) count, N-terminal pro-brain natriuretic peptide (NT-proBNP), and CRP.

Table 1 The Baseline Clinical Features of Participants According to C-Reactive Protein to Albumin Ratio

Variable	Low CAR Group (n = 131)	High CAR Group (n = 96)	p-value
Age, years	72(62,79)	77(67,85)	0.006
Male gender, n (%)	64(48.9)	60(62.5)	0.041
History, n (%)			
Coronary artery disease	63(48.1)	64(66.7)	0.005
Dilated cardiomyopathy	13(9.9)	10(10.4)	0.903
Hypertension	89(67.9)	70(72.9)	0.419
Hyperlipidemia	59(45.0)	38(39.4)	0.412
Diabetes	34(26.0)	34(35.4)	0.124
Atrial fibrillation	43(32.8)	39(40.6)	0.227
Valvular heart disease	18(13.7)	12(12.5)	0.785
Chronic pulmonary disease	18(13.7)	15(15.6)	0.691
Stroke or TIA	34(26.0)	38(39.6)	0.029
Smoking	32(24.4)	25(26.0)	0.782

(*Continued*)

Table 1 (Continued).

Variable	Low CAR Group (n = 131)	High CAR Group (n = 96)	p-value
Clinical profiles on admission			
BMI, kg/m2	23.43(20.94,27.32)	24.53(21.61,29.48)	0.049
Heart rate, beat/min	86(73,101)	93.5(78.5,108)	0.041
SBP, mmHg	131.74 ± 23.24	141.18 ± 22.13	0.002
Creatinine, umol/L	85(68,101)	86(73,101)	0.26
Sodium, mmol/L	139(138,142)	138(134,140)	<0.001
Potassium, mmol/L	3.87 ± 0.44	3.82 ± 0.43	0.446
WBC, 109/L	6.29 ± 1.50	6.86 ± 1.57	0.005
Hemoglobin, g/L	135.89 ± 14.11	129.38 ± 15.79	0.001
NT-proBNP, ng/L	2950(1490,4690)	6075(4282.5,8382.5)	<0.001
CRP, mg/L	1.68(0.99,2.36)	5.13(4.22,7.04)	<0.001
Albumin, g/L	39.7(35.6,41.4)	37.15(34.33,40.7)	0.006
CAR × 100	4.36(2.74,6.17)	13.55(11.18,18.74)	<0.001
LVEF, %	56(45,62)	48(37.25,60.00)	0.007
NYHA III–IV, n (%)	105(80.2)	88(91.7)	0.016
Medication, n (%)			
ACEI/ARB*	61(46.6)	38(39.6)	0.295
Beta-blocker*	105(80.2)	70(72.9)	0.200
Aldosterone antagonist*	121(92.4)	90(93.8)	0.687
Diuretic	125(95.4)	93(96.9)	0.579
Digitalis	65(49.6)	58(60.4)	0.107
Anti-platelet/Anticoagulant	128(97.7)	93(96.9)	0.698
Endpoint			
All-cause mortality	17(13)	47(49.0)	<0.001
Cardiac mortality	10(7.6)	38(39.6)	<0.001
Noncardiac mortality	7(5.3)	9(9.4)	0.241

Notes: *Dosage adjustments specified to achieve target or maximum tolerated doses.
Abbreviations: TIA,transient ischaemic attack; BMI, body mass index; SBP,systolic blood pressure; WBC, white blood cell count; NT-pro-BNP, N-terminal pro-brain natriuretic peptide; CRP, C-reactive protein; CAR, C-reactive protein to albumin ratio; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association; ACEI, angiotensinogen converting enzyme inhibitor; ARB, angiotensinogen receptor blocker.

Conversely, they had significantly lower serum sodium levels, hemoglobin levels, albumin levels, and left ventricular ejection fraction (LVEF). The high CAR group also demonstrated a significantly higher prevalence of New York Heart Association (NYHA) functional class III–IV HF. No significant differences were noted between the two groups in creatinine levels, potassium levels, or the use of medications.

Outcomes

During follow-up, 64 (28%) patients died, including 48 (21%) from cardiac causes and 16 (7%) from non-cardiac causes. The high CAR group had significantly higher all-cause and cardiac mortality rates compared to the low CAR group (49% vs 13%, $p < 0.001$; 39.6% vs 7.6%, $p < 0.001$, respectively). Non-cardiac mortality rates did not differ. Figure 2 illustrates the ROC curves for CAR and NT-proBNP in forecasting all-cause mortality (Figure 2A), cardiac mortality (Figure 2B) and non-cardiac mortality (Figure 2C). Hosmer-Limax test for goodness of fit was applied to the ROC curves of CAR ($p = 0.594$) and NT-proBNP ($p = 0.077$) in forecasting all-cause mortality, showed that the scoring model worked well. In forecasting all-cause mortality, the areas under the curve (AUC) for CAR and NT-proBNP were 0.78 (95% CI: 0.713–0.841, $p < 0.001$) and 0.76 (95% CI: 0.695–0.826, $p < 0.001$). DeLong's test $P = 0.649$. In forecasting cardiac mortality, the AUC for CAR and NT-proBNP were 0.80 (95% CI: 0.732–0.870, $p < 0.001$) and 0.74 (95% CI: 0.659–0.810, $p < 0.001$). DeLong's test $P = 0.083$. In forecasting non-cardiac mortality, the AUC for CAR and NT-

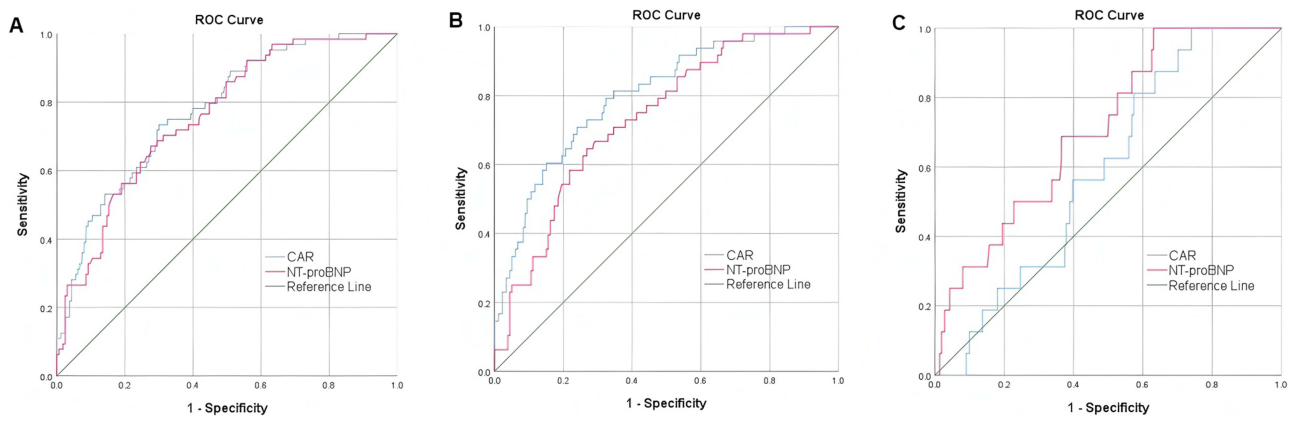

Figure 2 The receiver operating characteristic curves for CAR and NT-proBNP in forecasting all-cause mortality (**A**), cardiac mortality (**B**) and cardiac mortality (**C**).
Abbreviations: CAR, C-reactive protein to albumin ratio; NT-proBNP, N-terminal pro-brain natriuretic peptide.

proBNP were 0.59 (95% CI: 0.478–0.702, p = 0.230) and 0.71 (95% CI: 0.593–0.823, p = 0.006). DeLong's test P = 0.129. The results showed that CAR and NT-proBNP had good predictive power for all-cause mortality and cardiac mortality, and there was no difference. CAR had poor predictive power for non-cardiac mortality. A formal test of incremental predictive performance for integrated discriminant improvement (IDI) was conducted. The new model (NT-proBNP+CAR) demonstrated statistically superior performance compared to the previous model (NT-proBNP), with an improvement in overall discrimination IDI reaching 0.0955 (95% CI: 0.049–0.1447; p < 0.0001). This signifies a 9.55% enhancement in overall discrimination, statistically significantly better calibration accuracy (p < 0.0001), representing clinically significant improvements in risk stratification of all-cause mortality. Figure 3 was the risk stratification chart showed the incidence of events corresponding to different CAR quarters. The results showed that the higher the CAR, the higher the all-cause mortality rate.

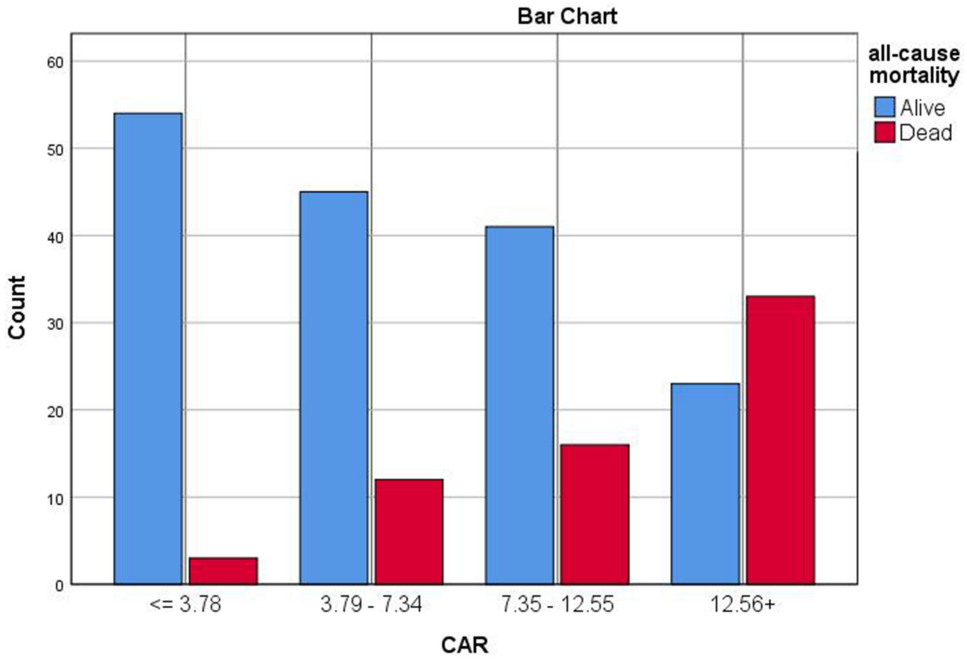

Figure 3 The risk stratification chart shows the incidence of events corresponding to different CAR quarters.
Abbreviation: CAR, C-reactive protein to albumin ratio.

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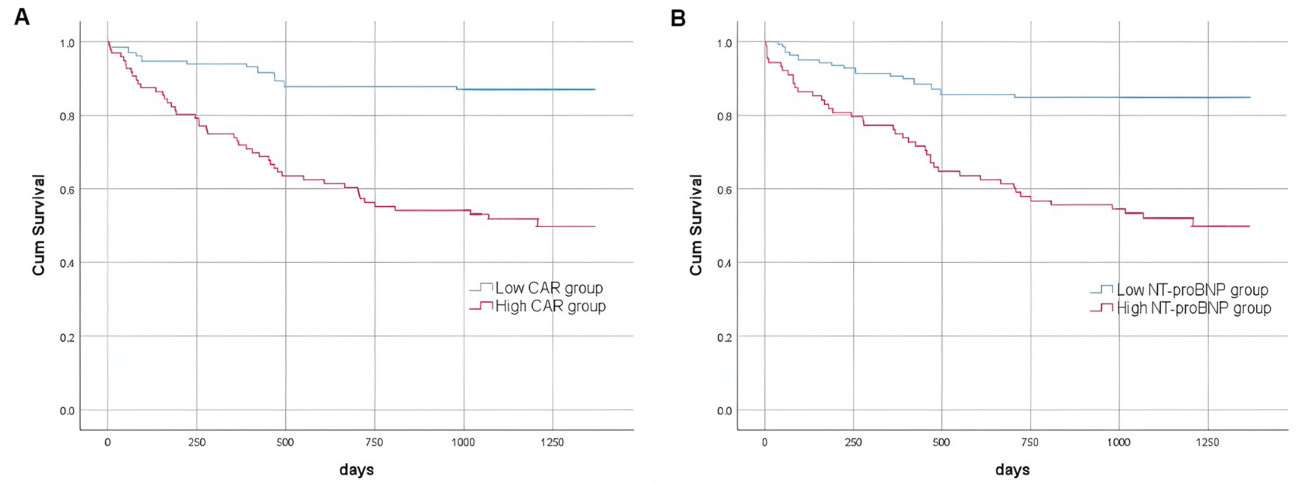

Figure 4 Kaplan–Meier survival curves for all-cause mortality according to CAR (**A**) and NT-proBNP (**B**).
Abbreviations: CAR, C-reactive protein to albumin ratio; NT-proBNP, N-terminal pro-brain natriuretic peptide.

The optimal cut-off values, determined by ROC curve analysis, were 8.62 for CAR (sensitivity: 73.4%; specificity: 69.9%) and 5120 ng/L for NT-proBNP (sensitivity: 67.2%; specificity: 72.4%). Bootstrap validation (n = 1000) confirmed the stability of the optimal CAR cut-off value, showing a mean of 9.139 (SD ± 1.327), 95% CI [7.490, 12.030], and coefficient of variation of 14.5%. Using these cut-off points, patients were further stratified into low NT-proBNP (<5120 ng/L) and high NT-proBNP (≥5120 ng/L) groups. Kaplan–Meier survival analysis demonstrated a significantly increased risk of all-cause mortality in patients with elevated CAR (≥8.62) or elevated NT-proBNP (≥5120 ng/L) levels during the follow-up period (Log rank test, $P < 0.001$ for both; Figure 4A and B). Univariate and multivariate Cox regression analyses were performed to identify predictors of all-cause mortality; the results are summarized in Table 2. Univariate analysis indicated that the following variables were significantly associated with all-cause mortality: age, valvular heart disease, chronic pulmonary disease, creatinine level, hemoglobin level, NT-proBNP level, CRP level, albumin level, CAR, LVEF, NYHA functional class III–IV, and the use of ACE inhibitors/angiotensin receptor blockers (ACEi/ARB) and beta-blockers.

Table 2 Univariate and Multivariate Cox Regression Analyses for Predictors of All-Cause Mortality

	Univariate Analysis		Multivariate Analysis	
	p Value	**HR (95% CI)**	**p Value**	**HR (95% CI)**
Age, years	<0.001	1.046 (1.021–1.072)	<0.001	1.053 (1.024–1.084)
Valvular heart disease	0.026	0.509 (0.281–0.921)	0.003	0.392 (0.212–0.723)
Chronic pulmonary disease	0.002	0.405 (0.230–0.714)	<0.001	0.228 (0.116–0.451)
Creatinine	<0.001	1.025 (1.014–1.036)		
Hemoglobin	<0.001	0.961 (0.945–0.977)		
NT-proBNP/100	<0.001	1.013 (1.009–1.017)	0.032	1.006 (1.001–1.011)
CRP	<0.001	1.264 (1.172–1.363)		
Albumin	<0.001	0.809 (0.756–0.866)	0.001	0.875 (0.809–0.947)
CAR × 100	<0.001	1.095 (1.068–1.123)	0.008	1.043(1.011–1.077)
LVEF, %	<0.001	0.958 (0.940–0.976)	<0.001	0.942 (0.917–0.968)
NYHA III–IV, n (%)	0.020	0.037(0.002–0.595)		
ACEI/ARB	<0.001	2.742 (1.556–4.830)		
Beta-blocker	0.005	2.106 (1.257–3.530)	0.008	2.167(1.225–3.833)

Abbreviations: NT-pro-BNP, N-terminal pro-brain natriuretic peptide; CRP, C-reactive protein; CAR, C-reactive protein to albumin ratio; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association; ACEI, angiotensinogen converting enzyme inhibitor; ARB, angiotensinogen receptor blocker.

Variables with a P-value <0.05 in the univariate analysis were incorporated into the multivariate Cox regression model. The final multivariate model, which included age, valvular heart disease, chronic pulmonary disease, NT-proBNP, albumin, LVEF, and use of beta-blockers, identified CAR as an independent predictor of all-cause mortality (hazard ratio [HR]: 1.043; 95% confidence interval [CI]: 1.011–1.047; P = 0.008).

Discussion

This study demonstrated that patients with elevated CAR levels had significantly higher rates of both all-cause mortality and cardiac mortality compared to those with lower CAR levels. Furthermore, a positive correlation was observed between increasing CAR levels and rising long-term all-cause mortality rates in AHF patients. Multivariate Cox regression analysis confirmed that an elevated CAR is an independent predictor of long-term all-cause mortality in patients with AHF.

Systemic inflammation is a well-established central pathophysiological component of both acute and chronic HF.[13] Since its identification, CRP has been recognized as a key inflammatory biomarker elevated in patients with HF.[14] Large-scale clinical studies have consistently demonstrated that elevated CRP levels predict morbidity and mortality in individuals with diagnosed HF.[2,15,16] Although the precise mechanisms driving this chronic immune activation in HF remain incompletely understood, potential contributors include activation of monocyte-macrophage and lymphocyte systems, along with involvement of the renin-angiotensin-aldosterone system and sympathetic nervous system.[17–20]

In both acute and chronic HF, albumin has been established as a critical factor influencing survival, with numerous studies identifying it as a significant prognostic indicator for clinical outcomes.[3,21–23] The development of hypoalbuminemia in HF patients is primarily attributed to reduced albumin synthesis and increased protein loss. These pathological changes may result from multiple factors, including hemodilution, chronic inflammation, hepatic congestion, malnutrition, cachexia secondary to volume overload, as well as proteinuria or intestinal disorders.[24] In HF, decreased albumin levels promote and exacerbate congestion by reducing intravascular colloidal osmotic pressure, while simultaneously increasing oxidative stress, systemic inflammation, and susceptibility to infections.[25–27]

The CAR is an integrative systemic inflammatory index that incorporates both CRP and albumin levels. This novel biomarker has garnered significant attention for its ability to predict adverse clinical outcomes, attributed to its accessibility, practicality, and cost-effectiveness. Recent investigations have demonstrated the prognostic utility of CAR across various clinical contexts, including acute medical conditions,[4,5] malignancies,[6] stable angina pectoris,[7] and acute coronary syndrome.[8] Emerging evidence indicates that CAR may also serve as a significant prognostic indicator in HF. Several studies have specifically examined the prognostic value of CAR in patients with chronic HF. Çinier et al[10] demonstrated that elevated CAR levels were associated with an increased risk of long-term all-cause mortality in patients with heart failure with reduced ejection fraction (HFrEF) who underwent implantable cardioverter-defibrillator (ICD) therapy. In a smaller prospective study of 77 patients with LVEF <50%, a higher CAR was associated with increased hospitalization rates, though no significant effect on mortality was observed.[11] Additionally, Tanık et al[12] reported that CAR was independently associated with long-term mortality in patients with chronic HFrEF over a median follow-up period of 30 months.

Evidence regarding the association between the CAR and clinical outcomes in AHF remains limited. Sonsoz et al[9] reported that admission CAR was associated with higher all-cause in-hospital mortality in patients hospitalized with acute decompensated HF. To the best of our knowledge, our study is the first to investigate the relationship between CAR and long-term clinical outcomes in patients with AHF. In our cohort, the median follow-up duration was 1074 days. Patients in the high CAR group demonstrated significantly higher all-cause mortality rates compared to those in the low CAR group (49% vs 13%, P < 0.001). The AUC for predicting all-cause mortality was 0.78 for CAR and 0.76 for NT-proBNP. DeLong's test revealed no statistically significant difference in the predictive performance between CAR and NT-proBNP for all-cause mortality (P = 0.649). We further assessed the incremental predictive value using the integrated discrimination improvement (IDI) method. The combined model (NT-proBNP + CAR) demonstrated a statistically significant improvement in risk stratification for all-cause mortality compared to the NT-proBNP-alone model, with an IDI of 0.0955 (95% CI: 0.0490–0.1447; P < 0.0001). Risk stratification analysis across CAR quartiles revealed a dose-response relationship, with progressively higher all-cause mortality rates observed in higher CAR quartiles. Kaplan–

Meier survival curves confirmed a significantly increased risk of all-cause mortality in patients with elevated CAR (≥8.62) or NT-proBNP (≥5120 ng/L) levels during follow-up (Log rank test, $P < 0.001$ for both). Both univariate and multivariate Cox regression analyses identified elevated CAR as an independent predictor of long-term all-cause mortality in AHF (multivariate analysis: HR: 1.043; 95% confidence interval [CI]: 1.011–1.047; $P = 0.008$). These findings collectively underscore the significant predictive value of CAR in the risk stratification of AHF patients.

Our findings confirm a positive correlation between increasing CAR levels and rising long-term all-cause mortality in AHF patients. This suggests that therapeutic strategies aimed at reducing CAR may potentially improve clinical outcomes in AHF. CAR reduction could be achieved through two complementary approaches: decreasing CRP levels (reflecting reduced inflammation) and increasing albumin concentrations. Emerging evidence indicates that specific HF therapies may modulate inflammation. Angiotensin Receptor-Neprilysin Inhibitor (ARNI) and Sodium-Glucose Cotransporter-2 Inhibitor (SGLT2i) therapies have been associated with reduced CRP levels, suggesting that anti-inflammatory mechanisms may contribute to their prognostic benefits in HF.[28–30] This supports the investigation of other anti-inflammatory therapeutic strategies in HF management. However, the approach to addressing hypoalbuminemia requires careful consideration. Current evidence suggests that albumin infusion may increase hospitalization mortality in HF patients with low serum albumin levels,[31,32] potentially due to fluid retention and increased cardiac workload. Therefore, alternative strategies for improving nutritional status and albumin synthesis, such as high-quality protein diets or oral protein supplementation, warrant further investigation to determine their potential benefits in AHF prognosis.

Limitations

This study has several limitations that should be considered when interpreting the results. First, this was a single-center, retrospective study with a limited sample size, which may affect the generalizability of our findings. Second, CRP and albumin measurements were not performed in all AHF patients as they were obtained at the discretion of treating physicians, potentially introducing selection bias. Third, although we adjusted for numerous confounding variables in our multivariate analysis, the potential influence of unmeasured or residual confounding factors cannot be completely excluded.

Additionally, our study did not categorize patients according to whether they underwent cardiac catheterization, which might represent a potential confounding variable. Furthermore, we did not classify AHF patients according to current HF subtypes—HFrEF, heart failure with mildly reduced ejection fraction (HFmrEF), and heart failure with preserved ejection fraction (HFpEF)—which represents another limitation of our study. Future studies with specific HF phenotype classifications are warranted to explore potential differential effects of CAR across these distinct pathophysiological entities.

Given these limitations, our findings should be considered hypothesis-generating. Larger-scale, multicenter, prospective cohort studies are needed to validate the association between CAR and clinical outcomes in AHF patients and to establish its potential clinical utility across different HF phenotypes.

Conclusions

In conclusion, our study demonstrates that the CAR serves as an independent predictor of long-term all-cause mortality in patients with AHF. These findings support the utility of this simple, inexpensive, and readily available biomarker for risk stratification in AHF populations. The accessibility of CAR measurement may be particularly valuable for prognostic assessment in resource-limited settings. Furthermore, our results suggest that therapeutic strategies aimed at reducing CAR—potentially through anti-inflammatory interventions or nutritional support—may represent a promising approach to improving outcomes in HF patients. This perspective provides new insights that may inform future treatment strategies for HF management.

Abbreviations

AHF, acute heart failure; CRP, C-reactive protein; CAR, C-reactive protein to albumin ratio; HF, heart failure; HR, hazard ratio; CAD, coronary artery disease; ROC, receiver operating characteristic; SD, standard deviation; NT-proBNP,

N-terminal pro-brain natriuretic peptide; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association; AUC, areas under the curve; IDI, integrated discriminant improvement; HFrEF, heart failure with reduced ejection fraction; ICD, implantable cardioverter-defibrillator.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no conflicts of interest.

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