



Research Article

C-reactive protein/albumin ratio in non-ST elevation myocardial infarction: Determining its predictive value for mortality

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Abstract

Objectives: Although the C-reactive protein/albumin ratio is accepted as a current biomarker in many diseases, such as myocardial infarction, studies on its clinical relevance in patients diagnosed with non-ST elevation myocardial infarction (NSTEMI) are limited. This study aimed to evaluate the prognostic significance of the C-reactive protein/albumin ratio in patients with NSTEMI.

Methods: This retrospective study included 300 patients diagnosed with NSTEMI. All patients were compared in terms of survival status and clinical, biochemical, and inflammatory markers. Logistic regression and receiver operating characteristic (ROC) curve analyses were used to determine the predictive value of CRP, albumin, and the CRP/albumin ratio.

Results: The CRP/ALB ratio was significantly higher in the mortality group than in the survivor group ($p=0.001$), whereas albumin levels were significantly lower ($p=0.001$). This ratio had a strong positive correlation with the CHA₂DS₂-VASc score ($p=0.711$, $p<0.001$) and independently predicted in-hospital mortality (OR=1.485, $p=0.046$). ROC analysis revealed an area under the curve (AUC) of 0.891 for the CRP/albumin ratio, which was significantly better than that of CRP or albumin alone ($p<0.001$). Although the combined CRP-albumin model had a slightly higher AUC (0.894), it was not significantly different from the C-reactive protein/albumin ratio.

Conclusion: The CRP/ALB ratio is a strong and independent predictor of in-hospital mortality in patients with NSTEMI. As an easily accessible and cost-effective biomarker, it provides valuable prognostic information and may improve early risk stratification and treatment strategies in clinical settings.

Keywords: Albumin, c-reactive protein, CRP/albumin ratio, inflammation, non-ST elevation myocardial infarction

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Non-ST-segment elevation myocardial infarction (NSTEMI) is a dangerous form of acute coronary syndrome (ACS) and remains one of the leading causes of morbidity and mortality. Although the specific changes on the electrocardiogram are less pronounced than those seen in ST-segment elevation myocardial infarction (STEMI), patients diagnosed with NSTEMI experience worse post-discharge outcomes than those with STEMI on their ECG [1]. For all these reasons, the urgent need for NSTEMI diagnosis, evidence-based risk stratification, and implementation of patient-specific treatment decisions is essential.

Risk stratification is crucial for NSTEMI treatment. Patients at high risk undergo invasive procedures earlier [2]. Therefore, risk stratification is highly effective in prognosis, and early and accurate implementation reduces mortality and morbidity [2, 3]. In recent years, the number of studies on inflammatory markers for risk assessment in patients with ACS has increased significantly [4–6]. Potentially useful biomarkers, such as C-reactive protein (CRP) and the CRP/albumin ratio (CAR), are available for measuring the clinical severity and predicting the prognosis of this group of diseases.

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CAR has been associated with thrombus burden and poor outcomes in patients [6–8]. Although some studies have been conducted on patients with NSTEMI, the results are more limited [9–12]. These studies have found a correlation between CAR and contrast-induced nephropathy, arterial occlusion, and hospital mortality in patients with STEMI elevation myocardial infarction. All studies and emerging evidence suggest that inflammation plays a direct or indirect role in the pathophysiology of NSTEMI.

Recent evidence supports the prognostic relevance of the CAR in cardiovascular diseases. A large prospective analysis from the UK Biobank demonstrated its association with cardiovascular outcomes and mortality [13]. A meta-analysis published in 2023 confirmed that an elevated CAR predicts adverse outcomes and mortality in different populations [14]. Furthermore, the CAR has been shown to be a useful prognostic biomarker in heart failure [15].

Therefore, this study aimed to investigate the prognostic value of the C-reactive protein/albumin ratio in patients diagnosed with NSTEMI. Unlike previous studies that mainly focused on STEMI populations, our study uniquely evaluated this biomarker specifically in NSTEMI patients, providing novel insights into its role in predicting in-hospital mortality.

Materials and Methods

The study protocol was approved by the Ankara Etlik City Hospital Ethics Committee (Date: 30/04/2025, No: 2025-0285) and conducted according to the Helsinki Declaration.

Study setting and study population

Our study evaluated patients aged 18–80 years who presented to the Etlik City Hospital with NSTEMI between January 2023 and December 2024. Three hundred patients with NSTEMI were retrospectively analyzed and divided into patient and control groups based on morbidity and mortality. Patients younger than 18 years, those diagnosed with MI other than NSTEMI, those with incomplete medical data, those with a previous diagnosis of AF, those with a history of antiarrhythmic therapy, those with end-stage renal disease, those with acute or chronic infections, and those with chronic inflammatory diseases were excluded from the study. Clinical characteristics, including a history of hypertension, diabetes mellitus, and heart failure, as well as medication use (β -blockers, ACE inhibitors or angiotensin receptor blockers, and statins), were recorded from the hospital electronic records. Patients with acute or chronic infections, chronic inflammatory or dermatologic diseases, malignancy, or those receiving corticosteroid or immunosuppressive therapy were excluded to minimize potential confounding effects on the inflammatory and biochemical parameters. CRP, albumin, mortality, length of hospital stay, CHA₂DS₂-VASc score, age, sex, blood pressure, body mass index, complete blood count parameters, biochemical parameters, and troponin parameters of the study population were retrospectively analyzed. The CHA₂DS₂-VASc score, which incorporates congestive heart failure, hypertension,

age, diabetes mellitus, prior stroke or transient ischemic attack, vascular disease, and sex category, was calculated for each patient to assess the overall cardiovascular risk burden.

Venous blood samples were collected from all patients within the first 24 h after admission, prior to the initiation of specific medical therapy. Samples were obtained from the antecubital vein after at least 8 h of fasting and drawn into serum separator tubes. After centrifugation at 3500 rpm for 10 min, the serum was promptly separated and analyzed.

All biochemical and cardiac parameters were measured using a Roche Cobas 8000 modular analyzer (Roche Diagnostics, Mannheim, Germany). Serum CRP levels were determined using an immunoturbidimetric method, while albumin concentrations and other routine biochemical parameters were measured using enzymatic colorimetric methods on the same system.

High-sensitivity troponin T (HsTropT) was analyzed using the Roche Cobas 8000 e801 module employing the electrochemiluminescence immunoassay (ECLIA) principle. Complete blood count (CBC) parameters were measured using a Sysmex XN-1000 automated hematology analyzer (Sysmex Corporation, Kobe, Japan).

Statistical analysis

All statistical analyses were performed using SPSS version 23 (IBM Corp., Armonk, NY, USA), MedCalc version 23.2.8 (MedCalc Software Ltd., Ostend, Belgium), the Analyze-it add-in for Microsoft Excel (Analyze-it Software, Ltd., Leeds, UK), and R version 4.3.1 (R Foundation for Statistical Computing, Vienna, Austria). Data visualization and multivariate modeling in R were conducted using the mixOmics and ggplot2 packages.

The normality of the distribution was assessed using the Shapiro–Wilk test. As most continuous variables did not follow a normal distribution, they were summarized using median and interquartile ranges (25th–75th percentiles) and compared between groups using the Mann–Whitney U test. Categorical variables are presented as counts and percentages and were compared using the chi-square test or Fisher's exact test, where appropriate.

To evaluate the strength and direction of associations between inflammatory markers (CRP, albumin, and CRP/albumin ratio) and clinical variables (e.g., CHA₂DS₂-VASc score, length of hospital stay, and age), Spearman's rank correlation coefficient (ρ) was used because of the nonparametric nature of the data. The complete correlation matrix was visualized as a heatmap using R.

The diagnostic performance of albumin, CRP, and the CRP-to-albumin ratio in predicting in-hospital mortality was assessed using Receiver Operating Characteristic (ROC) curve analysis. The area Under the Curve (AUC) values and their 95% confidence intervals were calculated and compared using the DeLong test for correlated ROC curves. Pairwise AUC comparisons were performed using MedCalc, and the optimal cutoff values for each biomarker were determined based on the Youden index.

Table 1. Distribution of CRP/albumin ratio and related clinical parameters in NSTEMI patients

	Median (25/75%)	95% CI of median	Min-max
Gender, n (%)			
Female	93 (31)		
Male	207 (69)		
Mortality, n (%)			
Alive	271 (90.3)		
Deceased	29 (9.76)		
Age (year)	64 (55–74)	63–66	32–100
CHA ₂ DS ₂ -VASc	3.5 (1–6)	3–4	0–7
Length of Hospital stay	19 (11–26)	18–22	2–39
WBC (×10 ⁹ /L)	12.1 (10.1–14)	11.7–12.7	8.3–15.9
HGB (g/dL)	13.6 (12.2–15)	13.3–14	11.2–16.2
Neutrophil (×10 ⁹ /L)	4.9 (3.4–6.4)	4.5–5.2	1.7–8.5
Lymphocyte (×10 ⁹ /L)	2.3 (1.4–3.3)	2.2–2.5	0.7–4.1
HsTropT (pg/mL)	41 (24–61.5)	38–47	7–81
HbA1c (%)	6.7 (6–7.4)	6.6–6.9	5.3–8.1
Glucose (mg/dL)	121 (85–155.5)	115–129	54–189
Total cholesterol (mg/dL)	196.5 (150.5–246)	189–208	95–291
HDL (mg/dL)	45 (37–51)	44–47	29–59
LDL (mg/dL)	143 (120–167)	141–148	94–191
ALT (U/L)	52 (43–61.5)	51–54	34–71
TG (mg/dL)	140 (107–172)	133–148	74–211
Albumin (g/dL)	3.3 (2.7–4)	3.2–3.5	2.1–4.5
CRP (mg/L)	49.5 (30–68.5)	45–55	4–93
CRP/albumin ratio	14.8 (8.86–20.7)	14.1–15.85	1.16–40.4

CRP: C-reactive protein; NSTEMI: non-ST elevation myocardial infarction; CI: Confidence interval; WBC: White blood cells; HGB: hemoglobin, HsTropT: High-sensitivity troponin T; HbA1c: Hemoglobin A1c; HDL: High-density lipoprotein; LDL: low-density lipoprotein; ALT: Alanine aminotransferase; TG: Triglyceride; CRP: C-reactive protein.

A binary logistic regression model was constructed to identify independent predictors of in-hospital mortality. The model included demographic, hematological, metabolic, hepatic, and inflammatory variables of the patients. The enter method was used, and odds ratios (OR) with 95% confidence intervals were reported for the results. Multicollinearity was checked using variance inflation factors (VIFs), and model calibration was evaluated using the Hosmer–Lemeshow goodness-of-fit test.

For multivariate class discrimination between alive and deceased patients, Partial Least Squares Discriminant Analysis (PLS-DA) was performed using the `mixOmics::plsda()` function in R. This supervised multivariate technique was selected because of its robustness in handling collinearity and its ability to model small sample sizes. The analysis focused on three variables: CRP, albumin, and the CRP/albumin ratio. The model was validated using leave-one-out cross-validation (LOOCV), and the variance explained by each component was reported as follows: Visualization included pairwise component scatter plots, biplots, and 2D/3D score plots to illustrate class separation.

Multiple hypothesis testing was adjusted using the Bonferroni correction, where appropriate, especially for post hoc comparisons after the univariate analyses. Statistical significance was set at $p < 0.05$.

Results

A total of 300 patients diagnosed with NSTEMI were included in the study. The majority of the study population was male (69%), and the median age was 64 years (IQR, 55–74). The overall in-hospital mortality rate was 9.7%. The distributions of the biochemical and inflammatory parameters are summarized in Table 1.

Hepatic enzyme (ALT) levels and triglyceride values also showed a wide distribution, which may have been influenced by comorbid metabolic conditions. In terms of inflammation and nutritional status, CRP and albumin levels varied significantly among individuals. In particular, the CRP/albumin ratio showed a wide range and interquartile range, suggesting significant differences in systemic inflammation and nutritional reserves among the patients. Cardiac injury markers, including Hs-TropT, showed elevated values consistent with acute myocardial injury, further supporting the diagnosis.

A significant and strong positive correlation was found between the CAR and the CHA₂DS₂-VASc score ($p = 0.711$, $p < 0.001$), whereas serum albumin showed a moderate negative correlation ($p = -0.307$, $p < 0.001$) (Table 2, Fig. 1). This indicates that patients with higher systemic inflammation exhibit higher cardiovascular risk profiles.

Table 2. Spearman correlation heatmap of CRP, albumin, and composite ratio with selected clinical variables

Spearman's rs	Age	CHA ₂ DS ₂ -VASc	Length of Hospital stay	Albumin	CRP	CRP/albumin ratio
Age	–	-0.121	-0.123	-0.004	-0.076	-0.077
CHA ₂ DS ₂ -VASc	-0.121	–	0.114	-0.307	0.689	0.711
Length of Hospital stay	-0.123	0.114	–	-0.066	0.115	0.142
Albumin	-0.004	-0.307	-0.066	–	-0.124	-0.430
CRP	-0.076	0.689	0.115	-0.124	–	0.930
CRP/albumin ratio	-0.077	0.711	0.142	-0.430	0.930	–
HsTropT	-0.096	0.073	0.086	-0.043	0.037	0.057
Neutrophil (×10 ⁹ /L)	-0.019	-0.028	-0.008	-0.021	-0.011	-0.020
Lymphocyte (×10 ⁹ /L)	-0.028	-0.011	-0.064	0.036	0.018	-0.003
Total cholesterol	0.092	-0.061	-0.070	0.080	-0.052	-0.076
HDL	0.062	0.086	0.003	-0.147	-0.025	0.026
LDL	-0.030	-0.008	0.046	-0.023	0.019	0.031
TG	-0.051	0.052	0.029	-0.012	0.053	0.056

CRP: C-reactive protein; HsTropT: high-sensitivity troponin T; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; TG: Triglyceride

Additionally, albumin levels exhibited a moderate inverse correlation with the CHA₂DS₂-VASc score ($p=-0.307$, $p<0.001$), further supporting the value of combining inflammatory and nutritional markers. The complete correlation matrix is presented in Appendix 1, which includes extended relationships among hematologic, metabolic, and inflammatory parameters.

These findings highlight the potential utility of the CAR as a composite biomarker that reflects both inflammatory status and cardiovascular risk, particularly in elderly patients with NSTEMI.

When patients were categorized according to in-hospital mortality status, 271 (90.3%) were survivors and 29 (9.7%) were non-survivors. As summarized in Table 3, patients in the deceased group had significantly higher CRP ($p=0.001$) and CRP/albumin ratio values ($p=0.001$), whereas albumin levels were significantly lower ($p=0.001$) compared with survivors. No statistically significant differences were observed between the two groups in terms of age ($p=0.415$), sex ($p=0.676$), glucose ($p=0.952$), or lipid profile parameters ($p>0.05$ for all).

These findings suggest that the CRP-to-albumin ratio may serve as a strong discriminator of in-hospital mortality in patients with NSTEMI, likely reflecting the combined impact of systemic inflammation and nutritional status on clinical outcomes.

In the multivariate logistic regression analysis, only the CAR remained an independent predictor of in-hospital mortality (OR=1.485, 95% CI=1.007–2.190, $p=0.046$), whereas CRP, albumin, and other biochemical markers were not statistically significant (Table 4).

This reinforces the utility of the CAR as a practical and independent biomarker for identifying patients at a higher risk of adverse outcomes during hospitalization.

Receiver operating characteristic (ROC) analysis demonstrated an AUC of 0.891 (95% CI: 0.850–0.924, $p<0.001$) for the CRP/albumin ratio, which was significantly higher than for CRP ($p<0.0001$) or albumin alone ($p=0.0028$). These results confirm the superior discriminative power of the CAR in predicting in-hospital mortality among patients with NSTEMI (Table 5, Fig. 2).

Pairwise comparisons of AUCs confirmed that the CAR was significantly better than albumin ($p=0.0028$) and CRP ($p<0.0001$). In contrast, there was no significant difference between the predictive abilities of albumin and CRP alone ($p=0.8296$), suggesting comparable but individually limited prognostic utilities. The combined model (CRP + albumin) yielded a slightly higher AUC than the CRP/albumin ratio alone (0.894 vs. 0.891), but this difference was not

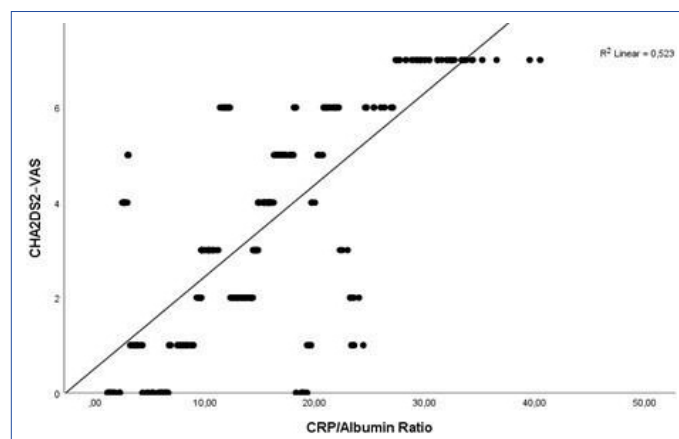


Figure 1. Scatter Plot depicting the association between systemic inflammation (CRP/albumin ratio) and cardiovascular risk burden (CHA₂DS₂-VASc).

CRP: C-reactive protein.

Table 3. Comparison of demographic, haematological, biochemical, and inflammatory parameters between alive and deceased NSTEMI patients

	Alive (n=271)	Deceased (n=29)	p
Gender, n (%)			
Female	85 (91.4)	8 (8.6)	0.676
Male	186 (89.8)	21 (10.3)	
Age (year)	64 (55–74)	64 (54–72)	0.415
CHA ₂ DS ₂ -VASc	3 (1–5)	7 (6–7)	0.001
Length of Hospital stay	18 (10–26)	27 (22–30)	0.001
WBC (×10 ⁹ /L)	12.1 (10.1–14.1)	12 (10.1–13.4)	0.803
HGB (g/dL)	13.6 (12.2–15.1)	13.4 (12.3–14.6)	0.506
Neutrophil (×10 ⁹ /L)	5.00 (3.50–6.40)	4.61 (3.12–5.21)	0.127
Lymphocyte (×10 ⁹ /L)	2.4 (1.4–3.3)	2 (1.5–2.8)	0.678
HsTropT (pg/mL)	40 (23–61)	50 (31–67)	0.082
HbA1c (%)	6.60 (5.90–7.40)	6.9 (6.2–7.3)	0.593
Glucose (mg/dL)	121 (87–154)	126 (82–163)	0.952
Total Cholesterol (mg/dL)	198(155–250)	185 (143–221)	0.293
HDL (mg/dL)	44 (37–50)	48 (42–54)	0.069
LDL (mg/dL)	144 (121–169)	142 (116–160)	0.560
ALT (U/L)	52 (43–62)	48 (41–56)	0.111
TG (mg/dL)	140 (106–171)	163 (112–182)	0.427
Albumin (g/dL)	3.4 (2.8–4)	2.6 (2.4–2.8)	0.001
CRP (mg/L)	46 (27–65)	75 (64–82)	0.001
CRP/albumin ratio	14.3 (8.25–18.29)	29.5 (21.79–33.3)	0.001

The results were expressed as Median (25–75%). Gender was expressed as count and %. NSTEMI: Non-ST elevation myocardial infarction; WBC: White blood cells; HGB: Hemoglobin; HsTropT: High-sensitivity troponin T; HbA1c: Hemoglobin A1c; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; ALT: Alanine aminotransferase; TG: Triglyceride; CRP: C-reactive protein.

statistically significant, indicating that the ratio itself may encapsulate much of the predictive value of the two parameters (Fig. 3).

The optimal threshold for the combined logistic model was calculated as 0.085, based on Youden's index. This cutoff provided a sensitivity of 89% (73–98%) and specificity of 79% (74–84%), indicating strong discriminatory performance.

Taken together, these results support the use of the CRP-to-albumin ratio as a single, practical, and statistically superior biomarker for risk stratification in patients with NSTEMI, with a performance comparable to that of a multivariate logistic model combining CRP and albumin.

PLS-DA was applied to assess the discriminative ability of albumin, CRP, and the CRP/albumin ratio in differentiating between patients who survived and those who died during hospitalization. The model was constructed using these three variables as predictors, with mortality as the outcome variable.

The pairwise scatter plot matrix (Fig. 4a) indicates that Component 1 alone captured the majority of the variance (100%) relevant for class discrimination, whereas Components 2 and 3 contributed minimally (0%). The dominance of Component 1 suggests that the combination of CRP and albumin-based variables contains strong discriminatory information.

The biplot (Fig. 4b) revealed distinct clustering of alive versus deceased patients along Component 1, with the CRP/albumin ratio emerging as the most influential variable in the model. This finding is consistent with previous ROC analyses, which showed the highest AUC for the CRP-to-albumin ratio.

In the 2D score plot (Fig. 4c), a clear separation between the alive (green) and deceased (red) groups was observed, despite some overlap between the groups. The 3D score plot (Fig. 4d) further confirmed this separation, suggesting that these biomarkers, when used in combination, provide a meaningful projection space for classifying mortality risk.

Overall, the PLS-DA model supports the hypothesis that combining CRP and albumin levels and their ratio enhances the ability to distinguish patients with poor in-hospital outcomes, underscoring their clinical relevance in early mortality risk stratification.

Panel A presents a pairwise scatter plot matrix that illustrates the distribution and relationships between the components extracted using PLS-DA. Notably, Component 1 accounted for 100% of the explained variance, indicating that it captured the most substantial discriminative information between patient groups. Panel B shows the biplot, where a distinct separation between deceased (red) and alive (green) patients is evident along the first principal component (PC1) axis. Among the

Table 4. Multivariate logistic regression analysis of demographic, hematologic, biochemical, and inflammatory variables for predicting in-hospital mortality in NSTEMI patients

	B	Wald	Significance	Odds ratio Exp(B)	95% CI for Exp(B)	
					Lower	Upper
Age	-0.004	0.037	0.848	0.996	0.955	1.038
CHA ₂ DS ₂ -VASc	-0.016	0.009	0.926	0.984	0.706	1.372
WBC	-0.002	0.000	0.988	0.998	0.780	1.278
HGB	-0.094	0.314	0.575	0.910	0.655	1.265
Neutrophil	-0.117	0.592	0.441	0.889	0.660	1.199
Lymphocyte	-0.107	0.191	0.662	0.899	0.557	1.450
HsTropT	0.013	1.045	0.307	1.013	0.988	1.039
HbA1c	0.534	2.234	0.135	1.705	0.847	3.434
Glucose	-0.007	1.079	0.299	0.993	0.979	1.006
Total cholesterol	0.002	0.188	0.665	1.002	0.993	1.012
HDL	0.028	0.734	0.392	1.028	0.965	1.096
LDL	-0.015	2.371	0.124	0.985	0.967	1.004
ALT	0.000	0.000	0.990	1.000	0.953	1.051
TG	-0.001	0.042	0.837	0.999	0.986	1.012
Albumin	0.501	0.134	0.715	1.650	0.112	24.244
CRP	-0.071	1.107	0.293	0.932	0.817	1.063
CRP/albumin ratio	0.395	3.974	0.046	1.485	1.007	2.190
Constant	-8.086	1.504	0.220	0.000		

NSTEMI: Non-ST elevation myocardial infarction; WBC: White blood cells; HGB: Hemoglobin; HsTropT: High-sensitivity troponin T; HbA1c: Hemoglobin A1c; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; ALT: Alanine aminotransferase; TG: Triglyceride; CRP: C-reactive protein.

Table 5. Diagnostic performance of albumin, CRP, CRP/albumin ratio, and CRP-albumin combination in predicting in-hospital mortality

Variable	Cut-off	Sensitivity	Specificity	AUC
Albumin	≤3.1	90 (72–93)	58 (52–64)	0.808 ^a (0.756–0.851)
CRP	>60	86 (62–96)	67 (61–73)	0.817 ^a (0.768–0.859)
CRP/albumin ratio	>20.28	89 (72–98)	80 (75–85)	0.891 ^b (0.85–0.924)
CRP-albumin combination	>0.085	89 (73–98)	79 (74–84)	0.894 ^b (0.85–0.931)

Different letters in the same column indicate statistically significant differences. CRP: C-reactive protein; AUC: Area under the curve.

variables, the CRP/Albumin Ratio appeared to be the most influential in driving this separation. Panel C displays the two-dimensional scores plot, which reveals a partial overlap between groups but also demonstrates a discernible clustering pattern, suggesting meaningful group differentiation along Component 1. Panel D illustrates the three-dimensional scores plot, offering a spatial view of the component distribution and further emphasizing the separation between alive and deceased patients in the multivariate space defined by the first three components.

Discussion

In our study, we demonstrated that the CRP-to-albumin ratio is a significant and independent predictor of mortality during hospitalization in patients with NSTEMI. This biomarker

is important and clinically relevant because it indicates systemic inflammation and malnutrition. Both factors are essential for understanding the clinical outcomes of acute coronary syndromes. In acute myocardial infarction, the body's physiological stress response can be determined much more accurately using this ratio than using CRP or albumin alone.

Patients who died had significantly higher CRP/albumin ratios and significantly lower albumin levels than those who survived did. This association has also been found in some NSTEMI cohort studies in 2022 and 2020, which reported that patients in these cohorts had significantly higher CRP/albumin ratios and significantly lower albumin levels than those without NSTEMI. [9, 16]. One of the many striking results of our study is that the correlation between the CRP/albumin ratio and the CHA₂DS₂-VASc score was stronger

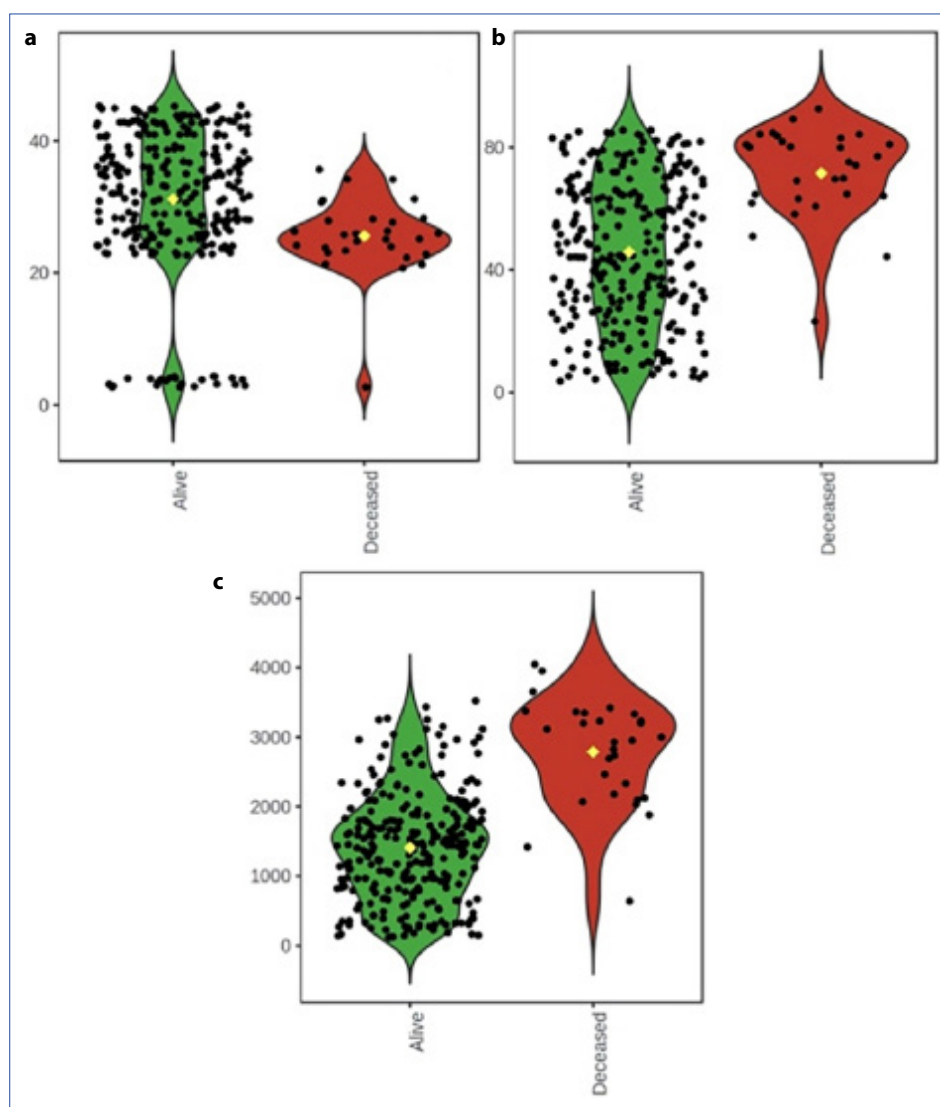


Figure 2. Violin plot comparison of albumin, CRP, and CRP/albumin ratio between alive and deceased non-survivors in NSTEMI patients.

CRP: C-reactive protein; NSTEMI: Non-ST elevation myocardial infarction.

than that between CRP and the $\text{CHA}_2\text{DS}_2\text{-VASc}$ score. Although the $\text{CHA}_2\text{DS}_2\text{-VASc}$ score has long been used to assess the risk of thromboembolic events in patients with atrial fibrillation, it is gaining increasing acceptance for the risk stratification of cardiovascular events. Consequently, the close correlation of a simple inflammatory ratio with a score increasingly accepted by the American College of Cardiology guidelines further highlights the potential utility of this test in clinical practice. Furthermore, although the high-sensitivity troponin T test plays a key role in the diagnosis of various types of myocardial injury, it was not found to be a strong predictor of in-hospital mortality in the patients included in our study.

Our ROC analysis provides additional support for the clinical utility of the CRP: ALB ratio. With an AUC value approaching 0.90, the ratio was found to be a significantly superior

biomarker compared to CRP or albumin alone. Although a logistic model including both CRP and albumin yielded a slightly higher AUC, the difference was not significant. Our findings support the conclusion of a similar report in STEMI patients that the CRP/albumin ratio is a current and accepted marker of poor prognosis in the ACS spectrum [7]. Additionally, the distinction between the neutrophil percentage/albumin ratio (NPAR), a newer marker, appears to be more similar to CRP and MCV than to CRP or albumin. Moreover, a comparison with CRP and albumin does not reflect the clinically desirable properties of parameters such as CRP and albumin.

Our findings further support the concept that systemic inflammation and hypoalbuminemia reflect the combined metabolic and immune responses in acute myocardial injury. The CRP/albumin ratio, which integrates these two processes, ap-

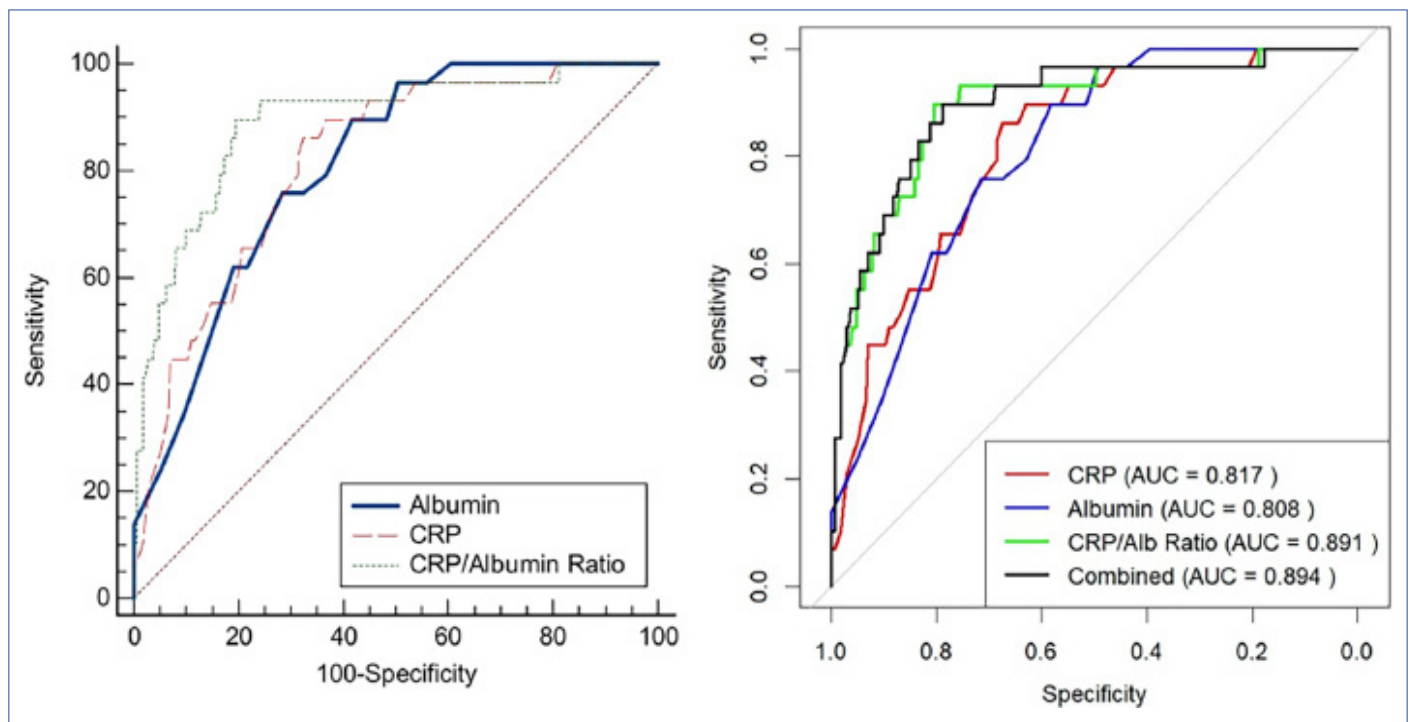


Figure 3. ROC curves comparing the predictive accuracy of albumin, CRP, CRP/albumin ratio, and combined logistic regression model for in-hospital mortality in NSTEMI patients.

AUC: Area under the curve; CRP: C-reactive protein; ROC: Receiver operating characteristic; NSTEMI: Non-ST elevation myocardial infarction.

pears to provide a more stable indicator of patient prognosis than either marker alone. This is consistent with recent reports in both STEMI and NSTEMI populations showing that higher CRP/albumin ratios predict increased in-hospital mortality and adverse cardiovascular events [9, 10, 16]. The present study strengthens this evidence by demonstrating that this ratio remains an independent predictor even after adjusting for conventional risk factors.

These results are consistent with the pathophysiological mechanisms. Plaque rupture alone does not cause NSTEMI. It is now more widely accepted that the body's inflammatory response, which also causes damage to the blood vessel wall, is linked to endothelial dysfunction. Metabolic stress associated with strenuous physical training and resting conditions plays a significant role [8].

In this study and similar publications, the CRP-to-albumin ratio was considered an important predictor of prognosis in patients with MI. It appears particularly useful in identifying high-risk patients who initially appear stable and may therefore be easily overlooked for intensive care or other types of interventions.

The CRP-to-albumin ratio can be easily calculated using routine laboratory test results. This could provide a risk assessment method that can be easily and conveniently integrated into resource-limited emergency departments and small hospitals. This ratio could be integrated into risk scoring systems, such as TIMI or GRACE, to reduce mortality and morbidity.

From a clinical perspective, the CRP/albumin ratio may be particularly useful in identifying high-risk subgroups of patients who initially present with stable features but are at an increased risk of systemic inflammation and impaired nutritional reserves. Such patients could benefit from close monitoring and early intervention. Moreover, it should be emphasized that low albumin levels may not only reflect inflammation but can also be influenced by malnutrition and liver dysfunction. This multifactorial nature should be considered when interpreting the CRP-to-albumin ratio in clinical settings.

Conclusion

Our study demonstrated that the CRP-to-albumin ratio is a strong and independent predictor of mortality in patients with NSTEMI. This ratio is associated with complex mechanisms related to inflammation and nutritional status, which are often overlooked in routine assessments of cardiovascular risk factors. As a simple, accessible, and effective marker, this ratio is a promising parameter for improving rapid risk stratification in patients with NSTEMI.

Study Limitations: Our study had several limitations. The single-center, retrospective design and relatively small mortality subgroup ($n=29$) limit the generalizability and statistical power of our findings. Although the overall cohort size ($n=300$) was adequate, we did not perform detailed subgroup analyses by sex, diabetes, hypertension, or other comorbidities. Moreover, patients aged ≥ 80 years and those

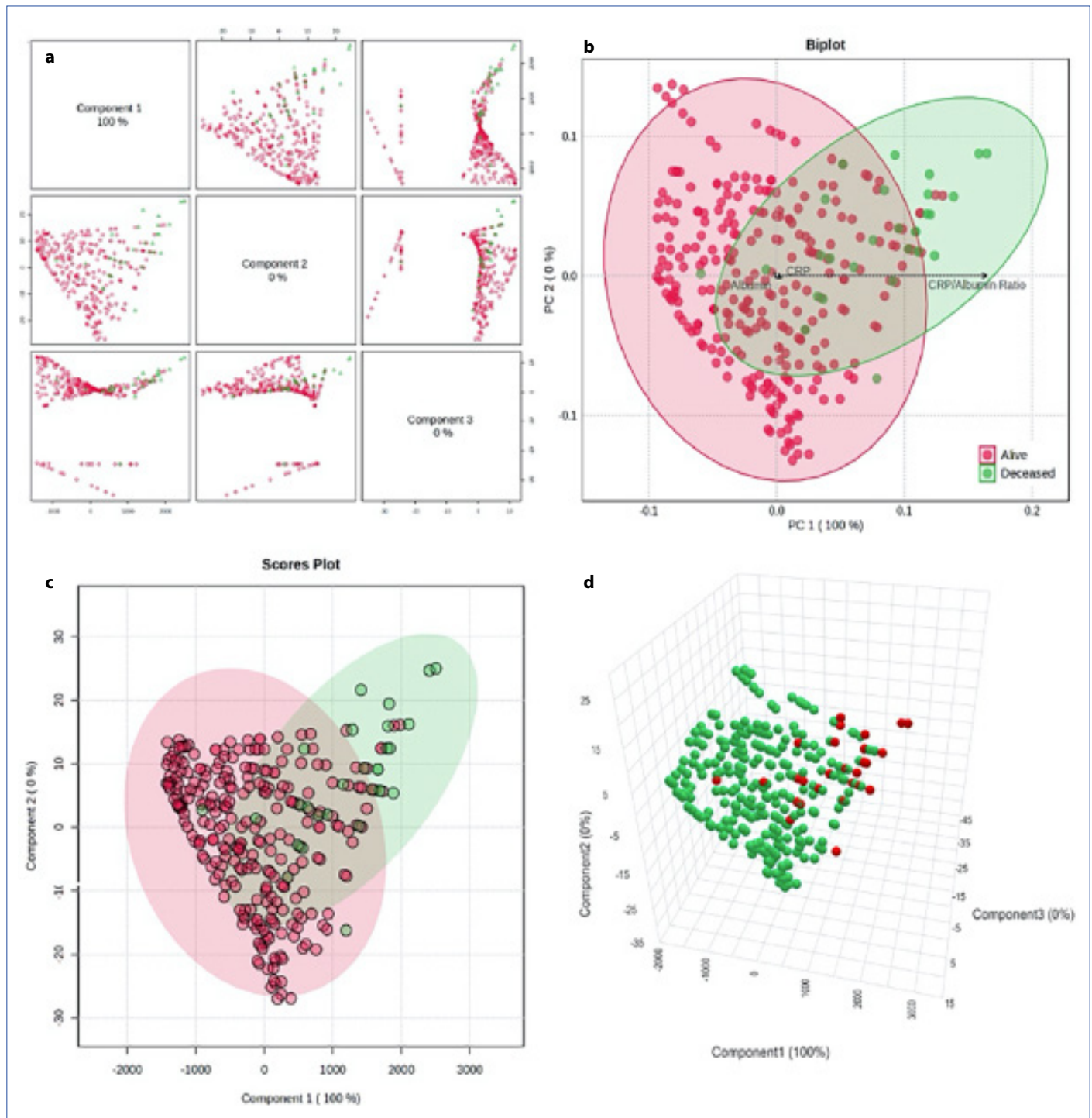


Figure 4. Partial least squares discriminant analysis (PLS-DA) plots based on albumin, CRP, and CRP/albumin ratio levels in relation to in-hospital mortality.

CRP: C-reactive protein.

with multiple comorbidities were excluded, which may reduce the representativeness of the real-world NSTEMI population in this study. Another limitation is that we focused primarily on CRP and albumin, without including additional inflammatory biomarkers such as IL-6, TNF- α , or high-sensitivity CRP, which could have provided a broader understand-

ing of the underlying mechanisms of sarcopenia. Finally, our comparisons were limited to the CHA₂DS₂-VASc score and did not extend to other established prognostic tools such as GRACE or TIMI. Therefore, large-scale, multicenter, prospective studies are needed to validate our results and explore these aspects in greater depth.

Online Appendix Files: [https://jag.journalagent.com/ijmb/abs_files/IJMB-02693/IJMB-02693_\(4\)_IJMB-02693_Appendix_1.pdf](https://jag.journalagent.com/ijmb/abs_files/IJMB-02693/IJMB-02693_(4)_IJMB-02693_Appendix_1.pdf)

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Informed Consent: Because our study was retrospective in design, informed consent was not obtained.

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