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RESEARCH ARTICLE

Blood urea nitrogen-to-albumin ratio and in-hospital mortality in patients with ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention

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In-hospital mortality remains an importantconcern in patients with ST-elevation myocardial infarction (STEMI), highlighting the need for simple and readily available markers for early risk stratification. This study investigated the prognostic value of the blood urea nitrogen-to-serum albumin ratio (BAR) for in-hospital mortality in patients with STEMI undergoing primary percutaneous coronary intervention (pPCI). This single-center retrospective cohort study included 388 consecutive patients who underwent pPCI in 2024. Admission BAR was calculated using blood urea nitrogen (mg/dL) and serum albumin (g/L). Its association with in-hospital mortality was evaluated using receiver operating characteristic analysis and exploratory multivariable logistic regression, with Firth-penalized regression and bootstrap validation performed as sensitivity analyses. In-hospital death occurred in 18 patients (4.6%). Patients who died had significantly higher BAR values than survivors (0.7 vs. 0.4, $P < 0.001$). BAR showed good discrimination for in-hospital mortality, with an area under the curve of 0.869 ($P < 0.001$); the bootstrap-corrected area under the curve was also 0.869. An exploratory cut-off of 0.475 yielded 83.3% sensitivity and 75.4% specificity. In the multivariable model, each 0.1-unit increase in BAR was associated with higher odds of in-hospital mortality (odds ratio 1.412, 95% confidence interval 1.161–1.718; $P = 0.0006$), with a similar estimate in the Firth-penalized analysis (odds ratio 1.346, 95% confidence interval 1.121–1.616; $P = 0.0008$). Admission BAR may represent a simple candidate marker for early mortality risk stratification in STEMI patients undergoing pPCI; however, its prognostic performance and clinical utility require validation in larger prospective multicenter cohorts.

Keywords: Blood urea nitrogen, albumin, BAR, myocardial infarction, in-hospital mortality.

Introduction

Acute myocardial infarction (AMI) remains a leading cause of global morbidity and mortality. Despite advanced reperfusion strategies, in-hospital mortality rates for AMI are approximately 5%, with ST-elevation myocardial infarction (STEMI) being an independent predictor of this mortality [1]. Early identification of high-risk STEMI patients, appropriate treatment strategies, and optimized clinical management are crucial for improving outcomes.

Current risk stratification systems, such as the Global Registry of Acute Coronary Events (GRACE) and Thrombolysis In Myocardial Infarction (TIMI) scores, predict in-hospital mortality in STEMI patients [2]. However, their reliance on multiple complex clinical and laboratory parameters limits practical application. This highlights the need for simpler, rapidly calculable, and easily accessible prognostic biomarkers.

The blood urea nitrogen-to-serum albumin ratio (BAR) is a novel prognostic biomarker [3, 4]. Blood urea nitrogen (BUN), a byproduct of protein metabolism, reflects renal function. Elevated BUN levels in AMI patients indicate neurohumoral activation due to renal hypoperfusion and decreased cardiac output, offering prognostic value independent of glomerular filtration rate (GFR) [5]. Albumin, besides indicating nutritional status, possesses antioxidant and anti-inflammatory properties, stabilizes the endothelial glycocalyx, and regulates vascular permeability [6]. Both BUN and albumin are widely used and readily accessible clinical biomarkers. The BAR comprehensively indicates renal function, inflammation, nutritional status, and endothelial health. Studies show that elevated BUN and low albumin levels correlate significantly with worse prognoses in AMI patients [3, 4, 7–9]. While the BAR predicts mortality in various cardiovascular populations, evidence for its specific role in predicting in-hospital mortality in STEMI patients undergoing primary percutaneous coronary intervention (pPCI) is limited. Similarly, other composite markers like the C-reactive protein-to-albumin ratio (CAR) and

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neutrophil-to-lymphocyte ratio (NLR) have shown predictive value for in-hospital mortality in AMI patients [10, 11].

This study aims to investigate the BAR's predictive value for in-hospital mortality in STEMI patients undergoing pPCI, offering new insights into early risk stratification and prognostic assessment for STEMI.

Materials and methods

Study design and patients

This single-center, retrospective, observational cohort study was conducted at the coronary intensive care unit (CICU) of a tertiary care hospital. Data were extracted from the hospital's electronic medical database. From January 1, 2024, to December 31, 2024, 423 consecutive patients admitted to the CICU with a STEMI diagnosis were screened. Inclusion criteria were age ≥18 years, STEMI diagnosis, and undergoing pPCI.

Exclusions included patients not undergoing pPCI ($n = 5$), those with missing BUN or albumin data ($n = 13$), known malignancy ($n = 6$), active infection or sepsis ($n = 2$), severe hepatic insufficiency ($n = 1$), end-stage renal disease requiring renal replacement therapy ($n = 6$), or significant nutritional deficiency/cachexia ($n = 2$). After exclusions, a final cohort of 388 patients was included. STEMI diagnosis followed European Society of Cardiology (ESC) guidelines [12].

Data collection

Demographic characteristics (age, gender), medical history (hypertension, diabetes mellitus, smoking status, previous revascularization), and clinical findings were obtained from the hospital database. Myocardial infarction type (anterior, inferior, other) and angiographic findings (culprit lesion: left anterior descending artery [LAD], circumflex artery [CX], right coronary artery [RCA], left main coronary artery [LMCA], or coronary artery bypass graft [CABG]) were recorded.

Laboratory parameters, including renal function markers (BUN, creatinine, GFR), electrolytes (sodium, potassium), albumin, liver function tests (alanine aminotransferase [ALT]), lipid profile, inflammatory markers (C-reactive protein [CRP]), hematologic indices (white blood cells [WBCs], hemoglobin, platelets, neutrophils, lymphocytes), and cardiac troponin, were collected from venous blood samples immediately upon CICU admission, prior to pPCI and contrast exposure. The BAR was calculated by dividing admission BUN (mg/dL) by serum albumin (g/L). While albumin is often reported as g/dL, our institution uses g/L, resulting in a 10-fold scale difference (0.1 coefficient) in BAR values. These values were normalized for literature comparison by accounting for this unit difference (mg/dL / g/L). Left ventricular ejection fraction (LVEF) was determined via transthoracic echocardiography using the Simpson biplane method within 24 h of admission.

Ethical statement

The study protocol was approved by the Ethics Committee of Health Science University, Antalya Training and Research Hospital (Approval Date: 05.02.2026, Approval Number: 3/18). All procedures adhered to the ethical standards of the 1964 Declaration of Helsinki and its later amendments, as well as Good Clinical Practice Guidelines. Informed consent was waived by the Ethics Committee due to the study's retrospective nature.

Statistical analysis

Data were analyzed using SAS v. 9.4 (SAS Institute, Cary). Descriptive statistics included frequencies and percentages for categorical variables and the mean with standard deviation (SD) or median with interquartile range (IQR) for continuous variables, depending on the Shapiro-Wilk test for normality. Categorical and continuous variables were compared using the chi-square test and appropriate parametric or non-parametric tests. Pearson's correlation coefficient assessed relationships between continuous variables.

Patients with missing primary laboratory data (BUN or albumin, $n = 13$) were excluded. For the final cohort ($n = 388$), rare missing data in secondary clinical and laboratory variables (<2.0% of observations; specifically LVEF, sodium, and platelet count) were handled using multiple imputation with 5 imputations via the approximate Bayesian bootstrap approach. Subsequent multivariable analyses, including standard stepwise logistic regression and Firth's penalized likelihood regression, were performed on the imputed datasets. Parameter estimates and standard errors were pooled across imputations using Rubin's rules to account for missing-data uncertainty.

In-hospital mortality was the response variable. Explanatory variables included demographic characteristics, medical history, and laboratory parameters (BUN, creatinine, GFR, electrolytes, albumin, ALT, lipid profile, inflammatory markers, hematologic indices, and cardiac troponin). Given the low event-to-variable ratio (18 deaths), multivariable stepwise logistic regression was used as an exploratory analysis to identify potential candidate covariates associated with mortality using Wald-Chi Square test selection criteria. To address potential overfitting, small-sample bias, and rare events, sensitivity analyses were conducted using Firth's penalized likelihood logistic regression and bootstrap validation (1,000 iterations). Standard maximum likelihood estimation (MLE) and Firth's penalized estimates are reported side-by-side for transparency. Multicollinearity in the final multivariable model was assessed using Variance Inflation Factor (VIF), with a threshold of <2.0 considered acceptable. Receiver operating characteristic (ROC) analysis determined the BAR's predictive value, with Youden's index identifying the exploratory cut-off value.

Statistical significance was set at $P < 0.05$.

Results

A total of 388 patients were included in this study, with in-hospital mortality occurring in 18 (4.6%) patients. Patients with fatal outcomes were significantly older ($P = 0.0093$) and more likely to be female ($P < 0.0001$) compared to survivors. Additionally, the distribution of culprit lesions differed significantly between the two groups ($P = 0.001$). Table 1 presents the baseline demographic, clinical, and laboratory characteristics of the study population, stratified by in-hospital mortality status.

Table 1. Baseline demographic, clinical, and laboratory characteristics of patients with acute myocardial infarction stratified by in-hospital mortality

	Survivors ($n = 370$)	Non-survivors ($n = 18$)	Total ($n = 388$)	*P* value
Sex, *n* (%)				**<.0001**
Female	49 (13.2%)	10 (55.6%)	59 (15.2%)	
Male	321 (86.8%)	8 (44.4%)	329 (84.8%)	
Age, median (IQR)	58.0 (51.0–66.0)	67.5 (56.0–84.0)	59.0 (51.0–67.0)	**0.0093**
MI Type, *n* (%)				0.3635
Anterior	163 (44.1%)	11 (61.1%)	174 (44.8%)	
Inferior	175 (47.3%)	6 (33.3%)	181 (46.6%)	
Other	32 (8.6%)	1 (5.6%)	33 (8.5%)	
Culprit lesion, *n* (%)				**0.0012**
LAD	166 (44.9%)	10 (55.6%)	176 (45.4%)	
CX	68 (18.4%)	2 (11.1%)	70 (18.0%)	
RCA	127 (34.3%)	3 (16.7%)	130 (33.5%)	
LMCA	3 (0.8%)	2 (11.1%)	5 (1.3%)	
CABG graft	6 (1.6%)	1 (5.6%)	7 (1.8%)	
DM, *n* (%)	121 (32.7%)	5 (27.8%)	126 (32.5%)	0.6630
HT, *n* (%)	134 (36.2%)	4 (22.2%)	138 (35.6%)	0.2259
Smoking status, *n* (%)				0.8086
No	80 (21.6%)	4 (22.2%)	84 (21.6%)	
Yes	252 (68.1%)	13 (72.2%)	265 (68.3%)	
Former smoker	38 (10.3%)	1 (5.6%)	39 (10.1%)	
Revascularization history, *n* (%)				0.5201
No	312 (84.3%)	14 (77.8%)	326 (84.0%)	
PCI	51 (13.8%)	3 (16.7%)	54 (13.9%)	
CABG	7 (1.9%)	1 (5.6%)	8 (2.1%)	
LVEF (%)	50.0 (40.0–55.0)	40.0 (30.0–50.0)	50.0 (40.0–55.0)	**0.0046**
Glucose, mg/dL	130.0 (106.0–189.0)	252.0 (115.0–343.0)	131.0 (106.0–196.5)	**0.0048**
BUN, mg/dL	15.0 (12.0–19.0)	23.5 (19.0–33.0)	15.0 (12.5–20.0)	**<.0001**
Creatinine, mg/dL	1.0 (0.9-1.1)	1.3 (1.0-1.7)	1.0 (0.9-1.2)	**0.0012**
GFR, mL/min/1.73 m^2	78.4 (63.7–90.7)	54.7 (30.9–65.1)	77.9 (62.8–90.4)	**0.0001**
Sodium, mmol/L	137.0 (135.0–139.0)	133.5 (128.0–136.0)	137.0 (135.0–139.0)	**<.0001**
Potassium, mmol/L	4.2 (3.9–4.5)	4.4 (3.8–4.7)	4.2 (3.9–4.5)	0.6160
Albumin, g/L	42.2 (39.5–44.9)	35.9 (33.0–38.9)	41.9 (39.2–44.6)	**<.0001**
ALT, U/L	25.0 (18.0–36.0)	41.5 (25.0–214.0)	25.0 (18.0–38.0)	**0.0031**
Cholesterol, mg/dL	206.0 (171.0–241.0)	160.5 (138.0–215.0)	205.0 (169.0–240.0)	**0.0036**
HDL, mg/dL	46.0 (38.0–53.0)	40.5 (29.0–47.0)	46.0 (38.0–53.0)	**0.0338**
LDL, mg/dL	131.0 (103.0–159.0)	105.5 (86.0–123.0)	130.0 (102.5–157.5)	**0.0232**
Triglyceride, mg/dL	149.0 (91.0–216.0)	154.0 (96.0–196.0)	149.0 (91.5–216.0)	0.9691
Uric acid, mg/dL	5.7 (4.5–6.9)	5.1 (4.4–6.0)	5.7 (4.5–6.8)	0.1486
CRP, mg/L	5.7 (2.2–13.1)	5.6 (1.6–13.7)	5.7 (2.2–13.1)	0.9708
WBC, $10^3/mm^3$	10.7 (8.6–13.5)	10.6 (9.0–12.9)	10.7 (8.6–13.3)	0.7739
Hemoglobin, g/dL	14.4 (12.9–15.5)	13.4 (11.5–14.6)	14.4 (12.8–15.5)	0.0334
Platelets, $10^3/mm^3$	248.5 (206.0–296.0)	265.5 (216.0–336.0)	250.0 (207.0–296.5)	0.3436

(Continued)

Table 1. Continued

	Survivors ($n = 370$)	Non-survivors ($n = 18$)	Total ($n = 388$)	*P* value
Neutrophil, $10^3/mm^3$	7.4 (5.4–10.2)	6.6 (5.3–9.2)	7.4 (5.3–10.2)	0.6032
Lymphocyte, $10^3/mm^3$	2.0 (1.4–2.8)	2.5 (1.5–3.0)	2.0 (1.4–2.8)	0.4474
Troponin, ng/L	846.0 (180.0–2781.0)	1257.0 (138.0–4584.0)	852.0 (178.5–2787.5)	0.5913
BAR	0.4 (0.3–0.5)	0.7 (0.5–0.9)	0.4 (0.3–0.5)	**<.0001**
CAR	0.1 (0.1–0.3)	0.2 (0.1–0.4)	0.1 (0.1–0.3)	0.7632
NLR	3.9 (2.2–6.1)	3.1 (2.1–5.3)	3.8 (2.2–6.1)	0.4159

Data with non-normal distributions are presented as medians and interquartile ranges. **Abbreviations**: ALT, alanine aminotransferase; BAR, blood urea nitrogen-to-albumin ratio; BUN, blood urea nitrogen; CABG, coronary artery bypass grafting; CAR, C-reactive protein-to-albumin ratio; CRP, C-reactive protein; CX, circumflex artery; DM, diabetes mellitus; GFR, glomerular filtration rate; HDL, high-density lipoprotein; HT, hypertension; IQR, interquartile range; LAD, left anterior descending artery; LDL, low-density lipoprotein; LMCA, left main coronary artery; LVEF, left ventricular ejection fraction; MI, myocardial infarction; NLR, neutrophil-to-lymphocyte ratio; PCI, percutaneous coronary intervention; RCA, right coronary artery; WBC, white blood cell.

Table 2. Pairwise cross-correlation of select laboratory findings

	BUN	Creatinine	GFR	Cholesterol	LDL	CRP	WBC	PLT	BAR
Creatinine	0.6133**	1.0000							
GFR	−0.6056**	−0.8038**	1.0000						
Cholesterol	−0.1695**	−0.1431**	0.1985**	1.0000					
LDL	−0.1470**	−0.1262*	0.1707**	0.8961**	1.0000				
CRP	0.0313	0.0409	−0.0374	0.0014	0.0410	1.0000			
WBC	−0.0524	−0.0323	0.0312	0.1298*	0.1160	0.1298*	1.0000		
PLT	0.0331	−0.0129	0.0124	−0.0237	−0.0169	−0.0493	0.3375**	1.0000	
BAR	0.9378**	0.5737**	−0.5753**	−0.2629**	−0.1832**	0.0216	−0.0698	0.0167	1.0000
CAR	0.0430	0.0528	−0.0508	−0.0214	0.0242	0.9888**	0.1254	−0.0508	0.0434

*$P < 0.05$; **$P < 0.01$. **Abbreviations**: BAR, blood urea nitrogen-to-albumin ratio; BUN, blood urea nitrogen; CAR, C-reactive protein-to-albumin ratio; CRP, C-reactive protein; GFR, glomerular filtration rate; LDL, low-density lipoprotein; PLT, platelets; WBC, white blood cell.

Table 2 displays the pairwise cross-correlation between laboratory findings. Moderate correlations were observed between renal function markers (BUN, creatinine, and GFR) and the BAR, with a particularly strong positive correlation between BUN and the BAR ($r = 0.9378$). Strong correlations were also found between cholesterol and low-density lipoprotein (LDL) cholesterol, and between CRP and the CAR, suggesting potential multicollinearity among these variables in predicting in-hospital mortality.

Table 3 presents the results of the exploratory multivariable stepwise logistic regression analysis alongside the Firth-penalized sensitivity analysis. Collinearity diagnostics for the final model showed all VIF values <2.0, confirming no significant multicollinearity. The model demonstrated good fit (Hosmer–Lemeshow test: chi-Square = 3.486, $P = 0.900$), retaining five candidate covariates: gender, LVEF, sodium, platelet count, and BAR. For each 0.1-unit increase in BAR, the standard maximum-likelihood model yielded an adjusted odds ratio (OR) of 1.412 (95% confidence interval [CI]: 1.161–1.718; $P = 0.0006$), while the Firth-penalized model yielded an OR of 1.346 (95% CI: 1.121–1.616; $P = 0.0008$). The two estimates were largely consistent; however, these findings should be interpreted as exploratory.

The area under the curve (AUC) for BAR values according to mortality status was 0.869 ($P < 0.001$). An exploratory cut-off value of 0.475 was identified using Youden's index within the study cohort. Bootstrap validation confirmed the robustness of the BAR, with a bootstrap-corrected AUC of 0.869. Restricted cubic spline analysis suggested a non-linear association between BAR and in-hospital mortality (Figure 1). Because the cut-off was derived and evaluated in the same cohort, the value of 0.475 should not be interpreted as a validated clinical threshold. At this cut-off value, sensitivity was 83.3% (95% CI: 60.8%–94.2%) and specificity was 75.4% (95% CI: 70.8%–79.5%). The ROC analyses are presented in Table 4 and Figure 2.

Discussion

This study investigated the predictive value of the admission BAR for in-hospital mortality in STEMI patients undergoing pPCI. BAR levels were significantly higher in patients with fatal outcomes compared to survivors. An exploratory

Table 3. Exploratory multivariable stepwise logistic regression and Firth's penalized sensitivity analysis for predicting in-hospital mortality

Predictor variable	Model type	Coefficient (B)	SE	*P* value	OR (95% CI) per 1.0 Unit	OR (95% CI) per 0.1 unit
Intercept	Standard MLE	27.968	9.457	0.0031	—	—
	Firth-penalized	25.142	8.865	0.0045	—	—
Sex (female vs. male)	Standard MLE	1.770	0.639	0.0056	5.874 (1.680–20.533)	—
	Firth-penalized	1.624	0.612	0.0080	5.073 (1.528–16.843)	—
LVEF (%)	Standard MLE	−0.105	0.031	0.0007	0.901 (0.848–0.957)	—
	Firth-penalized	−0.098	0.030	0.0011	0.907 (0.855–0.962)	—
Sodium (mmol/L)	Standard MLE	−0.234	0.069	0.0008	0.792 (0.690–0.908)	—
	Firth-penalized	−0.218	0.066	0.0010	0.804 (0.707–0.915)	—
Platelets (10^3/mm^3)	Standard MLE	0.009	0.004	0.0186	1.009 (1.002–1.017)	—
	Firth-penalized	0.008	0.004	0.0245	1.008 (1.001–1.016)	—
BAR*	Standard MLE	0.345	0.100	0.0006	31.470 (4.432–223.439)	1.412 (1.161–1.718)
	Firth-penalized	0.297	0.094	0.0008	19.551 (3.111–122.880)	1.346 (1.121–1.616)

*BAR coefficients (B) and SEs are scaled to represent a clinically meaningful 0.1-unit increase, aligning with our institution's albumin unit measurement of g/L, to avoid mathematical extrapolation bias. Profile-likelihood CIs are reported for the Firth-penalized sensitivity model, which was used to adjust for small-sample bias and the low event-to-variable ratio (18 deaths). The reference category for sex is male. **Abbreviations**: BAR, blood urea nitrogen-to-albumin ratio; CI, confidence interval; LVEF, left ventricular ejection fraction; MLE, maximum likelihood estimation; OR, odds ratio; SE, standard error.

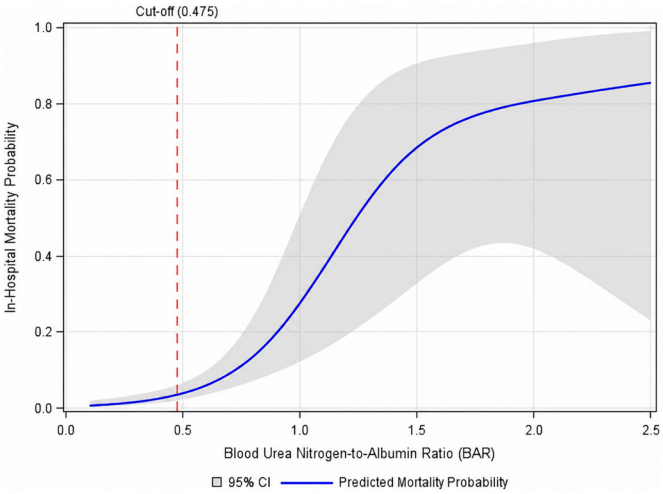


Figure 1. Restricted cubic spline curve for the association between admission BAR and in-hospital mortality. The solid blue curve represents the estimated probability of in-hospital mortality across admission BAR values, and the shaded area represents the 95% CI. The analysis suggests a nonlinear increase in mortality probability with increasing BAR. The vertical dashed line marks the exploratory BAR cut-off of 0.475, derived from ROC analysis using Youden's index. Because this cut-off was derived and evaluated in the same cohort, it should not be interpreted as a validated clinical threshold. **Abbreviations**: BAR, blood urea nitrogen-to-serum albumin ratio; CI, confidence interval; ROC, receiver operating characteristic.

cut-off value of 0.475 demonstrated high sensitivity (83.3%) and specificity (75.4%). Furthermore, the high negative predictive value (98.9%) and low negative likelihood ratio (0.22) observed at this exploratory cut-off may suggest potential utility for identifying patients at lower risk of in-hospital mortality. However, these findings should be interpreted cautiously, as the cut-off was derived and tested in the same cohort, and the high negative predictive value may be influenced by the low event rate.

Multivariable stepwise logistic regression identified gender, LVEF, sodium, platelet count, and BAR as candidate covariates associated with in-hospital mortality. While BAR was associated with in-hospital mortality in the exploratory model, the wide CI for the BAR estimate (95% CI: 4.432–223.439 per 1.0-unit increase) and low event-to-variable ratio suggest potential overfitting. Therefore, these multivariable regression results should be interpreted as exploratory and hypothesis-generating rather than definitive. Among demographic factors, only gender remained statistically significant in the exploratory model. A possible explanation is that age and culprit lesion location might influence mortality primarily through metabolic and hemodynamic parameters, which could be reflected by the BAR. Although several laboratory parameters showed significant univariate associations with mortality—including lower LVEF, elevated glucose and ALT, impaired renal function, hyponatremia, and lower lipid and hemoglobin levels—most did not retain statistical significance in the exploratory model. This pattern suggests that the BAR provides more comprehensive prognostic stratification by

Table 4. ROC analysis—predicting in-hospital mortality with the BAR

Biomarker	AUC (95% CI)	*P* value	Cut-off	Sensitivity (95% CI, %)	Specificity (95% CI, %)	PPV (%)	NPV (%)	LR+	LR−
BAR	0.869 (0.794–0.945)	<0.001	0.475	83.3 (60.8–94.2)	75.4 (70.8–79.5)	14.1	98.9	3.39	0.22

Abbreviations: AUC, area under the curve; BAR, blood urea nitrogen-to-albumin ratio; CI, confidence interval; LR+, positive likelihood ratio; LR−, negative likelihood ratio; NPV, negative predictive value; PPV, positive predictive value; ROC, receiver operating characteristic.

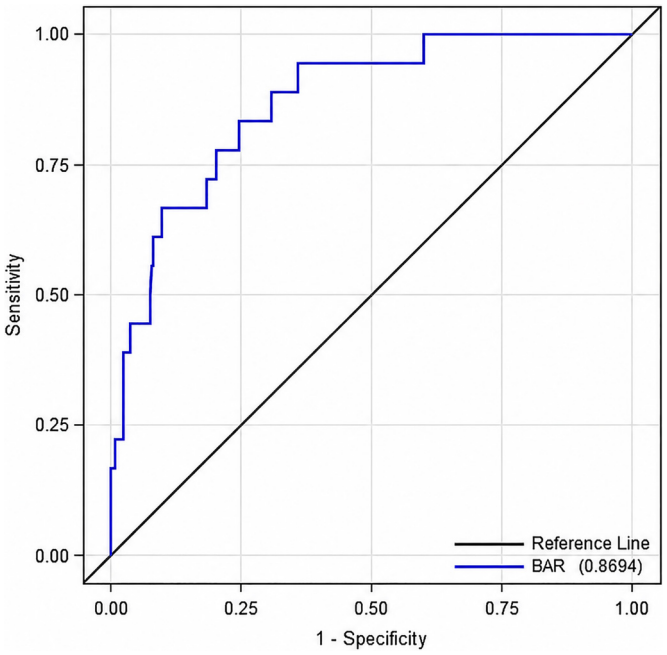


Figure 2. ROC curve of the admission blood urea nitrogen-to-serum albumin ratio for in-hospital mortality. The ROC curve shows the discriminatory performance of admission BAR in 388 patients with STEMI undergoing pPCI, including 18 in-hospital deaths. BAR yielded an AUC of 0.869 (95% CI, 0.794–0.945; $P < 0.001$). The exploratory cut-off value of 0.475, identified using Youden's index, provided 83.3% sensitivity and 75.4% specificity. Because this cut-off was derived and evaluated in the same cohort, it should not be interpreted as a validated clinical threshold. The diagonal reference line represents discrimination equivalent to chance. **Abbreviations**: AUC, area under the curve; BAR, blood urea nitrogen-to-serum albumin ratio; CI, confidence interval; pPCI, primary percutaneous coronary intervention; ROC, receiver operating characteristic; STEMI, ST-elevation myocardial infarction.

integrating renal function, neurohumoral activation, and metabolic status more effectively than individual parameters. While CAR and NLR have been reported to predict adverse outcomes in some AMI populations, these markers did not show significant associations with in-hospital mortality in our study, whereas the BAR showed an association with mortality in this exploratory analysis [10, 11]. Standard MLE and Firth-penalized estimates for BAR per 0.1-unit increase were broadly consistent (MLE OR = 1.412, 95% CI: 1.161–1.718, $P = 0.0006$; Firth OR = 1.346, 95% CI: 1.121–1.616, $P = 0.0008$); however, these findings should be interpreted as exploratory.

Our findings are largely consistent with current literature regarding the BAR's ability to predict mortality in cardiovascular events [3, 13]. The AUC value of 0.869 obtained in our study demonstrates that the BAR has higher predictive power for in-hospital mortality in STEMI patients compared to a previous report [3]. Beyond STEMI, the prognostic utility of the BAR has also been demonstrated in broader cardiovascular populations, including CICU patients with AMI and non-diabetic non-ST-elevation myocardial infarction (NSTEMI) patients, with comparable predictive performance [5, 6]. The most important distinction of our study from existing literature is its presentation of the BAR not as a short- or long-term risk marker, but as an early, rapid, and highly sensitive prognostic tool, specifically for acute in-hospital mortality in the STEMI-pPCI setting.

Although albumin units are commonly expressed as g/dL in the literature, our study used g/L units. When our identified cut-off value of 0.475 is normalized (4.75), it shows similarity to the value of 4.0 identified by Balcık et al. in STEMI patients, confirming that mortality risk significantly increases in the 4.0–5.0 BAR range in STEMI presentations [3]. The higher cut-off values identified in heart failure or general intensive care unit (ICU) patients (6.82 and 7.83) may reflect more advanced baseline metabolic dysfunction in these populations compared to acute STEMI patients [8, 13].

In addition to BUN, estimated GFR—another indicator of renal function—has been shown to be associated with AMI prognosis [14]. However, clinical studies have demonstrated that estimated GFR measured by creatinine is not a

precise indicator of renal function [15, 16]. BUN is normally an end product of protein metabolism excreted through the kidneys. Multiple other factors such as systemic hypoperfusion, decreased cardiac output, and neurohumoral activation also affect BUN concentration, frequently occurring in the acute phase of myocardial infarction [17]. Therefore, in AMI patients, BUN reflects not only cardio-renal function but also neurohumoral activation. Saygitov and colleagues demonstrated that elevated BUN levels, rather than creatinine, are a more important risk factor for adverse outcomes in acute coronary syndrome [18]. Serum albumin levels in clinical presentations such as AMI reflect not only nutritional status but also the body's vascular and metabolic resistance, serving as a multifunctional parameter [6]. Specifically, its independent prognostic value is driven by crucial antioxidant, anti-inflammatory, endothelial-protective, and anticoagulant activities [6]. Given these broad biological roles, comprehensive immunonutritional indices incorporating albumin have emerged as valuable prognostic tools. For instance, recent evidence demonstrates that the Controlling Nutritional Status (CONUT) score, which integrates serum albumin, total cholesterol, and lymphocyte count, independently predicts long-term mortality and exhibits superior prognostic performance compared to other nutritional indices in elderly patients with heart failure [19]. Severe systemic inflammation, which can develop after STEMI, suppresses albumin synthesis in the liver. Decreased serum albumin levels have been shown to be associated with endothelial dysfunction through increased blood viscosity, platelet aggregation, and platelet-mediated coronary artery narrowing [20, 21]. Low albumin levels have been found to be associated with in-hospital adverse events in STEMI patients [22, 23]. Consequently, the BAR functions as a holistic indicator simultaneously reflecting the patient's renal function, neurohumoral activation, nutritional status, vascular stability, and systemic inflammatory burden.

In this cohort, admission BAR was associated with in-hospital mortality. However, GRACE and TIMI scores were not calculated or compared in our study. Therefore, the prognostic performance of BAR relative to established risk scores cannot be determined. BAR may be a simple candidate prognostic marker that warrants prospective validation and direct comparison with established risk scores.

Limitations

This study has several limitations. First, its single-center, retrospective design limits the generalizability of our findings and restricted access to complete data on certain acute clinical and procedural variables, such as Killip class, symptom-to-door time, door-to-balloon time, and TIMI flow. Second, the relatively small number of mortality events ($n = 18$) introduces a risk of overfitting in our multivariable model, as reflected by the wide CIs. Consequently, our regression estimates are exploratory and require validation in larger cohorts. Third, GRACE and TIMI scores were not calculated or compared, preventing an assessment of BAR's prognostic performance relative to established risk scores. Fourth, BAR values were measured only once at admission; thus, the effects of dynamic changes in BUN and albumin levels during hospitalization on mortality could not be evaluated. Finally, only in-hospital mortality was examined, while long-term cardiovascular outcomes were not.

Conclusion

In this exploratory cohort study of STEMI patients undergoing pPCI, higher admission BAR levels were associated with an increased risk of in-hospital mortality. While the BAR represents a simple, rapid, and easily accessible candidate prognostic marker, its true predictive performance relative to established risk scores and its clinical utility must be confirmed in larger prospective, multi-center studies before integration into routine clinical practice.

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