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Review Article

Blood urea nitrogen as outcome predictor in acute coronary syndrome: A systematic review and meta-analysis

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ABSTRACT

Background: Predicting outcomes in acute coronary syndrome (ACS) remains challenging, as established risk scores often require variables that are unavailable in low-resource healthcare settings. Blood urea nitrogen (BUN) has demonstrated prognostic value in predicting cardiovascular disease outcomes, such as heart failure and infective endocarditis. However, no meta-analysis has yet evaluated its predictive role in ACS.
Objective: This study evaluated the prognostic utility of BUN for mortality and major adverse cardiac events (MACE) in ACS.
Methods: A systematic review and meta-analysis were conducted using literature from PubMed, Cochrane, and Web of Science. Risk of bias was assessed using the Risk Of Bias In Non-randomized Studies-of Exposures (ROBINS-E) tool. Pooled analysis of hazard ratios (HR) was calculated utilizing a random-effects model based on restricted maximum likelihood method. Subgroup analysis and meta-regression were performed. Sensitivity analysis was done using graphical display of study heterogeneity.
Results: Ten studies consisting of 7,238 participants were included. Elevated BUN was associated with heightened risk of MACE and mortality (HR: 1.05, 95% CI: 1.03–1.07, p=0.0011) and remained significant after excluding two outlier studies (HR: 1.04, 95% CI: 1.02–1.05, p=0.0002). Univariate meta-regression identified age, hypertension, and diabetes as potential covariates (p=0.112, 0.221, and 0.194). Multivariate analysis revealed no independent predictors.
Conclusion: BUN may serve as a promising biomarker for predicting MACE and mortality in ACS, particularly in resource-limited settings. Further research is needed to compare its performance with established biomarkers or traditional scoring systems.

1. Introduction

Acute coronary syndrome (ACS) encompasses a range of clinical manifestations arising from acute myocardial ischemia, including unstable angina, non-ST-segment elevation myocardial infarction (NSTEMI), or ST-segment elevation myocardial infarction (STEMI).[1] Myocardial infarction (MI) occurs when irreversible ischemic damage causes myocardial necrosis, resulting in greater morbidity and mortality than unstable angina.[2,3] ACS is an emerging predominant cause of death in the Asia-Pacific region, representing approximately half of the global burden. Its in-hospital mortality rate generally surpasses five percent.[4] Several traditional scoring systems have been developed, such as the Global Registry of Acute Coronary Events (GRACE) and Thrombolysis in Myocardial Infarction (TIMI) scores.[5,6] However, these scores require variables like troponin levels or angiography, which may be unavailable in low-resource settings.

Blood urea nitrogen (BUN) is a routine marker of renal function, inexpensive and widely available in many healthcare settings. BUN not only indicates renal function but also acts as a significant measure of neurohormonal activity.[7] Reduced renal clearance, as in acute or chronic renal failure or impairment, increases BUN level.[8] Its role as an emerging prognostic biomarker for cardiovascular diseases has been studied extensively.[9] Several studies have shown that BUN is a significant prognostic predictor for specific conditions such as heart failure[7,10] and infective endocarditis.[11]

Although the prognostic significance of BUN has been reflected in heart failure and infective endocarditis, the predictive role in ACS remains inadequately examined, particularly in meta-analyses. Previous research indicates that BUN is a more reliable predictor of ACS outcomes compared to creatinine; however, no meta-analysis has thoroughly assessed its predictive value in ACS.[12,13] Therefore, the objective of this study was to determine the prognostic value of BUN for mortality and adverse outcomes in ACS patients.

2. Method

Data Source and Search Methodology

This systematic review and meta-analysis referred to the Preferred Reporting Items for Systematic Reviews and Meta-Analysis 2020 (PRISMA 2020) guidelines. PubMed, Cochrane, and Web of Science were used to search the literature in April 2025 using predefined keywords listed in **Table S1**. PROSPERO registration was done under the registration number CRD420251031485.

Study Selection and Risk of Bias Assessment

The selection of eligible studies was done by one reviewer (GN). This review consisted of inclusion criteria that were established based on the objective of this study: 1) studies that examined the prognostic value of BUN as a numerical variable, 2) the population was diagnosed with acute coronary syndrome spectrum, such as STEMI, NSTEMI, and UAP. Retrospective, prospective, or prognostic study designs were sought as eligible. Studies that presented BUN as categorical data, included patients with end stage renal disease and mortality in less than 24 hours, study participants consisted of patients

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Table 1. Participant baseline data of included studies.

Characteristic	K	Mean or Proportion	95% CI*		I^2
			Lower	Upper	
Male	10	0.6787	0.6349	0.7195	90.7%
Smoking	7	0.4093	0.3461	0.4756	86.5%
Age	10	67.5888*	64.5828	70.5949	98.6%
SBP	6	94.8570*	73.5682	116.1458	99.9%
DBP	3	67.7621*	55.1206	80.4036	99.8%
Hypertension	8	0.4613	0.3046	0.6260	98.7%
Diabetes	9	0.3636	0.3095	0.4214	94.4%
Previous IHD	6	0.1369	0.0944	0.1945	85.3%
Creatinine	8	1.1744	1.0367	1.3121	96.4%

*: Data presented in mean

Table 2. Result of meta-regression in corresponding to major adverse cardiac event and mortality outcome. Age, hypertension, and diabetes were identified as covariates.

Covariate	K	Intercept	*p*-value	β	*p*-value	95% CI*		I^2
						Lower	Upper	
Male	10	0.0622	0.0194	-0.0165	0.4829	-0.0681	0.0352	96.98%
Smoking	7	0.0481	0.0268	-0.0406	0.2307	-0.1173	0.0360	91.51%
Age	10	-0.1842	0.1934	0.0034	0.1120	-0.0010	0.0079	95.41%
SBP	6	0.0123	0.8446	0.0005	0.4992	-0.0012	0.0021	90.74%
DBP	3	-0.1083	0.7155	0.0026	0.5761	-0.0397	0.0449	95.86%
Hypertension	8	0.0600	0.0014	0.0199	0.1067	-0.0058	0.0455	88.78%
Diabetes	9	0.0358	0.0040	-0.0116	0.0382	-0.0224	0.0008	85.67%
Previous IHD	6	0.0536	0.2652	0.0016	0.9397	-0.0541	0.0574	89.01%
Creatinine	8	0.0616	0.4271	-0.0153	0.8062	-0.1612	0.1306	97.44%

younger than 18 years old, and did not provide complete extractable data were excluded. Risk of bias (ROB) assessment was conducted using the Risk Of Bias In Non-randomized Studies - of Exposures (ROBINS-E) by all reviewers, and the judgement of risk was categorized into low, moderate, or high. A consensus meeting was arranged if there was a disagreement between reviewers. The characteristics of each study were also summarized in **Table S2.**

Data Extraction

Last name of the first author and year of publication, number of individuals with a smoking history, mean age (years), recorded systolic and diastolic blood pressure (mmHg), number of participants with previous history of ischemic heart disease (IHD), number of participants with hypertension, number of participants with diabetes, creatinine (mg/dL), hazard ratio and 95% confidence interval were extracted. Extraction of numerical data in mean and standard deviation (SD) were done and estimated using Wan et al. [14] formula for the numerical data that were reported in median and interquartile range or range.

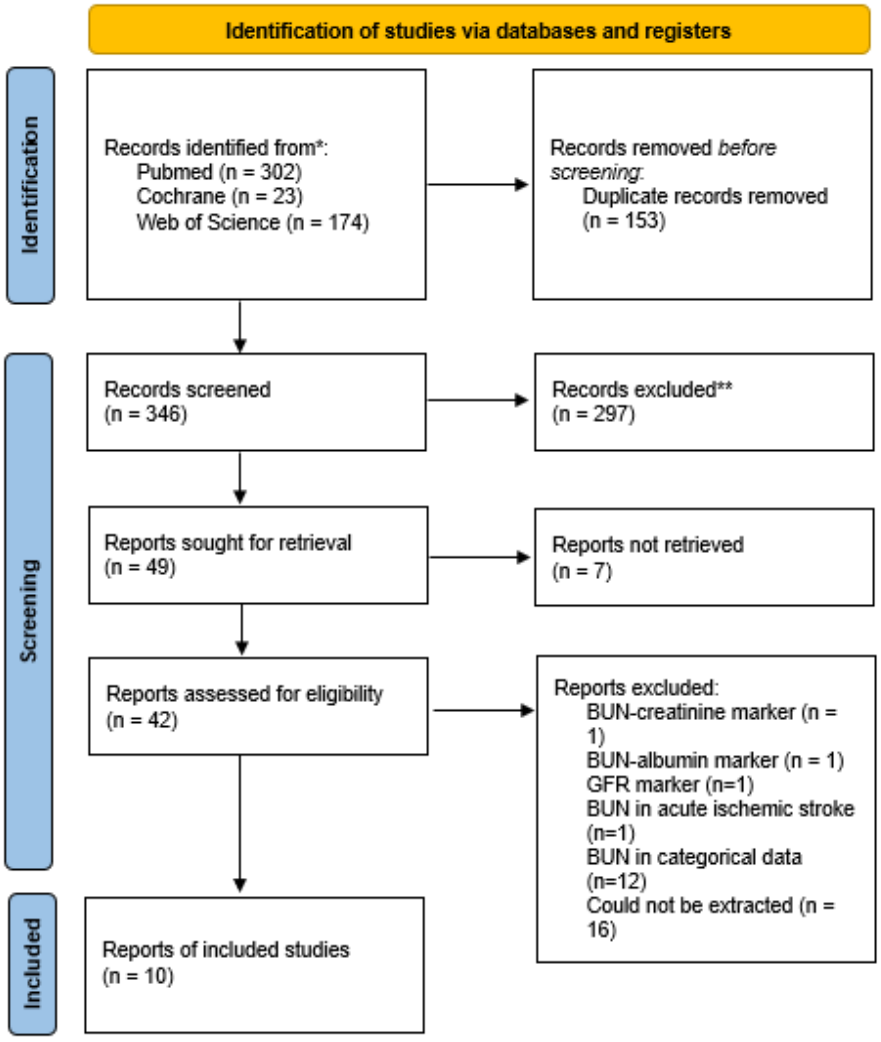
Data Analysis

All analyses were conducted using RStudio version 4.3.1 (R Foundation for Statistical Computing, Vienna, Austria). The analysis was conducted by two reviewers (GN, FH) utilizing the [meta], [metafor] and [dmetar] packages. A heterogeneity estimation of random-effects meta-analysis was conducted using restricted maximum likelihood (REML), and the Hartung-Knapp adjustment was applied under the assumption of unanticipated heterogeneity among participants. The pooled effect size was calculated using the generic inverse-variance method. To accommodate this, the standard errors of representing HR were obtained using the formula provided by The Cochrane Handbook for Systematic Reviews of Interventions. The significance level was established at 0.05. The heterogeneity of studies was estimated using the Cochrane Q and I^2 statistics. The heterogeneity was categorized according to I^2 values of 25-50% (low), 50-75% (moderate), and >75% (high), respectively. The significance level was set at 0.10. The evaluation of publication bias was demonstrated through a funnel plot and statistically analyzed via Egger's approach. A *p*-value lower than 10% indicated significant publication bias.

Meta-regression analysis was planned using numerical or categorical data – number of participants with a history of smoking, mean age, SBP and DBP, number of participants with a previous history of IHD, number of participants with hypertension, number of participants with diabetes, mean of creatinine –to observe the possible covariates that may influence the pooled effect size and heterogeneity.

Table 3. Multivariate analysis of significant variables in univariate analysis. No significant covariate was identified.

Covariate	K	Intercept	*p*-value	β	*p*-value	95%CI*		I^2
						Lower	Upper	
Age	10	-0.0665	0.4565	0.0018	0.2578	-0.0031	0.0067	66.32%
Hypertension	8			0.0827	0.1360	-0.0639	0.2294	
Diabetes	9			0.0033	0.6383	-0.0229	0.0296	
Smoking	7			-0.0041	0.7929	-0.0629	0.0547	



The multivariable meta-regression was conducted in three steps by including variables from the univariable meta-regression analysis with *p*-value <0.25. First, the collinear variable was explored using Pearson's correlation. If collinearity were detected, the analysis of these predictors would be done separately. Second, identify interacting variables; if any exist, they would be included in multivariable analysis. Third, a multivariable meta-regression analysis was conducted in a single step.

Sensitivity analysis was planned in four methods. Initially, fixed-effect analysis was employed to investigate the small-study impact. A random-effects model with Hartung-Knapp adjustment was employed. Third, studies deemed to possess significant or high risk of bias were omitted. Ultimately, papers identified as substantial contributions to the impact magnitude via graphical display of study heterogeneity (GOSH) were omitted.

3. Result

Study Selection and Study Characteristics

A sum of 499 articles was identified from 3 databases. Following the elimination of duplicates, 346 publications were evaluated based on their titles and abstracts, yielding 49 articles. Seven full articles were not retrieved, leaving 42 articles to be assessed for eligibility. Thirty-two articles did not fulfill the eligibility criteria for several reasons, as recorded in **Figure. 1.** The 10 remaining articles were observational studies.[15–24] The studies were mostly conducted in China, while the remaining studies conducted in Saudi Arabia, Turkey, and Taiwan. ROB assessment overall showed low risk of bias as shown in **Figure. 2** and **Figure. S1.**

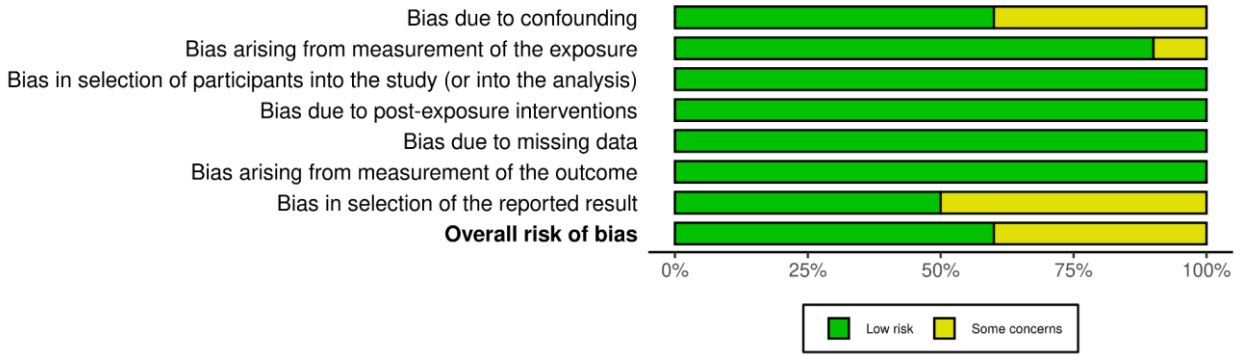


Figure 2. Summary of risk of bias assessment

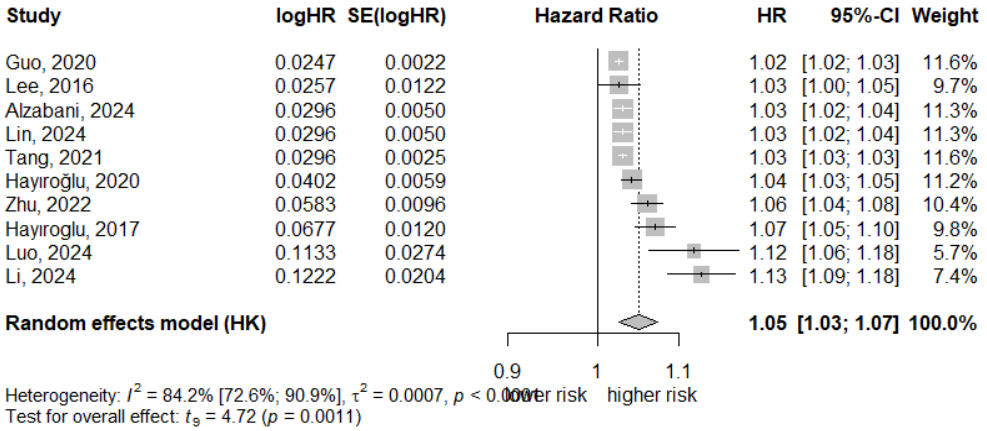


Figure 3. Forest plot of BUN level with risk of MACE and mortality. MACE and mortality are significantly higher as the BUN increases.

This study included 7,238 participants, predominantly male. The mean age of studied population was 67.59 years (95% CI 64.58-70.59). The means of SBP and DBP were 94.86 mmHg (95% CI 73.57-116.16) and 67.76 mmHg (95% CI 55.12-80.40), respectively. More than half of the participants did not suffer from hypertension, diabetes, or have a history of smoking. Around 13.69% of participants had no prior history of IHD. The mean creatinine level was 1.12 (95% CI 1.04-1.31), indicating that most of the participants had a slight increase in creatinine. Further baseline characteristic information is summarized in **Table 1.**

Association of Blood Urea Nitrogen with Major Adverse Cardiac Events and Mortality

In a meta-analysis including 10 articles, the association between MACE and mortality with BUN was analyzed. Higher BUN levels were associated with a 5% increase in risk (HR 1.05; 95% CI 1.03-1.07; **Figure. 3**) than the lower BUN levels. However, heterogeneity between studies was high (I^2 84.2%; p < 0.0001). Subgroup analysis of BUN level association with mortality and MACE risk was also conducted based on the type of ACS as shown in **Figure.4**. Six studies consisted of all type of ACS showed significant result of increased outcome as the increased of BUN level (HR 1.04; 95% CI 1.01-1.06; I^2 77.6%).[15,16,21–24] The association of BUN level with outcome within STEMI population was analyzed in three studies.[17–19] Pooled HR analysis revealed insignificant association with HR 1.05 (95% CI 0.99-1.1; I^2 69.1%). Pooled HR for NSTEMI population could not be analyzed due to the single publication only.[20]

Funnel plot asymmetry was assumed either by funnel plot visualization or Egger's (p=0.0019) indicating a presence of publication bias (**Figure. 5a**). Sensitivity analysis identified two studies that disproportionately contribute to the effect size, shown by the decrease of heterogeneity from 84.2% to 74.1% (**Figure. 5b**).[20,22] Nevertheless, after excluding those studies using GOSH analysis, the risk of MACE and mortality in high BUN remained higher as well as the heterogeneity (HR 1.04; 95% CI 1.02-1.05; I^2 74.1%; p=0.0002; **Figure. 5b**). GOSH analysis for subgroup population revealed decreased heterogeneity after excluding two studies in all population subgroup, yet the test for random-effects model remains significant (HR 1.03; 95% CI 1.02-1.05; I^2 68.4%; p=0.0026).[22] There was no significant contributor to the result in STEMI group.

Meta-regression Analysis

Meta-regression was carried out, identifying age, hypertension, and diabetes as covariates (**Table 2**). The p-values of them were respectively 0.11, 0.22, and 0.19. Those covariates were then proceeded to be analyzed for possible correlation with Pearson's correlation. **Figure 6.** showed no significant correlation between age, hypertension, and diabetes. All VIF values were <5, confirming that there was no significant correlation between the possible covariates, which allowed them to be analyzed for multivariate analysis in one go. **Table 3** summarizes the multivariate analysis result, which included covariates that had p-value <0.25 in the univariate analysis. Based on the analysis, no significant covariate was detected, which shows that age, hypertension, and diabetes did not contribute to increased risk of MACE and mortality in ACS.

4. Discussion

This study demonstrates a significant result that higher BUN levels are linked to higher risk of MACE and mortality in ACS. This finding underscores the prognostic significance of BUN, not only in renal function but also for cardiovascular outcome, which may serve as a simple yet inexpensive biomarker. Due to its low cost and accessibility, incorporating BUN measurement into early risk stratification for ACS may help identifying high-risk patients and predict the risk of poor outcomes. This bolsters the argument for including BUN as part of standard admission assessments in ACS settings. To our knowledge, this is the first meta-analysis to evaluate this association and estimate a quantitative pooled effect size across various ACS populations.

Blood urea nitrogen (BUN) is a serum byproduct resulting from protein metabolism. Urea is synthesized by the liver and transported via the bloodstream to the kidneys for elimination.[25]

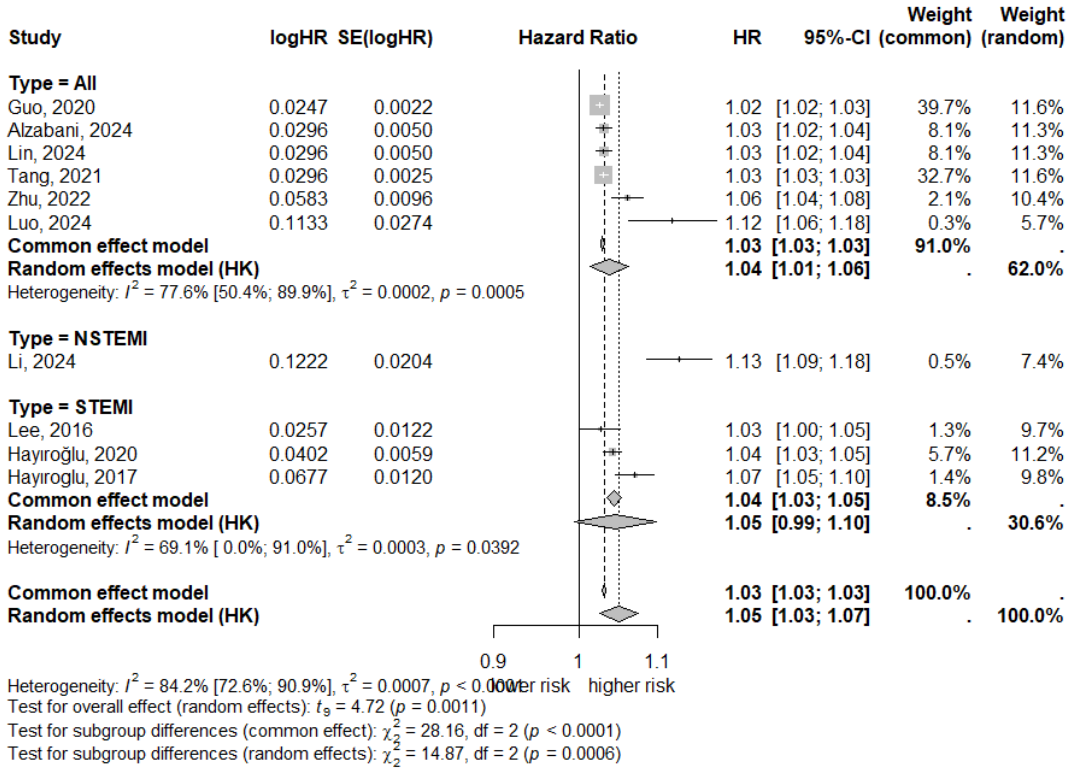


Figure 1. Forest plot of subgroup analysis based on type of ACS. BUN level was significantly associated with increased risk of MACE and mortality in all ACS but not for STEMI.

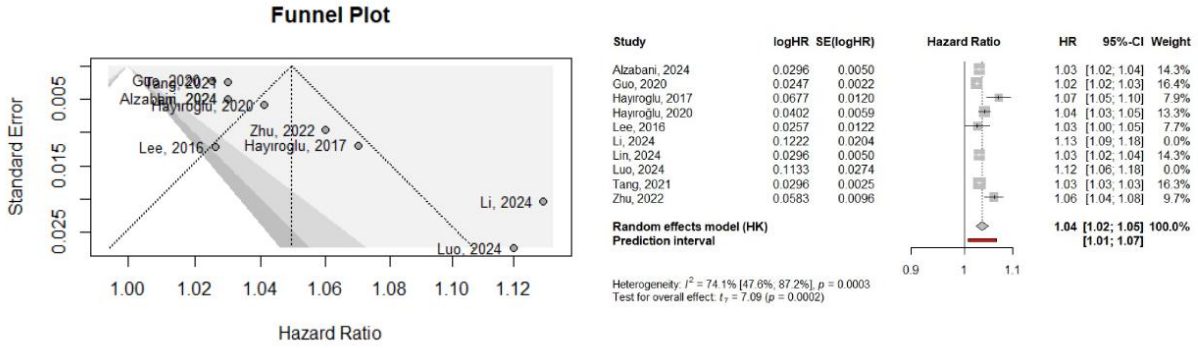


Figure 5a. Funnel plot of BUN level with risk of MACE and mortality. MACE and mortality were considered to be significantly increased as the BUN increased. Egger's test (p=0.0019) indicates the presence of publication bias. **Figure 5b.** GOSH analysis showed two significant contributor studies, which affected the heterogeneity.[16,23]

Elevated BUN level may reflect reduced glomerular filtration rate (GFR), activation of sympathetic nerve systems and renin-angiotensin-aldosterone (RAA), which may result from renal hypoperfusion as a consequence of hypovolemia, renovascular disease, and decreased cardiac output.[26] In the context of ACS, modifications in cardiac output, the use of angiotensin-converting enzyme inhibitors or diuretics, stimulation of neurohormonal vasoconstrictor systems, and the use of contrast media during revascularization procedures may result in an acute decline in renal function.[27]

The bidirectional relationship between BUN and ACS pathophysiology warrants attention. On one hand, increased BUN level might reflect renal dysfunction, which is proven to have an association with coronary artery disease (CAD).[28] Kidney disease has been recognized to induce endothelial dysfunction, which contributes to the mechanism of atherosclerosis.[29] Conversely, ACS induces hemodynamic changes and neurohormonal activation (e.g., vasopressin release), which causes reabsorption of BUN, creating a vicious cycle that exacerbates cardiovascular injury through promotion of oxidative stress affecting the vascular wall.[30] Therefore, this interplay suggests that increased BUN could serve as both a contributor and/or consequence of ACS.

Findings from Adam et al.[31] further enhance the hypothesis of BUN prognostic utility in ACS, revealing a significant positive association between BUN and Troponin-I, which suggests that elevated BUN may signify increased myocardial injury alongside renal hypoperfusion. The study also demonstrated that patients with higher GRACE scores had markedly elevated BUN levels, which support its role as an integrated marker of hemodynamic stress and neurohormonal activation. These results complement our meta-analysis by emphasizing that BUN captures both renal and cardiovascular dysfunction, strengthening the case for its incorporation into early risk stratification strategies for ACS.

Several studies have shown the superiority of BUN to predict outcome in acute coronary syndrome. Compared to creatinine, BUN demonstrates a more significant result for predicting mortality and in-hospital adverse outcome.[12,13,32] Zhu et al.[24] further demonstrated that BUN outperforms creatinine, BUN-to-creatinine ratio, and estimated

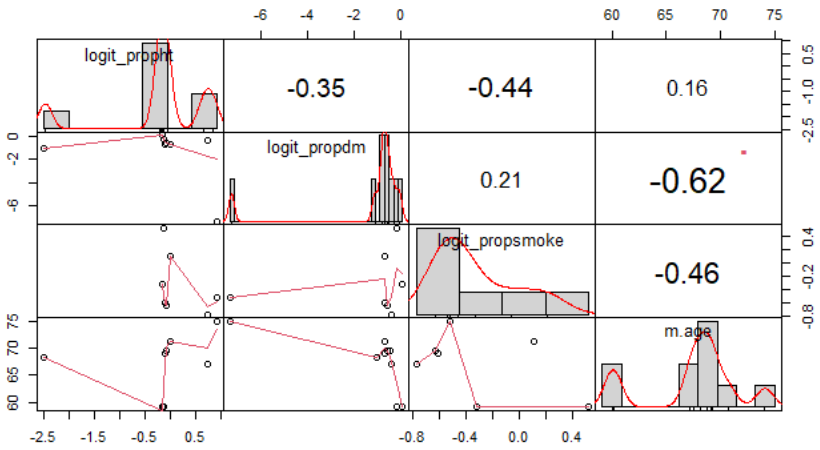


Figure 6. Pearson's correlation plot showed no significant correlation between age, hypertension, and diabetes

GFR (eGFR) in predicting short-term mortality in MI complicated by cardiogenic shock. It showed that BUN remains the most effective predictor compared to other biomarkers.

Compared with eGFR, BUN may better reflect acute hemodynamic changes and neurohormonal disturbance which make it a more sensitive predictor of adverse outcomes than eGFR. The rise of BUN not only occurs along with the deterioration of eGFR but also may reflect an increase of sodium and water reabsorption due to the activation of RAA system in insufficient cardiac output conditions. In this setting, the elevation of BUN may represent adjusted systemic hypoperfusion even when eGFR remains preserved.[24] In contrast, a decline of eGFR may reflect either acute kidney injury (AKI) or chronic kidney disease (CKD), which carries distinct prognostic implications, yet are often not differentiated in observational studies. The inclusion of participants with underlying CKD may confound the prognostic value of eGFR.

However, BUN levels may be affected by hydration status, dietary protein consumption, gastrointestinal bleeding, and catabolic conditions, necessitating cautious interpretation in clinical practice.[33] This further elaborates that the high heterogeneity in the studied populations might be influenced by the various spectra of ACS and its complicating factors, such as cardiogenic shock or GI bleeding. Gastrointestinal bleeding, particularly from the upper tract, is associated with elevated blood urea nitrogen levels due to the digestion of significant quantities of blood or protein in the gastrointestinal system, resulting in increased urea synthesis.[34,35] Meanwhile, in cardiogenic shock, several factors might contribute to AKI which decreased GFR and increased BUN due to renal hypoperfusion and use of drugs as mentioned before.[24,36]

This study has several limitations. Firstly, this study is a systematic review and meta-analysis study where the data available for extraction from the previous studies could be limited or incomplete. Those factors could affect the result of this study. Second, this study included MI which was complicated by several conditions, such as gastrointestinal bleeding and cardiogenic shock, which could confound its results. Future studies to determine the optimal cut-off value of BUN for prognostication and incorporate BUN level with the established scoring systems should be conducted to further add to its clinical value.

5. Conclusion

In summary, this study marks BUN as a simple, accessible, and effective biomarker for predicting MACE and mortality in ACS. Its strong association with the severity of cardiac injury and its superior prognostication ability compared to creatinine highlight its potential utility, especially in limited advanced testing settings. Due to its low cost and widespread availability, adding BUN into routine assessment may help to stratify risk and guide clinical decision-making. Nonetheless, larger number of studies are necessary to directly compare its predictive value with established cardiac biomarkers and standardized risk scores to elucidate its role in modern ACS management.

6 . Declaration

6.1 Ethics Approval and Consent to participate
Not applicable.

6.2. Consent for publication
Not applicable.

6.3 Availibility of data and materials
Data used in our study were presented in the main text.

6.4 Competing interests
Not applicable.

6.5 Funding Source
Not applicable.

6.6 Authors contributions
Idea/concept: GNC. Design: GNC. Control/supervision: FH. Data collection/processing: GNC. Extraction/Analysis/Interpretation: GNC, FH. Literature review: GNC, HP. Writing the article: GNC, FH, HP. Critical review: FH. All authors have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

6.7 Acknowledgements
None.

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