Clin. Lab. 2026;72:XXX-XXX

ORIGINAL ARTICLE

Increased Blood Urea Nitrogen-To-Creatinine Ratio is an Independent Predictor of Mortality in Patients with End-Stage Renal Disease

Hongyan Zhao [1, *], Hui Cheng [1, *], Chaofeng Huang [2, *], Maowen Huang [3, *], Dan Li [4], Fangchao Mei [1]

[*] *These are the co-first authors*
[1] *Department of Clinical Blood Transfusion, Huangshi Central Hospital, Affiliated Hospital of Hubei Polytechnic University, Huangshi, China*
[2] *Department of Health Management, Huangshi Central Hospital, Affiliated Hospital of Hubei Polytechnic University, Huangshi, China*
[3] *Molecluar Laboratory, The People's Hospital of Beilun District, Beilun Branch Hospital of The First Affiliated Hospital of Medical School Zhejiang University, Ningbo, China*
[4] *Pediatrics Department of Daye Maternal and Child Health Hospital, Huangshi, China*

ABSTRACT

Background: **End-stage renal disease (ESRD) is the final phase of chronic kidney disease, characterized by severe renal dysfunction that leads to metabolic disturbances in blood urea nitrogen (BUN) and serum creatinine (Scr). Currently, the relationship between the BUN/creatinine ratio (BCR) and all-cause mortality in patients with ESRD remains unclear. This study aimed to investigate the predictive value of BCR for short-term (60-day) and intermediate-term (365-day) all-cause mortality risk in patients with ESRD.**

Methods: **By using data from the MIMIC-IV database, we included patients with ESRD meeting ICD-9/ICD-10 diagnostic criteria, excluding those aged < 18 years, non-intensive care unit admissions, and those with incomplete information, ultimately enrolling 2,102 patients. The participants were stratified according to the BCR quartiles. Kaplan-Meier survival analysis and multivariate Cox regression were used to compare differences in all-cause mortality between the groups. Restricted cubic spline (RCS) analysis was used to examine the dose-response relationship between BCR and all-cause mortality. Subgroup analysis was used to assess result stability, and receiver operating characteristic (ROC) curves were used to compare the predictive performance of BCR versus individual markers (BUN or Scr).**

Results: **This study included 2,102 patients with ESRD. The results demonstrated a positive correlation between BCR and 60- and 365-day all-cause mortality rates. Multivariate Cox regression analysis revealed that each one-unit increase in BCR was associated with a 3% higher risk of 60-day all-cause mortality (HR = 1.03, 95% CI: 1.01 - 1.04) and 365-day all-cause mortality (HR = 1.03, 95% CI: 1.01 - 1.04). The RCS analysis showed a linear relationship between BCR and 60-day all-cause mortality (p-overall < 0.001, p-non-linear = 0.237), whereas a non-linear relationship was observed for 365-day all-cause mortality (p-overall < 0.001, p-non-linear = 0.018). The subgroup analysis revealed no significant interaction effects. ROC analysis indicated the superior predictive performance of BCR compared to BUN or Scr alone.**

Conclusions: **BCR serves as an independent predictor of all-cause mortality risk in patients with ESRD and may provide a novel reference index for clinical prognosis assessment.**

(Clin. Lab. 2026;72:xx-xx. DOI: 10.7754/Clin.Lab.2025.250736)

Correspondence:
Fangchao Mei
Department of Clinical Blood Transfusion
Huangshi Central Hospital
Affiliated Hospital of Hubei Polytechnic University
No. 141, Tianjin Road
Huangshi City, Hubei Province
China
Phone: + 86 7146257596
Fax: + 86 7143189999
Email: 280542194@qq.com

Manuscript accepted September 10, 2025

Dan Li
Pediatrics Department of Daye Maternal and Child Health Hospital
No. 22, East Yinjiahu Road
Daye City, Hubei Province
China
Phone: + 86 7148712997
Fax: + 86 7148712993
Email: 253750026@qq.com

KEYWORDS

blood urea nitrogen, chronic kidney disease, creatinine, end-stage renal disease, renal failure

INTRODUCTION

End-stage renal disease (ESRD) is the terminal phase of chronic kidney disease progression, marked by severe renal failure, which leads to impaired water-electrolyte balance and metabolic waste excretion [1]. These patients typically require renal replacement therapy (dialysis or kidney transplantation) for survival. Epidemiological data indicate that even with standard renal replacement therapy, the five-year survival rate for patients with ESRD remains only 39 – 60% [2], underscoring the urgent need for effective prognostic indicators.

In the current clinical practice, serum creatinine (Scr), blood urea nitrogen (BUN), and estimated glomerular filtration rate are the most commonly used laboratory parameters for renal function assessment [3,4]. These markers offer the advantages of simplicity and cost-effectiveness, making them suitable for implementation in healthcare facilities. However, individual renal function indicators have notable limitations: Scr levels are influenced by muscle mass and dietary creatine intake [5,6], while BUN concentrations vary with protein intake and catabolic state [7,8].

The BUN/creatinine ratio (BCR), a composite index, may compensate for these limitations. Existing research has demonstrated the prognostic value of BCR in various conditions: Wang et al. confirmed BCR as an independent predictor of all-cause mortality in patients with chronic heart failure [9], while another study found an association between BCR and long-term mortality in patients with renal insufficiency congestive heart failure [10]. These findings suggest the potential of BCR as a prognostic indicator for patients with ESRD.

Large-scale evidence regarding the association between BCR and mortality in patients with ESRD is lacking. Therefore, this study used the MIMIC-IV [11,12] database and Kaplan-Meier survival, multivariate Cox regression, and restricted cubic spline (RCS) analyses to systematically evaluate the relationship between BCR and short- (60-day) and intermediate-term (365-day) mortality in patients with ESRD, with the aim of providing new evidence for clinical prognosis assessment.

MATERIALS AND METHODS

Data source

This study used data from the MIMIC-IV database (version 3.1), which contains comprehensive clinical records of patients in the intensive care unit at Beth Israel Deaconess Medical Center in the United States between 2008 and 2022. The research protocol was approved by the institutional review board (ID: 40060500) and requirement for informed consent was waived due to the de-identified nature of the data.

Study population

Data extraction was performed using Structured Query Language in Navicat Premium software (version 17). Patient selection was based on International Classification of Diseases (ICD) diagnostic codes, including ICD-9 (403.01, 403.11, 403.91, 404.02, 404.03, 404.12, 404.13, 404.92, 404.93, 585.5, and 585.6) and ICD-10 (I12.0, I13.11, I13.2, N18.5, N18.6, and N26.1) codes. The exclusion criteria were as follows: non-ICU admissions, patients aged < 18 years, missing critical baseline data (e.g. gender), and duplicate records. A total of 2,102 eligible patients were included in the final analysis. The detailed patient selection process is illustrated in Figure 1.

This study collected the following variables:

- Demographic characteristics: age, gender, race
- Vital signs: systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), heart rate, respiratory rate, body temperature, and peripheral oxygen saturation (SpO_2)
- Laboratory parameters: complete blood count, white blood cell count (WBC), platelet count (PLT), hematocrit (HCT), and hemoglobin
- Electrolytes: sodium, potassium, calcium, phosphorus, bicarbonate, and chloride
- Renal function: BUN and Scr
- Other: glucose, anion gap, international normalized ratio (INR), and prothrombin time (PT)
- Comorbidities: myocardial infarction, congestive heart failure, peripheral vascular disease, dementia, rheumatic disease, peptic ulcer disease, paraplegia, chronic obstructive pulmonary disease (COPD), liver disease, and diabetes mellitus.

All laboratory values were averaged over the first 24 hours after ICU admission. Variables with < 10% missing data were imputed using the Random Forest method, whereas those with ≥ 10% missing data were excluded. Data analysis was performed using the SPSS software (version 27).

Exposure and outcomes

The primary exposure variable was the BCR, which was analyzed as follows:

1) As a continuous variable.
2) Categorized by quartiles:
Q1: BCR ≤ 6.28
Q2: 6.28 < BCR ≤ 8.97

Table 1. Impact of BCR and other factors on 60-day and 365-day mortality in ESRD patients.

	Model 1		Model 2		Model 3	
	HR (95% CI)	p-value	HR (95% CI)	p-value	HR (95% CI)	p-value
60-day mortality						
BCR	1.03 (1.01 - 1.04)	0.001	1.02 (1.01 - 1.04)	0.005	1.03 (1.01 - 1.04)	0.001
BCR quartiles						
Q1	ref		ref		ref	
Q2	1.34 (1.00 - 1.80)	0.047	1.10 (0.84 - 1.46)	0.482	1.14 (0.86 - 1.52)	0.367
Q3	1.32 (0.98 - 1.78)	0.072	1.17 (0.88 - 1.56)	0.271	1.18 (0.87 - 1.60)	0.275
Q4	1.03 (0.67 - 1.57)	0.905	0.92 (0.62 - 1.38)	0.692	0.90 (0.59 - 1.37)	0.610
p-value for trend	0.073		0.327		0.216	
365-day mortality						
BCR	1.02 (1.01 - 1.04)	< 0.001	1.02 (1.01 - 1.04)	< 0.001	1.03 (1.01 - 1.04)	< 0.001
BCR quartiles						
Q1	ref		ref		ref	
Q2	1.32 (1.08 - 1.60)	0.006	1.16 (0.95 - 1.41)	0.156	1.20 (0.98 - 1.47)	0.084
Q3	1.24 (1.00 - 1.53)	0.045	1.01 (0.81 - 1.25)	0.943	1.07 (0.85 - 1.34)	0.569
Q4	1.02 (0.75 - 1.38)	0.907	0.82 (0.60 - 1.12)	0.202	0.86 (0.62 - 1.19)	0.357
p for trend	0.011		0.063		0.052	

BCR: blood urea nitrogen/creatinine ratio.
Model 1: Unadjusted.
Model 2: Adjusted for gender, race, and age.
Model 3: Adjusted for gender, race, age, systolic blood pressure, diastolic blood pressure, mean arterial pressure, heart rate, respiratory rate, body temperature, peripheral oxygen saturation, white blood cell count, platelet count, hematocrit, hemoglobin, sodium, potassium, calcium, phosphorus, bicarbonate, chloride, blood urea nitrogen, serum creatinine, glucose, anion gap, international normalized ratio, prothrombin time, myocardial infarction, congestive heart failure, peripheral vascular disease, dementia, rheumatic disease, peptic ulcer disease, paraplegia, chronic obstructive pulmonary disease, liver disease, and diabetes mellitus.

Table 2. Comparative predictive performance of BUN, Scr, and BCR for 60- and 365-day mortality in patients with ESRD.

	AUC	p-value	95% CI	Cutoff value	Sensitivity	Specificity	Youden's index
60-day-mortality ROC							
BUN	0.492	0.602	0.463- 0 .521	113.25	0.075	0.942	0.017
Scr	0.403	< 0.001	0.376 - 0.430	47.10	0	1	0.000
BCR	0.573	< 0.001	0.545 - 0.602	8.96	0.603	0.517	0.120
365-day-mortality ROC							
BUN	0.482	0.156	0.457 - 0.507	157.75	0.016	0.993	0.009
Scr	0.409	< 0.001	0.385 - 0.434	1.38	0.977	0.003	0.007
BCR	0.557	< 0.001	0.532 - 0.581	6.24	0.816	0.277	0.093

BUN: blood urea nitrogen, Scr: serum creatinine, CR: blood urea nitrogen/creatinine ratio.

Q3: 8.97 < BCR ≤ 14.33
Q4: BCR >14.33
The primary outcomes were 60- and 365-day all-cause mortality.

Statistics

Continuous variables were assessed for normality using the Kolmogorov–Smirnov test. Normally distributed variables were presented as mean ± standard deviation and compared using Student's *t*-test, while non-normally

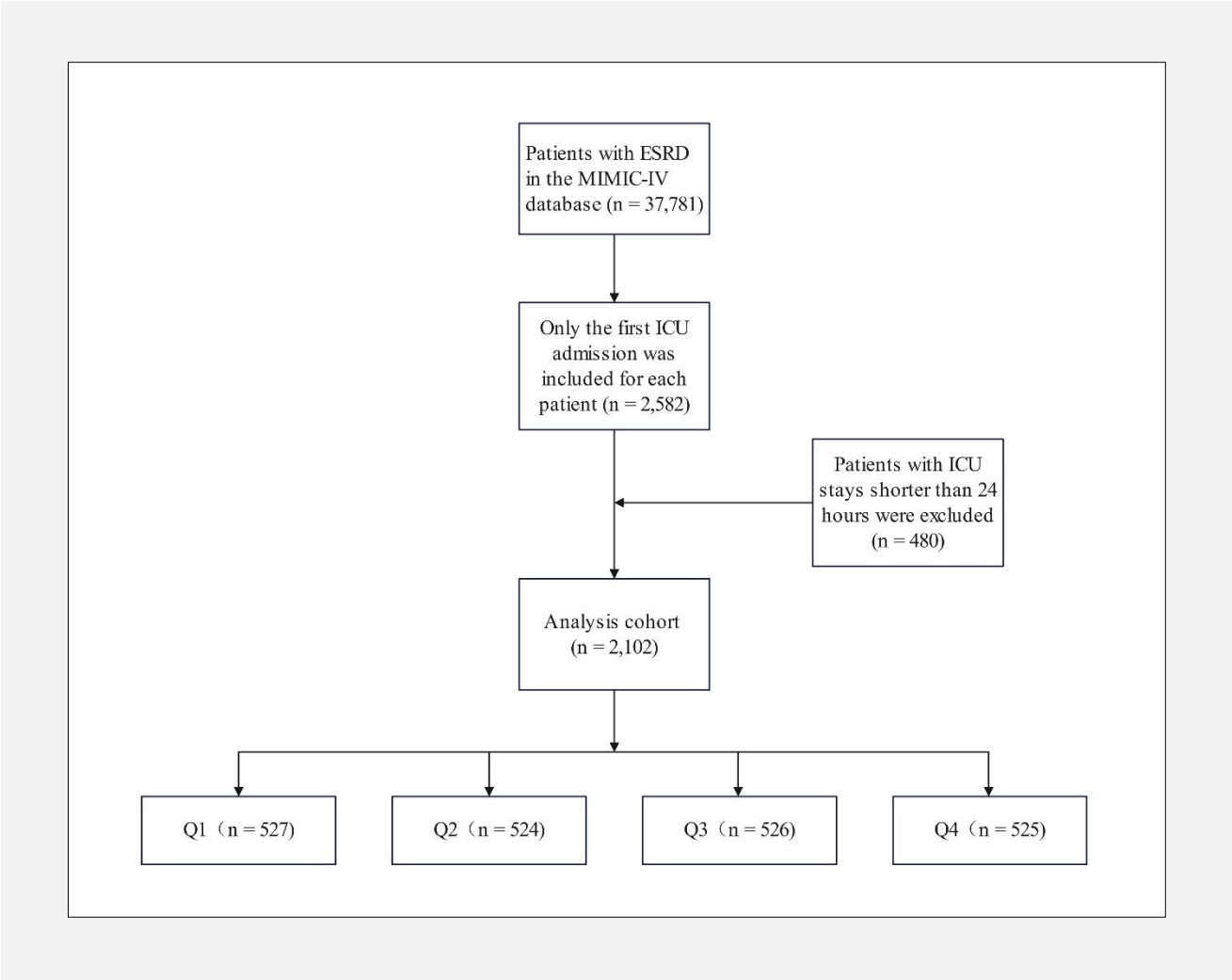

Figure 1. Study flowchart.

distributed variables were reported as median (interquartile range, Q1 - Q3) and analyzed using the Mann-Whitney U test. Categorical variables were expressed as counts (n) and percentages (%) and compared using chi-squared or Fisher's exact tests.

The association between BCR and mortality was evaluated using Kaplan–Meier survival analysis, multivariate Cox proportional hazards regression models (three progressively adjusted models), and RCS to examine potential non-linear relationships. Subgroup analyses were conducted to assess effect modification by gender, race, COPD, liver disease, and diabetes mellitus, and malignancy. Interaction terms were tested in these subgroup analyses. Receiver operating characteristic (ROC) curves were generated to compare the prognostic value of BCR with that of BUN or Scr alone.

Software used were:

- SPSS (version 27): primary statistical analyses and missing data imputation
- GraphPad Prism (version 10.1): survival curve generation
- R software (version 4.4.3): RCS analysis and plotting
- Microsoft Excel: forest plot creation for subgroup analyses

RESULTS

Baseline characteristics of patients with ESRD

Patients were categorized into four groups (Q1 - Q4) based on BCR quartiles, with comparative data presented in Supplemental Table 1. Demographic analysis revealed no statistically significant differences in gender distribution across groups, whereas higher BCR groups exhibited significantly older age ($p < 0.001$) and a greater proportion of Caucasian patients ($p < 0.001$). Laboratory parameters demonstrated significant associ-

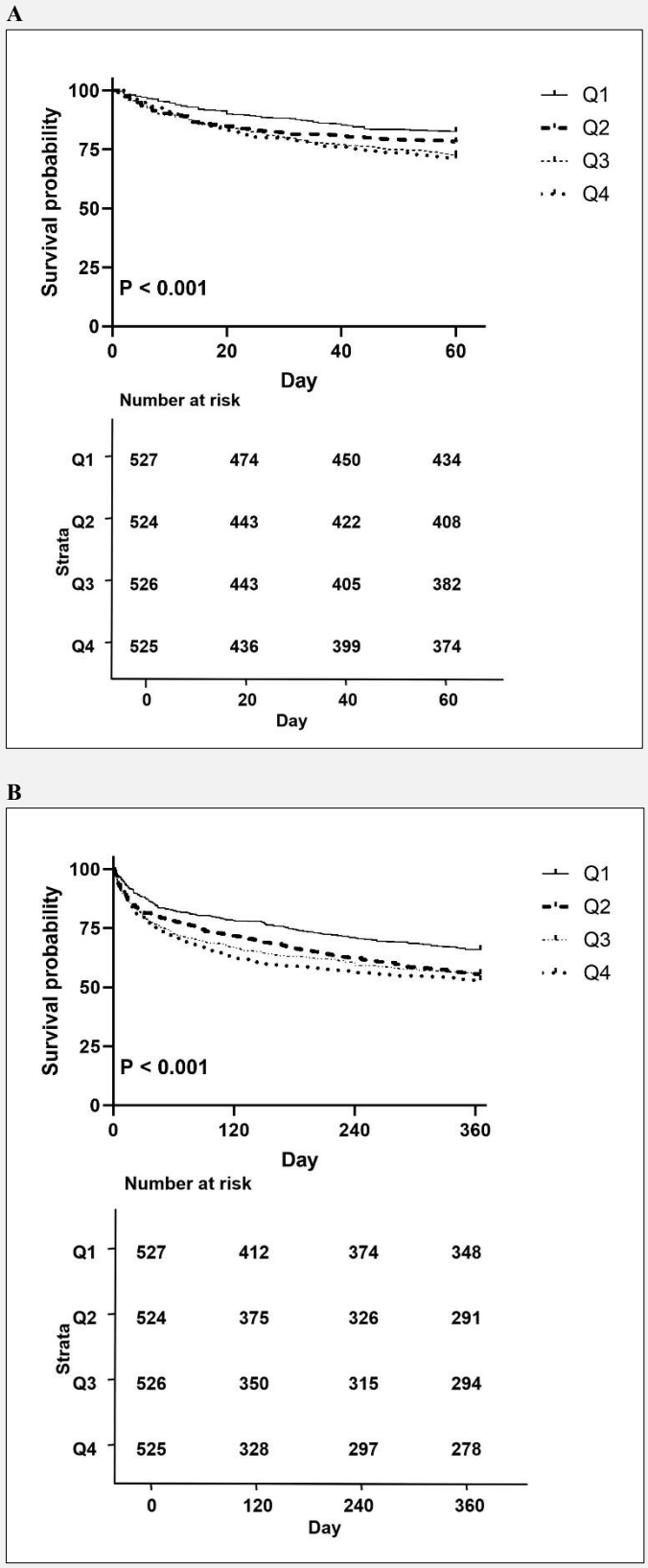

Figure 2. Kaplan–Meier survival analysis of patients with ESRD stratified using BCR quartiles.

A: 60-day mortality. B: 365-day mortality.
BCR: blood urea nitrogen-to-creatinine ratio, ESRD: end-stage renal disease.

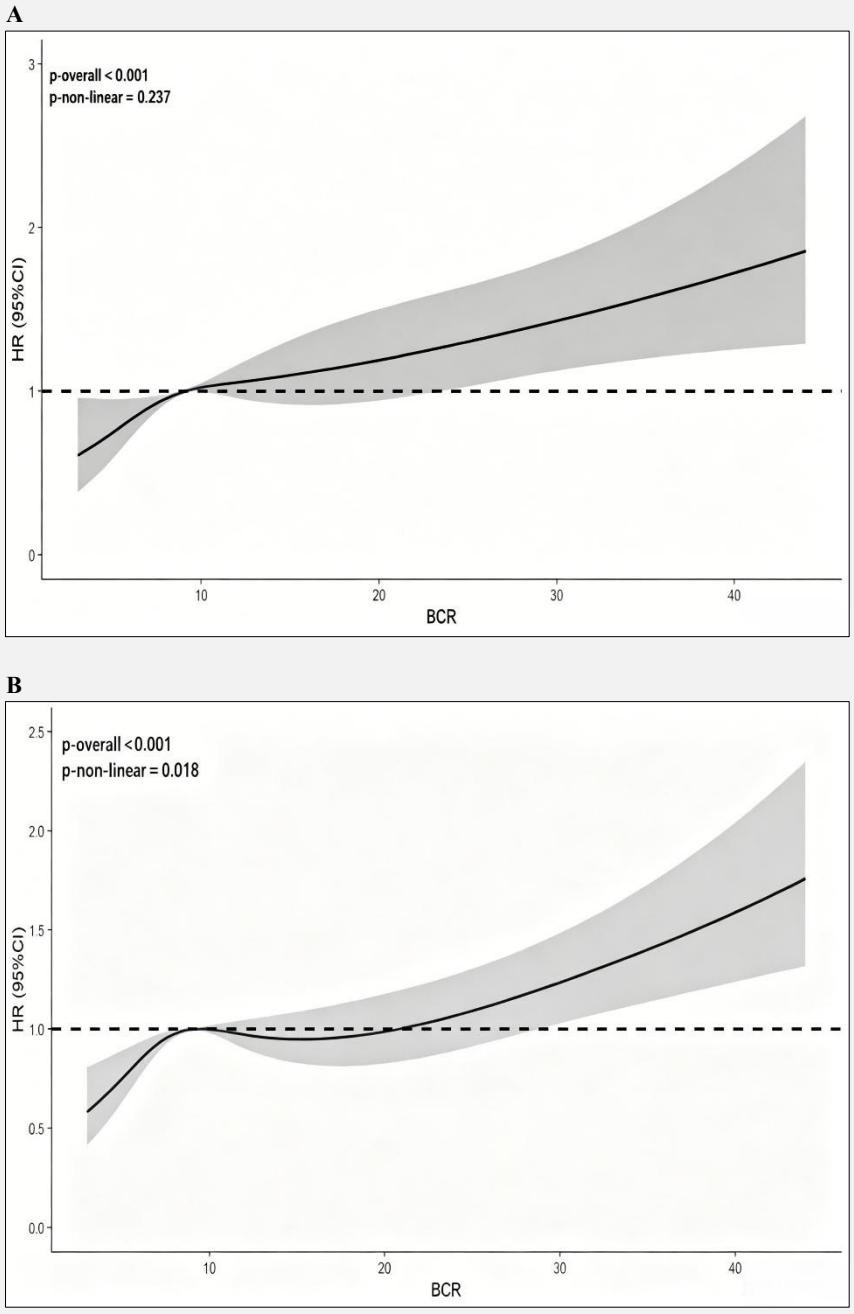

Figure 3. Restricted cubic spline analysis of the association between BCR and mortality risk.

A: Association between BCR and 60-day mortality risk. B: Association between BCR and 365-day mortality risk. BCR: blood urea nitrogen-to-creatinine ratio.

ations with BCR levels ($p < 0.05$): hematocrit, hemoglobin, bicarbonate, potassium, and Scr showed inverse correlations; chloride, glucose, and BUN displayed positive correlations; anion gap and phosphorus followed an initial increase then decrease pattern; calcium exhibited an initial decrease followed by an increase; and INR/PT remained stable in Q1 - Q3 but was elevated in Q4. Vital sign analysis showed progressive decreases in body temperature, increases in respiratory rate, and a biphasic pattern (initial increase and then decrease) in

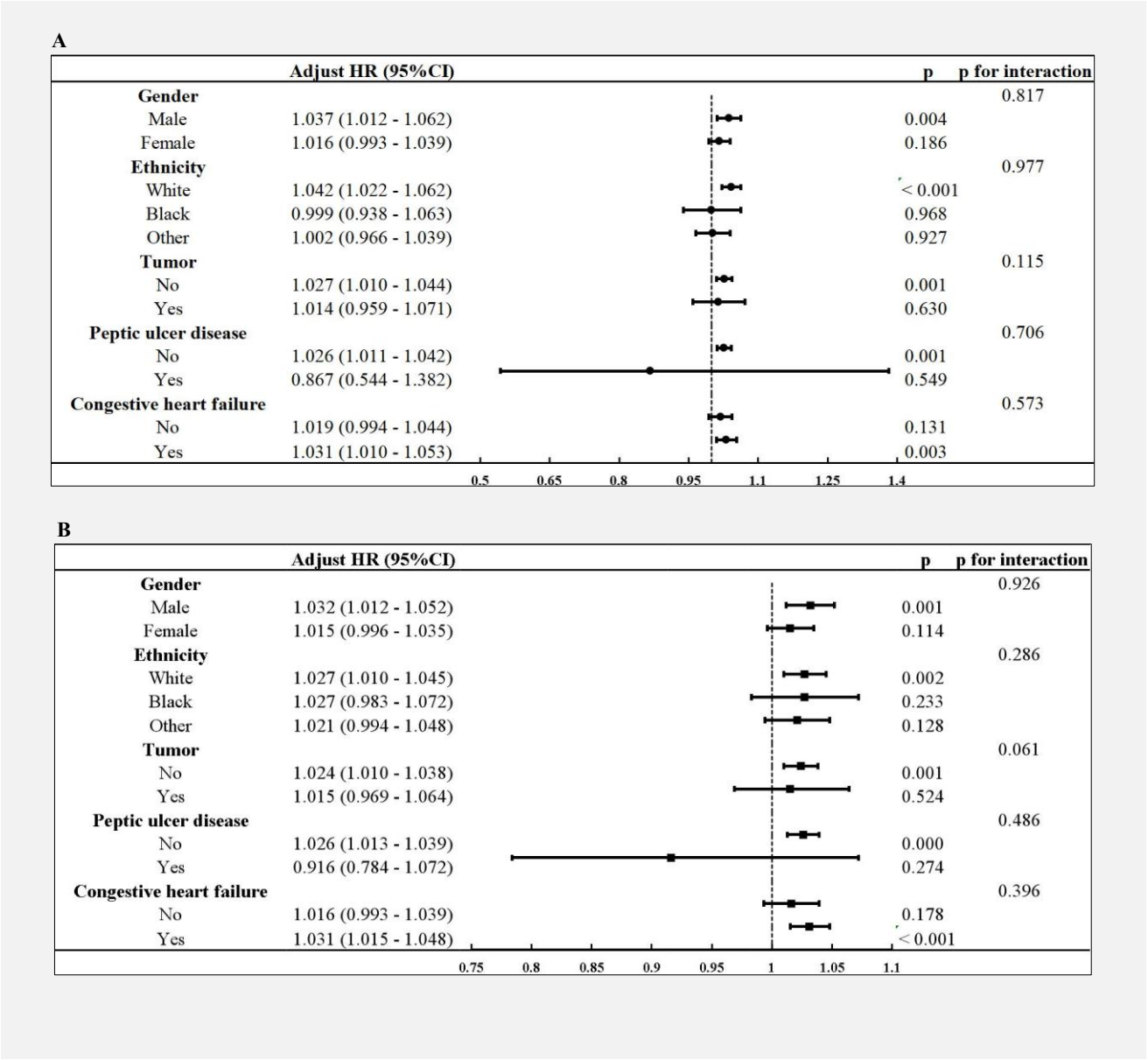

Figure 4. Subgroup analysis of the association between BCR and mortality in patients with ESRD patients.

A: 60-day mortality subgroup analysis. B: 365-day mortality subgroup analysis.
BCR: blood urea nitrogen-to-creatinine ratio, ESRD: end-stage renal disease.

SpO_2 with increasing BCR ($p < 0.05$). Comorbidity prevalence varied significantly ($p < 0.05$), with congestive heart failure, COPD, peptic ulcer disease, and liver disease showing increasing incidence, whereas diabetes mellitus demonstrated a decreasing incidence with higher BCR levels. Most notably, both 60- and 365-day mortality rates increased progressively across the BCR quartiles ($p < 0.001$).

Association between BCR and mortality in patients with ESRD

Kaplan-Meier analysis was used to evaluate the association between BCR values and both 60- and 365-day mortality rates (Figure 2). Figure 2A shows that the Q1 group exhibited significantly higher survival rates than those of the Q2, Q3, and Q4 groups ($p < 0.05$), whereas the Q2 group showed superior survival outcomes relative to the Q3 and Q4 groups ($p < 0.05$). Figure 2B shows that the Q1 group maintained a significantly greater survival probability than that of the Q2 - Q4 groups ($p < 0.001$).

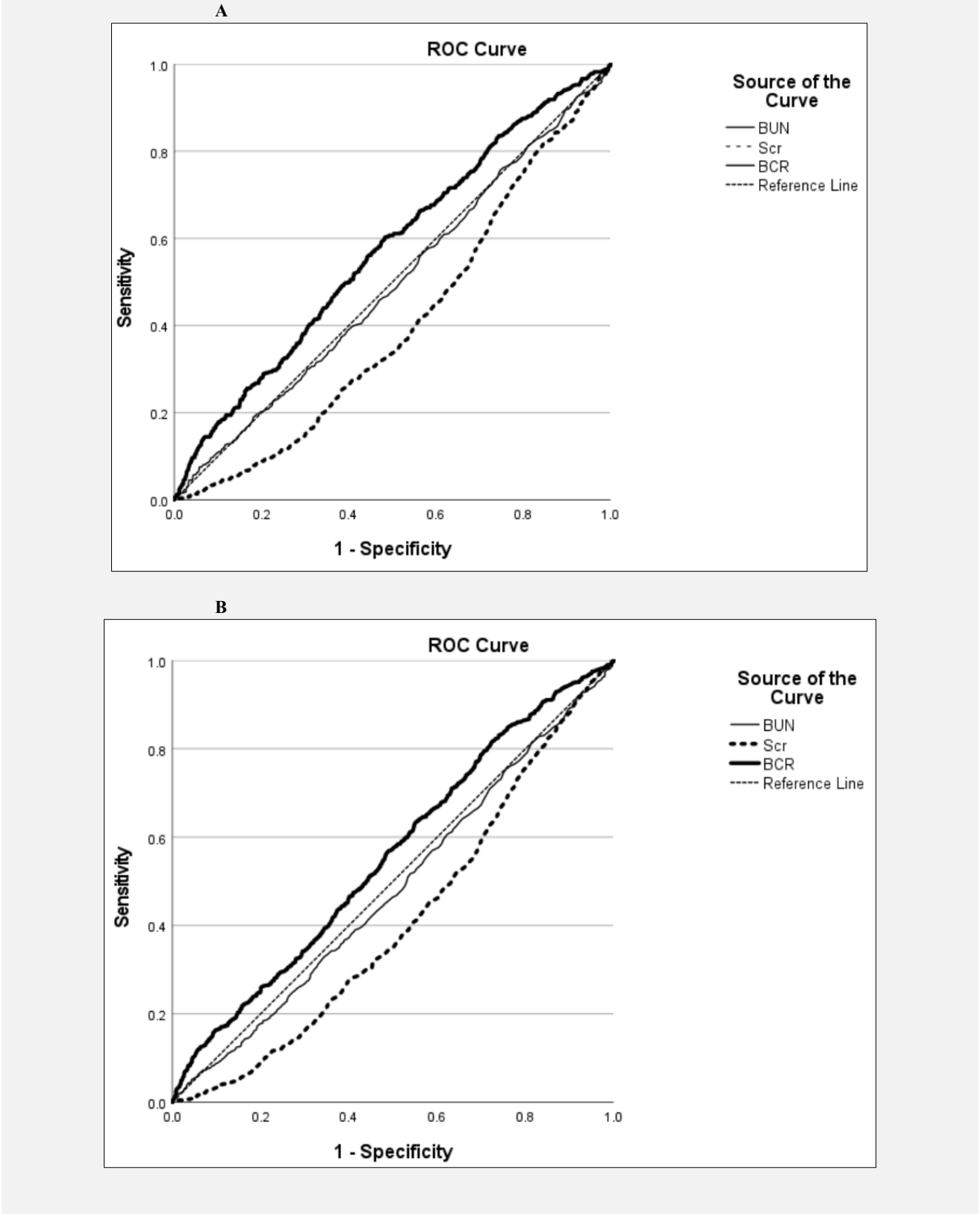

Figure 5. Receiver operating characteristic (ROC) curve analysis of BCR, BUN, and Scr for predicting mortality in patients with ESRD.

A: ROC curve for 60-day mortality. B: ROC curve for 365-day mortality.
BCR: blood urea nitrogen-to-creatinine ratio, BUN: blood urea nitrogen, ESRD: end-stage renal disease.

Subsequent multivariate Cox regression analysis was used to examine the relationship between BCR values and mortality outcomes while controlling for potential confounders (Table 1). The results confirmed significant associations between BCR and both 60- and 365-day mortality rates ($p < 0.05$). In Model 1 (unadjusted analysis), each one-unit increase in BCR was associated with a 3% increased risk of 60-day mortality and 2% increased risk of 365-day mortality ($p \leq 0.001$). Compared with Q1, the Q2 group demonstrated 34% and 32% increased risks of 60- and 365-day mortality, respectively ($p < 0.05$). After full adjustment in Model 3, each one-unit BCR increment remained significantly associated with a 3% increase in both 60- and 365-day mortality risks.

Linear association between the BCR and patient mortality

To further investigate the potential linear relationship between BCR and mortality, we performed RCS analysis (Figure 3). The RCS revealed a significant linear association between BCR and 60-day mortality (p-overall < 0.001, p-non-linear = 0.237), demonstrating a gradual but consistent increase in mortality risk with increasing BCR values. In contrast, BCR showed a significant inverted "S"-shaped non-linear relationship with 365-day mortality (p-overall < 0.001, p-non-linear = 0.018), characterized by an initial increase in mortality risk followed by a subsequent decrease and then another increase as BCR values ascended.

Subgroup analysis

Subgroup analysis demonstrated no significant interaction effects between BCR values and clinical characteristics (gender, race, malignancy, peptic ulcer disease, or congestive heart failure) on either 60- or 365-day mortality in patients with ESRD (all $p > 0.05$, Figures 4A, 4B); however, stratified analyses identified male gender, white race, absence of malignancy, absence of peptic ulcer disease, and presence of congestive heart failure as significant predictors of increased 60- and 365-day mortality risk (all $p < 0.05$, Figures 4A, 4B).

Figure 5 demonstrates the predictive performance of BUN, Scr, and BCR for 60- and 365-day mortality in patients with ESRD, revealing that BCR exhibited superior predictive accuracy at both time points ($p < 0.001$). The optimal cutoff values were 8.96 for 60-day mortality (sensitivity = 0.603, specificity = 0.517) and 6.24 for 365-day mortality (sensitivity = 0.816, specificity = 0.277), as detailed in Table 2.

DISCUSSION

The association between the BCR and 60- and 365-day mortality in patients with ESRD was examined. After adjustment for multiple confounders, BCR remained independently and positively associated with both short-term (60-day) and long-term (365-day) mortality. RCS analysis indicated a linear relationship between BCR and 60-day mortality (p-overall < 0.001), whereas the association with 365-day mortality was non-linear (p-overall < 0.001; p-non-linear = 0.018). ROC curve analyses confirmed that BCR predicted mortality more accurately than BUN or Scr alone.

As a key biochemical marker of amino acid and creatine metabolism [13,14], BCR has demonstrated prognostic value across diverse clinical settings. Shigehiko et al. [15] reported that elevated BCR was significantly associated with increased in-hospital mortality in patients with acute kidney injury. Furthermore, mortality risk was lowest within a specific BCR range, echoing the non-linear relationship we observed for 365-day mortality in ESRD. Additional studies have linked higher BCR to adverse outcomes in chronic kidney disease [16], liver cirrhosis [17], acute ischemic stroke [18], and septic shock [19], suggesting that BCR may also inform prognosis in non-ICU ESRD populations.

Key findings include the following. First, increasing BCR was not accompanied by a higher prevalence of diabetes. We hypothesize that patients with diabetic nephropathy who progress to ESRD have already experienced cardiovascular events [20], introducing survival bias in our cohort. Second, BCR exhibited an inverted S-shaped, non-linear association with 365-day mortality: risk was elevated when BCR was < 12 or > 20, whereas the intermediate range (12.9 - 17.6) conferred the lowest risk [21-24]. This pattern likely reflects distinct pathophysiologic states across BCR strata. Third, the Scr-only model performed poorly (AUC = 0.403), with low sensitivity, underscoring the limitations of using Scr alone. Finally, in the subgroup with peptic ulcer disease, confidence intervals markedly widened, possibly due to fluctuations in BUN driven by gastrointestinal bleeding [25,26].

An elevated BCR may result from increased BUN, decreased Scr, or both. In this cohort, BUN rose and Scr declined as BCR increased (both $p < 0.001$). Prior work indicates that BUN is influenced by dietary protein intake [27]; moderate protein restriction can slow renal function decline, whereas reduced Scr often signals muscle wasting [28]. Thus, BCR may serve as a proxy for metabolic and nutritional status [29], with higher values reflecting malnutrition, a well-established predictor of adverse outcomes [30]. Although we did not directly measure nutritional status, hemoglobin, used as a surrogate marker, partially supports this interpretation [31].

Strengths of our study include: 1) a large sample size (n = 2,102) derived from the MIMIC-IV database and 2) the use of multiple statistical methods to validate findings. Limitations include: the 1) reliance on a single database and 2) inherent constraints of retrospective design in controlling confounding. To minimize bias, we incorporated comprehensive covariates and employed multivariable Cox regression. Prospective studies are warranted to confirm the prognostic utility of BCR and to develop multi-biomarker prediction models.

In summary, BCR is an independent predictor of mortality in patients with ESRD and has potential clinical value. Its underlying mechanisms and optimal application require further elucidation.

Acknowledgment:
The authors thank all participants, developers, and investigators who have contributed to the Medical Information Mart for Intensive Care (MIMIC)-IV database.

Source of Funds:
Funding for this study was provided solely by the authors' personal funds.

Declaration of Interest:
The authors declare that they have no competing interests.

References:

1. Wouk N. End-stage renal disease: medical management. Am Fam Physician 2021 Nov 1;104(5):493-9. (PMID: 34783494)
2. Thurlow JS, Joshi M, Yan G, et al. Global epidemiology of end-stage kidney disease and disparities in kidney replacement therapy. Am J Nephrol 2021 52(2):98-107. (PMID: 33752206)
3. Mercado MG, Smith DK, Guard EL. Acute kidney injury: diagnosis and management. Am Fam Physician 2019 Dec 1;100(11): 687-94. (PMID: 31790176)
4. Liu X, Ou YN, Ma YH, Huang LY, Zhang W, Tan L. Renal function and neurodegenerative diseases : a two-sample Mendelian randomization study. Neurol Res 2023 May;45(5):456-64. (PMID: 36692889)
5. Vukovic V, Hantikainen E, Raftopoulou A, et al. Association of dietary proteins with serum creatinine and estimated glomerular filtration rate in a general population sample: the CHRIS study. J Nephrol 2023 Jan;36(1):103-14. (PMID: 35930180)
6. Vega J, Huidobro E JP. [Effects of creatine supplementation on renal function]. Rev Med Chil 2019 May;147(5):628-33. (PMID: 31859895)
7. Lee MY, Yang DK, Kim SJ. Alterations of Mg(2+) after hemorrhagic shock. Biol Trace Elem Res 2017 Nov;180(1):120-6. (PMID: 28315119)
8. Duan YC, Shi L, Jin Z, et al. Swimming exercise ameliorates hypertension-induced kidney dysfunction via alleviating renal interstitial fibrosis and apoptosis. Kidney Blood Press Res 2021;46(2): 219-28. (PMID: 33849006)
9. Wang Y, Xu X, Shi S, et al. Blood urea nitrogen to creatinine ratio and long-term survival in patients with chronic heart failure. Eur J Med Res 2023 Sep 14;28(1):343. (PMID: 37710326)
10. Parrinello G, Torres D, Testani JM, et al. Blood urea nitrogen to creatinine ratio is associated with congestion and mortality in heart failure patients with renal dysfunction. Intern Emerg Med 2015 Dec;10(8):965-72. (PMID: 26037394)
11. Johnson A, Bulgarelli L, Pollard T, et al. MIMIC-IV (version 3.1). PhysioNet 2024 (https://doi.org/10.13026/kpb9-mt58)
12. Johnson AEW, Bulgarelli L, Shen L, et al. MIMIC-IV, a freely accessible electronic health record dataset. Sci Data 2023 Jan 3;10(1):1. (PMID: 36596836)
13. Das AM. Urea cycle defects in adulthood: clinical presentation, diagnosis and treatment in genetically encoded hepatic metabolic disorders with a potential for encephalopathy. Metab Brain Dis 2025 Apr 26;40(5):192. (PMID: 40285952)
14. Fang J, Huang X, Guo C, et al. Creatinine promotes adipose tissue wasting in chronic kidney disease via creatine and futile creatine cycle. Mol Metab 2025 Jun 19; 98:102191. (PMID: 40543756)
15. Uchino S, Bellomo R, Goldsmith D. The meaning of the blood urea nitrogen/creatinine ratio in acute kidney injury. Clin Kidney J 2012 Apr;5(2):187-91 (PMID: 29497527)
16. Brookes EM, Power DA. Elevated serum urea-to-creatinine ratio is associated with adverse inpatient clinical outcomes in non-end stage chronic kidney disease. Sci Rep 2022 Dec 2;12(1):20827. (PMID: 36460694)
17. Yi Y, Li L, Chen Y, Luo Y. Interaction between age and blood urea nitrogen to creatinine ratio on mortality in patients with severe cirrhosis: a retrospective cohort study from the MIMIC database. Front Endocrinol (Lausanne) 2025 Mar 5:16:1544223. (PMID: 40110543)
18. Jiang WF, Deng ML. Prognostic impact of blood urea nitrogen/creatinine ratio changes in patients with acute ischemic stroke. Clin Neurol Neurosurg 2022 Apr;215:107204. (PMID: 35299071)
19. Han D, Zhang L, Zheng S, et al. Prognostic value of blood urea nitrogen/creatinine ratio for septic shock: an analysis of the MIMIC-III clinical database. Biomed Res Int 2021 May 22;2021: 5595042. (PMID: 34095304)
20. Jiang Y, Fine JP, Mottl AK. Competing risk of death with end-stage renal disease in diabetic kidney disease. Adv Chronic Kidney Dis 2018 Mar;25(2):133-40. (PMID: 29580577)
21. Peng R, Liu K, Li W, et al. Blood urea nitrogen, blood urea nitrogen to creatinine ratio and incident stroke: the Dongfeng-Tongji cohort. Atherosclerosis 2021 Sep;333:1-8 (PMID: 34390959)
22. Paulus MC, Melchers M, van Es A, Kouw IWK, van Zanten ARH. The urea-to-creatinine ratio as an emerging biomarker in critical care: a scoping review and meta-analysis. Crit Care 2025 May 2;29(1):175 (PMID: 40317012)
23. Matsue Y, van der Meer P, Damman K, et al. Blood urea nitrogen-to-creatinine ratio in the general population and in patients with acute heart failure. Heart 2017 Mar;103(6):407-13. (PMID: 27658757)
24. Shen S, Yan X, Xu B. The blood urea nitrogen/creatinine (BUN/cre) ratio was U-shaped associated with all-cause mortality in general population. Ren Fail 2022 Dec;44(1):184-90. (PMID: 35166184)
25. van Berlo CL, van de Bogaard AE, van der Heijden MA, et al. Is increased ammonia liberation after bleeding in the digestive tract the consequence of complete absence of isoleucine in hemoglobin? A study in pigs. Hepatology 1989 Sep;10(3):315-23. (PMID: 2788117)

26. Tomizawa M, Shinozaki F, Hasegawa R, et al. Patient characteristics with high or low blood urea nitrogen in upper gastrointestinal bleeding. World J Gastroenterol 2015 Jun 28;21(24):7500-5. (PMID: 26139996)

27. Hamidianshirazi M, Shafiee M, Ekramzadeh M, Torabi Jahromi M, Nikaein F. Diet therapy along with nutrition education can improve renal function in people with stages 3-4 chronic kidney disease who do not have diabetes: a randomised controlled trial. Br J Nutr 2023 Jun 14;129(11):1877-87. (PMID: 35795914)

28. Baxmann AC, Ahmed MS, Marques NC, et al. Influence of muscle mass and physical activity on serum and urinary creatinine and serum cystatin C. Clin J Am Soc Nephrol 2008 Mar;3(2): 348-54. (PMID: 18235143)

29. Tufan F, Yıldız A, Dogan I, Yıldız D, Sevinir Ş. Urea to creatinine ratio: a forgotten marker of poor nutritional state in patients undergoing hemodialysis treatment. Aging Male 2015 Mar;18(1): 49-53. (PMID: 24702599)

30. Sabatino A, Regolisti G, Karupaiah T, et al. Protein-energy wasting and nutritional supplementation in patients with end-stage renal disease on hemodialysis. Clin Nutr 2017 Jun;36(3):663-71. (PMID: 27371993)

31. Basaleem HO, Alwan SM, Ahmed AA, Al-Sakkaf KA. Assessment of the nutritional status of end-stage renal disease patients on maintenance hemodialysis. Saudi J Kidney Dis Transpl 2004 Oct-Dec;15(4):455-62. (PMID: 17642781)

Additional material can be found online at:
http://supplementary.clin-lab-publications.com/250736/