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Impact of the Geriatric Nutritional Risk Index on short-term prognosis of patients with sepsis-related acute kidney injury: analysis using the MIMIC-IV database



Kailun Cai^{1†}, Wenchao Mao^{1†}, Mingkun Yang^{2†}, Changqin Chen¹, Shijin Gong¹, Lifen Zheng³ and Changyun Zhao^{1*}

Abstract

Background In critically ill elderly patients, malnutrition is a common comorbidity. The Geriatric Nutritional Risk Index (GNRI) is a straightforward tool for evaluating the nutritional status of elderly individuals. The association between GNRI score and unfavorable health outcomes has been established. However, no studies have yet elucidated the relationship between GNRI score and sepsis-related acute kidney injury (S-AKI).

Methods We sourced patient data from the Medical Information Mart for Intensive Care IV (MIMIC-IV) database. All patients were divided into four groups based on their GNRI score using quartile analysis. The main objective of this study was to investigate the 28-day mortality rate. Secondary study outcomes were the incidence of severe AKI, length of stay in the intensive care unit, and days in the hospital. To evaluate the association between GNRI score and study outcomes, we used a Cox proportional hazards regression model and restricted cubic splines. Kaplan–Meier curves were used to compare the outcomes in each group.

Results A total of 4515 elderly patients with S-AKI were included in this study. Patients were categorized into four groups according to GNRI quartile: Q1 (<78.92), Q2 (78.92–84.88), Q3 (84.88–90.84), and Q4 (>90.84). Overall 28-day mortality was 29.5%. Patients with a low GNRI were predominantly women, and had a low body mass index. After controlling for confounding factors, GNRI score emerged as an independent predictor of 28-day mortality among elderly patients with S-AKI (Q4 vs. Q1: hazard ratio 0.74, 95% confidence interval 0.63–0.87; *p* < 0.001). Restricted cubic spline analysis revealed a linear relationship between GNRI and 28-day mortality (p for non-linearity = 0.207), and this association remained consistent across all subgroup analyses.

Conclusions The GNRI is an important nutritional assessment tool, and is useful in predicting the prognosis of critically ill elderly patients with S- AKI.

Keywords Geriatric Nutritional Risk Index, Sepsis, Sepsis-related acute kidney injury, Elderly

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Background

Sepsis is a complex condition characterized by an uncontrolled inflammatory response to infection, leading to organ dysfunction and, potentially, multiple organ failure. Acute kidney injury (AKI) frequently occurs as a complication of sepsis [1]. Cases in which both sepsis and AKI criteria are satisfied are classified as having sepsis-associated acute kidney injury (S-AKI) [2, 3]. In the intensive care unit (ICU), sepsis is associated with 40–60% of AKI cases [4, 5]. S-AKI is associated with a six- to eight-fold increased risk of in-hospital mortality [6] and a three-fold increased risk of chronic kidney disease among survivors [7]. Moreover, patients with S-AKI have significantly increased mortality compared with patients who have AKI with other etiologies [8].

Malnutrition is a significant risk factor for the development of AKI [9]. A recent study of data from 238 million emergency department visits showed that the prevalence of malnutrition in the elderly population increased from 2.5% in 2006 to 3.6% in 2014 [10]. Assessment of nutritional status is critical for identifying elderly patients who are vulnerable to AKI and at risk of death [11]. Traditional tools for nutritional screening focus on weight loss, reduced food intake, and laboratory values, but are unreliable for patients with AKI who are unable to provide these data, or may have water-electrolyte imbalances [12]. Additionally, elderly patients with AKI in the ICU often require volume resuscitation, which can result in rapid weight gain and even tissue edema. Thus, even data such as body mass index (BMI) and skinfold thickness often do not accurately reflect the nutritional status of patients with AKI.

The Geriatric Nutritional Risk Index (GNRI) has been widely recognized as a novel nutritional assessment index and a promising screening tool for easily identifying malnourished patients [13, 14]. Current research suggests that GNRI score could be indicative of a poor prognosis in those with AKI [15–17]. Among individuals with heart failure, a relationship exists between GNRI score and the incidence of AKI [18]. To date, no studies have assessed the predictive validity of the GNRI in critically ill elderly patients with S-AKI. Therefore, the aim of this study was to assess the ability of the GNRI to predict adverse outcomes in elderly patients with S-AKI in the ICU.

Materials and methods

Data source

In this study, we used data sourced from the Medical Information Mart for Intensive Care-IV (MIMIC-IV) database (version 3.0) [19], a large publicly available dataset comprising over 50,000 anonymized patient records from the ICU at Beth Israel Deaconess Medical Center in Boston, Massachusetts, USA, covering the period from 2008 to 2022. One author (Changyun Zhao) obtained access to the MIMIC-IV database (certification number: 65303679) after completion of the Collaborative Institutional Training Initiative program. The research was carried out in compliance with the principles outlined in the Declaration of Helsinki; the requirement for informed consent was waived because only anonymous data were analyzed. The institutional review board at Beth Israel Deaconess Medical Center granted approval for this study.

Study population

Patients aged \geq 65 years with a diagnosis of S-AKI were included. The exclusion criteria for this study were patients with an ICU stay of less than 24 h, those with repeat admissions, patients with end-stage renal disease, and patients lacking albumin, height, and weight data.

Data collection

Data extraction was performed using Structured Query Language (SQL). The SQL script codes were obtained from the GitHub website (https://github.com/MIT-LCP /mimic-iv). The extracted variables included demograph ic data (age, race and ethnicity, sex, weight, and height), vital signs on admission (heart rate, blood pressure [systolic, diastolic, and mean], and respiratory rate), as well as pre-existing comorbidities (diabetes mellitus type 2, chronic obstructive pulmonary disease, chronic kidney disease, hypertension, and stroke). Laboratory parameters collected within 24 h of ICU admission encompassed renal function markers (serum creatinine and blood urea nitrogen), liver function markers (aspartate aminotransferase [AST], alanine aminotransferase [ALT], and total bilirubin [TBiL]), nutritional/metabolic indicators (serum albumin, hemoglobin, glucose, bicarbonate, chloride, sodium, potassium, lactate, and anion gap), and inflammatory/hematologic profiles (white blood cell count, platelet count, and lymphocyte count). Severity scores, including the Sequential Organ Failure Assessment (SOFA), Acute Physiology and Chronic Health Evaluation III, Simplified Acute Physiology Score II, Oxford Acute Severity of Illness Score, and Charlson Comorbidity Index, were analyzed. Treatment-related variables comprised renal support (continuous renal replacement therapy [CRRT]), pharmacologic interventions (vasopressors, mechanical ventilation, and nephrotoxic drugs [e.g., non-steroidal anti-inflammatory drugs, aminoglycosides, vancomycin, iodinated contrast agents, calcineurin inhibitors; see Supplementary Table S1 for a complete list]), and duration of mechanical ventilation. BMI was calculated as weight (kg)/height (m²). All laboratory data were derived from the first measurement recorded within 24 h of ICU admission.

Definitions

S-AKI was defined as AKI occurring within 7 days of a sepsis episode (diagnosed according to the Renal Disease Improvement Global Outcome Criteria and Sepsis 3 criteria, respectively) [20]. Sepsis is characterized as a life-threatening condition involving organ dysfunction that arises from an inappropriate host response to an infection [21]. AKI was diagnosed based on an increase in serum creatinine (Scr) of ≥ 0.3 mg/dL within 48 h, a rise to ≥ 1.5 times the baseline value within the last 7 days, or a urine output of <0.5 mL/kg/hour for at least 6 consecutive hours [22]. Stage 1 AKI was classified as mild, whereas stages 2 and 3 were categorized as severe AKI.

Nutritional status was assessed via the GNRI, computed as $[1.489 \times \text{serum}$ albumin $(g/L)]+[41.7 \times (\text{current}$ weight in kg/ideal weight)]. Ideal weight was calculated using the Lorentz formulas [23]: height (cm)-100-[height (cm)-150]/4 for men and height (cm)-100-([height (cm)-150]/2.5 for women. When the current weight exceeded the ideal body weight, we used the current weight in kg/ideal weight=1. Additionally, GNRI scores were categorized into four classes based on GNRI score quartiles: Q1 (<78.92), Q2 (78.92–84.88), Q3 (84.88–90.84), and Q4 (>90.84). The GNRI is inversely correlated with the risk of malnutrition; a lower GNRI score indicates a greater likelihood of malnutrition [13].

Exposure and outcome ascertainment

The primary exposure of interest in this study was the GNRI. The primary endpoint of the study was the 28-day mortality rate. Additional endpoints, considered secondary outcomes, comprised the frequency of severe AKI, duration of the ICU stay, and the overall length of hospitalization measured in days.

Statistical analysis

Continuous variables with a normal distribution are presented as mean±standard deviation and were analyzed using analysis of variance for comparisons between groups. Skewed continuous variables are described as median with interquartile range, and were compared using the Kruskal-Wallis test. Categorical variables are reported as number and percentage, and were compared between groups using the chi-square test. Univariate Cox regression models were used to screen covariates, and univariate and multivariate Cox regression models were developed to explore the relationship between GNRI score and 28-day mortality in patients with S-AKI. Model 1 was a crude model, and Model 2 (demographically adjusted) was adjusted for age, sex, and ethnicity. Model 3 (fully adjusted) was adjusted for all baseline variables with p < 0.05 (Table 1) and clinically relevant prognostic factors, including ALT, AST, TBiL, CRRT, mechanical ventilation status, and nephrotoxic drug exposure. The non-linear association between GNRI score and in-hospital mortality was evaluated using an adjusted restricted cubic spline (RCS) model that included variables from Model 3. Cumulative survival rates among the four groups were evaluated using Kaplan–Meier survival curves. Subgroup analyses were performed to examine the relationship between GNRI score and in-hospital mortality across various subgroups, including the computation of p-values for interaction effects. The statistical analyses for this study were conducted using R software, version 3.3.2 (The R Foundation for Statistical Computing, Vienna, Austria). A two-tailed p-value of <0.05 was deemed statistically significant.

Results

Patient characteristics

Figure 1 presents the flowchart of participant screening. A total of 4515 elderly patients with S-AKI who met the inclusion and exclusion criteria participated in this study. Patients were divided into four groups according to GNRI score quartiles.

Table 1 provides an overview of the characteristics of each group. Patients in the high malnutrition risk group (low GNRI scores) were predominantly women and had low BMI. Additionally, these patients had a higher respiratory rate on admission. In terms of laboratory parameters, the high malnutrition risk group had higher white blood cell and platelet counts, urea nitrogen, creatinine, chloride, and lactate levels, as well as lower values for hemoglobin, lymphocytes, albumin, bicarbonate, and glucose. The use of vasoactive drugs was greater in the high malnutrition risk group, but there were no significant differences between the groups in terms of nephrotoxic drugs, CRRT, and ventilator support. Finally, SOFA scores and Acute Physiology and Chronic Health Evaluation III (APSIII) scores were significantly higher in the high malnutrition risk group.

GNRI and clinical outcomes

The overall 28-day mortality was 29.50%. Mortality was significantly higher in lower quartiles in the nutrition-related risk groups (Q1 vs. Q4: 39.50% vs. 22.78%; p < 0.001) (Table 2). Patients with a high nutritional risk had a greater incidence of severe AKI (Q1 vs. Q4: 88.68% vs. 82.63%; p < 0.001). The ICU and hospital lengths of stay were longer in the high nutritional risk group (Q1) than in the low nutritional risk group (Q4): 6.59 vs. 5.43 and 15.58 vs. 11.69, respectively ; both p < 0.001).

Survival curves of 28-day mortality according to GNRI quartile in patients with S-AKI

Figure 2 shows the Kaplan-Meier curves of GNRI scores and survival probability in quartile subgroups of patients

Table 1 Baseline characteristics of the study participants, overall and stratified by Geriatric Nutritional Risk Index score quartiles

Variable	Overall N=4,515	Q1 (<78.92) N=1,243	Q2(78.92-84.88) N=1,092	Q3(84.88–90.84) N=1,109	Q4(>90.84) N=1,071	<i>p</i> - value
Sex, n (%)						0.012
F	1,956 (43.32)	578 (46.50)	484 (44.32)	446 (40.22)	448 (41.83)	
M	2,559 (56.68)	665 (53.50)	608 (55.68)	663 (59.78)	623 (58.17)	
Race, n (%)						0.123
ASIAN	109 (2.41)	34 (2.74)	30 (2.75)	26 (2.34)	19 (1.77)	
BLACK	332 (7.35)	100 (8.05)	85 (7.78)	75 (6.76)	72 (6.72)	
OTHER	1,108 (24.54)	311 (25.02)	263 (24.08)	243 (21.91)	291 (27.17)	
WHITE	2,966 (65.69)	798 (64.20)	714 (65.38)	765 (68.98)	689 (64.33)	
Age, Median (Q1, Q3)	76.00 (70.00, 83.00)	76.00 (70.00, 83.00)	76.00 (71.00, 84.00)	76.00 (71.00, 83.00)	75.00 (70.00, 81.00)	0.012
BMI, Median (Q1, Q3)	27.78 (24.14, 32.50)	26.46 (22.41, 31.13)	28.07 (24.07, 32.68)	28.36 (24.80, 33.30)	28.44 (25.10, 32.94)	< 0.00
Diagnoses, comorbidities and treatments, n (%)						
Diabetes	1,572 (34.82)	381 (30.65)	396 (36.26)	417 (37.60)	378 (35.29)	0.002
COPD	579 (12.82)	173 (13.92)	132 (12.09)	159 (14.34)	115 (10.74)	0.040
Hypertension	1,943 (43.03)	528 (42.48)	459 (42.03)	456 (41.12)	500 (46.69)	0.044
Stroke	484 (10.72)	101 (8.13)	122 (11.17)	120 (10.82)	141 (13.17)	0.001
Chronic kidney disease,	1,242 (27.51)	315 (25.34)	323 (29.58)	329 (29.67)	275 (25.68)	0.022
CRRT	591 (13.09)	188 (15.12)	143 (13.10)	132 (11.90)	128 (11.95)	0.068
Ventilation	3,220 (71.32)	889 (71.52)	794 (72.71)	785 (70.78)	752 (70.21)	0.604
Vasopressors	2,070 (45.85)	720 (57.92)	528 (48.35)	456 (41.12)	366 (34.17)	< 0.00
Nephrotoxic drugs	3,799 (84.14)	1,030 (82.86)	927 (84.89)	944 (85.12)	898 (83.85)	0.416
/ital signs						
Hr(bpm), Median (Q1, Q3)	122.00 (106.00, 140.00)	127.00 (112.00, 143.00)	125.00 (108.00, 143.00)	118.00 (102.00, 136.00)	118.00 (103.00, 137.00)	< 0.00
Nbps(mmHg), Median (Q1, Q3)	157.00 (141.00, 174.00)	156.00 (140.00, 172.00)	158.00 (143.00, 175.00)	157.00 (141.00, 175.00)	157.00 (140.00, 175.00)	0.015
Nbpd(mmHg), Median (Q1, Q3)	99.00 (85.00, 115.00)	99.00 (84.00, 114.00)	99.50 (86.00, 115.00)	98.00 (84.00, 115.00)	99.00 (85.00, 115.00)	0.221
Nbpm(mmHg), Median (Q1, Q3)	111.00 (97.00, 126.00)	109.00 (96.00, 126.00)	112.00 (98.00, 127.50)	110.00 (96.00, 126.00)	111.00 (97.00, 127.00)	0.271
RR(insp/min), Median (Q1, Q3)	34.00 (30.00, 40.00)	35.00 (31.00, 41.00)	35.00 (30.00, 40.00)	34.00 (29.00, 39.00)	33.00 (29.00, 39.00)	< 0.001
Laboratory parameters						
Creatinine(mg/dL), Median (Q1, Q3)	1.20 (0.90, 1.90)	1.30 (0.90, 2.10)	1.20 (0.90, 1.90)	1.20 (0.90, 1.90)	1.10 (0.90, 1.70)	0.009
Hemoglobin(g/dL), Median (Q1, Q3)	10.30 (8.80, 12.00)	9.80 (8.50, 11.30)	10.20 (8.70, 11.65)	10.40 (9.00, 12.00)	11.10 (9.40, 12.70)	< 0.00
WBC(10^9/L), Median (Q1, Q3)	12.10 (8.50, 17.00)	13.20 (8.70, 18.80)	12.10 (8.65, 17.30)	12.10 (8.70, 16.80)	11.20 (8.20, 15.00)	< 0.001
Platelets(10^9/L), Median (Q1, Q3)	184.00 (129.00, 254.00)	194.00 (127.00, 283.00)	181.50 (124.00, 250.00)	177.00 (127.00, 241.00)	183.00 (136.00, 241.00)	0.001
Lymphocytes(10^9/L), Median (Q1, Q3)	10.48 (5.50, 11.00)	8.00 (4.10, 10.48)	10.10 (5.20, 11.00)	10.48 (6.20, 11.10)	10.48 (7.00, 12.90)	< 0.00
Urea nitrogen(mg/dL), Median (Q1, Q3)	26.00 (17.00, 43.00)	30.00 (19.00, 49.00)	27.50 (18.00, 43.00)	25.00 (17.00, 42.00)	23.00 (16.00, 35.00)	< 0.00
Albumin(g/dL), Median (Q1, Q3)	3.00 (2.50, 3.30)	2.30 (2.00, 2.50)	2.80 (2.70, 2.90)	3.10 (3.00, 3.30)	3.60 (3.50, 3.90)	< 0.00
Bicarbonate(mEq/L), Median (Q1, Q3)	22.00 (19.00, 25.00)	21.00 (18.00, 24.00)	22.00 (19.00, 24.00)	22.00 (19.00, 25.00)	23.00 (20.00, 25.00)	< 0.00
Glucose(mg/dL), Median (Q1, Q3)	139.00 (111.00, 181.00)	138.00 (106.00, 183.00)	142.50 (114.00, 185.00)	139.00 (113.00, 182.00)	137.00 (111.00, 174.00)	0.037
Chloride(mEq/L), Median (Q1, Q3)	104.00 (100.00, 109.00)	105.00 (101.00, 110.00)	105.00 (100.00, 109.00)	104.00 (100.00, 108.00)	104.00 (100.00, 107.00)	< 0.001
Sodium(mEq/L), Median (Q1, Q3)	139.00 (136.00, 142.00)	139.00 (135.00, 142.00)	139.00 (135.00, 142.00)	139.00 (136.00, 141.00)	139.00 (136.00, 141.00)	0.867
ALT(U/L), Median (Q1, Q3) AST(U/L), Median (Q1, Q3)	29.00 (16.00, 86.00) 45.00 (26.00, 125.00)	31.00 (16.00, 101.00) 48.00 (25.00, 145.00)	31.00 (16.00, 91.50) 46.00 (25.00, 123.00)	28.00 (16.00, 79.00) 43.00 (26.00, 125.00)	26.00 (16.00, 68.00) 42.00 (26.00, 105.00)	0.042 0.560

Table 1 (continued)

Variable	Overall N=4,515	Q1 (<78.92) N=1,243	Q2(78.92–84.88) N=1,092	Q3(84.88–90.84) N=1,109	Q4(>90.84) N=1,071	<i>p</i> - value
TBil(mg/dL), Median (Q1, Q3)	0.70 (0.40, 1.39)	0.70 (0.40, 1.39)	0.70 (0.40, 1.39)	0.70 (0.40, 1.39)	0.70 (0.50, 1.39)	0.636
Potassium(mEq/L), Median (Q1, Q3)	4.20 (3.80, 4.70)	4.20 (3.70, 4.80)	4.20 (3.80, 4.70)	4.20 (3.80, 4.70)	4.20 (3.80, 4.70)	0.646
Lactate(mmol/L), Median (Q1, Q3)	2.00 (1.30, 2.90)	2.10 (1.40, 3.10)	1.90 (1.30, 3.00)	2.00 (1.30, 2.80)	1.90 (1.30, 2.60)	0.002
Anion gap(mEq/L), Median (Q1, Q3)	15.00 (12.00, 18.00)	14.00 (12.00, 18.00)	14.00 (12.00, 17.00)	15.00 (12.00, 18.00)	15.00 (12.00, 17.00)	0.100
Score						
SOFA, Median (Q1, Q3)	3.00 (2.00, 5.00)	4.00 (3.00, 5.00)	4.00 (2.00, 5.00)	3.00 (2.00, 5.00)	3.00 (2.00, 5.00)	< 0.001
APSIII, Median (Q1, Q3)	53.00 (40.00, 69.00)	62.00 (49.00, 79.00)	55.00 (43.00, 70.00)	50.00 (38.00, 65.00)	45.00 (34.00, 60.00)	< 0.001
SAPSII, Median (Q1, Q3)	46.00 (38.00, 56.00)	50.00 (42.00, 61.00)	47.00 (39.00, 56.00)	44.00 (37.00, 53.00)	41.00 (35.00, 51.00)	< 0.001
OASIS, Median (Q1, Q3)	38.00 (32.00, 44.00)	40.00 (34.00, 46.00)	38.00 (33.00, 44.00)	37.00 (31.00, 42.00)	35.00 (30.00, 41.00)	< 0.001
GCS, Median (Q1, Q3)	15.00 (13.00, 15.00)	15.00 (13.00, 15.00)	15.00 (14.00, 15.00)	15.00 (13.00, 15.00)	15.00 (13.00, 15.00)	0.553
Charlson, Median (Q1, Q3)	6.00 (5.00, 8.00)	6.00 (5.00, 8.00)	6.00 (5.00, 8.00)	6.00 (5.00, 8.00)	6.00 (5.00, 8.00)	0.012

BMI, Body mass index; GCS, Glasgow Coma Scale; OASIS, Organ Assessment Score in the ICU; SAPSII, Simplified Acute Physiology Score II; APSIII, Acute Physiology and Chronic Health Evaluation III; SOFA, Sequential Organ Failure Assessment; WBC, White blood cell; HR, Heart rate; RR, Respiratory rate; NBPM, Non-invasive blood pressure mean; NBPD, Non-invasive blood pressure diastolic; NBPS, Non-invasive blood pressure systolic; CRRT, Continuous renal replacement therapy; CKD, Chronic kidney disease; COPD, Chronic obstructive pulmonary disease

Patients who aged ≥ 65 years and are admitted to the ICU for the first time with a diagnosis of sepsis in the MIMIC-IV database(n=35493)

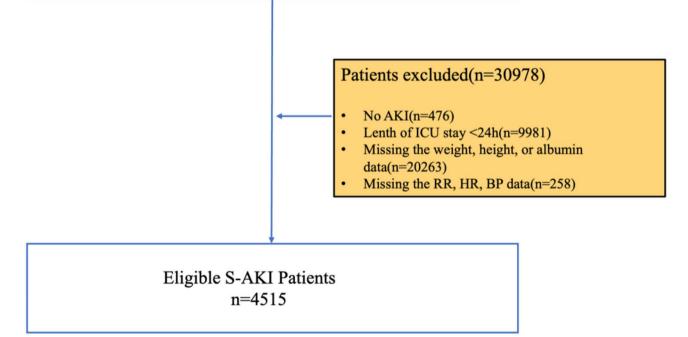
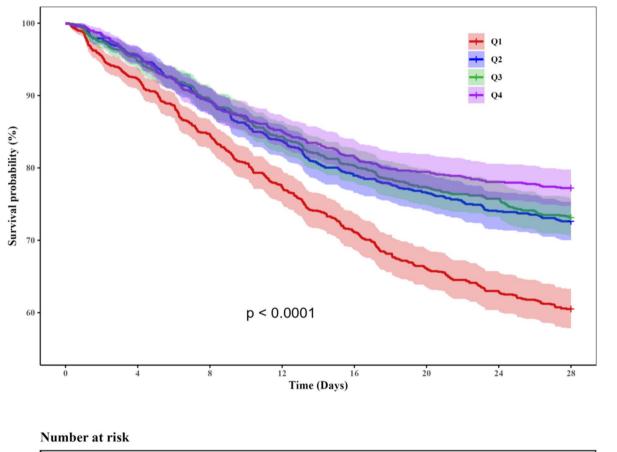


Fig. 1 Flowchart of participant screening

Table 2 Outcomes o	f patients with acute l	kidney injury stratified ac	cording to the Geriatric	Nutritional Risk Index

Overall N=4,515	Q1 (<78.92) N=1,243	Q2(78.92–84.88) N=1,092	Q3(84.88–90.84) N=1,109	Q4(>90.84) N=1,071	<i>p</i> -value
1,332 (29.50)	491 (39.50)	299 (27.38)	298 (26.87)	244 (22.78)	< 0.001
					< 0.001
633 (14.02)	141 (11.34)	148 (13.55)	158 (14.25)	186 (17.37)	
1,978 (43.81)	468 (37.65)	497 (45.51)	515 (46.44)	498 (46.50)	
1,904 (42.17)	634 (51.01)	447 (40.93)	436 (39.31)	387 (36.13)	
5.97 (3.24, 10.91)	6.59 (3.54, 11.64)	6.47 (3.61, 11.97)	5.47 (3.01, 9.96)	5.43 (3.00, 9.93)	< 0.001
13.16 (8.04, 22.09)	15.58 (8.88, 24.87)	13.79 (8.64, 22.95)	12.22 (7.95, 19.82)	11.69 (7.08, 19.38)	< 0.001
	N=4,515 1,332 (29.50) 633 (14.02) 1,978 (43.81) 1,904 (42.17) 5.97 (3.24, 10.91)	N=4,515 N=1,243 1,332 (29.50) 491 (39.50) 633 (14.02) 141 (11.34) 1,978 (43.81) 468 (37.65) 1,904 (42.17) 634 (51.01) 5.97 (3.24, 10.91) 6.59 (3.54, 11.64)	N=4,515 N=1,243 N=1,092 1,332 (29.50) 491 (39.50) 299 (27.38) 633 (14.02) 141 (11.34) 148 (13.55) 1,978 (43.81) 468 (37.65) 497 (45.51) 1,904 (42.17) 634 (51.01) 447 (40.93) 5.97 (3.24, 10.91) 6.59 (3.54, 11.64) 6.47 (3.61, 11.97)	N=4,515 N=1,243 N=1,092 N=1,109 1,332 (29.50) 491 (39.50) 299 (27.38) 298 (26.87) 633 (14.02) 141 (11.34) 148 (13.55) 158 (14.25) 1,978 (43.81) 468 (37.65) 497 (45.51) 515 (46.44) 1,904 (42.17) 634 (51.01) 447 (40.93) 436 (39.31) 5.97 (3.24, 10.91) 6.59 (3.54, 11.64) 6.47 (3.61, 11.97) 5.47 (3.01, 9.96)	N=4,515 N=1,243 N=1,092 N=1,109 N=1,071 1,332 (29.50) 491 (39.50) 299 (27.38) 298 (26.87) 244 (22.78) 633 (14.02) 141 (11.34) 148 (13.55) 158 (14.25) 186 (17.37) 1,978 (43.81) 468 (37.65) 497 (45.51) 515 (46.44) 498 (46.50) 1,904 (42.17) 634 (51.01) 447 (40.93) 436 (39.31) 387 (36.13) 5.97 (3.24, 10.91) 6.59 (3.54, 11.64) 6.47 (3.61, 11.97) 5.47 (3.01, 9.96) 5.43 (3.00, 9.93)

SD, Standard deviation



4	0	4	8	12	16 me (Days)	20	24	28	a	Q4
Q4 -	1071	1024	955	911	874	851	836	827		04
Q3 -	1109	1051	993	935	891	858	840	811	а	Q3
Q2 -	1092	1043	974	914	862	836	808	793	а	Q2
Q1 -	1243	1147	1052	962	884	822	782	752	а	Q1

Fig. 2 Kaplan-Meier curves for Geriatric Nutritional Risk Index quartiles in patients with sepsis-related renal injury

with S-AKI. The results showed that a lower GNRI score was associated with lower survival probability (p < 0.001).

Cox regression analyses of 28-day mortality in patients with S-AKI

We conducted multivariable regression analysis to confirm the independent relationship between GNRI score and worse outcomes (Table 3). In Model 1, higher GNRI score was associated with a reduced 28-day hospitalized mortality rate (Q4 vs. Q1: hazard ratio [HR] 0.50, 95% confidence interval [CI]: 0.43-0.58; p<0.001. In model 2, the association remained significant (Q4 vs. Q1: HR 0.51, 95% CI: 0.44–0.59; p<0.001). In Model 3, adjusted for additional confounders, a high GNRI still significantly reduced 28-day hospital mortality rates (Q4 vs. Q1: HR 0.74, 95% CI: 0.63-0.87; p<0.001. The GNRI was an independent risk factor for 28-day in-hospital mortality in patients with S-AKI. As a continuous variable, higher GNRI score was still associated with lower 28-day inhospital mortality (all p < 0.001). Model 3 showed that higher GNRI as a continuous variable was associated with reduced in-hospital mortality after adjusting for confounders (HR 0.99, 95% CI: 0.98–1.0; p<0.001). Figure 3 depicts the RCS model for the association between GNRI and 28-day mortality. After accounting for relevant confounding factors, Model 3 revealed a monotonically decreasing relationship between GNRI and 28-day mortality (non-linear p = 0.207), with a cutoff of 83.416. A

 Table 3
 Cox regression analyses of 28-day mortality in patients

 with sepsis-related acute kidney injury

	HR (95%CI)	P value	P for trend
Model 1			< 0.001
Q1	Reference		
Q2	0.64(0.55-0.74)	< 0.001	
Q3	0.59(0.51-0.69)	< 0.001	
Q4	0.50(0.43-0.58)	< 0.001	
Continuous	0.97(0.97–0.98)	< 0.001	
Model 2			< 0.001
Q1	Reference		
Q2	0.64(0.55-0.74)	< 0.001	
Q3	0.60(0.52-0.70)	< 0.001	
Q4	0.51(0.44-0.59)	< 0.001	
Continuous	0.97(0.97–0.98)	< 0.001	
Model 3			< 0.001
Q1	Reference		
Q2	0.79(0.68–0.92)	0.002	
Q3	0.81(0.70-0.95)	0.009	
Q4	0.74(0.63–0.87)	< 0.001	
Continuous	0.99(0.98-1.0)	< 0.001	

Model 1: Unadjusted

Model 2: Adjusted for age, sex, race and ethnicity

Model 3: Adjusted for all baseline variables with p < 0.05 (Table 1) and ALT, AST, TBiL, CRRT, mechanical ventilation status, and nephrotoxic drug exposure HR. Hazard ratio: CI. Confidence interval

significant reduction in the risk of in-hospital mortality was observed with increasing GNRI scores.

Subgroup analysis

The association between nutrition-related risk and inhospital mortality was found to be positive across all subgroups, with no significant interactions identified in the subgroup analysis except for the SOFA score subgroup, as illustrated in Fig. 4.

Discussion

This study provides the first evidence for the GNRI as a robust predictor of short-term mortality and adverse clinical outcomes in elderly patients with S-AKI. Our findings demonstrated that lower GNRI score was independently associated with higher 28-day mortality, increased severity of AKI, and a prolonged stay in the ICU or hospital, even after rigorous adjustment for confounders. These results underscore the critical role of nutritional status in modulating the outcomes of sepsisassociated renal injury, and highlight the utility of the GNRI as a pragmatic tool for risk stratification in this vulnerable population.

Previous studies have demonstrated the prognostic importance of the GNRI in various patient populations, including those with heart failure [18], cancer [24], contrast-induced nephropathy [25], and non-S-AKI [15-17]. The GNRI has been shown to effectively predict adverse outcomes in these groups. However, this was the first study to specifically explore the relationship between GNRI and S-AKI in elderly patients. We specifically addressed the interplay between malnutrition and sepsisdriven renal dysfunction. The linear relationship between GNRI score and mortality (p for non-linearity = 0.207) suggests that even marginal improvements in nutritional status may confer survival benefits in patients with S-AKI. This finding is particularly relevant given the lack of targeted therapies for S-AKI, positioning nutritional optimization as a modifiable intervention.

The GNRI demonstrates superior predictive capability compared with traditional nutritional markers (e.g., BMI, serum albumin) owing to its composite design, which integrates albumin levels with anthropometric parameters. Newer albumin-based indices (such as the albumin-bilirubin score [26], prognostic nutritional index [27], red cell distribution width-albumin ratio [28], and albumin-creatinine ratio [29]) incorporate inflammatory, hepatic, or renal markers to enhance prognostic sensitivity; however, their complexity limits clinical utility in elderly patients with S-AKI. For instance, the red cell distribution width-albumin ratio combines the red cell distribution width (reflecting inflammation/oxidative stress) and albumin, but is confounded by anemia or transfusion [28]; furthermore, the reliance of the albumin-creatinine

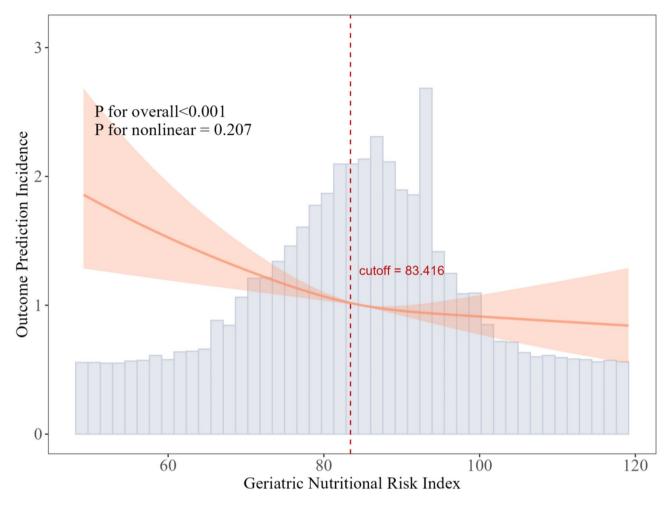


Fig. 3 Restricted cubic smoothing for 28-day mortality risk according to Geriatric Nutritional Risk Index score. Model 3 was used in the analysis

ratio on creatinine may lead to instability in the presence of AKI owing to rapid renal function fluctuations [29]. In contrast, the simplicity of the GNRI (requiring only albumin, weight, and height) facilitates rapid bedside assessment, even in resource-limited settings.

To effectively prevent S-AKI, the most important step is to identify sepsis patients at high risk and intervene against factors that could exacerbate the condition. Elderly people often have malnutrition [30, 31], a condition exacerbated by the natural aging process, which leads to physical frailty and a decrease in energy intake. Therefore, age is a pivotal factor contributing to the prevalence of malnutrition in this patient group. The GNRI is a simple, objective, and well-validated nutritional screening tool specifically designed for hospitalized elderly patients [13], and its application value in assessing elderly patients at risk of S-AKI should not be underestimated. Our analysis shows that, even after adjustment for confounding factors, there was a negative linear correlation between GNRI score and 28-day mortality. Furthermore, across all subgroups, higher nutritional risk was closely associated with a higher hospital mortality rate among elderly patients with S-AKI, thereby confirming the predictive reliability of the GNRI in assessing prognosis. Conducting early and comprehensive nutritional assessments for elderly patients with sepsis, devising personalized nutritional plans, and improving patients' nutritional status may be crucial in ameliorating poor outcomes of patients with S-AKI.

Our analysis has certain limitations. Despite the data being sourced from a large public database, they have not undergone external validation to confirm the predictive utility of the GNRI. Additionally, GNRI score was assessed only at the time of admission. Further research should be conducted to investigate the prognostic implications of dynamic changes in GNRI scores.

Conclusion

The GNRI score at admission can serve as a critical predictor of adverse outcomes in elderly patients with S-AKI in the ICU. Our study findings suggest that the GNRI can be instrumental in identifying elderly patients with S-AKI at higher risk of malnutrition, thereby ensuring timely and effective nutritional support.

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Variable	Count	Percent(%)		HR (95% CI)	P value	P for interaction
Age			i			0.386
>=75	2543	56.3	⊢ ⊷1	0.97 (0.96 to 0.98)	<0.001	
<75	1972	43.7	н	0.97 (0.97 to 0.98)	<0.001	
Gender						0.348
Female	1956	43.3	⊢ -	0.97 (0.96 to 0.98)	<0.001	
Male	2559	56.7	⊷ ¦	0.97 (0.97 to 0.98)	<0.001	
Race						0.583
ASIAN	109	2.4	i	0.95 (0.91 to 0.99)	0.025	
BLACK	332	7.4	⊢ł	0.98 (0.95 to 1.00)	0.027	
WHITE	2966	65.7	H=-1	0.97 (0.96 to 0.98)	<0.001	
OTHERS	1108	24.5	⊢−−	0.98 (0.97 to 0.99)	<0.001	
CKD						0.309
No	3273	72.5	H=-1	0.97 (0.96 to 0.98)	<0.001	
Yes	1242	27.5		0.98 (0.97 to 0.99)	<0.001	
Ventilation						0.758
No	1295	28.7	⊢ 1	0.97 (0.96 to 0.98)	<0.001	
Yes	3220	71.3	•	0.97 (0.97 to 0.98)	<0.001	
Vasopressor						0.212
No	2445	54.2	H-1	0.97 (0.96 to 0.98)	<0.001	
Yes	2070	45.8	H-1	0.98 (0.97 to 0.99)	<0.001	
SOFA						0.017
<=4	3106	68.8	H .	0.97 (0.96 to 0.97)	<0.001	
>4	1409	31.2		0.98 (0.97 to 0.99)	<0.001	
Overall	4515	100	H	0.97 (0.97 to 0.98)	<0.001	
			0.9 1	1.1		

Fig. 4 Subgroup analysis of the association between 28-day mortality and the Geriatric Nutritional Risk Index

Abbreviations

GNRI	Geriatric Nutritional Risk Index
S-AKI	Sepsis-related acute kidney injury
BMI	Body mass index
Scr	Serum creatinine
CRRT	Continuous renal replacement therapy
SOFA	Sequential organ failure assessment
APSIII	Acute physiology and chronic health evaluation III
SAPSII	Simplified acute physiology score II
OASIS	Organ assessment score in the ICU
GCS	Glasgow coma scale
HR	Hart rate
RR	Respiratory rate
NBPM	Non-invasive blood pressure mean
NBPD	Non-invasive blood pressure diastolic
NBPS	Non-invasive blood pressure systolic
CKD	Chronic kidney disease
COPD	Chronic obstructive pulmonary disease

Supplementary Information

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Supplementary Material 1

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Author contributions

CZ was responsible for designing the protocol, conducting the search and analyzing data from MIMIC-IV, interpreting the results, and creating summary tables of the findings. KC, WM, and MY were responsible for designing the review protocol and extracting data. KC, MY, and CC contributed to updating reference lists and provided feedback on the report. KC, WM, and SG contributed to analyzing data, interpreting the results, creating tables and figures, and writing the paper. All authors contributed to the article and approved the submitted version.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

The requirement for ethical approval for this study was waived by the Institutional Review Board of Zhejiang Hospital because the data were accessed from the MIMIC-IV database, a publicly available database. The

requirement for written informed consent was waived by the Institutional Review Board of Zhejiang Hospital, owing to the retrospective design of this study. All research methods were conducted in compliance with applicable guidelines and regulations.

Consent for publication

All authors have consented to the publication of the present manuscript.

Clinical trial number

Not applicable.

Competing interests

The authors declare no competing interests.

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References

- 1. Zarjou A. Sepsis and acute kidney injury. J Am Soc Nephrol. 2011;22(6):999–1006.
- Bellomo R, Kellum JA, Ronco C, et al. Acute kidney injury in sepsis. Intensive Care Med. 2017;43:816–28.
- Godin M, Murray P, Mehta RL. Clinical approach to the patient with AKI and sepsis. Semin Nephrol. 2015;35:12–22.
- Hoste EA, Bagshaw SM, Bellomo R, et al. Epidemiology of acutekidney injury in critically ill patients: the multinational AKI-EPIstudy. Intensive Care Med. 2015;41:1411–23.
- Bagshaw SM, Lapinsky S, Dial S, Cooperative Antimicrobial Therapy of Septic Shock (CATSS) Database Research Group. Acute kidney injury in septic shock: clinical outcomes and impact of duration of hypotension prior to initiation of antimicrobial therapy. Intensive Care Med. 2009;35:871–81.
- Kellum JA, Chawla LS, Keener C, et al. The effects of alternative resuscitation strategies on acute kidney injury in patients with septic shock. Am J Respir Crit Care Med. 2016;193(3):281–7.
- Wald R, Quinn RR, Luo J, et al. Chronic dialysis and death among survivors of acute kidney injury requiring dialysis. JAMA. 2009;302(11):1179–85.
- Bagshaw SM, Uchino S, Bellomo R et al. Beginning and ending supportive therapy for the kidney (BEST kidney) investigators, Septic acute kidney injury in critically ill patients: clinical characteristics and outcomes. Clin J Am Soc Nephrol. 2007;2:431–9.
- Xiang X, Zhu X, Zhang L. Association of malnutrition with risk of acute kidney injury: a systematic review and meta-analysis. Int Journal of Clinical Practice. 2023; 1:9910718.
- Lanctin DP, Merced-Nieves F, Mallett RM, Arensberg MB, Guenter P, Sulo S, Platts-Mills TF. Prevalence and economic burden of malnutrition diagnosis among patients presenting to united States emergency departments. Acad Emerg Med. 2021;28(3):325–35.
- Yu J, Li D, Jia Y, Li F, Jiang Y, Zhang Q, et al. Nutritional risk screening 2002 was associated with acute kidney injury and mortality in patients with acute coronary syndrome: insight from the REACP study. Nutr Metab Cardiovasc Dis. 2021;31(4):1121–8.
- 12. McCarthy MS, Phipps SC. Special nutrition challenges: current approach to acute kidney injury. Nutr Clin Pract. 2014;29(1):56–62.
- Cereda E, Pedrolli C. The use of the geriatric nutritional risk index (GNRI) as a simplified nutritional screening tool. Am J Clin Nutr. 2008;87(6):1966–7.
- 14. Zhao Y et al. Association between geriatric nutritional risk index and frailty in older hospitalized patients. Clin Interv Aging. 2021;16:1241–9.
- Zhao D, Zhou D, Li T, et al. The relationship between geriatric nutritional risk index (GNRI) and in-hospital mortality in critically ill patients with acute kidney injury (AKI). BMC Anesthesiol. 2024;24:313.

- Page 10 of 10
- 16. Liao D, et al. The prognostic effects of the geriatric nutritional risk index on elderly acute kidney injury patients in intensive care units. Front Med. 2023;10:1165428.
- Zhao X, Li J, Liu H, Shi K, He Q, Sun L, Xue J, Jiang H, Wei L. Association of geriatric nutritional risk index with short-term mortality in patients with severe acute kidney injury: a retrospective cohort study. Ren Fail. 2024;46(2):2374449.
- Zhang S, et al. Geriatric nutritional risk index is associated with the occurrence of acute kidney injury in critically ill patients with acute heart failure. Ren Fail. 2024;46(1):2349122.
- Johnson AEW, Bulgarelli L, Shen L, Gayles A, Shammout A, Horng S, Pollard TJ, Hao S, Moody B, Gow B, et al. MIMIC-IV, a freely accessible electronic health record dataset. Sci Data. 2023;10(1):1.
- Zarbock A, Nadim MK, Pickkers P, et al. Sepsis-associated acute kidney injury: consensus report of the 28th acute disease quality initiative workgroup. Nat Rev Nephrol. 2023;19(6):401–17.
- 21. Singer M, et al. The third international consensus definitions for sepsis and septic shock (Sepsis-3). JAMA. 2016;315:801–10.
- 22. Ad-hoc Ad-hoc working group of E, Fliser D, Laville M, Covic A, Fouque D, Vanholder R, et al. A European renal best practice (ERBP) position statement on the kidney disease improving global outcomes (KDIGO) clinical practice guidelines on acute kidney injury: part 1: definitions, Conservative management and contrast-induced nephropathy. Nephrol Dial Transpl. 2012;27:4263–72.
- Bouillanne O, Morineau G, Dupont C, Coulombel I, Vincent JP, Nicolis I, et al. Geriatric nutritional risk index: a new index for evaluating at-risk elderly medical patients. Am J Clin Nutr. 2005;82:777–83.
- 24. Lidoriki I, Schizas D, Frountzas M, et al. GNRI as a prognostic factor for outcomes in Cancer patients: a systematic review of the literature. Nutr Cancer. 2021;73(3):391–403.
- Li D, Chen Z, He W, et al. The association between nutritional risk and contrast-induced acute kidney injury in patients undergoing coronary angiography: a cross-sectional study. Nutr J Sep. 2022;16(1):56.
- Deng M, Ng SWY, Cheung ST, Chong CCN. Clinical application of Albumin-Bilirubin (ALBI) score: the current status. Surgeon. 2020;18(3):178–86. https:// doi.org/10.1016/j.surge.2019.09.002. Epub 2019 Oct 8. PMID: 31601470.
- Suzuki E, Kawata N, Shimada A, Sato H, Anazawa R, Suzuki M, Shiko Y, Yamamoto M, Ikari J, Tatsumi K, Suzuki T. Prognostic nutritional index (PNI) as a potential prognostic tool for exacerbation of COPD in elderly patients. Int J Chron Obstruct Pulmon Dis. 2023;18:1077–90. https://doi.org/10.2147/COPD. S385374. PMID: 37309393; PMCID: PMC10257926.
- Li N, Li J, Wang K. Association between red cell distribution width-albumin ratio and all-cause mortality in intensive care unit patients with heart failure. Front Cardiovasc Med. 2025;12:1410339. https://doi.org/10.3389/fcvm.2025.1 410339. PMID: 39901900; PMCID: PMC11788307.
- Wang J, Li N, Mu Y, Wang K, Feng G. Association between serum albumin creatinine ratio and all-cause mortality in intensive care unit patients with heart failure. Front Cardiovasc Med. 2024;11:1406294. https://doi.org/10.3389 /fcvm.2024.1406294. PMID: 39027002; PMCID: PMC11254761.
- Corish CA, Bardon LA. Malnutrition in older adults: screening and determinants. Proc Nutr Soc. 2019;78(3):372–9.
- Zhang Z, Pereira SL, Luo M, Matheson EM. Evaluation of blood biomarkers associated with risk of malnutrition in older adults: a systematic review and Meta-analysis. Nutrients. 2017;9(8):829.

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