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Prognostic value of the Geriatric Nutritional Risk Index in mortality prediction among critically ill acute myocardial infarction patients

Shanshan Tang[1†], Chengcheng Wu[1†], Yanbin Su[1†] and Yongle Li[1*]

Abstract

Background Nutritional status is a key factor influencing outcomes in critically ill patients with acute myocardial infarction (AMI). This study investigated the association between the Geriatric Nutritional Risk Index (GNRI) and mortality among ICU-admitted AMI patients, as well as GNRI's potential to improve the predictive accuracy of current scoring systems.

Methods In this retrospective cohort study, data from 5,506 ICU-admitted AMI patients were sourced from three open-access critical care databases. Based on GNRI scores, patients were grouped into two categories: GNRI ≤ 98 and GNRI > 98. Statistical tools such as logistic regression and Cox proportional hazards models assessed in-hospital and 30-day mortality. Kaplan-Meier survival curves and restricted cubic splines analyzed survival trends and dose-response relationships. Sensitivity analyses, including propensity score matching (PSM), inverse probability weighting (IPW), and dropping missing data analysis validated the robustness of findings. The receiver operating characteristic (ROC) curve compared GNRI's predictive ability with SOFA and APSIII scores. A sensitivity analysis was performed using a four-tier GNRI classification: no risk (> 98), low risk (92–98), moderate risk (82–<92), and major risk (< 82) to further explore its gradient relationship with mortality.

Results Patients with GNRI ≤ 98 showed higher mortality rates for in-hospital (21.8% vs. 10.4%) and 30-day (22.5% vs. 10.7%) outcomes. GNRI displayed an inverse correlation with in-hospital mortality (OR 0.51, 95% CI 0.43–0.60) and 30-day mortality (HR 0.57, 95% CI 0.50–0.66), even after adjusting for confounders. Subgroup analysis emphasized GNRI's reliability as a predictive marker, particularly in patients with eGFR ≥ 90. ROC analysis confirmed GNRI's predictive performance (AUC = 0.64) and its enhancement of SOFA (AUC = 0.72) and APSIII (AUC = 0.66) scores (all $p < 0.001$). Sensitivity analyses reinforced GNRI's link to mortality.

Conclusion GNRI serves as a robust predictor of in-hospital and 30-day mortality among critically ill AMI patients. Its integration with existing scoring systems improves risk stratification in this high-risk population.

Keywords Geriatric nutritional risk index, Acute myocardial infarction, Mortality, Nutritional status, Critical care, Risk prediction

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Introduction

Nutritional status is a cornerstone of clinical outcomes, particularly for critically ill patients diagnosed with acute myocardial infarction (AMI) [1]. The geriatric nutritional risk index (GNRI), a non-invasive tool using serum albumin levels and body weight, has been shown to predict mortality and assist in clinical decision-making in critically ill patients [2–7]. However, its application and potential significance in AMI patients within the ICU setting remain insufficiently studied.

Emerging studies have hinted at GNRI's potential to augment traditional scoring systems, underscoring the need for deeper exploration. The sequential organ failure assessment (SOFA) and acute physiology and chronic health evaluation III (APSIII) scores are used to assess illness severity in ICU patients but do not include nutritional status, a key factor in recovery and survival [8]. Integrating GNRI into these models could improve predictive accuracy. Emerging evidence suggests GNRI may provide additional insights into nutritional health in critically ill patients, warranting further investigation.

This multicenter cohort study aims to investigate the role of GNRI in refining risk stratification and predicting mortality among critically ill AMI patients. By comparing GNRI with SOFA and APSIII scores, the study seeks to enhance understanding of how nutritional health affects outcomes and refine ICU decision-making.

Materials and methods

Study design and setting

This study utilized a retrospective cohort design, analyzing anonymized data from patients hospitalized for AMI. The data were obtained from three publicly accessible critical care databases. The eICU Collaborative Research Database (eICU-CRD, version 2.0) includes information from over 200,000 ICU patients across 200 medical centers during 2014–2015 [9]. The Medical Information Mart for Intensive Care (MIMIC-III, version 1.4), and MIMIC-IV (version 3.1) databases contain data from the ICUs of Beth Israel Deaconess Medical Center (BIDMC), spanning 2001–2012 and 2008–2022, respectively [10].

Clinical variables were extracted using SQL in conjunction with PostgreSQL (version 13.0) [11] and Navicat (version 16.0), ensuring uniformity and standardization of data processing. For variables recorded multiple times during a single hospitalization, only the first recorded measurement was used to establish baseline values. To enhance transparency and reproducibility, the data extraction code has been made publicly available on GitHub (https://github.com/MIT-LCP/mimic-iv). Ethical approval was secured for this research, and informed consent was waived due to the retrospective nature of the analysis and the anonymized nature of the datasets.

Patients

Patients initially admitted to the ICU were identified from the databases. The inclusion criteria specified adult patients diagnosed with AMI, defined according to the International Classification of Disease (ICD), Ninth or Tenth Revision (see Table S1). Patients were excluded if they were younger than 65 years, had ICU stays shorter than 24 h, or lacked critical data, such as serum albumin, height, or weight. After applying these criteria, a total of 5,506 patients with AMI were included in the final analysis (Fig. 1).

Covariates

Covariates were chosen based on their clinical relevance and significance to outcomes in critically ill AMI patients, guided by existing literature and expert judgment. These included: (1) Demographics: age, gender, race, and body mass index (BMI), calculated as weight (kg)/height $(m)^2$; (2) Physical exam findings: respiratory rate, heart rate, and mean blood pressure (MBP); (3) The type of myocardial infarction: ST-elevation myocardial infarction (STEMI) and non-ST-elevation myocardial infarction (NSTEMI); (4) Comorbidities: hypertension, diabetes, heart failure, atrial fibrillation, stroke, renal failure, and cancer; (5) Laboratory tests: hemoglobin, white blood cell (WBC) count, platelet count, creatinine, and albumin measured within 24 h of ICU admission; and (6) Treatments: Use of angiotensin-converting enzyme inhibitors/angiotensin receptor blockers (ACEI/ARB), beta-blockers, statins, hemodialysis, and mechanical ventilation.

Additional factors influencing outcomes included two widely used illness severity scores: The SOFA score evaluates six organ systems (respiration, coagulation, liver, cardiovascular, central nervous system, and renal) on a 0–4 scale, with higher scores indicating greater dysfunction. The APSIII score incorporates physiological measurements, laboratory data, and chronic health conditions to predict mortality risk, with higher scores reflecting greater severity.

The primary variable assessing nutritional status was GNRI, calculated as [6]: GNRI = $(1.489 \times 10 \times$ serum albumin (g/dL)) + $(41.7 \times$ weight (kg)/ideal body weight (kg)). Ideal body weight (IBW) was determined using gender-specific formulas: IBW = 0.75 × height (cm) − 62.5 for males, and IBW = 0.60 × height (cm) − 40 for females. Patients were categorized as no risk (GNRI > 98) or at risk of malnutrition (GNRI ≤ 98) [12]. Additionally, patients were further stratified into four groups: no risk (GNRI > 98), low risk (GNRI 92–98), moderate risk (GNRI 82–<92), and major risk (GNRI < 82) [6].

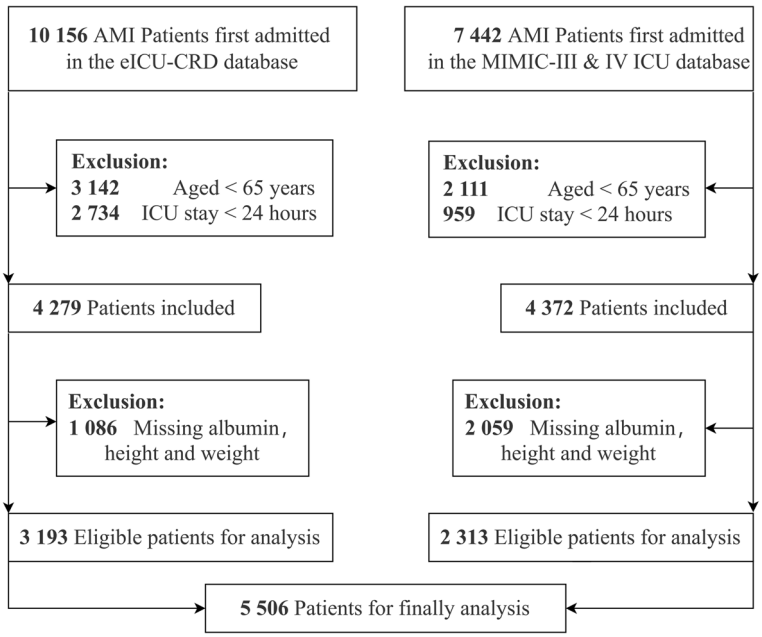

Fig. 1 The flowchart of patients' selection

The Prognostic Nutritional Index (PNI) was calculated as: PNI = serum albumin (g/L) + 5 × total lymphocyte count ($\times 10^9$/L) [13].

Outcome

The main outcomes were in-hospital mortality (death during hospitalization) and 30-day mortality (deaths within 30 days of admission, regardless of hospitalization status).

Statistical analysis

The distribution of variables was evaluated using histograms, Q-Q plots, and the Kolmogorov-Smirnov test to guide the choice of statistical methods. Continuous variables were reported as means with standard deviations (SD) for normally distributed data, and as medians with interquartile ranges (IQR) for non-normally distributed data. Categorical variables were summarized as frequencies and percentages. Group comparisons were performed using the Student's t-test or Mann-Whitney U test for continuous variables and the chi-square test or Fisher's exact test for categorical data, depending on the distribution.

Potential confounders were selected based on clinical significance, evidence from prior research, or statistical significance identified during univariate analysis. Dose-response relationships were analyzed using restricted cubic spline models, with knots positioned at the 25th, 50th, and 75th percentiles of GNRI. Receiver operating characteristic (ROC) curve analysis compared GNRI combined with SOFA and APSIII scores against their individual predictive power to determine GNRI's added value. Kaplan-Meier survival curves, stratified by malnutrition risk categories, were evaluated using the log-rank test.

Logistic regression models estimated odds ratios (ORs) with 95% confidence intervals (CIs) for in-hospital mortality, while Cox proportional hazards models were used to compute hazard ratios (HRs) and 95% CIs for 30-day mortality. Proportional hazards assumptions were verified using log-log plots and interaction terms with survival time as needed. Three models were developed for analysis: Model 1 was unadjusted; Model 2 adjusted for demographics and vital signs; Model 3 additionally included types of AMI, comorbidities, laboratory tests, and treatments.

Subgroup analyses examined GNRI's relationship with mortality across predefined variable categories.

Missing data were addressed using multiple imputation with chained equations, implemented via the R package mice [14], to reduce bias and preserve statistical power. For most variables, missing data rates were below 20% (see Table S2). Sensitivity analyses further validated the findings using complete-case data.

Robustness of the results was assessed through additional methods, including propensity score adjustment (PSA), propensity score matching (PSM), and inverse probability weighting (IPW). Effect sizes and *p*-values from these models were compared to confirm the consistency of GNRI's association with outcomes.

All analyses were conducted using R Statistical Software (version 4.2.2) and the Free Statistics Analysis Platform (version 2.1), with significance set at $p < 0.05$.

Results

Baseline characteristics

Baseline characteristics of patients, stratified by GNRI categories, are outlined in Table 1. The median age of the cohort was 80.8 years. Gender and race distributions showed no significant differences between groups. Patients in the GNRI ≤ 98 group were older and had lower BMI. This group also exhibited elevated respiratory and heart rates, though MBP differences were not statistically significant.

Laboratory findings revealed lower hemoglobin levels (10.1 ± 2.9 g/dL vs. 10.9 ± 2.7 g/dL) and albumin concentrations (2.8 ± 0.7 mg/dL vs. 3.6 ± 0.4 mg/dL) in the GNRI ≤ 98 group, along with higher WBC. Renal impairment was more pronounced, reflected by lower estimated glomerular filtration rates. (all $p < 0.001$)

Clinically, the GNRI ≤ 98 group had higher rates of hypertension, diabetes, renal failure, and cancer and was less likely to receive β-blockers or statins. Hospital stays were longer (8.3 vs. 7.6 days), and mortality rates were significantly higher, both in-hospital (21.8% vs. 10.4%) and at 30 days (22.5% vs. 10.7%). (all $p < 0.001$)

Restricted cubic splines analysis

Restricted cubic spline models revealed an L-shaped nonlinear relationship between GNRI and mortality outcomes. As shown in Fig. 2, declining GNRI levels were associated with higher odds ratios (ORs) for in-hospital mortality (panel A) and hazard ratios (HRs) for 30-day mortality (panel B), with p-values for nonlinearity < 0.05.

ROC curve analysis

ROC curves comparing the predictive performance of GNRI, PNI, SOFA, and APSIII for 30-day mortality are presented in Fig. 3. GNRI achieved an area under the curve (AUC) of 0.64 (95% CI: 0.62–0.66), exceeding that of PNI [0.62 (95% CI: 0.60–0.64)] and APSIII [0.60 (95% CI: 0.58–0.62)] but lower than SOFA [0.68 (95% CI: 0.66–0.70)] (panel A). Combining GNRI with SOFA increased the AUC to 0.72 (95% CI: 0.70–0.74) (panel B)., while integration with APSIII raised the AUC from 0.60 to 0.66 (95% CI: 0.64–0.68) (panel C).

Kaplan–Meier analysis

The Kaplan-Meier survival curves (Fig. 4A) show that patients with GNRI ≤ 98 experienced significantly lower 30-day survival rates compared to those with GNRI > 98 (log-rank test, $p < 0.001$). Using the four-tier GNRI categorization, recalculated survival curves (Fig. 4B) reveal a clear gradient: as GNRI scores decrease from "no risk" to "major risk," survival rates decline proportionally.

Association between GNRI and mortality in critically ill patients with AMI

Logistic regression models demonstrated a strong inverse relationship between GNRI levels and mortality outcomes (Table 2). In the unadjusted model (Model 1), higher GNRI levels were associated with reduced in-hospital mortality (OR 0.41, 95% CI: 0.36–0.48) and 30-day mortality (HR 0.45, 95% CI: 0.39–0.51). After adjusting for demographics and vital signs (Model 2), the association remained robust (in-hospital OR 0.46, 95% CI: 0.39–0.54; 30-day HR 0.49, 95% CI: 0.43–0.57). Further adjustments for types of AMI, comorbidities, laboratory tests, and treatments in Model 3 confirmed these findings, with in-hospital OR 0.51 (95% CI: 0.43–0.60) and 30-day HR 0.56 (95% CI: 0.48–0.65). Across all models, each 10-unit GNRI increase was consistently linked to significantly lower mortality rates ($p < 0.001$).

Additionally, a multivariate regression analysis was performed to examine in-hospital and 30-day mortality across the four GNRI categories. The results, presented in Table 3, demonstrate a consistent gradient reduction in risk as GNRI improves from "major risk" to "no risk".

Subgroup analysis

Subgroup analyses for in-hospital and 30-day mortality in AMI patients were conducted, including database, types of AMI, age, gender, race, heart failure, hypertension, diabetes, renal failure, stroke, and eGFR levels.

For in-hospital mortality (Figure S1), GNRI showed a protective effect across most subgroups, with the strongest effect in patients with eGFR ≥ 90 (OR 0.31, 95% CI 0.19–0.53, p for interaction < 0.001).

For 30-day mortality (Figure S2), GNRI remained predictive across all subgroups, with the strongest effect also in eGFR ≥ 90 (HR 0.32, 95% CI 0.20–0.53, $p < 0.001$) and a stronger effect in males (HR 0.47, 95% CI 0.39–0.58, p for interaction = 0.001).

Sensitivity analysis

Propensity score matching (PSM) balanced baseline characteristics, achieving standardized mean differences (SMD) below 0.1 for all covariates (Table S3).

Inverse probability weighting (IPW) confirmed GNRI's significant association with reduced in-hospital mortality (OR 0.55, 95% CI: 0.48–0.64, $p < 0.001$) and 30-day mortality (HR 0.59, 95% CI: 0.51–0.67, $p < 0.001$). Consistent results from PSM and additional sensitivity analyses that excluded patients with missing data further validated these findings (Table 4).

Table 1 Baseline characteristics of the study participants

Variables	Total ($n=5\,506$)	GNRI ≤ 98 ($n=2\,510$)	GNRI > 98 ($n=2\,996$)	*P* value
Demographics				
Age, (year)	80.8 ± 33.0	84.7 ± 39.8	77.5 ± 25.5	< 0.001
Gender male, n (%)	3218 (58.4)	1444 (57.5)	1774 (59.2)	0.207
Race, n (%)				0.106
White	4139 (75.2)	1861 (74.1)	2278 (76.0)	
Other	1367 (24.8)	649 (25.9)	718 (24.0)	
BMI, (kg/m^2)	27.6 ± 5.8	24.0 ± 4.1	30.6 ± 5.2	< 0.001
Vital signs				
Respiratory rate	21.0 (14.0, 31.0)	24.0 (15.0, 32.0)	19.0 (13.0, 29.0)	< 0.001
Heart rate, (min^{-1})	84.6 ± 18.8	86.8 ± 19.5	82.7 ± 18.0	< 0.001
MBP, (mmHg)	86.3 ± 20.9	86.0 ± 21.7	86.7 ± 20.2	0.235
Scores				
SOFA	3.0 (1.0, 6.0)	3.0 (1.0, 6.0)	3.0 (1.0, 6.0)	< 0.001
APSIII	10.0 (7.0, 16.0)	10.0 (7.0, 15.0)	10.0 (6.0, 17.0)	0.973
GNRI	100.0 ± 14.8	87.2 ± 8.4	110.6 ± 9.6	< 0.001
PNI	39.3 (32.9, 46.0)	34.8 (29.5, 40.7)	42.9 (37.0, 48.8)	< 0.001
Types of AMI, n (%)				0.330
NSTEMI	3884 (70.5)	1787 (71.2)	2097 (70.0)	
STEMI	1622 (29.5)	723 (28.8)	899 (30.0)	
Co-morbidities, n (%)				
Hypertension	3775 (68.6)	1633 (65.1)	2142 (71.5)	< 0.001
Diabetes	1418 (25.8)	523 (20.8)	895 (29.9)	< 0.001
Heart failure	1873 (34.0)	890 (35.5)	983 (32.8)	0.039
Atrial fibrillation	1463 (26.6)	658 (26.2)	805 (26.9)	0.584
Stroke	918 (16.7)	410 (16.3)	508 (17.0)	0.538
Renal failure	1074 (19.5)	547 (21.8)	527 (17.6)	< 0.001
Cancer	259 (4.7)	150 (6.0)	109 (3.6)	< 0.001
Laboratory tests				
Hemoglobin, (g/dL)	10.6 ± 2.8	10.1 ± 2.9	10.9 ± 2.7	< 0.001
WBC, (K/μL)	11.4 (8.5, 15.2)	11.8 (8.6, 15.8)	11.0 (8.4, 14.7)	< 0.001
Platelet, (K/μL)	189.0 (144.0, 244.0)	190.5 (143.0, 255.0)	187.0 (145.0, 237.0)	0.025
eGFR, ($mL/min/1.73\ m^2$)	60.0 (35.6, 86.2)	56.0 (31.7, 83.8)	64.6 (39.2, 90.0)	< 0.001
Albumin, (mg/dl)	3.3 (2.8, 3.7)	2.8 (2.5, 3.2)	3.6 (3.2, 3.9)	< 0.001
Treatments, n (%)				
ACEI/ARB	1085 (19.7)	456 (18.2)	629 (21.0)	0.009
β-blockers	2949 (53.6)	1222 (48.7)	1727 (57.6)	< 0.001
Anti-platelet	3422 (62.2)	1403 (55.9)	2019 (67.4)	< 0.001
Statin	2405 (43.7)	908 (36.2)	1497 (50.0)	< 0.001
Hemodialysis	365 (6.6)	189 (7.5)	176 (5.9)	0.014
Mechanical ventilation	2249 (40.8)	1027 (40.9)	1222 (40.8)	0.923
Length of hospital stay, (day)	7.9 (4.6, 13.1)	8.3 (5.0, 14.4)	7.6 (4.1, 12.0)	< 0.001
Mortality, n (%)				
In-hospital	859 (15.6)	548 (21.8)	311 (10.4)	< 0.001
30-day	888 (16.1)	566 (22.5)	322 (10.7)	< 0.001

Abbreviations: GNRI geriatric nutritional risk index,*BMI* body mass index, *MBP* mean arterial pressure, *SOFA* sequential organ failure assessment, *APSIII* acute physiology and chronic health evaluation III, *PNI* prognostic nutritional index, *NSTEMI* non-ST-segment elevation myocardial infarction, *STEMI* ST-segment elevation myocardial infarction, *WBC* white blood cell, *eGFR* estimated glomerular filtration rate, *ACEI/ARB* angiotensin converting enzyme inhibitors/angiotension receptor blockers

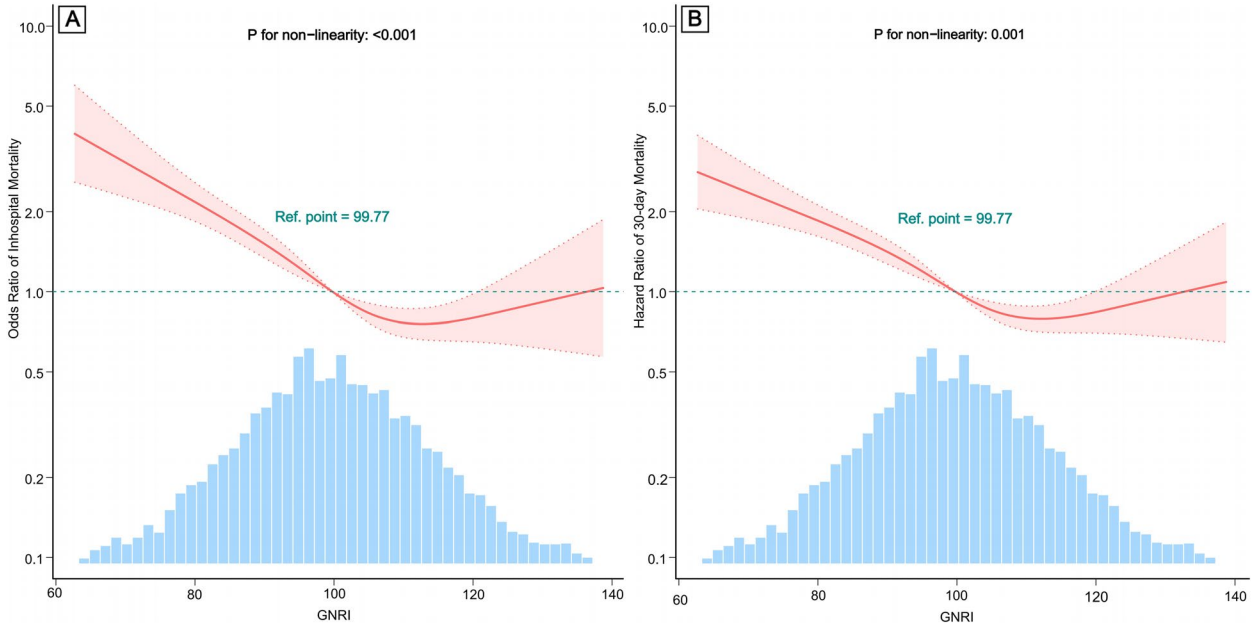

Fig. 2 Dose-response relationship between the GNRI and in-hospital (**A**), 30-day (**B**) mortality in AMI patients. Solid and dashed lines indicate the predicted value and 95% CI

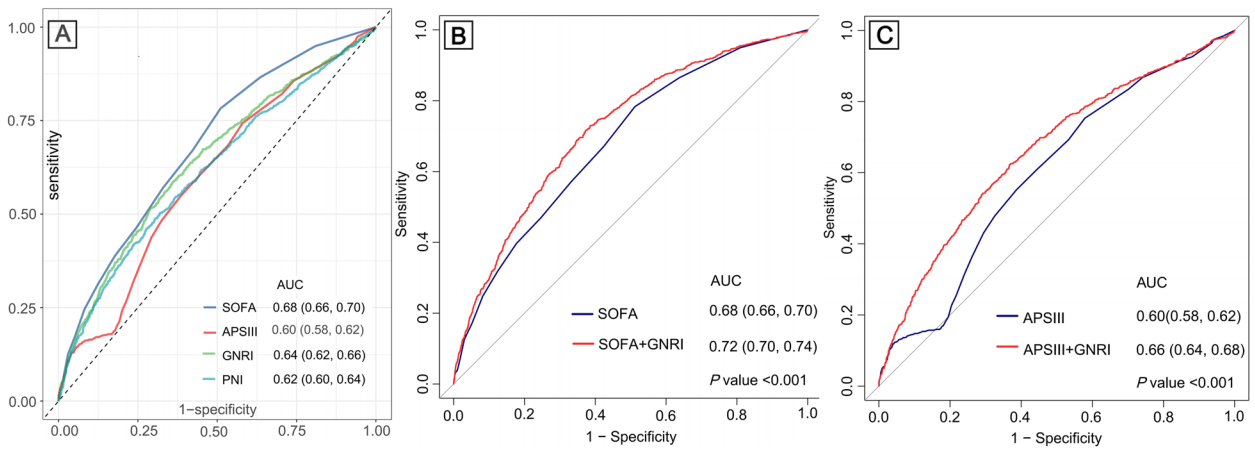

Fig. 3 ROC curves for the prediction of 30-day mortality. Abbreviations: GNRI, geriatric nutritional risk index; SOFA, sequential organ failure assessment; APSIII, acute physiology and chronic health evaluation III; PNI, prognostic nutritional index; AUC, the area under the curve

Discussion

This study identifies GNRI as a valuable predictor of in-hospital and 30-day mortality in critically ill patients with AMI. Patients with GNRI ≤ 98, indicative of high malnutrition risk, demonstrated significantly elevated mortality rates compared to those with GNRI > 98. Adjusted multivariable analyses further validated an inverse association between GNRI and mortality. Moreover, the analysis revealed a consistent gradient reduction in risk as GNRI improves from "major risk" to "no risk". Subgroup analyses highlighted the consistency of GNRI's predictive ability, with the strongest protective effect observed in patients with preserved renal function. Moreover, the addition of GNRI improved the predictive performance of SOFA (AUC 0.68 to 0.72) and APSIII (AUC 0.60 to 0.66), underscoring its utility in enhancing risk stratification.

GNRI offers superior predictive value for short-term mortality in critically ill AMI patients compared to PNI (AUC: 0.64 [0.62, 0.66] vs. 0.62 [0.60, 0.64]). By assessing both nutritional status and inflammation, GNRI provides a more comprehensive evaluation of the factors influencing AMI prognosis. While PNI focuses primarily on immune function, GNRI offers a broader

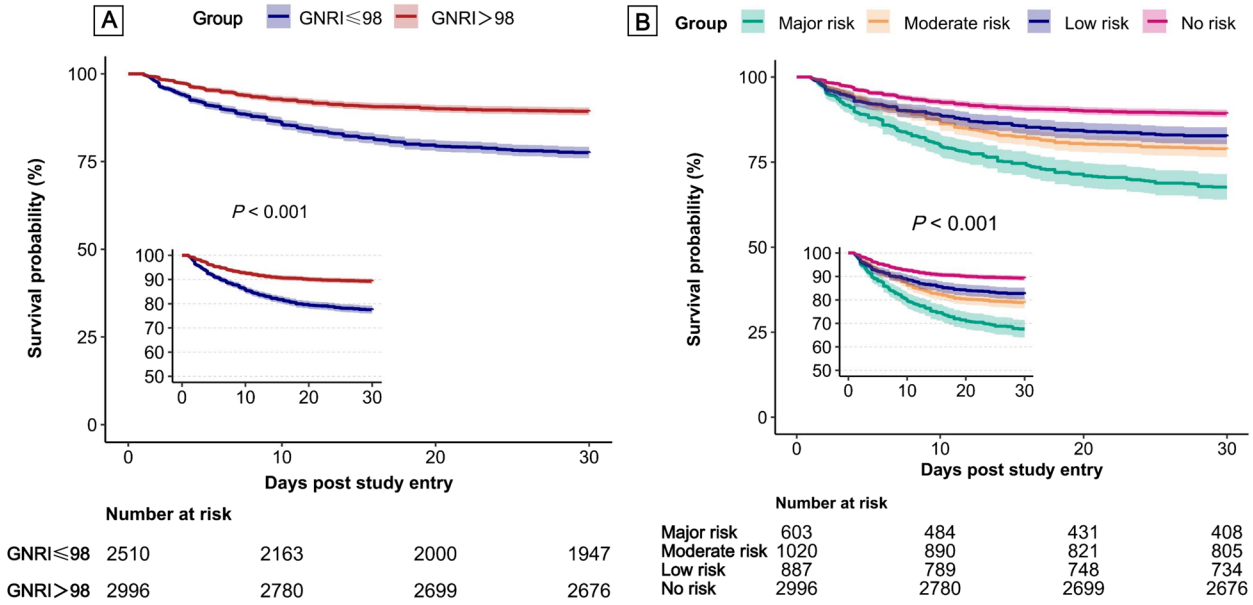

Fig. 4 Kaplan–Meier survival curves for 30-day mortality

Table 2 Association between GNRI and mortality in AMI patients

Mortality	Model 1	Model 2	Model 3
In-hospital, OR (95%CI)			
Per 10 unit increase	0.71 (0.67, 0.75)	0.74 (0.70, 0.78)	0.77 (0.73, 0.81)
GNRI ≤ 98 is used as reference	0.41 (0.36, 0.48)	0.46 (0.39, 0.54)	0.51 (0.43, 0.60)
30-day, HR (95%CI)			
Per 10 unit increase	0.73 (0.70, 0.77)	0.76 (0.72, 0.79)	0.81 (0.77, 0.85)
GNRI ≤ 98 is used as reference	0.45 (0.39, 0.51)	0.49 (0.43, 0.57)	0.56 (0.48, 0.65)

Abbreviations: GNRI geriatric nutritional risk index, *OR* odds ratio, *CI* confidential interval, *HR* hazard ratio

Model 1 adjust for: none

Model 2 adjust for: demographic information and vital signs

Model 3 adjust for: model 2 + types of AMI, comorbidities and laboratory tests, treatments

assessment, making it a more reliable tool for risk stratification in clinical practice. This underscores GNRI's potential to improve patient outcomes through the early identification of high-risk individuals.

The nonlinear L-shaped relationship between GNRI and mortality reveals critical thresholds for intervention. This pattern suggests that nutritional support may be most beneficial when targeted at patients below specific GNRI values, while also considering the risk of misclassification due to fluid imbalances or edema. We recommend combining GNRI with complementary assessment tools like NRS-2002 and implementing cautious, targeted interventions based on these thresholds. Future research should focus on validating these cutoff points in diverse critical care populations, particularly those with significant fluid shifts.

The findings show that GNRI predicts outcomes better in males, possibly due to differences in metabolism and inflammation, and in patients with preserved kidney function (eGFR ≥ 90), where it better reflects nutritional status. GNRI could be a useful tool for risk assessment in these groups, helping guide targeted interventions.

This study highlights GNRI's potential to guide clinical interventions and improve outcomes in ICU-admitted AMI patients. Nutritional strategies, such as protein-energy supplementation, enteral nutrition adjustments, and correcting deficiencies like vitamin D or omega-3 fatty acids, may help reduce risks associated with low

Table 3 Association between GNRI and mortality in AMI patients categorized by the four-tier GNRI classification

Mortality	Event (%)	Model 1	Model 2	Model 3
In-hospital, OR (95%CI)				
Major risk	186 (30.8)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
Moderate risk	213 (20.9)	0.59 (0.47, 0.74)	0.62 (0.49, 0.78)	0.67 (0.52, 0.86)
Low risk	149 (16.8)	0.45 (0.35, 0.58)	0.49 (0.38, 0.63)	0.56 (0.43, 0.73)
No risk	311 (10.4)	0.26 (0.21, 0.32)	0.30 (0.24, 0.37)	0.36 (0.28, 0.45)
30-day, HR (95%CI)				
Major risk	195 (32.3)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
Moderate risk	216 (21.2)	0.62 (0.51, 0.75)	0.64 (0.52, 0.77)	0.71 (0.58, 0.86)
Low risk	155 (17.5)	0.50 (0.41, 0.62)	0.54 (0.44, 0.67)	0.65 (0.52, 0.81)
No risk	322 (10.7)	0.30 (0.25, 0.35)	0.34 (0.28, 0.40)	0.43 (0.36, 0.52)

Abbreviations: GNRI geriatric nutritional risk index, *OR* odds ratio, *CI* confidential interval, *HR* hazard ratio

Model 1 adjust for: none

Model 2 adjust for: demographic information and vital signs

Model 3 adjust for: model 2 + types of AMI, comorbidities and laboratory tests, treatments

GNRI. Since GNRI relies on simple parameters like albumin levels and body weight, it can be easily incorporated into routine ICU assessments alongside tools such as the Nutritional Risk Screening 2002 to identify malnutrition early and prioritize interventions. By identifying nutritional and inflammatory risks, GNRI also provides a basis for initiating nutritional and anti-inflammatory therapies, making it a valuable tool for improving patient outcomes.

The findings align with previous research demonstrating GNRI's role in predicting outcomes in critically ill populations [1, 2, 15]. Consistent with studies on heart failure [16] and acute kidney injury [15, 17], this study affirms GNRI's relevance in predicting mortality and optimizing risk stratification for AMI patients.

In our study, we found a strong link between lower GNRI and short-term all-cause mortality. Yoo et al. showed that GNRI at hospital admission predicted in-hospital mortality (OR, 2.48; 95% CI, 1.55–3.95) and post-MI complications (OR, 2.13; 95% CI, 1.61–2.84;) in AMI patients [18]. A large Chinese study also revealed that in patients with non-ST-segment elevation acute coronary syndrome (NSTE-ACS) undergoing PCI, lower GNRI was associated with worse outcomes (HR, 1.159, 95% CI 1.130–1.189) during a 3-year follow-up [19]. However, these studies are relatively small and may not apply to the US population. Based on these findings, we suggest incorporating nutritional interventions in managing critically ill AMI patients, particularly those at high nutritional risk. Integrating GNRI with SOFA and APSIII scores improves predictive accuracy, especially in patients with preserved renal function. This approach offers a new framework for adding nutritional assessment to clinical decision-making. Early malnutrition detection using GNRI could enable targeted interventions to improve nutritional status and potentially enhance outcomes.

The mechanisms linking GNRI to adverse outcomes in AMI patients include: (1) Hypoalbuminemia and inflammation [20, 21]: Low GNRI reflects systemic inflammation and hypoalbuminemia, which exacerbate endothelial dysfunction, impair vascular stability, and heighten inflammatory responses, contributing to worse cardiovascular outcomes. Specifically, inflammation leads to an increase in pro-inflammatory cytokines, which further damage vascular integrity and promote the progression of atherosclerosis, heightening the risk of complications like myocardial ischemia and infarction. (2) Immune dysfunction [22, 23]: Malnutrition, as indicated by low GNRI, weakens immune responses by impairing lymphocyte function and cytokine production,

Table 4 Associations between GNRI and mortality in the sensitivity analyses

Analysis	In-hospital mortality		30-day mortality	
	OR (95% CI)	*P*	HR (95% CI)	*P*
Crude analysis	0.41 (0.36, 0.48)	< 0.001	0.45 (0.39, 0.51)	< 0.001
Multivariable analysis	0.51 (0.43, 0.60)	< 0.001	0.56 (0.48, 0.65)	< 0.001
With matching	0.59 (0.50, 0.70)	< 0.001	0.62 (0.53, 0.72)	< 0.001
With inverse probability weighting	0.55 (0.48, 0.64)	< 0.001	0.59 (0.51, 0.67)	< 0.001
Adjusted for propensity score	0.54 (0.46, 0.63)	< 0.001	0.58 (0.50, 0.66)	< 0.001
dropping missing data	0.49 (0.41, 0.59)	< 0.001	0.56 (0.48, 0.65)	< 0.001

Abbreviations: GNRI geriatric nutritional risk index, *OR* odds ratio, *CI* confidence interval, *HR* hazard ratio

Multivariable analysis adjusted for covariates included in demographics, vital signs, types of AMI, comorbidities, laboratory tests and treatments; GNRI≤98 is used as reference

increasing susceptibility to sepsis and ICU-related complications. Malnutrition further weakens the body's ability to mount an effective immune response, leading to higher morbidity and mortality rates. (3) Cardiac metabolism [24]: Nutritional deficits adversely impact myocardial energy metabolism, promoting cardiac cachexia, reducing functional reserve, and worsening AMI outcomes. The metabolic dysfunction linked to nutritional deficiencies impairs the heart's ability to recover and adapt after ischemic events, contributing to heart failure and poor clinical outcomes. (4) Renal function [2, 15]: GNRI's protective effect in patients with preserved renal function suggests a role in preventing renal-medullary hypoxia and tubular dysfunction, which otherwise elevate the risk of acute kidney injury. Malnutrition, particularly in AMI patients with renal preservation, may reduce kidney resilience, thus increasing the likelihood of AKI, a common and serious complication in critically ill patients. The preservation of renal function, supported by adequate nutrition, is crucial in improving survival and recovery in this population.

Strengths and limitations

This study has several strengths that enhance its findings. First, advanced statistical techniques like propensity score matching (PSM) and inverse probability weighting (IPW) helped mitigate confounding factors, improving result reliability. These methods address selection bias and residual confounding, providing a more accurate estimate of the relationship between the Geriatric Nutritional Risk Index (GNRI) and mortality outcomes.

Despite its strengths, this study has limitations. (1) Retrospective Design: The study's retrospective nature may introduce selection bias and residual confounding. Although advanced statistical methods were used to address these, unmeasured confounders could still affect the results. (2) Generalizability: The study's reliance on datasets from specific institutions (eICU-CRD, MIMIC-III, and MIMIC-IV) limits its generalizability to broader populations. Additionally, variability in patient demographics, clinical procedures, and time periods across these datasets may affect the consistency of outcomes. Further research is needed to assess how these factors influence results in diverse populations. (3) Fluid Imbalances, Edema, and Misclassification: GNRI, which uses body weight, may not accurately reflect nutritional status in patients with fluid imbalances or edema, leading to misclassification. These factors can distort nutritional assessments and increase misclassification risk. Future studies should explore other nutritional indices to account for these conditions. (4) Temporal Changes in GNRI: The study did not examine changes in GNRI over time or its interaction with other nutritional indicators. Future research should focus on how dynamic trends in GNRI and other nutritional tools, such as the PNI, affect long-term outcomes.

Conclusions

This study underscores GNRI as an essential tool for predicting short-term outcomes in critically ill AMI patients. Incorporating GNRI into established clinical scoring models significantly improves their predictive capabilities, emphasizing the importance of nutritional assessment in refining risk stratification and informing individualized care strategies. These findings highlight the potential of GNRI to advance critical care practices by prioritizing comprehensive evaluations of nutritional status in high-risk populations.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12872-025-04546-6.

Supplementary Material 1.

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Authors' contributions

S.T. contributed to the conception and design of the study， S.T. and C.W. are responsible for data extraction, data analysis, results visualization, and manuscript writing, Y.S. provided professional advice for the revision of the manuscript, Y.L. were responsible for the review and revision of the manuscript and the funding of the study.All contributors had unrestricted access to the data and collectively assumed responsibility for the accuracy and integrity of the information.

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Data availability

Publicly available datasets were analyzed in this study. The data are available on the MIMIC website at https://mimic.physionet.org/. The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate

The study was conducted in accordance with the guidelines of the Helsinki Declaration. The MIMIC database was supported by grants from the National Institute of Biomedical Imaging and Bioengineering (NIBIB) of the National Institutes of Health (NIH) under award numbers R01-EB001659 (2003–2013) and R01-EB017205 (2014–2022) and approved by the Institutional Review Boards of Beth Israel Deaconess Medical Center (Boston, MA) and the Massachusetts Institute of Technology (Cambridge, MA). As the data are publicly available, the ethical approval and requirement for informed consent were waived for this study.

The database used in this study was approved for research use by the Institutional Review Boards of the Massachusetts Institute of Technology and Beth Israel Deaconess Medical Center. All patient information in the database is

anonymized, and therefore, informed consent is not required. We completed online courses and exams and gained access to the database (record ID: 52219361).

Competing interests
The authors declare no competing interests.

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