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Association between the geriatric nutritional risk index and all-cause mortality in older critically ill patients with community-acquired pneumonia

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Abstract

Aim This study aimed to explore the association between the geriatric nutritional risk index (GNRI) and the risk of mortality in critically ill older patients with community-acquired pneumonia (CAP).

Methods This retrospective study included 1924 critically ill patients with CAP from the Medical Information Mart for Intensive Care-IV3.1 (MIMIC-IV3.1) database. The participants were grouped into four groups based on GNRI levels. The clinical outcome was 30-day, 90-day, 180-day, and 1-year mortality. Cox proportional hazards regression analysis and restricted cubic spline regression were used to evaluate the association between the GNRI and clinical outcomes in critically ill older patients with CAP.

Results A total of 1924 patients (56.9% male) were included in the study. The 30-day, 90-day, 180-day, and 1-year mortality were 37.7%, 47.7%, 54.0% and 59.1%, respectively. Multivariate Cox proportional hazards analysis showed that the GNRI was independently associated with all-cause mortality. After adjusting for confounders, GNRI remained significantly associated with both short- and long-term mortality. Restricted cubic splines revealed a linear association between GNRI and all-cause mortality in CAP patients.

Conclusion Our study indicates that the GNRI has a significant association with all-cause mortality in critically ill older patients with CAP. However, further confirmation of these findings requires larger prospective studies.

Keywords Geriatric nutritional risk index, All-cause mortality, Community-acquired pneumonia, Older patients

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Introduction

As a severe disease with high morbidity and mortality, Community-acquired pneumonia (CAP) is an infectious inflammation of the lung parenchyma that occurs outside the hospital. It also includes cases that arise after hospital admission but originate from infections contracted prior to hospitalization [1]. The overall mortality rate for hospitalized CAP patients is approximately 13%, while severe CAP can progress rapidly, with a fatality rate as high as 50% [2, 3]. In developing countries, the incidence of CAP ranges from 20 to 30%, significantly higher than in high-income nations [4]. CAP poses a substantial burden on global public health [5].

Due to weakened immunity, malnutrition, multiple comorbidities, and increased bacterial resistance, older adults are more susceptible to CAP and often experience more severe disease progression [6]. Studies have shown that the incidence of CAP is significantly higher among individuals aged 65 years and older, with increased mortality observed in those aged 60 years and older with comorbid conditions [7, 8]. The clinical presentation of elderly patients with CAP is often atypical, with fever and cough potentially absent. This atypical presentation can delay diagnosis and treatment, contributing to the higher mortality rate in this population [9]. Malnutrition is a common condition in older adults with CAP and a key predictor of adverse outcomes in this population [10]. Therefore, timely nutritional assessment is essential for improving the prognosis of older adults with CAP.

The GNRI (geriatric nutritional risk index) is an important nutritional screening tool for evaluating various clinical outcomes. It was first proposed by Bouillanne et al. in 2005 as a simple and reliable method to assess nutritional risk in hospitalized elderly patients [11]. Numerous studies have since demonstrated its effectiveness in prognostic assessment for older inpatients, as well as its significant role in managing and evaluating various chronic diseases, including chronic heart failure, chronic kidney disease, and cancer [12–14]. The GNRI is simple, easily obtainable, and affordable, calculated using routinely measured parameters such as height, weight, and serum albumin (ALB) levels [15]. Its simplicity and reliability make it particularly useful for early nutritional screening and risk stratification in vulnerable populations.

Materials and methods

Study population

This retrospective observational study utilized data from the publicly available Medical Information Mart for Intensive Care IV3.1 (MIMIC-IV3.1) database, specifically the records of ICU patients at the Beth Israel Deaconess Medical Center, United States. To comply with relevant regulations, the author, Lei Zhang obtained a Collaborative Institutional Training Initiative (CITI) license (Record ID 64101469) and the necessary permissions to utilize the MIMIC-IV3.1 database. This study adhered to the Declaration of Helsinki, and ethical approval was not required due to the database's standardized data and anonymized patient records.

The cohort consisted of patients admitted to the ICU due to CAP, with the CAP definition based on the ATS/IDSA guidelines [16]. The ICD-9 and ICD-10 codes for CAP included in the study are listed in Table S1 (Additional file 1). The exclusion criteria were: (1) patients aged less than 65 years at the time of first admission; (2) ICU stay of less than 24 h; (3) patients with multiple admissions to the ICU for CAP, for whom only from the first admission data were extracted; (4) missing height, Weight, albumin within 24 h of admission. The flowchart of this study is presented in Fig. 1.

Variable extraction

The software PostgreSQL (version 16.1.0) and Navicat Premium (version 17.1.9) were used to extract data from the MIMIC-IV3.1 database using Structured Query Language (SQL). Patient data from the first 24 h of ICU admission were extracted, including demographics, vital signs, severity at admission (measured by Simplified Acute Physiological Score II (SAPS II), Glasgow Coma Scale (GCS), Acute Physiology Score III (APS III), Oxford Acute Severity of Illness Score (OASIS) and Sequential Organ Failure Assessment (SOFA)), laboratory test results, clinical outcomes, and comorbidities. All laboratory parameters were measured at the first available time point after ICU admission. Follow-up began on the date of admission and ended on the date of death. GNRI at admission was calculated using the following formula: GNRI = 14.89 × serum albumin level (g/dL) + 41.7 × (actual body weight (kg)/ideal body weight (kg)), for males: ideal body weight = height (cm) – 100 – (height (cm) – 150)/4, for females: ideal body weight = height (cm) – 100 – (height (cm) – 150)/2.5 [11].

To avoid possible bias, variables with more than 20% missing values were excluded from the analysis. Variables with missing data less than 20% were processed by multiple imputation using a random forest algorithm (trained by other non-missing variables). This imputation process was conducted using the "mice" package in R (Additional file 2: Table S2).

Clinical outcomes

The outcomes of the present study were mortality within 30 days, 90 days, 180 days, and 1 year. These time points were chosen to capture short-, mid-, and long-term prognosis, which are clinically meaningful intervals in pneumonia and critical illness research. Patient mortality information for discharged patients was accessed from the U.S. Social Security Death Index.

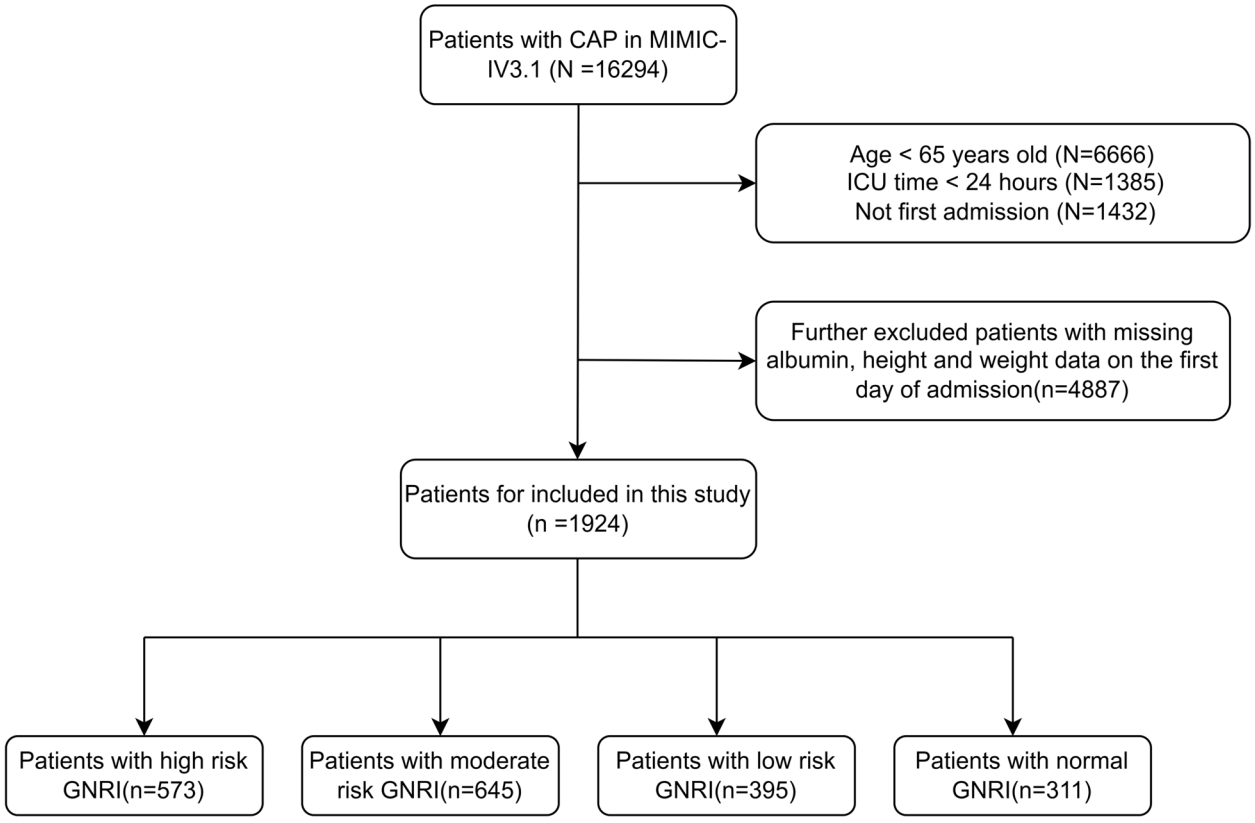

Fig. 1 Inclusion/exclusion criteria. MIMIC: Medical Information Mart for Intensive Care

Statistical analysis

As in previous studies, GNRI was categorized into four groups, Q1 (high risk): GNRI < 82, Q2 (moderate risk): 82 ≤ GNRI < 92, Q3 (low risk): 92 ≤ GNRI ≤ 98, and Q4 (normal): GNRI > 98 [17]. Categorical variables were analyzed using Fisher's exact or chi-square tests and presented as counts (percentages). For continuous variables, the Wilcoxon rank-sum test, Student's t-test, or one-way analysis of variance was employed.

Kaplan–Meier survival analysis was employed to assess the incidence rate of endpoints across GNRI groups, and their differences were assessed through log-rank tests. Cox proportional hazard models were used to calculate the hazard ratio (HR) and 95% confidence interval (CI) between the GNRI and endpoints, and also adjusted for some models. And clinically relevant and prognosis-associated variables were also enrolled in the multivariate model: model 1: unadjusted; model 2: adjusted for Age, Gender, Race; model 3 adjusted for Age, Gender, Race, Temperature, SBP, DBP, MBP, Heart Rate, Respiratory Rate, Heart Failure, Hypertension, APSIII, SAPSII, OASIS, SOFA, Hematocrit, Hemoglobin, WBC, Anion gap, Bicarbonate, Chloride, Glucose, Sodium, Basophil, Alp, Inr, Ptt, Pt, Calcium, Eosinophil, Bun, Lymphocyte, Monocyte, Neutrophil. Further, the linear associations between baseline GNRI and mortality at 30 days, 90 days, 180 days, and 1 year were explored using restricted cubic spline regression models. GNRI was analyzed both as a continuous variable and as an ordinal variable, with the high-risk (Q1) group serving as the reference. Trend analysis across GNRI quartiles was performed.

Subgroup analyses were conducted to evaluate potential differences across various patient subgroups. The stratification variables, including age (65–75 and ≥ 75 years), gender, and comorbidities including diabetes, hypertension, atrial fibrillation, heart failure, myocardial infarction, renal failure, and chronic obstructive pulmonary disease (COPD), were selected because they are well-established factors influencing pneumonia prognosis and overall mortality. These variables may modify the association between GNRI and outcomes, and therefore were included to provide a more comprehensive assessment of GNRI's prognostic value. Interactions between GNRI and stratification variables were examined using likelihood ratio tests.

All statistical analyses were performed using R software (version 4.4.2), with statistical significance set at $P < 0.05$ for two-tailed tests.

Table 1 Baseline clinical characteristics of older patients with CAP

Variable	Overall (*N* = 1924)	Q1 (*N* = 573)	Q2 (*N* = 645)	Q3 (*N* = 395)	Q4 (*N* = 311)	p
Age (years)	75.60(69.93,82.97)	75.36(69.98,82.64)	75.90(70.39,83.56)	75.82(69.59,83.46)	75.37(69.91,81.89)	0.698
Man n(%)	1094(56.9)	314(54.8)	368(57.1)	221(55.9)	191(61.4)	0.288
Race n(%)						0.002
White	1200(62.4)	351(61.3)	424(65.7)	258(65.3)	167(53.7)	
Black	223(11.6)	68(11.9)	66(10.2)	35(8.9)	54(17.4)	
Asian	69(3.6)	23(4.0)	22(3.4)	18(4.6)	6(1.9)	
Hispanic	56(2.9)	14(2.4)	13(2.0)	14(3.5)	15(4.8)	
Others	376(19.5)	117(20.4)	120(18.6)	70(17.7)	69(22.2)	
Height (cm)	168.00(160.00,175.00)	168.00(160.00,175.00)	168.00(160.00,175.00)	168.00(160.00,175.00)	168.00(160.00,175.00)	0.357
Weight (kg)	75.85(63.58,91.40)	70.00(57.73,86.46)	76.03(64.80,91.80)	78.20(66.45,93.40)	80.90(68.70,93.70)	< 0.001
Temperature (°C)	37.39(37.00,37.90)	37.33(36.94,37.83)	37.39(37.00,37.89)	37.39(37.06,38.00)	37.50(37.11,38.00)	0.002
SBP (mmHg)	146.00(133.00,162.25)	143.00(131.00,158.00)	147.00(133.00,160.00)	149.00(134.00,164.50)	152.00(137.00,171.50)	< 0.001
DBP (mmHg)	86.00(75.00,100.00)	83.00(72.00,96.00)	86.00(76.00,99.00)	89.00(76.50,102.50)	89.00(78.00,104.00)	< 0.001
MBP (mmHg)	102.00(91.00,116.00)	99.00(88.00,112.00)	101.00(92.00,115.00)	104.00(92.00,117.00)	105.00(94.00,122.00)	< 0.001
Heart rate	105.00(91.00,121.00)	108.00(93.00,124.00)	105.00(90.00,122.00)	103.00(89.00,118.00)	103.00(92.00,115.00)	0.005
Respiration rate	29.00(25.38,34.00)	29.00(26.00,34.00)	30.00(26.00,35.00)	29.00(25.00,33.00)	29.00(25.00,33.50)	0.029
Spo2(%)	100.00(99.00,100.00)	100.00(100.00,100.00)	100.00(99.00,100.00)	100.00(99.00,100.00)	100.00(99.00,100.00)	0.316
Atrial Fibrillation n(%)	911(47.3)	265(46.2)	317(49.1)	193(48.9)	136(43.7)	0.375
Diabetes n(%)	287(14.9)	72(12.6)	100(15.5)	70(17.7)	45(14.5)	0.16
Heart Failure n(%)	927(48.2)	232(40.5)	334(51.8)	225(57.0)	136(43.7)	< 0.001
Hypertension n(%)	652(33.9)	190(33.2)	205(31.8)	129(32.7)	128(41.2)	0.029
Myocardial Infarction n(%)	286(14.9)	73(12.7)	100(15.5)	63(15.9)	50(16.1)	0.395
Renal Failure n(%)	1168(60.7)	344(60.0)	402(62.3)	243(61.5)	179(57.6)	0.528
COPD n(%)	314(16.3)	89(15.5)	110(17.1)	64(16.2)	51(16.4)	0.914
SAPSII	46.93(14.07)	50.01(14.62)	47.42(13.38)	44.74(14.16)	43.04(12.92)	< 0.001
SOFA	7.00(4.00,9.00)	7.00(5.00,10.00)	7.00(4.00,10.00)	6.00(4.00,9.00)	6.00(3.00,9.00)	< 0.001
GCS	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)	0.464
APSIII	53.50(41.00,69.00)	62.00(47.00,78.00)	53.00(41.00,67.00)	50.00(38.00,63.00)	47.00(35.00,63.00)	< 0.001
OASIS	38.00(32.00,44.00)	39.00(34.00,45.00)	38.00(32.00,44.00)	36.00(31.00,43.00)	36.00(31.00,42.00)	< 0.001
Hematocrit n(%)	34.75(6.91)	32.54(6.36)	33.98(6.64)	36.27(6.42)	38.52(7.06)	< 0.001
Hemoglobin (g/dL)	10.90(9.38,12.70)	10.00(8.70,11.70)	10.60(9.20,12.30)	11.60(10.20,13.00)	12.50(10.85,14.00)	< 0.001
Platelets (K/uL)	216.50(152.00,295.00)	225.00(148.00,344.00)	210.00(142.00,289.00)	215.00(163.50,283.00)	217.00(163.00,270.00)	0.077
WBC (K/uL)	13.70(9.60,18.62)	14.70(9.90,20.30)	13.70(9.50,19.30)	12.70(9.10,17.55)	13.50(9.90,17.40)	0.004
Albumin (g/dL)	3.20(2.70,3.60)	2.50(2.20,2.70)	3.10(2.90,3.30)	3.50(3.45,3.60)	4.00(3.90,4.20)	< 0.001
Anion gap (mEq/L)	17.00(14.00,20.00)	16.00(13.00,19.00)	17.00(14.00,19.00)	17.00(15.00,20.00)	18.00(15.00,21.00)	< 0.001
Bicarbonate (mEq/L)	24.00(21.00,27.00)	24.00(20.00,27.00)	24.00(21.00,28.00)	24.00(22.00,27.50)	25.00(22.00,28.00)	< 0.001
Chloride (mEq/L)	105.00(100.00,110.00)	106.00(101.00,112.00)	105.00(100.00,109.00)	104.00(99.00,108.00)	105.00(100.00,108.00)	< 0.001
Creatinine(mg/dL)	1.40(1.00,2.30)	1.40(0.90,2.40)	1.50(1.00,2.40)	1.40(1.00,2.20)	1.40(1.00,2.05)	0.373
Glucose(mg/dL)	163.00(127.00,225.00)	156.00(119.00,219.00)	163.00(126.00,215.00)	168.00(130.50,239.50)	171.00(136.50,235.00)	0.002
Sodium(mEq/L)	140.00(137.00,143.00)	141.00(137.00,144.00)	140.00(137.00,143.00)	140.00(137.00,143.00)	141.00(138.00,143.00)	0.029
Potassium (mEq/L)	4.60(4.20,5.30)	4.60(4.20,5.20)	4.60(4.20,5.30)	4.60(4.20,5.30)	4.60(4.20,5.50)	0.307
Basophil (%)	0.02(0.01,0.05)	0.02(0.00,0.04)	0.02(0.01,0.05)	0.03(0.01,0.05)	0.03(0.01,0.05)	< 0.001
Total Bilirubin (mg/dL)	0.60(0.40,1.10)	0.60(0.40,1.20)	0.60(0.40,1.10)	0.60(0.40,1.00)	0.60(0.40,1.10)	0.7
Alt (U/L)	26.00(16.00,55.00)	27.00(15.00,59.00)	28.00(16.00,61.00)	25.00(16.00,51.00)	24.00(17.00,41.50)	0.353
Alp (U/L)	92.00(67.00,127.00)	100.00(71.00,144.00)	89.00(65.00,124.00)	87.00(64.50,117.50)	89.00(67.50,118.00)	< 0.001
Ptt	34.60(29.20,49.52)	36.70(30.70,51.50)	34.00(28.90,48.80)	34.50(29.20,51.10)	32.40(27.50,43.15)	< 0.001
Ast (U/L)	41.00(25.00,85.00)	42.00(25.00,88.00)	42.00(25.00,90.00)	39.00(25.00,77.00)	38.00(26.00,77.50)	0.629
Inr	1.40(1.20,1.90)	1.50(1.30,2.00)	1.40(1.20,1.90)	1.30(1.20,1.90)	1.30(1.10,1.70)	< 0.001
Pt	15.10(13.10,20.50)	16.10(13.90,21.50)	15.10(13.00,20.40)	14.60(12.90,20.30)	13.80(12.50,18.45)	< 0.001
Calcium (mg/dL)	8.60(8.10,9.10)	8.20(7.80,8.70)	8.50(8.10,9.00)	8.80(8.40,9.20)	9.10(8.60,9.50)	< 0.001
Bun (mg/dL)	33.00(21.00,53.00)	36.00(23.00,54.00)	34.00(22.00,56.00)	31.00(21.00,52.00)	27.00(19.00,44.00)	< 0.001
Eosinophil (%)	0.02(0.00,0.10)	0.02(0.00,0.09)	0.02(0.00,0.10)	0.02(0.00,0.10)	0.03(0.00,0.11)	0.022

Table 1 (continued)

Variable	Overall (*N* = 1924)	Q1 (*N* = 573)	Q2 (*N* = 645)	Q3 (*N* = 395)	Q4 (*N* = 311)	p
Lymphocyte (K/uL)	0.96(0.59,1.52)	0.87(0.52,1.44)	0.97(0.58,1.51)	0.96(0.64,1.53)	1.10(0.71,1.60)	0.001
Monocyte (K/uL)	0.64(0.38,1.05)	0.60(0.33,0.99)	0.69(0.39,1.08)	0.64(0.42,1.09)	0.64(0.40,1.04)	0.015
Neutrophil (K/uL)	10.15(6.71,14.72)	11.35(7.37,16.38)	10.54(6.64,15.39)	9.56(6.65,13.28)	9.28(6.48,13.47)	< 0.001
Mechanical ventilation n(%)	1491(77.5)	435(75.9)	509(78.9)	308(78.0)	239(76.8)	0.639
LOS Hospital day	12.90(7.69,22.01)	13.80(7.42,22.98)	12.88(7.59,21.38)	11.78(7.70,20.21)	12.81(7.91,22.19)	0.428
LOS ICU day	6.16(3.08,11.72)	5.77(3.05,11.69)	6.40(3.08,11.69)	6.15(3.16,11.69)	6.60(3.29,12.09)	0.494
30-day mortality n(%)	725(37.7)	269(46.9)	244(37.8)	130(32.9)	82(26.4)	< 0.001
90-day mortality n(%)	917(47.7)	340(59.3)	306(47.4)	163(41.3)	108(34.7)	< 0.001
180-day mortality n(%)	1039(54.0)	373(65.1)	348(54.0)	187(47.3)	131(42.1)	< 0.001
365-day mortality n(%)	1138(59.1)	396(69.1)	390(60.5)	205(51.9)	147(47.3)	< 0.001

Abbreviation:SOFA sequential organ failure assessment, *APSIII* acute physiology score III, *SAPSII* simplifed acute physiological score II, *OASIS* oxford acute severity of illness score, *GCS* glasgow coma scale, *WBC* white blood cell, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *MBP* mean blood pressure, *Spo2* pulse oximeter oxygen Saturation, *Alt* alanine aminotransferase, *Alp* alkaline phosphatase, *Ptt* partial thromboplastin time, *Ast* aspartate aminotransferase, *Inr* international normalized ratio, *Pt* prothrombin time, *Bun* blood urea nitrogen

Results

Baseline characteristics

A total of 1924 patients were included in the final data analysis. The median age of the cohort was 75.60 years (IQR: 69.93–82.97) years, and 1094 (56.9%) were male. The 30-day, 90-day, 180-day, and 1-year mortality were 37.7%, 47.7%, 54.0%, and 59.1%, respectively. Patients were categorized into four groups as follows: Q1: high risk (GNRI < 82; $n = 573$), Q2: moderate risk ($82 \le$ GNRI < 92; $n = 645$), Q3: low risk ($92 \le$ GNRI ≤ 98; $n = 395$), and Q4: normal (GNRI > 98; $n = 311$). The baseline characteristics of these patients are shown in Table 1. The Q4 group exhibited higher values for weight, temperature, SBP, DBP, MBP, hematocrit, hemoglobin, albumin, anion gap, glucose, basophil, calcium, and lymphocyte, higher prevalence of hypertension compared to the lower group. Additionally, Q4 had lower values of heart rate, SAPS II, SOFA, APS III, OASIS, ptt, inr, pt, bun, neutrophil, 30-day mortality, 90-day mortality, 180-day mortality, and 1-year mortality. Since malnutrition had a better association with all-cause mortality, we further compared the difference between Q4 and Q1-Q3. The analysis showed that different grouping approaches yielded similar results (Additional File 3, Table S3).

Clinical outcomes

The Kaplan–Meier survival analysis curves were employed to analyze the incidence of clinical outcomes among groups. The results showed that patients in the lowest GNRI group (Q1) had the poorest survival, with cumulative incidence rates of 30-day, 90-day, 180-day, and 1-year mortality consistently decreasing as GNRI increased (log-rank P all < 0.001, Fig. 2).

Multivariate Cox proportional hazards analysis was used to analyze the association between GNRI and 30-day, 90-day, 180-day, and 1-year mortality. The results demonstrated that GNRI was independently associated with a risk of 30-day mortality in model 1 [HR, 0.973(95% CI 0.968, 0.979) $P < 0.001$], model 2 [HR, 0.972(95% CI 0.965, 0.979) $P < 0.001$] and model 3 [HR, 0.973(95% CI 0.965, 0.981) $P < 0.001$] when the GNRI was a continuous variable. When GNRI was treated as a categorical variable (quartiles), patients in the higher GNRI quartiles exhibited a significantly lower risk of 30-day mortality compared to the lowest quartile (Q1), as shown in the three models: model 1 [HR, 0.487(95% CI 0.380,0.623) $P < 0.001$], model 2 [HR, 0.477(95% CI 0.372, 0.612) $P < 0.001$] and model 3 [HR, 0.617(95% CI 0.466, 0.816) P = 0.001]. A similar trend was observed in the multivariate Cox proportional risk analysis of the GNRI and 90-day, 180-day, and 1-year mortality (Table 2). Additionally, the risk of 30-day, 90-day, 180-day, and 1-year mortality demonstrated a consistent downward trend with increasing GNRI quartiles, with all trend *p*-values below 0.05 (Table 2, Fig. 3). Moreover, the results of the restricted cubic splines analysis demonstrated that the risks of 30-day, 90-day, 180-day, and 1-year mortality decreased linearly with increasing GNRI (Fig. 4).

Subgroup analysis

This study evaluated the heterogeneity of the relationship between GNRI and mortality at 30 days, 90 days, 180 days, and 1 year time points through stratified analyses (Figs. 5). The results showed that in all subgroup analyses—stratified by age (65–75 years vs. ≥ 75 years), gender (female vs. male), diabetes (absent vs. present), hypertension (absent vs. present), atrial fibrillation (absent vs. present), heart failure (absent vs. present), myocardial infarction (absent vs. present), COPD (absent vs. present), and renal failure (absent vs. present), GNRI was

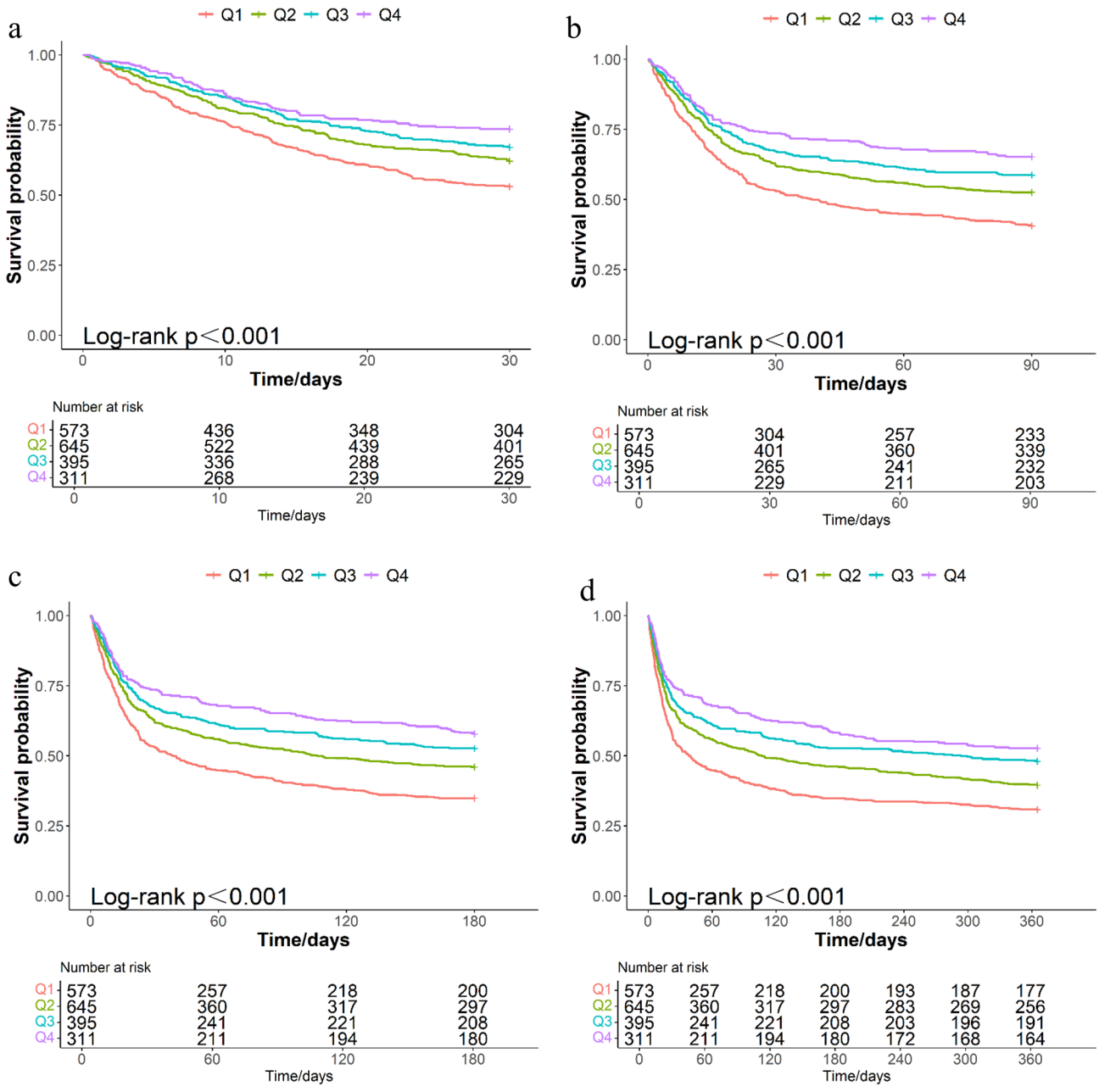

Fig. 2 Kaplan–Meier survival analysis curves for all-cause mortality according to groups at 30 days (**a**), 90 days (**b**), 180 days (**c**), and 1 year (**d**)

significantly associated with mortality at all time points (all subgroup *P* values < 0.05). Furthermore, interaction analysis demonstrated that atrial fibrillation was the only effect modifier that significantly influenced the association between GNRI and mortality at all time points (P for interaction < 0.05). In contrast, although the other stratification factors had significant main effects, their interactions did not reach statistical significance (all P for interaction > 0.05). These findings consistently showed that the association between GNRI values and mortality remained similar across most subpopulations.

Discussion

Nutritional issues in elderly CAP patients in the ICU are often overlooked, despite being a significant clinical challenge [18]. In the present study, we used the open-source MIMIC-IV database to evaluate the predictive capacity of GNRI for short- and long-term outcomes among critically ill older patients with CAP. The results indicated that a lower GNRI was strongly associated with increased all-cause mortality in this cohort. Even after adjustment for the confounding risk factors, the GNRI was still linearly and inversely associated with mortality at 30 days,

Table 2 Multivariable Cox regression analysis to assess the association between GNRI and mortality in patients with CAP

Outcome	Model1		Model2		Model3	
	HR (95% CI)	P	HR (95% CI)	P	HR (95% CI)	P
30-day mortality						
GNRI[a]	0.973(0.968,0.979)	< 0.001	0.972(0.965,0.979)	< 0.001	0.973(0.965,0.981)	< 0.001
Q1(< 82)	1 (Ref)					
Q2(82–92)	0.745(0.626,0.886)	0.001	0.753(0.633,0.896)	0.001	0.863(0.717,1.039)	0.120
Q3(92–98)	0.625(0.507,0.770)	< 0.001	0.628(0.509,0.775)	< 0.001	0.777(0.616,0.981)	0.034
Q4(> 98)	0.487(0.380,0.623)	< 0.001	0.477(0.372,0.612)	< 0.001	0.617(0.466,0.816)	0.001
P for trend		< 0.001		< 0.001		0.001
90-day mortality						
GNRI[a]	0.971(0.965,0.977)	< 0.001	0.971(0.965,0.977)	< 0.001	0.973(0.965,0.980)	< 0.001
Q1(< 82)	1 (Ref)					
Q2(82–92)	0.721(0.618,0.841)	< 0.001	0.727(0.623,0.849)	< 0.001	0.811(0.688,0.956)	0.013
Q3(92–98)	0.597(0.495,0.720)	< 0.001	0.600(0.498,0.724)	< 0.001	0.731(0.594,0.899)	0.003
Q4(> 98)	0.480(0.387,0.597)	< 0.001	0.472(0.380,0.587)	< 0.001	0.620(0.484,0.794)	< 0.001
P for trend		< 0.001		< 0.001		< 0.001
180-day mortality						
GNRI[a]	0.973(0.967,0.979)	< 0.001	0.973(0.967,0.979)	< 0.001	0.975(0.968,0.982)	< 0.001
Q1(< 82)	1 (Ref)					
Q2(82–92)	0.734(0.634,0.850)	< 0.001	0.738(0.638,0.855)	< 0.001	0.811(0.694,0.947)	0.008
Q3(92–98)	0.607(0.509,0.724)	< 0.001	0.609(0.511,0.727)	< 0.001	0.737(0.606,0.895)	0.002
Q4(> 98)	0.512(0.420,0.625)	< 0.001	0.505(0.413,0.617)	< 0.001	0.659(0.524,0.828)	< 0.001
P for trend		< 0.001		< 0.001		< 0.001
1-year mortality						
GNRI[a]	0.973(0.968,0.979)	< 0.001	0.973(0.968,0.979)		0.976(0.969,0.982)	
Q1(< 82)	1 (Ref)					
Q2(82–92)	0.766(0.666,0.881)	< 0.001	0.769(0.669,0.885)	< 0.001	0.835(0.720,0.968)	0.017
Q3(92–98)	0.614(0.518,0.727)	< 0.001	0.616(0.520,0.730)	< 0.001	0.737(0.611,0.889)	0.001
Q4(> 98)	0.529(0.437,0.639)	< 0.001	0.519(0.429,0.628)	< 0.001	0.672(0.541,0.836)	< 0.001
P for trend		< 0.001		< 0.001		< 0.001

Model 1: Unadjusted

Model 2: Adjusted Age, Gender, and Race

Model 3: Adjusted Age, Gender, Race, Temperature, SBP, DBP, MBP, Heart Rate, Respiratory Rate, Heart Failure, Hypertension, APSIII, SAPSII, OASIS, SOFA, Hematocrit, Hemoglobin, WBC, Anion gap, Bicarbonate, Chloride, Glucose, Sodium, Basophil, Alp, Inr, Ptt, Pt, Calcium, Eosinophil, Bun, Lymphocyte, Monocyte, Neutrophil. GNRI[a] was entered as a continuous variable per 1 unit

90 days, 180 days, and 1 year. Our results extended the application of the GNRI to the realm of critical illness, indicating its potential value as a decision-making tool for clinicians managing patients with CAP. These results highlighted the critical role of nutritional status and systemic inflammation in shaping clinical outcomes in critically ill elderly patients, reinforcing and expanding upon previous research in this area.

CAP poses a significant health threat to the elderly, with severe CAP drawing particular attention due to its high mortality and complication risks [19]. The vulnerability of elderly patients is largely attributed to immune dysfunction and malnutrition, which not only exacerbate disease progression but also increase the risk of multiple organ failure [20]. Given these challenges, early identification of high-risk nutritional status in elderly patients with CAP is crucial for mitigating disease severity, reducing medical burden, and improving prognosis [21].

Malnutrition and inflammation are well-established drivers of adverse outcomes in chronic and acute illnesses [22, 23]. The GNRI, integrating serum albumin, body weight, and height, serves as a composite marker of overall nutritional status and may also reflect the inflammatory burden. Previous studies have shown that GNRI effectively predicts all-cause mortality, especially in hospitalized elderly populations [11, 24]. Our findings align with previous research, demonstrating that higher GNRI scores are associated with improved survival rates.

The robust association between GNRI and CAP outcomes can be attributed to malnutrition's dual impact on immune dysfunction and tissue repair. A previous study indicated that malnutrition was characterized by

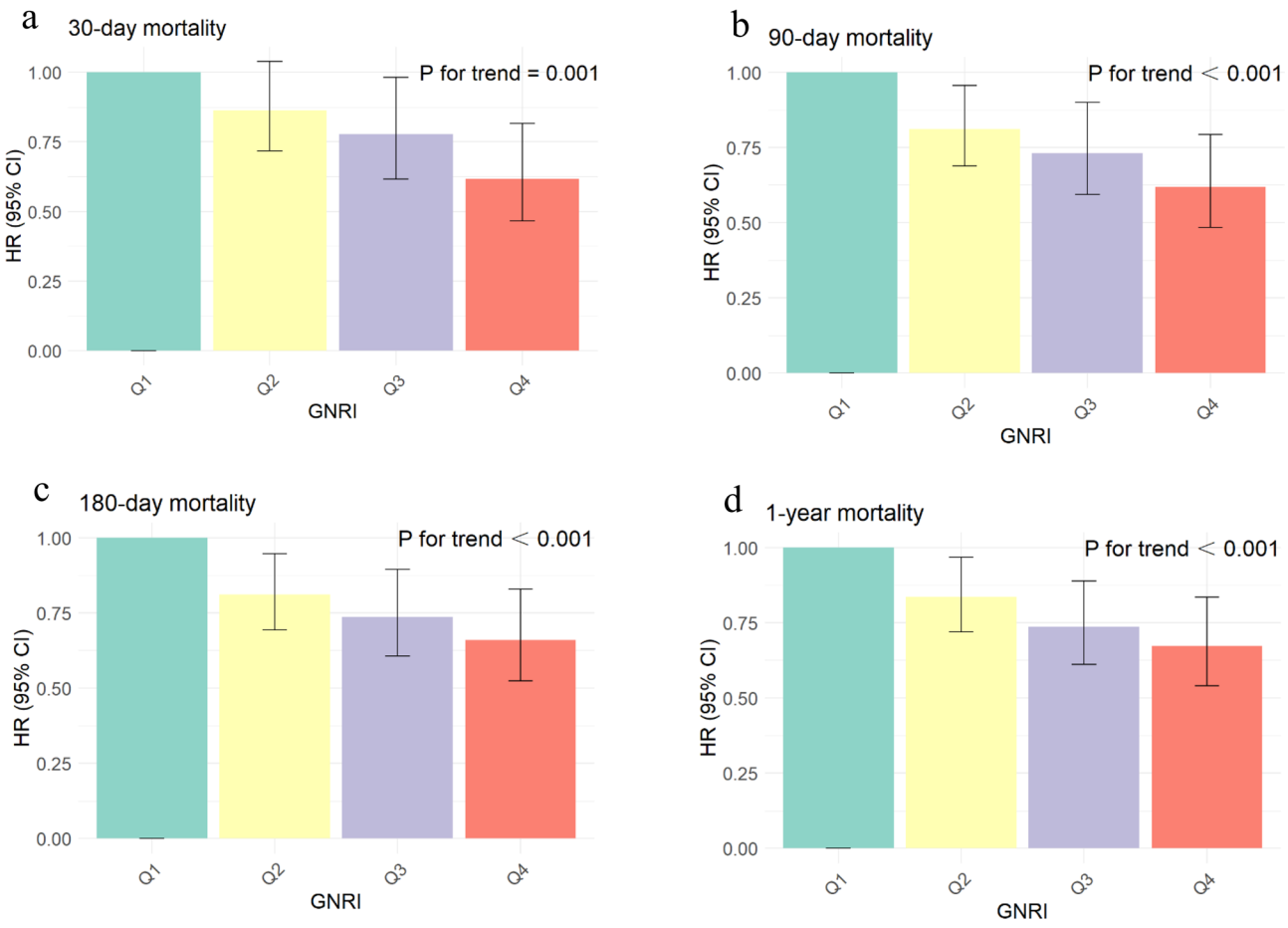

Fig. 3 The relationship for the levels of GNRI with 30-day, 90-day, 180-day, and 1-year mortality. **a**–**d** Hazard ratios (95% CIs) for 30-day, 90-day, 180-day, and 1-year mortality according to GNRI groups after adjusting for Age, Gender, Race, Temperature, SBP, DBP, MBP, Heart Rate, Respiratory Rate, Heart Failure, Hypertension, APSIII, SAPSII, OASIS, SOFA, Hematocrit, Hemoglobin, WBC, Anion gap, Bicarbonate, Chloride, Glucose, Sodium, Basophil, Alp, Inr, Ptt, Pt, Calcium, Eosinophil, Bun, Lymphocyte, Monocyte, Neutrophil. Error bars indicate 95% CIs. The first group is the reference

hypoalbuminemia and low body weight [25]. Hypoalbuminemia, a key GNRI component, is linked to impaired phagocytic activity and reduced antibody production, exacerbating infection severity [26]. Concurrently, reduced muscle mass (as reflected in low body weight) increases the risk of respiratory failure and prolonged immobilization [27]. These mechanisms align with studies emphasizing the prognostic role of low body weight and hypoalbuminemia in CAP [28, 29]. Furthermore, our findings extend the utility of GNRI beyond traditional scoring systems (e.g., SOFA, APS III), suggesting that GNRI offers complementary prognostic value in CAP management.

The consistency of GNRI's predictive power across most subgroups—including age, gender, and comorbidities—underscores its broad clinical applicability in elderly ICU patients. However, the significant interaction with atrial fibrillation merits further investigation. Atrial fibrillation patients exhibited a more pronounced mortality gradient across GNRI, possibly due to the synergistic effects of malnutrition and cardiac dysfunction. Malnutrition exacerbates electrolyte imbalances and endothelial dysfunction, potentiating arrhythmogenicity [30]. Additionally, atrial fibrillation-associated systemic inflammation may accelerate catabolism, amplifying nutritional risk [31]. The interplay between malnutrition and atrial fibrillation-related inflammation underscores the need for integrated cardiometabolic and nutritional management in atrial fibrillation patients with CAP.

Clinically, our findings support routine GNRI assessment in elderly CAP patients upon ICU admission. Early identification of high-risk individuals (GNRI < 82) could prompt targeted interventions, such as protein-calorie supplementation or immunonutrition, which have demonstrated mortality benefits in malnourished critically ill patients [32]. Additionally, the simplicity of GNRI allows

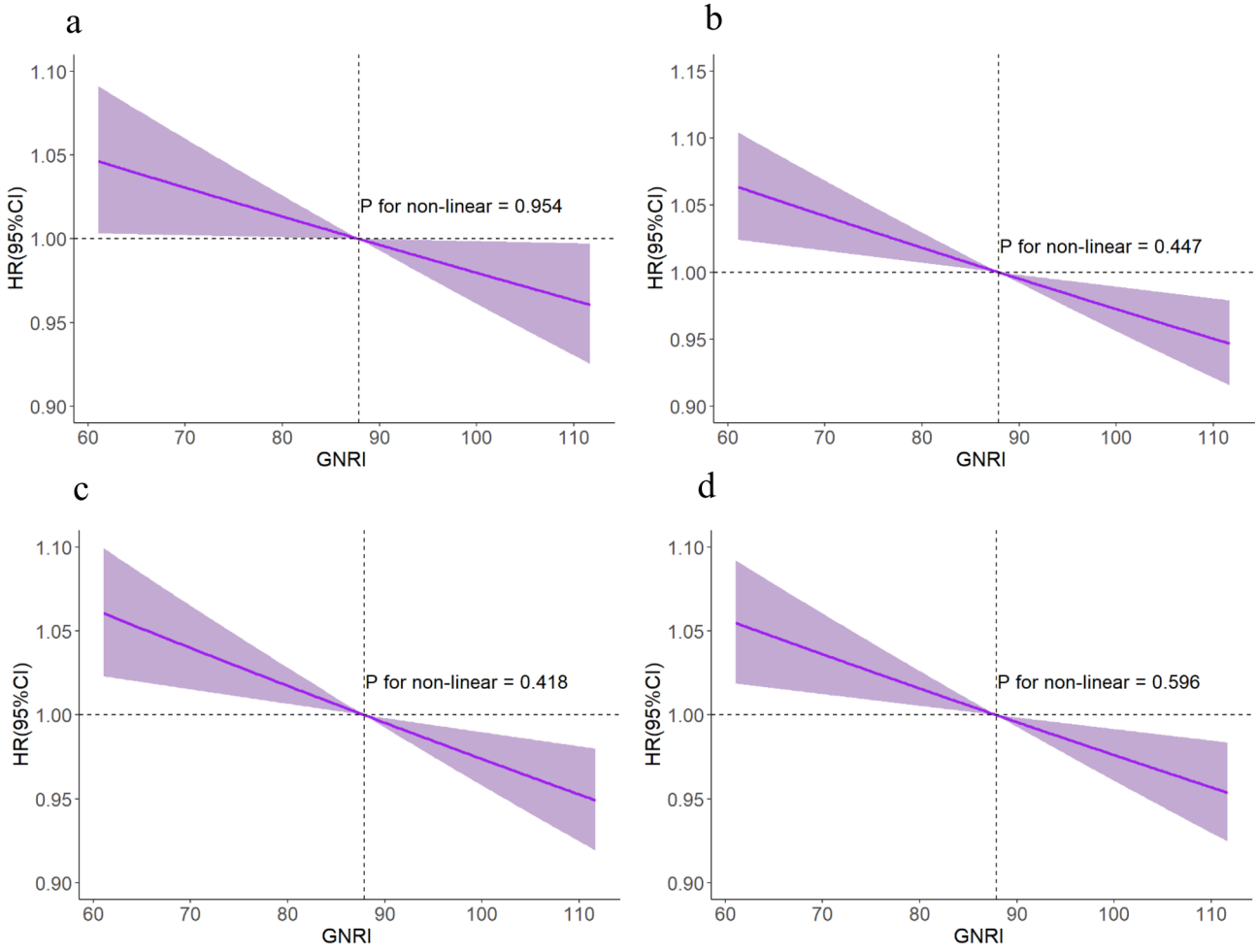

Fig. 4 Underlying the linear relationship between the GNRI and short- and long-term all-cause mortality. **a** Restricted cubic spline for 30-day mortality. **b** Restricted cubic spline for 90-day mortality. **c** Restricted cubic spline for 180-day mortality. **d** Restricted cubic spline for 1-year mortality

for rapid and efficient risk stratification, complementing existing tools like the CURB-65 score [33].

Several limitations of this study should be acknowledged. First, as an observational study, causal relationships between GNRI and CAP prognosis cannot be definitively established. Second, we relied on baseline GNRI measurements without accounting for dynamic changes during follow-up, which may have affected the accuracy of risk assessment. Third, GNRI includes serum albumin, which can decrease in the presence of systemic inflammation independent of actual nutritional status, potentially confounding the assessment of nutritional risk.

Despite these Limitations, this study has several notable strengths. It is based on the large, publicly available MIMIC-IV 3.1 database, which contains real-world clinical data from routine patient care, enhancing the generalizability of our findings. We accounted for a wide range of potential confounders through multivariable-adjusted Cox regression, stratified analyses, and interaction testing. In addition, GNRI is a simple yet comprehensive index that can be easily calculated using routinely measured parameters, making it a practical tool for nutritional risk assessment in critically ill elderly patients.

Future high-quality prospective studies are warranted to further validate the prognostic value of GNRI in CAP management. In addition, combining GNRI for early screening upon ICU admission with more comprehensive tools such as the Mini Nutritional Assessment (MNA) or MNA-Short Form (MNA-SF) once the patient's condition stabilizes could help balance timeliness and accuracy in nutritional evaluation. Longitudinal assessments of nutritional status may further enhance risk stratification and guide targeted interventions.

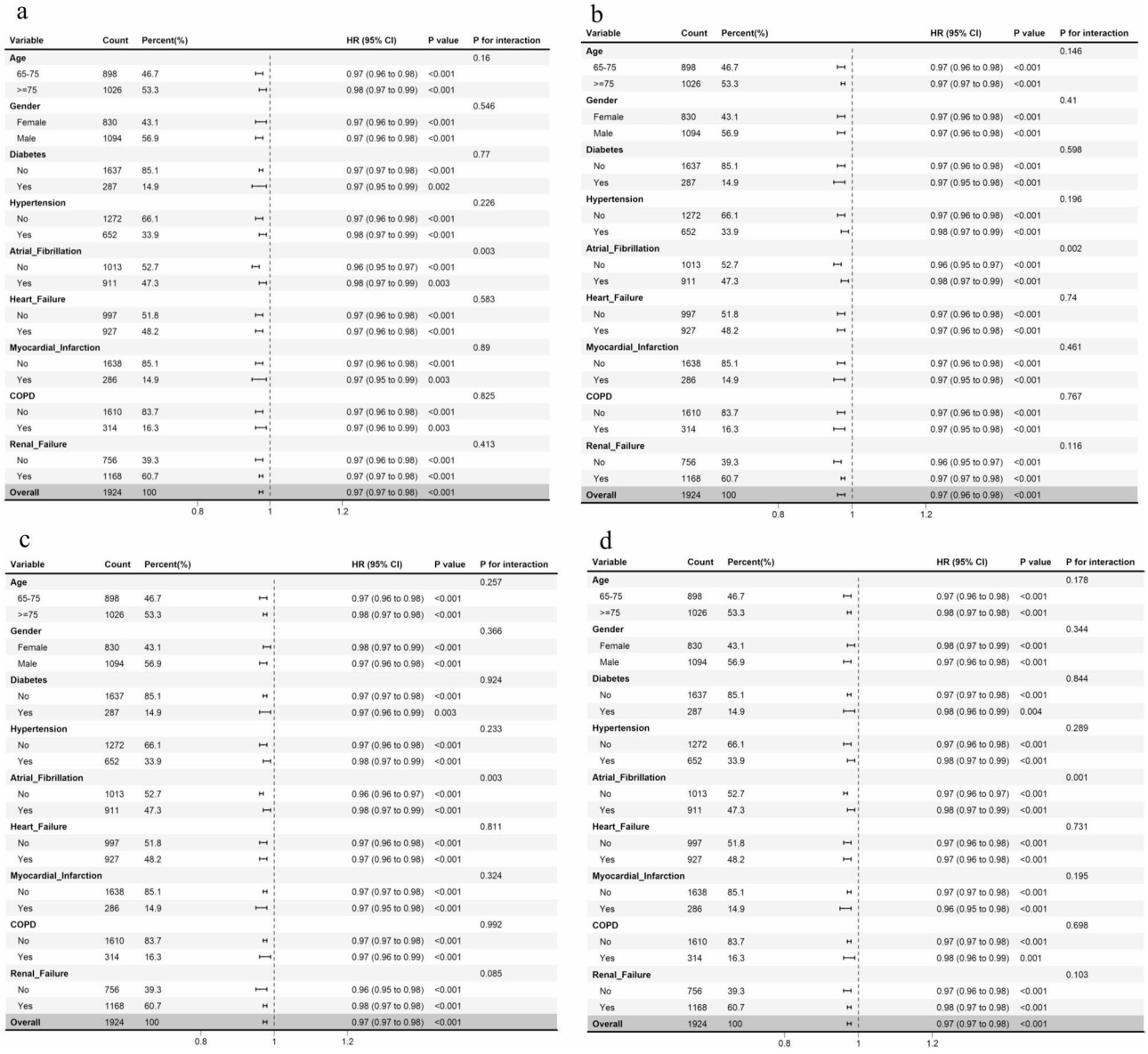

Fig. 5 Subgroup analyses of the association between GNRI and mortality at 30 days (**a**), 90 days (**b**), 180 days (**c**), and 1 year (**d**)

Conclusions

In conclusion, our results extended the utility of the GNRI to critically ill older patients with CAP, demonstrating its reliability and validity as a prognostic tool for assessing nutritional risk and predicting both short- and long-term mortality in this population. These results highlight the potential of GNRI in enhancing risk assessment and guiding targeted interventions. However, further high-quality studies are required to validate these findings and explore their broader clinical implications.

Supplementary Information

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Supplementary Material 1.

Supplementary Material 2.

Supplementary Material 3.

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Authors' contributions
Lei Zhang wrote the manuscript. Lei Zhang extracted, collected and analyzed data. Minye Li prepared tables and figures. Jianfei Liu, Hui Ma reviewed the results, interpreted data, and designed the study. All authors have made an intellectual contribution to the manuscript and approved the submission.

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Declarations

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Not applicable.

Consent for publication
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