BRITISH JOURNAL OF HOSPITAL MEDICINE

ARTICLE

A Correlation of Geriatric Nutritional Risk Index With the Progression of Sarcopenia in Patients With Lung Cancer

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Abstract

Aims/Background Malnutrition and sarcopenia frequently occur in individuals with lung cancer and are strongly associated with adverse clinical outcomes. The Geriatric Nutritional Risk Index (GNRI), which integrates serum albumin with body weight, offers a straightforward assessment of nutritional condition. Hence, this retrospective study aimed to investigate the association between GNRI scores and sarcopenia in a cohort of lung cancer patients.

Methods This retrospective study collected clinical data from lung cancer patients (n = 102) admitted between January 2023 and December 2024. Logistic regression analyses, both univariate and multivariate, were utilized to assess the association between GNRI and sarcopenia. Furthermore, the diagnostic utility of GNRI was evaluated using receiver operating characteristic (ROC) curve analysis.

Results Of the total recruited lung cancer patients (n = 102), 41 (40.2%) were diagnosed with sarcopenia. Multivariate logistic regression analysis identified GNRI as an independent predictor of sarcopenia (odds ratio [OR] = 0.912, 95% confidence interval [CI]: 0.865–0.961, $p < 0.001$). The ROC analysis yielded an area under the curve (AUC) of 0.805 (95% CI: 0.718–0.892), indicating that GNRI has excellent diagnostic performance for detecting sarcopenia.

Conclusion GNRI serves as an independent predictor of sarcopenia among lung cancer patients and demonstrates favorable clinical utility. As a practical and cost-effective screening measure, GNRI can facilitate early identification of patients at nutritional risk, informing timely intervention.

Key words: Geriatric Nutritional Risk Index; sarcopenia; lung neoplasms; skeletal muscle; nutrition assessment

Submitted: 31 March 2025 Revised: 6 August 2025 Accepted: 8 August 2025

Introduction

Lung cancer remains the leading cause of cancer-related deaths globally, posing a significant public health concern (Cao et al, 2021; Leiter et al, 2023). Sarcopenia is highly prevalent among cancer patients, with clinical evidence demonstrating a substantially higher incidence in lung cancer than in most other malignancies (Bossi et al, 2021; Yang et al, 2019). This condition is characterized by the progressive decline in skeletal muscle mass, muscle strength, and physical performance. It is associated with various adverse health outcomes, such as falls, disability, hospitalization, long-term care dependency, poor quality of life, and increased mortality.

How to cite this article:
Zhao X, Wu J, Jia L, Fang Q. A Correlation of Geriatric Nutritional Risk Index With the Progression of Sarcopenia in Patients With Lung Cancer. Br J Hosp Med. 2025. https://doi.org/10.12968/hmed.2025.0286

In advanced cancer, the severe loss of muscle mass is one of the most common and serious clinical manifestations, closely correlated with poor prognosis (Cruz-Jentoft et al, 2019; Cruz-Jentoft and Sayer, 2019). Therefore, early prediction and prompt intervention for sarcopenia are crucial for identifying high-risk patients and improving clinical outcomes.

Nutritional status is a key determinant in the onset and progression of sarcopenia (Sieber, 2019; Yuan and Larsson, 2023). The Geriatric Nutritional Risk Index (GNRI), which is calculated based on serum albumin levels and body weight, has been widely applied in assessing nutritional risk in hospitalized elderly patients (Sieber, 2019; Yuan and Larsson, 2023). Compared with other nutritional assessment tools, the GNRI is simple to calculate and has shown strong prognostic value across various disease settings (Shiroma et al, 2023; Dong et al, 2021; Chen et al, 2024). Furthermore, recent evidence shows that GNRI is strongly associated with prognosis in various cancer types (Gu et al, 2015; Bo et al, 2016; Tsukagoshi et al, 2024; Zhang et al, 2023).

Despite these findings, studies specifically examining the association between GNRI and sarcopenia in lung cancer patients remain scarce. Given the pivotal role of nutrition in the pathophysiology of sarcopenia, we hypothesized that the GNRI might be a useful indicator for assessing sarcopenia risk in this population. Therefore, the present study aimed to explore the relationship between GNRI and sarcopenia among patients with lung cancer, evaluate its predictive capacity, and provide evidence to support clinical nutrition assessment and guide early intervention approaches.

Methods

Study Population

This study enrolled 150 patients with pathologically confirmed lung cancer who were admitted to Yiwu Central Hospital, China, between January 2023 and December 2024. Eligibility criteria included patients aged $\geq$18 years with pathologically confirmed lung cancer who provided written informed consent. Patients were excluded if they had other concomitant malignancies, severe comorbidities potentially affecting muscle status (e.g., hepatic or renal failure, hematologic diseases), incomplete clinical data, or had received medications affecting muscle metabolism (e.g., corticosteroids) within the preceding three months. Following screening, 48 patients were excluded according to these criteria, and the remaining 102 patients were stratified into sarcopenia (n = 41) and non-sarcopenia (n = 61) groups based on established diagnostic standards. The patient selection process is summarized in Fig. 1.

Data Collection

Demographic and clinical data of all study subjects were obtained from the hospital's electronic health record system and anonymized to ensure patient privacy. The collected variables included age, sex, body mass index (BMI), smoking history, alcohol consumption, comorbidities (hypertension, diabetes mellitus, coro-

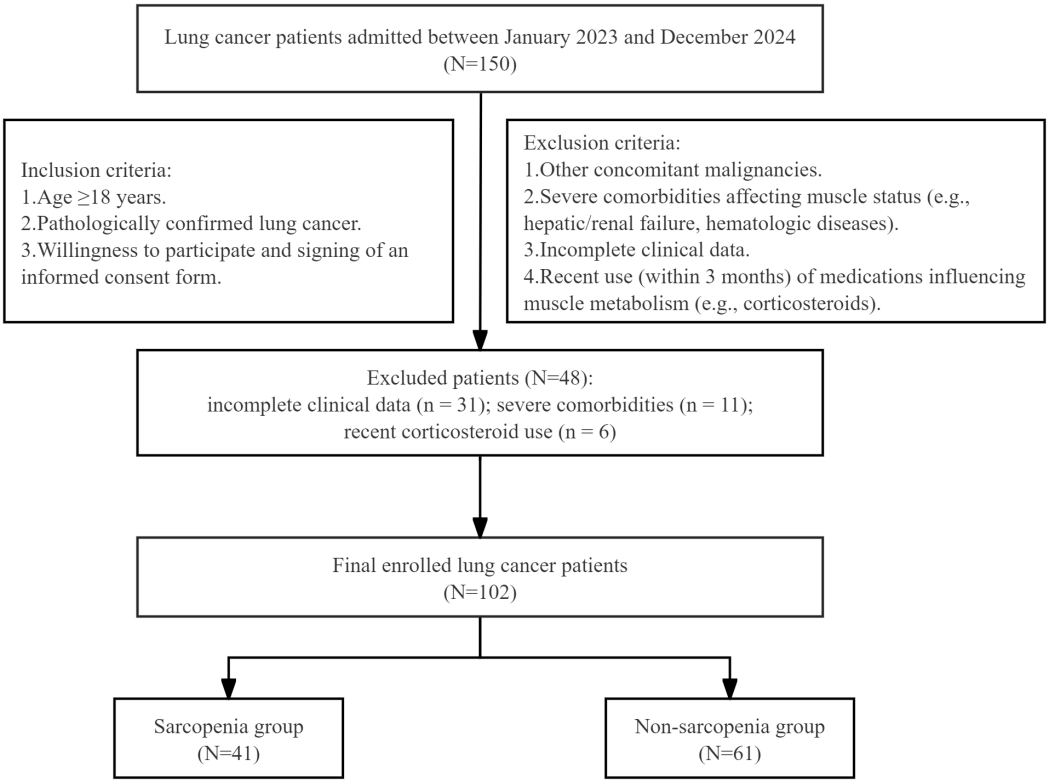

Fig. 1. A flowchart of the patient selection process and grouping.

nary heart disease), pathological type of lung cancer, and tumour-node-metastasis (TNM) stage according to the 8th edition of the American Joint Committee on Cancer (AJCC) staging manual (Maas et al, 2005).

Fasting venous blood samples, collected on the morning after admission, were assessed for laboratory indicators such as serum albumin, hemoglobin, white blood cell (WBC) count, neutrophil count, lymphocyte count, monocyte count, platelet count, and C-reactive protein (CRP). Furthermore, routine metabolic and cardiovascular parameters, including systolic blood pressure (SBP), diastolic blood pressure (DBP), fasting blood glucose (FBG), triglycerides (TG), high-density lipoprotein cholesterol (HDLC), total cholesterol (TC), and low-density lipoprotein cholesterol (LDLC), were documented to provide a comprehensive assessment of the patients' baseline metabolic and nutritional status. All values were obtained from standard laboratory and vital sign assessments.

GNRI Calculation

GNRI was determined using a previously established (Bouillanne et al, 2005). The formula is given as follows:

$$\text{GNRI} = (1.489 \times \text{serum albumin [g/L]}) + 41.7 \times \left(\frac{\text{actual weight}}{\text{ideal weight}}\right)$$

$$\text{Ideal weight} = 22 \times \text{height (m)}^2$$

If the actual body weight exceeded the ideal body weight, the weight ratio was given a value of 1.

Assessment of Sarcopenia

Based on the diagnostic criteria established by the European and Asian Working Groups on Sarcopenia (Chen et al, 2020), sarcopenia refers to a syndrome characterized by a progressive and widespread decline in skeletal muscle mass and muscular strength. Muscle mass was evaluated utilizing computed tomography (CT) at the level of the third lumbar vertebra (L3) and adjusted for height to calculate the skeletal muscle index (SMI, cm^2/m^2). Declined muscle mass was determined at an SMI of <38.5 cm^2/m^2 in females and <52.4 cm^2/m^2 in males. Muscle strength was assessed by measuring grip strength of the dominant hand using an electronic dynamometer, with low strength defined as less than 26 kg for men and less than 18 kg for women.

Treatment and Intervention

All patients received standard treatment regimens including chemotherapy, radiotherapy, and targeted therapy. The outcomes of the treatments were recorded, with a focus on the changes in GNRI scores after treatment and their relationship with sarcopenia.

Sample Size Calculation

The sample size was determined using G*Power 3.1.9.7 (Heinrich-Heine-Universität Düsseldorf, Düsseldorf, North Rhine-Westphalia, Germany) for a logistic regression model assessing the association between GNRI and sarcopenia. An a priori power calculation was performed for a two-tailed test with a significance level (α) of 0.05 and a power (1-β) of 0.80. Assuming an estimated sarcopenia prevalence of approximately 40% among lung cancer patients and anticipating the inclusion of 3–5 independent predictors in the multivariate model, the minimum required sample size was calculated to be 92 patients. The final cohort of 102 patients exceeded this requirement, thereby ensuring sufficient statistical power for the primary analyses. Due to the retrospective study design and limited patient availability, no prospective sample size calculation was conducted.

Statistical Analysis

Statistical analyses were performed using SPSS (version 27.0, IBM Corp., Armonk, NY, USA) and R (version 4.4.2, R Foundation for Statistical Computing, Vienna, Austria). The distribution of continuous variables was assessed using the Shapiro-Wilk test. Normally distributed continuous variables were expressed as mean $\pm$ standard deviation and compared using independent-sample *t*-tests. However, non-normally distributed variables were presented as medians with interquartile ranges and analyzed using the Mann-Whitney U test. Moreover, categorical variables were compared using the chi-square test. Variables with $p < 0.05$ in univariate logistic regression were included in multivariate regression analysis. To minimize collinearity, GNRI components such as albumin and BMI were excluded from the model, and multicollinearity was evaluated using variance inflation factor

Table 1. Comparison of baseline characteristics between the two study groups.

Variable	Total (n = 102)	Non-sarcopenia (n = 61)	Sarcopenia (n = 41)	Statistic	*p*-value
Sex, n (%)				$\chi^2 = 1.55$	0.213
Male	70 (68.63)	39 (63.93)	31 (75.61)		
Female	32 (31.37)	22 (36.07)	10 (24.39)		
Age, years	66.12 ± 10.08	64.92 ± 10.00	67.90 ± 10.05	$t = -1.47$	0.143
BMI, kg/m^2	22.06 ± 3.84	23.20 ± 3.73	20.36 ± 3.37	$t = 3.92$	<0.001
Hypertension, n (%)				$\chi^2 = 0.01$	0.909
No	64 (62.75)	38 (62.30)	26 (63.41)		
Yes	38 (37.25)	23 (37.70)	15 (36.59)		
Diabetes mellitus, n (%)				$\chi^2 = 0.00$	0.987
No	87 (85.29)	52 (85.25)	35 (85.37)		
Yes	15 (14.71)	9 (14.75)	6 (14.63)		
Coronary heart disease, n (%)				$\chi^2 = 0.03$	0.867
No	78 (76.47)	47 (77.05)	31 (75.61)		
Yes	24 (23.53)	14 (22.95)	10 (24.39)		
Pathological type, n (%)				$\chi^2 = 0.92$	0.822
Adenocarcinoma	38 (37.25)	21 (34.43)	17 (41.46)		
Squamous cell carcinoma	28 (27.45)	18 (29.51)	10 (24.39)		
Small cell carcinoma	15 (14.71)	10 (16.39)	5 (12.20)		
Other	21 (20.59)	12 (19.67)	9 (21.95)		
TNM stage, n (%)				$\chi^2 = 0.21$	0.647
I–II	42 (41.28)	24 (39.34)	18 (43.90)		
III–IV	60 (58.82)	37 (60.66)	23 (56.10)		
Smoke, n (%)				$\chi^2 = 0.13$	0.714
Yes	65 (63.73)	38 (62.30)	27 (65.85)		
No	37 (36.27)	23 (37.70)	14 (34.15)		
Drink, n (%)				$\chi^2 = 0.43$	0.513
Yes	84 (82.35)	49 (80.33)	35 (85.37)		
No	18 (17.65)	12 (19.67)	6 (14.63)		
GNRI, points	98.91 (87.95, 109.01)	105.47 (96.91, 111.06)	89.22 (80.05, 98.65)	$Z = -5.20$	<0.001
SBP, mmHg	125.71 ± 16.21	127.20 ± 17.47	123.49 ± 14.06	$t = 1.13$	0.259
DBP, mmHg	77.74 ± 10.85	79.02 ± 11.15	75.83 ± 10.21	$t = 1.46$	0.147
FBG, mmol/L	5.14 (4.56, 6.14)	5.12 (4.69, 6.15)	5.22 (4.34, 6.08)	$Z = -0.33$	0.738
TG, mmol/L	1.31 (0.88, 1.73)	1.29 (0.94, 1.65)	1.36 (0.85, 1.81)	$Z = 0.00$	1.000
TC, mmol/L	4.57 (3.58, 5.33)	4.62 (3.73, 5.41)	4.27 (3.15, 5.03)	$Z = -1.42$	0.155
HDLC, mmol/L	1.02 (0.82, 1.25)	1.12 (0.93, 1.25)	0.90 (0.68, 1.21)	$Z = -2.47$	0.014
LDLC, mmol/L	2.66 (1.94, 3.55)	2.75 (1.98, 3.60)	2.55 (1.92, 3.26)	$Z = -0.75$	0.456
WBC count, 10^9/L	6.41 (5.00, 9.70)	5.95 (4.97, 8.00)	7.92 (5.75, 11.85)	$Z = -2.43$	0.015
Neutrophil, 10^9/L	4.66 (3.09, 7.46)	3.91 (3.04, 6.08)	6.04 (3.79, 9.86)	$Z = -2.73$	0.006
Lymphocyte, 10^9/L	1.22 (0.84, 1.56)	1.31 (0.93, 1.61)	1.00 (0.64, 1.43)	$Z = -2.05$	0.041
Platelet count, 10^9/L	188.50 (158.25, 264.50)	201.00 (163.00, 265.00)	184.00 (154.00, 263.00)	$Z = -0.85$	0.397

Table 1. Continued.

Variable	Total (n = 102)	Non-sarcopenia (n = 61)	Sarcopenia (n = 41)	Statistic	*p*-value
Monocyte count, 10^9/L	0.45 (0.31, 0.64)	0.40 (0.30, 0.61)	0.52 (0.36, 0.76)	Z = –2.10	0.036
CRP, mg/L	7.55 (0.92, 40.25)	2.87 (0.57, 32.20)	20.74 (2.30, 55.72)	Z = –2.31	0.021
Albumin, g/L	38.35 (33.20, 43.00)	41.80 (36.80, 43.80)	34.20 (29.80, 38.80)	Z = –4.57	<0.001
Hemoglobin, g/L	120.91 ± 21.47	126.46 ± 19.98	112.66 ± 21.17	*t* = 3.34	0.001

t: *t*-test, Z: Mann-Whitney U test, χ^2: Chi-square test. BMI, body mass index; CRP, C-reactive protein; DBP, diastolic blood pressure; FBG, fasting blood glucose; GNRI, Geriatric Nutritional Risk Index; HDLC, high-density lipoprotein cholesterol; LDLC, low-density lipoprotein cholesterol; TC, total cholesterol; TG, triglycerides; WBC, white blood cell; SBP, systolic blood pressure; TNM, tumour-node-metastasis.

(VIF) values, with VIF <10 considered acceptable. Receiver operating characteristic (ROC) curves were generated to examine the diagnostic performance of GNRI, with sensitivity, specificity, and predictive values calculated employing the Youden index. Statistical significance was defined as a two-sided p-value < 0.05.

Results

Comparison of Baseline Characteristics Between the Two Study Groups

This study included 102 lung cancer patients, with a mean age of 66.12 ± 10.08 years, comprising 70 males and 32 females. However, sarcopenia was diagnosed in 41 (40.2%) patients. Baseline characteristics of the study participants are summarized in Table 1. There were no statistically significant differences between the sarcopenia and non-sarcopenia groups in terms of age, sex, smoking history, alcohol consumption, or the prevalence of comorbidities, such as hypertension, diabetes mellitus, and coronary heart disease (all $p > 0.05$). Similarly, the distribution of pathological types and TNM stages was comparable between the two groups, with approximately 59% of the overall cohort presenting with advanced-stage (III–IV) disease.

Furthermore, compared to patients without sarcopenia, those with sarcopenia had significantly lower BMI, serum albumin levels, and hemoglobin levels (all $p < 0.05$). The sarcopenia group also exhibited an inflammatory profile with considerably higher levels of WBC, neutrophils, monocytes, and CRP, alongside lower levels of lymphocytes and HDLC (all $p < 0.05$). Consequently, the GNRI was substantially lower in the sarcopenia group compared to the non-sarcopenia group ($p < 0.001$). Additionally, no statistically significant differences were observed in blood pressure, FBG, TG, TC, LDLC, or platelet count between the two groups ($p > 0.05$).

Table 2. Univariate analysis of factors affecting the presence of sarcopenia.

Variables	β	SE	Wald	*p*-value	OR (95% CI)
Sex					
Male					Reference
Female	0.559	0.361	2.391	0.122	1.749 (0.861–3.551)
BMI	–0.224	0.065	12.029	0.001	0.799 (0.704–0.907)
Hypertension, n (%)					
No					Reference
Yes	0.048	0.418	0.013	0.909	1.049 (0.462–2.381)
Diabetes mellitus, n (%)					
No					Reference
Yes	0.010	0.571	0.000	0.987	1.010 (0.330–3.089)
Coronary heart disease, n (%)					
No					Reference
Yes	0.080	0.474	0.028	0.867	1.083 (0.427–2.744)
Pathological type, n (%)					
Adenocarcinoma					Reference
Squamous cell carcinoma	0.076	0.549	0.019	0.889	1.079 (0.368–3.163)
Small cell carcinoma	–0.300	0.592	0.257	0.612	0.741 (0.232–2.362)
Other	–0.182	0.721	0.064	0.800	0.833 (0.203–3.422)
TNM stage, n (%)					
I–II					Reference
III–IV	0.188	0.410	0.210	0.647	1.207 (0.541–2.692)
Smoke					
No					Reference
Yes	0.155	0.422	0.134	0.714	1.167 (0.510–2.670)
Drink					
No					Reference
Yes	0.357	0.547	0.426	0.514	1.429 (0.489–4.172)
Age	0.031	0.021	2.131	0.144	1.031 (0.990–1.074)
GNRI	–0.103	0.023	22.192	<0.001	0.902 (0.864–0.942)
SBP	–0.014	0.013	1.281	0.258	0.986 (0.961–1.011)
DBP	–0.028	0.020	2.096	0.148	0.972 (0.936–1.010)
FBG	–0.074	0.113	0.425	0.514	0.929 (0.740–1.160)
TG	–0.109	0.202	0.291	0.590	0.897 (0.604–1.332)
TC	–0.155	0.135	1.323	0.250	0.857 (0.658–1.115)
HDLC	–1.498	0.644	5.404	0.020	0.224 (0.063–0.791)
LDLC	–0.093	0.175	0.284	0.591	0.911 (0.646–1.285)
WBC count	0.128	0.053	5.868	0.015	1.137 (1.025–1.261)
Neutrophil	0.147	0.056	6.752	0.009	1.158 (1.037–1.294)
Lymphocyte	–0.769	0.380	4.100	0.043	0.463 (0.220–0.976)
Platelet count	–0.002	0.002	0.737	0.391	0.998 (0.993–1.003)
Monocyte count	1.683	0.760	4.899	0.027	5.382 (1.212–23.890)
CRP	0.009	0.005	3.286	0.070	1.009 (0.999–1.018)
Albumin	–0.173	0.041	18.015	<0.001	0.841 (0.777–0.911)
Hemoglobin	–0.033	0.011	9.281	0.002	0.968 (0.948–0.988)

OR, odds ratio; CI, confidence interval.

Univariate Logistic Regression Analysis

Univariate logistic regression analysis revealed a significant association of several factors with the presence of sarcopenia (Table 2). Notably, a lower GNRI

Table 3. Multivariate analysis of influencing factors.

Variable	β	SE	Wald	p-value	OR (95% CI)
GNRI	–0.092	0.027	11.789	<0.001	0.912 (0.865–0.961)
HDLC	–0.117	0.744	0.025	0.875	0.890 (0.207–3.827)
Neutrophil	0.003	0.050	0.003	0.958	1.003 (0.910–1.105)
Lymphocyte	–0.141	0.445	0.100	0.752	0.869 (0.363–2.078)
Monocyte count	0.445	1.077	0.171	0.679	1.561 (0.189–12.872)
Hemoglobin	–0.003	0.014	0.032	0.858	0.997 (0.970–1.026)

showed a strong association with increased sarcopenia risk (odds ratio [OR] = 0.902, 95% confidence interval [CI]: 0.864–0.942, $p < 0.001$). An OR less than 1, as observed for GNRI, indicates a protective effect, suggesting that each one-unit increase in GNRI corresponds to a decrease in the odds of sarcopenia. Other significant protective factors included higher BMI (OR = 0.799), albumin (OR = 0.841), hemoglobin (OR = 0.968), lymphocyte (OR = 0.463), and HDLC (OR = 0.224). Conversely, factors associated with an increased risk of sarcopenia included increased WBC count (OR = 1.137), neutrophil count (OR = 1.158), and monocyte count (OR = 5.382) (all $p < 0.05$).

Multivariate Logistic Regression Analysis

To identify independent predictors of sarcopenia, significant variables in the univariate analysis ($p < 0.05$) were included in a multivariate logistic regression model. Albumin and BMI were excluded from the model due to their high collinearity with GNRI (both being components of the GNRI formula), which was confirmed by a VIF analysis. Additionally, WBC count was also excluded after VIF diagnosis, as its collinearity exceeded acceptable limits. The final model included GNRI, HDLC, neutrophils, lymphocytes, monocytes, and hemoglobin.

As detailed in Table 3, GNRI remained a significant and independent predictor of sarcopenia after adjusting for all other variables (OR = 0.912, 95% CI: 0.865–0.961, $p < 0.001$). The finding indicates that each one-point increase in GNRI was correlated with approximately an 8.8% decrease in the odds of sarcopenia. However, in this adjusted model, no other variables retained statistical significance.

Diagnostic Performance of GNRI for Sarcopenia

ROC curve analysis revealed that the GNRI had an area under the curve (AUC) of 0.805 (95% CI: 0.718–0.892, $p < 0.001$) for diagnosing sarcopenia (Fig. 2). The optimal cut-off value, as determined by the Youden index, was 101.94. At this threshold, GNRI demonstrated a sensitivity of 85.40%, a specificity of 63.90%, a positive predictive value of 61.40%, and a negative predictive value of 86.70%.

Discussion

In this study, we investigated the relationship between the GNRI and sarcopenia in lung cancer patients. Our results indicate that GNRI is an independent predictor of sarcopenia and possesses valuable diagnostic potential. These findings

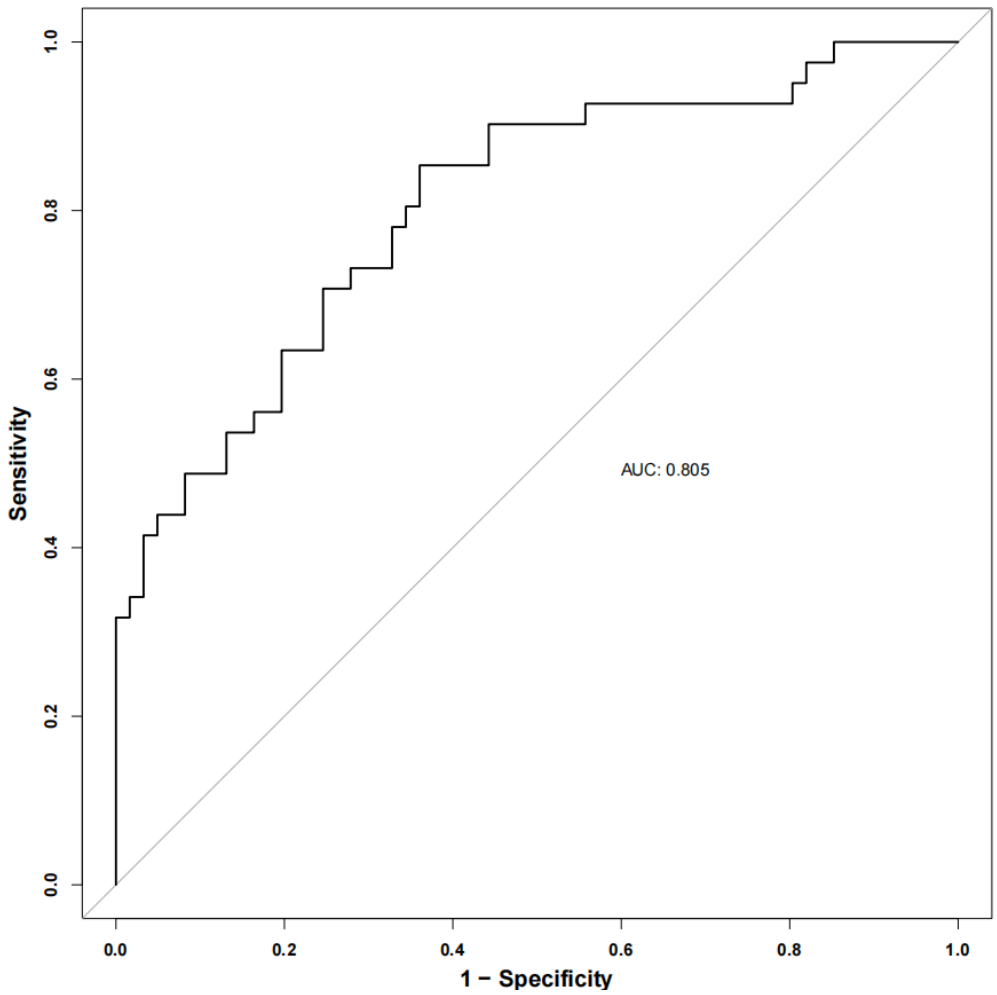

Fig. 2. Receiver operating characteristic (ROC) curve of GNRI for diagnosing sarcopenia. AUC, area under the curve.

underscore GNRI as a novel clinical tool for early screening and risk assessment of sarcopenia in lung cancer patients.

In our lung cancer cohort, the overall prevalence of sarcopenia was 40.2%, which is consistent with previous studies (Lin et al, 2022; Yang et al, 2019). Furthermore, patients with sarcopenia had significantly lower GNRI values compared to those without sarcopenia, and a low GNRI was independently associated with an increased risk of sarcopenia. This observation aligns with previous studies (Zhang et al, 2019; Xiang et al, 2022) and supports the prognostic significance of GNRI in cancer patients (Lidoriki et al, 2021). For instance, Zuo et al (2024) reported low GNRI as an independent predictor of sarcopenia in gastric cancer patients. Moreover, GNRI was found to correlate significantly with low muscle mass, reduced muscle strength, poor physical performance, and the presence of sarcopenia. Additionally, evidence reinforces the concept that systemic inflammation and nutritional status are key prognostic determinants in cancer (Li et al, 2021).

Several potential mechanisms may explain the observed association between GNRI and sarcopenia; however, our retrospective design prevents causal inference. Serum albumin—a key component of GNRI—is a critical marker of protein nu-

tritional status. Hypoalbuminemia reflects decreased protein synthesis and/or increased visceral protein catabolism (Keller, 2019). In lung cancer patients, complex metabolic changes, including central nervous system effects (e.g., anorexia, fatigue) and accelerated muscle protein breakdown, contribute to malnutrition and ultimately lead to sarcopenia (Arends, 2024). The link between low albumin levels and the development of sarcopenia has been confirmed in several studies (Picca et al, 2022; Zhou et al, 2024). Furthermore, inflammation, which plays an essential role in the pathogenesis of lung cancer (Liu et al, 2020; Ritter and Greten, 2019), is also closely linked to hypoalbuminemia and has been recognized as a prognostic biomarker in cancer (Fiala et al, 2016; Zhou et al, 2015; Kuang et al, 2024). As this study did not directly measure inflammatory markers, the precise mechanistic relationship remains speculative.

Second, the weight component of GNRI serves as a predictor of overall nutritional status. Cancer patients are at high risk for malnutrition, with reported incidence rates ranging from 20% to 70% depending on disease stage and clinical setting (Marshall et al, 2019; Khachaturian, 1987). Low muscle mass is a common feature of malnutrition (Sayer and Cruz-Jentoft, 2022). A meta-analysis has reported that the OR of malnutrition in patients with sarcopenia was 4.06 (95% CI: 2.43–6.80) (Ligthart-Melis et al, 2020). Inflammatory cytokines within the tumour microenvironment can promote muscle protein breakdown and accelerate sarcopenia, while a poor nutritional status may exacerbate the inflammatory response, creating a vicious cycle (Ji et al, 2022; Massironi et al, 2023). However, as our study did not measure inflammatory cytokines or direct markers of muscle protein breakdown, the suggested interaction between malnutrition and inflammation in driving sarcopenia remains hypothetical and requires further investigation.

Our study further demonstrated that the GNRI had a diagnostic AUC of 0.805 for sarcopenia, with an optimal cut-off value of approximately 101.94, providing a valuable reference for clinical evaluation. Compared with other sarcopenia assessment tools, the GNRI is simple to calculate—requiring only routinely measured serum albumin and body weight—and does not rely on specialized equipment, making it suitable for widespread clinical application. This is particularly valuable in resource-limited settings where Dual-energy X-ray Absorptiometry (DXA) or bioelectrical impedance analysis is not available. However, further prospective research, incorporating direct evaluations of inflammation and protein metabolism, is necessary to validate the clinical application of GNRI and elucidate the underlying causal mechanisms.

The clinical implications of this study are twofold. First, GNRI can serve as an economical and practical tool for assessing sarcopenia risk in lung cancer patients, facilitating the early identification of individuals at high risk. Second, patients with a GNRI below the cut-off value should undergo further evaluation of muscle mass and function. Early nutritional interventions and tailored exercise programs for these patients may help prevent or delay the onset and progression of sarcopenia, ultimately improving treatment tolerance and potentially enhancing survival outcomes.

Despite specific, valuable findings, this study has several limitations. First, its retrospective, single-center design precludes the establishment of causality and may limit the generalizability of the findings. Second, sarcopenia was diagnosed based on low muscle mass and strength but did not include a measure of physical performance (e.g., gait speed), deviating from the full European Working Group on Sarcopenia in Older People 2 (EWGSOP2) and Asian Working Group for Sarcopenia (AWGS) consensus criteria and potentially underestimating the true prevalence of sarcopenia. Third, the limited sample size impedes internal model validation (e.g., via cross-validation) and subgroup analyses to explore the consistency of our findings across strata, such as cancer stage or age. Fourth, this study was cross-sectional, with data collected at the time of admission. This design did not allow the analysis of the potential confounding effects of different treatment regimens, whose full impact on nutritional status and muscle mass may not yet have occurred. Finally, the absence of long-term follow-up data prevented assessment of GNRI's prognostic value for predicting survival or treatment outcomes.

During sample size estimation, we assumed a sarcopenia prevalence of approximately 40% among lung cancer patients and included 3–5 independent predictors in the multivariate regression model; however, the final model included 7 independent predictors, exceeding the initial assumptions. Moreover, retrospective studies often involve missing data, which can affect the stability and reliability of the model. Although adjustment using G*Power ensured that an adequate sample size was included to improve the statistical power of the study, the relatively small sample size and potential missing data remain limitations. Therefore, these results should be interpreted with caution, and further validation in extended, prospective studies is needed.

Conclusion

Our findings demonstrate that GNRI is an independent predictor of sarcopenia in lung cancer patients and exhibits moderate diagnostic performance. As a simple and practical nutritional assessment tool, GNRI can be readily applied for the initial screening of sarcopenia risk in clinical practice among lung cancer patients. Future prospective research should validate the predictive value of GNRI for both sarcopenia and overall prognosis, and explore individualized nutritional intervention approaches based on GNRI to improve clinical outcomes.

Key Points

- Sarcopenia is prevalent among lung cancer patients and is strongly associated with poor outcomes.
- GNRI serves as an independent predictor of sarcopenia in lung cancer patients.
- GNRI shows favorable diagnostic performance for sarcopenia, with an AUC of 0.805.
- GNRI is a simple and readily accessible tool for clinical utility.
- Early identification of sarcopenia risk can inform nutritional interventions in these patient populations.
- Future studies should validate these findings in larger, prospective cohorts.

Availability of Data and Materials

The data and materials in the current study are available from the corresponding author upon reasonable request.

Author Contributions

XBZ, JPW, LYJ and QF designed the study. XBZ and JPW conducted the literature search. LYJ and QF acquired the data. XBZ wrote the article. JPW performed data analysis. XBZ revised the article. All authors contributed to the important editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

This retrospective analysis adhered to the ethical standards outlined in the Declaration of Helsinki and was approved by the Ethics Committee of Yiwu Central Hospital (Approval No. K2025-IRB-015). Written informed consent was obtained from all participants enrolled in this study.

Acknowledgement

Not applicable.

Funding

This research received no external funding.

Conflict of Interest

The authors declare no conflict of interest.

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