Am J Transl Res 2025;17(7):5746-5756
www.ajtr.org /ISSN:1943-8141/AJTR0164650

Original Article

Association between nutritional risk and clinical outcomes in patients with acute exacerbation of chronic obstructive pulmonary disease

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Received March 20, 2025; Accepted July 7, 2025; Epub July 25, 2025; Published July 30, 2025

Abstract: Objective: To explore the relationship between nutritional risk and clinical outcomes in patients hospitalized for acute exacerbations of chronic obstructive pulmonary disease COPD (AECOPD). Methods: The medical records of 220 AECOPD patients hospitalized between June 2022 and June 2024 were retrospectively analyzed. The patients were categorized into two groups based on their Nutritional Risk Index (NRI): high-risk ([NRI] < 92) group and low-risk (NRI ≥ 92) group. Clinical outcomes assessed included albumin levels, arterial blood gas parameters, frequency of exacerbations, in-hospital mortality, length of hospital stays, readmission rates, and health-related quality of life (HRQoL). Pulmonary function recovery, including forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC), was also evaluated post-treatment. Results: Compared to the low-risk group, patients in the high-risk group exhibited significantly lower albumin levels (P = 0.007), increased frequency of exacerbations (P = 0.005), higher in-hospital mortality (P = 0.004), prolonged hospital stays (P = 0.001), and elevated readmission rates (P = 0.002). High-risk patients also reported significantly lower physical function (PF) and mental health scores. After treatment, improvements in FEV_1 and FVC were significantly greater in the low-risk group (P < 0.05). Conclusion: Nutritional risk is closely associated with the severity, prognosis, and recurrence of AECOPD. These findings underscore the importance of nutritional assessment and intervention in the management of hospitalized AECOPD patients.

Keywords: Chronic obstructive pulmonary disease (COPD), acute exacerbations, nutritional risk, hospitalization, mortality, quality of life

Introduction

Chronic obstructive pulmonary disease (COPD) is a prevalent respiratory condition characterized by persistent, irreversible airflow limitation and chronic pulmonary inflammation. The primary etiological factors include smoking and exposure to environmental pollutants [1-3]. COPD significantly compromises patients' health, leading to increased healthcare utilization, frequent hospitalizations, and considerable socioeconomic burden. Acute exacerbation of COPD (AECOPD), defined as sudden worsening of respiratory symptoms, often require urgent medical intervention and, in many cases, hospitalization [4, 5]. These episodes accelerate disease progression, impair quality of life, and contribute to higher healthcare costs and readmission rates [6, 7].

Nutritional status plays a crucial role in COPD management. Malnutrition, particularly prevalent during acute exacerbations, is often overlooked. Many patients experience involuntary weight loss and muscle wasting, a condition known as cachexia, driven by elevated energy expenditure, reduced dietary intake, and the effects of dyspnea, fatigue, and systemic inflammation. Research shows that malnutrition is associated with impaired pulmonary function, reduced exercise tolerance, and increased hospitalization risk among COPD patients [8-10]. However, further investigation is needed to clarify the extent to which nutri-

https://doi.org/10.62347/MCZH5861

tional risk influences in-hospital outcomes during AECOPD episodes.

Albumin is a commonly used marker of nutritional status. Hypoalbuminemia has been associated with adverse clinical outcomes, not only due to poor nutritional reserves but also as an indicator of systemic inflammation [11-13]. Nutritional status seems to may affect several physiological domains critical to COPD, including respiratory muscle strength, immune competence, and overall energy homeostasis [14]. Despite an apparent association between nutrition and COPD exacerbations, a comprehensive evaluation of how nutritional risk specifically influences clinical outcomes, such as disease severity, length of hospitalization, prognosis, and recurrence, during AECOPD remains limited.

This study therefore aims to elucidate the relationship between nutritional risk, as determined by established clinical markers, and critical in-hospital outcomes in patients with AECOPD.

Methods

Subjects selection

This retrospective cohort study included 220 individuals diagnosed with COPD who were consecutively admitted to the People's Hospital of Leshan between June 2022 and June 2024. Clinical data, including demographic characteristics, laboratory results, and clinical parameters, were retrieved from the hospital's electronic medical record system. The study was approved by the Ethics Committee and Institutional Review Board of the People's Hospital of Leshan.

Inclusion and exclusion criteria

Inclusion criteria: (1) confirmed diagnosis of COPD according to the American Thoracic Society (ATS) guidelines [15]; (2) diagnosis of AECOPD, defined by the presence of two or more principal symptoms (increased dyspnea, increased sputum purulence, and increased sputum volume) or one major symptom accompanied by at least one minor symptom (rhinorrhea, nasal congestion, wheezing, sore throat, or cough), with symptoms persisting for more than 48 hours; (3) age ≥ 18 years; and (4) complete clinical data available for analysis.

Exclusion criteria: (1) concurrent respiratory diseases such as bronchial asthma, pulmonary embolism, lung cancer, interstitial lung disease, or acute respiratory distress syndrome (ARDS); (2) severe dysfunction of other organs or comorbid conditions significantly affecting other systems; (3) psychiatric disorders; (4) electrolyte imbalance, specifically hyponatremia (sodium levels < 135 mmol/L) or hypernatremia (sodium levels > 145 mmol/L); and (5) severe hepatic impairment or end-stage renal disease (creatinine clearance < 15 mL/min).

Grouping and treatment methods

The Nutritional Risk Index (NRI) is a scoring system designed to evaluate the nutritional status of patients and to estimate the risk of adverse clinical outcomes associated with malnutrition. It incorporates serum albumin levels and body weight status, specifically the ratio of current to ideal body weight or recent weight loss. In this study, the NRI was evaluated upon hospital admission for each patient with AECOPD. For biochemical analysis, 5 mL of fasting venous blood was collected from the antecubital vein. The samples were subjected to centrifugation at 3,000 rpm for 10 minutes, and the resulting serum was stored at -80°C. Serum albumin (ALB) levels were measured using the biuret spectrophotometry method with a Beckman Coulter AU5800 instrument. The NRI was calculated using the formula: NRI = 1.519 × albumin (g/L) + 0.417 × (current body weight/ideal body weight) × 100, where ideal body weight was defined as 22 × height^2 (measured in meters), and body weight was measured in kilograms. An NRI score below 92 typically indicates high malnutrition risk, while a score of 92 or above suggests a lower risk. Based on this classification, 108 patients were assigned to the high-risk group and 112 to the low-risk group. All patients received standard treatment regimens, including antibiotics, bronchodilators, expectorants, and oxygen therapy. Supportive interventions such as smoking cessation counseling, postural drainage, and activities of daily living (ADL) training were also provided. Long-term prognosis was evaluated through follow-up.

Assessment tools

Complete blood count and biochemical profile: A 5 mL sample of fasting venous blood was col-

lected from each patient before 8 a.m. Hematological parameters, including red blood cells, white blood cells, neutrophils, lymphocytes, eosinophils, hemoglobin, and platelets, were analyzed using the blood analyzer (UniCel DxH800, Beckman Coulter, USA). Biochemical indices - including serum albumin, potassium, blood glucose, blood lipid profile, and markers of liver and renal function were measured using the Synchron X20 automated biochemical analyzer (Beckman Coulter, USA).

Assessment of dyspnea: The modified Medical Research Council (mMRC) dyspnea scale was used to evaluate the impact of dyspnea on daily activities. This self-reported scale consists of five grades (0-4). Level 0: breathlessness only during strenuous exercise; Level 1: shortness of breath when hurrying on level ground or walking up a slight hill; Level 2: Walks slower than peers due to breathlessness, or needs to pause for breath when walking at their own pace on level ground; Level 3: stops for breath after walking about 100 meters or just a few minutes on level ground; Level 4: Too breathless to leave the house or becomes breathless while getting dressed or undressed [16].

Arterial Blood Gas (ABG) analysis: Arterial blood samples (1-3 mL) were collected from the radial artery while patients were breathing at rest. Samples were anticoagulated and promptly analyzed using the RapidPoint 500 blood gas analyzer (Siemens Healthineers, USA). Parameters measured included pH, arterial partial pressure of carbon dioxide ($PaCO_2$), arterial partial pressure of oxygen (PaO_2), and arterial oxygen saturation (SaO_2).

Pulmonary Function Tests (PFTs): Pulmonary function parameters - including forced Vital Capacity (FVC), forced expiratory volume in one second (FEV_1), maximal inspiratory pressure (PImax), and maximal expiratory pressure (PEmax) were evaluated using a pulmonary function analyzer (MasterScreen CPX, Jaeger, Germany). Participants were directed to perform maximum respiratory efforts against an occluded airway. PImax was assessed starting from the residual volume (RV), whereas PEmax was measured beginning at total lung capacity (TLC).

Global Initiative for Chronic Obstructive Lung Disease (GOLD) classification: The severity of airway obstruction was classified according to the GOLD criteria, based on spirometry measurements. Stage I: $FEV_1 \geq 80\%$ of the predicted value; Stage II: an $50\% \leq FEV_1 < 80\%$ of predicted; Stage III: $30\% \leq FEV_1 < 50\%$ of predicted; and Stage IV: $FEV_1 < 30\%$ of predicted [17].

6-Minute Walk Test (6-MWT): The 6-MWT was utilized to assess functional exercise capacity and cardiopulmonary endurance. Patients were instructed to walk along a flat, straight corridor for six minutes at their fastest comfortable pace. The total distance walked was recorded. A distance less than 150 m suggests severe cardiac dysfunction, a distance between 150 and 425 meters indicates moderate cardiac dysfunction, and a distance between 426 and 550 meters suggests mild cardiac dysfunction.

Sequential Organ Failure Assessment (SOFA): The SOFA scoring system was designed to quantify the extent of organ dysfunction by evaluating six physiological systems: coagulation, respiratory, cardiovascular, hepatic, central nervous system, and renal. Each system was rated on a 0-4 scale, yielding a cumulative score ranging from 0 to 24. Higher total scores indicate more severe multi-organ failure and are associated with poorer clinical outcomes.

Health-Related Quality of Life (HRQoL): Health-related quality of life (HRQoL) was assessed using the 36-item Short Form Health Survey (SF-36), a validated instrument containing 36 questions distributed across eight domains: physical functioning (PF), physical role limitations (RP), bodily pain (BP), general health (GH), vitality (VT), social functioning (SF), emotional role limitations (RE), and mental health (MH). Each domain score ranges from 0 to 100, with higher scores indicating better quality of life. The SF-36 has demonstrated high internal consistency, evidenced by a Cronbach's alpha of 0.921 [18].

Primary outcomes included FEV1, in-hospital mortality, length of hospital stay, hospital readmission rates, PaO_2, and $PaCO_2$. Secondary outcomes included comorbidities, HRQoL scores, exercise capacity measured by the 6MWT, cardiopulmonary performance assessed via PImax and PEmax, frequency of acute exacerbations, and the incidence of multiple organ dysfunction syndrome (MODS).

Table 1. Comparison of general information between the two groups

	High risk group (n = 108)	Low risk group (n = 112)	t/χ^2	P
Gender (F/M)	50/58	54/58	0.081	0.776
Age (years)	53.89 ± 12.14	54.27 ± 11.93	0.235	0.815
BMI (kg/m^2)	26.56 ± 3.62	25.64 ± 3.47	1.915	0.057
Smoking history [n (%)]	65 (60.19%)	72 (64.29%)	0.394	0.530
High blood pressure [n (%)]	24 (22.22%)	26 (23.21%)	0.031	0.861
Dyslipidemia [n (%)]	17 (15.74%)	21 (18.75%)	0.348	0.555
Coronary artery disease [n (%)]	14 (12.96%)	19 (16.96%)	0.690	0.406
Diabetes [n (%)]	12 (11.11%)	15 (13.39%)	0.266	0.606
Osteoporosis [n (%)]	12 (11.11%)	14 (12.5%)	0.102	0.750
Anxiety [n (%)]	14 (12.96%)	15 (13.39%)	0.009	0.925
Depression [n (%)]	10 (9.26%)	13 (11.61%)	0.324	0.569
Degree of dyspnea (%)			0.302	0.583
≥ 2	58 (53.7%)	56 (50%)		
< 2	50 (46.3%)	56 (50%)		
Medical prescriptions				
Anticholinergics [n (%)]	54 (50%)	58 (51.79%)	0.070	0.791
Oral corticosteroids [n (%)]	46 (42.59%)	52 (46.43%)	0.328	0.567
Influenza vaccination [n (%)]	57 (52.78%)	56 (50%)	0.170	0.680
Respiratory rehabilitation [n (%)]	68 (62.96%)	64 (57.14%)	0.776	0.378
Long term oxygen therapy [n (%)]	56 (51.85%)	57 (50.89%)	0.020	0.887

F, female; M, male; BMI, body mass index.

Statistical analysis

Data were analyzed using SPSS 19 software (SPSS Inc., Chicago, IL, USA) and the R software package version 3.0.2 (Free Software Foundation, Inc., Boston, MA, USA). Continuous variables with normal distribution were expressed as mean ± standard deviation (SD). Comparisons of continuous variables between two groups were performed using unpaired t-tests for normally distributed data. Categorical data were reported as frequencies and percentages, and the chi-square test (χ^2) was used to compare proportions between groups. When expected cell counts were less than 5, Fisher's exact test was applied instead of the chi-square test to ensure. Both univariate and multivariate logistic regression analyses were employed to calculate odds ratio (OR) and 95% confidence interval (CI) for selected continuous variables. A two-tailed *P*-value < 0.05 was considered statistically significant.

Results

General information

Baseline information showed no statistically significant differences between the high-risk (n = 108) and low-risk (n = 112) groups in gender distribution (*P* = 0.776), mean age (*P* = 0.815), or BMI (*P* = 0.057) (**Table 1**). Similarly, no significant differences were observed in the prevalence of smoking history (*P* = 0.530), hypertension (*P* = 0.861), dyslipidemia (*P* = 0.555), coronary artery disease (*P* = 0.406), diabetes (*P* = 0.606), osteoporosis (*P* = 0.750), anxiety (*P* = 0.925), or depression (*P* = 0.569). The degree of dyspnea, as assessed by the mMRC scale, was also comparable between groups (*P* = 0.583). Regarding medical management strategies, the use of anticholinergics, oral corticosteroids, influenza vaccination, respiratory rehabilitation, and long-term oxygen therapy did not differ significantly between the two groups (all *P* > 0.05).

Laboratory parameters

The high-risk cohort exhibited markedly reduced albumin levels relative to the low-risk cohort (*P* = 0.007) (**Table 2**). Other laboratory parameters, including red blood cell count, white blood cell count, neutrophils, lymphocytes, eosinophils, platelet count, hematocrit, hemoglobin, creatinine, potassium, or blood urea nitrogen, did not show marked distinctions

Table 2. Comparison of laboratory parameters between the two groups

	High risk group (n = 108)	Low risk group (n = 112)	t	P
RBC ($\times 10^{12}$/L)	4.25 ± 0.68	4.29 ± 0.71	0.469	0.640
WBC ($\times 10^{9}$/L)	6.84 ± 1.49	6.51 ± 1.3	1.745	0.082
NEU ($\times 10^{9}$/L)	4.56 ± 1.28	4.60 ± 1.37	0.249	0.803
LYM ($\times 10^{9}$/L)	1.14 ± 0.58	1.23 ± 0.50	1.209	0.228
E ($\times 10^{8}$/L)	0.80 ± 0.05	0.79 ± 0.05	0.696	0.487
PLT ($\times 10^{9}$/L)	179.58 ± 58.46	180.54 ± 60.12	0.120	0.905
HCT (%)	42.05 ± 5.57	41.59 ± 7.34	0.529	0.598
HB (g/L)	138.64 ± 21.14	134.25 ± 19.81	1.589	0.113
Cr (g/L)	152.34 ± 30.45	149.57 ± 28.84	0.693	0.489
Potassium (mmol/L)	4.12 ± 1.18	4.21 ± 1.04	0.598	0.550
ALB (g/L)	32.64 ± 7.24	35.61 ± 8.91	2.716	0.007
BUN (mmol/L)	7.16 ± 2.61	6.58 ± 2.96	1.558	0.121

RBC, red blood cell; WBC, white blood cell count; NEU, neutrophil count; LYM, lymphocyte count; E, eosinophil; PLT, platelet count; HCT, hematocrit; HB, hemoglobin; Cr, creatinine; ALB, albumin; BUN, blood urea nitrogen.

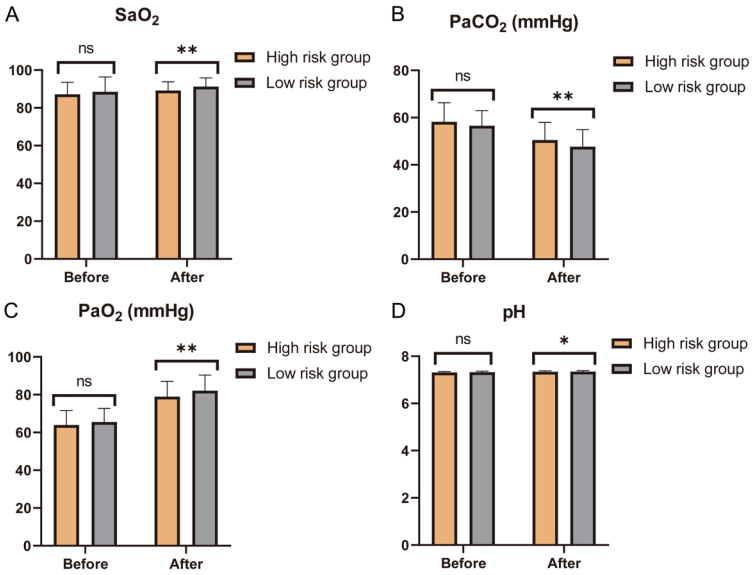

Figure 1. Comparison of arterial blood gas parameters between the two groups at admission and after therapy. A: SaO_2, arterial oxygen saturation; B: $PaCO_2$, arterial carbon dioxide partial pressure; C: PaO_2, arterial oxygen partial pressure; D: pH; ns: no significant difference; *: $P < 0.05$; **: $P < 0.01$.

between the two groups (all $P > 0.05$). These findings highlight hypoalbuminemia as a distinguishing marker of nutritional risk in AECOPD patients and suggest its potential relevance in clinical assessment and management.

Arterial blood gas

At admission, arterial blood gas parameters, including SaO_2, $PaCO_2$, PaO_2, and pH did not differ significantly between the two cohorts ($P > 0.05$) (**Figure 1**), indicating that, despite differences in nutritional status, the two groups had similar blood gas levels at admission.

After treatment, the low-risk cohort showed significantly greater improvements in arterial blood gas values. Specifically, SaO_2 levels increased significantly ($P = 0.001$), $PaCO_2$ decreased ($P = 0.005$), PaO_2 increased ($P = 0.004$), and pH values were more normalized ($P = 0.026$) compared to the high-risk group. Patients with lower nutritional risk experienced more favorable improvements in both respiratory gas exchange and acid-base balance following standard therapy for AECOPD.

Pulmonary function

At baseline, pulmonary function indicators, including FVC, FEV_1, FEV_1/FVC ratio, PE_{max}, and PI_{max} were similar between the high-risk and low-risk cohort ($P > 0.05$) (**Figure 2**), indicating no initial difference in lung function.

After treatment, the low-risk cohort demonstrated significantly greater improvements in

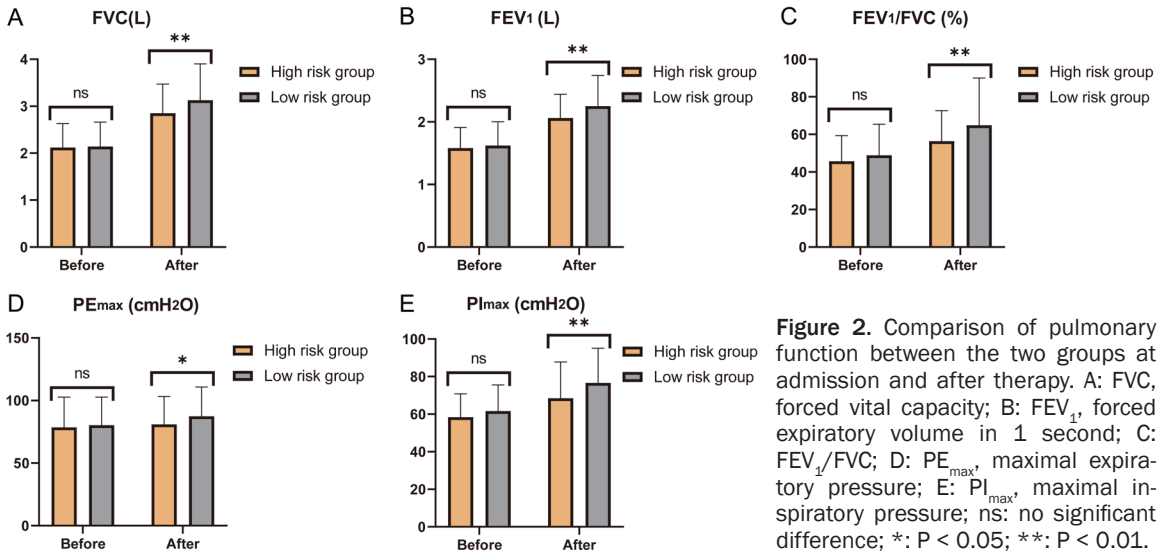

Figure 2. Comparison of pulmonary function between the two groups at admission and after therapy. A: FVC, forced vital capacity; B: FEV_1, forced expiratory volume in 1 second; C: FEV_1/FVC; D: PE_{max}, maximal expiratory pressure; E: PI_{max}, maximal inspiratory pressure; ns: no significant difference; *: P < 0.05; **: P < 0.01.

Table 3. Comparison of comorbidities, in-hospital mortality, and readmission rates between the two groups

	High risk group (n = 108)	Low risk group (n = 112)	t	P
Frequency of acute exacerbations (times/year)	3.16 ± 1.24	2.74 ± 0.93	2.823	0.005
In-hospital mortality (%)	20.21 ± 8.36	17.25 ± 6.54	2.916	0.004
Hospital lengths of stay (days)	13.32 ± 3.24	11.87 ± 3.45	3.223	0.001
Readmission rate (%)	22.98 ± 8.27	19.72 ± 7.42	3.080	0.002
MODS (%)	15.38 ± 4.25	13.74 ± 3.47	3.116	0.002
ARF (%)	26.54 ± 6.85	24.98 ± 6.51	1.730	0.085

MODS, multiple organ dysfunction syndrome; ARF, acute respiratory failure.

all pulmonary function parameters compared with the high-risk cohort. Specifically, the low-risk group showed greater gains in FVC (P = 0.005), FEV_1 (P = 0.001), and FEV_1/FVC ratio (P = 0.003). Additionally, PEmax (P = 0.036) and PImax (P = 0.002) were significantly higher in low-risk cohort after treatment. These findings indicate that patients with better nutritional status respond more favorably in terms of lung function after treatment for AECOPD.

Comorbidities, mortality and readmission

The high-risk cohort experienced a significantly higher frequency of acute exacerbations (P = 0.005) and in-hospital mortality (P = 0.004) compared to the low-risk group (**Table 3**). In addition, the high-risk cohort had a longer average hospital stay (P =0.001) and a higher readmission rates (P = 0.002). The incidence of MODS was markedly elevated in the high-risk group (P = 0.002). However, no significant difference was observed in the incidence of acute respiratory failure (ARF) between the groups (P = 0.085). These findings suggest a clear association between higher nutritional risk and adverse clinical outcomes, including increased exacerbation frequency, mortality, extended hospitalization, and higher readmission rate in patients with AECOPD.

GOLD classification, 6-MWT and SOFA

The distribution of GOLD stages did not show marked distinctions between the two cohorts (P = 0.072) (**Table 4**). The 6-MWT distance was significantly longer in the low-risk cohort compared to the high-risk cohort (P = 0.032), indicating better functional exercise capacity. Additionally, SOFA scores were markedly elevated in the high-risk cohort, reflecting more severe organ dysfunction (P < 0.001). These findings suggest that individuals with higher nutritional risk exhibit not only poorer function-

Table 4. Comparison of GOLD classification distribution, 6- MWT, and SOFA scores between the two groups

	High risk group (n = 108)	Low risk group (n = 112)	t/χ^2	P
GOLD classification, n (%)			6.99	0.072
Stage I	11 (10.19%)	24 (23.21%)		
Stage II	37 (34.26%)	42 (37.5%)		
Stage III	38 (35.19%)	28 (25%)		
Stage IV	22 (20.37%)	18 (14.29%)		
6-MWT (meters)	304.56 ± 102.31	335.22 ± 108.45	2.155	0.032
SOFA	6.05 ± 2.05	4.95 ± 1.72	4.318	< 0.001

GOLD, global Initiative for chronic obstructive lung disease; 6-MWT, 6-minute walk test; SOFA, sequential organ failure assessment.

Table 5. Comparison of HRQoL scores between the two groups

Scores	High risk group (n = 108)	Low risk group (n = 112)	t	P
PF	62.55 ± 25.12	69.41 ± 24.51	2.052	0.041
SF	74.49 ± 23.18	80.24 ± 25.74	1.740	0.083
VT	60.39 ± 19.12	61.52 ± 20.81	0.421	0.674
MH	66.07 ± 20.48	72.36 ± 22.44	2.169	0.031
BP	63.75 ± 21.88	66.42 ± 24.57	0.852	0.395
RE	74.61 ± 23.71	72.47 ± 22.14	0.693	0.489
RP	64.73 ± 23.14	67.42 ± 24.83	0.832	0.406
GH	63.61 ± 22.85	69.96 ± 24.52	1.986	0.048

PF, physical functioning; SF, social functioning; VT, vitality; MH, mental health; BP, bodily pain; RE, role limitations due to emotional problems; RP, role limitations due to physical health problems; GH, general health.

Table 6. Multivariate regression analysis of nutritional risk and primary outcomes

Influencing factors	OR (95% CI)	P
Nutritional risk (High/Low)-Hospital mortality	3.45 (1.78-6.69)	< 0.001
Nutritional risk (High/Low)-Length of stay	2.56 (1.32-4.95)	0.005
Nutritional risk (High/Low)-Readmission rates	2.89 (1.51-5.54)	0.001
Nutritional risk (High/Low)-FEV_1	1.79 (1.02-3.14)	0.042
Nutritional risk (High/Low)-PaO_2	2.15 (1.15-4.03)	0.017
Nutritional risk (High/Low)-$PaCO_2$	2.31 (1.21-4.42)	0.011

FEV_1, forced expiratory volume in 1 second; PaO_2, arterial oxygen partial pressure; B: $PaCO_2$, arterial carbon dioxide partial pressure.

al capacity but also greater organ function impairment during AECOPD.

HRQoL

The low-risk cohort scored significantly higher PF compared to the high-risk cohort ($P = 0.041$) (**Table 5**). MH and GH scores were also markedly improved in the low-risk cohort relative to the high-risk cohort ($P = 0.031$, $P = 0.048$). No significant differences were observed between the two groups in other SF-36 domains, including SF, VT, BP, RE, and RP (all $P > 0.05$). These findings suggest that nutritional risk was associated with poorer physical functioning, mental well-being, and general health status in patients hospitalized for AECOPD.

Multivariate regression analysis

Multivariate regression analysis revealed that high nutritional risk was an independent predictor of adverse clinical outcomes (**Table 6**). Patients in the high-risk group had significantly increased odds of in-hospital mortality (OR = 3.45, P < 0.001), prolonged length of hospital stay (OR = 2.56, P = 0.005), and higher readmission rates (OR = 2.89, P = 0.001) compared to those in the low-risk group. Furthermore, high nutritional risk correlated with impaired pulmonary function (OR = 1.79, P = 0.042), decreased PaO_2 (OR = 2.15, P = 0.017), and elevated $PaCO_2$ levels (OR = 2.31, P = 0.011). These results underscore the prognostic significance of nutritional risk in patients with AECOPD, highlighting its association with increased mortality, extended hospitalization, greater risk of readmission, and compromised respiratory function. The findings emphasizes

the critical need for nutritional risk screening and intervention in clinical practice to optimize patient prognosis and alleviate healthcare resource utilization.

Discussion

This study investigated the impact of nutritional risk on clinical outcomes among AECOPD patients, focusing on disease severity, hospitalization duration, prognostic indicators, and recurrence of exacerbation. Our results substantiate the critical contribution of nutritional status to these clinical endpoints.

A principal observation revealed an inverse relationship between serum albumin concentrations and the degree of nutritional risk, positioning hypoalbuminemia as a potential biomarker of suboptimal nutritional status in this cohort. Patients categorized as high nutritional risk exhibited significantly lower albumin levels relative to their low-risk counterparts. Albumin, a critical plasma protein, plays vital physiological roles beyond nutritional parameters, including preservation of colloid osmotic pressure, microvascular integrity, and transport of diverse endogenous and exogenous substances [19]. In COPD populations, hypoalbuminemia may reflect the combined effects of chronic systemic inflammation and poor dietary intake, both of which are common in patients with advanced disease. Inflammation can reduce albumin synthesis and accelerate protein catabolism, contributing to decreased serum levels [11, 20]. Our study suggests that strategies aimed at improving protein intake and reducing inflammation might help improve albumin levels, potentially leading to better clinical outcomes.

Our findings are consistent with previous literature, demonstrating that COPD patients with better nutritional status experience more significant improvements in SaO_2 and PaO_2 levels after treatment and faster recovery of respiratory function during hospitalization [21]. These results further support the importance of nutritional status in determining treatment outcomes of AECOPD. The more significant improvement in respiratory parameters among patients with low nutritional risk may be attributed to several biological mechanisms. First, adequate nutritional status is associated with enhanced immune function and antioxidant capacity, both of which help mitigate systemic inflammation and oxidative stress, thereby supporting pulmonary recovery. Second, sufficient nutrient intake ensures the availability of energy and substrates required for cellular repair and regeneration, which facilitates the healing of damaged lung tissues. In addition, proper nutrition also supports the musculoskeletal system, enhancing the strength and endurance of respiratory muscles and reducing fatigue [3, 22].

Patients at high nutritional risk often experience more frequent exacerbations. Malnutrition impairs immune system, increasing susceptibility to respiratory infections that can worsen COPD. Consequently, ensuring sufficient nutrient intake is essential to sustaining immune system function and reducing infection-related complications [23]. In particular, micronutrients like vitamins A, C, and E, along with trace elements are critical for immune regulation. A balanced diet helps prevent micronutrient deficiencies and supports overall immune resilience [24].

Our results also show that COPD patients with low nutritional risk experienced greater improvements in pulmonary function after treatment, suggesting that adequate nutritional status facilitates respiratory recovery. Nutrition plays a direct role in preserving skeletal muscle mass and strength, which are important for breathing. In cases of cachexia or severe muscle wasting, often seen in malnourished COPD patients, the respiratory muscles are weakened, leading to reduced exercise tolerance, compromised airway clearance, and impaired lung function [25]. Nutritional rehabilitation can support muscle regeneration and improve physical performance, ultimately contributing to better pulmonary outcomes.

We also noticed that patients with high nutritional risk had significantly longer hospital stays and higher readmission rate. This probably attributes to underlying physical and systemic vulnerabilities, including impaired immune responses, delayed wound healing, and muscle atrophy, all of which are commonly associated with malnutrition [26, 27]. Providing focused nutritional support could help reduce these risks by providing essential substrates for tissue repair, immune competence, and recovery.

Our study also identified a link between higher nutritional risk and the development of MODS, suggesting that malnutrition may worsen systemic inflammation, leading to organ dysfunction. Individuals with malnutrition are known to exhibit heightened oxidative stress and increased production of pro-inflammatory cytokines, both of which are central to the pathogenesis of MODS [28]. Addressing nutritional deficiencies could potentially protect patients from organ failure.

While the incidence of ARF did not differ significantly between groups, the observed relationship between nutritional risk and multiple adverse outcomes highlights the importance of incorporating nutritional evaluation and support into the comprehensive management of AECOPD. This underscores the multidimensional nature of COPD management, where addressing comorbidities and providing holistic care, including targeted nutritional support, remains essential [29, 30].

The observed association between higher nutritional risk and poor HRQoL indicates nutritional interventions benefit both clinical outcomes and overall quality of life. Malnutrition is often linked to symptoms such as fatigue, depression, and reduced physical and SF, all of which can adversely affect HRQoL [31]. Adequate nutrition plays a crucial role in maintaining energy homeostasis and mental health, and nutritional rehabilitation may lead to significant improvements in patient-perceived quality of life [31, 32].

In this study, we not only explored the relationship between nutritional risk and clinical outcomes in patients with AECOPD through logistic regression analyses. Multivariate regression analysis confirmed that even after adjusting for multiple potential confounding factors including age, gender, smoking history and comorbidities, high nutritional risk (NRI < 92) remained a significant predictor of poor clinical outcomes; including increased mortality, prolonged hospital stay, higher readmission rates, and impaired respiratory function. These findings emphasize nutrition's critical role in managing AECOPD, urging clinicians to prioritize assessing and improving nutritional status for better treatment outcomes.

Although offering valuable insights into nutritional risk and outcomes in AECOPD patients, this single-center study has inherent limitations. Its observational design prevents establishing causal relationships between nutritional status and clinical outcomes. Nutritional evaluation largely relied on selected biomarkers and clinical indicators, which may not fully capture the complexity of patients' nutritional states. Furthermore, the single-center cohort limits generalizability across diverse populations and healthcare settings. Important confounders including socioeconomic status and treatment adherence were not fully controlled, potentially influencing results. Another notable limitation is the absence of differentiation between acute and chronic nutritional deficiencies, both of which could have distinct implications for disease progression and recovery. Future research should employ multicenter, randomized controlled designs and incorporate multidimensional nutritional assessments to better elucidate the role of nutrition in AECOPD management.

Conclusion

Our study highlights that nutritional risk significantly impacts clinical outcomes in AECOPD patients. These findings support the integration of routine nutritional screening and timely intervention into standard management of COPD, particularly during acute exacerbations. Addressing nutritional needs may improve respiratory function, reduce hospital readmissions, and enhance quality of life. Future investigations should develop personalized nutritional strategies tailored to the specific metabolic demands of COPD patients, considering not only caloric adequacy but also the role of protein and micronutrients in modulating inflammation and supporting recovery. Further research into the biological mechanisms linking nutrition and COPD pathophysiology will better inform clinical practice.

Disclosure of conflict of interest

None.

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