



Comparative Prognostic Value of Serum Albumin, Prognostic Nutritional Index, and CRP-to-Albumin Ratio for In-Hospital Mortality in Hospitalized Pneumonia: A Retrospective Cohort Study

Pnömoni Nedeniyle Hastaneye Yatırılan Hastalarda Hastane İçi Mortalitenin Öngörülmesinde Serum Albumini, Prognostik Nutrisyonel Endeks ve CRP/Albumin Oranının Karşılaştırmalı Prognostik Değeri: Retrospektif Kohort Çalışması

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ABSTRACT

Aim: Pneumonia is a major cause of in-hospital mortality, particularly in older patients with multiple comorbidities. The comparative value of nutritional-inflammatory indices in this setting remains insufficiently clarified. We compared serum albumin, the Prognostic Nutritional Index (PNI), and the C-reactive protein/albumin ratio (CAR) as predictors of in-hospital mortality in adults hospitalized for pneumonia.

Materials and Methods: We retrospectively reviewed 294 adults (aged 18–90 years) hospitalized with pneumonia between January 2023 and January 2026. Patients with active malignancy, advanced hepatic failure, protein-losing conditions, or ongoing immunosuppression were excluded. Prognostic nutritional index was calculated as $(10 \times \text{albumin [g/dL]} + (0.005 \times \text{lymphocyte count [1/mm}^3])$ and CAR as $\text{CRP (mg/L)} / \text{albumin (g/L)}$. The primary endpoint was in-hospital all-cause mortality. Independent predictors were identified by logistic regression, with multicollinearity assessed by variance inflation factors (VIF). Discrimination was evaluated by ROC analysis with the Youden index.

Results: In-hospital mortality was 16.3% (48/294). Serum albumin showed the highest AUC (0.680; 95% CI 0.605–0.751; cutoff ≤ 32.5 g/L; sensitivity 67%, specificity 51%); PNI (AUC=0.611) and CAR (AUC=0.606) showed lower discrimination. Pairwise AUC comparisons (DeLong) were not performed, so the ordering is descriptive. In multivariable analysis, lower albumin (OR 0.840 per g/L, 95% CI 0.757–0.932; $p=0.001$), respiratory failure, and ICU admission were independent correlates of mortality; CAR was non-significant. No meaningful collinearity was observed between albumin and CAR (VIF=1.002 for both). Mortality was higher with PNI <45 than ≥ 45 (18.5% vs 5.9%; $p=0.027$), but PNI category was unrelated to etiologic distribution or resistant pathogen isolation.

Conclusion: Admission serum albumin was independently associated with in-hospital mortality and showed numerically higher discrimination than PNI and CAR. Composite indices added no measurable prognostic value beyond albumin. These findings support admission albumin as a practical early risk marker, pending prospective confirmation against validated severity scores.

Key words: pneumonia; serum albumin; prognostic nutritional index; CRP-to-albumin ratio; mortality; prognosis

ÖZET

Amaç: Pnömoni, ileri yaş ve yüksek komorbidite yükü olan hastalarda hastane içi mortalitenin önemli nedenlerindendir. Nutrisyonel durumun prognostik rolü bilinmekle birlikte, nutrisyonel-enflamatuvar endekslerin karşılaştırmalı değeri yeterince aydınlatılmamıştır. Bu çalışmada pnömoni tanısıyla yatırılan hastalarda serum albumin, Prognostik Nutrisyonel Endeks (PNI) ve CRP/albumin oranının (CAR) hastane içi mortalite ile ilişkisi araştırılmış ve prognostik performansları karşılaştırılmıştır.

Gereç ve Yöntem: Ocak 2023–Ocak 2026 tarihleri arasında pnömoni tanısıyla yatırılan 294 erişkin hasta retrospektif olarak değerlendirildi. Aktif malignite, ileri karaciğer yetmezliği, protein kaybettiren durumlar ve immünsüpresif tedavi dışlandı. PNI= $(10 \times \text{albumin [g/dL]} + (0,005 \times \text{lenfosit sayısı [1/mm}^3])$ formülüyle, CAR ise $\text{CRP (mg/L)} / \text{albumin (g/L)}$ oranı olarak hesaplandı. Birincil sonlanım hastane içi mortalite idi. Bağımsız prediktörler lojistik regresyonla belirlendi; albumin her iki endeksin de bileşeni olduğundan multikolinearite varyans inflasyon faktörleri (VIF) ile değerlendirildi. Prognostik performans ROC analizi ve Youden endeksi ile incelendi.

Bulgular: Hastane içi mortalite %16,3 ($n=48$) idi. Serum albumin en yüksek AUC değerini gösterdi (AUC=0,680; %95 GA 0,605–0,751; eşik $\leq 32,5$ g/L; duyarlılık %67,0; özgüllük %51,0); PNI (AUC=0,611) ve CAR (AUC=0,606) daha düşük diskriminasyon sergiledi. Formal AUC karşılaştırması (DeLong) yapılmadığından sıralama tanımlayıcıdır. Çok değişkenli analizde serum albumin (OR=0,840; %95 GA 0,757–0,932; $p=0,001$), solunum yetmezliği ve yoğun bakım yatışı mortalite ile bağımsız ilişkili bulundu; CAR anlamlılığa ulaşmadı. Albumin ile CAR arasında anlamlı kollinearite saptanmadı (VIF=1,002). Prognostik nutrisyonel endeks (PNI) <45 grubunda mortalite daha yüksek olmakla birlikte (%18,5'e karşın %5,9; $p=0,027$), PNI kategorisi etiyolojik dağılım veya dirençli mikroorganizma ile ilişkili değildi.

Sonuç: Yatış serum albumin düzeyi hastane içi mortalite ile bağımsız ilişkilidir ve PNI ile CAR'a kıyasla sayısal olarak daha yüksek diskriminatif performans göstermiştir; ancak formal AUC karşılaştırması yapılmamıştır. Bileşik endeksler bu kohortta albumin üzerine ölçülebilir katkı sağlamamıştır. Bulgular, albumin düzeyinin erken risk değerlendirmesindeki rolünü desteklemekte olup, şiddet skorlarını içeren ileriye dönük çalışmalarla doğrulanmalıdır.

Anahtar kelimeler: pnömoni; serum albumin; prognostik nutrisyonel endeks; CRP/albumin oranı; mortalite; prognoz

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Introduction

Pneumonia remains a leading cause of hospitalization, ICU admission, and death worldwide, particularly among elderly patients with significant comorbidities (1,2). Its course ranges from mild self-limiting illness to life-threatening respiratory failure, sustaining the need for prognostic tools identifying high-risk patients at admission.

Nutritional status is a key host determinant of outcome. Malnutrition is independently linked to impaired immunity, prolonged hospitalization, and excess mortality (3), and low admission serum albumin—reflecting both nutritional reserve and the acute inflammatory response—is consistently associated with adverse outcomes in community-acquired pneumonia (4).

The Prognostic Nutritional Index (PNI), originally developed in surgical patients (5), combines albumin with total lymphocyte count to reflect both nutritional and cellular immune status, and has shown prognostic value in sepsis and critical illness (6). The CRP-to-albumin ratio (CAR) adds a positive acute-phase reactant to albumin, aiming to integrate systemic inflammation with nutritional-synthetic reserve.

However, direct evidence for PNI and CAR in non-COVID pneumonia populations is limited, and their performance against albumin alone has not been systematically compared.

We therefore compared serum albumin, PNI, and CAR as predictors of in-hospital mortality in adults hospitalized with pneumonia, and examined whether composite indices offer incremental value over albumin alone.

Material and Methods

Study Design and Setting

This was a retrospective, single-center cohort study conducted at a tertiary care hospital. Patients admitted with pneumonia between January 2023 and January 2026 were reviewed. The study was approved by the local ethics committee (Decision No: 2796; 18/02/2026); informed consent was waived given the retrospective design and use of anonymized data. The study followed the Declaration of Helsinki.

Patient Identification and Diagnostic Verification

Cases were identified by ICD-10 codes (J12–J18) and independently reviewed by two pulmonologists blinded to nutritional parameters, microbiological results, and outcomes; cases without consensus were excluded. Pneumonia was defined by compatible clinical features (fever, cough, sputum, dyspnea, or pleuritic chest

pain) with a new or progressive pulmonary infiltrate on chest imaging. Management followed ATS/IDSA guidelines (2,7).

Eligibility Criteria

Adults aged 18–90 years with confirmed pneumonia and available admission serum albumin and lymphocyte count were eligible. Patients were excluded for: i) active malignancy or oncological therapy within the preceding six months (remote malignancy in remission for ≥ 6 months and off therapy was permitted); ii) advanced hepatic failure (Child-Pugh C); iii) chronic protein-losing conditions; iv) ongoing systemic immunosuppression (corticosteroids ≥ 15 mg/day methylprednisolone equivalent for > 15 days or other immunosuppressive agents; prior discontinued exposure was permitted); or v) incomplete data. In Table 1, “malignancy” refers to prior diagnosis in remission and “immunosuppression” to prior discontinued exposure.

Data Collection

Demographic characteristics, comorbidities, presenting symptoms, vital signs at admission, and laboratory and microbiological data were extracted from the electronic medical records. Comorbidity burden was quantified using the Charlson Comorbidity Index (8), and prior healthcare exposure was recorded.

Laboratory Assessment and Index Calculation

Blood samples were collected within 24 hours of admission. Prognostic nutritional index was calculated per Onodera et al. (5). The categorical PNI threshold of ≥ 45 was pre-specified from prior literature and not derived from ROC analysis of the present dataset (9).

$$\text{PNI} = (10 \times \text{Serum albumin [g/dL]}) + (0.005 \times \text{Total lymphocyte count [}/\text{mm}^3\text{]})$$

C-reactive protein/albumin ratio was calculated as CRP (mg/L) divided by serum albumin (g/L). Albumin behaves as a negative acute-phase reactant during infection (10), while lymphocyte count reflects cellular immune competence and may fall through apoptosis, migration, and PD-1/PD-L1 mediated exhaustion (11).

Microbiological Evaluation

Microbiological investigations followed routine clinical care. Sputum, endotracheal aspirates, and blood cultures were obtained as clinically indicated; respiratory specimens with > 10 squamous epithelial cells per low-power field on Gram stain were excluded. Distinction between infection and colonization was based on quantitative/semi-quantitative culture thresholds, Gram stain concordance, and the clinical judgment of the treating pulmonologist.

Culture positivity alone was not equated with pathogenicity. *Corynebacterium* spp., *Enterococcus* spp., and *Candida*/fungal isolates were considered colonizers unless supported by repeated isolation and a compatible clinical picture. Cases without microbiological identification were classified as culture-negative.

Outcome Definitions

The primary outcome was in-hospital all-cause mortality. Secondary outcomes were ICU admission, respiratory failure requiring invasive ventilation or high-flow oxygen, and length of hospital stay. Respiratory failure and ICU admission were defined by clinical status at admission and entered as baseline severity variables.

Statistical Analysis

Analyses used IBM Statistical Package for Social Sciences (SPSS) program version 26.0 (IBM Corp., Armonk, NY, USA). Normality was assessed by the Shapiro-Wilk test. Continuous variables are reported as mean $\pm$ SD or median (IQR); categorical variables as n (%). Groups were compared with the t-test, Mann-Whitney U, chi-square, or Fisher exact test as appropriate. Optimal cut-offs were determined by ROC analysis and Youden

index; pairwise AUC comparisons (DeLong) were not performed. Multivariable logistic regression identified independent mortality predictors, with entry at $p < 0.10$ in univariate analysis. Multicollinearity was assessed by VIF; albumin and PNI were not entered together. Significance was set at $p < 0.05$.

Results

Study Population

A total of 294 adult patients with confirmed pneumonia were included. The mean age was 72.87 ± 13.51 years, and 193 patients (65.6%) were male. Current and former smokers accounted for 20.7% and 46.9% of the cohort, respectively.

Comorbid conditions were present in 275 patients (93.5%); the most frequent were hypertension (56.8%), diabetes mellitus (36.4%), COPD (33.7%), and neurological disease/dementia (24.5%). A prior malignancy in remission was recorded in 46 (15.6%) and prior discontinued immunosuppressive exposure in 39 (13.3%). Supplemental oxygen was needed at admission in 143 (48.6%). In the preceding year, 120 (40.8%) had been hospitalized, and 51 (17.3%) had a prior ICU admission (Table 1).

Table 1. Baseline characteristics stratified by in-hospital mortality status (n=294)

Variable	Overall (n=294)	Survivors (n=246)	Non-survivors (n=48)	p value
Continuous variables				
Age (years), mean $\pm$ SD	72.87 $\pm$ 13.51	72.1 $\pm$ 13.8	77.6 $\pm$ 11.8	0.005
Serum albumin (g/L), mean $\pm$ SD	32.24 $\pm$ 5.01	32.76 $\pm$ 4.85	29.5 $\pm$ 4.92	<0.001
PNI, mean $\pm$ SD	35.69 $\pm$ 13.12	35.87 $\pm$ 13.91	35.12 $\pm$ 7.40	0.72
CAR, mean $\pm$ SD	4.37 $\pm$ 3.89	4.11 $\pm$ 3.70	5.71 $\pm$ 4.54	<0.001
SpO ₂ (%), mean $\pm$ SD	92.23 $\pm$ 5.42	92.5 $\pm$ 5.2	90.7 $\pm$ 6.4	0.033
DBP (mmHg), mean $\pm$ SD	70.13 $\pm$ 14.08	70.9 $\pm$ 14.0	66.3 $\pm$ 14.1	0.043
Urea (mg/dL), median (IQR)	48.1 (33.3–72.0)	42 (31.4–65.7)	73.1 (58.3–90.9)	<0.001
Hospital stay (days), mean $\pm$ SD	11.65 $\pm$ 9.84	9.9 $\pm$ 7.2	20.5 $\pm$ 15.5	<0.001
Categorical variables — n (%)				
Sex, male	193 (65.6)	161 (65.2)	32 (66.7)	0.84
Oxygen requirement at admission	143 (48.6)	109 (44.3)	34 (70.8)	<0.001
Any comorbidity	275 (93.5)	229 (92.7)	47 (97.9)	0.33
Hypertension	167 (56.8)	139 (56.5)	28 (58.3)	0.88
Diabetes mellitus	107 (36.4)	88 (35.8)	19 (39.6)	0.6
COPD	99 (33.7)	89 (36.2)	10 (20.8)	0.041
Neurological disease/dementia	72 (24.5)	53 (21.7)	19 (39.6)	0.007
Heart failure	62 (21.1)	49 (19.9)	13 (27.1)	0.26
Malignancy (in remission)	46 (15.6)	34 (13.9)	12 (25)	0.05
Past immunosuppressive exposure	39 (13.3)	31 (12.8)	8 (16.7)	0.44
Chronic kidney disease	19 (6.5)	12 (5.1)	7 (14.6)	0.021
Hospitalisation (last 1 year)	120 (40.8)	92 (37.4)	28 (58.3)	0.007
Prior ICU admission	51 (17.3)	34 (13.8)	17 (35.4)	<0.001
Resistant microorganism (admission)	136 (46.3)	100 (40.7)	36 (75)	<0.001
Respiratory failure	123 (41.8)	79 (32.2)	44 (91.7)	<0.001
ICU admission (index admission)	109 (37.1)	66 (26.8)	43 (89.6)	<0.001
PNI ≥ 45	51 (17.3)	48 (19.4)	3 (6.3)	0.027

Values are mean $\pm$ SD; median (IQR); or n (%). SpO₂: peripheral oxygen saturation; DBP: diastolic blood pressure; PNI: prognostic nutritional index; CAR: CRP-to-albumin ratio; COPD: chronic obstructive pulmonary disease; ICU: intensive care unit.

Mean serum albumin was 32.24 ± 5.01 g/L, mean PNI 35.69 ± 13.12 , mean CAR 4.37 ± 3.89 . Median CRP was 100.4 mg/L (IQR 44.1–186.2) and median WBC $11.460/\text{mm}^3$ (IQR 8, 680–15, 920).

Clinical Outcomes and Mortality-Associated Variables

In-hospital mortality was 16.3% (48/294). Respiratory failure was present in 123 patients (41.8%), and 109 (37.1%) required ICU admission at presentation.

Non-survivors were older (77.6 ± 11.8 vs 72.1 ± 13.8 years, $p=0.005$), with lower SpO_2 ($90.7 \pm 6.4\%$ vs $92.5 \pm 5.2\%$, $p=0.033$), lower diastolic blood pressure (66.3 ± 14.1 vs 70.9 ± 14.0 mmHg, $p=0.043$), and higher urea ($p<0.001$). Albumin and CAR differed between groups (both $p<0.001$); PNI did not. Non-survivors more often had prior ICU admission (35.4% vs 13.8%), a resistant microorganism (75.0% vs 40.7%), respiratory failure (91.7% vs 32.2%), and ICU admission during the index stay (89.6% vs 26.8%) (all $p<0.001$).

Microbiological Findings

A causative pathogen was identified in 147 patients (50.0%); the remainder were culture-negative. *Pseudomonas* spp. was the most frequent isolate (14.3%), followed by *Klebsiella* spp. (10.2%); a resistant microorganism was identified in 136 (46.3%). Etiologic distribution differed between survivors and non-survivors ($p<0.001$) (Table 2).

PNI Category Analysis

Using the pre-specified PNI ≥ 45 threshold, 51 (17.3%) were in the high-PNI group and 243 (82.7%) in the low-PNI group. Mortality was lower with high PNI (5.9% vs 18.5%; $p=0.027$) and recovery higher (94.1% vs 81.5%, $p=0.036$). Respiratory failure (37.3% vs 42.4%, $p=0.479$), resistant microorganism isolation (37.3% vs 48.1%, $p=0.163$), and etiologic distribution ($p=0.092$) did not differ (Table 3).

ROC Curve Analysis

Serum albumin had the highest AUC of 0.680 (95% CI 0.605–0.751); the Youden-optimal cut-off of ≤ 32.5 g/L yielded sensitivity 67.0% and specificity 51.0% (Youden index 0.18). Prognostic nutritional index had an AUC of 0.611 (95% CI 0.531–0.686), threshold ≤ 35 –36, sensitivity 79.0%, specificity 39.0%. C-reactive protein/albumin ratio had the lowest

Table 2. Microbiological findings and case-fatality rates ($n=294$)

Microorganism	Overall n (%)	Case-fatality (%)
<i>Pseudomonas</i> spp.	42 (14.3)	9.5
<i>Klebsiella</i> spp.	30 (10.2)	46.7
<i>Acinetobacter</i> spp.	16 (5.4)	43.8
<i>Escherichia coli</i>	15 (5.1)	0
<i>Corynebacterium</i> spp.	10 (3.4)	50.0
MRSA	8 (2.7)	12.5
<i>Enterococcus</i> spp.	7 (2.4)	14.3
<i>Stenotrophomonas</i> spp.	5 (1.7)	60.0
<i>Moraxella</i> spp.	5 (1.7)	0
Influenza virus	4 (1.4)	0
Fungal pathogens	3 (1.0)	66.7
<i>Streptococcus</i> spp.	3 (1.0)	0

CFR: case-fatality rate; MRSA: methicillin-resistant *Staphylococcus aureus*.
 $p<0.001$ for overall etiologic distribution by mortality status.

Table 3. Clinical outcomes stratified by PNI category ($n=294$)

Outcome	PNI <45 ($n=243$)	PNI ≥ 45 ($n=51$)	p-value
In-hospital mortality	45 (18.5%)	3 (5.9%)	0.027
Discharged with recovery	198 (81.5%)	48 (94.1%)	0.036
Respiratory failure	103 (42.4%)	19 (37.3%)	0.479
Resistant microorganism	117 (48.1%)	19 (37.3%)	0.163

PNI: prognostic nutritional index.
 Chi-square test for all comparisons.
 Cut-off of PNI ≥ 45 was adopted from prior literature.

AUC of 0.606 (95% CI 0.525–0.682), with a cut-off of ≥ 1.5 –2.0, sensitivity 65.0%, and specificity 50.0%. Pairwise AUC comparisons (DeLong) were not performed. All three markers showed poor-to-moderate discrimination with overlapping 95% CIs; albumin gave the numerically highest AUC (Fig. 1).

Multivariable Logistic Regression

The full multivariable model included albumin, CAR, urea, age, respiratory failure, and ICU admission. It was highly significant (Omnibus $\chi^2=118.634$, $df=6$, $p<0.001$; Nagelkerke $R^2=0.563$; Hosmer-Lemeshow $p=0.997$), with 88.8% overall classification. Collinearity was not meaningful: VIF was 1.002 (albumin), 1.002 (CAR), 1.040 (urea), 1.038 (age), 2.146 (respiratory failure), and 2.143 (ICU admission) —all below the conventional threshold of 5.

Three variables independently predicted mortality (Table 4): albumin (OR 0.840, 95% CI 0.757–0.932,

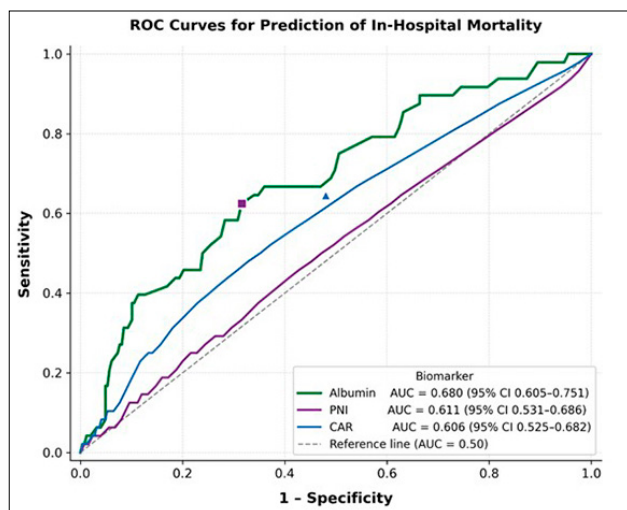


Figure 1. Receiver operating characteristic (ROC) curves of serum albumin, prognostic nutritional index (PNI) and C-reactive protein-to-albumin ratio (CAR) for predicting in-hospital mortality in 294 pneumonia patients (AUC: area under the curve; CI: confidence interval. Filled markers denote Youden-index-optimal cut-off points).

$p=0.001$), absence of respiratory failure (OR 0.066, 95% CI 0.020–0.218, $p<0.001$), and absence of ICU admission (OR 0.080, 95% CI 0.027–0.239, $p<0.001$). C-reactive protein/albumin ratio ($p=0.981$), urea ($p=0.150$), and age ($p=0.456$) were non-significant.

Discussion

This retrospective cohort study found that admission serum albumin was independently associated with in-hospital mortality in adults hospitalized with pneumonia and had the highest AUC among the three indices, with overlapping confidence intervals for PNI and CAR; since formal AUC comparisons were not performed, the ordering is descriptive. Respiratory failure and ICU admission were the strongest independent predictors, underscoring the dominant influence of admission severity, while albumin adds prognostic information not fully captured by severity alone.

The higher AUC of albumin relative to PNI is biologically plausible. Prognostic nutritional index was designed to augment albumin with lymphocyte count (5), but in acute pneumonia lymphocyte counts largely reflect the acute inflammatory response —through redistribution, apoptosis, and tissue migration— rather than the underlying immune reserve (11). Bermejo-Martin et al. showed that lymphopenia independently increased 30-day CAP mortality risk, independently of CURB-65 (12,13); Cilloniz et al. reported similar findings in CAP with sepsis (14). Consistent with this,

Table 4. Multivariable logistic regression: independent predictors of in-hospital mortality ($n=294$, full model)

Variable	OR	95% CI	p value
Serum albumin	0.840	0.757–0.932	0.001
CAR*	1.001	0.898–1.116	0.981
Urea	1.006	0.998–1.014	0.150
Age	1.015	0.977–1.054	0.456
Absence of respiratory failure**	0.066	0.020–0.218	<0.001
Absence of ICU admission**	0.080	0.027–0.239	<0.001

OR: odds ratio; CI: confidence interval; CAR: CRP-to-albumin ratio.

* CAR was pre-specified for entry but was non-significant ($p=0.981$); collinearity diagnostics showed no meaningful overlap with albumin (VIF=1.002 for both — see Discussion).

** Reference category is the presence of the condition; OR reflects the protective association of its absence.

Model: Omnibus $\chi^2=118.634$, $p<0.001$; Nagelkerke $R^2=0.563$; Hosmer-Lemeshow $p=0.997$; overall classification 88.8%; EPV=8.0.

Wang et al. found that within PNI the albumin component drove most of the discrimination (6), and De Rose et al. did not formally compare PNI with albumin alone (15).

The 16% reduction in mortality odds per g/L of albumin is consistent with its dual role as a nutritional marker and a negative acute-phase reactant, whose levels fall via IL-6-mediated hepatic suppression, capillary leak, and accelerated catabolism (10). Hypoalbuminemia thus reflects both pre-morbid vulnerability and the acute inflammatory response (4). Antioxidant, anti-inflammatory, and endothelial-stabilizing properties may further contribute (16,17).

C-reactive protein/albumin ratio had the lowest AUC and was not significant in the multivariable analysis. Variance inflation factor was near unity for both albumin (1.002) and CAR (1.002), so the two behaved as independent predictors. The non-significance of CAR is better explained clinically: CRP in acute pneumonia is directionally ambiguous —high levels may signal either a vigorous or an overwhelmed host response (18)— and albumin alone already integrates the nutritional and acute-inflammatory dimensions CAR is designed to combine.

The PNI ≥ 45 threshold identified a higher-mortality subgroup, but our Youden-optimal threshold (≤ 35 –36) was substantially lower. Most published PNI cut-offs in pneumonia derive from COVID-19 cohorts (19) or younger, less comorbid populations; Çınar et al. reported ≤ 43.7 in cardiovascular comorbidity (20). Chronic disease and pre-existing malnutrition likely compress the PNI distribution downward, so published cut-offs require local validation.

The strongest independent predictors of mortality were the absence of respiratory failure (OR 0.066) and the absence of ICU admission (OR 0.080). These reflect the severity of admission rather than later complications. Persistence of albumin as an independent predictor after adjusting for admission severity suggests nutritional status contributes through mechanisms only partly captured by acute severity, including susceptibility to secondary infections, altered antimicrobial pharmacokinetics, and reduced physiological reserve. Urea, age, and CAR were largely mediated by pathways already captured by albumin and disease severity.

Prognostic nutritional index category was not associated with etiologic distribution or with isolation of resistant pathogens (both $p > 0.05$), so nutritional-immune status does not preferentially predispose to specific species or resistance phenotypes. Low PNI or albumin should thus indicate outcome risk rather than guide empirical antibiotic use. The microbiological profile—*Pseudomonas* spp. predominant and resistant organisms in 46.3%—reflects a healthcare-associated spectrum expected in a high-comorbidity tertiary care population (7).

The inverse association between COPD and mortality is counterintuitive, since COPD is an established risk factor for pneumonia mortality (21). Rather than a true survival advantage, this likely reflects earlier hospital presentation and heightened symptom awareness (22), and possibly survival bias in a tertiary-care cohort; a modulatory role of inhaled corticosteroids (23) remains speculative.

Overall, our findings identify admission serum albumin as an inexpensive, readily available prognostic parameter that captures pre-morbid vulnerability not fully reflected by admission severity alone.

Limitations

This study has several limitations. Its retrospective single-center design carries risks of selection and information bias, and residual confounding cannot be excluded. Albumin is also influenced by hydration, hepatic function, capillary leak, and acute inflammation, and subclinical dysfunction or fluid shifts may have contributed to variability. Low Youden indices (0.15–0.18) mean thresholds should be used as risk-stratification aids, not diagnostic rules. The retrospective design also prevented reliable CAP-HAP differentiation and complete CURB-65/PSI calculation, so the incremental value of albumin beyond validated severity scores could not be assessed. The events-per-variable ratio of 8.0 carries a risk of model optimism, so estimates should be regarded as hypothesis-generating pending external validation. Finally, the 50% culture yield and predominance of healthcare-associated pathogens limit generalizability to lower-acuity CAP.

Conclusion

In this retrospective single-center cohort of adults hospitalized with pneumonia, admission serum albumin was independently associated with in-hospital mortality and had the highest—though descriptive—discriminative performance among the three indices. Composite indices added no measurable value beyond albumin; CAR's non-significance was not explained by collinearity ($VIF = 1.002$) but by the directional ambiguity of CRP in acute pneumonia. Clinically, admission albumin ≤ 32.5 g/L may serve as a practical alert value for closer monitoring and early nutritional assessment. Whether albumin adds value beyond CURB-65 or PSI, and whether correcting hypoalbuminemia is a modifiable prognostic target, should be tested in prospective multicenter studies.

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