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Gülhan AYHAN ALBAYRAK  
Mustafa İlteriş BARDAKÇI

<sup>1</sup>Şişli Hamidiye Etfal Training and Research  
Hospital, Chest diseases, İstanbul, Türkiye

## ORIGINAL ARTICLE

# INFLAMMATORY AND NUTRITIONAL BIOMARKERS FOR PREDICTING MORTALITY IN ELDERLY PATIENTS WITH COMMUNITY- ACQUIRED PNEUMONIA

## ABSTRACT

**Introduction:** Community-acquired pneumonia remains a leading cause of morbidity and mortality among older adults. Although conventional scoring systems, such as CURB-65 and pneumonia severity indexes, provide prognostic guidance, novel inflammation- and nutrition-based biomarkers may offer enhanced predictive accuracy. This study evaluated and compared the prognostic performance of six novel biomarkers; neutrophil-to-lymphocyte ratio, systemic immune-inflammation index, prognostic nutritional index, lymphocyte-to-monocyte ratio, C-reactive protein-to-albumin ratio, and modified Glasgow prognostic score in comparison with conventional scoring systems in elderly patients hospitalized with community-acquired pneumonia.

**Materials and Method:** A total of 105 patients aged  $\geq 65$  years who were hospitalized with community-acquired pneumonia between 2020 and 2025 were included in this retrospective cohort study, and their clinical, laboratory, and demographic data were collected. Prognostic performance for 30-day mortality was assessed using receiver-operating characteristic curves, Area Under the Receiver Operating Characteristic Curve and Kaplan–Meier survival analyses.

**Results:** C-reactive protein-to-albumin ratio demonstrated the highest discriminatory ability, followed by prognostic nutritional index and neutrophil-to-lymphocyte ratio. Although elevated platelet lymphocyte ratio was associated with reduced survival rates ( $p=0.038$ ), none of the novel biomarkers remained independent predictors in multivariate Cox regression analyses.

**Conclusion:** C-reactive protein-to-albumin ratio is a simple, cost-effective, and highly predictive biomarker for in-hospital mortality in elderly patients with community-acquired pneumonia, outperforming other indices and complementing conventional scores. Estimation of C-reactive protein-to-albumin ratio may facilitate early risk stratification and guide interventions in this population.

**Keywords:** Community-Acquired Pneumonia; Inflammation Mediators; Biomarkers; Prognosis; Aged.

## Correspondence

Gülhan AYHAN ALBAYRAK  
Phone : +905355416672  
e-mail : gulhanayhanalbayrak@gmail.com

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## INTRODUCTION

Community-acquired pneumonia (CAP) is a major cause of hospitalization and mortality, particularly affecting the elderly and those with chronic comorbidities globally (1,2). In older adults, immunosenescence and frailty compromise host responses, leading to atypical presentations, such as confusion or falls instead of classic symptoms which complicate early diagnosis and prognostication (3).

Traditional severity scores, such as PSI and CURB-65, provide useful prognostic information; however, their predictive accuracy is often limited in the geriatric population because of atypical presentations and dynamic inflammatory responses (4). Consequently, simple, objective, and readily available biomarkers that reflect systemic inflammation and nutritional status are needed.

Recent studies suggests neutrophil-to-lymphocyte ratio (NLR), systemic immune-inflammation index (SII), prognostic nutritional index (PNI), lymphocyte-to-monocyte ratio (LMR), C-reactive protein-to-albumin ratio (CAR), and modified Glasgow prognostic score (mGPS) as potential prognostic markers in CAP (5,6).

In elderly adults, CAP frequently presents with atypical or nonspecific features, complicating timely diagnosis and limiting the performance of established scoring systems (7). These limitations underscore the need for objective, easily applicable biomarkers that can enhance early prognostic assessment in this population. Accordingly, this study evaluated the prognostic performance of NLR, SII, PNI, LMR, CAR, and mGPS for predicting 30-day mortality among hospitalized elderly patients with CAP and compared their predictive accuracy with that of conventional scoring systems.

## MATERIALS AND METHOD

### Study Design

This retrospective cohort study was conducted at Şişli Etfal Training and Research Hospital, Istanbul,

Turkey. The study adhered to the principles of the WMA of Helsinki – Ethical Principles for Medical Research Involving Human Participants and was approved by the Institutional Ethics Committee (Approval No: 3193/28.10.2025). The retrospective design allowed for the evaluation of clinical and laboratory data extracted from previously recorded patient files over a defined study period.

Electronic medical records for patients with CAP at Şişli Hamidiye Etfal Training and Research Hospital between January 1, 2020 and January 1, 2025 were retrospectively reviewed. Diagnosis of CAP was established according to the Turkish Thoracic Society Pneumonia Guidelines, which require the presence of new pulmonary infiltrates on chest imagings together with compatible clinical symptoms, such as fever, cough, sputum production, or dyspnea.

Demographic characteristics (age, sex, and comorbidities), clinical outcomes (requirements for hospitalizations and 30-day all-cause mortality), and laboratory parameters, including white blood cell, neutrophil, lymphocyte, monocyte, and platelet counts, and C-reactive protein and albumin levels were systematically extracted from the electronic medical records for analysis.

### Study Participants

Eligible patients were aged  $\geq 18$  years, received a diagnosis of CAP confirmed according to ICD-10 codes, and had complete blood count and basic biochemical analyses were already performed upon admission. Patients who had hospital-acquired or ventilator-associated pneumonia, hematologic malignancies, active tuberculosis and received chemotherapy or immunosuppressive therapy within the preceding three months, , or those with missing laboratory data were excluded and remaining 105 patients were included in the study. Data about demographic characteristics, clinical outcomes, and laboratory parameters, including



white blood cell, neutrophil, lymphocyte, monocyte, and platelet counts, and C-reactive protein and albumin levels were systematically extracted from the electronic medical records for analysis.

### Calculation of Prognostic Scores

Conventional prognostic scoring systems (CURB-65 and PSI) and novel biomarker-based indices were calculated for each patient using demographic, clinical, and admission laboratory values. The novel scores, derived from routine blood tests, included the NLR = Neutrophil Count / Lymphocyte Count, PLR = Platelet Count / Lymphocyte Count, LMR = Lymphocyte Count / Monocyte Count, SII = Platelet Count X Neutrophil Count / Lymphocyte Count), PNI = (Albumin (g/dL) X 10) + (0.005 X Lymphocyte Count), and the CAR = CRP (mg/L) / Albumin (g/L). Additionally, the mGPS was categorized into 0, 1, or 2 points based on CRP and Albumin levels, while the PSI was calculated using its complex, established system incorporating age, sex, comorbidities, and various clinical/laboratory parameters (8).

### Statistical Analysis

Statistical analyses were performed with IBM SPSS Statistics for macOS, version 30.0 (IBM Corp.). Categorical variables were summarized as numbers and percentages, and continuous variables as means with standard deviations or medians with ranges. The distribution of continuous variables was evaluated using the Shapiro–Wilk test. Group comparisons were conducted using the Mann–Whitney U test for non-normally distributed continuous variables and chi-square or Fisher’s exact test for categorical variables. The discriminatory ability of NLR, SII, PNI, LMR, CAR, mGPS, and PSI for in-hospital mortality was assessed with receiver operating characteristic (ROC) curve analysis; area under the ROC curve values with 95% confidence intervals and optimal cut-off values were calculated. Subsequently, patients were categorized into high- and low-

risk groups according to these cut-off values. In-hospital survival, defined as the interval from admission to death or discharge, was analyzed with Kaplan–Meier methods, and between-group differences were tested with the log-rank test. Hazard ratios with 95% confidence intervals were reported. Statistical significance was set at  $P < 0.05$ .

## RESULTS

The analysis included 105 geriatric patients (mean age; 73 years, 33.3% women). Comorbid conditions were present in 37.4% of the cohort (Table 1). According to the CURB-65 scores, approximately 50% of the patients were classified as having low-to-moderate risk. Contrastingly, approximately 25% of them were categorized in the high or very high risk groups according to pneumonia severity indices (PSI), indicating considerable variation in baseline severity of CAP. Notably, 42.9% of the patients had an elevated mGPS, consistent with substantial systemic inflammation and highlighting the prognostic relevance of inflammation–nutrition biomarkers in this population.

The baseline demographic and clinical characteristics of survivors and non-survivors are presented (Table 2). Comorbidities were significantly more frequent among patients who died during hospitalization ( $P < 0.001$ ). All non-survivors were classified in the intermediate-risk CURB-65 category, and mortality increased markedly across higher PSI classes ( $P < 0.001$ ). Whereas most survivors fell into the low-to-moderate PSI groups, all deaths occurred in the very high-risk category. Higher modified Glasgow Prognostic Scores (mGPS scores) were more common among non-survivors ( $P < 0.001$ ). Length of hospital stay was significantly longer in patients who did not survive ( $P < 0.001$ ).

Laboratory findings and biomarker distributions according to survival status are shown (Table 3). Non-survivors had higher glucose, ALT, AST, CRP levels, leukocyte, and neutrophil counts ( $P < 0.05$ ), accompanied by lower lymphocyte and albumin

**Table 1.** Distribution of Demographic and Clinical Characteristics in Elderly Patients With Community-Acquired Pneumonia

Variables (N=105)	n (%)	Mean±SD	Median (Min-Max)
<b>Age (years)</b>		73±6.2	72 (65-92)
<b>Sex</b>			
Female	35 (33.3)		
Male	70 (66.7)		
<b>Comorbidities, n (%)</b>	37 (37.4)		
<b>Smoking status</b>			
Smoker	32 (30.5)		
Non-smoker	50 (47.6)		
Ex-smoker	23 (21.9)		
<b>Laboratory parameters</b>			
Fasting blood glucose (mg/dL)		129.5±57.3	113 (69-357)
BUN (mg/dL)		36.6±17.5	33 (14-141)
CR (mg/dL)		1±0.6	0.9 (0.3-5.6)
ALT ( IU/L)		28.5±20.3	22 (7-110)
AST ( IU/L)		35.4±26.4	27 (11-177)
Procalcitonin (ng/mL)		0.3±0.6	0.1 (0-3.8)
CRP (mg/dL)		49.5±59.2	23.4 (0.9-303)
WBC (× 10 <sup>9</sup> /L)		9.6±3.3	9.1 (3.4-19.9)
Neutrophil (× 10 <sup>9</sup> /L)		7.4±3.2	6.9 (1.9-18.9)
Lymphocyte (× 10 <sup>9</sup> /L)		1.4±0.7	1.3 (0.3-3.6)
Platelet (× 10 <sup>9</sup> /L)		222±95.7	197 (61-586)
Ferritin (µg/L)		287.3±281.3	189 (14-1983)
Monocyte (× 10 <sup>9</sup> /L)		0.6±0.3	0.5 (0.1-2.8)
Albumin (g/dL)		34.4±8.5	36.6 (18.9-54.9)
<b>Biomarkers</b>			
NLR		7.3±6.4	4.8 (1.1-33.7)
PLR		191.7±130.7	155 (58.2-857.1)
LMR		3.2±2.1	2.8 (0.4-10.9)
SII		1574±1875.8	1199.3 (143.5-16165.7)
PNI		41.3±10	43.9 (23.3-68.6)
CAR		1.7±2.2	0.7 (0-11.5)
<b>CURB-65</b>			
Low risk	56 (53.3)		
Moderate risk	49 (46.7)		
PSI			
Class II (Low)	57 (54.3)		
Class III (Moderate)	19 (18.1)		
Class IV (High)	4 (3.8)		
Class V (Very high)	25 (23.8)		
<b>mGPS</b>			
Systemic inflammation-none	30 (28.6)		
Moderately severe inflammation	30 (28.6)		
Marked inflammation	45 (42.9)		
Hospital stay (days)		16.7±5.6	15 (9-30)
<b>Patient's current medical condition</b>			
Survived	80 (76.2)		
Exited	25 (23.8)		

ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; BUN: Blood urea nitrogen; CAR: C-reactive protein-to-albumin ratio; CR: Creatinine; CRP: C-reactive protein; CURB-65 severity index: (confusion, uremia, respiratory rate, BP, age ≥ 65 years); LMR: lymphocyte-to-monocyte ratio; mGPS: modified Glasgow Prognostic Score; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; PNI: Prognostic Nutritional Index; PSI: Pneumonia severity index; SII: Systemic Immune-Inflammation Index; WBC: White blood cell



**Table 2.** Comparison of demographic and clinical characteristics between survival and non-survival patients

Variables (N=105)	Survived (n=80)	Non-survived (n=25)	p-value
	n (%) or Median (Min-Max)	n (%) or Median (Min-Max)	
Age (years)	72 (65-92)	72 (65-89)	0.431
<b>Sex</b>			1.000
Female	27 (33,8)	8 (32)	
Male	53 (66,3)	17 (68)	
<b>Presence of concomitant disease(s)</b>	18 (24,3)	19 (76)	<0.001
<b>Smoking status</b>			0.612
Smoker	24 (30)	8 (32)	
Non-smoker	40 (50)	10 (40)	
Quitter	16 (20)	7 (28)	
<b>CURB-65</b>			<0.001
Low risk	56 (70)	0 (0)	
Moderate risk	24 (30)	25 (100)	
<b>PSI</b>			<0.001
Class II (Low)	57 (71,3)	0 (0)	
Class III (Moderate)	19 (23,8)	0 (0)	
Class IV (High)	4 (5)	0 (0)	
Class V (Very high)	0 (0)	25 (100)	
<b>mGPS</b>			<0.001
Systemic inflammation-none	30 (37,5)	0 (0)	
Moderately severe inflammation	27 (33,8)	3 (12)	
Marked inflammation	23 (28,8)	22 (88)	
Hospital stay (days)	13 (9-24)	25 (18-30)	<0.001

CURB-65 severity index: (confusion, uremia, respiratory rate, BP, age  $\geq$  65 years); mGPS:modified Glasgow Prognostic Score; PSI: Pneumonia severity index

**Table 3.** Distribution of Patients' Biomarkers and Laboratory Parameters according to survival and non-survival patients

Variables (N=105)	Survived (n=80)	Exitus (n=25)	p-value
	Median (Min-Max)	Median (Min-Max)	
<b>Laboratory parameters</b>			
Fasting blood glucose, ( mg/dL)	108.5 (73-357)	126 (69-336)	0.007
BUN(mg/dL)	33 (15-141)	30 (14-55)	0.535
CR (mg/dL)	0.9 (0.3-2.8)	0.9 (0.5-5.6)	0.795
ALT ( IU/L)	19.5 (7-110)	29 (10-92)	0.006
AST (IU/L)	25.5 (11-163)	35 (15-177)	0.016
Procalcitonin (ng/mL)	0 (0-3.8)	0.2 (0-3.8)	<0.001
CRP (mg/dL)	14.2 (0.9-128)	92 (53-303)	<0.001
WBC ( $\times 10^9/L$ )	8.8 (3.4-19.6)	11.5 (5.7-19.9)	<0.001
Neutrophil ( $\times 10^9/L$ )	6.2 (1.9-18.9)	9.1 (4.1-16.7)	<0.001
Lymphocyte ( $\times 10^9/L$ )	1.4 (0.4-3.6)	0.9 (0.3-2.2)	0.002
Platelet ( $\times 10^9/L$ )	216 (61-586)	179 (101-425)	0.172
Ferritin $\mu g/L$	155.5 (14-1983)	367 (17-937)	0.021
Monocyte ( $\times 10^9/L$ )	0.5 (0.1-2.8)	0.5 (0.1-1.2)	0.721
Albumin (g/dL)	38.1 (21.2-54.9)	23.7 (18.9-47)	<0.001
<b>Biomarkers</b>			
NLR	4 (1.1-33.7)	10.2 (2.2-22.8)	<0.001
PLR	146.8 (58.2-857.1)	177 (73.9-652.3)	0.031
LMR	3 (0.4-10.5)	1.7 (0.7-10.9)	0.015
SII	950.4 (143.5-16165.7)	1784.5 (302.4-7000.4)	<0.001
PNI	45.3 (26.4-68.6)	28.1 (23.3-52.6)	<0.001
CAR	0.4 (0-4.8)	4 (2-11.5)	<0.001

ALT:Alanine aminotransferase; AST: Aspartate aminotransferase; BUN: Blood urea nitrogen; CAR: C-reactive protein-to-albumin ratio; CR: Creatinine; CRP: C-reactive protein;LMR: lymphocyte-to-monocyte ratio; mGPS:modified Glasgow Prognostic Score; NLR:Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; PNI:Prognostic Nutritional Index; PSI: Pneumonia severity index; SII:Systemic Immune-Inflammation Index; WBC: White blood cell

concentrations, indicating greater inflammatory burden and diminished nutritional reserve. NLR, PLR, LMR, SII, PNI, and CAR were all associated with mortality ( $P<0.05$ ).

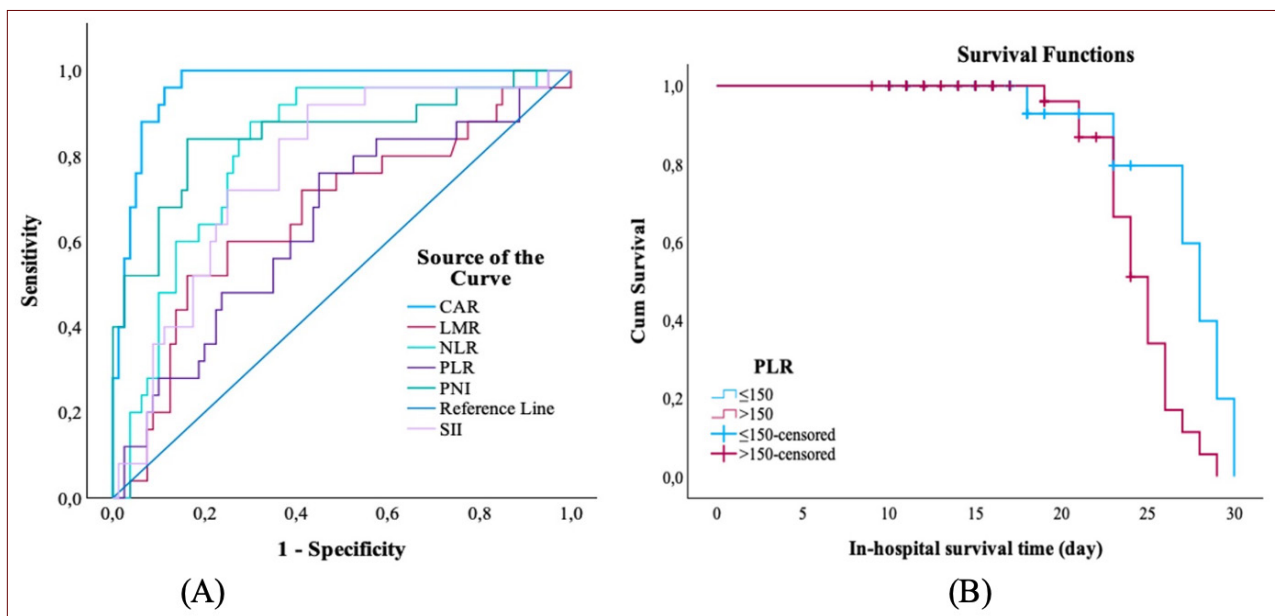
ROC curve analysis demonstrated that NLR, SII, and CAR had meaningful discriminative power for predicting in-hospital mortality (Table 4). CAR showed the highest accuracy rates with 100% sensitivity and 85% specificity. PLR and LMR

yielded more limited predictive performance. Kaplan–Meier analyses showed shorter in-hospital survival among patients with elevated NLR, PLR, SII, and CAR values. High PLR was associated with significantly reduced survival ( $24.5\pm 0.5$  vs.  $27.0\pm 1.2$  days,  $P=0.038$ ). No deaths occurred in the low-CAR group. Though not statistically significant survival was shorter in patients with low LMR and PNI (Figure 1).

**Table 4.** Results of ROC Analysis of Laboratory Parameters Predicting Mortality

Risk factors	AUC (95% CI)	Cut-off value	p-value	Sensitivity (%)	Specificity (%)
NLR	0.813 (0.720-0.905)	>5.2	<0.001	88.0	70.0
PLR	0.643 (0.518-0.768)	>150	0.025	76.0	55.0
LMR	0.662 (0.534-0.790)	$\leq 1.7$	0.013	52.0	83.8
SII	0.767 (0.666-0.868)	>997.7	<0.001	92.0	57.5
PNI	0.851 (0.751-0.951)	$\leq 36.1$	<0.001	84.0	83.8
CAR	0.964 (0.933-0.995)	>1.9	<0.001	100.0	85.0

CAR: C-reactive protein-to-albumin ratio; LMR: lymphocyte-to-monocyte ratio; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; PNI: Prognostic Nutritional Index; ROC: Receiver operating characteristic curve analysis; SII: Systemic Immune-Inflammation Index



**Figure 1.** A) Results of ROC Analysis of Laboratory Parameters Predicting Mortality. B) In-hospital survival (Kaplan–Meier) curve according to groups with high and low PLR indices

CAR: C-reactive protein-to-albumin ratio; LMR: lymphocyte-to-monocyte ratio; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; PNI: Prognostic Nutritional Index; SII: Systemic Immune-Inflammation Index



## DISCUSSION

CAP is a major cause of morbidity and mortality in older adults, owing to age-related physiological decline, multimorbidity, and immunosenescence. In this geriatric cohort, we demonstrated that traditional clinical severity scores (CURB-65 and PSI) and inflammation-nutrition-based biomarkers were strongly associated with in-hospital mortality. These findings reinforce the multifactorial determinants of prognosis in elderly CAP and highlight the need for risk-stratification tools that capture infectious burden, host resilience, and systemic inflammatory imbalance (7).

A central finding of our study is the superior prognostic accuracy of the CAR, which outperformed all other biomarkers and displayed greater discriminative ability than the PSI score. This observation aligns with the increasing evidence supporting CAR as a sensitive marker reflecting the dual impact of acute inflammation and nutritional compromise on prognosis of pneumonia (8). Because CRP rises rapidly in response to systemic inflammation while albumin declines with inflammatory stress and reduced physiologic reserve, CAR integrates both dimensions into a single metric, making it biologically plausible and clinically practical for early mortality prediction in vulnerable older adults (9).

The prognostic utility of NLR has been widely documented in CAP. Previous studies reported that high NLR values strongly correlate with short-term mortality and may perform comparably, or in some cases, superior, to conventional scoring systems (10-12). In a study focusing on elderly patients with CAP, NLR demonstrated a clear dose-response relationship, with mortality rates increasing sharply beyond a threshold value (11). Additional large-scale cohort data support a non-linear association, showing that mortality risk rises steeply with increasing NLR values, whereas incorporating NLR into PSI and CURB-65 improves model performance modestly (12). In this study, NLR showed significant

univariate predictive capacity; however, its effect was attenuated after adjustment for other biomarkers and clinical severity indices. This pattern mirrors recent literature data suggesting that although NLR is a sensitive indicator of systemic inflammation, its independent predictive power may diminish in biomarker-rich multivariate models (13).

Similarly, the SII, incorporating neutrophil, lymphocyte, and platelet counts, is a promising prognostic marker in pneumonia and other infectious diseases. Elevated SII values reflect amplified inflammatory responses and are associated with increased mortality, prolonged hospitalization, and ICU admission (14). In severe CAP, an "SII-PNI score," was shown to independently predict 28-day mortality, highlighting the synergistic prognostic value of inflammatory intensity and nutritional reserve (15). Although SII demonstrated significant associations with mortality in our univariate analyses, it did not remain independently predictive in multivariate modeling, possibly owing to the stronger performance of CAR, which captures similar pathophysiological pathways more robustly.

The mGPS, another inflammation-nutrition-based index, showed strong prognostic value in this elderly population. The performance of mGPS was comparable to that of the PSI, despite requiring fewer parameters and relying only on CRP and albumin levels. Previous research supports the prognostic value of mGPS in respiratory infections and inflammatory states, suggesting its potential as a practical alternative if complex scoring systems may not be feasible (16). The similar predictive ability of mGPS and PSI in our elderly cohort further underscores the importance of inflammatory-nutritional status in determining outcomes in CAP.

Furthermore, recent studies have examined combined indices to enhance prognostic precision. Evidence suggests that pairing inflammation-based scores with radiological severity assessments, nutritional markers, or comorbidity indices improves early identification of high-risk patients (17). For

instance, integrating computed tomography-based severity metrics with fibrinogen-albumin ratios or inflammation-nutrition indices has shown added value in predicting mortality and need for intensive care (18). Our findings complement this line of research by demonstrating that CAR, a simple and readily accessible biomarker, can offer prognostic performance equal to or greater than more complex multifactorial tools, especially in elderly populations in which reliability, speed, and practicality are essential.

Parameters used in this study including NLR, PLR, and SII across the broader spectrum of infectious disease, particularly in cases of pneumosepsis had firmly correlated with disease severity and mortality reflecting the hyperinflammatory state characteristic of advanced infection (19). Interestingly, our cohort comprised elderly patients with CAP without advanced sepsis; however, inflammation-related biomarkers still showed robust predictive value. This finding suggests that these indices may detect early inflammatory trajectories that precede clinical deterioration, providing an opportunity for timely intervention in older adults (20).

### Limitations

The study has several key limitations, primarily its retrospective design and the small sample size (n=105). These limitations restrict the generalizability of our findings. Further validation is warranted to enable the integration of these results into clinical guidelines and confirm the robustness of the prognostic superiority of CAR scores. Such studies are essential to solidify the prognostic value of CAR score as a definitive and widely accepted tool for accurate risk prediction in this population.

### CONCLUSION

In this geriatric cohort with CAP, particularly CAR demonstrated strong prognostic value and outperformed several widely used hematologic indices and traditional severity scores. CAR showed

superior discriminative accuracy and exhibited prognostic performance comparable to or greater than the PSI score, underscoring its clinical relevance as a simple, reliable, and readily accessible indicator of early risk of mortality. Along with the mGPS, CAR highlights the central role of systemic inflammation and nutritional reserve in determining short-term outcomes in older adults with CAP. Incorporating these biomarkers alongside established scoring systems may enhance early risk stratification, support timely therapeutic decision-making, and improve clinical management in vulnerable geriatric populations. Future multicenter prospective studies are warranted to validate these findings and explore biomarker-integrated prognostic models specifically tailored to the physiological characteristics of elderly patients.

**Statement of ethics:** This study was approved by the Şişli Hamidiye Etfal Training and Research Hospital, Istanbul, Türkiye (Approval No: 3193/28.10.2025).

**Conflict of interest statement:** The authors declare no competing interests.

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