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Incremental Value of Systemic Inflammatory Indices and Radiographic Features for Predicting Prolonged Hospitalization in Children with Atypical Pneumonia Determined by *Mycoplasma pneumoniae*

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Highlights

What are the main findings?

- Among routinely available inflammatory biomarkers, the platelet-to-lymphocyte ratio (PLR) showed the most consistent association with prolonged hospitalization in children with *Mycoplasma pneumoniae* pneumonia.
- Integrating clinical, inflammatory, and radiographic information modestly improved prediction of hospitalization longer than seven days, although the increase in discrimination was not statistically significant.

What are the implication of the main findings?

- Inflammatory indices may complement routine clinical assessment, but their limited standalone predictive performance does not support using them as independent decision-making tools.
- Multimodal risk assessment may help identify children requiring prolonged inpatient care, but prospective external validation is necessary before clinical implementation.

Abstract

Background/Objectives: Risk stratification in pediatric atypical pneumonia remains clinically challenging. Severity indices for pneumonia in adults are inadequately applicable to pediatric patients, and specific inflammatory biomarkers frequently exhibit restricted discriminatory efficacy. The aim of this study was to evaluate whether systemic inflammatory indices add prognostic information beyond clinical assessment for predicting hospitalization longer than 7 days in children with atypical pneumonia, and to determine whether radiographic features further improve prediction. Methods: Of 185 initially identified children, 159 with complete clinical, inflammatory, and radiographic data were included in the main analyses. Four predetermined hierarchical logistic regression models were evaluated: clinical, inflammatory, clinical combined with inflammatory, and clinical combined with inflammatory plus radiological factors. Performance was evaluated using AUROC, precision–recall AUC, Brier score, calibration, Youden-derived diagnostic indices, and decision-curve analysis. Results: Prolonged hospitalization occurred in 98/159 children

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(61.6%). Higher platelet-to-lymphocyte ratio (PLR), systemic immune-inflammation index (SII), and C-reactive protein (CRP) were associated with the outcome in univariable analyses. Following clinical and radiographic adjustment, PLR (OR 1.71, 95% CI 1.12–2.60; false-discovery-rate, FDR-adjusted $p = 0.037$) and SII (OR 1.55, 95% CI 1.04–2.30; FDR-adjusted $p = 0.045$) remained associated with the outcome. The full model achieved the highest area under the receiver operating characteristic curve, AUROC (0.592, 95% CI 0.510–0.674), precision–recall AUC (0.681), and the lowest Brier score (0.234). Conclusions: Inflammatory indices provided modest incremental prognostic value beyond clinical assessment. Radiographic variables were best handled through a separate screening step, with selective incorporation into predictive modeling.

Keywords: atypical pneumonia; pediatric population; inflammatory markers; prolonged hospitalization

1. Introduction

Community-acquired pneumonia (CAP) remains one of the leading causes of pediatric morbidity and hospitalization worldwide despite major advances in vaccination, antimicrobial therapy, and supportive care. Although mortality has declined substantially over recent decades, CAP continues to impose a considerable healthcare burden through frequent hospital admissions, prolonged caregiver burden, and increased healthcare resource utilization [1,2].

Within the spectrum of pediatric CAP, atypical pneumonia, predominantly caused by *Mycoplasma pneumoniae*, is characterized by a highly variable clinical course. While most children experience a self-limiting illness, a subset develops persistent fever, progressive pulmonary involvement, respiratory compromise, or prolonged hospitalization despite appropriate antimicrobial therapy. The increasing prevalence of macrolide-resistant *M. pneumoniae* and recurrent epidemic outbreaks further emphasize the need for early identification of children at risk of a complicated clinical course [3,4].

Early risk stratification remains challenging because clinical manifestations, conventional inflammatory markers, and chest radiography only incompletely reflect disease severity. Respiratory symptoms often overlap between mild and severe disease, radiographic abnormalities have limited prognostic specificity, and C-reactive protein (CRP) alone demonstrates only modest discriminatory performance for adverse outcomes [5,6].

Among clinically relevant outcomes, length of hospital stay (LOS) has emerged as a pragmatic indicator of disease severity, treatment response, and healthcare utilization. Unlike mortality or intensive care admission, which are relatively uncommon in contemporary pediatric cohorts, prolonged hospitalization is sufficiently frequent to permit meaningful prognostic investigation while capturing both clinical recovery and resource consumption [1,2].

Increasing evidence suggests that disease progression in atypical pneumonia depends not only on pathogen characteristics but also on the magnitude of the host inflammatory response. Thus, composite inflammatory biomarkers derived from routine complete blood counts have attracted growing interest because they integrate multiple components of systemic inflammation while remaining inexpensive, rapidly available, and universally accessible in routine clinical practice [7,8].

Among the inflammatory biomarkers investigated in pediatric respiratory infections, hemogram-derived inflammatory biomarkers have gained increasing attention because they integrate multiple components of the host immune response into inexpensive, reproducible, and readily available composite indices [9]. Unlike conventional inflammatory

markers, these biomarkers simultaneously reflect innate immune activation, adaptive immune regulation, and platelet-mediated inflammatory responses, thereby providing a more comprehensive assessment of systemic inflammation [7,8].

The most widely investigated indices include [10,11]: the neutrophil-to-lymphocyte ratio (NLR), reflecting the balance between neutrophilia and relative lymphopenia; the platelet-to-lymphocyte ratio (PLR), integrating platelet activation with lymphocyte depletion; the systemic immune-inflammation index (SII), combining platelet, neutrophil, and lymphocyte counts; and more recently, composite markers incorporating acute-phase proteins, such as the C-reactive protein-to-lymphocyte ratio (CLR) and the CRP-to-NLR ratio. These biomarkers provide complementary information regarding the magnitude and regulation of the systemic inflammatory response and may better characterize disease severity than isolated laboratory parameters [7,8].

Growing clinical evidence supports the prognostic value of these biomarkers in pediatric pneumonia. Florin et al. demonstrated that although CRP was independently associated with moderate-to-severe community-acquired pneumonia, its discriminatory performance remained modest (AUROC 0.53–0.64), suggesting that CRP is better interpreted within multivariable prediction models than as a standalone biomarker [5]. More recently, Hong et al. reported that PLR improved prediction of refractory *Mycoplasma pneumoniae* pneumonia when incorporated into multivariable clinical models [4]. Similarly, Wang et al. and Guo et al. demonstrated high prognostic performance of SII for severe *Mycoplasma pneumoniae* pneumonia (AUROC 0.88–0.94) [12,13]. However, these studies primarily investigated severe or refractory disease rather than prolonged hospitalization, limiting the generalizability of their findings to broader cohorts of hospitalized children.

Differences in study populations, clinical endpoints, biomarker thresholds, and analytical methods limit comparisons across studies, while most investigations have evaluated inflammatory biomarkers in isolation rather than within integrated prediction models. Consequently, the incremental prognostic value of hemogram-derived inflammatory biomarkers beyond routine clinical assessment remains incompletely understood, underscoring the need for comprehensive multimodal prediction models [7,14].

Despite recent advances, important knowledge gaps remain. Most studies have focused on severe pneumonia rather than prolonged hospitalization, while inflammatory biomarkers have largely been evaluated in isolation. Furthermore, few pediatric studies have examined their incremental value beyond routine clinical and radiographic assessment using comprehensive model evaluation, including discrimination, calibration, and clinical utility. Consequently, the role of routinely available inflammatory biomarkers within integrated multimodal prediction models remains poorly defined [14,15].

The aim of this study was to determine whether routinely available hemogram-derived inflammatory biomarkers improve the prediction of prolonged hospitalization in children with atypical pneumonia with *Mycoplasma pneumoniae* beyond conventional clinical assessment and chest radiographic findings by developing and evaluating hierarchical multimodal prediction models.

2. Materials and Methods

2.1. Study Design and Population

We conducted a retrospective observational study including children hospitalized with atypical pneumonia with *Mycoplasma pneumoniae* at the "Sfântul Ioan" Emergency Clinical Hospital for Children, Galați, between 2022 and 2025. Patients were eligible if they had a clinical and paraclinical diagnosis of atypical pneumonia with *Mycoplasma pneumoniae* and a documented length of hospital stay.

Mycoplasma pneumoniae infection was confirmed by real-time polymerase chain reaction (PCR) performed on respiratory specimens collected at hospital admission using the Bio-Rad CFX96 Real-Time PCR Detection System (Bio-Rad Laboratories, Hercules, CA, USA).

The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. The study protocol was reviewed and approved by the Institutional Ethics Committee of the "Sfântul Ioan" Emergency Clinical Hospital for Children, Galați (Approval No. 14123/07.08.2026).

2.2. Inclusion and Exclusion Criteria

Children were included if they were hospitalized with atypical pneumonia with *Mycoplasma pneumoniae*, had duration of hospitalization recorded, and had available clinical and/or laboratory data sufficient for at least one prespecified analysis.

Patients were excluded from the primary analysis if length of stay was unavailable and from model-specific analyses if required predictors were missing. Missing values were not imputed.

The initial cohort comprised 185 children, of whom 105 (56.8%) experienced prolonged hospitalization. Complete clinical, inflammatory, and radiographic data were available for 159 patients (98 events), who constituted the common analytical cohort for all logistic regression and hierarchical prediction analyses. Patients with incomplete data were excluded, and missing values were not imputed.

2.3. Outcomes

The primary outcome was prolonged hospitalization, defined a priori as length of stay greater than 7 days, which is the median duration for this center.

2.4. Predictors

The clinical reference model included age, sex, dyspnea, chest retractions, and wheezing. Inflammatory predictors included PLR, SII, and CRP. Continuous inflammatory variables were transformed using log1p and standardized before regression modeling.

2.5. Radiographic Feature Extraction

Radiographic features were extracted from chest-radiograph reports. Candidate features included interstitial pattern, perihilar and infrahilar involvement, bilateral and unilateral involvement, diffuse and focal pattern, consolidation, infiltrate, basal and lobar involvement, laterality, hilar prominence, pleural involvement, cardiomegaly, hyperinflation, adenopathy, and atelectasis. Features recorded as present or absent in fewer than ten patients were excluded from selection. The remaining features were ranked according to their association with prolonged hospitalization using Fisher's exact test, and the two most strongly associated variables were retained. This procedure was repeated within each cross-validation training subset to prevent information leakage.

2.6. Statistical Analysis

Continuous variables are presented as median and interquartile range (IQR), whereas categorical variables are expressed as frequencies and percentages. Group comparisons were performed using the Mann–Whitney U test for continuous variables and Pearson's χ^2 test or Fisher's exact test for categorical variables, as appropriate.

We evaluated the associations between therapeutic management and prolonged hospitalization. Inpatient systemic corticosteroid administration and macrolide exposure were assessed using Fisher's exact test and separate logistic regression models adjusted for age and sex. Antibiotic exposure was classified according to the antimicrobial agents docu-

mented in the medical records. A subgroup analysis was conducted among patients with documented antibiotic treatment before admission, who were categorized according to the recorded regimen into three groups: penicillin-class antibiotics alone, cephalosporin-class antibiotics alone, and mixed or other antimicrobial regimens. Associations with prolonged hospitalization were examined using logistic regression adjusted for age and sex, with penicillin-class antibiotics as the reference category. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated using logistic regression, and the Benjamini–Hochberg correction was applied to the two inpatient treatment comparisons.

Associations between inflammatory biomarkers and prolonged hospitalization were evaluated using logistic regression analyses. First, univariable logistic regression was performed with each inflammatory marker as the sole predictor. Second, each marker was evaluated after adjustment for age, sex, dyspnea, chest retractions, and wheezing. Third, the clinically adjusted models were extended by including two radiographic variables: right-sided and bilateral pulmonary involvement. Each inflammatory marker was entered separately rather than simultaneously with the other markers. Odds ratios (ORs) and Wald 95% confidence intervals (CIs) were expressed per one-standard-deviation increase in log1p-transformed marker values. To account for multiple comparisons, the Benjamini–Hochberg false discovery rate (FDR) correction was applied separately to the three marker-specific *p*-values obtained at each adjustment level.

To investigate the incremental prognostic value of inflammatory biomarkers and radiographic findings, four prespecified hierarchical prediction models were developed: (i) a clinical model including age, sex, dyspnea, chest retractions, and wheezing; (ii) an inflammatory model including PLR, SII, and CRP; (iii) a combined clinical plus inflammatory model; and (iv) a complete model integrating clinical, inflammatory, and two radiographic variables: right-sided and bilateral pulmonary involvement. Model performance was evaluated using repeated cross-validation with out-of-fold predictions to reduce optimism bias.

Prediction models were developed using L2-penalized logistic regression and evaluated through repeated cross-validation. The patients were randomly divided into five groups, maintaining a similar proportion of prolonged hospitalizations in each group. Models were trained on four groups and evaluated on the remaining group, ensuring that predictions were obtained for patients not used in model training. This procedure was repeated ten times. Within each training set, an additional four-fold cross-validation was performed to determine the optimal degree of penalization by minimizing logarithmic loss. Continuous variables were standardized, and radiographic predictors were selected using only the training data. The probability threshold for classification was determined by maximizing Youden's index using predictions generated during the internal cross-validation and was subsequently applied to the held-out patients. Model performance was calculated for each repetition, and the results were averaged across all ten repetitions.

Discriminative ability was assessed using the area under the receiver operating characteristic curve (AUROC) and the area under the precision–recall curve (PR-AUC). Sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were calculated using the classification thresholds determined within the training data. Model calibration was evaluated using calibration plots together with calibration intercept, calibration slope, and Brier score. Clinical utility was assessed by decision-curve analysis across clinically relevant threshold probabilities.

All statistical analyses were performed using R (R Foundation for Statistical Computing, Vienna, Austria). Statistical analyses were conducted using packages from the tidyverse ecosystem together with dedicated packages for regression modeling, predictive performance assessment, receiver operating characteristic analysis, calibration, and decision-

curve analysis. All statistical tests were two-sided, and p-values < 0.05 were considered statistically significant before adjustment for multiple testing.

3. Results

A total of 185 children hospitalized with atypical pneumonia were included in the analysis, of whom 105 (56.8%) experienced prolonged hospitalization (>7 days) (Table 1). The median age of the cohort was 9.75 years (IQR, 5.17–13.40), and 76 patients (41.1%) were male. Children with prolonged hospitalization tended to be older than those discharged within 7 days (median, 10.12 vs. 8.21 years). However, this difference did not reach statistical significance ($p = 0.093$). Also, the proportion of male patients was comparable between the two groups (37.1% vs. 46.2%; $p = 0.212$), indicating that neither age nor sex was significantly associated with prolonged hospitalization in this cohort.

Table 1. Baseline characteristics according to hospitalization duration in the entire cohort.

Characteristic	Overall	≤7 Days	>7 Days	*p*-Value
Age, years	9.75 (5.17–13.40)	8.21 (4.19–12.46)	10.12 (6.00–13.94)	0.093
Male sex	76/185 (41.1%)	37/80 (46.2%)	39/105 (37.1%)	0.212
Dyspnea	38/185 (20.5%)	16/80 (20.0%)	22/105 (21.0%)	0.874
Chest retractions	21/185 (11.4%)	6/80 (7.5%)	15/105 (14.3%)	0.149
Wheezing	11/185 (5.9%)	3/80 (3.8%)	8/105 (7.6%)	0.355
Length of stay, days	8.00 (6.00–9.00)	6.00 (5.00–7.00)	9.00 (8.00–10.00)	<0.001
CRP	2.28 (0.73–4.61)	1.73 (0.50–4.32)	2.80 (1.33–5.43)	0.016
PLR	170.28 (110.37–244.22)	146.25 (91.26–210.66)	176.05 (134.61–282.69)	0.007
SII	975.63 (546.08–1691.32)	924.25 (446.70–1454.10)	1129.33 (623.94–1898.62)	0.047
Radiographic interstitial pattern	106/159 (66.7%)	45/61 (73.8%)	61/98 (62.2%)	0.167
Radiographic consolidation	7/159 (4.4%)	2/61 (3.3%)	5/98 (5.1%)	0.708
Radiographic pleural involvement	2/159 (1.3%)	0/61 (0.0%)	2/98 (2.0%)	0.524

Legend: CRP, C-reactive protein; PLR, platelet-to-lymphocyte ratio; SII, systemic immune-inflammation index.

Baseline respiratory clinical manifestations were similarly distributed between groups. Dyspnea was present in 20.5% of the overall cohort and occurred with comparable frequency among children with prolonged and shorter hospital stays (21.0% vs. 20.0%; $p = 0.874$). Chest retractions were observed in 11.4% of patients (14.3% vs. 7.5%; $p = 0.149$), while wheezing was documented in only 5.9% of cases (7.6% vs. 3.8%; $p = 0.355$). None of these clinical variables demonstrated a statistically significant association with hospitalization duration, suggesting that routine clinical respiratory findings alone were insufficient to discriminate between children requiring prolonged inpatient care and those discharged earlier.

As expected, based on the study definition, the median length of hospital stay differed markedly between the two groups, increasing from 6.0 days (IQR, 5.0–7.0) among children hospitalized for ≤7 days to 9.0 days (IQR, 8.0–10.0) among those hospitalized for >7 days ($p < 0.001$).

On the other hand, inflammatory biomarkers differed significantly according to hospitalization duration. Median CRP concentrations were significantly higher in children with prolonged hospitalization than in those with shorter admissions (2.80 vs. 1.73 mg/dL; $p = 0.016$). Among the hematological inflammatory indices, the PLR demonstrated the strongest association, with median values increasing from 146.25 (IQR, 91.26–210.66) to 176.05 (IQR, 134.61–282.69) ($p = 0.007$). Similarly, the SII was significantly elevated in the prolonged hospitalization group (1129.33 vs. 924.25; $p = 0.047$).

Radiographic findings showed limited discriminatory value. An interstitial radiographic pattern was the most frequently observed finding, identified in 106 children (66.7%). Its prevalence was higher among patients hospitalized for ≤7 days than among those hospitalized for >7 days (73.8% vs. 62.2%), although the difference was not statistically significant ($p = 0.167$). Radiographic consolidation was uncommon, occurring in 7 patients (4.4%), with a higher frequency in the prolonged hospitalization group (5.1% vs. 3.3%; $p = 0.708$). Pleural involvement was documented in 2 patients (1.3%), both of whom required hospitalization for more than seven days (2.0% vs. 0.0%; $p = 0.524$).

The radiographic screening analysis identified two imaging characteristics significantly associated with prolonged hospitalization (Table 2). Right-sided pulmonary involvement was more frequent among children hospitalized for more than seven days than among those discharged earlier (16.3% vs. 3.3%), corresponding to an odds ratio (OR) of 4.76 ($p = 0.011$).

Table 2. Radiographic feature screen.

Radiographic Feature	Overall (N = 159)	≤7 Days (N = 61)	>7 Days (N = 98)	OR	*p*
Right-sided involvement	18 (11.3%)	2 (3.3%)	16 (16.3%)	4.76	0.011
Bilateral involvement	88 (55.3%)	44 (72.1%)	44 (44.9%)	0.32	<0.001
Left-sided involvement	13 (8.2%)	3 (4.9%)	10 (10.2%)	1.98	0.373
Infrahilar involvement	27 (17.0%)	14 (23.0%)	13 (13.3%)	0.52	0.131
Consolidation	7 (4.4%)	2 (3.3%)	5 (5.1%)	1.40	0.708
Interstitial pattern	106 (66.7%)	45 (73.8%)	61 (62.2%)	0.59	0.167
Perihilar involvement	61 (38.4%)	26 (42.6%)	35 (35.7%)	0.75	0.406
Pleural involvement	2 (1.3%)	0 (0.0%)	2 (2.0%)	3.19	0.524

Legend: OR, odds ratio.

Bilateral pulmonary involvement was identified in 88 children (55.3%) and was significantly less frequent among patients with prolonged hospitalization than among those hospitalized for seven days or less (44.9% vs. 72.1%; OR = 0.32, $p = 0.001$).

The remaining radiographic characteristics were not significantly associated with hospitalization duration. Left-sided involvement was documented in 13 patients (8.2%; OR = 1.98, $p = 0.373$), while infrahilar abnormalities were identified in 27 patients (17.0%; OR = 0.52, $p = 0.131$). Radiographic consolidation was uncommon, occurring slightly more frequently among children with prolonged hospitalization (5.1% vs. 3.3%; OR = 1.40, $p = 0.708$). An interstitial radiographic pattern was the most prevalent finding (66.7%) and was observed in 62.2% of children with prolonged hospitalization compared with 73.8% of those discharged earlier (OR = 0.59, $p = 0.167$). Perihilar involvement was documented in 61 patients (38.4%; OR = 0.75, $p = 0.406$), whereas pleural involvement was rare, occurring in only two patients (1.3%; OR = 3.19, $p = 0.524$).

We examined associations between therapeutic management and prolonged hospitalization (Table 3). Systemic corticosteroid administration was more frequent among children with prolonged hospitalization (63.3% vs. 42.6%) and remained associated with this outcome after adjustment for age and sex (OR 2.21, 95% CI 1.14–4.27; $p = 0.019$). Inpatient

macrolide administration showed no significant association (OR 0.69, 95% CI 0.35–1.35; $p = 0.273$).

Table 3. Exploratory associations between treatment-related factors and prolonged hospitalization.

Treatment Variable	N	≤7 Days, n/N (%)	>7 Days, n/N (%)	Adjusted OR (95% CI)	*p*
Inpatient treatment					
Systemic corticosteroids	159	26/61 (42.6%)	62/98 (63.3%)	2.21 (1.14–4.27)	0.019
Macrolide administration	154	36/57 (63.2%)	53/97 (54.6%)	0.69 (0.35–1.35)	0.273
Pre-admission antibiotic treatment					
Penicillin-class antibiotics only	22	10/22 (45.5%)	12/22 (54.5%)	Reference	
Cephalosporin-class antibiotics only	33	10/33 (30.3%)	23/33 (69.7%)	1.79 (0.55–5.80)	0.332
Mixed regimens or other antibiotics	10	4/10 (40.0%)	6/10 (60.0%)	1.57 (0.32–7.70)	0.578

Legend: OR, odds ratio; CI, confidence interval.

Pre-admission antibiotic treatment was documented in 65 patients, of whom 41 (63.1%) experienced prolonged hospitalization. Compared with children receiving exclusively penicillin-class antibiotics, those treated with cephalosporins had higher observed rates of prolonged hospitalization (69.7% vs. 54.5%), although the adjusted association was not statistically significant (OR 1.79, 95% CI 0.55–5.80; $p = 0.332$). Similarly, mixed or other antibiotic regimens were not significantly associated with prolonged hospitalization (OR 1.57, 95% CI 0.32–7.70; $p = 0.578$).

In the common complete-case cohort of 159 children, all three inflammatory indices were associated with prolonged hospitalization in univariate logistic regression analyses (Table 4). Each standard deviation increase in log-transformed PLR, SII, and CRP was associated with higher odds of hospitalization exceeding seven days (OR 1.58, 95% CI 1.11–2.24; OR 1.45, 95% CI 1.04–2.03; and OR 1.49, 95% CI 1.05–2.11, respectively), with all associations remaining significant after FDR correction. Following adjustment for demographic and clinical characteristics, PLR retained a nominal association with prolonged hospitalization (OR 1.60, 95% CI 1.07–2.40; $p = 0.022$), although none of the inflammatory indices remained significant after FDR correction. In models additionally adjusted for right-sided and bilateral radiographic findings, PLR (OR 1.71, 95% CI 1.12–2.60; FDR-adjusted $p = 0.037$) and SII (OR 1.55, 95% CI 1.04–2.30; FDR-adjusted $p = 0.045$) were associated with higher odds of prolonged hospitalization. In contrast, the association with CRP was attenuated and was no longer statistically significant (OR 1.28, 95% CI 0.88–1.86; $p = 0.204$).

The performance of the four hierarchical prediction models was evaluated in a common complete-case cohort of 159 children, of whom 98 (61.6%) had a hospital stay exceeding 7 days (Table 5). Internal validation was performed using stratified five-fold cross-validation repeated ten times. The clinical reference model, comprising age, sex, dyspnea, chest retractions, and wheezing, showed limited discrimination, with an AUROC of 0.485 (95% CI, 0.409–0.558). Sensitivity was 59.0% and specificity was 40.0%, with a PPV of 60.6% and an NPV of 38.4%. These results indicated that baseline demographic and respiratory clinical characteristics alone had poor discriminatory ability for prolonged hospitalization.

The inflammatory model, comprising the PLR, SII, and CRP, achieved an AUROC of 0.570 (95% CI, 0.486–0.655), compared with 0.485 for the clinical reference model. Average precision increased from 0.602 to 0.659, while the Brier score decreased from 0.239 to 0.237. This model achieved the highest sensitivity among the four models (61.4%), with a specificity of 46.4%, PPV of 64.8%, and NPV of 42.9%. Although inflammatory indices showed greater discriminatory ability than the selected clinical variables, their predictive performance remained modest.

Table 4. Logistic regression analyses for inflammatory indices.

Predictor	Model	N	Events	OR	95% CI	*p*	FDR *P*
PLR, per SD log1p	Univariate	159	98	1.58	1.11–2.24	0.011	0.028
SII, per SD log1p	Univariate	159	98	1.45	1.04–2.03	0.028	0.028
CRP, per SD log1p	Univariate	159	98	1.49	1.05–2.11	0.024	0.028
PLR, per SD log1p	Adjusted clinical	159	98	1.60	1.07–2.40	0.022	0.065
SII, per SD log1p	Adjusted clinical	159	98	1.43	0.98–2.09	0.065	0.065
CRP, per SD log1p	Adjusted clinical	159	98	1.44	0.99–2.09	0.055	0.065
PLR, per SD log1p	Adjusted clinical + radiology	159	98	1.71	1.12–2.60	0.012	0.037
SII, per SD log1p	Adjusted clinical + radiology	159	98	1.55	1.04–2.30	0.030	0.045
CRP, per SD log1p	Adjusted clinical + radiology	159	98	1.28	0.88–1.86	0.204	0.204

Legend: CI, confidence interval; CRP, C-reactive protein; FDR, false discovery rate; OR, odds ratio; PLR, platelet-to-lymphocyte ratio; SD, standard deviation; SII, systemic immune-inflammation index.

Table 5. Hierarchical model performance.

Model	N	Events	AUROC	95% CI	PR-AUC (AP)	Brier	Sens.	Spec.	PPV	NPV
Clinical	159	98	0.485	0.409–0.558	0.602	0.239	59.0%	40.0%	60.6%	38.4%
Inflammatory	159	98	0.570	0.486–0.655	0.659	0.237	61.4%	46.4%	64.8%	42.9%
Clinical + inflammatory	159	98	0.553	0.468–0.640	0.648	0.238	55.8%	49.7%	64.2%	41.2%
Clinical + inflammatory + radiology	159	98	0.592	0.510–0.674	0.681	0.234	57.2%	55.9%	67.7%	44.9%

Legend: AUROC, area under the receiver operating characteristic curve; CI, confidence interval; NPV, negative predictive value; PPV, positive predictive value; AP, average precision.

The combination of clinical and inflammatory predictors did not improve performance compared with the inflammatory model alone. The clinical plus inflammatory model yielded an AUROC of 0.553 (95% CI, 0.468–0.640), an AP of 0.648, and a Brier score of 0.238. Sensitivity was 55.8%, specificity was 49.7%, PPV was 64.2%, and NPV was 41.2%.

The addition of radiographic characteristics to the clinical plus inflammatory model resulted in the highest discrimination and lowest prediction error among the evaluated models. The full model achieved an AUROC of 0.592 (95% CI, 0.510–0.674), an AP of 0.681, and a Brier score of 0.234. Sensitivity and specificity were 57.2% and 55.9%, respectively, with a PPV of 67.7% and an NPV of 44.9%.

Radiographic feature selection was performed within the training folds to prevent information leakage. Bilateral involvement was selected in all 50 outer training folds, while right-sided involvement was selected in 42 folds (84.0%). Both variables were retained in the final model fitted to the complete-case cohort.

Compared with the clinical plus inflammatory model, the inclusion of radiographic information increased AUROC by 0.039 (conditional bootstrap 95% CI, −0.0004 to 0.0789). However, the confidence interval included zero, indicating uncertainty regarding the incremental discriminatory contribution of radiographic findings.

The receiver-operating-characteristic curves demonstrated a progressive, although modest, improvement in discrimination as inflammatory and radiographic information was added to the baseline clinical model (Figure 1).

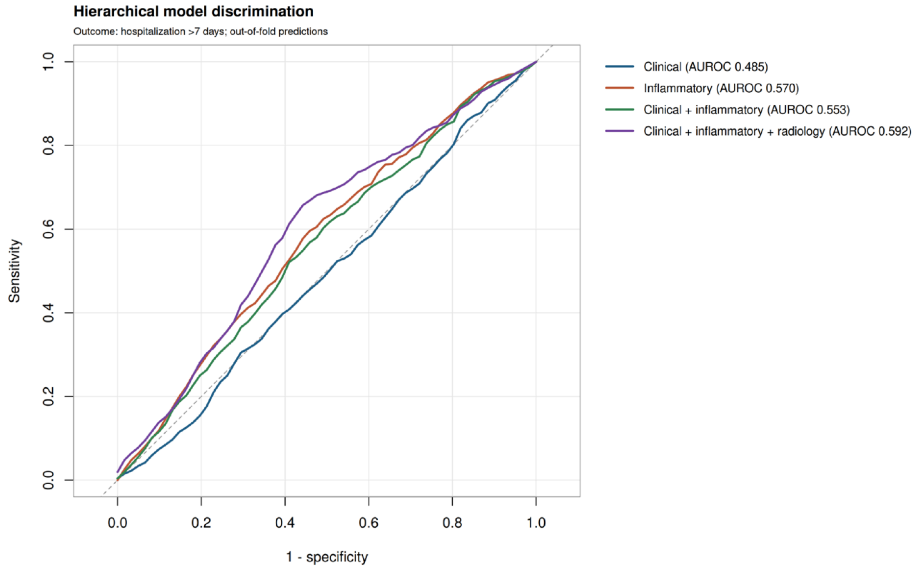


Figure 1. Hierarchical model discrimination.

Calibration performance was assessed using out-of-fold predictions from the ten repetitions of stratified five-fold cross-validation (Table 6). The observed proportion of prolonged hospitalizations was 61.6%, while mean predicted probabilities ranged from 61.6% to 61.8% across the four models. Calibration-in-the-large intercepts were close to zero (0.0006, −0.0076, −0.0054, and −0.0058 for the clinical, inflammatory, clinical plus inflammatory, and full models, respectively), indicating little systematic difference between the overall predicted and observed event frequencies. However, calibration slopes deviated substantially from the ideal value of 1.

Table 6. Calibration performance.

Model	N	Events	Observed Risk	Mean Predicted Risk	Brier Score	Intercept	Slope
Clinical	159	98	0.616	0.616	0.239	0.001	−0.879
Inflammatory	159	98	0.616	0.618	0.237	−0.008	0.320
Clinical + Inflammatory	159	98	0.616	0.618	0.238	−0.005	0.466
Full Model (+Radiology)	159	98	0.616	0.618	0.234	−0.006	0.561

The clinical model had a negative mean slope (−0.879), with considerable variation across cross-validation repetitions (SD, 1.000). Mean slopes were 0.320 for the inflammatory model, 0.466 for the clinical plus inflammatory model, and 0.561 for the full model, indicating persistent miscalibration despite the inclusion of additional predictors.

The full model had the lowest Brier score (0.2345), compared with 0.2387, 0.2366, and 0.2376 for the clinical, inflammatory, and combined models, respectively. However, its Brier score was only marginally lower than that of a prevalence-only prediction model (0.2365). Thus, although the full model showed the lowest overall prediction error, calibration remained suboptimal, limiting the reliability of individual risk estimates.

The calibration plots showed deviations from the ideal calibration line across the four hierarchical models (Figure 2). The clinical model exhibited particularly unstable calibration, with a negative mean calibration slope (−0.879). The inflammatory and clinical plus inflammatory models also showed substantial departures from ideal calibration, with mean

slopes of 0.320 and 0.466, respectively. The full model had the highest mean calibration slope (0.561) and the lowest Brier score (0.234), although its calibration remained suboptimal.

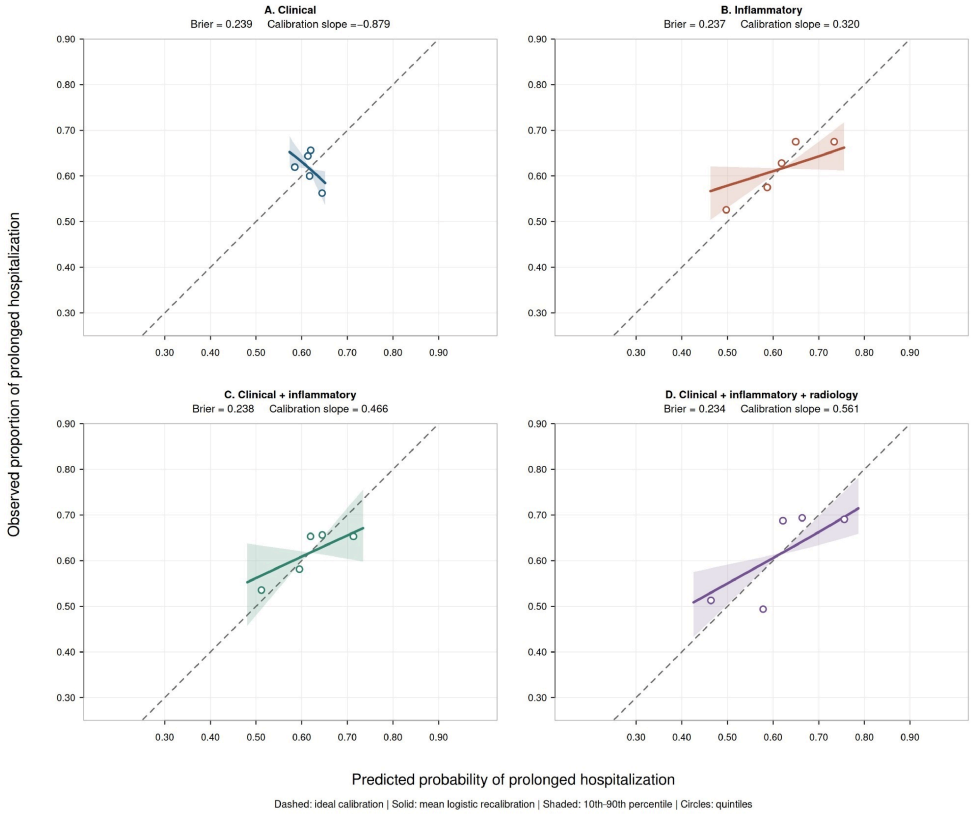


Figure 2. Calibration of hierarchical models. (**A**) Clinical, (**B**) Inflammatory, (**C**) Clinical + inflammatory, (**D**) Clinical + inflammatory + radiology.

Decision curve analysis demonstrated variation in net benefit across threshold probabilities (Table 7 and Figure 3). At lower thresholds (0.40–0.45), the treat-all strategy provided the highest net benefit, with performance nearly identical to that of the clinical model. At a threshold of 0.50, the clinical plus inflammatory model achieved the highest net benefit (0.2384), whereas the inflammatory model performed best at 0.55 (0.1711).

Table 7. Decision-curve analysis: net benefit across risk thresholds.

Threshold Probability	Treat None	Treat All	Clinical	Inflammatory	Clinical + Inflammatory	Full Model
0.40	0.0000	0.3606	0.3606	0.3564	0.3549	0.3541
0.45	0.0000	0.3025	0.3025	0.2989	0.2977	0.2979
0.50	0.0000	0.2327	0.2296	0.2377	0.2384	0.2340
0.55	0.0000	0.1475	0.1453	0.1711	0.1649	0.1551
0.60	0.0000	0.0409	0.0305	0.0890	0.0635	0.1230
0.65	0.0000	−0.0961	−0.0268	0.0174	−0.0021	0.0300
0.70	0.0000	−0.2788	−0.0088	−0.0187	−0.0258	−0.0092

The full model showed the greatest net benefit at thresholds of 0.60 and 0.65, reaching 0.1230 and 0.0300, respectively. At these thresholds, the incremental net benefit over the clinical plus inflammatory model was 0.0594 and 0.0321, respectively. The full model also exceeded both reference strategies at these thresholds. At a threshold of 0.70, all four prediction models yielded negative net benefit, favoring the treat-none strategy. The addition of radiographic features was associated with higher net benefit at selected thresholds,

particularly between 0.60 and 0.65, but did not provide a consistent advantage across the evaluated range.

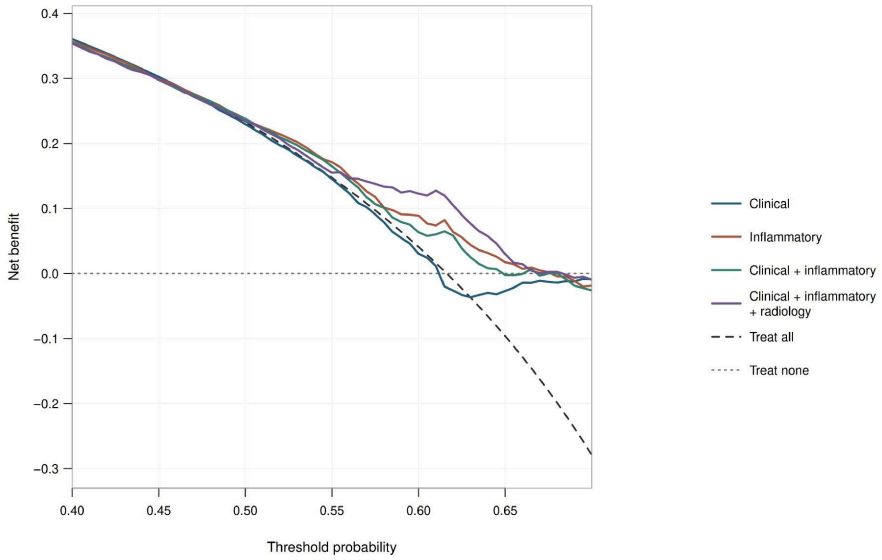


Figure 3. Decision curve analysis.

Figure 3 suggests that the combined models may offer limited but potentially meaningful clinical utility across an intermediate range of threshold probabilities, particularly approximately 0.60–0.65. No single model consistently dominated across the entire threshold range, although models incorporating inflammatory biomarkers generally performed more favorably than the clinical model alone.

4. Discussion

The present study investigated the association of routinely available inflammatory biomarkers and radiographic characteristics with prolonged hospitalization in children admitted with atypical pneumonia. Our study results indicated that PLR, SII, and CRP were associated with hospitalization exceeding seven days in univariable analyses. After adjustment for clinical and radiographic characteristics, PLR and SII retained statistically significant associations following correction for multiple comparisons, whereas CRP did not. The prediction models showed modest discriminatory performance, with the highest AUROC obtained when clinical, inflammatory, and radiographic information were combined. However, the incremental improvement associated with radiographic findings was limited, and model calibration remained suboptimal.

One of the most clinically relevant observations of the present study is the consistent relationship between admission inflammatory activity and prolonged hospitalization. The association between systemic inflammatory activity and prolonged hospitalization was most consistent for PLR and SII. Following adjustment for baseline clinical characteristics, PLR remained nominally associated with prolonged hospitalization, although the association did not retain statistical significance after FDR correction. Further adjustment for radiographic findings indicated significant associations for both PLR and SII. These findings suggest that platelet- and leukocyte-derived inflammatory indices capture aspects of the host inflammatory response associated with hospitalization duration.

These findings are biologically plausible and are increasingly supported by recent literature. PLR combines two complementary components of the host inflammatory response: reactive thrombocytosis, reflecting platelet activation and cytokine-mediated inflammation, and relative lymphopenia, reflecting stress-induced immune dysregulation and redistri-

bution of lymphocyte populations [16]. Unlike isolated leukocyte or platelet counts, PLR therefore integrates both inflammatory activation and adaptive immune suppression into a single composite marker. These biological characteristics may contribute to the observed association between PLR and prolonged hospitalization, although its individual predictive value remains limited.

Our results closely agree with those reported by Han et al., who evaluated 303 children hospitalized with community-acquired pneumonia and demonstrated significantly higher PLR values among patients with severe disease [17]. In that study, PLR remained an independent predictor of severe pneumonia in multivariable logistic regression despite only moderate discriminatory performance (AUC 0.721). The authors also concluded that PLR should not be interpreted as a standalone prognostic biomarker but rather as one component of comprehensive clinical assessment. This interpretation closely parallels our findings. In our study, the individual predictive performance of PLR remained modest, emphasizing that inflammatory biomarkers complement rather than replace routine clinical evaluation.

In contrast to PLR, the literature surrounding SII has expanded rapidly during the last two years and generally reports stronger prognostic performance than observed in our cohort. SII incorporates neutrophil, platelet, and lymphocyte counts simultaneously, thereby reflecting interactions between innate immune activation, platelet-mediated inflammatory amplification, and adaptive immune suppression. Guo et al. demonstrated that SII independently predicted severe *Mycoplasma pneumoniae* pneumonia in hospitalized children, achieving an AUC of 0.883 with 69.9% sensitivity and 88.1% specificity [12], while Wang et al. reported an even higher AUC of 0.940 for predicting severe *Mycoplasma pneumoniae* pneumonia in hospitalized children [13]. These performances exceed the discrimination observed in our study.

Several factors likely explain this apparent discrepancy. First, our primary endpoint was prolonged hospitalization, whereas both Guo and Wang investigated disease severity. Although related, these outcomes are not interchangeable. Disease severity primarily reflects biological aggressiveness during the acute phase of infection, whereas length of stay is additionally influenced by response to treatment, institutional discharge practices, social circumstances, physician decision-making, and recovery kinetics. Consequently, biomarkers that accurately identify severe pneumonia may exhibit considerably lower discrimination when predicting hospitalization duration. Second, our cohort included children with atypical pneumonia managed in routine clinical practice rather than exclusively severe *Mycoplasma pneumoniae* pneumonia, increasing biological heterogeneity and reducing the discriminative ability of any single inflammatory marker. Therefore, the lower AUROC values observed in our analyses should not necessarily be interpreted as contradictory to previous studies but rather as reflecting a fundamentally different clinical outcome.

In our study, CRP concentrations were significantly higher among children requiring prolonged hospitalization, and CRP remained associated with the outcome in univariable regression analyses. However, its independent contribution diminished after adjustment for clinical and radiographic variables. This observation is consistent with previous prospective pediatric evidence. In a multicenter cohort of 477 children with community-acquired pneumonia, Florin et al. demonstrated that CRP alone had limited ability to discriminate severe from non-severe disease, with AUROC values generally ranging between 0.53 and 0.64 despite statistically significant associations with moderate-to-severe illness after adjustment [5]. Similarly, our results indicate that CRP reflects inflammatory burden but does not adequately capture the complexity of disease progression when considered in isolation. Rather than functioning as a definitive prognostic biomarker, CRP appears to contribute incremental information when interpreted alongside clinical assessment and other inflammatory indices.

The biological mechanisms underlying these findings have been discussed extensively in recent literature. Elevated CRP reflects hepatic synthesis stimulated predominantly by interleukin-6, representing the magnitude of the acute-phase response. However, CRP primarily quantifies systemic inflammation without directly reflecting immune competence, endothelial activation, or thrombo-inflammatory interactions. Conversely, PLR and SII incorporate cellular immune responses that more comprehensively characterize host–pathogen interactions.

Islam et al. emphasized that platelet activation contributes directly to endothelial injury and cytokine amplification, while neutrophil–platelet aggregates promote inflammatory tissue damage [8]. Simultaneously, stress-induced lymphopenia reflects impaired adaptive immune regulation [18]. Consequently, composite indices such as PLR and SII may provide broader biological information than CRP alone, explaining their greater consistency across prognostic analyses.

The interpretation of hematological inflammatory indices in children should also consider physiological age-related changes in leukocyte and platelet distributions. Recent pediatric reference data have demonstrated age-dependent variation in PLR, SII, NLR, and LMR, emphasizing the limitations of applying uniform reference values across childhood [19]. Although age was included in the clinically adjusted analyses, the absence of locally validated age-specific reference intervals remains relevant when interpreting the absolute values of inflammatory indices in our cohort.

Another important aspect emerging from both our results and the contemporary literature is that none of the evaluated inflammatory biomarkers demonstrated sufficiently high discriminatory performance to justify independent clinical use. This finding should not be interpreted as evidence against their clinical value. On the contrary, recent systematic reviews consistently emphasize that inflammatory biomarkers perform best as components of integrated prognostic models rather than isolated predictors.

In a systematic review evaluating biomarkers in atypical pneumonia, Sahoo and Rout concluded that although PLR, SII, CRP, and related inflammatory indices consistently correlate with disease severity and adverse outcomes, substantial heterogeneity exists across patient populations, biomarker thresholds, and clinical endpoints [14]. The authors therefore recommended integrating inflammatory biomarkers with clinical and radiographic information rather than relying on individual laboratory parameters.

Prolonged hospitalization is inherently a multifactorial outcome reflecting not only inflammatory burden but also disease evolution, therapeutic response, imaging findings, supportive care requirements, and institutional discharge policies. Consequently, expecting a single biomarker to achieve excellent discrimination is unrealistic. Instead, the consistent but moderate prognostic value demonstrated by PLR, SII, and CRP should be interpreted as evidence that these inexpensive and universally available laboratory parameters capture meaningful biological information while simultaneously highlighting the need for integrated prediction models capable of combining complementary clinical domains.

Also, the use of hospitalization duration as a prognostic endpoint requires consideration of therapeutic factors that may influence the subsequent clinical course. In the present cohort, systemic corticosteroid administration was more frequent among children with prolonged hospitalization and remained associated with this outcome after adjustment for age and sex. This finding may reflect confounding by indication, as corticosteroids are generally considered in selected patients with more severe or refractory *Mycoplasma pneumoniae* pneumonia. On the other hand, pre-admission antibiotic treatment and inpatient macrolide administration were not significantly associated with hospitalization duration. However, these findings should be interpreted cautiously, given heterogeneity of antimicrobial regimens and the wide confidence intervals of the adjusted estimates. Moreover, the absence of

detailed information on treatment timing, duration, and macrolide resistance prevented a more comprehensive evaluation of therapeutic influences.

The hierarchical model comparison showed that the contribution of clinical, inflammatory, and radiographic information to prediction was not cumulative across all model combinations. The clinical model showed discrimination close to chance level (AUROC 0.485), whereas the inflammatory model achieved an AUROC of 0.570. Combining clinical and inflammatory predictors did not improve discrimination relative to the inflammatory model alone (AUROC 0.553). The addition of radiographic characteristics yielded the highest AUROC (0.592) and average precision (0.681), together with the lowest Brier score (0.234). However, the improvement in AUROC over the clinical–inflammatory model was small (ΔAUROC 0.039, 95% CI −0.0004 to 0.0789), with the confidence interval including zero. These results suggest that the available predictors capture only a limited proportion of the factors determining hospitalization duration.

These findings are consistent with recent developments in pediatric prognostic modeling. Increasingly, contemporary prediction models no longer rely on individual biomarkers but instead combine laboratory, clinical, and imaging variables into integrated multivariable algorithms. For example, Xue et al. developed a prognostic model for hospitalized children with community-acquired pneumonia incorporating CRP, procalcitonin, age, respiratory rate, oxygen saturation, and additional laboratory parameters [20]. Their machine-learning model achieved an external validation AUROC of approximately 0.84, substantially exceeding the performance of CRP considered alone. The authors concluded that inflammatory biomarkers derived their prognostic value primarily through integration with physiological variables rather than through independent prediction. This concept closely parallels our observations, where inflammatory indices alone demonstrated limited discrimination.

Similarly, Ning et al. proposed a multivariable nomogram integrating inflammatory biomarkers, biochemical variables, and radiographic findings for predicting severe pediatric community-acquired pneumonia [15]. Their model demonstrated excellent discrimination (training AUROC 0.863; validation AUROC 0.829), together with favorable calibration and decision-curve analysis. However, important differences between their study and ours should be acknowledged. First, Ning and colleagues evaluated severe pneumonia rather than hospitalization duration, a biologically more homogeneous endpoint that is generally easier to predict. Second, their model incorporated multiple cytokines and biochemical markers that are not routinely available in most clinical settings. By contrast, our study deliberately focused on inexpensive laboratory indices derived from standard complete blood counts and CRP, aiming to develop a prediction strategy readily applicable in routine pediatric practice.

Calibration assessment provided further evidence of the limitations of the prediction models. Although mean predicted probabilities were close to the observed frequency of prolonged hospitalization (61.6%) across all four models, this agreement at the population level was not accompanied by satisfactory calibration of individual risk estimates. The clinical model showed particularly unstable calibration, with a negative mean calibration slope, whereas the inflammatory and clinical–inflammatory models yielded slopes of 0.320 and 0.466, respectively. The full model had the highest mean calibration slope (0.561), although this remained substantially below the ideal value of 1. The Brier score was lowest for the full model (0.2345), but differed only marginally from that of a model predicting the observed event frequency for all patients (0.2365). These findings indicate that the numerical improvement in overall prediction error did not translate into sufficiently reliable individual risk estimates.

Similar observations have been reported by Ning et al. and Anteneh et al., whose pediatric pneumonia prediction models demonstrated that improvements in calibration fre-

quently accompany only moderate gains in discrimination [15,21]. These findings reinforce the concept that calibration should be considered an essential component of prognostic model evaluation rather than a secondary analysis.

Decision-curve analysis indicated that the incremental net benefit associated with radiographic information was dependent on the probability threshold considered. The full model showed higher net benefit than the clinical–inflammatory model at thresholds of 0.60 and 0.65, with absolute differences of 0.0594 and 0.0321, respectively, although this advantage was not sustained across the entire threshold range. From a clinical perspective, identifying children at increased risk of prolonged hospitalization could facilitate closer monitoring, earlier discharge planning, and more efficient allocation of hospital resources. Nevertheless, the evaluated thresholds were exploratory and did not correspond to established criteria for specific clinical interventions. Moreover, their proximity to the observed event rate of 61.6% warrants cautious interpretation, particularly given the modest discriminatory performance and suboptimal calibration of the models. Prospective validation is needed to establish clinically appropriate decision thresholds and determine whether model-guided strategies offer measurable advantages over standard clinical assessment.

Similar findings have been described by Ning et al. and Anteneh et al., both of whom demonstrated that multivariable prediction models retained positive net benefit across clinically relevant threshold ranges despite only moderate improvements in AUROC [15,21].

The contribution of baseline chest radiography deserves particular discussion. Conventional chest radiography remains one of the most widely performed investigations in children hospitalized with pneumonia, yet its independent prognostic value remains controversial. In our study, the right-sided and bilateral involvement emerged as the most frequently selected radiographic characteristics during cross-validation, being retained in 84% and 100% of the outer training folds, respectively. These findings suggest that the distribution of radiographic abnormalities may provide additional information beyond the selected clinical and inflammatory variables. However, the modest improvement in discrimination after incorporating radiographic findings indicates that their contribution was limited.

Previous pediatric studies have highlighted the limited specificity of conventional chest radiographic findings for distinguishing pneumonia etiology and severity. Using data from the multicenter EPIC study, Arnold et al. reported that radiographic consolidation was common across viral, atypical, and bacterial pneumonia, thereby limiting its specificity as an indicator of severe disease [22]. In contrast, pleural effusion represented the radiographic feature most strongly associated with bacterial pneumonia and more complicated clinical courses. Similarly, Cho et al. demonstrated that children with *Mycoplasma pneumoniae* pneumonia presenting with consolidation experienced more prolonged fever, greater hypoxemia, more frequent extrapulmonary manifestations, and longer hospitalization than those without consolidation [23]. Our study partially supports these observations. However, the relatively low frequency of consolidation and pleural involvement limited statistical power for detecting independent associations. The predominance of interstitial abnormalities in our cohort reflects the atypical etiology of pneumonia rather than severe parenchymal destruction, thereby explaining their limited prognostic significance.

An important strength of the present study is the exclusive use of biomarkers that are routinely available at hospital admission without additional financial burden. Unlike several recently published prediction models that incorporate cytokines, advanced inflammatory mediators, or specialized laboratory investigations, our models relied exclusively on complete blood count-derived inflammatory indices, CRP, standard clinical assessment, and conventional chest radiography. This strategy enhances potential clinical applicability, particularly in resource-limited healthcare settings where sophisticated biomarkers are

unavailable. Because complete blood count and CRP are universally performed during the initial evaluation of children hospitalized with pneumonia, calculation of PLR and SII requires no additional investigations or costs. Nevertheless, the modest discrimination and suboptimal calibration observed in the present study indicate that the proposed models require further development and independent validation before their application in routine clinical practice can be considered.

Our study should also be interpreted in light of several limitations. First, the retrospective single-center design inevitably introduces the possibility of selection bias and limits the generalizability of the findings. External validation in independent cohorts from different healthcare systems is therefore essential before clinical implementation.

Second, the relatively modest sample size reduced statistical power for evaluating uncommon radiographic abnormalities such as pleural involvement and consolidation.

Third, inflammatory biomarkers were measured only at hospital admission. Serial measurements during hospitalization may provide additional prognostic information by capturing the dynamic evolution of the inflammatory response and treatment effects.

Fourth, radiographic variables were extracted from the radiology reports. Although manual verification minimized misclassification, some residual information bias cannot be excluded.

Finally, prolonged hospitalization, while clinically meaningful, represents a composite outcome influenced by disease severity as well as institutional and organizational factors. Therefore, residual confounding cannot be excluded.

These limitations also identify several important directions for future research. Prospective multicenter studies should evaluate the reproducibility of the present findings across different patient populations and healthcare systems while incorporating external validation and temporal validation strategies. Future investigations should additionally examine whether serial assessment of inflammatory biomarkers improves prognostic performance compared with single admission measurements. Integration of quantitative imaging analysis, lung ultrasound findings, microbiological confirmation, and emerging machine-learning approaches may further enhance predictive accuracy while maintaining clinical interpretability.

Finally, future models should evaluate whether early identification of children at increased risk of prolonged hospitalization translates into improved clinical outcomes through individualized monitoring strategies, earlier therapeutic escalation, or optimized discharge planning.

5. Conclusions

Among the investigated laboratory parameters, PLR and SII remained associated with hospitalization exceeding seven days after adjustment for clinical and radiographic characteristics and correction for multiple comparisons, whereas the association with CRP was attenuated. These findings suggest that hematological inflammatory indices may provide information relevant to hospitalization duration, although their individual predictive value remains limited.

The integration of radiographic findings with clinical and inflammatory variables achieved the highest discriminatory performance among the evaluated models, but the incremental improvement over the clinical–inflammatory model was modest, calibration remained suboptimal, and decision-curve analysis indicated potential net benefit only at selected threshold probabilities.

Consequently, inflammatory biomarkers should not be interpreted as standalone prognostic tools but rather as complementary components of integrated multimodal prediction models. Prospective multicenter validation studies are recommended to determine whether

such models can facilitate early risk stratification and support personalized management strategies for children hospitalized with atypical pneumonia.

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