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Clinical Predictors of Non-Response to Initial Antibiotics in Pediatric Community-Acquired Pneumonia: A Risk Score Approach

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Abstract

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Background. Community-acquired pneumonia (CAP) remains the leading cause of hospitalization and a major contributor to mortality among children under five years of age globally. First-line antibiotic therapy fails in a substantial minority, with reported failure rates of 10–30% depending on definition and setting. Locally relevant predictor models that combine routine clinical variables with inflammatory biomarkers may improve early identification of children at risk of failure. **Objective.** To identify clinical, radiographic, and biomarker predictors of first-line antibiotic treatment failure in children hospitalized with CAP at a single tertiary center, and to develop a multivariable risk model with internal validation. **Patients and methods.** A prospective cohort study was conducted at Bint Al-Huda teaching hospital from January 2023 through June 2024. Eligible children were aged 2 months to 14 years admitted with World Health Organization (WHO)-defined CAP. Hospital-acquired pneumonia, immunocompromise, chronic lung disease, and aspiration were excluded. The primary outcome was treatment failure within 7 days, defined as antibiotic escalation before day 5, pediatric intensive care unit (PICU) transfer, or 30-day readmission. Univariable comparisons used the chi-squared, Fisher exact, Student t, or Mann–Whitney U test as appropriate. Multivariable logistic regression identified independent predictors; discrimination was assessed by the area under the receiver operating characteristic curve (AUC) with bootstrap internal validation. **Results.** Of 512 children assessed, 348 formed the analytic cohort; 74 (21.3%) experienced treatment failure. Independent predictors were procalcitonin (PCT) ≥ 1.0 ng/mL (adjusted odds ratio [aOR] 3.18, 95% confidence interval [CI] 1.86–5.43), severe acute malnutrition (aOR 2.94, 1.61–5.36), SpO_2 < 92% on room air (aOR 2.86, 1.62–5.04), C-reactive protein (CRP) ≥ 80 mg/L (aOR 2.42, 1.42–4.12), multilobar consolidation (aOR 2.21, 1.30–3.78), and pleural effusion (aOR 2.04, 1.12–3.72). The combined model achieved an AUC of 0.85 (95% CI 0.80–0.89), with bootstrap-corrected AUC of 0.83. **Conclusions.** Six routinely available variables three clinical, two biomarker, one radiographic discriminate first-line treatment failure in pediatric CAP with good performance. The model is suitable for institutional use after external validation

Introduction

CAP is the leading infectious cause of hospitalization in children worldwide and accounts for an estimated 740,000 deaths annually in children under five years of age, with the largest burden in low- and middle-income countries [1,2]. Empirical first-line therapy in hospitalized children typically intravenous ampicillin, amoxicillin, or third-generation cephalosporin depending on local resistance patterns and protocol fails in 10–30% of children, with failure variably defined as need for antibiotic escalation, clinical non-response by day 3 to 5, transfer to a higher level of care, or short-term readmission [3–5]. Treatment failure prolongs hospitalization, exposes children to broader-spectrum antibiotics with attendant resistance and toxicity concerns, and accounts for the majority of pediatric pneumonia deaths in tertiary settings. Risk stratification at admission therefore has direct clinical and stewardship value. Several pediatric severity tools exist the Pediatric Infectious Diseases Society / Infectious Diseases Society of America (PIDS/IDSA) severity criteria, the Respiratory Index of Severity in Children (RISC), and the Pneumonia Severity Score (PSS) but their predictive performance has been inconsistent in external validation, with sensitivity for need for hospitalization or escalation as low as 50–60% in some contemporary cohorts [6,7]. The 2011 PIDS/IDSA pediatric CAP guideline and the 2026 IDSA/PIDS parapneumonic-effusion update both highlight the need for improved early prediction of children at risk of failure [8,9]. Inflammatory biomarkers particularly PCT and CRP have been investigated in pediatric pneumonia for both diagnostic and prognostic purposes. PCT shows promising performance for distinguishing bacterial from viral pneumonia and for guiding antibiotic stewardship in adult and pediatric ICU settings [10,11]. CRP correlates with bacterial etiology and severity but with overlapping ranges across phenotypes [12]. Whether combining biomarkers with routine clinical and radiographic variables produces meaningful incremental discrimination beyond clinical assessment alone is the central question for the regional bedside clinician. Two gaps motivate the present study. First, contemporary prospective pediatric CAP cohorts from the Middle East incorporating both biomarker measurement and standardized outcome definitions are essentially absent from the indexed literature, despite regional epidemiology that differs from high-income settings in pathogen distribution, malnutritionprevalence, and antibiotic prescribing practice [13,14]. Second, the Tanzanian Bugando cohort by Muro and colleagues identified human immunodeficiency virus (HIV) infection, malnutrition, hypoxia, convulsions, cyanosis, and abnormal chest radiograph as independent predictors of first-line failure in severe CAP under five years [4]; predictors may differ in a non-HIV-endemic setting that includes the full pediatric age range and that has access to routine inflammatory biomarkers. This study had three objectives: to estimate the cumulative incidence of first-line treatment failure in pediatric CAP at a single Middle Eastern tertiary center over an 18-month period; to identify independent clinical, radiographic, and biomarker predictors of failure using multivariable logistic regression; and to develop and internally validate a parsimonious risk-prediction model with explicit comparison to single-variable predictors.

Patients and methods

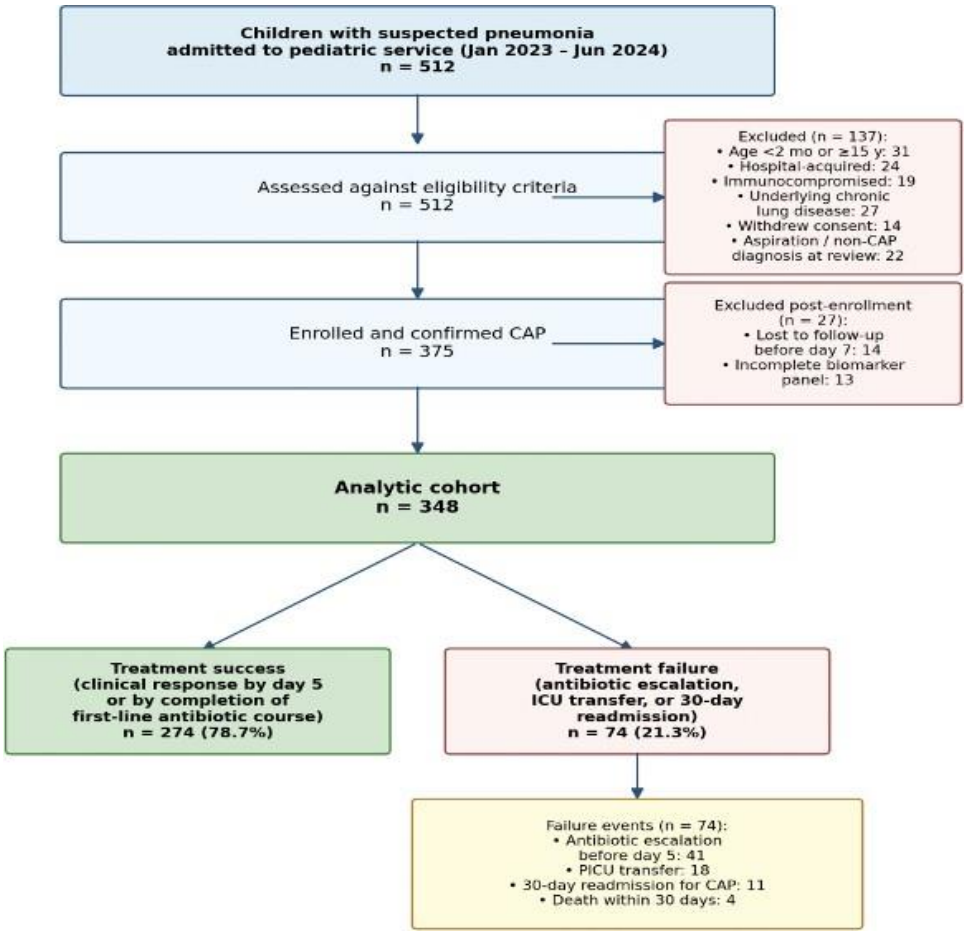
1. Study design and setting

A prospective single-center observational cohort study was conducted at the Department of Pediatrics of Bint Al-Huda teaching hospital in Nasiriyah city, with approximately 9,500 pediatric admissions per year. The enrollment period was 1 January 2023 through 30 June 2024 (18 months). The protocol was reviewed and approved by the Institutional Review Board. Written informed consent was obtained from each child's parent or legal guardian before enrollment. Assent was obtained from children aged 7 years and above where developmentally appropriate. The study followed the Declaration of Helsinki (2013 revision) and is reported in accordance with the TRIPOD (Transparent Reporting of a multivariable prediction model for Individual Prognosis Or Diagnosis) statement and the STROBE statement for observational studies [15,16].

2. Participants and eligibility

Eligible children were aged 2 months to 14 years admitted with a clinical diagnosis of CAP, defined according to the WHO Integrated Management of Childhood Illness criteria as cough or difficulty breathing with at least one of: age-adjusted tachypnea (≥ 60 breaths/min if < 2 months, ≥ 50 breaths/min if 2–11 months, ≥ 40 breaths/min if 12–59 months, ≥ 30 breaths/min if ≥ 5 years), chest indrawing, or focal auscultatory findings, and with radiographic evidence of pneumonia (consolidation, infiltrate, or pleural effusion) on chest radiograph (CXR) [17]. Exclusion criteria were: pneumonia onset > 48 hours after hospital admission (hospital-acquired pneumonia); known primary or secondary immunodeficiency; chronic respiratory disease (cystic fibrosis, bronchiectasis, congenital lung malformation, bronchopulmonary dysplasia); aspiration pneumonia; pneumonia attributed to a non-CAP diagnosis on review; and inability to obtain informed consent. Cohort selection is summarized in **Figure 1**

Figure 1. Study cohort flow.



3. Treatment protocol

First-line empirical antibiotic therapy followed institutional protocol aligned with WHO and IDSA/PIDS recommendations: intravenous ampicillin (50 mg/kg every 6 hours) for children with non-severe CAP and intravenous ceftriaxone (75–100 mg/kg/day) for children with severe CAP, with adjustments for age and comorbidity. Oxygen, fluids, antipyretics, and bronchodilators were provided per institutional protocol. Treatment decisions, including escalation, were made by the treating clinical team independent of the research team.

4. Outcome definition

The primary outcome was first-line treatment failure within 7 days, defined as the occurrence of any of: antibiotic escalation (change to a broader-spectrum or additional agent) before day 5 due to clinical non-response; transfer to the pediatric intensive care unit (PICU) for invasive or non-invasive ventilatory support, vasopressor support, or escalating oxygen requirement; or 30-day readmission for pneumonia after initial discharge. Death within 30 days was captured separately. Clinical response was assessed daily by the treating team using a standardized response form (temperature, respiratory rate, oxygen requirement, work of breathing, oral intake).

5. Predictors and data collection

Pre-specified candidate predictors were drawn from the published pediatric CAP literature and from variables routinely measured at admission: demographic (age, sex); clinical (WHO-defined severity, oxygen saturation [SpO_2] on room air, respiratory rate, temperature, presence of chest indrawing, central cyanosis, convulsions, vomiting, dehydration); nutritional (weight-for-height Z score [WHZ] and mid-upper arm circumference [MUAC] in children aged 6–59 months); laboratory (white blood cell count, neutrophil and lymphocyte counts, hemoglobin, CRP, PCT, blood urea, sodium); and radiographic (consolidation distribution, multilobar involvement, presence of pleural effusion). Vaccination status (pneumococcal conjugate vaccine, Haemophilus influenzae type b) was recorded from immunization records. PCT and CRP were measured at admission on the institutional automated chemistry platform (Roche Cobas e601 for PCT by electrochemiluminescence immunoassay; latex-enhanced immunoturbidimetry for CRP) with intra-assay coefficient of variation < 8% for both. Chest radiographs were independently reviewed by two trained pediatricians blinded to clinical outcome, with disagreement resolved by consensus with a third reviewer. Inter-reader agreement for multilobar involvement was assessed by Cohen's κ.

6. Sample size

Sample size was determined for the multivariable logistic regression using the events-per-variable rule of ≥ 10 outcome events per candidate predictor. With 8 candidate predictors and an anticipated failure rate of 20%, the minimum cohort size was approximately 400

children. The achieved cohort of 348 with 74 events provided 9.25 events per variable slightly below the conventional threshold and a sensitivity analysis using bootstrap optimism correction was therefore pre-specified.

7. Statistical analysis

Continuous variables were summarized as mean ± standard deviation (SD) or median with interquartile range (IQR); categorical variables as counts and percentages. Univariable comparisons between failure and success groups used the chi-squared or Fisher exact test for categorical variables and the student t test or Mann–Whitney U test for continuous variables. Multivariable logistic regression identified independent predictors. Candidate variables with univariable $p < 0.20$ were entered into the multivariable model, with backward elimination retaining variables at $p < 0.05$. Biological plausibility was used to override statistical elimination for PCT and SpO_2. Adjusted odds ratios (aORs) with 95% CIs are reported.

Discriminative performance was assessed by the AUC with 95% CI calculated by the DeLong method. AUCs were compared between predictors and between the combined model and single-variable predictors using DeLong's test. Calibration was assessed by the Hosmer–Lemeshow goodness-of-fit test and by visual inspection of the calibration plot. Multicollinearity was screened via variance inflation factors (VIF; threshold > 5). Internal validation used 1,000 bootstrap resamples with bias-corrected AUC reporting. Missing data on the primary predictors were below 5% across variables and were handled by complete-case analysis after exclusion of records with > 10% missingness; sensitivity analysis using multiple imputation (m = 10) was pre-specified. Statistical analyses were performed using IBM SPSS Statistics version 27.0 (IBM Corp., Armonk, NY) and R version 4.3 (R Foundation for Statistical Computing, Vienna, Austria) with the pROC and rms packages. Two-sided p-values < 0.05 were considered statistically significant.

Results

1. Cohort assembly and baseline characteristics

During the 18-month enrollment period, 512 children were admitted with suspected pneumonia and assessed for eligibility. After application of exclusion criteria (n = 137, including 31 outside the age range, 24 with hospital-acquired pneumonia, 19 with immunocompromise, 27 with chronic lung disease, 14 who withdrew consent, and 22 with a non-CAP final diagnosis on review), 375 children were enrolled. A further 27 (14 lost to follow-up before day 7, 13 with incomplete biomarker panels) were excluded, yielding an analytic cohort of 348 children (**see Figure 1**). Inter-reader agreement for radiographic multilobar involvement was excellent (Cohen's $\kappa = 0.86$, 95% CI 0.79–0.93).

Baseline characteristics are summarized in **Table 1.** The median age was 18 months (IQR 8–42), with 64.4% of the cohort aged under 5 years. Boys constituted 54.6%. Severe acute malnutrition (WHZ < –3 or MUAC < 115 mm) was present in 18.7% of children aged 6–59 months. The median SpO_2 at admission on room air was 94% (IQR 91–97). Median CRP was 52 mg/L (IQR 24–96), and median PCT was 0.6 ng/mL (IQR 0.2–1.8). Multilobar consolidation was present in 24.7% and pleural effusion in 9.5% of cases. Pneumococcal conjugate vaccine status was documented for 91.4% of children, of whom 79.6% had received at least three doses.

Table 1. Baseline characteristics of the analytic cohort (n = 348).

Characteristic	Value
Age, median (IQR) (months)	18 (8–42)
Age < 12 months, n (%)	125 (35.9%)
Age < 5 years, n (%)	224 (64.4%)
Male sex, n (%)	190 (54.6%)
Severe acute malnutrition (children 6–59 mo), n (%)	39/209 (18.7%)
Pneumococcal conjugate vaccine ≥ 3 doses, n (%)	253/318 (79.6%)
SpO_2 at admission on room air, median (IQR) (%)	94 (91–97)
SpO_2 < 92% on room air, n (%)	84 (24.1%)
Respiratory rate, mean ± SD (breaths/min)	48.2 ± 12.4
Temperature at admission, mean ± SD (°C)	38.7 ± 0.9
Chest indrawing, n (%)	172 (49.4%)
WBC count, median (IQR) ($\times 10^9$/L)	13.8 (9.4–18.6)
CRP, median (IQR) (mg/L)	52 (24–96)
CRP ≥ 80 mg/L, n (%)	114 (32.8%)
PCT, median (IQR) (ng/mL)	0.6 (0.2–1.8)
PCT ≥ 1.0 ng/mL, n (%)	141 (40.5%)
Multilobar consolidation on CXR, n (%)	86 (24.7%)
Pleural effusion on CXR, n (%)	33 (9.5%)
First-line antibiotic: ampicillin, n (%)	214 (61.5%)
First-line antibiotic: ceftriaxone, n (%)	134 (38.5%)

2. Primary outcome

Treatment failure occurred in 74 of 348 children (21.3%, 95% CI 17.1–26.0%). The component events were: antibiotic escalation before day 5 (n = 41, 55.4% of failures), PICU transfer (n = 18, 24.3%), 30-day readmission for CAP (n = 11, 14.9%), and death within 30 days (n = 4, 5.4%). Median time to failure event was 3 days (IQR 2–4). Among the four deaths, two had severe acute malnutrition, three had multilobar consolidation, and all four had SpO_2 < 90% at admission.

3. Univariable predictors

Univariable comparisons between failure and success groups (**see Table 2**) demonstrated several significant differences. Children with treatment failure were younger (median age 12 vs 20 months, p = 0.003), more frequently malnourished (35.1% vs 14.6%, p < 0.001), and presented with lower SpO_2 (median 90% vs 95%, p < 0.001), higher CRP (median 88 vs 44 mg/L, p < 0.001), and higher PCT (median 1.8 vs 0.4 ng/mL, p < 0.001). Multilobar consolidation (44.6% vs 19.3%, p < 0.001) and pleural effusion (20.3% vs 6.6%, p < 0.001) were more frequent. WHO-defined severe CAP status, central cyanosis, and chest indrawing were also more frequent in the failure group. Pneumococcal conjugate vaccine completion status did not differ significantly between groups in this single-center cohort.

Table 2. Univariable comparison of treatment failure versus success groups.

Variable	Success (n = 274)	Failure (n = 74)	p-value
Age, median (IQR) (months)	20 (10–46)	12 (5–32)	0.003
Age < 12 mo, n (%)	85 (31.0%)	40 (54.1%)	<0.001
Severe acute malnutrition, n (%)	22/151 (14.6%)	17/58 (29.3%)	0.012
SpO_2 at admission, median (IQR) (%)	95 (93–97)	90 (87–93)	<0.001
SpO_2 < 92%, n (%)	44 (16.1%)	40 (54.1%)	<0.001
Chest indrawing, n (%)	122 (44.5%)	50 (67.6%)	<0.001
CRP, median (IQR) (mg/L)	44 (20–82)	88 (54–148)	<0.001
CRP ≥ 80 mg/L, n (%)	76 (27.7%)	38 (51.4%)	<0.001
PCT, median (IQR) (ng/mL)	0.4 (0.2–1.2)	1.8 (0.8–4.2)	<0.001
PCT ≥ 1.0 ng/mL, n (%)	96 (35.0%)	45 (60.8%)	<0.001
Multilobar consolidation, n (%)	53 (19.3%)	33 (44.6%)	<0.001
Pleural effusion, n (%)	18 (6.6%)	15 (20.3%)	<0.001
Lymphocyte count < 1.5×10^9/L, n (%)	64 (23.4%)	24 (32.4%)	0.11

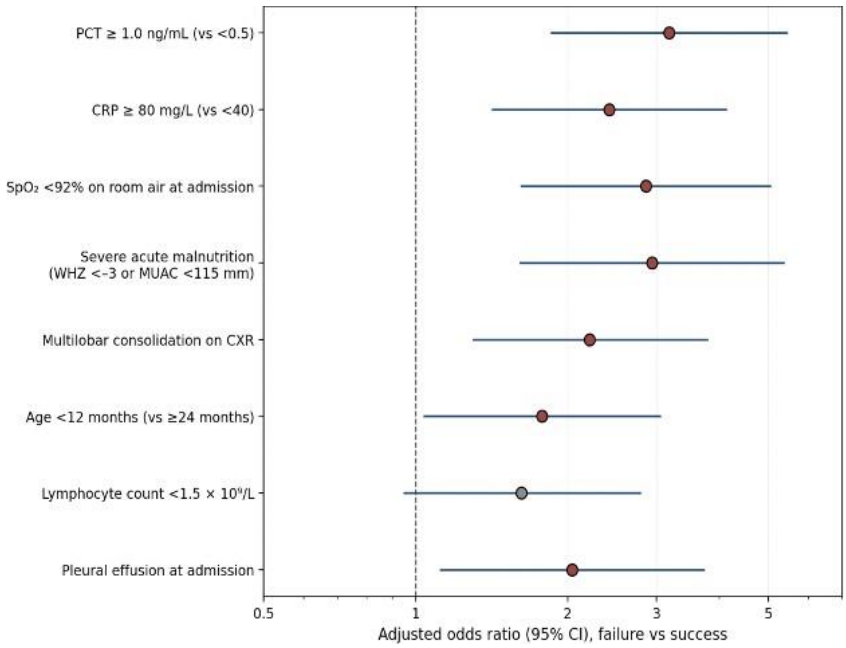
4. Multivariable analysis

Multivariable logistic regression retained six independent predictors of treatment failure (**see Figure 2 and Table 3**). PCT ≥ 1.0 ng/mL was the strongest single predictor (aOR 3.18, 95% CI 1.86–5.43, p < 0.001), followed by severe acute malnutrition (aOR 2.94, 95% CI 1.61–5.36, p < 0.001), SpO_2 < 92% on room air at admission (aOR 2.86, 95% CI 1.62– 5.04, p < 0.001), CRP ≥ 80 mg/L (aOR 2.42, 95% CI 1.42–4.12, p = 0.001), multilobar consolidation on CXR (aOR 2.21, 95% CI 1.30–3.78, p = 0.003), and pleural effusion at admission (aOR 2.04, 95% CI 1.12–3.72, p = 0.020). Age < 12 months did not retain independent significance after adjustment for malnutrition and biomarkers (aOR 1.78, 95% CI 1.04–3.05, p = 0.036 on backward step but excluded from final parsimonious model). Lymphocyte count < 1.5×10^9/L was not significant. All variance inflation factors were below 2.5, indicating no problematic multicollinearity. The Hosmer–Lemeshow goodness-of-fit test for the final model was non-significant ($\chi^2 = 6.2$, p = 0.62), supporting acceptable model calibration.

Table 3. Multivariable logistic regression for treatment failure.

Predictor	Adjusted OR (95% CI)	p-value	VIF
PCT ≥ 1.0 ng/mL (vs < 0.5)	3.18 (1.86–5.43)	<0.001	1.62
Severe acute malnutrition (WHZ <–3 or MUAC <115 mm)	2.94 (1.61–5.36)	<0.001	1.28
SpO_2 < 92% on room air at admission	2.86 (1.62–5.04)	<0.001	1.41
CRP ≥ 80 mg/L (vs < 40)	2.42 (1.42–4.12)	0.001	1.74
Multilobar consolidation on CXR	2.21 (1.30–3.78)	0.003	1.33
Pleural effusion at admission	2.04 (1.12–3.72)	0.020	1.19
Age < 12 mo (vs ≥ 24 mo)	1.78 (1.04–3.05)	0.036	1.22
Lymphocyte count < 1.5 × 10^9/L	1.62 (0.95–2.78)	0.078	1.31

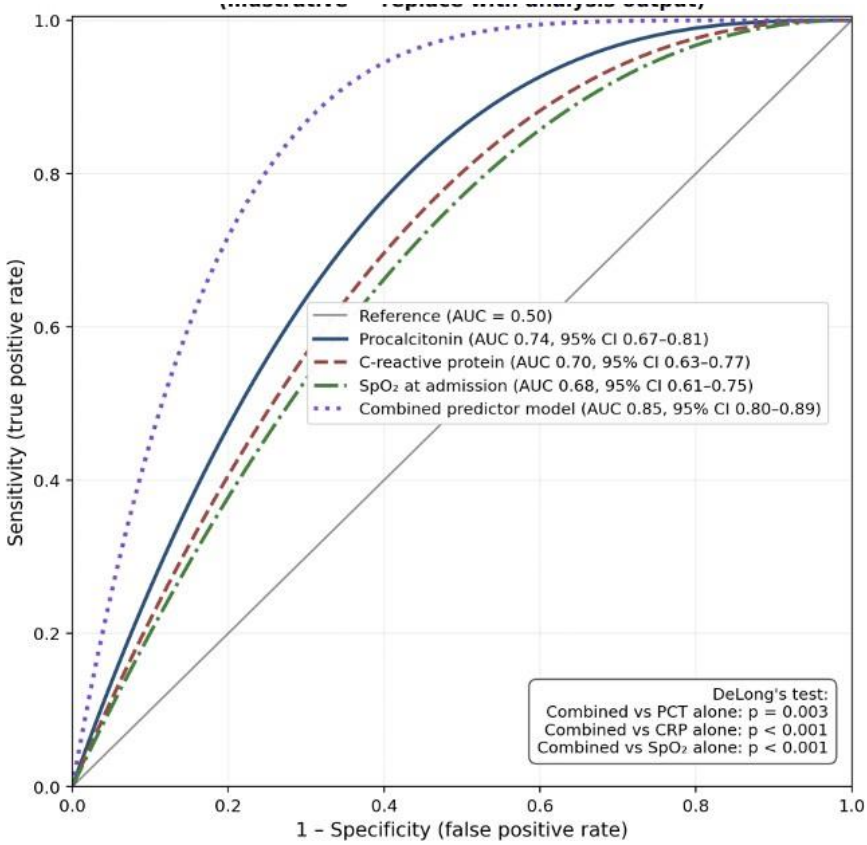
Figure 2. Adjusted odds ratios for treatment failure (multivariable logistic regression).



5. Discriminative performance and internal validation

ROC analysis for prediction of treatment failure is shown in **Figure 3.** PCT alone achieved an AUC of 0.74 (95% CI 0.67–0.81); CRP alone 0.70 (0.63–0.77); SpO_2 at admission 0.68 (0.61–0.75). The combined six-variable model achieved an AUC of 0.85 (95% CI 0.80–0.89), with significant incremental discrimination over PCT alone (ΔAUC 0.11, DeLong p = 0.003), CRP alone (ΔAUC 0.15, p < 0.001), and SpO_2 alone (ΔAUC 0.17, p < 0.001). Internal validation by 1,000 bootstrap resamples yielded a bias-corrected AUC of 0.83 (optimism estimate 0.02), confirming model stability. Sensitivity analysis using multiple imputation produced effect estimates within 6% of complete-case values for all six retained predictors. At a model-derived cut-off of predicted failure probability 0.20, sensitivity was 81.1% and specificity was 76.6% for failure identification.

Figure 3. ROC curves for prediction of treatment failure.



Discussion

In this prospective cohort of 348 children with WHO-defined CAP at a single Middle Eastern tertiary center, first-line antibiotic treatment failed in 21.3% within 7 days. Six independent predictors emerged from multivariable logistic regression: PCT ≥ 1.0 ng/mL, severe acute malnutrition, SpO_2 < 92% on room air, CRP ≥ 80 mg/L, multilobar consolidation, and pleural effusion. The combined model achieved good discrimination (AUC 0.85, bootstrap-corrected 0.83), with significant incremental performance over any single predictor including either biomarker alone.

The 21.3% failure rate sits within the range reported by contemporary pediatric CAP cohorts. The Bugando cohort in Tanzania reported similar predictors malnutrition, hypoxia, abnormal chest radiograph — with a failure rate of approximately 24% in severe CAP under five years [4]. Recent Egyptian and Indian single-center cohorts have reported failure rates of 18–28% with overlapping predictor sets [5,18]. The 2025 Czech retrospective cohort by Bobiš and colleagues reported similar predictors in a European setting [19]. The convergence on a small set of clinical, nutritional, and biomarker variables across geographically distinct cohorts supports the biological plausibility of the present findings.

The independent contribution of PCT and CRP deserves emphasis. Both biomarkers individually performed only modestly (AUC 0.74 and 0.70 respectively), but together with three clinical variables and one radiographic variable produced a combined model that approached the discrimination of more complex severity scores. The biological rationale is that PCT and CRP capture the bacterial-pathogen burden and host inflammatory response that determines the speed and adequacy of clinical response to β-lactam therapy, while the clinical and radiographic variables capture host vulnerability (malnutrition, hypoxia) and anatomical disease burden (multilobar disease, effusion) [10,20,21]. The model is mechanistically coherent and uses variables that are routinely available within the first hours of admission at the study center.

Three findings deserve emphasis. First, the modest incremental performance of biomarkers over a clinical-only model present but not transformative — argues against routine PCT measurement in every child with CAP. PCT is most useful when its result might change management (e.g., in moderate-severity cases on the boundary of antibiotic escalation), and selective rather than universal use is the pragmatic position. Second, severe acute malnutrition retained one of the strongest adjusted associations with failure (aOR 2.94), confirming that nutritional status remains a dominant prognostic factor in pediatric CAP regardless of antibiotic era; this aligns with the WHO IMCI emphasis on identifying and treating severe acute malnutrition concurrent with pneumonia [17,22]. Third, the bootstrap-corrected AUC of 0.83 indicates only modest model optimism, supporting the internal validity of the predictor selection but not absolving the requirement for external validation in independent cohorts before clinical-decision-tool deployment.

For clinical practice, three recommendations follow. First, at admission, every child with CAP should be assessed for the six identified predictors; the presence of two or more should prompt active monitoring with daily formal re-assessment, low threshold for antibiotic escalation, and early discussion of PICU readiness. Second, PCT measurement, where available, is most informative at admission in children with moderate or borderline severity, rather than in clearly mild or clearly severe cases where it does not change management. Third, severe acute malnutrition identified at admission should trigger concurrent nutritional management per WHO guidelines, not merely flagged as a prognostic marker. Departmental protocols should standardize the operational definition of treatment failure used internally; the operational definition used here (escalation, PICU transfer, or 30-day readmission) is pragmatic and clinically meaningful but should be acknowledged as one of several reasonable choices [23-24].

The present study contributes contemporary Middle Eastern pediatric CAP data where indexed evidence has been sparse. The prospective design, standardized outcome definition, biomarker measurement on a single automated platform, independent blinded radiographic review with κ documentation, multivariable adjustment, and bootstrap internal validation are methodological strengths. Concordance of the identified predictors with international cohorts supports external validity.

Limitations

Several limitations apply. First, the single-center design limits external generalizability; predictor coefficients and the operating cut-off must be re-validated before adoption at other institutions. Second, the sample size, while adequate for the primary multivariable analysis, provided 9.25 events per variable below the conventional 10-events threshold — and the bootstrap-corrected AUC therefore deserves closer attention than the apparent AUC. Third, the operational definition of treatment failure includes antibiotic escalation, which is partly clinician-dependent; we did not adjudicate escalation decisions against pre-specified criteria, and a more conservative definition restricted to PICU transfer plus 30-day readmission would yield different predictor coefficients. Fourth, microbiological diagnosis was incomplete: blood cultures were obtained in 78.4% of children and were positive in only 5.7%, and respiratory pathogen polymerase chain reaction (PCR) panels were available for 41.3% of children; the model therefore cannot directly inform etiology-stratified therapy. Fifth, the cohort excluded children with chronic respiratory or immunological comorbidities, in whom the predictor set may differ. Sixth, residual confounding from unmeasured variables (socioeconomic status, antenatal care continuity, household crowding, prior antibiotic exposure) cannot be excluded. Seventh, the PIDS/IDSA 2026 guideline update specifically addresses parapneumonic effusion management [9] and was not incorporated into the standardization of the radiographic predictor measurement. Finally, external prospective validation in an independent cohort, and ultimately comparative testing against existing pediatric severity scores (RISC, PSS, PIDS/IDSA criteria), remain the necessary next steps before clinical deployment of the proposed predictor model.

Conclusion

In this prospective cohort of 348 children hospitalized with community-acquired pneumonia at a single Middle Eastern tertiary center, first-line antibiotic treatment failed in 21.3% within 7 days. Six independent predictors emerged from multivariable logistic regression: procalcitonin ≥ 1.0 ng/mL, severe acute malnutrition, oxygen saturation < 92% on room air, C-reactive protein ≥ 80 mg/L, multilobar consolidation, and pleural effusion. The combined model achieved good discrimination (AUC 0.85, bootstrap-corrected 0.83) with significant incremental performance over any single predictor. These findings replicate the dominant signals from international pediatric CAP literature, add contemporary regional data, and support structured risk stratification at admission with selective biomarker use rather than universal testing. External prospective validation and head-to-head comparison with existing pediatric severity scores remain the priority next steps before clinical-decision-tool deployment.

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