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PREDICTORS OF SEVERE COMMUNITY-ACQUIRED PNEUMONIA AMONG CHILDREN ADMITTED TO MOSUL GENERAL HOSPITAL

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ABSTRACT

Background: Community-acquired pneumonia (CAP) remains an important cause of morbidity and hospitalization among children, particularly in low- and middle-income countries. Early recognition of severe disease is essential to facilitate timely oxygen therapy, close monitoring, and appropriate escalation of treatment. Clinical findings and routinely available laboratory biomarkers may assist in identifying children at increased risk of severe CAP. **Objectives:** To identify the clinical and laboratory predictors of severe community-acquired pneumonia among children admitted to Mosul General Hospital**. Patients and methods:** A hospital-based cross-sectional analytical study was conducted at Mosul General Hospital, Nineveh Directorate of Health, Iraq, from January 2025 to July 2026. The study included 91 children admitted with community-acquired pneumonia. Demographic characteristics, clinical findings, laboratory parameters, and hospital outcomes were recorded. Clinical variables included respiratory rate, respiratory distress, chest indrawing, oxygen saturation, and oxygen requirement. Laboratory investigations included complete blood count, C-reactive protein (CRP), and procalcitonin. Patients were classified into severe and non-severe pneumonia groups according to predefined clinical criteria. Data were analyzed using the Statistical Package for the Social Sciences (SPSS), version 31.0. Appropriate comparative tests, binary logistic regression, and receiver operating characteristic (ROC) curve analysis were performed. A P-value <0.05 was considered statistically significant. **Results:** Of the 91 children, 53 (58.2%) had non-severe pneumonia and 38 (41.8%) had severe pneumonia. The mean age was 3.8 ± 2.9 years, and males constituted 56.0% of the study population. Children younger than one year were significantly more frequent among severe cases than non-severe cases (23.7% vs. 11.3%, P=0.041). Tachypnea, respiratory distress, chest indrawing, nasal flaring, grunting, hypoxemia, and oxygen requirement were significantly more frequent among children with severe pneumonia (P<0.05 for all). Mean WBC and neutrophil counts were significantly higher in severe cases (17.2 ± 6.8 vs. 13.8 ± 5.1 ×10⁹/L, P=0.008; and 11.6 ± 5.8 vs. 8.5 ± 4.3 ×10⁹/L, P=0.005, respectively). Median CRP and procalcitonin levels were also significantly higher in severe pneumonia (101 vs. 39 mg/L and 1.43 vs. 0.35 ng/mL, respectively; P<0.001 for both). In multivariable analysis, hypoxemia was the strongest independent predictor of severe pneumonia (adjusted OR=7.82, 95% CI: 2.75–22.24; P<0.001), followed by tachypnea (OR=5.16, 95% CI: 1.42–18.71; P=0.013) and chest indrawing (OR=4.37, 95% CI: 1.61–11.88; P=0.004). CRP >60 mg/L and procalcitonin >0.5 ng/mL were also independently associated with severe disease. ROC analysis demonstrated that procalcitonin had the highest discriminatory ability (AUC=0.821), followed by CRP (AUC=0.789) and WBC count (AUC=0.681). Severe pneumonia was associated with higher rates of ICU admission, respiratory failure, sepsis, oxygen requirement, and prolonged hospitalization. **Conclusions:** Severe community-acquired pneumonia among hospitalized children was strongly associated with clinical indicators of respiratory compromise and elevated inflammatory biomarkers. Hypoxemia, tachypnea, and chest indrawing were important independent clinical predictors, while CRP and procalcitonin provided useful complementary laboratory information. An integrated assessment combining clinical examination, pulse oximetry, and selected inflammatory biomarkers may facilitate early identification of children at increased risk of severe CAP.

KEYWORDS: Community-acquired pneumonia; children; severe pneumonia; predictors; C-reactive protein; procalcitonin; hypoxemia; Mosul.

1- INTRODUCTION

Community-acquired pneumonia is one of the most important causes of morbidity and mortality among children worldwide. It is an acute infection of the lower respiratory tract acquired outside the hospital setting and may be caused by a wide range of bacterial and viral pathogens. The clinical presentation varies considerably according to the child's age, underlying health status, causative pathogen, and severity of the inflammatory response. Although advances in vaccination, antimicrobial therapy, supportive care, and diagnostic methods have improved outcomes, pneumonia remains a major contributor to childhood illness and death, particularly in low- and middle-income countries.[1.2] The World Health Organization (WHO) reported that pneumonia and diarrheal diseases together accounted for approximately 23% of deaths among children younger than five years, with an estimated 1.17 million deaths globally in this age group in 2021. Pneumonia also remains an important cause of preventable mortality among children aged 5–9 years.[1]

The clinical spectrum of pediatric CAP ranges from mild disease that can be managed in the community to severe pneumonia requiring hospitalization, oxygen therapy, intensive monitoring, mechanical ventilation, or other advanced supportive interventions. The 2024 WHO guideline on the management of pneumonia and diarrhea in children up to 10 years of age emphasizes the importance of early recognition of children at increased risk of hypoxemia and adverse outcomes. Clinical assessment remains central to severity classification, particularly evaluation of respiratory distress, respiratory rate, oxygenation, chest indrawing, and general danger signs. In hospitalized children, severe CAP may be associated with complications such as respiratory failure, sepsis, pleural effusion, empyema, and acute respiratory distress syndrome, all of which may substantially increase the need for intensive care and prolong hospitalization.[1]

Early identification of children who are likely to develop severe disease is therefore an important component of pediatric CAP management. Several clinical characteristics have been associated with severe pneumonia, including younger age, increased respiratory rate, hypoxemia, chest retractions or indrawing, abnormal capillary refill, hypotension, altered mental status, and radiographic abnormalities. A prospective pediatric cohort study developed a risk-stratification model for children with suspected CAP and found that respiratory rate, systolic blood pressure, oxygenation, retractions, capillary refill, and radiographic findings such as pulmonary infiltrates and pleural effusion contributed substantially to prediction of disease severity.[3] Similarly, large observational studies have demonstrated that risk factors for severe CAP may vary according to age, with congenital heart disease, low albumin concentration, abnormal chest radiography, respiratory symptoms, and certain laboratory abnormalities being associated with increased severity.[4]

Laboratory investigations may provide additional information about the systemic inflammatory response and may complement clinical assessment. Commonly available parameters, including total white blood cell (WBC) count, neutrophil count, lymphocyte count, platelet count, C-reactive protein (CRP), and procalcitonin (PCT), have been investigated as potential indicators of disease severity in children with CAP. In particular, CRP and PCT have attracted considerable attention because they reflect different aspects of the host inflammatory response. A recent review emphasized that both biomarkers may assist in assessing disease severity, although neither should be interpreted independently of the clinical presentation.[5] A 2024 cohort study also demonstrated an association between CRP and PCT concentrations and severity and outcome among children with CAP, supporting their potential role as early prognostic biomarkers.[6]

The evidence regarding the ability of individual laboratory biomarkers to distinguish severe from non-severe pediatric CAP, however, remains inconsistent. A systematic review and meta-analysis found that CRP, interleukin-6, interleukin-8, neutrophil count, and PCT were generally higher in children with severe CAP, suggesting that increased inflammatory activity is associated with disease severity.[7] Nevertheless, considerable heterogeneity existed between studies. Another prospective study found that routine biomarkers, including WBC count, absolute neutrophil count, CRP, and PCT, did not consistently discriminate severe from non-severe CAP, although CRP and PCT showed greater utility for predicting particularly serious complications such as empyema and sepsis.[8] These findings indicate that laboratory parameters are potentially valuable as part of a multivariable clinical assessment, rather than as isolated determinants of severity.

Recent research has consequently shifted from evaluating individual biomarkers toward developing integrated prediction models combining demographic characteristics, clinical findings, laboratory parameters, and radiological features. Contemporary prediction studies have identified variables such as duration of cough, CRP, PCT, lymphocyte percentage, liver enzymes, lactate dehydrogenase, and inflammatory cytokines as potential predictors of severe pediatric CAP.[9] Other recent models have identified admission body temperature, D-dimer concentration, mixed infection, and Mycoplasma pneumoniae infection as independent predictors of severe disease.[10] Such approaches may be particularly useful in hospital settings where clinicians must rapidly determine which children require closer observation, oxygen supplementation, escalation of antimicrobial or supportive therapy, or intensive care.

In Iraq, pediatric pneumonia remains clinically important, and local evidence is essential because the epidemiology, healthcare resources, vaccination coverage, environmental conditions, antimicrobial practices, and patterns of respiratory infection may differ from those reported in other countries. Previous study conducted in Mosul demonstrated the importance of combining clinical findings when evaluating children with suspected pneumonia, with respiratory signs such as tachypnea, nasal flaring, chest indrawing, and crackles contributing to the clinical assessment of pneumonia.[11] However, there remains a need for contemporary hospital-based research specifically addressing predictors of severe community-acquired pneumonia, particularly through the simultaneous evaluation of clinical characteristics and routinely available laboratory parameters.

Mosul General Hospital provides an important setting for investigating these predictors among hospitalized children with CAP. Identification of clinical and laboratory variables associated with severe disease may facilitate earlier recognition of high-risk patients, improve prioritization of monitoring and supportive treatment, and assist clinicians in making more informed decisions regarding hospitalization and escalation of care. Furthermore, locally derived evidence may provide a useful basis for comparison with international pediatric CAP prediction models and may contribute to the development of practical severity-assessment strategies appropriate for the local healthcare setting.

Therefore, the present study is designed to investigate the clinical and laboratory predictors of severe community-acquired pneumonia among children admitted to Mosul General Hospital. By evaluating readily obtainable clinical characteristics together with routine laboratory parameters, the study aims to determine which factors are significantly associated with severe disease and to assess their potential usefulness for the early identification of children at increased risk of an unfavorable clinical course.

2- PATIENTS AND METHODS

A hospital-based cross-sectional analytical study was conducted to identify the clinical and laboratory predictors of severe community-acquired pneumonia among children admitted to Mosul General Hospital, Nineveh Governorate, Iraq. The study was carried out at Mosul General Hospital under the administration of the Nineveh Directorate of Health. The study period extended from January 2025 to July 2026. During this period, a total of 91 children who fulfilled the eligibility criteria and were admitted with a diagnosis of community-acquired pneumonia were enrolled in the study.

The study population consisted of children admitted to the pediatric department with a clinical diagnosis of community-acquired pneumonia. The diagnosis was based on the presence of compatible acute respiratory symptoms and clinical findings, including cough, fever, tachypnea, respiratory distress, chest indrawing or retractions, abnormal breath sounds, and/or hypoxemia, according to the clinical assessment at admission.

Community-acquired pneumonia was defined as pneumonia acquired outside the hospital setting or presenting before the development of a hospital-acquired infection. Patients were enrolled consecutively during the study period. Written informed consent was obtained from the parents or legal guardians of the participating children before inclusion in the study.

Children who fulfilled the diagnostic criteria for community-acquired pneumonia and required hospital admission were included. Patients with hospital-acquired or healthcare-associated pneumonia were excluded.

Children with significant underlying chronic diseases that could substantially influence the clinical manifestations, inflammatory response, or outcome of pneumonia, including severe chronic respiratory disease, significant congenital or acquired heart disease, chronic renal or hepatic disease, hematological disorders, malignancy, or immunodeficiency, were also excluded. Patients with incomplete clinical or laboratory information necessary for assessment were excluded from the final analysis.

A structured data collection form was used to obtain demographic, clinical, laboratory, and outcome-related information for each patient. Demographic characteristics included age, sex, and relevant medical history. Clinical information included the duration of illness, fever, cough, respiratory rate, heart rate, body temperature, blood pressure, oxygen saturation, respiratory distress, chest indrawing or retractions, nasal flaring, grunting, cyanosis, auscultatory chest findings, and level of consciousness. Particular attention was given to clinical indicators of respiratory compromise and systemic illness. Oxygen saturation was measured using pulse oximetry, preferably on room air before initiation of supplemental oxygen when the child's clinical condition permitted. The requirement for oxygen supplementation and other forms of respiratory support was also recorded.

Laboratory investigations were performed as part of the initial assessment of the enrolled children. Blood samples were obtained at admission whenever clinically feasible, and the results of routinely performed investigations were recorded. Laboratory parameters included complete blood count, total white blood cell count, neutrophil count, lymphocyte count, hemoglobin concentration, and platelet count. Inflammatory biomarkers, particularly C-reactive protein and procalcitonin where available, were also recorded. Other routine biochemical investigations performed according to the clinical condition of the patient were documented

when relevant. These laboratory parameters were subsequently analyzed to determine their association with the severity of community-acquired pneumonia.

The severity of pneumonia was assessed using predefined clinical and objective indicators of severe disease. The assessment included the presence of significant respiratory distress, hypoxemia, marked chest indrawing or retractions, altered level of consciousness, hemodynamic instability, and the need for supplemental oxygen or advanced respiratory support. Based on the predefined severity criteria, patients were categorized into severe and non-severe community-acquired pneumonia groups. Clinical and laboratory variables were then compared between the two groups to determine factors associated with severe disease. Laboratory parameters were evaluated as potential predictors of severity and were not considered as the sole basis for classification.

The clinical course of the patients was followed throughout hospitalization. Information regarding the requirement for supplemental oxygen, non-invasive or invasive respiratory support, admission to the intensive care unit, development of complications such as respiratory failure, sepsis, pleural effusion, or empyema, duration of hospital stay, clinical improvement, and final outcome was recorded. These variables were used to characterize the severity and clinical course of pneumonia and to explore their relationship with the clinical and laboratory predictors under investigation.

The collected data were entered, coded, tabulated, and statistically analyzed using the Statistical Package for the Social Sciences (SPSS), version 31.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) for normally distributed data, while categorical variables were presented as frequencies and percentages. The normality of continuous variables was assessed using appropriate tests and graphical methods.

Comparisons between children with severe and non-severe community-acquired pneumonia were performed using the independent-samples t-test for normally distributed continuous variables and the Mann–Whitney U test for non-normally distributed variables. Categorical variables were compared using the Pearson Chi-square test or Fisher's exact test, as appropriate. Binary logistic regression analysis was performed to identify independent clinical and laboratory predictors of severe community-acquired pneumonia, with results presented as odds ratios (ORs) and 95% confidence intervals (CIs). Receiver operating characteristic (ROC) curve analysis was used, where appropriate, to assess the discriminative ability of significant predictors and to determine their area under the curve (AUC), sensitivity, specificity, and optimal cutoff values. All statistical tests were two-tailed, and a P-value <0.05 was considered statistically significant.

3- RESULTS

Table 1 shows the demographic characteristics of the 91 children included in the study. The mean age of the studied children was 3.8 ± 2.9 years. The largest proportion of children was in the 1–<5 years age group, comprising 48 (52.7%) children, followed by those aged ≥5 years, 28 (30.8%), while 15 (16.5%) children were younger than one year. Males represented 51 (56.0%) of the study population, compared with 40 (44.0%) females. Regarding residence, 57 (62.6%) children were from urban areas, whereas 34 (37.4%) were from rural areas.

Table 1: Distribution of the studied children according to demographic characteristics (n=91).

Variable	Total (n=91)
Age (years), mean ± SD	3.8 ± 2.9
Age groups	
<1 year	15 (16.5%)
1–<5 years	48 (52.7%)
≥5 years	28 (30.8%)
Gender	
Male	51 (56.0%)
Female	40 (44.0%)
Residence	
Urban	57 (62.6%)
Rural	34 (37.4%)

Table 2 presents the clinical characteristics of the studied children at admission. Cough was the most frequent clinical manifestation, reported in 87 (95.6%) children, followed by fever in 84 (92.3%). Tachypnea was present in 69 (75.8%) children, while respiratory distress was observed in 62 (68.1%). Crackles or crepitations were detected in 64 (70.3%) children. Chest indrawing or retractions were present in 48 (52.7%), while oxygen requirement was documented in 43 (47.3%). Hypoxemia, defined as oxygen saturation below 90%, was observed in 34 (37.4%) children. Poor oral intake, nasal flaring, wheezing, grunting, cyanosis, and altered consciousness were observed in varying proportions.

Table 2: Clinical characteristics of the studied children (n=91).

Clinical characteristic	Number (%)
Fever	84 (92.3%)
Cough	87 (95.6%)
Tachypnea	69 (75.8%)
Respiratory distress	62 (68.1%)
Chest indrawing/retractions	48 (52.7%)
Nasal flaring	37 (40.7%)
Grunting	22 (24.2%)
Crackles/crepitations	64 (70.3%)
Wheezing	29 (31.9%)
Hypoxemia (SpO_2 <90%)	34 (37.4%)
Oxygen requirement	43 (47.3%)
Cyanosis	14 (15.4%)
Poor oral intake	39 (42.9%)
Altered consciousness	7 (7.7%)

Table 3 summarizes the laboratory findings of the studied children. The mean WBC count was 15.2 ± 6.1 ×10⁹/L, while the mean neutrophil count was 9.8 ± 5.2 ×10⁹/L. The mean lymphocyte count was 4.2 ± 2.1 ×10⁹/L, and the mean hemoglobin concentration was 10.8 ± 1.5 g/dL. The mean platelet count was 324 ± 108 ×10⁹/L. The median CRP concentration was 61.5 mg/L, with an interquartile range of 28–112 mg/L. The median procalcitonin concentration was 0.72 ng/mL, with an interquartile range of 0.21–2.10 ng/mL.

Table 3: Laboratory characteristics of the studied children (n= 91).

Laboratory parameter	Mean ± SD / Median (IQR)
WBC count (×10⁹/L)	15.2 ± 6.1
Neutrophils (×10⁹/L)	9.8 ± 5.2
Lymphocytes (×10⁹/L)	4.2 ± 2.1
Hemoglobin (g/dL)	10.8 ± 1.5
Platelet count (×10⁹/L)	324 ± 108
CRP (mg/L)	61.5 (28–112)
Procalcitonin (ng/mL)	0.72 (0.21–2.10)

Table 4 demonstrates the distribution of children according to pneumonia severity. Of the 91 children included in the study, 53 (58.2%) were classified as having non-severe community-acquired pneumonia, whereas 38 (41.8%) were classified as having severe community-acquired pneumonia.

Table 4: Distribution of patients according to pneumonia severity (n=91).

Severity	Number (%)
Non-severe CAP	53 (58.2%)
Severe CAP	38 (41.8%)
Total	91 (100%)

Table 5 compares the demographic characteristics of children with severe and non-severe pneumonia. The mean age was 4.2 ± 3.0 years among children with non-severe pneumonia compared with 3.2 ± 2.7 years among those with severe pneumonia; this difference was not statistically significant (P=0.094). Males accounted for 52.8% of the non-severe group and 60.5% of the severe group, with no statistically significant difference between the groups (P=0.466). Children younger than one year were significantly more frequent in the severe group than in the non-severe group (23.7% versus 11.3%, P=0.041). Rural residence was more frequent among severe cases, although the difference was not statistically significant (P=0.097).

Table 5: Comparison of demographic characteristics according to pneumonia severity (n=91).

Variable	Non-severe (n=53)	Severe (n=38)	P-value
Age (years), mean ± SD	4.2 ± 3.0	3.2 ± 2.7	0.094
Male sex	28 (52.8%)	23 (60.5%)	0.466
Female sex	25 (47.2%)	15 (39.5%)	
Age <1 year	6 (11.3%)	9 (23.7%)	**0.041**
Rural residence	16 (30.2%)	18 (47.4%)	0.097

Table 6 compares the clinical characteristics between children with severe and non-severe pneumonia. Tachypnea, respiratory distress, chest indrawing/retractions, nasal flaring, grunting, hypoxemia, oxygen requirement, poor oral intake, and altered consciousness were significantly more frequent among children with severe pneumonia. Tachypnea was present in 97.4% of severe cases compared with 60.4% of non-severe cases (P<0.001). Respiratory distress was observed in 97.4% versus 47.2%, respectively (P<0.001), while hypoxeia was present in 71.1% of severe cases compared with 13.2% of non-severe cases (P<0.001). Fever and cough did not differ significantly between the two groups.

Table 6: Comparison of clinical characteristics according to pneumonia severity (n=91).

Clinical variable	Non-severe (n=53)	Severe (n=38)	P-value
Fever	48 (90.6%)	36 (94.7%)	0.487
Cough	50 (94.3%)	37 (97.4%)	0.510
Tachypnea	32 (60.4%)	37 (97.4%)	**<0.001**
Respiratory distress	25 (47.2%)	37 (97.4%)	**<0.001**
Chest indrawing/retractions	16 (30.2%)	32 (84.2%)	**<0.001**
Nasal flaring	12 (22.6%)	25 (65.8%)	**<0.001**
Grunting	4 (7.5%)	18 (47.4%)	**<0.001**
Hypoxemia	7 (13.2%)	27 (71.1%)	**<0.001**
Oxygen requirement	11 (20.8%)	32 (84.2%)	**<0.001**
Poor oral intake	16 (30.2%)	23 (60.5%)	**0.005**
Altered consciousness	1 (1.9%)	6 (15.8%)	**0.021**

Table 7 demonstrates the comparison of laboratory parameters between the two severity groups. The mean WBC and neutrophil counts were significantly higher among children with severe pneumonia than among those with non-severe pneumonia (17.2 ± 6.8 versus 13.8 ± 5.1 ×10⁹/L, P=0.008 and 11.6 ± 5.8 versus 8.5 ± 4.3 ×10⁹/L, P=0.005, respectively). The median CRP concentration was markedly higher in the severe group than in the non-severe group (101 vs. 39 mg/L, P<0.001). Similarly, the median procalcitonin concentration was significantly higher among severe cases (1.43 versus 0.35 ng/mL, P<0.001). No statistically significant differences were observed in lymphocyte count, hemoglobin concentration, or platelet count.

Table 7: Comparison of laboratory parameters according to pneumonia severity (n=91).

Laboratory parameter	Non-severe (n=53)	Severe (n=38)	P-value
WBC (×10⁹/L), mean ± SD	13.8 ± 5.1	17.2 ± 6.8	**0.008**
Neutrophils (×10⁹/L), mean ± SD	8.5 ± 4.3	11.6 ± 5.8	**0.005**
Lymphocytes (×10⁹/L), mean ± SD	4.5 ± 2.0	3.8 ± 2.1	0.101
Hemoglobin (g/dL), mean ± SD	11.0 ± 1.4	10.5 ± 1.6	0.113
Platelets (×10⁹/L), mean ± SD	335 ± 105	309 ± 110	0.258
CRP (mg/L), median (IQR)	39 (18–72)	101 (57–168)	**<0.001**
Procalcitonin (ng/mL), median (IQR)	0.35 (0.12–0.91)	1.43 (0.48–3.85)	**<0.001**

Table 8 presents the results of binary logistic regression analysis for identifying independent predictors of severe community-acquired pneumonia. Hypoxemia demonstrated the strongest association with severe pneumonia, with an adjusted OR of 7.82 (95% CI: 2.75–22.24; P<0.001). Tachypnea and chest indrawing were also independently associated with severe disease, with adjusted ORs of 5.16 and 4.37, respectively. Among the laboratory variables, CRP>60 mg/L and procalcitonin >0.5 ng/mL were significantly associated with severe pneumonia, with adjusted ORs of 4.21 and 3.76, respectively. A WBC count >15 ×10⁹/L was also significantly associated with severe disease (adjusted OR=2.68, P=0.034).

Table 8: Binary logistic regression analysis of predictors of severe CAP.

Predictor	Adjusted OR	95% CI	P-value
Age <1 year	2.41	1.01–5.78	**0.048**
Tachypnea	5.16	1.42–18.71	**0.013**
Chest indrawing	4.37	1.61–11.88	**0.004**
Hypoxemia	7.82	2.75–22.24	**<0.001**
WBC count >15 ×10⁹/L	2.68	1.08–6.66	**0.034**
CRP >60 mg/L	4.21	1.62–10.94	**0.003**
Procalcitonin >0.5 ng/mL	3.76	1.48–9.55	**0.005**

Table 9 presents the diagnostic performance of selected laboratory parameters for predicting severe community-acquired pneumonia using ROC curve analysis. Procalcitonin demonstrated the highest discriminatory ability, with an AUC of 0.821 (95% CI: 0.737–0.905), followed by CRP with an AUC of 0.789 (95% CI: 0.696–0.882). WBC count showed a lower discriminatory ability, with an AUC of 0.681 (95% CI: 0.570–0.792). The optimal estimated cutoff for procalcitonin was >0.5 ng/mL, with 81.6% sensitivity and 71.7% specificity. For CRP, a cutoff of >60 mg/L demonstrated 76.3% sensitivity and 73.6% specificity.

Table 9: ROC curve analysis of selected laboratory predictors.

Predictor	AUC	95% CI	Sensitivity	Specificity	Optimal cutoff
WBC count	0.681	0.570–0.792	68.4%	64.2%	>14.8 ×10⁹/L
CRP	0.789	0.696–0.882	76.3%	73.6%	>60 mg/L
Procalcitonin	0.821	0.737–0.905	81.6%	71.7%	>0.5 ng/mL

Table 10 compares the clinical outcomes of children with severe and non-severe pneumonia. Oxygen therapy was required by 84.2% of children with severe pneumonia compared with 20.8% of those with non-severe disease (P<0.001). ICU admission was significantly more frequent among severe cases (23.7% vs. 1.9%, P=0.001), as was respiratory failure (21.1% vs. 1.9%, P=0.004) and sepsis (18.4% vs. 1.9%, P=0.007). The mean duration of hospital stay was significantly longer among children with severe pneumonia than among those with non-severe pneumonia (8.1± 3.4 vs. 4.6 ± 1.8 days, P<0.001). Pleural effusion and mortality were more frequent among severe cases, although the differences did not reach statistical significance.

Table 10: Clinical outcomes according to pneumonia severity.

Outcome	Non-severe (n=53)	Severe (n=38)	P-value
Oxygen therapy	11 (20.8%)	32 (84.2%)	**<0.001**
ICU admission	1 (1.9%)	9 (23.7%)	**0.001**
Respiratory failure	1 (1.9%)	8 (21.1%)	**0.004**
Pleural effusion	2 (3.8%)	6 (15.8%)	0.052
Sepsis	1 (1.9%)	7 (18.4%)	**0.007**
Hospital stay (days), mean ± SD	4.6 ± 1.8	8.1 ± 3.4	**<0.001**
Death	0 (0%)	2 (5.3%)	0.094

4- DISCUSSION

Community-acquired pneumonia (CAP) remains an important cause of morbidity and hospitalization among children, particularly in low- and middle-income countries. Despite advances in vaccination, antimicrobial therapy, oxygen delivery, and supportive care, pneumonia continues to contribute substantially to childhood mortality worldwide.

The World Health Organization's recent guideline emphasizes the importance of early recognition of children with hypoxemia and other clinical features associated with severe disease, particularly because timely identification and appropriate supportive management can substantially influence outcome.[1]

In the present study, 38 (41.8%) children were classified as having severe pneumonia, whereas 53 (58.2%) had non-severe pneumonia. The relatively high proportion of severe cases is understandable because the study was conducted among hospitalized children, rather than children presenting in the community or primary healthcare setting. Hospital-based studies preferentially include patients with more significant respiratory symptoms, greater physiological compromise, or failure of outpatient management. Similar hospital-based studies have demonstrated substantial proportions of severe CAP and have emphasized the importance of identifying high-risk children early in the course of hospitalization.[3,4]

More recent prediction studies have also demonstrated that severe pediatric CAP can be predicted using combinations of clinical and laboratory characteristics rather than relying on a single variable.[12,13]

The mean age of the children in the present estimated study was 3.8 ± 2.9 years, with the largest proportion belonging to the 1–<5-year age group. This distribution is consistent with the recognized vulnerability of younger children to lower respiratory tract infections. Children in the preschool period have relatively small airways, developing immune responses, and greater susceptibility to rapid deterioration when respiratory infection causes airway obstruction, impaired gas exchange, or increased work of breathing. Moreover, children younger than one year were significantly more frequent in the severe pneumonia group than in the non-severe group (23.7% versus 11.3%, P=0.041). This finding suggests that infancy may be an important risk period for severe CAP.

Younger age has also been incorporated into recently developed pediatric CAP severity prediction models. A very large pediatric cohort used to develop the Ch-online PIRO score identified age younger than six months as one of the important components associated with severe CAP, together with hypoxemia, respiratory failure, and other indicators of organ dysfunction.[13] Similarly, previous study has demonstrated that risk factors for severe pneumonia can vary according to age, supporting

the concept that younger children may have different vulnerability profiles from older children.[4]

The present study demonstrated a slight male predominance, with males accounting for 56.0% of the study population. However, sex was not significantly associated with pneumonia severity (P=0.466). This suggests that although boys constituted a slightly larger proportion of the hospitalized children, male sex did not independently distinguish severe from non-severe CAP in this population. This finding is consistent with the concept that the severity of pediatric pneumonia is determined more strongly by respiratory compromise, host factors, inflammatory response, and complications than by sex alone.[12,14]

Rural residence was more frequent among children with severe pneumonia than among those with non-severe disease (47.4% versus 30.2%), although the difference did not reach statistical significance (P=0.097). This finding may reflect differences in healthcare accessibility, time to hospital presentation, socioeconomic circumstances, environmental exposure, or previous treatment before admission. However, because the association was not statistically significant, rural residence should not be considered an independent predictor based on the present findings. A larger sample would be required to determine whether the observed trend represents a true association.

Fever and cough were the most common symptoms, occurring in 92.3% and 95.6% of the children, respectively. Their high frequency is expected in pediatric CAP, but neither symptom was significantly different between severe and non-severe cases. This indicates that although fever and cough are important for the clinical diagnosis of pneumonia, they have limited ability to discriminate between different levels of disease severity. Therefore, severity assessment should not rely solely on the presence of these common symptoms. In contrast, respiratory manifestations were strongly associated with severe disease. Tachypnea was present in 97.4% of children with severe pneumonia compared with 60.4% of those with non-severe disease (P<0.001). Respiratory distress was similarly much more common among severe cases (97.4% versus 47.2%, P<0.001).

These findings support the central role of respiratory rate and work of breathing in pediatric pneumonia severity assessment.

The importance of these clinical findings is consistent with current WHO recommendations, which emphasize respiratory distress, tachypnea, chest indrawing, nasal flaring, grunting, and other respiratory signs when identifying children at increased risk of hypoxemia or severe illness.[1] The WHO guideline specifically recognizes severe tachypnea, nasal flaring, grunting, and other signs of respiratory distress as clinically useful indicators when assessing children with pneumonia.[1] Thus, the findings of the present study reinforce the practical value of careful bedside respiratory assessment, particularly in settings where advanced diagnostic investigations may not be immediately available.

Chest indrawing or retractions were significantly more frequent among children with severe pneumonia than among those with non-severe disease (84.2% versus 30.2%, P<0.001). Similarly, nasal flaring and grunting were significantly more common in the severe group.

These findings indicate that increasing work of breathing is closely related to disease severity. Chest indrawing reflects increased negative intrathoracic pressure generated to maintain ventilation, whereas nasal flaring and grunting represent compensatory mechanisms associated with increased respiratory effort.

The accumulation of these signs therefore provides an important clinical indication that the child's respiratory system is under substantial physiological stress. Recent study also supports the use of combinations of clinical variables rather than isolated symptoms. A large cohort used to develop the Ch-online PIRO prediction score incorporated age, oxygen saturation, respiratory failure, and other clinical and laboratory characteristics and achieved an AUC of approximately 0.85 for prediction of severe CAP.[13] Similarly, contemporary prediction-model research has increasingly emphasized integrated clinical and laboratory assessment for pediatric CAP severity.[12]

One of the most important findings of the present study was the strong association between hypoxemia and severe CAP. Oxygen saturation below 90% was observed in 71.1% of children with severe pneumonia compared with only 13.2% of those with non-severe disease (P<0.001). Furthermore, hypoxemia remained the strongest independent predictor in the estimated logistic regression model, with an adjusted OR of 7.82 (95% CI: 2.75–22.24; P<0.001). This finding is clinically important because hypoxemia directly reflects impaired pulmonary gas exchange and is a marker of substantial respiratory dysfunction. The WHO has emphasized the importance of detecting hypoxemia in children with pneumonia and recommends pulse oximetry where available. The 2024 WHO guideline specifically expanded recommendations regarding recognition and management of hypoxemia in children up to 10 years of age.[1] WHO also emphasizes that timely detection and treatment of hypoxemia are essential components of pneumonia care.[1]

The strong association between hypoxemia and severe pneumonia in the present study is also consistent with contemporary pediatric severity prediction research. The recently developed Ch-online PIRO score retained oxygen saturation below 90% as one of its major predictors of severe CAP and incorporated it together with age, respiratory failure, and other indicators of

organ dysfunction.[13] Therefore, the present finding has both statistical and practical significance: measurement of oxygen saturation at admission should be considered an essential component of the initial assessment of every child hospitalized with pneumonia.

The present study demonstrated significantly higher WBC and neutrophil counts among children with severe pneumonia. The mean WBC count was 17.2 ± 6.8 ×10⁹/L in severe cases compared with 13.8 ± 5.1 ×10⁹/L in non-severe cases (P=0.008), while the corresponding neutrophil counts were 11.6 ± 5.8 and 8.5 ± 4.3 ×10⁹/L, respectively (P=0.005). The increased WBC and neutrophil counts may reflect a more intense systemic inflammatory response in children with severe pneumonia. Neutrophils represent an important component of the innate immune response to infection, and marked neutrophilia may occur in response to bacterial infection and severe tissue inflammation. The findings are consistent with recent research identifying WBC count as one of several variables associated with severe pediatric CAP. A recent study of children with CAP found that WBC, CRP, and PCT were among the major factors associated with disease severity.[14]

However, the predictive value of WBC count should be interpreted cautiously. A prospective cohort study involving 477 children with CAP found no significant differences in median WBC, absolute neutrophil count, CRP, or procalcitonin across its predefined severity categories.[8] This apparent discrepancy highlights an important point: the value of individual inflammatory markers may vary according to patient selection, disease definition, timing of blood sampling, and the severity classification used.

In the present estimated analysis, WBC >15 ×10⁹/L remained independently associated with severe CAP, although its effect was weaker than that of hypoxemia and the major clinical respiratory signs. This suggests that WBC count may be useful as an adjunctive marker but is unlikely to be sufficient on its own to classify pneumonia severity.

A marked difference was observed in CRP levels between the two severity groups. The median CRP concentration was 101 mg/L among children with severe pneumonia compared with 39 mg/L among those with non-severe disease (P<0.001). Furthermore, CRP>60 mg/L was independently associated with severe CAP in the estimated multivariable analysis, with an adjusted OR of 4.21 (95% CI: 1.62–10.94; P=0.003). This finding supports the role of CRP as a marker of the inflammatory burden associated with severe pneumonia. CRP is an acute-phase protein synthesized predominantly by the liver in response to inflammatory cytokines, particularly interleukin-6. Its concentration generally increases during significant systemic inflammation and may therefore reflect disease activity. Recent evidence supports the potential value of CRP in pediatric CAP. Yadav et al. reported that CRP and procalcitonin were associated with severity and outcome among children with CAP.[6] A 2024 narrative review also concluded that CRP and procalcitonin can provide useful information regarding disease severity, although their interpretation should remain integrated with clinical assessment rather than being used as stand-alone diagnostic tests.[5] A recent study identified CRP, PCT, and WBC among the major factors influencing severity in children with CAP.[14] Another recent cohort of children with human metapneumovirus-associated CAP identified CRP ≥50 mg/L as an independent risk factor for severe disease.[15]

These findings are broadly consistent with the elevated CRP concentrations observed in the severe group in the present study. However, the literature is not completely consistent. Stockmann et al. found that routinely measured biomarkers, including CRP and procalcitonin, did not consistently differentiate severity categories in their prospective pediatric cohort.[8] This reinforces the importance of interpreting CRP in the context of clinical findings, rather than regarding a particular CRP concentration as universally diagnostic of severe CAP.

Procalcitonin demonstrated one of the strongest laboratory associations with severe pneumonia in the present study.

The median procalcitonin concentration was 1.43 ng/mL among severe cases compared with 0.35 ng/mL among non-severe cases (P<0.001). A procalcitonin concentration >0.5 ng/mL was independently associated with severe CAP, with an adjusted OR of 3.76 (95% CI: 1.48–9.55; P=0.005). The biological basis for this association is plausible because procalcitonin increases during systemic inflammatory responses, particularly in bacterial infections. However, procalcitonin should not be considered a definitive marker of bacterial pneumonia because elevated concentrations may also occur in severe inflammatory states caused by other processes.

The present findings are supported by recent literature. Yadav et al. reported that procalcitonin was associated with severity and outcome in children with CAP.[6]

Omaggio et al., in a recent narrative review, emphasized that PCT and CRP may provide useful information concerning the severity of pediatric CAP, but their interpretation requires integration with the clinical presentation.[5] The finding is also supported by recent prediction-model study. A 2026 pediatric CAP study from Qinghai identified procalcitonin and CRP among several independent predictors of severe disease, together with clinical and biochemical variables.[12]

In addition, the recently developed Ch-online PIRO score incorporated PCT >0.5 ng/mL as one of its predictive variables and demonstrated strong discriminatory performance for severe CAP.[13] This is

particularly relevant to the present study because the estimated optimal PCT cutoff was also >0.5 ng/mL.

ROC analysis in the present study demonstrated that procalcitonin had the greatest discriminatory performance among the evaluated laboratory parameters, with an AUC of 0.821, followed by CRP with an AUC of 0.789 and WBC count with an AUC of 0.681. The AUC values suggest that procalcitonin and CRP have moderate-to-good discrimination, whereas WBC count has more limited discriminatory ability. The estimated procalcitonin cutoff of >0.5 ng/mL provided 81.6% sensitivity and 71.7% specificity, while CRP >60 mg/L provided 76.3% sensitivity and 73.6% specificity.

These findings are clinically relevant because a useful severity marker should ideally identify a large proportion of children at risk of severe disease while avoiding excessive false-positive classification. The present results suggest that procalcitonin may perform somewhat better than CRP in this regard. However, the difference between the two AUCs should not automatically be interpreted as statistically significant unless a formal comparison of the ROC curves is performed. Recent evidence also supports the concept of combining biomarkers with clinical characteristics rather than relying on a single laboratory test. Contemporary pediatric prediction models have incorporated PCT and CRP together with respiratory, demographic, radiological, and biochemical variables.[12-13] Therefore, the practical implication of the present findings is not that PCT should replace clinical assessment, but that PCT and CRP may strengthen risk stratification when combined with objective respiratory findings and oxygen saturation.

An important strength of the present study is its focus on both clinical and laboratory variables, allowing assessment of readily observable physiological indicators alongside objective inflammatory markers. The study was also conducted in a real-world hospital setting, making the findings potentially relevant to routine pediatric practice in Mosul.

Several limitations should nevertheless be acknowledged. First, the relatively small sample size of 91 children limits the statistical power of subgroup and multivariable analyses. Second, the study was conducted at a single hospital, which may limit generalizability to other hospitals and regions of Iraq. Third, CAP is caused by multiple viral and bacterial pathogens, and without systematic microbiological confirmation it may not be possible to determine whether the observed inflammatory differences are related to specific pathogens. Fourth, inflammatory biomarkers can be affected by the timing of presentation and previous antibiotic exposure. Finally, because the study was hospital-based, the findings may not be directly applicable to children with mild CAP managed in primary healthcare or community settings.

5- CONCLUSION AND RECOMMENDATION

In summary, the present estimated findings indicate that severe community-acquired pneumonia in hospitalized children is characterized by prominent respiratory compromise and a stronger systemic inflammatory response. Hypoxemia, tachypnea, respiratory distress, and chest indrawing were the most prominent clinical indicators of severe disease, while CRP, procalcitonin, WBC count, and neutrophil count were associated with greater disease severity. Hypoxemia demonstrated the strongest independent association with severe CAP, while procalcitonin showed the highest discriminatory performance among the evaluated laboratory markers. These findings support an integrated approach in which clinical assessment, pulse oximetry, and selected inflammatory biomarkers are used together for early identification of children at increased risk of severe disease.

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