

Research Article

Predictors of Severe Outcomes in Children Hospitalised with Community-Acquired Pneumonia: A Prospective Observational Study from a Tertiary Care Centre in Bengaluru

Dr. Kavya S¹, Dr. Keerthana T N², Dr. Kartheeka M G^{3*}

^{1,2,3*}Associate Professor, Department of Paediatrics, Sapthagiri NPS University (SNPSU), Sapthagiri Institute of Medical Sciences and Research Centre (SIMSRC), Bangalore, Karnataka, India.

Corresponding Author: Dr. Kartheeka M G

Associate Professor, Department of Paediatrics, Sapthagiri NPS University (SNPSU), Sapthagiri Institute of Medical Sciences and Research Centre (SIMSRC), Bangalore, Karnataka, India.

Email: mgkartheeka@gmail.com

Received: 18.06.26, Revised: 21.07.26, Accepted: 23.09.26, Published: 24.09-2026

ABSTRACT

Background. Community-acquired pneumonia (CAP) remains a leading cause of hospitalisation and death among Indian children. Readily available bedside predictors of a severe in-hospital course are still incompletely defined in medium-volume tertiary paediatric units in South India.

Objective. To determine the frequency of severe in-hospital outcomes and to identify clinical, nutritional, and radiographic predictors among children hospitalised with CAP.

Methods. Consecutive children aged 2 months to 12 years admitted with World Health Organization (WHO)-defined CAP to the Department of Paediatrics, Sapthagiri Institute of Medical Sciences and Research Centre, Bengaluru, were enrolled over 12 months. The primary endpoint was a composite severe outcome: paediatric intensive care unit (PICU) admission, non-invasive or invasive ventilation, hospital stay ≥ 7 days, pneumonia complication, or in-hospital death. Associations were examined with χ^2 or Fisher exact tests and binary logistic regression.

Results. Of 78 children screened, 56 were analysed (mean age 28.4 ± 22.1 months; 34 boys, 60.7%). A severe outcome occurred in 18 children (32.1%), including two deaths (3.6%). Independent unadjusted associations included WHO danger signs (odds ratio [OR] 13.36; 95% confidence interval [CI] 3.28-54.39), hypoxaemia with $\text{SpO}_2 < 92\%$ (OR 8.86; 2.47-31.79), abnormal chest radiograph (OR 5.82; 1.17-28.96), age < 12 months (OR 4.69; 1.39-15.77), incomplete immunisation, lack of exclusive breastfeeding, and underweight (weight-for-age z-score [WAZ] < -2). In a three-variable model limited by event count, danger signs (adjusted OR [aOR] 8.12; 1.62-40.71) and hypoxaemia (aOR 5.94; 1.28-27.52) remained independent.

Conclusion. About one in three hospitalised children with CAP had a severe course. Pulse-oximetry hypoxaemia and WHO danger signs at admission should trigger PICU-level monitoring, even in a modest-volume tertiary ward.

Keywords: Community-Acquired Pneumonia, Children, Hypoxaemia, Danger Signs, Malnutrition, Immunization, Predictors, India.

INTRODUCTION

Pneumonia continues to account for a large share of deaths in children younger than 5 years worldwide, and India still contributes a substantial fraction of these deaths [2,17]. Hospital series from tertiary Indian centres report case-fatality approaching 8–11% in severe CAP, together with frequent need for antibiotic change, prolonged stay, or ventilatory support [4,5]. Reducing this burden depends on recognising, at the first hospital contact, the child who will deteriorate despite standard ward care.

The revised WHO Integrated Management of Childhood Illness (IMCI) guidance groups fast breathing and chest indrawing as “pneumonia” and reserves “severe pneumonia” for general danger signs, stridor in a calm child, severe malnutrition, or hypoxaemia [1,18]. Pulse oximetry is now regarded as an essential triage tool: hypoxaemia is both common in childhood pneumonia in low- and middle-income countries and one of the strongest markers of death [6,7]. British Thoracic Society (BTS) and Pediatric Infectious Diseases Society/Infectious Diseases Society of America (PIDS/IDSA) guidance, together with Indian acute-

treatment-unit studies, have used hypoxaemia ($\text{SpO}_2 < 92\%$ in BTS practice) as a hospitalisation and severity threshold [3,10,11].

Beyond respiratory signs, host factors modify risk. Underweight, incomplete universal immunisation, lack of exclusive breastfeeding, anaemia, and delayed presentation have all been linked to severe disease or prolonged hospitalisation [9,15–17]. Radiographic consolidation similarly predicted antibiotic change and longer stay in an All India Institute of Medical Sciences (AIIMS) severe-pneumonia cohort [4]. Severity scores derived in high-income emergency departments are not routinely applied on Indian paediatric wards [8,14], and data from private tertiary hospitals in South India—where the case-mix includes both referred and first-contact admissions—remain sparse.

This study was therefore designed to quantify the frequency of severe in-hospital outcomes and to identify readily available admission predictors in children hospitalised with CAP at a Bengaluru teaching hospital over one year. The working hypothesis was that hypoxaemia and WHO danger signs would show the strongest association with the composite severe endpoint, with nutritional and immunisation gaps contributing additional unadjusted risk.

METHODS

Design and setting

This prospective observational study was conducted in the Department of Paediatrics, Sathagiri Institute of Medical Sciences and Research Centre, Bengaluru, Karnataka, over 12 consecutive months. The department provides paediatric emergency, inpatient ward, and PICU services to urban and peri-urban children. Reporting follows the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement [12].

Participants

Children of either sex, aged 2 months to 12 years, hospitalised with CAP were eligible. CAP was defined as an acute illness (≤ 14 days) with cough or difficult breathing plus age-specific tachypnoea and/or chest indrawing, with or without radiographic infiltrate, occurring in the community or within 48 hours of hospital admission, consistent with revised WHO guidance [1,18].

Exclusion criteria were hospital-acquired pneumonia, haemodynamically significant congenital heart disease, known

immunodeficiency, pre-existing chronic lung disease, suspected tuberculosis on admission, incomplete medical records, and declined parental consent.

Sample size

A one-year consecutive sample of 50–60 children was planned to match expected CAP admissions to the unit. For an anticipated hypoxaemia prevalence of approximately 35% [6] and a severe-outcome rate of approximately 30%, detection of an odds ratio of 4.5 at two-sided $\alpha = 0.05$ and 80% power requires about 50 children. Fifty-six children completed the study and were included in the analysis.

Definitions

Hypoxaemia was defined as peripheral oxygen saturation (SpO_2) $< 92\%$ in room air, matching the BTS hospitalisation threshold also used in Indian acute-care work [3,10]. Underweight was a WHO weight-for-age z-score (WAZ) < -2 . Incomplete immunisation was any missing age-appropriate Universal Immunisation Programme (UIP) antigen. Exclusive breastfeeding was defined as breast milk only for the first 6 months of life, or until the age at enrolment if the infant was younger than 6 months. WHO general danger signs were inability to drink or breastfeed, vomiting everything, convulsions, lethargy or unconsciousness, or stridor in a calm child [1,18]. Anaemia was haemoglobin < 11 g/dL. An abnormal leukocyte count was a white-cell count < 5 or $> 15 \times 10^9/\text{L}$. An abnormal chest radiograph was consolidation, infiltrate, or pleural collection reported by the duty paediatrician and reviewed by a consultant.

The primary composite severe outcome was any of the following: PICU admission; non-invasive ventilation or invasive mechanical ventilation; length of stay ≥ 7 days; a pneumonia-related complication (pleural effusion or empyema, pneumothorax, sepsis, or respiratory failure); or in-hospital death. Individual components were also described. Categories were not mutually exclusive. Operational definitions are summarised in the Appendix.

Data collection and clinical management

A structured proforma captured demography, feeding and immunisation history, household crowding, duration of symptoms before admission, vital signs including pulse oximetry, anthropometry, danger signs, laboratory investigations (complete blood count and chest

radiograph in all; blood culture when clinically indicated), treatment, and daily progress until discharge or death. Children were managed according to Indian Academy of Pediatrics (IAP) and WHO antibiotic and oxygen pathways [13,18]. Oxygen was titrated to $\text{SpO}_2 \geq 94\%$. The decision for PICU transfer followed unit protocol (persistent hypoxaemia, work of breathing, haemodynamic instability, or need for ventilatory support).

Statistical analysis

Categorical variables were summarised as number (percentage) and compared with the χ^2 test or two-sided Fisher exact test as appropriate. Continuous variables were summarised as mean $\pm$ standard deviation or median (interquartile range). Unadjusted odds ratios with Woolf 95% confidence intervals were calculated from 2×2 tables. Variables with $P < 0.10$ on univariable analysis were considered for binary logistic regression. Because only 18 primary events occurred, the multivariable model was restricted a priori to three clinically pre-specified covariates (WHO danger signs, hypoxaemia, and $\text{WAZ} < -2$) to limit overfitting (events-per-variable ≈ 6). Model calibration was described with the

Hosmer–Lemeshow goodness-of-fit test and Nagelkerke R^2 . A two-sided $P < 0.05$ was considered statistically significant. Analyses were performed with IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA).

Ethics

The study protocol was approved by the Institutional Ethics Committee of Sathgiri Institute of Medical Sciences and Research Centre, Bengaluru. Written informed consent was obtained from parents or legally authorised representatives. Assent was obtained from children aged 7 years or older when clinically appropriate. Confidentiality of identifiers was maintained throughout.

RESULT

Enrolment

Seventy-eight children with suspected CAP were screened during the study year. Twenty-two were excluded: hospital-acquired pneumonia ($n = 5$), haemodynamically significant congenital heart disease ($n = 6$), chronic lung disease or immunodeficiency ($n = 3$), incomplete records ($n = 4$), and declined consent ($n = 4$). Fifty-six children were enrolled and analysed (Figure 1).

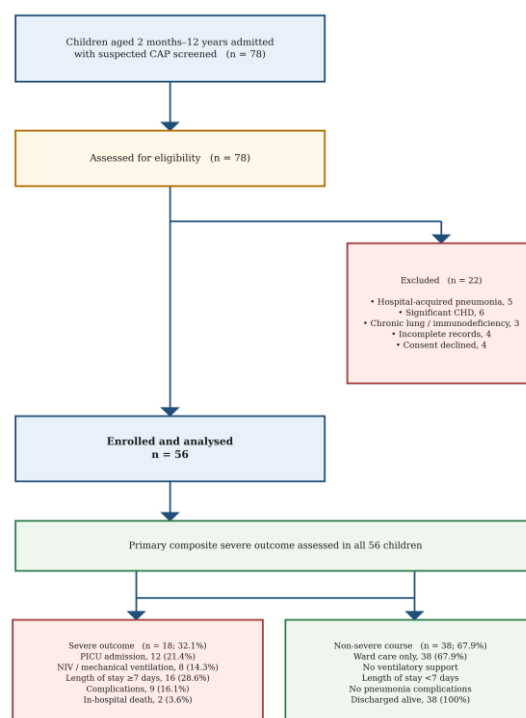


Figure 1. STROBE Flow of Study Participants.

CAP, community-acquired pneumonia; CHD, congenital heart disease; NIV, non-invasive ventilation; PICU, paediatric intensive care unit. Categories of severe outcome are not mutually exclusive.

Baseline profile

Mean age was 28.4 ± 22.1 months (median 24 months; interquartile range 9–42). Eighteen children (32.1%) were infants younger than 12 months, 28 (50.0%) were aged 12–59 months, and 10 (17.9%) were 5–12 years old. Boys comprised 34 of 56 (60.7%). Underweight was present in 21 (37.5%), incomplete immunisation in 16 (28.6%), and lack of

exclusive breastfeeding in 24 (42.9%). Symptom duration exceeded 3 days in 20 children (35.7%). Room-air hypoxaemia was recorded in 19 (33.9%) and WHO danger signs in 15 (26.8%). Anaemia was present in 23 (41.1%) and an abnormal chest radiograph in 38 (67.9%). Blood culture was obtained in 31 children (55.4%) and yielded a pathogen in 4 (7.1%) (Table 1).

Table 1. Baseline Demographic, Clinical, and Laboratory Characteristics of Children Hospitalised with Community-Acquired Pneumonia (n = 56)

Characteristic	n (%) or mean $\pm$ SD	95% CI / IQR where relevant
Demography		
Age, months, mean $\pm$ SD	28.4 $\pm$ 22.1	Median 24 (IQR 9–42)
Age <12 months	18 (32.1)	21.1–45.5
Age 12–59 months	28 (50.0)	37.3–62.7
Age 5–12 years	10 (17.9)	10.0–29.8
Male sex	34 (60.7)	47.6–72.4
Female sex	22 (39.3)	27.6–52.4
Host and social factors		
WAZ < -2 (underweight)	21 (37.5)	26.0–50.6
Incomplete immunisation (UIP)	16 (28.6)	18.4–41.5
No exclusive breastfeeding (first 6 months)	24 (42.9)	30.8–55.9
Symptom duration >3 days before admission	20 (35.7)	24.5–48.8
Admission physiology and signs		
SpO ₂ <92% in room air	19 (33.9)	22.9–47.0
WHO general danger signs present	15 (26.8)	16.9–39.7
Chest indrawing	41 (73.2)	60.4–83.0
Age-specific tachypnoea	48 (85.7)	74.3–92.6
Investigations		
Anaemia (haemoglobin <11 g/dL)	23 (41.1)	29.2–54.1
Abnormal leukocyte count (<5 or >15 $\times 10^9/L$)	21 (37.5)	26.0–50.6
Abnormal chest radiograph	38 (67.9)	54.8–78.6
Blood culture obtained	31 (55.4)	42.4–67.6
Pathogen isolated on blood culture	4 (7.1)	2.8–17.0

CI, confidence interval; IQR, interquartile range; SD, standard deviation; UIP, Universal Immunisation Programme; WAZ, weight-for-age z-score; WHO, World Health Organization. Binomial 95% CIs use the Wilson method.

In-hospital outcomes

A severe composite outcome occurred in 18 of 56 children (32.1%; 95% CI 21.4–45.2). PICU admission was required in 12 (21.4%), ventilatory support in 8 (14.3%), hospital stay ≥ 7 days in 16 (28.6%), and pneumonia-related complications in 9 (16.1%): pleural effusion or empyema in 5, sepsis in 3, and pneumothorax

in 1. Two children died (case fatality 3.6%); both were infants who presented with hypoxaemia and danger signs (Table 2). Median length of stay was 5 days (interquartile range 3–8) overall, 8 days (6–12) in the severe-outcome group and 4 days (3–5) among the remainder. All 38 children without a severe outcome were discharged alive.

Table 2. In-Hospital Outcomes among Children Hospitalised with Community-Acquired Pneumonia (n = 56)

Outcome	n	% of 56	95% CI (%)
Primary composite endpoint			

Any severe outcome*	18	32.1	21.4–45.2
Components of the composite (not mutually exclusive)			
PICU admission	12	21.4	12.7–33.8
Non-invasive or invasive ventilation	8	14.3	7.4–25.7
Length of stay ≥ 7 days	16	28.6	18.4–41.5
Any pneumonia-related complication	9	16.1	8.7–27.8
Pleural effusion / empyema	5	8.9	3.9–19.3
Sepsis	3	5.4	1.8–14.6
Pneumothorax	1	1.8	0.3–9.4
In-hospital death	2	3.6	1.0–12.1
Length of stay			
Median LOS, all children (IQR), days	5 (3–8)	—	—
Median LOS, severe-outcome group (IQR), days	8 (6–12)	—	—
Median LOS, non-severe group (IQR), days	4 (3–5)	—	—
Discharged alive	54	96.4	87.9–99.0

*Composite severe outcome = PICU admission, NIV/invasive ventilation, LOS ≥ 7 days, pneumonia complication, or death. CI, Wilson 95% confidence interval; LOS, length of stay; NIV, non-invasive ventilation; PICU, paediatric intensive care unit.

Unadjusted predictors

Severe outcome was significantly more frequent among infants younger than 12 months (55.6% vs 21.1%; OR 4.69, 95% CI 1.39–15.77, $P = 0.010$), children with WAZ < -2 (61.1% vs 26.3%; OR 4.40, 1.34–14.48, $P = 0.012$), incomplete immunisation (OR 4.43, $P = 0.015$), no exclusive breastfeeding (OR 4.33, $P = 0.013$), delayed presentation beyond 3 days (OR 3.50, $P = 0.033$), SpO₂ $< 92\%$ (66.7%

vs 18.4%; OR 8.86, 2.47–31.79, $P = 0.0004$), WHO danger signs (61.1% vs 10.5%; OR 13.36, 3.28–54.39, $P < 0.001$), anaemia (OR 3.40, $P = 0.036$), and an abnormal chest radiograph (OR 5.82, $P = 0.020$). Male sex and an abnormal leukocyte count were not statistically significant (Table 3). The corresponding forest plot is shown in Figure 2, and predictor-specific outcome frequencies are shown in Figure 3.

Table 3. Unadjusted Association of Admission Variables with the Composite Severe Outcome

Predictor	Severe n = 18	Non-severe n = 38	OR	95% CI	P
Age < 12 months	10 (55.6)	8 (21.1)	4.69	1.39–15.77	0.010
Male sex	12 (66.7)	22 (57.9)	1.45	0.45–4.70	0.530
WAZ < -2	11 (61.1)	10 (26.3)	4.40	1.34–14.48	0.012
Incomplete immunisation	9 (50.0)	7 (18.4)	4.43	1.29–15.23	0.015
No exclusive breastfeeding	12 (66.7)	12 (31.6)	4.33	1.31–14.32	0.013
Symptoms > 3 days	10 (55.6)	10 (26.3)	3.50	1.08–11.36	0.033
SpO ₂ $< 92\%$ in room air	12 (66.7)	7 (18.4)	8.86	2.47–31.79	0.0004
WHO danger signs	11 (61.1)	4 (10.5)	13.36	3.28–54.39	< 0.001
Anaemia (Hb < 11 g/dL)	11 (61.1)	12 (31.6)	3.40	1.06–10.96	0.036
Abnormal chest radiograph	16 (88.9)	22 (57.9)	5.82	1.17–28.96	0.020
Abnormal leukocyte count	10 (55.6)	11 (28.9)	3.07	0.96–9.83	0.055

Values are n (column %). Odds ratios are unadjusted (Woolf 95% CI). P values are from the χ^2 test or two-sided Fisher exact test. Rows in red tint are the two strongest unadjusted associations. Hb, haemoglobin; OR, odds ratio.

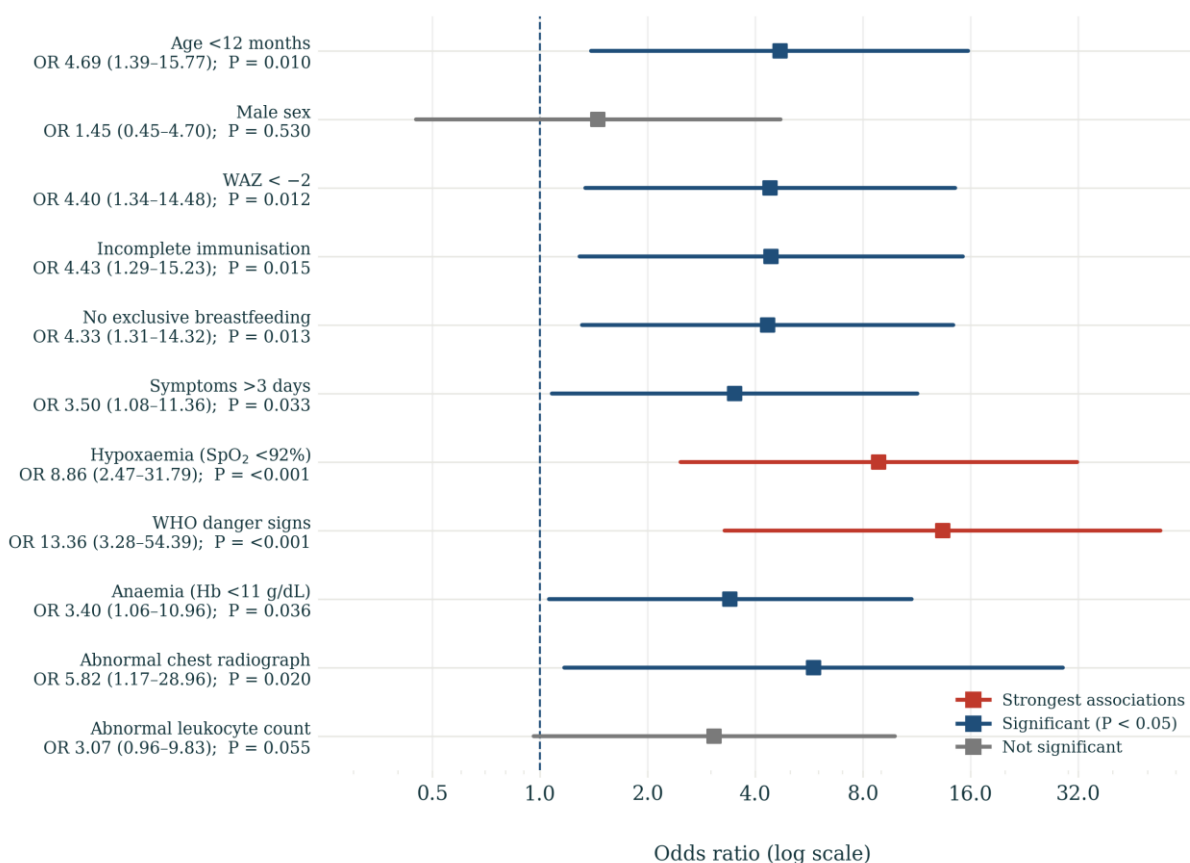


Figure 2. Forest Plot of Unadjusted Odds Ratios for the Composite Severe Outcome (n = 56). Squares mark the point estimate; horizontal bars are 95% confidence intervals on a logarithmic scale. The dashed vertical line indicates the null value (OR = 1). Red denotes the two strongest associations (WHO danger signs and hypoxaemia).

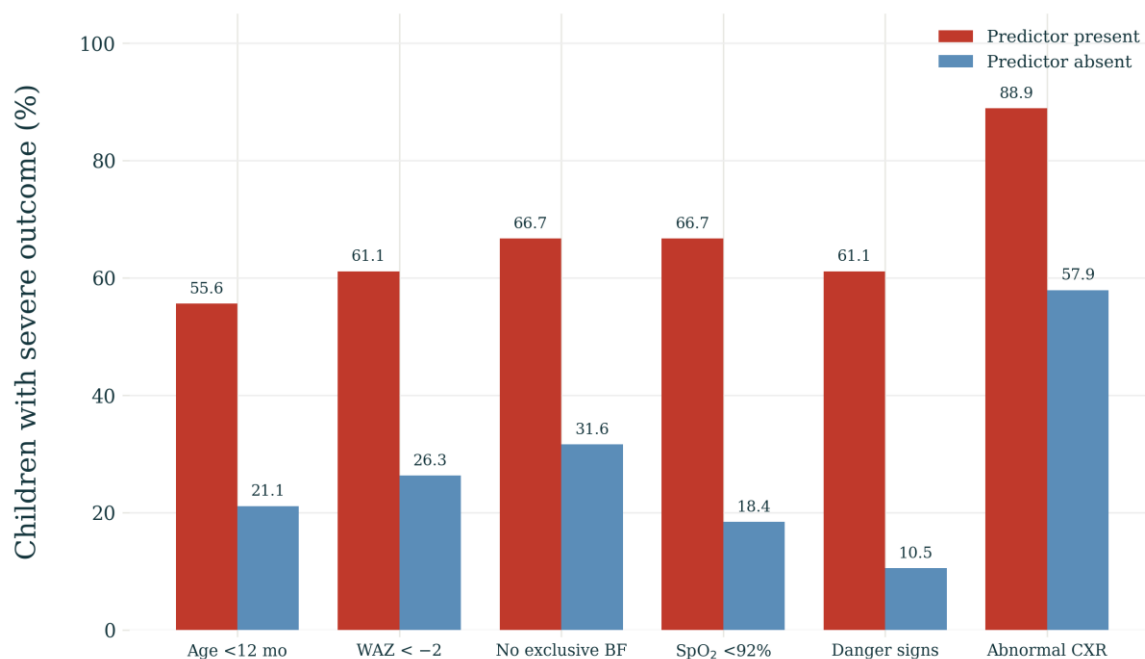


Figure 3. Frequency of the Composite Severe Outcome According to Presence or Absence of Six Admission Predictors. Percentages are row-specific (severe outcomes among children with versus without each predictor). BF, exclusive breastfeeding; CXR, chest radiograph; WAZ, weight-for-age z-score.

Multivariable analysis

In the pre-specified three-variable logistic model, WHO danger signs (aOR 8.12; 95% CI 1.62–40.71; $P = 0.011$) and hypoxaemia (aOR 5.94; 1.28–27.52; $P = 0.023$) were independently associated with the composite

severe outcome. Underweight was attenuated and no longer significant after adjustment (aOR 2.86; 0.68–12.05; $P = 0.152$) (Table 4). Nagelkerke R^2 was 0.42. The Hosmer–Lemeshow test did not indicate lack of fit ($P = 0.61$).

Table 4. Binary Logistic Regression for the Composite Severe Outcome (n = 56; 18 events)

Covariate	aOR	95% CI	Wald χ^2	P
WHO danger signs present	8.12	1.62–40.71	6.53	0.011
SpO ₂ <92% in room air	5.94	1.28–27.52	5.17	0.023
WAZ < -2	2.86	0.68–12.05	2.05	0.152
Model diagnostics	Nagelkerke $R^2 = 0.42$	Hosmer–Lemeshow $P = 0.61$	Events/variable ≈ 6	—

aOR, adjusted odds ratio. The model was limited a priori to three covariates because of 18 primary events. Entered covariates were pre-specified on clinical grounds (danger signs, hypoxaemia, underweight).

DISCUSSION

In this one-year tertiary-care cohort from Bengaluru, 32.1% of children hospitalised with CAP met a pragmatic severe-outcome definition, and 3.6% died. The case-mix—male predominance, high underweight prevalence, and hypoxaemia in about one-third of admissions—is consistent with Indian hospital series of childhood pneumonia [3–5]. Mortality was lower than in older north-Indian cohorts restricted to WHO “very severe” pneumonia (case fatality near 10%) [4], likely because the present sample included all hospitalised CAP rather than only the sickest stratum, and because PICU oxygen and non-invasive ventilation were available.

Danger signs and hypoxaemia were the strongest predictors (Table 3, Table 4, Figure 2), in keeping with IMCI logic and with evidence that hypoxaemia is among the most powerful mortality markers in childhood pneumonia [1,6,7,18]. Using SpO₂ <92% rather than <90% identified a larger at-risk group and is consistent with BTS and Indian acute-care practice [3,10]. An abnormal radiograph increased the odds of a severe course approximately six-fold, echoing the AIIMS observation that radiographic abnormality predicted antibiotic change and prolonged stay [4].

Modifiable host factors—underweight, incomplete immunisation, and lack of exclusive breastfeeding—were associated with severe outcome on unadjusted analysis (Figure 3). These associations are biologically coherent (impaired mucosal immunity, delayed care-seeking, greater bacterial burden) and have been reported from Indian and other low- and

middle-income datasets [9,15–17]. After adjustment, nutritional status lost conventional statistical significance, which is expected with only 18 events and collinearity with infant age and delayed presentation. The point estimate nevertheless remained in the harmful direction and should not be dismissed at the bedside.

Anaemia was associated with severe outcome univariately, consistent with the concept that reduced oxygen-carrying capacity compounds ventilation–perfusion mismatch in pneumonia [5]. Total leukocyte count was a weak discriminator, as expected in a mixed viral and bacterial CAP population in which a single white-cell threshold performs poorly [8].

Clinical implications

At admission, a calm SpO₂ <92% or any WHO danger sign should prompt early PICU review, controlled oxygen, and parenteral antibiotics, even if chest indrawing is the only respiratory sign [1,11,13,18]. Pulse oximetry must be universal and not reserved for children who “look sick” [6,7]. Nutrition and immunisation gaps identified on the same admission proforma are both prognostic markers and public-health targets [9,16]. In a unit of this size, a simple three-item screen (danger signs, SpO₂, WAZ) can be completed in minutes and does not require a laboratory turnaround.

Limitations

This was a single-centre study with 56 children and 18 primary events, so confidence intervals are wide and the multivariable model could include only three covariates. Viral polymerase-chain-reaction panels and induced-sputum microbiology were not obtained systematically. Chest radiographs were not double-read by a

blinded radiologist. The composite endpoint mixes intensity of care with biology; PICU admission can partly reflect local bed policy. High-income emergency-department severity models were not applied and would require local validation before use [8,14]. Findings should be confirmed in a larger Karnataka multicentre sample with aetiology and longer post-discharge follow-up.

CONCLUSION

Nearly one-third of children hospitalised with community-acquired pneumonia at this Bengaluru tertiary centre had a severe in-hospital course. WHO danger signs and room-air hypoxaemia (SpO₂ <92%) at presentation were independent predictors and are immediately actionable. Underweight, incomplete immunisation, lack of exclusive breastfeeding, anaemia, delayed presentation, and radiographic consolidation further stratify risk and should be recorded on every paediatric CAP admission sheet.

Acknowledgements

We thank the paediatric nursing staff, the PICU team, and the parents who consented to participation.

Declarations

Funding: This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflicts of interest: The authors declare that they have no competing interests.

Consent: Written informed consent was obtained from parents or legally authorised representatives; assent was obtained from children ≥7 years when appropriate.

Data availability: De-identified data are available from the corresponding author on reasonable request, subject to institutional policy.

REFERENCES

1. World Health Organization. Revised WHO classification and treatment of pneumonia in children at health facilities: evidence summaries. Geneva: World Health Organization; 2014.
2. Perin J, Mulick A, Yeung D, Villavicencio F, Lopez G, Strong KL, Prieto-Merino D, Cousens S, Black RE, Liu L. Global, regional, and national causes of under-5 mortality in 2000-19: an updated systematic analysis with implications for the Sustainable Development Goals. *Lancet Child Adolesc Health*. 2022;6(2):106-115.
3. Bhat JI, Charoo BA, Mukherjee A, Ahad R, Das RR, Goyal JP, Vyas B, Ratageri VH, Lodha R; ATU Group. Risk of hospitalization in under-five children with community-acquired pneumonia: a multicentric prospective cohort study. *Indian Pediatr*. 2021;58(11):1019-1023.
4. Tiewsoh K, Lodha R, Pandey RM, Broor S, Kalaivani M, Kabra SK. Factors determining the outcome of children hospitalized with severe pneumonia. *BMC Pediatr*. 2009;9:15.
5. Cao L, Ji Z, Zhang P, Wang J. Epidemiology and mortality predictors for severe childhood community-acquired pneumonia in ICUs: a retrospective observational study. *Front Pediatr*. 2023;11:1031423.
6. Rahman AE, Hossain AT, Nair H, Chisti MJ, Dockrell D, El Arifeen S, Campbell H. Prevalence of hypoxaemia in children with pneumonia in low-income and middle-income countries: a systematic review and meta-analysis. *Lancet Glob Health*. 2022;10(3):e348-e359.
7. Lazzerini M, Sonogo M, Pellegrin MC. Hypoxaemia as a mortality risk factor in acute lower respiratory infections in children in low and middle-income countries: systematic review and meta-analysis. *PLoS One*. 2015;10(9):e0136166.
8. Williams DJ, Zhu Y, Grijalva CG, Self WH, Harrell FE Jr, Reed C, Stockmann C, Arnold SR, Ampofo K, Anderson EJ, Bramley AM, Wunderink RG, McCullers JA, Pavia AT, Jain S, Edwards KM. Predicting severe pneumonia outcomes in children. *Pediatrics*. 2016;138(4):e20161019.
9. Chisti MJ, Tebruegge M, La Vincente S, Graham SM, Duke T. Pneumonia in severely malnourished children in developing countries: mortality risk, aetiology and validity of WHO clinical signs: a systematic review. *Trop Med Int Health*. 2009;14(10):1173-1189.
10. Harris M, Clark J, Coote N, Fletcher P, Harnden A, McKean M, Thomson A; British Thoracic Society Standards of Care Committee. British Thoracic Society guidelines for the management of community acquired pneumonia in children: update 2011. *Thorax*. 2011;66(Suppl 2):ii1-ii23.

11. Bradley JS, Byington CL, Shah SS, Alverson B, Carter ER, Harrison C, Kaplan SL, Mace SE, McCracken GH Jr, Moore MR, St Peter SD, Stockwell JA, Swanson JT. The management of community-acquired pneumonia in infants and children older than 3 months of age: clinical practice guidelines by the Pediatric Infectious Diseases Society and the Infectious Diseases Society of America. *Clin Infect Dis*. 2011;53(7):e25-e76.
12. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP; STROBE Initiative. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. *Lancet*. 2007;370(9596):1453-1457.
13. Sukumaran TU, Das RR, Warankar S; Indian Academy of Pediatrics. Community-acquired pneumonia. IAP Standard Treatment Guidelines 2022. Mumbai: Indian Academy of Pediatrics; 2022. Available from: <https://iapindia.org/pdf/Ch-011-Community-Acquired-Pneumonia.pdf>
14. Florin TA, Tancredi DJ, Ambroggio L, Babl FE, Dalziel SR, Eckerle M, Mintegi S, Neuman MI, Plint AC, Simon NJ, Kuppermann N; Pediatric Emergency Research Network. Predicting paediatric pneumonia severity in the emergency department: a multinational prospective cohort study of the Pediatric Emergency Research Network. *Lancet Child Adolesc Health*. 2025;9(6):383-392.
15. Dean P, Florin TA. Factors associated with pneumonia severity in children: a systematic review. *J Pediatric Infect Dis Soc*. 2018;7(4):323-334.
16. McCollum ED, King C, Hollowell R, Zhou J, Colbourn T, Nambiar B, Mukanga D, Burgess DCH. Predictors of treatment failure for non-severe childhood pneumonia in developing countries—systematic literature review and expert survey—the first step towards a community focused mHealth risk-assessment tool? *BMC Pediatr*. 2015;15:74.
17. Rudan I, O'Brien KL, Nair H, Liu L, Theodoratou E, Qazi S, Lukšić I, Fischer Walker CL, Black RE, Campbell H. Epidemiology and etiology of childhood pneumonia in 2010: estimates of incidence, severe morbidity, mortality, underlying risk factors and causative pathogens for 192 countries. *J Glob Health*. 2013;3(1):010401.
18. World Health Organization. Pocket book of hospital care for children: guidelines for the management of common childhood illnesses. 2nd ed. Geneva: World Health Organization; 2013.