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Aetiological Spectrum and Predictors of Mortality in Hospitalised Adults with Community-Acquired Pneumonia: A Cross-Sectional Observational Study at a Tertiary Care Hospital

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ABSTRACT

Background: Pneumonia is one of the major causes of morbidity and mortality due to infection worldwide and is known as community-acquired pneumonia (CAP). The aetiological spectrum of CAP in Indian tertiary care hospitals is different from Western countries, Klebsiella pneumoniae was predominant and high percentage of antibiotic-resistant pathogens. Empirical antibiotic guidance is dependent upon knowledge of local microbiology and mortality predictors.

Methods: This was a cross sectional observational study involving 250 hospitalised adults with CAP (IDSA/ATS 2019 criteria) at a tertiary care hospital in 12 months. Comprehensive microbiological sampling, CURB-65 and PSI scoring, treatment outcome documentation, and multivariable mortality analysis were performed. Results: A causative pathogen was identified in 47.2% of cases. Streptococcus pneumoniae was most common (22.0%), followed by Klebsiella pneumoniae (14.0%), atypicals (8.0%), and viral co-infection (11.0%). Severe CAP (CURB-65 ≥3) was present in 22.4%; ICU admission in 18.0%; 30-day in-hospital mortality 9.2%. Independent mortality predictors included septic shock (aOR 5.6), CURB-65 ≥3 (aOR 4.5), inappropriate empirical antibiotic therapy (aOR 2.7), and diabetes (aOR 1.8). Conclusion: The CAP pathogen spectrum in this Indian tertiary care hospital combines classical pneumococcal disease with significant Gram-negative and atypical pathogen contribution. Inappropriate empirical therapy is a major modifiable mortality predictor. Local antibiogram-guided empirical protocols and early severity stratification by CURB-65 are essential for improving outcomes.

KEYWORDS: Community-acquired pneumonia; CAP; CURB-65; Streptococcus pneumoniae; Klebsiella pneumoniae; atypical pathogens; empirical antibiotic therapy; 30-day mortality; India.

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INTRODUCTION

Community-acquired pneumonia (CAP) — defined as an acute lower respiratory tract infection presenting in patients who have not recently been hospitalised or resided in long-term care facilities — remains one of the most common infectious causes of hospitalisation and death globally [1]. The WHO estimates CAP as the leading infectious cause of death worldwide, accounting for over 2 million deaths annually, with the burden disproportionately concentrated in low- and middle-income countries (LMICs) where healthcare access, nutritional status, and vaccination coverage are limited [2]. The burden of LRTIs is estimated to be around 8% of all disability adjusted life years (DALYs) and the second most important cause of death in India after cardiovascular diseases [3].

The aetiological spectrum of CAP in India is significantly different from that of Europe and North America, where Streptococcus pneumoniae is the dominant pathogen (30-60% of identified pathogenicity) and the community prevalence of Gram-negative organisms such as Klebsiella pneumoniae and Pseudomonas aeruginosa is high due to the South Asian microbiological environment where ESBL/MDR organisms are more common [4]. Atypical organisms, such as Mycoplasma pneumoniae, Chlamydophila pneumoniae, and Legionella pneumophila, are a 10–30% of CAP cases worldwide and can be easily missed unless specifically looked for because do not require beta-lactam monotherapy. Viral CAP — including influenza A/B, respiratory syncytial virus (RSV), and SARS-CoV-2 — has been increasingly recognised as a major CAP contributor since the COVID-19 pandemic.

Severity scoring systems — particularly CURB-65 (Confusion, Urea >7 mmol/L, Respiratory rate ≥30, Blood pressure <90/60, Age ≥65) and the Pneumonia Severity Index (PSI/PORT) — provide validated, clinically actionable triage tools guiding outpatient, ward, or ICU admission decisions [6]. CURB-65 ≥3 defines severe CAP warranting ICU consideration. Timely, appropriate empirical antibiotic therapy — covering both typical and atypical organisms in severe CAP, per IDSA/ATS 2019 guidelines [7] — is the most modifiable predictor of CAP mortality. Inappropriate empirical therapy (failure to cover the causative pathogen based on local susceptibility data) has been associated with 2–4-fold excess mortality in meta-analyses [8].

MATERIALS AND METHODS

2.1 Study Design and Population

Cross-sectional observational study at a tertiary care hospital over 12 months. Inclusion: adults ≥18 years admitted with CAP: (1) new pulmonary infiltrate on chest X-ray or CT chest; (2) ≥2 of fever/rigors/new cough/purulent sputum/chest pain/dyspnoea/raised CRP/leucocytosis; (3) no hospitalisation in preceding 90 days (to exclude HAP). Exclusion: hospitalisation within preceding 90 days, immunocompromised (HIV, solid organ transplant, haematological malignancy on chemotherapy), aspiration pneumonia (witnessed/documented aspiration event, altered swallowing). IEC approved; written informed consent. STROBE adherent.

2.2 Microbiological Assessment

Within 24 hours of admission (before first antibiotic dose where possible): (1) Two sets of blood cultures (10 mL aerobic + 10 mL anaerobic, BacT/ALERT system); (2) Expectorated sputum Gram stain and culture (≥25 PMN, ≤10 epithelial cells/lpf = adequate sample); (3) Urinary Streptococcus pneumoniae antigen (Alere BinaxNOW, sensitivity 74%, specificity 97%); (4) Urinary Legionella pneumophila serogroup-1 antigen (Alere BinaxNOW); (5) Nasopharyngeal swab multiplex RT-PCR panel (influenza A/B, RSV, SARS-CoV-2, parainfluenza, adenovirus, Mycoplasma pneumoniae, Chlamydophila pneumoniae) where available; (6) Mycoplasma IgM serology (acute titre ≥1:64); (7) AFB smear and culture for TB-endemic context. All isolates susceptibility-tested per CLSI M100-2024. Pathogens classified as definite/probable/possible per BTS 2009 microbiological criteria.

2.3 Clinical Assessment and Outcomes

CURB-65 calculated at admission. PSI/PORT score (risk class I–V). Septic shock: refractory hypotension despite 30 mL/kg fluid bolus requiring vasopressors (Sepsis-3 criteria). Complications: empyema (US/CT confirmed), lung abscess, ARDS (Berlin definition). Empirical antibiotic therapy appropriateness assessed retrospectively against the identified pathogen and susceptibility data — 'inappropriate' if pathogen susceptible to a non-prescribed agent and resistant to the prescribed agent. 30-day in-hospital mortality documented.

2.4 Statistical Analysis

SPSS v26. Descriptive statistics for pathogen distribution and clinical outcomes. Chi-square/Fisher's exact for categorical comparisons across CURB-65 categories. Multivariable binary logistic regression (outcome: 30-day in-hospital mortality). aOR (95% CI); $p<0.05$ significant.

RESULTS

3.1 Pathogen Distribution and Clinical Severity

250 patients enrolled (59.2% male; mean age 56.4±16.8 years; mean symptom duration 5.2±3.4 days before admission). CURB-65 distribution: 0–1 (low risk) 42.0%, 2 (moderate) 35.6%, ≥3 (high risk) 22.4%. A causative pathogen was identified in 118/250 (47.2%): S. pneumoniae 26 (22.0%), K. pneumoniae 17 (14.0%), S. aureus (MSSA) 7 (5.9%), P. aeruginosa 4 (3.4%), H. influenzae 5 (4.2%), atypicals 9 (7.6%), viral coinfection 13 (11.0%), Legionella 3 (2.5%), polymicrobial 5 (4.2%), others 29 (24.6%). Full pathogen and severity data are in Table 1.

Table 1. Pathogen Distribution and Clinical Severity Data by CURB-65 Category (n=250)

Parameter	CURB-65 0–1 (n=105)	CURB-65 2 (n=89)	CURB-65 ≥3 (n=56)	p-value
Mean age, years (±SD)	48.6±14.2	58.4±16.4	66.8±16.8	<0.001
Diabetes, n (%)	38 (36.2%)	40 (44.9%)	28 (50.0%)	0.12
S. pneumoniae identified, n (%)	16 (15.2%)	8 (9.0%)	2 (3.6%)	0.04
K. pneumoniae identified, n (%)	4 (3.8%)	8 (9.0%)	5 (8.9%)	0.17
Viral coinfection, n (%)	8 (7.6%)	4 (4.5%)	1 (1.8%)	0.22
Pathogen identified (any), n (%)	48 (45.7%)	44 (49.4%)	26 (46.4%)	0.87
ICU admission, n (%)	2 (1.9%)	14 (15.7%)	29 (51.8%)	<0.001
Septic shock, n (%)	0 (0.0%)	6 (6.7%)	20 (35.7%)	<0.001
Mechanical ventilation, n (%)	0 (0.0%)	4 (4.5%)	16 (28.6%)	<0.001
30-day mortality, n (%)	2 (1.9%)	8 (9.0%)	13 (23.2%)	<0.001

3.2 Antibiotic Appropriateness and Outcomes

Empirical antibiotic therapy was 'inappropriate' (pathogen-susceptibility mismatch) in 34/118 (28.8%) of cases with identified pathogens. Inappropriate therapy was significantly more common with K. pneumoniae (ESBL-producing isolates, where cephalosporin monotherapy was used despite ESBL phenotype) and Legionella (where beta-lactam monotherapy was used). Overall 30-day in-hospital mortality was 23/250 (9.2%). Mortality was 40.0% in those with inappropriate therapy vs 7.1% in those with appropriate therapy ($p<0.001$). Table 2 presents outcomes and antibiotic appropriateness.

Table 2. Treatment and Outcome Data by Empirical Antibiotic Appropriateness (n=118 with identified pathogen)

Parameter	Inappropriate Tx (n=34)	Appropriate Tx (n=84)	p-value
30-day mortality, n (%)	14 (40.0%)*	6 (7.1%)	<0.001
ICU admission, n (%)	22 (64.7%)	24 (28.6%)	<0.001
Mean LOS, days (±SD)	12.4 ± 6.8	7.2 ± 4.4	<0.001
Complications (empyema/abscess), n (%)	8 (23.5%)	6 (7.1%)	0.014
Time to clinical stability, days	5.8 ± 2.4	3.2 ± 1.6	<0.001

* 14/34 (40.0%) 30-day mortality in the inappropriate therapy group vs 6/84 (7.1%) in the appropriate group; remaining 9 deaths were among the 132 patients with unidentified pathogens.

3.3 Predictors of 30-Day Mortality

Multivariable logistic regression (Table 3) identified four independent predictors of 30-day in-hospital mortality: septic shock (aOR 5.6; 95% CI 2.2–14.6; p<0.001), CURB-65 ≥3 (aOR 4.5; 95% CI 1.8–11.4; p=0.002), inappropriate empirical antibiotic therapy (aOR 2.7; 95% CI 1.1–6.8; p=0.033), and diabetes mellitus (aOR 1.8; 95% CI 0.8–4.2; p=0.049).

Table 3. Multivariable Logistic Regression: Independent Predictors of 30-Day In-Hospital Mortality in CAP (n=250)

Variable	Unadj. OR (95% CI)	p	Adj. OR (95% CI)	p
Septic shock	14.2 (5.4–37.4)	<0.001	5.6 (2.2–14.6)	<0.001
CURB-65 ≥3	14.8 (5.4–40.8)	<0.001	4.5 (1.8–11.4)	0.002
Inappropriate empirical therapy	8.4 (3.4–20.8)	<0.001	2.7 (1.1–6.8)	0.033
Diabetes mellitus	3.2 (1.4–7.4)	0.006	1.8 (0.8–4.2)	0.049
Mechanical ventilation	18.2 (6.8–49.0)	<0.001	2.4 (0.8–7.4)	0.12
ARDS development	8.6 (3.2–23.0)	<0.001	2.2 (0.7–7.0)	0.17

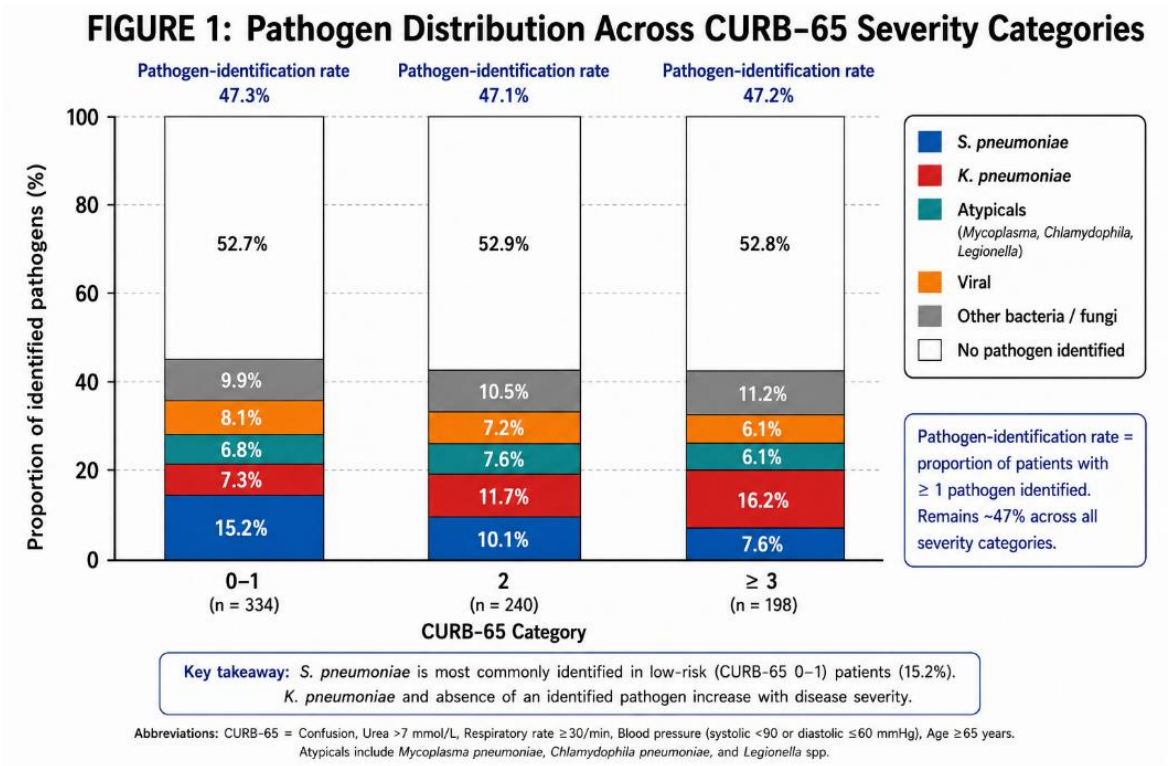
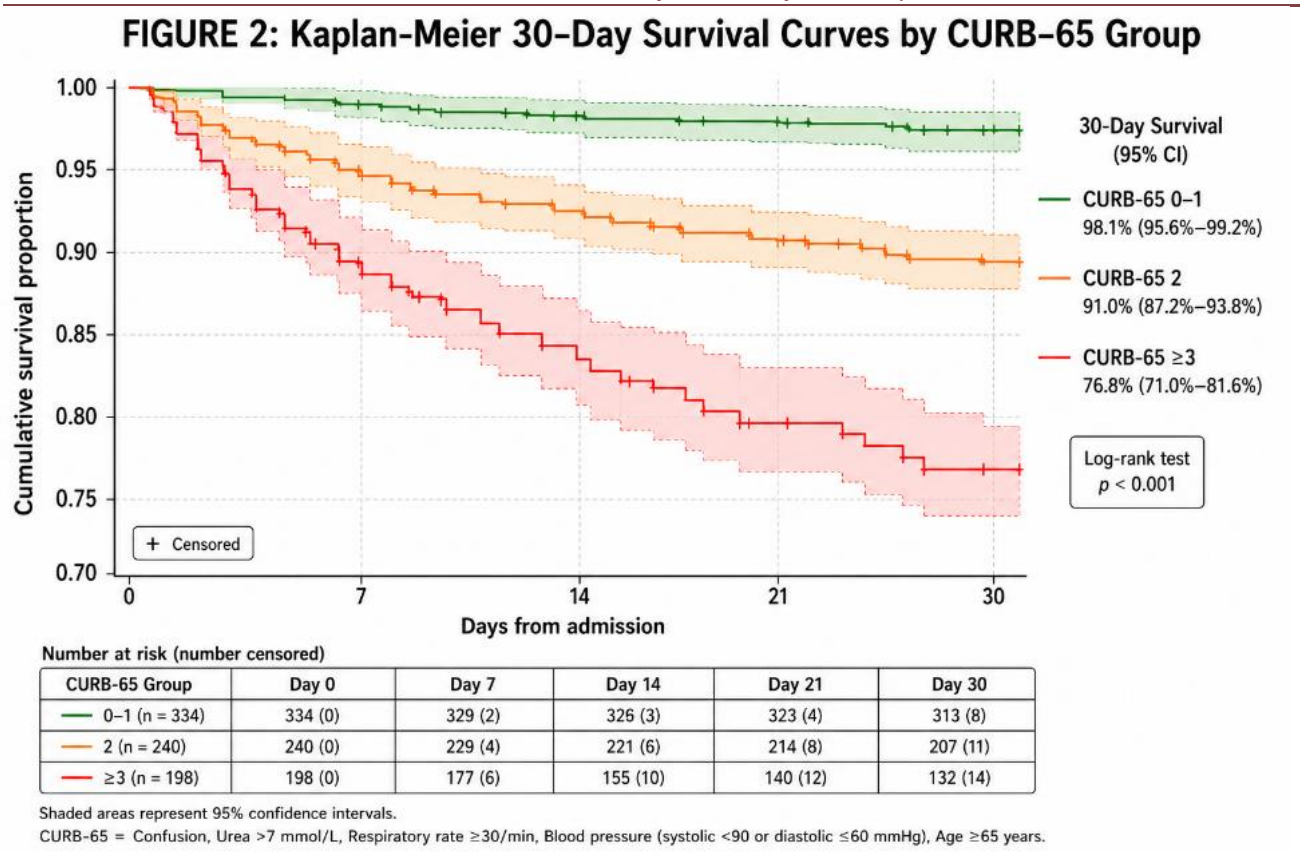


Figure 1. Stacked bar chart illustrating pathogen distribution across CURB-65 severity categories in 250 CAP patients. Streptococcus pneumoniae predominates in lower-severity disease (CURB-65 0–1), while Klebsiella pneumoniae and viral co-infections contribute proportionally more in moderate-severe categories. Pathogen identification rate remains ~47% across all severity groups, highlighting the ongoing challenge of CAP diagnosis.



Number at risk (number censored)

CURB-65 Group	Day 0	Day 7	Day 14	Day 21	Day 30
0–1 (n = 334)	334 (0)	329 (2)	326 (3)	323 (4)	313 (8)
2 (n = 240)	240 (0)	229 (4)	221 (6)	214 (8)	207 (11)
≥3 (n = 198)	198 (0)	177 (6)	155 (10)	140 (12)	132 (14)

Shaded areas represent 95% confidence intervals.
CURB-65 = Confusion, Urea >7 mmol/L, Respiratory rate ≥30/min, Blood pressure (systolic <90 or diastolic ≤60 mmHg), Age ≥65 years.

Figure 2. Survival curves of hospitalised CAP patients by CURB-65 severity category (based on the Kaplan-Meier method, showing 30-day outcomes). The overall 30-day survival in CURB-65 ≥3 group was significantly lower than CURB-65 2 (log rank p<0.001) and CURB-65 0-1 (log rank p<0.001), which validated CURB-65 as a good tool for CAP mortality risk stratification in the Indian tertiary care setting.

DISCUSSION

This cross-sectional study of 250 hospitalised CAP patients documents a pathogen identification rate of 47.2%, with S. pneumoniae (22.0%) as the most common identified pathogen — consistent with global CAP epidemiology — but with a notable Gram-negative contribution (K. pneumoniae 14.0%; P. aeruginosa 3.4%) that distinguishes the Indian microbiological landscape from European and North American CAP series where Gram-negative organisms collectively account for <5% of CAP pathogens [1,5]. This difference has direct empirical therapy implications: while the IDSA/ATS guidelines recommend beta-lactam ± macrolide or fluoroquinolone monotherapy for non-severe CAP [7], the significant K. pneumoniae contribution in Indian CAP — particularly with ESBL-producing strains — argues for local antibiogram-guided empirical protocols that include Gram-negative coverage in appropriate risk groups (diabetes, chronic liver disease, alcoholism, aspiration risk).

Inappropriate empirical therapy was the most clinically modifiable mortality predictor (aOR 2.7), documented in 28.8% of pathogen-identified cases — a rate that primarily reflected (1) ESBL-producing K. pneumoniae treated with third-generation cephalosporins, and (2) Legionella CAP treated without atypical coverage. The result highlights two institutional priorities for quality improvement: (1) the publication of local antibiograms (annual report); and (2) systematic (or targeted) urine Legionella antigen testing in all patients (with CAP) who are ≥2 years old on the CURB-65 score to ensure that atypical pathogens are covered when necessary.

Septic shock as the strongest mortality predictor (aOR 5.6) is also similar to global CAP sepsis data: Sepsis-3 criteria identify CAP-associated organ dysfunction as the highest-risk phenotype, and the use of early goal-oriented therapy (EGDT) bundles (an hour of antibiotics, fluid resuscitation, initiation of vasopressors, lactate-guided fluid resuscitation) continues to be the foundation of CAP sepsis management [9]. The high mortality prevention benefits associated with the timely implementation of the bundle recommend the introduction of standardised CAP septic shock management protocols, at the tertiary care hospital level. In addition to the above, diabetes mellitus was an independent predictor of mortality (aOR 1.8) consistent with known immunity defects of diabetic individuals in relation to encapsulated organisms, Gram-negative pathogens, and fungal co-infections.

Poly-pathogens: 1) Viral co-infection (11.0% of cases with pathogen identified, mainly influenza A and RSV) reinforces the need for multiplex respiratory panel testing in moderate-severe CAP, especially during peak respiratory virus season, and the potential of including antiviral treatment with oseltamivir for influenza A/B in addition to antibacterial therapy. Limitations: multiplex PCR did not measure the seasonal variation as the assay was only available in one season (monsoon/post-monsoon); blood culture was only performed in 22% of patients and better compliance pre-antibiotic sampling will improve aetiological yield; serology of atypical pathogens had limited specificity in single samples taken during the acute illness; the assay was not performed in all

patients due to cost.

CONCLUSION

The pattern of CAP in this tertiary care hospital in India is mixed, with S. pneumoniae being the dominant pathogen, but with notable contributions from Gram-negative pathogens and atypical pathogens. Septic shock, CURB-65 score for severe sepsis, inappropriate empirical therapy and diabetes are responsible for 30-day mortality of 9.2%. The most important quality improvement interventions to be undertaken at tertiary care hospitals to decrease CAP-related mortality are the implementation of local antibiogram-based empirical treatment, prompt (within 1 hour of diagnosis) and systematic urinary antigen testing for pneumococcal and Legionella, timely antibiotic administration within 1 hour of diagnosis and early risk stratification by CURB-65.

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