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Original Research Article

Clinical Profile and Microbiological Spectrum of Community-Acquired Pneumonia with Antimicrobial Susceptibility Pattern

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Abstract

Background: CAP continues to be a leading cause of hospitalisation and mortality and the microbial composition and susceptibility to antimicrobials are highly variable across geographical regions and local antibiotic pressure.

Objective: To describe the clinical characteristics, microbiological spectrum and antimicrobial susceptibility patterns of adults admitted to hospital with CAP and to explore the association between these and severity of illness.

Methods: This was a prospective observational study of 180 adults with clinical and radiographic CAP in a tertiary-care teaching hospital. Demographic data, symptoms, comorbidities, CURB-65 severity, laboratory and radiographic data, microbiological data, antimicrobial susceptibility, treatment course and outcomes were documented. Selected antigen and molecular tests were added to conventional cultures.

Results: The mean age was 56.8 ± 17.1 years and 112 (62.2%) participants were male. Common comorbidities included hypertension (39.4%), diabetes mellitus (34.4%) and chronic obstructive pulmonary disease (26.7%). In 104 (57.8%) patients, a microbiological etiology was found. A total of 92 isolates were obtained from 88 patients, with Klebsiella pneumoniae (26.1% of isolates) and Streptococcus pneumoniae (23.9%) being the most common, followed by Haemophilus influenzae (14.1%) and Pseudomonas aeruginosa (10.9%). Extended-spectrum beta-lactamase production was found in 45.8% of K. pneumoniae and 95.5% of S. pneumoniae isolates, while only 68.2% of S. pneumoniae isolates were susceptible to azithromycin. MDROs were found in 27 (15.0%) patients, and were more common in severe CAP than non-severe CAP (36.7% vs 10.7%, p=0.001). Severe CAP was also associated with longer hospitalization (10.8 ± 4.7 vs 6.1 ± 2.8 days, p<0.001), greater ICU admission (60.0% vs 4.0%, p<0.001), and higher in-hospital mortality (26.7% vs 2.0%, p<0.001).

Conclusions: CAP in hospitalized adults had a mixed spectrum of pneumococcal and Gram-negative bacteria, and clinically important resistance was seen, particularly with K. pneumoniae. Empiric therapy depends on local microbiological surveillance and susceptibility.

Keywords: Community-Acquired Pneumonia; Antimicrobial Resistance; Streptococcus Pneumoniae; Klebsiella Pneumoniae; Antimicrobial Susceptibility; CURB-65.

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Introduction

Community-acquired pneumonia (CAP) is a sudden infection of the pulmonary parenchyma that occurs in the community and remains a significant burden at all ages. The latest Global Burden of Disease study has shown that lower respiratory infections are still one of the leading infectious causes of morbidity and mortality globally, and that older people are at a higher risk of death from these infections [1]. Modern reviews also stress that CAP is not a single microbiological entity but a clinical syndrome that is caused by bacterial, viral, and atypical pathogens, and that the relative importance of these pathogens varies by age, comorbidity, vaccines, season, diagnostic technology, and geography [2]. Clinical management starts even before the causative organism is known. The current American Thoracic Society guidelines focus on personalized diagnosis and treatment, the

incorporation of respiratory viral testing, re-evaluating the length of antibiotic treatment after clinical stability, and judicious use of adjunctive therapy [3]. The previous ATS/IDSA guideline also recommended a risk-based strategy for microbiological testing, site-of-care decisions, and empiric antibiotic treatment, and advised against unnecessary empiric coverage for anti-MRSA and antipseudomonal drugs unless validated risk factors exist [4]. Local epidemiology is especially relevant due to the fact that empiric regimens that are effective in one area may not be in another.

The diagnosis of CAP is complex, as shown by the large prospective studies. In the U.S. EPIC study, a pathogen was found in a small number of hospitalized adults after extensive testing, and respiratory viruses were more likely to be found than bacteria [5]. Population-based data also indicate that the risk of hospitalization for CAP increases rapidly with age and that there is significant mortality associated with hospitalization for CAP, both short- and long-term [6]. Therefore, prognosis and management planning should take into account host factors and severity as well as pathogen identification.

Indian data show that there is significant heterogeneity in the etiology of CAP. In India, reviews of adult bacterial CAP have indicated that S. pneumoniae is still a significant pathogen, but several tertiary-care series have reported a significant contribution from Gram-negative bacilli, particularly Klebsiella pneumoniae and Pseudomonas aeruginosa [7].

A comprehensive North Indian study using culture, urinary antigens, serology and molecular methods identified S. pneumoniae most commonly, and also Legionella, influenza, Mycoplasma, Klebsiella, Staphylococcus aureus and Pseudomonas, showing the wide spectrum of possible pathogens [8]. A multi-centric study in New Delhi also identified clinically significant Mycoplasma pneumoniae and Legionella pneumophila infection in CAP patients [9]. Empiric treatment is becoming more difficult due to the rise of antimicrobial resistance (AMR). India has a high overall burden of antimicrobial consumption and resistance, and recent community-acquired respiratory pathogen susceptibility reviews show that there is an incomplete and geographically uneven susceptibility surveillance [10]. Local antibiograms are therefore vital to the balance of timely effective therapy and unnecessary exposure to wide-spectrum agents. The aim of the present study was to describe the clinical features and microbiological spectrum of hospitalized adults with CAP, to determine the antimicrobial susceptibility profile of the most common bacterial isolates, and to assess the relationship between antimicrobial resistance, severity of illness, and short-term clinical outcomes.

Materials and Methods

The study design and setting: A prospective observational study was carried out in the Departments of Medicine and Microbiology of a tertiary care teaching hospital in India for 12 months. The target sample size was calculated based on an expected microbiological positivity of about 45%, 95% confidence and an absolute precision of 8%, which resulted in 180 patients being included after allowing for incomplete specimens.

Eligibility Criteria: Eligible adults were those aged 18 years or older who had an acute respiratory illness with at least two of the following clinical features: fever or hypothermia, new or worsening cough, sputum production, dyspnea, pleuritic chest pain, or focal chest signs, and who had a new infiltrate on chest radiography or computed tomography at presentation or within 48 hours of admission. Patients were excluded if pneumonia occurred after 48 hours of hospitalisation, if there was a clear aspiration event, if there was already a diagnosis of pulmonary tuberculosis, if there was profound immunosuppression, or if there was an alternative non-infectious diagnosis which could account for the radiological opacity.

Clinical Assessment: A standardised case record form was used to record demographic details, smoking status, previous antibiotic use, vaccination history, comorbidities, symptoms, vital signs, oxygen saturation and examination findings. Complete blood count, renal and liver function tests, serum electrolytes, C-reactive protein and chest imaging were performed. CURB-65 score was determined on admission and divided into three groups: 0–1, 2, and ≥3. Severe CAP was considered as CURB-65 ≥3 or intensive care admission for respiratory or hemodynamic compromise.

Microbiological Methods

Specimens were collected prior to the first in-hospital dose of antibiotic when possible.

When microscopy indicated a satisfactory specimen from the lower respiratory tract, expectorated or induced sputum was accepted for culture. Sputum was Gram stained and cultured on suitable media, two sets of blood culture were taken and pleural fluid was cultured if present. Patients were tested for pneumococcal antigen, Legionella antigen in urine, viral molecular test of respiratory samples and Mycoplasma pneumoniae as clinically indicated and specimen available. Bacterial identification was done by conventional biochemical or automated methods.

Antimicrobial Susceptibility Testing: The antimicrobial susceptibility was tested by both standard disk diffusion and/or automated minimum inhibitory concentration (MIC) method and interpreted with the current laboratory breakpoints. Antibiotics were chosen based on the identity of the organism and were comprised of beta-lactam, beta-lactam/beta-lactamase inhibitor combinations, cephalosporins, macrolides, fluoroquinolones, aminoglycosides, carbapenems and anti-staphylococcal agents, as appropriate. Enterobacterales were recorded as ESBL phenotype. Multidrug resistance was considered as resistance to three or more antimicrobial categories.

Outcomes: The major results were microbiological yield, distribution of pathogens and antimicrobial susceptibility pattern. Secondary outcomes included length of hospital stay, admission to an intensive care unit (ICU), need for invasive mechanical ventilation, and in-hospital mortality.

Statistical Analysis: SPSS version 26.0 was used for data analysis. Continuous variables were presented as mean ± SD or median (IQR) depending on the distribution, and categorical variables were presented as frequency and percentage. Independent-samples t test or Mann–Whitney U test was used for continuous comparisons; chi-square or Fisher exact test was used for categorical comparisons. A two-sided p value <0.05 was considered statistically significant.

The number of deaths was small, so that mortality analyses were mainly univariable, to avoid unstable multivariable estimates.

Results

A total of 196 patients were screened; 16 were excluded because of hospital-acquired onset, established tuberculosis, aspiration pneumonia, or incomplete diagnostic criteria. The final analysis included 180 patients. Mean age was 56.8 ± 17.1 years (range 18–89 years), and 112 (62.2%) were male. At least one chronic comorbidity was present in 124 (68.9%) patients. Hypertension was most frequent (39.4%), followed by diabetes mellitus (34.4%) and chronic obstructive pulmonary disease (26.7%). Cough (91.1%), fever (86.7%), productive sputum (73.9%), and dyspnea (67.2%) were the dominant symptoms. Thirty patients (16.7%) had severe CAP by the prespecified definition, 24 (13.3%) required ICU admission, and overall in-hospital mortality was 6.1% (Table 1).

A microbiological etiology was established in 104 (57.8%) patients. Conventional bacterial cultures yielded 92 clinically significant isolates from 88 patients; four patients had two bacterial isolates. K. pneumoniae was the most frequent cultured organism (24 isolates; 26.1%), followed by S. pneumoniae (22; 23.9%), H. influenzae (13; 14.1%), P. aeruginosa (10; 10.9%), and S. aureus (9; 9.8%). Sixteen additional patients had a non-culture bacterial or viral etiology identified, including M. pneumoniae, influenza A, respiratory syncytial virus, and L. pneumophila (Table 2).

Among K. pneumoniae isolates, 11 of 24 (45.8%) demonstrated an extended-spectrum beta-lactamase phenotype and 4 (16.7%) were meropenem resistant. Susceptibility remained higher to amikacin (79.2%) and meropenem (83.3%) than to ceftriaxone (37.5%) or levofloxacin (54.2%). S. pneumoniae retained high susceptibility to ceftriaxone (95.5%) and amoxicillin-clavulanate (90.9%), whereas azithromycin susceptibility was lower (68.2%). All S. aureus isolates were susceptible to vancomycin and linezolid; three of nine (33.3%) were methicillin resistant. Detailed susceptibility results are presented in Table 3. Multidrug-resistant organisms were identified in 27 (15.0%) patients. MDR infection was significantly more frequent among severe CAP than non-severe CAP (36.7% vs 10.7%, p=0.001). Severe CAP patients were older (68.4 ± 12.8 vs 54.5 ± 16.9 years, p<0.001), more often had comorbidity (86.7% vs 65.3%, p=0.029), and more often had a Gram-negative bacterial pathogen (56.7% vs 32.7%, p=0.021). They also had longer hospitalization (10.8 ± 4.7 vs 6.1 ± 2.8 days, p<0.001), markedly higher ICU utilization (60.0% vs 4.0%, p<0.001), and greater mortality (26.7% vs 2.0%, p<0.001).

Table 1: Clinical and demographic profile of study participants (N=180)

Variable	Value
Age, years	56.8 ± 17.1
Male sex	112 (62.2%)
Current/former smoker	68 (37.8%)
Any comorbidity	124 (68.9%)
Hypertension	71 (39.4%)
Diabetes mellitus	62 (34.4%)
COPD	48 (26.7%)
Chronic kidney disease	16 (8.9%)
Fever	156 (86.7%)
Cough	164 (91.1%)

Productive sputum	133 (73.9%)
Dyspnea	121 (67.2%)
Pleuritic chest pain	39 (21.7%)
Confusion	21 (11.7%)
CURB-65 score 0–1	96 (53.3%)
CURB-65 score 2	54 (30.0%)
CURB-65 score ≥3	30 (16.7%)
ICU admission	24 (13.3%)
Invasive mechanical ventilation	14 (7.8%)
In-hospital mortality	11 (6.1%)

Table 2: Microbiological spectrum detected among adults with CAP

Pathogen	No. detected	Proportion
Klebsiella pneumoniae	24	26.1% of bacterial isolates
Streptococcus pneumoniae	22	23.9%
Haemophilus influenzae	13	14.1%
Pseudomonas aeruginosa	10	10.9%
Staphylococcus aureus	9	9.8%
Escherichia coli	6	6.5%
Acinetobacter baumannii	4	4.3%
Moraxella catarrhalis	4	4.3%
Mycoplasma pneumoniae	6	3.3% of all patients
Influenza A	5	2.8% of all patients
Respiratory syncytial virus	3	1.7% of all patients
Legionella pneumophila	2	1.1% of all patients

Note: Bacterial culture yielded 92 isolates from 88 patients; four patients had polymicrobial bacterial growth. Overall etiologic diagnosis was established in 104/180 (57.8%) patients.

Table 3: Antimicrobial susceptibility of major bacterial isolates

Organism	Antimicrobial tested	Susceptible, n (%)
K. pneumoniae (n=24)	Amoxicillin-clavulanate	8 (33.3%)
	Ceftriaxone	9 (37.5%)
	Piperacillin-tazobactam	15 (62.5%)
	Levofloxacin	13 (54.2%)
	Amikacin	19 (79.2%)
	Meropenem	20 (83.3%)
S. pneumoniae (n=22)	Penicillin	19 (86.4%)
	Amoxicillin-clavulanate	20 (90.9%)
	Ceftriaxone	21 (95.5%)
	Azithromycin	15 (68.2%)
	Levofloxacin	20 (90.9%)
H. influenzae (n=13)	Ampicillin	8 (61.5%)
	Amoxicillin-clavulanate	11 (84.6%)
	Ceftriaxone	13 (100%)
	Azithromycin	11 (84.6%)
	Levofloxacin	13 (100%)
P. aeruginosa (n=10)	Piperacillin-tazobactam	7 (70.0%)
	Ceftazidime	6 (60.0%)
	Cefepime	7 (70.0%)
	Amikacin	8 (80.0%)
	Levofloxacin	6 (60.0%)
	Meropenem	8 (80.0%)
S. aureus (n=9)	Cefoxitin	6 (66.7%)
	Clindamycin	6 (66.7%)
	Doxycycline	8 (88.9%)
	Linezolid	9 (100%)
	Vancomycin	9 (100%)

Additional resistance markers: ESBL phenotype in 11/24 (45.8%) K. pneumoniae isolates; meropenem resistance in 4/24 (16.7%); methicillin resistance in 3/9 (33.3%) S. aureus isolates.

Discussion

The results of this study highlight three clinically relevant characteristics of hospitalized CAP: the high prevalence of underlying disease, the presence of a polymicrobial flora, and the presence of a significant antimicrobial resistance problem. The microbiological yield (57.8%) was greater than in many previous series, but was less than in those studies using an extensive multimodal diagnostic panel. This is a typical pattern as factors such as prior outpatient antibiotic use, specimen quality, time of specimen collection and availability of molecular testing affect pathogen detection.

The predominance of K. pneumoniae and S. pneumoniae as the two most common cultured organisms was found, which is in accordance with the diverse Indian literature. Kalita et al. reported high prevalence of DRBIs among adults with CAP in Northeast India and emphasized the need of region-specific stewardship [11]. Khadanga et al. reported that the Gram-negative bacilli as a group were slightly more prevalent than pneumococcus and Pseudomonas and Klebsiella were found to be important pathogens [12]. Similarly, in an older hospitalized population from Kashmir, the Gram-negative organisms such as Pseudomonas, Escherichia coli and Klebsiella were found to be predominant [13]. The latter study by Para et al. in the North Indian region, however, had a wider spectrum of testing and pneumococcus was found to be the most common pathogen [8]. Combined, these reports indicate that Indian CAP is not a disease that can be managed based on the assumption of a single pathogen dominating the region and patient population.

Susceptibility to pneumococcal beta-lactams was relatively maintained in the current data set, with ceftriaxone having the highest rate of susceptibility, while susceptibility to azithromycin was lower. This pattern has implications for practice as macrolides are frequently used in atypical coverage, and occasionally as monotherapy in less severe respiratory infections. In a study of pneumococcal resistance in North India, significant resistance to cotrimoxazole, tetracycline and erythromycin was observed and no resistance to vancomycin was reported [14]. Recent genomic surveillance of pneumococcal lineages in Indian adults has revealed significant multidrug resistance and ongoing evolution of pneumococcal lineages [15]. Such surveillance populations are not the same as the hospitalized CAP populations, but the direction of the change is consistent with the need to continue local susceptibility monitoring and not assume that pneumococcal sensitivity is stable over time.

Gram-negative resistance was more of a concern. Almost half of the K. pneumoniae isolates were ESBL producers, susceptibility to ceftriaxone was low and a small proportion were carbapenem resistant. The clinical significance of this finding is that third generation cephalosporins are still frequently used in empiric inpatient CAP treatment. But indiscriminate escalation to carbapenems would be bad too. The Indian community-acquired respiratory AMR review highlights the importance of locally relevant susceptibility data and greater consistency in the implementation of guidelines [10]. Therefore, the initial antibiotic regimen should be modified according to the severity of illness, prior antibiotic use, structural lung disease, and recent microbiological history, with narrowing the antibiotic spectrum as soon as reliable culture results have been obtained.

In this study, non-culture diagnosis included Mycoplasma, influenza, respiratory syncytial virus and Legionella. They were not common, probably because molecular testing was not performed on all patients. Indian multicentric data have established M. pneumoniae and L. pneumophila as true pathogens of CAP [9] and larger modern CAP studies have shown that viral infection could contribute to a significant number of hospitalisations [5]. The results highlight the need to not define CAP microbiology solely by culture, especially in the setting of high seasonality and availability of rapid viral testing.

Clinical outcome was significantly affected by severity. Severe CAP patients were older, with more comorbidities, more frequent with Gram-negative and MDR infection, longer hospital stay, significantly higher use of ICU and higher mortality. These observations are consistent with current severe-CAP guidelines which focus on prompt identification of organ dysfunction and rapid escalation of treatment [16]. CURB-65 is simple and has been validated to correlate with mortality [17] while the Pneumonia Severity Index provides detailed risk stratification and is especially useful for identifying low-risk patients [18]. Severity scores should be used in conjunction with clinical judgment, particularly in patients with hypoxemia, rapidly progressive radiographic disease or significant frailty.

The overall mortality rate of 6.1% was within the wide range of mortality rates observed in CAP patients hospitalized and was observed among the severe subgroup. Small clinicomicrobiological series in India have also demonstrated that host factors, disease severity and etiologic diversity have an impact on outcome [19]. Importantly, the relationship between MDR pathogens and severe

CAP in this study does not imply that MDR pathogen is the cause of severity, as resistant infection may be a proxy for prior healthcare exposure, prior antimicrobial use, chronic lung disease, or other vulnerability.

The results have implications for stewardship. Therapy in non-severe CAP should be narrow and guideline-based, and should not include coverage of Gram-negative organisms unless the patient has proven epidemiologic or clinical risk. De-escalation can decrease unnecessary selection pressure in patients with cultures that show susceptibility to pneumococcus or H. influenzae. However, if the clinical response is inadequate, early detection of ESBL-producing or CPE Enterobacterales is important. Complex, pathogen-specific resistance mechanisms have been shown in major Gram-negative organisms, such as K. pneumoniae, P. aeruginosa, Acinetobacter baumannii, and Escherichia coli, in multicentric genomic work conducted in India, which justifies investment in rapid diagnostics and local antibiogram-based protocols.

There are some limitations of this study. It is one tertiary care center and may over-represent older, comorbid or more severe patients than community clinics. Culture yield may have been affected by previous antibiotic use. Not all patients had molecular and antigen testing, which may have under-estimated viral and atypical causes. The sample size was sufficient for descriptive aims, but small for modelling outcomes of organisms and multivariable mortality analysis. Lastly, susceptibility patterns are local and need to be updated regularly before being used to inform empiric treatment policy.

Conclusion

There was significant clinical variability and a wide spectrum of microorganisms cultured from hospitalized adults with CAP, with K. pneumoniae and S. pneumoniae the most common. The most worrying antimicrobial resistance profile was observed for Gram-negative isolates, and pneumococci had a relatively high beta-lactam susceptibility, but lower macrolide susceptibility. Early severity assessment, microbiological sampling, local antibiograms, and antibiotic de-escalation based on susceptibility were associated with improved resource use and adverse outcomes, highlighting the importance of early CAP severity assessment, microbiological sampling, local antibiograms, and antibiotic de-escalation based on susceptibility.

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