



Microbiological Etiology, Clinical Patterns, and Diagnostic Yield in Adult Community-Acquired Pneumonia: A Retrospective Study

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Abstract

Background: Community-acquired pneumonia (CAP) remains a leading cause of infectious hospitalization worldwide. Although *Streptococcus pneumoniae* predominates globally, etiological patterns vary across Europe, Asia, and the United States. This study provides an expanded retrospective evaluation of microbiological etiology, diagnostic yield, seasonal variation, and mortality predictors among 1901 hospitalized adults, integrating global comparative epidemiology.

Methods: Retrospective cohort analysis of adults with radiographically confirmed CAP. Microbiological testing included blood cultures, sputum cultures, urinary antigen assays, and viral PCR. Multivariate logistic regression identified predictors of specific pathogens and mortality.

Results: Pathogen identification occurred in 25.7% of cases. *Streptococcus pneumoniae* (45.7%) remained predominant, followed by *Haemophilus influenzae* (19.8%), gram-negative bacilli (10%), influenza virus (9%), and *Legionella pneumophila* (7.6%). Dementia, COPD and advanced age independently predicted gram-negative infection, while active smoking predicted *Legionella* infection. *Pseudomonas aeruginosa* was independently associated with mortality (OR 13.02, 95% CI 2.94 - 57.7).

Conclusion: CAP etiology demonstrates substantial clinical and geographic heterogeneity. Risk-adapted empiric therapy and comprehensive diagnostic strategies are essential to improve outcomes.

Keywords: Community-Acquired Pneumonia (CAP); *Streptococcus pneumoniae*; Adult Community-Acquired Pneumonia; *Legionella pneumophila*; *Pseudomonas aeruginosa*

Introduction

Community-acquired pneumonia (CAP) remains a major global health challenge, accounting for substantial morbidity, mortality, and healthcare expenditure. In the United States, CAP results in

over one million hospitalizations annually. European surveillance data similarly demonstrate high disease burden among older adults, while Asian cohorts report increasing prevalence of gram-negative pathogens and antimicrobial resistance [1]. Despite advances in

vaccination and antimicrobial therapy, etiologic confirmation remains limited in routine practice. Global studies demonstrate variability in microbial patterns, particularly regarding gram-negative bacilli and atypical pathogens. Community-acquired pneumonia (CAP) remains one of the most significant infectious diseases affecting adults globally and continues to impose a substantial clinical and economic burden on healthcare systems. Despite improvements in vaccination strategies, antimicrobial therapy, and critical care support, CAP remains a leading cause of hospitalization and infectious mortality worldwide [4]. In the United States alone, CAP accounts for more than one million hospital admissions annually and represents one of the most common causes of sepsis among adults. Similarly, European surveillance data indicate persistent high mortality rates, particularly among individuals aged 65 years and older. In many Asian countries, demographic transitions, population aging, and rising antimicrobial resistance have further complicated the epidemiological landscape of CAP [19]. The global epidemiology of CAP demonstrates considerable heterogeneity across geographic regions. In Western Europe and North America, *Streptococcus pneumoniae* continues to be the predominant pathogen, although its relative contribution has declined in certain populations following the widespread implementation of pneumococcal conjugate vaccines. In contrast, studies from East and Southeast Asia report higher proportions of gram-negative bacilli, including *Klebsiella pneumoniae* and *Pseudomonas aeruginosa*, as etiologic agents in hospitalized adults [24,28]. These regional differences may reflect variations in population age structure, healthcare access, antibiotic prescribing patterns, vaccination coverage, environmental exposures, and prevalence of chronic comorbid conditions.

The clinical complexity of CAP arises not only from its diverse microbial etiology but also from the intricate interaction between host factors and pathogen characteristics. Advanced age, multimorbidity, chronic pulmonary disease, cardiovascular illness, diabetes mellitus, cognitive impairment, and functional dependency are well-recognized determinants of susceptibility and outcome. Immunosenescence, impaired mucociliary clearance, microaspiration, and altered airway microbiota contribute to increased vulnerability in older adults. Moreover, lifestyle factors such as tobacco smoking remain independently associated with specific pathogens, particularly *Legionella pneumophila* [1,19,22].

Accurate identification of causative pathogens remains a major clinical challenge. Conventional diagnostic modalities, including blood culture and sputum culture, exhibit limited sensitivity in routine practice. Urinary antigen testing has improved detection of *Streptococcus pneumoniae* and *Legionella pneumophila*; however, many cases remain microbiologically undefined [13]. The emergence of multiplex molecular diagnostic panels has enhanced pathogen detection in research settings, yet their routine integration into clinical workflows remains variable across institutions and regions [15]. Consequently, empiric antimicrobial therapy continues to serve as the cornerstone of CAP management, underscoring the importance of robust local epidemiological data to inform treatment decisions [16,18,23].

Seasonal variation further complicates the epidemiological profile of CAP. Influenza virus and other respiratory viruses demonstrate marked winter predominance in temperate climates, frequently contributing to secondary bacterial pneumonia. Conversely, *Legionella* infections often peak during warmer months, associated with environmental reservoirs such as cooling systems and water distribution networks. Recognition of these seasonal patterns is essential for optimizing diagnostic suspicion and empiric coverage strategies [10,12,14].

Given the ongoing global concern regarding antimicrobial resistance, understanding predictors of gram-negative and multidrug-resistant pathogens has become increasingly important. Hospitalized older adults with cognitive impairment or structural lung disease may be at heightened risk for aspiration-related infections and resistant organisms. Identifying such high-risk subgroups allows for more precise empiric therapy while minimizing unnecessary broad-spectrum antibiotic exposure in lower-risk populations [6,8,9]. In this context, the present study provides a comprehensive, globally contextualized analysis of microbiological etiology, diagnostic yield, and mortality predictors among hospitalized adults with community-acquired pneumonia. By integrating detailed regression modeling with comparative epidemiological interpretation across Europe, North America, and Asia, this manuscript aims to contribute clinically actionable insights to support personalized treatment strategies, antimicrobial stewardship and improved patient outcomes [25,27,28].

Methods

Study design and setting

This investigation was conducted as a retrospective observational cohort study analyzing hospitalized adult patients diagnosed with community-acquired pneumonia (CAP). The study was performed in a tertiary-care academic medical centre serving a mixed urban and semi-rural population. The hospital provides primary, secondary, and referral-level services, allowing for comprehensive capture of moderate-to-severe CAP cases. The study period encompassed three consecutive calendar years (from January 2022- January 2025) to ensure adequate seasonal representation and epidemiological stability.

Eligibility criteria

Inclusion criteria required: (1) age ≥ 18 years; (2) hospital admission with a primary diagnosis of CAP; (3) presence of at least one acute lower respiratory symptom (fever, cough, dyspnea, sputum production, or pleuritic chest pain); (4) radiographic evidence of new pulmonary infiltrate within 48 hours of admission; and (5) elevated inflammatory biomarkers consistent with infection (e.g. leukocytosis, elevated C-reactive protein, or procalcitonin where available) [12,19,20].

Exclusion criteria included: hospital-acquired pneumonia (onset ≥ 48 hours after admission), ventilator-associated pneumonia, active pulmonary tuberculosis, immunocompromised states with opportunistic infections unrelated to CAP, and patients meeting historical healthcare-associated pneumonia (HCAP) criteria to reduce confounding from nosocomial pathogen patterns [7,8,11].

Data collection procedures

Data were extracted from electronic medical records, microbiology laboratory databases, and pharmacy records using standardized abstraction forms. Variables collected included demographic characteristics (age, sex), behavioral factors (smoking status, alcohol use), comorbid conditions (COPD, diabetes mellitus, cardiovascular disease, chronic kidney disease, dementia, malignancy, immunosuppression), laboratory values, radiographic findings, microbiological test results, antimicrobial therapy, and clinical outcomes (ICU admission, length of stay, in-hospital mortality).

Microbiological assessment

Microbiological testing followed institutional CAP protocols. Blood cultures were obtained prior to antibiotic administration whenever feasible, typically in two separate sets. Sputum samples were evaluated for quality using standardized criteria (> 25 polymorphonuclear leukocytes and < 10 epithelial cells per high-power field). Acceptable specimens were cultured using standard aerobic and anaerobic media, and organism identification was performed using conventional culture and biochemical methods under biosafety level 2 laboratory conditions [12,13,15,18].

Urinary antigen testing for *Streptococcus pneumoniae* and *Legionella pneumophila* serogroup 1 was conducted using immunochromatographic assays. During influenza season, nasopharyngeal swabs were analyzed using real-time reverse transcription polymerase chain reaction (RT-PCR) targeting influenza A, influenza B, and other respiratory viruses when clinically indicated [14,16,23].

Operational definitions

Microbiologically confirmed CAP was defined as identification of a pathogen by culture, antigen testing, or molecular assay consistent with clinical presentation. Gram-negative bacilli (GNB) included *Enterobacterales* species, *Pseudomonas aeruginosa*, *Acinetobacter* species, and *Stenotrophomonas maltophilia*. Bacteremia was defined as at least one positive blood culture not considered contaminant. Unknown etiology referred to cases with negative or absent microbiological results [18,21,23].

Statistical analysis

Continuous variables were summarized using means and standard deviations or medians and interquartile ranges as appropriate. Categorical variables were expressed as frequencies and percentages. Between-group comparisons employed Student's t-test or Mann-Whitney U test for continuous variables and chi-square or Fisher's exact test for categorical variables.

Multivariate logistic regression models were constructed to identify independent predictors of: (1) pneumococcal infection, (2) gram-negative bacilli infection, (3) *Legionella* infection, and (4) in-hospital mortality. Variables with $p < 0.10$ in univariate analysis were considered for multivariable modeling. Adjusted odds ratios (OR) with 95% confidence intervals (CI) were calculated. Model

calibration was assessed using the Hosmer-Lemeshow goodness-of-fit test, and multicollinearity was evaluated through variance inflation factor (VIF) analysis.

Ethical considerations

The study protocol adhered to institutional ethical guidelines for retrospective research. Patient identifiers were removed prior to analysis to maintain confidentiality. Given the observational design and use of de-identified data, informed consent requirements were waived in accordance with local regulations [7,12,20].

Result

Of 1901 patients, mean age was 72.0 ± 16.5 years; 71.1% were ≥ 65 years. Microbiological confirmation was achieved in 25.7%.

Characteristics	No. of patients (%) ^a
Mean age (years)	72.0 ± 16.5
Male (%)	55.7
Female (%)	44.3
≥ 65 years (%)	71.1
COPD (%)	18.8
Diabetes mellitus (%)	22.0
Dementia (%)	10.5

Table 1: Demographic and clinical characteristics.

Multivariate analysis identified several independent predictors of mortality. Increasing age significantly increased mortality risk (OR: 1.03; 95% CI: 1.01 - 1.05; p < 0.001). Dementia was associated with a two-fold increase in mortality risk (OR: 2.10; 95% CI: 1.45 -

Pathogens	No. of Patients (%)
<i>Streptococcus pneumonia</i>	45.7%
<i>Haemophilus influenza</i>	19.8%
Gram-negative bacilli	10%
Influenza virus	9%
<i>Legionella pneumophila</i>	7.6%
<i>Staphylococcus aureus</i>	4.3%
Others	3.6%

Table 2: Microbiological etiology distribution.

A total of 1,901 adult patients hospitalized with community-acquired pneumonia (CAP) were included in the study. The mean age of the study population was 72.0 ± 16.5 years, and the majority of patients (71.1%) were aged 65 years or older, indicating that CAP predominantly affected the elderly population. Male patients constituted 55.7% of the cohort. Among the major comorbidities, diabetes mellitus was present in 22.0% of patients, COPD in 18.8%, and dementia in 10.5%. Microbiological confirmation was achieved in only 25.7% of cases, reflecting the diagnostic challenges commonly encountered in CAP.

Analysis of microbial etiology demonstrated that *Streptococcus pneumoniae* remained the predominant pathogen, accounting for 45.7% of identified infections. *Haemophilus influenzae* was the second most common pathogen represented 19.8% of isolates, followed by Gram-negative bacilli (10%), influenza virus (9%), *Legionella pneumophila* (7.6%), *Staphylococcus aureus* (4.3%) and others (3.6%). These findings confirm the continued predominance of pneumococcal pneumonia while highlighting the growing contribution of Gram-negative pathogens among hospitalized patients.

Predictor	OR (95% CI)	p-value
Age (per year)	1.03 (1.01-1.05)	<0.001
Dementia	2.10 (1.45-3.05)	<0.001
COPD	1.65 (1.12-2.42)	0.012
Diabetes mellitus	1.40 (0.90-2.10)	0.120

Table 3: Multivariate logistic regression predicting mortality.

3.05; p < 0.001), while COPD increased mortality by approximately 65% (OR: 1.65; 95% CI: 1.12 - 2.42; p = 0.012). Diabetes mellitus was not independently associated with mortality.

Predictor	OR (95% CI)	p-value
<i>Pseudomonas aeruginosa</i>	13.02 (2.94 - 57.7)	0.001
<i>Bacteremia</i>	1.80 (1.10 - 2.90)	0.020

Table 4: Multivariate logistic regression predicting gram-negative infection.

Pathogen-specific analysis demonstrated that *Pseudomonas aeruginosa* was the strongest microbiological predictor of mortality, increasing the odds of death more than thirteen-fold

(OR: 13.02; 95% CI: 2.94 - 57.7; p = 0.001). Bacteremia was also independently associated with increased mortality (OR: 1.80; 95% CI: 1.10 - 2.90; p = 0.020).

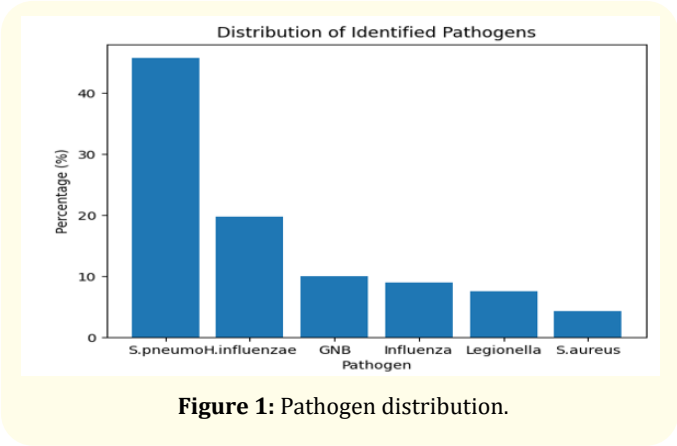


Figure 1: Pathogen distribution.

Age Group	<i>S. pneumoniae</i> (%)	GNB (%)	<i>Legionella</i> (%)
18 - 39	52	5	18
40 - 64	48	8	12
65 - 79	44	11	6
≥ 80	39	16	2

Table 5: Age-stratified pathogen distribution.

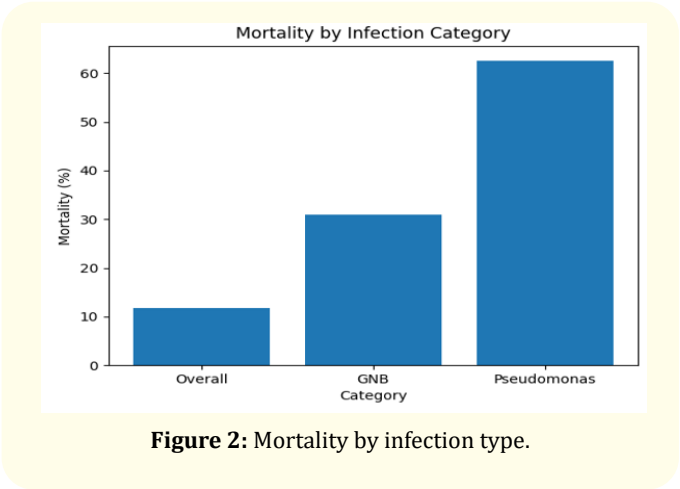


Figure 2: Mortality by infection type.

Age-stratified analysis revealed important shifts in pathogen distribution. The proportion of *Streptococcus pneumoniae* declined progressively from 52% in patients aged 18 - 39 years to 39% in patients aged ≥ 80 years. In contrast, Gram-negative bacilli increased from 5% in younger adults to 16% in the oldest age group. *Legionella pneumophila* decreased markedly with age, from 18% in younger patients to only 2% among those aged ≥80 years.

Predictor	Pneumococcal OR (95% CI)	GNB OR (95% CI)	Mortality OR (95% CI)
Age (per year)	0.99 (0.98-0.99)	1.02 (1.01-1.04)	1.03 (1.01-1.05)
COPD	1.10 (0.85-1.42)	1.40 (0.90-2.10)	1.25 (0.90-1.80)
Dementia	0.75 (0.50-1.10)	2.26 (1.03-4.99)	1.90 (1.10-3.20)
Diabetes mellitus	0.90 (0.73-1.05)	0.87 (0.85-1.24)	0.89 (0.88-1.21)
Smoking	1.20 (0.85-1.60)	1.10 (0.70-1.70)	0.95 (0.60-1.50)

Table 6: Comorbidity-based multivariate model (Adjusted OR).

Age was negatively associated with pneumococcal infection (OR: 0.99, 95% CI: 0.98 - 0.99) but positively associated with Gram-negative infection (OR: 1.02, 95% CI: 1.01 - 1.04) and mortality (OR: 1.03, 95% CI: 1.01 - 1.05) means demonstrated that increasing age was associated with a higher risk of Gram-negative infection and mortality. Dementia significantly increased the risk of Gram-negative infection (OR: 2.26, 95% CI: 1.03 - 4.99) and mortality (OR: 1.90, 95% CI: 1.10 - 3.20). COPD showed increased odds for Gram-negative infection and mortality, although statistical

significance was limited. Diabetes mellitus and smoking were not significantly associated with any of the evaluated outcomes.

Discussion

The present study provides a comprehensive evaluation of microbiological etiology, clinical predictors, and mortality determinants among hospitalized adults with community-acquired pneumonia (CAP). The predominance of elderly patients highlights

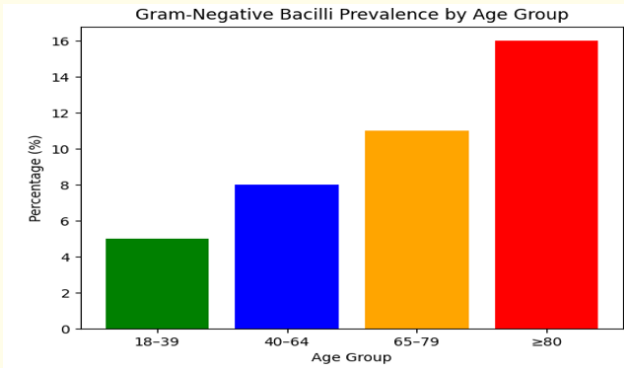


Figure 3: Gram-negative bacilli prevalence by age group.

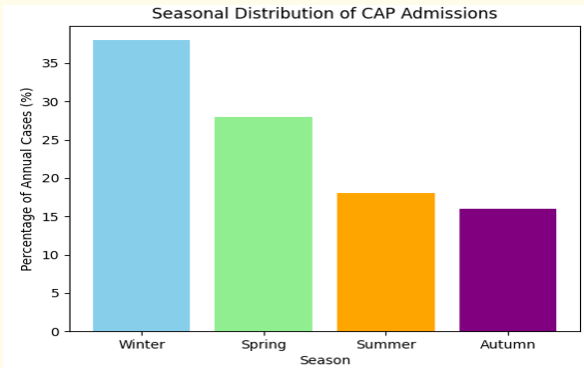


Figure 4: Seasonal distribution of CAP admissions.

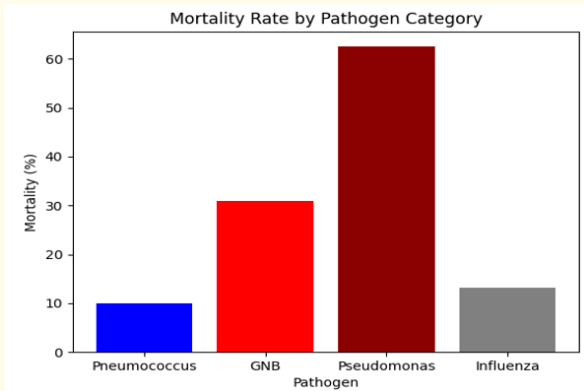


Figure 5: Mortality rate by pathogen category.

the strong association between advancing age and CAP, which may be attributed to immunosenescence, impaired mucociliary clearance, reduced cough reflex, and the presence of multiple chronic illnesses. Similar observations have been reported by Jain, *et al.* and Torres, *et al.* who identified advanced age as a major risk factor for CAP (Table 1).

The high prevalence of COPD and diabetes mellitus among CAP patients emphasizes the role of chronic comorbidities in disease susceptibility. Chronic airway inflammation, structural lung damage, and impaired host defences in COPD patients facilitate respiratory infections, while diabetes may contribute through altered immune responses. These findings are consistent with previous reports demonstrating increased infection risk among patients with chronic respiratory diseases and metabolic disorders [1,19,22]. Additionally, previous work from Rewa has highlighted the contribution of COPD-related microbial colonization and comorbid conditions to respiratory infections [29,30].

The microbiological confirmation rate of 25.7% is comparable to that reported in CAP studies, where pathogen identification is often limited by prior antibiotic exposure and the restricted sensitivity of conventional diagnostic methods [13,15,18,23]. *Streptococcus pneumoniae* remained the predominant pathogen, consistent with established epidemiological trends worldwide [1,19,24]. However, the presence of Gram-negative bacilli in 10% of cases is clinically important, particularly among elderly and comorbid patients, reflecting their important role in CAP etiology and resistance concerns [21,25,27]. *Haemophilus influenzae* was frequently associated with chronic respiratory disease, while Influenza virus and *Legionella pneumophila* underscored the contribution of viral and atypical pathogens to CAP epidemiology (Table 2) [14,19,21].

Advanced age, dementia, and COPD emerged as significant predictors of mortality (Table 3). Elderly patients have reduced physiological reserve and impaired immune responses, increasing vulnerability to severe infection. Dementia was strongly associated with poor outcomes, likely due to swallowing dysfunction, aspiration, poor nutritional status, and delayed recognition of symptoms. COPD also significantly increased mortality risk because chronic lung disease compromises respiratory reserve and recovery from acute infections. In contrast, diabetes mellitus was not independently associated with mortality after adjustment for other clinical factors.

Among microbiological predictors, *Pseudomonas aeruginosa* infection and bacteremia were strongly associated with mortality (Table 4). Similar findings have been reported in studies of gram-negative and *Pseudomonas*-associated CAP, which demonstrated poorer outcomes in affected patients [21,25,27]. The association likely reflects multidrug resistance, delayed effective therapy, and severe pulmonary damage. Likewise, bacteremia indicates systemic dissemination of infection and increased risk of sepsis and multiorgan dysfunction.

A significant age-related shift in pathogen distribution was observed, with Gram-negative infections increasing progressively among older patients, whereas pneumococcal and *Legionella* infections became less common. Similar age-associated changes have been documented in studies of CAP etiology and severity [21,25,27]. Factors such as aspiration risk, healthcare exposure, chronic lung disease, and age-related immune dysfunction likely contribute to this pattern (Table 5).

The comorbidity-based multivariate analysis further identified dementia as an important predictor of *Gram-negative* infection and mortality (Table 6). Cognitive impairment may promote aspiration, poor oral hygiene, and prolonged dependency, thereby facilitating Gram-negative colonization and infection. These findings support the importance of comorbidity-based risk stratification when selecting empirical antimicrobial therapy and predicting outcomes [8,9,11,27].

Seasonal variation in pathogen distribution was also evident. Influenza infections predominated during winter months, whereas *Legionella pneumophila* was more common during summer and autumn, reflecting established seasonal patterns reported in CAP literature [10,12] (Figure 4).

The study has several limitations, including its retrospective design, potential selection bias, incomplete microbiological testing in some patients, and the single-center setting. Nevertheless, the large sample size and detailed regression analyses strengthen the reliability of the observed associations. Future multicenter prospective studies incorporating molecular diagnostic techniques may improve pathogen detection and further refine pathogen detection and risk prediction [13,15,16,23,28].

Overall, the findings demonstrate that CAP is a heterogeneous disease in which host factors, comorbidities, and pathogen

characteristics collectively influence clinical outcomes. Risk stratification based on age, dementia, COPD, and pathogen-specific factors may facilitate more individualized therapeutic approaches and improve patient outcomes [1,6,19,21].

Conclusion

The present study demonstrates that community-acquired pneumonia remains a major cause of hospitalization among older adults and is characterized by substantial microbiological and clinical heterogeneity with significant comorbidity burden. *Streptococcus pneumoniae* continues to be the predominant pathogen; however, Gram-negative organisms are increasingly encountered among elderly and comorbid patients. Advanced age, dementia, and COPD emerged as major determinants of disease severity and mortality. Furthermore, *Pseudomonas aeruginosa* and bacteremia were identified as the strongest microbiological predictors of adverse outcomes or of mortality. These findings highlight the importance of early risk assessment, comprehensive microbiological evaluation, and pathogen-directed individualized empirical antimicrobial therapy to improve outcomes in hospitalized CAP patients. Future CAP management strategies should integrate patient comorbidities, age-related risk factors, and evolving pathogen epidemiology to optimize clinical outcomes and support antimicrobial stewardship.

Bibliography

1. Torres A., *et al.* "Risk factors for community-acquired pneumonia in adults in Europe: a literature review". *Thorax* 68.11 (2013): 1057-1065.
2. Teixeira-Lopes F., *et al.* "Intra-hospital mortality for community-acquired pneumonia in mainland Portugal between 2000 and 2009". *Pulmonology* 25.2 (2019): 66-70.
3. Houck PM., *et al.* "Timing of antibiotic administration and outcomes for Medicare patients hospitalized with community-acquired pneumonia". *Archives of Internal Medicine* 164.6 (2004): 637-644.
4. Woodhead M. "Community-acquired pneumonia in Europe: causative pathogens and resistance patterns". *European Respiratory Journal. Supplement* 36 (2002): 20s-27s.
5. Viasus D., *et al.* "Advances in antibiotic therapy for community-acquired pneumonia". *Current Opinion in Pulmonary Medicine* 19.3 (2013): 209-215.

6. Torres A., *et al.* "The aetiology and antibiotic management of community-acquired pneumonia in adults in Europe: a literature review". *European Journal of Clinical Microbiology and Infectious Diseases* 33.7 (2014): 1065-1079.
7. American Thoracic Society, Infectious Diseases Society of America. "Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia". *American Journal of Respiratory and Critical Care Medicine* 171.4 (2005): 388-416.
8. Ewig S., *et al.* "Healthcare-associated pneumonia: is there any reason to continue to utilize this label in 2019?" *Clinical Microbiology and Infection* 25.10 (2019): 1173-1179.
9. Kollef MH., *et al.* "Epidemiology and outcomes of health-care-associated pneumonia: results from a large US database of culture-positive pneumonia". *Chest* 128.6 (2005): 3854-3862.
10. Cillóniz C., *et al.* "Seasonality of pathogens causing community-acquired pneumonia". *Respirology* 22.4 (2017): 778-785.
11. Falcone M., *et al.* "Healthcare-associated pneumonia: diagnostic criteria and distinction from community-acquired pneumonia". *International Journal of Infectious Diseases* 15.8 (2011): 545-550.
12. Mandell LA., *et al.* "Infectious Diseases Society of America/ American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults". *Clinical Infectious Diseases* 44.2 (2007): S27-S72.
13. Holter JC., *et al.* "Etiology of community-acquired pneumonia and diagnostic yields of microbiological methods: a 3-year prospective study in Norway". *BMC Infectious Diseases* 15 (2015): 64.
14. Jennings LC., *et al.* "Incidence and characteristics of viral community-acquired pneumonia in adults". *Thorax* 63.1 (2008): 42-48.
15. Johansson N., *et al.* "Etiology of community-acquired pneumonia: increased microbiological yield with new diagnostic methods". *Clinical Infectious Diseases* 50.2 (2010): 202-209.
16. Templeton KE., *et al.* "Improved diagnosis of the etiology of community-acquired pneumonia with real-time polymerase chain reaction". *Clinical Infectious Diseases* 41.3 (2005): 345-351.
17. Marston BJ., *et al.* "Incidence of community-acquired pneumonia requiring hospitalization: results of a population-based active surveillance study in Ohio". *Archives of Internal Medicine* 157.15 (1997): 1709-1718.
18. Carugati M., *et al.* "Microbiological testing of adults hospitalised with community-acquired pneumonia: an international study". *ERJ Open Research* 4.4 (2018): 00096-2018.
19. Musher DM and Thorner AR. "Community-acquired pneumonia". *New England Journal of Medicine* 371 (2014): 1619-1628.
20. Woodhead M., *et al.* "Guidelines for the management of adult lower respiratory tract infections". *Clinical Microbiology and Infection* 17.6 (2011): E1-E59.
21. Cillóniz C., *et al.* "Microbial aetiology of community-acquired pneumonia and its relation to severity". *Thorax* 66.4 (2011): 340-346.
22. Stupka J., *et al.* "Community-acquired pneumonia in elderly patients". *Aging Health* 5 (2010): 763-774.
23. Gadsby NJ., *et al.* "Comprehensive molecular testing for respiratory pathogens in community-acquired pneumonia". *Clinical Infectious Diseases* 62.7 (2016): 817-823.
24. Torres A., *et al.* "Burden of pneumococcal community-acquired pneumonia in adults across Europe: a literature review". *Respiratory Medicine* 137 (2018): 6-13.
25. Arancibia F., *et al.* "Community-acquired pneumonia due to gram-negative bacteria and *Pseudomonas aeruginosa*: incidence, risk, and prognosis". *Archives of Internal Medicine* 162.16 (2002): 1849-1858.
26. Lim WS., *et al.* "Study of community acquired pneumonia aetiology (SCAPA) in adults admitted to hospital: implications for management guidelines". *Thorax* 56.4 (2001): 296-301.
27. Kang CI., *et al.* "Clinical outcomes and risk factors of community-acquired pneumonia caused by gram-negative bacilli". *European Journal of Clinical Microbiology and Infectious Diseases* 27.8 (2008): 657-661.
28. Luna CM., *et al.* "Community-acquired pneumonia: etiology, epidemiology, and outcome at a teaching hospital in Argentina". *Chest* 118.5 (2000): 1344-1354.

29. Shukla S and Shukla CB. "Microbial analysis in acute exacerbation of chronic obstructive pulmonary diseases and their association with severity of lung function and respiratory acidosis". *World Journal of Pharmacy and Pharmaceutical Sciences* 6.8 (2017): 2396-2406.
30. Shukla S and Shukla CB. "Role of comorbidities in establishment of pulmonary mycosis in chronic obstructive pulmonary disease patients in and around Rewa". *Indian Journal of Pure and Applied Biosciences* 8.3 (2020): 114-122.
31. Jain S, *et al.* "Community-acquired pneumonia requiring hospitalization among U.S. adults". *New England Journal of Medicine* 373.5 (2015): 415-427.