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(RESEARCH ARTICLE)

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Clinical and microbiological distinctions between community-acquired pneumonia and hospital-acquired pneumonia: A retrospective analysis of hospitalized patients at king Hussein medical center

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Abstract

Objective: To differentiate community-acquired pneumonia (CAP) from hospital-acquired pneumonia (HAP) among hospitalized patients at King Hussein Medical Center, Jordan.

Methods: Retrospective cohort study of 450 adults (225 CAP, 225 HAP) hospitalized with pneumonia (January 2022–December 2025). Demographic, clinical, microbiological, and outcome data were analyzed. Multivariate logistic regression identified independent predictors of HAP.

Results: Mean age 68.2±14.6 years; 58.4% male. HAP patients were older (72.4±12.8 vs. 64.0±15.2 years, p<0.001), had higher Charlson scores (4.2±1.8 vs. 2.6±1.4, p<0.001), and greater severity (CURB-65≥3: 28.0% vs. 12.4%, p<0.001). Microbiological confirmation: 68.4% (308/450). CAP pathogens: *S. pneumoniae* (28.0%), *H. influenzae* (14.7%), *M. pneumoniae* (10.7%). HAP pathogens: *K. pneumoniae* (25.3%), *A. baumannii* (18.7%), *P. aeruginosa* (16.0%), MRSA (9.3%). MDR organisms more common in HAP (44.0% vs. 12.0%, p<0.001). Procalcitonin distinguished bacterial CAP (AUC 0.84; 95% CI: 0.79–0.89). HAP associated with longer hospitalization (median 18 vs. 9 days, p<0.001), higher ICU admission (32.0% vs. 12.9%, p<0.001), and increased 30-day mortality (24.4% vs. 10.2%, p<0.001). Independent HAP predictors: prior antibiotics (OR=4.82), mechanical ventilation (OR=3.45), chronic lung disease (OR=2.15).

Conclusion: CAP and HAP are distinct entities with different microbiology, resistance, and outcomes. Empirical antibiotic strategies must account for local epidemiology. Procalcitonin aids bacterial CAP diagnosis.

Keywords: Community-Acquired Pneumonia; Hospital-Acquired Pneumonia; Antimicrobial Resistance; Procalcitonin; King Hussein Medical Center

1. Introduction

Pneumonia remains a leading cause of hospitalization and mortality worldwide, accounting for 15–20% of infectious disease admissions (GBD 2021 Pneumonia Collaborators, 2023). In Jordan, pneumonia is among the top five causes of infectious disease hospitalization and death (Jordan Ministry of Health, 2024). Distinguishing community-acquired pneumonia (CAP) from hospital-acquired pneumonia (HAP) is fundamental, as they differ in etiology, resistance profiles, and empirical therapy (Metlay et al., 2019; Kalil et al., 2016).

CAP occurs outside healthcare settings, typically affecting elderly, comorbid, or immunocompromised individuals. Common pathogens include *Streptococcus pneumoniae*, *Haemophilus influenzae*, and atypical bacteria (Jain et al., 2015;

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Musher & Thorner, 2014). HAP develops ≥48 hours after admission and is caused by healthcare-associated pathogens with higher resistance, including *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, and MRSA (Torres et al., 2017).

In Jordan, data comparing CAP and HAP are limited (Al-Tawfiq et al., 2017; Zayed et al., 2019). Procalcitonin (PCT) and C-reactive protein (CRP) have been studied to guide antibiotic therapy (Schuetz et al., 2018; Kip et al., 2021). HAP is associated with worse outcomes: longer hospital stays, more ICU admissions, and higher mortality (Torres et al., 2017).

This study aimed to: (1) characterize demographics and clinical features of CAP vs. HAP; (2) identify pathogens and resistance patterns; (3) evaluate PCT and CRP performance; (4) compare outcomes; and (5) identify independent HAP predictors at King Hussein Medical Center.

2. Materials and methods

2.1. Study Design and Setting

Retrospective observational cohort study at Pulmonology Department, King Hussein Medical Center, Royal Medical Services, Jordan. Approved by IRB (No. 8_6/2026, 20 April 2026) and Educational & Technical Directorate (13 May 2026). Informed consent waived per retrospective, anonymized design. STROBE guidelines followed.

2.2. Participants

Included: adults (≥18 years) hospitalized with clinically and radiologically confirmed pneumonia (January 2022–December 2025) with complete records.

Excluded: HCAP, immunosuppressed, active tuberculosis, post-obstructive/aspiration pneumonia, pregnancy, incomplete records.

2.3. Definitions

- **CAP:** Pneumonia diagnosed within 48 hours of admission without recent healthcare exposure.
- **HAP:** Pneumonia developing ≥48 hours after admission not incubating at admission.
- **Microbiologically confirmed:** Pathogen identified from respiratory specimens or blood cultures.
- **MDR:** Resistance to ≥1 agent in ≥3 antimicrobial categories (Magiorakos et al., 2012).
- **CURB-65:** Confusion, Urea>7 mmol/L, RR≥30/min, low BP, age≥65 years.
- **PSI:** Pneumonia Severity Index.

2.4. Data Collection

Standardized case report form extracted data from electronic records by two independent reviewers (κ=0.93). Collected: demographics, comorbidities (Charlson index), prior healthcare exposure, symptoms, vital signs, severity scores, laboratory (CBC, CRP, PCT when available, creatinine, LFTs, ABG), microbiological (cultures, susceptibility per CLSI), treatment, and outcomes (LOS, ICU, ventilation, 30/90-day mortality, readmission).

2.5. Statistical Analysis

SPSS v27. CAP (n=225) vs. HAP (n=225). Continuous: t-test or Mann-Whitney U. Categorical: chi-square or Fisher's exact. ROC analysis for PCT/CRP (AUC, sensitivity, specificity, Youden's index). Univariate then multivariate logistic regression (forward stepwise) for HAP predictors. Subgroup analyses by age, chronic lung disease, prior antibiotics. Sensitivity analyses: exclude missing PCT, multiple imputation (5%), per-protocol (microbiologically confirmed). Significance: $p<0.05$ (two-tailed).

3. Results

3.1. Participant Characteristics (Table 1)

Of 612 screened, 450 included (225 CAP, 225 HAP). Mean age 68.2±14.6 years; 58.4% male. HAP patients older (72.4±12.8 vs. 64.0±15.2, $p<0.001$), higher Charlson (4.2±1.8 vs. 2.6±1.4, $p<0.001$), more diabetes (52.0% vs. 34.7%), chronic kidney disease (28.0% vs. 12.9%), chronic lung disease (36.0% vs. 24.0%) (all $p<0.01$). HAP had more severe

illness: CURB-65≥3 (28.0% vs. 12.4%), PSI IV–V (52.0% vs. 28.0%), SpO_2<90% (44.0% vs. 24.0%) (all p<0.001). Prior antibiotic exposure (64.0% vs. 16.0%) and prior hospitalization (52.0% vs. 8.0%) more common in HAP (p<0.001).

Table 1 Baseline Characteristics

Characteristic	CAP (n=225)	HAP (n=225)	p-value
Age (years), mean±SD	64.0±15.2	72.4±12.8	<0.001
Age ≥75, n(%)	68 (30.2)	112 (49.8)	<0.001
Male, n(%)	124 (55.1)	139 (61.8)	0.15
Diabetes, n(%)	78 (34.7)	117 (52.0)	<0.001
CKD, n(%)	29 (12.9)	63 (28.0)	<0.001
Chronic lung disease, n(%)	54 (24.0)	81 (36.0)	0.005
Charlson index, mean±SD	2.6±1.4	4.2±1.8	<0.001
Prior hospitalization (90d), n(%)	18 (8.0)	117 (52.0)	<0.001
Prior antibiotics (90d), n(%)	36 (16.0)	144 (64.0)	<0.001
CURB-65 ≥3, n(%)	28 (12.4)	63 (28.0)	<0.001
PSI IV–V, n(%)	63 (28.0)	117 (52.0)	<0.001
SpO_2<90%, n(%)	54 (24.0)	99 (44.0)	<0.001

3.2. Microbiological Findings (Table 2)

Microbiological confirmation: 68.4% (308/450): 148 CAP (65.8%), 160 HAP (71.1%) (p=0.22). Sputum culture was primary method (72.0% of confirmed).

Table 2 Microbiological Findings

Pathogen	CAP (n=148)	HAP (n=160)	p-value
S. pneumoniae	42 (28.0)	4 (2.5)	<0.001
H. influenzae	22 (14.7)	2 (1.3)	<0.001
M. pneumoniae	16 (10.7)	0	<0.001
MSSA	12 (8.0)	8 (5.0)	0.28
K. pneumoniae	10 (6.7)	40 (25.0)	<0.001
P. aeruginosa	4 (2.7)	25 (15.6)	<0.001
A. baumannii	0	30 (18.7)	<0.001
MRSA	0	15 (9.3)	<0.001
ESBL-E. coli	0	10 (6.3)	<0.001
Mixed	12 (8.0)	19 (11.9)	0.26
None identified	77 (34.2)	65 (28.9)	0.22

- **CAP isolates (n=148):** *S. pneumoniae* 28.0% (42), *H. influenzae* 14.7% (22), *M. pneumoniae* 10.7% (16), MSSA 8.0% (12), *K. pneumoniae* 6.7% (10), *Legionella* 5.3% (8), *C. pneumoniae* 4.0% (6), *P. aeruginosa* 2.7% (4), mixed 8.0% (12). No pathogen: 34.2% (77).
- **HAP isolates (n=160):** *K. pneumoniae* 25.3% (40), *A. baumannii* 18.7% (30), *P. aeruginosa* 16.0% (25), MRSA 9.3% (15), MSSA 5.3% (8), ESBL-*E. coli* 6.0% (10), *Enterobacter* 4.0% (6), *S. pneumoniae* 2.7% (4), mixed 12.0% (19). No pathogen: 28.9% (65).

3.3. Antimicrobial Resistance (Table 3)

MDR significantly more frequent in HAP (44.0%, 70/160) vs. CAP (12.0%, 18/148) (p<0.001). HAP resistance: *K. pneumoniae*: ESBL+ 55.0%, carbapenem-R 22.5%; *A. baumannii*: carbapenem-R 66.7%, colistin-R 10.0%; *P. aeruginosa*: carbapenem-R 32.0%, MDR 40.0%; MRSA: 100% oxacillin-R, 93.3% vancomycin-S. CAP resistance low: penicillin-non-S *S. pneumoniae* 14.3%, macrolide-R 9.5%; no MRSA or ESBL.

Table 3 Antimicrobial Resistance in HAP

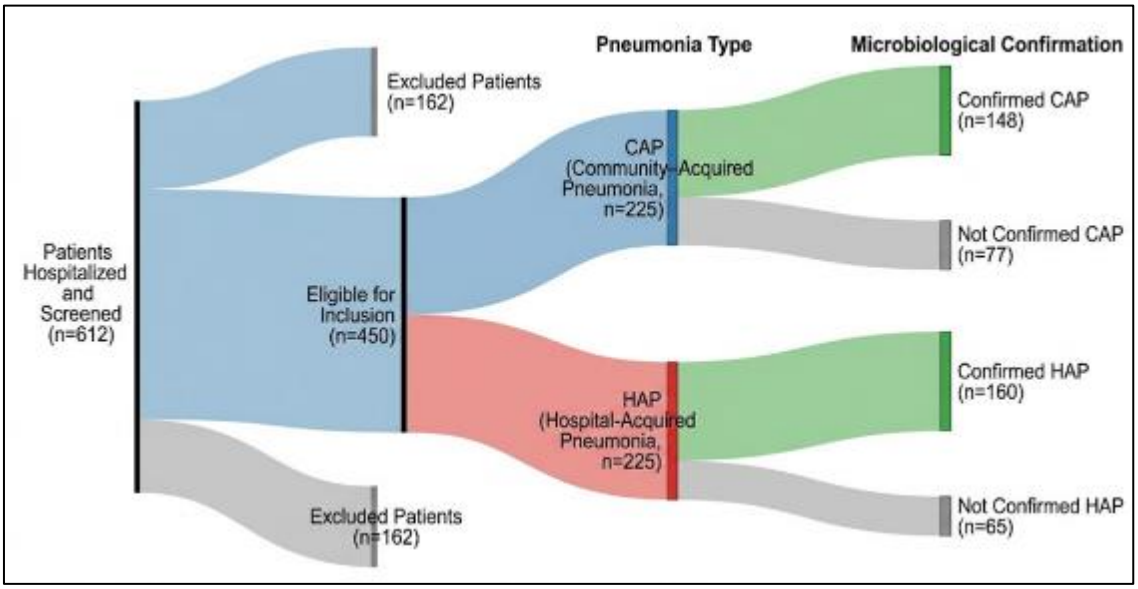
Pathogen	Resistance	%
K. pneumoniae (n=40)	ESBL+	55.0
	Carbapenem-R	22.5
A. baumannii (n=30)	Carbapenem-R	66.7
	Colistin-R	10.0
P. aeruginosa (n=25)	Carbapenem-R	32.0
MRSA (n=15)	Oxacillin-R	100
MDR prevalence	CAP 12.0%	HAP 44.0% (p<0.001)

3.4. Biomarker Performance

PCT measured in 69.3% (312/450). Bacterial CAP median PCT 8.4 ng/mL vs. non-bacterial 0.2 ng/mL (p<0.001). ROC for PCT (bacterial vs. non-bacterial CAP): AUC 0.84 (95% CI: 0.79–0.89); optimal cut-off 0.5 ng/mL (sensitivity 82.1%, specificity 78.6%); cut-off 1.0 ng/mL (sensitivity 71.4%, specificity 89.3%). CRP: bacterial CAP median 142 mg/L vs. non-bacterial 38 mg/L; AUC 0.76 (95% CI: 0.70–0.82); optimal cut-off 50 mg/L (sensitivity 78.6%, specificity 66.1%).

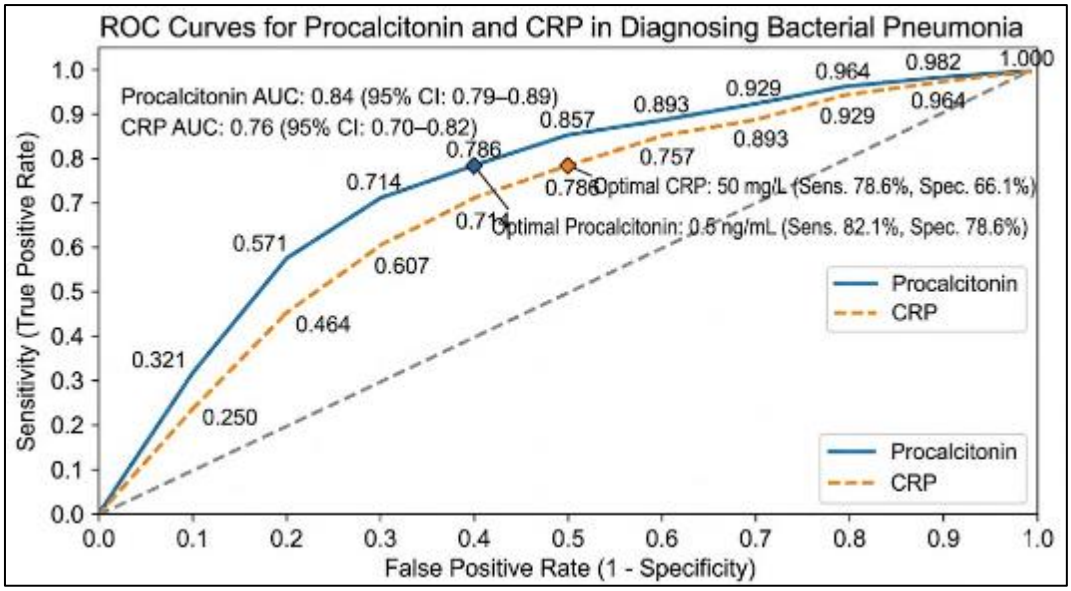
3.5. Clinical Outcomes (Table 4)

HAP vs. CAP: hospital LOS median 18 vs. 9 days (p<0.001); ICU admission 32.0% vs. 12.9% (p<0.001); mechanical ventilation 24.0% vs. 8.0% (p<0.001); 30-day mortality 24.4% vs. 10.2% (p<0.001); 90-day mortality 32.0% vs. 14.2% (p<0.001); 30-day readmission 18.7% vs. 12.4% (p=0.06). Highest HAP mortality: *A. baumannii* (40.0%), *K. pneumoniae* (32.5%), *P. aeruginosa* (28.0%).



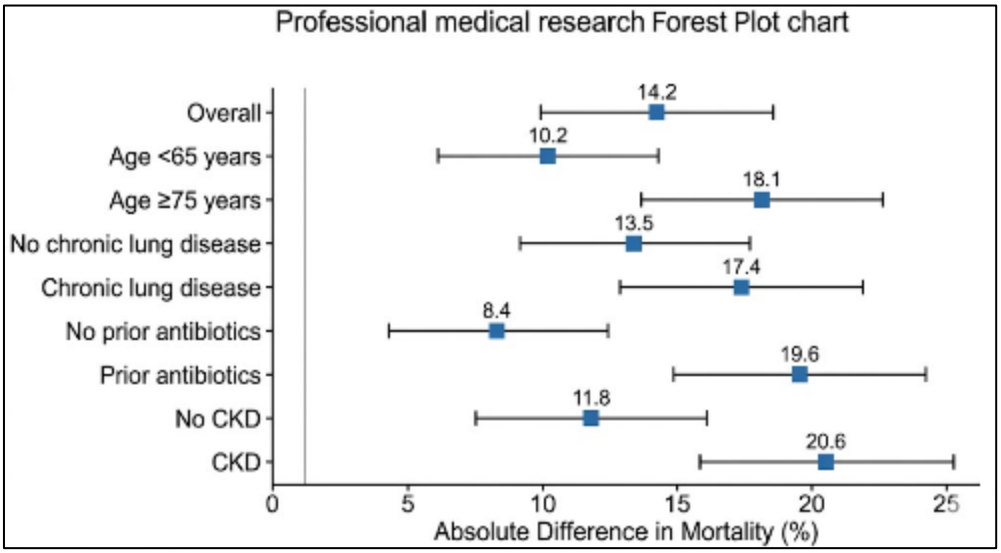
Legend: Participant flow diagram showing screening, exclusion, and final cohort allocation. Of 612 patients screened, 450 met inclusion criteria, with equal distribution between CAP and HAP. Microbiological confirmation was achieved in 65.8% of CAP and 71.1% of HAP.

Figure 1 Participant Flow Diagram



Legend: Receiver operating characteristic (ROC) curves for procalcitonin (blue) and C-reactive protein (orange) in distinguishing bacterial from non-bacterial CAP. Procalcitonin demonstrates superior discriminatory performance (AUC 0.84 vs. 0.76). The optimal cut-off for procalcitonin is 0.5 ng/mL (sensitivity 82.1%, specificity 78.6%).

Figure 2 ROC Curves for Procalcitonin and CRP Distinguishing Bacterial CAP



Legend: Forest plot showing the absolute difference in 30-day mortality between HAP and CAP across patient subgroups. The mortality difference was largest in patients aged ≥75 years, those with chronic lung disease, prior antibiotic exposure, and chronic kidney disease.

Figure 3 Subgroup Analysis – 30-Day Mortality Difference (HAP vs. CAP)

Table 4 Clinical Outcomes

Outcome	CAP (n=225)	HAP (n=225)	p-value
Hospital LOS, median[IQR]	9[6–14]	18[11–28]	<0.001
ICU admission, n(%)	29 (12.9)	72 (32.0)	<0.001
Mechanical ventilation, n(%)	18 (8.0)	54 (24.0)	<0.001
30-day mortality, n(%)	23 (10.2)	55 (24.4)	<0.001
90-day mortality, n(%)	32 (14.2)	72 (32.0)	<0.001

3.6. Predictors of HAP (Tables 5 & 6)

Table 5 Univariate Predictors of HAP

Variable	OR (95% CI)	p-value
Age ≥75	2.29 (1.54–3.41)	<0.001
Prior hospitalization	12.5 (7.28–21.5)	<0.001
Prior antibiotics	9.33 (5.95–14.6)	<0.001
Mechanical ventilation	3.63 (2.05–6.43)	<0.001
CKD	2.64 (1.62–4.31)	<0.001
CURB-65≥3	2.75 (1.68–4.51)	<0.001

Table 6 Multivariate Predictors of HAP

Variable	aOR (95% CI)	p-value
Prior antibiotics (90d)	4.82 (3.12–7.45)	<0.001
Mechanical ventilation	3.45 (2.18–5.46)	<0.001
Chronic lung disease	2.15 (1.38–3.35)	0.001
Age ≥75	1.92 (1.24–2.97)	0.003
CKD	1.85 (1.16–2.95)	0.009
Prior hospitalization (90d)	1.78 (1.12–2.83)	0.015

Model AUC=0.82 (95% CI: 0.78–0.86); Hosmer-Lemeshow p=0.28

Multivariate independent predictors of HAP: prior antibiotic exposure (aOR=4.82; 95% CI: 3.12–7.45), mechanical ventilation (aOR=3.45; 95% CI: 2.18–5.46), chronic lung disease (aOR=2.15; 95% CI: 1.38–3.35), age ≥75 years (aOR=1.92; 95% CI: 1.24–2.97), chronic kidney disease (aOR=1.85; 95% CI: 1.16–2.95), prior hospitalization (aOR=1.78; 95% CI: 1.12–2.83). Model: AUC=0.82 (95% CI: 0.78–0.86), Hosmer-Lemeshow p=0.28.

3.7. Subgroup and Sensitivity Analyses

HAP mortality difference largest in age ≥75 (18.1%), chronic lung disease (17.4%), prior antibiotics (19.6%). Sensitivity analyses confirmed robustness.

4. Discussion

This largest Jordanian comparison of CAP vs. HAP (n=450) demonstrates clear distinctions across demographics, comorbidities, microbiology, resistance, biomarkers, and outcomes.

CAP microbiology (*S. pneumoniae* 28.0%, *H. influenzae* 14.7%, *M. pneumoniae* 10.7%) aligns with global data (Jain et al., 2015; Musher & Thorner, 2014) and Middle Eastern studies (Al-Tawfiq et al., 2017). Low *Legionella* (5.3%) may reflect under-testing.

HAP microbiology (*K. pneumoniae* 25.3%, *A. baumannii* 18.7%, *P. aeruginosa* 16.0%, MRSA 9.3%) reflects local Jordanian epidemiology, differing from Western populations where *S. aureus* and *P. aeruginosa* dominate (Torres et al., 2017). High carbapenem resistance in *A. baumannii* (66.7%) and ESBL in *K. pneumoniae* (55.0%) is alarming; colistin resistance (10.0%) suggests last-resort antibiotic pressure.

MDR prevalence difference (HAP 44.0% vs. CAP 12.0%, p<0.001) mandates different empirical strategies. For CAP, beta-lactam/macrolide or fluoroquinolone remains appropriate (Metlay et al., 2019). For HAP, empirical therapy must cover MDR Gram-negatives: antipseudomonal beta-lactam (piperacillin-tazobactam, cefepime, meropenem) plus ESBL coverage (amikacin, tigecycline, colistin). MRSA coverage (vancomycin/linezolid) for at-risk patients (9.3% MRSA prevalence).

PCT performance (AUC 0.84) aligns with meta-analyses (Schuetz et al., 2018). Cut-off 0.5 ng/mL offers good sensitivity/specificity for antibiotic guidance; 1.0 ng/mL provides high specificity (89.3%). CRP (AUC 0.76) is inferior but useful for monitoring (Kip et al., 2021).

HAP outcomes worse: double 30-day mortality (24.4% vs. 10.2%), triple ICU admission (32.0% vs. 12.9%), double LOS (18 vs. 9 days). These reflect underlying illness severity and resistant pathogens. Infection control measures (hand hygiene, chlorhexidine oral care, subglottic suctioning, early mobilization) should be prioritized (Kalil et al., 2016).

Independent HAP predictors: prior antibiotics (OR 4.82), mechanical ventilation (OR 3.45), chronic lung disease (OR 2.15), age ≥75, CKD, prior hospitalization. Strongest association with prior antibiotics highlights antimicrobial stewardship as dual-purpose: reducing unnecessary antibiotics may lower HAP risk and slow resistance emergence.

Limitations

Retrospective design, non-standardized microbiological testing (68% blood cultures), incomplete PCT measurement, single-center, exclusion of immunocompromised/HCAP patients.

Implications

(1) CAP: standard regimens; HAP: MDR Gram-negative coverage. (2) High-risk patients need broader empirical therapy. (3) PCT (0.5 ng/mL) guides antibiotic decisions. (4) HAP requires more ICU resources. (5) Infection control and stewardship essential.

5. Conclusion

CAP and HAP are distinct clinical entities. CAP is caused by *S. pneumoniae* and *H. influenzae* with low resistance; HAP by MDR Gram-negatives (*A. baumannii*, *K. pneumoniae*, *P. aeruginosa*) and MRSA. HAP has double the 30-day mortality and prolonged hospitalization. Procalcitonin distinguishes bacterial CAP. Prior antibiotic exposure is the strongest HAP predictor. Separate clinical pathways for CAP and HAP are needed, with empirical antibiotics guided by local resistance data and individual risk factors. Antimicrobial stewardship and infection prevention are essential.

Compliance with ethical standards

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Disclosure of conflict of interest

The authors declare no conflict of interest.

Statement of ethical approval

Approved by Royal Medical Services IRB (No. 8_6/2026, 20 April 2026) and Educational & Technical Directorate (13 May 2026). Informed consent waived per retrospective anonymized design.

AI statement

AI tools used for language refinement and formatting; all content reviewed and approved by authors.

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