

Original Articles**Antimicrobial Management Following Positive Pneumococcal Urinary Antigen Testing in Hospitalized Community-Acquired Pneumonia: A Retrospective Cohort Study**

Maria Donga¹, Ana Gorgulho², Francisca Bartilotti², Dara Mbanze², Rita Menezes³, Cristóvão Figueiredo², Joana Fragoso², Sofia Nunes², Daniel Coutinho², Gabriela Abreu⁴, Tiago Teixeira² and Luís Malheiro^{1,2,5}.

¹ Department of Medicine, Faculty of Medicine, University of Porto, Alameda Professor Hernâni Monteiro, 4200-319 Porto, Portugal.

² Infectious Diseases Department, Unidade Local de Saúde de Gaia/Espinho, Rua Conceição Fernandes, s/n, 4434-502 Vila Nova de Gaia, Portugal.

³ Internal Medicine Department, Unidade Local de Saúde de Gaia/Espinho, Rua Conceição Fernandes, s/n, 4434-502 Vila Nova de Gaia, Portugal.

⁴ Department of Clinical Pathology, Unidade Local de Saúde de Gaia/Espinho, Rua Conceição Fernandes, s/n, 4434-502 Vila Nova de Gaia, Portugal.

⁵ Department of Medical Sciences, University of Aveiro, Campus Universitário de Santiago, 3810-193 Aveiro, Portugal.

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Abstract. Background: Pneumococcal urinary antigen testing (PUAT) rapidly supports pneumococcal etiology in community-acquired pneumonia (CAP), but how positive results are incorporated into antimicrobial management in routine care remains uncertain.

Objectives: To characterize antimicrobial stewardship actions implemented after a positive PUAT result became available in hospitalized adults with CAP, identify potential areas for improving guideline-concordant antimicrobial management, and compare clinical outcomes according to individual stewardship measures.

Methods: We conducted a retrospective observational cohort study at a Portuguese tertiary hospital including adults hospitalized with CAP and positive PUAT between January 2020 and December 2024. The primary endpoint was antimicrobial spectrum narrowing. Post-PUAT atypical coverage optimization and intravenous-to-oral switch were analyzed separately.

Results: Among 332 episodes, antimicrobial spectrum narrowing occurred in 30 (9.0%), after a median of 3 days (Q1–Q3, 2–4). De-escalation to ampicillin or amoxicillin occurred in 7 episodes (2.1%). Post-PUAT atypical coverage optimization occurred in 158 episodes (47.6%) and intravenous-to-oral switch in 71 (21.4%). Narrowing and intravenous-to-oral switch were associated with shorter length of stay in unadjusted analyses, although these associations were exploratory and potentially influenced by clinical stability.

Conclusions: In hospitalized PUAT-positive CAP, antimicrobial spectrum narrowing was uncommon, whereas atypical coverage optimization was the most frequent prescribing change. Structured post-result reassessment pathways may help improve guideline-concordant antimicrobial management.

Keywords: Community-acquired pneumonia; Pneumonia, pneumococcal; Antimicrobial stewardship; Anti-bacterial agents; *Streptococcus pneumoniae*.

Introduction. Community-acquired pneumonia (CAP) remains a leading cause of infectious morbidity and mortality worldwide, particularly among older adults and patients with substantial comorbidity.^{1,2} Chronic respiratory disease, chronic kidney disease (CKD), and immunosuppression are consistently associated with higher CAP incidence and more severe presentations.³ Because delayed effective therapy worsens outcomes, empirical antimicrobial therapy is usually initiated according to disease severity, risk factors, and local epidemiology.⁴

Streptococcus pneumoniae remains the most frequent bacterial cause of adult CAP and can progress to invasive disease.⁵ Conventional microbiological methods, including blood and sputum cultures, have limited sensitivity and are often affected by early antimicrobial exposure. The pneumococcal urinary antigen test (PUAT) provides rapid evidence supporting pneumococcal etiology and may remain positive after antimicrobial initiation.⁶ In principle, a positive PUAT should support antimicrobial stewardship by allowing pathogen-directed therapy once clinical stability has been achieved and alternative pathogens are not suspected, in line with international CAP guidance.⁷ Urinary-antigen-guided targeted regimens may achieve similar clinical outcomes to continued empiric therapy, while potentially reducing adverse events, *Clostridioides difficile* infection, and selection pressure for resistance.^{7,8}

However, the real-world stewardship value of PUAT is not automatic. Empirical CAP regimens already include pneumococcal coverage, clinicians may be reluctant to narrow treatment in severe or uncertain presentations, and urinary antigen results do not exclude viral or bacterial co-infection.⁸⁻¹⁰ Accordingly, PUAT results are more likely to influence prescribing when embedded in structured reassessment, including antimicrobial time-outs, predefined criteria for intravenous (IV)-to-oral switch, and explicit de-escalation triggers.^{7,11-16}

This study aimed to characterize antimicrobial stewardship actions implemented after a positive PUAT result became available in hospitalized adults with CAP, identify potential areas for improving guideline-concordant antimicrobial management, and explore clinical and therapeutic factors associated with the implementation or non-implementation of these measures. Antimicrobial spectrum narrowing was the

primary stewardship endpoint. Secondary objectives were to characterize post-result optimization of atypical coverage and, as a secondary descriptive stewardship measure, IV-to-oral switch, and to compare clinical outcomes according to these individual stewardship actions.

Material and Methods

Study design and setting. We conducted a retrospective observational cohort study at a tertiary care hospital in Northern Portugal. Adult patients hospitalized with CAP and a positive PUAT were included between January 1, 2020 and December 31, 2024. The study was approved by the institutional Ethics Committee on September 30, 2025, under approval number I27737-202506. Because of the retrospective design, informed consent was waived.

Case identification and eligibility. Potentially eligible cases were identified electronically through the institutional epidemiological surveillance/data extraction platform and the laboratory database. We screened all PUAT-positive results obtained in the emergency department or during the first 48 hours of hospital admission.

Inclusion criteria were age ≥ 18 years, hospitalization with a clinical diagnosis of CAP supported by chest imaging, and positive PUAT during the qualifying time window. We excluded duplicate records, repeated eligible episodes in the same individual, admissions in which the final adjudicated reason for hospitalization was not CAP, and patients who did not initiate antibacterial treatment because a palliative approach was instituted at admission or death occurred before antimicrobial therapy.

Data collection and definitions. Data were extracted from electronic health records and microbiology/laboratory databases. Variables included age, sex, comorbidities, respiratory viral polymerase chain reaction results for influenza, SARS-CoV-2 and other viruses, culture-based microbiology, markers of disease severity, antimicrobial treatment, stewardship actions, and clinical outcomes. Comorbidities included chronic obstructive pulmonary disease (COPD), other chronic pulmonary disease, type 2 diabetes mellitus (T2DM), congestive heart failure (CHF), CKD, active

malignancy, immunosuppression, and HIV infection. Disease severity variables included CURB-65 score, serum creatinine, C-reactive protein, procalcitonin, need and duration of supplemental oxygen, sepsis category as documented in the clinical record, and intensive care unit (ICU) admission. Antimicrobial variables included empiric core antimicrobial (beta-lactam or fluoroquinolone), macrolide use, total antimicrobial duration, IV therapy duration, and timing of antimicrobial changes. Empirical core antimicrobial categories were mutually exclusive, while azithromycin use was recorded separately and could occur in combination with the core regimen. The study period overlapped with the COVID-19 pandemic (2020 to 2022) and the post-pandemic period (2023 to 2024). Because respiratory viral circulation and pneumococcal epidemiology may have differed across calendar periods, pandemic timing was considered during interpretation. Data on pneumococcal vaccination status and adjudicated causes of death were not systematically available in the extracted dataset and were therefore not analyzed as formal study variables.

Antimicrobial management context. During the study period, empirical therapy for hospitalized CAP was selected by the treating team according to local practice, disease severity, comorbidities, and suspected coinfection. According to the local CAP protocol, empirical treatment generally consisted of a beta-lactam, most commonly ceftriaxone or amoxicillin/clavulanate, combined with azithromycin. Levofloxacin was used in patients with beta-lactam allergy. PUAT results were electronically available to the treating team. Routine antimicrobial stewardship or Infectious Diseases review following a positive PUAT was not performed, and no mandatory PUAT-specific de-escalation protocol was in place; antimicrobial spectrum narrowing and IV-to-oral switch were therefore performed according to clinical reassessment, microbiological results, and physician judgment.

Outcomes. The primary stewardship endpoint was antimicrobial spectrum narrowing occurring after availability of the positive PUAT result. Antimicrobial spectrum narrowing was defined as a reduction in antibacterial breadth while maintaining appropriate pneumococcal-directed therapy. This included discontinuation of empirical anti-MRSA or anti-pseudomonal coverage, replacement of broad-spectrum agents such as piperacillin/tazobactam, carbapenems, or fluoroquinolones with ceftriaxone or amoxicillin/clavulanate, and further narrowing from ceftriaxone or amoxicillin/clavulanate to cefuroxime, amoxicillin, or ampicillin when clinically appropriate.

Macrolide discontinuation alone was not counted as antimicrobial spectrum narrowing and was analyzed

separately as atypical coverage optimization. Atypical coverage optimization was evaluated only after the positive PUAT result had become available. It comprised two distinct situations: non-initiation of atypical coverage when the PUAT result was already known at the time of treatment selection, and early discontinuation of empirically initiated azithromycin after PUAT result availability, within the first 72 hours of treatment.

IV-to-oral switch was retained as a secondary descriptive stewardship measure and was not considered a PUAT-specific intervention, because it is primarily determined by clinical stability, gastrointestinal tolerance, and discharge planning rather than microbiological etiology alone. Other secondary outcomes included length of hospital stay (LOS), duration of IV therapy, 30-day all-cause mortality, 30-day readmission, and in-hospital *Clostridioides difficile* infection.

Statistical analysis. Continuous variables were summarized as medians and interquartile ranges (Q1-Q3), and categorical variables as counts and percentages. Patients who underwent antimicrobial spectrum narrowing were compared with those who did not. Secondary descriptive comparisons were also performed for post-PUAT atypical coverage optimization and for IV-to-oral switch. The two components of atypical coverage optimization, post-result non-initiation and early discontinuation of empirically initiated azithromycin, were described separately. Between-group comparisons were performed using the Mann-Whitney U test for continuous variables and the chi-square test or Fisher exact test for categorical variables, as appropriate. All between-group comparisons were exploratory. Given the number of unadjusted comparisons, p-values were interpreted descriptively and no individual univariate association was considered confirmatory. Because of the low number of antimicrobial spectrum narrowing events, no multivariable model was fitted for the primary endpoint. Associations involving narrowing were therefore interpreted as exploratory. Statistical significance was set at a two-sided $p < 0.05$. Analyses were conducted using IBM SPSS Statistics (IBM Corp., Armonk, NY, USA). Complete-case analysis was used for each comparison.

Results.

Study population. During the study period, 6,823 PUATs were performed and 477 (6.9%) yielded positive results. Twelve results were excluded as duplicates. After chart review, 465 episodes were screened; 130 were excluded because patients were discharged for outpatient management, and 3 were excluded because of palliative approach or death before antimicrobial initiation. The final analytic cohort comprised 332 hospitalized CAP episodes with positive PUAT.

Table 1. Baseline characteristics in hospitalized patients with community-acquired pneumonia and a positive pneumococcal urinary antigen test.

	Total (n=332)
Age in years, median (Q1-Q3)	75 (62.0-85.0)
Sex – Male, n (%)	185 (55.7)
Diagnosis during pandemic years, n (%)	180 (54.2)
Comorbidities	
COPD, n (%)	62 (18.7)
Other chronic pulmonary disease, n (%)	71 (21.4)
T2DM, n (%)	97 (29.2)
CHF, n (%)	84 (25.3)
CKD, n (%)	31 (9.3)
Cancer, n (%)	57 (17.2)
Immunosuppression, n (%)	46 (13.9)
HIV, n (%)	9 (2.7)
Microbiology	
Any coinfection, n (%)	93 (28.0)
Influenza virus, n (%)	20 (6.0)
SARS-CoV-2, n (%)	43 (13.0)
Other viruses, n (%)	7 (2.1)
Bacterial coinfection, n (%)	21 (6.3)
PC for <i>S. pneumoniae</i> , n (%)	42 (12.7)
PBC for <i>S. pneumoniae</i> , n (%)	37 (11.1)
Disease Severity	
CURB-65, median (Q1-Q3)	2 (1-3)
Serum Creatinine, mg/dL, median (Q1-Q3)	1.1 (0.8-1.6)
C-reactive protein, mg/dL, median (Q1-Q3)	16.5 (5.8-30.7)
Procalcitonin, µg/L, median (Q1-Q3)	2.6 (0.15-15.7)
Supplemental oxygen, n (%)	287 (86.4)
Duration of supplemental oxygen among patients requiring oxygen, days, median (Q1-Q3)	6 (3-10)
No sepsis, n (%)	227 (68.4)
Sepsis, n (%)	63 (19.0)
Septic shock, n (%)	42 (12.7)
ICU admission, n (%)	86 (25.9)
Empirical Treatment	
Amoxicillin/Clavulanate, n (%)	135 (40.7)
Ceftriaxone, n (%)	156 (47.0)
Other beta-lactams, n (%)	25 (7.5)
Fluoroquinolone, n (%)	16 (4.8)
Azithromycin, n (%)	200 (60.2)
Stewardship outcomes	
Antimicrobial spectrum narrowing, n (%)	30 (9.0)
Time to narrowing, days, median (Q1-Q3)	3 (2-4)

IV-to-oral switch, n (%)	71 (21.4)
IV duration in days, median (Q1-Q3)	6 (4-8)
Post-PUAT atypical coverage optimization, n (%)	158 (47.6)
Non-initiation of atypical coverage after PUAT result availability, n (%)	73 (22.0)
Early discontinuation of atypical coverage among those started, n/N (%)	85/200 (42.5)
Total ATB duration in days, median (Q1-Q3)	8 (7-11)
General Outcomes	
30-day mortality, n (%)	62 (18.7)
30-day readmission among patients discharged alive, n/N (%)	31/270 (11.5)
<i>C. difficile</i> infection, n (%)	2 (0.6)
LOS, median (Q1-Q3)	8 (5-14)

Percentages refer to the total number of cases unless otherwise stated. Legend: ATB – antimicrobial; CAP – community-acquired pneumonia; CHF – congestive heart failure; CKD – chronic kidney disease; COPD – chronic obstructive pulmonary disease; CRP – C-reactive protein; CURB-65 – Confusion, Urea, Respiratory rate, Blood pressure, age ≥ 65 score; HIV – human immunodeficiency virus; ICU – intensive care unit; IV – intravenous; O2 – oxygen; LOS – Length of hospital stay in days; PBC – positive blood culture; PC – positive culture; SARS-CoV-2 – severe acute respiratory syndrome coronavirus 2; T2DM – type 2 diabetes mellitus.

Table 1 describes the baseline characteristics, empirical treatment, stewardship actions, and clinical outcomes of the included patients. Patients were elderly, with a median age of 75 years (Q1-Q3, 62.0-85.0), and 185 (55.7%) were male. The most frequent comorbidities were T2DM (29.2%), CHF (25.3%) and COPD (18.7%). Other chronic pulmonary conditions were present in 71 patients and included asthma (n=19), bronchiectasis (n=12), emphysema and/or chronic bronchitis (n=8), pulmonary malignancy or pulmonary metastases (n=8), obstructive sleep apnea (n=6), fibrotic/interstitial lung disease (n=6), post-tuberculosis pulmonary sequelae (n=5), pneumoconiosis or other occupational lung disease (n=4), pulmonary nodules or micronodules (n=4), organizing pneumonia (n=3), pulmonary vascular disease (n=2), and other less frequent respiratory disorders. Some patients had more than one pulmonary condition. A documented viral or bacterial co-infection was present in 93 episodes (28.0%). Culture-confirmed *S. pneumoniae* was documented in 42 episodes (12.7%), including 37 positive blood cultures (11.1%). All *S. pneumoniae* isolates with available susceptibility testing were susceptible to penicillin (42/42, 100%). Disease severity was substantial: median CURB-65 was 2 (Q1-Q3, 1-3), 287 patients (86.4%) required supplemental oxygen, 63 (19.0%) had sepsis, 42 (12.7%) had septic shock, and 86 (25.9%) required ICU admission.

Empirical treatment most commonly included ceftriaxone (156/332, 47.0%) or amoxicillin/clavulanate (135/332, 40.7%). Other empirical beta-lactams were used in 25 episodes (7.5%), and empirical

Table 2. Empirical-to-final regimens among episodes classified as antimicrobial spectrum narrowing.

Empirical regimen	Final/narrowed regimen	n
Ceftriaxone	Amoxicillin/clavulanate	13
Ceftriaxone	Amoxicillin	4
Ceftriaxone	Cefuroxime	2
Piperacillin/tazobactam	Ceftriaxone	5
Piperacillin/tazobactam	Amoxicillin/clavulanate	1
Amoxicillin/clavulanate	Amoxicillin or ampicillin	3
Levofloxacin	Amoxicillin/clavulanate	2
Total		30

fluoroquinolones in 16 (4.8%). Azithromycin was used empirically in 200 episodes (60.2%). The median total antimicrobial duration was 8 days (Q1-Q3, 7-11). Median length of hospital stay was 8 days (Q1-Q3, 5-14). Thirty-day mortality occurred in 62 episodes (18.7%), 30-day readmission in 31 (11.5%), and in-hospital *Clostridioides difficile* infection in 2 (0.6%). The median time from PUAT result availability to antimicrobial spectrum narrowing was 3 days (Q1-Q3, 2-4).

Antimicrobial spectrum narrowing. Antimicrobial spectrum narrowing, the primary stewardship endpoint, occurred in 30 episodes (9.0%). Empirical-to-final regimen changes among episodes classified as antimicrobial spectrum narrowing represented varying degrees of spectrum reduction and were analyzed as a single pragmatic binary endpoint, without weighting according to the magnitude of de-escalation. The exact empirical-to-final regimen transitions are presented in **Table 2**. The median time to narrowing was 3 days (Q1-Q3, 2-4). De-escalation specifically to ampicillin or amoxicillin occurred in 7 episodes (2.1% of the total cohort and 23.3% of narrowing episodes).

Exploratory unadjusted comparisons between patients who did and did not undergo spectrum narrowing are presented in **Table 3**. No consistent differences in demographic characteristics or markers of disease severity were observed. Differences in selected comorbidities and empirical antimicrobial regimens were also observed; however, given the low number of narrowing events and the multiplicity of unadjusted comparisons, these findings were not interpreted as independent determinants of narrowing.

Other antimicrobial optimization actions. Post-PUAT atypical coverage optimization occurred in 158 episodes (47.6%). This included non-initiation of atypical coverage after the PUAT result became available in 73 patients (22.0%) and early discontinuation of empirically initiated azithromycin in 85 patients. Among the 200 patients who initially received azithromycin, 85 (42.5%) discontinued it early after PUAT result availability.

Compared with patients who did not undergo post-PUAT atypical coverage optimization, those who underwent optimization were older, had more documented co-infection, including SARS-CoV-2, and had lower severity markers, with less sepsis, septic shock, and ICU admission (**Table 3**).

IV-to-oral switch occurred in 71 episodes (21.4%) (**Table 1** and **Table 3**). Median IV therapy duration was 6 days (Q1-Q3, 4-8). Patients switched to oral therapy had a lower severity profile, including lower CURB-65 score, less frequent oxygen requirement, shorter oxygen duration, less sepsis and septic shock, and lower ICU admission. IV-to-oral switch was associated with shorter IV therapy duration (4 vs 8 days; $p<0.001$), shorter LOS (5 vs 10 days; $p<0.001$), and lower unadjusted 30-day mortality (1.4% vs 23.4%; $p<0.01$). No significant differences were observed in 30-day readmission or in-hospital *Clostridioides difficile* infection.

Discussion. In this retrospective cohort of hospitalized adults with CAP and a positive PUAT, post-PUAT antimicrobial spectrum narrowing was uncommon. De-escalation to ampicillin or amoxicillin, the narrowest pneumococcal-directed options evaluated, occurred even more rarely. These findings suggest that, although a positive PUAT provides microbiological information that may support pathogen-directed therapy in selected patients, antimicrobial management often remained broader than the narrowest pneumococcal-directed options evaluated.

Despite rapid PUAT result availability, with a median time from specimen collection to result availability of only 30 minutes, the median time to antimicrobial spectrum narrowing was 3 days.

This marked temporal gap suggests that rapid microbiological information alone may be insufficient to prompt early antimicrobial reassessment in the absence of a structured stewardship pathway, consistent with previous observational studies showing that, although PUAT is often proposed as a rapid diagnostic tool to support antimicrobial stewardship in CAP, a positive result does not reliably translate into targeted therapy in routine care.^{7,8,14-17}

Differences in spectrum narrowing were more closely related to the empirical antimicrobial regimen than to baseline disease severity. Narrowing was uncommon after initial amoxicillin/clavulanate but more frequent after broader-spectrum therapy, where the potential for de-escalation was greater. In these patients, narrowing to a pneumococcal-directed beta-lactam, including amoxicillin when appropriate, may be feasible.¹⁸ The 100% penicillin susceptibility observed among tested pneumococcal isolates supports the feasibility of narrower pneumococcal-directed therapy when clinically appropriate; however, as these isolates represented only 12.7% of the cohort, the finding should

Table 3. Patient characteristics stratified by individual stewardship actions, among PUAT-positive hospitalized CAP episodes.

	Narrowing (n=30)	No narrowing (n=302)	p-value	IV-to-oral (n=71)	No IV-to-oral (n=261)	p-value	Atypical coverage optimization, n (%) (n=158)	No atypical coverage optimization, n (%) (n=174)	p-value
Age in years, median (Q1-Q3)	69 (56-88)	75 (63-84)	0.932	74 (59-85)	75 (63-85)	0.849	77 (65-87)	72 (59-82)	0.023
Sex – Male, n (%)	16 (53.3)	169 (56.0)	0.782	38 (53.5)	147 (56.3)	0.674	87 (55.1)	98 (55.3)	0.818
Diagnosis during pandemic, n (%)	16 (53.3)	164 (54.3)	0.919	40 (56.3)	140 (53.6)	0.788	88 (55.7)	92 (52.9)	0.606
Comorbidities									
COPD, n (%)	6 (20.0)	56 (18.5)	0.808	12 (16.9)	50 (19.2)	0.665	28 (17.7)	34 (19.5)	0.671
Other chronic pulmonary disease, n (%)	8 (26.7)	63 (20.9)	0.485	12 (16.9)	59 (22.6)	0.229	35 (22.2)	36 (20.7)	0.746
T2DM, n (%)	13 (43.3)	84 (27.8)	0.092	18 (25.4)	79 (30.3)	0.419	52 (32.9)	45 (25.9)	0.158
CHF, n (%)	4 (13.3)	80 (26.5)	0.128	21 (29.6)	63 (24.1)	0.350	43 (27.2)	41 (23.6)	0.445
CKD, n (%)	7 (23.3)	24 (7.9)	0.014	3 (4.2)	28 (10.7)	0.095	18 (11.4)	13 (7.5)	0.220
Cancer, n (%)	9 (30.0)	48 (15.9)	0.072	16 (22.5)	41 (15.7)	0.176	30 (19.0)	27 (15.5)	0.402
Immunosuppression, n (%)	4 (13.3)	42 (13.9)	1.000	10 (14.1)	36 (13.8)	1.000	18 (11.4)	28 (16.1)	0.216
HIV, n (%)	1 (3.3)	8 (2.7)	0.579	1 (1.4)	8 (3.1)	0.690	2 (1.3)	7 (4.0)	0.120
Microbiology									
Any coinfection, n (%)	9 (30.0)	84 (27.8)	0.832	14 (19.7)	79 (30.3)	0.079	55 (34.8)	38 (21.8)	0.009
Influenza virus, n (%)	1 (3.3)	19 (6.3)	1.000	2 (2.8)	18 (6.9)	0.267	9 (5.7)	11 (6.3)	0.811
SARS-CoV-2, n (%)	4 (13.3)	39 (12.9)	1.000	5 (7.0)	38 (14.6)	0.094	27 (17.1)	16 (9.2)	0.032
Other viruses, n (%)	0	7 (2.3)	1.000	3 (4.2)	4 (1.5)	0.171	4 (2.5)	3 (1.7)	0.609
Bacterial coinfection, n (%)	1 (3.3)	20 (6.6)	0.707	4 (5.6)	17 (6.5)	0.787	14 (8.9)	7 (4.0)	0.071
PC for <i>S. pneumoniae</i> , n (%)	7 (23.3)	35 (11.6)	0.081	7 (9.8)	35 (13.4)	0.397	18 (11.4)	24 (13.8)	0.511
PBC for <i>S. pneumoniae</i> , n (%)	6 (20.0)	31 (10.4)	0.118	6 (8.6)	31 (12.1)	0.414	16 (10.3)	21 (12.3)	0.564
Disease Severity									
CURB-65, median (Q1-Q3)	2 (1-3)	2 (1-3)	0.299	2 (1-2)	3 (2-3)	<0.001	2 (1-3)	2 (1-3)	0.943
Supplemental oxygen, n (%)	26 (86.7)	261 (86.4)	1.000	56 (78.9)	231 (88.5)	0.036	131 (82.9)	156 (89.7)	0.073
Duration of O2 among patients requiring oxygen, days, median (Q1-Q3)	5 (4-10)	6 (3-10)	0.911	4 (2-6)	7 (4-11)	<0.001	5 (3-9)	7 (4-10)	0.046
No sepsis, n (%)	20 (66.7)	207 (68.5)	0.830	62 (87.3)	165 (63.2)	0.001	124 (78.5)	103 (59.2)	<0.001
Sepsis, n (%)	3 (10.0)	60 (19.9)	0.301	5 (7.0)	58 (22.2)	0.003	21 (13.3)	42 (24.1)	0.003
Septic Shock, n (%)	7 (23.3)	35 (11.6)	0.126	4 (5.6)	38 (14.6)	0.020	13 (8.2)	29 (16.7)	0.006
ICU admission, n (%)	8 (26.7)	78 (25.8)	1.000	10 (14.1)	76 (29.1)	0.010	27 (17.1)	59 (33.9)	<0.001
Empirical Treatment									
Amoxicillin/Clavulanate, n (%)	3 (10.0)	132 (43.7)	<0.001	32 (45.1)	103 (39.5)	0.394	65 (41.1)	70 (40.2)	0.866
Ceftriaxone, n (%)	19 (63.3)	137 (45.4)	0.083	27 (38.0)	129 (49.4)	0.088	71 (44.9)	85 (48.9)	0.475
Other beta-lactams, n (%)	6 (20.0)	19 (6.3)	0.007	4 (5.6)	21 (8.1)	0.500	20 (12.7)	5 (2.9)	0.002
Fluoroquinolone, n (%)	2 (6.7)	14 (4.6)	0.646	8 (11.3)	8 (3.1)	0.009	2 (1.3)	14 (8.0)	0.004
Stewardship outcomes									
Total ATB duration in days, median (Q1-Q3)	8 (7-10)	8 (7-10)	0.923	8 (7-10)	8 (7-10)	0.837	8 (7-10)	8 (7-10)	0.625
IV duration in days, median (Q1-Q3)	6 (4-10)	7 (6-10)	0.649	4 (3-6)	8 (7-10)	<0.001	7 (5-10)	7 (6-10)	0.170
General Outcomes									
30-day mortality, n (%)	4 (13.3)	58 (19.2)	0.623	1 (1.4)	61 (23.4)	< 0.01	33 (20.9)	29 (16.7)	0.325

30-day readmission, n/N (%) [*]	5/26 (19.2)	26/244 (10.7)	0.199	11/70 (15.7)	20/200 (10.0)	0.207	19/125 (15.2)	12/145 (8.3)	0.074
<i>C. difficile</i> infection, n (%)	0	2 (0.7)	1.000	0	2 (0.8)	1.000	1 (0.6)	1 (0.6)	0.954
Length of hospital stay in days, median (Q1-Q3)	6.5 (4-12)	9 (6-14)	0.034	5 (4-7)	10 (7-15)	<0.001	8 (6-14)	9 (6-14)	0.253

Legend: ^{*} Thirty-day readmission percentages were calculated among patients alive at discharge; ATB – antimicrobial; CAP – community-acquired pneumonia; *C. difficile* – *Clostridioides difficile*; CHF – congestive heart failure; CKD – chronic kidney disease; COPD – chronic obstructive pulmonary disease; CRP – C-reactive protein; CURB-65 – Confusion, Urea, Respiratory rate, Blood pressure, age ≥ 65 score; HIV – human immunodeficiency virus; ICU – intensive care unit; IV – intravenous; O₂ – oxygen; PBC – positive blood culture; PC – positive culture; PUAT – pneumococcal urinary antigen test; SARS-CoV-2 – severe acute respiratory syndrome coronavirus 2; T2DM – type 2 diabetes mellitus.

not be generalized to all PUAT-positive cases. However, several factors may limit narrowing after a positive PUAT, including the fact that empirical CAP regimens already provide pneumococcal coverage, the possibility of bacterial or viral co-infection, and clinician reluctance to de-escalate in patients with greater clinical complexity.^{7-10,12,19} In addition, without a structured PUAT-specific reassessment pathway, a positive result may not consistently trigger antimicrobial modification. Baseline severity was broadly similar between patients with and without narrowing, suggesting that de-escalation was not limited to those with milder disease.

Post-PUAT atypical coverage optimization was the most frequent stewardship action in our cohort. Both non-initiation and early discontinuation of atypical coverage were only considered when they occurred after the positive PUAT result became available. This suggests that atypical coverage may be more readily modified than the core antibacterial regimen during post-result reassessment. However, these decisions cannot be attributed solely to PUAT, as other microbiological and clinical factors may also have contributed. Structured guidance integrating PUAT results, viral testing, and clinical evolution may help standardize these decisions, consistent with previous evidence suggesting that PUAT may influence atypical coverage more readily than other forms of de-escalation.²⁰

IV-to-oral switch was more frequent than spectrum narrowing but occurred in only about one fifth of hospitalized PUAT-positive CAP cases. Patients undergoing IV-to-oral switch had lower disease severity, shorter IV therapy duration, and shorter hospital stay, suggesting that switching was primarily associated with clinical improvement rather than PUAT positivity alone. Both spectrum narrowing and IV-to-oral switch were associated with shorter length of stay, although these unadjusted associations should be interpreted cautiously because stewardship actions may have been preferentially undertaken in patients with a more favorable clinical course.

Previous studies suggest that antimicrobial de-escalation and early IV-to-oral switch can be safely implemented in selected patients with CAP without worsening mortality, readmission, or other adverse outcomes.^{17,21-23} Structured reassessment pathways may

help identify appropriate candidates by integrating microbiological results, co-infection assessment, oxygen requirements, oral tolerance, and clinical stability. Such pathways should distinguish two separate decisions: whether microbiological findings support antimicrobial spectrum narrowing and whether clinical evolution supports transition from intravenous to oral therapy.

This study has several limitations. Its retrospective, single-center design limits causal inference and generalizability, and prescribing decisions may have been influenced by unmeasured clinical factors. Stewardship actions were inferred from clinical documentation and prescribing patterns and may therefore be subject to misclassification. Because the cohort included only hospitalized patients with CAP and a positive PUAT, the study cannot assess the incremental effect of PUAT compared with PUAT-negative or untested patients. Although only antimicrobial changes occurring after PUAT result availability were classified as stewardship actions, PUAT could not be established as the sole driver of each decision, as other microbiological and clinical information may also have contributed. Pneumococcal vaccination status and causes of death were not systematically available. Clinical eligibility for spectrum narrowing could not be reliably adjudicated retrospectively in all patients, and the 9.0% narrowing rate should therefore not be interpreted as indicating that all remaining patients represented missed stewardship opportunities. In addition, the spectrum-narrowing endpoint included interventions of varying magnitude and should be interpreted as a pragmatic measure of de-escalation. Finally, associations between stewardship actions and clinical outcomes were exploratory and unadjusted and are subject to confounding by indication and clinical stability.

In hospitalized adults with CAP and a positive PUAT, antimicrobial spectrum narrowing after result availability was uncommon, and de-escalation to the narrowest pneumococcal-directed options was rare. Post-PUAT optimization of atypical coverage was more frequent, while IV-to-oral switch, evaluated as a secondary descriptive stewardship measure, primarily reflected clinical stability. These findings identify areas where structured post-result antimicrobial reassessment may improve guideline-concordant management, while

recognizing that the clinical suitability for de-escalation could not be retrospectively adjudicated in every patient.

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References:

1. File TM Jr, Ramirez JA. Community-acquired pneumonia. *N Engl J Med*. 2023;389:632-641. <https://doi.org/10.1056/NEJMcp2303286> PMID:37585629
2. Anderson R, Feldman C. The global burden of community-acquired pneumonia in adults, encompassing invasive pneumococcal disease and the prevalence of its associated cardiovascular events, with a focus on pneumolysin and macrolide antibiotics in pathogenesis and therapy. *Int J Mol Sci*. 2023;24:11038. <https://doi.org/10.3390/ijms241311038> PMID:37446214
3. Torres A, Peetermans WE, Viegi G, Blasi F. Risk factors for community-acquired pneumonia in adults in Europe: a literature review. *Thorax*. 2013;68:1057-1065. <https://doi.org/10.1136/thoraxjnl-2013-204282> PMID:24130229
4. Rider AC, Frazee BW. Community-acquired pneumonia. *Emerg Med Clin North Am*. 2018;36:665-683. <https://doi.org/10.1016/j.emc.2018.07.001>
5. Weiser JN, Ferreira DM, Paton JC. Streptococcus pneumoniae: transmission, colonization and invasion. *Nat Rev Microbiol*. 2018;16:355-367. <https://doi.org/10.1038/s41579-018-0001-8> PMID:29599457
6. Kaysin A, Viera AJ. Community-acquired pneumonia in adults: diagnosis and management. *Am Fam Physician*. 2016;94:698-706. <https://doi.org/10.1002/9780470727275.ch4> PMID:42587375
7. Metlay JP, Waterer GW, Long AC, Anzueto A, Brozek J, Crothers K, Cooley LA, Dean NC, Fine MJ, Flanders SA, Griffin MR, Metersky ML, Musher DM, Restrepo MI, Whitney CG. Diagnosis and treatment of adults with community-acquired pneumonia. An official clinical practice guideline of the American Thoracic Society and Infectious Diseases Society of America. *Am J Respir Crit Care Med*. 2019;200:e45-e67. <https://doi.org/10.1164/rccm.201908-1581ST> PMID:31573350
8. Schimmel JJ, Haessler S, Imrey P, Lindenauer PK, Richter SS, Yu PC, Rothberg MB. Pneumococcal urinary antigen testing in United States hospitals: a missed opportunity for antimicrobial stewardship. *Clin Infect Dis*. 2020;71:1427-1434. <https://doi.org/10.1093/cid/ciz983> PMID:31587039
9. Hyams C, Williams OM, Williams P. Urinary antigen testing for pneumococcal pneumonia: is there evidence to make its use uncommon in clinical practice? *ERJ Open Res*. 2020;6:00223-2019. <https://doi.org/10.1183/23120541.00223-2019>
10. Reyes LF, Conway Morris A, Serrano-Mayorga C, Derde LPG, Dickson RP, Martin-Loeches I. Community-acquired pneumonia. *Lancet*. 2025;406:2371-2388. [https://doi.org/10.1016/S0140-6736\(25\)01493-X](https://doi.org/10.1016/S0140-6736(25)01493-X) PMID:41110447
11. Viasus D, Vecino-Moreno M, De La Hoz JM, Carratala J. Antibiotic stewardship in community-acquired pneumonia. *Expert Rev Anti Infect Ther*. 2017;15:351-359. <https://doi.org/10.1080/14787210.2017.1274232> PMID:28002979
12. Vaughn VM, Dickson RP, Horowitz JK, Flanders SA. Community-acquired pneumonia: a review. *JAMA*. 2024;332:1282-1295. <https://doi.org/10.1001/jama.2024.14796> PMID:39283629
13. Lee RA, Centor RM, Humphrey LL, Jokela JA, Andrews R, Qaseem A. Appropriate use of short-course antibiotics in common infections: best practice advice from the American College of Physicians. *Ann Intern Med*. 2021;174:822-827. <https://doi.org/10.7326/M20-7355>
14. Kern M, Fally M, Kolte L, Ravn P, Benfield T, Israelsen SB. Impact of pneumococcal urinary antigen testing on clinical outcomes in patients hospitalized with community-acquired pneumonia. *Eur J Clin Microbiol Infect Dis*. 2025;44:2635-2644. <https://doi.org/10.1007/s10096-025-05226-1> PMID:40715669
15. Sorde R, Falco V, Lowak M, Domingo E, Ferrer A, Burgos J, Puig M, Cabral E, Len O, Pahissa A. Current and potential usefulness of pneumococcal urinary antigen detection in hospitalized patients with community-acquired pneumonia to guide antimicrobial therapy. *Arch Intern Med*. 2011;171:166-172. <https://doi.org/10.1001/archinternmed.2010.347> PMID:20876397
16. Bhatia M, Kim P, Inampudi J, Shapoo M, Scipione MR, Dhar S, Monday L. P-1599. Evaluating the impact of pneumococcal urinary antigen on antibiotic prescribing in an urban safety net health system. *Open Forum Infect Dis*. 2025;12(Suppl 1):ofae631.1766. <https://doi.org/10.1093/ofid/ofae631.1766>
17. Viasus D, Simonetti AF, Garcia-Vidal C, Niubo J, Dorca J, Carratala J. Impact of antibiotic de-escalation on clinical outcomes in community-acquired pneumococcal pneumonia. *J Antimicrob Chemother*. 2017;72:547-553. <https://doi.org/10.1093/jac/dkw441>
18. Falguera M, Ruiz-Gonzalez A, Schoenenberger JA, Touzon C, Gazquez I, Galindo C, Porcel JM. Prospective, randomised study to compare empirical treatment versus targeted treatment on the basis of the urine antigen results in hospitalised patients with community-acquired pneumonia. *Thorax*. 2010;65:101-106. <https://doi.org/10.1136/thx.2009.118588> PMID:19703825
19. Viasus D, Calatayud L, McBrowne MV, Ardanuy C, Carratala J. Urinary antigen testing in community-acquired pneumonia in adults: an update. *Expert Rev Anti Infect Ther*. 2019;17:107-115. <https://doi.org/10.1080/14787210.2019.1565994> PMID:30618315
20. Athlin S, Magnuson A, Spindler C, Hedlund J, Strålin K, Naclér P. Pneumococcal urinary antigen testing for antimicrobial guidance in community-acquired pneumonia: a register-based cohort study. *J Infect*. 2022;85:167-173. <https://doi.org/10.1016/j.jinf.2022.05.021>
21. van Heijl I, Schweitzer VA, Boel CHE, Oosterheert JJ, Huijts SM, Dorigo-Zetsma W, van der Linden PD, Bonten MJM, van Werkhoven CH. Confounding by indication of the safety of de-escalation in community-acquired pneumonia: a simulation study embedded in a prospective cohort. *PLoS One*. 2019;14:e0218062. <https://doi.org/10.1371/journal.pone.0218062> PMID:31560686
22. Abela-Alonso G, Rombauts A, Gudiol C, Garcia-Lerma E, Pallares N, Ardanuy C, Calatayud L, Niubo J, Tebe C, Carratala J. Effect of positive microbiological testing on antibiotic de-escalation and outcomes in community-acquired pneumonia: a propensity score analysis. *Clin Microbiol Infect*. 2022;28:1602-1608. <https://doi.org/10.1016/j.cmi.2022.06.021>
23. Deshpande A, Klompas M, Guo N, Imrey PB, Pallotta AM, Higgins T, Haessler S, Zilberberg MD, Lindenauer PK, Rothberg MB. Intravenous to oral antibiotic switch therapy among patients hospitalized with community-acquired pneumonia. *Clin Infect Dis*. 2023;77:174-185. <https://doi.org/10.1093/cid/ciad196> PMID:37011018

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