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Original Research Article

Bacterial etiology and multidrug resistance patterns in hospital-acquired pneumonia: predominance of Gram-negative pathogens

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ABSTRACT

Background: Hospital-acquired pneumonia (HAP) is a major nosocomial infection and is increasingly complicated by multidrug-resistant (MDR) pathogens. Center-specific data on bacterial etiology and susceptibility patterns are essential to optimize empiric therapy and antimicrobial stewardship. Objective was to determine bacterial etiology and MDR burden in HAP, and to describe organism-specific antibiotic susceptibility patterns, emphasizing Gram-negative predominance.
Methods: This cross-sectional study enrolled 100 adult inpatients with HAP (onset ≥48 hours after admission) in the department of respiratory medicine, Bangladesh Medical University, Dhaka, over 12 months, from August 2024 to August 2025. Sputum, induced sputum, or bronchoalveolar lavage specimens were cultured following quality screening, and antimicrobial susceptibility testing was performed by disc diffusion. Results were summarized using frequencies and percentages; MDR was defined as non-susceptibility to at least one agent in three or more antimicrobial classes.
Results: Bacterial growth was obtained in 78.0% of cases, with single-organism isolation in 68.0% and polymicrobial growth in 10.0%. A total of 89 isolates were recovered, predominantly Gram-negative (93.3%). Leading pathogens were *Klebsiella spp*. (33.7%), *Pseudomonas aeruginosa* (31.5%), and *Acinetobacter spp*. (24.7%). Overall MDR prevalence was 87.8%. Cephalosporin susceptibility was low among major Gram-negative organisms, while carbapenem susceptibility was moderate; colistin susceptibility varied across species.
Conclusions: HAP in this cohort was dominated by MDR Gram-negative pathogens with limited susceptibility to commonly used antibiotics, supporting the need for local antibiograms and stewardship-guided empiric therapy.

Keywords: Antibiotic susceptibility, Gram-negative bacilli, Hospital-acquired pneumonia, Multidrug resistance

INTRODUCTION

Hospital-acquired pneumonia (HAP), including the increasingly emphasized non-ventilator hospital-acquired pneumonia (NV-HAP) phenotype, remains a major source of avoidable inpatient harm because it occurs in medically complex patients, drives broad-spectrum antibiotic exposure, and is tightly linked to downstream outcomes such as prolonged hospitalization, ICU escalation, and death. Large electronic surveillance data from 284 US hospitals suggest that approximately 1 in 200 admissions meet NV-HAP criteria, with 22% in-hospital mortality, 8% discharge to hospice, and only 38% discharged directly home, and the authors estimated NV-HAP could be associated with up to 7.3% of hospital deaths, reinforcing that HAP prevention and early appropriate management are system-level priorities rather than niche ICU problems.[1] In parallel, contemporary ICU literature continues to describe healthcare-associated infections as an evolving epidemiologic landscape shaped by case-mix, device exposure, and antimicrobial pressure, where prevention and diagnostic strategies must be continuously

adapted to local ecology.[2] Evidence from low- and middle-income settings also highlights the dual challenge of substantial hospital-acquired infection burden alongside high antimicrobial utilization, a combination that predictably accelerates resistance selection and complicates empiric decision-making.[3] Against this background, the etiologic spectrum of HAP differs materially from community-acquired pneumonia, with repeated observations that hospital pneumonia is disproportionately driven by Gram-negative bacilli, while the contribution of Gram-positive organisms varies by institution and patient subgroups.[4] Recent center-level NV-HAP data from Bahrain illustrate this Gram-negative predominance clearly, with *Klebsiella pneumoniae* (38.59%) and *Pseudomonas aeruginosa* (26.09%) as leading pathogens among 184 culture-confirmed NV-HAP cases, accompanied by exceptionally high multidrug resistance, including 97.56% MDR in *K. pneumoniae*, underscoring how local hospital ecology can produce markedly constrained empiric options.[5] Comparable hospital and ICU cohorts outside the Gulf, for example from Paraguay, similarly frame HAP as a high-morbidity ICU problem with microbiologic profiles and outcomes that demand context-specific surveillance rather than reliance on imported antibiograms.[6] At the organism level, Pseudomonas remains a durable cause of nosocomial pneumonia with clinically meaningful resistance patterns over time, supporting the practice of presenting organism-specific susceptibility profiles rather than pooled antibiotic performance alone.[7] The clinical stakes of resistance are not abstract, in a tertiary-center VAP cohort, 25% of cases involved an MDR pathogen, and MDR isolation was associated with longer ICU stay (19 versus 16 days) and longer mechanical ventilation duration (18 versus 14 days), illustrating how resistance amplifies resource use and severity in hospital pneumonia syndromes.[8] More broadly, ICU microbiology is increasingly dominated by ESKAPE pathogens and carbapenemase-producing *Enterobacterales* in many regions, reinforcing why surveillance of Gram-negative predominance and MDR burden is central to HAP-focused reporting.[9] Therapeutically, MDR Gram-negative infections challenge standard empiric regimens by undermining the reliability of cephalosporins, fluoroquinolones, and even carbapenems in some settings, shifting practice toward last-line agents and combination strategies with toxicity, cost, and stewardship consequences, which makes local susceptibility data a prerequisite for rational empiric protocols.[10,11] In Bangladesh, the need for center-specific evidence is particularly acute because published ICU microbiology already signals heavy Gram-negative representation and high resistance, for example one tertiary-care ICU study reported frequent isolation of *Pseudomonas spp*. (35%), *E. coli* (28%), *Acinetobacter spp*. (24%), and *Klebsiella spp*. (18%), alongside high resistance to commonly used agents such as ceftriaxone and fluoroquinolones, underscoring the risk of mismatch between empiric choices and local susceptibility.[12] Prevention and stewardship are also linked to broader respiratory infection control capacity, including persistent gaps in influenza vaccination systems, with multicenter data showing low influenza vaccine uptake among Bangladeshi healthcare workers (13.8%, 283/2046), and national-level analyses emphasizing ongoing evidence and policy barriers to influenza prevention, both of which can indirectly sustain high antibiotic exposure and selective pressure in hospitals.[13,14] In this context, the present study aimed to determine the bacterial etiology of hospital-acquired pneumonia and to characterize multidrug resistance and antibiotic susceptibility patterns, focusing on the predominance of Gram-negative pathogens, to generate actionable, locally relevant evidence for empiric therapy, antimicrobial stewardship, and infection-prevention prioritization.

METHODS

This cross-sectional study was conducted in the department of respiratory medicine, Bangladesh Medical University, Dhaka, over 12 months, from August 2024 to August 2025, and enrolled 100 consecutively sampled adult inpatients (≥18 years) who met the working definition of hospital-acquired pneumonia, defined as a new pneumonia syndrome occurring ≥48 hours after hospital admission and not incubating at admission. Patients admitted with community-acquired pneumonia, those in whom an adequate lower respiratory tract sample could not be obtained, pregnant women, patients who were ventilated or had extubation within 48 hours prior to pneumonia onset, individuals with recent surgery within 1 month, and those aged <18 years were excluded. After written informed consent, sociodemographic variables (age, sex, occupation), smoking status, comorbidities, and prespecified HAP risk factors were recorded using a structured data collection sheet, including prior infection, prior antibiotic exposure within 90 days, immunosuppressive conditions, nutritional status (obesity, malnutrition), nebulization, nasogastric tube, depressed consciousness, neutropenia, prior hospital admission within 3 months, preventive vaccination history (influenza and pneumococcal), and duration of hospital stay. Respiratory specimens were collected as expectorated sputum, induced sputum, or bronchoalveolar lavage (BAL) fluid in patients undergoing fiber-optic bronchoscopy as part of clinical care; time from symptom onset to sample collection was documented. All samples underwent Gram staining, and specimens meeting acceptability criteria using Bartlett's composite Q score ≥1 were processed for culture; culture results were categorized as growth present, normal flora, or no growth, and growth characteristics were summarized as single-organism or polymicrobial isolation, with semi-quantitative colony assessment reported (scanty, moderate, profuse) in culture-positive cases. Bacterial culture was performed using routine media (including blood agar, chocolate agar, and MacConkey agar), and antimicrobial susceptibility testing was performed on Mueller-Hinton agar using standard disc diffusion methodology, with results interpreted as susceptible, intermediate, or resistant according to the laboratory

standard definitions used for testing; organism-specific susceptibility results were reported using isolate denominators that yielded a total of 89 isolates (reflecting polymicrobial infections among culture-positive patients). Multidrug resistance (MDR) was defined as non-susceptibility to at least one agent in three or more antimicrobial classes.[10] Overall MDR burden was summarized as a proportion of isolates. Data were checked, coded, and analyzed using SPSS (version 25), and results were presented primarily as frequencies and percentages, with continuous variables summarized using mean±SD and median (range), consistent with the descriptive tables and the isolate distribution figure used in the manuscript results.

RESULTS

The study enrolled 100 adult HAP patients with a mean age of 53.61±14.90 years; 64% were male, and the leading comorbidities were diabetes mellitus (55%), hypertension (36%), and heart failure (24%). The demographic characteristics of the cohort are summarized in Table 1. Microbiological specimens were predominantly expectorated sputum (82%), with induced sputum (10%) and bronchoalveolar lavage (8%) obtained where clinically indicated; mean time from symptom onset to sampling was 5.07±2.41 days. Bacterial growth was present in 78% of cases, with single-organism isolation in 68% and polymicrobial growth in 10%; 22% yielded no organism. A total of 89 isolates were recovered across culture-positive patients and form the basis of the etiology and susceptibility analyses below.

Table 1: Demographic characteristics of the study participants (n=100).

Variables	Category	N (%)
Age (years)	19-30	10 (10.0)
	31-40	11 (11.0)
	41-50	15 (15.0)
	51-60	31 (31.0)
	61-70	23 (23.0)
	>70	10 (10.0)
	Mean±SD	53.61±14.90
	Median (range)	55.0 (19.0-90.0)
Sex	Male	64 (64.0)
	Female	36 (36.0)
Occupation	Farming	7 (7.0)
	Business	23 (23.0)
	Service holder	26 (26.0)
	Housewife	25 (25.0)
	Other	19 (19.0)

The isolate distribution demonstrates a Gram-negative dominant etiology, led by *Klebsiella spp*. (n=30), *Pseudomonas aeruginosa* (n=28), and *Acinetobacter spp*. (n=22). Gram-positive pathogens were comparatively uncommon, with *Staphylococcus aureus* (n=6), while *Enterobacter spp*. (n=2) and *Escherichia coli* (n=1) were rarely isolated.

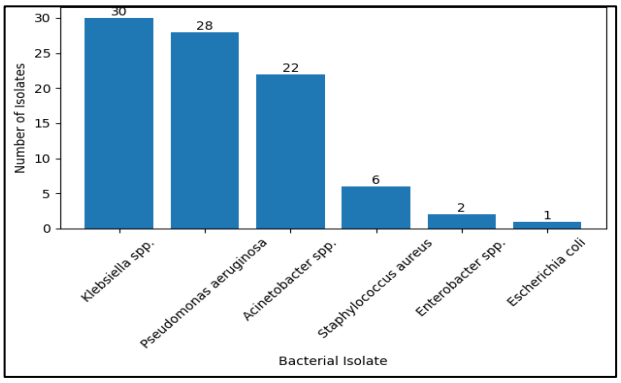

Figure 1: Distribution of bacterial isolates in HAP.

Table 2: Bacterial etiology in hospital-acquired pneumonia (total isolates n=89).

A total of 89 isolates were recovered, reflecting polymicrobial infections in a subset. Gram-negative organisms accounted for 83/89 (93.3%), indicating clear predominance, while Gram-positive organisms comprised 6/89 (6.7%). The leading pathogens were *Klebsiella spp*. 30 (33.7%), *Pseudomonas aeruginosa* 28 (31.5%), and *Acinetobacter spp*. 22 (24.7%), together contributing 80/89 (89.9%) of all isolates. Less frequent organisms included *Staphylococcus aureus* 6 (6.7%), *Enterobacter spp*. 2 (2.2%), and *Escherichia coli* 1 (1.1%).

Table 3: Multidrug resistance burden.

Variables	Category or statistic	Value
Multidrug resistance (MDR) prevalence	Overall	87.8%

Multidrug resistance was highly prevalent, with an overall MDR rate of 87.8% among isolates, indicating that the majority of bacterial pathogens recovered in this hospital-acquired pneumonia cohort met MDR criteria.

Antibiotic susceptibility varied by organism, with overall low susceptibility to third and fourth generation cephalosporins in key Gram-negative pathogens. For *Acinetobacter* (n=22), susceptibility was 18.2% to ceftriaxone and ceftazidime (each 4/22), and 4.5% to cefepime (1/22), while higher susceptibility was observed for amikacin 68.2% (15/22), meropenem 59.1% (13/22), and piperacillin-tazobactam 50.0% (11/22). For *Klebsiella* (n=30), susceptibility was 26.7% (8/30) to ceftriaxone, 6.7% (2/30) to ceftazidime, and 0.0% (0/30) to cefepime, whereas susceptibility was higher for meropenem 60.0% (18/30) and piperacillin-tazobactam 50.0% (15/30), with colistin 56.7% (17/30) and amikacin 46.7% (14/30). For *Pseudomonas* (n=28), susceptibility was 0.0% to ceftriaxone and cefepime (0/28 each) but higher to cefepime 64.3% (18/28) and piperacillin-tazobactam

67.9% (19/28), and meropenem 60.7% (17/28); susceptibility to amikacin and gentamicin was lower at 25.0% (7/28) and 14.3% (4/28), respectively. Fluoroquinolone susceptibility differed across organisms, with ciprofloxacin susceptibility 78.6% (22/28) in *Pseudomonas* compared with 16.7% (5/30) in *Klebsiella* and 36.4% (8/22) in *Acinetobacter*, while levofloxacin susceptibility was 0.0% in *Pseudomonas* and *Klebsiella*, and 4.5% (1/22) in *Acinetobacter*. Among *Staphylococcus aureus* (n=6), susceptibility was 100.0% (6/6) to vancomycin, 66.7% (4/6) to linezolid and oxacillin, and 33.3% (2/6) to clindamycin, with ceftriaxone susceptibility 16.7% (1/6). Minor isolates showed limited interpretability due to small denominators, with *Enterobacter* (n=2) showing 50.0% (1/2) susceptibility to several agents and *E. coli* (n=1) showing 100.0% (1/1) susceptibility across multiple tested antibiotics.

Table 4: Antibiotic susceptibility by pathogen, long format [susceptible N (%), total isolates n=89].

Variables (antibiotic)	Organisms	Susceptible, N (%)
Ceftriaxone	*Acinetobacter* (n=22)	4 (18.2)
	Klebsiella (n=30)	8 (26.7)
	E. coli (n=1)	1 (100.0)
	Pseudomonas (n=28)	0 (0.0)
	Staphylococcus aureus (n=6)	1 (16.7)
	Enterobacter (n=2)	1 (50.0)
Ceftazidime	*Acinetobacter* (n=22)	4 (18.2)
	Klebsiella (n=30)	2 (6.7)
	E. coli (n=1)	1 (100.0)
	Pseudomonas (n=28)	15 (53.6)
	Staphylococcus aureus (n=6)	0 (0.0)
	Enterobacter (n=2)	1 (50.0)
Cefepime	*Acinetobacter* (n=22)	1 (4.5)
	Klebsiella (n=30)	0 (0.0)
	E. coli (n=1)	1 (100.0)
	Pseudomonas (n=28)	18 (64.3)
	Staphylococcus aureus (n=6)	1 (16.7)
	Enterobacter (n=2)	1 (50.0)
Piperacillin-tazobactam	*Acinetobacter* (n=22)	11 (50.0)
	Klebsiella (n=30)	15 (50.0)
	E. coli (n=1)	1 (100.0)
	Pseudomonas (n=28)	19 (67.9)
	Staphylococcus aureus (n=6)	0 (0.0)
	Enterobacter (n=2)	0 (0.0)
Meropenem	*Acinetobacter* (n=22)	13 (59.1)
	Klebsiella (n=30)	18 (60.0)
	E. coli (n=1)	1 (100.0)
	Pseudomonas (n=28)	17 (60.7)
	Staphylococcus aureus (n=6)	0 (0.0)
	Enterobacter (n=2)	1 (50.0)
Amikacin	*Acinetobacter* (n=22)	15 (68.2)
	Klebsiella (n=30)	14 (46.7)
	E. coli (n=1)	1 (100.0)
	Pseudomonas (n=28)	7 (25.0)
	Staphylococcus aureus (n=6)	0 (0.0)
	Enterobacter (n=2)	1 (50.0)
Gentamicin	*Acinetobacter* (n=22)	9 (40.9)
	Klebsiella (n=30)	9 (30.0)
	E. coli (n=1)	1 (100.0)
	Pseudomonas (n=28)	4 (14.3)
	Staphylococcus aureus (n=6)	1 (16.7)
	Enterobacter (n=2)	1 (50.0)
Ciprofloxacin	*Acinetobacter* (n=22)	8 (36.4)

Continued.

Variables (antibiotic)	Organisms	Susceptible, N (%)
	Klebsiella (n=30)	5 (16.7)
	E. coli (n=1)	1 (100.0)
	Pseudomonas (n=28)	22 (78.6)
	Staphylococcus aureus (n=6)	0 (0.0)
	Enterobacter (n=2)	0 (0.0)
Levofloxacin	*Acinetobacter* (n=22)	1 (4.5)
	Klebsiella (n=30)	0 (0.0)
	E. coli (n=1)	0 (0.0)
	Pseudomonas (n=28)	0 (0.0)
	Staphylococcus aureus (n=6)	1 (16.7)
	Enterobacter (n=2)	0 (0.0)
Cotrimoxazole	*Acinetobacter* (n=22)	11 (50.0)
	Klebsiella (n=30)	10 (33.3)
	E. coli (n=1)	1 (100.0)
	Pseudomonas (n=28)	0 (0.0)
	Staphylococcus aureus (n=6)	2 (33.3)
	Enterobacter (n=2)	0 (0.0)
Tigecycline	*Acinetobacter* (n=22)	4 (18.2)
	Klebsiella (n=30)	0 (0.0)
	E. coli (n=1)	0 (0.0)
	Pseudomonas (n=28)	0 (0.0)
	Staphylococcus aureus (n=6)	3 (50.0)
	Enterobacter (n=2)	0 (0.0)
Colistin sulfate	*Acinetobacter* (n=22)	7 (31.8)
	Klebsiella (n=30)	17 (56.7)
	E. coli (n=1)	0 (0.0)
	Pseudomonas (n=28)	8 (28.6)
	Staphylococcus aureus (n=6)	0 (0.0)
	Enterobacter (n=2)	0 (0.0)
Linezolid	*Staphylococcus aureus* (n=6)	4 (66.7)
Vancomycin	*Staphylococcus aureus* (n=6)	6 (100.0)
Clindamycin	*Staphylococcus aureus* (n=6)	2 (33.3)
Oxacillin	*Staphylococcus aureus* (n=6)	4 (66.7)

DISCUSSION

In this cohort of 100 adults with hospital-acquired pneumonia, the demographic profile summarized in Table 1, including a mean age of 53.61±14.90 years, clustering in the 51-60 and 61-70 year bands, male predominance (64.0%), high prevalence of diabetes (55.0%), hypertension (36.0%), and heart failure (24.0%), and a mean hospital stay of 9.61±3.76 days, is consistent with the high-risk, medically complex inpatient population described in broader hospital pneumonia series where comorbidity burden and prolonged hospitalization shape colonization pressure, antibiotic exposure, and pathogen selection.[2,4,6] Prior antibiotic use within 90 days (16.0%), prior infection (34.0%), malnutrition (13.0%), and immunosuppressive conditions (10.0%) are exposures repeatedly linked to a higher likelihood of resistant hospital flora and broader empiric therapy requirements in HAP and ICU infection datasets, providing plausible host-level context for the high MDR burden observed microbiologically in this cohort.[3] Preventive vaccine uptake was low, with influenza vaccination at 15.0% and pneumococcal vaccination at 12.0%, which, while not a direct determinant of HAP bacteriology, aligns with regional prevention gaps and may contribute indirectly to higher respiratory infection burden and sustained antibiotic selection pressure in hospitals.[13,14] Culture positivity was 78.0%, with single-organism growth in 68.0% and polymicrobial growth in 10.0%, and profuse or moderate colony counts in the majority of culture-positive cases; this yield is comparable to several hospital-based series where sampling strategy and timing influence recovery rates, and it strengthens confidence that the isolate distribution reflects clinically relevant pathogens rather than sporadic colonizers alone.[5,15]

The central and most practice-relevant finding was the marked Gram-negative predominance, 83 of 89 isolates (93.3%), led by *Klebsiella spp*. (33.7%), *Pseudomonas aeruginosa* (31.5%), and *Acinetobacter spp*. (24.7%), together accounting for 89.9% of isolates, a pattern concordant with multiple contemporary HAP and ICU

pneumonia datasets that consistently place these organisms at the core of nosocomial pneumonia ecology.[15-17] Notably, Bahrain NV-HAP data similarly reported *Klebsiella* and *Pseudomonas* as dominant pathogens and demonstrated statistically significant organism distribution differences (p=0.014), reinforcing that Gram-negative predominance is robust across settings, although the exact rank order varies by center and care context.[5] The MDR burden in our isolates was extremely high at 87.8%, aligning with the broader literature describing MDR Gram-negative HAP and VAP as the modern default in many ICUs and high-pressure wards, where selective antibiotic pressure and transmission dynamics sustain resistant lineages.[9,11] Bangladesh ICU reports similarly describe high resistance to commonly used agents such as ceftriaxone, ceftazidime, and fluoroquinolones, supporting the clinical plausibility of the low cephalosporin susceptibility observed here, for example *Acinetobacter* cefepime 4.5% and *Klebsiella* cefepime 0.0%, with ceftriaxone susceptibility remaining low across key Gram-negatives.[12,18] In Ethiopia, *Pseudomonas*-focused HAP analyses also document very high resistance to antipseudomonal cephalosporins in some centers, while long-term data from Germany show ceftazidime resistance in nosocomial *Pseudomonas* can be significantly higher than in community-acquired cases (p=0.046), supporting the need to interpret *Pseudomonas* susceptibility within local hospital selection pressures rather than rely on assumptions of class-wide activity.[7,19] In our series, carbapenem susceptibility remained moderate for the three dominant Gram-negatives, approximately 59-61%, which contrasts with settings reporting much higher carbapenem resistance among *Acinetobacter* and other non-fermenters, including Vietnam ICU surveillance where carbapenem resistance in *A. baumannii* approached 89% and Korea ICU cohorts reporting MDR A. baumannii around 97%, highlighting how center-level antibiotic policy, infection control, and circulating clones can shift the baseline efficacy of carbapenems.[3,16] Colistin susceptibility was variable and comparatively low in *Acinetobacter* (31.8%) and *Pseudomonas* (28.6%), differing from several Bangladesh ICU VAP series that reported near-universal colistin susceptibility, a discrepancy that may reflect differences in patient mix, testing practices, local antimicrobial exposure, and small-sample instability in some comparator datasets, and it further supports the broader stewardship argument that local antibiograms, not external expectations, should drive empiric and targeted therapy in HAP.[10,20-22] Finally, Gram-positive burden was small, S. aureus 6.7%, with preserved glycopeptide activity (vancomycin 100.0%) and oxacillin susceptibility 66.7%, consistent with the variable contribution of *S. aureus* across hospital pneumonia cohorts and underscoring that, in this setting, the dominant clinical challenge remains Gram-negative MDR rather than Gram-positive coverage gaps.[4,11]

This was a single-center, cross-sectional study with a small sample size, so organism distribution and susceptibility patterns may not be generalizable to other hospitals or units. Culture-based diagnosis may have underestimated fastidious pathogens, and the MDR estimate was reported as an overall proportion without isolate-level stratification by organism or ward, limiting deeper resistance pattern inference.

CONCLUSION

In this hospital-acquired pneumonia cohort, bacterial etiology was overwhelmingly Gram-negative, dominated by *Klebsiella spp*., *Pseudomonas aeruginosa*, and *Acinetobacter spp*., and the overall MDR burden was very high. Susceptibility to third and fourth generation cephalosporins was poor among key Gram-negative pathogens, while carbapenem susceptibility remained only moderate, indicating a constrained empiric treatment landscape. These findings support the need for center-specific microbiological surveillance and susceptibility-guided therapy to improve empiric antibiotic selection and stewardship in hospital pneumonia management.

Recommendations

Routine, periodic hospital antibiogram updates specific to HAP should be institutionalized to guide empiric regimens, with stratification by unit where feasible. Antimicrobial stewardship measures should prioritize reducing unnecessary cephalosporin and fluoroquinolone exposure, strengthening de-escalation based on culture results, and ensuring rational use of reserve agents. Infection prevention interventions, including hand hygiene adherence, device-care bundles, and improved respiratory sampling quality, should be reinforced to reduce transmission of MDR Gram-negative organisms. Multicenter studies with isolate-level MDR classification and outcome linkage are recommended to refine empiric protocols and quantify the clinical impact of MDR in Bangladesh settings.

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