

Antibiotic Resistance and Sensitivity Patterns in Pneumonia Pathogens: A Comprehensive Study of Bacterial Isolates and Their Implications for Treatment in Diyala Province

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Antibiotic Resistance and Sensitivity Patterns in Pneumonia Pathogens: A Comprehensive Study of Bacterial Isolates and Their Implications for Treatment in Diyala Province

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Abstract

Background: Pneumonia is an inflammatory process affecting the airways and lung parenchyma, characterized by alveolar inflammation that leads to infiltration and consolidation of the lungs and disruption of gas exchange, which may result in respiratory distress.

Objectives: This study aims to identify the bacterial species that cause pneumonia and to determine their antibiotic resistance patterns.

Methods: This cross-sectional observational study assessed antibiotic resistance among patients with pneumonia in Diyala Province. Patients were identified from hospital and laboratory records over a four-month period (December 1st, 2024 - March 29th, 2025), yielding 100 patients, comprising 55 males and 45 females.

Results: The study population comprised 55% male and 45% female patients. The age distribution showed that most patients were 36 to 75 years old, with the 66-75 age group representing the largest group at nearly 20%. The 36-45 and 46-55 age groups each accounted for more than 15% of patients. Younger patients (under 16 years) and older patients (76-85 years) were the smallest groups, each with percentages under 5%. It was demonstrated that *Streptococcus pneumoniae* accounted for the highest proportion of pneumonia cases. Regarding bacterial resistance, *Pseudomonas luteola* showed significant resistance to ciprofloxacin (75%) and chloramphenicol (75%), while *Streptococcus pneumoniae* showed high resistance to azithromycin and erythromycin (87.5%).

Conclusion: Considerable variation in antibiotic resistance patterns among bacterial species. Seventy-five percent of the high resistance of *Pseudomonas luteola* to ciprofloxacin and chloramphenicol was shown in this study. In addition, it has been shown that *Streptococcus pneumoniae* is the most common cause of pneumonia and warrants further study to elucidate the factors that influence the spread of this infection.

Keywords: Antibiotic resistance, Pneumonia, Pathogenic bacteria, *Streptococcus pneumoniae*

1. Introduction

Pneumonia is an inflammatory process affecting the airways and lung parenchyma and the inflammation of alveoli, leading to infiltration and consolidation of the lungs and disruption of gas exchange, which may result in respiratory distress

[1]. The pathogens include bacterial, viral, fungal, or aspiration-related pathogens, producing an inflammatory response with vascular congestion, neutrophilic infiltration, and alveolar exudates. Pneumonia can be classified according to different characteristics related to its origin (such as community-acquired

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pneumonia, hospital-acquired pneumonia, and ventilator-associated pneumonia, the causative agent, and the radiographic pattern (lobar pneumonia, bronchopneumonia, and interstitial pneumonia) [2].

Pneumonia is a significant global cause of morbidity and mortality, with an annual incidence rate of 5–11 per 1,000. In contrast, the prevalence is higher in cases of infants, the elderly, and immunocompromised patients. It is responsible for 2.5 million deaths per year, with the greatest burden observed in low- and middle-income countries. HAP and VAP are particularly worrying because of the increased sepsis risk and organ dysfunction due to multidrug-resistant (MDR) pathogens, including *P. aeruginosa*, *A. baumannii*, and methicillin-resistant *S. aureus* [3, 4]. The pathogenesis of pneumonia varies with the causative agent, with bacterial pneumonia (e.g., *S. pneumoniae*, *K. pneumoniae*, and *M. pneumoniae*) characterized by capsule-mediated immune evasion, cytotoxicity mediated by pneumolysin, and the formation of alveolar exudate. In contrast, viral pneumonia (influenza virus, SARS-CoV-2, RSV, etc.) induces direct cytopathic effects, cytokine storms, and endothelial dysfunction that, in the latter case, cause acute respiratory distress syndrome [5, 6].

Diagnosis is primarily clinical, supplemented by imaging and microbiological studies; the gold standard is chest X-ray showing lobar consolidation, patchy infiltrates, or pleural effusion, whereas CT scans are helpful in complicated pneumonia (e.g., cavitory lesions, abscess, and empyema). Lab studies include CBC for leukocytosis (bacterial pneumonia) or normal/low WBC count (viral/fungal pneumonia); CRP and procalcitonin (bacterial infection markers) are elevated; ABG for hypoxemic status. Sputum Gram stain and culture, blood cultures, *Legionella* and *pneumococcal* urinary antigens, viral PCR, and bronchoalveolar lavage in immunocompromised patients guide the identification of pathogens [7]. Timely and precise diagnosis is vital for the administration of pathogen-directed antimicrobial therapy, supportive therapy with oxygen, and the prevention of deleterious sequelae such as septic shock, ARDS, and multi-organ failure [8].

Pneumonia is managed according to risk stratification, empiric antibiotic therapy, supportive care, and monitoring for complications. Initial evaluation and risk assessment is performed using CURB-65 scoring and the Pneumonia Severity Index to stratify the appropriate site of care, including outpatient management for mild cases (CURB-65 score 0-1), hospitalization for moderate cases (score 2-3) and ICU admission for severe cases (score ≥ 4) [9–12]. Follow-up chest X-rays are not routinely required but recommended in non-resolving pneumonia [13]. Timely diagnosis and treatment, including effec-

tive antibiotic use, supportive care, and ongoing assessment, contribute significantly to better clinical outcomes and lower morbidity and mortality rates in patients with pneumonia [14]. Antibiotic resistance, particularly of multidrug-resistant bacteria, is a significant challenge in clinical management of pneumonia, especially of hospital-acquired pneumonia and ventilator-associated pneumonia, often leading to treatment failure, increased length of hospital stays, increased mortality, and health-care costs [15, 16]. Pneumococcus resistant to multiple drugs, methicillin-resistant *S. aureus* extended-spectrum beta-lactamase-producing Gram-negative bacteria, carbapenem-resistant Enterobacterales, *P. aeruginosa*, and *A. baumannii* [17]. These organisms have multiple resistance mechanisms, including production of various beta-lactamases, efflux pumps that extrude antibiotics, modification of target sites, and mutations of porins [18, 19].

Invasive methicillin-resistant *S. aureus* is another organism associated with severe pneumonia; MRSA is resistant to beta-lactams, so treatment is with vancomycin or linezolid for lung penetration. ESBL-producing *K. pneumoniae* and *E. coli* hydrolyze most beta-lactams with the exception of the carbapenems [20–22]. CRE and *A. baumannii* are resistant to essentially all beta-lactams and require colistin, polymyxins, or combination therapy (cefiderocol, tigecycline, and aminoglycosides) as treatment. Multidrug-resistant *P. aeruginosa*: up to 80% of isolates are resistant because of acquisition or overexpression of efflux pumps and mutations in porins, which render strains non-susceptible to carbapenems, cephalosporins and fluoroquinolones; all isolates require piperacillin-tazobactam, cefepime, ceftolozane-tazobactam or aminoglycosides [23]. Prevention focuses on pneumococcal and influenza vaccination, the implementation of infection control measures, and limiting unnecessary antibiotic use in an attempt to prevent the emergence of resistance [24]. This study aimed to determine the bacterial species that cause pneumonia and determine their antibiotic resistance.

2. Materials and methods

2.1. Study design and setting

The current study is a cross-sectional observational analysis aimed at assessing antibiotic resistance among patients with pneumonia in Diyala province. Data were collected from paper-based hospital and laboratory records over four months, from December 1st, 2024, to March 29th, 2025.

The study included 100 patients diagnosed with pneumonia, comprising 55 males and 45 females.

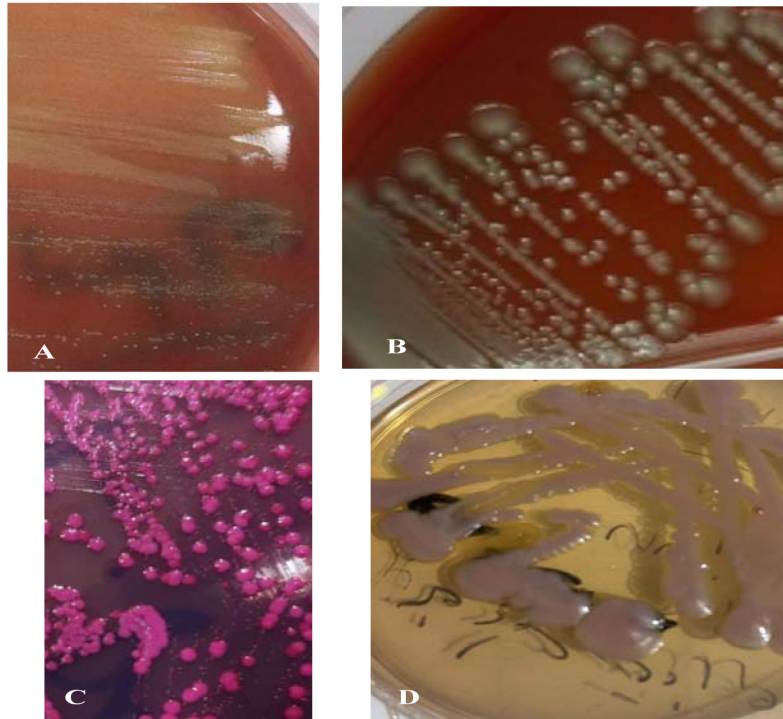


Fig. 1. Types of bacterial species that causes pneumonia in this study. A: *Streptococcus pneumoniae*, B: *Staphylococcus aureus*, C: *Klebsiella pneumoniae*, and D: *Pseudomonas aeruginosa*.

Data collected included age, gender, medical history, pneumonia severity. The specimens were cultured to isolate the bacterial species that cause pneumonia and antibiotic susceptibility test was done for each bacterial species. It was determine the multidrug-resistant (MDR), extensively drug-resistant (XDR), and pan-drug-resistant (PDR) bacteria. Bacterial isolates were identified using culture-based methods, Gram staining, and antimicrobial susceptibility testing (AST) by disk diffusion or automated systems (e.g., VITEK 2, BD Phoenix) [4, 23] (Fig. 1).

2.2. Statistical analysis

Data were analyzed using SPSS software. Frequencies and percentages were used to describe patient demographics (age, gender), pneumonia severity (mild, moderate, severe), and resistance patterns (MDR, XDR, PDR). Results were presented in tables and graphs to illustrate the distribution of antibiotic resistance across different bacterial isolates.

3. Results

The results showed the distribution of patients by gender. It indicates that 55% of patients are male and 45% are female (Fig. 2). The bar chart shows the distribution of patients according to their age groups.

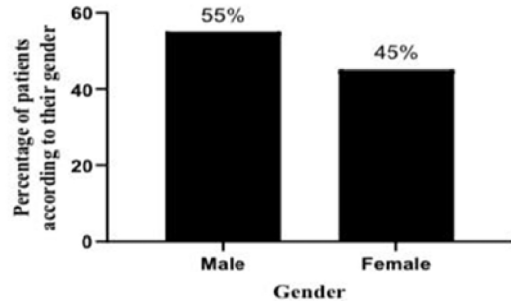


Fig. 2. The percentage of pneumonia patients according their gender.

The highest percentage of patients falls within the 66-75 age group, accounting for nearly 20% of the total population. The 36-45 and 46-55 age groups also show notable percentages, each slightly exceeding 15%. The groups of "less than 16" and "76-85" show the lowest percentages, below 5% (Fig. 3). It was shown in this study that there are different types of bacteria that cause pneumonia in Diyala province, as shown in Fig. 4.

Table 1 shows the antibiotic resistance and sensitivity of *Pseudomonas luteola* (4% of the cases). Amikacin demonstrates 100% sensitivity with no resistance. Gentamicin is 75% sensitive and 25% resistant. Ciprofloxacin has 25% sensitivity and 75% resistance. Meropenem, levofloxacin, and cefepime

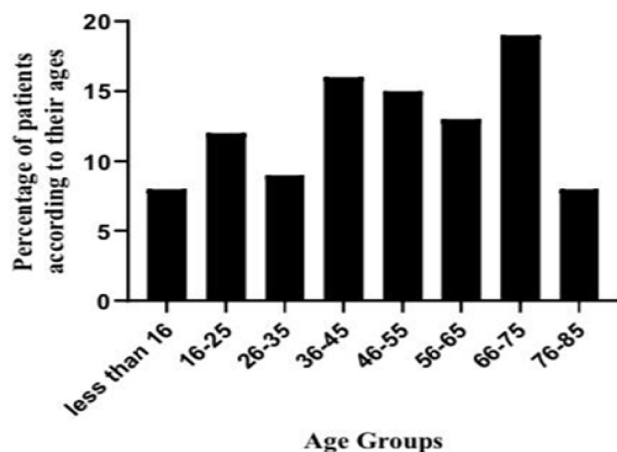


Fig. 3. The distribution of pneumonia patients according to their ages.

Table 1. Antibiotic resistance and sensitivity of *Pseudomonas luteola* in pneumonia patients.

Antibiotic	Sensitive	Resistant
amikacin	100%	
Gentamicin	75%	25%
Ciprofloxacin	25%	75%
Meropenem	50%	50%
Levofloxacin	50%	50%
cefepime	50%	50%
chloramphenicol	25%	75%

Table 2. Antibiotic resistance and sensitivity of *Pseudomonas aeruginosa* in pneumonia patients.

Antibiotic	Sensitive	Resistant
Amikacin	100%	
Levofloxacin	33.3%	66.6%
norfloxacin	66.6%	33.3%
Meropenem	100%	
Ofloxacin	33.3%	66.6%
Gentamicin	33.3%	66.6%
Ciprofloxacin	33.3%	66.6%

each show 50% sensitivity and 50% resistance. Chloramphenicol is 25% sensitive and 75% resistant.

Table 2 shows the antibiotic resistance and sensitivity of *P. aeruginosa*. Amikacin and meropenem are 100% sensitive with no resistance. Levofloxacin, ofloxacin, gentamicin, and ciprofloxacin each show 33.3% sensitivity and 66.6% resistance.

Table 3 shows the antibiotic resistance and sensitivity of *Staphylococcus.hemolytica* (1% of the cases). All antibiotics listed, including Rifampicin, Vancomycin, Gentamicin, Doxycycline, Levofloxacin, Azithromycin, and Erythromycin, show 100% sensitivity.

Table 4 shows the antibiotic resistance and sensitivity of *E. coli*. All antibiotics listed, including Amikacin, Cefepime, Ertapenem, Gentamicin,

Table 3. Antibiotic resistance and sensitivity of *S. hemolytica* in pneumonia patients.

Antibiotic	Sensitive	Resistant
Rifampicin	100%	
Vancomycin	100%	
Gentamicin	100%	
Doxycycline	100%	
Levofloxacin		100%
Azithromycin		100%
Erythromycin		100%

Table 4. Antibiotic resistance and sensitivity of *Escherichia coli* in pneumonia patients.

Antibiotic	Sensitive	Resistant
amikacin	100%	
Cefepime	100%	
Ertapenem	100%	
Gentamicin	100%	
Piperacillin /tazobactam	100%	
Imipenem	100%	
Levofloxacin		100%
Ciprofloxacin		100%
Ampicillin		100%
Trimethoprim /sulfamethaxazole		100%

Table 5. Antibiotic resistance and sensitivity of *Enterococcus faecium* in pneumonia patients.

Antibiotic	Sensitive	Resistant
Cefepime	100%	
Penicillin G	100%	
linezolid	100%	
Ampicillin	100%	
Teicoplanin	100%	
Levofloxacin		100%
Moxifloxacin		100%
Rifampicin		100%
Erythromycin		100%
Ceftriaxone		100%
Trimethoprim /sulfamethaxazole		100%

Piperacillin/ tazobactam, Imipenem, Levofloxacin, Ciprofloxacin, Ampicillin, and Trimethoprim/ sulfamethoxazole, show 100% sensitivity.

Table 5 shows the antibiotic resistance and sensitivity of *E. faecium*. All antibiotics listed, including Cefepime, Penicillin G, Linezolid, Ampicillin, Teicoplanin, Levofloxacin, Moxifloxacin, Rifampicin, Erythromycin, Ceftriaxone, and Trimethoprim/sulfamethoxazole, show 100% sensitivity.

The results also revealed that all *S. aureus* in pneumonia patients were highly resistant (100%) to Levofloxacin, Ofloxacin, Azithromycin, and Norfloxacin, but they were highly sensitive Gentamicin.

Regarding the antibiotic susceptibility of *Pseudomonas putida* in pneumonia patients, all isolates were totally resistant (100%) to all antibiotics tested in the study except for Doxycycline, Minocycline.

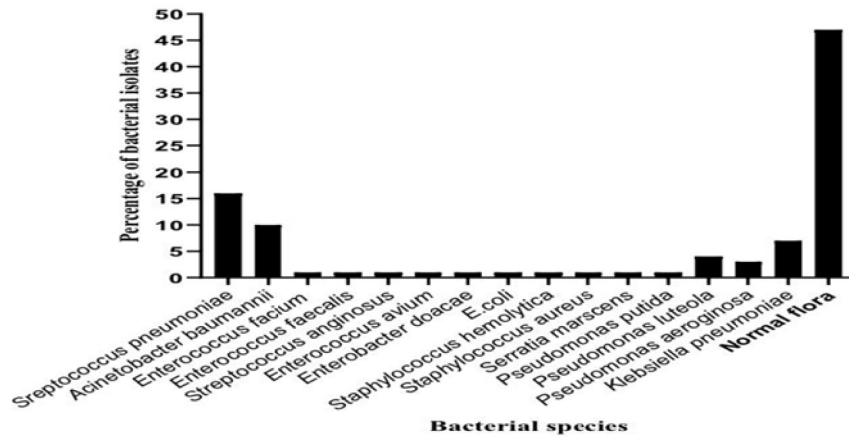


Fig. 4. The bacterial species that cause pneumonia in this study.

The results also found that *Serratia marcescens* isolates were highly resistant to Levofloxacin, Gentamicin, Ampicillin, and Sulbactam, but they showed high sensitivity Minocycline, Imipenem, and Meropenem.

The results also revealed that all *S. anginosus* isolates recovered from pneumonic patients were highly resistant to Cefepime, Imipenem, Ertapenem, Meropenem, and Ampicillin.

Regarding the antibiotic susceptibility of *Enterococcus spp.*, *Enterococcus faecalis* were resistant (100%) to Erythromycin, and Trimethoprim/sulfamethoxazole, but they were highly sensitive to Levofloxacin, Linezolid, and Nitrofurantoin. However, *Enterococcus avium*, isolates were totally resistant to all antibiotics tested in the study.

The results also revealed that all *E. cloacae* isolates were totally sensitive to Amikacin, Cefepime, Ampicillin /Sulbactam, Meropenem, Imipenem, Levofloxacin, Trimethoprim /Sulfamethaxazole, and Minocycline, whereas they were totally resistant to Chloramphenicol, Azithromycin, Cefotaxime, and Ampicillin.

Regarding the antibiotic susceptibility of *S. pneumoniae*, Azithromycin and Erythromycin each showed 12.5% sensitivity and 87.5% resistance. Levofloxacin showed 62.5% sensitivity and 37.5% resistance. Rifampicin showed 43.75% sensitivity and 56.25% resistance. Ofloxacin showed 25% sensitivity and 75% resistance. Doxycycline showed 18.75% sensitivity and 81.25% resistance. Ampicillin showed 93.75% sensitivity and 6.25% resistance.

Regarding the antibiotic susceptibility of *A. baumannii*, isolates showed resistance 80% for Cefepime, Levofloxacin, Amikacin, and Trimethoprim/sulfamethoxazole, whereas they showed 70% for Gentamicin, Meropenem, and Piperacillin/tazobactam.

Regarding the antibiotic susceptibility of *K. pneumoniae*. Doxycycline shows 28.6% sensitivity and 71.4%

resistance. Meropenem shows 71.4% sensitivity and 28.6% resistance. Levofloxacin, Amikacin, Gentamicin, and Cefepime each show 42.9% sensitivity and 57.1% resistance.

4. Discussion

Antimicrobial-resistances differed among the bacteria responsible for pneumonia, with *Pseudomonas luteola*, *Pseudomonas aeruginosa*, and *Klebsiella pneumoniae* as the three most-prolific pathogens in our study. Ciprofloxacin and chloramphenicol showed also the highest resistance rate of 75% for *Pseudomonas luteola*. Similar reports have shown that *Pseudomonas* species displayed high levels of resistance to fluoroquinolones and older antibiotics worldwide, as reported by Yimer et al. [25]. But amikacin showed 100% sensitivity which does not match with Hashim et al. [26], who also noted *Pseudomonas* species to be very sensitive to amikacin. It indicates that amikacin still has a therapeutic possibility rather than resistance to another commonly used antibiotic. Moreover, the moderate resistance to meropenem and levofloxacin (50%) [27], which reported the same resistance profile in *Pseudomonas* species.

For *P. aeruginosa*, the greatest resistance was observed with levofloxacin and ciprofloxacin, where 66.6% were resistant to both. This is in line with the findings of Tilahun et al. which also demonstrated high resistance to fluoroquinolones for *P. aeruginosa*. Nonetheless, amikacin was completely sensitive (100%) in our study, making it a therapeutic choice to treat *Pseudomonas* infections, as reported by Ayatollahi et al. [28]. It is critical to highlight this moderate resistance to meropenem (50%) because carbapenems are essential to treat multidrug-resistant infections. This growing resistance trend underscores the importance of judicious antibiotic

selection and additional efforts for preserving carbapenem utility.

For *K. pneumoniae*, resistance was highest to ampicillin (78.3%) and cefalotin (75%) and is consistent with the global trend of increasing beta-lactam resistance related to *Klebsiella* species. Hashim et al. reported similar findings [26] observed that resistance to ampicillin and other beta-lactams was high. However, *K. pneumoniae* remained highly sensitive to meropenem (82.8%) and cefepime (82%), which is consistent with Yimer et al., who observed comparable sensitivity to carbapenems. Our findings indicate that while *Klebsiella* infections continue to be susceptible to carbapenems and cephalosporins, it is necessary to remain vigilant as resistance levels continue to climb.

In summary, this study demonstrates the rising trend of resistance in *P. luteola*, *P. aeruginosa* and *K. pneumoniae* towards its commonly used antibiotics especially the fluoroquinolones and beta lactam antibiotics. In contrast, amikacin continues to be highly effective in vitro against all tested strains, highlighting its critical role as a treatment option despite the emergence of resistance to other antibiotics. The results also indicate high levels of carbapenem resistance, particularly from *P. aeruginosa* and *K. pneumoniae*, including very high levels in some areas, which is very worrisome since they are often our last resort when treating multidrug-resistant infections. These trends mirror a relative global issue which has also been highlighted by several studies worldwide [25, 26, 28, 29].

5. Conclusion

It was found that *S. pneumoniae* was the most common cause of pneumonia in Diyala city. Considerable variation in antibiotic resistance patterns among bacterial species. Seventy-five percent of the high resistance of *P. luteola* to ciprofloxacin and chloramphenicol was reported. In contrast, the most pertinent pathogenic isolates were *S. aureus* and *Enterococcus faecalis*, which were completely sensitive to most antibiotics, indicating that these pathogens remain susceptible to standard treatment. The patient-described pneumonia occurred more frequently in middle-aged to elderly people aged 36 to 75 years, which requires more systematic and age-appropriate treatment approaches.

Ethical approval

Approval for conducting the study was obtained from the Committee of College of Medicine, University of Diyala code no. (2025 ASM 907). All personal data were anonymized to protect patient confidential-

ity, and ethical guidelines for handling clinical data were strictly followed.

Source of funding

The study was funded by self-funding and without any other source of funding.

Conflict of interest

None.

Use of generative artificial intelligence

The authors state that they did not use any generative AI tools for creating or editing the language of the manuscript.

Author contribution

All authors contributed equally to the research. Anfal Shakir Motib designed the study, drafted the initial version, and reviewed the entire work. Anfal Abd Ulmunem Rasheed assisted with patient groupings, experimental procedures, specimen collection, and data analysis. Rafid Abdul-Mahdi Hasani was involved in collecting data on pneumonia patients, performing statistical analyses, interpreting data, and preparing figures and tables.

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