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Investigation of Antibiotic Resistance Profiles of *Acinetobacter* spp. Isolated from ICU Samples in Mashhad, Iran (October 2018–May 2019)

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Abstract

Background & Objectives: *Acinetobacter* spp. are non-fermentative, Gram-negative, opportunistic pathogens characterized by high levels of antibiotic resistance and are frequently associated with nosocomial infections in intensive care units (ICUs). This study aimed to investigate the antibiotic resistance profiles of *Acinetobacter* spp. isolated from ICU patients using the BD Phoenix system, which determines antimicrobial susceptibility based on the Minimum Inhibitory Concentration (MIC) method.

Materials & Methods: A descriptive, cross-sectional study was conducted from October 2018 to May 2019 involving 60 ICU patients at Mehr-e Hazrat Abbas Hospital in Mashhad, Iran. Clinical specimens—including blood, bronchoalveolar lavage, tracheal secretions, wound swabs, biopsies, pleural fluid, and sternum swabs—were collected under sterile conditions. *Acinetobacter* species were identified through standard culture techniques and confirmed via PCR (polymerase chain reaction) targeting the *blaOXA-51-like* gene. Antimicrobial susceptibility testing against 21 antibiotics was performed using the BD Phoenix system in accordance with CLSI guidelines. Data were analyzed using SPSS software employing chi-square tests ($p < 0.05$).

Results: All 60 isolates were confirmed as *Acinetobacter* spp. Colistin demonstrated the highest susceptibility rate (90%). The highest resistance rates were observed against aminoglycosides, carbapenems, and quinolones (each 55%), followed by cephalosporins, macrolides, and β-lactamase inhibitors (54%), sulfonamides (43%), and monobactams (40%).

Conclusion: The findings underscore the alarming rise in antibiotic resistance among *Acinetobacter* spp. and highlight the necessity of implementing targeted antibiotic stewardship programs and localized surveillance systems to optimize treatment outcomes and curb the spread of resistance.

Keywords: *Acinetobacter spp,* ICU, nosocomial infections, antibiotic-resistant pattern, Phoenix system

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Introduction

The genus *Acinetobacter* is a Gram-negative, non-motile, capsulated, obligate aerobic coccobacillus that lacks spores and is incapable of fermenting glucose (1). *Acinetobacter*, as a nosocomial pathogen, primarily affects patients in intensive care units (ICUs), particularly those with trauma, burns, or injuries and individuals requiring mechanical ventilation

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(2). This opportunistic pathogen is responsible for numerous infections, including pneumonia, meningitis, endocarditis, skin and soft tissue infections, conjunctivitis, burn wound infections, and bacteremia (3, 4). *Acinetobacter* spp. exhibit resistance to a wide range of antibiotics and can survive for extended periods in hospital environments. Reports indicate that among hospital-acquired Gram-negative bacteria, *Acinetobacter baumannii* has developed drug resistance at an accelerated rate (5), resulting in the emergence of extensively drug-resistant (XDR) and pan-drug-resistant (PDR) strains through the acquisition and utilization of multiple antibiotic resistance mechanisms (6).

Acinetobacter produces oxacillinase enzymes that hydrolyze carbapenems, leading to resistance to both carbapenems and penicillins. Other factors contributing to resistance in drug-resistant *Acinetobacter* strains include alterations in purine channels, modifications in penicillin-binding proteins, production of aminoglycoside-modifying enzymes, plasmid-mediated quinolone resistance, and activation of efflux pump mechanisms (7–9). Treatment of infections caused by these bacteria commonly involves β-lactams and fluoroquinolones; however, in recent years, the use of these agents has promoted the emergence of resistant strains (10). Antibiotic resistance has complicated the management of *Acinetobacter*-related infections, leading to prolonged hospitalization, increased healthcare costs, poorer prognoses, and higher mortality rates compared with infections caused by susceptible strains (6, 11).

Varying levels and patterns of antibiotic susceptibility have been reported among different *Acinetobacter* species, with *A. baumannii* showing a markedly higher prevalence of resistance than other species (12, 13). Rapid identification of antibiotic-resistant strains is essential for controlling and preventing the spread of drug-resistant isolates in healthcare facilities and for assisting clinicians in selecting appropriate antimicrobial therapy (14, 15).

Recent studies have further underscored the global threat posed by carbapenem-resistant *A. baumannii* (CRAB), emphasizing its rapid genomic adaptability and enhanced virulence in ICU environments (16). For example, Tagueha et al. (2025) demonstrated that recent respiratory isolates exhibit stronger biofilm formation and greater invasiveness than earlier strains, suggesting an evolutionary trend toward heightened bloodstream virulence. Similarly, Boutzoukas and Doi (2025) reviewed the worldwide epidemiology of CRAB, documenting its endemic presence in hospitals and its strong association with elevated mortality rates, particularly in bloodstream infections (17). Scoffone et al. (2025) identified emerging resistance mechanisms and highlighted the urgent necessity of developing alternative therapeutic strategies to combat this pathogen (18).

Identifying the antibiotic susceptibility patterns in each region is crucial for preventing the dissemination of antibiotic resistance. Therefore, this study aimed to determine the antibiotic resistance patterns of *Acinetobacter* spp isolated from ICU patients using the BD Phoenix system.

Materials and Methods

Patients and isolates

Study Design and Patient Selection

This descriptive cross-sectional study was conducted from October 2018 to May 2019 among 60 patients admitted to the ICU of Mehr-e Hazrat Abbas Hospital in Mashhad, Iran. Eligible participants were adults aged 18 years or older who had been hospitalized in the ICU for at least 48 hours and exhibited clinical symptoms of respiratory, bloodstream, or urinary tract infections. Patients with infections caused by organisms other than *Acinetobacter* or those admitted with pre-existing infections were excluded to ensure that the isolates represented true nosocomial infections.

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Sample Collection and Bacterial Isolation

Clinical specimens—including blood, urine, lung secretions, tissue biopsies, pleural fluid, bronchoalveolar lavage (BAL), sternum swabs, and blood cultures—were collected under sterile conditions and transported immediately to the microbiology laboratory. Standard microbial culture techniques were applied to isolate *Acinetobacter* species, and only samples yielding *Acinetobacter* growth were included for further analysis.

Phenotypic Identification

Initial identification was carried out using biochemical tests, including growth on MacConkey agar (non-lactose-fermenting colonies), oxidase test (negative), sulfide indole motility (SIM) medium (non-motile), triple sugar iron (TSI) agar (alkaline slant and butt), and absence of pigment production. Colonies consistent with Gram-negative bacilli were subjected to automated identification using the BD Phoenix™ system. Bacterial suspensions were standardized to 0.5 McFarland turbidity in nutrient broth and loaded into Gram-negative identification panels. The Phoenix system provided species-level identification and conducted antimicrobial susceptibility testing using the Minimum Inhibitory Concentration (MIC) method.

Molecular Identification of Acinetobacter Species

To confirm species-level identification of *Acinetobacter* isolates recovered from ICU samples, molecular assays were employed. Genomic DNA was extracted from pure cultures using QIAamp DNA Kits, and PCR (polymerase chain reaction) amplification targeting the *blaOXA-51-like* gene was performed to identify *A. baumannii*, as this gene serves as a reliable species-specific marker. Additionally, universal 16S rRNA primers were utilized for genus-level confirmation, and species-specific primers targeting genes such as *gyrB* were used to distinguish closely related *Acinetobacter* species. PCR reactions included appropriate positive and negative controls to ensure specificity, accuracy, and reproducibility.

Determination of Antibiotic Sensitivity

Antibiotic susceptibility was evaluated for 22 antimicrobial agents following the Clinical and Laboratory Standards Institute (CLSI) guidelines. The antibiotics tested included amikacin, gentamicin, ertapenem, meropenem, imipenem, cephalothin, cefuroxime, cefoxitin, ceftazidime, ceftriaxone, cefepime, aztreonam, ampicillin, amoxicillin–clavulanate, piperacillin–tazobactam, colistin, trimethoprim–sulfamethoxazole, nitrofurantoin, ciprofloxacin, levofloxacin, and tigecycline. Susceptibility results were classified as sensitive, intermediate, or resistant according to CLSI criteria.

Statistical Analysis

Statistical analysis was conducted using SPSS software version 26 (IBM Corp., Chicago, USA). Qualitative variables were expressed as frequencies and percentages. Comparisons between categorical variables were performed using the chi-square test, and a *p*-value less than 0.05 was considered statistically significant.

Results

A total of 60 *Acinetobacter* isolates were obtained from various clinical specimens collected from 60 ICU patients. Among these patients, 58% were male and 42% were female. The distribution of isolates based on the source of infection included 8 tracheal aspirates, 7 blood samples, 27 bronchoalveolar lavage (BAL) specimens, 4 wound swabs, 3 tissue samples, 3 lung secretion samples, 4 pleural fluid specimens, 3 blood cultures, and 2 sternum isolates. Based on species identification, 18 isolates were confirmed as *A. baumannii*, while 42 belonged to other *Acinetobacter* species. According to the antibiotic susceptibility profiles, gentamicin showed the highest resistance rate among aminoglycosides, with 55 resistant isolates. Within the carbapenem class, imipenem and meropenem exhibited

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the highest resistance rates (53 isolates each), whereas ertapenem demonstrated the lowest (37 isolates). Among cephalosporins, ceftazidime showed the greatest resistance (54 resistant isolates), whereas cefazolin exhibited the lowest (13 resistant isolates). Resistance to monobactams was observed in 40 isolates tested against aztreonam. In the aminopenicillin group, ampicillin displayed the lowest resistance rate, while amoxicillin–clavulanate demonstrated the highest. Piperacillin–tazobactam, the only β-lactam/β-lactamase inhibitor combination tested, exhibited resistance in 54 isolates. Colistin demonstrated the lowest resistance rate in the entire study, with only one resistant isolate. Trimethoprim–sulfamethoxazole, belonging to the sulfonamide class, showed a resistance rate of 43 isolates. In the quinolone group, ciprofloxacin exhibited the highest resistance rate (55 isolates), whereas levofloxacin demonstrated the lowest (52 isolates). A comprehensive summary of the antibiotic resistance patterns is presented in Table 1.

Discussion

Despite substantial advances in hospital care and the widespread use of antibiotics, *A. baumannii* remains one of the most persistent and formidable pathogens responsible for hospital-acquired infections, particularly in ICU settings

Table 1. Antibiogram Results for *Acinetobacter* Isolates

Antibiotics	Resistance isolates	Sensitive isolates	Intermediate isolates
Aminoglycosides			
Gentamicin	55	0	2
Amikacin	54	2	4
Carbapenems			
Ertapenem	37	0	0
Imipenem	53	1	2
Meropenem	53	1	3
Cephalosporins			
Cefazolin	13	0	0
Cephalothin	24	0	0
Cefuroxime	37	0	0
Cefoxitin	24	0	0
Ceftazidime	54	2	1
Cefepime	53	2	2
Monobactams			
Azetronam	40	0	0
Aminopenicillin			
Ampicillin	37	0	0
Amoxicillin Clavulanate	55	0	0
beta-lactam antibiotics			
Piperacillin/ Tazobactam	54	3	0
Polymyxin			
Colistin	1	50	0
Sulfonamide			
Trimethoprim sulfa	43	14	0
Macrolides			
Nitrofurantoin	55	0	0
Quinolone			
Ciprofloxacin	55	2	0
Levofloxacin	52	2	3

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(19-21). Its strong association with ventilator-associated pneumonia and high mortality rates highlights its profound clinical significance. The pathogen's ability to survive on abiotic surfaces, resist disinfectants, and rapidly acquire resistance genes has made it a critical global threat in healthcare environments. Lin and Lan emphasized that *A. baumannii* employs diverse resistance mechanisms, including beta-lactamase production and biofilm formation, which enhance its persistence under selective pressures and complicate treatment and eradication efforts (19, 22). Moreover, the presence of plasmid-borne resistance genes, which are prone to mutation and facilitate horizontal gene transfer via conjugation and transformation, accelerates the rapid dissemination of resistance traits across bacterial populations (23, 24). Vrancianu et al. further highlighted the role of carbapenemase genes such as OXA-type enzymes and NDM-1, reinforcing the need for molecular surveillance and innovative therapeutic strategies to contain its spread (25). As Peleg et al. noted, this genetic adaptability has enabled *A. baumannii* to evolve into a highly resilient nosocomial pathogen capable of evading conventional treatments (26).

Our study revealed alarmingly high resistance rates among *A. baumannii* isolates, consistent with global trends. Over 90% of strains exhibited resistance to carbapenems (Imipenem, Meropenem), cephalosporins (Ceftazidime, Ceftriaxone, Cefepime), and aminoglycosides (Amikacin). Additionally, resistance rates exceeded 80% for Levofloxacin and surpassed 40% for Gentamicin, Piperacillin/Tazobactam, and Ciprofloxacin. Notably, Colistin remained the most effective agent, with 98.2% sensitivity, despite its well-documented nephrotoxicity. These findings underscore the urgent necessity for alternative therapeutic strategies and reinforce the pressing importance of addressing multidrug resistance in clinical settings.

Recent global surveillance data have confirmed the alarming rise in multidrug-resistant (MDR) *A. baumannii*, particularly in regions such as Asia and the Mediterranean, where carbapenem resistance rates have exceeded 80% (27). These results are consistent with both regional and global surveillance findings. A meta-analysis of 795 studies conducted across 80 countries (1995–2023) reported global carbapenem resistance rates surpassing 70%, with peaks reaching 81% between 2020 and 2023 (20, 28). In China, resistance to carbapenems increased from 18% in 2012 to 60% in 2019, while Latin America reported rates between 50–75%, and East Africa showed a prevalence of 64.8% (20). In the Middle East, a 12-year retrospective study in the UAE (2010–2021) demonstrated a declining trend in resistance to Imipenem, Meropenem, and Amikacin, although carbapenem-resistant strains remained associated with higher mortality and longer hospital stays (27, 29).

The diversity of resistance patterns across geographical regions is largely influenced by local antibiotic usage practices. For instance, in our region, the resistance profile of *A. baumannii* appears more severe than that reported in previous studies. Hatami (2018) reported resistance rates above 70% for Ceftazidime and Ceftriaxone, and 88% sensitivity to Colistin (30). Maleki et al. found 100% sensitivity to Colistin and 83.3% to Tigecycline in isolates from burn patients, with complete resistance to Piperacillin/Tazobactam and Cephalosporins (31). Similarly, Vilason et al. in Spain observed multidrug resistance to several beta-lactams and fluoroquinolones, while maintaining preserved sensitivity to Colistin (32).

The World Health Organization's AMR Surveillance Report (2024) confirmed that Colistin remains the antibiotic of last resort for MDR *A. baumannii*, although emerging resistance is a growing concern, especially in South America and Southeast Asia (33). Our findings corroborate this observation, as Colistin showed the highest sensitivity among the tested agents. However, its

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nephrotoxicity and the potential for resistance development necessitate judicious use and close monitoring. Khaledi et al. reported 43% resistance to Imipenem, a drug traditionally used to treat *A. baumannii* infections. Their recommendation to use Colistin or Tigecycline, alone or in combination, aligns with our findings, wherein Colistin showed the lowest resistance rate (34). Almaghrabi et al. also confirmed resistance to carbapenems and preserved sensitivity to Colistin in Saudi isolates (35).

The emergence of New Delhi metallo-beta-lactamase (NDM)-producing strains further complicates treatment, as these bacteria exhibit resistance to fluoroquinolones, aminoglycosides, and beta-lactams, including carbapenems, and are often only susceptible to Colistin and Tigecycline. However, resistance to these last-line antibiotics is also rising globally. A systematic review (2004–2024) reported that Colistin resistance **rose** from 2% before 2011 to 5% after 2012, with South America showing the highest rates (29). In India, the Indian Council of Medical Research (ICMR) reported in 2025 that carbapenem resistance in *A. baumannii* isolates from tertiary hospitals exceeded 75%, with Colistin and Tigecycline remaining the only viable therapeutic options (36). This finding mirrors our results, where Tigecycline also demonstrated moderate levels of sensitivity, particularly in isolates from burn patients.

Gestal et al. corroborated our findings, reporting resistance to Ceftazidime, Imipenem, Meropenem, Ciprofloxacin, Cefepime, Gentamicin, Amikacin, and Ampicillin-Sulbactam, with retained sensitivity to Colistin (27). A 2025 study from India likewise confirmed extensive multidrug resistance, with Colistin and Tigecycline remaining effective (28).

Recent genomic studies from Italy (2010–2023) revealed that respiratory isolates of carbapenem-resistant *A. baumannii* have evolved enhanced biofilm formation and increased lung cell invasiveness, suggesting ongoing adaptation to clinical pressures and antibiotic exposure (24). Limongi et al. conducted a genomic analysis of respiratory isolates in Italy between 2010 and 2023, revealing that Colistin-resistant *A. baumannii* strains have developed stronger biofilm formation and greater lung cell invasiveness, indicating continued adaptation to clinical pressures and antibiotic exposure (37). These findings highlight the pathogen's ability not only to resist treatment but also to augment its virulence, posing a dual threat to patient outcomes.

Given the high prevalence of multidrug-resistant *A. baumannii* strains in Asia and the Mediterranean and the documented presence of various carbapenemase genes, it is imperative to implement robust infection control measures and region-specific antibiotic stewardship programs. Determining local resistance patterns is essential for guiding empirical therapy, optimizing treatment protocols, and curbing the overuse and misuse of antibiotics that drive resistance.

Actionable Recommendations

To mitigate the growing threat of multidrug-resistant *A. baumannii*, especially in ICU settings, the following strategies are recommended:

1. Implement carbapenem-use monitoring programs: Establish hospital-wide surveillance systems to monitor carbapenem prescriptions and ensure they are used only when clinically justified. Such monitoring can reduce selective pressure and slow the development of resistance.

2. Strengthen antibiotic stewardship protocols: Develop and enforce evidence-based guidelines for empirical therapy that reflect local resistance data. Promote de-escalation strategies and restrict the use of last-resort agents such as colistin and tigecycline.

3. Enhance infection-control measures: Institute rigorous protocols for hand hygiene, environmental cleaning, and the isolation of colonized or infected patients to limit nosocomial transmission.

4. Expand molecular surveillance: Incorporate

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PCR-based detection of resistance determinants and consider whole-genome sequencing to track the emergence and dissemination of high-risk clones and novel resistance mechanisms.

5. Promote regional data sharing: Collaborate with neighboring hospitals and public health authorities to establish a regional resistance database, thereby enabling more accurate forecasting and coordinated response strategies.

6. Educate healthcare personnel: Conduct regular, targeted training for clinicians, nurses, and microbiologists on current antimicrobial-resistance trends, diagnostic stewardship, and appropriate antibiotic prescribing.

7. Encourage research into alternative therapies: Support clinical and translational studies investigating bacteriophage therapy, antimicrobial peptides, and rational combination regimens to expand therapeutic options for resistant *Acinetobacter* infections.

Conclusion

A. baumannii remains a critical and persistent challenge in ICU settings due to its high prevalence, extensive multidrug resistance, and capacity to survive in harsh environmental conditions. Our study, which aligns with regional and international surveillance data, documents alarmingly high resistance rates to key antibiotic classes, including carbapenems, cephalosporins, aminoglycosides, and fluoroquinolones, while confirming that colistin remains the most effective therapeutic option despite its known toxicity. The emergence of carbapenemase-producing and NDM-positive strains further complicates treatment and underscores the urgent need for enhanced infection control, vigilant antibiotic stewardship, and sustained surveillance. Tailoring empirical therapy to local resistance patterns is essential for improving patient outcomes and limiting resistance spread. Ultimately, addressing the dual threat of antimicrobial resistance and increased virulence in *A. baumannii* requires a multifaceted approach that integrates clinical vigilance, genomic surveillance, and global collaboration.

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Conflict of interests

The authors declare that there are no conflicts of interest.

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Ethical Considerations

This study was reviewed and approved by the Ethics Committee of Mashhad University of Medical Sciences. No human interventions were performed during the study.

Code of Ethics

This study was conducted in accordance with the approval of the Ethics Committee of Mashhad University of Medical Sciences (IR.MUMS.REC.1399.331).

Authors' Contributions

Tahmineh Ghanei Yazdi and Nazanin Ataee contributed to data collection and laboratory analyses. Parastoo Tajzadeh conceptualized and supervised the study and was responsible for manuscript preparation. Masoud Chaboksavar assisted with data interpretation and critically revised the manuscript. All authors read and approved the final version of the manuscript.

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