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Phenotypic co-occurrence of resistance to aminoglycosides and fluoroquinolones in clinical isolates of *Acinetobacter baumannii*

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Abstract

Background and Aim: *Acinetobacter baumannii* is a major nosocomial pathogen associated with multidrug resistance, limiting therapeutic options in hospital settings. This study aimed to investigate antimicrobial susceptibility patterns and the co-occurrence of resistance to aminoglycosides and fluoroquinolones among clinical isolates of A. baumannii recovered from a tertiary hospital in Shahroud, Iran.

Methods: In this descriptive cross-sectional study, six non-duplicate clinical isolates of A. *baumannii* were collected from hospitalized patients. Species identification was confirmed by phenotypic methods and PCR detection of the intrinsic blaOXA-51-like gene. Antimicrobial susceptibility testing was performed using the Kirby–Bauer disk diffusion method and interpreted according to CLSI M100 (2024) guidelines. Data were analyzed descriptively, and hierarchical clustering was used for exploratory visualization of resistance patterns.

Results: All isolates (6/6, 100%) were classified as multidrug-resistant (MDR), and two isolates (2/6, 33.3%) met the criteria for extensively drug-resistant (XDR). All isolates (6/6, 100%) were resistant to ciprofloxacin. High resistance rates were observed among aminoglycosides, including amikacin (5/6, 83.3%), gentamicin (5/6, 83.3%), and tobramycin (5/6, 83.3%). Resistance to ceftazidime was observed in 5/6 isolates (83.3%), while resistance rates for cefepime, piperacillin–tazobactam, and ampicillin–sulbactam were 3/6 (50.0%) each. Resistance to carbapenems was detected in 4/6 isolates (66.7%) for meropenem and 3/6 (50.0%) for imipenem. Concurrent resistance to fluoroquinolones and aminoglycosides was observed across all isolates, indicating co-occurrence of resistance phenotypes.

Conclusion: The present study demonstrates a high burden of multidrug resistance and frequent co-occurrence of resistance to aminoglycosides and fluoroquinolones among clinical isolates of A. baumannii. These findings highlight the need for continued regional antimicrobial resistance surveillance and strengthened infection control strategies.

Keywords: *Acinetobacter baumannii*, co-resistance, aminoglycosides, fluoroquinolones, multidrug resistance

1. Introduction

Acinetobacter baumannii is a Gram-negative, aerobic, coccobacillus of the family Moraxellaceae. It has become one of the most troublesome opportunistic nosocomial pathogens in the world, especially in intensive care units (ICUs) where it causes serious infections such as ventilator-associated pneumonia, bloodstream infection, wound infection, meningitis, and urinary tract infection (1-3). This organism is very environmentally resilient and can survive for weeks to months on dry surfaces, and is resistant to many hospital disinfectants, making it a major contributor to its persistence and spread in hospitals. Multidrug-

resistant (MDR) and extensively drug-resistant (XDR) A. baumannii strains have become a significant threat to public health and have gained widespread prevalence in the last 20 years. A. baumannii is a critical pathogen that requires new antibiotics, according to the World Health Organization (WHO) (4, 5). This bacterium is extremely resistant due to intrinsic and acquired resistance mechanisms such as the production of a variety of β-lactamases (including carbapenemases), changes in outer membrane porins, changes in antibiotic target sites, and overexpression of multidrug efflux pumps (6, 7).

Aminoglycosides and fluoroquinolones have been historically important agents in the treatment of A. baumannii infections among other therapeutic options that remain. Aminoglycosides are bactericidal agents that bind to 30S ribosomal subunit and inhibit protein synthesis, while fluoroquinolones inhibit DNA gyrase and topoisomerase IV, which block DNA replication. However, many studies around the world have reported the development of cross-resistance between these two structurally different classes of antibiotics (7-9). Cross-resistance is mainly due to the overexpression of Resistance-Nodulation-Division (RND) family efflux pumps, particularly AdeABC and AdeIJK, and to the presence of co-existing genetic determinants like mutations in gyrA/parC and genes for aminoglycoside-modifying enzymes (AMEs) often found on mobile genetic elements (9-11). Although this cross-resistance has been well documented in international literature, there are significant gaps in the literature, especially in LMICs. Several studies have reported high frequencies of antimicrobial resistance in A. baumannii isolates in Iran (12-14), but most studies have been conducted on carbapenem resistance or the overall MDR pattern. Information on the degree and mode of cross-resistance between aminoglycosides and fluoroquinolones is scarce, particularly in the northeast provinces like Semnan. This dearth of local epidemiological data limits the ability to establish empirical treatment guidelines based on evidence and to implement effective antimicrobial stewardship programs in the region. Hence, the present study was carried out to overcome this gap in knowledge. The primary aims of this study were to determine antimicrobial susceptibility patterns of clinical A. baumannii isolates and to evaluate the co-occurrence of resistance between aminoglycosides and fluoroquinolones using descriptive statistical methods. between resistance phenotypes of both classes of antibiotics. The purpose of this study is to make more rational decisions on therapeutic management by clinicians, reinforce infection control measures and provide a basis for future, larger-scale molecular epidemiological studies in the area.

2. Methods

Study Design and Bacterial Isolates

This descriptive cross-sectional study was conducted in 2025 at Imam Hossein Hospital, Shahroud, Iran. Six non-duplicate clinical isolates of *Acinetobacter baumannii* (ABI082, ABI028, ABH080, ABI115, ABH025, and ABI003) were recovered from hospitalized patients in different wards, including intensive care unit (ICU), internal medicine, and surgery. The isolates were obtained from urine, wound exudates, blood, and respiratory secretions.

Isolation and Phenotypic Identification

Initial bacterial isolation was performed using Blood Agar and MacConkey Agar (Merck, Germany). Colonies were selected based on the typical morphology of A. baumannii, including smooth, convex, non-pigmented, and non-hemolytic appearance. Presumptive identification was carried out using standard biochemical tests including catalase activity (3% H_2O_2), oxidase test, non-motility in SIM medium, absence of lactose fermentation on MacConkey agar, and growth at 44°C.

Molecular Identification

Species-level confirmation was performed by PCR amplification of the intrinsic blaOXA-51-like gene, a reliable molecular marker for *A. baumannii*. Genomic DNA extraction for all six isolates was performed using a single standardized protocol. Briefly, bacterial colonies were suspended in sterile distilled water and subjected to thermal lysis (boiling at 100°C for 10 min), followed by centrifugation at 12,000 rpm for 10 min. The supernatant was used as DNA template. For all isolates, DNA quality was sufficient for downstream PCR analysis; therefore, no commercial extraction kit was required or used in this study. PCR amplification was performed in a total volume of 25 µL containing Master Mix (Amplicon, Denmark), template DNA, and primers. The forward and reverse primers were 5′-TAATGCTTTGATCGGCCTTG-3′ and 5′-TGGATTGCACTTCATCTTGG-3′, respectively. Thermal cycling products were visualized by electrophoresis on 1.5% agarose gel, and a 353 bp band confirmed the presence of the blaOXA-51-like gene. *A. baumannii* ATCC 19606 was used as a positive control and nuclease-free water as a negative control.

Antimicrobial Susceptibility Testing

Antimicrobial susceptibility testing was performed using the Kirby–Bauer disk diffusion method on Mueller–Hinton agar (Merck, Germany) according to the CLSI M100 (2024) guidelines. A 0.5 McFarland

bacterial suspension (approximately 1.5 × 10^8 CFU/mL) was prepared and inoculated onto Mueller–Hinton agar plates. Antibiotic disks (Mast Diagnostics, UK) were applied, and plates were incubated at 37°C for 18–24 h. Zone diameters were measured and interpreted as susceptible (S), intermediate (I), or resistant (R) strictly according to the CLSI-approved breakpoints for *A. baumannii-calcoaceticus* complex. Only antibiotics with established CLSI interpretive criteria were included in the final analysis; these included: amikacin, gentamicin, tobramycin, ciprofloxacin, imipenem, meropenem, ceftazidime, cefepime, piperacillin–tazobactam, ampicillin–sulbactam, trimethoprim–sulfamethoxazole, and tetracycline. Quality control strains included *Escherichia coli* ATCC 25922 and *Pseudomonas aeruginosa* ATCC 27853. Intermediate susceptibility results were reported separately and were not categorized as resistant when calculating antimicrobial resistance rates.

Definition of MDR and XDR

Multidrug-resistant (MDR) and extensively drug-resistant (XDR) isolates were classified according to the international expert proposal for standardized definitions of acquired resistance described by Magiorakos et al. (2012). MDR was defined as non-susceptibility to at least one agent in three or more antimicrobial categories. XDR was defined as non-susceptibility to at least one agent in all but two or fewer antimicrobial categories, meaning that bacterial isolates remained susceptible to only one or two antimicrobial categories (15).

Statistical Analysis

Data were analyzed using SPSS version 26.0 for descriptive statistics (frequencies and percentages). Hierarchical cluster analysis was performed in GraphPad Prism version 9 as an exploratory visualization tool to illustrate similarities in resistance profiles among the isolates. Due to the small sample size (n=6), no inferential statistical tests were conducted.

3. Results

Six non-duplicate clinical isolates of Acinetobacter baumannii (ABI082, ABI028, ABH080, ABI115, ABH025, and ABI003) were recovered from hospitalized patients at Imam Hossein Hospital, Shahroud, from various clinical specimens including urine, wound exudates, blood, and respiratory secretions. All isolates were initially identified using conventional phenotypic methods and subsequently confirmed as A. baumannii by PCR amplification of the intrinsic blaOXA-51-like gene. Based on antimicrobial susceptibility profiles and according to the standardized international definitions proposed by Magiorakos et al., all six isolates (6/6, 100%) were classified as multidrug-resistant (MDR). Among these, two isolates (ABH080 and ABI115) met the criteria for extensively drug-resistant (XDR) phenotypes. Antimicrobial susceptibility testing demonstrated high levels of resistance to several clinically relevant antimicrobial agents. All isolates (6/6, 100%) were resistant to ciprofloxacin. Resistance to aminoglycosides was also common, with five isolates (5/6, 83.3%) resistant to amikacin, gentamicin, and tobramycin. Among β-lactam agents, resistance was observed to ceftazidime in five isolates (5/6, 83.3%), while resistance to cefepime, piperacillin–tazobactam, and ampicillin–sulbactam was detected in three isolates (3/6, 50.0%) each. Resistance to carbapenems was identified in four isolates (4/6, 66.7%) for meropenem and three isolates (3/6, 50.0%) for imipenem. Resistance to trimethoprim–sulfamethoxazole and tetracycline was observed in five isolates (5/6, 83.3%) for each agent. The antimicrobial resistance profiles varied among isolates. ABI115 exhibited the broadest resistance pattern and was resistant to nearly all tested antimicrobial categories, whereas ABH025 showed a comparatively lower level of resistance and remained susceptible to several β-lactam agents and amikacin. A phenotypic co-occurrence of resistance to fluoroquinolones and aminoglycosides was observed among the studied isolates, characterized by universal resistance to ciprofloxacin together with high resistance rates to amikacin, gentamicin, and tobramycin. Because molecular mechanisms were not investigated, these findings should be interpreted solely as phenotypic resistance patterns and not as evidence of mechanistic cross-resistance. Hierarchical cluster analysis was used as an exploratory visualization tool to assess similarities among antimicrobial resistance profiles. Two major clusters were identified. The first cluster (ABI115, ABH080, and ABI028) comprised isolates with higher overall resistance levels, whereas the second cluster (ABH025, ABI003, and ABI082) included isolates with comparatively lower resistance profiles. Detailed antimicrobial susceptibility results are presented in Table 1.

Table 1. Antimicrobial susceptibility profiles of six clinical *Acinetobacter baumannii* isolates based on CLSI M100 (2024) interpretive criteria

Antibiotic	Class	ABI082	ABI028	ABH080	ABI115	ABH025	ABI003
Ampicillin-sulbactam	β-lactam/β-lactamase	R	I	R	R	S	I

	inhibitor						
Piperacillin-tazobactam	β-lactam/β-lactamase inhibitor	R	I	R	R	S	I
Ceftazidime	Cephalosporin	R	R	R	R	I	R
Cefepime	Cephalosporin	R	I	R	R	S	I
Imipenem	Carbapenem	S	S	R	R	S	R
Meropenem	Carbapenem	R	S	R	R	S	R
Ciprofloxacin	Fluoroquinolone	R	R	R	R	R	R
Amikacin	Aminoglycoside	R	R	R	R	S	R
Gentamicin	Aminoglycoside	R	R	R	R	I	R
Tobramycin	Aminoglycoside	R	R	R	R	I	R
Trimethoprim-sulfamethoxazole	Folate inhibitor	R	R	R	R	I	R
Tetracycline	Tetracycline	R	R	R	R	I	R

Table 1. S: susceptible; I: intermediate; R: resistant. Antimicrobial susceptibility testing was performed using the Kirby–Bauer disk diffusion method and interpreted according to Clinical and Laboratory Standards Institute M100 (2024) guidelines. Only antibiotics with established CLSI interpretive breakpoints for Acinetobacter baumannii-calcoaceticus complex were included in this table. Polymyxin agents (colistin and polymyxin B) were excluded from disk diffusion interpretation due to the absence of CLSI-recommended breakpoints and are not reported in this analysis.

4. Discussion

All of the six clinical isolates of Acinetobacter baumannii from Imam Hossein Hospital, Shahroud were multidrug resistant (MDR) and two of them (ABI115 and ABH080) were extensively drug resistant (XDR). Resistance to fluoroquinolones, especially ciprofloxacin (100%), and aminoglycosides, such as amikacin (83.3%), gentamicin (83.3%), and tobramycin (83.3%) was found to be high. This was also confirmed by hierarchical cluster analysis which showed that the fluoroquinolones and the aminoglycosides were clustered. The results demonstrate a pattern of co-occurrence of resistance between aminoglycosides and fluoroquinolones among the studied isolates. The observed co-resistance is clinically significant as it significantly restricts the few treatment choices currently available for A. baumannii infections. Potential mechanisms underlying the observed co-resistance may include efflux pump overexpression, aminoglycoside-modifying enzymes, and quinolone resistance mutations, although these mechanisms were not investigated in the current study. These pumps actively pump out both fluoroquinolones and aminoglycosides from the bacterial cell, which lowers the level of drug in the cell. Mutations in the regulatory genes adeR and adeS may further enhance the expression of pumps, leading to multidrug-resistant (MDR) phenotypes with a broad spectrum of drugs (14, 16, 17). Plasmids and transposons frequently carry resistance genes which can be horizontally transferred. Aminoglycoside resistance is often conferred by the production of aminoglycoside-modifying enzymes (AMEs) like AAC(6′)-Ib, APH(3′)-VI and ANT(2″)-Ia and fluoroquinolone resistance is mainly due to point mutations in the quinolone resistance-determining regions (QRDRs) of gyrA and parC (7, 18, 19). These mechanisms have been reported in the literature and may coexist in the same strains.

The findings of the present study corroborate previous reports of the country and abroad. The MDR A. baumannii isolates that overexpress AdeABC have been reported to be resistant to both classes of antibiotics, aminoglycosides and fluoroquinolones (20-22). Presence of efflux pumps and plasmid mediated resistance genes has been reported to be a major factor in the spread of multidrug resistance among clinical A. baumannii strains in Iran (10, 23). The resistance to ciprofloxacin and high level of resistance to amikacin observed in this study is consistent with the growing resistance to fluoroquinolones and aminoglycosides in Iranian hospitals in recent years. Polymyxin susceptibility was not included in the final analysis because current Clinical and Laboratory Standards Institute recommendations require broth microdilution rather than disk diffusion testing for accurate interpretation of Colistin and Polymyxin B susceptibility. Because polymyxin susceptibility testing was not performed, no conclusions can be drawn regarding the activity of colistin or polymyxin B in these isolates. Future studies should incorporate MIC-based testing for polymyxins to provide more reliable susceptibility data. This serves as a reminder of the continued need for the use of polymyxins as therapeutic agents in MDR and XDR A. baumannii infection (24, 25). Yet, plasmid mediated colistin resistance (mcr genes) is emerging worldwide and resistance is being reported in some areas, requiring continued surveillance and very limited use of these critical antibiotics (26-28). The high degree of co-resistance found in this study suggests that resistance to one class of antibiotics could arise from the other class due to the selective

pressure of excessive use of one of the two classes (aminoglycosides or fluoroquinolones). This is significant for AMS programs. Intra-hospital transmission of these resistant strains remains an important area of infection prevention and control, with contact precautions and extensive environmental cleaning remaining important measures to reduce transmission. It is important to note that the mechanistic explanations proposed in this study (overexpression of AdeABC/AdeIJK efflux pumps, presence of AMEs, and QRDR mutations) are inferred from phenotypic patterns and existing literature. Future genomic and transcriptomic studies are required to confirm these mechanisms in the local isolates.

5. Conclusion

This preliminary study identified frequent phenotypic co-occurrence of aminoglycoside and fluoroquinolone resistance among six clinical *Acinetobacter baumannii* isolates recovered from a tertiary hospital in Shahroud, Iran. All isolates were classified as multidrug-resistant, and two isolates met the criteria for extensively drug-resistant phenotypes. Given the very small sample size and the absence of molecular investigations, no conclusions regarding underlying resistance mechanisms or true cross-resistance can be drawn. Larger multicenter studies incorporating genomic analyses are required to validate these findings and further characterize resistance patterns in regional *A. baumannii* populations.

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Ethical Considerations and Compliance with Ethical Guidelines

The Ethics Committee of Damghan University approved this study with the Approval Code: IR.DU.REC.1403.016. Since the research was based on bacterial isolates and not patient identifiable information, informed consent was not required.

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Conflict of interest

The authors declare no conflict of interest.

AI Using Declaration

In the preparation of this manuscript, the authors used ChatGPT (OpenAI) strictly as a language-polishing tool to improve grammar, sentence structure, and readability. The core research, ideas, data, analysis, and scientific reasoning are entirely the authors' original work. The AI was not used to generate any part of the research content, conclusions, or data interpretations. The authors take full responsibility for the integrity and originality of the scholarly content presented herein. This manuscript was conceived, researched, analyzed, and drafted by the human authors. AI assistance was limited to post-drafting language enhancement, and all AI-influenced sections have been carefully reviewed, revised, and finalized by the authors to ensure alignment with their intended meaning and academic standards.

Author's contributions

All authors equally contributed to preparing this article.

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