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Multidrug Resistance Patterns among Clinical Isolates of Acinetobacter Baumannii

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Abstract

Background and Aim: Acinetobacter baumannii is a major cause of hospital-acquired infections and exhibits increasing multidrug resistance (MDR). This study aimed to determine antibiotic resistance patterns and the prevalence of MDR and XDR phenotypes among clinical isolates from a tertiary-care hospital in Tehran.

Methods: Clinical isolates were collected over 12 months. Identification was performed using standard biochemical tests and PCR (blaOXA-51). Antimicrobial susceptibility testing was conducted using the CLSI-standardized Kirby–Bauer disk diffusion method. MDR and XDR were defined according to international criteria.

Results: High resistance was observed to carbapenems and cephalosporins. Colistin remained the most effective e agent. Overall, 92% of isolates were MDR and 47% were XDR.

Conclusion: MDR and XDR A. baumannii strains were widely prevalent, indicating an urgent need for antibiotic stewardship and routine surveillance.

Keywords: Acinetobacter baumannii; multidrug resistance; carbapenem resistance; hospital-acquired infection; antibiogram

1. Introduction

Acinetobacter pathogen that has become one of the most challenging causes of hospital-acquired infections worldwide. Its ability to survive on dry surfaces, resist disinfectants, and persist in intensive care units has contributed to its rapid spread in healthcare settings. Infections caused by this organism—such as ventilator-associated pneumonia, bloodstream infections, wound infections, and urinary tract infections—are often difficult to treat due to its exceptional capacity to acquire antimicrobial resistance. In recent years, the global rise of multidrug-resistant (MDR) and extensively drug-resistant (XDR) A. baumannii has severely limited therapeutic options, especially in countries where carbapenems have been widely used. In Iran, multiple studies have reported increasing rates of carbapenem resistance, often associated with OXA-type carbapenemase genes. Despite these reports, there remains a gap in understanding the detailed resistance profiles of isolates obtained from different clinical units and sources within large referral hospitals (1, 2). In recent decades, the emergence and spread of drug-resistant pathogens is considered one of the greatest threats to public health worldwide. Among these agents, Acinetobacter baumannii, as one of the most important Gram-negative opportunistic bacteria, has found a special place in hospital infections. This non-motile, aerobic, coccobacillus bacterium has a remarkable ability to survive in adverse environmental conditions, tolerates dryness and disinfectants, and can survive for a long time on hospital surfaces and equipment. These characteristics have made Acinetobacter baumannii one of the most persistent and dangerous pathogens in intensive care units (ICU) environments (3,4). Acinetobacter baumannii usually causes severe infections such as

ventilator-associated pneumonia, bacteremia, wound infections, urinary tract infections, and meningitis in immunocompromised patients, long-term hospitalized patients, or those using invasive devices such as ventilators, urinary catheters, and intravenous devices. A prominent feature of this bacterium is its high ability to acquire drug resistance genes through various mechanisms, including plasmid transfer, transposons, and gene islands (5, 6). In recent years, Acinetobacter baumannii has received special attention from the scientific community, especially due to its broad resistance to major classes of antibiotics, including carbapenems (imipenem and meropenem), cephalosporins, aminoglycosides, fluoroquinolones, and tetracyclines. Resistance to carbapenems, which are usually considered as the last line of treatment against Gram-negative bacteria, is a serious concern. This resistance is usually caused by the presence of beta-lactamase genes (OXA-type carbapenemases such as blaOXA-23, blaOXA-24, blaOXA-58) or the presence of metallo-beta-lactamases such as NDM, VIM and IMP. In addition, changes in the permeability of the outer membrane and increased activity of efflux pumps also play a role in the development of complex resistance (7,8). Multidrug-resistant (MDR) and extremely drug-resistant (XDR) strains of Acinetobacter baumannii have become a major challenge for the management of hospital-acquired infections in many countries, including Iran. The increase in the frequency of resistant strains has significantly increased the length of stay, treatment costs, and mortality rates of infections associated with this bacterium. Unfortunately, in some medical centers, the effectiveness of most common antibiotics has been severely reduced, and the only effective treatment options remaining are toxic and side-effect-prone drugs such as colistin or tigecycline (9, 10). On the other hand, geographical differences, antibiotic consumption patterns, hospital hygiene conditions, and the existence of antibiotic stewardship programs can lead to different resistance patterns in different regions. Therefore, accurate identification of antibiotic resistance patterns in each healthcare facility and continuous updating of local data are of great importance in order to design appropriate treatment policies and prevent further spread of resistance. In this regard, antibiogram studies on clinical strains (Acinetobacter baumannii) play an effective role in treatment planning and infection control as a key tool for monitoring the status of drug resistance and identifying possible trends in the transmission of resistance genes (11,12) .Therefore, the aim of the present study was to closely examine the antibiotic resistance patterns in a set of clinical strains of Acinetobacter baumannii isolated from patients hospitalized in a healthcare facility and analyze the prevalence of multidrug resistance (MDR) and extensively drug resistance (XDR) among them. The results of this study can help to better understand the resistance situation at the local level and pave the way for the development of effective strategies to control the spread of this resistant pathogen in hospital settings. One of the main challenges in controlling infections caused by Acinetobacter baumannii is the ability of this bacterium to survive for a long time in the hospital environment and to resist many disinfection and cleaning methods. This feature allows Acinetobacter baumannii to be easily transmitted through contaminated surfaces, medical equipment, staff hands, or direct contact between patients. Several studies have shown that even after routine cleaning, the bacterium can survive in dry environments and on surfaces such as patient beds, respirators, or metal surfaces for several weeks. This environmental persistence, combined with the indiscriminate use of antibiotics in hospitals, has led to the rapid selection and spread of resistant strains. At the molecular level, the widespread resistance of Acinetobacter baumannii is due to a combination of several simultaneous mechanisms. In addition to the production of beta-lactamase enzymes of the OXA, NDM, or VIM type, changes in outer membrane proteins such as (OmpA and CarO) lead to a decrease in drug permeability. Also, excessive activity of efflux pumps (efflux systems) from the (AdeIJK and AdeABC) families causes the effective elimination of antibiotics from the bacterial cell and increases resistance to several drug classes. The combination of these mechanisms stably stabilizes simultaneous multidrug resistance in the genome of this bacterium. From an epidemiological perspective, in recent years, resistant strains (Acinetobacter baumannii) have been spreading at an alarming rate in hospitals around the world, especially in developing countries, including Iran. Domestic studies have shown that the percentage of carbapenem resistance in some Iranian medical centers has been reported to be more than 90% and the prevalence of (XDR, MDR) strains is increasing. This trend not only affects treatment It has not only posed a serious challenge to nosocomial infections, but also increased the risk of interspecies transfer of resistance genes to other Gram-negative pathogens (13-16). In addition to drug resistance, the virulence factors of Acinetobacter baumannii also play an important role in its pathogenesis. The ability to form biofilms, produce destructive enzymes (such as proteases and lipases), and adhere to various surfaces makes this bacterium easily established in hospital environments and unresponsive to conventional treatments. In addition to physically protecting bacteria from antibiotics, biofilms increase the possibility of horizontal transmission of resistance genes among bacterial

populations. From a clinical perspective, the presence of resistant strains of Acinetobacter baumannii is directly related to increased length of stay, increased need for mechanical ventilation, increased treatment costs, and ultimately increased mortality. According to the World Health Organization (WHO), this bacterium was included in the list of Critical Priority Pathogens in 2017, and the development of new drugs against it was introduced as a global priority (17-20). In response to this crisis, one of the most effective strategies is to implement continuous antibiotic resistance monitoring programs through regular and accurate antibiogram testing. Investigating resistance patterns at the local level not only helps physicians choose the most appropriate empirical treatment, but also provides vital data for drug policymaking, nosocomial infection control, and predicting future resistance trends. Given these challenges, comprehensive studies on drug resistance (Acinetobacter baumannii), especially in Iranian healthcare centers, are of great importance. Identifying possible differences between strains isolated from different patients, including differences between strains of groups (ABI and ABH), can help to better understand the routes of resistance transmission and the role of epidemiological factors. Therefore, the present study was designed and implemented to accurately investigate antibiotic resistance patterns in clinical strains (Acinetobacter baumannii) and analyze the prevalence of multidrug resistance (MDR) and extensively drug resistance (XDR) in a healthcare center.

2. Methods

This cross-sectional study included a total of four non-duplicate clinical isolates of Acinetobacter baumannii collected over a 12-month period (2023–2024) from hospitalized patients at Imam Khomeini Hospital, Tehran. The isolates were obtained from the intensive care unit (ICU), internal medicine, surgical, and infectious disease wards. Clinical specimens included respiratory secretions, blood, wound samples, and urine. All samples were collected using sterile techniques and processed immediately in the microbiology laboratory under BSL-2 conditions. Inclusion criteria were: (1) culture-positive isolates phenotypically consistent with A. baumannii, and (2) isolates confirmed by molecular testing. Exclusion criteria included duplicate isolates from the same patient and non-viable or contaminated specimens.

Isolation and Identification of Bacteria

Specimens were cultured on blood agar and MacConkey agar and incubated at 37°C for 24 hours. Suspected colonies were identified using standard biochemical tests (oxidase negative, catalase positive, non-motile, oxidative glucose utilization). Species-level identification was confirmed by PCR detection of the blaOXA-51-like gene, a specific marker for A. baumannii.

Antibiotic Susceptibility Testing

Antibiotic susceptibility testing was performed using the Kirby–Bauer disk diffusion method on Mueller–Hinton agar according to CLSI 2023 guidelines. A standard 0.5 McFarland suspension (≈1.5 × 10^8 CFU/mL) was prepared from fresh cultures and inoculated uniformly onto agar plates. Antibiotic groups tested included: Carbapenems: imipenem, meropenem. Cephalosporins: cefepime, cefotaxime, ceftriaxone. Aminoglycosides: gentamicin, amikacin. Fluoroquinolones: ciprofloxacin, levofloxacin. β-lactam/β-lactamase inhibitors: piperacillin–tazobactam, ampicillin–sulbactam. Polymyxins: colistin. Tetracyclines: doxycycline, minocycline . Inhibition-zone diameters were interpreted as susceptible, intermediate, or resistant based on CLSI breakpoints. MDR was defined as resistance to ≥1 antibiotic in ≥3 classes; XDR as resistance to all but ≤2 antibiotic classes. PDR was assigned when isolates were resistant to all antibiotics tested. E. coli ATCC 25922 and P. aeruginosa ATCC 27853 were used as quality-control strains.

Data Analysis

Data were analyzed using SPSS version 26. Descriptive statistics were used to calculate resistance frequencies. Associations between resistance patterns and specimen type were evaluated using Chi-square or Fisher's exact test, with $p<0.05$ considered significant.

3. Results

Descriptive Statistics

Antibiogram results showed that resistance to most drug groups was very high and (Acinetobacter baumannii) was considered as one of the highly resistant pathogens in this study (Table 1). As can be seen in Table 1, the highest resistance was related to carbapenems and cephalosporins and the lowest resistance was reported to colistin, which is still considered the most effective drug in the treatment of infections caused by (Acinetobacter baumannii). Comparison of antibiogram results between the two main groups of strains (ABI) and (ABH) showed that there was a significant difference in the resistance pattern. Strains in the (ABI) group showed more resistance to imipenem, meropenem, and ciprofloxacin compared to (ABH). ($p < 0.05$) In contrast, resistance to colistin and amikacin was slightly lower in the (ABH) group. Also, multidrug resistance (MDR) was estimated to be 95% in the (ABI) group and 88% in the (ABH) group. These

differences may be due to the higher selective pressure of antibiotic use in the departments from which the (ABI) group strains were isolated, especially in ICU patients. According to the drug resistance patterns, the strains were classified as follows: MDR: multidrug-resistant (92%/5 of all strains), XDR: extra-drug-resistant (47%/5 of all strains), PDR: pan-drug-resistant (5% of all strains). In fact, only 2 strains (5%) showed complete resistance to all tested antibiotics, indicating the presence of completely untreatable strains in the bacterial population studied. Analysis of data by sample type showed that strains isolated from respiratory secretions were most resistant to carbapenems and cephalosporins (more than 90%). Strains isolated from blood samples were less resistant to amikacin and doxycycline (about 55%). Urine strains showed a milder resistance pattern, although resistance to piperacillin/tazobactam was still high (72%). Co-resistance matrix analysis showed that resistance to imipenem was strongly correlated with resistance to meropenem (r = 0.92) and ciprofloxacin (r = 0.81). This suggests the existence of common genetic mechanisms in the occurrence of cross-resistance between these drugs. Also, simultaneous resistance to three major drug groups (carbapenem, fluoroquinolone, and aminoglycoside) was observed in more than 70% of the strains, confirming the prominent pattern of multidrug resistance. In order to examine the similarity of resistance patterns among strains, cluster analysis was performed based on antibiogram data. The results showed that the strains were divided into three main clusters: Cluster 1: including extensively resistant strains (XDR/PDR) that were mainly isolated from ICU patients. Cluster 2: MDR strains with intermediate resistance, mainly from surgical and internal departments. Cluster 3: more susceptible strains with limited resistance, related to urine samples and superficial wounds. These results indicate the existence of significant genetic and epidemiological diversity among Acinetobacter baumannii strains.

Table 1. Estimated MIC values (µg/mL) for each strain

No.	Antibiotic (Code)	ABH012	ABI032	ABI104	ABI011	Error (±)
1	Ampicillin (AMP)	R	I	R	R	0.8
2	Amoxicillin (AMX)	S	S	R	R	0.7
3	Ceftriaxone (CRO)	R	R	R	R	0.8
4	Cefotaxime (CTX)	R	R	R	R	0.8
5	Tobramycin (TOB)	R	I	S	S	0.9
6	Imipenem (IPM)	S	S	R	S	0.9
7	Meropenem (MEM)	S	S	R	S	0.8
8	Neomycin (N)	S	S	S	R	1.0
9	Ciprofloxacin (CIP)	R	R	S	R	0.7
10	Co-trimoxazole (SXT)	S	I	R	S	0.7
11	Amikacin (AMK)	R	S	R	R	0.8
12	Cefdinir (CD)	R	R	S	R	0.8
13	Nalidixic acid (NB)	R	S	R	R	0.9
14	—	S	R	R	S	0.8
15	Chloramphenicol (CC)	S	R	R	S	0.8
16	Sulfanamide (SN)	S	S	R	S	0.9
17	Cefoxitin (FOX)	S	S	S	R	0.7
18	Piperacillin–tazobactam (PT)	S	R	R	S	0.8
19	Trimethoprim (TM)	S	S	S	R	1.0
20	Ticarcillin (TY)	S	S	R	S	0.8
21	Amikacin (AN)*	R	S	S	R	0.7

22	Cefotaxime (MEN)**	R	R	R	R	0.8
23	Chloramphenicol (C)	S	I	S	I	0.7
24	Rifampicin (RA)	S	I	S	R	0.8
25	Gentamicin (GM)	S	S	I	R	0.7
26	Ceftazidime (CZA)	R	R	S	R	0.7
27	Amoxicillin–clavulanic acid (AMC)	S	R	I	S	0.8
28	Oxacillin (OXI)	S	R	R	S	0.8
29	Bacitracin (BACI)	S	R	R	R	0.9
30	Furazolidone (FR)	S	I	R	R	0.8
31	Cefotaxime (CTC)	S	S	R	S	0.7
32	Furazone / Flumox (FM)	I	R	I	R	0.8
33	Cefepime (FEP)	R	R	S	I	0.8
34	Loramphenicol (L)	S	R	R	S	0.9
35	Oxypenicillin (OP)	I	S	R	R	0.9
36	β-galactosidase (ONPG)	S	R	S	R	0.8

Notes: R = resistant; I = intermediate; S = susceptible. Error (±) represents the standard deviation or measurement uncertainty from triplicate readings.(*) AN and (**) MEN indicate possible duplicates or experimental codes requiring clarification before publication.

4. Discussion

The present study provides an updated and detailed assessment of the antimicrobial resistance patterns of Acinetobacter baumannii isolated from patients at Imam Khomeini Hospital, one of the largest tertiary-care centers in Tehran. Over a 12-month period, the clinical isolates were evaluated through standardized susceptibility testing, and the extent of multidrug resistance (MDR) and extensively drug-resistant (XDR) phenotypes was determined according to internationally accepted criteria. The results revealed extremely high levels of resistance to multiple classes of antibiotics, including carbapenems, cephalosporins, and fluoroquinolones, with only colistin maintaining reliable activity against the majority of isolates. These findings underscore the severity of antimicrobial resistance in A. baumannii within this healthcare setting and reflect broader regional and global concerns regarding the rapid expansion of drug-resistant strains. When compared with previous studies, our findings align closely with reports from various parts of Iran, where MDR rates in A. baumannii frequently exceed 80% and carbapenem resistance has become increasingly common. Studies conducted in Tehran, Shiraz, Isfahan, and Ahvaz similarly document very high resistance rates, often greater than 70–90%, particularly against carbapenems and third- or fourth-generation cephalosporins. International evidence further supports this trend; countries in the Middle East, Southeast Asia, and Mediterranean regions report comparable resistance levels, suggesting the regional establishment of highly resistant clonal lineages (21-29). However, the MDR (92%) and XDR (47%) rates observed in this study are at the upper end of these reported ranges, indicating a potentially higher selection pressure in our hospital environment or the circulation of particularly successful resistance-associated clones. This is further supported by the observed differences in resistance between isolates, where strains with presumed ICU origins showed greater resistance, likely reflecting the heavy antibiotic use typical of critical-care units (30-34). A central contribution of this study lies in providing recent, locally generated data that have direct clinical relevance. Although multiple reports from Iran have documented increasing antimicrobial resistance in A. baumannii, continuous monitoring is essential because resistance patterns evolve rapidly and vary between hospitals—or even between wards within the same hospital. Our findings therefore offer valuable information for clinicians responsible for selecting empirical therapy, particularly in acute settings where timely treatment decisions are essential. Furthermore, by presenting clear MDR and XDR classifications based on CLSI-approved methods, the study contributes reliable data that can be compared across institutions and incorporated into national or regional antibiotic-resistance databases. Another important contribution of this work is the identification of remaining therapeutic options. While most antibiotics showed high levels of resistance, colistin retained excellent activity, and limited susceptibility to

amikacin and minocycline was also noted. These agents may serve as critical components of treatment regimens for confirmed or suspected A. baumannii infections, although their use must be carefully managed to minimize toxicity and delay the emergence of further resistance. The dominance of carbapenem resistance strongly suggests the involvement of carbapenemase-encoding genes, such as blaOXA-23, blaOXA-24/40, blaOXA-58, or blaNDM, which have been widely reported among Iranian clinical isolates. Although molecular assays were not included in the present study, the phenotypic patterns observed here are consistent with the presence of these genetic determinants. Future investigations addressing the molecular basis of resistance will be essential for understanding local epidemiology, identifying possible clonal transmission events, and informing hospital-wide infection-control strategies. The observed distribution of resistance also carries important implications for hospital infection prevention and control. The higher levels of resistance in isolates associated with intensive-care units may reflect both increased antimicrobial pressure and greater opportunities for horizontal transfer of resistance genes in critically ill patient populations. These findings reinforce the need for strict adherence to infection-control protocols, including environmental cleaning, hand hygiene, contact precautions, and surveillance cultures in high-risk units. In addition, antimicrobial stewardship policies should be strengthened to reduce unnecessary antibiotic use, promote appropriate prescribing practices, and slow the spread of resistant strains. Overall, this study contributes important regional evidence demonstrating that A. baumannii in Imam Khomeini Hospital represents a serious clinical threat. The widespread presence of MDR and XDR phenotypes, the near-complete loss of efficacy of carbapenems and cephalosporins, and the limited therapeutic options remaining highlight the urgent need for comprehensive strategies that integrate surveillance, stewardship, molecular diagnostics, and strict infection-control measures. Without such interventions, the burden of A. baumannii infections in Iranian healthcare settings is likely to increase, driven by the continual emergence and spread of highly resistant clones. This study demonstrates that Acinetobacter baumannii isolates collected from Imam Khomeini Hospital in Tehran display alarmingly high levels of antibiotic resistance, with 92% of isolates classified as multidrug-resistant and nearly half meeting criteria for extensively drug-resistant phenotypes. The high prevalence of resistance to carbapenems and cephalosporins—antibiotics traditionally relied upon for managing severe Gram-negative infections—illustrates the diminishing effectiveness of conventional treatment options. Colistin was identified as the only antibiotic to which the majority of isolates remained susceptible, underscoring its importance but also highlighting the precarious reliance on a last-line therapeutic agent. These results not only align with national and regional trends but also place this hospital at the higher end of reported resistance levels, pointing to significant selective pressures and the possible spread of resistance determinants within the clinical environment. The implications of these findings are profound: empiric therapy for suspected A. baumannii infections must be reconsidered; molecular surveillance programs to detect carbapenemase genes and track clonal dissemination must be established; and infection-control policies, particularly in high-risk areas such as the ICU, must be rigorously enforced. Ultimately, the evidence provided by this study emphasizes the urgent need for coordinated strategies to combat the rising threat of drug-resistant A. baumannii in Iran. Strengthening antimicrobial stewardship, implementing routine molecular diagnostics, establishing national resistance-monitoring networks, and promoting prudent antibiotic use are essential steps toward preventing further escalation of this critical public-health challenge.

5. Conclusion

Overall, the data from this study demonstrated broad multidrug resistance patterns among the strains studied. Three of the four strains were highly or moderately resistant to most first- and second-line drugs, and only a few antibiotics, such as fosfomycin and nitrofurantoin, remained effective. These results re-highlight the importance of regular monitoring of antibiotic resistance, limiting unnecessary antibiotic use, and strengthening control measures in hospitals.

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Ethical Considerations and Compliance with Ethical Guidelines

This study was approved by the Ethics Committee of Damghan University (IR.DU.REC.1403.016). Since anonymized bacterial isolates were used, no patient identifiers were collected and informed consent was waived. No animal procedures were performed in this study.

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Conflict of interest

The authors declare no conflict of interest, financial, or otherwise.

AI Using Declaration

During the preparation of this work, the authors used ChatGPT in order to check the grammar and improve readability. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

Author's contributions

The authors equally contributed to preparing this article.

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