

Incidence of drug resistance and expression of *bla*_{OXA-51} and *adeA* genes among *Acinetobacter baumannii* strains isolated from hospitalized patients at a government hospital in Irbid, Jordan

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ABSTRACT

Background and Objectives: *Acinetobacter baumannii* (*A. baumannii*) is an opportunistic bacterial pathogen principally related with hospital-acquired infections. This study aimed to isolate and identify *A. baumannii* strains, investigate their resistance to various antibiotics, and characterize *A. baumannii* at the molecular level.

Materials and Methods: A total of 100 samples were obtained from various hospital departments, including the intensive care unit (ICU), emergency room, kidney dialysis and surgery units. The incidence of drug resistance was studied using the Vitek 2 Compact system and further using molecular techniques such as polymerase chain reaction to analyze the genes responsible for resistance.

Results: The study exhibited a high prevalence of multidrug-resistant (MDR) *A. baumannii* isolates, especially in ICU patients. The males were the predominant group, accounting for 60% whereas females were 40%. The most frequent samples were from urine (43%) and skin (24%). Majority of samples were from the ICU (42%) and emergency departments (20%). The tested isolates exhibited the highest resistance (66%) to oxacillin, whereas the maximum sensitivity (52%) was recorded for Erythromycin. Molecular analysis revealed the occurrence of resistance genes *bla*_{OXA-23}, *bla*_{OXA-24}, *bla*_{OXA-51}, and *bla*_{OXA-58}, which contribute to carbapenem resistance.

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Conclusion: The findings emphasize that *A. baumannii* remains a formidable nosocomial pathogen, and there is pressing requirement for enhanced infection control procedures and antibiotic stewardship. Through improved molecular observation, judicious use of antibiotics and improved infection control practices, healthcare providers can alleviate the impact of MDR *A. baumannii* infections and improve the prognosis for affected patients in Jordan and beyond.

Keywords: *Acinetobacter baumannii*; Electrophoresis; Agar gel; Carbapenemase; Drug resistance; Efflux pump; Erythromycin; Oxacillin

INTRODUCTION

Acinetobacter baumannii (*A. baumannii*) is a Gram-negative bacterium that is commonly associated with hospital-acquired infections, especially those in intensive care units (ICUs). This microorganism is recognized for its ability to develop multiple-drug resistance (MDR), which poses a significant challenge for healthcare practitioners (1). The prevalence of *A. baumannii* in ICU settings has risen significantly, contributing to higher morbidity and mortality rates among critically ill patients (2). "Governmental hospitals in Jordan have observed a rise in infections caused by *A. baumannii*. Considering the severity of cases in ICUs and the pathogen's robust resistance mechanisms, the epidemiological investigation and antibiotic resistance profiles of *A. baumannii* and its molecular characterization are utmost needed for implementing effective strategies for disease management (3)."

A. baumannii is a highly aerobic, nonfermenting coccobacillus belonging to the Moraxellaceae family, initially identified in 1911 as *Micrococcus calcoaceticus* (4). *Acinetobacter*, a genus of opportunistic pathogens, is becoming more common in both community-acquired and nosocomial infections, especially among patients in ICUs and high-dependency units (5). *A. baumannii* has garnered significant attention due to its capability to persist in hospital environments and acquire resistance to multiple classes of antibiotics (4). Its ability to form biofilms on medical devices, coupled with inherent defense mechanisms and the propensity for horizontal gene transfer, establishes it as a formidable pathogen responsible for various infections (6). *A. baumannii* could be a causative agent for ventilator-associated pneumonia, skin and severe bloodstream infections, infections related to prosthetic devices, and pneumonia, especially in immunocompromised and critically ill patients (6, 7).

The increasing prevalence of multidrug-resistant bacteria in healthcare settings poses a major global

challenge. These bacteria render standard antibiotics ineffective, underscoring the urgent need for continuous monitoring and advanced diagnostic solutions. In Jordanian governmental hospitals, where healthcare complexities are compounded by resource limitations, analyzing the molecular characteristics and antibiotic susceptibilities of *A. baumannii* is crucial for effective clinical management and infection control strategies. These techniques provide insights into genetic variation, distribution patterns, and potential treatment strategies in combating infection (8). These efforts are crucial for guiding antibiotic therapy, informing infection control strategies, enhancing patient outcomes, and preventing the dissemination of resistant strains within healthcare environments."

Current study aims to address knowledge gaps through a systematic analysis to determine the prevalence of *A. baumannii* isolates obtained from hospitalized patients in Jordanian government hospital and to evaluate their antibiotic susceptibilities and further, to identify the genetic factors contributing to antibiotic resistance in *A. baumannii* strains.

MATERIALS AND METHODS

Sample collection and ethical considerations.

From July to November 2024, one hundred samples were collected from various departments at Irbid Islamic Hospital, including the emergency room, ICU, kidney dialysis and surgery units. The hospital granted official permission to collect samples, isolates, and data.

Samples were obtained from both inpatients and outpatients undergoing pathological investigation for any clinical situation, with positive *A. baumannii* isolates identified. The criteria for exclusion and inclusion were based on demographic data including nationality, age, gender, and source of the sample. This study was approved by the research ethical committee at Zarqa University and the Ministry of Health; ethical approval number; (MOH/REC/2024/382) be-

fore collecting samples.

The isolates were sub-cultured on MacConkey agar plates (Mast Group, UK). The plates were incubated at $35^{\circ}\text{C} \pm 2^{\circ}\text{C}$ under ambient air for 20-24 hours. A single un-colored colony from each isolate was chosen for purification. The pure culture of each isolate was preserved in 20% glycerol (Cryobank, Mast Group, UK) and frozen at -80°C for further investigation.

Identification of *A. baumannii*. According to the general characteristics of *A. baumannii*, an oxidase test was performed on the pure culture growth of the isolates. The isolates that showed negative oxidase results were confirmed using the automated instrument Vitek 2 Compact (BioMérieux, France) with GN ID Cards (BioMérieux, France) in compliance with the WHO's recommendation for bacterial phenotypic detection and identification.

Antimicrobial susceptibility test. The susceptibility testing of *A. baumannii* isolates to various antibiotics was determined using the Vitek 2 Compact instrument (BioMérieux, France), with cards AST-GN222 and AST-XN05 (BioMérieux, France). These cards are used for *A. baumannii* by following the recommendation of the Clinical Laboratory Standards Institute (CLSI 2024) guidelines (9). The breakpoints for MIC of antibiotics used for *A. baumannii* isolates were followed as mentioned in the guidelines of CLSI 2024.

DNA extraction. Genomic DNA of *A. baumannii* isolates was extracted using the Quick-DNA Mini-prep Plus Kit (Zymo Research, USA) according to manufacturer's quick protocol instructions.

Detection of multi drug resistance (MDR) genes. Conventional PCR was executed using a programmable thermocycler (S1000 thermal cycler BIO-RAD, USA) for detection of OXA genes encoding *bla*_{OXA-23}, *bla*_{OXA-24}, and *bla*_{OXA-51}, and for efflux genes encoding AdeA, AdeB, AdeC, and AdeS. PCR was carried out in 25 μL reaction volumes with 10 μL of master mix (GoTaq Green Master Mix Promega, USA), 2 μL each of diluted forward and backward primers (Genewiz, USA), and 2 μL of extracted genomic DNA of *A. baumannii* isolates and then nuclease-free water (9 μL) was added to reach the final volume. The primers used in this survey were according to references mentioned in Table 1. The annealing temperatures for each primer pair and amplicon size are included in

Table 1. The amplification condition of polymerase chain reaction was as follows: pre-denaturation temperature at 95°C for 5 min, 30 cycles at 95°C for 30 sec, followed by annealing at 53°C for 35 sec, extension at 72°C for 5 min, and final extension cycle at 72°C for 5 minutes. In each run, tubes containing master mix without template DNA were considered as negative control. PCR products were separated on 1% Agarose gel. The eluted DNA was stored at -20°C (10).

A. baumannii NCTC 13301 was used as a positive control for OXA carbapenemase genes, and *Klebsiella pneumonia* NCTC 13443 was used as a positive control for Metallo- β -lactamase (NDM-1). The cultures were obtained from the National Collection of Type Cultures (NCTC) (11).

Agarose gel electrophoresis. Using 1% agarose gel electrophoresis containing 15% RedSafe stain, the amplified products and the PCR DNA marker were separated for 40-50 minutes at 120 volts and then visualized using a gel documentation system including UV camera, screen, and printer (Alpha Innotech, USA) as shown in Table 1.

Statistical analysis. Statistical study analysis was made using Statistical Package for Social Sciences (SPSS) software, version 21, and a p-value less than 0.05 was considered statistically significant for the analysis of variance (ANOVA). The correlations between variables in this study were examined using Pearson's correlation coefficient to evaluate the strength and direction of the linear relationships. The dependent variables are sample type and patient diagnosis, whereas independent variables are gender and age. A significance level of $p\text{-value} \leq 0.05$ was considered statistically significant.

RESULTS

Demographic data. Fig. 1 shows the distribution of *A. baumannii* samples according to sex. Males were the predominant group, accounting for 60 (60%), while females were 40 (40%). The participants' ages ranged from 7 to 95, with an average age of about 39.7. The median age was 44, and the most common (mode) age was 45, as illustrated in Fig. 1. This wide age range can help to analyze how different age groups respond to treatments or conditions.

Table 1. Primers used with their respective amplicon size, validation, specificity and sources

Genes	Primers sequences	Annealing Temperature °C for 30 sec	Amplicon Size	References
<i>bla</i> _{OXA-51}	F: TAATGCTTTGATCGGCCTTG R: TGGATTGCACTTCATCTTGG	55	760	(3, 11)
<i>bla</i> _{OXA-23}	F: GATCGGATTGGAGAACCAGA R: ATTTCTGACCGCATTCCAT	53	774	(3, 11)
<i>bla</i> _{OXA-24}	F: TTCCCCTAACATGAATTTGT R: GTACTAATCAAAGTTGTGAA	49	828	(3, 11)
<i>adeA</i>	F: GGCGTATTGGGCAATCTTTTGT R: GTCACCGACTTTCAAGCCTTTG	61	1157	(3, 10)
<i>adeB</i>	F: TGGCGGAATGAAGTATGT R: GCAGTGCGGCAGGTTAG	49	1323	(3, 10)
<i>adeC</i>	F: GACAATCGTATCTCGTGGACTC R: AGCAATTTTCTGGTCAGTTTCC	59	1331	(3, 10)
<i>adeS</i>	F: GTGGACGTTAGGTCAAGTTCTG R: TGTTATCTTTTGCGGCTGT ATT	57	949	(3, 10)

*F= Forward Primer *R= Reverse Primer

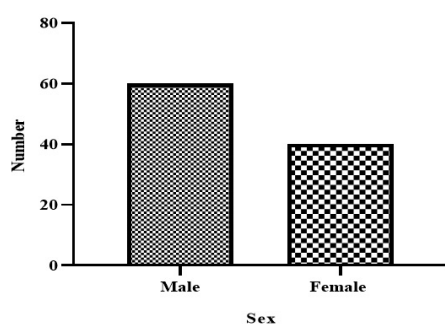


Fig. 1. Distribution of *A. baumannii* isolates according to the sex.

Samples characteristics. Fig. 2 shows that *A. baumannii* samples most frequently came from urine (43%) and skin (24%). The next most frequent sources were pus (12%), ulcers (5%), catheter (4%), nose (4%), and the cerebrospinal fluid (CSF) (2%). There was only one sample each from blood, sputum, and wounds.

Prevalence of *A. baumannii* among the Hospital departments. Fig. 3 shows that the majority of samples were collected from the ICU (42%) and emergency departments (20%). Notably, a significant proportion of samples were also collected from kidney dialysis units (15%).

Patients diagnosis. Fig. 4 shows that the most common diagnoses related with *A. baumannii* among par-

ticipants were meningitis (20%), pneumonia (20%), and bronchitis (18%). This emphasizes the prevalence of respiratory and infectious diseases. Sepsis was also notable, accounting for 12% of cases and reflecting its clinical importance.

Correlation coefficient analysis. The Pearson correlation coefficient (*r*) ranges from -1 to +1. Values closer to +1 or -1 indicate a stronger relationship.

Gender and sample type. A statistically significant (*p* = 0.04) Pearson correlation coefficient of 0.206 was identified between gender and sample type. Although the correlation is weak, it suggests slight variations in sample type based on gender, as shown in Table 2.

Age and diagnosis. The Pearson correlation between age and diagnosis is not statistically significant (*r* = 0.150, *p* = 0.136). This indicates that there is no strong relationship between participants' ages and their diagnoses. This suggests that diagnosis may be relatively independent of age in this dataset, as shown in Table 3.

Antimicrobial susceptibility testing for *A. baumannii*. Table 4 shows that seven antibiotics were tested against *A. baumannii*, including oxacillin, tigecycline, erythromycin, amikacin, gentamycin, rifampin, and levofloxacin. These antibiotics were tested for resistance (R), sensitivity (S), and intermediate

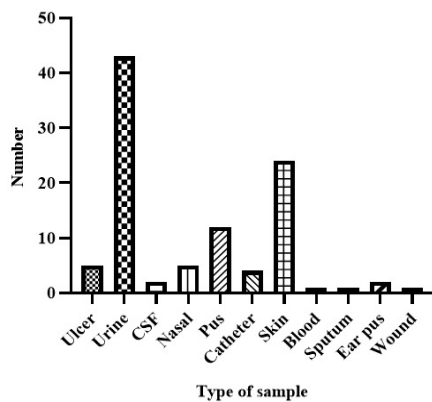


Fig. 2. Distribution of *A. baumannii* isolates according to the type of samples.

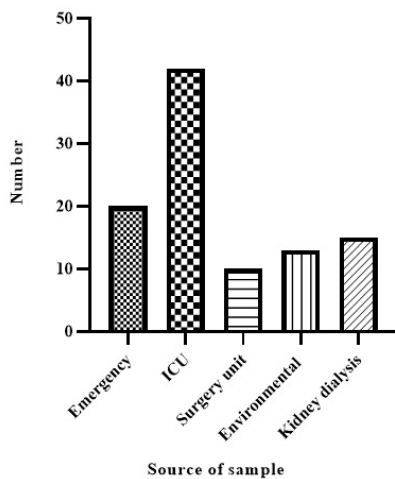


Fig. 3. Distribution of *A. baumannii* according to the source of samples.

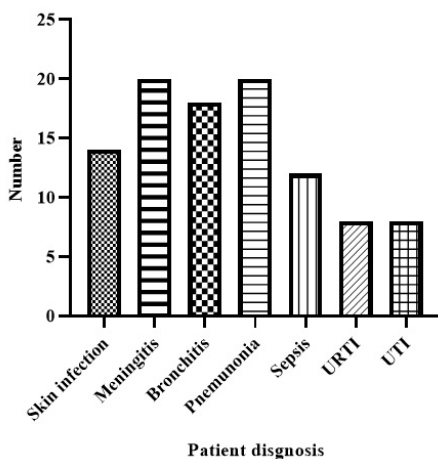


Fig. 4. Distribution of *A. baumannii* according to the patient diagnosis.

Table 2. Correlation between patient's gender and sample type

		Gender	Sample Type
Gender	Pearson Correlation	1	.206*
	Sig. (2-tailed)		.040
	N	100	100
Sample Type	Pearson Correlation	.206*	1
	Sig. (2-tailed)	.040	
	N	100	100

*Correlation is significant at the 0.05 level (2-tailed).

Table 3. Correlation between patient's age and diagnosis.

		Age	Diagnosis
Age	Pearson Correlation	1	.150
	Sig. (2-tailed)		.136
	N	100	100
Diagnosis	Pearson Correlation	.150	1
	Sig. (2-tailed)	.136	
	N	100	100

resistance (I) against each isolate. Oxacillin showed the highest rate of resistance (66%), while the highest sensitivity rate was for erythromycin (52%).

Detection of Oxa and efflux genes among the isolates. PCR was used to amplify and identify specific antibiotic resistance genes within the *A. baumannii* isolates. Conventional PCR was used to detect carbapenem resistance, a vital component of MDR *A. baumannii*, by targeting genes including *bla*_{OXA-23}, *bla*_{OXA-24}, and *bla*_{OXA-51}, as well as efflux genes *adeA*, *adeB*, *adeC*, and *adeS*, as shown in Table 5. The PCR amplification adhered to a standardized technique, with meticulously regulated conditions, including particular annealing temperatures for each primer pair, facilitating precise amplification of the targeted resistance genes. The examination of PCR results by agarose gel electrophoresis confirmed the presence of these genes. This has suggested a high prevalence of carbapenemase-producing isolates, as shown in Figs. 5a and b. This has provided significant understanding into the genetic basis of resistance, emphasizing the importance of molecular monitoring in combating MDR diseases in healthcare settings.

Table 4. Antimicrobial susceptibility profile for *A. baumannii*

Antibiotic	Sensitivity	Frequency	Percentage	Valid Percentage	Cumulative Percentage
Oxacillin	R	66	66.0	66.0	66.0
	S	34	34.0	34.0	100.0
	I	0	0.0	0.0	
Tigecycline	R	52	52.0	52.0	52.0
	S	39	39.0	39.0	91.0
	I	9	9.0	9.0	100.0
Erythromycin	R	48	48.0	48.0	48.0
	S	52	52.0	52.0	100.0
	I	0	0.0	0.0	
Amikacin	R	47	47.0	47.0	47.0
	S	49	49.0	49.0	96.0
	I	4	4.0	4.0	100.0
Gentamycin	R	50	50.0	50.0	50.0
	S	47	47.0	47.0	97.0
	I	3	3.0	3.0	100.0
Rifampin	R	53	53.0	53.0	53.0
	S	47	47.0	47.0	100.0
	I	0	0.0	0.0	
Levofloxacin	R	67	67.0	67.0	67.0
	S	33	33.0	33.0	100.0
	I	0	0.0	0.0	

Table 5. Detection of OXA and efflux genes in *A. baumannii* isolates

Target genes	Prevalence Rate%	P-value
OXA genes		
<i>bla</i> _{OXA-51}	15%	0.06
<i>bla</i> _{OXA-23}	80%	0.005
<i>bla</i> _{OXA-24}	77%	0.02
Efflux genes		
<i>adeA</i>	5%	0.07
<i>adeB</i>	8%	0.75
<i>adeC</i>	14%	0.15
<i>adeS</i>	7%	0.75

DISCUSSION

The present study aimed to examine the occurrence of drug resistance and MDR genes among the isolates of *A. baumannii* in hospitalized patients at a government hospital in Irbid, Jordan. The findings indicated higher incidence of MDR *A. baumannii* infections, particularly in ICU patients. A majority of isolates exhibited resistance to multiple antibiotics, with the highest resistance observed for Oxacillin

(66%). The findings of this study align with research from various geographic regions, including the Arab Peninsula, demonstrating that *A. baumannii* continues to pose a significant challenge in hospital environments worldwide (12, 13). A similar study by Bertini et al. (14) in Jordan noted an alarming rise in the occurrence of *A. baumannii* in ICUs, where 90% of the isolates were multidrug-resistant. This is comparable to the increased resistance observed in our study, and emphasizes the role of ICU settings as high-risk environments due to the common use of invasive medical devices and the high patient turnover.

Internationally, studies conducted in European and Asian countries have revealed similar resistance patterns. For example, a study by Dijkshoorn et al. (15) in Europe found that *A. baumannii* showed widespread resistance to aminoglycosides, fluoroquinolones, and carbapenems, comparable to the resistance patterns observed in the current study. The widespread dissemination of carbapenem-resistant *A. baumannii* in Europe and Jordan suggests a potential link between international healthcare networks and the worldwide spreading of resistant strains caused by travel or medical tourism.

Similar to our findings, a study conducted in Greek

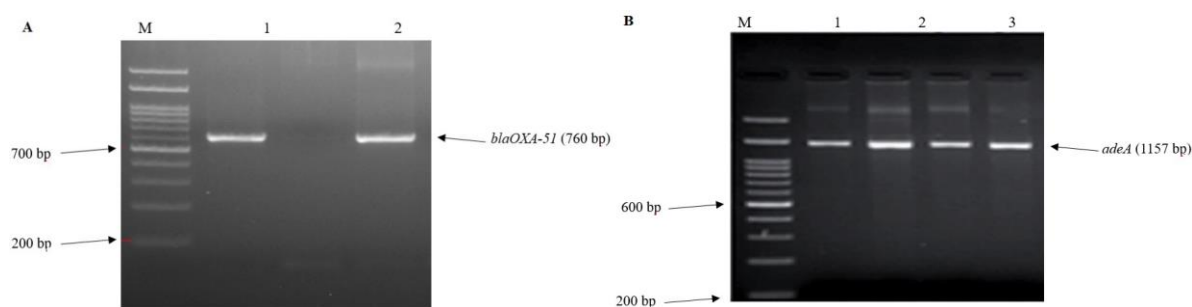


Fig. 5. (a). Detection of *bla*_{OXA-51} gene using gel electrophoresis. Agarose gel electrophoresis of PCR products targeting anti-microbial resistance genes. Amplified DNA fragments were separated on a 1.5% agarose gel stained with red safe stained and visualized under UV light. Lane M: 100 bp molecular weight DNA ladder. lanes 1 and 3 represents positive PCR amplification of resistance genes for *bla*_{OXA-51} (760 bp), and lane 2 represents the negative control. (b). Detection of *adeA* gene using gel electrophoresis. Agarose gel electrophoresis of PCR products targeting antimicrobial resistance genes. Amplified DNA fragments were separated on a 1.5% agarose gel stained with red safe stained and visualized under UV light. Lane M: 100 bp molecular weight DNA ladder. lanes 1, 2, 3 and 4 represent positive PCR amplification of resistance genes for *adeA* (1157 bp).

hospitals revealed a significant incidence of infections from *A. baumannii* among ICU patients who exhibited substantial resistance to various antibiotic classes, including carbapenems (16). Our study has highlighted emergence of *A. baumannii* as a critical nosocomial pathogen, especially in ICUs. This could be largely attributed to the immunocompromised state of patients and their frequent exposure to invasive procedures, including mechanical ventilation and catheterization (17).

To effectively address and alleviate the commonness of this pathogen, ongoing surveillance efforts and the implementation of advanced molecular diagnostic technologies are crucial. One of the key findings of the present study is the commonness of carbapenemase-producing *A. baumannii* strains, particularly those harboring *bla*_{OXA-23}, and *bla*_{OXA-24} genes. These resistance mechanisms are concerned as they limit the efficacy of carbapenem antibiotics, which are frequently used as a last option for combating severe infections (18). A study by Al-Tamimi et al. (19) investigating isolates from various Jordanian hospitals reported that the *bla*_{OXA-51} gene was recorded in all *A. baumannii* samples, highlighting the extensive distribution of this gene within Jordanian healthcare settings. These findings are consistent with our study, which also identified *bla*_{OXA-51} in *A. baumannii* isolates, suggesting that this gene is a prevalent molecular marker in the region and likely contributing to the dissemination of carbapenem-resistant *A. baumannii* (8). Presence of *bla*_{OXA} genes, such as *bla*_{OXA-51}

*bla*_{OXA-23}, and *bla*_{OXA-24} are leading cause of resistance mechanisms of *A. baumannii*. These genes encode carbapenemase enzymes and are located on both chromosomal DNA and plasmids, facilitating their widespread dissemination (16). Research by Wong et al. (20) has revealed that among the clinical isolates of *A. baumannii*, carbapenem resistance is predominantly driven by the overexpression of OXA-23 or OXA-51. This increased expression is linked to the insertion of the ISAbal element into the promoter regions of these genes, which enhances their transcriptional activity and contributes to resistance.

In addition, our study exhibited expression of Ade genes among the test isolates. It is to be mentioned here that the enhanced activity of efflux pumps plays a synergistic role alongside beta-lactamases in driving antibiotic resistance (21). The overexpression of the Ade ABC efflux pump in *A. baumannii* is strongly linked to resistance against carbapenems and cephalosporins (22). This pump is a member of the resistance-nodulation-division (RND) family and operates as a three-component system. The Ade B component functions as the primary transporter, actively expelling antibiotics from the bacterial cell. Ade A acts as a membrane fusion protein, whereas Ade C serves as an outer membrane protein (22, 23). Notably, the Ade B component has broad substrate specificity, possess ability of transporting a range of molecules including hydrophilic and hydrophobic compounds, as well as positively charged or neutral antibiotics (23). Harding et al. (24) conducted a study

in the United States highlighting that that efflux pumps were the predominant mechanism driving resistance in *A. baumannii*, with less emphasis on the role of *bla*_{OXA} genes. This finding contrasts with the

current study, which focuses on carbapenemase-producing genes such as *bla*_{OXA-23} and *bla*_{NDM-1}. The variation suggests potential geographic differences in the primary resistance mechanisms of *A. baumannii*, reflecting the influence of local selective pressures and antimicrobial usage patterns (8).

In conclusion, the high prevalence of MDR genes including *bla*_{OXA-23} among *A. baumannii* isolates obtained from Jordanian hospital, particularly in the ICU, highlights the pressing necessity for stringent infection control actions and the advancement of targeted treatment protocols to mitigate the spread of MDR infections in healthcare settings. Our study offers valued understandings into the molecular epidemiology of *A. baumannii* in a government hospital in Jordan. However, future research should expand the scope to include multiple healthcare facilities in different regions. This would provide a more comprehensive insights of the epidemiological trends and mechanisms of resistance among the isolates of *A. baumannii* in Jordan.

However, the study limitations include lack of whole-genome sequencing, need of single-center study design and absence of patient outcome data. Future studies should incorporate the whole-genome sequencing (WGS) to provide more detailed insights

into the clonal relationships and transmission dynamics of *A. baumannii* within hospital environments.

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