Original Article

Microbial Biosystems 9(2) (2024) 2024.1206
10.21608/mb.2024.337802.1206

Contents lists available at Egyptian Knowledge Bank
Microbial Biosystems
Journal homepage: http://mb.journals.ekb.eg/

MICROBIAL BIOSYSTEMS
INTERNATIONAL SCIENTIFIC JOURNAL OF MICROBIAL BIOLOGY

Genetic diversity and molecular epidemiology of carbapenem-resistant *Acinetobacter baumannii* in hospitalized patients of the Iraqi Kurdistan region: Insights from ERIC-PCR

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ARTICLE INFO

Article history
Received 11 November 2024
Received revised 23 November 2024
Accepted 22 December 2024
Available online 30 December 2024

Corresponding Editors:
Izzat, S.
Subhi, H.
Abdel-Azeem, A. M.

Keywords
ERIC-PCR,
genotyping,
human health,
multidrug-resistant,
resistance genes.

ABSTRACT

Many strains of *Acinetobacter baumannii* are multidrug-resistant (MDR) or carbapenem-resistant, which is very bad for public health. The prevalence of multidrug-resistant (MDR) and carbapenem-resistant strains of *A. baumannii* poses a serious threat to public health. Rapid identification and genotyping are crucial for controlling its spread in hospitals. There was a study that looked at the genetic diversity and molecular epidemiology of CRAB isolates from ICU patients in the Kurdistan region of Iraq. We collected a total of 283 clinical samples from hospitals. We tested susceptibility to 14 antibiotics using the VITEK 2 system. Carbapenem resistance genes (*blaOXA-51*, *blaOXA-23*, *bla*IMP, and *bla*NDM) were identified by PCR, and genetic relationships were analyzed using ERIC-PCR. Among 49 *A. baumannii* isolates, resistance to meropenem (77.6%) and imipenem (75.5%) was high, while colistin remained effective. MDR rates ranged from 55.1% to 79.6%, and XDR from 12.3% to 65.3%. We detected the blaOXA-51 and blaOXA-23 genes in 98% of isolates, while we found *bla*IM*P* and *bla*NDM *i*n 67.4% and 24.5%, respectively. ERIC-PCR identified 11 clusters, with C6, C9, and C1 showing the highest resistance. While tigecycline and polymyxin remained effective in most clusters. *A. baumannii* isolates from the area showed a lot of genetic diversity and high resistance to carbapenem. The widespread presence of *bla*OXA and MBL genes highlights the urgent need for enhanced infection control measures.

Published by Arab Society for Fungal Conservation

Introduction

Acinetobacter baumannii, an emerging gram-negative opportunistic pathogen, has received increased attention in the medical community due to its classification as an ESKAPE (Enterococcus faecium, *Staphylococcus aureus, Klebsiella pneumoniae, A. baumannii*, *Pseudomonas aeruginosa*, and *Enterobacter* spp.) pathogen, a group of microorganisms notorious for their ability to evade antimicrobial interventions (Ching et al. 2024).

This bacterium imposes a significant clinical burden by causing invasive infections in hospitalized individuals and those with compromised immune systems (Ganjo et al. 2016). Clinical manifestations of *A. baumannii* infections span a spectrum of severe conditions, including pneumonia, sepsis, meningitis, urinary tract infections, and wound infections, constituting a significant contributor to nosocomial mortality, with estimates attributing up to 35% of such mortality to this pathogen (Nocera et al. 2021).

A. baumannii is a major threat to healthcare because of its ability to acquire resistance to numerous medications. The global spread of multidrug-resistant (MDR) *A. baumannii* strains has increased morbidity, death, and healthcare expenditures (Kyriakidis et al.,

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2021). Carbapenems, such as imipenem and meropenem, are critical therapies; however, carbapenem-resistant *A. baumannii* (CRAB) complicates therapy and has been associated with significant mortality in bacteremia patients. The carbapenem resistance mechanisms largely involve Class B metallo-β-lactamases (MBLs) such as blaNDM and blaIMP and class D oxacillinases (OXAs) including *bla*OXA-23, *bla*OXA-51, and *bla*OXA-58 (Su et al., 2023). The resistance of *A. baumannii* to carbapenems has reached up to 90% in regions like the Middle East, Southern Europe, and North Africa, with high infection rates reported in the UAE, Saudi Arabia, Palestine, and Lebanon. This growing resistance poses serious challenges for treatment and infection control, raising significant concerns in the medical community (Al-Rashed et al., 2023). Iraqi Kurdistan Region cities have multiple tertiary care facilities where multidrug- and carbapenem-resistant *A. baumannii* infections pose a substantial clinical issue (Qurbani et al., 2024). Despite attempts to reduce the spread of these pathogens, little information is known about the genetic diversity and transmission dynamics of *A. baumannii* strains circulating in this environment (Ganjo et al. 2016, Shayea &Ali, 2022; Khlaif & Hussein, 2022; Abduljabar & Mawlood, 2023).

Understanding the genetic relatedness and epidemiological dynamics of *A. baumannii* isolates is critical for determining transmission routes, locating outbreak clusters, and implementing targeted infection control strategies. Among the existing molecular typing techniques, Enterobacterial Repetitive Intergenic Consensus PCR (ERIC-PCR) has emerged as a viable tool for genotyping A. baumannii strains because of its high discriminating power, repeatability, and convenience. ERIC- PCR analyzes the genetic fingerprints of bacterial isolates to detect clonal outbreaks, identify transmission routes, and track strain development over time (Maleki et al. 2022; Osanloo et al. 2023).

This study aims to investigate the genotypic diversity of carbapenemase-resistant *A. baumannii* strains isolated from hospitalized patients using ERIC-PCR. By analyzing the genetic fingerprints generated by ERIC-PCR, we seek to delineate the clonal relationship among isolates and characterize the population structure of CRAB.

Materials and Methods

Bacterial isolation, culture, and identification.

A total of 283 clinical samples, including blood, urine, sputum, wound swabs, burn swabs, and CSF, were obtained from ICU and general ward patients (after obtaining consent) in some hospitals in the Kurdistan region of Iraq from August 2023 to April 2024. The specimens were examined according to standard methods of bacteriology, including culturing, Gram staining, culture morphology assessment, and then bacterial identification and confirmation using the VITEK 2 compact system. Patient demographics and culture types were recorded throughout, and an ATCC strain was a control (by Medya Diagnostic Center).

Antibiotic susceptibility testing

Antimicrobial susceptibility analysis was performed utilizing the VITEK 2 compact system. The test was against ceftazidime (CAZ), imipenem (IMI), meropenem (MPR), piperacillin (PIP), amikacin (AM), tigecycline (TG), tetracycline (TC), colistin (Col), gentamicin (GM), tazobactam (TZP), tobramycin (TOB), ciprofloxacin (CIP), trimethoprim/sulfamethoxazole (STX), and levofloxacin (LEV). Clinical and Laboratory Standards Institute (CLSI) breakpoints were used to analyze data, and the results were recorded as susceptible (S), intermediate (I), or resistant (R) (Patel et al., 2015).

DNA extraction and molecular detection of carbapenemase resistance genes

DNA was isolated from bacterial isolates and the reference strain ATCC using the Qiagen extraction kit according to the manufacturer's instructions. For PCR, each reaction mixture of 20 µL was prepared, consisting of deionized water, PCR Master Mix (Eppendorf, Germany), forward and reverse primers (*blaOXA-51, blaOXA-23, blaI*MP, or *blaNDM*, Table 1), and DNA template. The PCR program consisted of an initial denaturation step at 94°C for 5 minutes and a final extension at 72°C for 10 minutes, Table 1. PCR results were then examined using gel electrophoresis in 1% agarose.

ERIC-PCR - Molecular Typing

ERIC-PCR was conducted using universal primers (ERIC1 5′-ATGTAAGCTCCTGGGGATTCAC-3′ and ERIC2 5′-AAGTAAGTGACTGGGGTGAGC-3′) to assess the heterogeneity of bacterial isolates (Ralte et al., 2022). The reactions were performed in 20 µl volumes, each containing 1 µl of each primer, 10 µl of master mix, 1 µl of template DNA, and 7 µl of deionized water. The PCR conditions included an initial denaturation at 94°C for 5 minutes, with the next 35 cycles consisting of denaturation at 95°C for 1 minute, annealing at 53°C for 1 minute and extension at 72°C for 3 minutes, with a final extension at 72°C for 10 minutes. PCR products were gel electrophoresed on a 1.6% agarose gel at 80 V for 180 minutes and then observed under UV light with a gel documentation system.

Table 1 Primer sequence, size, and PCR condition for carbapenase resistance gene in the present study.

Primer name	Nucleotide sequence (5′ → 3′)	Size (bp)	PCR condition 30 cycles	Reference
*bla*OXA-51	F: TAATGCTTTGATCGGCCTTG R: TGGATTGCACTTCATCTTGG	353	94°C, 1 min, 53°C, 30 sec; 72°C, 1 min	(Hou &Yang, 2015)
blaOXA-23	F: GATCGGATTGGAGAACCAGA R: ATTCTTGACCGCATTTCCAT	501	94°C, 1 min; 53°C, 40 sec; 72 °C, 1 min	
bla*IMP*	F: GGAATAGAGTGGCTTAAYTCTC R: CCAAACYACTASGTTATCT	188	94°C, 1 min; 53°C, 30 sec; 72 °C, 1 min	(Abduljabar & Mawlood, 2023)
blaNDM	F: CACCTCATGTTTGAATTCGCC R: CTCTGTCACATCGAAATCGC	984	94°C, 1 min; 52°C, 60 sec; 72°C, 1 min	(Nordmann et al. 2011)

Statistical Analysis

Frequencies and percentages were used to describe the variables in this study.

For Molecular Typing, ERIC-PCR - Band patterns were analyzed using a zero-one manual method to count the bands, and the dendrogram was created by entering data into a designated website: http://insilico.ehu.es/dice_upgma/ for hierarchical clustering and phylogenetic analysis among carbapenem-resistance *A. baumannii* isolates.

Results

Patients and bacterial Isolates

Forty-eight *A. baumannii* isolates were recovered from 283 total clinical samples from hospitalized patients in this study. The age of the patients ranged from 5 months to 62 years; there were 28 males (58.3%) and 20 females (41.6%). The most common sources of *A. baumannii* isolation from ICU patients were surgical site wound infection (39.6%), followed by burn (27.1%), urine and sputum each (10.4%), blood (8.3%), and CSF (4.2%), (Table 2).

Antimicrobial resistance pattern of MDR and XDR A. baumannii isolates

Table 3 depicts the resistance rate of *A. baumannii* isolates to 14 antibiotics of various classes. *A. baumannii* isolates are resistant to piperacillin (98%), trimethoprim/sulfamethoxazole (89.8%), ceftazidime (87.8%), tazobactam (85.7%), gentamicin (83.7%), levofloxacin (79.6%), amikacin (75.5%), meropenem (77.6%), imipenem (75.5%), ciprofloxacin (65.3%), and tobramycin (63.3%). They were more susceptible to colistin (98%) and tigecycline (67.3%).

Furthermore, the isolates, with the majority classified as either multidrug-resistant (MDR), showed 14 distinct patterns ranging from 55.1% to 79.6% resistance, or extensively drug-resistant (XDR), displaying 7 patterns with resistance rates ranging from 12.3% to 65.3% among isolates. Notably, the isolates in the XDR pattern that exhibited 12.3% were resistant to all used antibiotics: Ceftazidime (CAZ), Tazobactam (TZP), Piperacillin (PIP), and trimethoprim/sulfamethoxazole. Despite significant resistance, this pattern does not match the pan drug-resistant (PDR) requirements due to its remaining vulnerability to polymyxin. No pattern was PDR (Table 4).

Molecular Characterization of the Carbapenem-Resistance Determinants

In the present study, all isolates underwent PCR screening for metallo β-lactamase (MBL) and class D carbapenem hydrolyzing enzyme (Table 5), which revealed that the β-lactamase blaOXA-51 and blaOXA-23 genes were detected in 98% of isolates, whereas MBL, blaIMP, and blaNDM were found in 67.4% and 24.5%, respectively.

In this investigation, multiple resistance genes coexist among carbapenem-resistant *A. baumannii* isolates. The most prevalent combinations included blaOXA-23/blaOXA-51 (95.9%), blaOXA-23/blaIMP (67.4%), and blaOXA-51/blaIMP (65.3%). Also, blaOXA-23/blaOXA-51/blaIMP was found in 63.3% of the isolates, followed by blaOXA-51/blaNDM in 24.5%. Less common combinations included blaOXA-23/blaNDM and blaOXA-23/blaOXA-51/blaNDM, each found in 22.5%, and blaOXA-23/blaOXA-51/blaIMP/blaNDM was detected in 16.4% of the isolates (Table 5).

Table 2. Characterization of patients and distribution of *A. baumannii* recovered from clinical specimens.

Sex NO. (%)	Age groups (Years)					Hospitality	
	(>20)	(21-31)	31-40	41-50	>50	ICU	Ward
Male (M)= 28 (58.3)	4	8	6	1	9	26	2
Female (F)=20 (41.6)	5	3	3	7	2	17	3
Total=48 (100) + 1 lab strain	9 (18.8)	11 (22.9)	9 (18.8)	8 (16.6)	11 (22.9)	43 (89.6)	5 (10.4)
Source of specimen (S) NO.							
Wound= 19 (39.6) (M=10, F=9)	3	5	4	2	5	18	1
Burn = 13 (27.1) (M=7, F=6)	2	2	2	4	4	12	1
Urine= 5 (10.4) (M=4, F=1)	1	2	1	0	1	2	3
Sputum= 5 (10.4) (M=3, F=2)	1	2	1	2	0	5	0
Blood= 4 (8.3) (M=3, F=1)	1	1	1	0	1	4	0
CSF= 2 (4.2) (m=1, F=1)	1	0	0	0	0	2	0
Total=48 (100) + 1 lab strain	9 (18.8)	11 (22.9)	9 (18.8)	8 (16.6)	11 (22.9)	5 (10.4)	5 (10.4)
Hospital location of (S) NO. (%)							
Ward=5	3	2	0	0	0		
ICU=43	6	9	9	8	11		
Total=48 (100) + 1 lab strain	9 (18.8)	11 (22.9)	9 (18.8)	8 (16.6)	11 (22.9)		

Table 3. Resistance of antibiotic classes among clinical isolates

Antibiotic Class	Antimicrobial agent	Resistance %	Intermediate %	Sensitive %	Total NO. %
Penicillins	Piperacillin	48 (98)	1 (2)	0 (0)	49 (100)
Folate pathway inhibitors	trimethoprim/sulfamethoxazole	44 (89.8)	0 (0)	5 (10.2)	49 (100)
3rd generation Cephalosporins	Ceftazidime	43 (87.8)	0 (0)	6 (12.2)	49 (100)
β-lactamase inhibitors	Tazobactam	42 (85.7)	2 (4.1)	5 (10.2)	49 (100)
Carbapenem	Meropenem	38 (77.6)	3 (6.1)	8 (16.3)	49 (100)
	Imipenem	37 (75.5)	3 (6.1)	9 (18.4)	49 (100)
Fluoroquinolones	Ciprofloxacin	32 (65.3)	10 (20.4)	7 (14.3)	49 (100)
	Levofloxacin	39 (79.6)	1 (2.0)	9 (18.4)	49 (100)
	Amikacin	37 (75.5)	3 (6.1)	9 (18.4)	49 (100)
Aminoglycosides	Gentamicin	41 (83.7)	4 (8.2)	3 (6.1)	49 (100)
	Tobramycin	31 (63.3)	6 (12.2)	12(24.5)	49 (100)
Tetracyclines	Tetracycline	16 (32.7)	25 (51)	8 (16.3)	49 (100)
	Tigecycline	0 (0)	16 (32.7)	33(67.3)	49 (100)
Polymyxin	Colistin	0 (0)	1 (2.0)	48 (98)	49(100)

Table 4. Patterns of resistance of *A. baumannii* isolates in this present study.

Type of resistance	Antibiotic Classes NO.	Antimicrobial resistance patterns	Resistance NO. %
MDR	4	CAZ, TZP, PIP, STX	39/49 (79.6)
	4	CAZ, PIP, STX, GM	39/49 (79.6)
	4	CAZ, TZP, PIP, LEV	35/49 (71.4)
	4	CAZ, PIP, STX, IMI, MRP	34/49 (69.4)
	4	TZP, PIP, STX, IMI, MRP	33/49 (67.4)
	4	CAZ, TZP, PIP, STX, IMI	33/49 (67.4)
	5	CAZ, PIP, STX, LEV, GM	36/49 (73.5)
	5	CAZ, PIP, STX, IMI, MRP, LEV	35/49 (71.5)
	5	CAZ, TZP, PIP, STX, IMI, MRP	33/49 (67.5)
	5	CAZ, PIP, STX, MRP, LEV, GM	33/49 (67.5)
	5	CAZ, TZP, PIP, STX, LEV, CIP	29/49 (59.2)
	6	CAZ, TZP, PIP, STX, IMI, MRP, GM	31/31 (63.3)
	6	CAZ, TZP, PIP, STX, IMI, MRP, CIP	27/49 (55.1)
	6	CAZ, TZP, PIP, STX, IMI, MRP, CIP, LEV	27/49 (55.1)
XDR	7	CAZ, TZP, PIP, STX, IMI, MRP, LEV, GM	32/49 (65.3)
	7	CAZ, TZP, PIP, STX, IMI, MRP, LEV, GM, AK	29/49 (59.2)
	7	CAZ, TZP, PIP, STX, IMI, MRP, CIP, LEV, GM	27/49 (55.1)
	7	CAZ, TZP, PIP, STX, IMI, MRP, CIP, LEV, GM, AK	27/49 (55.1)
	7	CAZ, TZP, PIP, STX, IMI, MRP, CIP, LEV, GM, AK, TOB	17/49 (34.7)
	8	CAZ, TZP, PIP, STX, IMI, MRP, CIP, LEV, GM, AK, TOB, TC	7/49 (14.3)
	8	CAZ, TZP, PIP, STX, IMI, MRP, CIP, LEV, GM, AK, TOB,	6/49 (12.3)
PDR	8	CAZ, TZP, PIP, STX, IMI, MRP, CIP, LEV, GM, AK, TOB, TC, TG	0/49 (0)
	9	CAZ, TZP, PIP, STX, IMI, MRP, CIP, LEV, GM, AK, TOB, TC, TG, Col	0/49 (0)

IMI: Imipenem, MRP: Meropenem, CAZ: Ceftazidime, TZP: Tazobactam, PIP: Piperacillin, TG: Tigecycline, LEV: Levofloxacin, CIP: Ciprofloxacin, GM: Gentamycin, TOB: Tobramycin, AK: Amikacin, TC: Tetracycline, Col: Colistin, STX: Trimethoprim/Sulfamethoxazole.

Table 5. Carbapenemase-resistant genes distribution among *A. baumannii* in this study.

Gene Name	Carbapenems Resistance isolate NO. %	Carbapenems Sensitive isolate NO. %
*bla*OXA-51	48/49 (98)	1/49 (2)
*bla*OXA-23	48/49 (98)	1/49 (2)
*bla*IMP	33/49 (67.4)	16/49 (32.6)
*bla*NDM	12 (24.5)	37/49 (75.5)
Carbapenem resistance gene combination		**Number of isolates NO. %**
*blaO*XA-23/ *bla*OXA-51		47/49 (95.9)
*blaO*XA-23/ *bla*IMP		33/49 (67.4)
*bla*OXA-51/ *bla*IMP		32/49 (65.3)
*blaO*XA-23/ *blaOXA-51*/ *bla*IMP		31/49 (63.3)
*bla*OXA-51/ *bla*NDM		12/49 (24.5)
*blaO*XA-23/ *bla*NDM		11/49 (22.5)
*blaO*XA-23/ *bla*OXA-51/ *bla*NDM		11/49 (22.5)
*blaO*XA-23/ *bla*OXA-51/ *bla*IMP/ *bla*NDM		8 (16.4)

Molecular typing of A. baumannii

Figure 1 depicts the findings of ERIC-PCR fingerprinting of carbapenem-resistant *A. baumannii* isolates using a 1.6% agarose gel. The banding patterns were digitized using a binary method (presence/absence represented as 1/0), and the Dice similarity coefficient was applied to create a dendrogram, allowing for cluster analysis of the band profiles.

Figure 2 shows a phylogenetic tree that further elucidates the genetic diversity of the carbapenem-resistant *A. baumannii* strains, classifying them into eleven separate groups (C1, C2, C3, C4, C5, C6, C7, C8, C9, C10, and C11).

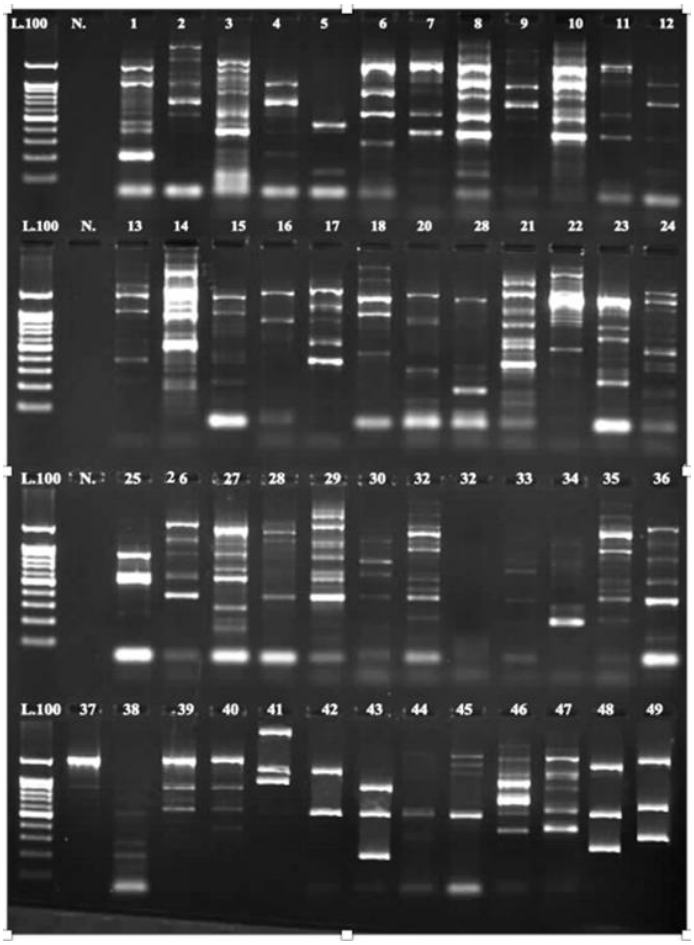

Fig. 1. Eric-PCR 1.6-agarose gel electrophoresis of *A. baumannii* strains, Lane (L100), DNA marker (100-bp), N, Negative control; 6= positive control (Lab strain ATCC)

All isolates from clusters C2, C3 (except for the control), C6, C7, and C8 were from ICU patients across various sources, including burns, wounds, sputum, and urine (Figure 2, Table 6, Supplementary). Regarding the distribution of carbapenemase genes among CRAB isolates within each cluster, all isolates in the clusters were found positive for both *bla*OXA-51 and *bla*OXA-23, except for clusters C2 and C1. In C2, 66.7% (2/3) of isolates were positive for *bla*OXA-51, whereas in C1, 83.3% (5/6) were positive for *bla*OXA-23. Furthermore, the *bla*IMP gene was found in 100% of isolates in clusters C5, C8, and C11. Additionally, all isolates in cluster C9 were 100% positive for the *bla*NDM gene, indicating a broad range of positive results for *bla*NDM across this cluster (Figure 1 & Table 6, Supplementary file).

Table 7 presents the resistance profiles of isolates across 11 clusters against various antibiotic classes, highlighting the prevalence of MDR and XDR phenotypes. Notably, clusters C6 and C9 exhibited the highest resistance, with isolates classified as XDR showing 100% resistance to all tested antibiotic classes except for tigecycline and polymyxin. Cluster C1 also demonstrated significant resistance, with 100% of the isolates resistant to meropenem, imipenem, piperacillin, trimethoprim/sulfamethoxazole, ceftazidime, tazobactam, levofloxacin, and 83.3% resistant to ciprofloxacin and gentamicin, as well as C5, which showed complete resistance (100%) to all used antibiotics, except for tobramycin, tigecycline and colistin. Across all clusters, tigecycline and polymyxin retained efficacy against most of the isolates.

Discussion

CRAB strains are among the most difficult to manage and treat in clinical settings (Tacconelli et al., 2018; Goic-Barisic et al. 2021; Al-Rashed et al. 2023). In this study, 48 *A. baumannii* isolates were obtained from 283 clinical samples from hospitalized patients aged 5 months to 62 years. Of these patients, 58.3% were male and 41.6% female. The isolates from ICU patients were most frequently found in surgical-site wound infections (39.6%), followed by burns (27.1%), urine and sputum (each 10.4%), blood (8.3%), and cerebrospinal fluid (4.2%).

The results of the current study demonstrate that the majority of clinical *A. baumannii* isolates were resistant to the utilized antibiotic classes, with considerable resistance to piperacillin (98%), trimethoprim/sulfamethoxazole (89.8%), ceftazidime (87.8%), tazobactam (85.7%), gentamicin (83.7%), levofloxacin (79.6%), amikacin (75.5%), meropenem (75.5%), imipenem (73.4%), ciprofloxacin (65.3%), and tobramycin (63.3%). There were still a few antimicrobial drugs in this study that were successful against the majority of CRAB, such as colistin (98%) and tigecycline (67.3%), respectively. Similar findings were previously published in investigations done in Erbil, Babylon, and Baghdad, which revealed that *A. baumannii* resistance rates to these medicines varied little. Nonetheless, overall susceptibility to colistin and tigecycline was observed (Al-Kadmy et al. 2018; Radhi & Al-Charrakh, 2019; Hassan & Khider, 2019; Abduljabar & Mawlood, 2023).

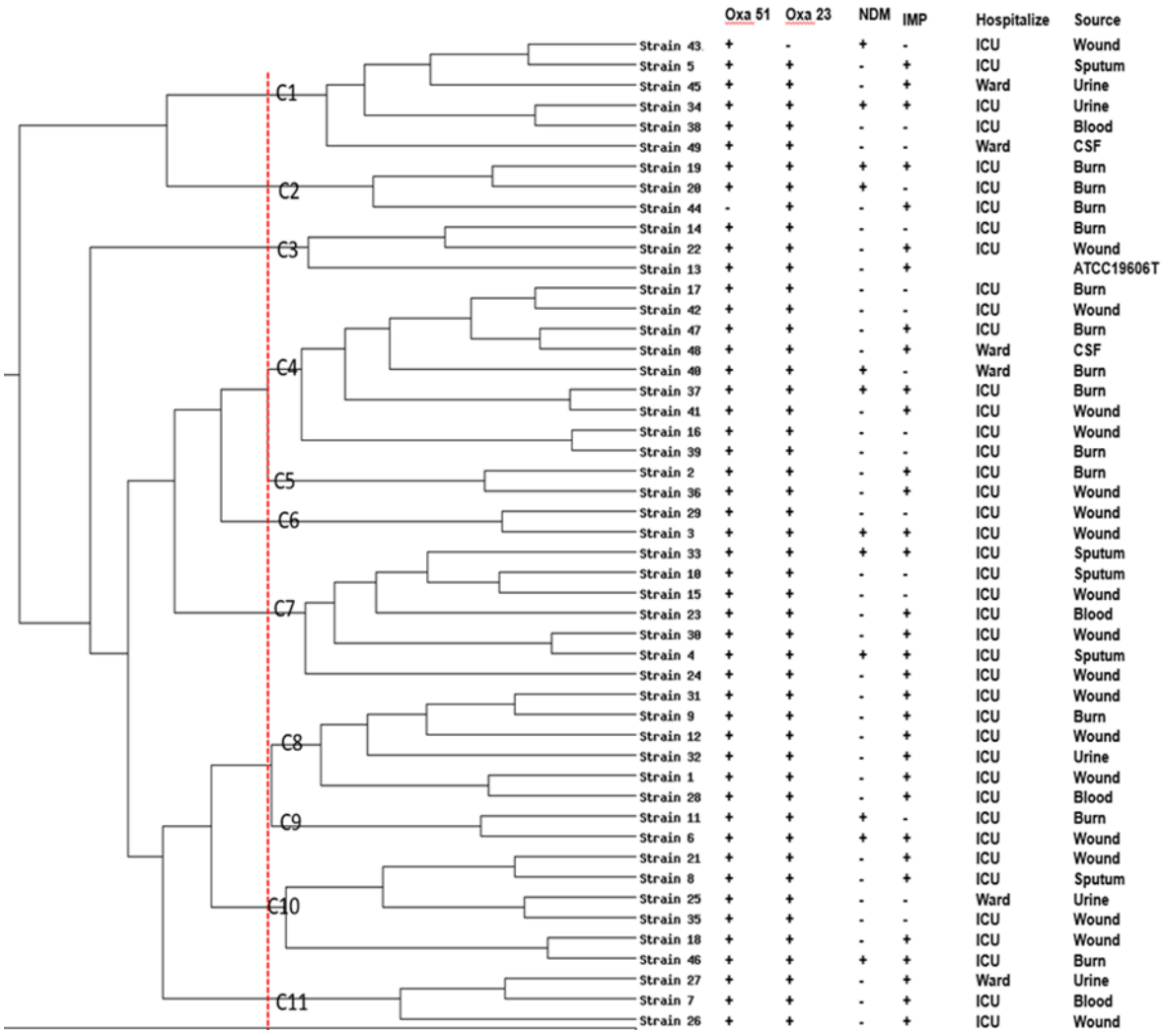

Fig. 2. A dendrogram of the strains investigated in this research. Cluster: C; Strain: S; **C1**: (S (43, S 5), S 45), S 34, S 38)), S 49); **C 2**: (S 19, S 20), S 44); **C 3**: (S 14, S 22), S 13); **C 4**: (S 17, S 42), (S 47, S 48)), S 40), (S 37, S 41)), (S 16, S 39); **C 5**: (S 2, S 36); **C 6**: (S 29, S 3); **C 7**: (S 33, (S 10, S 15)), S 23), (S 30, S 4)), 24); **C 8**: (S 31, S 9), S 12), S 32), (S 1, S 28); **C 9**: (S 11, S 6); **C 10**: (S 21, S 8), (S 25, S 35)), (S 18, S 46); **C 11**: (S 27, S 7), S 26).

Table 7. Distribution of antibiotic-resistant among CRAB within clusters

Cluster (C) NO	Antimicrobial Susceptibility test: R, I, S. %													
	PIP	**STX**	**MRP**	**IMP**	**CAZ**	**TZM**	**LEV**	**CIP**	**GM**	**TOB**	**AK**	**TC**	**TG**	**Col**
C1= 6	100	100	100	100	100	100	100	83.3	83.3	50	66.7	33.4, 50	50, 50	100
C2= 3	100	66.7	66.7	66.7	66.7	33.4	66.7	66.7	66.7	66.7	66.7	33.4	33.4	100
C3= 3	100	100	66.7	66.7	66.7	66.7	66.7	66.7	100	66.7	100	66.7	66.7	100
C4= 9	100	77.8	66.7	55.6	77.8	77.8	55.6	55.6	66.7	77.8	77.8	33.4	77.8	100
C5= 2	100	100	100	100	100	100	100	100	100	50, 50	100	100	50, 50	100
C6= 2	100	100	50	50	100	50	100	50	100	100	50	50, 50	100	100
C7= 7	85.7	85.7	85.7	100	85.7	100	85.7	57.2	85.7	42.9	71.5	71.5	85.7	100
C8= 6	100	100	83.4	83.4	83.4	100	83.4	50	100	83.4	83.4	50	83.4	100
C9= 2	100	100	100	100	100	100	100	100	100	100	100	100	50	100
C10= 6	100	83.4	50	50	100	83.4	66.7	66.7	66.7	66.7	50	33.4	83.4	100
C11= 3	100	100	100	100	100	100	100	100	100	66.7	100	33.4	66.7	100

PIP: Piperacillin, STX: Trimethoprim/Sulfamethoxazole, MRP: Meropenem, IMI: Imipenem, CAZ: Ceftazidime, TZP: Tazobactam, LEV: Levofloxacin, CIP: Ciprofloxacin, GM: Gentamycin, TOB: Tobramycin, AK: Amikacin, TC: Tetracycline, , TG: Tigecycline, Col: Colistin. **R= Resistant, I= intermediate, S= Sensitive.**

For this reason, the combination of colistin and tigecycline with carbapenems or other antibiotics is currently considered the best treatment option for multidrug-resistant A. baumannii infections and CRAB, despite rising resistance to these drugs (Aldali, 2023).

The study found that most *A. baumannii* isolates were classified as either multidrug-resistant (MDR) in 14 patterns (55.1%–79.6%) or extensively drug-resistant (XDR) in 7 patterns (12.3%-65.3%). Notably, 12.3% of isolates were broadly resistant to antibiotics like ceftazidime, tazobactam, piperacillin, and meropenem, but remained susceptible to polymyxin, thus not qualifying as pan-drug resistant (PDR). No PDR patterns were observed. Recently, the increasing rate of multidrug-resistant *A. baumannii* and CRAB has been predominantly distributed worldwide due to excessive antibiotic usage and inadequate infection control methods (Elbehiry et al. 2023). In addition, several studies have reported significant incidences of MDR and XDR A. *baumannii* isolates in different regions, including Iran (Abbasi et al. 2023, Kadivarian et al. 2023; Hazhirkamal et al. 2021), Iraq (Ahmad & Mohammad, 2020; Al Marjania et al. 2021; Shali et al. 2022), and China (Chen et al. 2022). Further research highlights trends in MDR *A. baumannii* infections in the United Kingdom and Egypt, with a notable rise in incidence in Egypt and alarmingly high levels of antibiotic resistance (Elwakil et al. 2023). The widespread use of carbapenems in Iraqi hospitals likely contributes to this resistance (Radhi & Al-Charrakh, 2019). Differences in carbapenemase gene distribution across countries may reflect ecological factors, antibiotic use policies, and antibiotype variations (Azimi et al. 2015).

Carbapenems are effective antibiotics for treating severe hospital-acquired infections, especially those caused by multidrug-resistant bacteria such as A. baumannii (Nguyen and Joshi, 2021). *A. baumannii*'s β-lactam resistance is mostly caused by enzyme degradation by β-lactamases, which include oxacillinases (OXA-type) and metallo-*β*-lactamases (MBLs), both of which exhibit carbapenemase activity, contributing significantly to carbapenem resistance (Massik et al. 2021). In a recent study, the sequencing of 313 genome carbapenem-resistant *A. baumannii* isolates that belonged to nearly 50 countries was performed, providing insights into the global distribution of these bacterial clones and their carbapenemase genes (Castillo-Ramírez, 2023). In the present study, all isolates underwent PCR screening for metallo *β*-lactamase (MBL) and class D carbapenem hydrolyzing enzyme, which revealed that the *β*-lactamase blaOXA-51 and blaOXA-23 genes were detected in 98% of isolates, whereas MBL, *bla*IMP, and *bla*NDM were found in 67.4% and 24.5%, respectively. The previous study highlights significant regional variations in the prevalence of different carbapenemase genes, particularly noting a 91.6% prevalence of *bla*OXA-23-like in America (Chávez Rodríguez et al. 2024).

In the line of this study, several studies were conducted on molecular epidemiology and antimicrobial resistance patterns of carbapenem-resistant *A. baumannii* (CRAB); for instance, one study investigated *A. baumannii* isolates from ICU patients in Hamadan, Iran. Out of 100 isolates, 84% carried the *bla*OXA-23 gene, while the incidence of the *bla*OXA-58 gene was only 3% (Kafshnouchi et al. 2022). Another study identified the *bla*IMP gene in this bacterium from burn patients in Duhok, Iraq, with 78.6% of isolates resistant to imipenem, highlighting MBL production as a significant contributor to antibiotic resistance (Khalid, 2024). Moreover, a study in Tunisia characterized the prevalence of *β*-lactamase-encoding genes in CRAB and found that all strains harbored *bla*OXA-51-like and *bla*OXA-23 genes, with some also carrying *bla*NDM-1 (Raddaoui et al. 2024). Another study that investigated the prevalence of carbapenem resistance genes of *A. baumannii* in Taiwan revealed that a significant majority of the isolates carried the *bla*OXA-51 gene, with various other resistance genes also identified, highlighting the importance of understanding these mechanisms for controlling antibiotic-resistant pathogens (Su et al. 2023). The study investigates the phenotypic and genotypic characteristics of carbapenem-resistant *A. baumannii* (CRAB) isolates in Thailand, revealing that 57.56% of the isolates are biofilm producers and identifying the predominant antibiotic resistance gene as *bla*OXA-51-like (Santajit et al. 2023).

Similarly, research in Iran found that *bla*OXA-51/*bla*OXA-23 was the most prevalent gene pattern among the studied *A. baumannii* isolates (Kafshnouchi et al. 2022). In contrast to the current study, metallo-*β*-lactamase *bla*IMP was found to be defective at a low rate in the research conducted by (Anoar et al. 2014) in Sulaimani City, Iraq, where 10.7% and 2.8% of CRAB isolates contained *bla*IMP and *bla*VIM, respectively, and *bla*NDM was not discovered in any *A. baumannii* isolate. On the other hand, the presence of *bla*OXA-51 shows significant carbapenemase activity just following the overexpression, so carbapenem resistance in *A. baumannii* requires the co-existence of other acquired oxacillinases like blaOXA-23, *bla*OXA-143, *bla*OXA-24, and *bla*OXA-143 enzymes (Abduljabar & Mawlood, 2023).

The ERIC-PCR fingerprinting analysis of carbapenem-resistant *A. baumannii* isolates was performed. The banding patterns were digitized in a binary format (presence/absence as 1/0), and the Dice similarity coefficient was used to create a dendrogram, allowing for thorough cluster analysis of genetic profiles. The phylogenetic tree reveals the vast genetic variety of these

carbapenem-resistant *A. baumannii* strains, which are divided into eleven different groups (C1-C11). This categorization highlights significant variation across isolates, indicating a high level of genetic diversity within isolates. Several studies have used the ERIC-PCR approach to understand the epidemiology and genotyping of *A. baumannii* isolates worldwide. This method has proven useful in tracking the spread of resistant strains, providing valuable insights into *A. baumannii*'s genetic diversity and transmission dynamics, and facilitating a better understanding of the mechanisms underlying antibiotic resistance in various regions around the world (Aljindan et al. 2018; Falah et al. 2019).

All isolates in clusters C2, C3 (excluding the control), C6, C7, and C8 were derived from ICU patients across diverse sources, including burns, wounds, sputum, and urine. Analysis of carbapenemase gene distribution revealed that *bla*OXA-51 and *bla*OXA-23 were prevalent (100%) across most clusters, with exceptions: in cluster C2, only 66.7% of isolates harbored *bla*OXA-51, and in cluster C1, 83.3% contained *bla*OXA-23. Furthermore, the blaIMP gene was universally present in all isolates within clusters C5, C8, and C11, while cluster C9 exhibited 100% positivity for the *bla*NDM gene, indicating a high prevalence of *bla*NDM within this group. Furthermore, Table 7 displays the resistance profiles of isolates across all 11 clusters against various antibiotic classes, highlighting the prevalence of multidrug-resistant (MDR) and extensively drug-resistant (XDR) phenotypes. Clusters C6 and C9 showed the highest resistance levels, with XDR isolates exhibiting 100% resistance to all tested antibiotic classes except for tigecycline and polymyxin. Cluster C1 also showed significant resistance, with 100% of isolates resistant to meropenem, imipenem, piperacillin, trimethoprim/sulfamethoxazole, ceftazidime, tazobactam, and levofloxacin; 83.3% were resistant to ciprofloxacin and gentamicin. Cluster C5 demonstrated complete resistance (100%) to all antibiotics tested, except tobramycin, tigecycline, and colistin. Notably, tigecycline and polymyxin remained effective against most isolates across all clusters.

Similar to the current study, several studies have investigated the molecular typing and genetic diversity of MDR *A. baumannii* isolates using methods such as ERIC-PCR, MLVA, and PCR-based techniques. Osanloo et al. found that 91% of isolates were extensively drug-resistant, demonstrating the effectiveness of ERIC-PCR and MLVA for assessing genetic diversity and clonal relationships (Osanloo et al. 2023). Similarly, Falah et al. highlighted the significant genetic diversity among *A. baumannii* isolates in burn hospitals using PCR and ERIC-PCR techniques (Falah et al. 2019). Sepahvand et al. (2022) evaluated colistin-resistant and sensitive strains, categorizing them into five and six groups using BOX-PCR and ERIC-PCR, underscoring the need for rapid control measures (Sepahvand et al. 2022). Nayak et al. compared PCR-based typing methods, with RAPD revealing the highest genetic similarity at 91%, while outer membrane protein profiles helped differentiate isolates (Nayak et al. 2023). Hashemizadeh et al. examined the role of transposable elements in carbapenem resistance and contributed significantly to resistance gene development (Hashemizadeh et al. 2022). Fatmawati et al. used RAPD-PCR and antibiograms to detect a potential outbreak of multidrug-resistant A. baumannii, correlating genetic and antibiotic susceptibility patterns, which could assist in outbreak management without advanced laboratory resources (Fatmawati et al. 2023).

Conclusion

Carbapenem-resistant was noted in a large majority of the isolates recovered. Furthermore, our data confirmed the significant frequency of the *blaOXA-51* and *blaOXA-23* genes in carbapenem-resistant *A. baumannii.* The quantity of XDR isolates identified in this investigation is concerning. Isolates have a high degree of diversity. The study's findings are anticipated to help improve our understanding of the epidemiology of *A. baumannii* infections and aid the development of specific techniques for infection management and prevention in hospitals. This study highlights the critical role of blaOXA and MBL genes in the spread of carbapenem resistance.

Conflict of interests.

No conflict of interests.

Ethical approval

This study received approval from the Research Ethics Committee of the University of Raparin, with the reference number 30, dated 26 June 2023.

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