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Molecular Analysis and Genotyping of Drug-Resistant *Acinetobacter baumannii* Isolates from Clinical Specimens

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***Abstract*—*Acinetobacter baumannii* is a globally concerning hospital infection because it has developed resistance to many antibiotics, including last-resort carbapenems. In this study, 46 non-duplicate *A. baumannii* isolates from hospitalized patients are identified by the Phoenix BD Diagnostic System, which are used for bacterial identification and antimicrobial susceptibility profiles. Various clinical specimens, including endotracheal aspiration, urine, wound, blood, burns, and cerebrospinal fluid, were collected between 2023 and 2024 at different hospital wards. To provide further understanding of the epidemiology of multidrug-resistant (MDR) *A. baumannii* isolates, this study attempts to; (1) utilize enterobacterial repetitive intergenic consensus polymerase chain reaction (ERIC-PCR) DNA fingerprinting to estimate genetic diversity, which reveals a significant level of genetic relationship is established amongst *A. baumannii* isolates from hospitalized patients, suggesting cross-transmission, and (2) clarifies the genetic characteristics of the antimicrobial resistance profiles contributing to the antibiotics widely used for MDR *A. baumannii* isolates. All isolates are classified as MDR (54.3%), extensively drug-resistant (39.1%), and pandrug-resistant (6.5%). According to Clinical and Laboratory Standards Institute-2024 standards, 93.4% and 91.3% of isolates are resistant to meropenem and imipenem, respectively, while colistin and tigecycline are the most effective antibiotics. Furthermore, the most common of the genes present among clinical isolates are *blaOXA-51* (100%) and *pmrC* (97.80%), while the less common detected genes are *blaIMP* (0.0%) and *blaOXA-58* (46%). ERIC-PCR could provide a rapid and dependable scheme to recognize clonal relationships among isolates from a multiplicity of clinical samples. Controlling endemic *A. baumannii* strains, particularly in intensive care unit settings, is essential.**

***Index Terms*: *Acinetobacter baumannii,* Antibiotic resistance gene, Enterobacterial repetitive intergenic consensus polymerase chain reaction, Extensively drug-resistant, Multidrug-resistant, Pan drug-resistant**

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I. Introduction

Acinetobacter baumannii (*A. baumannii*) has been identified as a highly concerning nosocomial pathogen on a worldwide scale. Being one of the most successful multidrug-resistant (MDR) organisms endangering modern antibiotic treatment, its remarkable ability to acquire or upregulate multiple resistance determinants has been a major issue in its clinical relevance (Ali, Al-Haideri, and Al Hishimi, 2022; Kyriakidis, et al., 2021). In addition to this intriguing development of resistance, *A. baumannii* has a variety of survival mechanisms that allow it to thrive in a variety of settings, increasing its potential to spread across hospitals (Sehree, Al-Kaysi and Abdullah, 2023; Shayea and Ali, 2022). The World Health Organization considers *A. baumannii* to be a critical priority pathogen, indicating that it represents a major threat to human health and that new medicines are desperately needed (Tagliabue and Rappuoli, 2018).

Increased proof of *A. baumannii* isolates that are both extensively drug-resistant (XDR) and pan-drug-resistant (PDR) is also emerging in multiple countries (Assimakopoulos, et al., 2019; Saelim, et al., 2018). The necessity to learn more about and assess the pathogen's mechanisms of antibiotic resistance as well as illness consequences for clinical and public health reasons is evident. There is now more interest in using colistin (CL) because of the quick development of *A. baumannii* resistance to most antibiotics, including carbapenems. At present, polymyxin resistance in *A. baumannii* is on the rise (Kumburu, et al., 2019; Abbasi, Hajihashemi and Shokri, 2024). Polymyxins have an amphipathic structure, cationic properties, and primarily react with the negative charge of the lipid a component of lipopolysaccharide (LPS). Controlled addition of positively charged residues, such as phosphoethanolamine (PetN) to LPS, diminishes negative charge on the bacterial surface and subsequently inhibits the adhesion of the polymyxin to the LPS (Moffatt, Harper and Boyce, 2019). *A. baumannii's* resistance to CL was initially chromosomal and is related to alterations in the *pmrCAB* operon, which limits its fast dissemination and distribution (Lima, et al., 2018). PetN transferase is encoded by the *pmrC* gene, whereas a two-component system (TCS) is encoded by the *pmrA* and *pmrB* genes. Mutations in the *pmrAB* TCS

system cause *pmrC* to be overexpressed, which modifies lipid A with PetN and results in CL resistance (Lima, et al., 2018; Trebosc, et al., 2019). Apparently, when *pmrAB* is mutated, *pmrC* expression rises, and this is linked to greater CL minimum inhibitory concentrations (MICs) (Abbasi, Hajihashemi and Shokri, 2024; Charretier, et al., 2018), which raises attention to the pathogen's strong propensity for spread and emphasizes the necessity to determine the true extent of CL resistance worldwide (Moubareck and Halat, 2020).

In infection control, various genotyping techniques have been established to better understand the clinical significance and epidemiological relationship among bacterial strains of *A. baumannii* outbreaks worldwide (Aljindan, Alsamman and Elhadi, 2018; Hamzah, 2018). Presently, biotyping and serotyping, as examples of phenotypic procedures, have now been replaced by molecular typing methods, including intragenic consensus Enterobacterial repetitive intergenic consensus polymerase chain reaction (ERIC-PCR) (Falah, Shokoohizadeh and Adabi, 2019). Repetitive sequences of the gene in *A. baumanni* are called symmetric elements and are often realized in the non-coding sequence of the DNA (Shayea and Ali, 2022). Concerning the variable length and number of these repeat sequences obtained for each isolate, some primers have been designed. The enterobacterial repetitive intergenic consensus (ERIC) amplicon's location and length are used as genetic indicators for the genotyping of *A. baumannii* according to the assortment of the bands (Falah, Shokoohizadeh and Adabi, 2019; Shayea and Ali, 2022).

In our domain, we have seen a significant increase in *A. baumannii* outbreak cases. Consequently, this study was directed to determine the ERIC-PCR DNA fingerprinting to clarify the epidemiology of MDR *A. baumannii* and the genetic characteristics of the antimicrobial resistance profiles contributing to the antibiotics widely used for MDR *A. baumannii* isolates.

II. Materials and Methods

A. Isolation and Identification of Bacterial Isolates

All 46 *A. baumannii* clinical isolates recovered from various clinical specimens, including endotracheal aspiration, urine, wound, blood, burns, and cerebrospinal fluid, between 2023 and 2024 at different hospitals in the Sulaymaniyah governorate, Iraq, were collected and identified using MacConkey agar, nutrient and blood base agar media (Himedia, India), *Acinetobacter* CHROMagar (CHROMagar, Paris, France), and Phoenix; BD Diagnostic Systems, Franklin Lakes, NJ, USA. Their final confirmation was performed by polymerase chain reaction (PCR) of the *blaOXA-51*-like gene, which is intrinsic to this species (Turton, et al., 2006). *blaOXA-51-negative* samples were excluded. The *A. baumannii* ATCC19606 was selected as a positive control. The isolates were then preserved in a Trypton Soy Broth medium (Himedia, India); 20% glycerol was added as a supplement and stored at −70°C until further analysis.

B. Antibiotic Susceptibility Test

MICs of 14 antibiotics: Ampicillin/sulbactam, piperacillin/tazobactam (P/T), Ceftazidime, cefepime (CEF), ceftriaxone (CTR), imipenem (IMP), meropenem (MER), amikacin (AK), CL, levofloxacin (LEV), ciprofloxacin, gentamicin (GEN), tigecycline (TIG), and trimethoprim-sulfamethoxazole (SXT) against *A. baumannii* isolates were determined using the ID/AST combo panel, NMIC/ID55 in (Phoenix; BD Diagnostic Systems, Franklin Lakes, NJ, USA). The MICs of all the antibiotics were interpreted by the aforementioned system according to the CLSI 2024 guidelines (*M100 Performance Standards for Antimicrobial*). Antimicrobial susceptibility of isolates was assessed applying the centers for disease control and prevention and European center for disease prevention and control definitions and categorized as MDR, XDR, and PDR antimicrobial categories (Alagna, et al., 2020).

C. ERIC-PCR Genotyping

To investigate the genetic variation and clonal relationships among isolates, specific ERIC-PCR primers and PCR conditions were achieved as displayed in "Table I" and used to determine the ERIC-type arrangements of isolates. The ERIC patterns and the dendrogram of isolates were analyzed by the XLSTAT software program.

D. PCR Screening for Antibiotic Resistance-Related Genes

The genomic DNA extraction of bacterial isolates was executed by the boiling method (Falah, Shokoohizadeh, and Adabi, 2019; Hou and Yang, 2015). All PCRs were executed in a final volume of 25 μL. Containing Taq Master Mix (addbio, Korea). Screening for the three groups of Oxacillinase (OXA)-type genes (*blaOXA-51*, *blaOXA-23*, and *blaOXA-58*) and Metallo Beta Lactamase (MBL) (*blaVIM*, *blaIMP, and blaNDM*), the isolates were also performed for CL Resistance *CoR* genes, *pmrCAB* (*pmrA, pmrB, and pmrC*), using primers and PCR conditions previously published in "Table I." 3 μL of *A. baumannii* whole DNA extract was used as a template for the PCR (10 μL of nuclease-free water, 10 μL of master mix ×2, and 1 μL per primer). In a thermocycler (Applied Biosystems, Singapore), a standard PCR amplification program was employed as follows: initial heating to 95°C for 5 min, followed by 30 cycles of amplification. Each cycle included three phases: denaturation at 95°C for 30 s, annealing at a proper temperature for 30 s, and extension at 72°C for the right time, tracked by 5 min at 72°C as a final elongation step. Finally, the PCR product was stored at 4°C until they were analyzed. To determine the elongation time, the length of the anticipated amplified DNA was utilized. A 1.5% agarose gel was used to electrophorese the PCR products, using ethidium bromide as a stain, and the results of the specific amplified target gene were visualized using a ultra-violet transilluminator (BIO-RAD, California, USA). Furthermore, Capillary Gel Electrophoresis, as an advanced technique, was used for the separation of PCR products, and DNA was detected by fluorescent labeling (QIAxcel Advanced, Germany).

TABLE I
PRIMER SEQUENCES, AMPLIFICATION SIZES, ANNEALING TEMPERATURES, AND ELONGATION TIMES OF ANTIBIOTIC RESISTANCE GENES IN CLINICAL ISOLATES OF *ACINETOBACTER BAUMANNII*

Targeted genes	Primer sequences (5'-3')	Product size (bp)	Annealing Temperature	Elongation time	References
blaOXA-51	F: TAATGCTTTGATCGGCCTTG R: TGGATTGCACTTCATCTTGG	353 bp	53°C	30 s	(Hou and Yang, 2015)
blaOXA-23	F: GATCGGATTGGAGAACCAGA R: ATTCTTGACCGCATTTCCAT	501 bp	53°C	40 s	(Hou and Yang, 2015)
blaOXA-58	F: AAGTATTGGGGCTTGTGCTG R: CCCCTCTGCGCTCTACATAC	599 bp	58°C	40 s	(Tawfeeq, et al., 2020)
blaIMP	F: GGAATAGAGTGGCTTAAYTCTC R: CCAAACYACTASGTTATCT	188 bp	53°C	30 s	(Poirel, et al., 2011)
blaNDM	F: CACCTCATGTTTGAATTCGCC R: CTCTGTCACATCGAAATCGC	984 bp	52°C	60 s	(Nordmann, et al., 2011)
blaVIM	F: GATGGTGTTTGGTCGCATA R: CGAATGCGCAGCACCAG	390 bp	55°C	40 s	(Poirel, et al., 2011)
pmrA	F: GATGGTTTAAATTTGGGTGCAGAT R: TTGACTCGCAAGTTGAGCTTCT	120 bp	58°C	45 sec	(Abbasi, Hajihashemi and Shokri, 2024)
pmrB	F: GCCATTATTCGTCGTGGTTTAAA R: GCGCTCAAAAAGACGGTTCA	150 bp	57°C	45 s	(Abbasi, Hajihashemi and Shokri, 2024)
pmrC	F: CCATTTGGCTAGGTGCAATTT R: CCGCATAATAGGTAGCAACAAG	132 bp	57°C	45 s	(Abbasi, Hajihashemi and Shokri, 2024)
ERIC	F: ATGTAAGCTCCTGGGGATTCAC R: AAGTAAGTGACTGGGGTGAGCG	Variable	53°C	30 s	(Aljindan, Alsamman and Elhadi, 2018)

ERIC: Enterobacterial repetitive intergenic consensus

E. Ethical Statement

The research proposal was submitted to the scientific and ethics committee of the College of Science, and official permission was obtained for sample collection from different hospitals in Sulaymaniyah City. After receiving assurances that their identities would remain anonymous, all patients agreed to participate in the trial.

F. Statistical Analysis

GraphPad Prism software version 10.0 (San Diego, CA, USA) was used to create percentages and frequencies for the variables.

III. RESULTS

The identity of the isolates (n = 46) as *A. baumannii* was collected from hospitalized patients from different ward, intensive care unit (ICU) 26 (56.5%), followed by burn and plastic surgery and outpatient department 12 (26.0%), and 8 (17.39%), respectively, from clinical specimens as follows: 10 (21.73%) for each endotracheal aspirate (ETA), burn, and urine; wound 7 (15.21%); sputum 6 (13.04%); and 1 (2.17) for each blood, synovial fluid, and central venous line. The ages of the patients varied from 12 years to 81 years, with a mean of 46.4 years, comprising 20 males (43.47%) and 26 females (56.52%).

A. Antibiotic Susceptibility Test

The antibiotic susceptibilities of isolates are demonstrated in "Fig. 1." All isolates were classified as MDR 25 (54.3%), XDR 18 (39.1%), and PDR 3 (6.5%). *A. baumannii* isolates have a high resistance rate to CEF, ceftazidime, and P/T (97.8%); LEV and CTR were both 95.6%. In addition to β-lactam medications, the isolates showed high resistance to the majority of antimicrobial medicines, including MER (93.4%), SXT (95.6%), and GEN (86.9%). The three most potent antibiotics were CL (6.5% resistance), TIG (54.4% susceptibility), and AK (82.7% susceptibility). Three isolates (6.5%), which were categorized as PDR, exhibited resistance to CL, with a MIC breakpoint of ≥ 4 mg/L (CLSI).

B. PCR Screening of Antibiotic Resistance Genes

PCR screening analysis was applied to detect genes responsible for class B MBL, OXA-type class D carbapenem hydrolyzing enzyme, and *pmrCAB* genes in *A. baumannii* isolates, like that seen in Fig. 2. The intrinsic β-lactamase *blaOXA-51* gene was found in all isolates, while *blaOXA-23* and *blaOXA-58* were found in 23 (50%) and 21 (45.6%) of isolates, respectively. Among the *MBL* genes, it was demonstrated that 28 (60.8%) were positive for *blaVIM* genes, and 40 (86.9%) of the isolates carried *blaNDM*, even though none of the isolates had *blaIMP*. Concerning CL resistance genes, *pmrA*, *pmrB*, and *pmrC* were detected in 36 (78.2%), 37 (80.4%), and 45 (97.8%) of the samples, respectively, as illustrated in Fig. 3.

C. Genotyping

The ERIC-PCR molecular typing method was applied to classify *A. baumannii* isolates. It formed 12 The typical range of DNA amplicon bands is <100–1500 bp. Clonal variation identified 28 different ERIC patterns (ERIC-type arrangements) categorized into three main phylogenetic classes (C1, C2, and C3), including 9 common types and 19 unique types with a similarity ≥95%. C1 was identified as the dominant class, with 22 (47.8%), including all three PDR isolates belonging to it. C3 18 (39.1%) contains 3 clusters and 11 unique types. While C2, as the smallest class, contains 6 (13.0%) with 1 cluster and 2 single isolates "Fig. 4."

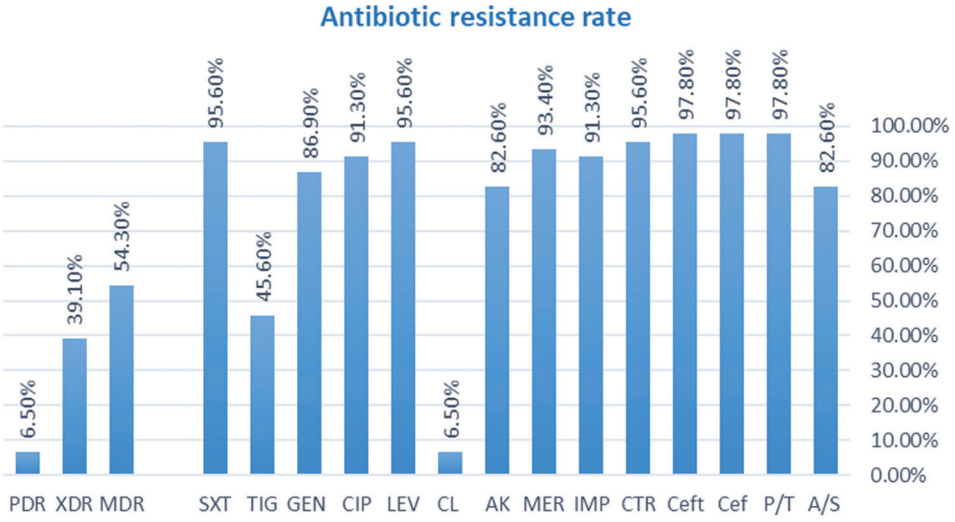


Fig. 1. Antibiotic resistance profiles of *Acinetobacter baumannii* isolates. MDR (54.3%) is Not susceptible to ≥1 agent in ≥3 antibiotic categories; XDR (39.1%) is susceptibility restricted to ≤2 categories; PDR (6.5%) refers to nonsusceptibility to all agents across all types of antimicrobials (Magiorakos, et al., 2012). The antibiotic susceptibility of *A. baumannii* isolates in this investigation. As a whole, the most prevalent resistance antibiotics were CEF, CEFT, and P/T (79.8%). The two most effective antibiotics were TIG (45.6%) and CL (6.5%). A/S: Ampicillin-sulbactam, P/T: Piperacillin/tazobactam, CEFT: Ceftazidime, CEF: Cefepime, MER: Meropenem, IMP: Imipenem, GEN: Gentamicin, AK: Amikacin, CIP: Ciprofloxacin, SXT: Trimethoprim-sulfamethoxazole, TIG: Tigecycline, and CL: Colistin, MDR: Multidrug-resistant, XDR: Extensively drug resistant, PDR: Pan drug resistant.

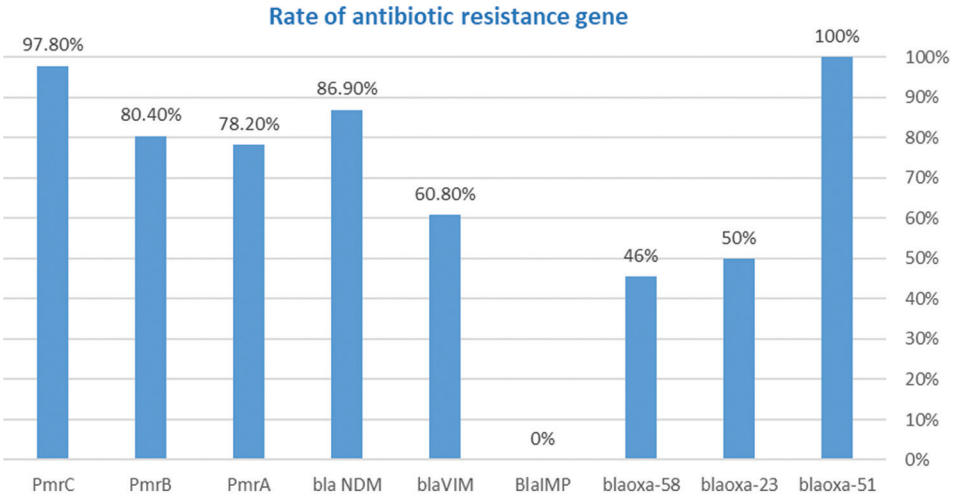


Fig. 2. Observation rate of screening antibiotic-resistant-related genes among *Acinetobacter baumannii* isolates.

IV. Discussion

The emergence of a harmful antibiotic-resistant *A. baumannii* strain is one of the most significant issues facing the healthcare system at that moment. In our investigation, 46 *A. baumannii* isolates were isolated in sputum, ETA, burns, urine, wounds, blood, and cerebrospinal fluid from patients with nosocomial infections, particularly in ICUs and burn and plastic surgery emergencies. Our outcomes show that most isolates obtained were from ETA and sputum, 16 (34.7%); this indicates that respiratory tract infections were the most common types of *A. baumannii* clinical isolates, followed by burn and wound 7 (15.21%) for each type. Our results matched those of other studies (Shayea and Ali, 2022; Smai and Ganjo, 2020; Chawsheen, Al-Naqshbandi and Abdulqader, 2020). Furthermore, our findings are in line with studies that show *A. baumannii* is the most prevalent member that is associated with hospital-acquired infections globally (Abban, et al., 2023; Jiang, et al., 2022). Previously restricted to medical settings, these species are now widespread throughout (Abduljabar and Mawlood, 2023; Shayea and Ali, 2022). Medical devices, including mechanical ventilation, intravascular catheterization, and endotracheal intubation, have all been linked to transmission and infections (Shayea and Ali, 2022; Smai and Ganjo, 2020). *A. baumannii* is particularly capable of surviving for a long time both in the hospital setting, mainly on inanimate surfaces, and in the hands of healthcare workers, which could lead to potential spread and the persistence of endemic bacterial strains in hospitals (Chapartegui-González, et al., 2018). In our region, like other regions of the world,

A. baumannii is one of the bacteria involved in hospital infections leading to several health issues for hospitalized patients.

Clinical isolates of *A. baumannii* were sensitive to common antibiotics, including ampicillin, GEN, chloramphenicol, and nalidixic acid, throughout the 1970s. After that, it became an important nosocomial pathogen, mainly attributed to the use of broad-spectrum antibiotics in hospitals (Towner, 2009). It now exhibits resistance to almost all first-line antibiotics. The only antibiotics that remain effective against MDR *A. baumannii* are CL; nevertheless, CL-resistant strains of *A. baumannii* have been identified in various regions of the world (Abduljabar and Mawlood, 2023; Novović and Jovčić, 2023a). The results of our research indicate *A. baumannii* isolates established resistance to the most commonly administered antimicrobial drugs, including extended-spectrum penicillin, in combination with beta lactamase inhibitors, cephalosporins, aminoglycosides, fluoroquinolones, and carbapenems. The isolates were then classified into three classes based on their antibiotic resistance pattern: MDR 25/46 (54.30%), XDR 18/46 (39.10%), and PDR 3/46 (6.50%). According to our findings**,** this is comparable to recent studies carried out in nearby places in Erbil and Baghdad cities (Abduljabar and Mawlood, 2023; Shayea and Ali, 2022; Al-Kadmy, et al., 2018). This indicates that *endime* possesses many resistance mechanisms, including target alterations, multidrug efflux pumps, permeability defects, and enzymatic drug degradation, which allow the bacteria to successfully evade the effects of the most commonly used antibacterial drugs (Lin and Lan, 2014; Vázquez-López, et al., 2020). Furthermore, our findings highlight the importance and severity of XDR in *A. baumannii*, especially in ICU patients, as the majority of the isolates tested displayed resistance to antibacterial drugs. Carbapenem resistance was high (93%) in our finding; this result shows the elevated likelihood of carbapenem treatment failure in *A. baumannii* infections and is in line with global reports (Müller, et al., 2023; Rezaei, et al., 2018).

Different molecular methods have exposed genotypic assortment to path the prevalence and progression of MDR bacteria, including PFGE, MLST, MLVA, and ERIC-PCR (Khalid, 2024; Falah, Shokoohizadeh and Adabi, 2019; Fattouh and El-Din, 2014). Within *A. baumannii*, ERIC-PCR was utilized in our study. All forty-six *A. baumannii* isolates were typed using ERIC-PCR. Three classes (C1, C2, and C3) with 9 common cluster types of *A. baumannii* isolates with high relatedness or genetic similarity and a high degree of DNA fingerprints among strains of *A. baumannii* (95%) were isolated from the same period and situation, demonstrating that epidemiological connection has an impact on *A. baumannii* strain clustering; this shows cross-transmission occurred among infectious isolates, as shown by this result. The conclusions of this finding agree with those of previous studies (Shayea and Ali, 2022; Aljindan, Alsamman and Elhadi, 2018). Our results indicate that the ERIC-PCR approach is a fast and accurate way to show the clonal relatedness of *A. baumannii* recovered from a spectrum of samples isolated from a variety of patients. As a result, this genotyping method is more quick, suitable, and less expensive than other genotyping approaches.

Genes encoding selected antibiotic resistance (*blaOXA-51*, *blaOXA-23*, *blaOXA-58*, *blaVIM*, *blaNDM*, and *pmrCAB*) were detected using PCR techniques, are shown in "Fig. 3." The results of this study demonstrated that all isolates of *A. baumannii* possess *blaOXA-51* by 100% as an intrinsic gene (Evans, 2009). The existence of blaOXA-51 has limited carbapenemase activity, but when this gene is overexpressed, it displays significant carbapenemase activity. Overexpression can occur through several mechanisms, such as mutations or modifications in regulatory machines. An ISAba1 element

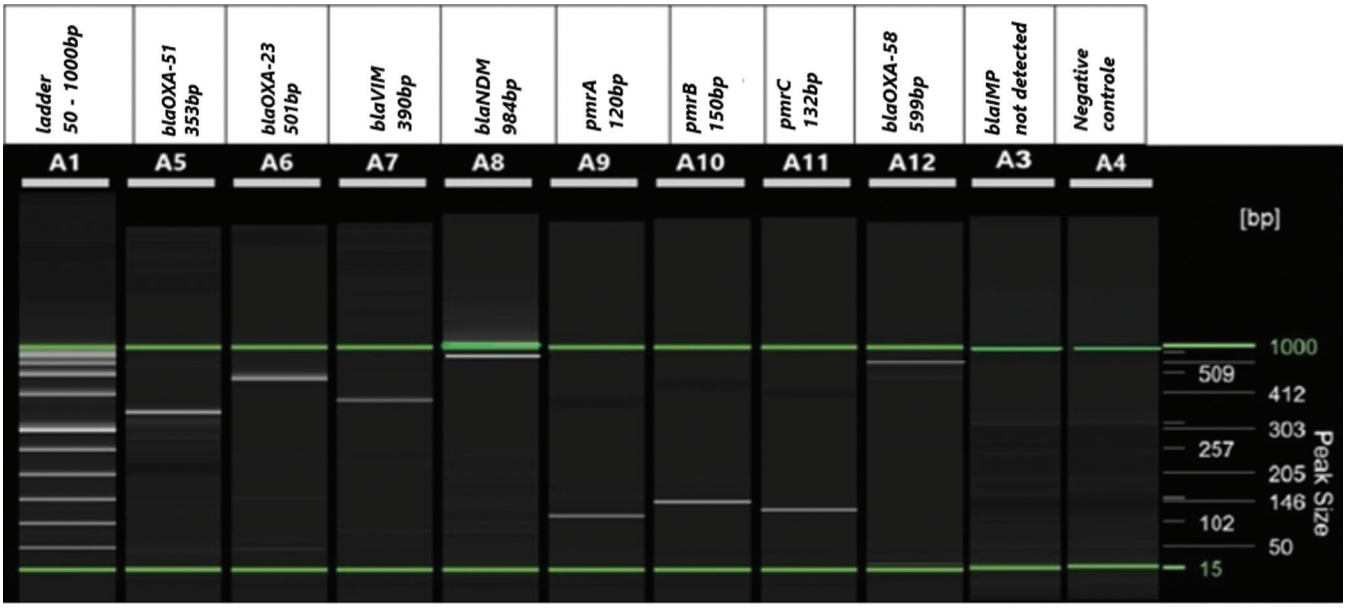
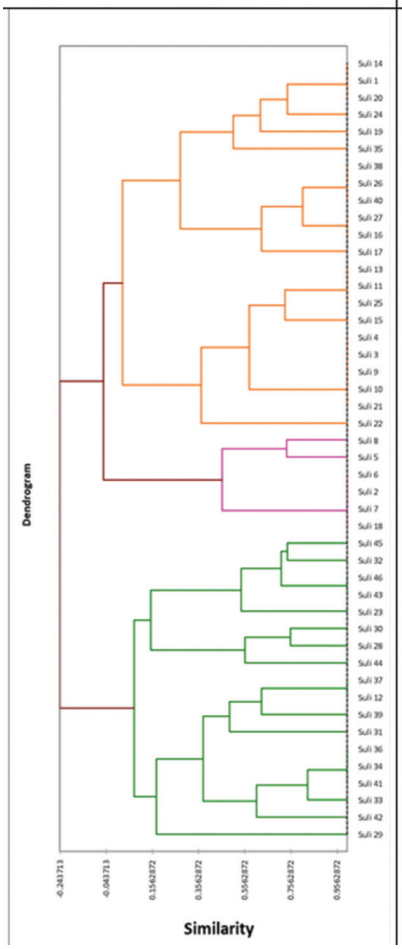
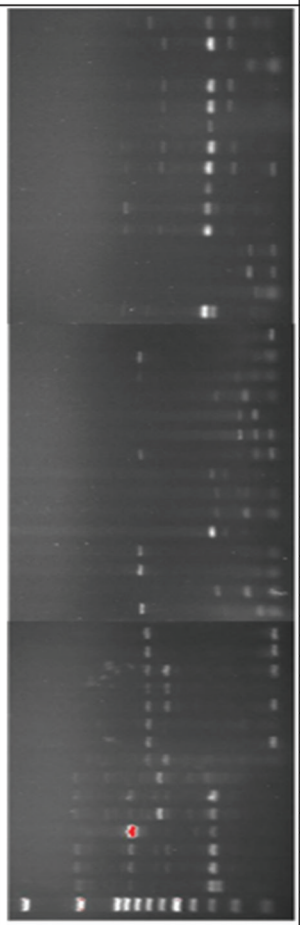


Fig. 3. Capillary gel electrophoresis (QIAGEN, Germany), showing the amplified product of antibiotic resistance-related genes. A1: ladder (50 bp–1000 bp); A5: *blaOXA-51* 353 bp; A6: *blaOXA-23* 501 bp; A7: *blaVIM* 390 bp; A8: *blaNDM* 984 bp; A9: *pmrA* 120 bp; A10: *pmrB* 150 bp; A11: *pmrC* 132 bp; A12: *blaOXA-58* 599 bp; A3: *blaIMP* not detected; A4: negative control.





Isolate	Gen/ Age	Ward/Specimen	Phenotype	Resistance Gene
Suli14	F / 51	ICU / Wound	MDR	blaoxa51, blaVIM, blaNDM, ampC
Suli1	F / 47	ICU/ETA	PDR	blaoxa51, blaoxa23, blaVIM, blaNDM, armA, armB, ampC
Suli20	M / 13	Emergency / Burn	XDR	blaoxa51, blaNDM, armA, armB, ampC
Suli24	M / 22	Emergency / Burn	MDR	blaoxa51, armA, armB, ampC
Suli19	M / 41	ICU / ETA	MDR	blaoxa51, blaNDM armA, armB, ampC
Suli35	M / 65	ICU / sputum	XDR	blaoxa51, blaoxa23, blaVIM, blaNDM, armA, armB, ampC
Suli38	M / 78	ICU / sputum	XDR	blaoxa51, blaoxa23, blaVIM, blaNDM, armA, armB, ampC
Suli26	M / 17	ICU / urine	MDR	blaoxa51, blaNDM, armA, armB, ampC
Suli40	F / 81	ICU / CV line	MDR	blaoxa51, blaoxa23, armA, armB, ampC
Suli27	F / 54	ICU/ ETA	XDR	blaoxa51, blaNDM, armA, armB, ampC
Suli16	F / 61	ICU / ETA	MDR	blaoxa51, blaNDM, armA, armB, ampC
Suli17	F / 58	ICU / Urine	PDR	blaoxa51, blaNDM, armA, armB, ampC
Suli13	F / 41	Emergency / Burn	MDR	blaoxa51, blaoxa23, blaVIM, blaNDM, ampC
Suli11	F / 61	ICU / ETA	MDR	blaoxa51, blaoxa23, blaVIM, blaNDM, ampC
Suli25	F / 36	OPD / Urine	MDR	blaoxa51, blaoxa23, blaVIM, blaNDM, armA, armB, ampC
Suli15	M / 51	ICU/ ETA	MDR	blaoxa51, blaVIM, blaNDM, ampC
Suli4	F / 39	ICU / ETA	MDR	blaoxa51, blaoxa23, blaVIM, blaNDM, armA, armB, ampC
Suli3	M / 63	ICU/ ETA	XDR	blaoxa51, blaoxa23, blaVIM, blaNDM, armA, armB, ampC
Suli9	M / 67	ICU / ETA	MDR	blaoxa51, blaNDM armB, , ampC
Suli10	M / 35	Emergency / Burn	PDR	blaoxa51, blaoxa23, blaVIM, blaNDM, armA, armB, ampC
Suli21	M / 61	ICU / Urine	MDR	blaoxa51, blaNDM, armA, armB, ampC
Suli22	M / 35	Emergency / Burn	MDR	blaoxa51, blaNDM, armA, armB, ampC
Suli8	F / 53	ICU/ETA	MDR	blaoxa51, blaoxa23, blaNDM, ampC
Suli5	F / 45	ICU / Wound	MDR	blaoxa51, blaoxa23, blaNDM, ampC
Suli6	F / 12	Emergency / Burn	XDR	blaoxa51, blaNDM, ampC
Suli2	F / 54	Internal / Blood	MDR	blaoxa51, armA, armB, ampC
Suli7	M/ 24	ICU/ Blood	MDR	blaoxa51, blaNDM, ampC
Suli18	F / 23	Emergency / Burn	MDR	blaoxa51, blaNDM, armA, armB, ampC
Suli45	F / 31	OPD / Urine	XDR	blaoxa51, blaVIM, blaNDM, armA, armB, ampC
Suli32	M / 70	ICU / Urine	XDR	blaoxa51, blaoxa23, blaVIM, blaNDM, armA, armB, ampC
Suli46	M/ 43	OPD / Sputum	XDR	blaoxa51, blaoxa23, blaVIM, blaNDM, armA, armB, ampC
Suli43	F / 65	Emergency / Burn	XDR	blaoxa51, blaVIM, blaNDM, armA, armB, ampC
Suli23	F / 24	Emergency / Burn	MDR	blaoxa51, blaoxa23, armA, armB, ampC
Suli30	F / 19	OPD/ Urine	MDR	blaoxa51, blaoxa23, blaVIM, armA, armB, ampC
Suli28	F / 21	OPD / wound	MDR	blaoxa51, blaoxa23, blaVIM, blaNDM, armA, armB, ampC
Suli44	M / 55	Emergency / burn	XDR	blaoxa51, blaVIM, blaNDM, armA, armB, ampC
Suli37	F / 63	OPD / Wound	XDR	blaoxa51, blaoxa23, blaVIM, blaNDM, armA, armB, ampC
Suli12	F / 54	Internal / Wound	MDR	blaoxa51, blaVIM, blaNDM, armA, armB, ampC
Suli39	M / 59	ICU / Urine	XDR	blaoxa51, blaoxa23, blaVIM, blaNDM, armA, armB, ampC
Suli31	F / 43	ICU/Synovial Fluid	XDR	blaoxa51, blaoxa23, blaVIM, blaNDM, armA, armB, ampC
Suli36	M / 69	ICU / Sputum	XDR	blaoxa51, blaoxa23, blaVIM, blaNDM, armA, armB, ampC
Suli34	F / 73	ICU / Sputum	XDR	blaoxa51, blaoxa23, blaVIM, blaNDM, armA, armB, ampC
Suli41	F / 30	OPD /Urine	XDR	blaoxa51, blaoxa23, blaVIM, blaNDM, armA, armB, ampC
Suli33	M / 73	ICU / Sputum	XDR	blaoxa51, blaVIM, blaNDM, armA, armB, ampC
Suli42	F / 42	Emergency / Burn	XDR	blaoxa51, blaVIM, blaNDM, armA, armB, ampC
Suli29	M / 17	OPD / Urine	MDR	blaoxa51, blaNDM, blaPmrA, armB, ampC
Ladder	Ladder 100bp DNA ladder (100 – 3,000)			

Fig. 4. Dendrogram of enterobacterial repetitive intergenic consensus polymerase chain reaction analysis for 46 *Acinetobacter baumannii* isolates from different clinical samples.

inserted upstream of the *blaOXA-51* gene, which acts as a strong promoter, drives the overexpression of the gene and thereby confers carbapenem resistance (Takebayashi, et al., 2021; Yazdansetad, et al., 2019). Therefore, other acquired oxacillinases, including *blaOXA-23*, *blaOXA-24,* and *blaOXA-143* enzymes, were necessary for *A. baumannii* to be resistant to carbapenem (Abd El-Baky, et al., 2020). Overall, *blaOXA-23* was found in 23 (50%) and *blaOXA-58* 21 (45.6%) of isolates. These results are similar to research that found among *A. baumannii* isolates, OXA-type carbapenemases were more abundant (Abduljabar and Mawlood, 2023). Furthermore, our findings are in line with this result; a high prevalence of bla_{OXA-23} carrying *A. baumannii* isolates has been described in West Asian patients (Joshi, et al., 2017). This could be the result of conjugation between *A. baumannii* and the *blaOXA-23* gene found on the plasmid. Consequently, the number of antibiotic-resistant bacteria has been raising quickly worldwide (Bertini, et al., 2010). Moreover, compared to the findings of this analysis, an applied study including 196 clinical isolates revealed that 48.4% were *blaOXA-51*, 46.3% were *blaOXA-23*, and 5.3% were *blaOXA-58* (Hou and Yang, 2015).

In addition to OXA-type oxacillinases, the *MBL* genes were screened in the present research. Recently, the *blaNDM* harboring *A. baumannii* has been detected in a number of nations in Europe, South America, and Asia (Rodrigues, et al., 2024; Joshi, et al., 2017; Nordmann, et al., 2011). There is evidence that the *blaNDM* gene is a chimera gene assembled by the combination of the mannose-binding lectin gene with an aminoglycoside resistance gene, aphA6. This occurrence most likely takes place in *Acinetobacter* spp., meaning that this gene most likely originated in these bacteria (Nordmann, et al., 2011). In the present study, we identified a high prevalence of 40 (86.9%) of the isolates carrying the bla_{NDM} gene, which is greater than the previous study in Erbil city, which identified 26% of the *A. baumannii* harboring the same gene (Abduljabar and Mawlood, 2023). Furthermore, it was higher than a study performed in West Asia, 13.6% (Joshi, et al., 2017). These findings suggested the possibility of plasmid-mediated transmission of the Bla_{NDM} gene from one strain to another and that plasmids are essential to the pandrug-resistant *A. baumannii* epidemic (Shi, et al., 2024). Regarding *blaVIM* our study identified that 28 (60.8%) isolates were positive for this gene, which is in line with other reports directed previously (Khalid, 2024; Abduljabar and Mawlood, 2023). In contrast to *blaVIM and blaNDM, blaIMP* was not detected in any of the *A. baumannii* isolates. Our finding was agreed with by another study, which discovered that the *A. baumannii* isolates do not have the bla_{IMP} gene

in their genome. Similar to our results, a study also reported that the finding of the same gene in *A. baumannii* isolates was 0.0% (Al-Hindawi, 2018). Moreover in line with other data, there was no detectable *blaIMP* gene in carbapenem-resistant isolates (Fattouh and El-Din, 2014).

According to our results, the lowest rate of resistance was given to CL (6.5%), which belonged to the PDR category *A. baumannii* with MIC values ≥4 μg/mL. The most effective antibiotics for *A. baumannii* infections are still CL when compared to other tested antibiotics. These results are consistent with research indicating that around 7% of isolates of *A. baumannii* are CL-resistant (Abbasi, Hajihashemi and Shokri, 2024; Behera, Swain and Chandra, 2017). The progress of CL resistance in *A. baumannii* is essentially influenced by *pmrCAB* genes. The resistance of *A. baumannii* to CL is believed to be due to an assortment of various mechanisms. The most common of these is the modification of the lipid A component of the outer membrane's LPS layer, which is essentially rendered achievable by the *pmrCAB* operon (Novović and Jovčić, 2023b). The addition of pEtN to lipid A is triggered by the *pmrC* gene; *pmrC* expression is regulated by the *pmrA* and *pmrB* (*pmrAB*) TCS. As a result, the outer membrane's negative charge reduces, which affects CL binding and protects the cell membrane's stability and structure intact (Moffatt, Harper and Boyce, 2019; Chen and Groisman, 2013). Here, we identified all three CoR *A. baumannii* isolates harboring the *pmrCAB* genes. Comprehending the function of *pmrCAB* genes in CL-resistant *A. baumannii* is essential for creating new approaches to fight antibiotic resistance (Al-Shamiri, et al., 2021).

Variation in the prevalence of distinct OXA-type, MBL, and CL resistance genes in different nations may be related to a variety of antibiotic therapy programs, ecological states, and variant antibiotype patterns (Azimi, et al., 2015). Our study has identified numerous intrinsic and acquired *OXA*-type, *MBL*, and *CoR* genes that co-exist. The fact that 46/46 (100%) of all isolates have multiple resistance genes indicates that an increase in the combination of these genes may lead to a rise in antibiotic resistance in *A. baumannii* isolates (Shi, et al., 2024). Variation in antibiotic resistance gene detection among different investigations could be attributed to several factors, including that bacterial antibiotic resistance is often acquired and disseminated through the movement of mobile genetic elements, such as conjugative plasmids, insertion sequences, transposons, and integrons, as well as the size and methodology of the study (Noel, Petrey and Palmer, 2022). Furthermore, widespread resistance to most classes of antibiotics among the population may be due to their common use (Radhi and Al-Charrakh, 2019). These outcomes show that the resistance of *A. baumannii* is ever-evolving. Therefore, in order to avoid infections with these bacteria in hospital settings, it is imperative to establish a suitable treatment plan and a precise methodology.

V. Conclusion

The present research discovered a considerable level of MDR in *A. baumannii* isolates in our study, with a high prevalence of OXA-type, BML, and *pmrCAB* genes, which have been identified as important antibiotic resistance genes among isolates. The results also indicate that ERIC-PCR could provide a quick and reliable method to identify clonal similarities between isolates from a variety of clinical samples. The findings also advise monitoring the incidence of clinical isolates of *A. baumannii*, which hence requires the use of molecular and genotyping methods. Consequently, controlling the endemic strains of *A. baumannii* is required, especially in ICUs and burn hospitals, and effective strategies to stop managing the infection's spread are urgently required.

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