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Research Article

Identification of class I, II, and III Integron genes in multidrug-resistant *Acinetobacter baumannii* strains

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Abstract
Integrons are one genetic factor that can contribute to the high prevalence of antibiotic resistance among *Acinetobacter baumannii* isolates and their spread. This research aimed to study the prevalence of class 1,2 and 3 integron in *A. baumannii* isolates and their relatedness to virulence factors of antibiotics resistance. Seventy clinical isolates of *A.baumannii* were isolated from several sources such as blood, urinary tract infection (UTI), sputum, urine, and cerebrospinal fluid (CSF). First, the isolates were identified and characterized according to certain morphology, cultural and biochemical tests. Second genotypic ally Identification of the isolates was confirmed by Polymerase chain reaction (PCR), which was performed by housekeeping gene *16sRNA* and by (*blaOxa 51*gene) for *A. baumannii* species. The antimicrobial susceptibility technique was evaluated through disk diffusion methods. These isolates resistant to various antibiotics were analyzed for integron class I, II and III content and sequences of the amplification products by PCR. The findings showed that the predominant *A. baumannii* isolates were multidrug resistant and they were most resistant to 15 antibiotics. The higher resistance of *A. baumannii* to Gatifloxacin and lower resistance to polymyxin. Sixty clinical Multidrug resistance (MDR) isolates of *A. baumannii* had class I integron and five class III integron, but class II integron was not detected in the isolates. This revealed that the dissemination of MDR among *A. baumannii* may be associated with the presence of integrons class I and class III. These data indicate that integrons are gene cassettes containing antibiotic-resistance genes that play a major role in the virulence characteristics of MDR and Extensively-drug resistance (XDR) in *Acinetobacter baumannii.*

Keywords: Multidrug resistance (MDR), Extensively-drug resistance (XDR), Pan–drug resistance, *Acinetobacter baumannii,* Integron class 1 ,2 and 3

INTRODUCTION

Gram-negative *Acinetobacter baumannii* is a major opportunistic nosocomial pathogen that primarily affects critically ill patients who are being treated in intensive care units for conditions such as pneumonia, urinary tract infections, bacteremia, and meningitis; in these patients, it is associated with a significantly increased risk of death due to *Acinetobacter baumannii* infection. (Theaker *et al.,* 2003; Yu, G *et al.* (2013). The vast majority of *A. baumannii* infections are brought on by outbreak strains, which have the potential to quickly and widely spread amongst patients (Anderson *et al.,* 2020). Antibiotics are widely applied in the prevention as well as treatment of infections in both human beings and animals. Antibiotic overuse and improper medication practices can both contribute to the development of antibiotic resistance in bacterial strains, which makes it more difficult to treat bacterial infections (Hao *et al.,* 2016; Salaheen *et al.,* 2019). The development of multidrug resistance in gram-negative bacteria, particularly *Acinetobacter baumannii*, has emerged as one of the most significant threats to both human and animal health (Hao *et al.,* 2016; Hou *et al.,* 2015; Aslam *et al.,* 2018).

Multidrug resistance (MDR) *Acinetobacter spp*. refer to an isolate of the genus *Acinetobacter* that is resistant to at least three different classes of antimicrobial agents. These classes include fluoroquinolones, aminoglycosides, penicillins, and cephalosporins. Extensively-drug resistance (XDR) *Acinetobacter spp*. refers to an isolate of *Acinetobacter spp*. resistant to carbapenem in the three antibiotic classes described above (MDR). The XDR Acinetobacter spp. strains resistant to poly-

myxins and tigecycline must be referred to as Pan drug resistance Acinetobacter spp. The aforementioned criteria were written with the currently understood and considered resistance mechanisms and the many *Acinetobacter spp*. infections being treated with antimicrobials Bolton *et al.,* (2018). These definitions aid in further clarifying the degree of resistance and appropriate antimicrobial therapy (Aslam *et al.,* 2018). These gram-negative bacteria have a large capability for acquiring resistance genes, most commonly through horizontal gene transfer, as a result of antimicrobial resistance (AMR), which is caused by both intrinsic and acquired resistance mechanisms. AMR is a result of these bacteria's ability to fend off antimicrobials. The fast spread of antibiotic-resistance genes between (Gram-negative species (GNB species) is facilitated in large part by a technique known as horizontal gene transfer (Chamosa *et al.,* 2017; Leungtongkam *et al.,* 2018).

Various mechanisms for acquired resistance genes, including bacteriophages, transposons, plasmids, and integrons, are involved in bacteria's diffusion of resistance genes. This has been the case because these acquired resistance mechanisms can pass resistance genes on to other bacteria (Domingues *et al.,* 2012). These mechanisms can be found in bacteria. Integrons are one type of genetic factor that has the potential to contribute to the widespread prevalence as well as the spread of antibiotic resistance (Sallman and Ali, 2018; Ghaly *et al.,* 2019). These elements can seize, integrate, and then mobilize antibiotic-resistant gene cassettes in their DNA. Based on the degree of genetic similarity between each integron's integrase intI gene sequence, the integrons were organized into three significant classes: 1, 2, and 3, respectively. Class 1 integrons are the most common, and they may be found in GNB (Jones-Dias *et al.,* 2016). Because these strains are also resistant to multiple antibiotics, it has been hypothesized that the existence of integrons may be linked to the epidemic potential of *A. baumannii* among its many isolates. Antibiotic resistance genes have been discovered on integrons in several species of *Acinetobacter*, according to several investigations (Ridha *et al.,*2019 Ali and Majeed (2021). Since integrons have an integrase gene (a site-specific recombinase) at their 5' end, Koeleman *et al.* (2001) hypothesized that PCR detection of this may be utilized as a straightforward approach to distinguish between different strains of *A. baumannii*. Their findings were certainly the case, with three-quarters of the epidemic isolates yielding positive results. This provided evidence that the hypothesis was correct. This study aimed to detect drug resistance and its presence by disseminating three classes of integrons in *A. baumannii* clinical isolates recovered from hospitalized patients in intensive care units.

MATERIALS AND METHODS

Bacterial isolates

Between January to April 2022, a total of 70 non-repetitive *A. baumnnii* isolates were obtained from 250 clinical specimens such as cerebrospinal fluid (CSF), blood, sputum and urine from patients (ranging in age from 3 to 65 years old) were hospitalized in Intensive care units (ICUs) of 3 medical centers in different regions of Iraq, Bagdad . The samples selection (sputum, urine, CSF, blood) was not random but based on whether the patients suffered problems in respiratory, urinary, or nervous problems. After multiple laboratory tests, *A. baumnnii* bacteria were detected. In addition to this, the sample size had an important effect, not less than 3 ml.

Identification of *Acinetobacter* species is complicated due to a lack of standard identification techniques. Final identification for all isolates was done at three levels. The primary identification was conducted according to morphology under the light microscope and cultural on Blood agar, CHROM agar, MacConkey agar, Kligler iron agar and Cetrimide agar plates in aerobic conditions at 37°C for 24h and biochemical test. The Genetically identification of *A.baumannii* using PCR technique for all isolates has been done by housekeeping gene *16sRNA* to detect genus of *Acinetobacter* and $blaOxa_{51}$ for detection of the species of *Acinetobacter baumannii.* All seventy isolates gave positive results for $blaOxa_{51}$, on CHROM agar showed light purple with a halo around the colonies showed after 24h. at 37°C, indicating that the isolates were *A. baumannii* and not another species of this bacteria. According to clinical source, the high percentage of bacteria were isolated

Table 1. Number and percentage of *Acinetobacter baumannii* isolates according to specimen's type

Source	Total number of isolates	No. of positive A. baumannii Isolates	Percentage (%) A. baumannii Isolates
Sputum	27	24	28.23
Blood	25	22	25.88
Urine	23	21	24.70
CSF	10	3	3.52
Total	85	70	82.34
Chi-Square	8.31	16.28	
P value	0.039*	0.001**	

* Significant at < 0.05 ** Significant at < 0.01

from sputum and lower percentage from CSF specimens. Table 1 shows that there was a significant value showing a real infection of *A.baumannii*. A among the infected patients whose ages ranged from 15 to 65 years.

Standard disc diffusion technique

An antibiotic susceptibility test was conducted on *A.baumannii* isolates using a disc diffusion test of 15 antibiotics in different concentrations. All antibiotics were classified into three class : Carbapenem class : Meropenem (MEM) 10 mg, Imipenem (IMP) 10 mg and Fluro -quinolone, which included: Ciprofloxacin (CIP) 10 mg and Levofloxacin (LEV) 5 mg, Aminoglycoside : Gentamicin (CN) 10 mg, and Beta-lactam class: Cefotaxime (ctx) 30 mg, Tigecycline (TGC) 15 mg, Polymyxin (PB) 100 mg, Colistin sulfate (CS) 10 mg , Amoxicillin /clavulanic acid (AMC) 20/10 mg, Ceftriaxone (CRO) 30 mg, Doripenem DOR 10 mg, Amikacin AK 30 mg, Minocycline TE 30 mg and Gatifloxacine GET 5 mg. Interpretation of antibiotic susceptibility to evaluate MDR , XDR and PDR isolates was performed based on the European Centre for Disease Prevention and Control (ECDC) instructor as well as Ridha and Jassim (2019).

Genetic relatedness and dendrogram construction

All of the isolates' dendrograms from cluster analyses were evaluated. The pattern of antibiotic resistance served as the foundation for creating the dendrogram. Each strain's binary table or haplotype matrix was created by linearly composing The program was used to conduct a statistical analysis of the resistance (1)/ sensitive (0) data obtained from examining the antibiogram sensitivity.

Preparation of bacterial DNA

Briefly, DNA templates were extracted by boiling method; a few isolates overnight growth colonies of bacteria were dissolved in 0.5ml sterile distilled water and boiled in a water bath for 10 minutes, then centrifuged at 10000 rpm for another 10 minutes; the suspensions were taken as a template (Zaki *et al.,* 2018).

Polymerase chain reaction amplification procedure

The identification (housekeeping genes of *A.baumannii*) and Primers used for the detection of Integron class I, II, and III genes were performed by PCR amplification technics' as: displayed in Table (1) and PCR amplification program in Table (2).The detection PCR products was performed on 1% agarose gels by electrophoresis and visualized under UV light.

RESULTS AND DISCUSSION

Isolation and identification

There were 70 clinical isolates from 250 clinical specimens, primarily diagnosed as Gram-negative non-fermenters *Acinetobacter spp.* obtained from three hospitals (The City of Medicine, Al-Hurok Hospital, Baghdad Hospital) in Baghdad. The primary diagnoses were distributed according to sources of isolation: 50% sputum, 5% blood, 10% urine, and 5% CSF.The isolates were initially diagnosed using a combination of bacteriological and biochemical tests. Based on these tests, it was ascertained that there was a difference between the results of isolation from the hospitals. The chemical and agricultural tests have been synchronized with the

Table 2. Primers used in the present study for integron gene detection

Gene	Primer name	(5'-3') Sequence	Size product	Reference
Housekeeping gene of *A. baumannii*	$blaOxa_{51}$	F-TAATGCTTTGATCGGCT R-TGGATTGCATTCATCTTGG	353bp	(Hou and Yang 2015)
Intrgron genes	Class 1	F- CAGTGGACATAAGCCTGTTC R- CCCGAGGCATAGACTGTA	160 bp	Gundogdu *et al.,* (2011)
	Class 2	F- CACGGATATGCGACAAAAAGGT R- GATGACAACGAGTGACGAAATG	788 bp	
	Class 3	F- GCCTCCGGCAGCGACTTTCAG R- ACGGATCTGCCAAACCTGACT	979 bp	Gundogdu *et al.,* (2011)

Table 3. PCR Reaction program for integrons and housekeeping genes

Genes	Initial denaturation	No. of cycles	Denaturation	Primer annealing	Primer extension	Final extension
bla_{OXA51}	95 ̊c 5m	35	94 ̊c 30sec	52 ̊c 30min	72̊c 1m	72 ̊C 10m
Int 1	95 ̊C 5m	35	94 ̊C 30sec	60 ̊C 30sec	72 ̊C 1m	72 ̊C 6m
Int 2	95 ̊C 5m	30	94 ̊C 30sec	60 ̊C 30 sec	72 ̊C 1m	72 ̊C 6m
Int 3	95 ̊C 5m	35	94 ̊C 30sec	55 ̊C 30 sec	72 ̊C 1m	72 ̊C 6m

genetic diagnoses, which confirms that they give the final detection rather than laboratory culture and biochemical tests when a sample is obtained from patients. The identification and characterization of the isolates were carried out according to certain morphology as Gram-negative diplococci, cultural on CHROM agar Blood agar non-hemolysis, MacConkey agar non-lactose fermenter pale color, Kligler iron agar and Cetrimide agar plates in aerobic conditions at 37°C for 24h. and biochemical test(positive and negative for catalase and oxidase test) . The genetic identification of *A.baumannii* for all isolates was done by housekeeping gene *16sRNA* to detect the genus of *Acinetobacter* and *blaOxa51* for detection of the species of *Acinetobacter baumannii* using PCR technique. PCR assay had 100% specificity and sensitivity for their intended targets (Fig. 1).
According to clinical sources, a high percentage of bacteria were isolated from sputum (10.8%) n=27) and from blood specimens (9.2 %) n=23); urine specimens constituted 8.8% (n= 22 ; and CSF specimens (1.2%)n= 3).

Antibiotic sensitivity test

Standerd disk diffusion (Antibiotype)

Data submitted in Fig. 2 display a high resistance of clinical isolates to the greatest number of antibiotics. The current study revealed the highest resistance of *A.baumannii* to gatifloxacine GET (n=69,98.57%), cefotaxime (ctx) (n=68,97.13%), amoxicillin/clavulanic acid (AMC) (n=68,97.13%), ceftriaxone (CRO) (n=68,97.13%), amikacin AK (n=67,95.71%),doripenem DOR (n=67,95.71%), minocycline(95%) ,meropenem (MEM) (n=66,94.27%), colistin sulfate (CS) (n=66,94.27%) .This value was higher than other research, ceftriaxone (79%), cefotaxime (74.3%), minocycline(69.5%), amoxicillin /clavulanic acid (AMC) (61.9%), doripenem(60%), and ceftazidime (55.2%) were observed by Ridha *et al,* (2019) for *A.bauamnnii* isolates, sputum specimen from the respiratory system, mid resistance to imipenem (IMP) (n=61,87.14%), gen-

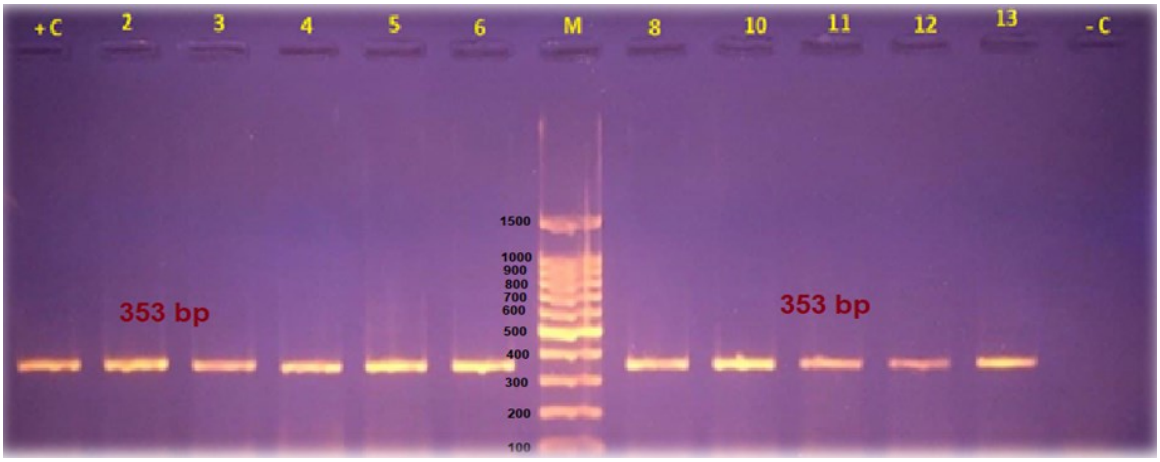

Fig. 1. *blaOXA-51 gene product size (band 353 bp) detected using agarose gel electrophoresis (1% agarose, 7v/cm^2) with Ethidium bromide staining. Utilizing the template DNA prepared using the boiling method, Showing molecular size of the DNA ladder as 100 bp, located in the middle (M). DNA isolated from A. baumannii samples showing positive PCR, with a positive control on the left (lene + C) and negative control on the right (lene – C).*

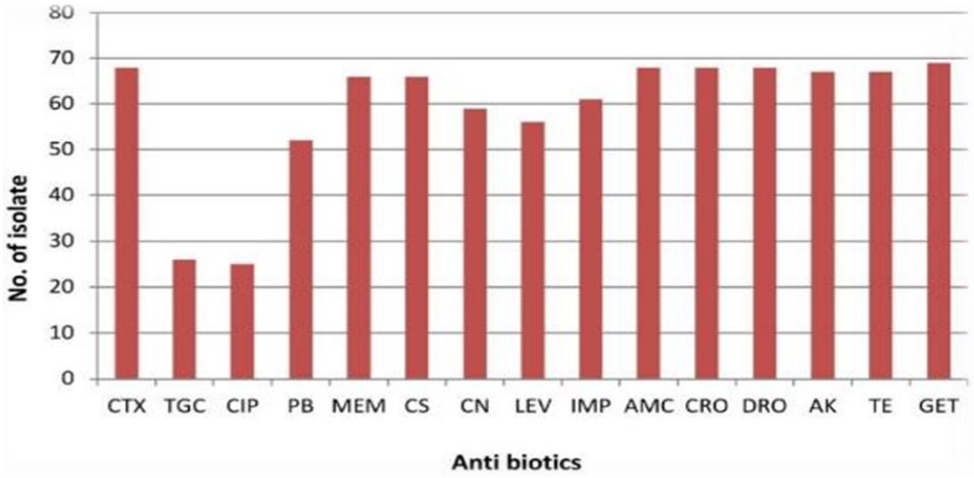

Fig. 2. *Antibiotic resistance of Acinetobacter baumannii isolates*

tamicin (CN) (n=59.84.28%), levofloxacin (LEV) (n=56,80%) and polymyxin (PB) n=(n=52,74.3%). While most *A.baumanni* isolates were sensitive to ciprofloxacin (n=25,35.71%) and tigecycline (TGC) (n=26,37.21%). In contrast, Both ciprofloxacin and tigecycline were the most effective antibiotics against *A. baumannii*. PCR was used to check resistant isolates for the presence of class I integron genes. The isolates had to be resistant to at least one antimicrobial agent.

All *Acinetobacter baumannii* isolates were subjected to hierarchical cluster analysis (Fig. 3). The dendrogram was based on dice coefficient values, and there were two major clusters when the dendrogram was generated based on their antimicrobial sensitivity against 15 antibiotics (Fig. 3). Among antibiogram patterns of the 70 *Acinetobacter baumannii* isolates of different origins, Uniplex resistance patterns A was classified into two clusters and among A group, the isolates clusters into six sub groups; A1 contained 3 isolates (53,54.18) and A2 sub group noticed 46 (4,2 ….28), A3 sub group noticed 8(57,9,7…23), A4 sub group noticed 7 isolates (30,39…52), A5 sub group noticed 2(67,68) isolates, and A6 noticed 2(66,58) isolates while final isolate was unique clone (51).

Subgroups A5 and A6 have been characterized as resistance patterns to antimicrobial class, especially aminoglycoside, fluoroquinolone and beta-lactam class.Further to that, the unique clone 51 number isolate was characterized as resistant to all used classe. Antimicrobial class included aminoglycoside, fluoroquinolone, beta-lactam and carbapenem class due to the prescience gene cassette integron I and III encoding the important gene responsible for this resistance. The results agree with Kadhim and Mun'im (2014), who showed that molecular typing for identifying clonal relationships between isolates has been quite frequent for simplicity and efficiency.

Among 70 clinical isolates of *A. baumannii*, 95.71% isolates have been MDR especially were isolated from sputum and blood such as (1S,2S,3S,4S,5S,6S,10S,15S, 42b,50b and 60b) and 92.85%. In general, antimicrobial-inactivating enzymes, reduced outer membrane permeability caused by the loss or reduced production of porins, overexpression of multidrug efflux pumps, and mutations that affect targets or cellular activities are the mechanisms of antimicrobial resistance in *A. baumannii* (alterations in penicillin-binding proteins; PBPs) (Ridha *et al.,* 2019; Ali *et al.,* 2019; Fernández-Cuenca *et al.,* 2003). It was noted that other gram-negative bacteria, such as *A. baumannii*, also had a combination of many processes, suggesting that this may also be the case with the microbe in issue.

Detection of integron genes

Amplification of an internal portion of the integrase genes was used to determine the presence of integrons in 85 % (60) of *A. baumannii* isolates, demonstrating that these elements are widely distributed among multiresistant isolates of this species. 60 of the 70 clinical isolates were positive for an intI1 gene. The intI II gene could not be located, and the intI III gene was the only isolated one. Previous research has shown that there was a high incidence of multi-resistant gram-negative isolates that included integrons, and class 1 integrons were discovered in 68 % of all worldwide nosocomial isolates of *A. baumannii* (Sunde *et al.,* 2005)

PCR was used to screen for integrons using three different sets of primers designed to be unique to the intI1, intI2, and intI3 genes. Integrons were then mapped using primers that were complementary to conserved sequences. When the 3' conserved section was absent, the cassettes were cloned with antibiotic resistance as the selection marker (Fig.4 A,B,C).

In recent years, there has been an increase in the prevalence of antimicrobial resistance in clinical isolates, which poses a serious threat to public health (Ridha

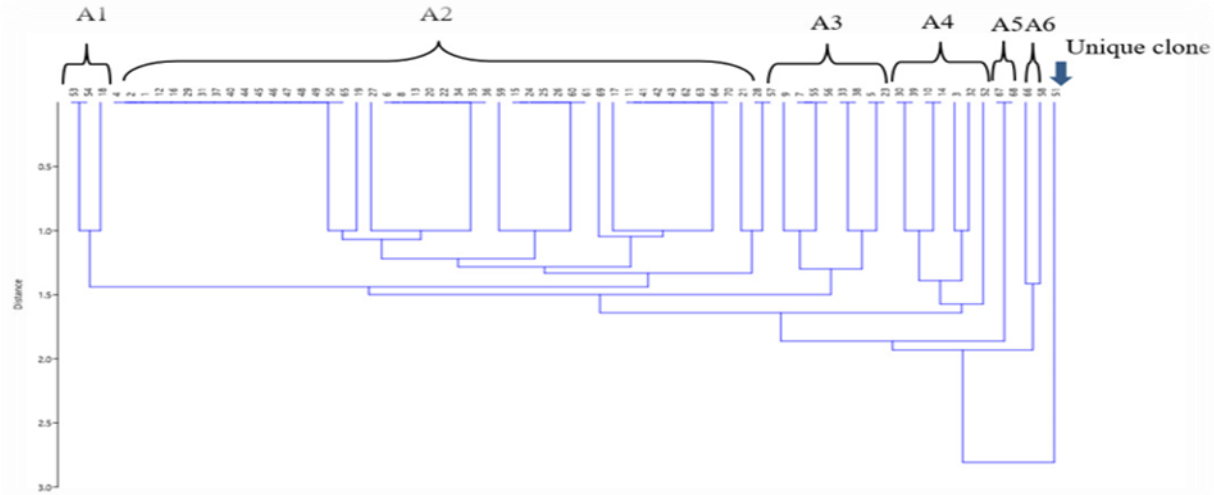

Fig. 3. *Dendrogram obtained from antibiogram data for Acinetobacter spp. included A. baumannii*

and Jassim 2019). Because clinical isolates have likely been subjected to much stronger antibiotic evolutionary changes or antimicrobials with a wider spectrum, they are considered the primary focus (Sunde *et al.*, 2005). Nevertheless, studies examining the antibiotic resistance and integron prevalence of non-pathogenic commensal bacteria are uncommon.

Integron role in antibiotic resistance

Gene cassettes that code for resistance to antibiotics of newer generations have been classified as belonging to class I and III integrons. Therefore, the presence of extended-spectrum beta-lactamases (ESBLs) and metallo -beta-lactamases (MBLs) that hydrolyze third and fourth -generation cephalosporins and carbapenems leads to resistance to these classes of antibiotics. This conclusion is based on the structure of class 1 integron gene cassettes (Veena, 2019). This wass because these enzymes hydrolyze the cephalosporins and carbapenems. Integrons, being mobile elements, can transmit and carry the resistance genes from one organism to another; this challenge is highly relevant in the livestock and poultry industries because the efficacy of infection control programs is low (Fletcher, 2015; Ridha *et al.,* 2019). Nevertheless, it would appear that many different factors have more significant effects on the frequency of the class 1 integron.

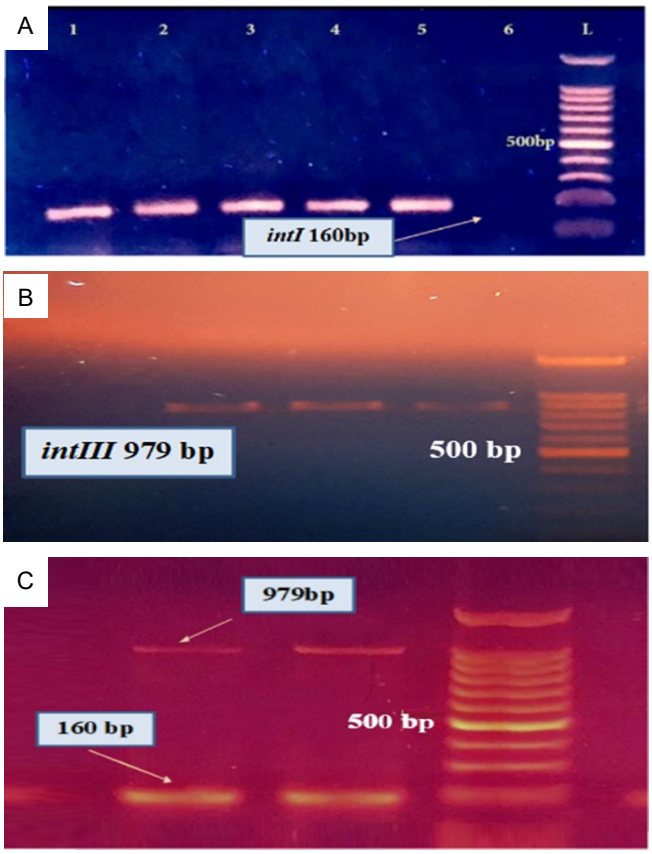

Fig. 4. *Agarose gel Electrophoresis 1% agarose to detect: A- intI (amplified size 160 bp,) B- int 3(amplified size 979bp) C- multiplex intland int 3 of Acinetobacter baumannii by using 100 bp (Promega) ladder.*

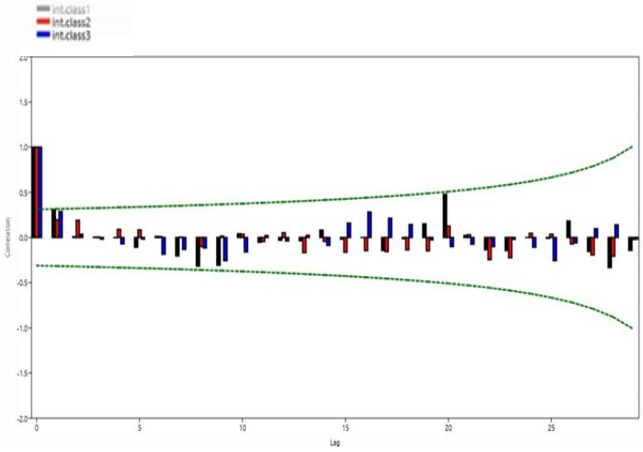

Fig. 5. *Dendrogram obtained from antibiogram data for Acinetobacter spp. included Acinetobacter baumannii*

Fig. 5 dendrogram explains the strong correlation and relationship between the presence of integron class I gene cassette and Antimicrobial resistance MDR and XDR. These elements can capture, integrate, and then mobilize antibiotic-resistant gene cassettes. Table 3 shows that among 60 isolates may have integron I and resistance to antibiotics (59 resist to CTX, 19 to TGC,30 to CIP,42 to PB, 59 to MEM,56 to CS, 51 to CN,47 to LEV,53 to IMP,58 to AMC, 59 to CRO, 58 to DRO, 57 to AK, 57 to TE,59 to GET). While some clinical isolates had integron I but were sensitive to antibodies (1 to CTX, 41 to TGC, 30 to CIP, 18 to PB, 1 to MEM, 4 to CS, 9 to CN, 13 to LEV, 7 to IMP, 2 to AMC, 1 to CRO, 2 to DRO, 3 to AK and TE and 1 to GET).

Some clinical isolates did not have integron I but resistant to antibodies (9 to CTX, 7 to TGC, 5 to CIP, 10 to PB, 7 to MEM,10 to CS, 8 to CN, 9 to LEV, 8 to IMP, 10 to AMC,9 to CRO, 10 DRO, AK and TE and GET) and Some clinical isolates did not have integron I sensitive to antibodies (1 to CTX, 3 to TGC, 5 to CIP, 3 to MEM, 2 to CN, 1 to LEV, 2 to IMP and 1 to CRO).

Among the isolates, 5 isolates may have integron III and resistance to antibiotics (5 resist to CTX, 1 to TGC,3 to CIP,3 to PB, 3 to MEM,5 to CS, 4 to CN,4 to LEV,4 to IMP,5 to AMC, 5 to CRO ,4 to DRO, 5 to AK, 5 to TE and 5to GET), while some clinical isolates had integron III but sensitive to antibodies (4 to TGC, 2 to CIP, 2 to PB, 1 to CN, 1 to LEV, 1 to IMP and 1 to DRO). Some clinical isolates did not have integronIII but resistance to antibodies (63 to CTX, 25to TGC, 32 to CIP, 39 to PB, 61to MEM,61 to CS, 55 to CN, 42 to LEV, 47 to IMP, 63 to AMC,63 to CRO, 64 DRO, 62 to AK and TE and 64 to GET) and some clinical isolates did not have integronIII and sensitive to antibodies (2 to CTX, 40 to TGC, 33 to CIP, 164 to MEM, 4 to CS, 10 to CN, 13 to LEV, 8to IMP, 2 to CRO and AMC, 1 to DRO and GET, 3 to AK and TE).

Table 3. Antibiotic susceptibility pattern of class I, III integron positive and integron negative of *Acinetobacter baumannii* strains

Class	Antibiotics	integron positive I n=60		Integon negative I n=10		integron positive III n=5		Integrin negative III n=65		p-value
		Resistance (n %)	Sensitive (n %)	Resistance (n %)	Sensitive (n %)	Resistance (n %)	Sensitive (n %)	Resistance (n %)	Sensitive (n %)	
Carbapenem class	Meropenem MEM	59, 84.2 %	1, 1.42%	7, 10%	34, 28%	5 , 7.14%		61, 87.14%	4, 5.71%	<0.001
	Doripenem DOR	58, 82.85%	2, 2.85%	10, 14.28%	-	4, 5.71%	1, 1.42%	64, 91.42%	1, 1.42%	<0.001
	Imipenem IMP	53, 75.71%	7, 10%	8, 11.42%	2, 2.85%	4, 5.71%	1, 1.42%	47, 67.14%	8, 11.42%	<0.001
Fluroquino-lone class	Ciprofloxacin CIP	30, 42.85%	30, 7.14%	5, 7.14%	5, 7.14%	3, 4.28%	2, 2.85%	32, 45.71%	33, 47.14%	<0.001
	Levofloxacin LEV	47, 67.14%	13, 42.85%	9, 12.85%	1, 1.42%	4, 5.71%	1, 1.42%	42, 60%	13, 18.57%	<0.001
	Gemifloxacin GAT	59, 84.28%	1, 1.42%	10 , 14.28%	-	5, 7.14%	-	64,91.42%	1, 1.42%	<0.001
Aminoglyco-side class	Amikacin AK	57, 81.42%	3, 4.28%	10 , 14.28%	-	5, 7.14%	-	62, 88.57%	3, 4.28%	<0.001
	Minocycline TE	57, 81.85%	34, 28%	10, 14.28%	-	5, 7.14%	-	62, 88.57%	3, 4.28%	<0.001
	Tigecycline TGC	19,27.14%	41, 58.57%	7, 10%	3, 4.28	1, 1.42%	4,5.71%	25, 35.71%	40, 57.14%	<0.001
Betalactam class	Cefotaxime CTX	59, 84.28%	1, 1.42 %	9 , 12.85%	1, 1.42%	5, 7.14%	-	63, 90%	2, 2.85%	<0.001
	Amoxicillin / clavulanic acid	58, 82.85%	2, 2.85%	10 , 14.28%	-	5, 7.14%	-	63, 90%	2, 2.85%	<0.001
	Ceftriaxone CRO	59, 84.28%	1, 1.42%	9 , 12.85%	1, 1.42%	5, 7.14%	-	63, 90%	2, 2.85%	<0.001
	Colistin sul-fate CS	56, 80%	4 , 5.71%	10, 14.28%	-	5,7.14%	-	61, 87.14%	4, 5.71%	<0.001
	Polymyxin PB	42, 60%	18, 25.71%	10, 14.28%	-	3,4.28%	2, 2.85%	39, 55.71%	16,22.85%	<0.001

Conclusion

The integrons are gene cassettes containing antibiotic-resistance genes that play a major role in the MDR and XDR virulence characteristics in *A. baumannii.* The findings highlighted continued surveillance's importance in detecting integrons among *A. baumannii* strains. The comparative genomic analysis revealed extensive genomic variation in the *A. baumannii* genome. Integrons I, II and III are the main contributors to the plasticity of the *A. baumannii* genome and play a critical role in facilitating antibiotic resistance development in the clinical isolates. The presence of strains harboring acquired AMR genes makes them more dangerous. Acquired resistance genes and chromosomal gene mutation are successful routes for disseminating AMR determinants among *A*. *baumannii*. Identification of chromosomal and plasmid-encoded AMR in the genome of *A*. *baumannii* may help understand the mechanism behind the genetic mobilization and spread of AMR genes. The PCR results provideed insight into the pathobiology of *A. baumannii*, which exhibited multiple virulence factors and natural transformation, which may contribute to its success as a hospital-dwelling pathogen. New anti-virulence-based medicines can be created by designing tiny compounds that can modulate these processes.

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Conflict of interest

The authors declare that they have no conflicts of interest.

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