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Molecular Detection Of Virulence And Antibiotics Resistance Gene Of Acinetobacter Baumannii Isolated From Burned Wound Patients

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Abstract

Background: *Acinetobacter baumannii is identified as one of the most common infections in burn units globally.* ***Objective***: *The purpose of this study was to assess antibiotic resistance and apply molecular method to clinical isolates of A. baumannii that were obtained from burned wound.* ***Methods***: *A total of 150 specimens (burned wound swab) were collected from patients who admit in burns units throughout the period of December 2023 to the Februry 2024. The collected specimens were streaked directly on culture media incubated for 24 hrs at 37°C. All bacteria isolates were examined by confirm by vitik 2 system. Antibiotic susceptibility test was prepared by vitik 2 system. Acinetobacter baumannii isolates was performed by using Polymerase chain reaction to detect blaoxa 51 Bap ,OmpA, CsuE and PgaA genes.* ***Results***: *Flame were higher in females,while Electric was higher in males. Burn surface area < 15% and 15 - 25 % were higher in males. Second / degree burns was higher percentage injury from other degree of burn. Patients injury by flame was more infected with A. baumannii bacteria by 80 (53.3%). A total of 30 isolates of A. baumannii have been discovered. A PCR was conducted on 15 Multi-Drug Resistant (MDR),(XDR) isolates to identify the presence of genes associated with virulence factors. The results showed that the bap ompA, pgaA blaOXA-51-like genes were found to be present in all 15 isolates A. baumannii clinical, while CsuE pili was 0%.* ***Conclusions***: *Our findings revealed a correlation between genes of antimicrobial resistance and significant biofilm formation.*

Keywords: ***A.baumannii , Resistance Gene,Causes of Burns***

INTRODUCTION

Burn infections are regarded as one of the most serious consequences of thermal injury [1]. Burn infections can be fatal if bacteria penetrate the tissue layers underlying the dermis burn injuries can be caused by high temperatures, electricity, friction, radiation, or chemicals [2]. Burn injuries can be defined based on a variety of variables, including their depth, aetiology, and percentage of body surface area injured; the combination of these categories determines the degree of burn injury; burns can be classed as "partial-thickness" or "full-thickness" [3]. In addition to the form of the burn infections, the number of bacteria inhabiting them is significant, as it can lead to bacteremia, sepsis, and multiple-organ failure syndrome [4]. Due to their considerable antibiotic resistance, Gram-negative bacteria have been identified as the primary pathogens of burn infections, Acinetobacter baumannii is identified as one of the most common infections in burn units globally [5]Acinetobacter spp., notably Acinetobacter baumannii, have emerged as opportunistic pathogens, causing a wide range of severe, life-threatening nosocomial infections in immunocompromised people, particularly burn patients [6]. The bacteria are capable of surviving in dehydrated and severe environmental circumstances for extended periods of time and may colonize in hospitalized patients, causing delayed wound healing, infection, and even death [7]. In recent years, the widespread appearance of multi- and pan-drug-resistant A. baumannii strains has demonstrated this organism's ability to rapidly adapt to environmental changes [8]. Biofilm development is one of the distinguishing features of opportunistic infections such as A. baumannii [9]. A biofilm is a population of bacterial cells that are attached to biotic or abiotic surfaces and interact intimately with one another. A. baumannii may create a wide range of virulence factors in the biofilm mode, which contribute to the various stages of biofilm cell adhesion to biotic or abiotic surfaces [10].

Bacteria in the biofilm state have more antibiotic tolerance than bacteria in the planktonic mode, allowing them to tolerate 100 to 1000 times higher concentrations of antimicrobial agents [11]. Many investigations in most countries have revealed the ability of A. baumannii strains to produce biofilms, as

well as the frequency of genes associated with it [12]. In recent decades, clinical isolates of A. baumannii have developed antimicrobial resistance to frequently prescribed antimicrobial drugs, primarily in hospitalized patients worldwide [13]. It has been accurately described that one of the most important factors contributing to the high mortality of patients with nosocomial infections caused by A. baumannii is the ability to acquire a wide range of antibiotic resistance genes and the rapid development of multidrug-resistant (MDR), extensively drug-resistant (XDR), and even pan-drug resistant (PDR) strains [14]. The introduction and dissemination of drug-resistant A. baumannii strains has drastically reduced the number of viable therapeutic choices for treating infections caused by the bacteria, resulting in a worse clinical outcome [15]. A. baumannii develops multidrug resistance by many mechanisms, including decreased membrane permeability, loss of porins, acquisition of extended-spectrum β-lactamase, and multidrug efflux systems [16].

MATERIAL And METHODS:

2.1 Specimens collecti

An amount of 150 specimens (burned wound swabs) were collected from patients admitted to the burns unit at Baquba Teaching Hospital, Khanaqin General Hospital, and outpatients between December 2023 and February 2024.

Isolation of Acinetobacter baumannii

Under aseptic conditions, specimens were streaked on blood agar and MacConkey agar and incubated at 37°C for 24 hours. The non-hemolytic opaque creamy colonies on blood agar and non-lactose fermenting colonies on MacConkey agar and chrom agar were subcultured and incubated for an additional 24 hours at 37°C. Bacterial isolates were tested for gram stainability and growth at 44°C. The Vitik 2 system was used to confirm the isolates and produce an antibiotic susceptibility test [17].

Genotype confirmation of A. baumannii by PCR

Acinetobacter baumannii isolates was performed by using Polymerase chain reaction (PCR) to detect blaoxa 51 Bap ,OmpA, CsuE and PgaA and the sequences of primers pair used were shown in table 1 and 2, DNA of each isolate was extracted using a commercial purification system (BIONEER/ Korea).

Table 1: Sequences of primers pair of genes

Gene	Sequences (5'_3')	Product size/bp
blaoxa 51	TAATGCTTTGATCGGCCTTG	353
	TGGATTGCACTTCATCTTGG	
bap	TGCTGACAGTGACGTAGAACCACA	184
	TGCAACTAGTGGAATAGCAGCCCA	
csuE	ACCAATGCTCAGACCGGAG	751
	CTTGTACCGTGACCGTATCTTG	
ompA	GAGTCGTATTGCACTTGCTAC	594
	GCAGGCTTCAAGTGACCACC	
pgaA	ATTCAAAAGTCAGTTGATGGGC	460
	CCCCTGCTCATCATAATGTAAG	

Template DNA was prepared by the boiling method according to [18] and supernatant was used as template DNA in PCR analyses.

Table 2:Amplification program of primers

Amplified	Initial denaturation	No.of cycle	Denaturation	Annealing	Elongation	Final extension
blaoxa 51	95°C/ 5min	35	95°C/ 30 sec	54°C/ 30 sec	72°C/ 30 sec	72°C/ 7 min
bap	95°C/ 5min	35	95°C/ 30 sec	54°C/ 30 sec	72°C/ 30 sec	72°C/ 7 min
csuE	95°C/ 5min	35	94°C/ 1min	59°C/ 1min	72°C/ 1min	72°C/ 10 min

ompA	95°C/ 5min	35	94°C/ 1min	59°C/ 1min	72°C/ 1min	72°C/ 10 min
pgaA	95°C/ 5min	35	94°C/ 1min	59°C/ 1min	72°C/ 1min	72°C/ 10 min

RESULTS

The distribution of males and females among Study groups

The results shown in table (3A) showed cause of burn among males and females as follow: Flame (35 (45. 75 %) male , 45 (56. 25 %) female) , Hot liquid (26 (48 . 15 %) male , 28 (51. 85 %) female), Electric (15 (93 . 75 %) male , 1 (6. 25 %) female) with significant was < 0.001. The results shown in table (3B) showed burn surface area among males and females as follow: < 15 % (33 (22 %) male , 19 (12 .7 %) female), 15 - 25 % 28 (18 . 7 %) male , 24 (16 %) female), 26 - 50 % (13 (8 . 7 %) male , 20 (13 . 3 %) female), > 50 % 2 (1. 3%) male , 11 (7. 3%) female) with significant was < 0.001 . The results shown in table (3C) showed Burn degree among males and females as follow: First / degree burns 11 (7. 3 %) male , 5 (3 . 3 %) female), Second / degree burns 39 (2 . 6 %) male , 37 (14 . 7 %) female), Third / degree burns 25 (16. 7 %) male , 33 (22 %) female) with significant was < 0.001 .

Table 3: Distribution of male and female among Study groups

Characteristic	Male ,N 76 (50 .7 %)	Female ,N 74 (49 .3 %)	Total ,N 150 (100 %)	P . value
(A) Cause of burn				
Flame	35 (45 . 75 %)	45 (56 . 25 %)	80 (53 . 3 %)	
Hot liquid	26 (48 . 15 %)	28 (51 . 85 %)	54 (36 %)	
Electric	15 (93 . 75 %)	1 (6 . 25 %)	16 (10 .7 %)	< 0.001
(B) Burn Surface area (%) TBSA				
< 15 %	33 (22 %)	19 (12 . 7 %)	52 (34 . 7 %)	
15 - 25 %	28 (18 . 7 %)	24 (16 %)	52 (34 . 7 %)	< 0.001
26 - 50 %	13 (8 . 7 %)	20 (13 . 3 %)	33 (22 %)	
> 50 %	2 (1 . 3 %)	11 (7 . 3 %)	13 (8 . 6 %)	
(C) Burn degree				
First / degree burns	11 (7 . 3 %)	5 (3 . 3 %)	16 (10 . 6 %)	
Second / degree burns	39 (2 . 6 %)	37 (14 . 7 %)	76 (50 . 7 %)	< 0.001
Third / degree burns	25 (16 . 7 %)	33 (22 %)	58 (38 . 7 %)	

Isolation and identification of Acinetobacter baumannii isolates

All isolates from a total of 150, 30 specimens were Gram-negative coccobacilli, occasionally grouped in diplococci. All isolates were confirm diagnosised by vitik 2 system table 4(A,B). Also, when A. baumannii isolates were cultivated on MacConkey agar, they appeared as small, pale, lactose-free fermenter colonies, on blood agar, they appeared as opaque, creamy, and non-hemolytic colonies, whereas on chrom agar appeared as light purple with a halo around the colonies. All A. baumannii isolates demonstrated good growth at 44°C.This test was used to distinguished A. baumannii (which wasable to grow at this temperature degree) from other Acinetobacter species which unable to grow at this temperature degree [19]. Antibiotics sensitivity test of A. baumannii was done by vitik 2 system .

Table 4 A: Biochemical test of vitik for A. baumannii

Hasan Lab.
bioMérieux Customer: Microbiology Chart Report Printed January 4, 2024 5:05:52 PM CST

Patient Name: 16. . Patient ID: 175
Location: Physician:
Lab ID: 175 Isolate Number: 1

Organism Quantity:
Selected Organism : Acinetobacter baumannii complex

Source: Collected:

Comments:

Identification Information	Analysis Time:	5.85 hours	Status:	Final
Selected Organism	99% Probability	Acinetobacter baumannii complex		
	Bionumber:	0201010103500210		
ID Analysis Messages				

Biochemical Details

2	APPA	-	3	ADO	-	4	PyrA	-	5	lARL	-	7	dCEL	+	9	BGAL	-
10	H2S	-	11	BNAG	-	12	AGLTp	-	13	dGLU	+	14	GGT	-	15	OFF	-
17	BGLU	-	18	dMAL	-	19	dMAN	-	20	dMNE	+	21	BXYL	-	22	BAlap	-
23	ProA	-	26	LIP	-	27	PLE	-	29	TyrA	+	31	URE	-	32	dSOR	-
33	SAC	-	34	dTAG	-	35	dTRE	-	36	CIT	+	37	MNT	+	39	5KG	-
40	ILATk	+	41	AGLU	-	42	SUCT	+	43	NAGA	-	44	AGAL	-	45	PHOS	-
46	GlyA	-	47	ODC	-	48	LDC	-	53	IHIsa	-	56	CMT	+	57	BGUR	-
58	O129R	+	59	GGAA	-	61	IMLTa	-	62	ELLM	-	64	ILATa	-			

Table 4 B: Antibiotics sensitivity test of A. baumannii

Patient Name: 12, . Patient ID: 185
Location: Physician:
Lab ID: 185 Isolate Number: 1

Organism Quantity:
Selected Organism : Acinetobacter baumannii

Source: Burned wound Collected:

Comments:

Susceptibility Information | Analysis Time: 7.52 hours | Status: Final

Antimicrobial	MIC	Interpretation	Antimicrobial	MIC	Interpretation
Ticarcillin	>= 128	R	Amikacin		
Ticarcillin/Clavulanic Acid	>= 128	R	Gentamicin	8	I
+Mezlocillin		R	Tobramycin	>= 16	R
Piperacillin	>= 128	R	Ciprofloxacin	>= 4	R
Piperacillin/Tazobactam	>= 128	R	Pefloxacin		
Ceftazidime	>= 64	R	Minocycline	<= 1	S
Cefepime	>= 64	R	Colistin	<= 0.5	S
Aztreonam			Rifampicin		
Imipenem	>= 16	R	Trimethoprim/ Sulfamethoxazole	>= 320	R
Meropenem	>= 16	R			

AI's Findings

Confidence: Consistent

]

The Relation of A. baumannii with Cause of burn

The results shown in table 5 and figure 1 showed A. baumannii bacteria infected with Cause of burn as follow: Flame 80 (14 (46 . 7%)), Hot liquids 54 (12 (40%)), Electricity 16 (4 (13.3%)) with significant was > 0.05.

Table 5 : Distribution of A. baumannii according Cause of burn

Origin of Sample Collection	Flame	Hot liquids	Electricity	Total	P value
No . Sample %	80 (53 .3%)	54 (36%)	16 (10. 7%)	150 (100 %)	< 0.001
No . A. baumannii	14 (46 . 7%	12 (40 %)	4 (13. 3 %)	30 (100 %)	> 0.05

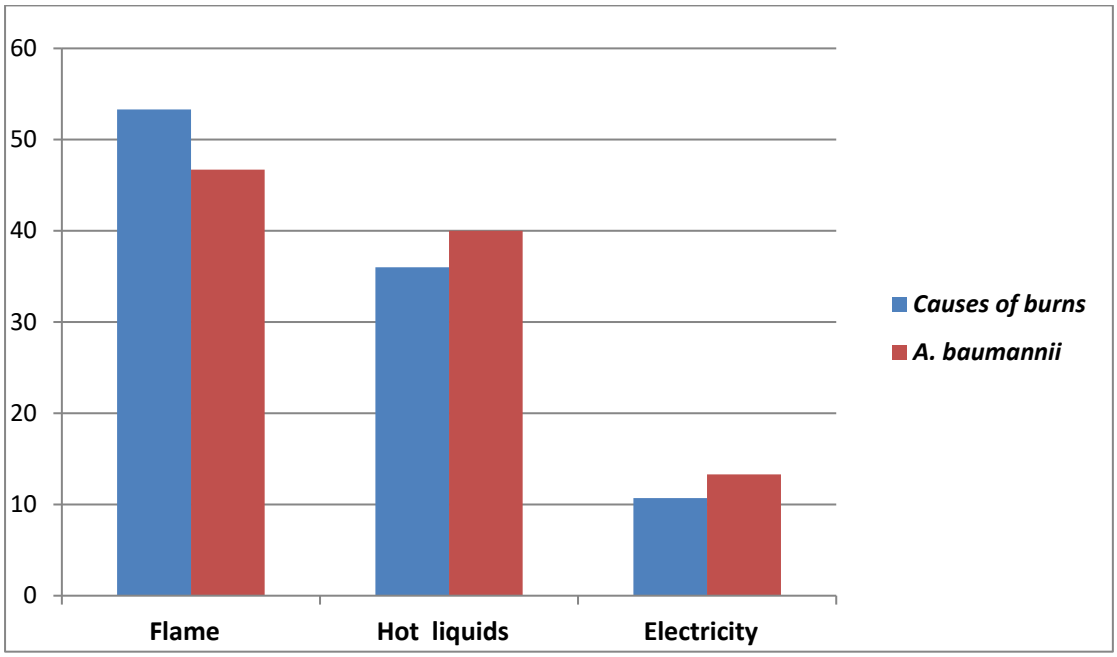

Figure 1: Prevalence of A. baumannii according to causes of the burns

Identification of Acinetobacter baumannii by Polymerase Chain Reaction (PCR)

The bap ompA, pgaA blaOXA-51-like genes were found to be present in all 15 (100%) A. baumannii clinical , while CsuE pili was 0% in all A. baumannii as shown in table 6 and environmental studied isolates as shown in figures 2a,2b,3a,3b,4 < 0.001 .

Table 6 : Frequency of the virulence and antibiotic resistance gene of Acinetobacter baumannii isolated from burned wound patients

Gene	Present No.%	Absent No.%	Total No.%	P.value
blaoxa 51	15 (100%)	0 (100%)	15 (100%)	< 0.001
bap	15 (100%)	0 (100%)	15 (100%)	< 0.001
csuE	0(100%)	15 (100%)	15 (100%)	< 0.001
ompA	15(100%)	0 (100%)	15 (100%)	< 0.001
pgaA	15 (100%)	0 (100%)	15 (100%)	< 0.001

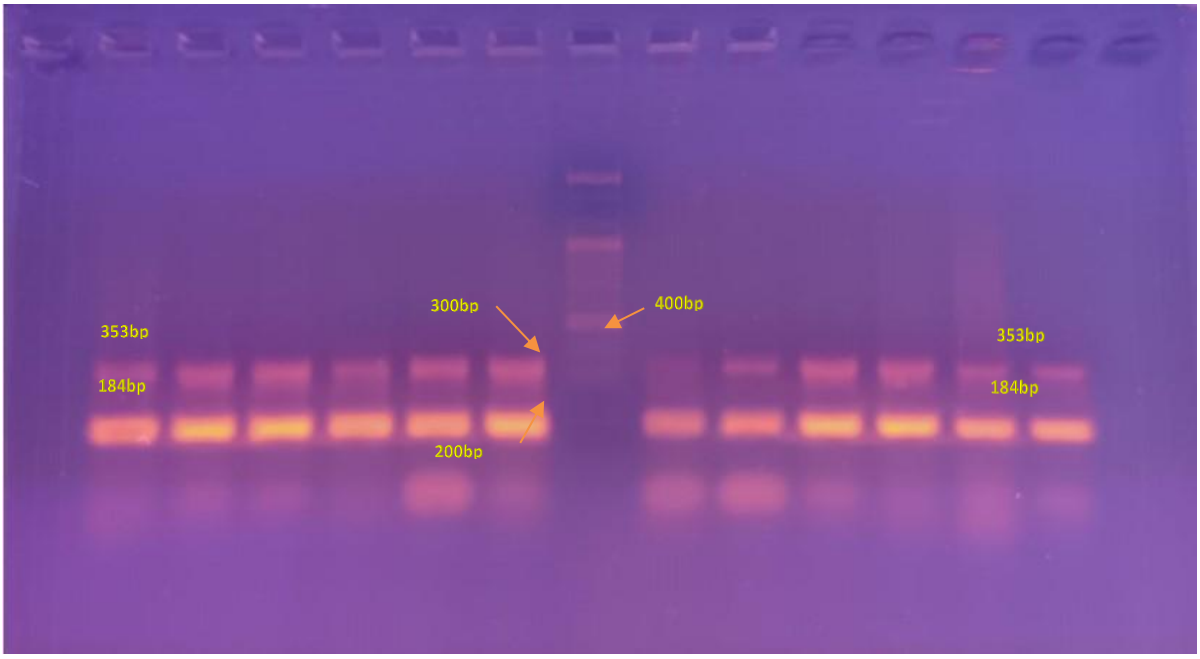

Figure 2A: Agaros gel electrophoresis (1.5% agarose,7v/cm^2 for 60 min) for blaoxa51 gene (353bp amplicon) and bap gene (184bp amplicon) lane7 represent M100bp DNA Ladder, lanes 1-6 and 8-13 represent of bands.

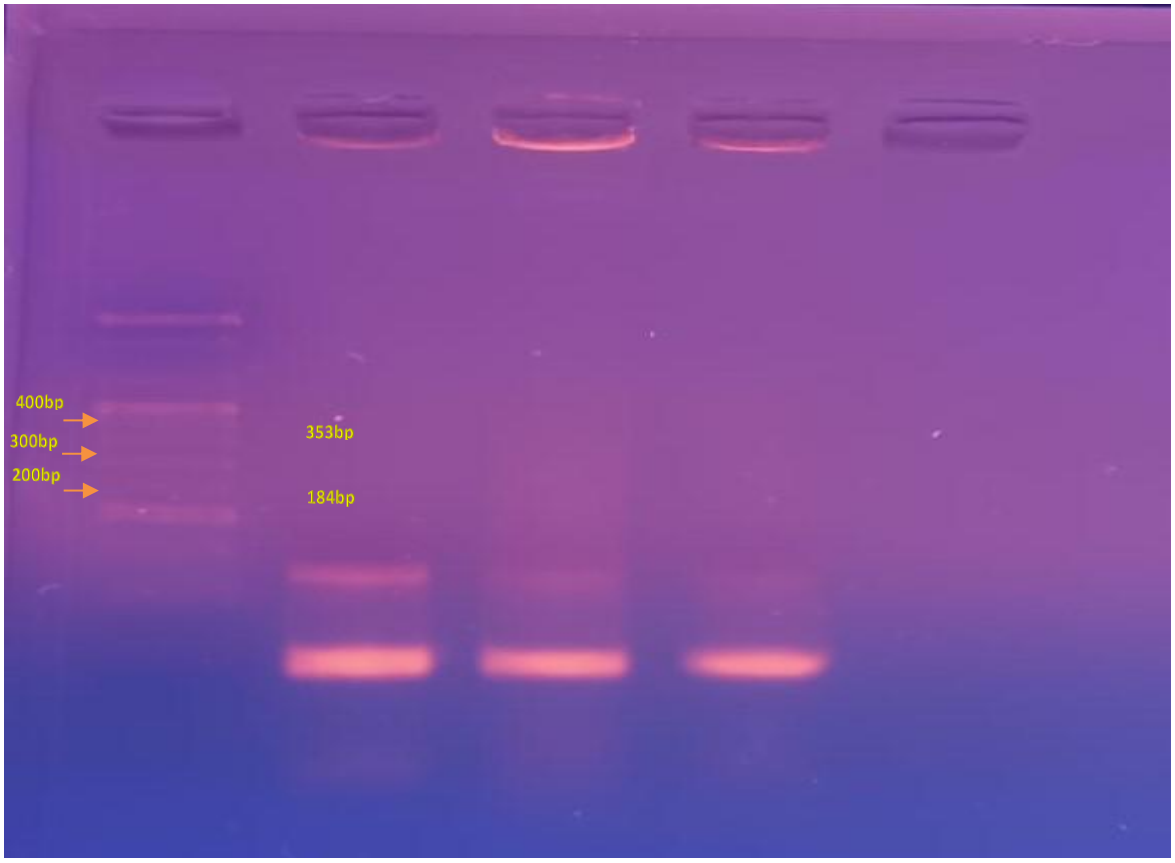

Figure 2B: Agaros gel electrophoresis (1.5% agarose,7v/cm2 for 60 min) for blaoxa51 gene (353bp amplicon) and bap gene (184bp amplicon) lane1 represent M100bp DNA Ladder, lanes 2-4 represent of bands.

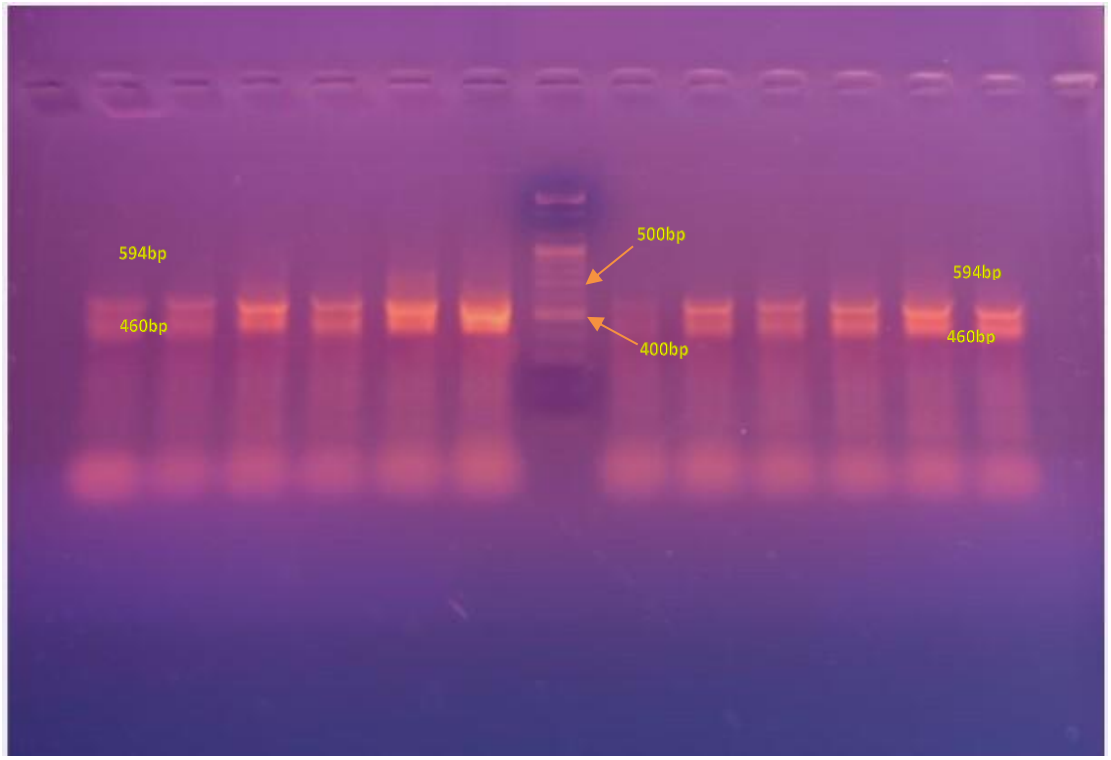

Figure 3A: Agaros gel electrophoresis (1.5% agarose,7v/cm2 for 60 min) for ompA gene (594bp amplicon) and pgaA gene (460bp amplicon) lane7 represent M100bp DNA Ladder, lanes 1-6 and 8-13 represent of bands.

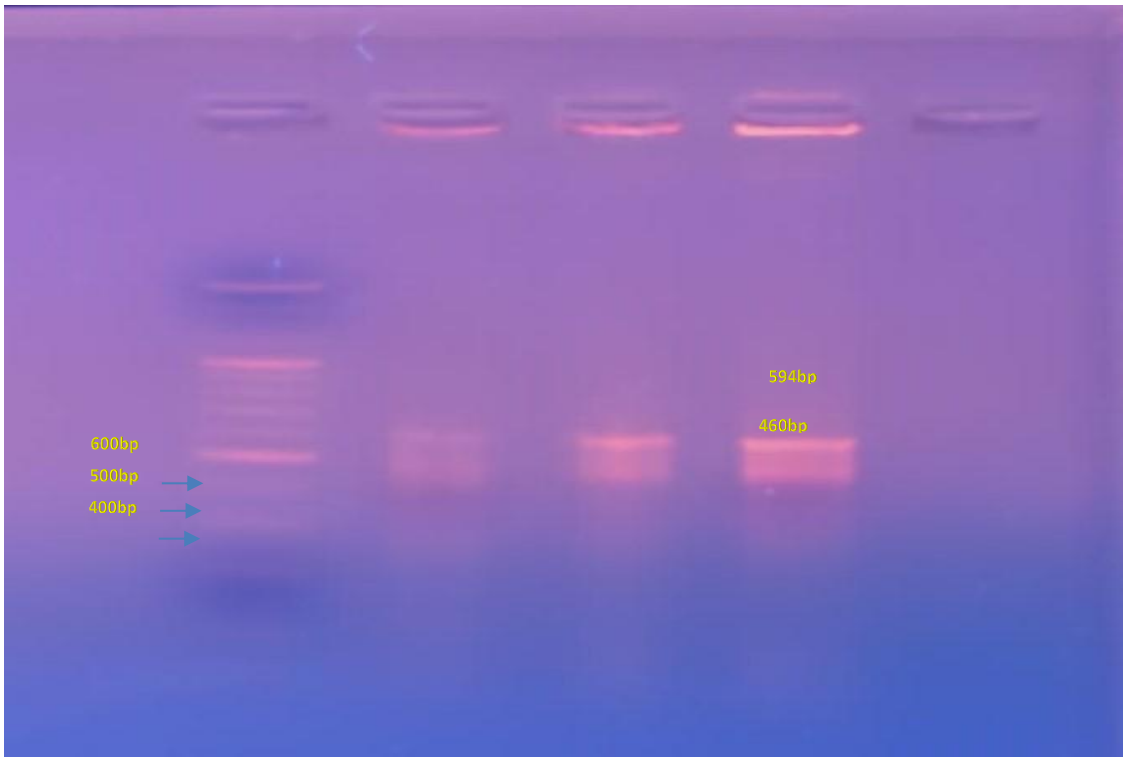

Figure 3B: Agaros gel electrophoresis (1.5% agarose,7v/cm^2 for 60 min) for ompA gene (594bp amplicon) and pgaA gene (460bp amplicon) lane7 represent M100bp DNA Ladder, lanes 2-4 represent of bands.

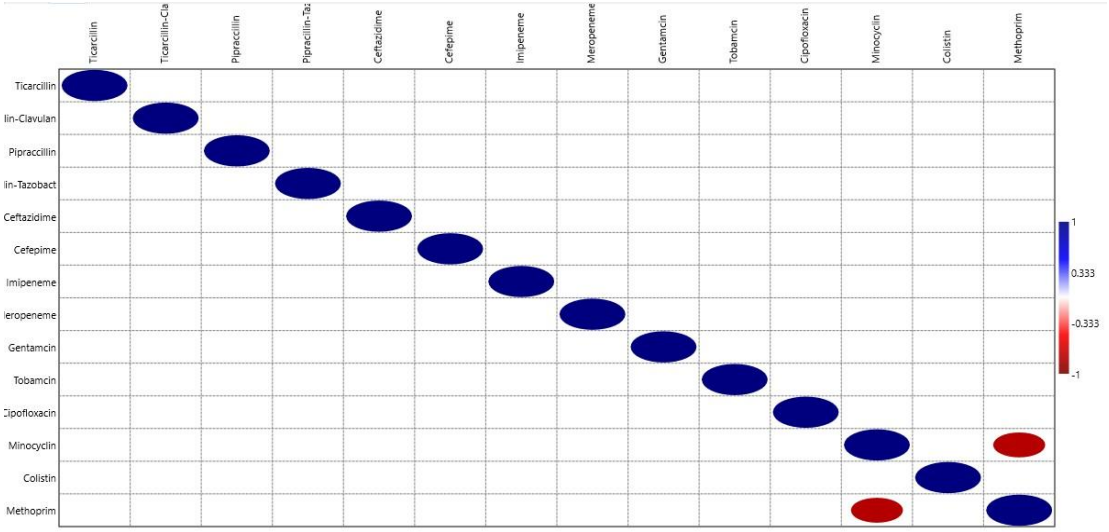

Figure 4 : Showing the correlation between antibiotics and the susceptibility of isolates of Acinetobacter baumannii

DISCUSSION

Burns are among the most common unintentional injuries in the world [20]. The current results displayed in table 2 provided cause of burn among males and females, flame and hot liquid were greater in female than male, while electric was higher in male than female. Many studies agree with our results. According to Oseni et al.[21], contact with hot liquids that cause burns (57%), such as cooking oil, appeared to cause the greatest number of burn injuries, followed by flame (25%), and contact with hot objects (16%). In terms of burn causes, this study found that burning fires were the most common, accounting for 58.5%, followed by hot water (32.1%) [22]. Jeschke et al. [23] demonstrated that males were more frequently impacted by electrical injuries (100% and 55.0%, respectively); yet, females are more susceptible to self-inflicted burns or heater injuries than males. Similar to numerous studies,10 this investigation demonstrated that burn injuries were more common in males across all age categories (54.5% vs 45.5%), demonstrating the considerable influence of gender on injury risk [24].Liu et al. [25] discovered that thermal burns involve the skin, scalds caused by hot fluid or steam, contact burns caused by hot solids or objects such as hot pressing irons or cooking utensils, as well as lighted cigarettes, and flame burns caused by flames or incandescent fires, such as those started by lighted cigarettes, candles, lamps, or stoves, and electrical burns caused by an electrical current passing through the body. In underdeveloped nations, burn injuries are most commonly caused by fire-related mishaps in the home setting; a substantial proportion of burns occur in households where women are more likely to work with flames and stoves, particularly for cooking [26].According to table 3a, males had larger burn surface area (15-25%) than females, which is consistent with a research by Rezaee et al. [27]. According to table 3b, the present data demonstrate that second/degree burns cause a larger percentage of injury than other degrees of burns; various studies support our findings. Pirbonyeh et al. [28] conducted a study in Shiraz, Iran, and discovered that out of 713 patients, 510 (71.5%) were male and 203 (28.5%) were female, with 304 (42.6%) having second or higher degree burns. Another study found that the degree of burning was superficial, with redness (first degree) in 69.2%, second degree in 29.7%, and only 1% in the third degree [29]. However, another study discovered that 45.5% of instances were second degree, followed by mixed degree 38.6%, deep second 9%, third 4.8%, and only 1.6% were first degree [30]. In terms of the relationship between burns and gender, multiple studies indicated that females had 79.4% more burns than males (20.6%), and there was a significant association ($p = 0.04$) [31]. Shokouhi et al.[26] found that females were more affected than males by 74% to 26%, and that females are more responsible for everyday household duties than males. This is the reason that more females sustain flame burn compared to males. Similarly, a notable feature of this study is that gas- or oil-related fire injuries were more common among female than male patients, which can be explained by the fact that some women in this area prefer to be housewives or have limited autonomy to engage in public or social activities on their own [25]. It is also

likely that low literacy, the flammability of women's local clothing, and the unsafe application/design of stoves and heaters, which are the primary sources of cooking and heating in the region, impose a greater load on female injuries than males [28]. In terms of the relationship between the cause of burns and A. baumannii bacteria as shown in table 5 and figure 1, patients injured by flame were more infected with A. baumannii bacteria by 80 (14 (46. 7%)) than those injured by other causes. These findings are consistent with a study conducted by El Hamzaoui et al. [32], which found that the main sources of burns infected with A. baumannii were flames (52.38%) and hot water (28.57%).Burn wounds are typically a vulnerable site for colonization by opportunistic organisms of either endogenous or external origin [33,34]. Both facultative and aerobic gram-negative bacilli and aerobic gram-positive cocci can be isolated from burn wound cultures. Acinetobacter baumannii is one of the causal organisms being isolated from burn wound infections at an increasing rate [31]. This bacterium is a major opportunistic pathogen that causes a wide range of nosocomial infections, including bacteremia, urinary tract infection, secondary meningitis, surgical site infection, nosocomial ventilator-associated pneumonia, and dirty wound infections [28].Identification of A. baumannii by genes shown in table 6 and figures 2a,2b,3a,3b in A. baumannii, the genes encoding ompA, bap, pgaA, and abaI are recognized to be critical in biofilm formation [12]. Al-Shamiri et al.[35] discovered that the gene profiles of bap, csuE, ompA, pgaA, and abaI were present in all strong biofilm producers as well as some moderate producers. The gene patterns of csuE, ompA, pgaA, and abaI were found in both weak and moderate biofilm producers; these findings suggest that, in addition to the presence of these genes, their expression levels play an important role in determining biofilm formation capacity [36]. Some genes important in biofilm production were found in non-biofilm producing strains; however, the expression levels of these genes were higher in biofilm producers as compared with non-producers [37].The pgaABCD operon produces poly-β-1,6-Nacetylglucosamine (PNAG), which helps build biofilm [38]. Khoshnood et al.[39] demonstrated that the presence of all biofilm-related genes (ompA, ptk, ataA, csuE, and csuA) predicted robust biofilm formation and varying antimicrobial resistance. Wallace reported an investigation of A. baumannii's biofilm-related genes, [40], and discovered that abaI and csuE were present in 59.8% of the samples and ompA in 100% of the samples. Khoshnood et al. [13] found that ompA-mediated adhesion plays a substantial role in biofilm development in A. baumanni-associated nosocomial infections, particularly in burn and VAP patients. Previous research has found a link between the presence of biofilm-related genes like ompA and antimicrobial resistance. OmpA is the most abundant porin in A. baumannii, and its role in drug resistance was more prominent in disruption mutants of the gene, which showed reduced resistance to imipenem, meropenem, nalidixic acid, and chloramphenicol [41]. Amin et al.[12] found that a strong potential for biofilm formation was significantly linked with the presence of all the studied biofilm-related genes.Qi et al.[42] discovered that XDR A. baumannii strains have a greater ability for biofilm formation than MDR strains. Several investigations have suggested that XDR bacterial strains generate stronger biofilms than antimicrobial-sensitive strains, which is consistent with our findings that XDR strains have a higher biofilm production capability than MDR strains [43]. The csuE gene, is commonly found in many Acinetobacter species and plays a significant role in biofilm formation, it's possible for certain isolates within the genus Acinetobacter to lack this operon or have mutations that render it non-functional. Overall, the absence of the csu operon in certain Acinetobacter isolates could be attributed to genetic variation, horizontal gene transfer, environmental factors. It highlights the genetic diversity and adaptability of bacterial species, even within the same genus.According to the work by Özkul and Hazırolan [44], the blaoxa-51-like gene, an intrinsic enzyme marker, was used as a trustworthy marker for the identification of A. baumannii. The primary cause of carbapenem resistance in A. baumannii is the synthesis of genes encoding β-lactamases of the OXA type. The ability to treat nosocomial infections has been shown to be significantly impacted clinically by the influence of OXA β-lactamases in conferring carbapenem resistance [45].From figure 4, The blue circles in the image represent the correlation coefficients between the different antibiotics. The higher the correlation coefficient, the more closely the antibiotics are correlated. This means that they tend to have similar effects on bacteria, and are often either effective or ineffective against the same bacteria. The red tint shows a high positive association, indicating that the antibiotic is efficient against the isolate [42]: distinct Mechanisms of Action: Antibiotics target distinct regions of a bacterial cell. If two antibiotics have very different modes of action,

a bacterium may become resistant to one while staying sensitive to the other. Resistance Mechanisms: Some bacteria have acquired resistance mechanisms to specific antibiotic classes. An isolate resistant to one antibiotic may be sensitive to another by a different route, and vice versa [46].

CONCLUSION

This study found a high frequency of genes implicated in biofilm development. There was a strong negative connection between antibiotic resistance and biofilm formation in A. baumannii isolated from burns. Our findings revealed a correlation between antimicrobial resistance and significant biofilm formation, implying that resistance mechanisms are transmitted across bacterial strains within the biofilm niche.

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