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New Isolates of *Proteus Mirabilis* and *Klebsiella Pneumoniae* Associated with Burn Surface Infection

Nada K. Younus[1*], Nadhim A. AL-Shaheri[2]
[1]*Department of Pharmacy, Alnoor University, Alshallalat Road, 41002, Ninevah Province, Iraq*
[2]*Anesthesia Department, Alnoor University, Alshallalat Road, 41002, Ninevah Province, Iraq*

Abstract

Burn patients suffer from breach of the protective skin layer which would lead to the association of complications including nosocomial bacterial infection. Klebsiella pneumoniae and Proteus mirabilis are among the most common bacterial infections. They are characterised by the expression of virulence factors leading to multi-drug resistance (MDR) the present study aimed to identify the association of virulence genes with burn bacterial infection. After a fire broke out in an accident in an Al-Hamdaniya town (Iraq), a total of 250 clinical isolates (swabs) were collected from patients. Identification of 184 (73.6%) and P. mirabilis 66 (26.4%) were made as the commonest Gram-negative bacteria to be studied. All isolates were carried out depending on microscopic examination, cultures, and genetic technique. A susceptibility test was achieved for all clinical isolates using the disk diffusion method. Ten antibiotics disks (imipenem, gentamycin, cefotaxime, sulphamethoxazole, pipracillin, cefixime, amoxycillin, trimethoprim, ciprofloxacin, and amikacin. For both K. pneumoniae and P. mirabilis, sensitivity was the highest with IMP (65% and 100%, respectively and lowest with PRL (0% and 4.6%, respectively). expressed mrkD, uge, fimH-, bla CTXM-1, and bla NDM-1, while the sample of P. mirabilis expressed aclb, bla TEM, atpD, ureC, rsbA, zabA, bla CTX, and bla OXA-1, these genes supposed that they evade the antibiotic therapy and thereby commence antibiotic-resistance. This study observed that some Iraqi isolates contain many different genes for both K. pneumoniae and P. mirabilis the causative agent of burn wound infections. These bacteria were resistant to multiple drugs with the highest sensitivity being associated with imipenem and the lowest with piperacillin.

Keywords: *Burn, Extended-spectrum Beta-lactamases, Klebsiella Pneumoniae, Multi-drug Resistance, Proteus Mirabilis.*

Introduction

Burn is one of the health-challenging crises because the dermal barriers are breached leading to tissue exposure to external environments including pathogens [1]. The wound release of fluids called exudate serves as an extra-promoting factor for pathogen multiplication [2]. The involvement of bacteria in the wound injury is reciprocally correlated with the hospital stay [3]. The most common pathogens involved are methicillin-resistant *Staphylococcus aureus, Klebsiella pneumoniae, Proteus mirabilis,* and Pseudomonas aeruginosa [3]. Most of these bacteria were reported to be resistant to antibiotics [4].

Gram-negative bacilli are highly virulent pathogens involved in burn injury, *K. pneumoniae, and P. mirabilis,* are Gram-negative rods belonging to the Enterobacteriaceae order [5]. These bacteria are responsible for inducing infections, such as

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****Corresponding Author: nada.khairi@alnoor.edu.iq***

urinary tract infections, wound infections, meningitis in infants, rheumatoid, endocarditis, septicemia, and cystic fibrosis [3]. The pathogenicity is likely related to high virulence capacity which is partly linked to released virulence factors or bacterial behaviours motility or inhibition of immune response or tackling phagocytosis [4, 6–8]. These properties make *K. pneumoniae, and P. mirabilis* resistant to many antibiotics, resulting in multidrug resistance (MDR) [4, 5, 9].

Antibiotic resistance becomes harder when MDR exists, especially in Extended-spectrum beta-lactamases (ESBL), with admitted patients having a higher chance of exposure to such a mixed population of bacteria, which is common in burn patients [9]. These ESBL-encoded genes are transmitted through plasmids over bacterial generations, leading to the generation of more virulent bacteria with MDR [10].

K. pneumoniae mostly contains extended-spectrum beta-lactamase genes (SHV, TEM and C-TXM) that are encoded by a plasmid responsible for MDR in these bacteria [11]. Extended-spectrum beta-lactamases arise mainly due to mutations in beta-lactamases encoded by the bla SHV, bla TEM, and bla CTX-M genes. More than 300 different ESBL variants have been described [12]. Over the last 20 years, strategies that were usually used by researchers were standard PCR and gene sequencing which is still the most widely used technique [13]. Therefore, a new generation of cephalosporin (e.g. cefotaxime, ceftazidime, and ceftriaxone) were introduced to treat nosocomial infection due to their capacity to eliminate bacteria with MDR [11, 14–16]. The present study aimed to find the new strain of ESBL K. pneumoniae and *P. mirabilis* among pathogenic isolates isolated from Iraqi patients who have attended hospitals during a fire breakout at an Iraq wedding, to detect their drug resistance pattern and to identify many resistance genes in such organisms.

Patients and Methods

Study design and settings: A total of 250 isolates from burn wound infection were collected during the Al-Hamdaniya town fire break out (Iraq) at an accident that happened during a wedding party on 26 Sep 2023. Samples were collected from patients who attended Al-Hamdaniya Hospital and Ibn-Sina Hospital (Ninevah Province, Iraq). Samples from hospital-admitted burn patients were collected by sterile swabs and streaked onto the surface of MacConkey agar and blood agar (Oxoid, UK) and incubated (37°C, 24h). All isolated *K. pneumoniae* and *P. mirabilis* strains were identified in the clinical samples by conventional microbiological methods, including Gram staining and biochemical tests (Table 1).

Table 1. Biochemical Tests for Identification *of K.pneumoniae* and *P. mirabilis* Strains

Test name	***K. pneumoniae* (184)**	***P. mirabilis* (n=66)**
Urease	+ve	+ve
Oxidase	+ve	-ve
Triple sugar iron	+ve	
Simmons Citrate Agar	+ve	
Indol	+ve	-ve
Motility	Non-motile	Motile
Capsule	-ve	-ve
Catalase		+ve
Citrate		+ve
Gelatin hydrolysis		+ve

Methyl red		+ve
Nitrate reduction		+ve
Pigment		-ve
Voges-Proskauer		-ve

Antimicrobial susceptibility testing: Samples of isolates were harnessed for the pattern of antimicrobial susceptibilities by an agar disc diffusion method using paper discs (ABBiodisk, Solna, Sweden) on Mueller–Hinton (MH) agar, including susceptibility to the following antibiotics: imipenem (IPM), gentamycin (CN), sulphamethoxazole (SXT), pipracillin (PRL), cefixime (CFM), amoxycillin (AMC), trimethoprim (TMP), ciprofloxacin (CIP), cefotaxime (CTX), and amikacin (AK). After overnight incubation at 37°C, inhibition zone diameters were read. The results of a disc diffusion test are interpreted by comparing the measured zone diameter with the interpretive criteria.

Polymerase chain reaction (PCR) for detection of resist genes: DNA was extracted by a boiling method as follows: Three to five pure and fresh colonies were suspended in (300µl) of distilled water, then cells were lysed by heating at 100°C for 20 min (in a water bath), immediately the cells were placed in ice for 30 min and the other cellular components was removed by centrifugation (8500rpm, 10min). Finally, the supernatant was used as the DNA template [16, 17].

The polymerase chain reaction (PCR) method was applied for all isolates and all primers used are listed in Table 2 and Table 3 for *K. pneumoniae* and P. mirabilis, respectively. Thermocycling conditions are listed in Table 4 and Table 5 for *K. pneumoniae* and P. mirabilis, respectively. The tested genes for the sample of *K. pneumoniae* are those involved in the virulence and pathogenicity of this bacteria including magA (microviscosity-associated gene), rmpA (regulators of mucoid phenotype A), mrkD (bacterial adhesion gene), uge (UDP galacturonate 4-epimerase), fimH-1(bacterial adhesion gene), bla KPC (*K. pneumoniae* carbapenemase), bla CTXM-1 (extended-spectrum β-lactamase gene), bla NDM-1 (Carbapenemase gene), and OXA (Carbapenemase) [18–21]. The tested genes for the sample of P. mirabilis are those involved in the virulence and hindering the antimicrobial mechanisms of this bacteria including bla NDM-1, and bla CMY [14] were not expressed. The expression of aclb [15], bla TEM, atpD, ureC, rsbA, zabA, bla CTX, and bla OXA-1 [22].

Table 2. Primers were used in this Study for Isolates

Primers	**Forward**	**Reverse**	**Annealing Temp°C**	**Product size(bp)**
magA	GGTGCTCTTTACATCATTGC	GCAATGGCCATTTGCGTTAG	49	1280
rmpA	ACTGGGCTACCTCTGCTTCA	CTTGCATGAGCCATCTTTCA	50	535
mrkD	CCACCAACTATTCCCTCGAA	ATGGAACCCACATCGACATT	43	226
uge	TCT TCA CGC CTT CCT TCA CT	GAT CAT CCG GTC TCC CTG TA	54	534
fimH-1	GCCAACGTCTACGTTAACCTG	ATATTTCACGGTGCCTGAAAA	43	180
blaKPC	TGTCACTGTATCGCCGTC	CTCAGTGCTCTACAGAAAACC	57	900
bla CTXM-1	GACGATGTCACTGGCTGAGC	AGCCGCCGACGCTAATACA	55	500
blaNDM-1	GGTGCATGCCCGGTGAAATC	ATGCTGGCCTTGGGGAACG	52	661
OXA	ATATCTCTACTGTTGCATCTCC	AAACCCTTCAAACCATCC	56	619

Table 3. Primers used in the Current Study for P. Mirabilis Isolates

Primers	Forward	Reverse	Annealing Temp ℃	Product size(bp)
aacIb	TGCGATGCTCTATGAGTGGCTA	CTCGAATGCCTGGCGTGTTT	54	482
blaTEM	TACGATACGGGAGGGCTTAC	TTCCTGTTTTTGCTCACCCA	52	716
atpD	GTATCATGAACGTTCTGGGTAC	TGAAGTGATACGCTCTTGCAG	58	595
ureC	GTTATTCGTGATGGTATGGG	ATAAAGGTGGTTACGCCAGA	94	317
rsbA	TTGAAGGACGCGATCAGACC	ACTCTGCTGTCCTGTGGGTA	94	467
zabA	ACCGCAGGAAAACATATAGCCC	GCGACTATCTTCCGCATAATCA	94	540
blaCTX-M	TTTGCGATGTGCAGTACCAGTAA	CGATATCGTTGGTGGTGCCATA	55	499
blaNDM-1	GGCGGAATGGCTCATCACGA	CGCAACACAGCCTGACTTTC	94	287
blaOXA-1	ATATCTCTACTGTTGCATCTCC	AAACCCTTCAAACCATCC	94	619
blaCMY	CGAAGAGGCAATGACCAGAC	TACAGTGCGGGTGGT	52	538

Table 3. PCR Thermo Cycling Condition for Isolates to Amplify the DNA

Gene	Initial denaturation	Denaturation	Annealing (Repeated cycles)	Extension	Final extension	Product size (bp)
Mag A	94℃, 3min	94℃, 1min	49℃, 45sec(35 cycles)	74℃, 1:30min	72℃, 3min	1280
rmp A	95℃, 3min	95℃, 45sec	50℃, 45sec(35 cycles)	72℃, 1min	72℃, 3min	535
OXA-1	94℃, 3min	94℃, 50sec	47℃, 45sec(35 cycles)	72℃, 1min	72℃, 3min	619
bla CTXM-1	94℃, 3min	94℃, 45sec	55℃, 45sec(35 cycles)	72℃, 1min	72℃, 3min	500
bla KPC	95℃, 3min	95℃, 1min	51℃, 45sec(35 cycles)	72℃, 1min	72℃, 3min	900
bla NDM-1	95℃, 3min	95℃, 45sec	56℃, 45sec(35 cycles)	72℃, 1min	72℃, 3min	661
Uge	94℃ 3min	94℃, 45sec	54℃, 45sec(35 cycles)	72℃, 1min	72℃, 3min	534
fim H-1	94℃, 3min	94℃, 30sec	48℃, 45sec(35 cycles)	72℃, 1min	72℃, 3min	180
Mrk D	95℃, 3min	94℃, 45sec	52℃, 45sec(35 cycles)	72℃, 1min	72℃, 3min	228

Table 4. PCR Thermo Cycling Condition for P. Mirabilis Isolates to Amplify the DNA

Gene	Initial denaturation	Denaturation	Annealing (Repeated cycles)	Extension	Final extension	Product size (bp)
aacIb	94℃, 3min	94℃, 45sec	55℃, 45sec(35 cycles)	72℃, 45sec	72℃, 4min	482
bla TEM	94℃, 3min	94℃, 45sec	52℃, 45sec(35 cycles)	72℃, 60sec	72℃, 4min	716
atpD	94℃, 3min	94℃, 45sec	50℃, 45sec(35 cycles)	72℃, 45sec	72℃, 4min	595
ureC	94℃, 3min	95℃, 45sec	48℃, 45sec(35 cycles)	72℃, 45sec	72℃, 4min	317
rsb A	94℃, 3min	94℃, 45sec	55℃, 45sec(35 cycles)	72℃, 45min	72℃, 4min	467
zabA	95℃, 3min	95℃, 45sec	52℃, 45sec(35 cycles)	72℃, 45sec	72℃, 4min	540
*bla*CTXM	94℃, 3min	95℃, 45sec	54℃, 45sec(35 cycles)	72℃, 45sec	72℃, 4min	543
*bla*NDM-1	94℃, 3min	94℃, 45sec	55℃, 45sec(35 cycles)	72℃, 45sec	72℃, 4min	287
*bla*OXA	94℃, 3min	94℃, 45sec	48℃, 45sec(35 cycles)	72℃, 45sec	72℃, 4min	619
*bla*CMY	94℃, 3min	95℃, 45sec	52℃, 45sec(35 cycles)	72℃, 45sec	72℃, 4min	538

Results

The present study has been designed to test the most common antibiotics used for the treatment of burn infection and/or prophylaxis, this includes imipenem (IPM), gentamycin (CN), sulphamethoxazole (SXT), pipracillin (PRL), cefixime (CFM), amoxycillin (AMC), trimethoprim (TMP), ciprofloxacin (CIP), cefotaxime (CTX), and amikacin (AK). The culture sensitivity test has confirmed that *P. mirabilis* is the most resistant to antibiotics showing MDR to 9 commonly used antibiotics in clinical settings. Moreover, *K. pneumoniae* has also shown MDR to 8 commonly used antibiotics for burn infection (Figure 1, Table 5).

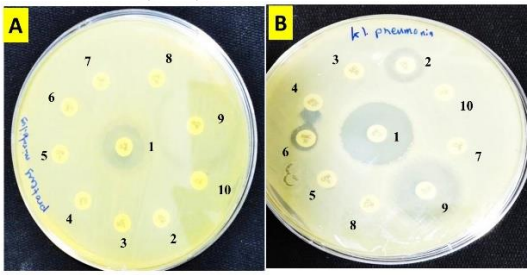

Figure 1. A Representative Image of the Antibiotic Inhibition Zone for (A) P. Mirabilis Showed MDR with Complete Resistance to 9 Types of Antibiotics. (b) K. Pneumoniae Shower MDR with Complete Resistance to 8 Types of Antibiotics. 1=Imipenem, 2=Gentamicin, 3=Sulfamethoxazole, 4=Pipracillin, 5=Cefixime, 6=Amoxycillin, 7=Trimethoprim, 8=Ciprofloxacin, 9=Cefotaxime, 10=Amikacin

To test the efficacy of commonly used antibiotics in clinical practice (Table 5), including imipenem (IPM), gentamycin (CN), sulphamethoxazole (SXT), pipracillin (PRL), cefixime (CFM), amoxycillin (AMC), trimethoprim (TMP), ciprofloxacin (CIP), cefotaxime (CTX), and amikacin (AK). Regarding *K. pneumoniae*, sensitivity was the highest with IMP (65%) and lowest with PRL (0%), TMP (0.6%), SXT (4.4%), CFM (8.2%), and AMC (15.8%). *K. pneumoniae* immediately responded to AK (45.7%), CTX (44.6%), CIP (34.8%), and CN (29.4%). Regarding *P. mirabilis*, sensitivity was the highest with IMP (100%) and lowest with PRL (4.6%), AK (9.1%), CN (12.2%), SXT (22.8%), and CFM (28.8%). *K. pneumoniae* immediately responded to CTX (78.8%), AMC (63.7%), TMP (39.4%) and CIP (30.4%).

Table 5. The Pattern of Resistance K. Pneumoniae and P. Mirabilis against Antibiotics

Antibiotics	**Conc used (µ/ml)**	**(184 isolates)**		***P. mirabilis* (66 isolates)**	
		Resistance	**Sensitive**	**Resistance**	**Sensitive**
		n (%)	**n (%)**	**n (%)**	**n (%)**
IPM	10	64 (34.8)	120(65.2)	0(0)	66(100)
CN	10	130(70.6)	54(29.4)	58(87.8)	8(12.2)
SXT	25	176(95.6)	8(4.4)	51(77.2)	15(22.8)
PRL	100	184(100)	0(0)	63(95.4)	3(4.6)
CFM	5	169(91.8)	15(8.2)	47(71.2)	19(28.8)
AMC	30	155(84.2)	29(15.8)	24(36.3)	42(63.7)
TMP	10	183(99.4)	1(0.6)	40(60.6)	26(39.4)
CIP	10	120(65.2)	84(34.8)	46(69.6)	20(30.4)
CTX	30	102(55.4)	82(44.6)	14(21.2)	52(78.8)
AK	10	100(54.3)	84(45.7)	60(90.9)	6(9.1)

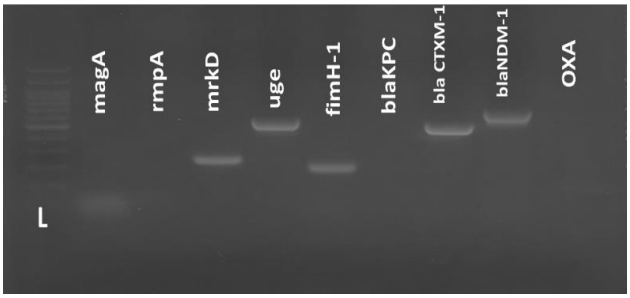

Figure 2. Products of PCR are Positive for MrkD, Uge, FimH, Ctx1, and NDM Resist Genes (lanes 3,4,5,7 and 8), Respectively. The PCR Results are Negative for MageA, rmpA, Kpc, and OxA1 Resist Genes (lanes 1,2,6 and 9), Respectively. Lane L is the DNA Ladder

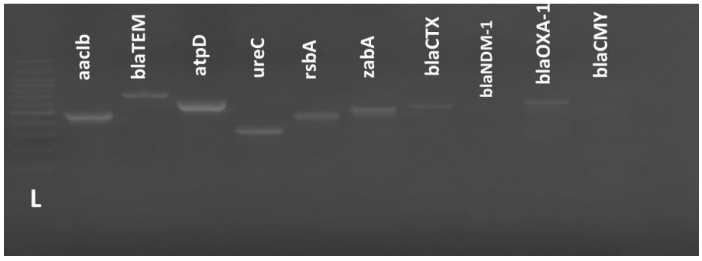

Figure 3. Products of PCR are Positive for P. Mirabilis aacIb, TEM, atp, Urec, rsbA, Zab, Ctx, and Oxa Resist Genes (Lanes 1-7 and 9), Respectively. PCR is Negative – for NAM and CMy Resist Genes (Lanes 8 and 10), Respectively. Lane L is the DNA Ladder

Discussion

Hospital-acquired infections are the most challenging issue for clinicians in the burn ward, due to the availability of optimum conditions for infection and bacterial colonization; which are responsible for three-quarters of mortality in burn patients admitted to the hospital [23]. The reason for this morbidity and mortality is the loss of protective skin surface integrity and vitiated immune responses, which prone the patients to opportunistic infections in areas with a high rate of exposure to a mixture of bacteria at the hospital [24]. Management of burn infection is crucial for saving patients' lives, however, managements need long-term therapy and multiple drug use; making the use of antibiotics challenging the fate of facing resistance bacteria to multiple drugs [9, 25]**.** Moreover, the isolation of new bacterial isolate from burn patients further complicates the condition making treatment with effective antibiotics impossible. The present study confirmed MDR of *K. pneumoniae* and *P. mirabilis*. These bacteria have been sensitive to imipenem. *K. pneumoniae*, was resistant for PRL (0%), TMP (0.6%), SXT (4.4%), CFM (8.2%), and AMC (15.8%). *P. mirabilis*, was resistant for PRL (4.6%), AK (9.1%), CN (12.2%), SXT (22.8%), and CFM (28.8%). Similar MDR has been reported in a previous Indian study [16, 26]. These resistances were related to the hospital environment which flourish bacterial growth and transmission [27, 28]. The percentage of resistance to drugs used in our study is nearly similar to other previous studies [1, 26, 29], however, the resistance in the present study was much lower than that conducted by Gupta et al. [16]. In an alternative study, *K. pneumoniae* and *P. mirabilis* respond weakly to imipenem [30].

The resistance of *K. pneumoniae* reported in the present study is matched with the global prevalence of 70% [31] with most cases being reported as an MDR [32]. The resistance is mainly due to plasmid genetic transmission of extended-spectrum beta-lactamase or carbapenemases hindering the management of

infections [33], increasing the challenges of healthcare providers and overburdening the health system due to increased the rate of hospitalization with an increased possibility of mortality rate [21]. In the present study, the sample of *K. pneumoniae* expressed mrkD, uge, fimH-, bla CTXM-1, and bla NDM-1, these genes are supposed they destroy the antibiotics and hence ensure antibiotic resistance. Nonetheless, magA, rmpA, bla KPC, and OXA were not expressed. The expression of mrkD, fimH-1, magA, rmpA [34–37], bla KPC, bla CTXM-1, bla NDM-1 [38–41], and urg genes [42] have been reported in previous studies. These genes together are responsible for the virulence and pathogenicity of *K. pneumoniae* in burn patients. Since, these factors help the bacteria from escaping the immune response, resist phagocytosis, inhibition of complement lysis, and opsonization [4, 6–8].

In the present study, the sample of *P. mirabilis* expressed aclb, bla TEM, atpD, ureC, rsbA, zabA, bla CTX, and bla OXA-1, these genes supposed that they evade the antibiotic therapy and thereby commence antibiotic-resistance. Nonetheless, bla NDM-1 and bla CMY [14] were not expressed. The expression of aclb [15], bla TEM, atpD, ureC, rsbA, zabA, bla CTX, and bla OXA-1 [22] genes have been corporated in previous studies. These genes participate in the infectivity of *P. mirabilis* in burn patients. Since, these factors help the bacteria from escaping the immune response, tackle phagocytosis, modulate the complement lysis, and formation of biofilm [6].

Conclusion

The study highlighted the great contribution of *K. pneumoniae* and *P. mirabilis* in burn infectivity. Resistance genes were expressed by both bacteria increasing the chance of virulence activity and antimicrobial resistance. These genes were determined to be involved in the production of toxic proteins and contribute to host damage hindering healing. The bacteria species isolated were highly resistant to multiple antibiotics, sensitivity was only high with imipenem.

Conflict of Interest

To authors declare no conflict of interest.

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