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ORIGINAL RESEARCH ARTICLE

Detection of Carbapenemase genes in MDR Environmental and Clinical *Klebsiella pneumoniae* isolates

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ABSTRACT

Background: Carbapenem-resistant *K. pneumoniae* (CRKP) as well as the emergence of other resistance genes such as extended-spectrum beta-lactamases (ESBLs). These bacteria are linked to complicated infection management and contribute to higher morbidity and mortality

Objectives: In this study, phenotypic and molecular analyses of multidrug and carbapenem resistance are performed in *Klebsiella pneumoniae* isolated from clinical and environmental sources in Baghdad, to determine and characterize the prevalence of resistance genes.

Methods: A total of 50 clinical isolates were collected from different hospitals and from water sources across various communities; 22 environmental isolates were obtained in Baghdad. The study underscores the extensive occurrence of multidrug-resistant *K. pneumoniae* in both healthcare and environmental contexts in Baghdad. (56%) and (31.8%) of clinical and environmental carbapenem-resistant *Klebsiella pneumoniae* isolates, respectively, were metallo-β-lactamase (MBL) producers.

Results: Environmental isolates revealed high resistance to ceftazidime (90%) and ampicillin (100%), with *bla*NDM (17%) and *bla*$_O$XA-48 (12 %) genes being the most prevalent resistance genes. Carbapenem resistance genes, *bla*NDM and *bla*OXA-48 genes, were detected in 19% and 14% of clinical isolates, respectively, whereas the *blaI*MP gene was not detected.

Conclusions: These results highlight the critical importance of strengthening surveillance systems, infection control practices, and antimicrobial stewardship programs to control the spread of resistant *K. pneumoniae* strains.

Keywords: **Carbapenem genes, Environment, Water, *Klebsiella pneumoniae***

1. INTRODUCTION

A primary cause of nosocomial infections, including pneumonia, blood infections, and urinary tract infections, is *Klebsiella pneumoniae*. Also, complicated situations in clinical practice due to rapid development and spread of carbapenem-resistant *K. pneumoniae* (CRKP) as well as emergence of other resistance genes such as extended-spectrum beta-lactamases (ESBLs) in this bacterium are linked to complicated infection management and contribute to the higher morbidity and mortality [1-2]

Within the One Health framework, particularly the role of wastewater systems spreading of resistant microorganisms is increasingly recognized for the environmental dimension of antibiotic resistance. This reveals the role of human, animal, and environmental health in the prevalence of antimicrobial resistance (AMR)[3]. Wastewater systems originating from human, agricultural, and industrial sources serve as convergence points for antibiotics, antibiotic-resistant bacteria (ARB), and antibiotic resistance genes (ARGs) [4].

Soil and water serve as important contributors to the spread of AMR genes, which act as environmental reservoirs [5]. Carbapenemase enzymes, mainly New Delhi metallo-β-lactamase (NDM), OXA-48-like enzymes, and, less frequently, IMP-type carbapenemases facilitate *K. pneumoniae* resistance to carbapenem [6]. IMP-type enzymes like zinc-dependent metallo-β-lactamase NDM that hydrolyze most β-lactams, and OXA-48 enzymes are class D β-lactamases with changing carbapenem activity. Bearing in mind that these enzymes are not yet common, they also play a role in carbapenem resistance [7].

Klebsiella isolates of environmental origin have been described by several studies to be nearly identical to clinical isolates with respect to several phenotypic properties, but the pathogenic potential of environmental *K. pneumoniae* isolates is essentially unknown [8]. The critical antibiotic resistance prevalence and dissemination in water and sludge environments necessitate a comprehensive understanding; this study aimed to determine antimicrobial resistance phenotypes and resistance gene prevalence in *K. pneumoniae* isolates from clinical and environmental sources of Baghdad by phenotypic antibiotic susceptibility testing and PCR-based molecular detection of resistance genes.

2. MATERIALS AND METHODS

2.1 Bacterial Isolates

A total of 50 clinical *Klebsiella pneumoniae* isolates were collected from patients admitted to different hospitals in Baghdad, and 22 environmental isolates were collected from water. Water samples were cultured to detect bacterial isolates. The pour-plate method was used for the enumeration and isolation of bacteria from polluted water samples according to the procedures described in Standard Methods for the Examination of Water and Wastewater

2.1.2 Bacterial Identification

The inoculated plates were incubated aerobically at 37°C for 24 hrs. Following incubation, bacterial colonies recovered from Nutrient agar were selected and subcultured on MacConkey agar. The MacConkey agar plates were incubated at 37°C for 24 hrs to recover presumptive Gram-negative bacterial isolates. Initial identification was performed using conventional bacteriological methods, as well as the VITEK-2 compact system (bioMérieux, France).

2.2. Antimicrobial Susceptibility Testing

The following (12) antimicrobial agents discs were used: imipenem (IMI 10 µg), ceftazidime (CAZ 30 µg), cefepime (FEP 30 µg), meropenem (MRP 10µg), tetracycline (TE 30 µg), ceftriaxone (CRO 30 µg), Amikacin (AK 30 µg), gentamicin (CN 10 µg), Ampicillin (AMP 10 µg), ciprofloxacin (CIP 5 µg), Trimethoprim-Sulfamethoxazole (SXT 25 µg), Nitrofurantoin (F 300 µg). A 0.5 McFarland bacterial suspension was inoculated using a cotton swab onto Mueller-Hinton agar. Then the

antibiotic discs were deposited, after 24 hrs of incubation at 37°C. The critical diameter was determined after the reading was done, and the strains were classified as resistant, intermediate, or susceptible according to Clinical and Laboratory Standards Institute (CLSI) guidelines [8].

2.3 Detection of Carbapenemase enzymes

Modified carbapenem inactivation method (mCIM) was used to identify bacteria resistant to carbapenems and producing carbapenemases [9]. A single colony of *K. pneumoniae* was transferred into a tryptic soy broth tube, and a meropenem disc was placed to conduct the test. The meropenem disc is placed on a Mueller-Hinton agar plate that has been infected with meropenem-susceptible *E. coli* ATCC 25922 during a 4 hrs incubation period at 37°C. To ascertain the outcome, the diameter of the inhibitory zone surrounding the disc is measured. For the synthesis of carbapenemase, a zone diameter of 6–15 mm is positive, 16–18 mm is moderate, and ≥19 mm is negative.

2.3.1 Molecular Identification of Carbapenemase Genes

The boiling method was used for DNA extraction [10]. 2-4 single colonies of *K. pneumoniae* were resuspended in 0.5 mL of distilled sterile water in a 1.5 mL Eppendorf tube. A boiling water bath was subsequently used to heat the Eppendorf tube to 100 °C for 10 minutes, followed by immediate centrifugation at 14,000 rpm for 10 minutes in a cooling centrifuge at 4 °C. Amplification was performed on the supernatant containing DNA.

*2.3.2. Molecular Identification of bla*NDM, *blaOXA-48, and bla*IMP

All clinical and environmental *K. pneumoniae* isolates were tested by PCR using oligonucleotide primers specific for carbapenem resistance genes as shown in Table 1. Genes were amplified by polymerase chain reaction (PCR) using a thermal cycler. The reaction mixture consisted of a total volume of 25 µL in Table 2. Thermal cycling conditions were optimized for each gene. For *bla*NDM, amplification was included in Table 3. For blaOXA-48 and *bla*IMP, amplification as shown in Table 4. After PCR amplification, PCR products were electrophoresed on 1.5% agarose gel for 60 min at 50V-100V in 0.5X TBE buffer, and 5 µL of Red Safe stain in the Gel tank. Amplified DNA bands were visualized using a UV transilluminator

Table 1. Primers used for the detection of carbapenemase genes in *K. pneumoniae* isolates

Primer	Sequence (5'to 3')	Product Size (bp)	Reference
NDM	F: TGGCAGCACACTTCCTATC R: AGATTGCCGAGCGACTTG	488	11
OXA-48	F: GCGTGGTTAAGGATGAACAC R: CATCAAGTTCAACCCAACCG	438	13
IMP	F: GGAATAGAGTGGCTTAAYTCTC R: GGTTTAAYAAAACAACCACC	232	14

Table 2. The PCR mixture contents and volumes.

NO	Content	Volume (µL)
1	Master Mix	12.5
2	Forward primer	1
3	Reverse Primer	1
4	nuclease-free water	5.5
5	template DNA	5

Table 3. Thermal cycling conditions for *bla*NDM, gene amplification for 35 cycles.

Steps		Temp.(ºC)	Time
Initial Denaturation		95	5 min
Each cycle	Secondary Denaturation	95	35 sec
	Annaeling	58	35 sec
	Extension	72	40 sec
Final Extension		72	10 min

Table 4. Thermal cycling conditions for blaOXA-48 and *bla*IMP gene amplification for 36 cycles.

Steps		Temp.(ºC)	Time
Initial Denaturation		94	10 min
Each cycle	Secondary Denaturation	94	30 sec
	Annaeling	52	40 sec
	Extension	72	50 sec
Final Extension		72	5 min

3. RESULTS

A total of 50 clinical *Klebsiella pneumoniae* isolates and 22 from the environment were identified. The majority of isolates were from urine samples (33.04%), followed by blood (22.6%), sputum (20.86%), and wound swabs (10.43%). Table 5 includes the main Biochemical test results for the identification of the clinical and environmental *K. pneumoniae* isolates

Table 5. Biochemical test results for the identification of the clinical and environmental *K. pneumoniae* isolates

Biochemical test	Result
Glucose fermentation, Catalase, Citrate utilization, Voges-Proskauer (VP), Urease	Positive
Oxidase, Methyl red (MR), H_2S production, indole, PDA	Negative

The antimicrobial susceptibility test showed that 48% and 36.3% of clinical and environmental *K. pneumoniae* isolates, respectively, were multidrug-resistant (MDR). High resistance rates in clinical isolates were observed for Ampicillin (98%), Cefepime (80%), and Ceftazidime (86%). In addition, resistance to imipenem and meropenem was identified in (68% and 66%) of isolates, respectively as shown in Table 6. The susceptibility of water isolates of *K.pneumoniae* was as follows: Nitrofurantoin (68%), Ampicillin (100%), Ceftazidime (90%), Gentamicin (45.4%), and tetracycline (40.9%), as seen in Table 6. 63.6% of environmental isolates were found to be imipenem-resistant *K. pneumoniae*.

Table 6: Antimicrobial susceptibility profile of clinical and environmental *K. pneumoniae* isolates.

Antimicrobial Class	Antimicrobial agents	resistance (%) In clinical isolates	Resistance (%) in environmental isolates
$\beta - lactam$	Imipenem (IMI)	(68 %)	(63.6%)
	Meropenem (MRP)	(66%)	(59%)
	Ceftazidime (CAZ)	(86%)	(90%)
	Ampicillin (AMP)	(98%)	(100%)
	Ceftriaxone (CRO)	(80%)	(81.8%)
	Cefepime (FEP)	(80%)	(77.2%)
Nitrofurans	Nitrofurantoin (F)	(74%)	(68%)
Tetracyclines	Tetracycline (TE)	(66%)	(40.9%)
Quinolones	Ciprofloxacin (CIP)	(70%)	(72.7%)
Sulfonamides	Trimethoprim-sulfamethoxazole (SXT)	(82%)	(40.9%)
Aminoglycosides	Amikacin (AK)	(60%)	(45.4%)
	Gentamicin (CN)	(56%)	(45.4%)

The mCIM assays were used to detect phenotypic carbapenemase production. 56% and 31.8 % of clinical and environmental carbapenem-resistant Klebsiella pneumoniae isolates, respectively, were metallo-β-lactamase (MBL) producers .

In clinical isolates he amplification of *bla*NDM (19%) and *bla*OXA-48 (14 %) genes was confirmed by agarose gel electrophoresis, and *bla*NDM (17%) and *bla*OXA-48 (12 %) in environmental isolates as seen in Figure 1. Whereas molecular analysis showed that all *K. pneumoniae* isolates were negative for the *bla*IMP gene.

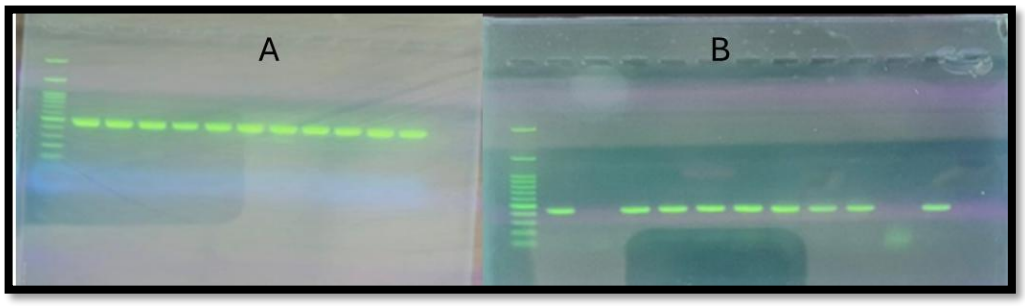

Figure 1: Agarose gel electrophoresis of PCR amplification of the *bla*NDM (a) *bla*OXA-48 (b) genes in *K. pneumoniae* isolates. L: DNA ladder (100-3k bp); C: negative control

4. DISCUSSION

The distribution of most clinical isolates was from urine; these results are partially consistent with the study results by [11] and [12], which reported that *K. pneumoniae* was the most common pathogen among clinical samples isolated in urinary and respiratory infections. This can be attributed to the capacity of the organism to develop biofilms and colonize the urinary tract, leading to persistence and resistance. 63.6% of environmental isolates were found to be imipenem-resistant *K. pneumoniae*.

This incidence seems much higher than [13] reported, 8% of clinical isolates were carbapenem-resistant. This variation can be credited to either of the two sources of selective pressure, and local use

of antibiotics, all of which have been known to enhance the prevalence and persistence of carbapenem-resistant strains in the clinical setting.

The antimicrobial resistance profiles found in the current study are consistent with previous reports. Moustafa *et al*. [14] found 100% resistance to ampicillin and a high level of resistance to ceftriaxone and ceftazidime, whereas Mermer and Çağlayan [15] had similarly reported carbapenem resistance rates also similar to our findings. Similarly, Tsolakidou *et al.* [16] showed high resistance to trimethoprim-sulfamethoxazole and colistin, also being consistent with the high levels observed in this study. Barkhordari *et al*., [17], found that CRKP isolates were highly resistant to cefepime, ciprofloxacin, and trimethoprim-sulfamethoxazole, and less resistant to amikacin and piperacillin-tazobactam, again similar to the moderate resistance to aminoglycosides.

Sewage often acts as channel and reservoir for antibiotics and its active antibiotic metabolites. Hence, it plays a pivotal role in dissemination of antibiotic resistance. Before environmental discharge inadequate treatment of such effluents enables the release of ARGs and ARB into the natural water bodies, promoting their integration into environmental microbiomes [18].

Previous studies have shown a abundance and higher diversity of ARGs in hospitals effluents compared to other water sources, underscoring the critical need for surveillance and management strategies targeting such resistance point sources. For example, a study showed that highest diversity of ARGs in effluent (1,520 unique genes) compared with lake water and river water (247 unique genes), highlighting the effluents role as reservoirs of ARGs [19]. Water surveillance offers a cost effective strategy to identify emerging trends of resistance and evaluate the mitigation measures effectiveness [20].

Coexistence of carbapenemase-producing mechanisms was also observed. Studies by [21], and [22] revealed that serine-β-lactamases and metallo-β-lactamases were found in the clinical isolates. A major clinical problem with such enzyme classes is their co-occurrence, which expands the range of β-lactam hydrolysis and constrains the treatment choices.

The detection of carbapenem resistance genes in environmental isolates underscores the role of environmental reservoirs in the spread of AMR. The *bla*NDM and *bla*OXA-48 genes are prevalent in the present study, which is in line with [23] and [24], indicating the ubiquitous nature of the identified genes in the Middle East. The lack of *bla*IMP is also consistent with findings by [25], indicating that this gene is not as widespread as NDM and OXA-48.

5. CONCLUSION

The study offers important lessons about the local epidemiology of AMR and the need for proactive measures to help control the environmental spread of resistant pathogens and maintain the effectiveness of the last-resort antibiotics. In particular, routine monitoring of hospital effluents and upgrading wastewater treatment infrastructure are critical actions to take to combat this emerging public health threat.

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CONFLICTS OF INTEREST

The authors declare no conflict of interest.

AUTHOR CONTRIBUTIONS

M.F.A. conceptualized the study and wrote the whole manuscript. H.YA. contributed to the isolation and identification of isolates. H.K.N provided the necessary primers for molecular detection of antimicrobial resistance genes. M.M.K. conducted the molecular experiments, and S.Q.M. conducted the isolation experiments. All authors reviewed and approved the final version of the manuscript.

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DATA AVAILABILITY STATEMENT

All data are included in the manuscript

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Ethical approval for the study was secured from the Clinical Laboratory/Microbiology Department at the College of Science, Mustansiriyah University

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