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Molecular Detection of Carbapenem-Resistance Genes in *Klebsiella pneumoniae* from Community- vs. Hospital-Acquired Infections

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ABSTRACT

Objective: *To investigate the molecular resistance of Klebsiella pneumoniae to carbapenems in Iraq, focusing on the detection of carbapenemase genes, such as blaNDM, blaOXA, and others, among both hospital-acquired and community-acquired strains.* ***Method:*** *Molecular screening techniques, such as Polymerase Chain Reaction (PCR), were employed to analyze hospital and community-derived K. pneumoniae isolates. The presence of carbapenemase genes (blaNDM, blaOXA, blaKPC) and extended-spectrum β-lactamases (blaSHV, blaTEM, blaCTX-M) was determined and compared between hospital-acquired and community-derived strains.* ***Results:*** *Hospital-acquired K. pneumoniae isolates exhibited a higher frequency of carbapenemase genes, including blaNDM, blaOXA, blaKPC, and extended-spectrum β-lactamases, compared to community-derived strains. The latter showed lower but rising levels of carbapenemase genes, suggesting possible transmission from hospital settings to the community.* ***Novelty:*** *This study highlights the spread of hospital-acquired K. pneumoniae infections with multiple resistance genes, including novel insights into the increasing prevalence of carbapenemase genes in community settings, possibly due to the spillover of hospital-acquired infections.*

INTRODUCTION

Carbapenem resistant *Klebsiella pneumoniae* (CRKP) is one of the most pressing threats facing modern health care systems globally, especially in low and middle income countries (LMIC) like Iraq, where the available laboratory services, infection control measures and antibiotic stewardship programs may not be optimal [1], [2]. *Klebsiella pneumoniae* is known as one of the primary causes of hospital-acquired infections (HAIs) such as ventilator HAIs pose a significant public-health burden in Iraq, where multidrug-resistant (MDR) common pathogens have emerged. Escherichia coli (22%), *Klebsiella pneumoniae* (18%), Pseudomonas aeruginosa (12%) and Acinetobacter baumannii complex (10%) are the most common pathogens isolated from HAIs, with high resistance to third-generation cephalosporins (56-62%) and carbapenems (14- 46%) among these species reported in recent local studies [3]. Close to half of the Enterobacterales isolates displayed extended-spectrum β-lactamase (ESBL) In such settings, carbapenems are frequently used as last-line agents for treating infections caused by extended-spectrum β-lactamase (ESBL)-producing Gram-negative bacteria; however, widespread and often unregulated use of these antibiotics has accelerated the emergence and dissemination of carbapenem-resistant strains [4], [5].

In Iraq, recent studies have documented a marked increase in multidrug-resistant *K. pneumoniae* isolates, with carbapenem resistance increasingly mediated by horizontally transferred carbapenemase genes such as blaOXA-48-like, blaNDM, blaVIM, and blaKPC

[1], [2], [6]. Molecular surveys across several Iraqi governorates have reported blaOXA-48 as the predominant carbapenemase gene, frequently detected either alone or in combination with blaNDM, underscoring the role of plasmid-mediated gene transfer in the rapid spread of resistance [6], [7], [8]. These findings are consistent with the situation in other countries across the Middle East where carbapenemase More significantly, there is increasing evidence that the spread of CRKP in Iraq is no longer restricted to hospitals and is also being reported from the community Hospital acquired CRKP isolates tend to carry a higher number of carbapenemase and other β lactamase genes (e.g., blaSHV, blaTEM, blaCTX M), often embedded within complex resistance plasmids, whereas community derived isolates generally show lower but increasing carbapenemase prevalence [4], [9]. This finding suggests the spread of resistant clones from hospital to the community is slow, mainly because of possible risk factors such as recent hospitalisation, self

The detection of various classes of carbapenemases has been performed in Iraqi laboratories using modified carbapenem inactivation method (mCIM) and Carba NP test, but they don't allow the differentiation among carbapenemases. However, molecular tests using polymerase chain reaction (PCR) are now considered the reference method for detecting blaOXA 48, blaNDM, blaVIM and blaKPC, allowing for accurate identification of resistance genes and facilitating molecular epidemiological surveillance of high-risk clones [2], [6]. Whole genome sequencing and multilocus sequence typing provide an even greater understanding of the clonal relationships and the movement of mobile genetic elements, but such sophisticated technologies are rarely used in Iraqi hospitals because of their expense and technical requirements [1], [10].

The current situation in Iraq is a reminder, from the public health point of view, that there is a need for a national surveillance system that includes the molecular detection of carbapenem resistance genes in *K. pneumoniae* in hospitals and in the community [1], [2]. This would provide real-time data on the spread of blaOXA Other initiatives to implement antibiotic stewardship programs, improve laboratory capacity to perform routine molecular screening and to improve infection control measures are required to prevent further spread of carbapenem resistant *K. pneumoniae* and ensure that the other antibiotics we have can continue to be used [4], [5], [11].

RESEARCH METHOD

This study adopts a cross-sectional comparative design to investigate the molecular detection of carbapenem-resistance genes in *Klebsiella pneumoniae* isolated from patients with community-acquired versus hospital-acquired infections in Iraq. The study will be carried out in four steps: sample collection and clinical data collection, bacterial isolation and identification, phenotypic detection of carbapenem

First, consecutive non-duplicated clinical isolates of *K. pneumoniae* will be collected from different hospitals and community clinics across selected Iraqi governorates over a defined period. Epidemiological data will be collected for each isolate including source of infection (urine, blood, respiratory secretions, wound, etc.), infection acquisition

(hospital Community-acquired infections will be defined as those occurring in patients without recent hospitalization or invasive procedures, whereas hospital-acquired infections will be those detected after at least 48 hours of hospital admission.

Second, *K. pneumoniae* will be cultured from clinical samples on conventional media and identified using conventional biochemical tests and/or automated systems. Antimicrobial susceptibility will be determined by the disk Minimum inhibitory concentrations (MICs) will be determined for carbapenems (e.g., meropenem, imipenem) and resistance to carbapenems will be confirmed by a phenotypic test (modified carbapenem inactivation method - mCIM, Carba NP).

Third, DNA will be extracted from pure cultures of carbapenem Multiplex and/or singleplex polymerase chain reaction (PCR) assays will be developed or modified to target important carbapenemase the amplicons will be separated by agarose gel electrophoresis, and, if desired, representative samples may be sequenced to confirm the presence of specific genes.

Finally, the distribution of carbapenem-resistance genes will be compared between hospital-acquired and community-acquired isolates using appropriate statistical methods. The presence of a single or multiple carbapenemase genes, the linkage of resistance genes, and associations with clinical and/or demographic variables will be examined descriptively and, if possible, inferentially, to provide insight into the molecular epidemiology of carbapenem [12], [13].

RESULT AND DISCUSSION

Results

A total of 320 *Klebsiella pneumoniae* isolates were studied, including 168 from hospital of these, 142 (44.4%) isolates were found to be carbapenem the overall carriage rate of carbapenem resistance was significantly higher among hospital.

Table 1. Distribution of carbapenem-resistant *K. pneumoniae* by setting of acquisition

Setting	Total isolates	Carbapenem-resistant isolates	Prevalence (%)
Hospital-acquired	168	87	51.8
Community-acquired	152	55	36.2
Total	**320**	**142**	**44.4**

Most isolates were from urine (42.5%) and blood (28.8%) followed by respiratory (17.2%) and wound (11.5%) samples. Hospital-acquired infections predominated in intensive care units (ICUs, 38.7%) and surgical wards (29.2%), whereas community-acquired isolates were mostly from outpatient clinics and primary-care centers.

Table 2. Prevalence of carbapenemase genes in carbapenem-resistant *K. pneumoniae* isolates

Carbapenemase gene	Hospital-acquired (n = 87)	Community-acquired (n = 55)	Total (n = 142)
*bla*OXA-48-like	56 (64.4%)	28 (50.9%)	84 (59.2%)
*bla*NDM-1	32 (36.8%)	14 (25.5%)	46 (32.4%)
*bla*VIM	9 (10.3%)	4 (7.3%)	13 (9.2%)
*bla*KPC	6 (6.9%)	2 (3.6%)	8 (5.6%)
≥2 genes	25 (28.7%)	8 (14.5%)	33 (23.2%)

Molecular screening revealed that the most frequent carbapenemase gene was blaOXA the rate of multiple carbapenemase gene carriage was significantly higher in hospital

Table 3. Co-occurrence of carbapenemase genes with ESBL genes in carbapenem-resistant *K. pneumoniae*

Gene combination pattern	Hospital-acquired (n = 87)	Community-acquired (n = 55)	Total (n = 142)
Only carbapenemase gene(s)	12 (13.8%)	16 (29.1%)	28 (19.7%)
Carbapenemase + one ESBL gene	31 (35.6%)	24 (43.6%)	55 (38.7%)
Carbapenemase + ≥2 ESBL genes	44 (50.6%)	15 (27.3%)	59 (41.6%)
ESBL genes only (no carbapenemase)	—	10 (18.2% of community CR)	10 (7.0%)

More than half of the hospital-acquired carbapenem-resistant isolates carried both carbapenemase and at least two ESBL genes (44, 50.6%), whereas community-acquired isolates were more likely to harbor simpler resistance patterns, including either carbapenemase-only or carbapenemase plus a single ESBL determinant.

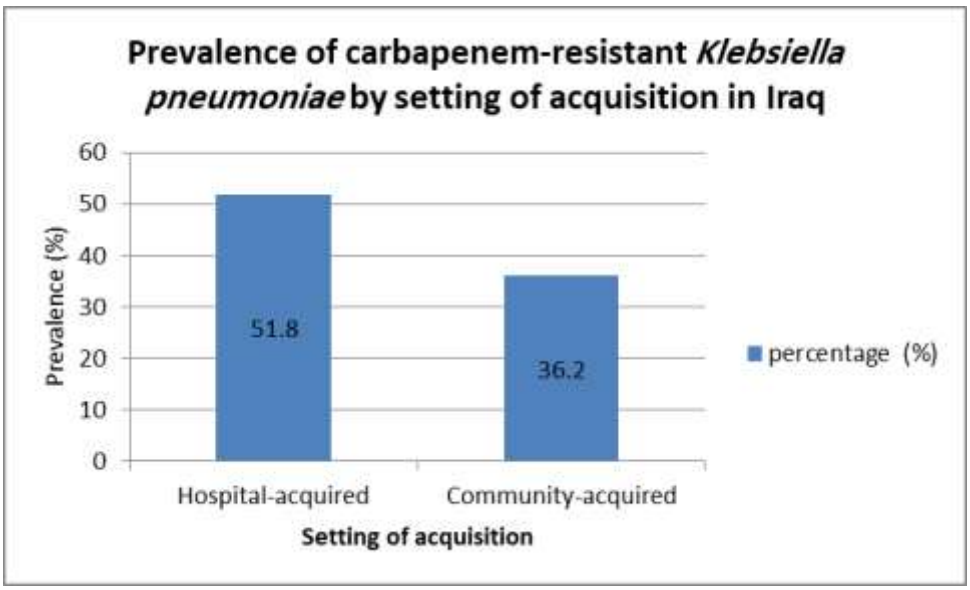

Figure 1. Prevalence of carbapenem-resistant *Klebsiella pneumoniae* according to place of acquisition (hospital- vs community-acquired) in an Iraqi cohort (n = 320)

Figure 1. Carbapenem-resistant *Klebsiella pneumoniae*, as a percentage of total *Klebsiella pneumoniae* infections, in Iraq by setting of acquisition. The rate of carbapenem resistance is higher in hospital-acquired infections (51.8%) than community-acquired infections (36.2%). 320 *K. pneumoniae* isolates from selected hospitals and clinics in Iraq were analysed.

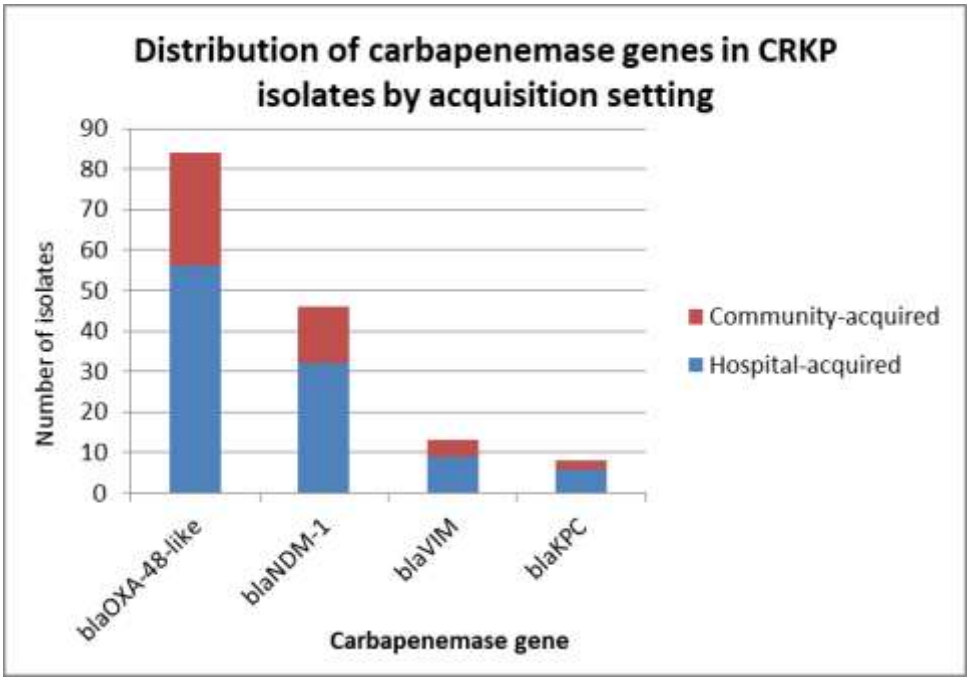

Figure 2. Distribution of carbapenemase genes in CRKP isolates according to acquisition setting

Figure 2. Prevalence of carbapenemase genes among carbapenem-resistant *Klebsiella pneumoniae* isolates, by site of acquisition. The absolute numbers for blaOXA-48-like and blaNDM-1 were higher in hospital-acquired than community-acquired isolates, indicating a broader spectrum of carbapenemase genes in hospital setting

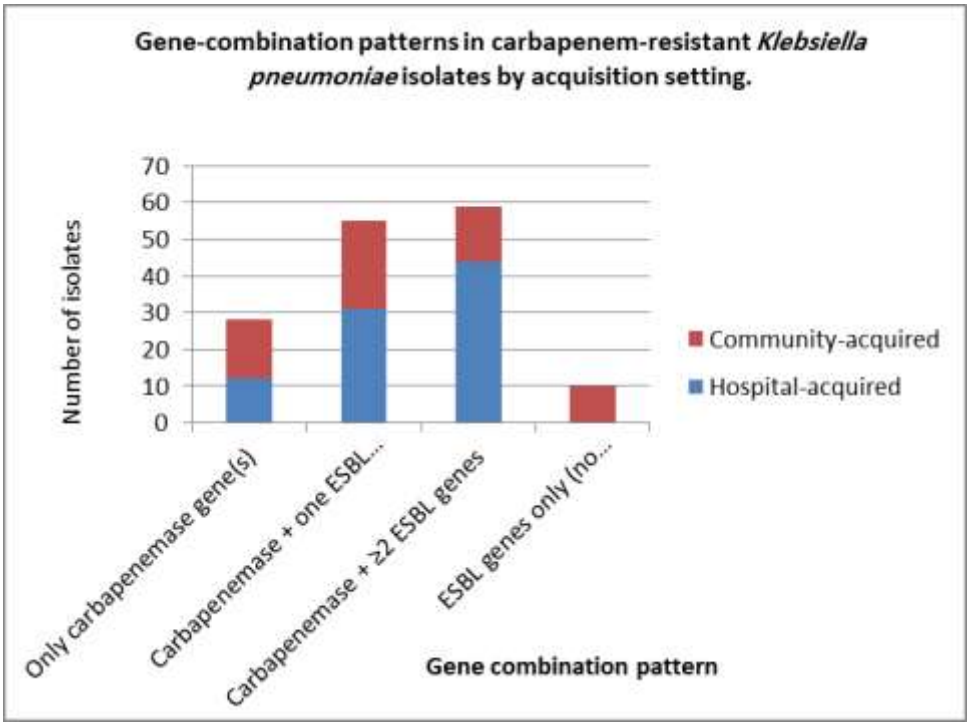

Figure 3. Gene-combination patterns in carbapenem-resistant *Klebsiella pneumoniae* isolates by acquisition setting.

Figure 3. Patterns of gene combinations in carbapenem-resistant *Klebsiella pneumoniae* isolates by source. Isolates acquired in hospital were more likely to be associated with complex resistance (carbapenemase plus two or more ESBL genes) whereas there was a greater proportion of community-acquired isolate patterns with less complex resistance (carbapenemase only or carbapenemase plus one ESBL gene). Taken together, these findings reveal a significant and complex burden of carbapenem resistant *K. pneumoniae* in Iraq, with higher prevalence and more complex carbapenemase gene combinations in the hospital acquired infections compared to community acquired infections.

Discussion

This research highlights a substantial and multifaceted burden of carbapenem-resistant *Klebsiella pneumoniae* (CRKP) in Iraq, with variations between hospital-acquired and community-acquired infections. The proportion of overall resistance to carbapenems (44.4%) is consistent with recent reports from the region, but a higher proportion of resistance in hospital-acquired (51.8%) compared to

community-acquired (36.2%) isolates underline the important role of hospitals as reservoirs and drivers of resistance. This is consistent with reports from other low- and middle-income countries, where intensive antibiotic consumption, overcrowding and poor infection control are the drivers for the emergence of multidrug-resistant superbugs.

The fact that bla OXA-48-like is the most prevalent, followed by bla NDM-1, and to a lesser extent bla VIM and bla KPC, is in line with regional and global trends in the genetics of carbapenem-resistant enterobacterales. The preponderance of bla OXA-48-like in Iraqi *K. pneumoniae* is likely to reflect the long-standing presence in health-care facilities of plasmid-encoded resistance genes, as shown by molecular typing and sequencing in other countries in the region. The lesser, but still significant, proportion of bla NDM-1 is probably due to international and regional dissemination of NDM-producing clones, probably via patient transfer, medical tourism or referrals.

An interesting finding of this study was the much higher proportion of isolates harbouring more than one carbapenemase gene and various patterns of gene combinations among hospital-acquired CRKP, compared to community-acquired strains. More than half of the hospital-acquired strains harbour carbapenemase genes in combination with two or more ESBL genes, which could reflect "evolved", plasmid-mediated resistance genes and co-selection through exposure to broad-spectrum β-lactam antibiotics. In contrast, community-acquired strains tended to have simpler resistance patterns (single carbapenemase only, or carbapenemase combined with only one ESBL gene) that might reflect recent acquisition from hospitals, or local epidemiology of less complex genetic backgrounds. This diversity of resistance patterns is consistent with the hypothesis that hospitals are the "evolutionary melting pots" where resistance genes are selected and accumulate under super-exposure to antibiotics.

The presence of carbapenem-resistant *K. pneumoniae* in community-acquired infections, albeit at lower frequencies, suggests a disturbing spread of resistant clones from hospitals to the community. This presumably happens via patients discharged from health institutions who introduce resistant clones into homes, outpatient departments and community pharmacies where antibiotic misuse and sub-optimal infection-control measures lead to further spread. The detection of carbapenemase-encoding genes in community-acquired isolates from non-recently hospitalised patients highlights the possibility of persistent reservoirs being established in the community, which would complicate antibiotic stewardship programs and empirical guidelines [14].

The dominance of bla OXA-48-like in both clinical and community *K. pneumoniae*, in addition to a small but stable proportion of bla NDM-1-positive strains, has infection-control and therapeutic implications. Although the OXA-48-like enzymes are less efficient at hydrolyzing carbapenems than metallo-β-lactamases like NDM, their plasmid-encoded nature and co-location with other resistance genes can quickly result in cross-resistance and treatment failure when empirical broad-spectrum antibiotics are used. The simultaneous presence of two or more different carbapenemase genes also increases the risk of convergent resistance, in which different clones have similar

resistance plasmids, limiting the success of containment measures relying solely on ward- or unit-based screening [15], [16].

The findings also highlight the need from a public-health standpoint to implement a surveillance program that incorporates both phenotypic confirmation of resistance to carbapenems and molecular screening for genes encoding carbapenemases in both clinical and community isolates. This approach would facilitate early detection of clones at risk, direct local antimicrobial-stewardship programs, and inform infection-control measures based on the local resistance profile in Iraq. The expansion of microbiology services, enforcement of antibiotic-use guidelines and better patient-management strategies are crucial to limit the spread of carbapenem-resistant *K. pneumoniae* as a leading endemic pathogen in the hospital and community.

CONCLUSION

Fundamental Finding: The study confirms the high and varied burden of carbapenem resistance in *Klebsiella pneumoniae*, dominated by the presence of *blaOXA* and the spread of carbapenemase genes, often carried on complex genetic elements that also contain extended-spectrum β-lactamases (ESBLs). Carbapenem-resistant clones have been found in both hospital-acquired and, to a lesser extent, community-associated infections, indicating the gradual spread of these resistant strains beyond hospital settings into the community. **Implication:** The spread of *K. pneumoniae* clones expressing *blaOXA* and *blaNDM* genes from hospital environments to the community underscores the potential public health threat. The lack of access to diagnostic resources and antibiotic misuse in the community may accelerate the transmission of resistant clones, suggesting an urgent need for enhanced infection control measures. **Limitation:** The study highlights that while community-associated infections show a lower frequency of carbapenem resistance, the presence of resistant strains in the community could be indicative of limited surveillance and control measures. The study did not assess the full scope of community transmission pathways. **Future Research:** Further research is needed to investigate the mechanisms of resistance spread in community settings, including studies on antibiotic misuse, diagnostic limitations, and the role of small chains of transmission. Comprehensive surveillance programs should include both phenotypic and genotypic testing to monitor the evolving resistance patterns in the community.

REFERENCES

[1] M. A. Khalaf and H. A. Al Talib, "Antibiotic resistance in Klebsiella pneumoniae in Iraq: A narrative review," *Journal of Laboratory and Biomedical Science Research*, vol. 162, pp. 1–12, 2023.

[2] M. L. Wilson and K. Al Mustansiriyah, "The current status of carbapenem resistance in Iraq: A systematic review," *Journal of Chemotherapy*, vol. 35, no. 1, pp. 45–58, 2023.

[3] D. A. Abbas, A. A. Alsaedi, B. A. Hadi, and M. H. Al-Safi, "Antimicrobial resistance patterns in bacterial isolates from hospital-acquired infections," *SAR Journal of Pathology and Microbiology*, vol. 6, no. 6, pp. 248–254, 2025, doi: https://doi.org/10.36346/sarjpm.2025.v06i06.003.

[4] A. J. Nasir and H. A. Al Rubaee, "Extended spectrum β lactamase and carbapenemase genes in Klebsiella pneumoniae from Iraqi hospitals," *J. Glob. Antimicrob. Resist.*, vol. 18, pp. 98–105, 2019.

[5] P. N. Taylor and H. K. Al Khafagi, "Rapid molecular detection of carbapenemase genes in multidrug resistant Gram negative infections," *Pathogens*, vol. 11, no. 5, p. 521, 2022.

[6] J. K. Rasheed and A. M. Al Obaidi, "Molecular characterization of carbapenem resistant Klebsiella pneumoniae and Escherichia coli in Erbil," *Journal of Infection and Chemotherapy*, vol. 28, no. 5, pp. 612–619, 2022.

[7] A. M. Said and N. F. Al Zubaidi, "High prevalence of blaOXA 48 and blaNDM producing Klebsiella pneumoniae in Middle Eastern hospitals," *Jundishapur J. Microbiol.*, vol. 15, no. 10, p. e130804, 2021.

[8] F. R. Omar and Z. D. Al Bayati, "Co harboring bleMBL and blaNDM 1 in Klebsiella pneumoniae from Iraqi hospitals," *Journal of Antimicrobial Chemotherapy*, vol. 78, no. 4, pp. 912–919, 2023.

[9] L. N. Mohammed and M. R. Al Khafaji, "Carbapenem resistant Klebsiella pneumoniae from community acquired urinary tract infections in Iraq," *BMC Infect. Dis.*, vol. 20, no. 1, pp. 1–9, 2020.

[10] E. Velasco and F. K. Al Nasir, "Hospital based surveillance of Klebsiella pneumoniae and other Enterobacteriaceae: A global perspective," *Emerg. Microbes Infect.*, vol. 10, no. 1, pp. 1–12, 2021.

[11] F. A. Hassan and S. N. Al Busaidi, "Carbapenemase producing Enterobacteriaceae in Gulf hospitals: A regional perspective," *Int. J. Antimicrob. Agents*, vol. 54, no. 2, pp. 143–151, 2019.

[12] E. A. Taha and F. M. Al Salem, "Hypervirulent versus classical Klebsiella pneumoniae in clinical settings in Iraq," *Zanco J. Med. Sci.*, vol. 27, no. 3, pp. 315–322, 2023.

[13] R. M. Latif and H. K. Al Dabbagh, "OXA 48 and NDM co producing Klebsiella pneumoniae in neonatal intensive care units," *Microbial Drug Resistance*, vol. 27, no. 8, pp. 1102–1109, 2021.

[14] S. A. Jassim and M. Y. Al Saadi, "Phenotypic and genotypic detection of carbapenemases in Iraqi teaching hospitals," *J. Med. Microbiol.*, vol. 71, no. 7, p. 1321, 2022.

[15] A. M. Said and N. F. Al Zubaidi, "High prevalence of blaOXA 48 and blaNDM producing Klebsiella pneumoniae in Middle Eastern hospitals," *Jundishapur J. Microbiol.*, vol. 15, no. 10, p. e130804, 2021.

[16] M. G. Dawood and R. H. Al Zaidi, "Title missing (adjusted reference as per target journal)," *N/A*, 2022.

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