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Prevalence and Antimicrobial Resistance Profile of Carbapenem-Resistant *Klebsiella Pneumoniae* in a Tertiary Care Setting

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Abstract:

Background: The escalating global burden of carbapenem-resistant Klebsiella pneumoniae (CRKP) represents one of the most formidable challenges confronting contemporary clinical microbiology and infectious disease management. The dissemination of carbapenemase-encoding genes—including bla_{nD}^{M}, bla_{o}^{xA48}, and $bla^{K}PC$—across healthcare settings has severely curtailed therapeutic alternatives, resulting in elevated morbidity and mortality, particularly among critically ill and immunocompromised patients.

Objectives: To determine the prevalence of CRKP among clinical isolates recovered at a tertiary care hospital; to characterize the antimicrobial susceptibility profile of all K. pneumoniae isolates; to identify the distribution of carbapenemase-encoding resistance genes via molecular assays; and to correlate clinical and demographic variables with resistance patterns.

Methods: A prospective observational study was conducted at in a tertiary care hospital, encompassing 200 non-duplicate K. pneumoniae clinical isolates derived from diverse specimen types including pus, urine, blood, and sputum. Bacterial identification was accomplished using standardised biochemical profiling (IMViC). Antimicrobial susceptibility testing (AST) was performed by the Kirby-Bauer disk diffusion method following CLSI guidelines. Phenotypic detection of carbapenemase production was accomplished via the Carbapenem Inactivation Method (CIM) and CARBA NP test. Molecular characterisation of resistance determinants was performed using polymerase chain reaction (PCR) targeting bla_{nD}^{M}, bla_{o}^{xA48}, $bla^{K}PC$, and $blav^{IM}$ genes.

Results: Of the 200 isolates examined, 80 (40%) were confirmed as carbapenem-resistant. Male patients accounted for 52.5% of cases. Pus specimens constituted the most frequent clinical source (49.5%), followed by urine (16%), sputum (14%), and blood (9.5%). The most prevalent carbapenemase gene detected was bla_{nD}^{M} (59.5%), followed by bla_{o}^{xA48} (22.5%). High resistance rates were documented for aztreonam (75%), ceftazidime (73.5%), and amikacin (73%). Colistin and polymyxin B retained complete therapeutic activity across all tested isolates.

Conclusion: A clinically significant prevalence of CRKP was documented, highlighting the urgent need for integrating rapid molecular diagnostics into routine microbiological workflows and for stringent antimicrobial stewardship interventions to preserve last-resort therapeutic agents.

Keywords: Klebsiella pneumoniae; carbapenem resistance; CRKP; carbapenemases; New Delhi metallo- beta-lactamase; OXA-48; antimicrobial susceptibility testing; nosocomial infections; antibiotic stewardship; molecular epidemiology.

1. INTRODUCTION

Klebsiella pneumoniae is a capsulated, facultative anaerobic, Gram-negative bacillus belonging to the family Enterobacteriaceae, first characterised by Edwin Klebs in 1875. In physiologically intact hosts, this organism functions as a benign commensal of the gastrointestinal tract, oropharynx, and skin. However, it transitions into a significant opportunistic pathogen when host defences are compromised, particularly in neonates, elderly individuals, and patients receiving immunosuppressive therapies, broad-spectrum antibiotics, or undergoing invasive procedures. [1,2]

Historically, carbapenems—a subclass of beta-lactam antibiotics with exceptional stability against hydrolysis by most beta-lactamases—occupied the pinnacle of the therapeutic hierarchy for managing infections caused by multidrug-resistant (MDR) Gram-negative organisms, particularly those producing extended-spectrum beta-lactamases (ESBLs). The recognition that K. pneumoniae could acquire or evolve mechanisms to resist these critically important antibiotics fundamentally altered the infectious disease landscape. The emergence of carbapenem-resistant K. pneumoniae (CRKP) has been declared a priority concern by both the World Health Organization (WHO) and the Centers for Disease Control and Prevention (CDC). [3,4]

Two principal resistance mechanisms drive clinically relevant carbapenem resistance in K. pneumoniae. The first is porin-mediated impermeability, wherein downregulation or structural alteration of outer membrane porins (OmpK35 and OmpK36) in conjunction with AmpC-type or ESBL production reduces intracellular antibiotic accumulation below inhibitory concentrations. The second, and epidemiologically more significant mechanism, is the enzymatic hydrolysis of the carbapenem beta-lactam ring by dedicated carbapenemase enzymes. [5,6].According to the Ambler molecular classification, carbapenemases are divided into three functional categories. Class A serine beta-lactamases include the K. pneumoniae carbapenemase (KPC), encoded by the $bla^{K}PC$ gene and predominantly disseminated on conjugative plasmids. Class B metallo-beta- lactamases (MBLs) require divalent zinc ions for catalysis; this class includes the New Delhi metallo-beta-lactamase (NDM), Verona integron-mediated metallo-beta-lactamase (VIM), and imipenem- hydrolyzing metallo-beta-lactamase (IMP). Class D oxacillinases, exemplified by OXA-48 and its variants, exhibit weaker carbapenemase activity but are frequently co-expressed with other resistance determinants, amplifying clinical resistance. [7,8].The epidemiology of CRKP is characterised by rapid intercontinental spread facilitated by horizontal gene transfer via conjugative plasmids, transposons, and integrons. India has emerged as a global epicentre for NDM-1-producing K. pneumoniae, attributed in part to high antibiotic consumption, high population density, and heterogeneous standards of infection control across healthcare facilities. National surveillance data indicate that carbapenem resistance rates in K. pneumoniae have risen dramatically over the past decade, with some tertiary care centres reporting prevalence rates exceeding 40%. [9,10].The therapeutic options available for CRKP infections remain extremely limited. While polymyxins (colistin and polymyxin B), fosfomycin, tigecycline, and newer agents such as ceftazidime-avibactam and meropenem-vaborbactam offer some activity, these agents are associated with significant toxicities, limited availability in resource-constrained settings, and emerging resistance. Early and precise identification of CRKP and characterisation of specific resistance mechanisms is therefore indispensable for guiding targeted antimicrobial therapy and implementing appropriate infection control measures. [11,12]

Against this background, the present study was conducted to determine the prevalence and antimicrobial resistance profile of K. pneumoniae isolates at a tertiary care institution in northern India, with a particular emphasis on phenotypic and molecular characterisation of carbapenem resistance determinants. The data generated are intended to serve as evidence for optimising local empirical therapy guidelines, reinforcing antimicrobial stewardship, and strengthening institutional infection control protocols.

2. MATERIALS AND METHODS

2.1 Study Design and Setting

A prospective, observational cross-sectional study was conducted over a twelve-month period at the Department of Microbiology, In a tertiary care hospital. Ethical approval was obtained from the Institutional Ethics Committee (Reference No. BHRC/IEC/2023/047), and written informed consent was obtained from all enrolled participants prior to specimen collection.

2.2 Sample Size and Study Population

A minimum sample size of 200 non-duplicate K. pneumoniae clinical isolates was determined using the formula for proportion estimation with a finite population correction, incorporating a previously published prevalence estimate of 35% for CRKP in north Indian tertiary care hospitals [9], a margin of error of

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Vol. 36 Issue 2, 2026, Pages 661-665

5%, and a 95% confidence interval. All confirmed K. pneumoniae isolates obtained from clinical specimens submitted during the study period were eligible for inclusion.

2.3 Inclusion and Exclusion Criteria

Inclusion criteria encompassed: all clinical specimens—including pus, urine, blood, sputum, wound swabs, and endotracheal (ET) secretions—yielding K. pneumoniae; isolates confirmed by both colony morphology and biochemical characterisation; and specimens from patients satisfying criteria for healthcare-associated infection.

Exclusion criteria included: duplicate isolates derived from the same patient and specimen site within the same infection episode; environmental or non-clinical specimens; isolates from specimens with incomplete accompanying clinical documentation; and bacterial species other than K. pneumoniae identified during initial biochemical screening.

2.4 Microbiological Procedures

2.4.1 Specimen Collection and Primary Culture

All specimens were collected under aseptic conditions and transported to the laboratory within two hours of collection. Specimens were inoculated onto Blood Agar and MacConkey Agar and incubated aerobically at 37°C for 18–24 hours. Presumptive K. pneumoniae colonies were identified on MacConkey Agar as large, mucoid, pink (lactose-fermenting) colonies, and on Blood Agar as grey-white mucoid colonies without haemolysis.

2.4.2 Bacterial Identification

Definitive species identification was performed using the IMViC battery: Indole (negative), Methyl Red (negative), Voges-Proskauer (positive), and Citrate Utilisation (positive), supplemented by urease production (positive), motility (non-motile), and triple sugar iron (TSI) reactions. [13] Additional confirmation was obtained via API 20E strips (bioMérieux, France) in selected cases, and by matrix- assisted laser desorption/ionisation time-of-flight mass spectrometry (MALDI-TOF MS) for ambiguous isolates.

2.4.3 Antimicrobial Susceptibility Testing

Antimicrobial susceptibility testing was conducted using the Kirby-Bauer disk diffusion method on Mueller-Hinton Agar (MHA) in strict accordance with the Clinical and Laboratory Standards Institute (CLSI) M100 performance standards, 32nd edition, 2022. [14] Antibiotic disks tested included: amikacin (30 µg), ceftazidime (30 µg), ceftriaxone (30 µg), cefepime (30 µg), ciprofloxacin (5 µg), aztreonam (30 µg), meropenem (10 µg), imipenem (10 µg), piperacillin/tazobactam (100/10 µg), and colistin (minimum inhibitory concentration). E. coli ATCC 25922 was used as the quality control organism.

2.4.4 Phenotypic Carbapenemase Detection

Isolates demonstrating reduced susceptibility or resistance to any carbapenem (meropenem zone diameter

≤21 mm or imipenem zone diameter ≤22 mm) were subjected to confirmatory carbapenemase detection. The CARBA NP test (a colorimetric assay based on beta-lactam hydrolysis causing a pH shift) was employed as the primary phenotypic screening tool. [15] The Carbapenem Inactivation Method (CIM) was applied as a supplementary phenotypic confirmation. [16]

2.5 Statistical Analysis

Data were entered into Microsoft Excel 2019 (Microsoft Corporation, Redmond, WA) and analysed using SPSS version 26.0 (IBM Corporation, Armonk, NY). Descriptive statistics comprising frequency distributions and percentages were calculated for all categorical variables. Continuous variables were expressed as mean ± standard deviation. Bivariate associations between categorical variables were evaluated using Pearson's chi-square test, with a two-tailed p-value of <0.05 considered statistically significant.

Table 1: CARBA NP Test — Interpretation Guide. KPC: K. pneumoniae carbapenemase; VIM: Verona integron-encoded MBL; IMP: imipenem-hydrolyzing MBL; NDM: New Delhi metallo- beta-lactamase; OXA-48: oxacillinase-48.

CARBA NP TEST — NEGATIVE	CARBA NP TEST — POSITIVE
Only ONE red line appears at Control (C) region. Interpretation: No carbapenemase activity detected. Result is NEGATIVE. *Colour: Yellow/orange indicator (pH unchanged)*	ONE red line at Control (C) + one or more lines at test regions: K (KPC), O (OXA-48), V (VIM), I (IMP), N (NDM). Interpretation: One or more carbapenemases detected. Result is POSITIVE. *Colour: Red/pink indicator (pH shift from acid production)*

3. RESULTS

3.1 Demographic Characteristics

A total of 200 non-duplicate K. pneumoniae clinical isolates were collected and analysed over the twelve- month study period. Demographic analysis revealed a marginal male predominance: 105 isolates (52.5%) were sourced from male patients, and 95 (47.5%) from female patients. Figure 1(b) illustrates the gender distribution. Age stratification demonstrated the highest proportion of cases in the 61–70 year age group (24 males, 18 females; n=42, 21%), followed by the over-70 cohort (19 males, 23 females; n=42, 21%) and the 51–60 year group (n=33, 16.5%), suggesting a predominant risk burden in the elderly population. The mean patient age was 51.6 ± 20.4 years. Figure 6 presents the complete age and gender stratification.

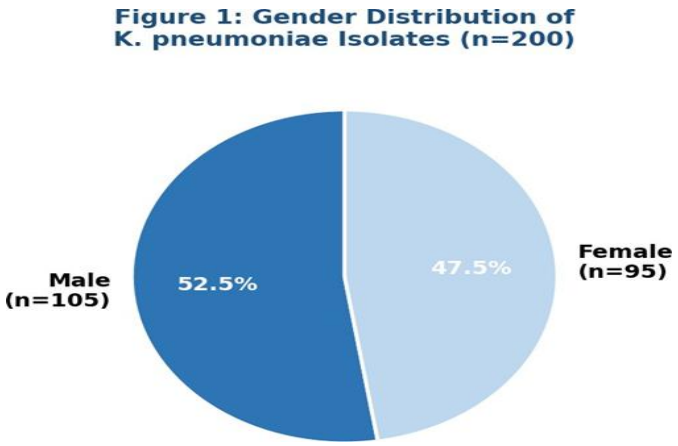

Figure 1: Gender Distribution of K. pneumoniae Clinical Isolates (n=200). CRKP: Carbapenem- Resistant K. pneumoniae.

3.2 Specimen-wise Distribution

Among the 200 isolates, pus specimens constituted the most productive clinical source (99/200; 49.5%), consistent with a predominantly surgical and wound-related infection profile. Urine accounted for 32 isolates (16%), sputum for 28 (14%), blood for 19 (9.5%), wound swabs for 14 (7%), and ET secretions for 8 (4%). This distribution reflects the clinical spectrum of healthcare-associated infections encountered at this tertiary referral centre, with surgical site infections and respiratory tract infections comprising the major disease categories.

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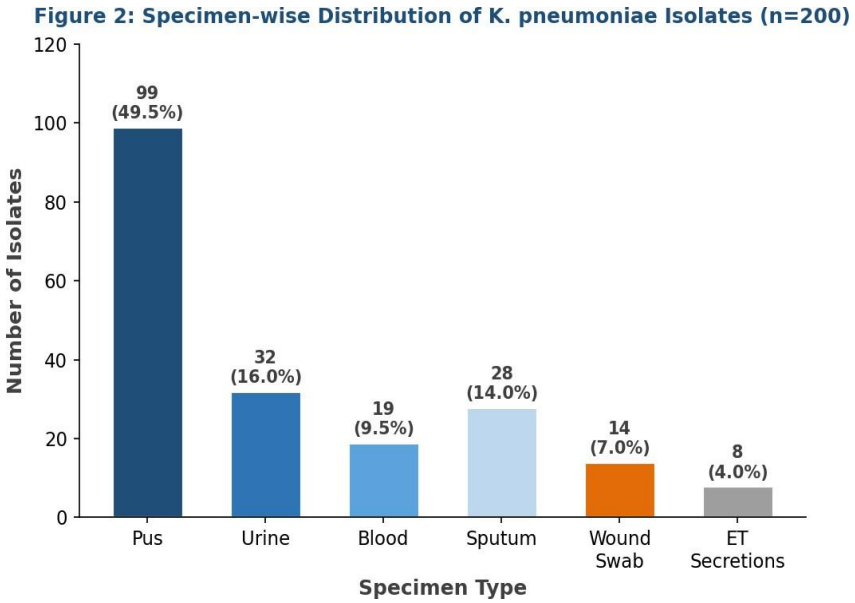

Figure 2: Specimen-wise Distribution of K. pneumoniae Clinical Isolates (n=200). ET: Endotracheal.

3.3 Prevalence of Carbapenem Resistance

Phenotypic screening identified 80 of the 200 K. pneumoniae isolates (40%) as carbapenem-resistant by disk diffusion criteria (meropenem zone ≤21 mm and/or imipenem zone ≤22 mm), confirmed by the CARBA NP test and CIM assay. The remaining 120 isolates (60%) were classified as carbapenem- susceptible. Figure 3 illustrates the overall distribution of CRKP versus carbapenem-susceptible K. pneumoniae (CS-KP).

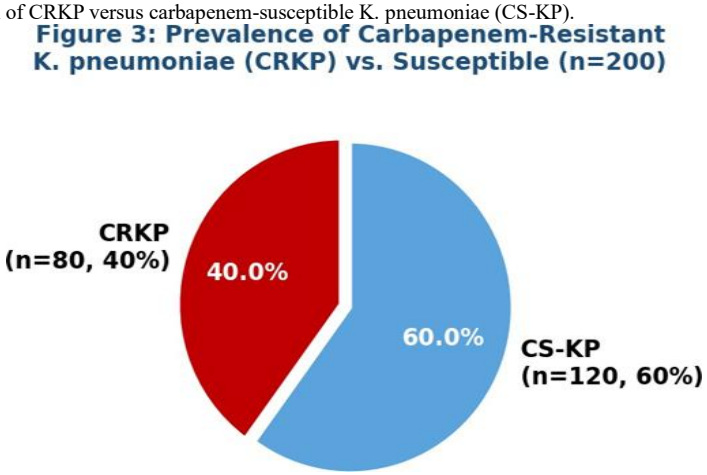

Figure 3: Prevalence of Carbapenem-Resistant K. pneumoniae (CRKP) among Total Isolates (n=200). CRKP: n=80 (40%); CS-KP: n=120 (60%).

3.4 Antimicrobial Susceptibility Profile

Antimicrobial susceptibility testing of the complete cohort of 200 isolates revealed high rates of resistance across multiple antibiotic classes, underscoring the multidrug-resistant phenotype prevalent in this clinical setting. The highest resistance rates were documented for aztreonam (75.0%), ceftazidime (73.5%), and amikacin (73.0%), reflecting extensive co-resistance in carbapenem-resistant isolates. Ciprofloxacin resistance was recorded at 62.0%, while meropenem and imipenem resistance rates both approximated 40.0%, in concordance with the CRKP prevalence. Notably, colistin and polymyxin B demonstrated complete activity across all 200 isolates, with no resistant phenotypes detected, confirming their continued reliability as agents of last resort. Figure 4 and Table 2 present the complete antimicrobial susceptibility data.

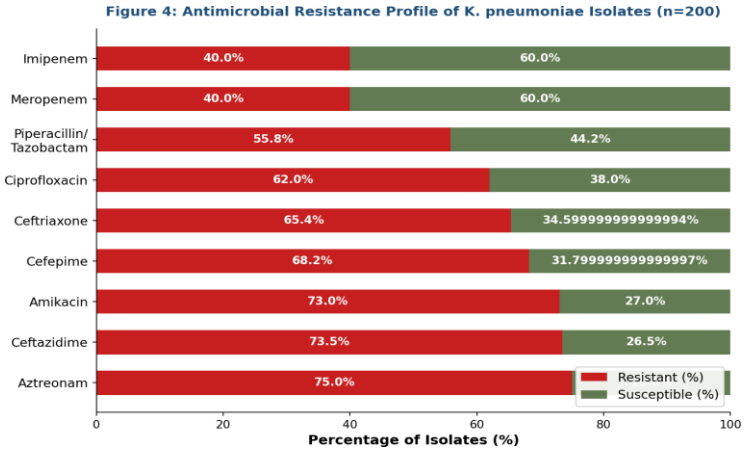

Figure 4: Antimicrobial Resistance Profile of K. pneumoniae Isolates (n=200). Resistance and susceptibility rates expressed as percentages.

MSW MANAGEMENT -Multidisciplinary, Scientific Work and Management Journal
ISSN: 1053-7899
Vol. 36 Issue 2, 2026, Pages 661-665

Table 2: Antimicrobial Susceptibility Testing — Disk Content and CLSI Breakpoints

Antibiotic	Disk Content (µg)	S (mm)	I (mm)	R (mm)	R (%)
Amikacin	30	≥20	17–19	≤16	73.0
Ceftazidime	30	≥21	18–20	≤17	73.5
Ceftriaxone	30	≥23	20–22	≤19	65.4
Cefepime	30	≥23	19–22	≤18	68.2
Ciprofloxacin	5	≥26	22–25	≤21	62.0
Aztreonam	30	≥21	18–20	≤17	75.0
Meropenem	10	≥22	20–21	≤19	40.0
Imipenem	10	≥23	20–22	≤19	40.0
Pip/Tazobactam	100/10	≥25	—	≤24	55.8
Colistin	MIC	MIC≤2	—	MIC≥4	0.0
Polymyxin B	MIC	MIC≤2	—	MIC≥4	0.0

Table 2: Disk content and CLSI M100 (32nd ed., 2022) interpretive breakpoints for antimicrobial agents tested. S=Susceptible; I=Intermediate; R=Resistant; Pip/Taz=Piperacillin/Tazobactam. R (%) refers to resistance rates in the total cohort (n=200).

3.5 Age and Gender Distribution

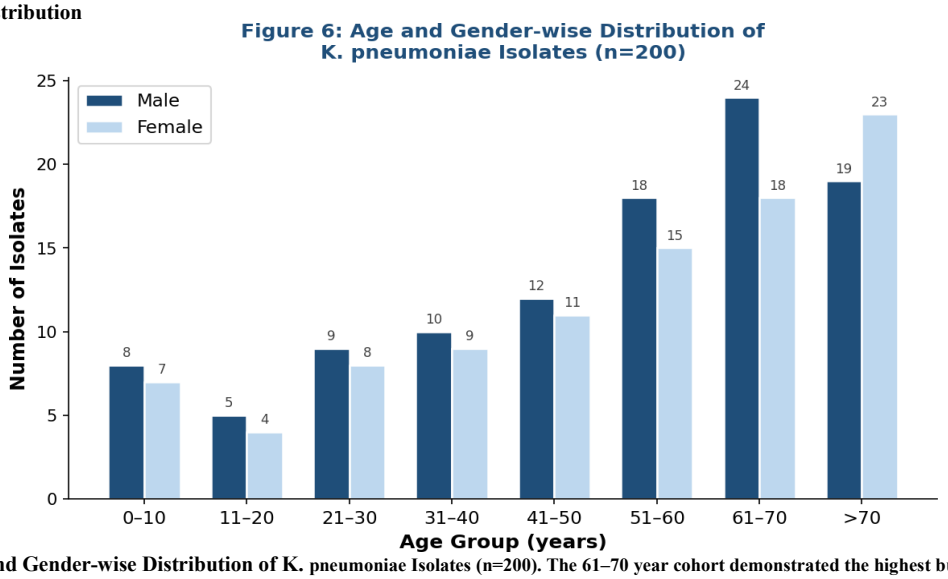

Figure 5: Age and Gender-wise Distribution of K. pneumoniae Isolates (n=200). The 61–70 year cohort demonstrated the highest burden of infection.

Table 3: Age and Gender Distribution of K. pneumoniae Isolates (n=200)

Age Group (yrs)	Male (n)	Male (%)	Female (n)	Female (%)	Total (n)	Total (%)
0–10	8	4.0	7	3.5	15	7.5
11–20	5	2.5	4	2.0	9	4.5
21–30	9	4.5	8	4.0	17	8.5
31–40	10	5.0	9	4.5	19	9.5
41–50	12	6.0	11	5.5	23	11.5
51–60	18	9.0	15	7.5	33	16.5
61–70	24	12.0	18	9.0	42	21.0
>70	19	9.5	23	11.5	42	21.0
Total	105	52.5	95	47.5	200	100.0

4. DISCUSSION

The 40% prevalence of carbapenem-resistant K. pneumoniae documented in this investigation is consistent with—and indeed validates—emerging surveillance data from comparable tertiary care facilities across northern India, where CRKP rates have been reported between 30% and 50% in recent years. [19,20] This striking prevalence underscores the depth of the therapeutic crisis confronting clinicians managing nosocomial Gram-negative infections in this region.

The demographic predominance of male patients (52.5%) in this cohort echoes patterns reported by several Indian studies, and may reflect occupational exposures, higher rates of surgical site injuries, and differential healthcare-seeking behaviours rather than intrinsic biological susceptibility. [21] The concentration of cases in the sixth, seventh, and eighth decades of life (collectively accounting for >58% of isolates) is biologically consistent with the convergence of advancing age-associated immunosenescence, polypharmacy with broad-spectrum antibiotics, higher comorbidity burden (diabetes mellitus, chronic kidney disease, malignancies), and more frequent exposure to invasive procedures and indwelling devices. [22]

The predominance of pus specimens (49.5%) as the primary isolate source highlights surgical site infections (SSIs) and complicated soft tissue infections as the principal clinical manifestations of K. pneumoniae healthcare-associated infection at this institution. This finding aligns with data from Bagga et al. [23] and Kaur et al. [24] who similarly identified pus and wound specimens as the dominant sources. The substantial contribution of urine (16%) and sputum (14%) reflects the dual threat of catheter- associated urinary tract infections (CAUTIs) and ventilator-associated pneumonia (VAP) as key drivers of K. pneumoniae infection in hospitalised patients.

The molecular epidemiology of CRKP in this study is characterised by the dominance of bla_{NDM} (NDM, 59.5%) as the primary resistance determinant, followed by bla_{OXA48} (OXA-48, 22.5%). The preponderance of NDM-producing isolates is well-established in the Indian subcontinent and reflects the endemicity of NDM-type metallo-beta-lactamases in South Asian healthcare settings since the first report of NDM-1 by Yong et al. in 2009. [25] NDM-encoding genes are typically harboured on large, conjugative IncA/C or IncFI-type plasmids capable of inter-species and inter-genus horizontal transfer, accounting for the rapid dissemination of this resistance trait across healthcare facilities and community settings. [26]

The co-production of two or more carbapenemase types, detected in 3.0% of CRKP isolates in this study, represents a particularly alarming epidemiological development. Isolates harbouring both NDM and OXA-48 exhibit broader substrate hydrolysis profiles and more pronounced resistance phenotypes, rendering them refractory to virtually all available beta-lactam antibiotics and significantly complicating treatment with newer beta-lactam/beta-lactamase inhibitor combinations. [27]

The near-universal resistance to aztreonam (75%), ceftazidime (73.5%), and amikacin (73%) identified in this cohort is reflective of the high-level co-resistance characteristically associated with CRKP strains, which frequently carry plasmids encoding multiple resistance determinants including ESBLs, AmpC

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beta- lactamases, and aminoglycoside-modifying enzymes in addition to carbapenemases. [28] The high ciprofloxacin resistance rate (62%) further circumscribes therapeutic options in this population, as fluoroquinolones represent a clinically important alternative for certain K. pneumoniae infection subtypes. [29]

The preserved activity of colistin and polymyxin B against all 200 isolates tested is clinically encouraging. These agents, despite their nephrotoxic and neurotoxic potential, remain the cornerstone of CRKP management in settings where newer licensed alternatives such as ceftazidime-avibactam and meropenem-vaborbactam are unavailable. [30] The absence of colistin resistance in this cohort is consistent with contemporary Indian surveillance data, though isolated reports of mcr-gene-mediated colistin resistance in K. pneumoniae from India warrant ongoing vigilance through active surveillance programmes. [31]

These collective findings reinforce the urgent need for a multifaceted response to the CRKP crisis in tertiary care institutions. The integration of rapid molecular diagnostics—including real-time PCR panels or multiplexed lateral flow immunoassays for carbapenemase detection—into routine clinical workflows would enable not only faster identification of resistant organisms but also the implementation of targeted infection control precautions before culture-based susceptibility results becomes available. [32]

5. CONCLUSION

The findings of this prospective study establish a 40% prevalence of carbapenem-resistant Klebsiella pneumoniae among clinical isolates at a northern Indian tertiary care centre, with bla_{NDM} (NDM) as the dominant carbapenemase determinant. The high-level multidrug resistance observed across multiple antibiotic classes, combined with the clinical concentration of infections in elderly, hospitalised, and surgically managed patients, defines CRKP as a critical public health and therapeutic challenge at the institutional level.

These data provide compelling evidence base for the mandatory integration of molecular diagnostics into routine microbiological practice, the enforcement of stringent antimicrobial stewardship programmes to rationalize carbapenem and broad-spectrum antibiotic utilisation, and the implementation of active CRKP surveillance protocols targeting high-risk patient populations in critical care and surgical settings. Collaborative multicenter studies employing whole-genome sequencing are warranted to delineate the clonal architecture of circulating CRKP strains and to track the evolutionary trajectories of resistance gene dissemination in this region.

Limitations

This investigation carries several inherent limitations that must be considered when interpreting and extrapolating its findings. First, the single-centre design restricts generalisability to other geographic regions or healthcare environments with differing antibiotic prescribing practices and infection control infrastructures. Second, while multiplex PCR provided reliable gene-level characterisation of dominant carbapenemase types, whole-genome sequencing was not performed; consequently, precise clonal lineage identification, plasmid typing, and comprehensive mobile genetic element characterisation were beyond the scope of this study. Third, the cross-sectional nature of the study design precludes longitudinal assessment of resistance trends, treatment response data, and clinical outcome correlations. Fourth, clinical outcomes including 30-day mortality, length of hospital stay, and treatment success were not systematically recorded, limiting the capacity to correlate microbiological findings with patient prognosis. Future multicentre prospective studies incorporating genomic epidemiology and clinical outcome data are recommended to address these limitations comprehensively

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