Sahel Journal of Life Sciences FUDMA (SAJOLS)
June 2026 Vol. 4(2): 232-241
ISSN: 3027-0456 (Print)
ISSN: 1595-5915 (Online)
DOI: *https://doi.org/10.33003/sajols-2026-0402-24*

Research Article

Molecular Detection of *magA* Gene and Antibiotic Susceptibility Pattern of Uropathogenic *Klebsiella pneumoniae* Isolated from Patients Attending Federal Teaching Hospital Katsina, Nigeria

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ABSTRACT
Urinary tract infections (UTIs) caused by *Klebsiella pneumoniae* pose significant public health challenges due to increasing antimicrobial resistance and virulence determinants. This study aimed to molecularly confirm *K. pneumoniae* isolates, determine the occurrence of the *magA* virulence gene, and evaluate antimicrobial susceptibility patterns among patients attending Federal Teaching Hospital Katsina, Nigeria. A cross-sectional study was conducted involving 200 urine specimens from patients with suspected UTIs. *K. pneumoniae* was confirmed by PCR targeting the *yhaI* gene, and the *magA* virulence gene was detected. Antimicrobial susceptibility was determined by the Kirby-Bauer disc diffusion method according to CLSI guidelines. Molecular confirmation identified 11 (5.5%) *K. pneumoniae* isolates. Only one isolate. (9.0 %) was positive for the *magA* virulence gene. Antimicrobial susceptibility testing revealed complete susceptibility (100%) to amikacin, high susceptibility to ciprofloxacin (81.8%) and meropenem (81.8%), but significant resistance to cefotaxime (90.9%), amoxicillin-clavulanic acid (81.8%), and ampicillin (63.6%). Resistance was observed in 90.9% of isolates, with MAR indices ranging from 0.25 to 0.63. This study confirms the presence of Uropathogenic *K. pneumoniae* carrying the *magA* virulence gene with significant resistance patterns. The coexistence of virulence determinants and antimicrobial resistance underscores the need for routine molecular diagnostics, continuous surveillance, and strengthened antimicrobial stewardship programs to guide appropriate therapy and prevent the spread of resistant strains.

Keywords: Antimicrobial resistance; *Klebsiella pneumoniae*; *magA* gene; Nigeria; Urinary tract infections; *yhai* gene

Citation: Aliyu, Y., Ifediora, S., & Tukur, I. M. (2026). Molecular Detection of *magA* Gene and Antibiotic Susceptibility Pattern of Uropathogenic *Klebsiella pneumoniae* Isolated from Patients Attending Federal Teaching Hospital Katsina, Nigeria. *Sahel Journal of Life Sciences FUDMA*, 4(2): 232-241. DOI: *https://doi.org/10.33003/sajols-2026-0402-24*

INTRODUCTION

Urinary tract infections (UTIs) remain one of the most prevalent bacterial infections worldwide and constitute a major public health concern due to their high morbidity, increasing recurrence rates, and growing antimicrobial resistance. They account for over 150 million cases annually and are among the most common healthcare-associated infections, contributing substantially to prolonged hospitalization, increased healthcare expenditure, and adverse clinical outcomes (Stamm, 2002; Gobernado *et al.,* 2007). Among Gram-negative uropathogens, *Klebsiella pneumoniae* has emerged as an important opportunistic pathogen responsible for both community-acquired and hospital-associated UTIs. *Klebsiella pneumoniae* is an encapsulated, Gram-negative, non-motile bacillus belonging to the family *Enterobacteriaceae*. Although it normally colonizes the gastrointestinal tract, *K. pneumoniae* readily establishes infection in susceptible individuals, particularly hospitalized and

immunocompromised patients, where it can cause UTIs pneumonia, septicaemia, and other invasive diseases (Murdoch, 2001; Amaraie *et al.,* 2004; Brisse *et al.,* 2006).

The increasing clinical significance of *K. pneumoniae* is largely attributed to its extensive repertoire of virulence determinants and its remarkable ability to acquire antimicrobial resistance. Major virulence factors, including capsular polysaccharides, lipopolysaccharides, fimbriae, siderophores, and outer membrane proteins, facilitate bacterial adherence, immune evasion, persistence within host tissues, and disease progression (Ariffin *et al.,* 2000; Brisse *et al.,* 2006; Paczosa and Mecsas, 2016). Consequently, infections caused by this pathogen are frequently associated with more severe clinical manifestations and poorer therapeutic outcomes than those caused by many other urinary pathogens (Cheng *et al.,* 2010; Chen and Wen, 2011)

Recent advances in molecular microbiology have significantly improved the accurate identification of *K. pneumoniae*. Conventional culture and biochemical methods remain useful for preliminary diagnosis; however, polymerase chain reaction (PCR)-based detection provides greater sensitivity and specificity for confirming bacterial identity. Molecular confirmation minimizes the possibility of misidentification among closely related members of the *Enterobacteriaceae*, thereby improving epidemiological surveillance and facilitating appropriate clinical management (Amani *et al.,* 2016; Yusuf *et al.,* 2018).

Beyond species confirmation, characterization of bacterial virulence genes has become increasingly important in understanding the pathogenic potential of clinical isolates. One of the most extensively investigated virulence-associated genes is mucoviscosity-associated gene A (*magA*), which contributes to capsular polysaccharide synthesis and the hypermucoviscous phenotype characteristic of hypervirulent *K. pneumoniae*. The presence of *magA* enhances resistance to phagocytosis and complement-mediated killing while promoting bacterial persistence within host tissues (Fang *et al.,* 2004; Struve *et al.,* 2005; Lee *et al.,* 2006). Furthermore, studies have associated the gene with increased biofilm formation and enhanced virulence, thereby increasing the likelihood of invasive infections and treatment failure (Fodah *et al.,* 2014; Wu *et al.,* 2018).

Equally concerning is the rapid emergence of antimicrobial-resistant *K. pneumoniae*. The organism has developed resistance to several commonly prescribed antibiotics, including penicillins, cephalosporins, fluoroquinolones, and, increasingly to carbapenems, thereby limiting therapeutic options for clinicians (Gupta *et al.,* 2011; Shon *et al.,* 2013; Pitout *et al.,* 2015). The coexistence of virulence determinants and antimicrobial resistance has intensified the clinical burden of *K. pneumoniae* infections and underscores the need for continuous molecular surveillance and antimicrobial susceptibility monitoring (Caneiras *et al.,* 2019; Wyres *et al.,* 2019).

Although several studies have investigated the epidemiology of *K. pneumoniae*, information regarding the molecular confirmation of isolates, distribution of the *magA* gene, and antimicrobial susceptibility profiles among urinary isolates in Northwestern Nigeria remains limited. Generating such data is essential for strengthening diagnostic accuracy, guiding empirical antimicrobial therapy, and supporting infection prevention and antimicrobial stewardship programmes.

Therefore, this study aimed to molecularly confirm *Klebsiella pneumoniae* isolates recovered from urine specimens of patients and evaluate their antimicrobial susceptibility patterns and determine the occurrence of *magA* gene.

MATERIALS AND METHODS

Study Design and Area

A hospital-based cross-sectional study was conducted to molecularly characterize *Klebsiella pneumoniae* isolates recovered from patients with suspected (UTIs) attending the Microbiology Laboratory of Federal Teaching Hospital, Katsina, Nigeria. The Hospital is a tertiary healthcare institution that serves as a major referral Centre for Katsina State and neighbouring states in northwestern Nigeria, providing specialized diagnostic and clinical services. The cross-sectional design was considered appropriate because it permits the simultaneous assessment of bacterial isolation, molecular detection of virulence genes, and antimicrobial susceptibility profiles within a defined study population (Setia, 2016).

Study Population and Sample Size

The study population comprised patients presenting with clinical manifestations suggestive of UTIs who were referred to the Microbiology Laboratory during the study period. Only consenting participants who fulfilled the inclusion criteria were recruited, whereas patients without symptoms of UTIs or those who declined participation were excluded.

The minimum sample size was estimated using the standard formula for prevalence studies based on a previously reported prevalence of 14.7% for *K. pneumoniae* urinary tract infections (Abdulfatai *et al.,* 2023). A minimum sample size of 191 participants was obtained; however, this was increased to 200 to enhance statistical reliability and reduce sampling error. Ethical approval for this study was obtained from the Ethics Committee of Federal Teaching Hospital Katsina before commencement of the study.

Collection and Processing of Clinical Specimens

Approximately 10 mL of clean-catch midstream urine was collected aseptically from each participant into sterile universal containers following standard clinical procedures. The specimens were transported immediately to the Microbiology Laboratory of Federal Teaching Hospital Katsina for bacteriological analysis to minimize contamination and preserve bacterial viability.

Urine samples were cultured on MacConkey agar using the calibrated loop streaking technique and incubated aerobically at 37°C for 18–24 hours. Lactose-fermenting mucoid colonies suggestive of *K. pneumoniae* were subcultured repeatedly until pure cultures were obtained. Preliminary identification was performed using colony morphology, Gram staining characteristics, and conventional biochemical tests, including citrate utilization, indole production, methyl red, Voges–Proskauer, and motility tests following established microbiological procedures (Cheesbrough, 2006; MacWilliams, 2015; Wright *et al.,* 2017). Pure isolates were preserved on Nutrient agar slants at 4°C pending molecular analysis.

Genomic DNA Extraction

Genomic DNA was extracted from purified bacterial isolates using the conventional boiling lysis technique as previously described by Yusuf *et al.* (2018). Briefly, a single bacterial colony was suspended in sterile distilled water and subjected to boiling for 10 minutes to lyse the bacterial cells. The lysate was rapidly cooled on ice to stabilize the released nucleic acids before centrifugation at 10,000 rpm for 10 minutes. The resulting supernatant containing genomic DNA served as the template for polymerase chain reaction (PCR) assays. The boiling method remains a rapid, economical, and reliable approach for extracting bacterial DNA intended for conventional PCR applications (Dashti *et al.,* 2009; Yusuf *et al.,* 2018).

Molecular Confirmation of *Klebsiella pneumoniae*

Species confirmation was achieved by amplification of the *yhaI* gene using conventional PCR with species-specific primers described by Poirier *et al.* (2022). The PCR amplification was performed in a 20 µL reaction mixture containing PCR buffer, deoxynucleotide triphosphates (dNTPs), Taq DNA polymerase, forward and reverse primers, template DNA, and nuclease-free water. Thermal cycling conditions consisted of an initial denaturation step followed by repeated cycles of denaturation, primer annealing, extension, and a final extension stage according to the optimized protocol described by Yusuf *et al.* (2018).

Amplified products were separated by agarose gel electrophoresis, stained with ethidium bromide, and visualized under ultraviolet transillumination. Isolates producing the expected 249 bp amplicon were considered molecularly confirmed as *K. pneumoniae* (Poirier *et al.,* 2022).

Detection of Virulence (*magA*) Gene

The PCR-confirmed *K. pneumoniae* isolates were further screened for the presence of the (*magA)* virulence gene using gene-specific primers previously validated by Zamani *et al.* (2013). The PCR reactions were prepared in a total volume of 25 µL comprising PCR buffer, dNTP mixture, Taq DNA polymerase, forward and reverse primers, template DNA, and nuclease-free water.

Amplification was carried out using optimized cycling conditions consisting of initial denaturation, 30 amplification cycles involving denaturation, primer annealing, extension, and a final elongation step. PCR products were resolved on 1% agarose gel electrophoresis and examined under ultraviolet illumination. Isolates producing the expected 198 bp amplification product were regarded as positive for the *magA* gene (Zamani *et al.,* 2013; Poirier *et al.,* 2022).

Antimicrobial Susceptibility Testing

Antimicrobial susceptibility testing was performed on all PCR-confirmed *K. pneumoniae* isolates using the Kirby–Bauer disc diffusion method on Mŭeller–Hinton agar according to the Clinical and Laboratory Standards Institute (CLSI, 2024) recommendations. Fresh bacterial colonies were suspended in sterile normal saline and adjusted to the turbidity equivalent of a 0.5 McFarland standard (approximately 1.5×10^8 CFU/mL). Standardized bacterial suspensions were inoculated uniformly onto Mŭeller–Hinton agar plates using sterile cotton swabs. Commercial antibiotic discs representing commonly prescribed antimicrobial classes were placed aseptically on the inoculated plates. The antibiotics evaluated included amikacin (30 µg), meropenem (10 µg), ciprofloxacin (5 µg), chloramphenicol (30 µg), ampicillin (10 µg), cefotaxime (30 µg), tetracycline (30 µg), and

amoxicillin–clavulanic acid (30 µg). Following incubation at 37°C for 18–24 hours, zones inhibition was measured in millimetres and interpreted as susceptible, intermediate, or resistant according to CLSI (2024) guideline.

RESULTS

A total of 200 urine specimens obtained from patients with suspected UTIs were processed during the study period. Molecular confirmation by PCR targeting the *yhaI* gene confirmed 11 isolates (5.5% of all urine specimens) (Figure1). as *K. pneumoniae* plate 1. Among the PCR-confirmed isolates, only one isolate (9.1%) was positive for the *magA* Virulence gene following amplification of the expected 198 bp fragment plate II.

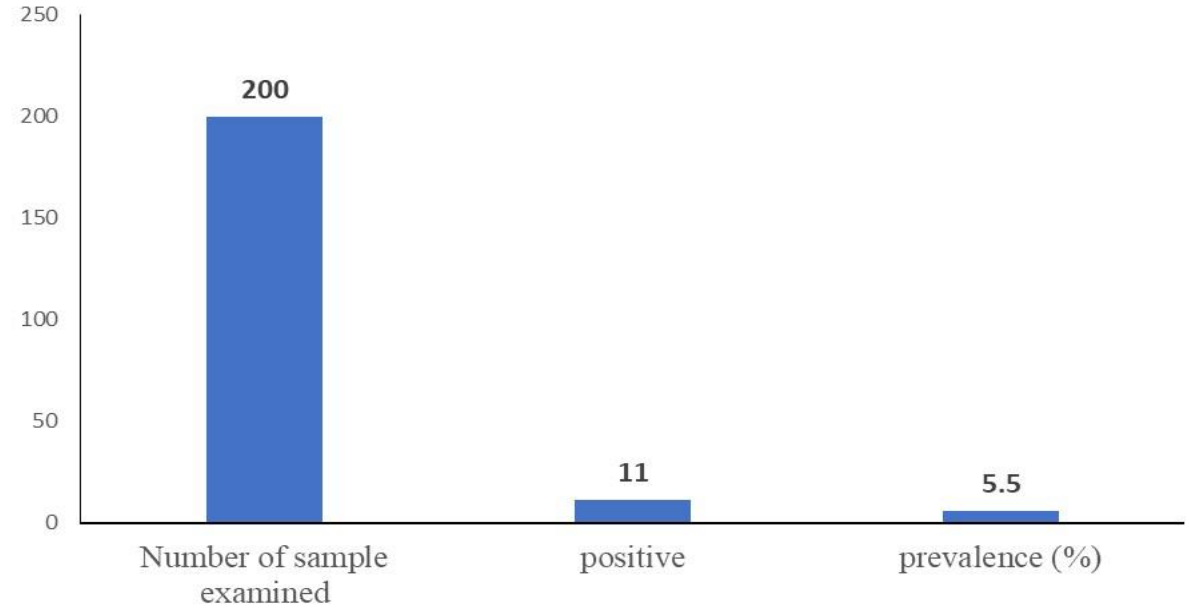

Figure 1: Overall prevalence of *Klebsiella pneumoniae* in urine of patients attending Federal Teaching Hospital Katsina

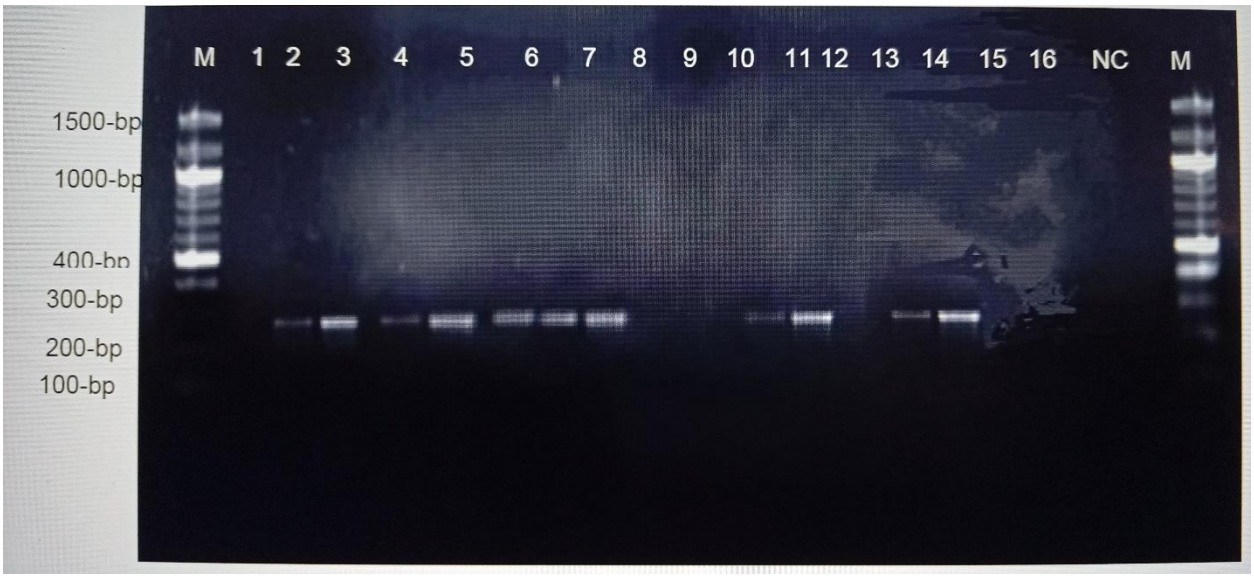

Plate I: Amplified fragment of *yhaI* geneof *Klebsiella pneumoniae*
Lanes 2, 3,4,5,6, 7,8, 12, 13,15 and 16 = positive; Expected size (249bp)
M = Molecular marker size of (100bp)
NC = Nuclease free water as negative control

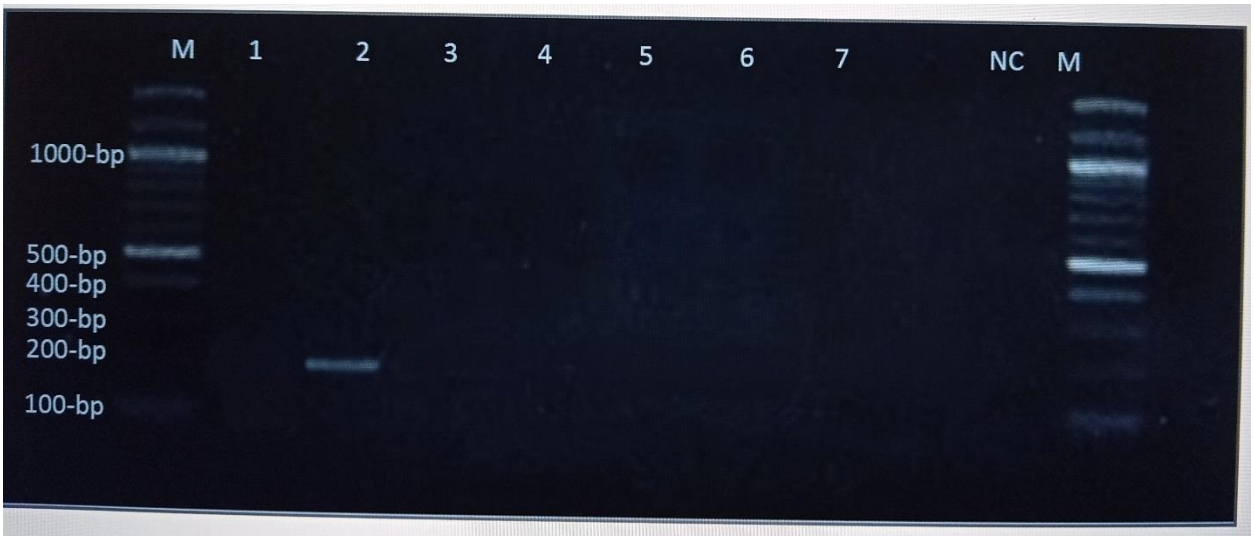

Plate II: Detection of magA in *Klebsiella pneumoniae isolated* from urine
Lane 2 = positive
Expected size (198 bp)
M = Molecular marker size of 100bp)
NC = Nuclease free water as negative control

Antimicrobial Susceptibility Pattern of PCR-Confirmed *Klebsiella pneumoniae*

The antimicrobial susceptibility profiles of the eleven PCR-confirmed *K. pneumoniae* isolates are presented in Table 1**.** Complete susceptibility (100%) was observed to amikacin, indicating High susceptibility of 81.81% was recorded for ciprofloxacin and meropenem each. Conversely, substantial resistance was observed against several commonly prescribed antibiotics. The isolates were most resistant to cefotaxime (90.0%), followed by amoxicillin–clavulanic acid (81.8%) and ampicillin (63.6%); but moderately resistant to chloramphenicol (54.5%) and tetracycline (54.5%).

Multiple Antibiotic Resistance (MAR) Index

The distribution of multiple antibiotic resistance among the PCR-confirmed *K. pneumoniae* isolates is summarized in Table 2. Every isolate exhibited resistance to at least two antimicrobial agents. The MAR index values ranged from 0.20 to 0.63. Four isolates (36.4%) demonstrated resistance to four of the eight antibiotics evaluated, corresponding to a MAR index of 0.50. Only one isolate (9.1%) showed resistance to two antibiotics (MAR index = 0.20), whereas the remaining isolates exhibited higher resistance burdens. Overall, 10 of the 11 isolates (90.9%) had MAR indices greater than 0.30.

Table 1: Antimicrobial susceptibility profile of PCR-confirmed *Klebsiella pneumoniae* isolates (n = 11)

Antibiotic	Susceptible n (%)	Resistant n (%)
Amikacin (30 µg)	11 (100.0)	0 (0.0)
Meropenem (10 µg)	9 (81.8)	2 (18.2)
Ciprofloxacin (5 µg)	9 (81.8)	2 (18.2)
Chloramphenicol (30 µg)	5 (45.5)	6 (54.5)
Ampicillin (10 µg)	4 (36.4)	7 (63.6)
Cefotaxime (30 µg)	1 (9.1)	10 (90.9)
Tetracycline (30 µg)	5 (45.5)	6 (54.5)
Amoxicillin–clavulanic acid (30 µg)	2 (18.2)	9 (81.8)

Table 2: Multiple antibiotic resistance (MAR) patterns of PCR-confirmed *Klebsiella pneumoniae* isolates

Isolate code	Number of Antibiotics Resisted	Resistance Pattern	MAR Index
KP102	2	ME, AMP	0.25
KP16	3	TE, CTX, AMC	0.38
KP180	3	TE, AMP, CTX	0.38
KP45	3	CIP, AMC, CTX	0.38
KP42	4	TE, AMP, AMC, CTX	0.50
KP73	4	CTX, C, ME, AMC	0.50
KP6	4	AMP, CTX, C, AMC	0.50
KP9	4	C, AMP, CTX, AMC	0.50
KP157	5	CTX, C, TE, CIP, AMC	0.63
KP196	5	TE, AMP, C, CTX, AMC	0.63
KP25	5	TE, AMP, C, CTX, AMC	0.63

Keys: ME = Meropenem; TE = Tetracycline; CIP = Ciprofloxacin; AMC = Amoxicillin–clavulanic acid; AMP = Ampicillin; CTX = Cefotaxime; C = Chloramphenicol; KP = *Klebsiella pneumoniae*.

DISCUSSION

The present study detected *magA* (virulence) gene in uropathogenic *Klebsiella pneumoniae* among patients attending Federal Teaching Hospital Katsina, and determined their antibiotic susceptibility pattern.The findings provide important evidence on the circulating virulence determinants and antimicrobial resistance profiles of *K. pneumoniae* in urinary tract infections (UTIs) within the study setting. Molecular confirmation using PCR enhanced the accuracy of identification compared to conventional biochemical methods, which are often limited by misidentification of closely related Enterobacterales. This supports previous reports that molecular techniques improve diagnostic precision and epidemiological tracking of *K. pneumoniae* in clinical infections (Mirzaie and Ranjbar, 2021). The application of PCR-based confirmation in this study therefore strengthens the reliability of the isolates used for downstream virulence and resistance analysis.

The detection of the *magA* gene (which code for virulence) among uropathogenic isolates is clinically significant. The *magA* gene has been strongly associated with capsule formation, serum resistance, and enhanced invasive potential in *K. pneumoniae*, particularly in hypervirulent strains linked to severe infections such as liver abscess and septic complications (Cheng *et al.,* 2010; Hyun *et al.,* 2024). Its presence in urinary isolates in this study suggests the circulation of potentially hypervirulent strains beyond classical invasive sites. This raises concern because such strains may increase the risk of complicated UTIs, ascending infection, and possible bloodstream dissemination if not promptly managed.

The antibiotic susceptibility profile revealed notable resistance to commonly used antibiotics, particularly beta-lactams and fluoroquinolones. This aligns with global evidence showing that *K. pneumoniae* has become increasingly resistant due to extended-spectrum beta-lactamase (ESBL) production and other resistance mechanisms (Mirzaie and Ranjbar, 2021; Yousuf *et al.,* 2026). The high resistance profile observed may be attributed to indiscriminate antibiotic use, self-medication, and weak antimicrobial stewardship practices in many healthcare settings in low- and middle-income countries.

Conversely, higher susceptibility to carbapenems and aminoglycosides suggests that these agents remain relatively effective therapeutic options in the study environment. However, global reports indicate an increasing emergence of carbapenem-resistant *K. pneumoniae*, which threatens current treatment strategies and limits available therapeutic options (Yousuf *et al.,* 2026; CDC, 2024). Continuous surveillance is therefore essential to prevent further escalation of resistance.

The coexistence of virulence factors such as *magA* with resistance patterns is particularly alarming. Recent studies have demonstrated that hypervirulent and multidrug-resistant *K. pneumoniae* strains are increasingly emerging, often harbouring both resistance genes and virulence determinants, which significantly complicates clinical management and worsens patient outcomes (Hyun *et al.,* 2024). This convergence represents a major public health threat; as such strains are capable of causing severe, hard-to-treat infections.

Overall, the findings highlight the dual challenge posed by virulent and drug-resistant *K. pneumoniae* in urinary tract infections. The detection of *magA*-positive isolates combined with resistance underscores the need for routine molecular

diagnostics, strict infection control practices, and strengthened antimicrobial stewardship programs within healthcare facilities.

Despite these important contributions, the study is limited by its single-center design and relatively small sample size, which may limit generalizability. Future studies incorporating whole-genome sequencing and multi-center surveillance would provide deeper insights into the genetic diversity, transmission dynamics, and evolutionary trends of uropathogenic *K. pneumoniae* in Nigeria.

The antibiotic susceptibility pattern of *Klebsiella pneumoniae* isolates demonstrated varying degrees of resistance and susceptibility to the tested antimicrobial agents. High resistance was observed against Ampicillin (63.63%), amoxicillin–clavulanic acid (81.81%), and cefotaxime (90.90%). Similar findings have been reported by Ashefo *et al.* (2023), who documented resistance rates of 94.74% and 73.60% to ampicillin and amoxicillin–clavulanic acid, respectively. Likewise, Varghese *et al.* (2016) and Hamza *et al.* (2016) reported complete resistance of *K. pneumoniae* isolates to Ampicillin. The observed resistance is largely attributed to the production of β-lactamase enzymes, including AmpC and extended-spectrum β-lactamases (ESBLs), as well as alterations in penicillin-binding proteins, efflux pump activity, reduced membrane permeability, and horizontal gene transfer (Pitout and Laupland, 2008; Dallenne *et al.,* 2010; Blair *et al.,* 2015; Bush, 2018). Similar high resistance to Amoxicillin–Clavulanic acid has been reported by Muhammad *et al.* (2021) in Nigeria and Ghenea (2021) in Romania.

Conversely, high susceptibility was recorded for Amikacin (100%), Meropenem (81.81%), and Ciprofloxacin (81.81%). These findings agree with previous reports demonstrating excellent activity of Amikacin against *K. pneumoniae* isolates (Giske *et al.,* 2012; Simanjuntak, 2014). The effectiveness of Amikacin is largely due to its resistance to enzymatic modification and its ability to inhibit bacterial protein synthesis by targeting the 30S ribosomal subunit (Xiao and Hu, 2012). Similarly, the susceptibility of *K. pneumoniae* to Meropenem observed in this study is consistent with reports from Kaduna State, Nigeria (Muhammad *et al.,* 2021), Northwest Ethiopia (Ameshe *et al.,* 2022), and India (Varghese *et al.,* 2016). The sustained efficacy of carbapenems may be associated with the relatively lower prevalence of carbapenemase-producing strains compared with ESBL-producing isolates (Tzouvelekis *et al.,* 2012; Jean, 2018). The high susceptibility to Ciprofloxacin is also in agreement with the findings of Osundiya *et al.* (2013), reflecting its potent inhibition of bacterial DNA gyrase and topoisomerase IV (Hooper and Jacoby, 2016).

Moderate resistance was observed against Chloramphenicol (54.54%) and Tetracycline (54.54%). The widespread use and misuse of these antibiotics in both human and veterinary medicine may have contributed to the development of resistance through mechanisms such as reduced membrane permeability and active efflux systems (Butaye *et al.,* 2003; Aminov *et al.,* 2004).

Multiple Antibiotic Resistance (MAR) index analysis revealed values ranging from 0.25 to 0.63, indicating that the isolates originated from environments with frequent antibiotic exposure. According to Hemen *et al.* (2012), MAR values greater than 0.2 suggest high-risk contamination sources. In this study, 90.90% of the isolates exhibited resistance to three or more antibiotic as such were seen to have multiple antibiotic resistance. This prevalence is higher than those reported by Osundiya and Oladele (2013) and Olonitola *et al.* (2007). The high resistance rate may be attributed to the acquisition of multiple resistance genes encoding ESBLs, carbapenemases, and multidrug efflux pumps, thereby limiting available treatment options and increasing the risk of therapeutic failure (Paterson *et al.,* 2003; Nikaido, 2009; Nobrega *et al.,* 2013). These findings highlight the growing challenge of antimicrobial resistance and underscore the need for continuous surveillance, rational antibiotic use, and strengthened infection prevention and control measures.

CONCLUSION

This study demonstrates the presence of molecularly confirmed uropathogenic *Klebsiella pneumoniae* among patients attending Federal Teaching Hospital Katsina, with notable detection of the *magA* virulence gene and significant antibiotic resistance patterns. The identification of *magA*-positive isolates indicates the circulation of potentially hypervirulent strains capable of enhanced pathogenicity and severe clinical outcomes. The observed resistance, particularly against commonly prescribed antibiotics such as beta-lactams and fluoroquinolones, highlights a growing therapeutic challenge in the management of urinary tract infections in the study area. Although some antibiotics such as carbapenems and aminoglycosides retained relatively higher activity, their effectiveness remains threatened by the global emergence of resistant strains.

Overall, the coexistence of virulence determinants and antimicrobial resistance underscores the urgent

need for improved diagnostic accuracy, continuous surveillance, and evidence-based treatment strategies to mitigate the spread and impact of these pathogens.

Routine implementation of molecular diagnostic techniques, including PCR for the detection and characterization of *Klebsiella pneumoniae* and important virulence determinants such as the *magA* gene, should be adopted to improve the accurate identification of uropathogenic strains. Continuous antimicrobial susceptibility surveillance and strengthened antimicrobial stewardship programs should guide appropriate antibiotic use and help curb the emergence of resistance. Healthcare facilities should reinforce infection prevention and control measures to prevent the spread of multidrug-resistant and hypervirulent *K. pneumoniae* strains, while public health education should discourage self-medication and indiscriminate antibiotic use. Additionally, large-scale multicenter studies incorporating whole-genome sequencing are recommended to further elucidate the prevalence of the *magA* virulence gene, genetic diversity, antimicrobial resistance mechanisms, and molecular epidemiology of uropathogenic *K. pneumoniae* in Nigeria.

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