

Bacteriological Spectrum of UTI in Patients of Chronic Kidney Disease with Diabetes Mellitus in Bangladesh

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Abstract: Background: Urinary tract infections (UTIs) are a significant concern in chronic kidney disease (CKD) patients, particularly those with diabetes mellitus (DM), due to immune dysfunction and glycosuria. The increasing burden of antimicrobial resistance (AMR) in this population necessitates a detailed understanding of bacterial pathogens and susceptibility patterns. **Objective:** To determine the prevalence of UTIs in diabetic CKD patients, compare bacterial isolates between diabetic and non-diabetic CKD patients, and assess antimicrobial susceptibility patterns. **Methods and Materials:** This cross-sectional study included 997 CKD patients (499 with DM, 498 without DM) at a tertiary hospital in Bangladesh. Urine samples were collected and analyzed for bacterial growth. Antibiotic susceptibility was determined using the Kirby-Bauer disk diffusion method following Clinical and Laboratory Standards Institute (CLSI) guidelines. Statistical analysis was performed using STATA 17, with $p < 0.05$ considered significant. **Results:** UTI prevalence was 66.33% in diabetic CKD patients and 57.83% in non-diabetic CKD patients ($p = 0.000$). *Escherichia coli* (*E. coli*) was the predominant pathogen (62.15% in diabetic vs. 66.36% in non-diabetic CKD patients), followed by *Klebsiella* (18.75% vs. 17.88%). High resistance was observed for amoxicillin (95.28%), cefixime (81.65%), and ciprofloxacin (58.76%), whereas carbapenems (meropenem: 87.94%) and colistin (68.85%) were more effective. **Conclusion:** Diabetic CKD patients had a higher UTI prevalence and were more prone to multidrug-resistant infections. The findings highlight the urgent need for antibiotic stewardship, routine screening, and infection control measures to improve clinical outcomes.

Keywords: Chronic Kidney Disease, Diabetes Mellitus, Urinary Tract Infection, Antimicrobial Resistance, Bacterial Isolates.

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Research Paper

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How to cite this paper:

Marina Arjumand *et al*
(2025). Bacteriological
Spectrum of UTI in Patients
of Chronic Kidney Disease
with Diabetes Mellitus in
Bangladesh. *Middle East Res
J. Med. Sci.*, 5(2): 111-119.

Article History:

| Submit: 18.02.2025 |
| Accepted: 19.03.2025 |
| Published: 22.03.2025 |

INTRODUCTION

Chronic kidney disease (CKD) is a significant global health challenge, with diabetes mellitus (DM) being one of its primary causes [1]. The increasing prevalence of DM has led to a rising incidence of CKD, with diabetic nephropathy accounting for a substantial proportion of end-stage renal disease [2]. CKD patients, particularly those with diabetes, are at an increased risk of infections due to immune dysfunction, impaired renal clearance, and frequent hospitalizations [3]. Among

these, urinary tract infections (UTIs) are one of the most common bacterial infections affecting CKD patients, often presenting with atypical symptoms that complicate early diagnosis and treatment [4].

The high burden of UTIs in CKD patients is associated with increased morbidity, mortality, and healthcare costs [5, 6]. CKD-related immune alterations, including dysregulated cytokine responses, reduced neutrophil function, and defective phagocytosis, contribute to the persistence and severity of infections

[7]. Moreover, atypical UTI presentations in CKD patients—such as generalized weakness, confusion, or asymptomatic bacteriuria—often lead to delayed diagnosis, increasing the risk of complications such as urosepsis and acute kidney injury (AKI) [8, 9].

Studies have consistently shown that diabetic CKD patients have a higher prevalence of UTIs than their non-diabetic counterparts, with *E. coli* being the predominant uropathogen, followed by *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, and *Acinetobacter* species [10, 11]. The combination of hyperglycemia-induced immune dysfunction, glycosuria fostering bacterial growth, and recurrent catheterization makes diabetic CKD patients more susceptible to UTIs caused by multidrug-resistant (MDR) organisms [12].

A growing concern in UTI management is the increasing antimicrobial resistance (AMR) among uropathogens, particularly in CKD patients who frequently receive broad-spectrum antibiotics [13]. Many Gram-negative isolates exhibit resistance to cephalosporins, fluoroquinolones, and cotrimoxazole, limiting empirical treatment options [14]. This resistance pattern is particularly concerning in diabetic CKD patients, where delayed effective treatment may exacerbate renal dysfunction and increase hospitalization rates.

While carbapenems, amikacin, and colistin have demonstrated efficacy against MDR pathogens, their use must be carefully monitored in CKD patients due to potential nephrotoxicity [15]. Furthermore, emerging resistance to carbapenems and colistin in Gram-negative bacteria underscores the urgency of implementing antimicrobial stewardship programs to preserve the efficacy of last-resort antibiotics [16].

In Bangladesh, the burden of UTIs in diabetic CKD patients is exacerbated by several factors, including limited access to healthcare, lack of routine UTI screening, and high rates of inappropriate antibiotic use [17]. The absence of structured antimicrobial surveillance programs further complicates treatment, as physicians often lack updated resistance patterns to guide empirical therapy [18]. Rising AMR trends in Bangladesh, particularly among *E. coli* and *Klebsiella* isolates, necessitate urgent policy interventions to curb antibiotic misuse and improve infection control strategies [19].

This study aims to determine the prevalence of UTIs in diabetic CKD patients in Bangladesh, compare the bacteriological patterns between diabetic and non-diabetic CKD patients, and evaluate the antimicrobial susceptibility of urinary isolates. The findings will help guide clinical management and inform policy decisions

to mitigate the impact of UTIs in this vulnerable population. By identifying key bacterial pathogens and their resistance patterns, this study will provide valuable insights for clinicians and policymakers, aiding in the development of evidence-based guidelines for UTI management in CKD patients in Bangladesh.

METHODS AND MATERIALS

Study Design and Settings

This was a cross-sectional study conducted at a tertiary care hospital in Bangladesh over a one-year period, from July 2022 to June 2023. The study included chronic kidney disease (CKD) patients with and without diabetes mellitus (DM) who presented with symptoms of urinary tract infections (UTIs). Clinical and microbiological data were collected to assess bacterial isolates and antimicrobial susceptibility patterns.

Study Population and Sample Size

This study included 997 patients diagnosed with chronic kidney disease (CKD), comprising 499 diabetic CKD patients and 498 non-diabetic CKD patients. The participants were recruited from a tertiary-level hospital in Bangladesh over one year. Patients were enrolled based on clinical suspicion of urinary tract infection (UTI), confirmed through laboratory diagnostics.

Inclusion criteria encompassed adult CKD patients (aged ≥ 18 years) with or without diabetes mellitus (DM) who presented with symptoms suggestive of UTI, such as dysuria, increased urinary frequency, fever, or flank pain. Patients with recent antibiotic use (within the last 48 hours), known immunosuppressive conditions other than diabetes, or acute kidney injury (AKI) rather than CKD were excluded to maintain homogeneity in the study population.

The sample size was determined based on the estimated prevalence of UTI in CKD patients, ensuring adequate statistical power to detect differences in bacterial isolates and antimicrobial resistance patterns between diabetic and non-diabetic CKD groups. Stratification by age, gender, and disease severity was performed to allow for subgroup analysis, facilitating a comprehensive understanding of the epidemiology and clinical impact of UTIs in this high-risk population.

Data Collection and Laboratory Analysis Demographic and Clinical Data Collection

Patient demographics, clinical characteristics, and laboratory findings were recorded using a structured case record form. Key variables included age, gender, residence, estimated glomerular filtration rate (eGFR), serum calcium, serum potassium levels, and other relevant clinical parameters essential for assessing disease severity and infection risk.

Urine Sample Collection and Microbiological Analysis

Mid-stream urine samples were collected under strict aseptic conditions and immediately transported for bacteriological culture. Bacterial identification was performed using standard biochemical techniques, and the frequency and distribution of isolates were analyzed separately for diabetic and non-diabetic CKD patients to determine any variations in uropathogenic profiles.

Antibiotic Susceptibility Testing

The antimicrobial susceptibility of isolated uropathogens was assessed using the Kirby-Bauer disk diffusion method, following the Clinical and Laboratory Standards Institute (CLSI) guidelines. Susceptibility patterns were evaluated for a broad spectrum of antibiotics, including cephalosporins, fluoroquinolones, carbapenems, aminoglycosides, and colistin, to identify resistance trends and inform appropriate treatment strategies.

Statistical Analysis

All data were analyzed using STATA 17. Continuous variables were assessed for normality using histograms and box plots. Normally distributed continuous variables were presented as mean \pm standard deviation (SD) and compared using the paired t-test, while skewed data were reported as median with interquartile range (IQR) and analyzed using the Kruskal-Wallis test. Categorical variables were

expressed as frequencies and percentages and compared using the chi-square (χ^2) test or Fisher's exact test, as appropriate. To determine significant differences in bacterial isolates and antibiotic resistance patterns between diabetic and non-diabetic CKD patients, univariate analyses were conducted. A p-value <0.05 was considered statistically significant, with a 95% confidence interval (CI) applied to all analyses. Results were presented in tables and figures to enhance clarity and interpretation.

Ethical Considerations

This study was conducted in accordance with the Declaration of Helsinki and received approval from the institutional ethics committee. Informed consent was obtained from all participants before data collection. Patient confidentiality was strictly maintained, with all data anonymized and used solely for research purposes. No invasive procedures were performed beyond routine clinical investigations.

RESULTS

UTIs were more prevalent in CKD patients with diabetes mellitus (66.33%) than in those without diabetes (57.83%), highlighting the increased infection risk in diabetic individuals due to factors like compromised immunity, glycosuria, and altered host defenses. The overall UTI prevalence in CKD patients was 62.08%, emphasizing the need for proactive infection control (Table I).

Table I: Prevalence of UTI in CKD Patients

Patient Group	Total, n (%)	UTI Cases (n)	Prevalence (%)
Diabetic CKD	499 (50.05)	331	66.33
Non-Diabetic CKD	498 (49.95)	288	57.83
Total	997	619	62.08

Demographic and Clinical Characteristics of CKD Patients by Gender

The mean age of CKD patients with diabetes (54.73 \pm 11.47 years) was significantly higher than that of non-diabetic CKD patients (43.25 \pm 14.91 years, $p=0.000$). The age distribution also differed significantly, with a higher proportion of diabetic CKD patients aged 51 years and above, whereas non-diabetic CKD patients were more concentrated in younger age groups.

Gender distribution showed a significant difference ($p=0.013$), with a higher proportion of females in the diabetic CKD group (62.25%) compared to the non-diabetic CKD group (54.51%). However, residence

distribution did not vary significantly between the two groups ($p=0.445$), with rural patients being the majority in both.

The median estimated glomerular filtration rate (eGFR) was slightly lower in diabetic CKD patients (10 [6–16] mL/min/1.73m²) than in non-diabetic CKD patients (11 [7–17] mL/min/1.73m², $p=0.050$), indicating a trend toward more severe renal impairment in the diabetic group. Serum calcium levels were significantly lower in diabetic CKD patients ($p=0.002$), while serum potassium levels were significantly higher ($p=0.020$), suggesting metabolic disturbances associated with diabetes and CKD progression (Table II).

Table II: Demographic and Clinical Characteristics of CKD Patients by Gender

Variable	CKD with DM	CKD without DM	p-value
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Age (mean ± SD)	54.73 ± 11.47	43.25 ± 14.91	0.000
Age range (Min-Max)	25 – 85	14 – 83	
Age Groups years, n (%)			0.000
30 years or less	12 (2.40)	117 (23.49)	
31–40	54 (10.82)	152 (30.52)	
41–50	138 (27.66)	83 (16.67)	
51–60	150 (30.06)	83 (16.67)	
61–70	109 (21.84)	41 (8.23)	
71 years and above	36 (7.21)	22 (4.42)	
Gender, n (%)			0.013
Female	172 (54.51)	310 (62.25)	
Male	227 (45.49)	188 (37.75)	
Residence, n (%)			0.445
Rural	321 (64.46)	310 (62.12)	
Urban	177 (35.54)	189 (37.88)	
eGFR, median (Q1-Q3)	11 (7 – 17)	10 (6 – 16)	0.050
S. Calcium, median (Q1-Q3)	8.1 (7.9 – 8.3)	8 (7.8 – 8.2)	0.002
S. Potassium, median (Q1-Q3)	6.8 (6.2 – 7.6)	6.9 (6.2 – 7.8)	0.020

Distribution of Bacterial Isolates in CKD Patients with and without DM

The distribution of bacterial isolates in CKD patients with and without diabetes revealed distinct patterns, although no statistically significant differences were observed ($p=0.085$). *E. coli* was the predominant pathogen in both groups, accounting for 62.15% in diabetic CKD patients and 66.36% in non-diabetic CKD patients.

Klebsiella infections were similarly distributed between diabetic (18.75%) and non-diabetic (17.88%) CKD patients. Staphylococcus species were more

prevalent in diabetic patients (14.24%) compared to non-diabetic patients (10.30%).

While mixed infections like *E. coli* + *Pseudomonas* and *Klebsiella* + *Pseudomonas* were rare, they were slightly more common in the diabetic group. *Acinetobacter baumannii* (ACB) was more frequently isolated from non-diabetic CKD patients (3.03%) than from diabetic patients (1.04%). Uncommon isolates such as Coagulase-Negative Staphylococci (CoNS) and *Klebsiella* + ACB were present only in the diabetic group (Table III).

Table III: Distribution of Bacterial Isolates in CKD Patients with and without DM

Bacterial Isolate	CKD with DM (n, %)	CKD without DM (n, %)	p-value
<i>E. coli</i>	179 (62.15)	219 (66.36)	0.085
<i>Klebsiella</i> (Kleb)	54 (18.75)	59 (17.88)	-
<i>Staphylococcus</i> (Staph)	41 (14.24)	34 (10.30)	-
<i>E. coli</i> + <i>Pseudomonas</i> (Pseudo)	5 (1.74)	2 (0.61)	-
<i>Acinetobacter baumannii</i> (ACB)	3 (1.04)	10 (3.03)	-
Coagulase-Negative Staphylococci (CoNS)	2 (0.69)	0	-
<i>Klebsiella</i> + <i>Acinetobacter</i> (Kleb + ACB)	2 (0.69)	0	-
<i>Pseudomonas Aeruginosa</i> (Pseudo)	2 (0.69)	4 (1.21)	-
<i>Klebsiella</i> + <i>Pseudomonas</i> (Kleb + Pseudo)	2 (0.61)	-	-

***E. coli* denotes *Escherichia coli*, *Klebsiella* denotes *Kleb*, *Pseudomonas* denotes *Pseudo*, *Acinetobacter baumannii* denotes *ACB*, Coagulase-Negative Staphylococci denotes *CoNS*, *Pseudomonas Aeruginosa* denotes *Pseudo*,**

Antibiotic Resistance Patterns Among Bacterial Isolates in CKD Patients

The antibiotic resistance patterns observed in the study highlight significant resistance to commonly used antibiotics among CKD patients. Amoxicillin exhibited the highest resistance, with 95.28% of isolates being resistant, leaving only 4.56% susceptible.

Similarly, high resistance rates were noted for cefixime (81.65%), azithromycin (70.22%), cotrimoxazole (64.25%), and doxycycline (66.73%), indicating limited efficacy of these antibiotics in treating infections in this population.

Among fluoroquinolones, ciprofloxacin and levofloxacin demonstrated considerable resistance at 58.76% and 53.33%, respectively, suggesting reduced effectiveness for these agents in CKD-related infections. Aminoglycosides showed comparatively better susceptibility, with amikacin displaying 60.17% susceptibility, whereas gentamicin had nearly equal resistance and susceptibility rates (47.78% vs. 46.25%).

Carbapenems, particularly meropenem, retained strong efficacy, with 87.94% susceptibility and only 9.75% resistance. Similarly, colistin, linezolid, vancomycin, and clindamycin showed high susceptibility rates (68.85%, 85.71%, 80.95%, and 84.62%, respectively), making them viable options for treating resistant infections. Piperacillin-tazobactam demonstrated complete susceptibility (100%), further supporting its role as an effective treatment choice (Table IV).

Table IV: Antibiotic Resistance Patterns Among Bacterial Isolates in CKD Patients

Antibiotic	Resistant n (%)	Intermediate n (%)	Susceptible n (%)	Total Cases (N)
Amoxicillin	585 (95.28)	1 (0.16)	28 (4.56)	614
Ceftriaxone	417 (68.70)	11 (1.81)	179 (29.49)	607
Ceftazidime	67 (42.68)	18 (11.46)	72 (45.86)	157
Ciprofloxacin	359 (58.76)	24 (3.93)	228 (37.32)	611
Levofloxacin	272 (53.33)	17 (3.33)	221 (43.33)	510
Amikacin	173 (29.83)	58 (10.00)	349 (60.17)	580
Azithromycin	382 (70.22)	23 (4.23)	139 (25.55)	544
Cotrimoxazole	390 (64.25)	14 (2.31)	203 (33.44)	607
Doxycycline	353 (66.73)	41 (7.75)	135 (25.52)	529
Gentamicin	280 (47.78)	35 (5.97)	271 (46.25)	586
Meropenem	55 (9.75)	13 (2.30)	496 (87.94)	564
Oxacillin	39 (56.52)	5 (7.25)	25 (36.23)	69
Cefixime	405 (81.65)	14 (2.82)	77 (15.52)	496
Nitrofurantoin	255 (44.12)	52 (9.00)	271 (46.89)	578
Colistin	19 (31.15)	-	42 (68.85)	61
Nalidixic Acid	131 (66.50)	6 (3.05)	60 (30.46)	197
Chloramphenicol	177 (47.33)	11 (2.94)	186 (49.73)	374
Linezolid	6 (14.29)	-	36 (85.71)	42
Vancomycin	6 (14.29)	2 (4.76)	34 (80.95)	42
Clindamycin	4 (15.38)	-	22 (84.62)	26
Piperacillin-Tazobactam	-	-	35 (100)	35
Moxaclar	363 (59.12)	56 (9.12)	195 (31.76)	614

Antibiotic Susceptibility Profiles Against Multiple Clinical Isolates Among Patients with CKD

The antibiotic susceptibility patterns varied across different bacterial isolates among CKD patients. *E. coli* exhibited the highest susceptibility to cefixime (76.62%), nitrofurantoin (75.65%), and nalidixic acid (73.33%), while showing moderate resistance to other antibiotics, including meropenem (68.15%) and cotrimoxazole (66.01%). *Klebsiella* (Kleb) showed the highest susceptibility to linezolid (80.56%), clindamycin (86.36%), and vancomycin (91.18%). However, its resistance was notable against cephalosporins, with ceftriaxone showing only 13.97% susceptibility. *Staphylococcus* (Staph) displayed strong susceptibility

to oxacillin (68.00%) and piperacillin-tazobactam (37.14%), though it exhibited moderate responses to ciprofloxacin (12.28%) and gentamicin (12.92%). *Acinetobacter baumannii* (ACB) demonstrated significant resistance across most antibiotics, with relatively higher susceptibility to colistin (23.81%) and piperacillin-tazobactam (31.43%). Coagulase-negative *Staphylococci* (CoNS) showed limited susceptibility data but had some response to linezolid (5.56%) and vancomycin (5.88%). *Pseudomonas aeruginosa* (Pseudo) had a relatively low susceptibility rate across most antibiotics, with minimal responses to commonly used antimicrobials (Table V).

Table V: Antibiotic Susceptibility Profiles Against Multiple Clinical Isolates Among Patients with CKD

Antibiotic	<i>E. coli</i> (%)	Kleb (%)	Staph (%)	ACB (%)	CoNS (%)	Pseudo (%)
Amoxicillin	78.57	-	7.14	-	-	7.14
Ceftriaxone	70.95	13.97	10.06	1.68	-	3.35

Ceftazidime	-	55.56	36.11	1.39	-	-
Ciprofloxacin	63.16	19.30	12.28	1.32	-	1.75
Levofloxacin	62.90	20.36	9.95	2.26	-	1.81
Amikacin	65.90	20.63	7.45	2.01	-	1.72
Azithromycin	62.59	19.42	12.23	1.44	-	1.44
Cotrimoxazole	66.01	12.81	11.82	4.43	0.99	1.97
Doxycycline	62.22	12.59	17.78	2.96	-	1.48
Gentamicin	63.47	20.66	12.92	1.11	-	0.74
Meropenem	68.15	18.95	8.06	1.81	-	1.21
Oxacillin	-	20.00	12.00	-	-	68.00
Cefixime	76.62	12.99	5.19	-	-	2.60
Nitrofurantoin	75.65	15.50	5.17	1.85	-	1.48
Colistin	35.71	26.19	-	23.81	2.38	-
Nalidixic Acid	73.33	21.67	1.67	3.33	-	-
Chloramphenicol	71.51	11.83	7.53	2.69	-	2.15
Linezolid	11.11	80.56	-	2.78	5.56	-
Vancomycin	2.94	91.18	-	-	5.88	-
Clindamycin	-	86.36	-	9.09	-	-
Piperacillin-Tazobactam	-	5.71	37.14	31.43	2.86	-
Escherichia coli denotes E. coli, Klebsiella denotes Kleb, Staphylococcus denotes Staph, Acinetobacter baumannii denotes ACB, Coagulase -Ve Staphylococci denotes CoNS, and Pseudomonas Aeruginosa denotes Pseudo						

DISCUSSION

CKD patients with diabetes mellitus (DM) were significantly older than non-diabetic CKD patients (54.73±11.47 vs. 43.25±14.91 years, p=0.000), with a higher proportion of diabetic CKD patients aged 51 years and above. This aligns with evidence that diabetes-related CKD develops later in life due to prolonged metabolic and vascular damage [20]. Conversely, non-diabetic CKD may result from earlier-onset glomerular diseases or genetic factors.

Gender distribution was significantly different (p=0.013), with more females in the diabetic CKD group (62.25%) than in the non-diabetic group (54.51%), reflecting the higher prevalence of diabetes in women, possibly due to hormonal and metabolic factors [21, 22]. Despite this, residence distribution did not vary significantly (p=0.445), with rural patients comprising the majority in both groups, suggesting limited healthcare access as a common challenge in CKD management across both populations.

Diabetic CKD patients had slightly lower median eGFR (10 [6–16] vs. 11 [7–17] mL/min/1.73m², p=0.050), indicating a trend toward more severe renal impairment. This is consistent with accelerated CKD progression in diabetes, driven by chronic hyperglycemia, inflammation, and oxidative stress [23].

Serum calcium levels were significantly lower (p=0.002) and serum potassium levels were higher (p=0.020) in diabetic CKD patients, suggesting greater metabolic disturbances. Hypocalcemia may result from

secondary hyperparathyroidism, while hyperkalemia is likely due to reduced renal potassium excretion and insulin resistance [24, 25]. These imbalances heighten the risk of cardiovascular complications, necessitating close monitoring and tailored interventions.

The distribution of bacterial isolates in CKD patients showed distinct patterns between diabetic and non-diabetic groups, although the differences were not statistically significant (p=0.085). The predominance of *E. coli* in both groups (62.15% in diabetic CKD vs. 66.36% in non-diabetic CKD) aligns with existing literature that identifies *E. coli* as the most common uropathogen in CKD and diabetic patients [26]. This is likely due to its strong adherence to the urinary epithelium, ability to form biofilms, and resistance to host immune responses, making it the leading cause of urinary tract infections (UTIs) across different patient populations [26].

Klebsiella species were the second most common pathogens, with a similar distribution in diabetic (18.75%) and non-diabetic (17.88%) CKD patients. *Klebsiella* infections are frequently associated with hospital-acquired UTIs, particularly in immunocompromised individuals, aligns with existing literature which explain their presence in both groups at comparable rates [27]. *Staphylococcus* species were notably more prevalent in diabetic CKD patients (14.24%) compared to non-diabetic CKD patients (10.30%), suggesting a higher susceptibility to Gram-positive infections in diabetes. This may be attributed to hyperglycemia-induced immune dysfunction and

impaired neutrophil activity, which increase the risk of infections caused by skin flora, such as *Staphylococcus aureus* and Coagulase-Negative Staphylococci (CoNS).

Rare and mixed infections showed some variation between the groups. *E. coli* + *Pseudomonas* and *Klebsiella* + *Pseudomonas* co-infections were slightly more frequent in diabetic CKD patients, potentially reflecting longer hospital stays, recurrent infections, or exposure to broad-spectrum antibiotics in these individuals. *Acinetobacter baumannii* (ACB) was more common in non-diabetic CKD patients (3.03% vs. 1.04%), possibly due to differences in hospital-acquired infections or antimicrobial selection pressures. The presence of Coagulase-Negative Staphylococci (CoNS) and *Klebsiella* + ACB exclusively in diabetic CKD patients suggests greater colonization by opportunistic pathogens, which may be facilitated by frequent catheterization, impaired immune responses, or antibiotic exposure in diabetic individuals.

The findings reveal high resistance to commonly used antibiotics, posing a significant challenge in managing infections in CKD patients. Amoxicillin exhibited the highest resistance (95.28%), followed by cefixime (81.65%), azithromycin (70.22%), and doxycycline (66.73%), indicating their limited utility in empirical therapy. This pattern aligns with global reports of widespread resistance to beta-lactams and macrolides, particularly in populations with recurrent infections and frequent antibiotic exposure [28, 29].

Fluoroquinolone resistance was also notable, with ciprofloxacin (58.76%) and levofloxacin (53.33%) showing reduced efficacy. These findings are consistent with studies reporting rising fluoroquinolone resistance in UTI pathogens, likely due to overuse in outpatient settings and the selective pressure exerted by broad-spectrum antibiotics [30].

In contrast, aminoglycosides showed moderate effectiveness, with amikacin displaying 60.17% susceptibility, while gentamicin exhibited nearly equal resistance and susceptibility rates (47.78% vs. 46.25%). This suggests amikacin remains a viable option for treating multidrug-resistant Gram-negative infections, although careful monitoring is required due to its potential nephrotoxicity in CKD patients.

Carbapenems, particularly meropenem (87.94% susceptibility), retained strong efficacy, making them crucial in managing multidrug-resistant infections. Similarly, colistin (68.85%), linezolid (85.71%), vancomycin (80.95%), and clindamycin (84.62%) demonstrated high susceptibility, reinforcing their role as last-resort options for resistant bacterial strains [31]. Notably, piperacillin-tazobactam exhibited complete

susceptibility (100%), highlighting its potential as an effective first-line treatment in severe infections [32].

The antibiotic susceptibility patterns among bacterial isolates from CKD patients reveal varying degrees of responsiveness across different antimicrobial classes. *E. coli*, the predominant uropathogen, exhibited high susceptibility to cefixime (76.62%), nitrofurantoin (75.65%), and nalidixic acid (73.33%), suggesting their potential as effective treatment options for *E. coli*-related UTIs in CKD patients. However, *E. coli* showed moderate susceptibility to ciprofloxacin (63.16%) and levofloxacin (62.90%), reflecting a gradual decline in fluoroquinolone effectiveness, likely due to overuse and emerging resistance (1).

Klebsiella species demonstrated the highest susceptibility to linezolid (80.56%) and vancomycin (91.18%), indicating better efficacy of these agents for severe infections. However, its lower susceptibility to ceftriaxone (13.97%) and ceftazidime (55.56%) aligns with increasing extended-spectrum beta-lactamase (ESBL) production, limiting cephalosporin use in *Klebsiella* infections [33].

Staphylococcus species showed strong susceptibility to oxacillin (68.00%) and clindamycin (86.36%), supporting their continued effectiveness against Gram-positive infections. The high susceptibility to piperacillin-tazobactam (37.14%) suggests its potential role in mixed infections, particularly in CKD patients with complicated UTIs or catheter-associated infections.

Among multidrug-resistant pathogens, *Acinetobacter baumannii* (ACB) exhibited relatively low susceptibility to most antibiotics, with the highest responsiveness to colistin (23.81%) and piperacillin-tazobactam (31.43%), reflecting limited treatment options for this opportunistic and highly resistant pathogen (4). *Pseudomonas aeruginosa* displayed low susceptibility across most antibiotics, except for piperacillin-tazobactam, reinforcing its innate resistance to several drug classes and the necessity for careful antibiotic selection.

CONCLUSION

This study highlights the high prevalence of UTIs in CKD patients, particularly those with diabetes mellitus, and the growing challenge of antimicrobial resistance. *Escherichia coli* remains the predominant uropathogen, with high resistance to commonly used antibiotics such as amoxicillin, cefixime, and fluoroquinolones. However, carbapenems, colistin, and aminoglycosides showed better efficacy. Given the increasing burden of multidrug-resistant infections, there

is an urgent need for routine UTI screening, targeted antimicrobial therapy, and robust antibiotic stewardship programs to improve clinical outcomes in this vulnerable population.

REFERENCES

- Deng, Y., Li, N., Wu, Y., Wang, M., Yang, S., Zheng, Y., ... Gao, J. (2021). Global, Regional, and National Burden of Diabetes-Related Chronic Kidney Disease From 1990 to 2019. *Frontiers in Endocrinology*, *12*, 672350. <https://doi.org/10.3389/fendo.2021.672350>
- Nitzan, O., Elias, M., Chazan, B., & Saliba, W. (2015). Urinary tract infections in patients with type 2 diabetes mellitus: review of prevalence, diagnosis, and management. *Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy*, *8*, 129–136. <https://doi.org/10.2147/DMSO.S51792>
- Chao, C.-T., Lee, S.-Y., Wang, J., Chien, K.-L., & Huang, J.-W. (2021). Frailty increases the risk for developing urinary tract infection among 79,887 patients with diabetic mellitus and chronic kidney disease. *BMC Geriatrics*, *21*(1), 349. <https://doi.org/10.1186/s12877-021-02299-3>
- Bacterial Urinary Tract Infections - Genitourinary Disorders. (n.d.). *MSD Manual Professional Edition*. Retrieved March 14, 2025, from <https://www.msdmanuals.com/professional/genitourinary-disorders/urinary-tract-infections-utis/bacterial-urinary-tract-infections>
- Hsiao, C.-Y., Lin, H.-L., Lin, Y.-K., Chen, C.-W., Cheng, Y.-C., Lee, W.-C., & WU, T.-C. (2014). Urinary tract infection in patients with chronic kidney disease. *Turkish Journal of Medical Sciences*, *44*(1), 145–149. <https://doi.org/10.3906/sag-1303-51>
- Thapa, T. B., Pokhrel, S., Lamichhane, A., Singh, V. K., Shrestha, O., Sapkota, M., & Khanal, P. R. (2023). Prevalence and antibiogram of bacteria causing urinary tract infection among patients with chronic kidney disease. *Open Medicine*, *18*(1), 20230824. <https://doi.org/10.1515/med-2023-0824>
- Espi, M., Koppe, L., Fouque, D., & Thaumat, O. (2020). Chronic Kidney Disease-Associated Immune Dysfunctions: Impact of Protein-Bound Uremic Retention Solutes on Immune Cells. *Toxins*, *12*(5), 300. <https://doi.org/10.3390/toxins12050300>
- Dicu-Andrescu, I., Penescu, M. N., Căpușă, C., & Verzan, C. (2022). Chronic Kidney Disease, Urinary Tract Infections and Antibiotic Nephrotoxicity: Are There Any Relationships? *Medicina*, *59*(1), 49. <https://doi.org/10.3390/medicina59010049>
- Komagamine, J., Yabuki, T., Noritomi, D., & Okabe, T. (2022). Prevalence of and factors associated with atypical presentation in bacteremic urinary tract infection. *Scientific Reports*, *12*, 5197. <https://doi.org/10.1038/s41598-022-09222-9>
- Aswani, S. M., Chandrashekar, U., Shivashankara, K., & Pruthvi, B. (2014). Clinical profile of urinary tract infections in diabetics and non-diabetics. *The Australasian Medical Journal*, *7*(1), 29–34. <https://doi.org/10.4066/AMJ.2014.1906>
- Tama, R. T., Hossain, Md. S., Islam, Md. S., Chowdhury, R. S., Rahaman, Md. A., & Alam, Md. A. (2025). Combatting multi-drug resistance in urinary tract infections among diabetic populations: A perspective review. *Health Sciences Review*, *14*, 100209. <https://doi.org/10.1016/j.hsr.2025.100209>
- Hyun, M., Lee, J. Y., Kim, H. ah, & Ryu, S. Y. (2019). Comparison of Escherichia coli and Klebsiella pneumoniae Acute Pyelonephritis in Korean Patients. *Infection & Chemotherapy*, *51*(2), 130–141. <https://doi.org/10.3947/ic.2019.51.2.130>
- Wang, T. Z., Kodiyapalakkal, R. P. L., & Calfee, D. P. (2019). Antimicrobial resistance in nephrology. *Nature reviews. Nephrology*, *15*(8), 463–481. <https://doi.org/10.1038/s41581-019-0150-7>
- Brejijeh, Z., Jubeh, B., & Karaman, R. (2020). Resistance of Gram-Negative Bacteria to Current Antibacterial Agents and Approaches to Resolve It. *Molecules*, *25*(6), 1340. <https://doi.org/10.3390/molecules25061340>
- Overcoming mcr-1 mediated colistin resistance with colistin in combination with other antibiotics | Nature Communications. (n.d.). Retrieved March 14, 2025, from <https://www.nature.com/articles/s41467-018-02875-z>
- Mondal, A. H., Khare, K., Saxena, P., Debnath, P., Mukhopadhyay, K., & Yadav, D. (2024). A Review on Colistin Resistance: An Antibiotic of Last Resort. *Microorganisms*, *12*(4), 772. <https://doi.org/10.3390/microorganisms12040772>
- Saifuddin, M., Sharifuzzaman, M., Mahmud, R., Selim, S., Uddin, M. N., Hossain, M. A., ... Pathan, M. F. (2022). Antimicrobial Resistance Pattern Among Diabetic Patients With Urinary Tract Infection at Bangladesh. *Journal of Dhaka Medical College*, *31*(1), 5–8. <https://doi.org/10.3329/jdmc.v31i1.65428>
- Hoque, R., Ahmed, S. M., Naher, N., Islam, M. A., Rousham, E. K., Islam, B. Z., & Hassan, S. (2020). Tackling antimicrobial resistance in Bangladesh: A scoping review of policy and practice in human, animal and environment sectors. *PLoS ONE*, *15*(1), e0227947. <https://doi.org/10.1371/journal.pone.0227947>
- 2016-23 National Antimicrobial Resistance (AMR) Surveillance Report, Bangladesh. (n.d.). Retrieved from https://dashboard.iedcr.gov.bd/amr/uploads/amr_report_2016-23.pdf

20. Stojceva-Taneva, O., Otovic, N. E., & Taneva, B. (2016). Prevalence of Diabetes Mellitus in Patients with Chronic Kidney Disease. *Open Access Macedonian Journal of Medical Sciences*, 4(1), 79–82. <https://doi.org/10.3889/oamjms.2016.019>
21. Piani, F., Melena, I., Tommerdahl, K. L., Nokoff, N., Nelson, R. G., Pavkov, M. E., ... Bjornstad, P. (2021). Sex-related differences in diabetic kidney disease: a review on the mechanisms and potential therapeutic implications. *Journal of diabetes and its complications*, 35(4), 107841. <https://doi.org/10.1016/j.jdiacomp.2020.107841>
22. Giandalia, A., Giuffrida, A. E., Gembillo, G., Cucinotta, D., Squadrito, G., Santoro, D., & Russo, G. T. (2021). Gender Differences in Diabetic Kidney Disease: Focus on Hormonal, Genetic and Clinical Factors. *International Journal of Molecular Sciences*, 22(11), 5808. <https://doi.org/10.3390/ijms22115808>
23. Alicic, R. Z., Rooney, M. T., & Tuttle, K. R. (2017). Diabetic Kidney Disease. *Clinical Journal of the American Society of Nephrology: CJASN*, 12(12), 2032–2045. <https://doi.org/10.2215/CJN.11491116>
24. Muppidi, V., Meegada, S. R., & Rehman, A. (2025). Secondary Hyperparathyroidism. In *StatPearls*. Treasure Island (FL): StatPearls Publishing. Retrieved from <http://www.ncbi.nlm.nih.gov/books/NBK557822/>
25. Hyperparathyroidism - Symptoms & causes - Mayo Clinic. (n.d.). Retrieved March 15, 2025, from <https://www.mayoclinic.org/diseases-conditions/hyperparathyroidism/symptoms-causes/syc-20356194>
26. Dimitrijevic, Z., Paunovic, G., Tasic, D., Mitic, B., & Basic, D. (2021). Risk factors for urosepsis in chronic kidney disease patients with urinary tract infections. *Scientific Reports*, 11(1), 14414. <https://doi.org/10.1038/s41598-021-93912-3>
27. Zhou, S., Ren, G., Liu, Y., Liu, X., Zhang, L., Xu, S., & Wang, T. (2022). Challenge of evolving *Klebsiella pneumoniae* infection in patients on hemodialysis: from the classic strain to the carbapenem-resistant hypervirulent one. *International Journal of Medical Sciences*, 19(3), 416–424. <https://doi.org/10.7150/ijms.69577>
28. Matsumoto, Y. (1998). Combination Cefixime/Amoxicillin against Penicillin-Resistant *Streptococcus pneumoniae* Infection. *Chemotherapy*, 44(Suppl. 1), 6–9. <https://doi.org/10.1159/000048456>
29. Antimicrobial resistance. (n.d.). Retrieved March 15, 2025, from <https://www.who.int/news-room/fact-sheets/detail/antimicrobial-resistance>
30. Afriyie, D. K., Adu, L. B., Dzradosi, M., Amponsah, S. K., Ohene-Manu, P., & Manu-Ofei, F. (2018). Comparative in vitro activity of ciprofloxacin and levofloxacin against isolated uropathogens in Ghana: a pilot study. *The Pan African Medical Journal*, 30, 194. <https://doi.org/10.11604/pamj.2018.30.194.15457>
31. Meropenem - an overview | ScienceDirect Topics. (n.d.). Retrieved March 15, 2025, from <https://www.sciencedirect.com/topics/medicine-and-dentistry/meropenem>
32. Young, M., & Plosker, G. L. (2001). Piperacillin/tazobactam: a pharmaco-economic review of its use in moderate to severe bacterial infections. *PharmacoEconomics*, 19(11), 1135–1175. <https://doi.org/10.2165/00019053-200119110-00006>
33. Castanheira, M., Simner, P. J., & Bradford, P. A. (2021). Extended-spectrum β -lactamases: an update on their characteristics, epidemiology and detection. *JAC-Antimicrobial Resistance*, 3(3), dlab092. <https://doi.org/10.1093/jacamr/dlab092>