IJPSR (2026), Volume 17, Issue 6 **(Research Article)**

INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES AND RESEARCH

Received on 04 January 2026; received in revised form, 25 February 2026; accepted, 27 February 2026; published 01 June 2026

PREVALENCE AND ANTIMICROBIAL SUSCEPTIBILITY OF GRAM-NEGATIVE BACTERIA IN THE URINE SAMPLE OF DIABETIC PATIENTS IN TERTIARY CARE HOSPITAL

T. Raghupathy [* 1], N. Paramasivan [2], Rubavathi Anandhan [3] and P. Saravanan [1]

Department of Pharmacology [1], Government Medical College and Hospital, Namakkal - 637003, Tamil Nadu, India.
Department of Pharmacology [2], Nandha Medical College and Hospital, Erode - 638052, Tamil Nadu, India.
Department of Microbiology [3], Periyar University, Salem - 636011, Tamil Nadu, India.

Keywords:

Urinary tract infection, Diabetes mellitus, Gram-negative bacteria, Anti-microbial resistance, ESBL

Correspondence to Author:
Dr. T. Raghupathy

Assistant Professor
Department of Pharmacology,
Government Medical College and Hospital, Namakkal - 637003, Tamil Nadu, India.

E-mail: rubavathi040197@gmail.com

ABSTRACT: To determine the prevalence and antimicrobial susceptibility patterns of Gram-negative uropathogens isolated from diabetic patients with urinary tract infection (UTI) attending a tertiary care hospital in Namakkal, Tamil Nadu. A hospital-based cross-sectional study was conducted on 225 midstream urine samples collected from clinically suspected diabetic UTI patients, of which 132 showed significant bacteriuria. Bacterial identification was carried out using standard microbiological methods. Antimicrobial susceptibility testing was performed by the Kirby–Bauer disc diffusion method following CLSI 2023 guidelines, and extended-spectrum β-lactamase (ESBL) production was evaluated. Gram-negative bacteria constituted the majority of uropathogens. *Escherichia coli* was the most frequently isolated organism, followed by *Klebsiella pneumoniae* and *Pseudomonas aeruginosa*. Carbapenems and amikacin demonstrated the highest antimicrobial activity, whereas commonly prescribed oral antibiotics such as ampicillin, ciprofloxacin, and cotrimoxazole showed poor effectiveness. A considerable proportion of isolates were ESBL producers, indicating a high burden of multidrug resistance. UTIs were more prevalent among female diabetic patients, particularly those with longer disease duration. These findings highlight the predominance of multidrug-resistant Gram-negative uropathogens in diabetic UTIs and emphasize the importance of routine culture-guided therapy to ensure effective treatment and limit the emergence of resistance.

INTRODUCTION:

Background and Global Burden of Diabetes Mellitus: Diabetes mellitus (DM) is a group of chronic metabolic disorders characterized by hyperglycemia due to defects in insulin secretion, insulin action, or both (American Diabetes Association, 2008) [2].

QUICK RESPONSE CODE

DOI: 10.13040/IJPSR.0975-8232.17(6).1950-57

This article can be accessed online on www.ijpsr.com

DOI link: https://doi.org/10.13040/IJPSR.0975-8232.17(6).1950-57

DM represents a growing global health burden, with over 366 million people affected worldwide a number projected to rise to 552 million by 2030 (Whitinga *et al.*, 2011) [39]. India is at the forefront of this epidemic, currently home to an estimated 77 million diabetic individuals, a figure expected to surpass 134 million by 2045 (Pradeepa and Mohan, 2021) [29].

Among the many complications associated with diabetes, urinary tract infections (UTIs) remain one of the most prevalent. Diabetic patients are particularly susceptible to UTIs due to glycosuria (glucose in urine), impaired immune response, and

autonomic neuropathy leading to urinary retention and stasis [3, 20]. The presence of glucose in the urine serves as a growth medium for bacterial pathogens, further increasing the risk of colonization and infection [7, 33].

Burden of Urinary Tract Infections: UTIs are among the most common bacterial infections globally, affecting approximately 150 million people each year [11]. These infections can involve any part of the urinary system urethra, bladder, ureters, and kidneys and may present as either lower (cystitis) or upper (pyelonephritis) UTIs. Women are disproportionately affected due to anatomical and physiological factors, though men and older adults, especially those with comorbidities like diabetes, are also at significant risk [13, 15].

UTIs in Diabetic Patients: Diabetic individuals are two to four times more likely to develop UTIs than non-diabetics [27]. The incidence of asymptomatic bacteriuria in diabetic women is reported at 9–27%, compared to 0.7–3% in non-diabetic women [16]. UTIs in diabetic patients tend to be more severe, recurrent, and complicated. Common risk factors include impaired neutrophil function, decreased cytokine response, glycosuria, bladder dysfunction due to autonomic neuropathy, and poor glycemic control.

Etiology and Pathophysiology of UTIs in Diabetics: Gram-negative bacteria are the predominant uropathogens in diabetic individuals. Escherichia coli is the most frequently isolated organism, followed by *Klebsiella pneumoniae*, *Proteus mirabilis*, *Pseudomonas aeruginosa*, and *Enterobacter* spp. [17, 34]. In cases of complicated UTIs, organisms such as *Enterococcus* spp., *Staphylococcus aureus*, coagulase-negative *staphylococci* (CoNS), and *Candida* spp. may also be involved [18]. Diabetic patients often have compromised immunity due to hyperglycemia-induced dysfunction of neutrophils and other immune cells, which makes them more susceptible to infections.

Bladder dysfunction due to autonomic neuropathy and elevated glucose concentrations in urine create an ideal environment for microbial colonization and proliferation. These infections can become complicated, leading to recurrent UTIs, bacteremia, and kidney damage if left untreated.

Antimicrobial Resistance: A major challenge in the treatment of UTIs is the rising prevalence of antimicrobial resistance. Frequent and empirical use of antibiotics without proper sensitivity testing has led to the emergence of multidrug-resistant (MDR) uropathogens. This is particularly concerning in developing countries, where antibiotic misuse is widespread, compounded by the presence of substandard medications and lack of infection control measures [12, 41]. Resistance to commonly used antibiotics like ampicillin, ciprofloxacin, and trimethoprim-sulfamethoxazole has been documented, whereas drugs like nitrofurantoin and amikacin may retain better efficacy [40, 42].

Clinical Presentation and Classification of UTIs: Clinically, UTIs are classified as uncomplicated or complicated. Uncomplicated UTIs occur in otherwise healthy individuals, typically presenting as cystitis or pyelonephritis. Complicated UTIs are associated with underlying structural or functional abnormalities, immunosuppression, or comorbidities like diabetes mellitus. These infections often involve multidrug-resistant organisms (MDROs) and require more intensive treatment [13, 22]. Catheter-associated urinary tract infections (CAUTIs) are a subtype of complicated UTIs and are particularly prevalent in hospital settings. Diabetic patients undergoing catheterization are at increased risk, and CAUTIs are the leading cause of secondary bloodstream infections in hospitalized patients [23].

Gram-negative Bacteria in UTIs and Resistance Mechanisms: Gram-negative bacilli such as *E. coli*, *Klebsiella* spp., *Proteus* spp., and *Pseudomonas* spp. are responsible for the majority of UTIs, especially in diabetics. These organisms are often highly adaptive and capable of acquiring resistance mechanisms such as extended-spectrum beta-lactamase (ESBL) production, efflux pumps, and porin mutations, making them formidable pathogens in clinical practice [24, 26].

UTIs caused by multi-resistant Gram-negative bacteria are increasingly reported in community and hospital settings. Treatment options are limited,

and therapy often requires second-line or last-resort antibiotics, contributing to healthcare costs and complications.

Empirical Treatment and the Need for Surveillance: UTI treatment often begins empirically, especially in resource-limited settings. However, due to the dynamic nature of antimicrobial resistance, such empirical strategies may lead to treatment failure and recurrence. Surveillance of local antimicrobial resistance patterns is essential to guide rational therapy. Novel approaches, such as anti-virulence therapies that target bacterial pathogenesis without affecting the normal flora, are also being explored [21].

Current Trends in Antimicrobial Therapy for UTIs: The traditional first-line antibiotics for UTIs include trimethoprim-sulfamethoxazole, ciprofloxacin, and ampicillin. However, resistance to these agents has increased, necessitating susceptibility testing before initiating treatment. Newer approaches are exploring anti-virulence strategies that target pathogen-specific mechanisms without affecting the commensal microbiota.

Despite these advancements, many regions, especially rural or semi-urban settings in developing countries, still rely on empirical treatment without microbiological confirmation. This practice exacerbates resistance and complicates the management of UTIs.

Impact and Importance of Local Studies: There is limited data from rural and semi-urban areas in India regarding the prevalence and antimicrobial susceptibility patterns of Gram-negative uropathogens in diabetic patients. Most existing studies have been conducted in urban tertiary care centers, potentially overlooking regional differences in microbial profiles and resistance patterns. This highlights the importance of localized studies to inform public health policies and clinical guidelines.

In the Indian context, Tamil Nadu has witnessed increasing rates of DM and associated infections. The Tertiary care hospital caters to a semi-urban and rural population, making it an important center for studying regional disease patterns and antimicrobial susceptibility profiles. A previous study conducted in a tertiary care hospital in South Tamil Nadu identified common UTI pathogens and their resistance patterns, underscoring the necessity of continuous surveillance (FMHACA, 2014). However, there is limited published data specifically focusing on diabetic patients from Namakkal. Given the growing prevalence of diabetes and AMR, region-specific data are essential for tailoring empirical antibiotic therapy and infection control strategies.

Rationale and Objectives of the Study: Given the high burden of diabetes and the increasing resistance of uropathogens to common antibiotics, this study aims to investigate the prevalence and antimicrobial susceptibility patterns of Gram-negative bacteria isolated from urine samples of diabetic patients attending Tertiary care hospital. This research will help identify the predominant uropathogens affecting diabetic individuals, assess their antibiotic susceptibility and resistance profiles, and provide valuable data to support local treatment guidelines and antimicrobial stewardship efforts. The rising incidence of diabetes and its associated complications, including UTIs, combined with the threat of antimicrobial resistance, presents a critical public health challenge. Understanding the microbiological and resistance profiles of pathogens in specific populations is essential for effective infection management. This study will contribute valuable insights into the patterns of Gram-negative bacterial infections in diabetic patients in Tertiary care hospital, Namakkal informing both clinical practice and public health policy.

MATERIALS AND METHODS:

Study Area: This cross-sectional descriptive study was conducted in the Department of Microbiology, Tertiary Care Hospital, Namakkal, Tamil Nadu, India. The study was carried out over a three-month period from August to October 2025.

Sampling and Data Collection: A total of 225 urine samples were collected from consecutive diabetic patients with clinically suspected urinary tract infections (UTIs) who attended the Tertiary Care Hospital, Namakkal, during the study period. Patients were classified as having suspected UTI based on the presence of at least one urinary symptom (dysuria, increased urinary frequency, urgency, suprapubic pain, flank pain, or fever)

along with a clinician's request for urine culture. Only symptomatic patients were included in the study. Cases of asymptomatic bacteriuria, defined as significant bacterial growth in the absence of urinary symptoms, were excluded. No eligible patient was excluded during the study period. Thus, 225 represents all suspected diabetic UTI cases investigated. Of these, 132 samples (58.7%) showed significant bacteriuria and were considered culture-positive UTIs. Among the culture-positive samples, 109 (82.6%) yielded Gram-negative bacterial isolates and were included for organism distribution and antimicrobial susceptibility analysis.

Source of Urine Samples: Urine specimens were obtained either as midstream clean-catch samples or from patients with indwelling urinary catheters using aseptic catheter-port aspiration. Catheterized samples were included only when clinical features of UTI were present. Routine screening samples from catheterized but asymptomatic patients were excluded.

Pre-analytical Handling of Urine Samples: All urine specimens were transported to the Microbiology laboratory in insulated cold boxes maintained at 4–8 °C and processed within 4–6 hours of collection. Refrigerated transport was used to prevent bacterial overgrowth and preserve original colony counts. No chemical preservatives were added. When immediate processing was not possible, samples were stored at 4 °C and cultured within the recommended time limits according to standard clinical microbiology guidelines.

Culture and Isolation of Uropathogens: Each urine sample was inoculated using a calibrated 0.001 mL loop onto CLED agar and MacConkey agar for quantitative culture and primary isolation. CLED agar was used to prevent Proteus swarming and permit accurate colony counting, while MacConkey agar was used for differentiation of lactose-fermenting and non-lactose-fermenting Gram-negative bacteria. Plates were incubated at 37 °C for 18–24 hours and colony counts were performed. Distinct colonies were sub-cultured onto Nutrient Agar to obtain pure isolates. Identification of Gram-negative bacteria was carried out using Gram staining and standard biochemical tests including indole, citrate, urease, triple sugar iron (TSI), and motility tests.

Quantitative Urine Culture and Definition of Significant Bacteriuria: Urine was cultured using a calibrated 0.001 mL (1 µL) loop onto CLED and MacConkey agar plates. After incubation at 37 °C for 18–24 hours, colonies were counted and the bacterial load was calculated by multiplying the colony count by 1,000 to obtain colony-forming units per milliliter (CFU/mL). A bacterial count of $\geq 10^5$ CFU/mL was considered significant bacteriuria.

Definition and Handling of Contaminated or Mixed-Growth Samples: Urine cultures showing growth of two or more morphologically distinct bacterial species without a predominant uropathogen were considered contaminated and excluded from analysis. Samples showing mixed growth suggestive of improper collection were not subjected to antimicrobial susceptibility testing and were not included in prevalence or resistance analysis.

Statistical Analysis: Data were analyzed using descriptive statistics and are presented as frequencies and percentages.

RESULTS: Among the 109 Gram-negative isolates, *Escherichia coli* was the most frequently identified pathogen, accounting for 54 isolates (49.5%), followed by *Klebsiella pneumoniae* with 28 isolates (25.7%). *Pseudomonas aeruginosa* constituted 14 isolates (12.8%), while *Proteus mirabilis* and *Enterobacter* spp. accounted for 8 (7.3%) and 5 (4.6%) isolates, respectively.

TABLE 1: DISTRIBUTION OF GRAM-NEGATIVE BACTERIAL ISOLATES (N = 109) FROM CULTURE-POSITIVE DIABETIC UTI PATIENTS

Bacterial species	Number of Isolates	Percentage (%)
Escherichia coli	54	49.5
Klebsiella pneumoniae	28	25.7
Pseudomonas aeruginosa	14	12.8
Proteus mirabilis	8	7.3
Enterobacter spp.	5	4.6
Total	109	100

Antimicrobial susceptibility testing of the 109 Gram-negative isolates showed the highest overall activity for carbapenems and aminoglycosides. Imipenem demonstrated susceptibility in 101 isolates (92.7%), followed by meropenem in 99 isolates (90.8%) and amikacin in 97 isolates (88.9%). Piperacillin–tazobactam also showed good activity with 92 susceptible isolates (84.4%), while cefepime was active against 86 isolates (78.9%). Moderate susceptibility was observed for ceftazidime (75 isolates, 68.8%), gentamicin (77 isolates, 70.6%), and nitrofurantoin (66 of tested isolates, 70.2%). Lower susceptibility rates were recorded for levofloxacin (56 isolates, 51.4%) and ceftriaxone (42 of tested isolates, 44.7%). High resistance was observed to ciprofloxacin (69 isolates, 63.3%), cotrimoxazole (56 of tested isolates, 58.1%), and ampicillin (77 of tested isolates, 84.7%). Among *E. coli* isolates (n = 54), the highest susceptibility was observed for imipenem (92.6%), meropenem (90.7%), and amikacin (88.9%), whereas resistance was highest to ampicillin (87.0%) and ciprofloxacin (63.0%). *Klebsiella pneumoniae* (n = 28) showed similar susceptibility to imipenem (92.9%), meropenem (89.3%), and amikacin (89.3%), but high resistance to ampicillin (82.1%) and ciprofloxacin (60.7%). *Pseudomonas aeruginosa* (n = 14) demonstrated high susceptibility to imipenem and meropenem (92.9% each) and amikacin (85.7%), with resistance to ciprofloxacin observed in 64.3% of isolates. *Proteus mirabilis* (n = 8) showed high susceptibility to carbapenems, aminoglycosides, and piperacillin–tazobactam, but marked resistance to ampicillin (87.5%). *Enterobacter* spp. (n = 5) exhibited 100% susceptibility to imipenem, meropenem, amikacin, and cefepime, while 80% were resistant to ampicillin. Extended-spectrum beta-lactamase (ESBL) production was detected in 34 of the 109 Gram-negative isolates (31.2%), with ESBL positivity observed in 16 *E. coli* (29.6%), 9 *K. pneumoniae* (32.1%), 3 *P. mirabilis* (37.5%), and 2 *Enterobacter* spp. (40%) isolates.

TABLE 2: ANTIMICROBIAL SUSCEPTIBILITY PATTERN OF GRAM-NEGATIVE ISOLATES (N = 109)

Antibiotic	Pattern	*E. coli* n=54	*K. pneumoniae* n=28	*P. aeruginosa* n=14	*P. mirabilis* n=8	*Enterobacter* spp. n=5	Total (n=109)
Imipenem	S	50 (92.6	26 (92.9)	13 (92.9)	7 (87.5)	5 (100)	101 (92.7)
	R	4 (7.4)	2 (7.1)	1 (7.1)	1 (12.5)	0	8 (7.3)
Meropenem	S	49 (90.7)	25 (89.3)	13 (92.9)	7 (87.5)	5 (100)	99 (90.8)
	R	5 (9.3)	3 (10.7)	1 (7.1)	1 (12.5)	0	10(9.2)
Amikacin	S	48 (88.9)	25 (89.3)	12 (85.7)	7 (87.5)	5 (100)	97 (88.9)
	R	6 (11.1)	3 (10.7)	2 (14.3)	1 (12.5)	0	12 (11.1)
Piperacillin–	S	46 (85.2)	24 (85.7)	12 (85.7)	7 (87.5)	3 (60)	92 (84.4)
Tazobactam	R	8 (14.8)	4 (14.3)	2 (14.3)	1 (12.5)	2 (40)	17 (15.6)
Cefepime	S	43 (79.6)	21 (75)	11(78.6)	6 (75.0)	5 (100)	86 (78.9)
	R	11(20.4)	7 (25)	3 (21.4)	2 (25.0)	0	23 (21.1)
Ceftazidime	S	37 (68.5)	19 (67.9)	10 (71.4)	5 (62.5)	4 (80)	75 (68.8)
	R	17 (31.5)	9 (32.1)	4 (28.6)	3 (37.5)	1 (20)	34 (31.2)
Gentamicin	S	38 (70.4)	19 (67.9)	10 (71.4)	6 (75.0)	4 (80)	77 (70.6)
	R	16 (29.6)	9 (32.1)	4 (28.6)	2 (25.0)	1 (20)	32 (29.4)
Levofloxacin	S	28 (51.9)	15 (53.6)	7 (50)	4 (50.0)	2 (40)	56 (51.4)
	R	26 (48.1)	13 (46.4)	7 (50)	4 (50.0)	3 (60)	53 (48.6)
Ciprofloxacin	S	20 (37)	11(39.3)	5 (35.7)	2 (25.0)	2 (40)	40 (36.7)
	R	34 (63)	17 (60.7)	9 (64.3)	6 (75.0)	3 (60)	69 (63.3)
Nitrofurantoin	S	39 (72.2)	20 (71.4)	-	7 (87.5)	-	66 (70.2)*
	R	15 (27.8)	8 (28.6)	-	1 (12.5)	-	24 (29.8)*
Ceftriaxone	S	24 (44.4)	13 (46.4)	-	3 (37.5)	2 (40)	42 (44.7)*
	R	30 (55.6)	15 (53.6)	-	5 (62.5)	3 (60)	53 (55.3)*
Cotrimoxazole	S	22 (40.7)	12 (42.9)	-	3 (37.5)	2 (40)	39 (41.9)*
	R	32 (59.3)	16 (57.1)	-	5 (62.5)	3 (60)	56 (58.1)*
Ampicillin	S	7 (13)	5 (17.9)	-	1 (12.5)	-	13 (15.3)*
	R	47 (87)	23 (82.1)	-	7 (87.5)	-	77 (84.7)*
ESBL Positive	-	16 (29.6)	9 (32.1)	-	3 (37.5)	2 (40)	34 (31.2)

Among the 132 culture-positive diabetic patients, 81 (61.4%) were female and 51 (38.6%) were male. The age of the patients ranged from 18 to over 70 years, with a mean age of 54.6 ± 13.2 years. The

largest proportion of patients belonged to the 41–50-year age group (30 patients, 22.7%), followed by 31–40 years (28 patients, 21.2%) and 51–60 years (24 patients, 18.2%). Patients aged 18–30 years accounted for 22 cases (16.7%), while 18 patients (13.6%) were in the 61–70-year group and 10 patients (7.6%) were older than 70 years. Regarding the duration of diabetes, most patients had a disease duration of 5–10 years, comprising 78 cases (59.1%). Patients with a duration of less than 5 years accounted for 35 cases (26.5%), while 19 patients (14.4%) had diabetes for more than 10 years.

TABLE 3: DEMOGRAPHIC CHARACTERISTICS OF DIABETIC PATIENTS WITH CULTURE-POSITIVE UTIS (N = 132)

Characteristic	Category	n (%)
Gender	Female	81(61.4)
	Male	51 (38.6)
Agegroup (years)	18–30	22(16.7)
	31-40	28 (21.2)
	41-50	30 (22.7)
	51-60	24 (18.2)
	61-70	18 (13.6)
	>70	10 (7.6)
Meanage±SD	-	54.6±13.2
Durationof diabetes	<5years	35 (26.5)
	5–10 years	78 (59.1)
	>10years	19(14.4)

DISCUSSION: The present study demonstrates that more than half of diabetic patients with clinically suspected UTIs had significant bacteriuria, with Gram-negative organisms accounting for over 80% of isolates. This finding is consistent with previous reports showing that Gram-negative uropathogens dominate UTIs in diabetic patients due to their ability to adhere to uroepithelial cells and thrive in glucose-rich urine [16, 17].

E. coli was the predominant pathogen, similar to findings reported in Ethiopia, India and other regions [5, 40, 42]. The high proportion of *K. pneumoniae* also aligns with earlier studies [19, 30]. The relatively higher prevalence of *P. aeruginosa* in this study compared with some reports may reflect increased hospital exposure and catheter use in diabetic patients [8].

The antimicrobial susceptibility pattern revealed that carbapenems and amikacin remain highly effective against Gram-negative uropathogens. Similar high carbapenem activity has been reported from India and Africa [4, 32, 37]. Piperacillin–tazobactam and cefepime also demonstrated good activity, suggesting their usefulness in hospital-acquired infections. In contrast, high resistance to ampicillin, ciprofloxacin and cotrimoxazole was observed, which reflects the widespread misuse of these agents as empirical therapy. This pattern is consistent with global antimicrobial resistance trends reported by WHO (2017) and earlier diabetic UTI studies [1, 17].

The ESBL prevalence of 31.2% is within the range reported in previous Indian and regional studies [35, 36], indicating a significant burden of multidrug-resistant organisms. This highlights the importance of routine ESBL screening and strict infection-control measures.

Female predominance and the concentration of cases in middle-aged and elderly patients are consistent with earlier reports [17, 30, 42]. The higher risk among patients with longer diabetes duration may be attributed to chronic hyperglycemia, impaired immune responses and bladder dysfunction [16, 17].

Overall, the findings emphasize the need for culture-guided therapy rather than empirical use of fluoroquinolones, cotrimoxazole or ampicillin in diabetic UTIs, in order to limit treatment failure and the spread of resistant organisms.

CONCLUSION: This study demonstrated that Gram-negative bacteria, predominantly *Escherichia coli* and *Klebsiella pneumoniae*, were the leading causes of urinary tract infections among diabetic

patients at a tertiary care hospital in Namakkal, Tamil Nadu. More than half of the clinically suspected cases showed significant bacteriuria, and a substantial proportion of isolates exhibited resistance to commonly used antibiotics such as ampicillin, ciprofloxacin, and cotrimoxazole. In contrast, higher in-vitro susceptibility was observed for carbapenems and amikacin, while nitrofurantoin and piperacillin–tazobactam showed moderate activity. The detection of ESBL-producing organisms highlights the growing burden of multidrug resistance in this setting. These findings emphasize the importance of culture-guided therapy for the management of UTIs in diabetic patients. However, as this was a single-center study with a limited study period, the results should be interpreted within this context.

ACKNOWLEDGEMENTS: Nil

CONFLICTS OF INTEREST: The authors declare that there are no conflicts of interest. Written informed consent for publication was obtained from all participants.

REFERENCES:

1. Al-Rubeaan K, Youssef AM, Subhani SN, Ahmad NA, Al-Sharqawi AH, Al-Mutlaq HM, David SK and Al-Naqeb D: Urinary tract infection among diabetic patients: A retrospective analysis in Saudi Arabia. PLoS ONE 2014; 9(2): 88956. https://doi.org/10.1371/journal.pone.0088956.
2. American Diabetes Association: Diagnosis and classification of diabetes mellitus. Diabetes Care 2008; 21(1): 62–S67.https://doi.org/10.2337/dc08-S055.
3. Baqai R, Aziz M and Rasool G: Urinary tract infection in diabetic patients and biofilm formation of uropathogens. Infectious Diseases Journal of Pakistan 2008; 17(1): 7–9.
4. Bellolemoto OI, Tejumade DO, Fakayode SO, Motayo BO and Adebiyi BO: Antibiotic susceptibility pattern of urinary tract pathogens in Ibadan, Nigeria. African Journal of Clinical and Experimental Microbiology 2015; 16(1). https://doi.org/10.4314/ajcem.v16i1.3.
5. Borj MA: Prevalence and antibiotic resistance of urinary tract pathogens in diabetic patients. Journal of Clinical Microbiology and Infectious Diseases 2017; 29(1): 45–52.
6. Cheesbrough M: Morphology and characterization of E. coli and *S. aureus*. District laboratory practice in tropical countries part II. Cambridge University 2004; 157-179.
7. Chita T, Timar B and Muntean D: Urinary tract infections in Romanian patients with diabetes: prevalence, etiology, and risk factors. Therapeutics and Clinical Risk Management 2016; 13: 1–7. https://doi.org/10.2147/TCRM.S123226.
8. Cottalorda A, Dahyot S and Soares A: Phenotypic and genotypic within- host diversity of *Pseudomonas aeruginosa* urinary isolates. Scientific Reports 2022; 12: 5421. https://doi.org/10.1038/s41598-022-09234-5.
9. Cowan ST and Steel KJ: Manual for the identification of medical bacteria. 4th edn. Cambridge University Press. London 1985; 217.
10. Fawole MO and Oso BA: (1988). Laboratory manual for microbiology, 1st edn. Spectrum Book Ltd., Ibadan.22- 45.
11. Flores-Mireles, AL, Jennifer N Walker, Michael Caparon, Scott J Hultgren: Urinary tract infections: epidemiology, mechanisms of infection and treatment options. Nature Reviews Microbiology 2015; 13: 269–284. https://doi.org/10.1038/nrmicro3432.
12. Food Medicine and Healthcare Administration and Control Authority of Ethiopia (FMHACA) Standard Treatment Guidelines for General Hospitals. 3rd. Addis Ababa, Ethiopia: FMHACA 2014.
13. Foxman B: The epidemiology of urinary tract infection. Nature Reviews Urology 2010; 7: 653–660. https://doi.org/10.1038/nrurol.2010.190.
14. Foxman B: Urinary tract infection syndromes: occurrence, recurrence, bacteriology, risk factors, and disease burden. Infectious Disease Clinics of North America 2014; 28: 1–13. https://doi.org/10.1016/j.idc.2013.09.003.
15. Gabriel, HB and Shehu F: Prevalence and antibiotic susceptibility patterns of bacterial etiologies of urinary tract infections among students attending Sick-Bay of Ahmadu Bello University Nigeria. Edorium Journal of Microbiology 2016; 2: 7–12. https://doi.org/10.5348/M08-2016-4-OA-2.
16. Geerlings SE, Stolk RP, Camps MJ, Netten PM, Hoekstra JB and Bouter KP: Asymptomatic bacteriuria may be considered a complication in women with diabetes. Advances in Experimental Medicine and Biology 2000; 485: 309–314. https://doi.org/10.1007/0-306-46840-9_41.
17. Hamdan H, Kubbara E, Adam A, Hassan O, Suliman S and Adam I: Urinary tract infections and antimicrobial sensitivity among diabetic patients at Khartoum, Sudan. Annals of Clinical Microbiology and Antimicrobials 2015; 14(26): 1–6. https://doi.org/10.1186/s12941-015-0082-4.
18. Jacobsen SM, Stickler DJ, Mobley HL and Shirtliff ME: Complicated catheter-associated urinary tract infections due to Escherichia coli and Proteus mirabilis. Clinical Microbiology Reviews 2008; 21: 26–59. https://doi.org/10.1128/CMR.00019-07.
19. Kebamo S, Dabsu R, Deressa A and Gebre M: Urinary tract infection: Bacterial etiologies, drug resistance profile and associated risk factors among diabetic patients attending Nekemte Referral Hospital. American Journal of Current Microbiology 2017; 5(1): 19–32. http://ivyunion.org/index.php/ajcmicrob/.
20. Ketema HW, Geleta DA, Tulu KD, Aber NA, Legese MH and Fenta GM: Common uropathogens and their antibiotic susceptibility pattern among diabetic patients. BMC Infectious Diseases 2019; 19: 43. https://doi.org/10.1186/s12879-018-3669-5.
21. Kostakioti M, Hultgren SJ and Hadjifrangiskou M: Molecular blueprint of uropathogenic *Escherichia coli* virulence provides clues toward the development of anti-virulence therapeutics. Virulence 2012; 3: 592–594. https://doi.org/10.4161/viru.22364.
22. Lichtenberger P and Hooton TM: Complicated urinary tract infections. Current Infectious Disease Reports 2008; 10: 499–504. https://doi.org/10.1007/s11908-008-0081-0.
23. Lo E: Strategies to prevent catheter-associated urinary tract infections in acute care hospitals: Infection Control and Hospital Epidemiology 2014: 35: 464–479. https://doi.org/10.1086/675718.
24. McManus MC: Mechanisms of bacterial resistance to antimicrobial agents. American Journal of Health-System Pharmacy 1997; 54: 1420-1433. https://doi.org/10.1093/ajhp/54.12.1420.

25. Mishra R, Pandey J, Singh AK and Jasuja K: Bacteriological profile and sensitivity pattern of microorganisms causing Urinary Tract Infection at a tertiary care center in eastern Uttar Pradesh. International Journal of Biomedical and Advance Research 2016; 7(6): 292. https://doi.org/10.7439/ijbar.v7i6.3366.
26. Neu HC: The crisis in antibiotic resistance. Sci 1992; 257: 1064-1073. https://doi.org/10.1126/science.257.5073.1064.
27. Nitzan O, Elias M, Chazan B and Saliba W: Urinary tract infections in patients with type 2 diabetes mellitus: review of prevalence, diagnosis, and management. Diabetes, Metabolic Syndrome and Obesity 2015; 8: 129–136. https://doi.org/10.2147/DMSO.S51792.
28. Patel HB: Causative agents of urinary tract infections and their antimicrobial susceptibility patterns in a tertiary care setting. Journal of Family Medicine and Primary Care 2019; 8(10). https://doi.org/10.4103/jfmpc.jfmpc_203_18.
29. Pradeepa R and Mohan V: Epidemiology of type 2 diabetes in India. Indian Journal of Ophthalmology 2021; 69(11): 2932–8. https://doi.org/10.4103/ijo.IJO_1627_21.
30. Prakash O and Saxena RK: Profile and outcome of urinary tract infection in diabetic patients: A hospital-based study. Indian J of Medical Microbiology 2013; 31(1): 21–23.
31. Ramesh P, Camacho-Moll R, Vijayakumar G and Viswanathan GV: Prevalence of lower urinary tract infection in South Indian type 2 diabetic. Journal Indian Journal of Nephrology 2011; 22(3). https://doi.org/10.4103/0971-4065.57107
32. Ravishankar U, Sathyamurthy P and Thayanidhi P: Antimicrobial resistance among uropathogens: Surveillance report from South India. Cureus 2021; 13(1): 12913. https://doi.org/10.7759/cureus.12913.
33. Ribera R, Pascual R, Orozco D, Perez B, Pedrera V and Gil V: Incidence and risk factors associated with urinary tract infection in diabetic patients with and without asymptomatic bacteriuria. European Journal of Clinical Microbiology and Infectious Diseases 2006; 25(6): 389–399. https://doi.org/10.1007/s10096-006-0148-5.
34. Ronald A: The etiology of urinary tract infection: traditional and emerging pathogens. The American Journal of Medicine 2002; 113(1): 14–19. https://doi.org/10.1016/S0002-9343(02)01174-8.
35. Shaikh S, Fatima J, Shakil S, Syed Mohd Danish Rizvi and Mohammad Amjad Kamal: Prevalence of multidrug resistant and extended spectrum beta-lactamase producing Pseudomonas aeruginosa in a tertiary care hospital. Saudi Journal of Biological Sciences 2015; 22(1): 62-4. https://doi.org/10.1016/j.sjbs.2014.06.001.
36. Shilpakar A, Ansari M, Raj Rai K, Rai G and Shiba Kumar R: Prevalence of multidrug-resistant and extended-spectrum beta-lactamase producing Gram-negative isolates from clinical samples in a tertiary care hospital of Nepal. Tropical Medicine and Health 2021; 49: 23. https://doi.org/10.1186/s41182-021-00313-3.
37. Somashekara SC, Deepalaxmi S, Jagannath N, Ramesh B, Lavees MR and Govindadas D: Retrospective analysis of antibiotic resistance pattern to urinary pathogens in a tertiary care hospital in South India. Journal of Basic and Clinical Pharmacy 2014; 5(4): 105–108. https://doi.org/10.4103/0976-0105.141948.
38. Timothy OO, Olusesan FJ, Adesola BO, Temitayo AA, David FO and Ige OO: Antibiotic resistance pattern of bacterial isolates from cases of urinary tract infections among hospitalized and out-patients at a tertiary health facility in South Western Nigeria. Annals of Tropical Medicine & Public Health 2014; 7: 130–135. https://doi.org/10.4103/1755-6783.146403
39. Whitinga D, Guariguataa L, Weil C and Shaw J: IDF diabetes atlas: global estimates of the prevalence of diabetes for 2011 and 2030. Diabetes Research and Clinical Practice 2011; 94(3): 311–321. https://doi.org/10.1016/j.diabres.2011.10.029.
40. Worku GY, Alamneh YB and Abegaz WE: Prevalence of bacterial urinary tract infection and antimicrobial susceptibility patterns among diabetes mellitus patients attending Zewditu Memorial Hospital, Addis Ababa, Ethiopia. Infection and Drug Resistance 2021; 14: 1441–1454. https://doi.org/10.2147/IDR.S298176.
41. World Health Organization: Global priority list of antibiotic-resistant bacteria to guide research, discovery, and development of new antibiotics 2017. https://doi.org/10.1016/S1473-3099(17)30753-3.
42. Yismaw G, Asrat D, Woldeamanuel Y and Unakal CG: Urinary tract infection: bacterial etiologies, drug resistance profile and associated risk factors in diabetic patients attending Gondar University hospital, Gondar, Ethiopia. European Journal of Experimental Biology 2012; 2(4): 889–898. https://doi.org/236143050.

How to cite this article:
Raghupathy T, Paramasivan N, Anandhan R and Saravanan P: Prevalence and antimicrobial susceptibility of gram-negative bacteria in the urine sample of diabetic patients in tertiary care hospital. Int J Pharm Sci & Res 2026; 17(6): 1950-57. doi: 10.13040/IJPSR.0975-8232.17(6).1950-57.