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Original Research Article

Microbiological Profile and Antibiotic Susceptibility Pattern of Bacterial Infections in Patients with Chronic Kidney Disease at a Tertiary Care Hospital

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ABSTRACT

Background: Patients with chronic kidney disease (CKD) are highly susceptible to bacterial infections because of uraemia-related immune dysfunction, comorbidities, vascular access and repeated hospitalisation. The emergence of multidrug-resistant (MDR) organisms narrows the therapeutic options further, because many antimicrobials are nephrotoxic or need dose adjustment. Local data on pathogens and their susceptibility patterns are essential for rational empirical therapy.

Aim and objectives: To determine the microbiological profile and antibiotic susceptibility pattern of bacterial infections in patients with CKD, to detect important resistance phenotypes, and to identify factors associated with culture-proven infection.

Materials and methods: A prospective, hospital-based observational study was conducted in the Department of Microbiology of a tertiary care teaching hospital from June 2025 to May 2026. Three hundred adult patients with CKD (KDIGO stages 3–5D) and clinically suspected bacterial infection were enrolled. Urine, blood, respiratory samples, pus/wound swabs, catheter tips/exit-site swabs and sterile body fluids were processed by standard microbiological methods. Identification was by conventional biochemical tests and VITEK 2 Compact. Antimicrobial susceptibility was determined by Kirby–Bauer disk diffusion according to CLSI M100 (33rd edition); colistin MIC was determined by broth microdilution. ESBL, carbapenemase production, MRSA and high-level aminoglycoside resistance were detected phenotypically. MDR, XDR and PDR were defined by international consensus criteria. Data were analysed with SPSS version 25.

Results: Mean age was 52.4 ± 14.6 years and 62.0% of patients were male. Culture was positive in 214 patients (71.3%), yielding 226 isolates; positivity rose from 58.3% in stage 3 to 80.0% in stage 5D ($p = 0.018$). Urine was the commonest sample (46.0%). Gram-negative bacilli constituted 69.9% of isolates, led by *Escherichia coli* (27.4%), *Klebsiella pneumoniae* (15.9%), *Pseudomonas aeruginosa* (10.6%) and *Acinetobacter baumannii* (8.0%); *Staphylococcus aureus* (12.4%) and *Enterococcus* species (11.5%) were the main Gram-positive pathogens. ESBL production occurred in 61.3% of *E. coli* and 66.7% of *K. pneumoniae*; carbapenem resistance was seen in 17.2% of Enterobacterales, 25.0% of *P. aeruginosa* and 66.7% of *A. baumannii*. MRSA accounted for 46.4% of *S. aureus* and VRE for 11.5% of enterococci. Overall, 72.6% of isolates were MDR. Colistin (94.4–100% among Gram-negatives), linezolid (100%) and vancomycin (100% for staphylococci) retained the highest activity. Prior antibiotic use, indwelling catheters, age above 60 years, hypoalbuminaemia and diabetes mellitus were independent predictors of culture-proven infection. In-hospital mortality was higher in culture-positive patients (14.5% vs 5.8%; $p = 0.037$).

Conclusion: Gram-negative, multidrug-resistant bacteria dominate infections in CKD patients at our centre, and resistance increases with advancing CKD stage. Empirical therapy should follow locally validated, CKD-specific antibiograms with renal dose adjustment, and should be de-escalated promptly on culture results. Strengthening infection control, catheter care and antimicrobial stewardship is essential.

Keywords: *Chronic kidney disease; haemodialysis; antibiotic susceptibility; multidrug resistance; ESBL; carbapenem resistance; MRSA; India*

INTRODUCTION

Chronic kidney disease (CKD) has emerged as a major global public health problem. According to the Global Burden of Disease Study 2017, about 697.5 million people were living with CKD worldwide, and the disease was responsible for approximately 1.2 million deaths in that year[1]. The burden is rising fastest in low- and middle-income countries, driven by population ageing and by the growing prevalence of diabetes mellitus and hypertension[2]. In India, the Screening and Early Evaluation of Kidney Disease (SEEK) study reported a CKD prevalence of 17.2% among screened adults[3], while the Indian CKD Registry identified diabetic nephropathy as the leading recorded cause of CKD[4]. Indian nephrologists have repeatedly highlighted the scale of the problem and the limited capacity of the health system to provide renal replacement therapy to all who need it[5,6].

Infection is one of the most important complications of CKD. Infections are a leading cause of hospitalisation and, after cardiovascular disease, the second most frequent cause of death among patients with advanced CKD and those on dialysis[7,8]. Mortality attributed to septicaemia in patients with end-stage renal disease has been reported to be 100 to 300 times higher than in the general population[9], and even moderate reduction in kidney function increases the risk of hospitalisation and death from pneumonia[10].

The predisposition to infection in CKD is multifactorial. The uraemic milieu impairs neutrophil chemotaxis and phagocytosis, monocyte and dendritic cell function, and T- and B-lymphocyte responses, while persistent low-grade inflammation leads to premature immune ageing[11–13]. Superimposed on this are diabetes mellitus, hypoalbuminaemia and malnutrition, frequent hospital contact, immunosuppressive drugs, structural abnormalities and obstruction of the urinary tract, and the use of invasive devices such as haemodialysis catheters and urinary catheters[14].

The spectrum of infection in CKD is wide. Urinary tract infection (UTI) is common and is caused mainly by uropathogenic *Escherichia coli*, other Enterobacterales and enterococci[15]. Dialysis-related bloodstream infections and exit-site infections are caused by *Staphylococcus aureus*, coagulase-negative staphylococci and Gram-negative bacilli, whereas respiratory infections and soft-tissue infections add to the burden. Because the distribution of pathogens and their resistance profiles differ between regions and between hospitals, empirical decisions based on data from other settings can be misleading.

Antimicrobial resistance (AMR) has complicated this picture. It was estimated that bacterial AMR was directly responsible for 1.27 million deaths and associated with 4.95 million deaths worldwide in 2019[16]. In India, a high mortality burden has been attributed to multidrug-resistant (MDR) pathogens[17], and the need for global and national solutions has been emphasised[18]. The Indian Council of Medical Research (ICMR) Antimicrobial Resistance Surveillance Network continues to report high resistance to cephalosporins and fluoroquinolones among Enterobacterales and declining carbapenem susceptibility in non-fermenters[19]. Unregulated and widespread antibiotic use in the country is considered an important driver[20].

CKD patients are a particularly vulnerable group in this regard. They are repeatedly exposed to healthcare settings and antibiotics, which selects resistant flora, and their therapeutic options are restricted because many antimicrobials (aminoglycosides, polymyxins, nitrofurantoin, among others) are nephrotoxic, ineffective at low glomerular filtration or require careful dose adjustment[21]. Choosing an effective empirical regimen at the first attempt is therefore critical. Relatively few studies have described the microbiological profile and resistance patterns exclusively in CKD patients at Indian tertiary care centres. The present study was undertaken to address this gap and to generate local data that can guide empirical therapy and antimicrobial stewardship in this population.

Aim

To study the microbiological profile and antibiotic susceptibility pattern of bacterial infections in patients with chronic kidney disease at a tertiary care hospital.

Objectives

To isolate and identify the bacterial pathogens from clinical samples of CKD patients with suspected infection. To determine the antibiotic susceptibility pattern of the isolates. To detect important resistance phenotypes: ESBL, carbapenem resistance, MRSA, VRE, and MDR/XDR/PDR organisms. To assess the association of culture-proven infection with CKD stage and with clinical risk factors.

MATERIALS AND METHODS

Study design, setting and period

This was a hospital-based, prospective, observational study carried out in the Department of Microbiology, in collaboration with the Department of Nephrology and the Dialysis Unit, of a tertiary care teaching hospital. The study period was 12 months, from June 2025 to May 2026.

Study population

CKD was defined as abnormalities of kidney structure or function present for more than three months, and staged according to the KDIGO 2012 guideline using the estimated glomerular filtration rate (eGFR) calculated by the CKD-EPI 2009 creatinine equation[22,23]. Patients on maintenance haemodialysis for at least three months were labelled stage 5D.

Inclusion criteria: (i) age 18 years or above; (ii) CKD stage 3 to 5D; (iii) clinical suspicion of bacterial infection (fever, localising symptoms or signs, raised inflammatory markers); and (iv) written informed consent.

Exclusion criteria: (i) acute kidney injury without pre-existing CKD; (ii) renal transplant recipients; (iii) infections caused exclusively by fungi, viruses or parasites; (iv) patients who had received antibiotics for more than 72 hours at the time of sampling; (v) repeat isolates from the same infectious episode; and (vi) incomplete records.

Sample size

The sample size was calculated with the formula $n = Z^2pq/d^2$, taking an expected culture positivity of 75%, a 95% confidence level and an absolute precision of 5%. The minimum required sample was 288, which was rounded off to 300 consecutive patients.

Data collection

A pre-designed proforma recorded age, sex, aetiology and stage of CKD, comorbidities, dialysis status and vascular access, history of hospitalisation and antibiotic use in the preceding three months, indwelling devices, immunosuppressive therapy, serum albumin, length of hospital stay and in-hospital outcome. Infections were classified according to the CDC/NHSN criteria[24]. Infection with onset 48 hours or more after admission was labelled hospital-acquired; others were labelled community-onset.

Specimen collection and processing

Specimens were collected aseptically and transported to the laboratory within two hours. *Urine:* clean-catch midstream samples, or samples drawn from the catheter port after disinfection, were cultured semi-quantitatively with a calibrated loop (0.001 mL) on CLED and MacConkey agar. A count of ≥10^5 CFU/mL was considered significant for voided samples[25] and ≥10^3 CFU/mL for catheter-collected samples from symptomatic patients[26]. *Blood:* two sets of 8–10 mL from separate sites were inoculated into automated blood culture bottles (BacT/ALERT 3D) and incubated for seven days. In suspected catheter-related bloodstream infection, paired catheter and peripheral cultures were taken and a differential time to positivity of at least two hours was considered significant[27]. *Catheter tip and exit-site samples:* tips were cultured by the semi-quantitative roll-plate method, with ≥15 CFU considered significant[28]. *Respiratory samples:* sputum was accepted if it showed more than 25 neutrophils and fewer than 10 squamous epithelial cells per low-power field; endotracheal aspirates were processed quantitatively (≥10^5 CFU/mL). *Pus/wound swabs* and *peritoneal dialysate or other sterile fluids* (inoculated into blood culture bottles and as centrifuged deposit) were processed by standard methods.

Identification of isolates

All samples were Gram-stained and inoculated on 5% sheep blood agar and MacConkey agar (chocolate agar in 5% CO_2 for respiratory samples) and incubated at 35 ± 2 °C for 18–24 hours. Isolates were identified by colony morphology, Gram reaction and standard biochemical tests, including catalase, coagulase, bile-esculin,

oxidase, triple sugar iron, indole, citrate, urease and sugar fermentation tests[29]. Identification of doubtful isolates was confirmed using the VITEK 2 Compact system (bioMérieux).

Antimicrobial susceptibility testing

Susceptibility was tested by the Kirby–Bauer disk diffusion method[30] on Mueller–Hinton agar with a 0.5 McFarland inoculum and interpreted according to CLSI M100, 33rd edition[31]. Gram-negative isolates were tested against ampicillin, amoxicillin–clavulanate, piperacillin–tazobactam, ceftriaxone, ceftazidime, cefepime, cefoperazone–sulbactam, gentamicin, amikacin, ciprofloxacin, levofloxacin, co-trimoxazole, imipenem, meropenem and, for urinary isolates, nitrofurantoin. Gram-positive isolates were tested against penicillin/ampicillin, cefoxitin, erythromycin, clindamycin, gentamicin (high-level, 120 µg, for enterococci), ciprofloxacin, levofloxacin, co-trimoxazole, doxycycline, nitrofurantoin (urinary isolates), linezolid, vancomycin and teicoplanin. Colistin susceptibility was determined by broth microdilution and vancomycin MIC for staphylococci and enterococci by agar dilution. *E. coli* ATCC 25922, *P. aeruginosa* ATCC 27853, *K. pneumoniae* ATCC 700603, *S. aureus* ATCC 25923 and *Enterococcus faecalis* ATCC 29212 were used as quality control strains.

Detection of resistance phenotypes and definitions

ESBL production was confirmed by the combined disk method (ceftazidime and cefotaxime, with and without clavulanic acid; an increase in zone diameter of ≥5 mm). Carbapenem-resistant Enterobacterales (CRE) were defined by meropenem non-susceptibility and screened for carbapenemase production by the modified carbapenem inactivation method[32]. Methicillin resistance in *S. aureus* and coagulase-negative staphylococci (CoNS) was detected by cefoxitin (30 µg) disks, and inducible clindamycin resistance by the D-test. Isolates were classified as MDR (non-susceptible to at least one agent in three or more antimicrobial categories), XDR (susceptible to only one or two categories) or PDR (non-susceptible to all agents)[33].

Statistical analysis

Data were entered in Microsoft Excel and analysed with SPSS version 25 (IBM Corp., Armonk, NY). Categorical variables are expressed as numbers and percentages, and continuous variables as mean ± standard deviation. The chi-square test or Fisher's exact test was used to compare proportions and the independent t-test to compare means. A chi-square test for trend was used for ordered categories. Variables with $p < 0.10$ in univariate analysis were entered into a multivariate logistic regression model to identify independent predictors of culture-proven infection, and results are given as adjusted odds ratios (aOR) with 95% confidence intervals (CI). A p value < 0.05 was considered statistically significant.

Ethical considerations

The study was approved by the Institutional Ethics Committee. Written informed consent was obtained from all participants or their legal guardians, and confidentiality of patient data was maintained throughout.

RESULTS

Characteristics of the study population

A total of 300 CKD patients with suspected bacterial infection were enrolled. Their mean age was 52.4 ± 14.6 years (range 19–81 years), with the largest proportion in the 46–60-year group (39.3%). There were 186 males (62.0%) and 114 females (38.0%), a male-to-female ratio of 1.6:1. Diabetic nephropathy (42.0%) and hypertensive nephrosclerosis (26.0%) were the commonest causes of CKD (Table 1). One hundred patients (33.3%) were on maintenance haemodialysis, of whom 62 dialysed through a central venous catheter (41 non-tunnelled, 21 tunnelled) and 38 through an arteriovenous fistula.

Characteristic	Number	Percentage (%)
Age group (years)		
18–30	21	7.0
31–45	62	20.7
46–60	118	39.3
61–75	83	27.7
>75	16	5.3
Sex		
Male	186	62.0
Female	114	38.0
Aetiology of CKD		
Diabetic nephropathy	126	42.0
Hypertensive nephrosclerosis	78	26.0
Chronic glomerulonephritis	48	16.0
Chronic interstitial nephritis / obstructive uropathy	30	10.0
Autosomal dominant polycystic kidney disease	6	2.0
Unknown / others	12	4.0
Total	**300**	**100.0**

Table 1: Demographic and clinical characteristics of the study population (n = 300)

Culture positivity and CKD stage

Of the 300 patients, 214 (71.3%) had a positive culture and 86 (28.7%) showed no growth. Monomicrobial growth was seen in 202 patients (94.4%) and polymicrobial growth in 12 (5.6%), giving a total of 226 bacterial isolates. Infection was hospital-acquired in 126 culture-positive patients (58.9%) and community-onset in 88 (41.1%). Culture positivity increased significantly with advancing CKD stage, from 58.3% in stage 3 to 80.0% in patients on haemodialysis (χ^2 = 10.12, df = 3, p = 0.018) (Table 2).

CKD stage (eGFR, mL/min/1.73 m²)	Patients (n)	Culture positive n (%)	Culture negative n (%)
Stage 3 (30–59)	48	28 (58.3)	20 (41.7)
Stage 4 (15–29)	72	46 (63.9)	26 (36.1)
Stage 5, non-dialysis (<15)	80	60 (75.0)	20 (25.0)
Stage 5D (haemodialysis)	100	80 (80.0)	20 (20.0)
Total	**300**	**214 (71.3)**	**86 (28.7)**

Table 2: Culture positivity according to CKD stage

χ^2 = 10.12, df = 3, p = 0.018.

Distribution of clinical samples

Urine was the commonest sample (46.0%), followed by blood (20.7%) and respiratory samples (12.7%). Culture positivity was highest in pus/wound swabs (87.5%) and sputum or endotracheal aspirates (78.9%) and lowest in blood (61.3%) and sterile body fluids (58.3%) (Table 3).

Sample	Number (%)	Culture positive n (%)	No growth (n)
Urine	138 (46.0)	98 (71.0)	40
Blood	62 (20.7)	38 (61.3)	24
Sputum / endotracheal aspirate	38 (12.7)	30 (78.9)	8
Pus / wound swab	32 (10.7)	28 (87.5)	4
Catheter tip / exit-site swab	18 (6.0)	13 (72.2)	5
Peritoneal dialysate / other sterile fluid	12 (4.0)	7 (58.3)	5
Total	**300 (100)**	**214 (71.3)**	**86**

Table 3: Distribution of clinical samples and culture positivity (n = 300)

Bacterial isolates

Gram-negative bacilli accounted for 158 isolates (69.9%) and Gram-positive cocci for 68 (30.1%). *Escherichia coli* was the commonest isolate (27.4%), followed by *Klebsiella pneumoniae* (15.9%), *Staphylococcus aureus* (12.4%), *Pseudomonas aeruginosa* (10.6%) and *Acinetobacter baumannii* (8.0%). Enterococci comprised 11.5% of all isolates (Table 4). *E. coli* alone made up 42.2% of the 102 urinary isolates (Table 5), whereas *S. aureus* and CoNS were important in blood (34.2% of 38 blood isolates), pus and catheter-related samples. *Acinetobacter baumannii* and *P. aeruginosa* predominated in respiratory samples (60.6% of 33 isolates).

Organism	Number	Percentage of isolates (%)
Gram-negative bacilli (n = 158)		
Escherichia coli	62	27.4
Klebsiella pneumoniae	36	15.9
Pseudomonas aeruginosa	24	10.6
Acinetobacter baumannii	18	8.0
Proteus mirabilis	8	3.5
Enterobacter species	6	2.7
Citrobacter species	4	1.8
Gram-positive cocci (n = 68)		
Staphylococcus aureus	28	12.4
Enterococcus faecalis	20	8.8
Coagulase-negative staphylococci	10	4.4
Enterococcus faecium	6	2.7
Streptococcus species	4	1.8
Total	**226**	**100.0**

Table 4: Distribution of bacterial isolates (n = 226)

Organism	Urine	Blood	Sputum / ETA	Pus / swab	Catheter	Fluid	Total
E. coli	43	6	0	9	2	2	62
K. pneumoniae	20	6	6	3	1	0	36
P. aeruginosa	8	2	10	2	1	1	24
A. baumannii	3	4	10	0	1	0	18
P. mirabilis	5	0	0	3	0	0	8
Enterobacter spp.	3	1	1	0	1	0	6
Citrobacter spp.	3	0	0	1	0	0	4
S. aureus	2	8	3	10	4	1	28
E. faecalis	11	4	0	2	1	2	20
E. faecium	3	2	0	0	0	1	6
CoNS	1	5	0	1	3	0	10
Streptococcus spp.	0	0	3	1	0	0	4
Total	**102**	**38**	**33**	**32**	**14**	**7**	**226**

Table 5: Distribution of the isolates according to sample type (number of isolates)

ETA = endotracheal aspirate; Catheter = catheter tip / exit-site swab; Fluid = peritoneal dialysate / other sterile fluid; CoNS = coagulase-negative staphylococci.

Antibiotic susceptibility of Gram-negative isolates

Susceptibility of the four commonest Gram-negative pathogens is shown in Table 6. Susceptibility to third-generation cephalosporins and fluoroquinolones was poor in Enterobacterales (ceftriaxone 22.6% and 16.7%; ciprofloxacin 17.7% and 27.8% for *E. coli* and *K. pneumoniae*, respectively). Amikacin (74.2% and 63.9%), cefoperazone–sulbactam (62.9% and 47.2%) and the carbapenems (83.9–85.5% for *E. coli*; 72.2–75.0% for *K. pneumoniae*) were the more reliable options. Nitrofurantoin was active against 79.1% of urinary *E. coli* but only 45.0% of urinary *K. pneumoniae*. *A. baumannii* showed the poorest susceptibility profile, with fewer than 40% of isolates susceptible to carbapenems, piperacillin–tazobactam and cefoperazone–sulbactam. Colistin remained active against 94.4–100% of isolates.

Antibiotic	*E. coli* (n = 62)	*K. pneumoniae* (n = 36)	*P. aeruginosa* (n = 24)	*A. baumannii* (n = 18)
Ampicillin	6.5	–	–	–
Amoxicillin–clavulanate	24.2	19.4	–	–
Piperacillin–tazobactam	51.6	38.9	58.3	16.7
Ceftriaxone	22.6	16.7	–	–
Ceftazidime	25.8	19.4	50.0	11.1
Cefepime	32.3	25.0	54.2	16.7
Cefoperazone–sulbactam	62.9	47.2	58.3	38.9
Gentamicin	41.9	36.1	41.7	22.2
Amikacin	74.2	63.9	70.8	44.4
Ciprofloxacin	17.7	27.8	41.7	16.7
Levofloxacin	21.0	30.6	45.8	22.2
Co-trimoxazole	27.4	30.6	–	27.8
Nitrofurantoin†	79.1	45.0	–	–
Imipenem	83.9	72.2	70.8	38.9
Meropenem	85.5	75.0	75.0	33.3
Colistin‡	100.0	94.4	95.8	94.4
Tigecycline	96.8	83.3	–	–

Table 6: Antibiotic susceptibility pattern of Gram-negative isolates (% susceptible)

– = not tested or not applicable (intrinsic resistance / no CLSI breakpoint). †Urinary isolates only (E. coli n = 43; K. pneumoniae n = 20). ‡Broth microdilution.

Antibiotic susceptibility of Gram-positive isolates

Among *S. aureus* isolates, 46.4% were methicillin-resistant (MRSA), and 60.0% of CoNS were methicillin-resistant. All staphylococci were susceptible to linezolid, and all *S. aureus* isolates to vancomycin and teicoplanin. Resistance to erythromycin (57.1%), ciprofloxacin (60.7%) and levofloxacin (57.1%) was high. Among enterococci, ampicillin susceptibility was 75.0% in *E. faecalis* but only 16.7% in *E. faecium*; high-level gentamicin resistance was found in 45.0% and 50.0%, respectively. Three enterococcal isolates (1 *E. faecalis* and 2 *E. faecium*) were vancomycin-resistant (VRE, 11.5% of 26 enterococci), and all enterococci were linezolid-susceptible (Table 7). All four *Streptococcus* isolates were susceptible to penicillin, ceftriaxone and vancomycin.

Antibiotic	*S. aureus* (n = 28)	*E. faecalis* (n = 20)	*E. faecium* (n = 6)	CoNS (n = 10)
Penicillin / ampicillin§	10.7	75.0	16.7	10.0
Cefoxitin (methicillin)	53.6	–	–	40.0
Erythromycin	42.9	–	–	30.0
Clindamycin	57.1	–	–	50.0
Gentamicin (high-level for enterococci)	60.7	55.0	50.0	50.0
Ciprofloxacin	39.3	35.0	16.7	30.0
Levofloxacin	42.9	40.0	16.7	40.0
Co-trimoxazole	64.3	–	–	50.0
Doxycycline / tetracycline	71.4	30.0	33.3	60.0
Nitrofurantoin¶	–	90.9	33.3	–
Linezolid	100.0	100.0	100.0	100.0
Vancomycin	100.0	95.0	66.7	100.0
Teicoplanin	100.0	95.0	66.7	90.0

Table 7: Antibiotic susceptibility pattern of Gram-positive isolates (% susceptible)

§Penicillin for staphylococci; ampicillin for enterococci. ¶Urinary isolates only (E. faecalis n = 11; E. faecium n = 3). – = not tested / not applicable.

Resistance phenotypes and multidrug resistance

ESBL production was detected in 61.3% of *E. coli*, 66.7% of *K. pneumoniae* and 3 of 8 *P. mirabilis* isolates. Carbapenem resistance was found in 20 of 116 Enterobacterales (17.2%), and the modified carbapenem inactivation method confirmed carbapenemase production in 18 of these 20 isolates. Carbapenem resistance was far more frequent in *A. baumannii* (66.7%) than in *P. aeruginosa* (25.0%). Colistin resistance was uncommon (4 of 140 isolates of the four main Gram-negative species, 2.9%) (Table 8).

Phenotype	Organism(s)	n / N	%
ESBL producers	*E. coli*	38 / 62	61.3
	K. pneumoniae	24 / 36	66.7
	P. mirabilis	3 / 8	37.5
	All (three species)	65 / 106	61.3
Carbapenem-resistant Enterobacterales	*E. coli*	9 / 62	14.5
	K. pneumoniae	9 / 36	25.0
	Enterobacter spp.	2 / 6	33.3
	All Enterobacterales	20 / 116	17.2
Carbapenem-resistant non-fermenters	*P. aeruginosa*	6 / 24	25.0
	A. baumannii	12 / 18	66.7
Colistin-resistant Gram-negatives	*K. pneumoniae*, *P. aeruginosa*, *A. baumannii*	4 / 140	2.9
Methicillin resistance	*S. aureus* (MRSA)	13 / 28	46.4
	CoNS	6 / 10	60.0
Vancomycin-resistant enterococci	*E. faecalis* + *E. faecium*	3 / 26	11.5
High-level gentamicin resistance	*E. faecalis* + *E. faecium*	12 / 26	46.2

Table 8: Important resistance phenotypes among the isolates

ESBL = extended-spectrum β-lactamase; CoNS = coagulase-negative staphylococci; MRSA = methicillin-resistant S. aureus. The denominator for colistin includes E. coli (n = 62, all susceptible).

Overall, 164 of 226 isolates (72.6%) were MDR (79.7% of Gram-negative and 55.9% of Gram-positive isolates). Twenty-one isolates (9.3%) were XDR and two (0.9%) PDR (*A. baumannii* and *K. pneumoniae*, one each); nine of the XDR isolates were *A. baumannii* (Table 9). At patient level, 150 of 214 culture-positive patients (70.1%) harboured at least one MDR isolate, and the proportion rose progressively with CKD stage: 50.0% in stage 3, 63.0% in stage 4, 73.3% in stage 5 (non-dialysis) and 78.8% in stage 5D (chi-square for trend, p = 0.004).

Organism	Isolates (n)	MDR *n (%)*	XDR (n)	PDR (n)
E. coli	62	48 (77.4)	2	0
K. pneumoniae	36	31 (86.1)	6	1
P. aeruginosa	24	17 (70.8)	4	0
A. baumannii	18	17 (94.4)	9	1
P. mirabilis	8	5 (62.5)	0	0
Enterobacter spp.	6	5 (83.3)	0	0
Citrobacter spp.	4	3 (75.0)	0	0
S. aureus	28	15 (53.6)	0	0
E. faecalis	20	9 (45.0)	0	0
E. faecium	6	5 (83.3)	0	0
CoNS	10	7 (70.0)	0	0
Streptococcus spp.	4	2 (50.0)	0	0
Total	**226**	**164 (72.6)**	**21 (9.3%)**	**2 (0.9%)**

Table 9: Multidrug-resistant (MDR), extensively drug-resistant (XDR) and pandrug-resistant (PDR) isolates

MDR includes XDR and PDR isolates.

Factors associated with culture-proven infection

On univariate analysis, age above 60 years, diabetes mellitus, dialysis dependence, central venous catheter use for haemodialysis, indwelling urinary catheter, hospitalisation and antibiotic use in the preceding three months, and serum albumin below 3.0 g/dL were significantly associated with culture-positive infection, whereas sex, hypertension and immunosuppressive therapy were not (Table 10). On multivariate logistic regression, prior antibiotic use, presence of an indwelling catheter (urinary or central venous), age above 60 years, hypoalbuminaemia and diabetes mellitus remained independent predictors (Table 11; Hosmer–Lemeshow p = 0.62).

Risk factor	Culture positive (n = 214) n (%)	Culture negative (n = 86) n (%)	Odds ratio (95% CI)	p value
Age >60 years	82 (38.3)	17 (19.8)	2.52 (1.39–4.59)	0.002
Female sex	80 (37.4)	34 (39.5)	0.91 (0.55–1.53)	0.73
Diabetes mellitus	104 (48.6)	28 (32.6)	1.96 (1.16–3.31)	0.012
Hypertension	186 (86.9)	72 (83.7)	1.29 (0.64–2.59)	0.47
Dialysis dependence (stage 5D)	80 (37.4)	20 (23.3)	1.97 (1.11–3.49)	0.020
Central venous catheter for haemodialysis	52 (24.3)	10 (11.6)	2.44 (1.18–5.06)	0.017
Indwelling urinary catheter	62 (29.0)	12 (14.0)	2.52 (1.28–4.95)	0.008
Hospitalisation in previous 3 months	112 (52.3)	30 (34.9)	2.05 (1.22–3.44)	0.007
Antibiotic use in previous 3 months	136 (63.6)	32 (37.2)	2.94 (1.75–4.94)	<0.001
Serum albumin <3.0 g/dL	100 (46.7)	26 (30.2)	2.02 (1.19–3.45)	0.010
Immunosuppressive therapy	24 (11.2)	7 (8.1)	1.43 (0.59–3.44)	0.43

Table 10: Univariate analysis of risk factors for culture-positive infection

Variable	Adjusted OR (95% CI)	p value
Antibiotic use in previous 3 months	2.61 (1.52–4.48)	<0.001
Indwelling catheter (urinary or central venous)	2.38 (1.34–4.22)	0.003
Age >60 years	2.19 (1.22–3.94)	0.009
Serum albumin <3.0 g/dL	1.91 (1.11–3.29)	0.020
Diabetes mellitus	1.78 (1.04–3.05)	0.036
Dialysis dependence (stage 5D)	1.49 (0.80–2.77)	0.21
Hospitalisation in previous 3 months	1.62 (0.95–2.76)	0.076

Table 11: Multivariate logistic regression: independent predictors of culture-proven infection

Clinical outcome

The mean hospital stay was significantly longer in culture-positive patients (14.6 ± 6.8 days) than in culture-negative patients (9.2 ± 4.5 days; p < 0.001). In-hospital mortality was 14.5% (31 of 214) among culture-positive patients and 5.8% (5 of 86) among culture-negative patients (p = 0.037). Among culture-positive patients, mortality was 17.3% (26 of 150) in those with an MDR isolate and 7.8% (5 of 64) in those without, a difference that did not reach statistical significance (p = 0.07).

DISCUSSION

This study describes the bacteriological profile and resistance pattern in 300 CKD patients with suspected bacterial infection at a tertiary care hospital. Three main observations emerged. First, culture positivity increased progressively with advancing CKD stage and was highest in patients on maintenance haemodialysis. Second, Gram-negative bacilli, led by *E. coli* and *K. pneumoniae*, accounted for nearly 70% of isolates and showed high rates of ESBL production and carbapenem resistance. Third, almost three-quarters of the isolates were MDR, and prior antibiotic exposure, indwelling catheters, age above 60 years, hypoalbuminaemia and diabetes were independent predictors of culture-proven infection.

Culture positivity was 71.3% overall and rose from 58.3% in stage 3 to 80.0% in stage 5D (p = 0.018). This gradient is biologically plausible. As uraemia progresses, defects in neutrophil, monocyte and lymphocyte function and chronic inflammation increasingly blunt host defence and registry-based studies have shown that the risk of infection-related hospitalisation rises as eGFR falls. In dialysis-dependent patients, vascular access adds a direct portal of entry; catheter-related bloodstream infection remains the most important preventable complication of haemodialysis[34,35]. In our cohort, 62% of haemodialysis patients dialysed through a central venous catheter, and catheter use was significantly associated with culture-positive infection (OR 2.44).

Urine was the commonest sample (46.0%) and *E. coli* the commonest urinary isolate (42.2% of urinary isolates), in keeping with the established role of uropathogenic *E. coli* in UTI. In CKD, urinary stasis, obstruction, diabetic bladder dysfunction, catheterisation and reduced urinary antibacterial activity facilitate infection and favour more resistant and less typical pathogens. A predominance of *E. coli* followed by *Klebsiella* has been consistently reported by Indian studies of uropathogens from community and hospital settings[36–38]. *Enterococcus faecalis* contributed 10.8% of our urinary isolates; enterococci are well known to be prominent in catheterised patients in India[39].

Gram-negative bacilli constituted 69.9% of all isolates, with *E. coli*, *K. pneumoniae*, *P. aeruginosa* and *A. baumannii* leading the list. This distribution matches national surveillance data, in which Enterobacterales and non-fermenters dominate Gram-negative isolates from Indian hospitals[40]. Gram-positive cocci, mainly *S. aureus* and CoNS, were concentrated in blood, pus and catheter-related samples. Together they represented 34.2% of blood isolates, which reflects skin flora entering through vascular access. Nasal and skin carriage of *S. aureus* is an important source of infection in dialysis patients[41], and meta-analysis shows that MRSA colonisation increases the risk of

subsequent infection in this group[42]. *S. aureus* bacteraemia is a serious infection with substantial mortality, and prompt source control is vital[43].

ESBL production was found in 61.3% of *E. coli* and 66.7% of *K. pneumoniae*. These rates explain the poor susceptibility to third-generation cephalosporins (22.6% and 16.7%). ESBLs have become widespread among Enterobacterales, with CTX-M enzymes predominating[44], and a multicentre Indian study of *E. coli* and *K. pneumoniae* from tertiary hospitals confirmed the dominance of the CTX-M-15 type[45]. ESBL and AmpC β-lactamases have also been demonstrated in a substantial proportion of Indian isolates from complicated UTI[46]. Because CKD patients typically have complicated infections, this is directly relevant to empirical choices.

Susceptibility to fluoroquinolones was below 25% in *E. coli* (ciprofloxacin 17.7%, levofloxacin 21.0%), and co-trimoxazole was active against only 27.4%. Rising fluoroquinolone resistance in *E. coli* has been documented in Indian centres and our data suggest that fluoroquinolones and co-trimoxazole can no longer be relied on empirically in this population. Activity of β-lactam/β-lactamase inhibitor combinations was intermediate (piperacillin–tazobactam 51.6%, cefoperazone–sulbactam 62.9% in *E. coli*). Indian in-vitro comparisons have shown wide variation in the activity of these combinations against Gram-negative bacilli[47], which reinforces that local data, rather than the drug class, should guide selection.

Amikacin remained active against 74.2% of *E. coli* and 63.9% of *K. pneumoniae*, but its nephrotoxicity and ototoxicity limit its use in CKD to selected situations with dose adjustment and drug-level monitoring. Nitrofurantoin retained 79.1% activity against urinary *E. coli* but only 45.0% against *Klebsiella*. Its use in patients with reduced eGFR is debated: the traditional contraindication below a creatinine clearance of 60 mL/min is not firmly evidence-based, but efficacy in the urinary tract falls as renal function declines[48]. Since most of our patients were in stage 4 or 5, we would not recommend nitrofurantoin for them.

Carbapenem resistance was seen in 17.2% of Enterobacterales (25.0% in *K. pneumoniae*), and carbapenemase production was confirmed in 90% of the resistant isolates by phenotypic testing. The global spread of carbapenemase-producing Enterobacterales is a major concern[49]. Emerging carbapenem resistance in Indian tertiary hospitals was documented early[50], and the NDM-1 enzyme, first described from India, has since spread worldwide[51]. Indian bloodstream isolates of carbapenem-resistant *K. pneumoniae* are likewise reported to carry NDM and OXA-48-type carbapenemases[52]. For such infections, colistin and tigecycline are the options left. Colistin was active against 94.4–100% of our Gram-negative isolates, but its nephrotoxicity is a particular worry in CKD[53], and careful renal dose adjustment is mandatory.

P. aeruginosa showed only 50.0–58.3% susceptibility to antipseudomonal β-lactams and 70.8–75.0% to carbapenems. The organism combines intrinsic resistance with chromosomal AmpC, efflux pumps and porin loss, which together allow rapid emergence of resistance during therapy[54]. Resistance rates in Indian hospitals have been substantial[55]. *A. baumannii* was the most resistant species in our study: 94.4% were MDR, 66.7% were carbapenem-resistant, and nine of the 21 XDR isolates and one of the two PDR isolates belonged to this species. Most were from respiratory samples, consistent with hospital-acquired and ventilator-associated infection. *A. baumannii* is a successful nosocomial pathogen able to survive on surfaces and to accumulate resistance mechanisms[56], and Indian reviews have called for strict infection control to contain it[57]. Device-associated infection rates in Indian intensive care units remain high[58], and this probably contributes to the burden we observed.

Methicillin resistance was present in 46.4% of *S. aureus* and 60.0% of CoNS, which is within the range reported across Indian centres[59]. All staphylococci were susceptible to linezolid and all *S. aureus* to vancomycin, so these remain the drugs of choice for serious MRSA infection, with vancomycin dosing timed to dialysis sessions. Among enterococci, ampicillin resistance was notable in *E. faecium* (83.3%), and high-level gentamicin resistance (46.2%) removes the possibility of aminoglycoside synergy. VRE accounted for 11.5% of enterococci; enterococci are intrinsically resistant to many drugs and readily acquire further resistance

determinants[60]. Linezolid remained fully active against all enterococci in this series.

Overall, 72.6% of isolates were MDR, 9.3% XDR and 0.9% PDR, and the proportion of patients carrying an MDR isolate climbed from 50.0% in stage 3 to 78.8% in stage 5D (trend p = 0.004). Resistance of this magnitude is consistent with the global and Indian estimates of AMR burden. Repeated healthcare contact, antibiotic exposure, indwelling devices and prolonged hospital stay in advanced CKD probably select for resistant organisms, and the widespread, often inappropriate use of antibiotics in India aggravates the problem. In our cohort 58.9% of infections were hospital-acquired, pointing to a significant role for nosocomial transmission.

Prior antibiotic use (aOR 2.61), indwelling catheters (aOR 2.38), age above 60 years (aOR 2.19), hypoalbuminaemia (aOR 1.91) and diabetes mellitus (aOR 1.78) were independent predictors of culture-proven infection. Diabetes is known to increase the risk of many types of infection[61], and diabetic nephropathy was the commonest cause of CKD in our cohort (42.0%), as in the Indian CKD registry. Culture-positive infection was associated with a longer hospital stay and higher in-hospital mortality (14.5% vs 5.8%). The numerically higher mortality with MDR isolates (17.3% vs 7.8%) did not reach significance, possibly because of the limited number of events.

Our findings suggest that empirical regimens for CKD patients with serious infection should be selected from a CKD-specific, locally generated antibiogram rather than from general hospital data. Fluoroquinolones, third-generation cephalosporins and co-trimoxazole are unreliable empirical agents for Gram-negative infection in this setting, and where sepsis is severe or risk factors for ESBL or CRE are present, a carbapenem or a guided combination may be required, followed by de-escalation as soon as culture results are available. Every antimicrobial should be dosed for the patient's renal function and, where applicable, for dialysis . The ICMR treatment guidelines provide a framework for syndromic therapy[62], while hospital antimicrobial stewardship programmes[63] and the National Action Plan on AMR[64] provide the institutional and policy basis for curbing resistance. Preventive measures matter equally: preference for arteriovenous fistulae over catheters, strict aseptic catheter care, early removal of urinary catheters, hand hygiene and decolonisation strategies in selected patients.

Strengths and limitations

The strengths of the study are its prospective design, the inclusion of all CKD stages with a dialysis subgroup, uniform laboratory methods with quality control, and the use of standard definitions for MDR, XDR and PDR. Its limitations are the single-centre design, the phenotypic characterisation of resistance mechanisms, exclusion of fungal, anaerobic and mycobacterial infections, the lack of data on antibiotic dosing and clinical response, and the use of in-hospital mortality as the only outcome. Multicentre studies with molecular typing and longer follow-up are needed to confirm and extend these findings.

CONCLUSION

Bacterial infections in patients with CKD at our centre were caused predominantly by Gram-negative bacilli, chiefly *E. coli* and *K. pneumoniae*, with *S. aureus* and CoNS prominent in bloodstream and catheter-related infections. Culture positivity and the burden of multidrug resistance both increased with advancing CKD stage, and almost three-quarters of the isolates were MDR, with a considerable proportion of ESBL, carbapenem-resistant and MRSA isolates. Colistin, linezolid, vancomycin and, to a lesser degree, carbapenems and amikacin retained the best activity, but their use in CKD is limited by toxicity and the need for renal dose adjustment. Prior antibiotic exposure, indwelling catheters, advanced age, hypoalbuminaemia and diabetes were independent predictors of infection, and infection was associated with longer hospitalisation and higher mortality. Regular CKD-specific antibiograms, rational and renally adjusted antimicrobial therapy, strict infection control and catheter care, and robust antimicrobial stewardship are essential to improve outcomes in this vulnerable population.

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