



Microbiological Evaluation of Urinary samples in A Cohort of Patients with Chronic Renal Failure

**Gitali Bhagawati^{1*}, Yasir Rizvi², Lakshmi Kant Jha³, Bhavana Chauhan⁴,
Domathoti Yashwanth⁵ and Rekha Saji Kumar⁶**

¹MBBS, MD (Microbiology), PGDHM, CIC, Senior Consultant and Clinical Lead, Department of Microbiology, and Infection Control, Narayana Health, Dharamshila Narayana Superspeciality Hospital, Delhi, India

²MBBS, MD (Internal Medicine), DM (Nephrology), Senior Consultant and Director Nephrology, Narayana Health, Dharamshila Narayana Superspeciality Hospital, Delhi, India

³MBBS, MD - General Medicine, DM - Nephrology, Senior Consultant and Director, Nephrology, Narayana Health, Dharamshila Narayana Superspeciality Hospital, Delhi, India

⁴MBBS, MD (Microbiology), Senior Resident, Department of Microbiology, Narayana Health, Dharamshila Narayana Superspeciality Hospital, Delhi, India

⁵Infection Control Nurse, Narayana Health, Dharamshila Narayana Superspeciality Hospital, Delhi, India

⁶Technical Supervisor, Department of Microbiology, Narayana Health, Dharamshila Narayana Superspeciality Hospital, Delhi, India

***Corresponding Author:** Gitali Bhagawati, MBBS, MD (Microbiology), PGDHM, CIC, Senior Consultant and Clinical Lead, Department of Microbiology, and Infection Control, Narayana Health, Dharamshila Narayana Superspeciality Hospital, Delhi, India.

DOI: 10.31080/ASMI.2025.08.1485

Received: December 13, 2024

Published: January 28, 2025

© All rights are reserved by
Gitali Bhagawati, et al.

Abstract

Introduction: Urinary tract infections (UTIs) is one of the most common healthcare-associated infections (HAI) in patients with chronic renal failure (CKD).

Objective: The objective of the study was to evaluate the incidence of UTI and possible risk factors among patients with CKD. Uropathogens and their antimicrobial susceptibility pattern (AST) were also evaluated in this group of patients.

Methods: This retrospective study was carried out over a period of 22 months in a tertiary care hospital in Delhi. A total of 310 urine samples were received in the department of microbiology from patients of nephrology unit with CKD. Urine samples were processed on Cysteine Lactose Electrolyte Deficient (CLED) agar and incubated aerobically at 37°C for 24-48 hrs. Complete work-up was done in case of significant growth (SG) with colony count $\geq 10^4$ CFU/ml.

Results: Indwelling catheter, previous history of UTI and pyuria (≥ 10 / high power field) were found to have strong association with SG (p value < 0.05). On the contrary, hyperglycaemia, dialysis, and grade of CKD were not found to have statistically significant correlation with SG (p value > 0.05). The most common urinary isolate was *E. coli*, 36 (28.57%) followed by *Enterococcus spp.* 22 (17.46%). Among enterobacterales, meropenem resistance was 49.12% (31/57) while colistin resistance was found to be 5.71% (2/35).

Conclusion: CKD is associated with a proinflammatory milieu with impaired innate and acquired immunity leading to infections. Minimal invasive procedures along with judicious use of antibiotics based on local antibiogram can provide better outcome among these patients.

Keywords: Chronic Kidney Disease (CKD); Estimated Glomerular Filtration Rate (eGFR); Urinary Tract Infections (UTIs); Antimicrobial Susceptibility (AST); Estimated Glomerular Filtration Rate (eGFR); *Escherichia coli*

Introduction

Chronic kidney disease (CKD) has become a serious public health concern leading to an important contributor of morbidity and mortality; infectious complications are approximately three times higher in this group of patients as compared to the non-CKD population [1]. Impairment of the normal reaction of the innate and adaptive immune systems (IAIS) in CKD predisposes patients to an increased risk of infections, along with poor vaccine response [2]. CKD itself is a risk factor for the development of healthcare-associated infections (HAI); urinary tract infections (UTIs) remain one of the most common HAIs in patients with CKD [3]. There are many patient-related risk factors associated with CKD for developing UTI which include advanced age, impaired immune defence, diabetic nephropathy, nephrotic syndrome etc. [4]. Previous history of UTI and an increasing number of invasive urologic procedures for both diagnosis and treatment have also been related to the high rates of UTIs among these patients [5]. Alteration of intestinal microbiota and urinary microbiome also plays important role in causing UTI among this cohort of patients [4,6]. Metabolic acidosis, volume overload with intestinal wall congestion, retention of uremic toxins, intestinal ischaemia etc. induce dysbiosis inside the gut lumen leading to overgrowth of pathobiont that induces inflammation and loss of barrier function that in turn promotes increased translocation of bacteria and bacterial components into the host's internal environment [6]. Healthy individual's urinary tract (UT) is hosting 'urinary microbiome', which is speculated that it might have implications for maintaining urinary health. This suggests that UTI results from an 'imbalance' in the UT microbiome [4].

UTI comprises of mild cystitis to life-threatening sepsis and multiple organ failure (MOF); the term complicated urinary tract infection (cUTI) is widely used for an infection that occurs in a patient with a structural or functional abnormality of the genitourinary tract. Therefore, all UTIs in patients with CKD are considered cUTI [1]. Apart from significant colony count in culture (10⁵ CFU/mL), asymptomatic bacteriuria and pyuria are also significant in these patients because they can reveal an increased risk for the progression of CKD [7].

The study was undertaken to evaluate the incidence of UTI and possible risk factors among patients in a cohort of patients attend-

ing nephrology department with the evidence of CKD. Uropathogens and their antimicrobial susceptibility pattern (AST) were also evaluated in this group of patients.

Material and Methods

Study design

This descriptive, retrospective study was carried out over a period of 22 months, January 2022 to October 2023 in a 200 bedded tertiary care hospital in Delhi. Patients' data were collected from Laboratory Information System (LIS), requisition forms and files from the department of Medical Records Department (MRD). Data on identification and AST pattern of organisms were retrieved from Vitek 2 compact system and WHONET 5.6 software. Approval for the research was taken with no. DNSH/HR/ PER/D110103.

Inclusion criteria

A total of 310 urine samples were received in the department of Microbiology from both indoor and outdoor patients of nephrology unit with CKD. Estimated Glomerular filtration rate (eGFR) is generally accepted as the best index of kidney function. Classification of CKD is based on eGFR; Grade I include kidney damage with no or increased GFR while Grade II includes kidney damage with mild reduction in GFR. GFR <60 ml/min/1.73 m² is considered as moderate reduction in GFR (Grade III) while GFR <15 ml/min/1.73 m² (Grade V) is considered as kidney failure [8,9].

Urine sample collection

In non-catheterized patients, mid-stream urine samples were collected whereas in catheterized patients, samples were collected through the sampling port after clamping the catheter below the port following proper aseptic precautions. Samples were immediately processed for culture following standard guideline. When delay of more than 2 hours is unavoidable, specimens were stored in a refrigerator at 4° C [10].

Urine culture and microscopy

In the Microbiology laboratory, urine samples were processed for culture initially. With standard calibrated loop, 1µl (0.001ml) of urine was inoculated on Cysteine Lactose Electrolyte Deficient (CLED) agar and incubated aerobically at 37°C for 18-24hrs. In case of no growth, plates were re-incubated for another 24 hours [10].

Complete work-up was done in case of significant growth (SG) [Samples with significant bacteriuria (SB) with colony count $\geq 10^5$ CFU/ml and samples with doubtful significance (DS) with colony count $\geq 10^4$ CFU/ml] of a single potential pathogen or for each of two potential pathogens [11].

Pyuria is defined as the presence of 3 or more white cells per high-power field of unspun urine [12]. We defined pyuria as higher than ≥ 10 white blood cells/high-power field (WBC/HPF) in a random centrifuged urine sample [13]. Asymptomatic bacteriuria is defined as more than 10^5 CFU/mL, but with no signs or symptoms of UTI [11].

Rest was considered as either no growth (NG), insignificant growth (IG) when colony count is $< 10,000$ CFU/ml or mixed growth (MG) when growth is \geq three organisms [11].

Identification and antimicrobial susceptibility test

Identification of bacteria was done by Gram stain, colony morphology, motility test and standard biochemical tests. For yeast and yeast like fungus (YYLF) subculture was done on Sabouraud’s Dextrose Agar (SDA) slants and incubated further for 48 to 72 hours [10]. Identification and AST was done by Vitek 2 Compact System 8.01 (bioMérieux, Inc. Durham, North Carolina/USA). Control strains used were *Staphylococcus aureus* ATCC 29213, *Escherichia coli* ATCC 25922, *Candida albicans* ATCC 14053 and *Candida parapsilosis* ATCC 22019.

Factors	All	Culture showing SG	Culture showing NG/IG/MG	p- value
Age (years)	62.15 ± 14.44	66.52 ± 13.02	59.72 ± 14.14	0.4480
Gender				
Male	143 (46.13%)	61 (54.95%)	82 (41.20%)	0.0239
Female	167 (53.87%)	50 (45.04%)	117 (58.79%)	
eGFR (CKD Grade)				
≥ 90 (Grade I)	18 (5.81%)	7 (6.31%)	11 (5.53%)	0.8031
60-89 (Grade II)	14 (4.52%)	5 (4.50%)	9 (4.52%)	1
30-59 (Grade III)	36 (11.61%)	16 (14.41%)	20 (10.05%)	0.2702
15-29 (Grade IV)	70 (22.58%)	22 (19.82%)	48 (24.12%)	0.4
< 15 (Grade V)	172 (55.48%)	61 (54.95%)	111 (55.78%)	0.9056
Indwelling catheter				
Yes	150 (48.38%)	71 (63.97%)	79 (39.69%)	0.0001
No	160 (51.61%)	40 (36.04%)	120 (60.30%)	
Urine sample collection and Catheterisation				
After 48 hours of catheterization	64 (42.66%)	39 (63.93%)	25 (28.09%)	0.0001
Before catheterization or within 48 hours of catheterization	86 (57.33%)	22 (36.06%)	64 (71.91%)	
Pus cells in Urine /HPF				
≥ 10	125 (40.32%)	80 (72.07%)	45 (22.61%)	0.0001
< 10	185 (59.68%)	31 (27.93%)	154 (77.38%)	
Admission status				
In-patient	249 (80.32%)	85 (76.57%)	164 (82.41%)	0.2348
Out-patient	61 (19.68%)	26 (23.42%)	35 (17.58%)	
Previous history of UTI				

Yes	91 (29.35%)	55 (49.55%)	36 (18.09%)	0.0001
No	219 (70.64%)	56 (50.45%)	163 (81.91%)	
Hyperglycaemia				
Yes	85 (27.42%)	35 (31.53%)	50 (25.12%)	0.2346
No	225 (72.58%)	76 (68.47%)	149 (74.87%)	
Dialysis				
Yes	115 (37.09%)	44 (39.64%)	71 (35.68%)	0.5402
No	195 (62.90%)	67 (60.36%)	128 (64.32%)	

Table 1: Demographic profile with risk factors for Significant Bacteriuria in culture among patients with Chronic Renal Failure.

Note: NG: No growth, IG: Insignificant growth, MG: Mixed growth, SG: Significant growth; eGFR: Estimated Glomerular filtration rate; HPF: high power field; IP: UTI: urinary tract infections.

Statistical analysis

The binary variables of the study were compared between the groups using Chi-square test. The outcome was determined to be statistically significant different if the observed P value was <0.05. All analyses were performed using Software Statistical Package for the Social Sciences (SPSS) Version 27 (Armonk, USA) [14].

Results

A total 310 urine samples received from patients with CKD ranging from CKD grade I to V from nephrology cohort are included in the study. Average age of patients in this category was 62.15±14.44 years. SG was found in age group 66.52 ± 13.02 years as compared to 59.72 ± 14.14. UTI with SG in urine was found more in males, 61 (54.95%) than females, 50 (45.04%) with statistically significant *p* value, 0.0239. Category of CKD with respect to eGFR was not found to be statistically significant with the SG in urine culture, *p* value ranging from 0.27 to 1 (>0.05). Indwelling catheter had strong association with SG in urine (*p* value 0.0001) however, outpatient or in-patient status of patients had no such association with the SG in urine, *p* value 0.09. Duration of urine sample collection after catheterisation has a strong association with urinary growth (*p* value 0.0001). Out of 64 samples that were collected after 48 hours of catheterisation, 39 (81.25%) showed SG; on the other hand, out of 86 urine samples collected prior to catheterisation, only 22 (25.58%) samples showed SG. Similarly, evidence of previous history of UTI and pus cells more than 10/ high power field has a strong correlation with significant bacteriuria (*p* value 0.0001). However, raised random blood sugar (RBS) at the time of sample

collection and pre-dialysis status were not found to be statistically significant [Table 1].

Urine culture showed no growth among 120 (38.71%) patients while SG with colony count ≥10,000 CFU/ml was found among 111 (35.81%) patients; either with double isolates 12 (10.81%) or with single isolates 99 (89.19%). Although urinary samples with SG was 111 (35.81%), total number of isolates in urine were 123 [Table 2].

The most common isolate among the patients with CKD was *E. coli*, 36 (28.57%) followed by *Enterococcus* spp. 22 (17.46%) and *Klebsiella pneumoniae* 20 (15.87%). Out of 22 isolates of *Enterococcus* spp., *E. faecium* 14 (63.64%) was predominating over *E. faecalis* 8 (36.36%) [Figure 1].

Among the uropathogens, 20% (25/126) belonged to yeast and yeast like fungus (YYLF). Among *Candida* spp., non-albicans group of *Candida*, predominated 24 (87.50%) over *Candida albicans* 3 (12.50%). A single *Trichosporon asahii* was isolated in urine sample in a CKD Grade IV, non-diabetic patient on dialysis [Figure 1].

Although number of urine samples with SG was found to have no significant correlation with the range of eGFR (*p* value >0.5), number of isolates was found be maximum among patients with eGFR<15, 53% (67/126) followed by eGFR 30-59, 19% (24/126) [Table 1, Figure 2].

Urine culture finding	No.		%		
No Growth (NG)	120		38.71		
Insignificant Growth (IG)	54		17.42		
Mixed Growth (MG)	25		8.06		
Significant Growth (SG)	111		35.81		
SG with no. of isolates	No.	%	No. of Isolates	No.	%
SG with double isolates	12	10.811	No. of isolates (12x2)	24	19.51
SG with Single isolate	99	89.189	No. of isolates (99x1)	99	80.49
Total	111	100	Total no. of isolates	123	100

Table 2: Distribution of Urine Culture Findings in all patients with Chronic Renal Failure.

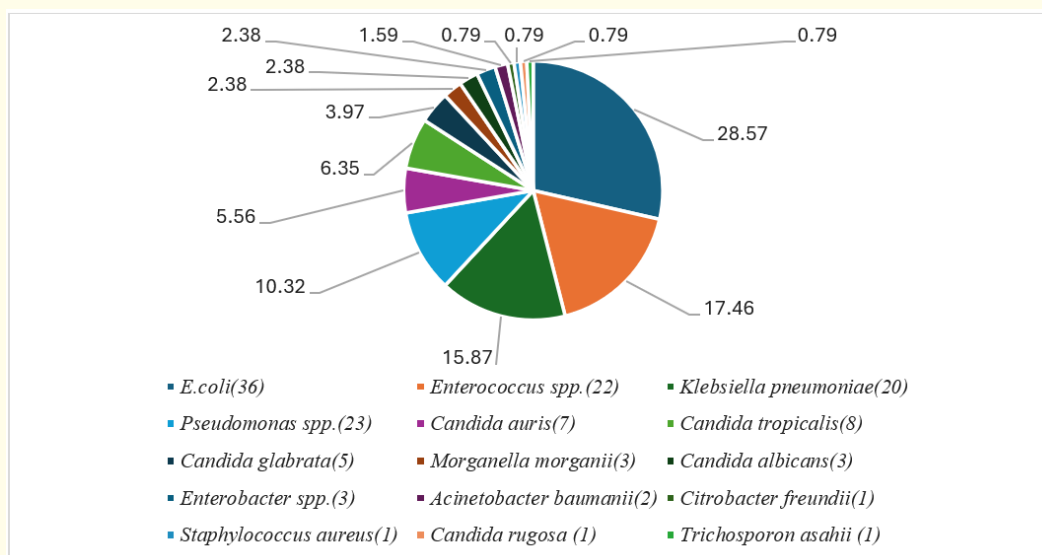


Figure 1: Urinary isolates in all patients with Chronic Renal Failure.

AST pattern of enterobacterales 63 (51.22%) (*E. coli*, *Klebsiella pneumoniae*, *Morganella morganii*, *Enterobacter spp.* and *Citrobacter freundii*) showed 97.22% (35/36) resistance to ampicillin and 78.95% resistance to ceftriaxone. Fluoroquinolone resistance ranged from 94.74% (54/57) in ciprofloxacin and 92.31% (48/52) in ofloxacin. Carbapenem resistance ranged from 49.12% (31/57) in meropenem to 54.39% (28/57) in ertapenem. Although nitrofurantoin resistance was found to be 43.64% (24/55), fosfomycin resistance was only 5.56% (2/36). Colistin resistance was found to be 5.71% (2/35) [Figure 2].

Discussion

Due to anatomical differences, the complexity of UTI in men and women is significantly different. However, we showed that both genders shared similar risk factors, and no differences in UTI was observed [Table 1] Pyuria can differentiate between true infection and transient colonization. It has been reported that in CKD cohort, 25–45% patients with pyuria could present with UTI, mostly associated at its advanced grade [9,15,16]. In this study, 64% (80/125) patients with pyuria had UTI; 52.5% (42/80) belonged to Grade V.

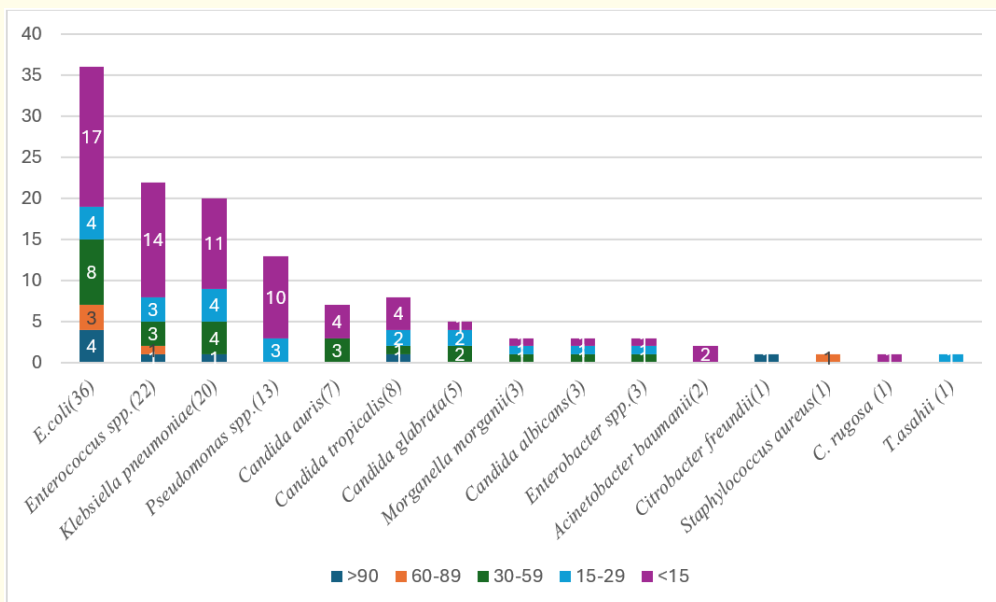


Figure 2: Distribution of urinary isolates with respect to eGFR among patients with Chronic Renal Failure.

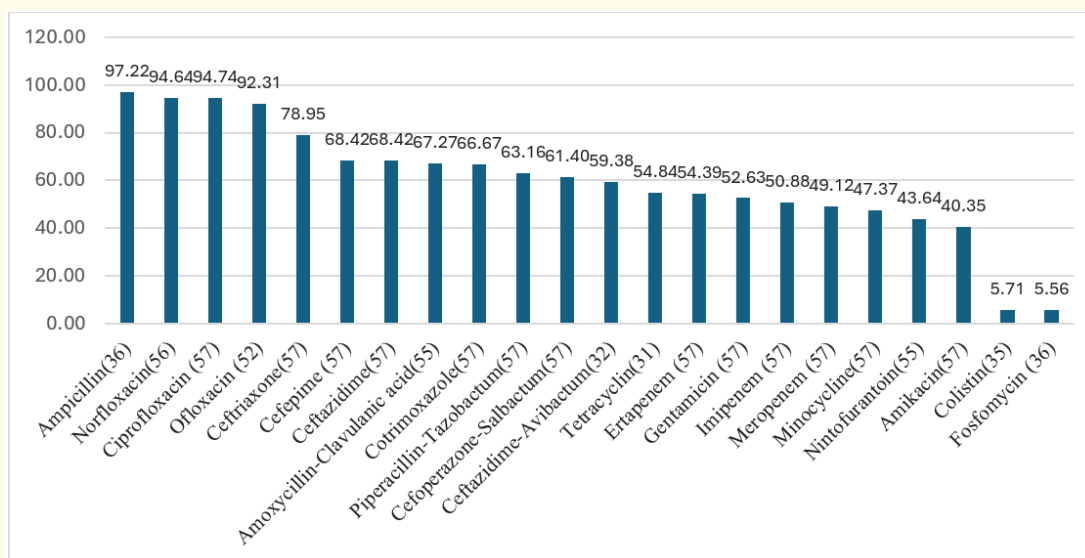


Figure 3: Antimicrobial Susceptibility Pattern of Enterobacterales (n = 63) isolated from urinary samples of patients with Chronic Renal Failure.

Sterile pyuria was found to be 36% corresponding with the findings of previous studies that reveal 31-72% prevalence of sterile pyuria among CKD patients because of chronic renal parenchymal inflammation related to diabetes, hypoalbuminemia etc [9,17,18] [Table 1].

When we compared the isolates with respect to eGFR, majority of the isolates belonged to eGFR <15 ml/min/1.73 m² irrespective of Gram-negative bacilli, Gram positive cocci and YYLFs. This finding corresponds with the finding of another literature [19].

UTI has been commonly associated with the commensal members of the gut microbiota e.g., *Escherichia coli*, *Enterococcus* spp. etc. It is presumed that there must be a correlation between an increase in the intestinal abundance of these genera and a higher prevalence of UTI [20]. In this study, *E. coli* 36 (28.57%) was found to be the most common uropathogens which is almost like other studies [1,4,12].

Catheter-associated urinary tract infection (CAUTI) represents the most common healthcare-associated infection (HAI) globally, with a fourfold risk of UTI as compared to those without a urinary catheter [4]. *Enterococcus* is the most common Gram-positive organism causing CAUTI which provokes a robust infection in the bladder, in the kidneys, and on the catheter material itself, where it forms a biofilm that promotes persistent infection in the platform of intense catheter related inflammation [1,12,21] In our study, *Enterococcus* spp., 22 (17.46%) was found to be the most 2nd most common isolate in our study; out of these, 13 (59%) were related to catheter-associated infection. Therefore, urinary catheters should only be used if clinically indicated in CKD patients [Figure 1].

Patients with inflammatory kidney disease, patients with indwelling urinary catheter are prone to develop candiduria, characteristically after inappropriate antibiotic therapy. Even asymptomatic funguria may trigger potentially life-threatening candidaemia [12]. In our study, non-albicans group of candida, 21 (87.50%) predominated over *Candida albicans*, 3 (12.50%). Our findings are not in concordance with few literatures with respect to the prevalent species of *Candida* [*C. albicans* 51.8%, 50%, 50-

70%, [22-24]]. It has been suggested that there has been a shift of *Candida* spp causing candiduria; we found non-albicans *Candida* is predominating over *C. albicans*, the most prevalent being *C. tropicalis*, 32% which is similar to other literatures (*C. tropicalis*-43% [25], 30.76% [26]). Catheterization was found to be the major risk factor for candiduria, 80% (20/25) which is in concordance with other studies [78%] [27].

Impaired immune defence associated with CKD, altered intestinal microbiome, underlying kidney disease, vascular access complications, catheterizations in combination with repeated courses of antimicrobial therapy finally leads to overgrowth of microorganisms with augmented antimicrobial resistance (AMR) [1,12]. In this study, Enterobacterales were found to be 67.67% resistant to amoxicillin-clavulanic acid; quinolone resistant ranged from 92.31% to 94.64%, ceftriaxone resistant 78.95% which is like the finding of another literature [28]. Carbapenem resistance, in our study, ranged from 49.12% in meropenem to 54.39% in ertapenem which is not in concordance to the finding of Mythri *et. al.* (16.7% for ertapenem, 12.35% for meropenem). Among *Enterococcus* spp., 41% (9/22) isolates were found to be vancomycin resistant enterococcus (VRE) which is slightly higher than the findings of other literature (Freitas MC., *et. al.* [29], 13.6%; Nguyen DB., *et. al.* [30] 11.4%). However, no VRE isolated among 3 *Enterococcus* spp. by Mythri., *et. al.* [28]. In our study, only a single methicillin resistant *Staphylococcus aureus* (MRSA) was isolated from a patient of Grade II CKD with DM, not on dialysis. This finding was correlating with the finding of a similar study [28]. However, this was not in concordance with many literatures where MRSA isolates were higher in numbers among patients with impaired renal function [31,32].

Conclusion

There is paucity of publications on the microbial isolates among patients with renal insufficiency. CKD is associated with a proinflammatory milieu with impaired innate and acquired immunity leading to infections. Altered urinary microbiome, chronic inflammation, invasive procedures provide a favourable platform to multiply the drug-resistant bugs to cause UTI. Minimal invasive procedures along with judicious use of antibiotics based on local antibiogram can provide better outcome among these patients.

Bibliography

1. Zorica Dimitrijevic., *et al.* "Risk factors for urosepsis in chronic kidney disease patients with urinary tract infections". *Scientific Reports* 11 (2021): 14414 .
2. Maaz Syed-Ahmed and Mohanram Narayanan. "Immune Dysfunction and Risk of Infection in Chronic Kidney Disease". *Advances in Chronic Kidney Disease* 26.1 (2019): 8-15.
3. Graciana Maria de Moraes Coutinho., *et al.* "Urinary tract infection in patients with chronic kidney disease under conservative treatment". *Revista Brasileira de Enfermagem* 75.3 (2022): e20210065.
4. Jürgen E Scherberich., *et al.* "Urinary tract infections in patients with renal insufficiency and dialysis - epidemiology, pathogenesis, clinical symptoms, diagnosis, and treatment". *GMS Infectious Disease* 9 (2021).
5. AHM Sanjedul Haque Sumon., *et al.* "Multidrug Resistance Urinary Tract Infection in Chronic Kidney Disease Patients: An Observational Study". *Cureus* 15.5 (2021): e38571.
6. Hans-Joachim Anders., *et al.* "The intestinal microbiota, a leaky gut, and abnormal immunity in kidney disease". *Kidney International* 83 (2013): 1010-1016.
7. Ioana Dicu-Andreescu., *et al.* "Chronic Kidney Disease, Urinary Tract Infections and Antibiotic Nephrotoxicity: Are There Any Relationships?" *Medicina (Kaunas)* 59.1 (2023): 49.
8. Andrew S Levey., *et al.* "Definition and classification of chronic kidney disease: A position statement from kidney disease: Improving Global Outcomes (KDIGO)". *Kidney International* 67 (2005): 2089-2100.
9. Lina Almaiman., *et al.* "Prevalence and Significance of Pyuria in Chronic Kidney Disease Patients in Saudi Arabia". *Journal of Personalized Medicine* 11.9 (2021): 831.
10. JG Collee., *et al.* "Laboratory Strategy in Diagnosis of Infective Syndromes: Urinary Tract Infection: Mackie and Mc Cartney". *Practical Medical Microbiology* 14 (2009): 84-90
11. Betty A Forbes., *et al.* "Infections of Urinary Tract". Chapter 57. Bailey and Scott's Diagnostic Microbiology. 12th Edition (2007).
12. Gilbert J Wise and Peter N Schlegel. "Sterile Pyuria". *The New England Journal of Medicine* 372 (2021): 11.
13. Rasheda Amin., *et al.* "Chapter 7 - Kidney and urinary tract disorders". Biochemical and Molecular Basis of Pediatric Disease (Fifth Edition) (2021): 167-228
14. IBM Corp. Released 2020. IBM SPSS Statistics for Windows, Version 27.0. Armonk, NY: IBM Corp (2020).
15. Kuo IC., *et al.* "Urinary tract infection and renal outcome in patients with chronic kidney disease stage 3-5". *Scientific Report* 10 (2020): 1-9.
16. Kwon YE., *et al.* "Prevalence and Clinical Characteristics of Asymptomatic Pyuria in Chronic Kidney Disease". *Annals of Laboratory Medicine* 40 (2020): 238-244.
17. Cabaluna CC., *et al.* "Urinalysis in patients on chronic hemodialysis". *Urology* 10 (1977): 103-104.
18. Fasolo LR., *et al.* "Diagnostic relevance of pyuria in dialysis patients". *Kidney International* 70 (2006): 2035-2038.
19. Guobin Su., *et al.* "Association of Kidney Function with Infections by Multidrug-Resistant Organisms: An Electronic Medical Record Analysis". *Scientific Reports* 8 (2018): 13372.
20. Virginia Perez-Carrasco., *et al.* "Urinary Microbiome. Yin and Yang of the Urinary Tract". *Frontiers in Cellular and Infection Microbiology* 11 (2021): 617002.
21. Weiner L M., *et al.* "Antimicrobial-resistant pathogens associated with healthcare-associated infections: summary of data reported to the National Healthcare Safety Network at the Centers for Disease Control and Prevention, 2011-2014". *Infection Control and Hospital Epidemiology* 37 (2016): 1288-1301.

22. Kauffman CA, *et al.* "Prospective multicenter surveillance study of funguria in hospitalized patients". *Clinical Infectious Disease* 30.1 (2000): 14-18.
23. Sobel JD, *et al.* "Candiduria: A randomized, double-blind study of treatment with fluconazole and placebo". *Clinical Infectious Disease* 30.1 (2000): 19-24.
24. Carol A Kauffman. "Candiduria". *Clinical Infectious Diseases* 41.6 (2005): S371-S376.
25. Paul N, *et al.* "Emerging microbiological trends in candiduria". *Clinical Infectious Disease* 39.11 (2004): 1743-1744.
26. Mythreyi Shekar Rishpana and Jyoti S Kabbin. "Candiduria in Catheter Associated Urinary Tract Infection with Special Reference to Biofilm Production". *Journal of Clinical and Diagnostic Research* 9.10 (2015): DC11-DC13.
27. Bukhary Zakeya Abdulbaqi. "Candiduria A Review of Clinical Significance and Management". *Saudi Journal of Kidney Diseases and Transplantation* 19.3 (2008): 350-360.
28. Mythri Shankar, *et al.* "Urinary Tract Infection in Chronic Kidney Disease Population: A Clinical Observational Study". *Cureus* 13.1 (2021): e12486.
29. Freitas MC, *et al.* "Prevalence of vancomycin-resistant Enterococcus fecal colonization among kidney transplant patients". *BMC Infectious Disease* 6 (2006): 133.
30. Nguyen DB, *et al.* "National Healthcare Safety Network (NHSN) Dialysis Event Surveillance Report for 2014". *Clinical Journal of the American Society of Nephrology* 12 (2017): 1139-1146.
31. Richard Fluck, *et al.* "Chapter 12: Epidemiology of Methicillin Resistant Staphylococcus Aureus Bacteraemia Amongst Patients Receiving Dialysis for Established Renal Failure in England in 2008: a joint report from the UK Renal Registry and the Health Protection Agency". *Nephron Clinical Practice* 115 (2010): c261-c270.
32. Kailash Singha, *et al.* "Methicillin-Resistant Staphylococcus aureus Infections in Patients with Renal Disorders: A Review". *World Journal of Nephrology and Urology* 8.1 (2019): 8-13.