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A COMPARATIVE STUDY ON MICROBIAL SENSITIVITY AND RESISTANCE PATTERN AMONG PATIENTS AFFECTED WITH URINARY TRACT INFECTION AT A TERTIARY CARE HOSPITAL

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ABSTRACT

Urinary tract infection (UTI) is among the most prevalent bacterial infections worldwide and represents a significant healthcare challenge. Since treatment is frequently initiated before culture results become available, understanding the local distribution of uropathogens and their antimicrobial susceptibility patterns is crucial for selecting appropriate empirical therapy. This study combined retrospective and prospective analyses conducted over a period of 10 months. A total of 1,382 urine samples were evaluated in the retrospective phase and 1,343 samples in the prospective phase. Patient demographic data, including age and gender, were collected, and urine specimens were processed using standard microbiological and biochemical techniques. Antimicrobial susceptibility testing was performed using the Kirby–Bauer disc diffusion method. In the retrospective analysis, significant bacterial growth was observed in 294 samples, with male patients accounting for 51% and female patients 49% of positive cases. The highest prevalence of UTI was noted in the 60–80-year age group. Escherichia coli was identified as the leading causative organism (51%), followed by Klebsiella species (15%). Similarly, in the prospective phase, 307 samples demonstrated significant growth, with females comprising 51.79% and males 48.21% of culture-positive cases. E. coli remained the predominant pathogen (52%), while Klebsiella species accounted for 15% of isolates. Antimicrobial susceptibility testing revealed that E. coli and Klebsiella species exhibited the greatest sensitivity to β-lactam/β-lactamase inhibitor combinations and

carbapenems. Enterococcus species showed high susceptibility to fosfomycin and vancomycin. These findings highlight the necessity of routine culture and susceptibility testing, along with effective antibiotic stewardship strategies, to optimize antimicrobial therapy and limit the emergence of resistant uropathogens.

KEYWORDS: Urinary tract infection, antimicrobial susceptibility, antibiotic resistance, Escherichia coli, Klebsiella species, carbapenems, antibiotic stewardship.

1. INTRODUCTION

Urinary tract infection (UTI) is a frequently encountered bacterial infection that affects millions of individuals globally. It develops when pathogenic microorganisms, predominantly bacteria, colonize and proliferate within any part of the urinary system, including the kidneys, ureters, bladder, and urethra.[1,2] Owing to its high prevalence and associated healthcare burden, UTI remains an important public health issue. It contributes substantially to patient morbidity, increased healthcare expenditure, and hospital admissions, ranking among the most common infectious diseases worldwide.[1,2]

UTIs can occur across all age groups, from infancy to old age. However, women are more susceptible than men because of anatomical characteristics such as a shorter urethra and its proximity to the perianal region, which facilitate bacterial entry into the urinary tract.[3] Epidemiological studies indicate that more than half of all women experience at least one episode of UTI during their lifetime[4], while recurrent infections are also common. Several predisposing factors have been associated with the development of UTIs, including diabetes mellitus, pregnancy, obesity, sexual activity, urinary catheterization, immunocompromised states, structural abnormalities of the urinary tract, and a previous history of infection.[6]

Based on clinical and anatomical characteristics, UTIs are commonly categorized as uncomplicated or complicated infections.[6] Uncomplicated UTIs generally occur in otherwise healthy individuals with normal urinary tract function, whereas complicated infections are associated with underlying conditions such as urinary tract obstruction, renal impairment, indwelling catheters, neurological disorders, pregnancy, or renal transplantation.[6] Anatomically, infections may involve the lower urinary tract, resulting in cystitis, or the upper urinary tract, leading to pyelonephritis.[6] Common clinical manifestations include painful urination, increased urinary frequency and urgency, suprapubic discomfort, fever, flank pain, and turbid urine.

A diverse range of microorganisms can cause UTIs, although uropathogenic Escherichia coli (UPEC) remains the principal etiological agent responsible for the majority of cases.[3,6] Other frequently isolated pathogens include Klebsiella species, Proteus species, Pseudomonas aeruginosa, Enterobacter species, Citrobacter species, Staphylococcus species, and Enterococcus species.[3,6] In addition, fungal urinary tract infections, particularly those caused by Candida species, are increasingly recognized among hospitalized patients, especially those receiving prolonged antibiotic therapy or possessing risk factors such as diabetes, catheterization, or organ transplantation.[1]

Accurate diagnosis of UTI relies on both clinical assessment and laboratory confirmation. Urine culture of a properly collected midstream urine sample remains the definitive diagnostic method because it enables identification of the causative organism and determination of bacterial load.[1,8] Significant bacteriuria is generally defined according to established clinical guidelines and varies depending on the type and severity of infection.[1,8] The presence of significant bacterial growth in the absence of symptoms is referred to as asymptomatic bacteriuria, a condition commonly observed among women and older adults.[1]

Antimicrobial therapy remains the cornerstone of UTI management, with agents such as nitrofurantoin, trimethoprim-sulfamethoxazole, fluoroquinolones, cephalosporins, and aminopenicillins frequently prescribed.[6] Nevertheless, the growing prevalence of antimicrobial resistance among uropathogens has emerged as a major challenge in clinical practice.[7,12] The increasing occurrence of multidrug-resistant organisms, including extended-spectrum β-lactamase-producing and carbapenem-resistant bacteria, has significantly reduced the effectiveness of conventional treatment options.[1,6] Furthermore, resistant Enterococcus strains have complicated the management of Gram-positive urinary tract infections.[1]

The emergence and spread of antimicrobial resistance are largely attributed to the inappropriate and excessive use of antibiotics.[9] Consequently, the development of novel antimicrobial agents and β-lactam/β-lactamase inhibitor combinations has become increasingly important. Newer therapeutic options have demonstrated encouraging activity against resistant Gram-negative pathogens.[6] Continuous monitoring of local antimicrobial susceptibility patterns, combined with evidence-based prescribing practices and effective antibiotic stewardship programs, is essential for optimizing patient outcomes and limiting the spread of resistant microorganisms.[10,12]

2. AIM

To assess the microbial sensitivity and resistance pattern among patients affected with urinary tract infection.

3. OBJECTIVES

To identify the prevalence of uropathogen causing UTI.

To analyze the sensitivity and resistance pattern of uropathogen causing UTI.

To compare the sensitivity and resistance pattern of uropathogenic data obtained from both retrospective and prospective study.

4. MATERIALS AND METHODS

Site of Study

The study was conducted at a 1000-bedded private tertiary care multi-specialty hospital in Coimbatore. The hospital provides advanced healthcare services in various specialties including General Medicine, Urology, Nephrology, Cardiology, Neurology, Oncology, Pediatrics, Orthopedics, and Critical Care. It is equipped with modern diagnostic and treatment facilities such as CT, MRI, ultrasound, ICCU, ICU, dialysis, kidney transplantation unit, and advanced operation theaters.

Department Selected for the Study

The study was carried out in the Departments of General Medicine and Urology, as pilot study findings showed a higher prevalence of UTI cases in these departments. Knowledge regarding the prevalence and antimicrobial sensitivity pattern of uropathogens helps healthcare professionals in selecting appropriate antibiotics for rational therapy. The study was conducted under the guidance of Clinical Pharmacy professionals and senior physicians after obtaining permission from the concerned departments.

Study Duration and Design

Study Duration: 10 months.

Study Design: Prospective, retrospective, and comparative study.

Literature Survey

A detailed literature survey was conducted throughout the study period to collect evidence related to UTIs, uropathogens, and antimicrobial resistance. Information was obtained from

various national and international journals including Indian Journal of Medical Microbiology, International Journal of Urology and Nephrology, and Ethiopian Journal of Health Sciences.

Ethical Approval and Consent

Approval for the study was obtained from the hospital Ethics Committee. Patient information forms and written consent forms were prepared to explain the study objectives, ensure confidentiality, and obtain informed consent from participants or bystanders.

Data Entry Form

A specially designed data collection form was used to record patient demographics, laboratory investigations, identified uropathogens, and their antimicrobial sensitivity and resistance patterns.

Study Population

Inclusion Criteria

- Patients of both sexes with symptoms of UTI and positive urine culture showing bacterial growth.

Exclusion Criteria

- Pregnant women.
- Urine cultures with mixed organisms.
- Patients unwilling to participate.

Sample Size: 601 patients.

Data Collection

Data were collected from the microbiology laboratory and during ward rounds in the Departments of General Medicine and Urology. Patients meeting the inclusion criteria were enrolled, and relevant clinical and laboratory details were documented using the data collection form.

5. RESULTS AND DISCUSSION

During the retrospective study period, 1,382 culture samples were analyzed, of which 294 (21%) showed positive microbial growth and 1,089 (79%) were negative. In the prospective period, 1,343 samples were tested; 307 (23%) were positive and 1,036 (77%) were negative. The prevalence of UTI was 21% in the retrospective group and 23% in the prospective group,

with an overall prevalence of 22%. This finding is consistent with a study by Mohua B et al., which also reported a 22% prevalence, correlating with our results. (Table 1)

Table 1: Culture result of study population.

Culture Result of urine sample	(Retrospective) No of patient (n = 1382)	(Prospective) No of patient (n=1343)
Positive [presence of microbial growth]	294 (21%)	307 (23%)
Negative [absence of microbial growth]	1089 (79%)	1036 (77%)

The study population was categorized based on their status of admission. (Table 2).Among the retrospective population of the study, there were 154 (52%) inpatients and 140 (48%) were outpatients and among the prospective population of the study, there were 172 (56%) inpatients and 135 (44%) were outpatients.

Table 2: Patient categorization of study population.

Department	No. of patients in retrospective (n=294)	No. of patients in prospective (n=307)
In Patient	154 (52%)	172 (56%)
Out patient	140 (48%)	135 (44%)

The study populations were categorized based on their age and gender. [Table 3] In the retrospective population 150 (51%) were male and 144 (49%) were female and in the prospective population 159 (52%) were female and 148 (48%) were male. (Table 3) Overall prevalence of the study shows that UTI were slightly more prevalence in females than in males which correlate with findings from other studies (Ezenobi N.O et al, Pritam P).

Table 3: Gender classification of study population.

Gender	No of patient in retrospective population (n=294)	No of patient in prospective population (n=307)	Total (n=601)
Male	150(51%)	148(48%)	298(49%)
Female	144(49%)	159(52%)	303(51%)

The gram staining data of the study sample were analyzed and the results reveals that 84% of retrospective sample shows gram negative bacilli and 83% of prospective sample shows gram negative bacilli followed by Fungi and gram positive cocci. (Table 4)

Table 4: Categorization of sample based on staining.

Organism	No of organism in Retrospective (n=294)	No of organism in Prospective (n=307)
Gram negative bacilli	248 (84%)	255 (83%)
Fungi	26 (9%)	33 (11%)
Gram positive cocci	20 (7%)	19 (6%)

The results were analyzed and isolated organisms were categorized. (Table 5) Out of total uropathogens isolated in retrospective population, E.coli found to be affecting 51% patient followed by K. pneumoniae (15%), P. Aeruginosa (11%) and Candida (9%) and prospective population also shows that, 52% of the isolated organism were E.coli followed by K. pneumoniae (15%), Candida (11%) and P. Aeruginosa (9%).

Escherichia coli was found to be the most common cause of UTI in both prospective (52%) and retrospective study (51%). This result is consistent with reports from other studies by Insaf B et al, Pritam P (18) reported 46%, 53.77% of E. coli in their study sample respectively. Other isolated bacteria's in our study were K. pneumonia (15%), P. aeruginosa (10%), and candida (10%) etc. These results also correlate with other studies in which Klebsiella spp. was reported as the second most frequently isolated organism in UTI.

Table 5: Categorization of isolated organism.

Type of organism	No of isolated organism (retrospective) (n=294)	No of isolated organism (prospective) (n= 307)	Total (n= 601)
E.coli	151 (51%)	160 (52%)	311 (52%)
K. pneumoniae	44 (15%)	47 (15%)	91 (15%)
P. Aeruginosa	32 (11%)	28 (9%)	60 (10%)
Candida	26 (9%)	33 (11%)	59 (10%)
Enterococcus faecalis	20 (9%)	19 (6%)	39 (7%)
Proteus mirabilis	12 (4%)	9 (3%)	21 (3%)
Citrobacter	4 (1%)	4 (2%)	8 (1%)
Acinetobacter	1 (0.34%)	5 (2%)	6 (1%)
Enterobacter	1 (0.34%)	0 (0%)	1 (0.2%)
Providencia staturtii	1 (0.34%)	0 (0%)	1 (0.2%)
Morganella morganli	1 (0.34%)	0 (0%)	1 (0.2%)
Salmonella	0 (0%)	1 (0.32%)	1 (0.2%)
Serratia Marcescens	0 (0%)	1 (0.32%)	1 (0.2%)

It's essential to categorize the variants of bacteria to know their sensitivity and resistance towards various antibiotics. The E. coli sample were further categorized based on its enzymatic variance. The ESBL strain of E.coli was found to be more predominant in both

retrospective (45%) and prospective samples (44%). The least found variant was E. coli (ESBL + AMP C) 4.5%. The other variants identified were E. coli (AMP C) 11%, E.coli (AMP C + CR) 10.5% and non-variant E. coli was present is 31% of the sample. (Table 6)

Table 6: Variant Of E.Coli In The Study Population.

Variant of E. coli	No of patient (n=151) Retrospective	No of patient (n=160) Prospective	TOTAL (n=311)
E. coli	46 (30%)	52 (32%)	98 (31%)
E. coli (ESBL)	68 (45%)	71 (44%)	139 (44.5%)
E. coli (Amp C)	16 (11%)	17 (11%)	33 (11%)
E. coli (Amp C + Cr)	13 (9%)	19 (12%)	31 (10.5%)
E. coli (ESBL + Amp C)	8 (5%)	1 (1%)	9 (3%)

Non-variant E. coli showed complete sensitivity to cephalosporins, carbapenems, beta-lactamase inhibitors, and colistin in retrospective samples, with lowest sensitivity to ampicillin (57%). Prospective samples showed a similar pattern but with increased resistance to fluoroquinolones, cephalosporins, cotrimoxazole, and ampicillin. Although empirical therapy recommends fluoroquinolones, cotrimoxazole, or ampicillin for uncomplicated UTI, our study indicates rising resistance to these drugs. E. coli was most sensitive to carbapenems (imipenem, meropenem, ertapenem), followed by beta-lactamase inhibitors, consistent with Mohua B et al. (Table 7)

Table 7: sensitivity and resistance pattern non variant E.coli.

ANTIBIOTICS	E. coli (n=46) (Retrospective)		E. coli (n=52) (Prospective)	
	Sensitivity	Resistance	Sensitivity	Resistance
Cefoperazone/Sulbactam	46 (100%)	0 (0.00%)	52 (100%)	0 (0.00%)
Piperacillin/Tazobactam	46 (100%)	0 (0.00%)	52 (100%)	0 (0.00%)
Cefuroxime	46 (100%)	0 (0.00%)	45 (87%)	7 (13%)
Ceftazidime	46 (100%)	0 (0.00%)	45 (87%)	7 (13%)
Ceftriaxone	46 (100%)	0 (0.00%)	45 (87%)	7 (13%)
Cefotaxime	46 (100%)	0 (0.00%)	45 (87%)	7 (13%)
Cefepime	46 (100%)	0 (0.00%)	49 (94%)	3 (6%)
Ertapenem	46 (100%)	0 (0.00%)	52 (100%)	0 (0.00%)
Meropenem	46 (100%)	0 (0.00%)	52 (100%)	0 (0.00%)
Imipenem	46 (100%)	0 (0.00%)	52 (100%)	0 (0.00%)
Amikacin	44 (96%)	2 (4%)	51 (98%)	1 (2%)
Gentamicin	44 (96%)	2 (4%)	43 (83%)	9 (17%)
Netilmicin	45 (98%)	1 (2%)	46 (88%)	6 (12%)
Tobramycin	40 (87%)	6 (13%)	51 (98%)	1 (2%)
Norfloxacin	28 (61%)	18 (39%)	25 (48%)	27 (52%)
Ofloxacin	29 (63%)	17 (37%)	23 (44%)	29 (56%)
Trimethoprim/Sulfamethoxazole	32 (70%)	14 (30%)	33 (63%)	19 (37%)

Fosfomycin	43 (93%)	3 (7%)	48 (92%)	4 (8%)
Colistin	46 (100%)	0 (0.00%)	52 (100%)	0 (0.00%)
Nitrofurantoin	39 (85%)	13 (15%)	47 (90%)	5 (10%)
Doxycycline	34 (74%)	12 (26%)	37 (71%)	15 (29%)
Ampicillin	26 (57%)	20 (43%)	28 (54%)	24 (46%)

Both prospective and retrospective samples shown that ESBL producing E. coli is most sensitive to Carbapenems (Imipenem, Meropenem, and Ertapenem). This finding is consistent with the study carried out by Bander B (22) also reported that ESBL producing E. coli are more sensitive to carbapenem. (Table 8)

Table 8: sensitivity and resistance pattern of E.Coli (ESBL).

Antibiotics	**E. coli (ESBL) (n=68) (Retrospective)**		**E. coli (ESBL) (n=71) (Prospective))**	
	Sensitivity	Resistance	Sensitivity	Resistance
CEFOPERAZONE/SULBACTAM (CFS)	68 (100%)	0 (0.00%)	69 (97%)	2 (3%)
PIPERACILLIN/TAZOBACTAM (PT)	68 (100%)	0 (0.00%)	71 (100%)	0 (0%)
CEFUROXIME (CU)	0 (0.00%)	68 (100%)	0 (0%)	71 (100%)
CEFTAZIDIME (CAZ/CA)	0 (0.00%)	68 (100%)	0 (0%)	71 (100%)
CEFTRIAXONE (CI)	0 (0.00%)	68 (100%)	0 (0%)	71 (100%)
CEFOTAXIME (CE)	0 (0.00%)	68 (100%)	0 (0%)	71 (100%)
CEFEPIME (CPM)	0 (0.00%)	68 (100%)	2 (3%)	69 (97%)
ERTAPENEM (ETP)	68 (100%)	0 (0.00%)	71 (100%)	0 (0%)
MEROPENEM (MR)	68 (100%)	0 (0.00%)	71 (100%)	0 (0%)
IMIPENEM (I)	68 (100%)	0 (0.00%)	71 (100%)	0 (0%)
AMIKACIN (AK)	62 (91%)	6(9%)	69 (97%)	2 (3%)
GENTAMICIN (G)	42 (62%)	26 (38%)	52 (73%)	19 (27%)
NETILMICIN (NT)	62 (92%)	6(8%)	63 (89%)	10 (11%)
TOBRAMYCIN (TB)	55 (81%)	13 (19%)	51 (72%)	20 (28%)
NORFLOXACIN (NX)	14 (21%)	54 (79%)	24 (34%)	47 (66%)
OFLOXACIN (OF)	14 (21%)	54 (79%)	24 (34%)	47 (66%)
TRIMETHOPRIM/SULFAMETHOXAZOLE (COT)	23 (34%)	45 (66%)	27 (38%)	44 (62%)
FOSFOMYCIN (FO)	68 (100%)	0 (0.00%)	71 (100%)	0 (0%)
COLISTIN (CL)	68 (100%)	0 (0.00%)	71 (100%)	0 (0%)
NITROFURANTOIN (NF)	59 (86%)	9 (14%)	61 (86%)	10 (14%)
DOXYCYCLINE (DO)	31 (46%)	37 (54%)	44 (62%)	27 (38%)
AMPICILLIN (A)	0 (0.00%)	68 (100%)	0 (0%)	71 (100%)

In contrast to standard E. coli, the AMP C variant was 100% resistance towards Cephalosporins, beta lactamase and ampicillin. The AMP C strain was 100% sensitive to Carbapenems Fosfomycin and Colistin. The empirical antibiotics used in UTI such as fluoroquinolones found to be less sensitive for AMP C strain while nitrofurantoin highly sensitive to AMP C strain. (Table 9)

Table 9: sensitivity and resistance pattern of E.Coli (AMPC)

ANTIBIOTICS	E. coli (AMPC) (n=16) (Retrospective) ve)		E. coli (AMPC) (n=17) (Prospective)	
	SSensitivity	RResistance	Sensitivity	Resistance
CEFOPERAZONE/SULBACTAM (CFS)	0 (0.00%)	16 (100%)	0 (0%)	17 (100%)
PIPERACILLIN/TAZOBACTAM (PT)	0 (0.00%)	16 (100%)	0 (0%)	17 (100%)
CEFUROXIME (CU)	0 (0.00%)	16 (100%)	0 (0%)	17 (100%)
CEFTAZIDIME (CAZ/CA)	0 (0.00%)	16 (100%)	0 (0%)	17 (100%)
CEFTRIAXONE (CI)	0 (0.00%)	16 (100%)	0 (0%)	17 (100%)
CEFOTAXIME (CE)	0 (0.00%)	16 (100%)	0 (0%)	17 (100%)
CEFEPIME (CPM)	7 (44%)	9 (56%)	10 (59%)	7 (41%)
ERTAPENEM (ETP)	16 (100%)	0 (0.00%)	17 (100%)	0 (0%)
MEROPENEM (MR)	16 (100%)	0 (0.00%)	17 (100%)	0 (0%)
IMIPENEM (I)	16 (100%)	0 (0.00%)	17 (100%)	0 (0%)
AMIKACIN (AK)	16 (100%)	0 (0.00%)	17 (100%)	0 (0%)
GENTAMICIN (G)	14 (87%)	2 (13%)	14 (82%)	3 (18%)
NETILMICIN (NT)	16 (100%)	0 (0.00%)	16 (94%)	1 (6%)
TOBRAMYCIN (TB)	16 (100%)	0 (0.00%)	15 (88%)	2 (12%)
NORFLOXACIN (NX)	5 (31%)	11 (69%)	5 (29%)	12 (71%)
OFLOXACIN (OF)	5 (31%)	11 (69%)	5 (29%)	12 (71%)
TRIMETHOPRIM/SULFAMETHOXAZOLE (COT)	5 (31%)	11 (69%)	4 (24%)	13 (76%)
FOSFOMYCIN (FO)	16 (100%)	0 (0.00%)	17 (100%)	0 (0%)
COLISTIN (CL)	16 (100%)	0 (0.00%)	17 (100%)	0 (0%)
NITROFURANTOIN (NF)	14 (87%)	2 (13%)	16 (94%)	1 (6%)
DOXYCYCLINE (DO)	9 (56%)	7 (44%)	8 (47%)	9 (53%)
AMPICILLIN (A)	0 (0.00%)	16 (100%)	0 (0%)	17 (100%)

In contrast to standard E. coli, the AMP C + CR variant was 100% resistance towards Cephalosporins, Carbapenems, beta lactamase and ampicillin. The AMP C + CR strain was 100% sensitive to only Fosfomycin and Colistin. The empirical antibiotics used in UTI such as fluoroquinolones found to be 100% resistance for AMP C + CR strain. (Table 10)

Table 10: Sensitivity and resistance pattern of E.Coli (AMPC+CR)

ANTIBIOTICS	E. coli (AMPC+ CR) (n=13) (Retrospective)		E. coli (AMPC+ CR) (n=19) (Prospective).	
	Sensitivity	Resistance	Sensitivity	Resistanceistance
CEFOPERAZONE/SULBACTAM (CFS)	0 (0.00%)	13 (100%)	0 (0%)	19 (100%)

PIPERACILLIN/TAZOBACTAM (PT)	0 (0.00%)	13 (100%)	0 (0%)	19 (100%)
CEFUROXIME (CU)	0 (0.00%)	13 (100%)	0 (0%)	19 (100%)
CEFTAZIDIME (CAZ/CA)	0 (0.00%)	13 (100%)	0 (0%)	19 (100%)
CEFTRIAXONE (CI)	0 (0.00%)	13 (100%)	0 (0%)	19 (100%)
CEFOTAXIME (CE)	0 (0.00%)	13 (100%)	0 (0%)	19 (100%)
CEFEPIME (CPM)	0 (0.00%)	13 (100%)	0 (0%)	19 (100%)
ERTAPENEM (ETP)	0 (0.00%)	13 (100%)	0 (0%)	19 (100%)
MEROPENEM (MR)	0 (0.00%)	13 (100%)	0 (0%)	19 (100%)
IMIPENEM (I)	0 (0.00%)	13 (100%)	0 (0%)	19 (100%)
AMIKACIN (AK)	4 (31%)	9 (69%)	13 (68%)	6 (32%)
GENTAMICIN (G)	3 (23%)	10 (77%)	12 (63%)	7 (37%)
NETILMICIN (NT)	4 (31%)	9 (69%)	13 (68%)	6 (32%)
TOBRAMYCIN (TB)	3 (23%)	10 (77%)	12 (63%)	7 (37%)
NORFLOXACIN (NX)	0 (0.00%)	13 (100%)	1 (5%)	18 (95%)
OFLOXACIN (OF)	0 (0.00%)	13 (100%)	1 (5%)	18 (95%)
TRIMETHOPRIM/SULFAMETHOXAZOLE (COT)	0 (0.00%)	13 (100%)	2 (11%)	17 (89%)
FOSFOMYCIN (FO)	13 (100%)	0 (0.00%)	19 (100%)	0 (0%)
COLISTIN (CL)	13 (100%)	0 (0.00%)	19 (100%)	0 (0%)
NITROFURANTOIN (NF)	9 (69%)	4 (31%)	12 (63%)	7 (37%)
DOXYCYCLINE (DO)	5 (38%)	8 (62%)	7 (37%)	12 (63%)
AMPICILLIN (A)	0 (0.00%)	13 (100%)	0 (0%)	19 (100%)

The K. pneumoniea sample were also further categorized based on its enzymatic variance. The Amp c and CR producing K. pneumoniea was found to be more predominant in both retrospective (36%) and prospective sample (38%). The least found variant was ESBL producing K. pneumoniea 9%. The other variants identified were K. pneumoniea (ESBL + Amp c) 11%, K. pneumoniea (Amp c) 10%, K. pneumoniea (ESBL + Amp c + Cr) 9% and non-variant K. pneumoniea (24%). (Table 11)

Table 11: Variant of K. pneumoniea in the study population.

Variant of K. pneumoniae	No of patient (n = 44) Retrospective	No of patient (n = 47) Prospective	Total (n = 91)
K. pneumoniae	12 (27%)	10 (21%)	22 (24%)
K. pneumoniae (Amp c + Cr)	16 (36%)	18 (38%)	34 (37%)
K. pneumoniae (ESBL + Amp c)	7 (16%)	3 (6%)	10 (11%)
K. pneumoniae (Amp c)	3 (7%)	6 (13%)	9 (10%)
K. pneumoniae (ESBL + Amp c + Cr)	4 (9%)	4 (9%)	8 (9%)
K. pneumoniae (ESBL)	2 (5%)	6 (13%)	8 (9%)

The sensitivity and resistance pattern of standard non- variant K. pneumonia was analysed. In case standard K. pneumonia isolated was completely sensitive towards cephalosporins, carbapenems, beta lactamase inhibitors and Colistin. The standard K. pneumonia has least sensitive (100%) towards ampicillin. Similar sensitivity pattern were seen among the

prospective sample with some marginal deviation. Increased resistance was observed among, aminoglycosides and Clotrimazole**.** Our study shows that K. pneumonia is most sensitive to Carbapenems (Imipenem, Meropenem, Ertapenem), followed by beta lactamase inhibitor's. This result correlates with the study result by Mohua B et al which also reveals K. pneumonia show maximum sensitivity towards Carbapenem antibiotics. (Table 12)

Table 12: Sensitivity and Resistance pattern of K. pneumonia.

ANTIBIOTICS	K. pneumonia (n=12) (Retrospective)		K. pneumonia (n=10) (Prospective)	
	Sensitivity	Resistance	Sensitivity	Resistance
Cefoperazone/Sulbactam	12 (100%)	0 (0.00%)	10 (100%)	0 (0%)
Piperacillin/Tazobactam	12 (100%)	0 (0.00%)	10 (100%)	0 (0%)
Cefuroxime	12 (100%)	0 (0.00%)	10 (100%)	0 (0%)
Ceftazidime	12 (100%)	0 (0.00%)	10 (100%)	0 (0%)
Ceftriaxone	12 (100%)	0 (0.00%)	10 (100%)	0 (0%)
Cefotaxime	12 (100%)	0 (0.00%)	10 (100%)	0 (0%)
Cefepime	12 (100%)	0 (0.00%)	10 (100%)	0 (0%)
Ertapenem	12 (100%)	0 (0.00%)	10 (100%)	0 (0%)
Meropenem	12 (100%)	0 (0.00%)	10 (100%)	0 (0%)
Imipenem	12 (100%)	0 (0.00%)	10 (100%)	0 (0%)
Amikacin	12 (100%)	0 (0.00%)	9 (90%)	1 (10%)
Gentamicin	12 (100%)	0 (0.00%)	9 (90%)	1 (10%)
Netilmicin	12 (100%)	0 (0.00%)	7 (70%)	3 (30%)
Tobramycin	11 (92%)	1 (8%)	9 (90%)	1 (10%)
Norfloxacin	11 (92%)	1 (8%)	10 (100%)	0 (0%)
Ofloxacin	12 (100%)	0 (0.00%)	10 (100%)	0 (0%)
Trimethoprim/ Sulfamethoxazole	11 (92%)	1 (8%)	8 (80%)	2 (20%)
Fosfomycin	12 (100%)	0 (0.00%)	10 (100%)	0 (0%)
Colistin	12 (100%)	0 (0.00%)	10 (100%)	0 (0%)
Nitrofurantoin	6 (50%)	6 (50%)	4 (40%)	6 (60%)
Doxycycline	11 (92%)	1 (8%)	9 (90%)	1 (10%)
Ampicillin	0 (0.00%)	12 (100%)	0 (0%)	10 (100%)

The sensitivity and resistance pattern of K. pneumonia (Amp C + CR) variant was analysed. In contrast to standard K. pneumonia, Amp C + CR variant was 100% resistance towards cephalosporins, beta lactamase inhibitors, carbapenems, and fluoroquinolones. Amp C + CR strain was 100% sensitive to only Colistin. (Table 13)

Table 13: Sensitivity and Resistance pattern of K. pneumonia(AMPC+CR).

ANTIBIOTICS	K. pneumonia(AMPC+CR) (n=16) (Retrospective)		K. pneumonia(AMPC+CR) (n=18) (Prospective)	
	Sensitivity	Resistance	Sensitivity	ReResistance
CEFOPERAZONE/SULBACTAM (CFS)	0 (0%)	16 (100%)	0 (0%)	18 (100%)

PIPERACILLIN/TAZOBACTAM (PT)	0 (0%)	16 (100%)	0 (0%)	18 (100%)
CEFUROXIME (CU)	0 (0%)	16 (100%)	0 (0%)	18 (100%)
CEFTAZIDIME (CAZ/CA)	0 (0%)	16 (100%)	0 (0%)	18 (100%)
CEFTRIAXONE (CI)	0 (0%)	16 (100%)	0 (0%)	18 (100%)
CEFOTAXIME (CE)	0 (0%)	16 (100%)	0 (0%)	18 (100%)
CEFEPIME (CPM)	0 (0%)	16 (100%)	0 (0%)	18 (100%)
ERTAPENEM (ETP)	0 (0%)	16 (100%)	0 (0%)	18 (100%)
MEROPENEM (MR)	0 (0%)	16 (100%)	0 (0%)	18 (100%)
IMIPENEM (I)	0 (0%)	16 (100%)	0 (0%)	18 (100%)
AMIKACIN (AK)	0 (0%)	16 (100%)	4 (22%)	14 (78%)
GENTAMICIN (G)	1 (6%)	15 (94%)	4 (22%)	14 (78%)
NETILMICIN (NT)	1 (6%)	15 (94%)	4 (22%)	14 (78%)
TOBRAMYCIN (TB)	0 (0%)	16 (100%)	4 (22%)	14 (78%)
NORFLOXACIN (NX)	0 (0%)	16 (100%)	0 (0%)	18 (100%)
OFLOXACIN (OF)	0 (0%)	16 (100%)	0 (0%)	18 (100%)
TRIMETHOPRIM/SULFAMETHOXAZOLE (COT)	0 (0%)	16 (100%)	0 (0%)	18 (100%)
FOSFOMYCIN (FO)	13 (81%)	3 (19%)	17 (94%)	1 (6%)
COLISTIN (CL)	16 (100%)	0 (0%)	18 (100%)	0 (0%)
NITROFURANTOIN (NF)	1 (6%)	15 (94%)	3 (17%)	15 (83%)
DOXYCYCLINE (DO)	14 (88%)	2 (12%)	13 (72%)	5 (28%)
AMPICILLIN (A)	0 (0%)	16 (100%)	0 (0%)	18 (100%)

P. aeruginosa (58%) followed by carbapenem resistance producing P. Aeruginosa (42%). (Table 14)

Table 14: Variant of P. aeruginosa in the study population.

Variant of P. aeruginosa	No of patient (n = 32) Retrospective	No of patient (n = 28) Prospective	Total (n = 60)
P. aeruginosa	21 (67%)	14 (50%)	35 (58%)
P. aeruginosa (Cr)	11 (33%)	14 (50%)	25 (42%)

The sensitivity and resistance pattern of standard P. aeruginosa was analysed. (Table 15) In case standard P. aeruginosa isolated was completely sensitive towards Aztreonam, carbapenems and Colistin while other antibiotics show minimal resistance. Ahmat, A.M et al (27) conducted a similar study which reveals that P. aeruginosa is highly susceptible to Colistin and Imipenem which correlates with our study result which also reveals P. aeruginosa highly susceptible to Colistin and Imipenem.

Table 15: Sensitivity and Resistance pattern of P. aeruginossa.

Antibiotics	P. Aeruginosa N=21 (Retrospective)		P. Aeruginosa N=14 (Prospective)	
	Sensitivity	Resistance	Sensitivity	Resistance
Cefoperazone/Sulbactam	20 (95%)	1 (5%)	14 (100%)	0 (0%)

Piperacillin/Tazobactam	20 (95%)	1 (5%)	14 (100%)	0 (0%)
Ceftazidime	19 (90%)	2 (10%)	14 (100%)	0 (0%)
Cefepime	18 (86%)	3 (14%)	13 (93%)	1 (7%)
Aztreonam	21 (100%)	0 (0%)	14 (100%)	0 (0%)
Meropenem	21 (100%)	0 (0%)	14 (100%)	0 (0%)
Imipenem	21 (100%)	0 (0%)	14 (100%)	0 (0%)
Amikacin	20 (95%)	1 (5%)	9 (64%)	5 (36%)
Gentamicin	17 (81%)	4 (19%)	12 (86%)	2 (14%)
Netilmicin	20 (95%)	1 (5%)	14 (100%)	0 (0%)
Tobramycin	20 (95%)	1 (5%)	14 (100%)	0 (0%)
Levofloxacin	19 (90%)	2 (10%)	13 (93%)	1 (7%)
Norfloxacin	19 (90%)	2 (10%)	13 (93%)	1 (7%)
Ofloxacin	19 (90%)	2 (10%)	13 (93%)	1 (7%)
Fosfomycin	19 (90%)	2 (10%)	9 (64%)	5 (36%)
Colistin	21 (100%)	0 (0%)	14 (100%)	0 (0%)
Piperacillin	19 (90%)	2 (10%)	12 (86%)	2 (14%)

The sensitivity pattern of Candida was analysed. Candida spp was 100% sensitive to all the anti- fungal drugs (Amphotericin B, Clotrimazole, Fluconazole, Itraconazole, Ketoconazole and Nystatin) with no resistance on tested anti-fungal agents. (Table 16)

Table 16: Sensitivity and Resistance pattern of Candida spp.

ANTI-FUNGAL	Candida spp (n=26) (Retrospective)		Candida spp (n=33) (Prospective)	
	Sensitivity	Resistance	Sensitivity	Resistance
Amphotericin	26 (100.00%)	0 (0.00%)	33 (100%)	0 (0.00%)
Clotrimazole	26 (100.00%)	0 (0.00%)	33 (100%)	0 (0.00%)
Fluconazole	26 (100.00%)	0 (0.00%)	33 (100%)	0 (0.00%)
Itraconazole	26 (100.00%)	0 (0.00%)	33 (100%)	0 (0.00%)
Ketoconazole	26 (100.00%)	0 (0.00%)	33 (100%)	0 (0.00%)
Nystatin	26 (100.00%)	0 (0.00%)	33 (100%)	0 (0.00%)

6. CONCLUSION

The findings of this study highlight the critical role of urine culture and antimicrobial susceptibility testing in the management of urinary tract infections. Performing culture sensitivity testing enables accurate identification of the causative uropathogens and helps clinicians select the most effective antimicrobial agents for treatment. Since pathogens such as Escherichia coli possess multiple strains with varying resistance and susceptibility profiles, empirical antibiotic therapy may not always produce satisfactory clinical outcomes. Therefore, culture and sensitivity testing should be considered essential, particularly in patients with recurrent, persistent, or complicated UTIs, to ensure appropriate treatment and reduce the risk of disease progression and associated complications.

In addition, routine susceptibility testing contributes to the development of local antibiograms, which serve as valuable tools for monitoring antimicrobial resistance trends and guiding evidence-based antibiotic prescribing. Regular evaluation of antibiotic utilization patterns and timely updates to institutional treatment guidelines are necessary to promote the judicious use of antimicrobial agents. The implementation of comprehensive antibiotic stewardship programs within healthcare facilities can further optimize antimicrobial therapy, improve patient outcomes, and help curb the emergence and spread of antibiotic-resistant pathogens across a wide range of infectious diseases.

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