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ORIGINAL ARTICLE

Phenotypic, genotypic characterization and antimicrobial resistance profiling of uropathogenic *Escherichia coli* in a tertiary care hospital, Puducherry, India

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ABSTRACT

Background and Objectives: Uropathogenic *Escherichia coli* (*E. coli)* (UPEC) accounts for 70-95% of community-acquired urinary tract infections (UTIs) and a significant proportion of nosocomial UTIs. This study aimed to characterize the phenotypic and genotypic characteristics of *E. coli* isolates from symptomatic UTI patients and evaluate their antimicrobial susceptibility patterns.

Materials and Methods: A hospital-based observational study was conducted at Aarupadai Veedu Medical College and Hospital, Puducherry, India, from August 2022 to April 2024. A total of 106 UPEC isolates were obtained from symptomatic UTI patients. Antimicrobial susceptibility testing (AST) was performed using the Kirby-Bauer method, and virulence genes (*hlyA, fimH, papC*) were detected using PCR.

Results: The mean age of patients was 49.7 years, with a female predominance (69.8%). Diabetes mellitus was the most common comorbidity (29.2%). Fever (60.4%) and dysuria (38.7%) were the most common symptoms. AST showed high susceptibility (>90%) to amikacin, nitrofurantoin, meropenem, and piperacillin/tazobactam, while >60% resistance was observed to cefotaxime and ceftazidime. Phenotypically, 30.2% of the isolates produced mannose-resistant hemagglutinins, and 17.9% produced hemolysin. ESBL production was found in 46.3%. Biofilm production was moderate in 65.1%, weak in 30.2% and strong in 4.7% and significantly correlated with multidrug resistance ($p<0.05$). Genotypically, 80.2% had *fimH*, 51.9% had *papC* and 20.8% had *hlyA*. *papC* was associated with reduced cefotaxime susceptibility ($p<0.05$).

Conclusion: The study highlights the significance of phenotypic and genotypic characterization in understanding UPEC virulence and resistance patterns, and emphasizes the need for targeted empiric therapy to improve UTI management.

Keywords: Urinary tract infection; Uropathogenic *Escherichia coli*; Antibiotic susceptibility testing; Antimicrobial resistance

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INTRODUCTION

Urinary tract infections (UTIs) are infections occurring in the bladder, ureters, kidneys, and urethra. The pathogenesis of infections in the urinary tract usually begins with the infiltration of microorganisms into the urinary tract, where they begin to multiply (1). Among bacterial infections, UTIs are one of the most common causes, with 230,000 deaths and an estimated 400 million cases worldwide as of 2019. The most common pathogens causing UTIs are *Escherichia coli*, *Klebsiella pneumoniae*, *Enterococcus faecalis*, and *Proteus mirabilis.* Among these, *E. coli* accounts for approximately 70% to 95% of community-acquired UTIs and a substantial proportion of nosocomial UTIs (2). A recently published systematic review/meta-analysis revealed that, in India, the most common pathogens are *E. coli*, with a prevalence rate of 49.6%, and *Klebsiella* spp., with 12.8% (3). Uropathogenic *E. coli* (UPEC) is responsible for 65% of complicated and 75% of uncomplicated UTIs. UPEC is one of the four pathotypes of extraintestinal pathogenic *E. coli* (ExPEC). ExPEC pathotypes are classified based on their isolation site and the virulence genes that enable them to cause disease outside of the intestinal tract. This highlights the ability of UPEC to grow and cause infection in host sites beyond the intestine (4).

UPEC isolates harbour pathogenicity islands, which are mobile genetic elements hosting specialized virulence genes. Common virulence traits among UPEC isolates include adhesins associated with fimbriae (P and type 1 fimbriae), mechanisms to evade host defences (capsules and lipopolysaccharides), nutrient acquisition mechanisms (siderophores), and toxins (hemolysin, cytotoxin necrotizing factor) (5). These virulence factors facilitate their invasion, colonization, and persistence within the urinary tract, with bacterial adhesion to the uroepithelium being the first step in UTI development (6). Genes encoding adhesion molecules serve as crucial markers of the organism's virulence (7).

Typically, patients presenting with clinical symptoms of UTI are promptly administered antibiotics without antibiotic susceptibility testing, potentially leading to the development of multidrug-resistant (MDR) microorganisms. The treatment protocol for UTIs follows different guidelines in different regions of the world, further complicating the escalating prevalence of antibiotic-resistant organisms (8). Since the mid-2000s, there has been a consistent upward trend in multidrug-resistant UPEC case rates, challenging the efficacy of clinical management, and thus emerging as a public health concern worldwide (9). The management of UTI necessitates timely administration of appropriate antibiotics. This requires regional antibiotic susceptibility patterns, an understanding of biofilm formation ability, virulence profile, and comprehensive knowledge of the association between these factors. This approach will ensure a more targeted and successful treatment strategy for UTIs caused by *E. coli* (10). Therefore, this study is undertaken to evaluate the different virulence markers among UPEC isolates, determine the antibiotic susceptibility pattern, and determine the correlation between virulence markers and their antibiotic resistance pattern.

MATERIALS AND METHODS

This observational study was conducted between August 2022 and April 2024 among patients in the outpatient department and the inpatient wards of Aarupadai Veedu Medical College and Hospital, a tertiary teaching healthcare facility, in Puducherry, India. Institutional Human Ethics Committee (IHEC), Aarupadai Veedu Medical College, Puducherry approved the study. The study included 106 non-duplicated UPEC isolates obtained from symptomatic UTI patients.

Sample collection and processing. Midstream clean catch urine was collected in a sterile, leakproof container and transported to the laboratory for processing according to standard microbiological procedures using media from Hi Media Laboratory Pvt Ltd, Mumbai, India. A wet mount preparation of centrifuged urine was examined under a microscope to look for significant pyuria (more than 10 pus cells/HPF). Culture was performed using the semiquantitative method on MacConkey agar to look for significant bacteriuria (100000 CFU/ml). Antimicrobial susceptibility testing (AST) was performed using the Kirby Bauer disk diffusion method (11), with a range of antibiotic disks (HiMedia) as per CLSI guidelines 2022(M100 edition 32) and hospital availability.

ESBL screening. Extended-spectrum beta-lactamase (ESBL) screening was performed using a combined disk diffusion test (CDDT) for all the iso-

lates. The 0.5 McFarland preparation of the isolate was plated on the Mueller Hinton Agar (MHA) plate. The disks ceftazidime (30 µg), ceftazidime-clavulanic acid (30/10 µg) and cefotaxime (30 µg), cefotaxime-clavulanic acid (30/10 µg) were kept on the same plate and incubated at 37°C for 16 to18 hours. Zone size was measured and compared. An increase of 5 mm or more in zone diameter when tested in combination with v/s was considered an ESBL producer. ATCC 25922 *E. coli* was used as the control strain (12).

Identification of virulence factors by phenotypic method. An isolated colony of the *E. coli* was streaked onto 5% human blood agar to test for beta hemolysis. Isolated colonies inoculated into nutrient broth were incubated at 37°C for 48 hours to achieve complete fimbriation. A drop of 3% RBC suspension was added to a drop of bacterial suspension on a clean slide and incubated for 5 minutes at room temperature. If agglutination was present, a drop of 2% w/v of D-mannose was added to test for mannose-sensitive/resistant haemagglutination (MSHA/MRHA) (13). The biofilm-forming capacity was assessed by the tube method. A loopful of test organisms was inoculated into 10ml trypticase soy broth (TSB) with 1% glucose in test tubes and incubated at 37°C for 24 hours. Tubes were decanted and washed with phosphate buffer saline (PBS) (pH 7.3) and dried. Tubes were stained with 0.1% crystal violet and incubated for 30 minutes at room temperature. Excess stain was washed off with deionized water and the tubes were dried in an inverted position. The observation was made based on visible film lining the sides and bottom of the tubes. Scores were given as weak, moderate, or strong biofilm producers (14).

Identification of virulence genes. A commercially available Origin bacteria DNA kit (Kerala) was used for DNA extraction. The procedure was carried out according to the strict instructions of the manufacturer. The virulence genes such as *hly* A (alpha-hemolysin) encoding hemolysin, *fim H* (Type 1 fimbrin D-mannose specific adhesin) and *pap C* (pilus associated with pyelonephritis C) encoding adhesins responsible for the adhesion of organisms to the uroepithelial cells were detected by the conventional method of polymerase chain reaction using appropriate primers (Eurofins) as mentioned in Table 1 and visualized by gel electrophoresis. The total reaction volume of PCR was 10 µl per reaction (15).

Statistical analysis. The data obtained were manually entered into Microsoft Excel and analyzed using the Statistical Package for Social Sciences (SPSS) v23. All the categorical variables were summarised using frequencies and percentages. Continuous variables were summarized using mean (standard deviation) and/or median (interquartile range) (based on the results of data normality, tested using the Kolmogorov–Smirnov test and the Shapiro–Wilk test). The Chi-square test or Fisher exact test (for categorical variables) and the independent "t" test or Mann-Whitney U test (for continuous variables) was used to test statistical significance. Statistical significance was considered when the p-value was less than 0.05.

RESULTS

Distribution of baseline characteristics among study participants. The mean age of the patients was 49.7 years. Almost half the patients (42.5%) were between 41 and 60 years of age. More than two-thirds of patients (69.8%) were females. More than one-third of patients with UTI had underlying diseases (38.7%) with diabetes mellites (29.2%) being the most common. The distribution of patients by presenting symptoms showed that fever was the most common presenting symptom (60.4%), followed by pain on micturition (38.7%), loin/groin pain (36.8%), and polyuria (27.4%). The distribution of patients by significant history/predisposing factors showed that 6.6% of patients had a history of instrumentation, 5.7% had a history of renal calculi, 5.6% were pregnant women, 4.7% had benign prostatic hypertrophy, and 3.7% had post-LSCS status (Table 2).

Resistance pattern among the UPEC isolates. Results of antibiotic susceptibility testing showed significant resistance to amoxycillin/clavulanic acid (40.6%), ciprofloxacin (49.1%), ceftazidime (61.3%), and cefotaxime (62.3%). Gentamicin, cefepime and cotrimoxazole showed comparatively low resistance, with resistance rates ranging from approximately 25% to 40%. The resistance rates of amikacin, meropenem, piperacillin/tazobactam, and nitrofurantoin were 5.7%, 5.7%, 6.6%, and 8.5% respectively (Fig.1).

Phenotypic identification of virulence factors. Mannose-resistant haemagglutination was seen in 30.2% and 11 isolates (10.4%) had mannose-sensitive

Table 1. Primers and optimized temperature used for molecular characterization of genes

Genes	Primer sequence (5'-3')	Annealing temperature	Amplicon size	Reference
uid A (Beta D Glucouronidase)	F:TCACCGTGGTGACGCATGTCGC R:CACCACGATGCCATGTTCATCTGC	56°C	486 bp	Zeb et al. (27)
hly A (α-hemolysin)	F: AACAAGGATAAGCACTGTTCTGGCT R:ACCATATAAGCGGTCATTCCCGTCA	58°C	1177 bp	
pap C (P pili)	F-GACGGCTGTACTGCAGGGTGTGGC R-ATATCCTTTCTGCAGGGATGCAATA	62°C	328 bp	Basu et al. (28)
fim H (fimbrial antigen)	F-TGCAGAACGGATAAGCCGTGG R-GCAGTCACCTGCCCTCCGGTA	58°C	508 bp	

F-Forward primer, R-Reverse primer, *uid A* = Beta D Glucouronidase, *hly A* =hemolysin A, *pap C* =P pili, *fim H* = fimbrial antigen

haemagglutination. Importantly, 59.4% of the isolates were negative for agglutination. Most of the isolates were non-hemolytic (82.1%), whereas 17.9% of isolates were beta-hemolytic (Table 3).

ESBL screening and biofilm production. The results showed that ESBL production was present in 46.3% of the isolates and absent in 53.7% of the isolates. All the isolates were biofilm producers, with two-thirds (65.1%) of the isolates showing moderate biofilm production, 30.2% showing weak biofilm production, and 4.7% showing strong biofilm production (Table 3). Among MDR isolates,79.54 % isolates were moderate biofilm producers, and 43.54% of non-MDR isolates were weak biofilm producers showing a statistically significant association between MDR isolates and biofilm production (Table 4).

Identification of genes by PCR assay. The phenotypically identified isolates were further confirmed as *E. coli* by the presence of the *uid* A gene. The other virulence genes identified were *hly A, pap C* and *fim H* (Fig. 2). The *hly A* gene was positive in 20.8% of the isolates, *fim H* in 80.2%, and *pap C* in 51.9% of the isolates (Table 3).

Comparison of resistance pattern and virulence genes. The frequency of resistance to amoxycillin/clavulanate, gentamicin, amikacin, cefotaxime, cefepime, ceftazidime, ciprofloxacin, nitrofurantoin, cotrimoxazole, meropenem and piperacillin/tazobactam did not vary significantly by presence or absence of the *hly A* and *fim H* genes ($p>0.05$). The comparison of AST patterns for the isolates with *pap C* gene did not vary significantly except for cefotaxime. The isolates that did not have *pap C* gene showed more sensitivity to cefotaxime, which was found to be statistically significant ($p<0.05$) (Table 5).

DISCUSSION

One of the most common bacterial infections affecting 150 million people worldwide each year globally is urinary tract infections (UTIs). Uropathogenic *E. coli* is one of the most common causative agents of uncomplicated UTI. This may be due to the adaptability of UPEC to the harsh environment of the urinary tract with flexible and broad metabolic capabilities (1).

The prevalence rate of infection in our study was 10%, which is low compared to other studies conducted in India where a higher infection rate like 34.5% and 36.68% were observed (16). This could be due to the small sample size and different demographics conditions of the study area. Our study also proved that the most common bacterial uropathogen was *E. coli* which accounted for 59% of UTI. This finding was consistent with previous literature, which demonstrated that *E. coli* is the common cause of UTI, both in community-acquired and hospital-acquired cases (2, 3).

The mean age of the patients in this study was 49.7 years, with a standard deviation of 17.1 years. The age distribution showed that UTIs were prevalent in all age groups but were particularly common in individuals aged 41 to 60 (42.5%). This finding is consistent with previous studies such as the one

Table 2. Baseline characteristics of patients with urinary tract infections

		Number (N = 106) (n)	Percent (%)
Age (in years) Mean (SD)		49.7 (17.1)	
Age (in years)	<20	3	2.8
	21 to 40	29	27.4
	41 to 60	45	42.5
	61 to 80	26	24.5
	>81	3	2.8
Gender	Female	74	69.8
	Male	32	30.2
Underlying diseases	Diabetes mellitus	31	29.2
	Chronic kidney disease	7	6.6
	Carcinoma bladder	1	0.9
	Carcinoma cervix	1	0.9
	Hypothyroidism	1	0.9
	Total	41	38.6
Presenting symptoms	Fever	64	60.4
	Chills and rigor	16	15.1
	Loin and groin pain	39	36.8
	Polyuria	29	27.4
	Oliguria	2	1.9
	Pain during micturition	41	38.7
	Haematuria	7	6.6
	Others (vomiting, headache)	3	2.8
Predisposing factors	History of instrumentation	7	6.6
	History of renal calculi	6	5.7
	Uterine prolapse	2	1.8
	Pregnancy	6	5.6
	Obstructive uropathy	3	2.8
	Post LSCS	4	3.7
	Post TURP	1	0.9
	BPH	5	4.7

SD, Standard deviation; BPH, Benign prostatic hypertrophy; LSCS, Lower segment cesarean section; TURP, Transurethral resection of the prostate

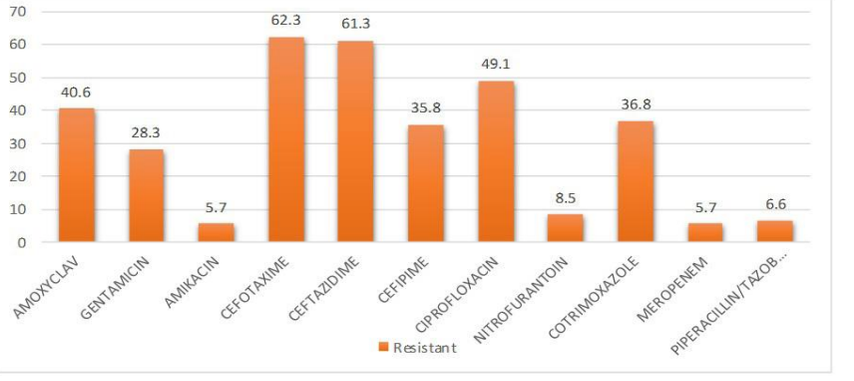

Fig. 1. Chart showing resistance pattern among *E. coli* isolates

Table 3. Distribution of *E. coli*, by phenotypic and genotypic characteristics

		Number (N = 106) (n)	Percent (%)
Phenotypic characteristics			
Haemagglutination	MRHA	32	30.2
	MSHA	11	10.4
	No agglutination	63	59.4
Hemolysin production	Beta hemolytic	19	17.9
	Non-hemolytic	87	82.1
Biofilm production	Weak	32	30.2
	Moderate	69	65.1
	Strong	5	4.7
ESBL production			
ESBL production	ESBL present	49	46.3
	ESBL absent	57	53.7
Genotypic characteristics			
Virulence genes	*hly A* gene	22	20.8
	fim H	85	80.2
	pap C	55	51.9

MRHA, Mannose-resistant haemagglutinins; MSHA, Mannose-sensitive haemagglutinins; ESBL, Extended-spectrum beta-lactamases

Table 4. Frequency of biofilm formation among MDR and non-MDR isolates

Biofilm forming capacity	**Total UPEC isolates N=106 n (%)**	**MDR isolates N=44 n (%)**	**Non-MDR isolates N=62 n (%)**	
Negative	0 (0)	0 (0)	0 (0)	P value
Weak	32 (30.2)	5 (11.36)	27 (43.54)	0.006*
Moderate	69 (65.1)	35 (79.54)	34 (54.83)	
Strong	5 (4.7)	4 (9.09)	1 (1.61)	

*Statistically significant at $p<0.05$

for Odoki et al. (17), which reported that young and middle-aged adults are at higher risk for UTIs due to factors such as hormonal changes, sexual activity and the presence of underlying diseases (5). There was a higher prevalence of UTIs in women as compared to men. This may be due to the female urethra being short and wide and structurally less effective in preventing bacterial entry from commensal flora. Moreover, with *E. coli* being part of normal flora in the large intestine, it can easily access the urinary tract via fecal contamination (18). Another study by Pardesi et al. (19) also demonstrated that female to male ratio was 2:1 for UTI prevalence.

Underlying medical conditions were present in one-third of the study participants, with diabetes mellitus being the most common. The association between diabetes and increased susceptibility to UTI is well-established, due to factors such as impaired immune response, glycosuria and neurogenic bladder dysfunction. In addition, the most prevalent infection among diabetes patients is also UTIs. Our findings corroborate those reported by Ahmed et al. (20). Other significant underlying conditions included chronic kidney disease and immunocompromising conditions such as carcinomas as discussed in the study conducted by Hyun et al. (5). They documented similar findings and noted that these conditions may also contribute to urinary tract infections. The

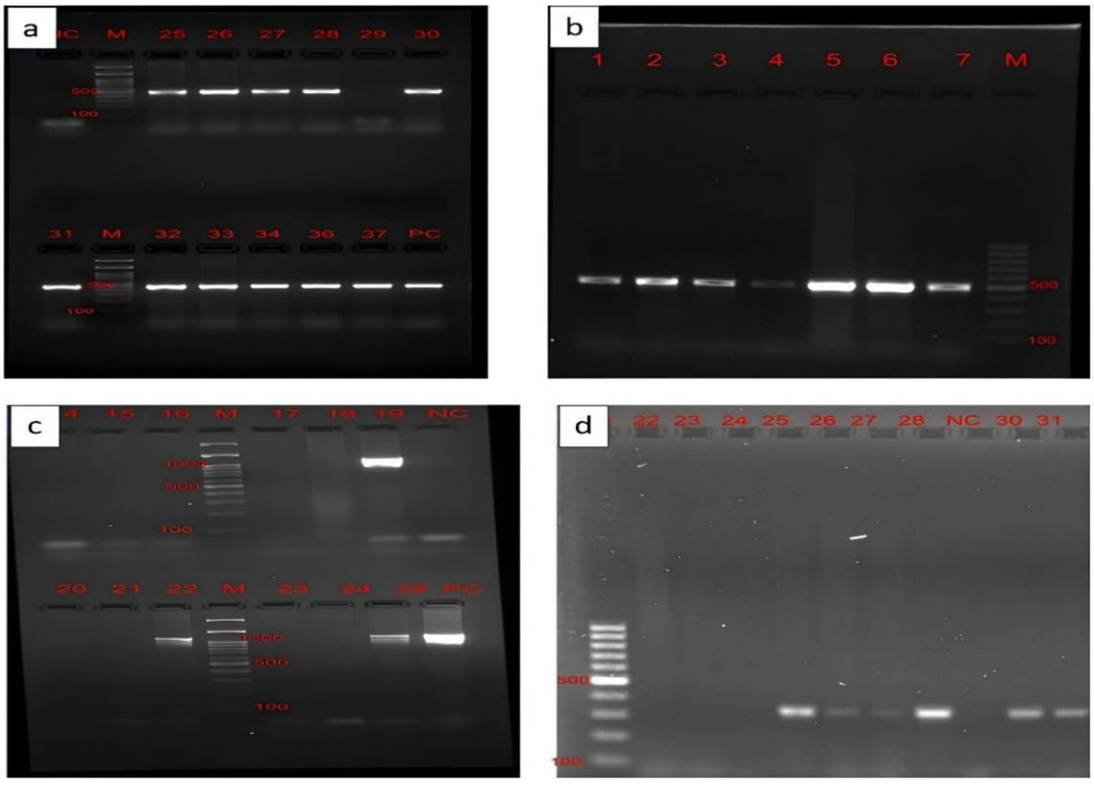

Fig. 2. Gene identification by PCR: a-*uid A* (486 bp); b-*fim H* gene (508 bp); c-*hly A* gene (1177 bp); d-*pap C* gene (328 bp)

Table 5. Comparison of resistance pattern and the virulence genes studied among *E. coli* isolates

Antibiotics	*hly A* gene		P value	*fim H* gene		P value	*pap C* gene		P value
	Present N = 22 n (%)	Absent N = 84 n (%)		Present N = 85 n (%)	Absent N = 21 n (%)		Present N = 55 n (%)	Absent N = 51 n (%)	
Amoxycillin/clavulanate (20/10 mcg)	9 (40.9)	34 (40.5)	0.971	33 (38.8)	10	0.462	25 (45.5)	18 (35.3)	0.287
Gentamicin (10 mcg)	6 (27.3)	24 (28.6)	0.904	25 (29.4)	5 (23.8)	0.610	17 (30.9)	13 (25.5)	0.536
Amikacin (30 mcg)	0 (0.0)	6 (7.1)	0.197	6 (7.1)	0 (0.0)	0.210	3 (5.5)	3 (5.9)	0.924
Cefotaxime (30 mcg)	11 (50.0)	55 (65.5)	0.182	53 (62.4)	13	0.970	39 (70.9)	27 (52.9)	0.044*
Cefepime (30 mcg)	7 (31.8)	31 (36.9)	0.658	30 (35.3)	8 (38.1)	0.811	22 (40.0)	16 (31.4)	0.355
Ceftazidime (30 mcg)	11 (50.0)	54 (64.3)	0.221	52 (61.2)	13	0.951	38 (69.1)	27 (52.9)	0.088
Ciprofloxacin (5 mcg)	7 (31.8)	45 (53.6)	0.069	41 (48.2)	11	0.734	26 (47.3)	26 (51.0)	0.703
Nitrofurantoin (300 mcg)	2 (9.1)	7 (8.3)	0.910	9 (10.6)	0 (0.0)	0.119	5 (9.1)	4 (7.8)	0.818
Cotrimoxazole (1.25/23.75 mcg)	8 (36.4)	31 (36.9)	0.963	28 (32.9)	11	0.098	19 (34.5)	20 (39.2)	0.618
Meropenem (10 mcg)	0 (0.0)	6 (7.1)	0.197	5 (5.9)	1 (4.8)	0.842	3 (5.5)	3 (5.9)	0.924
Piperacillin/Tazobactam (100/10 mcg)	0 (0.0)	7 (8.3)	0.161	5 (5.9)	2 (9.5)	0.547	5 (9.1)	2 (3.9)	0.284

*Statistically significant at $p<0.05$

most common presenting symptom in our study was fever, followed by dysuria, loin/groin pain and polyuria. These symptoms are characteristic of UTI and reflect the inflammatory response of the urinary tract to bacterial infection. The common symptoms such as fever, flank pain and dysuria were similar to other studies such as Paudel et al. (21) and Dilleban et al. (22). The study also highlighted the importance of significant history and precipitating factors in the development of UTIs, such as the history of instrumen-

tation/catheterization and renal calculi, as reported by Medina et al. (23).

The results of the antibiotic susceptibility testing indicate the lowest resistance to amikacin, nitrofurantoin, meropenem and piperacillin/tazobactam, with sensitivity rates exceeding 90%. This is consistent with previous studies by Kadry et al. and Alshaikh et al. showing less than 20% resistance to these drugs (24, 25). These antibiotics are highly reliable against UPEC and other *Enterobacteriaceae* due to their broad-spectrum activity and resistance to common resistance mechanisms. Our current study showed a high prevalence of extended-spectrum beta-lactamases (ESBL) production in 46.3% of isolates and this signifies a significant resistance mechanism that compromises the efficacy of beta-lactam antibiotics. Ehsan et al. (11) in their study proved that 48% of isolates were ESBL producers and a similar finding was reported by Shaikh et al. (12).

About 30.2% of the isolates exhibited mannose-resistant haemagglutination indicating the presence of P fimbriae which are associated with upper urinary tract infections and are key virulence factors in UPEC, and 10.4% exhibited mannose-sensitive haemagglutination indicating the presence of type 1 fimbriae which are crucial for initial colonization of the bladder epithelium. This suggests that a significant proportion of UPEC isolates possess adhesive properties that facilitate colonization and persistence in the urinary tract. Hemolysin production, a marker of virulence, was detected in 17.9% of the isolates in our study, with the majority being non-hemolytic (82.1%). Our findings are consistent with the study by Tabasi et al. for hemolysin production (36%) (26). However, MSHA (60.2%) was more frequently identified phenotypically compared to MRHA (37.2%) in the study by Tabasi et al. which was different from our study. This could be due to the difference in the expression of virulence properties in the study population. However, findings similar to our study were reported by Naveen et al. (13) documenting that hemolysin, particularly alpha-hemolysin, contributes to tissue damage and immune evasion, facilitating the progression of infection.

A significant proportion of UPEC isolates exhibited moderate biofilm production (65.1%), followed by weak (30.2%) and strong (4.7%) biofilm production in our study. A significant association was noted between the MDR isolates and biofilm producers. This distribution points out the ability of UPEC to adhere and form biofilms on urinary tract surfaces, contributing to the chronicity and recurrence of urinary tract infections (UTIs). Similar findings were also noted by Castillo et al. in community-acquired UTIs (15).

The *hlyA* gene, encoding for hemolysin, was present in 20.8% of the isolates. This was similar to the study by Zeb et al. (27). The *fimH* gene, encoding type 1 fimbriae, and the *papC* gene, encoding P fimbriae, were present in 80.2% and 51.9% of the isolates, respectively. Basu et al. (28), and Chakraborty et al. (2). also concluded similar findings. These virulence genes play crucial roles in the pathogenesis of UTIs, promoting bacterial adherence to host cells, invasion of tissues and evasion of immune responses.

The correlation between virulence genes and antimicrobial resistance is uncertain and also depends on the strain's interaction between the resistance determinant and the phylogenetic group of the strain. The results of our current study indicate that the presence or absence of the *hlyA, fimH,* or *papC* genes did not significantly influence the antibiotic susceptibility profiles of the UPEC isolates for a range of antibiotics tested. In contrast, Radera et al. (29). found a significant association between the presence of *fim H* and MDR *E. coli.* Although the *fim H* gene was present in 80% of the isolates in our study, its association with the AST pattern was not significant.

Interestingly, the presence of the *papC* gene was found to be significantly associated with reduced susceptibility to cefotaxime, a third-generation cephalosporin antibiotic. Specifically, a lower proportion of the isolates with the *papC* gene were sensitive to cefotaxime compared to isolates without the gene in the current study. This association highlights a potential link between P fimbriae-mediated adhesion and resistance to certain antibiotics, particularly cephalosporins. While the exact mechanism underlying this association requires further investigation, the expression of P fimbriae may confer selective advantages, including enhanced adherence and colonization, which could influence the response to antibiotic therapy.

CONCLUSION

The findings of the present study highlight the prevalence of virulence factors and their role in an-

tibiotic resistance in the study population. It emphasizes the importance of empirical antibiotic therapy based on local antibiogram data, targeted treatment after culture, sensitivity, and tailored regimens to prevent multidrug-resistant strains, thus improving UTI management and treatment efficacy.

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