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Original Article

Virulence Factor Profiles of Uropathogenic *Escherichia coli* in Community-Acquired and Health-Care Associated Urinary Tract Infections

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ABSTRACT

Background: Urinary tract infections are among the most common bacterial infections worldwide, with most cases caused by a single bacterium, uropathogenic *Escherichia coli* (*E. coli*). The study aimed to compare virulence factor patterns and antibiotic resistance profiles of Uropathogenic *E. coli* (UPEC) isolates from community-acquired and healthcare-associated UTIs. **Methods & Materials:** This cross-sectional analytical study was carried out at Sir Salimullah Medical College, Dhaka, Bangladesh from January to December 2020, including a total of 164 urine specimens collected from patients with clinically suspected UTI. Group I consisted of 82 outpatients with CA-UTI, and Group II consisted of 82 inpatients with HA-UTI (catheterized and non-catheterized subgroups). The *E. coli* isolates were tested for virulence factors such as haemolysin production, haemagglutination (mannose-sensitive and mannose-resistant), serum resistance, gelatinase production, and fimH gene detection by PCR. Antimicrobial susceptibility test was performed by the Kirby-Bauer disc diffusion method. **Results:** *E. coli* was isolated from 65.9% of CA-UTI cases compared to 46.3% of HA-UTI cases. Virulence factors were significantly more prevalent in CA-UTI isolates: haemolysin production (22% vs 3.7%), serum resistance (20.7% vs 6.1%), mannose-resistant haemagglutination (13.4% vs 2.4%), and fimH gene (56.1% vs 40.2%). Multiple virulence factors (≥3 factors) were observed in 25.6% of CA-UTI isolates versus 7.3% in HA-UTI. Carbapenems demonstrated the highest sensitivity rates (meropenem 61% and imipenem 58.5% in CA-UTI), while high resistance was noted to cephalosporins and amoxiclav. **Conclusion:** Community-acquired strains of UPEC showed a significantly higher expression of virulence factors than their healthcare-associated counterparts, thus indicating different pathogenic mechanisms. The correlation between virulence profiles and the source of infection may have implications for both therapeutic strategies and infection control measures.

Keywords: *Escherichia coli, Uropathogenic, Urinary tract infections.*

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INTRODUCTION

Urinary tract infections pose a significant public health burden, with an estimated 150 million cases occurring annually worldwide and standing out as one of the most common indications for antibiotic prescription [1]. Uropathogenic *E. coli* is responsible for 70-95% of community-acquired UTIs and for 50-65% of healthcare-associated ones, thus representing the predominant uropathogen across diverse clinical settings [2]. The clinical spectrum of UTI encompasses conditions that range from uncomplicated cystitis to severe pyelonephritis and urosepsis, while management complexity increases owing to rising antimicrobial resistance and variability in bacterial virulence attributes [3]. This burden is further increased in low- and middle-income countries due to limitations in diagnostics, empirical use of antibiotics, and high rates of catheter use, all of which accelerate the emergence of drug-resistant strains and complicate patient outcomes. The pathogenesis of UPEC infections is mediated through a complex array of virulence factors, which enable colonization, invasion, and persistence within the urinary tract [4]. These include adhesins like type 1 fimbriae, encoded by the fimH gene, and P fimbriae; toxins like haemolysin; iron acquisition systems; capsular polysaccharides that confer serum resistance; and various enzymes such as gelatinase [5]. Type 1 fimbriae allow bacteria to attach to uroepithelial cells via mannose-sensitive interactions, while P fimbriae mediate a mannose-resistant tropism to kidney tissue, both of these being important for establishing ascending infection [6]. The pore-forming toxin haemolysin causes tissue injury and promotes bacterial dissemination, while serum resistance mechanisms confer protection against complement-mediated bacterial killing in the bloodstream, a factor particularly pertinent to life-threatening UTI cases [7]. These virulence elements work in concert, enabling UPEC to efficiently establish infection despite host immune defenses. It is emerging that virulence factor profiles are very different between community-acquired and healthcare-associated UTIs, impacting on the severity of disease, the outcome of treatments, and epidemiological surveillance [8]. Community-acquired UPEC strains usually have a full virulence repertoire, allowing infection of an anatomically normal urinary tract, while healthcare-acquired strains depend more on host compromise and instrumentation than on intrinsic virulence mechanisms [9]. This is particularly relevant in the case of catheter-associated UTI, where biofilm formation, frequent exposure to antibiotics, and extended urinary catheterization do provide ecological niches different from those of non-catheterized infections [10]. These differences mold the behavior of the pathogen, its antimicrobial susceptibility, and the presence of persistence or recurrence. In Bangladesh, UTIs pose an important clinical problem, data on virulence determinants of circulating UPEC strains are scant, and information regarding any differences between community and hospital settings is particularly limited [11]. The few local studies have focused primarily on antimicrobial resistance patterns, with limited investigation of the interplay between resistance patterns and virulence traits. Information on these virulence profiles is critical for the forecasting of disease progress, guiding

empirical therapy, and formulating targeted prevention strategies. A more precise view of the differences between strains can support evidence-based decisions on catheter care, antimicrobial stewardship, and infection control practices. Therefore, this study aimed to characterize and compare the virulence factor profiles of UPEC isolates from community-acquired versus healthcare-associated UTIs and to assess their antimicrobial susceptibility patterns in a tertiary care setting in Dhaka, Bangladesh.

METHODS & MATERIALS

This cross-sectional analytical study was conducted in the Department of Microbiology at Sir Salimullah Medical College, Dhaka, Bangladesh from January to December 2020, enrolling patients of any age and sex presenting with clinically suspected urinary tract infections at Mitford Hospital. The study population comprised two groups: outpatients with community-acquired UTI (Group I) and inpatients with healthcare-associated UTI (Group II), the latter further divided into catheterized and non-catheterized subgroups. Inclusion criteria for Group I required a clinically diagnosed UTI without hospitalization within the preceding 28 days, while Group II participants were hospitalized for at least 72 hours (non-catheterized) or catheterized for a minimum of 48 hours, with relevant urinary symptoms. Patients on antibiotics or those who had taken antibiotics within 14 days were excluded. Purposive sampling yielded 82 samples per group, totaling 164 urine specimens. Data were collected using a structured questionnaire capturing demographic and clinical variables. Midstream urine was collected aseptically from non-catheterized patients, whereas catheterized samples were drawn using sterile technique proximal to the catheter-urobag junction. All samples were cultured on MacConkey and blood agar, followed by incubation at 37°C for 24 hours [12]. Bacterial isolates were identified through colony morphology, Gram stain, and standard biochemical tests. Antimicrobial susceptibility testing was performed by Kirby-Bauer disc diffusion on Mueller-Hinton agar in accordance with CLSI 2019 guidelines, using *E. coli* ATCC 25922 as the control strain [13,14]. Identified *E. coli* isolates were preserved on Mueller-Hinton agar slants and evaluated for virulence determinants, including haemolysin production, haemagglutination (mannose-sensitive and mannose-resistant), serum resistance, and gelatinase production using standard procedures. The fimH gene was detected by conventional PCR following genomic DNA extraction, primer preparation, amplification, and agarose gel electrophoresis [15]. Data were entered and analyzed using SPSS version 26.

RESULTS

Table I shows that the positivity rate of culture was 82.9% in Group I (community-acquired UTI) and 68.3% in Group II (healthcare-associated UTI). Isolation rates for *E. coli* were significantly different between groups (65.9% in community-acquired cases vs 46.3% in healthcare-associated cases). Culture negativity was more common among healthcare-associated infection cases (31.7%) than community infections (17.1%).

Table I
Growth Culture Results of UTI Cases among the study population.

Growth	Group-I (n=82)	Group-II (n=82)
	n (%)	n (%)
Culture Positive	68 (82.9%)	56 (68.3%)
Culture Negative	14 (17.1%)	26 (31.7%)
E. coli	54 (65.9%)	38 (46.3%)

Table II shows that *E. coli* predominated in both groups but had a higher prevalence in community-acquired UTI, 65.9% compared with healthcare-associated UTI, 46.3%. Healthcare-associated infections, however, manifested greater bacterial diversity, including increased proportions of *Klebsiella* spp., 12.2% versus 8.5%, and *Pseudomonas* spp., 8.5% versus 3.7%. Community isolates included a few unique organisms, such as *Citrobacter*, *Serratia*, and *Staphylococcus aureus*, although the overall distribution reflected typical patterns of uropathogenic species in different acquisition settings.

Table II
Distribution of Bacterial Isolates of UTI Patients.

Bacterial Isolates	Group-I (n=82) (n) (%)	Group-II (n=82) (n) (%)
E. coli	54 (65.9%)	38 (46.3%)
Klebsiella spp.	07 (8.5%)	10 (12.2%)
Pseudomonas spp.	03 (3.7%)	07 (8.5%)
Citrobacter spp.	01(1.2%)	00 (0%)
Proteus spp.	01(1.2%)	01 (1.2%)
Serratia spp.	01(1.2%)	00 (0%)
Staphylococcus aureus	01(1.2%)	00 (0%)

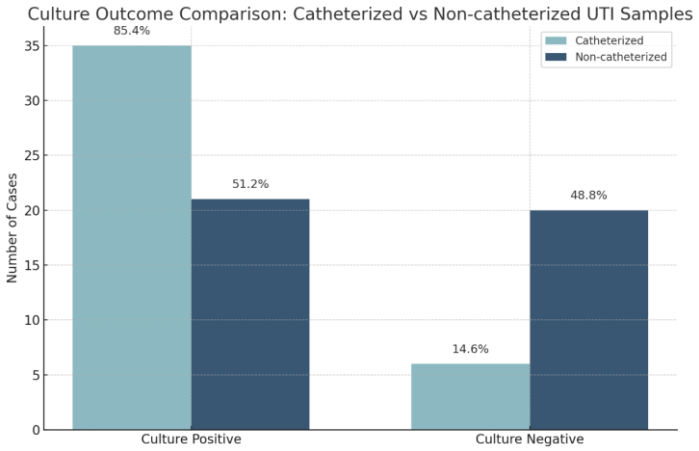

Figure 1 Culture Outcome Comparison between Catheterized and Non-Catheterized UTI Samples.

Figure 1 illustrates bacterial distribution between catheterized and non-catheterized samples within healthcare-associated UTI cases. *E. coli* had a marked predominance in catheterized samples, 65.9% versus 26.8% in non-catheterized samples. By contrast, the non-*E. coli* organisms, especially the species *Pseudomonas* and *Klebsiella*, were more common in non-catheterized samples. Such a distribution pattern reflects the differential bacterial ecology related to catheterization versus spontaneous healthcare-acquired infections.

Table III demonstrates that *E. coli* isolations among healthcare-associated infections in catheterized patients were 65.9%, significantly higher than that found among non-catheterized patients at 26.8%. *Pseudomonas* spp. had comparable prevalence between the subgroups, 12.2% catheterized versus 14.6% non-catheterized. *Klebsiella* spp. was lower in the catheterized population compared to the non-catheterized at 4.9% and 9.8%, respectively. This distribution would seem to suggest that the catheterization selects for *E. coli* colonization despite the hospital environment.

Table III
Distribution of Bacterial Isolates among Catheterized and Non-Catheterized UTI Patients.

Isolates	Growth from catheterized urine (n=41)	Growth from non-catheterized urine (n=41)
	n (%)	n (%)
Escherichia coli	27 (65.9%)	11 (26.8%)
Pseudomonas spp.	05 (12.2%)	06 (14.6%)
Klebsiella spp.	02 (4.9%)	04 (9.8%)
Proteus spp.	01 (2.4%)	00 (0%)

Table IV shows that carbapenems had the highest susceptibility rates with meropenem at 61% CA-UTI, 39% HA-UTI, and imipenem at 58.5% CA-UTI, 37.8% HA-UTI, showing superior efficacy. Resistance to cephalosporins was significant in both groups, with ceftazidime resistance at 37.8% in the community and 30.9% in healthcare settings. Fluoroquinolone susceptibility was moderate, with levofloxacin performing better (42.7% CA-UTI) than ciprofloxacin (31.7% CA-UTI). Nitrofurantoin maintained reasonable sensitivity at 50% CA-UTI and 29.3% HA-UTI, while amoxicillin showed high resistance rates of over 36% in both groups.

Table IV
Antimicrobial Susceptibility Patterns of Uropathogens in Community-Acquired and Health-Care Associated UTI Cases.

Antimicrobial Agents	Group-I (n=82)			Group-II (n=82)		
	Sensitive	Intermediate	Resistant	Sensitive	Intermediate	Resistant
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
Mecillinam	29 (35.5%)	3 (3.7%)	22 (26.8%)	22 (26.8%)	3 (3.7%)	13 (15.9%)
Ceftazidime	20 (24.4%)	3 (3.7%)	31 (37.8%)	9 (10%)	4 (4.9%)	25 (30.9%)
Cefuroxime	15 (18.3%)	3 (3.7%)	36 (43.9%)	24 (29.3%)	21 (25.6%)	13 (15.9%)
Ceftriaxone	23 (28%)	2 (2.4%)	29 (35.4%)	10 (12.2%)	3 (3.7%)	25 (30.9%)
Cefixime	18 (22%)	2 (2.4%)	34 (41.5%)	11 (13.4%)	3 (3.7%)	24 (29.3%)
Meropenem	50 (61%)	0 (0%)	4 (4.9%)	32 (39%)	0 (0%)	6 (7.3%)
Imipenem	48 (58.5%)	1 (1.2%)	5 (6.1%)	31 (37.8%)	0 (0%)	7 (8.5%)
Amoxiclav	14 (17.1%)	2 (2.4%)	38 (46.3%)	5 (6.1%)	3 (3.7%)	30 (36.6%)
Ciprofloxacin	26 (31.7%)	3 (3.7%)	25 (30.9%)	16 (19.5%)	2 (2.4%)	20 (14.4%)
Levofloxacin	35 (42.7%)	3 (3.7%)	16 (19.5%)	17 (20.7%)	2 (2.4%)	19 (23.2%)
Nalidixic acid	12 (14.6%)	4 (4.9%)	38 (46.3%)	15 (18.3%)	4 (4.9%)	19 (23.2%)
Gentamicin	40 (48.9%)	1 (1.2%)	13 (15.9%)	17 (20.7%)	3 (3.7%)	18 (22%)
Amikacin	47 (57.3%)	0 (0%)	7 (8.5%)	32 (39%)	0 (0%)	6 (7.3%)
Co-trimoxazole	26 (31.7%)	2 (2.4%)	26 (31.7%)	16 (19.5%)	3 (3.7%)	19 (23.2%)
Nitrofurantoin	41 (50%)	0 (0%)	13 (15.9%)	24 (29.3%)	1 (1.2%)	13 (15.9%)
Aztreonam	16 (19.5%)	3 (3.7%)	35 (42.9%)	22 (26.8%)	3 (3.7%)	13 (15.9%)

Table V denotes that within the healthcare-associated infections, the samples from catheterized patients showed broader antibiotic resistance, with ceftazidime resistance at 46.3%, compared to 14.6% in the non-catheterized ones. Carbapenem sensitivity remained the highest in both subgroups (meropenem 53.7% catheterized vs 24.4% non-catheterized), but the absolute sensitivity was lower in catheterized patients. Amoxiclav demonstrated particularly high resistance in catheterized samples (51.2%), while nitrofurantoin maintained reasonable efficacy (43.9% sensitive in catheterized patients), suggesting its potential utility in catheter-associated infections.

Table V
Comparative Antimicrobial Susceptibility Patterns between Catheterized and Non-Catheterized UTI Samples.

Antimicrobial Agents	Catheterized urine (n=41)			Non-catheterized urine (n=41)		
	Sensitive	Intermediate	Resistant	Sensitive	Intermediate	Resistant
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
Mecillinam	15 (36.9%)	2 (4.9%)	10 (24.4%)	7 (17%)	1 (2.4%)	3 (7.3%)
Ceftazidime	6 (14.6%)	2 (4.9%)	19 (46.3%)	3 (7.3%)	2 (4.9%)	6 (14.6%)
Cefuroxime	16 (39%)	0 (0%)	11 (26.8%)	8 (19.5%)	1 (2.4%)	2 (4.9%)
Ceftriaxone	7 (17%)	2 (4.9%)	18 (43.9%)	3 (7.3%)	1 (2.4%)	7 (17%)
Cefixime	8 (19.5%)	2 (4.9%)	17 (41.5%)	3 (7.3%)	1 (2.4%)	7 (17%)
Meropenem	22 (53.7%)	0 (0%)	5 (12.2%)	10 (24.4%)	0 (0%)	1 (2.4%)
Imipenem	22 (53.7%)	0 (0%)	5 (12.2%)	9 (22%)	0 (0%)	2 (4.9%)
Amoxiclav	4 (9.8%)	2 (4.9%)	21 (51.2%)	1 (2.4%)	1 (2.4%)	9 (22%)
Ciprofloxacin	12 (29.7%)	2 (4.9%)	13 (31.7%)	4 (9.8%)	0 (0%)	7 (17%)
Levofloxacin	13 (31.7%)	2 (4.9%)	12 (29.7%)	4 (9.8%)	1 (2.4%)	6 (14.6%)
Nalidixic acid	10 (24.4%)	3 (7.3%)	14 (34.1%)	5 (12.2%)	1 (2.4%)	5 (12.2%)
Gentamicin	14 (34.1%)	2 (4.9%)	11 (26.8%)	3 (7.3%)	1 (2.4%)	7 (17%)
Amikacin	22 (53.7%)	0 (0%)	5 (12.2%)	10 (24.4%)	0 (0%)	1 (2.4%)
Co-trimoxazole	11 (26.8%)	2 (4.9%)	14 (34.1%)	5 (12.2%)	1 (2.4%)	5 (12.2%)
Nitrofurantoin	18 (43.9%)	1 (2.4%)	8 (19.5%)	6 (14.6%)	0 (0%)	5 (12.2%)
Aztreonam	15 (36.9%)	2 (4.9%)	10 (24.4%)	7 (17%)	1 (2.4%)	3 (7.3%)

Table VI shows that community-acquired *E. coli* isolates showed significantly higher rates of virulence factors such as haemolysin production (22% vs 3.7%), serum resistance (20.7% vs 6.1%), and mannose-resistant haemagglutination (13.4% vs 2.4%). The fimH gene was present in 56.1% of strains from the community versus 40.2% from healthcare. As many as 25.6% of community strains but only 7.3% of healthcare strains expressed multiple virulence factors ≥3, indicating that community-acquired UPEC possess more extensive virulence repertoires.

Table VI
Comparative analysis of virulence factor profiles of *E. coli.*

Variable	Category	**Group-I (n=82)** n (%)	**Group-II (n=82)** n (%)
Virulence Factors	Haemolysin production	18 (22%)	3 (3.7%)
	Serum resistance	17 (20.7%)	5 (6.1%)
	Gelatinase production	3 (3.7%)	0 (0%)
	fimH gene	46 (56.1%)	33 (40.2%)
Haemagglutination	Mannose-Sensitive Haemagglutination	38 (46.3%)	26 (31.7%)
	Mannose-Resistant Haemagglutination	11 (13.4%)	2 (2.4%)
Virulence Factors	All VF	1 (1.2%)	0 (0%)
	Any 4 VF	10 (12.2%)	1 (1.2%)
	Any 3 VF	10 (12.2%)	5 (6.1%)
	Any 2 VF	26 (31.7%)	22 (26.8%)
	Any 1 VF	6 (7.3%)	6 (7.3%)
	No Virulence Factors	1 (1.2%)	4 (4.9%)

Table VII unveils the prevalence of the fimH gene was higher in catheterized samples among healthcare-associated infections than among non-catheterized samples, at 58.5% compared to 22%, indicating the importance of adhesin-mediated colonization in catheter-associated UTI. Mannose-sensitive haemagglutination was highly increased in catheterized samples (51.2% vs 12.2%), reflecting type 1 fimbriae expression. Haemolysin production and serum resistance showed minimal differences between subgroups and generally remained low, showing that healthcare-associated strains depend less on classical virulence mechanisms independent of catheterization.

Table VII
Virulence Factors in Catheterized vs Non-Catheterized Samples.

Virulence Factors	Catheterized urine (n=41)	Non-catheterized (n=41)
	n (%)	n (%)
Haemolysin production	1 (2.4%)	2 (4.9%)
Haemagglutination	-	-
Mannose-Sensitive Haemagglutination	21 (51.2%)	5 (12.2%)
Mannose-Resistant Haemagglutination	0 (0%)	2 (4.9%)
Serum resistance	3 (3.7%)	2 (4.9%)
Gelatinase production	0 (0%)	0 (0%)
fimH gene	24 (58.5%)	9 (22%)

DISCUSSION

This study demonstrates the differences in the virulence factor profiles between community-acquired and healthcare-associated uropathogenic *E. coli*, where community strains had significantly higher expression of classical virulence determinants. The differences observed reflect a fundamental divide in pathogenic strategy, wherein community-acquired UPEC must maintain complete virulence arsenals to initiate infection in otherwise immunocompetent hosts possessing an anatomically normal urinary tract, whereas healthcare-associated strains can leverage host compromise and instrumentation [16]. These data reinforce the notion that UPEC virulence is context-dependent, not absolute, driven by ecological pressures and by the host environment. In the community environment, only strains capable of adhesion, invasion, and evasion of host immunity are naturally selected. In contrast, the hospital setting, characterized by prolonged catheterization, antibiotic pressure, and host comorbidities, provides a selective environment for the predominance of less intrinsically virulent but highly resistant organisms. Infection rates of *E. coli* were higher in community-acquired UTI (65.9%) compared to healthcare-associated UTI (46.3%), consistent with Tandogdu et al. [17]. The higher bacterial diversity of healthcare-associated infections, particularly increased *Klebsiella* and *Pseudomonas* species, reflects nosocomial ecological pressures and selective antimicrobial exposure typical of the hospital environment [18]. This diversification has therapeutic implications, given that these opportunistic pathogens often exhibit enhanced antimicrobial resistance and distinct virulence mechanisms compared to community *E. coli* strains [19]. Their presence complicates empirical therapy and emphasizes the need for risk stratification based on the infection setting. The virulence factor analysis showed striking differences between infection sources. Haemolysin production, present in 22% of community-acquired isolates versus only 3.7% of healthcare-associated strains, is an important tissue-damaging toxin associated with severe disease manifestations such as pyelonephritis and bacteremia [20]. Similarly, serum resistance (a correlate of invasive disease potential) was threefold more prevalent among community strains (20.7% vs 6.1%), consistent with Sartor et al., who reported increased complement resistance among community UPEC [21]. These findings indicate that healthcare-associated strains may have experienced fitness cost trade-offs, losing metabolically expensive virulence factors in order to acquire antimicrobial resistance determinants [22]. Such trade-offs have been described as evolutionary responses to sustained antibiotic pressure and biofilm-associated growth. The presence of the fimH gene, encoding the adhesin component of type 1 fimbriae, was detected in 56.1% of community isolates and 40.2% in healthcare isolates, but notably higher expression was found in catheterized patients, at 58.5%. This paradox-lower overall prevalence in healthcare settings but highest expression in the catheterized subgroup-suggests that though the healthcare-associated non-catheterized UTIs may involve less virulent opportunistic strains, the catheter-associated infections still depend on adhesin-mediated biofilm formation for persistence [23]. This mechanism is further supported by the mannose-sensitive haemagglutination pattern, which shows 51.2% positivity in catheterized samples versus 12.2% in noncatheterized healthcare samples [24]. These findings thus support the central role of surface adhesins in the establishment of catheter biofilms as a key driver of chronic and recurrent infections. The trend for antimicrobial resistance was a cause of concern, with high rates of resistance to commonly used agents. Community-acquired infection rates of resistance to cephalosporins exceeded 35% for several agents, considerably higher than those reported for developed countries but consistent with reports by Shaifali et al. [25]. Carbapenem sensitivity remained relatively preserved at 61% for community infections with meropenem, although the 39% sensitivity in healthcare settings has raised concerns about emerging carbapenem resistance in hospital settings [26]. The inverse relationship between virulence factor expression and antimicrobial resistance provides evidence of evolutionary trade-offs in which healthcare-associated strains sacrifice virulence determinants for survival mechanisms [27]. This complicates infection management, as strains may be less virulent yet more difficult to eradicate. These findings have significant clinical implications. Empirical therapy for CA-UTI must be based on local resistance patterns, while being cognizant of the increased virulence potential of such strains, which may require more aggressive management in complicated infections. Management of HA-UTI, however, requires consideration of broader resistance profiles and the possibility of polymicrobial infection [28]. Distinct virulence profiles, as discussed above, support differential approaches to diagnosis and therapy based on the setting of acquisition of infection and emphasize the importance of effective infection control, antimicrobial stewardship, and catheter management. Enhancing routine diagnostic capability, including the introduction of virulence markers into infection surveillance programs, and strategies for empirical treatment, may make a significant difference in resource-constrained settings.

LIMITATIONS

This single-center cross-sectional design limits the generalizability. Further, the lack of molecular characterization of resistance genes limits the understanding of resistance mechanisms. The sample size is relatively small and may not reflect complete bacterial diversity; seasonal variations in pathogen distribution were also not assessed.

CONCLUSION

Community-acquired uropathogenic *E. coli* demonstrates considerably higher expression of virulence factors such as the production of haemolysin, serum resistance, and adhesin determinants, in comparison with healthcare-associated strains. This virulence-resistance dichotomy reflects distinct evolutionary pressures, with community strains needing comprehensive pathogenic mechanisms while healthcare strains prioritize antimicrobial survival. The findings provide a basis for tailored therapeutic strategies and infection control

measures depending on the setting of acquisition and catheterization status.

RECOMMENDATIONS

Whole-genome sequencing characterizing the virulence and resistance gene repertoire should be included in future studies. Prospective, multicenter studies should also correlate virulence profiles with clinical outcomes such as treatment failure and recurrence rates. Investigation of biofilm formation capacity and clonal relatedness would enhance the understanding of nosocomial transmission dynamics.

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CONFLICT OF INTEREST

None declared

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