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Antibiotic Resistance and Detection of the fimH Gene in E. coli Isolated from Urinary Tract Infection Patients in Surabaya, Indonesia

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ABSTRACT

Background: *E. coli is a pathogenic bacterium most closely associated with fatal infections worldwide, including uropathogenic E. coli (UPEC), which causes Urinary Tract Infections (UTIs). UPEC has a specific set of virulence genes, one of which is the type 1 pilus (fimH adhesin), which mediates bacterial adhesion to the bladder epithelium, resulting in bacterial growth and colonization. fimH also has potential as a vaccine target for UTI to reduce the high rate of multidrug resistance. The aim of this research is (1) to determine the resistance level of E. coli isolates from UTI patients to amoxicillin, ciprofloxacin, and gentamicin antibiotics, and (2) to evaluate the presence of the fimH gene encoding the adhesion factor in these isolates.*

Methods*: This study is an experimental laboratory study. The determination of antibiotic resistance levels to amoxicillin, ciprofloxacin, and gentamicin uses the CLSI's disc diffusion methods. The presence of the fimH gene (gene ID: 948847) was determined by Polymerase Chain Reaction (PCR).*

Results*: The tested UPEC showed the greatest resistance to amoxicillin (92%), followed by ciprofloxacin (88%) and gentamicin (56%). Additionally, molecular identification showed that 49 of 50 isolates (98%) were positive for fimH.*

Conclusion*: Escherichia coli isolated from patients with urinary tract infections showed the highest resistance to amoxicillin, followed by ciprofloxacin and gentamicin. At the same time, the fimH gene encoding the adhesion factor was also highly prevalent.*

INTRODUCTION

Escherichia coli is a pathogenic bacterium that has been extensively studied and is among the microorganisms most frequently associated with fatal infections worldwide [1]. *E. coli* is the most common cause of Gram-negative bacteremia, with urinary tract infections (UTIs) serving as the primary source of *E. coli* bacteremia in more than 50% of cases, and its incidence is increasing

in several regions [2]. In addition, *E. coli* is the dominant pathogen in both community-acquired and hospital-acquired infections [3]. UTIs represent a major public health concern, causing significant morbidity and, in vulnerable populations such as infant boys, older men, and women of all ages, even mortality. The global burden of UTIs continues to rise, with an estimated 404.6 million cases reported in 2019, a situation further exacerbated by the emergence of antimicrobial resistance (AMR) [4].

E. coli, the causative agent of UTI, belongs to the uropathogenic group (UPEC) with a specific set of virulence genes. The majority of UPEC virulence genes are found in the pathogenicity island (PAI), where many genes are transferred from other species. UPEC strains can utilize these genes to survive in the urinary tract of human hosts [5]. There are three different types of organelles that UPEC uses to perform irreversible attachment, including conjugative pili, curli fibers, and type 1 fimbriae. [6]. UPEC also possesses chaperone-usher pathway (CUP) pili, hair-like fibers with adhesin tips that bind to receptors with stereochemical specificity, mediating tropism, colonization, invasion, and biofilm formation. One subset of CUP pili is the type 1 pilus, which is most studied due to its crucial role in UTI pathogenesis. [7]. Type 1 pili, or the fim H adhesin, mediate bacterial attachment to several glycoproteins and non-glycosylated peptide epitopes in the bladder epithelium, resulting in bacterial internalization and the formation of intracellular bacterial communities [8]. Type 1 pili are long, thin filamentous appendages composed primarily of hundreds of FimA monomers encoded by the fimA gene. The tip of the type 1 pilus has *fimH* adhesin, which binds to mannosylated groups, including those found on urethral and bladder epithelial cells [9].

Previous studies have shown that type 1 fimbriae, through the *fimH* adhesin subunit located at the terminal end of the fimbriae, will bind urothelial uroplakin, facilitating biofilm formation during UTI [10]. Single-nucleotide polymorphism (SNP) studies have revealed three *fimH* alleles or variants (H30, H30-R, and H30-Rx) in UPEC clones [11] and thus can be used as a screening tool for epidemiological typing of UPEC. The key virulence factor *fimH* is a promising target for vaccines against UPEC through the immunogenicity of mRNA-based nanoparticle vaccines against UPEC [12]. In a phase 1A/1B clinical trial, the study demonstrated the potential of fimH-targeted therapy to prevent infection by two of the most common UTI pathogens. The effectiveness of *fimH* vaccination is related to the antibody response that inhibits *fimH* binding [13].

Several studies have shown the presence of UPEC resistance to antibiotics, including genomic investigation of ST167 as a multidrug-resistant UPEC clone associated with UTI [14]. The increasing prevalence of quinolone-resistant *E. coli* in urinary tract infections in Central Inner Mongolia, China, is the result of a combination of resistance gene mutations and strong biofilm-forming ability [15]. The global prevalence of nitrofurantoin-resistant UPEC isolates is quite high, with a higher prevalence in low- and middle-income countries (LMIC) [16].

This study was conducted to determine the antibiotic resistance of E. coli isolated from patients with UTI to amoxicillin, ciprofloxacin, and gentamicin, and to assess the expression of the fimH gene, which encodes an adhesion factor. The presence of virulence genes and high levels of antibiotic resistance make this a serious and challenging health problem. It is crucial to raise public awareness at both the individual and global levels of drug resistance, thereby necessitating the careful use of antibiotics.

METHODS

Bacterial isolates

This study was conducted on 50 UPEC isolates collected by researchers [17] and stored in the Gastroenteritis and Salmonellosis Laboratory, ITD Unair. The isolates were obtained from urine samples of patients with suspected UTIs treated at a Surabaya Regional General Hospital. The urine samples were cultured on EMB agar and then incubated overnight at 35°C. Isolated bacteria with colony counts exceeding 10^5 CFU/mL were considered UTI agents. Identified isolates were stored on semisolid nutrient agar for further processing.

Determination of resistance levels to the antibiotics amoxicillin, ciprofloxacin, gentamicin, and definition of MDR

After isolation and identification of *E. coli*, antibiotic resistance testing was performed using the disc diffusion method in accordance with the CLSI guidelines (2017). Inoculum density was standardized using the McFarland standard. Three antibiotics were used for this study: amoxicillin (AMX), ciprofloxacin (CIP), and gentamicin (GEN). After disc application and incubation at 37°C for 24 hours, each petri dish was examined for the presence of a confluent growth zone with a clear zone of inhibition. Isolates were identified as resistant or susceptible based on the zone-of-inhibition diameter (mm) against UPEC. Determination of sensitive, intermediate, and resistant categories according to CLSI guidelines (in mm units): For amoxicillin antibiotics: Sensitive: ≥ 11, Intermediate: 12-13, and Resistant: ≤ 14, For ciprofloxacin antibiotics: Sensitive: ≥ 21, Intermediate: 16-20, and Resistant: ≤ 15. For gentamicin antibiotics: Sensitive: ≥15, Intermediate: 13-14, and Resistant: ≤12. The antibiotics tested included amoxicillin (β-lactam), gentamicin (aminoglycoside), and ciprofloxacin (fluoroquinolone). Multidrug resistance (MDR) was defined based on the criteria proposed by Magiorakos et al [18]namely, resistance to at least one antimicrobial agent from three different antimicrobial classes. Isolates showing intermediate susceptibility were not classified as resistant for MDR determination.

DNA extraction, PCR amplification, and virulence factor detection

All of the UPEC isolates were then analyzed for the fimH gene. DNA extraction was performed using the NEXprep TM Cell/Tissue genomic DNA preparation kit. Each PCR reaction consists of 8,4 µL of DNA template, 0,8 µL of forward primer, 0,8 µL of reverse primer, and 10 µL

of PCR master mix (Nex[tm] Diagnostics). The DNA was then amplified by 35 successive cycles of denaturation at 95°C for 1 min, primer annealing at 53°C for 1 min, and DNA chain extension at 72°C for 1 min. Also, the initial denaturation was 95°C for 150s, and the final extension was 72°C for 5 min. The presence of DNA was confirmed by horizontal electrophoresis containing 1% Merck agarose gel in 0.5X Tris-Borate-EDTA buffer and stained with DNA-safe marker. The virulence gene fimH (357 base pairs, gene ID 948847) was detected using specific primers: F:CTACTCTGTTTCCTTTATGGCGA and R: GCAGGGATAGCTTTAACATTAACC (Macrogen Singapore). PCR products were loaded onto an agarose gel to visualize the amplified gene, with a 100 bp DNA marker to confirm the size of the fimH PCR amplicon in base pairs.

RESULTS

Resistance testing for amoxicillin, ciprofloxacin, and gentamicin antibiotics

UPEC isolates were tested for their antimicrobial susceptibility to three antibiotics using the disc diffusion method. The results of the UPEC antibiotic resistance test using the disc diffusion method, indicated by the presence of zones of inhibition, are shown in Figure 1. Isolates that produced clear zones of inhibition around several antibiotic discs were considered sensitive to those antibiotics. Conversely, isolates that did not produce zones of inhibition around the antibiotic discs were considered resistant to those antibiotics.

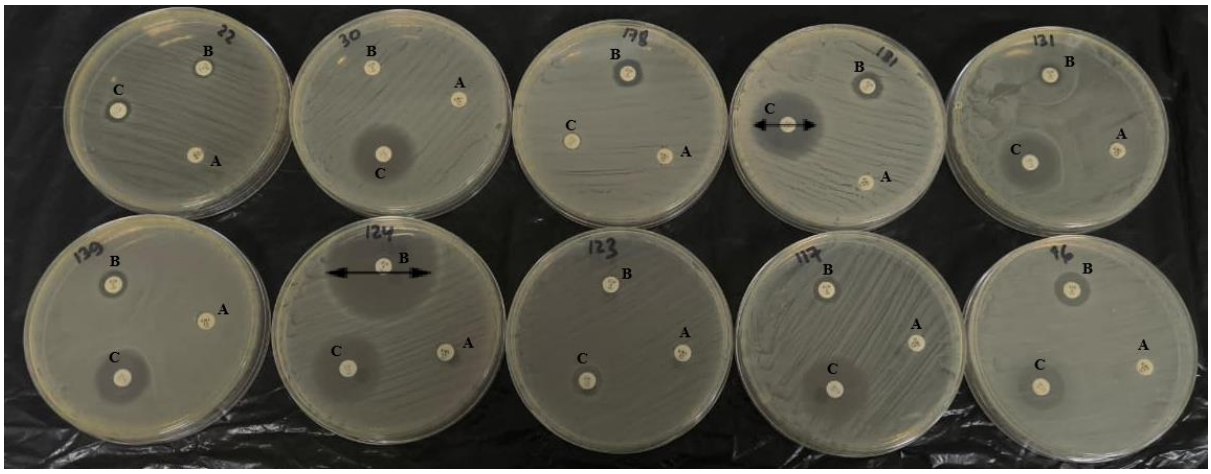

Figure 1. UPEC antibiotic resistance test using the disc diffusion method showing the inhibition zone (⟷): A. amoxicillin, B. ciprofloxacin, C. gentamicin

The UPEC resistance profile to amoxicillin, ciprofloxacin, and gentamicin antibiotics is shown in Table 1. All UPEC isolates tested showed that the resistant category had the largest percentage for amoxicillin, ciprofloxacin, and gentamicin. Classification of UPEC isolates based on multidrug resistance (MDR) is shown in Table 2. The data in Table 2 shows that UPEC MDR and non-MDR have the same percentage, namely 50%.

Table 1. UPEC resistance profile to the antibiotics amoxicillin, ciprofloxacin, and gentamicin

Antibiotics	Sensitive		Intermediate		Resistant	
	Number of samples	%	Number of samples	%	Number of samples	%
Amoxicillin	3	6	1	2	46	92
Ciprofloxacin	3	6	3	6	44	88
Gentamicin	13	26	9	18	28	56

Table 2 Classification of UPEC isolates based on multidrug resistance (MDR)

MDR Status	Isolate Codes	n (%)
MDR	1, 3, 22, 23, 62, 106, 109, 114, 116, 118, 123, 131, 133, 137, 139, 145, 152, 157, 158, 160, 167, 168, 170, 175, 178	25 (50%)
Non-MDR	6, 30, 35, 40, 46, 50, 52, 56, 59, 78, 93, 99, 113, 117, 124, 150, 154, 155, 156, 163, 169, 171, 172, 176, 181	25 (50%)
Total		50 (100%)

Detection of the FimH Virulence Gene in UPEC

The results of electrophoresis visualization of the fimH gene in UPEC bacteria isolated from UTI patients are shown in Figure 3. Molecular identification showed that 49 of 50 isolates (98%) were positive for fimH. The relationship between the presence of the fimH virulence gene and antibiotic susceptibility testing is shown in Table 3. Isolates that tested positive for the fimH gene showed a significant tendency to be resistant to all tested antibiotics (amoxicillin, ciprofloxacin, and gentamicin) compared with negative isolates.

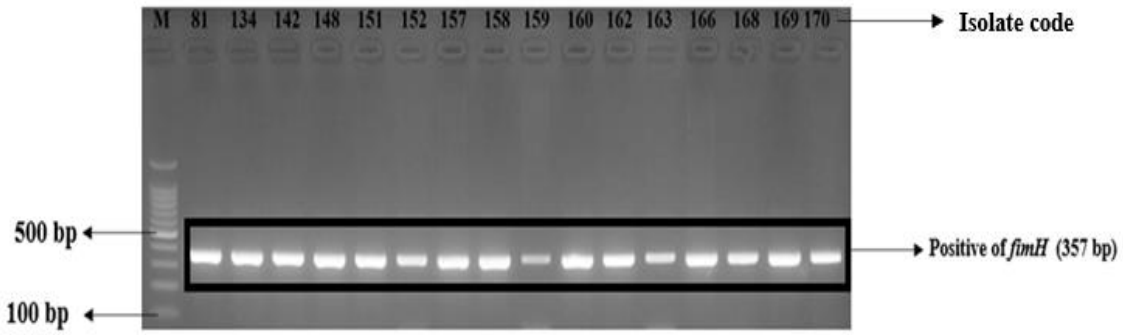

Figure 2. Results of electrophoresis visualization of the *fimH* gene in UPEC bacteria isolated from UTI patients.

Table 3. Relationship between the presence of the *fimH* virulence gene and antibiotic susceptibility testing

Antibiotics		*fimH*	
		Negative	Positive
Amoxicillin	Sensitive	0 (0%)	3 (6%)
	Intermediate	0 (0%)	1 (2%)
	Resistant	1 (2%)	45 (90%)
Ciprofloxacin	Sensitive	0 (0%)	3 (6%)
	Intermediate	0 (0%)	3 (6%)
	Resistant	1 (2 %)	43 (86 %)
Gentamicin	Sensitive	0 (0%)	13 (26%)
	Intermediate	1 (2%)	8 (16%)
	Resistant	0 (0%)	28 (56%)

DISCUSSION

In this study, we examined the antibiotic resistance of E. coli isolated from patients with UTI to amoxicillin, ciprofloxacin, and gentamicin, and determined the expression of the *fimH* gene, which encodes an adhesion factor. The antibiotics amoxicillin, ciprofloxacin, and gentamicin are frequently used to treat UTIs. In this study, UPEC isolates showed high resistance to amoxicillin (92%), followed by ciprofloxacin (88%) and gentamicin (56%). Several studies have also demonstrated high resistance in UPEC, including amoxicillin (53.3%) and cephalexin (53.3%). Moderate resistance was found to ampicillin/sulbactam (40.0%), ciprofloxacin (33.3%), and trimethoprim-sulfamethoxazole (33.3%). Lower levels of resistance were noted with most cephalosporins and β-lactam/β-lactamase inhibitor combinations [19]. Antibiograms of ESBL-producing isolates showed higher levels of resistance to cephalosporins compared to non-cephalosporin antibiotics. Low levels of resistance were also observed to aminoglycosides and carbapenems [20]. Inappropriate and excessive antibiotic use will increase *E. coli* resistance, so appropriate use of available antibiotics is essential. Furthermore, educational programs and regular monitoring of antimicrobial susceptibility are crucial to reduce antibiotic resistance [21]. Ciprofloxacin cannot be recommended as a first-line antibiotic for the treatment of UTI. However, this antibiotic is commonly used in empirical therapy. Our study showed ciprofloxacin resistance in UPEC isolates of 88%[22]

In this study, the tested UPEC bacteria still showed sensitivity to Gentamicin, although at a low percentage of around 26%. Sensitivity to gentamicin may be due to its lower frequency of use. Resistance to Amikacin and Gentamicin remained low, but increased to Cephalexin, Co-Amoxiclav, and Nitrofurantoin [23]. Gentamicin showed the highest sensitivity (74.1%) among the cultures, as two-thirds of *E. coli* were sensitive to it. The sensitivity pattern also showed that the majority of Staphylococcus aureus and Enterococcus faecalis isolates were significantly sensitive to

gentamicin [24]. Ampicillin (38%), ceftriaxone (32.9%), ciprofloxacin (28.3%), and trimethoprim-sulfamethoxazole (TMP/SMX) (24.4%) had the highest levels of resistance. Resistance to antibiotics such as meropenem, amikacin, and gentamicin was also lower [25].

In this study, a considerable proportion of UPEC isolates were classified as multidrug-resistant (MDR). The resistance profile of Escherichia coli in East Java shows a high level of resistance to amoxicillin, as well as the presence of resistance to ciprofloxacin in some isolates from native chicken and meat sources [26, 27]. These findings are consistent with other reports from Indonesia indicating high resistance to β-lactam and fluoroquinolone antibiotics and a high prevalence of multidrug-resistant (MDR) isolates, particularly among ESBL-producing strains[28]. Clinically, although some clinical isolates remain susceptible to ciprofloxacin, the occurrence of resistance in community and food-related sources represents a significant threat to public health.

The coexistence of antibiotic resistance and virulence genes highlights the increasing clinical challenge posed by uropathogenic Escherichia coli (UPEC) and underscores the need for strengthened antibiotic stewardship and infection control measures [29]. Virulence gene profiles can serve as indicators of pathogen behavior; in particular, severe or persistent lesions are associated with highly virulent strains [30]. Virulence gene profiles are recognized as indicators of pathogen behavior, with more severe or persistent infections often associated with highly virulent strains [30]. In this context, the reported association between phenotypic antibiotic resistance and specific virulence genes suggests that resistance and virulence traits may be co-selected under antimicrobial pressure [31]. The results of this study showed a high frequency of *fimH* (98%) in UPEC isolates, suggesting a role in causing UTI in patients. Most strains of uropathogenic *Escherichia coli* (UPEC) encode long, thin filamentous appendages called type 1 pili. Type 1 fimbriae (*FimH*), in particular, play a crucial role in initial attachment to bladder epithelial cells, enabling bacterial invasion and the formation of intracellular bacterial communities (IBCs). *FimH* binds to mannosylated glycoproteins on bladder epithelial cells, promoting invasion and biofilm formation, which contribute to recurrent UTI [32]. The primary natural receptor for *FimH* is the glycoprotein uroplakin Ia, which is present in high concentrations on urinary tract epithelial cells. Other *FimH* receptors include the Tamm-Horsfall glycoprotein (THP), β1 and α3 integrins, CD48, collagen, laminin, fibronectin, and abiotic surfaces. *FimH* detects a wide variety of high-mannose glycoproteins. Therefore, it targets renal proximal tubule cells, buccal cells, bladder epithelial cells, intestines, lungs, and various inflammatory cells [33]. Biofilm-associated growth enhances bacterial persistence and reduces antibiotic susceptibility by limiting antimicrobial penetration and promoting adaptive stress responses, providing a biological explanation for the frequent co-occurrence of high virulence and antibiotic resistance in UPEC isolates.

The results of this study indicate that the prevalence of f*imH* among UPEC isolates is similar to other studies, including the highly heterogeneous expression of *fimH*, ihf, upaB, and upaH in clinical isolates, both at transcript levels and in response to suspension or biofilm conditions [34]. Molecular analysis showed that the majority of UPEC tested (98.6%; 69/70) contained the *FimH* gene. A significant correlation was found between *FimH* and antimicrobial resistance traits of UPEC [35]. The results of the study reported the presence of the studied genes in a high percentage (95.6%) for the *fimH*-encoded gene, while in a moderate percentage (47%) for the tosA-encoded gene. Thus, *fimH* and tosA can be used as possible diagnostic markers in addition to indicating the level of pathogenicity of UPEC [36]. The most frequently found virulence genes among all *E. coli* isolates were *fimH* (53/60, 88.3%) and fyuA (42/53, 70%), followed by sfa (33/60, 55%) and usp (24/60, 40%). Pap was the least frequently detected, found in only 10 isolates. The *fimH* gene was present in 100% of fecal isolates from UTI patients compared to 70% of fecal isolates from healthy controls [37]. Biofilm-forming UPEC, compared with commensal E. coli, exhibited significantly greater antibiotic resistance, with a 128-fold decrease in ciprofloxacin susceptibility. In addition, the *fimH* gene was detected in 98.33% of UPEC isolates [38].

CONCLUSION

E. coli isolated from urinary tract infection patients showed the highest resistance to amoxicillin, followed by ciprofloxacin and gentamicin. Detection of the *fimH* gene, which encodes an adhesion factor, was also highly prevalent. The presence of antibiotic resistance and virulence genes is a concern. Further research should analyze the relationship between virulence genes and antibiotic resistance.

DECLARATIONS

Ethics approval

This study was approved by Ethical approval obtained from the Health Research Ethics Committee of Fakultas Kedokteran Universitas Islam Malang, No. 089/LE.003/V/04/2024

Conflict of interest.

The authors declare no conflict of interest

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