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ORIGINAL ARTICLE

Molecular Detection of the Virulence *ureC* Gene of *Proteus mirabilis* in Urine Samples from Catheterized Patients

Nasreen Abdulgabbar Ahmad[1], Nehan Bahaaldden Jafar [1*] , Sanaa H.Mohammed [1]

[1]*Department of Biology, College of Sciences, University of Kirkuk, Kirkuk, Iraq.*

[*]*Corresponding author email: nihan@uokirkuk.edu.iq*

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ABSTRACT

Background: *Proteus mirabilis* is a facultative anaerobe in the family Enterobacteriaceae and a frequent cause of catheter-associated urinary tract infection (CAUTI) owing to swarming motility, biofilm formation, and urease activity. This study aimed to detect the *ureC* gene among catheterized patients admitted to hospitals in Kirkuk.

Methods: A total of 235 urine specimens were collected from catheterized patients at three hospitals in Kirkuk Province (Al-Amal Center, Artificial Kidney Unit at Kirkuk Teaching Hospital, and Azadi Teaching Hospital) from March through July 2024. Isolates were identified using conventional biochemical tests with automated confirmation (VITEK 2/DL-120). Antimicrobial susceptibility of *P. mirabilis* isolates was assessed by the Kirby–Bauer disk diffusion method. Detection of *ureC* was performed by PCR.

Results: Of 235 samples, 56 (23.8%) yielded *P. mirabilis*. Urease activity on urea agar was observed in 52/56 isolates (92.9%), and PCR confirmed the presence of *ureC* in 52/56 (92.9%). Among 16 tested antimicrobials, resistance was 100% to ceftazidime, ampicillin, and trimethoprim–sulfamethoxazole, whereas meropenem showed the lowest resistance (3.6%).

Conclusion: *Proteus mirabilis* was frequently isolated from catheterized patients in Kirkuk and exhibited a high prevalence of urease positivity with concordant *ureC* detection. PCR-based identification of *ureC* complements phenotypic urease testing and represents a practical marker of virulence potential and diagnostic value in CAUTI surveillance.

Key words: *Proteus mirabilis*; Catheter-associated urinary tract infection; *UreC* gene; Antimicrobial resistance.

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INTRODUCTION

Proteus mirabilis is a Gram-negative, facultatively anaerobic bacterium within the family Enterobacteriaceae and a major cause of catheter-associated urinary tract infections (CAUTIs). A central driver of its pathogenesis is urease activity, which hydrolyzes urea to ammonia and carbon dioxide, increases urinary pH, and promotes precipitation of struvite and hydroxyapatite. These mineral deposits seed crystalline biofilms that encrust and obstruct catheters, leading to urinary retention, ascending infection, and complications including pyelonephritis, bacteremia, and endotoxic shock [1, 2].

Beyond urease, *P. mirabilis* expresses additional virulence traits—including swarming motility, fimbrial adhesins, hemolysins, and siderophores—that facilitate rapid surface colonization, biofilm formation, and evasion of host defenses [3].

Acting in concert within the catheterized urinary tract, these factors sustain persistence and recurrence of infection.

Clinically, *P. mirabilis* is among the most frequently isolated organisms in CAUTI and is associated with recurrent encrustation and difficult-to-eradicate biofilms. Antimicrobial susceptibility patterns vary by setting; reduced susceptibility to several β-lactams has been reported, whereas carbapenems and aminoglycosides often retain activity—underscoring the importance of prevention and targeted therapy [4, 5].

At the genetic level, urease expression is encoded by the *ureDABCEFG* operon and regulated by the transcriptional activator UreR. Within this cluster, the *ureC* gene encodes the catalytic α subunit and serves as a reliable genotypic marker for molecular detection of urease-producing *P. mirabilis* [6, 7]. Genotypic confirmation via *ureC* complements phenotypic assays and strengthens attribution of urease-mediated pathogenic mechanisms in CAUTI.

This study aimed to isolate *P. mirabilis* from catheterized patients in Kirkuk Province and to assess phenotypic urease activity alongside genotypic detection of the *ureC* gene, providing complementary evidence for urease-driven pathogenesis in CAUTI.

MATERIALS AND METHODS

The study sample was collected from March to July 2024; a total of 235 urine specimens were collected from catheterized patients with clinical signs and symptoms of urinary tract infection (UTI) attending the Al-Amal Center, the Artificial Kidney Unit at Kirkuk Teaching Hospital, and Azadi Teaching Hospital (Kirkuk, Iraq). Participants included both sexes (151 females, 84 males; age 15–75 years). Specimens were obtained aseptically from the catheter sampling port (not the collection bag): the catheter was clamped for 15–30 min, the port disinfected with 70% alcohol, and 5–10 mL of urine was aspirated with a sterile syringe into a sterile container. Samples were transported to the laboratory within 2 h or stored at 4 °C and processed within 24 h. Demographic variables (age and sex) were recorded. The study protocol was approved by the College of Science, Department of Biology, University of Kirkuk (Approval No. 418; 24 April 2024).

All 235 specimens were processed aseptically. Samples were inoculated onto blood agar, nutrient agar, cystine–lactose–electrolyte-deficient (CLED) agar, and MacConkey agar, then incubated at 37,°C for 24–48,h. Colonies suggestive of *Proteus mirabilis* were initially identified by standard morphological and biochemical characteristics, and confirmation was performed using the VITEK 2 Compact system (bioM'erieux, France) with the DL-120 card (ZHUHAL DL BIOTECH, Germany). Urease activity was assessed on urea agar.

All *P. mirabilis* isolates (n=56) underwent antimicrobial susceptibility testing by the Kirby–Bauer disk diffusion method on Mueller–Hinton agar, with results interpreted according to CLSI (2021) criteria. Zone diameters were read using a calibrated digital zone reader (DL-120; ZHUHAL DL BIOTECH, Germany). Isolates were tested against amikacin, ampicillin, cefazolin, cefepime, cefotaxime, cefoxitin, ceftazidime, cefuroxime, chloramphenicol, gentamicin, imipenem, levofloxacin, meropenem, piperacillin–tazobactam, and trimethoprim–sulfamethoxazole. Ceftazidime–clavulanate was employed solely for ESBL confirmation and was not included in therapeutic susceptibility percentages.

Isolates were subcultured in brain–heart infusion broth and incubated at 37 °C for 24 h. Genomic DNA was extracted using the FavourPrep Blood/Cultured Cell Genomic DNA Extraction Mini Kit (Favorgen Biotech, Taiwan) according to the manufacturer's instructions. DNA concentration and purity were measured with a Quantus™ fluorometer (Promega, USA). The *ureC* gene was detected by PCR using specific primers, with cycling conditions summarized in Table 1. A schematic overview of the workflow is presented in Figure 1.

Analyses were performed using SPSS (version 17.0). Categorical comparisons used Pearson's chi-square (χ^2) test, with Fisher's exact test applied when expected cell counts were < 5. All p-values are two-sided with $\alpha = 0.05$. Where multiple independent comparisons were conducted (sex, age group, department), multiplicity was controlled using the Holm–Bonferroni method. Proportions are reported with counts and percentages, and exact p-values are provided.

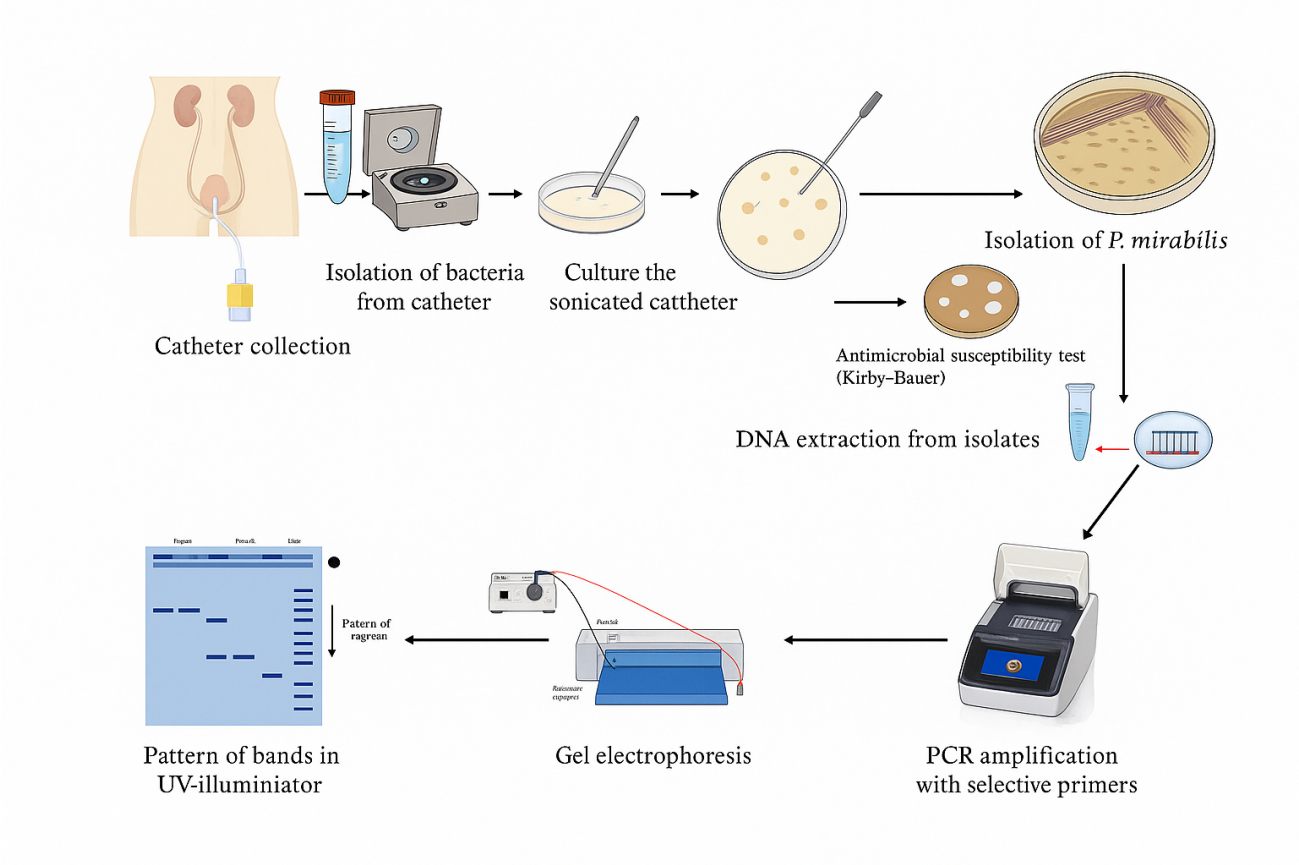

Figure 1. Graphical abstract of the methodology.

Table 1. Primer details and PCR program for amplification of *ureC*.

Primer details				PCR program		
Gene	**T_m (°C)**	**Primer sequence (5′→3′)**	**Amplicon (bp)**	**Step**	**Temp. (°C)**	**Time**
ureC	61.8	F: CAAGCCCAAGAAGGTCTCGT R: CAAGATGCTCGTCCACGGTA	517	Initial denaturation	94	3 min
				Denaturation	94	1 min
				Annealing	63	9 s
				Extension	72	1 min
				Final extension	72	7 min
				Steps 2–4 were repeated for 40 cycles.		

RESULTS

Conventional and biochemical tests

Of 235 urine specimens, 56 (23.8%) were presumptively identified as *P. mirabilis* based on colony morphology and biochemical features. On blood agar, colonies exhibited characteristic β-hemolysis and swarming motility, forming concentric, thin, film-like layers radiating from the inoculation point and producing a distinctive fishy odor (Figure 2b). On MacConkey agar, colonies appeared pale and smooth, consistent with non-lactose fermentation; on CLED agar, colonies showed similar morphology with a characteristic swarming pattern (Figure 2d). All 56 presumptive isolates were confirmed as *P. mirabilis* by VITEK 2 with the DL-120 card. Urease production on urea agar (yellow → pink) was observed in 52/56 isolates (92.9%) (Figure 2e).

Table 2. Demographics, antimicrobial resistance, and virulence factors among *Proteus mirabilis* isolates (n=56).

Variable / Item	n (%)	*p*-value*
Sex		
Female	37 (66.1)	**<0.001**
Male	19 (33.9)	
Age (years)		
15–35	7 (12.5)	
36–55	12 (21.4)	**<0.001**
≥56	37 (66.1)	
Department		
Urology	47 (83.9)	
Resuscitation	7 (12.5)	**0.020**
Burns	2 (3.6)	
Antimicrobial resistance (number resistant)		
Amikacin	11 (19.6)	—
Ampicillin	56 (100.0)	—
Cefazolin	39 (69.6)	—
Cefepime	42 (75.0)	—
Cefotaxime	23 (41.1)	—
Cefoxitin	45 (80.4)	—
Ceftazidime	56 (100.0)	—
Cefuroxime	38 (67.9)	—
Chloramphenicol	45 (80.4)	—
Gentamicin	42 (75.0)	—
Imipenem	20 (35.7)	—
Levofloxacin	28 (50.0)	—
Meropenem	2 (3.6)	—
Piperacillin–tazobactam	6 (10.7)	—
Trimethoprim–sulfamethoxazole	56 (100.0)	—
ESBL confirmatory test†		
ESBL positive (CAZ–CLA synergy)	20 (35.7)	—
Virulence factors		
Urease enzyme	52 (92.9)	**0.08**
ureC gene	52 (92.9)	

* Pearson's χ^2 or Fisher's exact test (two-sided) as appropriate; multiplicity controlled by Holm–Bonferroni across demographic blocks. P-values do not apply to per-antibiotic descriptive resistance counts.
† ESBL status determined by ceftazidime–clavulanate synergy; reported as number positive (not a therapeutic susceptibility result).

Demographic characteristics

Sex, age group, and admitting department are summarized in Table 2. Isolation was higher in females than males (37/56 [66.1%] vs 19/56 [33.9%]; χ^2, $p < 0.001$). By age, the 56–75 year group accounted for 37/56 (66.1%), compared with 12/56 (21.4%) in 36–55 years and 7/56 (12.5%) in 15–35 years (χ^2, $p < 0.001$). By department, the largest proportion was from Urology (47/56; 83.9%), followed by Resuscitation (7/56; 12.5%) and Burns (2/56; 3.6%), with a significant association across departments (χ^2, $p = 0.020$). Where applicable, Fisher's exact test was used when expected counts were < 5.

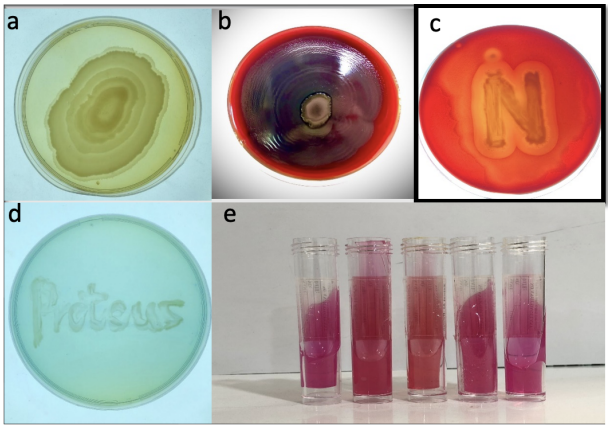

Figure 2. Colony morphology and biochemical traits of *Proteus mirabilis*. (a) Swarming motility on nutrient agar; (b) growth on blood agar; (c) β-hemolysis on blood agar; (d) growth on CLED agar; (e) urease production on urea agar (pink = positive).

Antimicrobial susceptibility

Antimicrobial susceptibility profiles for the 56 isolates across 15 therapeutic agents are shown in Figure 3. Resistance was universal (100%) to ceftazidime, ampicillin, and trimethoprim–sulfamethoxazole. The lowest resistance was observed for meropenem (2/56; 3.6%). Intermediate resistance levels were observed for gentamicin (42/56; 75.0%), cefepime (42/56; 75.0%), cefoxitin (45/56; 80.4%), and chloramphenicol (45/56; 80.4%). Piperacillin–tazobactam showed relatively favorable activity (6/56; 10.7% resistant).

Molecular detection of *ureC*

Conventional PCR targeting *ureC* yielded the expected 517 bp amplicon in 52/56 isolates (92.9%), concordant with phenotypic urease positivity and VITEK 2 identification. Representative products are shown in Figure 4.

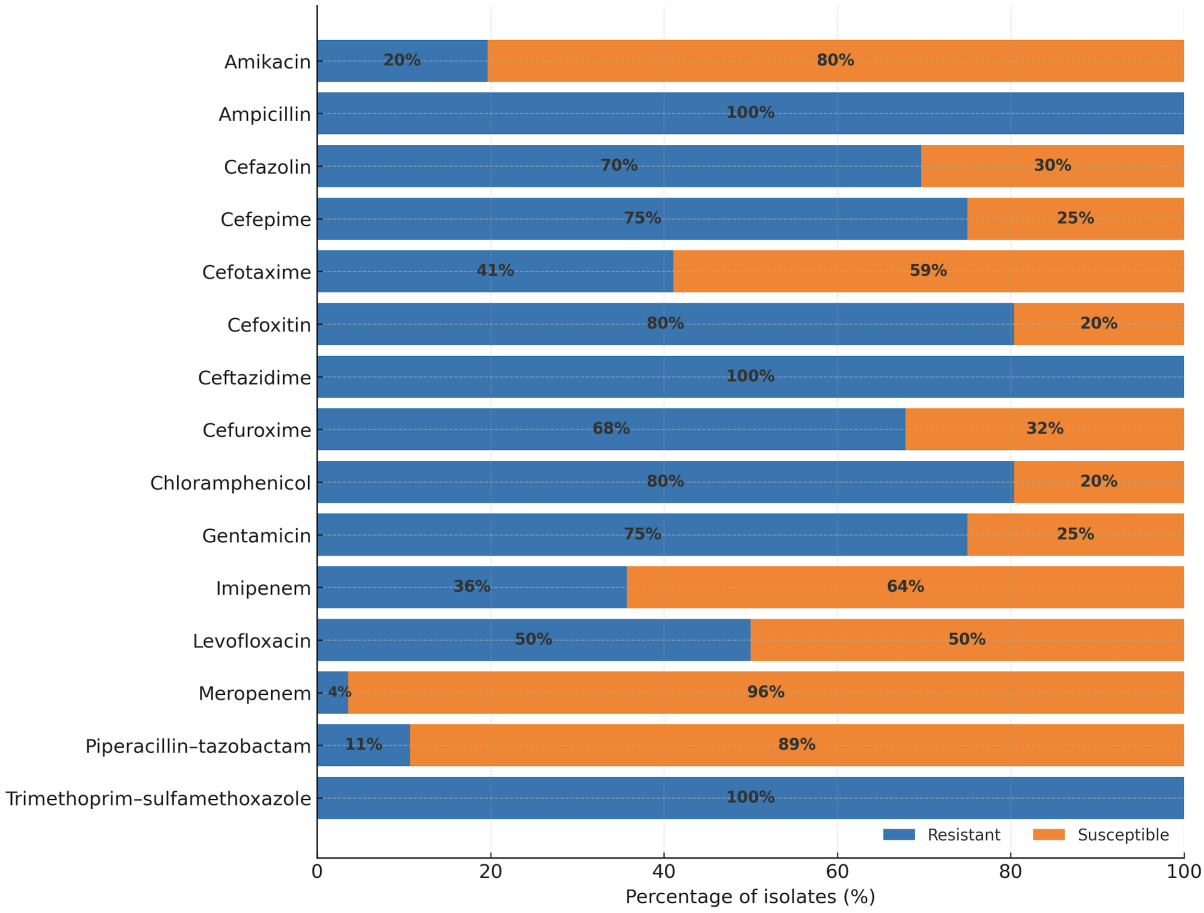

Figure 3. Antibiotic susceptibility of *Proteus mirabilis* isolates (n=56). Horizontal 100% stacked bars display resistant and susceptible proportions for each therapeutic agent; the ESBL confirmatory agent (ceftazidime–clavulanate) is excluded.

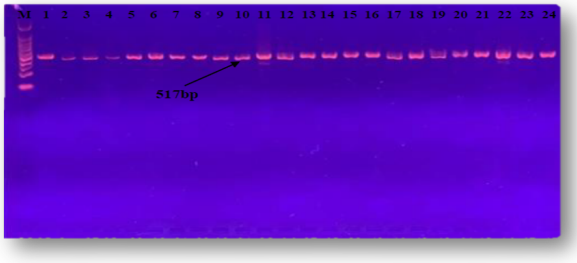

Figure 4. PCR amplification of the *ureC* gene (517 bp) in *Proteus mirabilis* isolates. Amplicons were resolved on a 1.2% agarose gel and visualized post-staining (75 V, 60 min). M, 100 bp DNA ladder; lanes 1–24, selected clinical isolates; positive amplification indicated by bands at 517 bp.

DISCUSSION

From all clinical specimens processed, 56 isolates (23.8%) were identified as *Proteus mirabilis* based on characteristic colony morphology on blood, nutrient, and MacConkey agars. On blood agar, the isolates displayed swarming motility with a characteristic fishy odor, and on MacConkey agar they produced smooth, pale (non-lactose-fermenting) colonies [8]. Phenotypic identification was confirmed using the VITEK 2 automated system with the DL-120 card [9]. Urease production was demonstrated in 52 isolates on urea agar by a pink color shift, reflecting hydrolysis of urea to ammonia and carbon dioxide with alkalinization of the medium [10, 11].

Sex-specific distribution showed that 37/56 (66.1%) of the isolates were from females and 19/56 (33.9%) from males, a difference that was statistically significant ($p < 0.001$). This female predominance agrees with prior reports (53.37%, 61.2%, and 63.7%) [4, 12, 13]. *P. mirabilis* urinary tract infection is frequently associated with catheterization [14, 15]; intermittent periurethral colonization from the intestinal tract can seed the urinary tract and, during catheterization, facilitate hospital-acquired infection. Biofilm formation on catheter surfaces is clinically important, and the *ureC* gene contributes to encrustation and urinary stone formation within these biofilms [16]. The higher burden among females is plausibly explained by anatomical factors (shorter, wider urethra and proximity of the urethral meatus to the anus and reproductive tract) and hygiene practices [17], whereas prostatic secretions rich in zinc may confer some protection in males.

Age-stratified analysis showed the highest isolation rate among patients aged 56–75 years (37/56; 66.1%), which was statistically significant ($p < 0.001$). Comparable age-related patterns have been reported (56.25% and 82.5% in older groups) [4, 13], although one study found a higher proportion among patients <18 years (40%) [18], underscoring contex-

tual differences in case mix and exposure.

By hospital location, isolation was greatest in Urology (47/56; 83.9%) and lowest in Burns (2/56; 3.6%), with a significant association across departments (p = 0.02). These distributions likely reflect referral patterns and procedure-related risk; together with community exposures, they may indicate a contribution of community acquisition in the Kirkuk setting, although dedicated epidemiologic data would be required to confirm this.

Antimicrobial susceptibility testing was performed by the disk diffusion method and interpreted according to CLSI 2021 [19]. Amikacin, an aminoglycoside that binds the 30S ribosomal subunit and inhibits protein synthesis [20], and meropenem exhibited the highest susceptibility rates in this cohort, consistent with earlier studies [12, 13, 21]. In contrast, the lowest susceptibility was observed for trimethoprim–sulfamethoxazole and ampicillin [22, 23]. Resistance to gentamicin was 75%, aligning with a reported rate of 76% [24] but differing from other series (41.2% and 7.5%) [20, 25], which may reflect local prescribing practices, breakpoints used, and resistance ecology.

The *ureC* gene encodes a structural subunit of urease, a metalloenzyme central to nitrogen metabolism. Urease catalyzes the hydrolysis of urea to ammonia and carbon dioxide, elevating local pH, promoting crystal precipitation, injuring the uroepithelium, and thereby facilitating persistence of *P. mirabilis* within the urinary tract [26]. In the present work, PCR detected *ureC* in 52/56 isolates (amplicon 517 bp), concordant with phenotypic urease results and comparable to prior detection rates (93%) [18]. These findings reinforce the role of *ureC*-mediated urease activity in the pathogenesis of catheter-associated infection, encrustation, and stone disease.

This study has limitations. *ureC* carriage was confirmed by PCR, but functional expression (e.g., *ureC* mRNA by RT-qPCR) and enzymatic activity in vitro/in vivo were not assessed; thus, genotype–phenotype linkage cannot be fully established. Variability in sampling, storage, and processing of urine or catheter-biofilm material may also affect DNA integrity and PCR yield. Future work should incorporate longitudinal sampling, quantitative assessment of biofilm burden and catheter encrustation, and gene-expression or activity measurements (e.g., RT-qPCR for *ureC* or urease assays) to clarify the temporal relationship among urease expression, biofilm development, and clinical outcomes.

CONCLUSION

PCR-based detection of the *ureC* gene provided rapid and reliable genotypic confirmation in *Proteus mirabilis* isolates, in agreement with phenotypic urease activity. The high frequency of *ureC* carriage and its established link to urease-mediated alkalinization, catheter encrustation, and stone formation underscore its central role in virulence and pathogenicity. Collectively, these findings support *ureC* as a practical diagnostic marker for laboratory identification and as an indicator of pathogenic potential. Integration of *ureC*-targeted PCR into routine workflows and surveillance panels is supported, with future studies aimed at quantifying *ureC* expression and correlating it with clinical outcomes to refine risk stratification.

ETHICAL DECLARATIONS

Ethics Approval and Consent to Participate

Ethical approval was granted by the College of Science, Department of Biology, University of Kirkuk (Document No. 418; 24 April 2024). Written informed consent was secured from all participants, with assurances of confidentiality and secure handling of patient information throughout all phases of the study.

Consent for Publication

Non.

Availability of Data and Material

The datasets are available from the corresponding author upon reasonable request.

Competing Interests

The authors declare that there is no conflict of interest.

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Use of Generative Artificial Intelligence

The authors declare that no generative AI tools were used in the preparation, writing, or editing of this manuscript.

Authors' Contributions

All authors contributed to the literature review, study design, data collection, statistical analysis, and manuscript preparation. All authors have read and approved the final version of the manuscript.

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