

Uropathogen Distribution and Antimicrobial Resistance in Patients With Diabetes, Other Chronic Diseases, and No Comorbidities: A Retrospective Cohort Study

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Abstract

Background

Diabetes mellitus and other chronic diseases are associated with urinary infection, catheterization, and other urological and healthcare exposures that may influence both uropathogen distribution and antimicrobial resistance. However, binary diabetic versus non-diabetic comparisons often combine otherwise healthy adults with patients who have substantial non-diabetic comorbidity. We compared bacterial isolates and antimicrobial resistance across three clinically distinct comorbidity groups.

Methods

This retrospective, laboratory-based study included 835 eligible positive bacterial urine-culture records from adults evaluated by the Departments of Internal Medicine, Urology, and Infectious Diseases at a tertiary hospital in Istanbul, Türkiye, between 1 January 2021 and 31 December 2025. Records were classified into three mutually exclusive groups: diabetes mellitus, non-diabetic chronic disease, and no documented comorbidity. Group differences in demographic characteristics, urine-collection method, uropathogen distribution, and antimicrobial resistance among *Escherichia coli* isolates were evaluated. Because complete culture dates and a consistently reliable anonymized patient identifier were unavailable, analyses were performed at the culture-record level.

Results

Among 835 eligible positive bacterial urine-culture records, 56 (6.7%) were from the diabetes group, 153 (18.3%) from the non-diabetic chronic disease group, and 626 (75.0%) from the no documented comorbidity group. Median age and the frequency of catheter-collected specimens were highest in the diabetes group (both $P < 0.001$). *Escherichia coli* was the predominant organism but accounted for a lower proportion of records in the diabetes, non-diabetic chronic disease, and no documented comorbidity groups, respectively (32.1%, 41.2%, and 54.2%; $P < 0.001$). *Klebsiella* spp. and *Pseudomonas aeruginosa* were proportionally more frequent in the diabetes group, although the between-group differences did not reach statistical significance. Among evaluable *E. coli* isolates, gentamicin resistance was 35.3%, 13.3%, and 8.7% across the diabetes, non-diabetic chronic disease, and no documented comorbidity groups, respectively ($P = 0.003$). Corresponding nitrofurantoin resistance was 25.0%, 6.9%, and 5.0% ($P = 0.012$). Resistance to ceftriaxone, fluoroquinolones, trimethoprim-sulfamethoxazole, and ertapenem was numerically higher in the diabetes group, but these differences were not statistically significant and several estimates were based on small tested denominators.

Conclusions

Uropathogen distribution and selected *E. coli* resistance patterns differed across comorbidity groups in this tertiary-care culture-record cohort. Adults with diabetes had greater representation of non-*E. coli* organisms and higher resistance to gentamicin and nitrofurantoin, but they were also older and more frequently catheterized. These findings support comorbidity-aware interpretation of local microbiological

surveillance and individualized urological risk assessment; they do not establish diabetes as an independent cause of antimicrobial resistance.

Introduction

Urinary tract infections (UTIs) are among the most common bacterial infections encountered in outpatient and inpatient clinical practice and represent a major driver of antimicrobial prescribing worldwide [1–3]. *Escherichia coli* remains the predominant uropathogen, particularly in community-acquired infections; however, *Klebsiella* spp., *Proteus* spp., *Pseudomonas aeruginosa*, *Enterococcus* spp., and other Gram-negative and Gram-positive organisms are frequently encountered in older patients and those with complicated urinary conditions or substantial healthcare exposure [1, 4–6]. The distribution of urinary pathogens varies according to age, sex, geographical region, clinical setting, previous antimicrobial exposure, and local prescribing practices [3, 5–7]. In Türkiye, Gözüküçük et al. previously reported antibiotic susceptibility patterns among *Escherichia coli* isolates from community-acquired UTIs, providing an earlier local benchmark for empirical treatment [8]. Consequently, institution-specific surveillance data are important for informing empirical treatment decisions and antimicrobial stewardship.

The management of UTIs has become increasingly challenging because of rising antimicrobial resistance among urinary pathogens [9]. Resistance to trimethoprim-sulfamethoxazole, fluoroquinolones, aminoglycosides, beta-lactam/beta-lactamase inhibitor combinations, and third-generation cephalosporins has been reported across both community and hospital settings [7, 10, 11]. Extended-spectrum beta-lactamase-producing and multidrug-resistant Enterobacterales, particularly *E. coli* and *Klebsiella pneumoniae*, are of particular concern because they may limit oral treatment options and increase the need for broad-spectrum or parenteral therapy [5, 10–12]. However, resistance estimates vary considerably between institutions and patient populations. Laboratory-based surveillance may overestimate resistance in uncomplicated community infections because urine cultures are more likely to be requested from patients with recurrent infection, treatment failure, comorbidities, or previous antimicrobial exposure [13, 14]. Local resistance findings must therefore be interpreted in relation to the clinical and epidemiological characteristics of the population from which the isolates were obtained.

Diabetes mellitus is an important host-related factor associated with an increased risk of bacteriuria, symptomatic UTI, recurrent infection, and complicated urinary disease [15–17]. Several mechanisms may contribute to this susceptibility, including impaired innate and adaptive immune responses, glycosuria, autonomic bladder dysfunction, incomplete bladder emptying, microvascular disease, and more frequent contact with healthcare services [15, 16]. Patients with diabetes may also have greater exposure to antimicrobial agents, urinary catheterization, and urological procedures, potentially increasing the likelihood of infection or colonization with resistant organisms.

Nevertheless, whether diabetes itself influences the urinary pathogen spectrum and antimicrobial resistance remains uncertain. Some studies have reported a greater frequency of non-*E. coli* organisms

or resistant Enterobacterales among patients with diabetes, whereas others have found more limited differences after considering age, sex, catheterization, hospitalization, and previous antimicrobial use [18]. A recent systematic review and meta-analysis confirmed that *E. coli* remains the predominant urinary isolate among patients with diabetes but also demonstrated substantial heterogeneity in pathogen prevalence and antimicrobial resistance across studies [19]. These findings suggest that microbiological patterns attributed to diabetes may partly reflect differences in case mix, healthcare exposure, and local antimicrobial pressure.

Chronic diseases other than diabetes may have similar effects. Chronic kidney disease, cardiovascular disease, malignancy, neurological disorders, and chronic pulmonary disease are frequently accompanied by advanced age, repeated hospitalization, urinary instrumentation, and antimicrobial exposure. These characteristics have been associated with resistant urinary organisms and multidrug resistance [20–22]. However, many studies use a binary diabetic versus non-diabetic comparison, thereby including otherwise healthy individuals and patients with substantial non-diabetic comorbidity in the same reference group. Other studies classify these heterogeneous populations under the broad category of complicated UTI [6]. These approaches make it difficult to determine whether observed microbiological differences are specifically associated with diabetes or reflect the broader consequences of chronic disease, advanced age, and healthcare exposure.

Direct comparisons among patients with diabetes, patients with non-diabetic chronic diseases, and patients without documented comorbidities remain limited. Separating these three groups may provide a more clinically informative assessment of the relationship between comorbidity status, urinary pathogen distribution, and antimicrobial resistance. It may also clarify whether patients with non-diabetic chronic diseases demonstrate an intermediate or distinct microbiological profile compared with the other two groups.

This study aimed to compare bacterial uropathogen distribution and antimicrobial resistance across three mutually exclusive groups of adults with positive urine cultures: those with diabetes mellitus, those without diabetes but with at least one documented chronic disease, and those with no documented comorbidity. Secondary objectives were to compare resistance among *E. coli* isolates and to assess how the observed microbiological patterns should be interpreted in relation to age, sex, urine-collection method, and clinically relevant urological exposures.

Materials and Methods

Study design and setting

This retrospective, laboratory-based observational study was conducted at Hisar Intercontinental Hospital, a tertiary-care hospital in Istanbul, Türkiye. Electronic microbiology and clinical records from the Departments of Internal Medicine, Urology, and Infectious Diseases were screened for positive urine cultures obtained from adults between 1 January 2021 and 31 December 2025. The study was reported

in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement.

Eligibility criteria and study population

The source population consisted of adults who underwent urine culture testing in the participating departments during the study period. The unit of analysis was an eligible positive urine-culture record.

Inclusion criteria

Records were eligible when all of the following criteria were met:

1. The patient was aged 18 years or older at the time of specimen collection;
2. The urine culture demonstrated growth of at least one bacterial organism reported by the microbiology laboratory;
3. At least one interpretable antimicrobial susceptibility result was available;
4. Age and sex were available; and
5. The available diagnostic information permitted assignment to one of the three prespecified comorbidity groups.

Exclusion criteria

Records were excluded for any of the following reasons:

1. Patient age younger than 18 years;
2. No bacterial growth or a negative urine culture;
3. Mixed growth reported by the laboratory as contamination or a non-interpretable polymicrobial culture;
4. An organism reported as a probable contaminant without sufficient microbiological evidence to retain it as a urinary isolate;
5. Absence of an interpretable antimicrobial susceptibility result;
6. *Ureaplasma* spp. or *Mycoplasma* spp. testing, because these records did not represent conventional bacterial urine cultures evaluated with the routine susceptibility panel;
7. Exact duplicate records originating from the same laboratory result; or
8. Insufficient diagnostic information to assign the record to a prespecified comorbidity group.

The available dataset did not contain complete culture dates and a consistently reliable anonymized patient identifier for all records. Consequently, repeat positive cultures from the same patient could not be systematically deduplicated by a predefined time interval, and the primary analysis was performed at the culture-record level. This analytical feature was considered when interpreting precision and generalizability and was included among the study limitations.

Definition of comorbidity groups

Eligible records were classified into three mutually exclusive groups using diagnoses documented in the available electronic records:

1. **Diabetes mellitus group:** patients with documented type 1 or type 2 diabetes mellitus, irrespective of the presence of additional chronic diseases;
2. **Other chronic disease group:** patients without diabetes who had at least one documented chronic disease; and
3. **No documented comorbidity group:** patients for whom no chronic disease was documented in the available dataset.

Diabetes was assigned hierarchical priority; therefore, a patient with both diabetes and another chronic disease was included in the diabetes group. Chronic diseases included hypertension or other cardiovascular disease, chronic kidney disease, chronic pulmonary disease, chronic liver disease, neurological disease, malignancy, thyroid disease, rheumatological or autoimmune disease, and other persistent disorders requiring continuing follow-up or treatment. Acute diagnoses, transient laboratory abnormalities, vitamin deficiencies without an associated chronic disorder, and the urinary condition prompting culture collection were not classified as chronic diseases.

Because diagnostic documentation was incomplete for some records, the third group was designated “no documented comorbidity” rather than “no comorbidity.” This terminology indicates absence of a recorded chronic diagnosis and does not establish that chronic disease was definitively excluded by comprehensive clinical assessment.

Record selection

A total of 895 positive culture records were assessed for eligibility. Sixty records involving Ureaplasma or Mycoplasma testing were excluded from the conventional bacterial analysis. The final analytical dataset comprised 835 eligible positive bacterial urine-culture records: 56 in the diabetes mellitus group, 153 in the non-diabetic chronic disease group, and 626 in the no documented comorbidity group.

Data collection

Available demographic, clinical, and microbiological variables were extracted from the hospital records. These included age, sex, specimen collection method when recorded, documented diabetes mellitus, documented non-diabetic chronic diseases, isolated microorganism, and antimicrobial susceptibility results. Glycated haemoglobin and C-reactive protein values were extracted when available but were not used as principal explanatory variables because of extensive missingness. Direct personal identifiers were removed before statistical analysis.

Ethical considerations

The study protocol was reviewed and approved by the Hisar Hospital Intercontinental Medical and Ethics Advisory Board (approval no. 26–63; 13 February 2026). The study involved retrospective analysis of routinely collected, anonymized clinical and laboratory records, with no direct patient contact or study-related intervention. The study was conducted in accordance with the Declaration of Helsinki and applicable national regulations.

Results

Study population and baseline characteristics

After the exclusion of Ureaplasma- and Mycoplasma-related records, 835 conventional culture-positive bacterial records were included in the analysis. Of these, 56 (6.7%) were classified in the diabetes mellitus group, 153 (18.3%) in the non-diabetic chronic disease group, and 626 (75.0%) in the group without documented comorbidities.

The groups differed significantly in age, sex distribution, and urine-collection method. Median age was 74.3 years (IQR, 64.2–81.6) in the diabetes group, 68.6 years (IQR, 54.3–77.4) in the non-diabetic chronic disease group, and 45.5 years (IQR, 32.4–70.4) in the group without documented comorbidities ($P < 0.001$). Women accounted for 48.2%, 66.0%, and 83.9% of the three groups, respectively ($P < 0.001$). Catheter-collected specimens were more frequent in the diabetes group than in the non-diabetic chronic disease and no-comorbidity groups (41.1%, 24.8%, and 8.5%, respectively; $P < 0.001$) (Table 1).

Table 1
Baseline characteristics of culture-positive records according to comorbidity group

Characteristic	Diabetes mellitus (n = 56)	Other chronic diseases (n = 153)	No documented comorbidity (n = 626)	P value
Records, n (%)	56 (6.7)	153 (18.3)	626 (75.0)	—
Age, years, median (IQR)	74.3 (64.2–81.6)	68.6 (54.3–77.4)	45.5 (32.4–70.4)	< 0.001
Female sex, %	48.2	66.0	83.9	< 0.001
Catheter-collected specimen, %	41.1	24.8	8.5	< 0.001

Data are presented as n (%) or percentage unless otherwise indicated. Abbreviation: IQR, interquartile range. P values compare the three comorbidity groups.

Uropathogen distribution

The overall distribution of conventional bacterial uropathogens differed significantly among the three comorbidity groups ($P = 0.003$). *Escherichia coli* was the most frequently identified microorganism in each group. However, its proportional contribution decreased with increasing comorbidity burden: *E. coli* accounted for 18 of 56 records (32.1%) in the diabetes group, 63 of 153 (41.2%) in the non-diabetic chronic disease group, and 339 of 626 (54.2%) in the group without documented comorbidities ($P < 0.001$).

Klebsiella spp. were identified in 26.8% of records in the diabetes group, 22.2% in the non-diabetic chronic disease group, and 16.9% in the no-comorbidity group. Although a numerical gradient was observed, the overall difference did not reach statistical significance ($P = 0.084$). Similarly, *Pseudomonas aeruginosa* was more frequent in the diabetes group than in the other chronic disease and no-comorbidity groups (12.5%, 6.5%, and 5.0%, respectively), although the difference was of borderline statistical significance ($P = 0.058$).

The frequency of *Enterococcus* spp. was 3.6% in the diabetes group, 8.5% in the non-diabetic chronic disease group, and 5.9% in the group without documented comorbidities ($P = 0.366$). *Streptococcus agalactiae* accounted for 3.6%, 1.3%, and 5.9% of isolates, respectively ($P = 0.052$), while *Proteus* spp. showed similar frequencies across the groups (1.8%, 2.0%, and 1.8%; $P = 1.000$). Staphylococcal species, including coagulase-negative staphylococci, were more frequent in the diabetes and non-diabetic chronic disease groups than in the no-comorbidity group (14.3%, 12.4%, and 6.7%, respectively; $P = 0.016$). The group-specific distribution of the principal uropathogens is presented in Table 2 and Fig. 1.

Table 2
Distribution of principal uropathogens according to comorbidity group

Uropathogen	Diabetes mellitus (n = 56)	Other chronic diseases (n = 153)	No documented comorbidity (n = 626)	P value
<i>Escherichia coli</i>	32.1	41.2	54.2	< 0.001
<i>Klebsiella</i> spp.	26.8	22.2	16.9	0.084
<i>Pseudomonas aeruginosa</i>	12.5	6.5	5.0	0.058
<i>Enterococcus</i> spp.	3.6	8.5	5.9	0.366
<i>Streptococcus agalactiae</i>	3.6	1.3	5.9	0.052
<i>Proteus</i> spp.	1.8	2.0	1.8	1.000
Staphylococcal species	14.3	12.4	6.7	0.016
Values are percentages of culture-positive records within each comorbidity group. The overall uropathogen distribution differed among the three groups (P = 0.003). Staphylococcal species include coagulase-negative staphylococci. P values in the table represent organism-specific comparisons across groups.				

Antimicrobial resistance among *Escherichia coli* isolates

Antimicrobial resistance analyses were restricted to *E. coli* isolates with internally consistent and interpretable susceptibility results. The number of isolates tested differed among antimicrobial agents because susceptibility panels were not uniform.

Resistance to gentamicin differed significantly among the three groups. Gentamicin resistance was detected in 6 of 17 evaluable isolates (35.3%) in the diabetes group, 8 of 60 (13.3%) in the non-diabetic chronic disease group, and 29 of 333 (8.7%) in the group without documented comorbidities (P = 0.003).

Nitrofurantoin resistance was also highest among patients with diabetes. Resistance was present in 4 of 16 tested isolates (25.0%) in the diabetes group, compared with 4 of 58 (6.9%) in the non-diabetic chronic disease group and 16 of 321 (5.0%) in the no-comorbidity group (P = 0.012).

Ceftriaxone resistance showed a similar numerical pattern, occurring in 10 of 18 isolates (55.6%) in the diabetes group, 21 of 61 (34.4%) in the non-diabetic chronic disease group, and 102 of 332 (30.7%) in the group without documented comorbidities. However, the overall difference did not reach statistical significance (P = 0.084).

Fluoroquinolone resistance was numerically higher in the diabetes group. Ciprofloxacin resistance was 47.1% in the diabetes group, 38.3% in the non-diabetic chronic disease group, and 29.7% in the group

without documented comorbidities (P = 0.165). Corresponding levofloxacin resistance rates were 61.5%, 46.8%, and 38.3%, respectively (P = 0.163).

Resistance to trimethoprim-sulfamethoxazole was detected in 47.1% of evaluable isolates in the diabetes group, 29.5% in the non-diabetic chronic disease group, and 29.6% in the no-comorbidity group (P = 0.307). Ampicillin resistance remained high in all three groups, with rates of 66.7%, 46.5%, and 56.2%, respectively (P = 0.438).

Amikacin activity was largely preserved. Resistance was detected in 1 of 16 isolates (6.2%) in the diabetes group, none of 50 isolates in the non-diabetic chronic disease group, and 2 of 287 isolates (0.7%) in the no-comorbidity group (P = 0.182). Ertapenem resistance was numerically higher in the diabetes group (20.0%) than in the non-diabetic chronic disease (4.0%) and no-comorbidity groups (5.0%), but the overall difference did not reach statistical significance using the fixed-margin Monte Carlo analysis (P = 0.057). Fosfomycin resistance did not differ among the groups (33.3%, 28.6%, and 33.3%, respectively; P = 1.000), although relatively few isolates were tested for this agent.

The complete antimicrobial resistance findings are shown in Table 3, and the group-specific resistance patterns are visualized in Fig. 2.

Table 3
Antimicrobial resistance among *Escherichia coli* isolates according to comorbidity group

Antimicrobial agent	Diabetes mellitus	Other chronic diseases	No documented comorbidity	P value
Gentamicin	6/17 (35.3)	8/60 (13.3)	29/333 (8.7)	0.003
Nitrofurantoin	4/16 (25.0)	4/58 (6.9)	16/321 (5.0)	0.012
Ceftriaxone	10/18 (55.6)	21/61 (34.4)	102/332 (30.7)	0.084
Ciprofloxacin	47.1%	38.3%	29.7%	0.165
Levofloxacin	61.5%	46.8%	38.3%	0.163
Trimethoprim-sulfamethoxazole	47.1%	29.5%	29.6%	0.307
Ampicillin	66.7%	46.5%	56.2%	0.438
Amikacin	1/16 (6.2)	0/50 (0.0)	2/287 (0.7)	0.182
Ertapenem	20.0%	4.0%	5.0%	0.057
Fosfomycin	33.3%	28.6%	33.3%	1.000
Values are resistant/tested isolates, n/N (%), when denominators were reported in the available manuscript text; otherwise, resistance percentages are shown. Susceptibility panels were not uniform, and the number of evaluable isolates therefore varied by antimicrobial agent. The ertapenem comparison used fixed-margin Monte Carlo analysis.				

Discussion

In this retrospective study of positive urine-culture records, bacterial distribution and antimicrobial resistance patterns differed across adults with diabetes mellitus, adults with other chronic diseases, and adults with no documented comorbidity. *Escherichia coli* remained the predominant organism, but its proportional contribution was lowest in the diabetes group. *Klebsiella* spp. and *Pseudomonas aeruginosa* were relatively more frequent among records from patients with diabetes. Among *E. coli* isolates, resistance to gentamicin and nitrofurantoin differed significantly and was highest in the diabetes group. These findings are relevant to urological practice because the diabetes group was older and substantially more likely to have catheter-collected specimens, but the same differences prevent attribution of the microbiological pattern to diabetes alone.

The predominance of *E. coli* is consistent with community- and hospital-based surveillance studies in which this organism has remained the leading bacterial isolate recovered from urine cultures [1–4, 7, 13]. Nevertheless, its relative frequency varies according to age, sex, healthcare exposure, specimen source, comorbidity burden, and whether the study population represents uncomplicated community infections or patients treated in tertiary-care settings [4–6, 10, 13]. Wu et al. reported that *E. coli* and *Klebsiella pneumoniae* accounted for 35.1% and 24.2% of bacterial isolates from positive urine cultures, respectively [5]. Similarly, Mahjabin et al. identified *E. coli*, *K. pneumoniae*, and *P. aeruginosa* as the predominant organisms among patients with complicated UTIs [6]. Although differences in setting and case definition limit direct numerical comparisons, the pathogen spectrum in our cohort is broadly consistent with these reports.

The lower proportion of *E. coli* and relative enrichment of *Klebsiella* spp. and *P. aeruginosa* in patients with diabetes may reflect a more complicated clinical profile rather than an isolated biological effect of diabetes. Patients in the diabetes group were older, more frequently male, and substantially more likely to have catheter-collected specimens than patients with no documented comorbidity. Older age, male sex, urinary catheterization, recent healthcare exposure, previous antimicrobial use, and recurrent urinary infection have been associated with resistant organisms and non-*E. coli* urinary pathogens [20–22]. Bischoff et al. identified urinary catheterization, nursing-home residence, recent hospitalization, and previous antimicrobial therapy as important predictors of antimicrobial resistance [21]. Therefore, the unadjusted differences observed in our study should not be interpreted as evidence that diabetes alone causes a shift toward *Klebsiella* or *Pseudomonas*.

Previous investigations of diabetes and urinary pathogens have produced heterogeneous findings. Bonadio et al. reported that diabetes influenced some aspects of the microbial spectrum and resistance profile among older adults, although sex and other clinical characteristics also contributed to the observed differences [18]. A recent systematic review and meta-analysis found that *E. coli* accounted for approximately half of urinary isolates from patients with diabetes, but substantial between-study heterogeneity was present [19]. Variation in healthcare setting, glycaemic control, prior antimicrobial exposure, urinary tract abnormalities, catheter use, and microbiological procedures may explain some of

this inconsistency. The relatively low proportion of *E. coli* in our diabetes group should therefore be interpreted in the context of its older and more frequently catheterized population.

Among *E. coli* isolates, gentamicin resistance was significantly higher in patients with diabetes than in the other groups. Resistance to ceftriaxone, ciprofloxacin, levofloxacin, trimethoprim-sulfamethoxazole, and ertapenem was also numerically higher. Diabetes has been associated with recurrent healthcare contact, antimicrobial exposure, hospitalization, urinary instrumentation, and impaired host defence, all of which may increase the likelihood of colonization or infection with resistant organisms [15–18]. However, information on previous antimicrobial use, recent hospitalization, recurrent infection, and previous resistant isolates was unavailable. The observed associations may therefore be partly attributable to unmeasured healthcare exposure and should not be interpreted as demonstrating an independent effect of diabetes.

The resistance patterns observed in this study further emphasize the importance of local surveillance. Large multicentre datasets have documented substantial resistance to fluoroquinolones and trimethoprim-sulfamethoxazole among urinary Enterobacterales isolates, together with increasing extended-spectrum beta-lactamase-associated resistance [10, 11]. Conversely, studies conducted in primary care populations have generally reported lower resistance levels [13, 23]. This difference may partly reflect selection bias because cultures are more likely to be requested from patients with treatment failure, recurrent infection, complicated disease, or previous antimicrobial exposure. Our findings are consequently most applicable to adults undergoing urine culture in a tertiary-care hospital and should not be extrapolated directly to all patients with uncomplicated community-acquired cystitis.

The higher nitrofurantoin resistance observed among diabetic *E. coli* isolates warrants attention but requires cautious interpretation. Nitrofurantoin activity against *E. coli* has generally remained well preserved in surveillance studies [5, 13, 19]. In the present cohort, however, only a limited number of diabetic *E. coli* isolates were tested against nitrofurantoin. Moreover, patients with diabetes were older and more frequently had catheter-collected specimens, suggesting a greater prevalence of complicated urinary conditions for which nitrofurantoin may be clinically unsuitable regardless of the *in vitro* susceptibility result. This finding should therefore be regarded as exploratory and requires confirmation in a larger cohort with standardized susceptibility testing.

Ertapenem resistance was recorded in 20% of the diabetic *E. coli* isolates tested, compared with approximately 4–5% in the other groups, although the between-group difference did not reach statistical significance. This estimate was based on a small denominator, and confirmatory information on extended-spectrum beta-lactamase production or carbapenemase status was unavailable. It should therefore not be interpreted as a precise estimate of carbapenem resistance among patients with diabetes. Verification of the original susceptibility reports and microbiological confirmation of resistant isolates would be required before drawing firm clinical or epidemiological conclusions. This is particularly important because recent surveillance has demonstrated marked organism-specific

variation, with carbapenem resistance generally being more frequent in *K. pneumoniae* than in *E. coli* urinary isolates [5].

An important feature of this study was the separation of patients with diabetes from those with other chronic diseases. Many previous studies have used a diabetic versus non-diabetic comparison without distinguishing otherwise healthy individuals from patients with substantial non-diabetic comorbidity. In our study, patients with other chronic diseases frequently showed intermediate pathogen and resistance patterns. This observation suggests that a binary comparison may obscure the effects of age, multimorbidity, catheterization, and healthcare exposure within the non-diabetic reference group. Nevertheless, this finding remains descriptive because individual chronic diseases and their severity could not be analysed separately.

From a urological perspective, the results support local microbiological surveillance and individualized risk assessment when selecting empirical antimicrobial treatment. Diabetes status alone should not determine antibiotic choice. Clinical decisions should also consider urinary symptoms and infection site, renal function, urinary retention or obstruction, stone disease, previous culture results, recent antimicrobial exposure, catheterization, and recent urological procedures, together with local resistance thresholds and current treatment guidelines [14, 24, 25]. Once culture and susceptibility results are available, empirical therapy should be reassessed and narrowed whenever clinically appropriate.

Strengths and limitations

This study included a relatively large collection of positive urine cultures obtained over a five-year period and directly compared three clinically relevant comorbidity groups. Pathogen distribution and organism-specific resistance were evaluated separately, and resistance results were reported using antibiotic-specific tested denominators. This is particularly important when susceptibility panels are not uniform across organisms or study groups.

The study also has several limitations. First, its retrospective design relied on the completeness and accuracy of routine clinical records. A substantial proportion of patients had no recorded diagnosis; therefore, the reference group represents patients with no documented comorbidity rather than patients in whom chronic disease was definitively excluded. Misclassification between groups is possible and would most likely attenuate true differences.

Second, clinical symptoms, pyuria, fever, infection site, clinical diagnosis, and treatment information were unavailable. Positive urine cultures could not therefore be reliably classified as symptomatic UTI, asymptomatic bacteriuria, or catheter-associated colonization [26]. This limitation is especially relevant because catheter-collected specimens were substantially more frequent in the diabetes group. Accordingly, the study describes bacterial isolates recovered from positive urine cultures and should not be interpreted as estimating the incidence or clinical severity of UTI.

Third, a stable patient identifier and complete culture dates were not available in the analysed dataset. Some patients may have contributed more than one culture, potentially over-representing recurrent episodes and resistant organisms and violating the statistical assumption of independent observations. The present results should therefore be regarded as culture-record-level findings unless patient-level deduplication can be completed.

Fourth, antimicrobial susceptibility testing was not uniform across all isolates. Antibiotic-specific denominators were sometimes small, particularly in the diabetes subgroup, resulting in imprecise resistance estimates and limited statistical power. The nitrofurantoin and ertapenem findings are especially sensitive to this limitation. Fifth, confirmatory data on extended-spectrum beta-lactamase production, carbapenemase production, minimum inhibitory concentrations, and the laboratory interpretive standard used during each study year were unavailable. Changes in laboratory methods or breakpoints during the study period may have introduced additional variability.

Sixth, potentially important confounders—including previous antimicrobial exposure, recent hospitalization, residence in a long-term care facility, recurrent UTI, urinary tract obstruction, urinary stones, neurogenic bladder, urological procedures, renal function, and glycaemic control—could not be incorporated into the analysis. HbA1c measurements were available for only a small subset of patients and were therefore insufficient for evaluating whether glycaemic control was associated with pathogen distribution or resistance. Consequently, an independent association between diabetes and antimicrobial resistance cannot be established from the current dataset. Finally, multiple organism and antimicrobial comparisons were performed without adjustment for multiplicity. Statistically significant findings should therefore be considered exploratory, particularly when based on small tested denominators.

Future research

Future studies should use a patient-level design with a stable anonymized identifier and complete culture dates, applying a predefined deduplication rule such as the first eligible isolate per patient or the first isolate within a specified surveillance interval. Prospective or enhanced retrospective data collection should distinguish symptomatic UTI, asymptomatic bacteriuria, and catheter-associated infection and should record infection site, community versus healthcare acquisition, recent hospitalization, previous antimicrobial exposure, recurrent infection, urinary tract abnormalities, urological interventions, renal function, and diabetes control. Susceptibility testing should follow a consistent CLSI or EUCAST standard, with explicit reporting of antibiotic-specific denominators and confirmatory ESBL and carbapenemase testing. Larger multicentre cohorts would permit separate evaluation of individual chronic diseases and adequately adjusted patient-level regression analyses. Such studies could determine whether diabetes independently predicts resistant urinary isolates or primarily acts as a marker of advanced age, multimorbidity, catheterization, and healthcare exposure.

Conclusions

Among adults with positive urine cultures, *E. coli* was the predominant bacterial organism across all comorbidity groups, although its proportional frequency was lower among records from patients with diabetes and other chronic diseases than among those with no documented comorbidity. The diabetes group had greater representation of *Klebsiella* spp. and *P. aeruginosa* and higher *E. coli* resistance to gentamicin and nitrofurantoin. However, this group was older and more frequently catheterized, and the culture-record design could not distinguish symptomatic UTI from asymptomatic bacteriuria or colonization. The findings support comorbidity-aware interpretation of local resistance data and individualized urological risk assessment but do not establish diabetes as an independent predictor of antimicrobial resistance.

Declarations

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Author Contribution

B.C. conceived and designed the study and supervised the research. S.A. and S.G. collected and curated the clinical and microbiological data. S.A., S.G., and B.C. contributed to the study methodology, data interpretation, and critical evaluation of the findings. S.A. and B.C. drafted the main manuscript text. S.G. and B.C. critically revised the manuscript for important intellectual content. B.C. prepared the tables and figures. All authors read and approved the final manuscript and agreed to be accountable for all aspects of the work.

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Figures

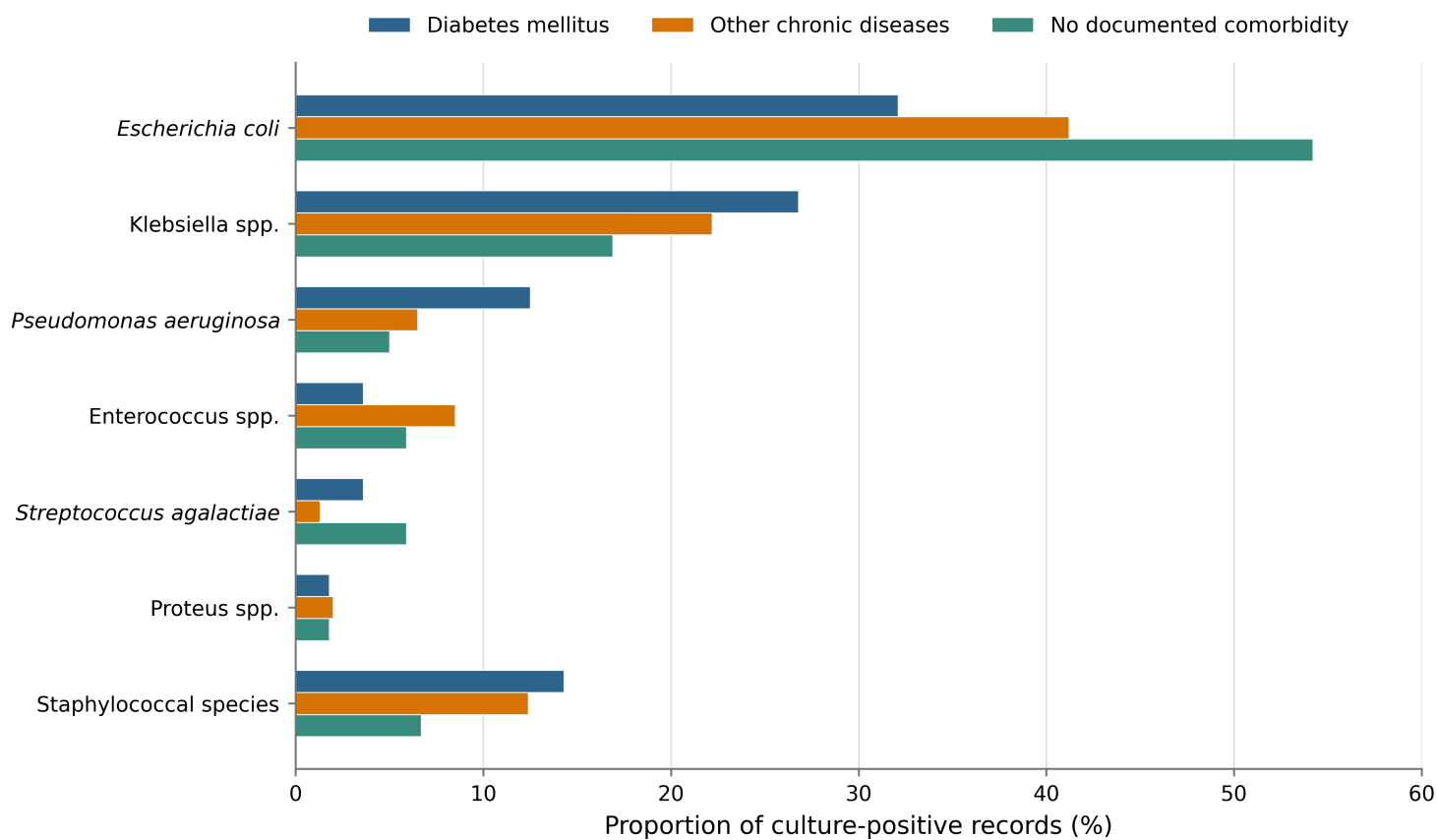


Figure 1

Uropathogen Distribution



Figure 2

Ecoli Resistance Heat map