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# Antimicrobial Utilization and Uropathogen Profile in Diabetic versus Non-Diabetic Patients with Urinary Tract Infection: A Retrospective Comparative Study from a Tertiary Care Centre in South India

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**ABSTRACT**

Urinary tract infection (UTI) remains one of the commonest reasons for antibiotic prescribing, and diabetes mellitus is widely held to alter both the organisms responsible and the way these infections behave. We set out to compare how antibiotics were used and which pathogens were recovered in diabetic and non-diabetic patients treated for UTI at a tertiary nephrology centre. Case records of 140 adults diagnosed with UTI at Abirami Kidney Care Centre, Erode, were reviewed retrospectively. Demographic data, presenting symptoms, urine culture findings, prescribed antimicrobials, whether therapy was modified after sensitivity reporting, and the eventual outcome were abstracted onto a structured form, and the cohort was split by diabetic status. Women outnumbered men, and patients older than sixty made up the largest single age band (38.6%). Type 2 diabetes accounted for almost all of the diabetic cases (36.4% of the whole group versus 3.6% with type 1). Cefoperazone–sulbactam was the antibiotic chosen most often (33%), followed by piperacillin–tazobactam (13%) and levofloxacin (12%), reflecting a heavy reliance on broad-spectrum agents. Where an organism was grown, Escherichia coli predominated (21.4%), with Klebsiella (7.9%) and Pseudomonas aeruginosa (5.7%) next; just over half of the samples (53.6%) yielded no growth. Empirical therapy was left unchanged in roughly two-thirds of patients after culture, clinical improvement was recorded in 95.7%, and recurrence affected 17.1%. The findings reinforce the dominance of E. coli and the routine use of broad-spectrum cover, and they argue for tighter culture-led de-escalation as a practical stewardship target in this setting.

**Keywords:** Urinary tract infection; diabetes mellitus; antimicrobial utilization; uropathogens; Escherichia coli; antimicrobial stewardship.

## 1. INTRODUCTION

A urinary tract infection is any infective process that takes hold along the urinary system – the kidneys, the ureters, the bladder or the urethra – once pathogenic microbes settle and multiply within the urine or the tissues that line the tract, usually with symptoms to match.[1] By convention these infections are split anatomically. Lower-tract disease is confined to the bladder and urethra; it is uncomfortable and disruptive but seldom dangerous, with cystitis typically producing dysuria, frequency, urgency and suprapubic discomfort once organisms such as E. coli attach to the bladder lining through their fimbriae.[2,3] Upper-tract disease reaches the kidney and presents as acute pyelonephritis, marked by flank pain, high fever with rigors and systemic upset; left untreated it can scar the kidney or tip into sepsis.[4]

Globally UTIs are among the most frequent bacterial infections, with somewhere between 150 and 200 million episodes estimated each year, and women bear the larger share because of their shorter urethra and its closeness to the perineum.[5] India carries a particularly heavy load: a dense population, uneven access to clean water and sanitation, a very large diabetic population and the widespread, often unsupervised use of antibiotics have together pushed resistance to worrying levels, with

extended-spectrum beta-lactamase (ESBL) producers now common in routine isolates.[6,7] Recurrence is frequent, and a drift towards complicated infections has increased reliance on costly reserve drugs such as the carbapenems.[8]

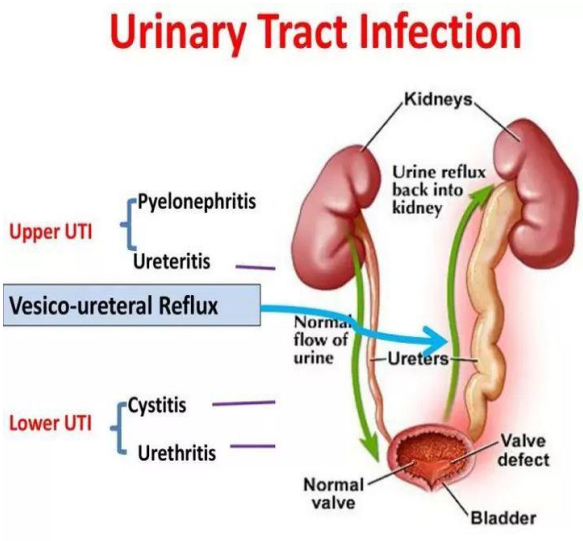


**Figure - 1: Anatomy of the urinary tract.**

Globally UTIs are among the most frequent bacterial infections, with somewhere between 150 and 200 million episodes estimated each year, and women bear the larger share because of their shorter urethra and its closeness to the perineum.[5] India carries a particularly heavy load: a dense population, uneven access to clean water and sanitation, a very large diabetic population and the widespread, often unsupervised use of antibiotics have together pushed resistance to worrying levels, with extended-spectrum beta-lactamase (ESBL) producers now common in routine isolates.[6,7] Recurrence is frequent, and a drift towards complicated infections has increased reliance on costly reserve drugs such as the carbapenems.[8]

Diabetes is repeatedly singled out as an independent risk factor for UTI, and the reasons are biological rather than coincidental. Sustained hyperglycaemia blunts neutrophil function – chemotaxis, phagocytosis and intracellular killing are all impaired – and dampens the cytokine signals needed to mount a brisk inflammatory response.[9,10] Glucose spilling into the urine once the renal threshold is crossed turns the urinary tract into a nutrient-rich medium that favours bacterial growth and biofilm formation, while autonomic neuropathy leaves urine pooling in an incompletely emptied bladder.[11,12] The net effect is that diabetic patients tend to develop more severe, more often recurrent and more frequently complicated infections, and they are more likely to harbour resistant or non-E. coli organisms.[13]

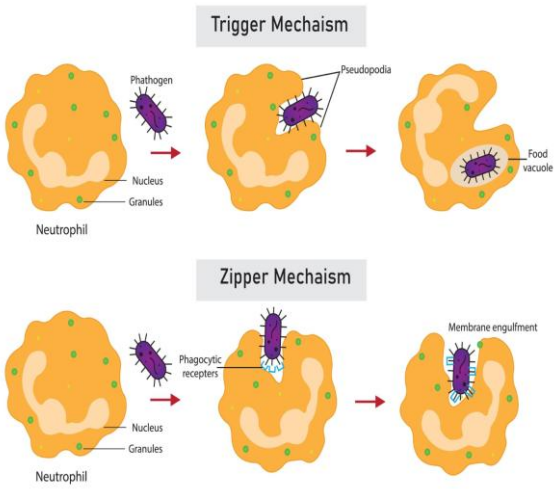


**Figure - 2: Neutrophil phagocytosis (trigger and zipper mechanisms) impaired in diabetes.**

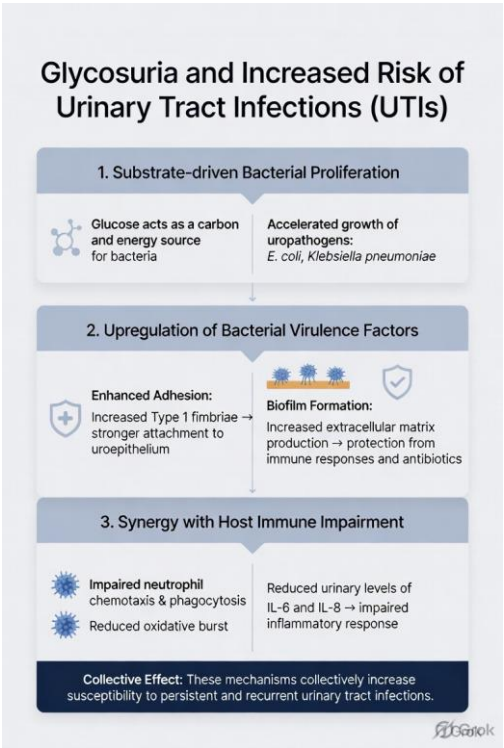


**Figure - 3: How glycosuria predisposes to urinary tract infection.**

Antibiotic treatment of UTI is usually a two-step affair: empirical cover is started at once on the strength of the likely organism and local resistance data, then narrowed once culture and susceptibility results return.[14] In practice the second step is often skipped. Broad-spectrum agents are reached for even in straightforward cases, courses run longer than necessary, and asymptomatic bacteriuria is sometimes treated as though it were symptomatic disease – all of which feed resistance.[15] Most published work has examined either the spectrum of organisms or their resistance patterns, and relatively few

studies have looked at diabetic and non-diabetic patients side by side in the same Indian setting. That gap, together with the practical need to inform empirical prescribing locally, prompted the present comparison.[16]

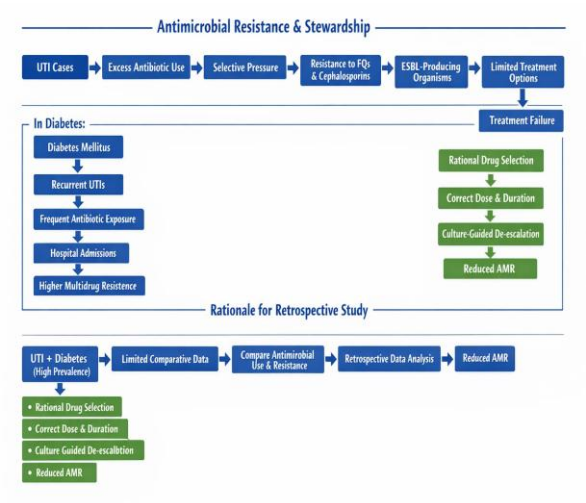


**Figure - 4: Antimicrobial resistance and the rationale for stewardship in UTI.**

### 1.1. Aim and objectives

The study aimed to evaluate and compare antimicrobial utilization and the uropathogen profile in diabetic and non-diabetic patients with UTI at a tertiary care hospital. Specific objectives were to identify the organisms recovered in each group, to describe resistance and prescribing patterns, to judge how appropriate empirical and culture-guided choices were, and to record clinical outcomes such as improvement and recurrence.

## 2. MATERIALS AND METHODS

### 2.1. Study design and setting

This was a retrospective, comparative, observational study carried out at Abirami Kidney Care Centre, Erode – a tertiary referral unit offering both nephrology and urology services to inpatients and outpatients. The protocol was cleared by the Institutional Ethics Committee, and because only existing records were examined the requirement for individual consent was waived; no personal identifiers were extracted and patient anonymity was preserved throughout.

### 2.2. Patients

Records of adults treated for UTI during the study period were retrieved from the Medical Records Department. A diagnosis of UTI was accepted when documented symptoms – dysuria, frequency, urgency, suprapubic or flank pain, or fever – were supported by urine routine examination and a urine culture and sensitivity report. Patients were eligible if they were at least eighteen years old, had a confirmed diagnosis with an available culture report, and had complete, retrievable notes. Records were excluded where notes were incomplete, where no culture sensitivity report existed, or where the patient was on long-term prophylactic antibiotics. After screening, 140 patients met all criteria and formed the final cohort.

For the comparison the cohort was divided into two groups: Group A comprised patients with a documented diagnosis of diabetes mellitus or on antidiabetic treatment at the time of their UTI, and Group B comprised those with no record of diabetes.

### 2.3. Data collection

A structured proforma was used to capture, for each patient, demographic details (age, sex, inpatient or outpatient status), presenting symptoms and comorbidities, urine routine findings, culture results with the organism isolated, the susceptibility profile, and full prescribing information – the antibiotic used, its dose, route and duration, and whether the choice was revised once sensitivity results were available. Outcomes recorded were clinical improvement and any recurrence noted during the study window. Results were summarised as frequencies and percentages.

## 3. RESULTS

### 3.1. Sex distribution

Of the 140 patients, women clearly outnumbered men. Non-diabetic women were the single largest group at 40.71%, with non-diabetic men close behind at 36.43%; diabetic women and diabetic men made up 12.86% and 10.00% respectively (Table 1, Figure 5). The pattern is consistent with the well-recognised female predominance in UTI.

**Table - 1: Sex distribution of patients (n = 140).**

| Category | No. of subjects | Percentage (%) |
|---|---|---|
| Diabetic – Male | 14 | 10.00 |
| Diabetic – Female | 18 | 12.86 |
| Non-diabetic – Male | 51 | 36.43 |
| Non-diabetic – Female | 57 | 40.71 |
| **Total** | **140** | **100** |

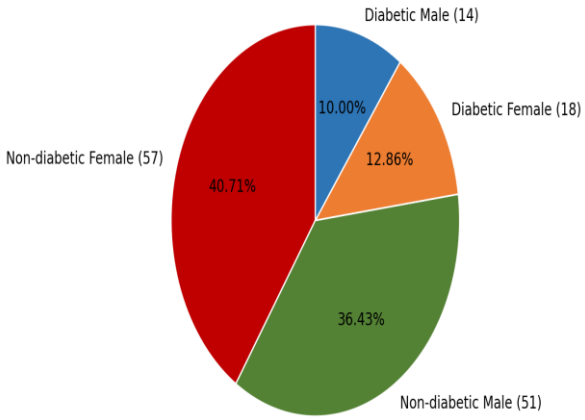


*Figure 5: Sex distribution of patients.*

### 3.2. Age distribution

Infection was most common in older patients. Those above sixty accounted for 38.6% of cases, the largest band by some margin, followed by the 30–39 year group at 20.0%. The 50–59, 19–29 and 40–49 year groups contributed 12.1%, 11.4% and 11.4% respectively, while only 2.1% fell in the 12–18 year range (Table 2, Figure 6). The skew towards the elderly likely reflects waning immunity, accumulating comorbidity and structural changes in the ageing urinary tract.

**Table 2: Age distribution of patients (n = 140).**

| Age group (years) | No. of subjects | Percentage (%) |
|---|---|---|
| 12–18 | 3 | 2.1 |
| 19–29 | 16 | 11.4 |
| 30–39 | 28 | 20.0 |
| 40–49 | 16 | 11.4 |
| 50–59 | 17 | 12.1 |
| >60 | 54 | 38.6 |
| **Total** | **140** | **100** |

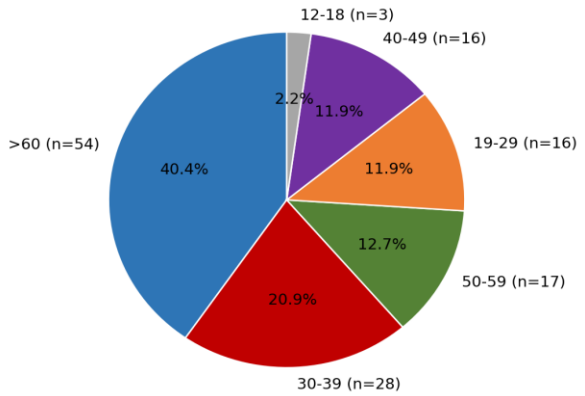


**Figure - 6: Age distribution of patients.**

### 3.3. Inpatient and outpatient distribution

Most infections were managed in hospital. Non-diabetic inpatients formed the largest segment at 48.57% (68 patients), with diabetic inpatients next at 35.00% (49 patients). Outpatients were far fewer – 12.14% non-diabetic and only 4.29% diabetic (Table 3, Figure 7). The concentration of cases among inpatients fits a tertiary referral setting where more severe or complicated infections present.

**Table - 3: Inpatient versus outpatient distribution (n = 140).**

| Category | No. of subjects | Percentage (%) |
|---|---|---|
| Diabetic – Inpatient | 49 | 35.00 |
| Diabetic – Outpatient | 6 | 4.29 |
| Non-diabetic – Inpatient | 68 | 48.57 |
| Non-diabetic – Outpatient | 17 | 12.14 |
| **Total** | **140** | **100** |

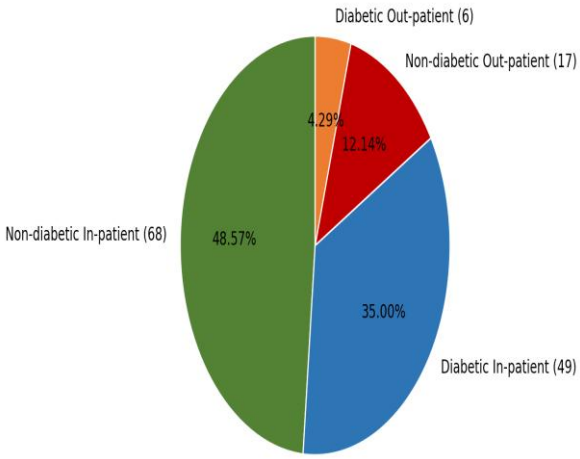


**Figure - 7: Distribution of diabetic and non-diabetic patients by care setting.**

### 3.4. Antimicrobial utilization

Prescribing leaned firmly towards broad-spectrum cover. The combination of cefoperazone and sulbactam was used most often, in a third of patients (33%), followed by piperacillin–tazobactam (13%) and levofloxacin (12%). Ceftriaxone (6%), meropenem (5%), amikacin (3%) and cefixime (3%) accounted for smaller shares, and a miscellaneous group of other agents made up the remaining 25% (Table 4, Figure 8). The reliance on potent combination beta-lactams

is typical of empirical treatment begun before culture results are to hand.

**Table 4: Antibiotics prescribed to patients.**

| Antibiotic | Percentage (%) |
|---|---|
| Cefoperazone + Sulbactam | 33 |
| Piperacillin + Tazobactam | 13 |
| Levofloxacin | 12 |
| Ceftriaxone | 6 |
| Meropenem | 5 |
| Amikacin | 3 |
| Cefixime | 3 |
| Others | 25 |
| **Total** | **100** |

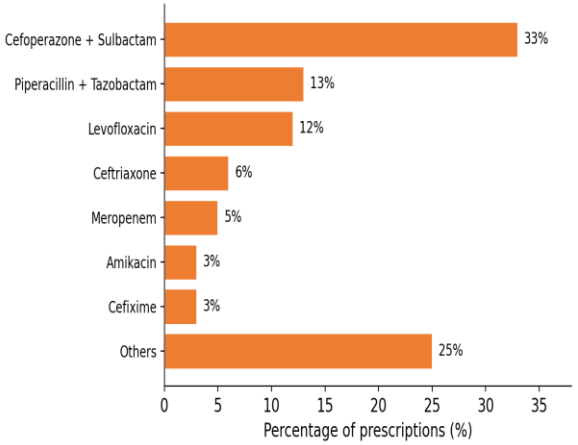


**Figure - 8: Antibiotics used in patients.**

### 3.5. Type of diabetes

Among the whole cohort, 60.0% had no diabetes. Within the diabetic patients, type 2 disease dominated almost entirely – 36.4% of the total – against just 3.6% with type 1 (Table 5, Figure 9), mirroring the relative frequency of the two types in the general population and the strong association of type 2 diabetes with infection.

**Table - 5: Distribution by type of diabetes (n = 140).**

| Type of diabetes | No. of subjects | Percentage (%) |
|---|---|---|
| None | 84 | 60.0 |
| Type 1 | 5 | 3.6 |
| Type 2 | 51 | 36.4 |
| **Total** | **140** | **100** |

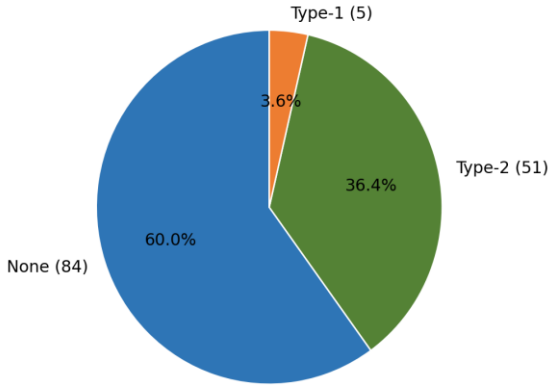


**Figure - 9: Types of diabetes among patients.**

### 3.6. Modification of therapy after culture

In 49 patients (34.7%) the antibiotic was switched once culture and sensitivity results were available, while in the remaining 91 (65.3%) the original empirical regimen was carried through unchanged (Table 6, Figure 10). That the initial choice held in roughly two-thirds of cases suggests empirical prescribing was broadly appropriate, though it also leaves clear room for more deliberate culture-led de-escalation.

**Table - 6: Antibiotic therapy changed after culture (n = 140).**

| Antibiotic changed after culture | No. of subjects | Percentage (%) |
|---|---|---|
| Yes | 49 | 34.7 |
| No | 91 | 65.3 |
| **Total** | **140** | **100** |

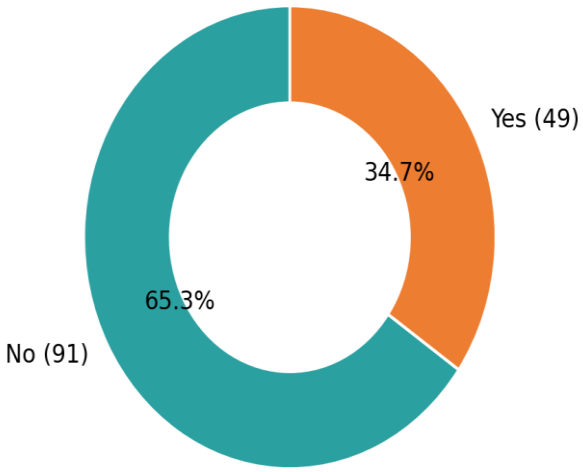


**Figure - 10: Proportion of patients whose antibiotic was changed after culture.**

### 3.7. Uropathogen profile

No organism was isolated in 53.6% of samples – a sizeable fraction that may reflect prior antibiotic exposure, low colony counts or the limits of routine culture. Where a pathogen did grow, Escherichia coli was easily the most common at 21.4%, followed by Klebsiella species (7.9%) and Pseudomonas aeruginosa (5.7%). Staphylococcus aureus (5.0%) and Enterococcus (4.3%) appeared less often, with a handful of rarer isolates making up the remainder (Table 7, Figure 11). The dominance of E. coli is in line with the wider UTI literature.

**Table - 7: Frequency of organisms isolated (n = 140).**

| Organism | No. of subjects | Percentage (%) |
|---|---|---|
| No growth | 75 | 53.6 |
| E. coli | 30 | 21.4 |
| Enterococcus | 6 | 4.3 |
| Staphylococcus aureus | 7 | 5.0 |
| Klebsiella | 11 | 7.9 |
| Bacillus | 1 | 0.7 |
| Pseudomonas aeruginosa | 8 | 5.7 |
| Klebsiella pneumoniae | 1 | 0.7 |
| Others | 1 | 0.7 |
| **Total** | **140** | **100** |

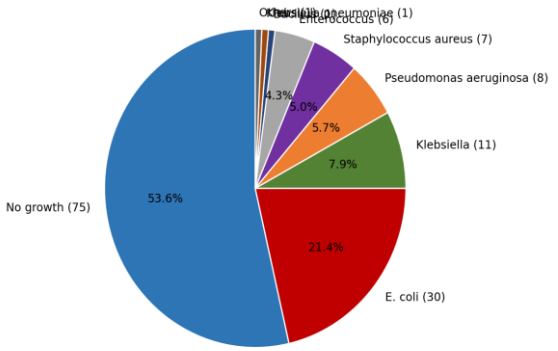


**Figure - 11: Distribution of organisms isolated from patients.**

### 3.8. Clinical improvement

Treatment was effective in the great majority of patients. Clinical improvement was documented in 134 of 140 (95.7%), and only 6 patients (4.3%) failed to improve over the treatment period (Table 8, Figure 12).

**Table - 8: Clinical improvement among patients (n = 140).**

| Clinical improvement | No. of subjects | Percentage (%) |
|---|---|---|
| Yes | 134 | 95.7 |
| No | 6 | 4.3 |
| **Total** | **140** | **100** |

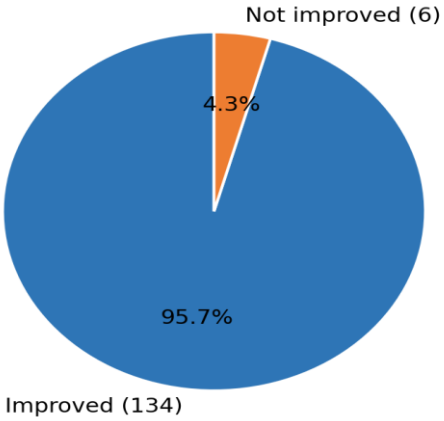


**Figure - 12: Frequency of clinical improvement.**

### 3.9. Recurrence

Recurrent infection was seen in 24 patients (17.1%) during the study period, while 116 (82.9%) had no recurrence (Table 9, Figure 13). Recurrence in this minority may relate to diabetes, incomplete clearance of the original infection or underlying structural abnormality.

**Table 9: Frequency of recurrent UTI (n = 140).**

| Recurrent UTI | No. of subjects | Percentage (%) |
|---|---|---|
| Yes | 24 | 17.1 |
| No | 116 | 82.9 |
| **Total** | **140** | **100** |

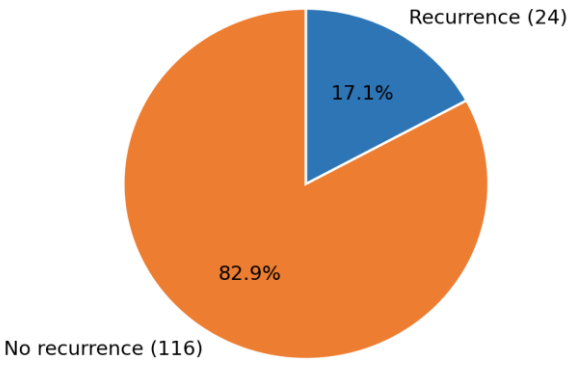


**Figure - 13: Frequency of recurrent UTI.**

## 4. DISCUSSION

This review of 140 UTI episodes set out to see how prescribing and the organisms recovered differed between diabetic and non-diabetic patients in a single tertiary unit. The headline demographic findings – a female predominance and a peak in the elderly – echo what has been reported repeatedly elsewhere. Women accounted for the bulk of cases, which is usually explained by the shorter female urethra and its proximity to the anus, and patients over sixty formed the largest age band, plausibly because immunity wanes and comorbidities and urinary tract abnormalities accumulate with age.

Hospitalised patients made up most of the cohort, with non-diabetic inpatients the largest group, a distribution that suits a referral centre handling more severe disease and catheter-associated infection. Diabetes was present in 40% of patients, and almost all of these had type 2 disease – again consistent with its far greater population frequency and its recognised tendency to predispose to infection through impaired immunity and glycosuria.

The prescribing picture was dominated by broad-spectrum agents, led by cefoperazone–sulbactam, with piperacillin–tazobactam and levofloxacin next. This is understandable when empirical therapy must be started before sensitivities are known, particularly in sicker inpatients, but heavy front-line use of such agents is exactly the pressure that drives ESBL emergence, and it underlines the value of structured de-escalation. In this cohort the empirical choice was revised after culture in about a third of patients and continued unchanged in the rest, which suggests the initial selection was usually reasonable while still leaving scope for more active narrowing once results return.

Microbiologically, E. coli was the predominant isolate, with Klebsiella and Pseudomonas following – a hierarchy that matches the established UTI literature. The high proportion of culture-negative samples is notable and probably reflects earlier antibiotic use, low bacterial loads or the inherent limitations of routine culture. Outcomes were good: nearly all patients improved and only about one in six experienced a recurrence, the latter again likely tied to diabetes, incomplete eradication or anatomical factors.

## 5. LIMITATIONS

- The retrospective design relied on previously recorded hospital data, so a causal link between diabetes and resistance could not be established.
- Data came from a single tertiary centre, which limits how widely the results can be generalised, and the sample was relatively small.
- Some records held incomplete clinical information, and glycaemic control measures such as HbA1c were not available for analysis.
- Long-term follow-up was not possible, and confounders such as prior antibiotic exposure, catheterisation and other comorbidities could not be fully accounted for.

## 6. CONCLUSION

In this tertiary care cohort, UTI fell most heavily on women and on older patients, and Escherichia coli was the leading pathogen wherever an organism could be grown. Treatment leaned strongly on broad-spectrum antibiotics, and although empirical therapy worked in most patients, culture-guided narrowing was applied in only a minority. Early diagnosis, appropriate empirical selection informed by local data, and disciplined culture-led de-escalation remain the practical levers for improving outcomes and slowing resistance – priorities that apply with particular force to diabetic patients.

## 7. FUTURE DIRECTIONS

- Prospective, multicentre studies with larger samples would strengthen external validity and statistical power.
- Correlating glycaemic control (HbA1c) with resistance, and adding molecular detection of resistance genes, would sharpen the diabetes–resistance question.
- Longer follow-up, formal evaluation of stewardship interventions and pharmacoeconomic analysis would help translate findings into diabetes-specific treatment guidance for UTI.

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