

Frontiers in Multidisciplinary Research and Studies

journal homepage: <https://openj.edwiserinternational.com/index.php/fmrs>

Review Article

Type 2 Diabetes Mellitus and Urinary Tract Infection: Epidemiology, Pathophysiology, Diagnosis, Antimicrobial Resistance and Contemporary Management

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ARTICLE INFO

Received: 29 July 2026

Revised: 30 August 2026

Available Online: 18 September 2026

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ABSTRACT

Urinary tract infection (UTI) is an important infectious complication in people with type 2 diabetes mellitus (T2DM), with risk influenced by hyperglycaemia, impaired innate immunity, glycosuria, autonomic neuropathy, bladder dysfunction, renal disease and recurrent antimicrobial exposure. The available evidence indicates that the burden is heterogeneous across populations, but a systematic review and meta-analysis involving 827,948 participants estimated an overall UTI prevalence of 11.5% among people with T2DM. Diabetes is also associated with increased asymptomatic bacteriuria (ASB), recurrent infection, upper urinary tract involvement and antimicrobial-resistant organisms. *Escherichia coli* remains the leading uropathogen, while *Klebsiella*, *Proteus*, *Enterobacter*, *Enterococcus* and *Pseudomonas* are clinically important in selected patients; extended-spectrum β -lactamase (ESBL)-producing and other multidrug-resistant organisms increasingly complicate treatment. Diagnosis should integrate symptoms, physical examination, urinalysis and, particularly in patients with risk factors for complicated or systemic infection, urine culture with susceptibility testing. Current guidance does not support routine screening or treatment of ASB in people with diabetes, except in specific circumstances such as pregnancy or selected urological procedures. Management of symptomatic infection requires appropriate antimicrobial selection, renal-function assessment, source control and attention to local resistance patterns. Prevention depends on optimized glycaemic management, correction of urinary obstruction or retention, avoidance of unnecessary catheterization and antibiotics, and patient education. SGLT2 inhibitors deserve particular attention: current evidence does not demonstrate a consistent increase in severe UTI, although genital infections are more clearly associated with the drug class. Future research should define risk-stratified diagnostic pathways, improve surveillance of resistant uropathogens and evaluate non-antibiotic preventive strategies.

Keywords: Type 2 Diabetes Mellitus; Urinary Tract Infection; Recurrent UTI; Asymptomatic Bacteriuria; Glycosuria; Antimicrobial Resistance; *Escherichia Coli*; SGLT2 Inhibitors.

Introduction

Type 2 diabetes mellitus is a chronic metabolic disorder characterized predominantly by insulin resistance together with progressive β -cell dysfunction and persistent hyperglycaemia. Beyond its vascular and neurological complications, diabetes alters host susceptibility to infection. Urinary tract infection is particularly relevant because the urinary tract depends on coordinated mechanical, epithelial and immune defences, all of which can be affected by diabetes. The project on which this review is based describes UTI as a clinically important complication of T2DM and identifies glycosuria, defective neutrophil function, autonomic neuropathy, urinary stasis and diabetic nephropathy as major biological links [1].

UTIs range from localized bladder infection to systemic infection such as pyelonephritis and urosepsis. The traditional terminology of “uncomplicated” and “complicated” UTI remains widely used in the literature and is retained where it is necessary for comparison with older studies. However, the European Association of Urology (EAU) has moved toward a clinically oriented classification distinguishing localized UTI from systemic UTI while separately assessing patient risk factors that can predict treatment failure or a difficult course. Diabetes, renal impairment, urinary tract abnormalities, previous antibiotic exposure, resistant organisms and urinary catheters are among these risk factors [2].

The clinical importance of UTI in T2DM extends beyond dysuria and frequency. Recurrent or inadequately treated infection may progress to pyelonephritis, renal abscess, emphysematous infection, bacteremia and urosepsis. At the same time, indiscriminate treatment of bacteriuria can increase antimicrobial exposure and accelerate resistance. The central clinical challenge is therefore to identify symptomatic infection promptly, distinguish it from ASB, select therapy using microbiological and patient-specific information, and reduce recurrence without unnecessary antibiotic use.

Review Methodology

This narrative review was developed from the through web-based searches of PubMed-indexed literature, the Infectious Diseases Society of America (IDSA), EAU guidance and American Diabetes Association (ADA) Standards of Care. The project provided the foundational discussion of epidemiology, pathophysiology, symptoms, diagnosis, antimicrobial resistance and management, while contemporary sources were used to update areas in which terminology or evidence has evolved. The source

project itself emphasizes early diagnosis, culture-guided antimicrobial therapy, glycaemic control, antimicrobial stewardship and multidisciplinary care [3].

The review is narrative rather than a formal systematic review; therefore, no quantitative meta-analysis was performed by the authors. Where numerical estimates are reported, they are explicitly attributed to the cited study or meta-analysis. Statements from the project that reflect older guidelines have been reconciled with newer guidance where necessary, particularly for ASB and UTI classification.

Epidemiology and Clinical Burden

The frequency of UTI in T2DM varies substantially according to population, sex, age, setting, definition of infection and whether culture-confirmed or clinically diagnosed infection is measured. The project summarizes several large database studies showing higher UTI incidence among people with diabetes than among matched non-diabetic controls and a pronounced sex difference, with women consistently experiencing a greater burden [4].

A 2022 systematic review and meta-analysis of 15 studies including 827,948 participants estimated the overall prevalence of UTI among people with T2DM at 11.5% (95% CI 7.8–16.7%). The authors also reported relationships with age and study characteristics, illustrating why single-center prevalence figures should not be generalized to all diabetic populations [5].

ASB is also more frequent in diabetes. The project reports estimates of approximately 8–26% across studies and cites a meta-analysis showing a point prevalence of 12.2% among diabetic participants compared with 4.5% in healthy controls. It also notes that prevalence increases with longer diabetes duration [6]. Importantly, ASB should not be equated with symptomatic UTI and should not automatically trigger antibiotic treatment. The 2019 IDSA guideline strongly recommends against screening for or treating ASB in patients with diabetes because available evidence does not show clinical benefit and unnecessary therapy promotes antimicrobial resistance [7].

Upper urinary tract disease may be more severe in diabetes. The project summarizes evidence of higher hospitalization rates for acute pyelonephritis in diabetic women and men and highlights increased risk of prostatitis and prostatic complications in diabetic men. These observations support a lower threshold for careful evaluation when systemic symptoms, renal

impairment, urinary obstruction or recurrent infection are present.

Table 1: Epidemiology and clinical implications of UTI in T2DM.

Epidemiological feature	Key evidence	Clinical implication
Overall UTI prevalence in T2DM	11.5% pooled prevalence in 15 studies; 827,948 participants	UTI represents a substantial comorbidity burden.
Sex	Women have consistently higher rates than men	Female sex should be incorporated into risk assessment.
ASB	Reported approximately 8–26% in diabetes; pooled estimates exceed healthy controls	Do not treat bacteriuria alone in most nonpregnant diabetic patients.
Resistant organisms	ESBL and other MDR pathogens are increasingly reported	Culture and susceptibility testing are important in high-risk cases.
Severe infection	Diabetes is associated with greater risk of upper-tract and systemic complications	Early recognition of pyelonephritis and sepsis is essential.

Pathophysiology: Why Diabetes Predisposes to UTI

The relationship between T2DM and UTI is multifactorial. No single mechanism explains the increased susceptibility. Instead, metabolic, immune, neurological, vascular and urological abnormalities interact to impair bacterial clearance and increase the probability that colonization will progress to symptomatic infection.

Hyperglycaemia and Glycosuria

Persistent hyperglycaemia increases urinary glucose when the renal threshold is exceeded. Glycosuria has traditionally been proposed to provide a nutrient-rich environment that facilitates bacterial growth. The project identifies urinary glucose as a potential contributor but also appropriately notes that HbA1c is not consistently associated with UTI in every study. Thus, glycaemic control is biologically and clinically important, but UTI risk should not be reduced to HbA1c alone.

Impaired Innate Immunity

Diabetes can impair neutrophil chemotaxis, phagocytosis and intracellular killing and may alter cytokine responses. The project cites reduced urinary interleukin-6 and interleukin-8 in diabetic patients with ASB, supporting the concept of altered local immune surveillance. These abnormalities may allow uropathogens to persist despite apparently modest local symptoms.

Autonomic Neuropathy and Diabetic Bladder Dysfunction

Diabetic autonomic neuropathy may produce impaired detrusor activity, increased post-void residual volume and urinary retention. Reduced urinary flushing removes an important mechanical defence and creates a niche in which bacteria can multiply. The project reports that bladder dysfunction may occur in a substantial proportion of diabetic women and recommends considering bladder dysfunction in diabetic patients with UTI. In recurrent infection, assessment for incomplete emptying or other functional abnormalities is therefore clinically relevant.

Nephropathy, Microvascular Disease and Structural Factors

Diabetic nephropathy and vascular disease may reduce tissue resilience and coexist with chronic kidney disease, which in turn affects drug selection and dosing. Obstruction, stones, prostatic enlargement, urinary tract instrumentation and indwelling catheters provide additional risk. These factors are particularly important because successful management may require source control rather than escalation of antibiotics alone.

Bacterial Adhesion, Intracellular Persistence and Biofilm

Uropathogenic bacteria first colonize the periurethral region and bladder and then adhere to uroepithelial cells using adhesins. Uropathogenic *E. coli* can form biofilms and intracellular bacterial communities, facilitating persistence and recurrence. Catheter-associated infection adds another biofilm surface, with organisms protected within extracellular matrices. The project describes adhesion, biofilm formation and

ascending infection as central steps in progression from lower UTI to pyelonephritis and bacteremia [8].

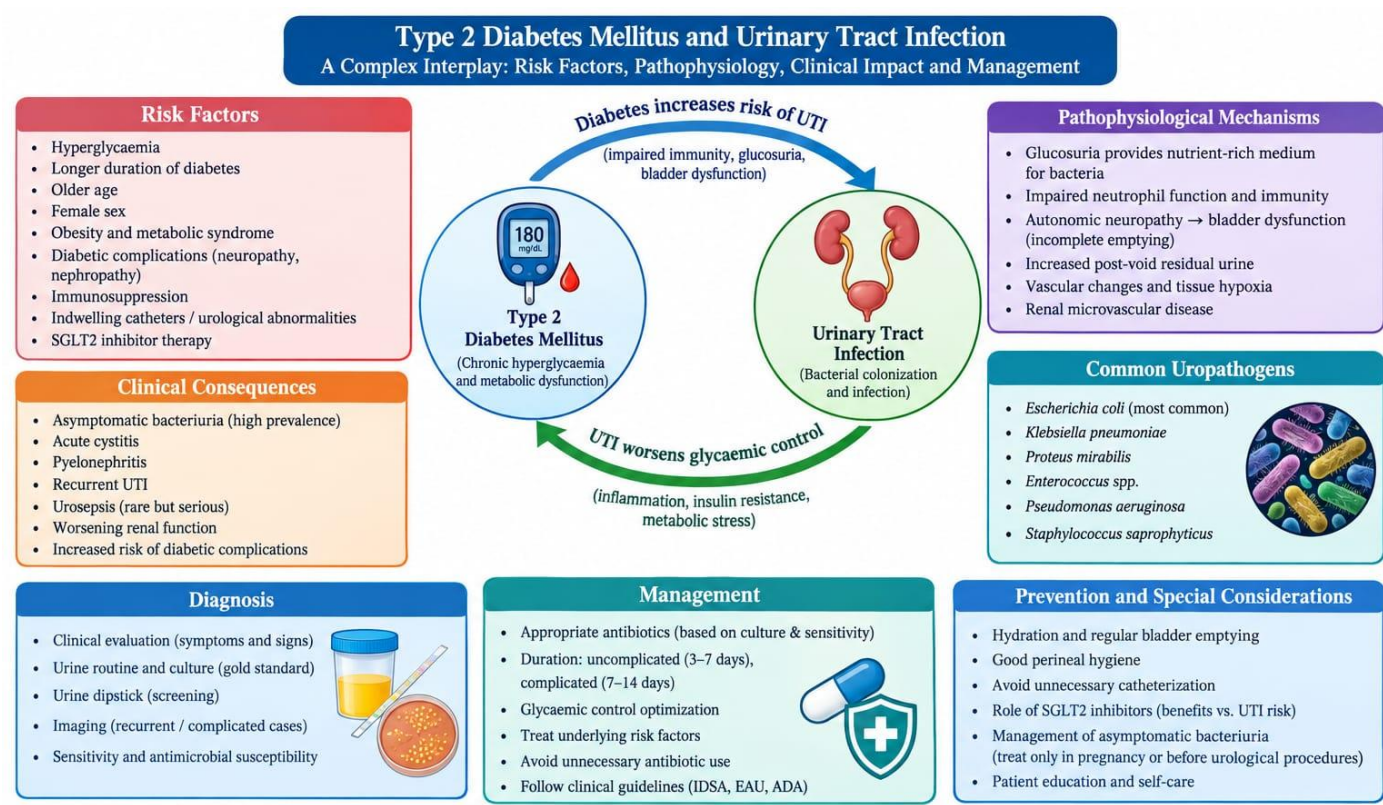


Figure 1: Conceptual model of the pathophysiological continuum linking T2DM to recurrent and systemic UTI.

Risk Factors for UTI in T2DM

Risk factors can be grouped into patient-related, diabetes-related, urinary tract and treatment-related factors. The project repeatedly identifies female sex, older age, longer duration of diabetes, poor metabolic control, obesity, nephropathy, previous UTI and neuropathy as important contributors [9]. Contemporary EAU guidance similarly emphasizes diabetes, renal impairment, urinary tract abnormalities, residual urine, prior antibiotics, catheters and resistant organisms as risk factors for a difficult course [10].

Causative Microorganisms and Antimicrobial Resistance

E. coli remains the dominant uropathogen in diabetic patients, but the microbial spectrum is broader than in otherwise healthy women with uncomplicated cystitis. *Klebsiella* spp., *Proteus* spp., *Enterobacter* spp., *Enterococcus* spp. and *Pseudomonas* spp. become more relevant with recurrent infection, healthcare exposure, urinary abnormalities or catheterization. The project specifically emphasizes ESBL-producing Enterobacterales, fluoroquinolone-resistant organisms, carbapenem-resistant Enterobacterales and

vancomycin-resistant enterococci as important resistance phenotypes [11].

Indian data illustrate the potential scale of resistance. A cross-sectional study of 504 adults with diabetes in a tertiary hospital in eastern India reported a high culture-defined UTI burden and identified *E. coli* as the leading isolate; resistance was substantial, reinforcing the importance of local antibiograms rather than importing susceptibility assumptions from other countries [12].

A 2024 study of 500 diabetic patients reported UTI in 37.2% and found that uncontrolled glycaemia was associated with more infections. Among *E. coli* isolates, 58.4% were multidrug resistant. The study should not be interpreted as a population-wide prevalence estimate because it represents a selected study population, but it demonstrates the clinically important intersection of glycaemic control and antimicrobial resistance [13].

Antimicrobial resistance is promoted by repeated antibiotic exposure, treatment of ASB, inadequate adherence, hospital-acquired infection, catheterization and transmission of resistant clones. The project correctly highlights antimicrobial stewardship as a core component of care [14]. In practice, empiric therapy

should be guided by local resistance patterns and previous culture results, then narrowed or changed when susceptibility results become available.

Clinical Presentation and Differential Diagnosis

Lower UTI commonly presents dysuria, frequency, urgency and suprapubic discomfort, sometimes accompanied by hematuria. Systemic UTI is suggested by fever, chills, flank pain, costovertebral-angle tenderness, nausea or vomiting. The project notes that diabetic neuropathy may blunt typical symptoms, making clinical assessment more challenging [15].

Symptoms alone are not perfectly specific. Dysuria and frequency can occur with vulvovaginitis, sexually transmitted infections, genitourinary atrophy and noninfectious bladder disorders. Conversely, older or neuropathic patients may have atypical presentations. Therefore, clinicians should interpret symptoms in context and use urinalysis and culture when the probability of complicated or systemic infection is meaningful.

Recurrent UTI is commonly defined as at least two symptomatic episodes within six months or at least three within 12 months. Relapse refers to recurrence with the same organism soon after therapy and should prompt consideration of persistent infection, inadequate treatment, anatomic abnormality or a protected bacterial reservoir. [16].

Diagnostic Approach

Urinalysis

Urinalysis provides rapid supportive evidence. Pyuria reflects urinary inflammation but is not synonymous with symptomatic UTI, particularly in patients with ASB or urinary devices. Leukocyte esterase supports the presence of white cells, while nitrite positivity supports infection with nitrate-reducing organisms. A negative nitrite result does not exclude UTI because bacterial burden may be low and several organisms do not reliably produce nitrite[17].

Urine culture and susceptibility testing

Culture is particularly important when diabetes coexists with recurrent infection, systemic features, prior resistant organisms, recent antibiotic exposure, renal impairment, catheterization or suspected pyelonephritis. The submitted project recommends a clean-catch midstream sample before antibiotics in suspected diabetic UTI, with catheter replacement before sampling in patients with long-term indwelling catheters when appropriate. This principle remains

clinically sound, although current management should follow local microbiology protocols and contemporary guideline definitions.

Blood tests and imaging

Blood glucose and HbA1c provide information about metabolic status. In systemic infection, complete blood count, renal function, electrolytes, and inflammatory markers may assist severity assessment and antimicrobial dosing. Blood cultures are appropriate when bacteremia or sepsis is suspected. Imaging is not required for every episode of cystitis, but renal ultrasonography or cross-sectional imaging should be considered when obstruction, stones, abscess, emphysematous infection, persistent fever or treatment failure is suspected.

The diagnostic principle is therefore risk-stratified rather than indiscriminate: confirm infection microbiologically when the likelihood of resistant or complicated disease is high, investigate anatomic or functional abnormalities when recurrence suggests a reservoir, and avoid turning isolated bacteriuria into a diagnosis of symptomatic UTI.

Asymptomatic Bacteriuria: A Critical Distinction

ASB is defined by significant bacterial growth in urine in the absence of urinary symptoms attributable to infection. The presence of pyuria does not by itself justify antimicrobial treatment. Diabetes increases the likelihood of ASB, but randomized evidence has not demonstrated benefit from routine eradication in diabetic adults [18].

The IDSA 2019 guideline therefore recommends screening for or treating ASB in patients with diabetes. Exceptions are situations in which treatment is known to improve outcomes, most notably pregnancy and selected invasive urological procedures. The 2026 EAU guidance similarly states that treatment of ASB is not beneficial in well-regulated diabetes and emphasizes that poorly regulated diabetes is a risk factor for symptomatic UTI and complications [19].

This distinction has major stewardship implications. Treating ASB exposes patients to adverse drug effects and ecological consequences without clear clinical benefit. In diabetic populations, where resistant organisms are already a concern, avoiding unnecessary antibiotics is a key component of prevention.

Management of Symptomatic UTI in T2DM

General Principles

Management should begin with assessment of severity and localization. Patients with localized cystitis can often be treated as outpatients when clinically stable, whereas fever, flank pain, vomiting, hypotension, altered mental status, acute kidney injury or other systemic signs require evaluation for systemic UTI and possible hospital-based management. In diabetic patients, the threshold for culture, renal-function assessment and evaluation for complicating factors is generally lower.

Antibiotic selection should consider the site of infection, previous culture results, local antibiogram, allergy history, renal function, recent antimicrobial exposure, drug interactions and the likelihood of resistant pathogens. Therapy should be narrowed to the most appropriate agent once susceptibility results are available. The project emphasizes culture-guided therapy and stewardship rather than indiscriminate broad-spectrum treatment [20].

Table 2: Clinical Management of UTI in T2DM.

Clinical situation	Core management approach	Important cautions
Localized cystitis	Use an evidence-based oral agent appropriate for local resistance and patient factors; obtain culture when risk is high.	Avoid agents unsuitable for suspected upper-tract infection.
Systemic UTI / pyelonephritis	Culture before therapy when feasible; start appropriate empiric therapy promptly; adjust to susceptibility.	Assess sepsis, renal function, obstruction and need for hospitalization.
Recurrent UTI	Confirm symptomatic episodes; review cultures, adherence, anatomy, residual urine and modifiable risk factors.	Avoid repeated empiric antibiotics without microbiological confirmation.
ASB in diabetes	Do not routinely screen or treat.	Treat only guideline-supported exceptions such as pregnancy or selected urological procedures.
Catheter-associated infection	Assess necessity of catheter, obtain appropriate culture and address catheter/source control.	Biofilm makes eradication difficult; avoid treating colonization alone.

Oral Agents for Localized Cystitis

Historical IDSA recommendations summarized in the project include nitrofurantoin, trimethoprim-sulfamethoxazole and fosfomycin as preferred agents for uncomplicated cystitis in appropriate patients, with β -lactams or fluoroquinolones reserved for selected circumstances. These regimens should not be transplanted uncritically into every diabetic patient because T2DM, renal impairment, male sex, systemic symptoms and resistant organisms may alter the diagnosis and treatment pathway.

Nitrofurantoin is useful for bladder-limited infection when renal function and other patient factors permit, but it should not be relied upon for pyelonephritis because adequate renal tissue concentrations are not achieved. Fosfomycin is also primarily a lower-tract option. Trimethoprim-sulfamethoxazole can be effective when the organism is susceptible and local resistance is acceptable. Fluoroquinolones are valuable in selected upper-tract infections because of tissue penetration but should be used judiciously due to adverse effects and resistance. Exact drug choice and duration should follow current national/local guidance rather than a fixed historical regimen.

Systemic UTI and Pyelonephritis

Patients with suspected pyelonephritis require therapy capable of achieving therapeutic concentrations in renal tissue. Empiric choices vary with severity and local resistance and may include a third-generation cephalosporin, fluoroquinolone where appropriate, β -lactam/ β -lactamase inhibitor, or broader agents in patients at high risk for ESBL or other resistant organisms. Culture-directed de-escalation is essential. Obstruction or abscess requires urgent source-control assessment because antibiotics alone may fail.

Renal Function and Drug Safety

Diabetic kidney disease is common and may alter antimicrobial pharmacokinetics. Renal function should therefore be checked when infection is significant or when the selected antimicrobial requires dose adjustment. The project discusses nitrofurantoin and renal impairment in detail; modern practice should follow current product information and guideline thresholds rather than relying on older creatinine-clearance cutoffs. Medication review should also consider interactions with glucose-lowering drugs.

SGLT2 Inhibitors and UTI Risk

SGLT2 inhibitors lower plasma glucose by increasing urinary glucose excretion, creating a theoretical

concern that glycosuria could increase UTI. The project reflects this concern but also notes that available clinical data have not consistently demonstrated an important increase in UTI, particularly for canagliflozin, while genital mycotic infection is a more reproducible adverse effect [21].

A 2024 systematic review of landmark randomized trials involving 38,723 participants reported UTI in 1.04% of SGLT2 inhibitor recipients versus 1.15% in placebo recipients, whereas genital infections were substantially more frequent with SGLT2 inhibitors. More recent real-world evidence is reassuring regarding severe UTI: a 2025 meta-analysis of 11 cohort studies involving 679,617 people with T2DM found lower odds of severe UTI with SGLT2 inhibitors compared with composite glucose-lowering therapies and DPP-4 inhibitors, with no significant difference compared with GLP-1 receptor agonists [22].

These findings suggest that a mild UTI alone should not automatically prompt permanent discontinuation of an SGLT2 inhibitor when the drug provides substantial cardiovascular or renal benefit. However, acute illness, poor oral intake or acute kidney injury may require temporary medication adjustment. The 2026 ADA Standards specifically advise considering holding SGLT2 inhibitors when oral intake cannot be maintained or when acute kidney injury is a concern. Clinical decisions should be individualized.

Recurrent UTI and Prevention

Recurrent UTI requires a different mindset from repeated treatment of isolated episodes. First, the diagnosis should be confirmed whenever possible with culture. Second, clinicians should determine whether recurrence represents relapse or reinfection. Third, modifiable drivers such as hyperglycaemia, residual urine, obstruction, stones, catheter use and unnecessary antibiotics should be addressed.

Hydration and appropriate voiding are reasonable supportive measures, although evidence for individual behavioral interventions varies. Postmenopausal women with recurrent UTI may benefit from vaginal estrogen when clinically appropriate and not contraindicated. Non-antibiotic approaches such as cranberry products, probiotics, D-mannose and immunomodulatory strategies have attracted interest, but evidence remains heterogeneous and they should not replace proven management of structural abnormalities or symptomatic infection. The project appropriately describes these approaches as promising but inconclusive [23].

Long-term antibiotic prophylaxis can be effective in carefully selected patients with recurrent uncomplicated infection, but in diabetes it should be individualized because repeated exposure may select resistant organisms. Culture history, adverse-effect risk, renal function and patient preference should inform any prophylactic strategy.

Role of Glycaemic Control and Comprehensive Diabetes Care

Glycaemic management is central to reducing infection susceptibility and improving recovery. The project consistently identifies poor glycaemic control as a modifiable risk factor and recommends integration of diabetes management with UTI care. The ADA 2026 Standards emphasize individualized glycaemic targets and active management during intercurrent illness; acute illness can produce both hyperglycaemia and hypoglycaemia and may precipitate severe metabolic decompensation [24].

During significant infection, glucose monitoring may need to be intensified. Patients should receive clear advice about hydration, medication adjustments during poor intake, recognition of ketones when clinically indicated, and when to seek urgent care. In hospitalized patients, the ADA recommends structured monitoring and individualized insulin-based strategies when needed [25].

Antimicrobial Stewardship and the Role of the Clinical Pharmacist

Antimicrobial stewardship is especially important in diabetic UTI because repeated infections, prior antibiotic exposure and healthcare contact can create a cycle of resistance and therapeutic failure. Stewardship includes avoiding antibiotics for ASB, obtaining cultures when indicated, selecting the narrowest effective drug, adjusting therapy to susceptibility, using the shortest evidence-supported duration and reviewing clinical response. Clinical pharmacists can contribute by reviewing antimicrobial selection and dose, checking renal function and drug interactions, monitoring adverse effects, counseling on adherence, identifying duplicate or unnecessary antibiotics, interpreting susceptibility reports and supporting patient education. The submitted project explicitly identifies pharmacists as important members of a multidisciplinary team [26].

Special Populations and Clinical Considerations

Women with diabetes, particularly older women, may have overlapping risks from menopause-related urogenital changes, recurrent UTI and glycaemic

disease. Men with diabetes require consideration of prostatitis and prostatic obstruction when infection recurs. Pregnancy represents a distinct setting because ASB screening and treatment are recommended to prevent complications; diabetic pregnancy therefore requires coordination between obstetric and diabetes care. Patients with chronic kidney disease require careful antimicrobial selection and monitoring [27,28].

Catheterized patients are at high risk for bacteriuria and biofilm formation. A positive culture alone does not establish symptomatic cautions. The clinical picture and catheter status should be considered together, and unnecessary catheterization should be avoided. When infection is confirmed, source control and appropriate culture collection are critical.

Future Directions

Several priorities emerge from the literature. First, diabetes-specific risk scores could identify patients who benefit most from early culture and imaging. Second, regional surveillance in India is needed because resistance patterns vary substantially between hospitals and communities. Third, molecular diagnostics may shorten the time to pathogen identification and resistance detection, although cost and implementation remain barriers. Fourth, research should clarify which non-antibiotic preventive strategies are genuinely effective in diabetic populations. Fifth, future studies should distinguish lower-tract UTI, systemic UTI and ASB rather than combining them into a single outcome.

The evolving classification proposed by the EAU may improve clinical communication by emphasizing localization and systemic features while separately documenting risk factors [29,30]. Such a framework is particularly useful in diabetes because the same patient may have localized symptoms but a high-risk background or may develop systemic disease with relatively subtle urinary symptoms.

Conclusion

UTI is a clinically important complication of T2DM arising from the interaction of metabolic, immune, neurological and urological factors. The burden is substantial, particularly among women and older adults, and includes symptomatic infection, recurrent UTI, ASB and severe upper-tract disease. *E. coli* remains the predominant pathogen, but resistant Enterobacterales and other healthcare-associated organisms increasingly complicate management. The project evidence and contemporary literature converge on several practical principles: recognize high-risk patients early; distinguish ASB from symptomatic

UTI; obtain urine culture when complicated or systemic infection is suspected; use local resistance data and prior cultures to guide antimicrobial selection; assess renal function and urinary tract abnormalities; optimize glycaemic control; and practice antimicrobial stewardship. Current evidence does not support routine discontinuation of SGLT2 inhibitors solely because of uncomplicated UTI, although treatment should be individualized and temporary interruption may be appropriate during acute illness with poor intake or kidney injury. Long-term improvement will depend on integrated diabetes care, responsible antibiotic use, patient education and collaboration among physicians, pharmacists, nurses and other healthcare professionals. The submitted project similarly concludes that a multidisciplinary approach is essential for reducing recurrence, preventing renal complications and improving long-term outcomes.

Acknowledgement

The author would like to thank Nibha Institute of Pharmaceutical Sciences, Rajgir for providing necessary facility to carry out the project.

Funding

No financial assistance was provided for this project.

Conflict of Interest

None declared.

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