

Relationship Between Diabetes Mellitus and Urinary Tract Infections: Pathogenesis, Risk Factors, and Management Strategies

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ABSTRACT

Background: Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycemia, insulin resistance, and progressive β -cell dysfunction. In addition to its metabolic consequences, diabetes substantially increases susceptibility to urinary tract infections (UTIs) through multiple mechanisms, including impaired immune function, autonomic bladder dysfunction, and enhanced bacterial colonization of the urinary tract. The increasing prevalence of multidrug-resistant (MDR) uropathogens further complicates the management of UTIs in diabetic patients. **Objective:** This review summarizes current evidence regarding the relationship between diabetes mellitus and urinary tract infections, with particular emphasis on disease pathogenesis, major risk factors, clinical complications, diagnostic considerations, and contemporary management strategies. **Main Findings:** Hyperglycemia promotes glycosuria, immune dysfunction, and bladder dysfunction, thereby facilitating bacterial growth and recurrent UTIs. Individuals with diabetes are at increased risk of complicated infections, including pyelonephritis, renal abscesses, and urosepsis. Appropriate glycemic control, culture-guided antimicrobial therapy, optimization of bladder function, and lifestyle modifications remain the cornerstone of management. Emerging therapeutic approaches, including probiotics, stem cell-based therapies, and advanced glucose-lowering agents, may provide additional benefits in selected patients. **Conclusion:** Effective prevention and management of UTIs in patients with diabetes require an integrated approach combining optimal metabolic control, early diagnosis, appropriate antimicrobial therapy, and individualized preventive strategies. Continued research into novel therapeutic interventions and antimicrobial resistance is essential to improve clinical outcomes and reduce the long-term burden of urinary tract infections in diabetic populations.

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INTRODUCTION

Diabetes mellitus (DM) is one of the most significant global public health challenges of the twenty-first century. The prevalence of diabetes continues to increase worldwide, with projections indicating that more than 550 million people will be affected by 2030, of whom over 90% will have type 2 diabetes mellitus (T2DM). More than 80% of diabetes-related deaths occur in low- and middle-income countries, highlighting the substantial socioeconomic and healthcare burden associated with this chronic disease (1). Diabetes mellitus is a heterogeneous metabolic disorder characterized by persistent hyperglycemia resulting from impaired insulin secretion, insulin resistance, or both,

leading to disturbances in carbohydrate, lipid, and protein metabolism (2–4). Chronic hyperglycemia induces progressive biochemical and structural alterations in multiple tissues and organs, contributing to the development of microvascular and macrovascular complications involving the kidneys, retina, nervous system, and cardiovascular system (5,6).

Diabetes is broadly classified into four major categories: type 1 diabetes mellitus (T1DM), type 2 diabetes mellitus (T2DM), gestational diabetes mellitus (GDM), and other specific forms of diabetes resulting from genetic defects, pancreatic disorders, endocrinopathies, or medication-induced hyperglycemia (7). Type 1 diabetes is characterized by autoimmune destruction of pancreatic β -cells, leading to absolute insulin deficiency, whereas type 2 diabetes primarily results from insulin resistance accompanied by progressive β -cell dysfunction (8–10). Persistent hyperglycemia promotes glucosuria, osmotic diuresis, polyuria, polydipsia, and polyphagia while simultaneously increasing susceptibility to infectious and non-infectious complications (11–13). A comparative summary of the principal types of diabetes mellitus, including their etiologies, characteristic clinical manifestations, standard management strategies, and approximate prevalence, is presented in Table 1.

Table (1): Clinical characteristics, etiologies, and management of the major types of diabetes mellitus.

Diabetes type	Typical age at onset	Etiology	Major clinical features	Standard management	Approximate prevalence
Type 1 diabetes mellitus	Childhood or adolescence	Autoimmune destruction of pancreatic β -cells resulting in absolute insulin deficiency	Polyuria, polydipsia, weight loss, fatigue, dehydration	Lifelong insulin therapy, dietary management, glucose monitoring	5–10%
Type 2 diabetes mellitus	Usually adulthood	Insulin resistance with progressive β -cell dysfunction; obesity, sedentary lifestyle, genetic susceptibility	Polyuria, polydipsia, fatigue, slow wound healing, blurred vision	Lifestyle modification, oral antihyperglycemic agents, insulin when indicated	85–90%*
Gestational diabetes mellitus	Pregnancy	Pregnancy-associated insulin resistance	Often asymptomatic; detected by routine antenatal screening	Dietary modification, exercise, insulin when required	5–10% of pregnancies
Specific types of diabetes	Variable	Monogenic disorders, pancreatic disease, endocrinopathies, medications, genetic syndromes	Variable according to the underlying condition	Treatment directed toward the underlying cause with appropriate glycemic management	<1%

Among the infectious complications associated with diabetes, urinary tract infections (UTIs) are among the most common bacterial infections encountered in clinical practice. Individuals with diabetes experience a significantly higher incidence of both primary and recurrent UTIs than non-diabetic individuals, resulting in increased healthcare utilization, antimicrobial exposure, and medical costs (14,15). Several mechanisms contribute to this increased susceptibility, including impaired innate and adaptive immune responses, autonomic bladder dysfunction leading to urinary retention, and elevated urinary glucose concentrations that promote bacterial growth and colonization of the urinary tract (11,14,15).

In addition to increasing the frequency of infection, diabetes predisposes patients to more severe clinical outcomes, including pyelonephritis, renal abscesses, bacteremia, and urosepsis. Furthermore, the widespread use of antimicrobial agents in diabetic patients has contributed to the emergence of multidrug-resistant (MDR) uropathogens, thereby increasing the complexity of treatment and emphasizing the importance of evidence-based prevention and management strategies (15).

Therefore, this review aims to summarize current evidence regarding the relationship between diabetes mellitus and urinary tract infections, with particular emphasis on disease pathogenesis, major risk factors, bacterial profiles, diagnostic considerations, clinical complications, and contemporary prevention and management strategies. By integrating recent advances in both diabetology and infectious diseases, this review seeks to provide a comprehensive overview that may assist clinicians and researchers in improving the prevention, diagnosis, and management of UTIs in patients with diabetes.

Definition and diagnosis of Diabetes

Diabetes mellitus is a general expression that includes more than one disease. It is the result of metabolic disorders of hyperglycemia with carbohydrates disorders in the metabolism, proteins, and fats. Diabetes development increases because of the destruction of insulin-secreting beta cells in the pancreas, resulting in a deficiency or cessation of insulin production (16). According to The World Health Organization (WHO), more than 220 million people worldwide have diabetes, around 1.1 million deaths around the world are associated with diabetes annually, more than 5 million cases are undiagnosed, and about 54 million have early diabetes or are at risk of developing this disease (17). In diabetes, excess glucose is filtered from the blood by the renal tubules more than it is reabsorbed and excreted in the urine. High concentrations of glucose in the urine lead to an osmotic effect that reduces the reabsorption of water by the kidneys, causing excessive urination (18). Water loss in the bloodstream stimulates thirst in patients with diabetes, causing increased urination and severe thirst (18). Also, loss of glucose in the blood causes increased appetite (polyphagia), fatigue, weakness, and weight loss. Leading to serious complications such as glucosuria, blurred vision, thinness, cramps, and skin fungal diseases (12), inflammations, and the most common of these are infections Vaginal and urinary infections (13).

There are two important and life-threatening factors for patients with diabetes: hyperglycemia and increased acidity of the blood (acidosis). If insulin deficiency is not treated, it leads to severe hyperglycemia and increased breakdown of fats with increased release of intermediate fatty acids from adipose tissue (19). Most of the excess fatty acids in the liver are converted to ketoacids beta hydroxybutyric acid and acetic acid (19). The excessive release of fatty acids and ketoacids from muscle and liver tissues raises the acidity level of the blood. The incorporation of hyperglycemia with acidosis is named Diabetic Ketoacidosis, which cause serious complications that lead to coma and then death (20). A study has shown that hyperglycemia causes oxidative stress in tissues that are sensitive to diabetes complications, including peripheral nerves (21). The most serious complications are those resulting from high blood glucose with increased ketone levels causing retinopathy. High blood sugar levels lead to the destruction of blood vessels around the retina, which leads to the deposition of proteins and fats, forming deposits that interfere with vision. Also, the destroyed blood vessels will become ineffective in transporting oxygen to the retina, causing damage to the retina (12). Hyperglycemia also leads to kidney disease that ends in kidney failure, and it also causes peripheral and central nervous diseases that lead to foot ulcers, amputations, and joint atrophy, which may be accompanied by an increased risk of developing diseases Cardiovascular, peripheral vascular, and spinal vascular disorders with gastrointestinal disturbances (22).

DEFINITION AND DIAGNOSIS OF DIABETES

Diabetes mellitus (DM) is a group of chronic metabolic disorders characterized by persistent hyperglycemia resulting from defects in insulin secretion, insulin action, or both. These abnormalities disrupt the metabolism of carbohydrates, lipids, and proteins and contribute to the progressive development of systemic complications affecting multiple organs (16). According to the World Health Organization (WHO), diabetes represents one of the leading causes of morbidity and mortality worldwide, with its prevalence continuing to increase because of population aging, urbanization, sedentary lifestyles, and the growing prevalence of obesity (17).

Persistent hyperglycemia results in glucose concentrations that exceed the renal threshold, leading to glucosuria and osmotic diuresis. Consequently, patients commonly develop polyuria, polydipsia, polyphagia, fatigue, weight loss, and generalized weakness. Chronic hyperglycemia also contributes to blurred vision, recurrent skin and mucosal infections, and increased susceptibility to urinary tract infections owing to impaired immune function and elevated urinary glucose concentrations (12,13,18).

Uncontrolled diabetes may progress to life-threatening metabolic emergencies, particularly diabetic ketoacidosis (DKA), which is characterized by severe insulin deficiency, accelerated lipolysis, excessive ketone body production, metabolic acidosis, and dehydration (19,20). In addition, chronic oxidative stress induced by prolonged hyperglycemia plays a central role in the development of diabetic complications by promoting endothelial dysfunction, inflammation, and cellular injury (21). These mechanisms contribute to the development of diabetic retinopathy, nephropathy, neuropathy, cardiovascular disease, and peripheral vascular disease, which collectively account for a substantial proportion of diabetes-related morbidity and mortality (22).

The diagnosis of diabetes mellitus is established using standardized biochemical criteria recommended by international organizations, including the American Diabetes Association (ADA) and the World Health Organization (WHO). Diabetes can be diagnosed by a fasting plasma glucose level ≥ 126 mg/dL (7.0 mmol/L), a 2-hour plasma glucose level ≥ 200 mg/dL (11.1 mmol/L) during a 75-g oral glucose tolerance test, a glycated hemoglobin (HbA1c) level $\geq 6.5\%$, or a random plasma glucose level ≥ 200 mg/dL in the presence of classic symptoms of hyperglycemia (4,16). Early diagnosis is essential because timely glycemic control reduces the risk of long-term complications and improves overall clinical outcomes.

CLASSIFICATION OF DIABETES

Diabetes mellitus is classified into four major categories according to the World Health Organization (WHO) and the American Diabetes Association (ADA): type 1 diabetes mellitus (T1DM), type 2 diabetes mellitus (T2DM), gestational diabetes mellitus (GDM), and specific types of diabetes resulting from other identifiable causes (23). Each category differs in etiology, pathophysiology, clinical presentation, and therapeutic management.

1. Type 1 Diabetes Mellitus (T1DM)

Type 1 diabetes mellitus is an autoimmune disorder characterized by immune-mediated destruction of pancreatic β -cells, resulting in absolute insulin deficiency. The disease most commonly develops during childhood or adolescence, although it may occur at any age. Patients require lifelong insulin replacement therapy to maintain glycemic control and prevent life-threatening complications such as diabetic ketoacidosis (24,25).

Although classical autoimmune T1DM accounts for the majority of cases, less common forms, including type 1B diabetes and monogenic disorders such as maturity-onset diabetes of the young (MODY), have also been described. MODY results from inherited mutations affecting pancreatic β -cell function and differs clinically from autoimmune diabetes because it is not associated with immune-mediated β -cell destruction (26).

2. Type 2 Diabetes Mellitus (T2DM)

Type 2 diabetes mellitus is the most prevalent form of diabetes, accounting for approximately 90–95% of all cases worldwide. The disorder is characterized by insulin resistance combined with progressive pancreatic β -cell dysfunction, leading to inadequate insulin secretion over time. Major risk factors include obesity, physical inactivity, advancing age, family history, and genetic susceptibility (27).

Unlike T1DM, patients with T2DM are frequently asymptomatic during the early stages of the disease, resulting in delayed diagnosis and increasing the likelihood of chronic vascular and metabolic complications before treatment is initiated.

3. Gestational Diabetes Mellitus (GDM)

Gestational diabetes mellitus is defined as glucose intolerance first recognized during pregnancy. Hormonal changes associated with pregnancy induce physiological insulin resistance, which may exceed the compensatory capacity of pancreatic β -cells in susceptible women. Consequently, hyperglycemia usually develops during the second or third trimester. Although blood glucose levels often normalize after delivery, women with a history of GDM remain at increased risk of developing type 2 diabetes later in life (27).

4. Specific Types of Diabetes

Specific forms of diabetes comprise a heterogeneous group of disorders caused by identifiable genetic, endocrine, pancreatic, or drug-related factors. These include monogenic diabetes syndromes, diseases affecting the exocrine pancreas, endocrinopathies, and diabetes induced by medications such as glucocorticoids. Although relatively uncommon, accurate identification of these forms is clinically important because treatment strategies depend on the underlying etiology (27).

RISK FACTORS ASSOCIATED WITH URINARY TRACT INFECTIONS IN PATIENTS WITH DIABETES

Patients with diabetes mellitus are at a substantially higher risk of developing urinary tract infections (UTIs) than the general population. This increased susceptibility results from the interaction of metabolic abnormalities, immune dysfunction, urinary tract abnormalities, and patient-related factors that facilitate bacterial colonization and infection (28).

Persistent hyperglycemia is one of the principal contributors to UTI development in diabetic patients. Elevated blood glucose levels promote glucosuria, creating a nutrient-rich environment that enhances bacterial growth within the urinary tract. Chronic hyperglycemia also impairs neutrophil chemotaxis, phagocytosis, and intracellular bacterial killing, thereby weakening the host's innate immune defense against invading microorganisms (29,30).

Female sex is another well-established risk factor because women have a shorter urethra and a closer anatomical relationship between the urethral opening and the gastrointestinal tract, facilitating ascending bacterial infection. The risk is further increased in postmenopausal women owing to estrogen deficiency, which alters the normal vaginal microbiota and reduces natural antimicrobial protection (31).

Autonomic diabetic neuropathy contributes to bladder dysfunction by impairing detrusor muscle contractility and bladder emptying. Residual urine promotes bacterial persistence and recurrent infections by providing favorable conditions for microbial proliferation (32). Similarly, structural abnormalities of the urinary tract, urinary obstruction, nephrolithiasis, and indwelling urinary catheters significantly increase the likelihood of complicated UTIs in diabetic patients (33).

Additional factors associated with an increased risk of UTIs include advanced age, prolonged duration of diabetes, poor glycemic control, chronic kidney disease, obesity, immunosuppressive therapy, previous episodes of UTI, and hospitalization. Collectively, these factors increase both the incidence of infection and the risk of recurrent or complicated disease, emphasizing the importance of early identification and appropriate preventive strategies (34,35).

PATHOGENESIS OF URINARY TRACT INFECTIONS IN PATIENTS WITH DIABETES MELLITUS

The pathogenesis of urinary tract infections (UTIs) in patients with diabetes mellitus is multifactorial and results from the combined effects of metabolic abnormalities, impaired host immune defenses, autonomic dysfunction, and enhanced bacterial virulence. Chronic hyperglycemia creates an environment that favors microbial colonization while simultaneously reducing the body's ability to eliminate invading pathogens (36).

One of the principal mechanisms underlying this increased susceptibility is glucosuria. Elevated urinary glucose concentrations provide a favorable nutrient source for uropathogenic microorganisms, facilitating their growth, adherence, and persistence within the urinary tract. Hyperglycemia also impairs several components of innate immunity, including neutrophil chemotaxis, phagocytosis, intracellular killing, and complement activity, thereby weakening the initial host response to bacterial invasion (37,38).

Diabetic autonomic neuropathy further contributes to infection by impairing bladder sensation and detrusor muscle contractility, resulting in incomplete bladder emptying and urinary retention. Residual urine serves as a reservoir for bacterial proliferation, increasing the risk of persistent and recurrent infections. Microvascular damage associated with chronic diabetes may additionally reduce tissue perfusion and delay the local inflammatory response, compromising tissue repair and bacterial clearance (39).

The development of UTIs also depends on bacterial virulence factors. Uropathogenic microorganisms possess adhesins, fimbriae, biofilm-forming capacity, toxins, and iron-acquisition systems that promote attachment to the uroepithelium, facilitate colonization, and protect bacteria from host immune responses and antimicrobial therapy. Biofilm formation is particularly important in recurrent and catheter-associated UTIs because it enhances bacterial persistence and antimicrobial tolerance (40,41).

The interaction between host susceptibility and microbial pathogenicity ultimately determines the severity and clinical outcome of infection. Poor glycemic control, prolonged duration of diabetes, structural abnormalities of the

urinary tract, and infection with multidrug-resistant organisms further increase the likelihood of complicated UTIs, recurrent infections, and severe outcomes such as pyelonephritis and urosepsis (42).

COMMON UROPATHOGENS IN DIABETIC PATIENTS

Urinary tract infections (UTIs) in patients with diabetes mellitus are caused by a wide range of microorganisms. Although the spectrum of pathogens is generally similar to that observed in non-diabetic individuals, diabetic patients are more likely to develop infections caused by opportunistic and multidrug-resistant (MDR) organisms because of impaired host immunity, frequent healthcare exposure, and repeated antimicrobial use (43).

Escherichia coli remains the predominant causative organism, accounting for the majority of community-acquired and many hospital-acquired UTIs in diabetic patients. Its pathogenicity is largely attributed to virulence factors such as type 1 and P fimbriae, adhesins, toxins, siderophores, and biofilm formation, which facilitate adhesion to the uroepithelium, bacterial persistence, and evasion of host immune defenses (44,45).

Other important Gram-negative pathogens include *Klebsiella pneumoniae*, *Proteus mirabilis*, *Pseudomonas aeruginosa*, *Enterobacter* species, and *Citrobacter* species. These organisms are more frequently isolated from patients with complicated UTIs, urinary tract abnormalities, prolonged hospitalization, indwelling urinary catheters, or previous antimicrobial exposure. Several possess intrinsic or acquired antimicrobial resistance mechanisms, making treatment increasingly challenging (46,47).

Among Gram-positive organisms, *Enterococcus faecalis*, *Staphylococcus saprophyticus*, and other coagulase-negative staphylococci are recognized causes of UTIs, particularly in hospitalized patients and those with urinary catheterization or structural urinary tract abnormalities (48). In addition, fungal pathogens, especially *Candida* species, are encountered more frequently in diabetic patients because persistent hyperglycemia, glucosuria, prolonged antibiotic therapy, and urinary catheterization create favorable conditions for fungal colonization and infection (49).

The emergence of multidrug-resistant organisms, including extended-spectrum β -lactamase (ESBL)-producing Enterobacterales, carbapenem-resistant Gram-negative bacilli, and vancomycin-resistant enterococci, has become an important clinical concern. These pathogens are associated with prolonged hospitalization, higher treatment costs, increased morbidity, and reduced therapeutic options. Consequently, accurate microbiological diagnosis, antimicrobial susceptibility testing, and rational antibiotic stewardship are essential for optimizing treatment outcomes in diabetic patients with UTIs (50,51).

DIAGNOSIS OF URINARY TRACT INFECTIONS IN PATIENTS WITH DIABETES MELLITUS

The diagnosis of urinary tract infections (UTIs) in patients with diabetes mellitus requires the integration of clinical assessment, laboratory investigations, microbiological confirmation, and, when indicated, radiological evaluation. Early and accurate diagnosis is essential because diabetic patients are at increased risk of recurrent infections, antimicrobial resistance, and severe complications such as pyelonephritis, emphysematous infections, and urosepsis (52).

Clinical manifestations of UTIs in diabetic patients are similar to those observed in the general population and commonly include dysuria, urinary frequency, urgency, suprapubic pain, and cloudy or malodorous urine. Upper urinary tract involvement may present with fever, chills, flank pain, nausea, vomiting, and costovertebral angle tenderness. However, diabetic patients, particularly older adults and those with diabetic neuropathy, may exhibit atypical or less pronounced symptoms, making laboratory confirmation especially important (53).

Urinalysis is the initial laboratory investigation and may demonstrate pyuria, bacteriuria, leukocyte esterase positivity, nitrites, microscopic hematuria, and proteinuria. Although these findings support the diagnosis of UTI, urine culture with antimicrobial susceptibility testing remains the diagnostic gold standard because it confirms the causative organism and guides appropriate antimicrobial therapy, particularly in complicated or recurrent infections (54,55).

Additional laboratory investigations, including complete blood count, inflammatory markers, renal function tests, and blood cultures in selected patients, may be required to evaluate the severity of infection and identify systemic involvement. Imaging studies, such as ultrasonography or computed tomography (CT), are recommended when

urinary tract obstruction, nephrolithiasis, renal abscess, emphysematous pyelonephritis, or other structural abnormalities are suspected (56).

Accurate diagnosis not only facilitates prompt initiation of targeted antimicrobial therapy but also enables the identification of underlying risk factors, assessment of disease severity, and implementation of strategies to prevent recurrence and long-term complications. A comprehensive diagnostic approach is therefore fundamental to improving clinical outcomes in diabetic patients with urinary tract infections (57).

Table (2): Diagnostic methods for urinary tract infections in patients with diabetes mellitus

Diagnostic method	Purpose	Typical findings / Clinical significance
Clinical assessment	Identify symptoms and assess disease severity	Dysuria, urinary frequency, urgency, suprapubic pain, flank pain, fever, chills, or atypical presentations in older adults and patients with diabetic neuropathy
Urinalysis	Initial laboratory evaluation	Pyuria, bacteriuria, positive leukocyte esterase, nitrites, hematuria, and proteinuria support the diagnosis of UTI
Urine culture	Gold standard for microbiological diagnosis	Confirms the causative microorganism and quantifies bacteriuria
Antimicrobial susceptibility testing	Guide targeted antimicrobial therapy	Determines susceptibility or resistance to antimicrobial agents and supports appropriate antibiotic selection
Blood investigations	Assess systemic involvement	Complete blood count, inflammatory markers, renal function tests, and blood cultures in suspected severe infection or sepsis
Imaging studies	Detect complications or structural abnormalities	Ultrasonography or computed tomography (CT) identifies urinary obstruction, nephrolithiasis, renal abscess, emphysematous pyelonephritis, and other complicated infections

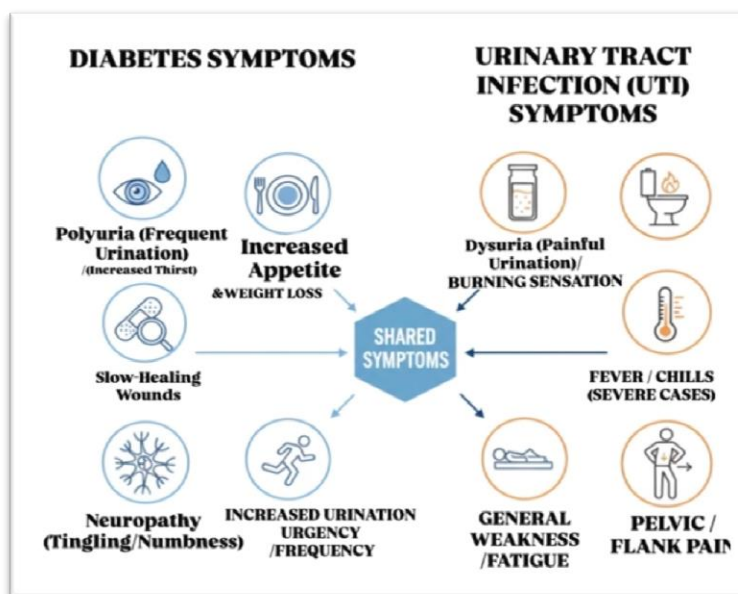


Figure (1): Schematic illustration of the bidirectional relationship between diabetes mellitus and urinary tract infections (UTIs), demonstrating shared symptoms and increased infection risk associated with hyperglycemia.

Patients with diabetes may develop several forms of urinary tract infections, including asymptomatic bacteriuria, acute cystitis, complicated lower UTIs (such as catheter-associated infections), uncomplicated pyelonephritis, and complicated pyelonephritis or urosepsis.

MANAGEMENT AND TREATMENT OF URINARY TRACT INFECTIONS IN PATIENTS WITH DIABETES MELLITUS

The management of urinary tract infections (UTIs) in patients with diabetes mellitus requires a comprehensive approach that combines appropriate antimicrobial therapy, optimization of glycemic control, correction of predisposing factors, and prevention of recurrent infection. Because diabetic patients are at increased risk of complicated UTIs, multidrug-resistant (MDR) pathogens, and treatment failure, management should be individualized according to the severity of infection, microbiological findings, renal function, and patient comorbidities (58).

Empirical antimicrobial therapy should be initiated in symptomatic patients based on the severity of infection, local antimicrobial resistance patterns, and patient-specific risk factors. Once urine culture and antimicrobial susceptibility results become available, treatment should be adjusted to the narrowest effective antimicrobial agent to maximize clinical efficacy while minimizing the emergence of antimicrobial resistance (59,60).

Effective glycemic control is a fundamental component of treatment because persistent hyperglycemia impairs immune function, promotes glucosuria, and facilitates bacterial growth within the urinary tract. Optimization of blood glucose levels improves host immune responses and may reduce the risk of persistent or recurrent infection. Adequate hydration, timely bladder emptying, and correction of urinary tract obstruction or catheter-related problems are additional measures that contribute to successful treatment (61).

Patients with complicated UTIs, pyelonephritis, recurrent infections, renal impairment, or systemic manifestations such as sepsis may require hospitalization, intravenous antimicrobial therapy, and close clinical monitoring. Imaging studies should be considered when structural abnormalities, urinary obstruction, emphysematous infections, or renal abscesses are suspected.

Preventive strategies are equally important in reducing the burden of UTIs among diabetic patients. Maintaining optimal glycemic control, encouraging adequate fluid intake, minimizing unnecessary urinary catheterization, promoting appropriate personal hygiene, and implementing antimicrobial stewardship programs can substantially reduce infection rates and limit the development of antimicrobial resistance. Patient education regarding early symptom recognition and prompt medical evaluation is also essential for preventing disease progression and recurrent infections.

BACTERIAL PROFILE OF URINARY TRACT INFECTIONS IN PATIENTS WITH DIABETES MELLITUS

Urinary tract infections (UTIs) in patients with diabetes mellitus are caused by a diverse range of bacterial pathogens. Although the overall bacterial spectrum resembles that observed in the non-diabetic population, diabetic patients exhibit a higher prevalence of opportunistic and multidrug-resistant (MDR) organisms because of impaired immune defenses, persistent hyperglycemia, frequent healthcare exposure, recurrent infections, and repeated antimicrobial use (43).

Escherichia coli remain the predominant uropathogen, accounting for approximately 70–90% of community-acquired UTIs and a substantial proportion of hospital-acquired infections. Its pathogenicity is mediated by multiple virulence factors, including type 1 and P fimbriae, adhesins, hemolysin, siderophores, and biofilm formation, which facilitate adhesion to uroepithelial cells, bacterial persistence, tissue invasion, and evasion of host immune responses (44,45).

Among other Gram-negative bacteria, *Klebsiella pneumoniae*, *Proteus mirabilis*, *Pseudomonas aeruginosa*, *Enterobacter* spp., and *Citrobacter* spp. are frequently isolated from diabetic patients, particularly those with recurrent or complicated UTIs, urinary catheterization, structural urinary tract abnormalities, or prolonged hospitalization. Many of these organisms possess intrinsic or acquired resistance mechanisms that complicate antimicrobial therapy and contribute to poorer clinical outcomes (46,47).

Gram-positive bacteria also contribute to UTIs in diabetic patients. *Enterococcus faecalis* is commonly associated with healthcare-associated and complicated infections, whereas *Staphylococcus saprophyticus* is an important pathogen in uncomplicated UTIs, particularly among younger women. Other coagulase-negative staphylococci may also be isolated in selected clinical settings, especially in patients with urinary catheters or underlying urinary tract abnormalities (48).

The increasing prevalence of multidrug-resistant bacteria has become a major therapeutic challenge. Extended-spectrum β -lactamase (ESBL)-producing Enterobacterales, carbapenem-resistant Gram-negative bacilli, and vancomycin-resistant enterococci are increasingly reported among diabetic patients with recurrent or healthcare-associated UTIs. These pathogens are associated with prolonged hospitalization, increased healthcare costs, higher morbidity, and limited treatment options, highlighting the importance of urine culture, antimicrobial susceptibility testing, and antimicrobial stewardship in clinical practice (49,50).

Knowledge of the local bacterial epidemiology and antimicrobial resistance profile is essential for selecting appropriate empirical therapy and optimizing patient outcomes. Continuous microbiological surveillance and periodic updates of institutional antibiotic policies are therefore critical for improving the management of UTIs in patients with diabetes mellitus (51).

Table (3): Major bacterial pathogens causing urinary tract infections in patients with diabetes mellitus

Bacterial pathogen	Gram stain	Clinical significance	Major virulence factors	Common resistance mechanisms
<i>Escherichia coli</i>	Gram-negative	Most common cause of community- and hospital-acquired UTIs	Type 1 fimbriae, P fimbriae, adhesins, hemolysin, biofilm, siderophores	ESBL production, fluoroquinolone resistance
<i>Klebsiella pneumoniae</i>	Gram-negative	Complicated and catheter-associated UTIs	Capsule, biofilm, siderophores	ESBLs, carbapenemases
<i>Proteus mirabilis</i>	Gram-negative	Catheter-associated UTIs and urinary stones	Urease, fimbriae, biofilm	β -lactam resistance
<i>Pseudomonas aeruginosa</i>	Gram-negative	Healthcare-associated and recurrent UTIs	Biofilm, exotoxins, secretion systems	Multidrug resistance, carbapenem resistance
<i>Enterobacter</i> spp.	Gram-negative	Opportunistic infections	Biofilm, adhesins	AmpC β -lactamases, ESBLs
<i>Citrobacter</i> spp.	Gram-negative	Complicated UTIs	Adhesion factors, biofilm	ESBLs, AmpC β -lactamases
<i>Enterococcus faecalis</i>	Gram-positive	Complicated and healthcare-associated UTIs	Aggregation substance, biofilm	Vancomycin resistance (VRE)
<i>Staphylococcus saprophyticus</i>	Gram-positive	Uncomplicated UTIs, particularly in young women	Adhesins, urease	Variable resistance to β -lactams

Conclusion

Diabetes mellitus significantly increases both the incidence and severity of urinary tract infections (UTIs) through a complex interplay of persistent hyperglycemia, impaired immune function, autonomic bladder dysfunction, and structural abnormalities of the urinary tract. These pathophysiological changes facilitate bacterial colonization, promote recurrent infections, and increase the likelihood of complications, including pyelonephritis, emphysematous urinary tract infections, and urosepsis. Furthermore, the growing prevalence of multidrug-resistant uropathogens presents an additional challenge in the clinical management of diabetic patients.

Early recognition of UTIs, accurate microbiological diagnosis, and timely initiation of targeted antimicrobial therapy are fundamental to reducing morbidity and preventing long-term renal complications. Optimizing glycemic control, correcting modifiable risk factors, and implementing antimicrobial stewardship are equally important components of comprehensive patient management. In addition, patient education and preventive measures should be integrated into routine diabetes care to reduce the frequency of recurrent infections and improve overall clinical outcomes.

Continued surveillance of bacterial epidemiology and antimicrobial resistance patterns, together with advances in rapid diagnostic techniques and novel therapeutic approaches, will further improve the prevention and management of UTIs in patients with diabetes mellitus. Future research should focus on identifying predictive biomarkers, evaluating personalized treatment strategies, and developing interventions that reduce recurrent infections while preserving antimicrobial effectiveness. Collectively, these efforts have the potential to improve patient outcomes and reduce the global healthcare burden associated with diabetes-related urinary tract infections.

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العلاقة بين مرض السكري و التهاب المسالك البولية

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الخلاصة

خلفية البحث : داء السكري مرض مزمن واسع الانتشار يتميز بارتفاع مستمر في مستوى السكر في الدم، ومقاومة الأنسولين، واختلال تدريجي في وظيفة خلايا بيتا. بالإضافة إلى تأثيراته الأيضية، يزيد داء السكري من قابلية الإصابة بالعديد من الحالات الثانوية، بما في ذلك التهابات المسالك البولية. يرتبط ارتفاع معدل الإصابة بالتهابات المسالك البولية لدى مرضى السكري بشكل رئيسي بارتفاع مستويات الجلوكوز، وضعف الاستجابة المناعية، واعتلال الأعصاب اللاإرادي الذي يؤثر على وظيفة المثانة. في هذه الفئة من المرضى، قد تتطور التهابات المسالك البولية إلى مضاعفات خطيرة مثل تكوّن خراجات الكلى والتهاب الحويضة والكلية. يعتمد الوقاية من التهابات المسالك البولية وعلاجها لدى مرضى السكري على ضبط مستوى السكر في الدم بشكل مناسب، واختيار العلاج المضاد للميكروبات المناسب، وتناول كميات كافية من السوائل، وتعديل نمط الحياة. علاوة على ذلك، يتطلب وجود خلل في وظيفة المثانة وتزايد مشكلة مسببات الأمراض المقاومة للأدوية المتعددة استراتيجيات وقائية وعلاجية فردية. **الهدف من البحث:** تستعرض هذه المقالة العلاقة بين داء السكري والتهابات المسالك البولية، مع التركيز على عوامل الخطر الكامنة، والجوانب التشخيصية، وأساليب العلاج الحالية التي تهدف إلى الحد من عبء التهابات المسالك البولية لدى مرضى السكري. **الاستنتاج:** يُبرز الارتباط بين داء السكري والتهابات المسالك البولية ضرورة الإدارة الدقيقة والمستمرة للمرض. يُعدّ مرضى السكري، وخاصةً المصابين بالنوع الثاني، أكثر عرضةً للإصابة المتكررة بالتهابات المسالك البولية نتيجةً لارتفاع مستوى السكر في الدم بشكل مستمر، وضعف جهاز المناعة، ومضاعفات داء السكري. يلعب التشخيص المبكر للعدوى، والتحكم الجيد في مستوى السكر في الدم، والاستخدام المناسب للمضادات الحيوية دورًا هامًا في الحدّ من تكرار وشدة التهابات المسالك البولية. بالإضافة إلى ذلك، يُساهم الاهتمام بوظائف المثانة، والنظافة الشخصية، واتباع نمط حياة صحي بشكل كبير في الوقاية. يجري حاليًا دراسة خيارات علاجية جديدة، بما في ذلك استخدام البروبيوتيك والأساليب القائمة على الخلايا الجذعية، والتي قد تُقدّم فوائد إضافية في المستقبل. يُمكن أن يُساهم دمج هذه الإجراءات في الممارسة السريرية الروتينية في تحسين نتائج المرضى، والحدّ من المضاعفات، ودعم جودة حياة أفضل للأفراد المصابين بداء السكري.

الكلمات المفتاحية: مرض السكري ، التهابات المسالك البولية ، ارتفاع سكر في الدم ، مقاومة الانسولين ، خلل في خلايا بيتا.