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Recurrent Urinary Tract Infections in Patients with Type 2 Diabetes Mellitus: A Narrative Review

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ABSTRACT

Recurrent urinary tract infections (rUTIs) represent a frequent and clinically challenging complication among patients with type 2 diabetes mellitus (T2DM). Impaired glycemic control alters host defense mechanisms, promotes glycosuria, and facilitates bacterial adhesion and colonization within the urinary tract, thereby increasing susceptibility to recurrent infections. This narrative review summarizes current evidence regarding the epidemiology, pathophysiology, microbial characteristics, diagnostic considerations, and management strategies of rUTIs in individuals with T2DM. Escherichia coli remains the predominant uropathogen; however, diabetic patients are disproportionately affected by multidrug-resistant organisms, complicating therapeutic decision-making. Autonomic neuropathy and bladder dysfunction further contribute to urinary stasis and infection recurrence. Diagnosis relies on careful clinical assessment supported by urine microscopy and culture, which is particularly important in diabetic patients to guide appropriate antimicrobial therapy. Management principles are generally similar to those applied in non-diabetic individuals but require classification of most infections as complicated, emphasizing culture-guided antibiotic selection, appropriate treatment duration, and vigilant monitoring. Non-pharmacological measures, strict glycemic control, and evaluation for structural or functional urinary tract abnormalities are essential components of care. Emerging therapies, including sodium–glucose cotransporter-2 inhibitors and non-antibiotic preventive approaches such as probiotics, warrant further investigation. An integrated, multidisciplinary approach combining optimized diabetes management with judicious antimicrobial use is critical to reducing recurrence, preventing complications, and limiting antimicrobial resistance in this vulnerable population.

1. Introduction

Urinary tract infections (UTIs) are among the most common bacterial infections encountered in clinical practice. Patients with T2DM exhibit a higher incidence of both uncomplicated and complicated UTIs compared with non-diabetic individuals [1]. Chronic hyperglycemia contributes to immune dysfunction, microvascular damage, and autonomic neuropathy, all of which predispose diabetic patients to recurrent urinary tract infections (rUTIs). The pathogenesis of rUTI in diabetic individuals may be caused by a number of immune system abnormalities, poor metabolic control of diabetes, and incomplete bladder evacuation brought on by autonomic neuropathy[2]. Diabetes alters the natural host system, which may contribute to the development of UTIs[3]. Limited research indicates that defective bladder contractions may result in stagnant urine pools, which provide a conducive environment for bacterial proliferation. Additional research suggests that urine with elevated glucose levels fosters a rise in bacterial clusters and their colonization within the urinary system[4]. Concerning the findings of therapeutic approaches for asymptomatic bacteriuria in diabetic individuals, limited clinical trials have been performed. The findings of these clinical trials demonstrate that treatment durations of 2 weeks and 6 weeks exhibit equivalent efficacy, the incidence of rUTIs remains elevated, despite prolonged antibiotic therapy, the majority of re-infections occurred recurrently (4/8 weeks post-therapy) and these recurrences were not caused by the same microorganism. Physicians should be cognizant of the elevated incidence of underlying structural genitourinary abnormalities in bacteriuric women with diabetes mellitus[5]. In addition to increasing infection risk, diabetes is associated with more severe clinical presentations and higher rates of complications, including emphysematous cystitis and pyelonephritis [6]. This review provides an updated narrative overview of the mechanisms, diagnosis, and management of rUTIs in patients with T2DM.

2. Pathophysiology of rUTIs in T2DM

2.1 Impaired host defense mechanisms

In diabetic patients, the elevated risk of rUTI may be attributed to a variety of potential mechanisms that are distinctive to diabetes[5]. The proliferation of pathogenic bacteria may be facilitated by elevated glucose concentrations in urine[7]. Elevated renal parenchymal glucose concentrations foster an environment conducive to the proliferation of bacteria, potentially serving as a precipitating factor for pyelonephritis and renal consequences, including emphysematous pyelonephritis[8]. Various abnormalities in the immune system, including humoral, cellular, and innate immunity, may contribute to the pathogenesis of infections of the urinary system in patients with diabetes [9]. Patients with diabetes and asymptomatic bacteriuria exhibited reduced levels of interleukin-6 and interleukin -8 compared to their non-diabetic counterparts with asymptomatic bacteriuria[10]. Autonomic neuropathy affecting the genitourinary system leads to impaired voiding and urine retention, which diminishes physical bacterial elimination by urination, consequently promoting bacterial proliferation[11] Bladder dysfunction impacts 26%–85% of diabetic women, depending on age, severity of neuropathy, and duration of diabetes[12]. The emergence of new anti-diabetic sodium glucose cotransporter-2 inhibitors SGLT2 inhibitors, which promote glycosuria, has elicited worries about a possible increase in urinary tract infections [13]. Thus, the most frequent side effect of SGLT2 inhibitors therapy seems to be genitourinary tract infections. Increased urinary glucose can lead to a faster rate of microbial development, which raises the risk of ASB and UTI. Urine that is subsequently deposited on the vulvovaginal tissues after voiding can further raise the risk of urogenital infections[14].Non-DM individuals with difficult UTIs had similar microorganisms to those recovered from DM patients with UTIs. Enterobacter spp., Proteus spp., E. coli, Klebsiella spp., Group B Streptococci, and Enterococcus faecalis are the most commonly isolated pathogens [15]. Figure1 summarizes the steps

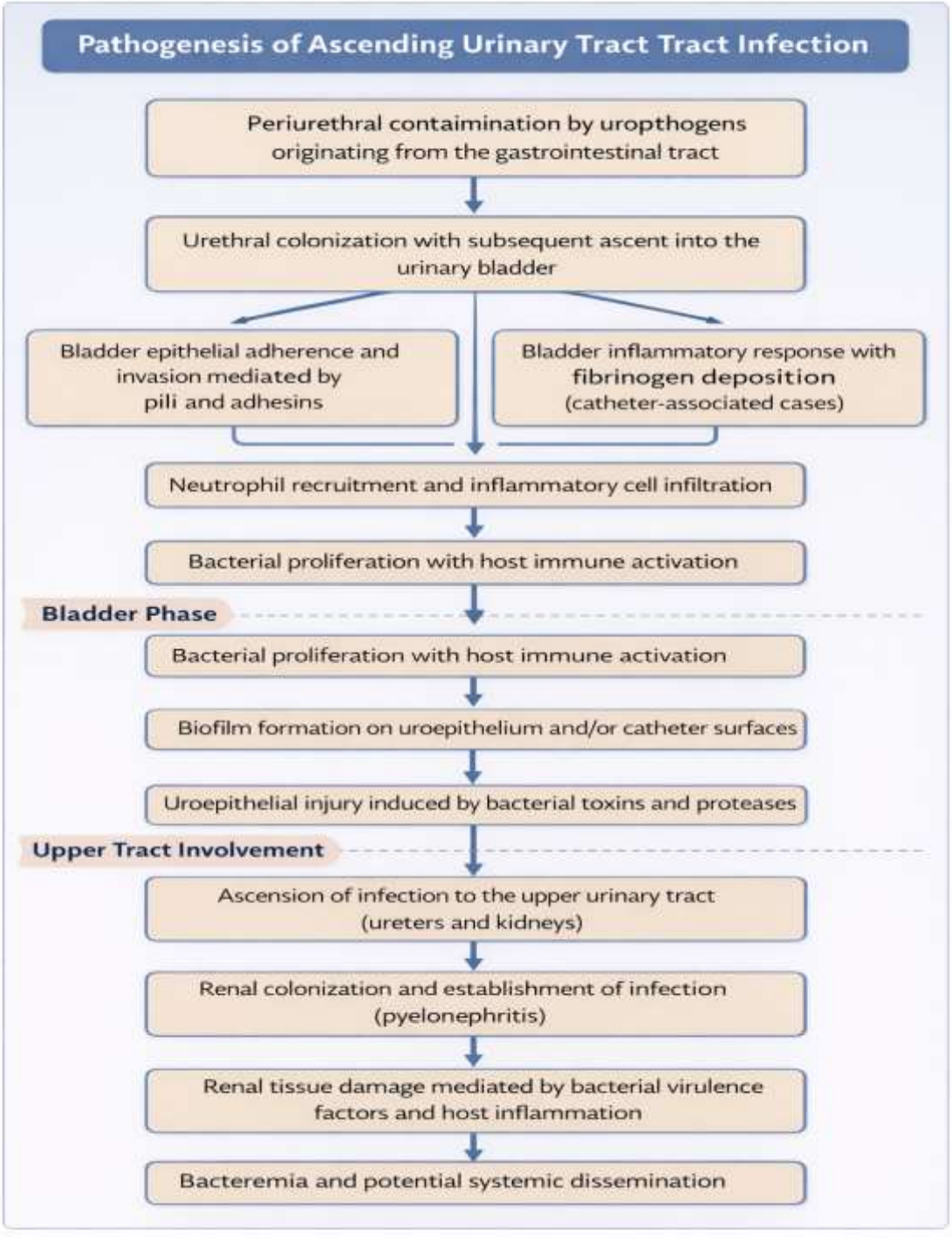

Figure 1. *The pathophysiology of urinary tract infections. ,*

that contribute to the pathophysiology of UTIs.

2.2 Glycosuria and bacterial proliferation

Persistent hyperglycemia results in glycosuria, creating a favorable environment for bacterial growth within the urinary tract . Elevated glucose concentrations in renal parenchyma and urine enhance bacterial adhesion and colonization, increasing the risk of ascending infections and severe renal complications [16].

2.3 Bladder dysfunction and urinary stasis

[Diabetic autonomic neuropathy affects bladder sensation and contractility, leading to incomplete bladder emptying and increased post-void residual volume Bladder dysfunction has been reported in up to 87% of diabetic women and represents a key contributor to urinary stasis and recurrent infection [17].

3. Microbiological Patterns

The spectrum of uropathogens isolated from diabetic patients with rUTIs is broadly similar to that observed in non-diabetic populations. Escherichia coli remains the most frequently identified organism, followed by Klebsiella species, Proteus species, Enterococcus faecalis, and Group B Streptococci [18].

4. Diagnosis of Recurrent UTIs in T2DM

4.1 Clinical features

The clinical manifestation of rUTI is identical to that of sporadic acute cystitis. Local genitourinary symptoms of dysuria, frequency, urgency, and hesitation manifest abruptly [19]). In addition, simple cystitis may show with suprapubic discomfort and gross hematuria. The symptoms are frequently the same as in earlier episodes, and ambulatory women with recurrent, simple UTIs are quite accurate in diagnosing themselves [19]

A UTI diagnosis should be taken into consideration for any diabetic patient displaying symptoms suggestive of a UTI. According to a recent multi-center study on women with community-acquired acute pyelonephritis in South Korea, diabetic patients experienced significantly fewer episodes of lower urinary tract infection symptoms, costovertebral angle soreness, and flank pain than their non-diabetic counterparts [20] Patients with T2DM and UTIs may exhibit hypo- or hyperglycemia, non-ketotic hyperosmolar condition, or ketoacidosis, necessitating the immediate elimination of infectious triggering factors, including UTIS [21].

4.2 Laboratory investigations

Urinalysis typically demonstrates pyuria, which can be detected by microscopy or leukocyte esterase testing. Urine culture is strongly recommended prior to initiating antimicrobial therapy in diabetic patients to distinguish infection from colonization and to guide antibiotic selection . In recurrent cases, imaging studies may be required to evaluate for underlying structural abnormalities[22].

5. Management Strategies

5.1 Non-pharmacological management

General preventive measures include adequate hydration, regular and complete bladder emptying, avoidance of unnecessary catheterization, and modification of behavioral risk factors such as spermicide use .Cranberry products and probiotics have demonstrated modest preventive benefits in selected populations, although evidence specific to diabetic patients remains limited [23].

5.2 Pharmacological management

Antimicrobial medications are frequently used to treat symptomatic UTIs. Individualized treatment should be provided based on the antibiotic sensitivity of the urine culture, or a broad-spectrum antimicrobial medication may be used as empirical or preventive therapy [24]. Commonly utilized antibiotics

comprise trimethoprim/sulfamethoxazole, cotrimoxazole, amoxicillin, quinolones, and nitrofurantoin [25]. For people with acute uncomplicated cystitis, the first-line treatment is oral nitrofurantoin for five days; fosfomycin is an alternate. These two medications, however, do not work well on renal parenchyma and are therefore not advised for pyelonephritis; instead, ciprofloxacin and trimethoprim/sulfamethoxazole are utilized [26].

people with diabetes who have acute pyelonephritis are more likely to experience more serious consequences, such as bacteremia, extended hospitalization, or death, than people without diabetes. [27]. Bilateral acute pyelonephritis occurs more frequently in individuals with T2DM [28]. Management of pyelonephritis in diabetic patients presenting with pronounced symptoms—such as nausea, vomiting, or metabolic disturbances—requires initial administration of intravenous antibiotics during hospitalization, while those with milder symptoms can be treated with oral antibiotics [21]. Effective glycemic control, whether through insulin or other hypoglycemic agents, is a vital component of therapy [29]. Furthermore, surgical interventions including drainage, debridement, or computed tomography (CT)-guided procedures may be necessary, depending on clinical severity[30]. In critical presentations such as sepsis, broad-spectrum antibiotics must be administered urgently—ideally within the first hour—alongside fluid resuscitation and prompt diagnostic evaluation via blood and urine cultures. [31].

6. Conclusions

Recurrent UTIs in patients with T2DM represent a multifactorial clinical challenge influenced by metabolic, immunological, and functional abnormalities. Effective management requires an integrated approach combining strict glycemic control, appropriate diagnostic evaluation, and judicious antimicrobial use. Future research should focus on individualized prevention strategies, optimization of non-antibiotic therapies, and the long-term impact of novel antidiabetic agents on urinary tract infection risk.

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