

Risk Factors and Causes of Womens Urinary Tract Infections

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Abstract:

Urinary tract infections (UTI) are common among the female population. It has been calculated that about one-third of adult women have experienced an episode of symptomatic cystitis at least once. It is also common for these episodes to recur. If predisposing factors are not identified and removed

Keywords: urinary tract, Escherichia coli, staphylococcus, gram-negative, uropathogenic, pregnancy, klebsiella.

Introduction

UTI can lead to more serious consequences, in particular kidney damage and renal failure. The aim of this review was to analyze the factors more commonly correlated with UTI in women, and to see what possible solutions are currently used in general practice and specialized areas, as well as those still under investigation. A good understanding of the possible pathogenic factors contributing to the development of UTI and its recurrence will help the general practitioner to interview the patient, search for causes that would otherwise remain undiscovered, and to identify the correct therapeutic strategy.

Escherichia coli and *Staphylococcus saprophyticus* account for about 80% of community-acquired uncomplicated urinary infections (UTI), particularly in women under 50 years of age. *E. coli* is a Gram-negative commensal of the distal colon which also harbors other anaerobic bacteria, including *Bacteroides* and *Bifidobacteria*. Uropathogenic *E. coli* differs from intestinal pathogenic *E. coli* with regard to the presence of specific virulence factors. Among the various serotypes of *E. coli*, 01, 02, 04, 06, 07, 08, 016, 018, 025, and 075 are commonly recovered from patients with UTI. About 80% of uropathogenic *E. coli* express P fimbriae which anchor to the glycolipid of the outer membranes of urothelial cells localized in the kidney. P fimbriae are frequently associated with cases of acute pyelonephritis.

Another important virulence factor is Type 1 fimbriae. These are very important in the mechanism of bacterial adhesion to the uroepithelium. They are comprised of several subunits, the most important of which is an adhesion protein known as FimH which plays a principal role in the pathogenic mechanism of *E. coli* at the level of the urinary tract. It mediates both cellular invasion of *E. coli* and adhesion to mannose-containing glycoproteins. In vivo and in vitro studies have shown that the pathogenicity of *E. coli* is mainly related to mechanisms of colonization and invasion of the bladder epithelium and the ability to form intracellular bacterial communities. FimH enables uropathogenic *E. coli* to escape the innate immune response by internalization within urothelial cells, mediated by the activation of host signal transduction cascades via protein tyrosine kinases, phosphoinositide-3 kinase, and localized host actin cytoskeletal rearrangements. After invasion, *E. coli* is harbored within vesicles inside infected urothelial cells, from which it cannot be expelled. FimH also plays a role in the modulation of apoptosis in epithelial cells and in the mechanisms of cytoplasmic replication. Therefore, it is a very powerful factor in invasion and virulence. When FimH is not present, the uroepithelium is usually able to clear *E. coli* colonies via expulsion and mechanisms of the cellular immune response.

S. saprophyticus is a common UTI pathogen in younger women. It is reported to colonize the rectum and, to a lesser extent, the cervix and urethra in a small proportion of women. *E. coli* is the main organism responsible for nosocomial UTI, but other Gram-negative pathogens, including *Pseudomonas* spp, *Enterobacter* spp, *Serratia* spp, *Citrobacter* spp, and urease-producing *Klebsiella* spp, *Proteus* spp, *Corynebacterium urealyticum*, and *Providencia* spp are also involved in this type of infection. They are commonly involved in nosocomial UTI due to the inability of antibiotics to penetrate into the biofilm formed around and within the infectious stone. Also, Gram-positive bacteria, including *Enterococcus* spp and *Staphylococcus* spp, can cause nosocomial UTI due to selective pressure from the antimicrobial agents used in hospitalized patients. Anaerobic bacteria are also described in UTI, but their role is not well-defined. Also increasing are the numbers of fungal UTI, particularly those caused by *Candida* spp, and, to a lesser extent, by *Aspergillus* spp and *Cryptococcus neoformans*. In one study, positivity for *Candida* spp was found in 5% of urine specimens from a general hospital and in 10% from a tertiary-level care center. Most UTI caused by *Candida* spp are associated with the use of indwelling urinary devices, including Foley catheters, internal stents, and percutaneous nephrostomy drains. Diabetics are particularly prone to fungal UTI. Normally this kind of infection is acquired by patients with severe neutropenia, drug abuse, recent surgery (chest, abdomen), and systemic infection.

Altered vaginal biota

Lactobacilli are the dominant bacteria in the vaginal biota and possess antimicrobial properties that regulate other urogenital microbiota by maintaining an acidic pH in the vagina and producing hydrogen peroxide. Incomplete cure and recurrence of genitourinary tract infection leads to a shift in the local flora from a predominance of lactobacilli to coliform uropathogens. In a study by Kirjavainen et al it was confirmed that, in bacterial vaginosis, the normal lactobacillus microbiota of the vagina shifts to a more mixed composition, which is also typical during disease-free periods in women prone to UTI. While the vaginal flora were not dominated by the most common uropathogens per se, they were likely to be defective in terms of resisting infection because of the lack of colonization resistance-enhancing properties conferred by lactobacillus accumulation. The use of lactobacillus-containing probiotics has been suggested for the treatment and prophylaxis of bacterial urogenital infection. However, the results of UTI prophylaxis studies using lactobacilli

remain inconclusive. An alteration in vaginal flora is also observed during the postmenopausal period, because estrogens stimulate the proliferation of lactobacilli and reduce the local pH.

Pregnancy

In a prospective study by Haylen et al 1140 women aged 18–98 years were examined for recurrent UTI in different physiologic and pathologic conditions. A correlation was observed between nulliparity and recurrent UTI, particularly in woman younger than 50 years. The authors suggested that stretch and relaxation of the birth canal caused by pregnancy and delivery may be beneficial in preventing recurrent UTI by reducing the frictional effect of intercourse. A reduced frequency of intercourse and number of sexual partners may also account for these findings. In this study, there was a positive relationship between recurrent UTI and a postvoid residual urine volume of >30 mL. Pregnancy is associated with asymptomatic bacteriuria in 4%–7% of cases and pyelonephritis in 0.5%–2%. Many studies have reported that pyelonephritis is more common during the second half of pregnancy. This is thought to result from increasing mechanical compression by the enlarging uterus. A history of recurrent UTI, diabetes, and anatomical abnormalities of the urinary tract increase the risk of developing a UTI during pregnancy. One of the most important risk factors for symptomatic infection is asymptomatic bacteriuria. Asymptomatic bacteriuria is the main risk factor for development of acute pyelonephritis. During pregnancy, development of UTI is correlated with stasis of urine in the ureters, which impairs emptying of the bladder, with an increased postvoid residual urine volume, vesicoureteral reflux, and increased urinary pH.

Menopause

The incidence of UTI in women increases with advancing age. Bacteriuria occurs in about 10%–15% of women aged 65–70 years and in 20%–50% of women aged over 80 years. These values are higher than the 5% rate of bacteriuria reported in premenopausal women. In a study conducted by Raz et al in a group of 149 postmenopausal women, a strong association was noticed between anatomical and functional alterations of bladder emptying and recurrent UTI. Inherited factors and a history of premenopausal UTI have also been suggested as predisposing factors for the development of recurrent UTI. After menopause, there is a significant reduction in estrogen secretion by the ovary, which is often associated with vaginal atrophy. Estrogens stimulate proliferation of *Lactobacillus* in the vaginal epithelium, causing reduction of vaginal pH, thereby preventing vaginal colonization by *Enterobacteriaceae*. In addition, the absence of estrogens decreases the volume of the vaginal muscles, resulting in slackness of the ligaments holding the uterus, pelvic floor, and bladder, resulting in prolapse of the internal genitalia. A placebo-controlled, double-blind study demonstrated a correlation between reduced estrogenic hormone levels after menopause and the development of recurrent UTI. Application of topical vaginal estrogens markedly reduced the incidence of recurrent UTI in this study. In older institutionalized women, the main factors for development of recurrent UTI are deterioration in functional status and urethral catheterization.

Diabetes

Diabetes is another condition correlated with the development of asymptomatic bacteriuria and recurrent UTI. In a multicenter study of 589 women aged 18–75 years and affected by either Type 1 or 2 diabetes, the risk of developing a symptomatic UTI was increased by the same risk factors as those in younger women (sexual intercourse and oral contraceptive use) for Type 1 diabetes and by the presence of asymptomatic bacteriuria for Type 2 diabetes. For Type 1 diabetes mellitus, risk factors for asymptomatic bacteriuria included a longer duration of diabetes, peripheral neuropathy, and microalbuminuria. For Type 2 diabetes, the risk factors were higher age, microalbuminuria, and

a recent symptomatic UTI. The same study showed a weak correlation between increased risk of UTI and poor regulation of diabetes, presence of diabetic cystopathy, neuropathy, microalbuminuria, or macrovascular complications. Glycosuria does not seem to be a risk factor for the development of asymptomatic bacteriuria or symptomatic UTI in vivo. Neuropathy involving the urinary tract may be a potential mechanism that could increase the risk of UTI in diabetics, because it may result in dysfunctional voiding and urinary retention. However, efforts to determine a relationship between high postvoid residual urine and increased risk of UTI have been inconclusive. No relationship between diabetes and uropathogens has been observed. However, first episodes of asymptomatic bacteriuria seem to be less often caused by *E. coli*, and more often caused by *Klebsiella* and *Enterococcus faecalis*. This suggests that asymptomatic bacteriuria and symptomatic UTI may be different disease processes, and that bacterial colonization in the absence of symptoms differs in patients with diabetes.

In conclusion, UTI is more difficult to treat in women than in men because of its multifactorial etiology and the limitations posed by the possibility of pregnancy. The first objective for the physician is to identify women at risk for recurrence of UTI as well as to recognize and remove individual risk factors.

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