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# Urinary Tract Infections in Pregnancy

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Urinary tract infections (UTIs) represent the most common bacterial infection in pregnant and nonpregnant women [1,2]. Eight million women visit a physician annually for evaluation of UTIs [3] at a direct cost of $659 million [4] and aggregate cost of $1.6 billion [4,5]. Physiologic changes of pregnancy increase a woman's susceptibility to UTI. Progesterone effects and mechanical compression by the gravid uterus impair emptying of the bladder and lead to increased bladder residual volume and vesicoureteral reflux. Relative stasis of urine in the ureters results in hydronephrosis. Furthermore, pregnancy-related changes in glomerular filtration rate increases the urinary glucose concentration and alkalinity, thereby facilitating bacterial growth [6]. In addition, alterations in maternal immunologic defense mechanisms occur in pregnancy [7]. The signs and symptoms of UTIs vary by the type of infection. UTI in pregnancy is classified by the site of bacterial proliferation as follows: asymptomatic bacteriuria (ASB; urine), cystitis (bladder), pyelonephritis (kidney).

## Asymptomatic bacteriuria

ASB is defined as significant bacterial colonization of the lower urinary tract without symptoms. Traditional diagnostic criteria of significant bacteriuria include culture of $10^5$ colony forming units (CFUs)/mL of a single uropathogen on two consecutive clean catch urine specimens [6,7]. Recent evidence suggests that

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lower colony counts ($\geq 10^2$–$10^3$ CFUs/mL) may demonstrate active infection and eventually lead to pyelonephritis in pregnant women [8–10]. The incidence of ASB during pregnancy is 2% to 14%—similar to that of nonpregnant women—and translates into 80,000 to 400,000 cases in the United States each year [10,11]. Predisposing factors to ASB include low socioeconomic status, increasing age, multiparity, sexual behavior, and a history of childhood UTIs (with or without scarring). The prevalence of ASB also is increased markedly in certain preexisting medical conditions, such as diabetes mellitus, sickle cell disease, immunocompromised states (eg, AIDS), urinary tract anatomic anomalies, and spinal cord injuries. UTI before pregnancy is a predictor of the diagnosis of ASB at the first prenatal visit [12].

Without treatment, ASB progresses to pyelonephritis in 20% to 40% of pregnant women. In contrast, progression to pyelonephritis in nonpregnant women is only 1% to 2%. Furthermore, the incidence of pyelonephritis in pregnant women without ASB complicating early pregnancy is less than 1%. With appropriate treatment in pregnancy, progression to pyelonephritis can be decreased to 3% [13].

The causative organisms that are isolated in ASB, cystitis, and pyelonephritis are similar in pregnant and nonpregnant women. Enterobacteriae, a group of gram-negative rods, encompass most colonizing organisms, including *Escherichia coli*, the primary pathogen in 80% to 90% of initial UTIs and 70% to 80% of recurrent infections [6,12,14,15]. Other gram-negative pathogens include *Klebsiella pneumoniae* and *Proteus mirabilis*. Further pathogens include *Pseudomonas aeruginosa* and gram-positive organisms, *Streptococcus agalactiae*, and *Staphylococcus saphrophyticus*. The most virulent strains of *E coli* possess toxins and adhesins, pili, or fimbriae to allow adherence to uroephithelium [12]. These protect the bacteria from urinary lavage and allow bacterial multiplication and renal tissue invasion. Specific O-serotypes of *E coli* have been epidemiologically related to the occurrence of acute pyelonephritis, recurrent infection, parenchymal scarring, and renal failure [16]. Fimbriae P, found in uropathogenic strains of *E coli*, aids in adherence to vaginal and renal epithelium and causes upper UTI [17]. Recently, the class of DR adhesins also has been associated with pyelonephritis in pregnancy, and a high rate of preterm delivery in mice [18].

Screening for ASB in pregnancy is recommended by the U.S. Preventative Services Task Force and the American College of Obstetricians and Gynecologists [19,20]. A urine culture should be obtained between 12 and 16 weeks of pregnancy. Appropriate therapy for positive urine culture at this time leads to the highest number of bacteria-free weeks in pregnancy. This recommendation is based on a large epidemiologic study from Sweden [13]. Urine culture detects approximately 80% of cases of ASB. The average cost of urine culture ranges from $16 to $45. In a cost analysis, screening with urine culture is cost-effective if the risk of ASB is greater than 2%, the risk of resultant pyelonephritis is greater than 13%, or if the efficacy of treatment in preventing pyelonephritis is 38% [21]. In populations with a prevalence of ASB of at least 9%, urine culture was the