REVIEW ARTICLE

# Asymptomatic bacteriuria in pregnancy

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**ABSTRACT**

Asymptomatic bacteriuria (ABU) is common in pregnant women and around 4-7% pregnant women actually suffer from this condition. A history of previous urinary tract infections and low socioeconomic status are risk factors for bacteriuria in pregnancy. *Escherichia coli* is the most common aetiologic agent in asymptomatic infection (also in symptomatic infection) and a quantitative (semiquantitative) culture is the gold standard for diagnosis. Treatment of asymptomatic bacteriuria has been shown to reduce the rate of pyelonephritis in pregnancy and therefore screening for and treatment of asymptomatic bacteriuria has become a standard of obstetrical care. Antibiotic treatment of asymptomatic bacteriuria is associated with a decrease in the incidence of low birth weight though there are some controversies in the conclusions drawn by different studies. Empirical antibiotic treatment is the mainstay of therapy in non-reccurrent type ABU. However, there is no clear consensus in the literature on antibiotic choice or duration of therapy for infection. With increasing antibiotic resistance, consideration of local resistance rates is necessary when choosing empirical therapy.

**Keywords:** Asymptomatic bacteriuria, urinary tract infection, UTI, pregnancy, pyelonephritis, drug resistance, screening for bacteriuria.
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Pregnancy is a distinctive state with both anatomical and physiological, largely reversible, alterations in the urinary tract. There is almost no difference between incidence rates of bacteriuria in pregnant and non-pregnant women, but the risk of former progressing to acute pyelonephritis is significant [1]. Bacteriuric pregnant women also have increased susceptibility to other pregnancy related complications compared to pregnant women without bacteriuria. Asymptomatic bacteriuria (ABU) in a non-pregnant woman is considered benign [2]. Earlier Kincaid-Smith et al had concluded that women with asymptomatic bacteriuria during pregnancy are more likely to deliver premature or low-birth-weight infants and have a 20- to 30-fold increased risk of developing pyelonephritis during pregnancy compared with women without bacteriuria [3].

A Cochrane systematic review found that treatment of asymptomatic bacteriuria in pregnancy decreases the risk of subsequent pyelonephritis from a range of 20 to 35 percent to a range of 1 to 4 percent [4]. Antimicrobial treatment of asymptomatic bacteriuria also improves fetal outcomes, with decreases in the

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frequency of low-birth-weight infants and preterm delivery [5, 6].

Urinary tract infections in pregnancy can be either asymptomatic or symptomatic. Asymptomatic variety, also called asymptomatic bacteriuria i.e. ABU, is defined as significant bacteriuria (as explained by Kaas etc – see below) without the symptoms of acute urinary tract infection. Symptomatic urinary tract infections are divided into lower tract (usually acute cystitis) and upper tract (acute pyelonephritis) infections. Cystitis is defined as significant bacteriuria with associated bladder mucosal invasion, whereas pyelonephritis is defined as significant bacteriuria with associated inflammation of the renal parenchyma, calices and pelvis [7].

Screening and treatment of ABUs in pregnancy is an integral part of modern obstetric care. In fact every antenatal care guidelines include routine screening for ABU [8-13]. Despite these, there is an ongoing debate regarding the role of asymptomatic bacteriuria in perinatal outcomes. Controversy also exists regarding treatment of ABU as well as whether antibiotic treated pyelonephritis leads to adverse pregnancy outcomes or not [14].

**Pathophysiology**

UTI as a whole, including both ABU and symptomatic cases, is the most common medical complication of pregnancy. Covert bacteriuria increases the risk of preterm birth and low-birth weight (LBW) infants [6,15,16]. Urinary tract (except the distal urethra) is generally sterile. Ascension of bacteria up urethra into bladder result is ABU. The source of bacteria is usually the normal flora of the GI tract, vagina, or periurethral area. Instrumentation too can introduce bacteria into bladder. In patients with ABU, bacteria persist in the urinary tract but cannot elicit sufficient enough host response to result in either symptoms or eradication of bacteria. Interestingly, *E coli* strains, the most common pathogen of urinary tract, isolated from women with asymptomatic bacteriuria are characterized by fewer virulence characteristics than are those isolated from women with symptomatic infection [17]. Apart from bacterial virulence, factors like host susceptibility, incomplete bladder emptying, obstruction, foreign bodies (e.g. catheter) etc. are important for establishment of infection including ABU [14].

Kass et al documented for the first time that significant bacteriuria can occur in the absence of symptoms or signs of UTIs [18]. Other studies found persistent ABU in 6% antenatal patients and 40% of these people, upon pacebo treatment, went on to develop acute pyelonephritis [19,20]. On the other hand, if bacteriuria was eradicated, pyelonephritis never occurred. Neonatal death rates and prematurity rates were 2-3 times greater in bacteriuric women on placebo compared to treated group/non-bacteriuric group [21,22].

Several factors play roles in replication and ascension of microbe along urinary tract in a pregnant woman [23, 24].

Due to action of progesterone, muscle tone and peristalsis reduces considerably; upper ureter and renal pelvis dilates; and there is a reduced rate of urine passage throughout the urinary collecting system. Entire process leads to a state of physiological hydronephrosis

Enlarging uterus results in a mechanical obstruction to flow.

Pregnancy associated urinary bladder changes like decreased tone, increased capacity, incomplete emptying etc may culminate into vesicoureteric reflux, further facilitating the ascending migration of bacteria

Urinary pH elevation during pregnancy encourages bacterial growth.

Glycosuria, common in pregnancy, favors an increase in the rate of bacterial replication.

The increased urinary excretion of estrogen may be a factor in the pathogenesis of UTI [25,26]

The renal medulla is found to be susceptible to infection because its hypertonic environment inhibits leukocyte migration, phagocytosis, and complement activity [26-28].

Majority ABU of pregnancy will be detected at the initial antenatal visit itself, as only miniscule of them actually develop bacteriuria in later stage of pregnancy [1,19,29,30]. Evidences are galore that bacteriuria preludes the

pregnancy. Studies proved that incidence rate of ABU in non-pregnant population are similar to that in pregnant women in a given locality. Pregnancy related cases may have acquired ABU early in their life with incidence of bacteriuria increasing as a result of sexual activity. Although pregnancy per se does not cause any major increase in bacteriuria, it does predispose to the development of acute pyelonephritis [1,23,24].

**ABU rate in pregnancy**

Studies have confirmed the prevalence rate of ABU in pregnancy to be 2% -11% with majority investigators reporting it to be between 4% to 7% [1,3,19,31-63]. Socio-economic factors may be an important decider in the incidence of ABU in pregnant subjects. Infection is found to be five times more in impoverished woman. Sickle cell disease and Diabetes mellitus enhances the risk of bacteriuria, with the latter is also significantly associated with non-*E coli* infections (e.g. Klebsiella, Proteus etc) [64,65]. Previous UTI too can be an important risk factor for occurrence of ABU in pregnancy [66]. Bacteriuria in female is also associated with lower availability of medical care, increased parity etc [14].

Untreated ABU leading to acute pyelonephritis (in 20-30% pregnant lady) is a well-known fact and hence adequate screening and management steps for ABU is an integral part of all modern antenatal care guidelines [23]. On the other hand claims such as ABU antedating anaemia [45,51,60,61]. Hypertension, preeclampsia [36,37,49,54,61]. Chronic renal disease, Amnionitis [62], endometritis [63] etc. are controversial and need further evidence to be convincing to one and all. Though initially considered controversial, the association between bacteriuria and prematurity and LBW infants are established fact [6,15,16].

**Pathogens**

*E. coli* is isolated in almost 60%-90% ABU in pregnant women in different studies carried out all over the world, at different periods of time. Other common agents include *Proteus mirabilis, Klebsiella pneumoniae, Enterococcus,* Group B beta-haemolytic streptococci [61], *Staphylococcus saprophyticus* etc [68,69].

***Bacteriuria and Pyelonephritis***

One amongst the common medical complications of pregnancy i.e. Acute pyelonephritis is a threat to both maternal and the fetal wellbeing [23,70]. Studies carried out during both in the pre-antibiotic era as well as after discovery of antibiotics convincingly prove the association between acute pyelonephritis and an increased risk of premature delivery [19,36,71-83]. Study by Millar et al even noted an additional confounding factor socioeconomic status as it is related to both UTI and prematurity [23].

Earlier several explanations for this association were put forwarded, including (a) pyrogens increase myometrial activity [84]; (b) ureteric contractions result in reflex myometrial contractions [71]; (c) endotoxin of Gram-negative bacteria associated with pyelonephritis has a direct oxytocic effect on the myometrium [54,86] ; or (d) endotoxin crosses the placenta, resulting in fetal effects culminating in preterm labor. Other theories include bacterial enzymes (such as proteases and collagenase) may lead to rupture of membrane and initiating the onset of labor [87]. Additionally Bacterial products such as phospholipase or endotoxins may stimulate synthesis of prostaglandins from the membranes or decidua leading to labor [88]. These bacterial products have the potential to activate appropriate cells (e.g. monocytes, macrophages) of the immune system to release cytokines (e.g.IL-1, IL-6, tumor necrosis factor, platelet activating factor etc) and thus triggering prostaglandin production [86].

The concept of quantitative urine cultures enabled detection of UTI in individuals without symptoms or signs and it was also a major contribution to the understanding of the pathogenesis of pyelonephritis. Kass's initial studies determined that the presence of untreated/placebo treated ABU was the most significant factor associated with the development of acute pyelonephritis in pregnancy [18,19].

Subsequent studies confirmed that screening pregnant ABU cases actually implies detection of a group of patients at high risk to develop acute

pyelonephritis during pregnancy. About 13.5% to 65% of pregnant women with untreated ABU subsequently developed acute pyelonephritis during pregnancy [3,31,36,42,43,47-52,56,80,87]. Detection and treatment of pregnant women with ABU significantly reduces the risk of developing pyelonephritis.

Many investigators found that only a few women without bacteriuria at the first antenatal visit will develop pyelonephritis. The reason for this could be that approximately 1% of pregnant women who do not have bacteriuria at the first antenatal visit acquire ABU later in pregnancy. These women are then at risk for developing pyelonephritis [14]. In pregnant women with treated bacteriuria, the incidence of pyelonephritis ranged from 0% to 5.3%, with an average of 2.9%. Additionally, few women with bacteriuria will develop pyelonephritis before their first antenatal visit. Detection and eradication by treatment of bacteriuria early in pregnancy may not completely eliminate pyelonephritis, it should prevent at least 70% to 80% of the cases of pyelonephritis in pregnancy [14].

Widespread screening for ABU in pregnancy has significantly reduced the incidence of pyelonephritis in pregnancy [89-91]. Wadland et al noted that screening for ABU in pregnancy was cost-effective when the prevalence of bacteriuria was more than 2% [92]. Rouse et al. performed a cost-effectiveness and cost-benefit analysis and concluded that screening for and treating ABU to prevent pyelonephritis in pregnancy is cost beneficial [93].

***Bacteriuria & Anemia***

Though there were some suggestion of an association between ABU in pregnant women and presence of anemia, many other studies failed in documenting such association [1,32,34,44,45,48,51,59,94]. It is a fact that bacteriuric women with subclinical renal disease would have a greater risk of developing anemia. Socioeconomic deprivation, bacteriuria, and anemia are features common to patients going to prenatal clinics. Hence causal relationship between bacteriuria and anemia remains to be convincingly demonstrated.

***Bacteriuria & Hypertension***

A higher incidence of hypertensive disease of pregnancy has been suggested to exist in pregnant women with ABU. Some investigators have confirmed this postulate, but majority studies have failed to confirm this association [3,31,32,34-36,42-45,49,60]. Even those studies supporting the association/causal relationship could not conclude convincingly that eradication of bacteriuria can result in reduction of hypertensive disease of pregnancy among bacteriuric women [3,22,34,42-44].

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***Bacteriuria & Chronic Renal Disease***

Studies estimated that 10% to 15% of bacteriuric pregnant women are destined to have evidence of chronic pyelonephritis 10 to 12 years after delivery, and that renal failure will ultimately develop in 1 of 3,000 pregnant women with bacteriuria [89,96].

There are sufficient evidences that women with untreated ABU during pregnancy have persistent bacteriuria over the year post delivery in 35% to 80% of cases [52,60,96-97]. Other groups have noted that bacteriuria still persisted in 20% to 30% of patients, even when the bacteriuria had been treated during pregnancy [41,49,80,96]. In contrast, only 5% of women who were not bacteriuric during pregnancy had significant bacteriuria at the 10- to 12-year follow-up examination.

It has been suggested that the patients with evidence of underlying renal involvement are the group at high risk to have persistent bacteriuria after delivery. Studies of antibody-coated bacteria have demonstrated that one half of such ABU cases at least, are renal in origin [99]. Intravenous pyelography as a follow up in asymptomatic bacteriuria during pregnancy has proved radiological evidences consistent with chronic pyelonephritis in about 8%-33% cases [3,32,35,41,43,52,60,96,97,98]. In these cases there were also high incidence of abnormalities like congenital anomalies of the urinary tract, renal calculi, and ureteric dilatation. The highest incidence of radiologic evidence of chronic pyelonephritis was noted in patients with localized upper urinary tract infection or in

bacteriuria during pregnancy which was difficult to eradicate.

Because of the incidence of persistent bacteriuria, abnormal renal function, and radiologic evidence of chronic pyelonephritis, documented in follow-up studies of patients with ABU of pregnancy, long-term follow-up of mothers with bacteriuria is essential. They should be closely followed with periodic urine cultures and treatment if bacteriuria persists or recurs. Patients with relapses and difficult to treat ABUs should undergo (after pregnancy) intravenous pyelograms to detect urinary tract abnormalities that may be correctable or chronic pyelonephritis. With such close surveillance and management, the progression to end-stage renal disease may hopefully be delayed or prevented [14].

***Preterm Delivery & Low-Birth weight Infants***

Though the association between acute pyelonephritis in pregnancy and increased risk of preterm labour/delivery is well documented and accepted, the relationship of ABU to preterm delivery, low-birthweight, small for gestational age babies, and fetal mortality were not without controversy. Few studies initially reported reported association between ABU and prematurity, and that eradication of bacteriuria significantly reduced the rate of premature delivery [18,22,76]. Later on many workers have confirmed the original finding by Kass [3,32,37,42-45,51,54,59,60], but some other investigators have failed to establish an unambiguous relationship between ABU and preterm delivery or low-birth weight infants [1,31,34,36,42,48,52,99]. Although some workers noted that antibiotic treatment of bacteriuria did not significantly reduce the rate of occurrence of low-birth weight infants [3,34], others have reported a reduction in the incidence of premature births when bacteriuria was eradicated with antimicrobial therapy [20,42-45,54,60]. Kincaid-Smith et al suggested that underlying renal disease was the major risk factor for the prematurity or low birth weight among the infants of the bacteriuric women [3]. Gruneberg et al noted that an increased rate of prematurity and a decrease in infants' birth weight occurred in bacteriuric women who were either refractory to treatment or in whom bacteriuria had recurred [33]. Earlier it was reported that bacteriuric patients refractory to treatment are likely to have subclinical renal involvement 48,100]. These were clear support for the hypothesis that women with subclinical renal involvement are at risk to deliver preterm or low-birth weight infants. However, many variables can influence the etiology of prematurity, and bacteriuria is only one of the many factors [15]. Also as the incidence of both pregnancy related bacteriuria and prematurity increases with decreasing socioeconomic status, any relationship between bacteriuria and gestational length and birth weight is complex and difficult to establish.

Romero et al. in a metaanalysis, found that, in pregnancy compared to untreated ABU cases, nonbacteriuric women have significantly less risk of LBW as well as preterm deliveries [6]. Moreover, antibiotic treatment can reduce the risk of LBW. Schieve et al. in a retrospective study found that women exposed to antepartum UTI were at greater risk of delivering infants with LBW, premature infants, preterm infants, and infants who were small for their gestational age [16]. Meta-analysis by Smail and many other large multivariate analyses confirmed these findings [14]. Increased frequency of abortions and stillbirths in pregnant women with bacteriuria has been reported by some investigators [3,32,51,54,60] but not confirmed by others [47,53] An association between maternal bacteriuria and congenital abnormalities has also been proposed [3,45,53]. However, establishment of such causal relationship await further investigations.

***Diagnosis of ABU in Pregnancy***

*Specimen collection and Quantitative /Semi-quantitative culture:*

Ideally the diagnosis of ABU is based on two consecutive midstream urine cultures containing >100,000 CFUs/mL of a single uropathogen. In practice only a single urine specimen is obtained as the expense of using two cultures as a screening test could be too much to bear apart from operational difficulties [23]. Two successive positive urine cultures detect approximately 95% of the cases of ABU,

approaching the accuracy of catheterization [20,101,102]. Use of a single positive culture detects 80% of asymptomatic bacteriuria cases. While it is recommended that pregnant women should be screened for ABU by urine culture at least once in early pregnancy, no recommendation/consensus has emerged for or against repeated screening of culture negative women in later stage of pregnancy. Most common approach is not to rescreen culture negative asymptomatic women later in pregnancy except in those with a history of recurrent UTIs prior to or during pregnancy [14].

Obtaining a good specimen for urine culture requires careful instruction to the patient to minimize contamination from the vagina, distal urethra, and labia. The midstream urine specimen should be obtained ideally after the external genitalia is washed two or three times with an appropriate cleansing solution, from vagina toward anus. A minimum of 4 hours should have elapsed since the last voiding [103].

**Falsely high bacterial counts**

Irrespective of method of collection, bacterial numbers may be increased by delays in culturing the specimen. Since bacteria may divide every 20-30 min, organisms that originally were present in small numbers (as the result of urethral or vulval contamination) can reach "significant" levels rapidly if there is delay in transporting the specimen to the laboratory or in setting up the culture (e.g. a 6-hour delay at room temperature will result in $10^3$ bacteria per milliliter increasing to $10^5$ bacteria per milliliter, urine being a good culture media at room temperature) [14]; or contaminant organisms may overgrow true infection. Keeping the urine specimen at 4°C does not alter colony counts significantly for at least 48 hr. Another method of avoiding such fallacious results is to use slides coated with culture medium ("dip slides") that are dipped into freshly passed urine and incubated immediately [103].

**Falsely low bacterial counts**

Reduced or no bacterial growth may be obtained from an infected urinary tract if the urine contains a bacteriostatic agent. A single 500-mg dose of ampicillin may reduce the concentration from $10^9$ / ml to less than $10^5$/ ml for 3--4 days before the concentration again rises above $10^5$/ ml . If the laboratory knows that the urine contains an antibiotic, penicillinase or other relevant agents can be placed in the culture medium to aid maximum appropriate bacterial growth. Chlorhexidine or other antiseptics used for cleaning the vulva before an MSU is obtained may get into the specimen and effectively sterilize it, so preparative cleaning is best done with saline or water. Diuresis or increased frequency of micturition may dilute the urinary concentration of bacteria or not allow sufficient time for bacterial multiplication to occur so that only low bacterial counts are found [103].

*In the lab*

A urine culture specimen should be processed immediately, because at room temperature, bacteria can begin to multiply, yielding a false-positive result (as explained above).

*Rapid Screening tests*

Because cultures are expensive and require 24 to 48 hours for results, inexpensive, rapid, office-based screening tests have been introduced [23].

Direct Microscopic urinalysis has a poor sensitivity (detects only 25% to 67% of culture positive UTIs), but excellent specificity (97% to 100%) [104,105]

Nitrite Dipstick test has similar credential (sensitivity 50%. specificity from 97% to 100%) [104,105]

Biochemical tests: Griess test, tetrazolium reduction, glucose oxidase, catalase

Gram stain is one of the superior screening test with a sensitivity of 90% and a specificity of 88%. However, this is relatively expensive and technician dependent [23,104]. A Gram stain of a well-mixed, unspun urine demonstrating 2 bacteria per high-power field has 90% correlation with results of quantitative bacteriologic cultures. On the other hand, demonstration of pyuria (more than 5 WBC per high-power field of a centrifuged urine specimen) has variable sensitivity (pyuria is present in 90% of symptomatic UTIs, but in only

50% of asymptomatic bacteriuria) and poor specificity [75,106].

Uricult dipslide paddle,

Cult-Dip Plus

Uristat test

Bioluminescence assays [23]

Uriscreen (sensitivity of 100%, a specificity of 81%) [84]

Despite the fact that rapid tests are less costly, culture of urine remains the screening test of choice for detecting ABU in pregnant patients. Screening for ABU with a midstream clean-catch urine culture should be obtained at the first antenatal visit [23,107-109]. Among women whose cultures are negative at the initial screen, only 1% to 1.5% acquires bacteriuria later in pregnancy [1,20]. Thus, repeated screening may not be recommended.

*Localization:*

ABU may be due either to the *de novo* acquisition of bacterial replication via ascending urethral infection or seeding of the urine from above due to chronic pyelonephritis. The distinction is of great therapeutic importance. In the case of the former, eradication of the bacteria can be readily achieved with short-term administration antibiotics. Chronic disease requires long-term therapy. The major diagnostic problem is how to distinguish between these two entities. Localization as to the site of ABU can be achieved by ureteral catheterization, bladder washout techniques, and the analysis of IgG antibody-coated bacteria. The former two are invasive techniques of purely academic interest. Only the antibody-coated bacteria determination is a practical noninvasive technique. It is predicated upon the fact that with parenchymal involvement and the elicitation of an inflammatory response, the body responds by elaborating specific antibodies which ultimately adhere to the bacterial surface. Specific bacterial fluorescence can be demonstrated by using an anti-IgG, fluorescence-tagged antibody. Unfortunately, the occurrence of false-positive and false negative results have limited this test's usefulness [110].

***Management***

Detection and treatment of ABU can prevent significant medical complications of pregnancy. Screening at the early antenatal visit with appropriate treatment in bacteriuria cases, may result in prevention of majority antenatal acute pyelonephritis cases [23,24]. This and possible waning in maternal and fetal risk are sufficient enough logic for a universal urine culture screening followed by treatment programme. The expected decrease in the rate of preterm births and low-birth weight infants provides some more justification [6,91].

*Goal:*

Idea of treatment is to maintain sterile urine throughout pregnancy with the shortest possible course of antimicrobial agents in order to minimize the drug toxicity to mother and fetus.

*Antimicrobials:*

Many antibacterial agents have a renal excretion mechanism, as a consequence of which therapeutic concentrations are readily achieved in urine. Drugs like Nitrofurantoin cannot reach therapeutic level in serum but easily reach that in urine. Regimen and drug choice can vary depending on multiple factors [23,111]. Common drugs used in management of ABU in pregnancy are outlined in table 1 [14].

*Duration:*

A continuous therapy until delivery was advised earlier with the belief that this would eradicate the bacteriuria as well as ward of the problem of recurrence, which was considered an issue with short course therapy [20,32,34,44]. Now-a-days it is established that short courses of treatment (e.g. 14 days) is quite effective [33,38,40,43,47,112]. Short courses are preferred as - (i) duration of initial therapy does not affect recurrence (ii) minimizes the adverse effect of drug both in mother and developing fetus in utero (iii) chance of emergence of drug resistance is minimized (iv) costs are kept minimized [14].

| **Table1. Treatment of ABU in pregnancy** | |
|---|---|
| Antimicrobial agent | Regimen |
| Single dose treatment | |
| Ampicillin* | 2g |
| Amoxicillin* | 3g |
| Nitrofurantoin | 200mg |
| Trimethoprim-sulfamethoxazole | 320/1600mg |
| 3-day or 7-day treatment | |
| Ampicillin* | 250mg 4times daily |
| Amoxicillin* | 500mg 3 times daily |
| Cephalexin* | 250 – 500mg 4 times daily |
| Nitrofurantoin | 100mg 2 times daily |
| Sulfasoxazole | Loading 2g then 1 g 4 times daily |
| Trimethoprim-sulfamethoxazole | 160/800mg 2 times daily |
| Suppressive therapy ** | |
| Nitrofurantoin | 100mg at bedtime (during pregnancy) |
| Trimethoprim-sulfamethoxazole | 160/800 mg at bedtime (during pregnancy) |
| *In geographic areas with low level drug resistance to E coli<br>** Therapy for recurrent/persistent ABU | |

Single-dose therapy is not as effective in pregnant patients as it is in nonpregnant patients, and it is not as effective as 3-day or 7-day courses [113-115]. Though either 7-days or 3-days regimens are commonly employed, a recent Cochrane systematic review concluded that there was insufficient evidence to recommend duration of antimicrobial therapy for pregnant women among the single dose, 3-days, 4-days and 7-days treatment regimen [14]. Treatment of ABU is empirical and in vitro susceptibility may not be recommended for the initial positive culture.

One pressing issue in the therapy of UTI/ABU is the emergence of drug resistance in the common pathogens. Even the most common pathogen of UTI i.e. *E coli*, 25% to 70% of isolates demonstrate *in vitro* resistance to many beta lactams as well as sulphonamides [23,24,116]. This has huge implications in devising empiric therapy, which is the mainstay in management of ABU. The drug resistant/sensitive pattern or database of common pathogens could be a crucial factor in devising empiric treatment regimen in a given locality or Institution. Antibiotic policies need to be formulated carefully based on such database [117].

The recurrence of bacteriuria during the same pregnancy has been detected in 16% to 33% of women. Ideally patients with recurrent ABU should be treated with antimicrobials on the basis of the microorganism's sensitivities and then should remain on suppressive antimicrobial therapy (see table 1) for the remainder of the pregnancy and for 2 weeks postpartum. There are instances of treating recurrence successfully with a second short period of therapy. The effectiveness of therapy for ABU is best documented by reports demonstrating that treatment of ABU significantly decreases the incidence of acute pyelonephritis [23,24,90,92,114] and preterm birth and LBW [6,91].

**Prevention**

As ABU predates pregnancy and not generally acquired during pregnancy, no prevention strategy is available. Recurrent ABU is noticed in upto 30% cases. Close monitoring with frequent urine culture subsequent to the diagnosis and treatment of ABU in early pregnancy can prevent recurrent ABU. Diagnosis, treatment and eradication of ABU in pregnant women substantially reduce acute pyelonephritis and preterm birth cases [14].

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