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Group B streptococcal infection in pregnant women

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INTRODUCTION — Group B streptococcus (GBS; Streptococcus agalactiae) is a gram-positive coccus that frequently colonizes the human genital and gastrointestinal tracts, as well as the upper respiratory tract of young infants [1,2]. It is an important cause of illness in infants, pregnant women, and adults with underlying medical conditions [3].

In pregnant and postpartum women, GBS is a frequent cause of asymptomatic bacteriuria, urinary tract infection, upper genital tract infection (ie, intraamniotic infection or chorioamnionitis), postpartum endometritis (8 percent), pneumonia (2 percent), puerperal sepsis (2 percent), and bacteremia without a focus (31 percent). It also can cause focal infection such as meningitis and endocarditis, albeit rarely. The serotype distribution of invasive GBS infection in pregnant women is similar to that of early-onset neonatal disease [4].

GBS infection in pregnant women will be reviewed here. The microbiology of GBS; GBS infection in neonates, young infants, and nonpregnant adults; and prevention strategies through chemoprophylaxis and vaccination are discussed separately. (See "Group B streptococcus: Virulence factors and pathogenic mechanisms" and "Group B streptococcal infection in neonates and young infants" and "Group B streptococcal infections in nonpregnant adults" and "Neonatal group B streptococcal disease: Prevention" and "Management of the infant whose mother has received group B streptococcal chemoprophylaxis" and "Vaccines for the prevention of group B streptococcal disease".)

EPIDEMIOLOGY — GBS infections in pregnant women include urinary tract infection, upper genital tract infection, intraamniotic infection, endometritis, and bacteremia [5,6]. Invasive maternal infection with GBS is associated with pregnancy loss and preterm delivery [4,7]. Prior to the widespread use of maternal intrapartum chemoprophylaxis, maternal colonization with GBS conferred an increased risk of chorioamnionitis, and early postpartum infection [8,9]. There does not appear to be an association between maternal GBS colonization during pregnancy and preterm delivery [10].

In the Centers for Disease Control and Prevention (CDC) surveillance study including data collected from 1999 to 2005, the rate of invasive infection (defined as isolation of GBS from a blood or other usually sterile body site, excluding urine) in pregnant women was 0.12 per 1000 live births (range 0.11 to 0.14 per 1000 births) [4]. Upper genital tract infection accounted for approximately one-half of cases, isolated bacteremia occurred in one-third of cases, and GBS was isolated from maternal blood in approximately one-half of cases. Among women for whom pregnancy outcome data were available, approximately one-half of the maternal GBS infections led to fetal death, neonatal infections, neonatal death, or pregnancy loss.

COLONIZATION — Colonization of pregnant women by GBS is a major risk factor for neonatal GBS infection. Issues related to management of colonization and antibiotic prophylaxis for prevention of neonatal infection are discussed separately. (See "Neonatal group B streptococcal disease: Prevention".)

INFECTIONS

Urinary tract — GBS is a frequent cause of asymptomatic bacteriuria, cystitis, and pyelonephritis during pregnancy. Meta-analyses of the impact of asymptomatic bacteriuria in pregnancy demonstrate an association between untreated, asymptomatic bacteriuria (independent of the bacterial species) with progression to pyelonephritis, and with low birth weight or preterm delivery [11,12]. The risk of adverse outcome is decreased with antibiotic treatment of asymptomatic bacteriuria in pregnancy [11,12].

Asymptomatic GBS bacteriuria in pregnancy is a marker for heavy genital colonization with GBS and, as such, is associated with increased risk of upper genital tract infection and postpartum endometritis [6,13]. Although Escherichia coli is the most frequently isolated organism in bacteriuria, cystitis, and pyelonephritis in pregnancy, GBS is isolated in 7 to 30 percent of pregnancy-associated cases of asymptomatic bacteriuria [14,15]. (See "Approach to the adult with asymptomatic bacteriuria", section on 'Pregnancy'.)

Asymptomatic bacteriuria — Asymptomatic bacteriuria is identified by screening urine cultures that are obtained during prenatal visits. At least one screening culture should be obtained during early pregnancy [16]. Asymptomatic bacteriuria in pregnancy is as defined separately for nonpregnant adults. (See "Approach to the adult with asymptomatic bacteriuria".)

The utility of treating GBS bacteriuria at colony counts ≤10^5 prior to 35 weeks gestation is controversial; some favor this approach to prevent the subsequent development of pyelonephritis and to prevent preterm delivery [17]. We recommend treatment if ≥10^4 colonies per mL are detected; this is the cutoff for reporting that is recommended in CDC and ACOG guidelines [3,18] and is consistent with obstetric expert opinion. We still recommend intrapartum prophylaxis regardless of the quantity of GBS detected. In a prospective study of 69 women at 27 to 31 weeks of gestation with GBS bacteriuria, treatment at all colony counts significantly decreased the rates of preterm labor (5 versus 38 percent) and preterm rupture of the membranes (11 versus 53 percent) [19]. In a retrospective study of 305 women in early pregnancy (122 with bacteriuria of any colony count and 183 without bacteriuria), an association was observed between untreated GBS bacteriuria and chorioamnionitis at delivery (adjusted odds ratio 7.2; 95% CI 2.4-21.2) [13].

Treatment consists of antibiotic therapy with amoxicillin, penicillin, or cephalexin. These drugs have not been associated with an increased risk of adverse pregnancy outcome or teratogenic effects. For patients who have a severe IgE-mediated hypersensitivity to penicillins and cephalosporins, clindamycin is the only oral alternative, if the isolate is susceptible. For cases in which the isolate is resistant to clindamycin, investigation and confirmation of the nature of the allergy is critical, and ultimately desensitization may be warranted. (See "Penicillin allergy: Immediate reactions".)

The recommended duration of therapy is three to seven days [16]. Sterile urine must be documented after treatment, and periodic screening cultures should be obtained throughout the pregnancy during routine visits to identify recurrent bacteriuria.

Genital colonization with GBS persists despite adequate therapy for GBS bacteriuria. Women with documented GBS bacteriuria should not be screened for GBS rectal/vaginal colonization later in pregnancy but should be considered GBS colonized and receive intrapartum chemoprophylaxis at the time of delivery. (See "Neonatal group B streptococcal disease: Prevention".)

Cystitis — Cystitis is diagnosed by a positive urine culture in the clinical setting of urinary frequency, urgency, and dysuria without fever. It is treated with the same oral antibiotic regimens as asymptomatic GBS bacteriuria (discussed in the preceding section). A repeat urine culture demonstrating clearance of the organism, and periodic repeat cultures for bacteriuria should be obtained at each subsequent routine prenatal visit.

Pyelonephritis — Pyelonephritis during pregnancy is diagnosed by a positive urine culture in the clinical setting of fever, urinary symptoms, nausea/vomiting, flank pain, and/or costovertebral angle tenderness. In a series of 440 cases of pyelonephritis in pregnancy, GBS accounted for 10 percent of cases [20]. Treatment includes intravenous hydration and intravenous antibiotics (empirical ampicillin plus gentamicin or a cephalosporin). If GBS is identified as the cause of pyelonephritis, treatment with penicillin G may be administered for a total duration of 10 days, tailored to evidence of clinical improvement [21]. In otherwise uncomplicated cases, treatment can be continued with an oral agent (such as amoxicillin or cephalexin) after resolution of fever and other severe symptoms.

In the setting of a serious IgE mediated allergy to penicillin and cephalosporins, vancomycin should be employed until there is a clinical and microbiological response (ie, negative urine culture). Oral clindamycin can be used to complete the course of therapy (eg, 10 to 14 days depending on clinical response). Even though clindamycin is not a typical agent for pyelonephritis, it is a reasonable option in this limited situation. In the setting of known or suspected clindamycin resistance, oral cephalexin is an alternative if the patient is able to tolerate a test dose before hospital discharge. Otherwise, intravenous vancomycin can be used for the complete course. (See "Urinary tract infections and asymptomatic bacteriuria in pregnancy" and "Acute uncomplicated cystitis and pyelonephritis in women".)

Intraamniotic infection — Intraamniotic infection (IAI), also called chorioamnionitis, is an infection of the amniotic fluid, membranes, placenta, and/or decidua [22]. Clinical manifestations include fever, uterine tenderness, maternal and fetal tachycardia, purulent amniotic fluid, and maternal leukocytosis.

Microbiologic and pathologic criteria for GBS intraamniotic infection include isolation of GBS from culture of placenta, amniotic fluid or amniotic membranes, or from fetal parts in case of pregnancy loss. However, these tissues are frequently contaminated during delivery. An uncontaminated amniotic fluid culture can be obtained by amniocentesis prior to rupture of the fetal membranes. After delivery, the best procedure for placental culture is to peel the amnion off the chorion for a significant amount of fetal surface, and then swab the exposed (and untouched) surface with a sterile swab several times before using the swab for culture inoculation. Fetal cultures can be performed on blood from the umbilical vessels or tissues/body fluids collected at autopsy.

Treatment is discussed separately. (See "Intra-amniotic infection (clinical chorioamnionitis or triple I)", section on 'Maternal management'.)

Endometritis — Colonization with GBS significantly increases the risk of developing postpartum endometritis [7]. In studies of endometritis, GBS has been identified as a single pathogen in 2 to 14 percent of cases but is more commonly a component of polymicrobial infections [23]. Endometritis is treated with broad-spectrum antibiotics including anaerobic coverage (ampicillin and clindamycin plus gentamicin or cefoxitin alone). In cases of life-threatening endometritis or incipient sepsis, broader antibiotic coverage, for example with a carbapenem and/or vancomycin should be considered. (See "Postpartum endometritis", section on 'Treatment'.)

Bacteremia — In a study of obstetric patients in the 1970s, GBS was the second most common cause of bacteremia [24]. In a Finnish review of women with peripartum sepsis in the 1990s, GBS was the single most common organism isolated [25]. Both studies demonstrated a variety of aerobic and anaerobic gram-positive and gram-negative pathogens other than GBS, suggesting that empiric therapy for suspected bacteremia must consist of broad-spectrum therapy that includes anaerobic coverage. Since implementation of maternal intrapartum GBS chemoprophylaxis, data on the distribution of organisms causing peripartum bacteremia have been lacking. A study of 195 peripartum bacteremia bacterial isolates in the era of screening-based GBS prophylaxis (2000-2008) demonstrated that only 4 percent of blood culture isolates were due to GBS. E. coli and enterococci accounted for over half of all isolates, and 13 percent of isolates were anaerobic species [26].

GBS bacteremia is discussed further separately. (See "Group B streptococcal infections in nonpregnant adults".)

Other infections — GBS rarely has been associated with a variety of unusual peripartum infections such as maternal meningitis (both antepartum and postpartum), endocarditis, abdominal abscess, and necrotizing fasciitis [23,27,28], following both live births and elective pregnancy termination [29,30]. These are discussed further separately. (See "Group B streptococcal infections in nonpregnant adults".)

INFORMATION FOR PATIENTS — UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword (s) of interest.)

- Basics topics (see "Patient education: Group B streptococcal disease (The Basics)")
- Beyond the Basics topics (see "Patient education: Group B streptococcus and pregnancy (Beyond the Basics)")

SUMMARY

- Group B streptococcus (GBS; Streptococcus agalactiae) is a gram-positive coccus that frequently colonizes the human genital and gastrointestinal tracts, as well as the upper respiratory tract of young infants. It is an important cause of illness in infants, pregnant women, and adults with underlying medical conditions. (See 'Introduction' above.)
- Invasive maternal infection with GBS is associated with pregnancy loss and preterm delivery. Prior to the widespread use of maternal intrapartum chemoprophylaxis, maternal colonization with GBS conferred an increased risk of chorioamnionitis, and early postpartum infection. It is not clear whether there is an association between maternal GBS colonization during pregnancy and fetal loss or preterm delivery. Issues related to management of colonization and antibiotic prophylaxis for prevention of neonatal infection are discussed separately. (See 'Epidemiology' above and "Neonatal group B streptococcal disease: Prevention".)
- GBS is a frequent cause of asymptomatic bacteriuria, cystitis, and pyelonephritis during pregnancy. There is an association between untreated, asymptomatic bacteriuria (independent of the bacterial species) with progression to pyelonephritis, and with low birth weight or preterm delivery. The risk of adverse outcome is decreased with antibiotic treatment of asymptomatic bacteriuria in pregnancy. (See 'Urinary tract' above.)
- Asymptomatic GBS bacteriuria in pregnancy is a marker for heavy genital colonization with GBS and, as such, is associated with increased risk of upper genital tract infection and postpartum endometritis. Asymptomatic bacteriuria is identified by screening urine cultures that are obtained during prenatal visits. At least one screening urine culture should be obtained during early pregnancy. (See 'Urinary tract' above and 'Asymptomatic bacteriuria' above.)
- Treatment of asymptomatic bacteriuria with GBS consists of antibiotic therapy with amoxicillin, penicillin, or cephalexin. The recommended duration of therapy is three to seven days. Sterile urine must be documented after treatment, and periodic screening cultures should be obtained throughout the pregnancy to identify recurrent bacteriuria. (See 'Asymptomatic bacteriuria' above.)
- Genital colonization with GBS persists despite adequate therapy for GBS bacteriuria. Women with documented GBS bacteriuria should not be screened for GBS rectal/vaginal colonization later in pregnancy but should be considered persistently GBS colonized and receive intrapartum chemoprophylaxis at the time of delivery. (See "Neonatal group B streptococcal disease: Prevention".)
- Cystitis is diagnosed by a positive urine culture in the clinical setting of urinary frequency, urgency, and dysuria without fever. It is treated with the same oral antibiotic regimens as asymptomatic GBS bacteriuria. A repeat urine culture

demonstrating clearance of the organism and periodic repeat cultures for bacteriuria should be obtained. (See 'Cystitis' above.)

- Pyelonephritis during pregnancy is diagnosed by a positive urine culture in the clinical setting of fever, urinary symptoms, nausea/vomiting, flank pain, and/or costovertebral angle tenderness. Treatment includes intravenous hydration and intravenous antibiotics. If GBS is identified as the cause of pyelonephritis, treatment with penicillin G may be administered for a total duration of 10 days, tailored to evidence of clinical improvement. (See 'Pyelonephritis' above.)

- Other infections associated with GBS include intraamniotic infection (chorioamnionitis), endometritis, and bacteremia. Rare peripartum infections include maternal meningitis (both antepartum and postpartum), endocarditis, abdominal abscess, and necrotizing fasciitis. (See 'Intraamniotic infection' above and 'Endometritis' above and 'Bacteremia' above and 'Other infections' above.)

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REFERENCES

1. Edwards MS, Nizet V, Baker CJ. Group B Streptococcal Infections. In: Infectious Diseases of the Fetus and Newborn Infant, 7th ed, Remington JS, Klein JO, Wilson CB, et al (Eds), Elsevier Saunders, Philadelphia 2011. p.419.
2. Eichenwald EC. Perinatally transmitted neonatal bacterial infections. Infect Dis Clin North Am 1997; 11:223.
3. Verani JR, McGee L, Schrag SJ, Division of Bacterial Diseases, National Center for Immunization and Respiratory Diseases, Centers for Disease Control and Prevention (CDC). Prevention of perinatal group B streptococcal disease--revised guidelines from CDC, 2010. MMWR Recomm Rep 2010; 59:1.
4. Phares CR, Lynfield R, Farley MM, et al. Epidemiology of invasive group B streptococcal disease in the United States, 1999-2005. JAMA 2008; 299:2056.
5. Regan JA, Klebanoff MA, Nugent RP, et al. Colonization with group B streptococci in pregnancy and adverse outcome. VIP Study Group. Am J Obstet Gynecol 1996; 174:1354.
6. Krohn MA, Hillier SL, Baker CJ. Maternal peripartum complications associated with vaginal group B streptococci colonization. J Infect Dis 1999; 179:1410.
7. Zaleznik DF, Rench MA, Hillier S, et al. Invasive disease due to group B Streptococcus in pregnant women and neonates from diverse population groups. Clin Infect Dis 2000; 30:276.
8. Schrag SJ, Zywicki S, Farley MM, et al. Group B streptococcal disease in the era of intrapartum antibiotic prophylaxis. N Engl J Med 2000; 342:15.
9. Prevention of perinatal group B streptococcal disease: a public health perspective. Centers for Disease Control and Prevention. MMWR Recomm Rep 1996; 45:1.
10. Valkenburg-van den Berg AW, Sprij AJ, Dekker FW, et al. Association between colonization with Group B Streptococcus and preterm delivery: a systematic review. Acta Obstet Gynecol Scand 2009; 88:958.
11. Romero R, Oyarzun E, Mazor M, et al. Meta-analysis of the relationship between asymptomatic bacteriuria and preterm delivery/low birth weight. Obstet Gynecol 1989; 73:576.
12. Mittendorf R, Williams MA, Kass EH. Prevention of preterm delivery and low birth weight associated with asymptomatic bacteriuria. Clin Infect Dis 1992; 14:927.
13. Anderson BL, Simhan HN, Simons KM, Wiesenfeld HC. Untreated asymptomatic group B streptococcal bacteriuria early in pregnancy and chorioamnionitis at delivery. Am J Obstet Gynecol 2007; 196:524.e1.
14. Wood EG, Dillon HC Jr. A prospective study of group B streptococcal bacteriuria in pregnancy. Am J Obstet Gynecol 1981; 140:515.
15. Persson K, Bjerre B, Elfström L, et al. Group B streptococci at delivery: high count in urine increases risk for neonatal colonization. Scand J Infect Dis 1986; 18:525.
16. Nicolle LE, Bradley S, Colgan R, et al. Infectious Diseases Society of America guidelines for the diagnosis and treatment of asymptomatic bacteriuria in adults. Clin Infect Dis 2005; 40:643.
17. Aungst M, King J, Steele A, Gordon M. Low colony counts of asymptomatic group B streptococcus bacteriuria: a survey of practice patterns. Am J Perinatol 2004; 21:403.
18. American College of Obstetricians and Gynecologists Committee on Obstetric Practice. ACOG Committee Opinion No. 485: Prevention of early-onset group B streptococcal disease in newborns. Obstet Gynecol 2011; 117:1019.
19. Thomsen AC, Mørup L, Hansen KB. Antibiotic elimination of group-B streptococci in urine in prevention of preterm labour. Lancet 1987; 1:591.
20. Hill JB, Sheffield JS, McIntire DD, Wendel GD Jr. Acute pyelonephritis in pregnancy. Obstet Gynecol 2005; 105:18.

21. ACOG educational bulletin. Antimicrobial therapy for obstetric patients. Number 245, March 1998 (replaces no. 117, June 1988). American College of Obstetricians and Gynecologists. Int J Gynaecol Obstet 1998; 61:299.
22. Hitti J, Tarczy-Hornoch P, Murphy J, et al. Amniotic fluid infection, cytokines, and adverse outcome among infants at 34 weeks' gestation or less. Obstet Gynecol 2001; 98:1080.
23. Isada NB, Grossman JH. Perinatal Infections. In: Obstetrics: Normal and Problem Pregnancies, Gabbe SG, Niebyl JR, Simpson JL (Eds), Churchill Livingstone, New York 1991. p.1276.
24. Blanco JD, Gibbs RS, Castaneda YS. Bacteremia in obstetrics: clinical course. Obstet Gynecol 1981; 58:621.
25. Kankuri E, Kurki T, Carlson P, Hiilesmaa V. Incidence, treatment and outcome of peripartum sepsis. Acta Obstet Gynecol Scand 2003; 82:730.
26. Cape A, Tuomala RE, Taylor C, Puopolo KM. Peripartum bacteremia in the era of group B streptococcus prophylaxis. Obstet Gynecol 2013; 121:812.
27. Guerin JM, Leibinger F, Mofredj A, Ekherian JM. Streptococcus B meningitis in post-partum. J Infect 1997; 34:151.
28. Braun TI, Pinover W, Sih P. Group B streptococcal meningitis in a pregnant woman before the onset of labor. Clin Infect Dis 1995; 21:1042.
29. Deziel PJ, McGuire N, Brown PD. Group B streptococcal meningitis complicating elective abortion: report of 2 cases. Clin Infect Dis 2000; 31:E23.
30. Palys EE, Li J, Gaut PL, Hardy WD. Tricuspid valve endocarditis with Group B Streptococcus after an elective abortion: the need for new data. Infect Dis Obstet Gynecol 2006; 2006:43253.

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