Group B streptococcal infection in neonates and young infants

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INTRODUCTION

Group B *Streptococcus* (GBS; or *Streptococcus agalactiae*) is gram-positive diplococcus that commonly colonizes the gastrointestinal and genital tracts. GBS colonization in pregnant women is generally asymptomatic. However, maternal colonization is the primary risk factor for GBS infection in neonates and young infants.

GBS infection in neonates and young infants will be reviewed here. The microbiology of GBS infections, prevention of GBS infection in neonates, assessment of newborns at risk for GBS, and diagnosis and management of neonatal sepsis broadly are discussed separately:

- (See "Group B streptococcal infections in nonpregnant adults", section on 'Microbiology'.)
- (See "Vaccines for the prevention of group B streptococcal disease".)
- (See "Prevention of early-onset group B streptococcal disease in neonates".)
- (See "Management of neonates at risk for early-onset group B streptococcal infection".)
- (See "Clinical features, evaluation, and diagnosis of sepsis in term and late preterm neonates".)
- (See "Management and outcome of sepsis in term and late preterm neonates".)

TERMINOLOGY

Group B streptococcal (GBS) infection in neonates and young infants is classified by age at onset [1].

- **Early-onset GBS** – Early-onset GBS generally presents at or within 24 hours of birth [2] but can occur through day 6 after birth.

- **Late-onset GBS** – Late-onset GBS usually occurs at four to five weeks of age (range 7 to 89 days).

- **Late, late-onset GBS** – Late, late-onset GBS (also called very late-onset GBS or GBS beyond early infancy) occurs in infants older than three months of age. Late, late-onset GBS infections are most common in infants who are born before 28 weeks gestation or in children with a history of immunodeficiency [3,4].

MICROBIOLOGY AND PATHOGENESIS

Group B Streptococcus (GBS; *Streptococcus agalactiae*) is a gram-positive coccus that frequently colonizes the human genital and gastrointestinal tracts. It is an important cause of infection in neonates.

- **Serotype distribution** – GBS serotype corresponds to the capsular polysaccharide. There are 10 GBS serotypes: Ia, Ib, and II through IX [1,5].

  Serotypes Ia, Ib, II, III, IV, and V account for >95 percent of early-onset cases in the United States and >97 percent of late-onset cases [6]. Serotype III has a propensity to cause meningitis and is responsible for a high proportion of late-onset infections [6,7].

  The distribution of serotypes and surface proteins among GBS isolates has important implications for the development of vaccines to prevent GBS disease. (See "Vaccines for the prevention of group B streptococcal disease".)

- **Transmission** – Neonatal early-onset GBS infection is acquired in utero through clinically apparent or "silent" intraamniotic infection or rupture of membranes, as well as during passage through the vagina. Evidence suggests that vaginal colonization with a high inoculum (>10^5 colony-forming units/mL) of GBS during pregnancy increases the risk of vertical transmission [8,9] and early-onset disease in neonates. After discharge from the hospital, neonates and young infants can acquire GBS horizontally from mother or colonized household contacts and go on to develop late-onset bacteremia, meningitis, or other focal infections.

Virulence factors and pathogenic mechanisms of GBS are discussed in detail separately. (See "Group B streptococcal infections in nonpregnant adults", section on 'Microbiology'.)

EPIDEMIOLOGY

Incidence — The estimated incidence of infant GBS disease worldwide is approximately 0.5 per 1000 live births [10,11]. Rates of GBS disease vary from region to region, with the highest rates in Africa and lowest rates in Asia [10].

In the United States, the combined incidence of early- and late-onset GBS disease has declined since the 1990s and early 2000s, from approximately 2 cases per 1000 live births in that era to approximately 0.5 cases per 1000 live births in 2018 (figure 1) [12-14]. Similar declines have been reported in other parts of the world, including Europe and Australia [15-18].

With both early- and late-onset GBS disease, there is an ongoing racial disparity, with Black infants at greater risk of infection than White infants [6,19]. Racial disparities in prematurity may partly explain this finding.

- **Early-onset disease** – Most of the decline of GBS disease is accounted for by a reduction in the incidence of early-onset disease (figure 1). The overall decline in early-onset GBS disease has been attributed to universal screening of pregnant women for GBS colonization and the widespread use of intrapartum antibiotic prophylaxis (IAP) [20-23]. (See "Prevention of early-onset group B streptococcal disease in neonates".)

  The incidence of early-onset GBS disease declined from 1.8 cases per 1000 live births in 1990 to 0.25 cases per 1000 live births in 2018 (figure 1) [6,12-14,24]. Despite implementation of prevention policies, early-onset neonatal GBS disease in the United States continues to occur and has plateaued since the mid-2000s [25-29]. Prior to routine maternal screening and use of IAP, the majority of early-onset GBS disease occurred in term neonates; however, in the contemporary era, approximately 25 percent of cases occur in preterm neonates, among whom the risk of mortality is substantially higher [6,26-28]. (See "Clinical features and diagnosis of bacterial sepsis in preterm infants <34 weeks gestation".)

- **Late-onset disease** – The incidence of late-onset GBS disease in the United States has remained stable since 1990, at approximately 0.3 to 0.4 per 1000 live births (figure 1) [14]. Late-onset disease is not prevented by maternal intrapartum chemoprophylaxis [6,12,13,24,30-33]. Roughly one-third of infants with late-onset disease are born before 37 weeks gestation, with a median gestational age of 30 weeks [6,32].

Risk factors

- **Clinical risk factors** – Important clinical risk factors for early-onset neonatal GBS infection include [20-22,34-37]:

  - Maternal GBS colonization (ie, positive GBS vaginal or rectal screening culture or GBS bacteriuria during pregnancy)
  - Prior delivery of an infant with GBS disease
  - Delivery at <37 weeks of gestation
  - Rupture of membranes for ≥18 hours before delivery
  - Preterm prelabor rupture of membranes
  - Intraamniotic infection (previously called chorioamnionitis)
  - Intrapartum fever (temperature ≥38°C [100.4°F])

  These risk factors are incorporated into guidelines for prevention of early-onset neonatal GBS disease (ie, by screening pregnant women and administering IAP), which are discussed in detail separately. (See "Prevention of early-onset group B streptococcal disease in neonates".)

  Multiple-gestation pregnancy was associated with increased risk of GBS disease (early- or late-onset) in some studies [38,39] but not others [40,41].

- **Bacterial and immunologic risk factors** – Bacterial and immunologic risk factors include [8,9,42-46]:

  - GBS strain with enhanced virulence
  - Heavy maternal colonization (vaginal inoculum >10^5 colony-forming units/mL)
  - Deficient maternal GBS capsular type-specific immunoglobulin G (IgG) at term delivery

  These are discussed in greater detail separately. (See "Group B streptococcal infections in nonpregnant adults", section on 'Pathogenesis'.)

- **Impact of IAP** – The rate of transmission from colonized mothers to infants without IAP is approximately 50 percent [34,47]. However, only 1 to 2 percent of all infants born to colonized pregnant women develop early-onset GBS disease [2,34]. As discussed above, the routine use of IAP has substantially reduced the incidence of early-onset neonatal GBS disease. (See 'Incidence' above.)

  IAP has had no impact on the incidence of late-onset disease, which is not surprising given that IAP eliminates or diminishes neonatal exposure only during labor and delivery; women given IAP continue to be colonized postpartum. This suggests that the GBS exposures of infants with late-onset disease occur in the home from colonized parents or siblings, or in the community, and that these exposures are important in the pathogenesis of late-onset disease [48-50].

CLINICAL MANIFESTATIONS

Early-onset disease — Early-onset GBS infection most commonly manifests as generalized sepsis, pneumonia, or meningitis [6]. In >90 percent of cases, clinical signs are apparent in the first 24 hours after birth.

Infants whose mothers receive intrapartum antibiotic prophylaxis (IAP) are less likely to have sepsis, need assisted ventilation, or have documented GBS bacteremia [51]. Exposure to IAP does not appear to alter the time of onset of clinical signs of infection [47,52].

Sepsis — Sepsis without a focus of infection occurs in 80 to 85 percent of cases of early-onset GBS disease [6]. Signs of sepsis are nonspecific and include irritability, lethargy, respiratory symptoms (eg, tachypnea, grunting, hypoxia), temperature instability, poor perfusion, and hypotension (table 1). Many infants presenting at <24 hours after birth do not have fever, although fever can occur in term infants on the second or third day after birth. Sepsis syndromes range from nonspecific signs to profound septic shock. Early-onset disease can be associated with persistent pulmonary hypertension of the newborn (PPHN). (See "Clinical features, evaluation, and diagnosis of sepsis in term and late preterm neonates" and "Persistent pulmonary hypertension of the newborn (PPHN): Clinical features and diagnosis".)

Pneumonia — Pneumonia accounts for approximately 10 percent of cases of early-onset disease [6]. Signs of pneumonia include tachypnea, grunting, hypoxia, and increased work of breathing. GBS pneumonia also may be associated with PPHN in term infants. (See "Neonatal pneumonia" and "Persistent pulmonary hypertension of the newborn (PPHN): Clinical features and diagnosis".)

Radiographic findings of GBS pneumonia include a diffuse alveolar pattern that can be difficult to distinguish from hyaline membrane disease or transient tachypnea of the newborn [53]. Pleural effusions are more common in GBS pneumonia than in hyaline membrane disease [35,54,55]. (See "Clinical features and diagnosis of respiratory distress syndrome in the newborn" and "Management of respiratory distress syndrome in preterm infants" and "Transient tachypnea of the newborn".)

Meningitis — GBS meningitis accounts for approximately 5 to 10 percent of cases of early-onset disease [6]. Presenting signs are usually nonspecific and may include respiratory abnormalities (eg, tachypnea, grunting, apnea), irritability or lethargy, and poor feeding or vomiting [56,57]. (See "Bacterial meningitis in the neonate: Clinical features and diagnosis", section on 'Clinical features'.)

Late-onset disease — Late-onset GBS disease most often presents as bacteremia without a focus; however, meningitis and other focal infections can occur [58].

Bacteremia — Bacteremia without a focus accounts for approximately 65 percent of late-onset cases [6,59]. Affected infants typically present with fever. These infants may have a history of a preceding or intercurrent upper respiratory infection. Other clinical findings can include irritability, lethargy, poor feeding, tachypnea, grunting, and occasionally apnea.

Meningitis — Meningitis accounts for 25 to 30 percent of cases of late-onset GBS disease [47]. The clinical presentation of neonatal meningitis typically is indistinguishable from that of neonatal sepsis without meningitis. The most commonly reported clinical signs are temperature instability, irritability or lethargy, and poor feeding or vomiting. Signs of central nervous system inflammation (eg, bulging fontanel, nuchal rigidity, focal neurologic findings, seizures) are more common in late-onset than early-onset GBS meningitis [60]. In 20 to 30 percent of late-onset cases, there are preceding signs of upper respiratory infection [34,56,61]. (See "Bacterial meningitis in the neonate: Clinical features and diagnosis", section on 'Clinical features' and "Bacterial meningitis in children older than one month: Clinical features and diagnosis", section on 'Clinical features'.)

Other focal infection — Late-onset disease also can present as pneumonia, septic arthritis, osteomyelitis, and cellulitis-adenitis [58]. Rare presentations of late-onset GBS disease include endocarditis, myocarditis, pericarditis, pyelonephritis, endophthalmitis, and brain abscess, among others [47].

- **Bone/joint infection** – Late-onset GBS infection manifests as septic arthritis and osteomyelitis in approximately 5 percent of cases [47]. GBS septic arthritis generally has an acute presentation and involves the lower extremity; the mean age of onset is 20 days. GBS osteomyelitis typically has an insidious onset; the most frequent sites include the humerus (especially the right proximal humerus), femur, and tibia; the mean age of onset is 31 days. These manifestations of late-onset disease have decreased in frequency, most likely due to more prompt diagnosis and empiric treatment of GBS bacteremia without a focus.

  Decreased movement of the involved extremity and pain with manipulation are important clues to GBS bone and joint infection [47,62]. Fever is absent in the majority of patients. Concomitant bacteremia is present in more than one-half of cases [62].

- **Cellulitis-adenitis** – Late-onset GBS infection can manifest as cellulitis and/or adenitis, most commonly involving the face or submandibular area, though other areas may be affected [47]. Cellulitis-adenitis accounted for 4 percent of late-onset GBS infections in an 11-year series from one institution [63]. Associated examination findings can include ipsilateral otitis media; infected or thyroglossal duct cyst also has been reported [64].

In a review of 32 cases of GBS cellulitis-adenitis, bacteremia was documented in 91 percent of cases [65]. Among the 25 infants in whom cerebrospinal fluid (CSF) was obtained, six grew GBS from the CSF; three infants died (two had CNS involvement and one had fulminant GBS septicemia). Lack of fever and well appearance at presentation did not predict subsequent clinical course.

These observations suggest that lumbar puncture (LP) should be performed in young infants with cellulitis-adenitis and that empiric antibiotics should include coverage for GBS meningitis until CNS infection is excluded [65]. (See 'Evaluation' below and 'Empiric therapy' below.)

Late, late-onset disease — Late, late-onset GBS disease most commonly occurs in preterm infants born at <28 weeks of gestation [3,47,59]. It typically manifests as bacteremia without a focus, but focal sites of infection may be noted, including the CNS, soft tissues, bones and joints, and intravascular catheters [3,6,47].

GBS infection after six months of age can be the first sign of immune deficiency, including HIV infection [3,66,67].

EVALUATION

Clinical suspicion — Infants with signs of sepsis require prompt evaluation and initiation of antibiotic therapy. Because the signs of sepsis in neonates and young infants are subtle and nonspecific (table 1), newborn care providers use established risk factors for neonatal early-onset sepsis together with the newborn clinical condition to identify newborns at highest risk of infection. (See "Clinical features, evaluation, and diagnosis of sepsis in term and late preterm neonates", section on 'Evaluation and initial management'.)

- **Early-onset** – For patients presenting within the first six days after birth (early-onset disease), it is crucial to review the pregnancy, labor, and delivery history to identify factors associated with increased risk of sepsis (eg, maternal intraamniotic infection, inadequate maternal intrapartum antibiotic prophylaxis [IAP], preterm delivery, prolonged duration of rupture of membranes) [68,69].

  For term neonates with risk factors for early-onset GBS disease, accepted approaches to risk assessment include the "categorical" approach (algorithm 1), the "clinical observation" approach (algorithm 2), and the early-onset sepsis calculator. Risk assessment in preterm neonates (ie, <35 weeks gestational age) considers additional factors (ie, the reason for preterm delivery, whether delivery was vaginal or via caesarean section, etc) (algorithm 3). These approaches are discussed in a

separate topic review.(See "Management of neonates at risk for early-onset group B streptococcal infection".)

- **Late-onset** – GBS status of the mother is also important to consider in infants presenting beyond six days of age (late-onset disease); however, factors such as maternal IAP and duration of rupture of membranes are not weighed in decision-making in late-onset disease. Maternal IAP does not decrease the risk of late-onset GBS disease. Decisions to evaluate and initiate empiric treatment for late-onset sepsis are based chiefly on clinical appearance and signs of illness. (See "Clinical features, evaluation, and diagnosis of sepsis in term and late preterm neonates", section on 'Late-onset sepsis' and "The febrile infant (29 to 90 days of age): Outpatient evaluation", section on 'Evaluation'.)

Tests to perform in the index patient — Evaluation for clinically suspected GBS infection includes:

- Blood culture.
- Complete blood count (CBC) with differential:
  - Early onset (first six days after birth) – The white blood cell count does not perform well in predicting risk of early-onset infection and should not be used alone to determine whether a newborn should be treated empirically with antibiotics [70-75]. (See "Clinical features, evaluation, and diagnosis of sepsis in term and late preterm neonates".)
  - Late onset (≥7 days after birth) – When used in conjunction with other assessments, the CBC with differential may be useful in the evaluation of infants with suspected late-onset GBS disease [76]. (See "Clinical features, evaluation, and diagnosis of sepsis in term and late preterm neonates" and "The febrile infant (29 to 90 days of age): Outpatient evaluation", section on 'Inflammatory markers'.)
- Lumbar puncture (LP) – An LP should be performed if there is clinical suspicion for sepsis. LP is usually not necessary when evaluating well-appearing term infants who are assessed as at risk of early-onset GBS disease based upon maternal risk factors. (See "Management of neonates at risk for early-onset group B streptococcal infection", section on 'Diagnostic evaluation'.)

  Cerebrospinal fluid (CSF) should be sent for cell count, protein and glucose concentration, Gram stain, and culture. The LP should be performed before starting antibiotic therapy unless the infant is clinically unstable, in which case, the LP can be deferred. (See "Bacterial meningitis in the neonate: Clinical features and diagnosis", section on 'Lumbar puncture'.)

- Chest radiograph (if respiratory signs present).
- Urine culture (if the infant is >1 week of age) by sterile collection method (urinary catheter or suprapubic aspirate).

Additional studies may be necessary if osteoarticular infection is suspected. These include radiographs of the involved site, magnetic resonance imaging, bone biopsy, or joint aspiration with culture and susceptibility testing. (See "Hematogenous osteomyelitis in children: Evaluation and diagnosis", section on 'Diagnostic approach' and "Bacterial arthritis: Clinical features and diagnosis in infants and children", section on 'Evaluation'.)

Evaluation of a twin sibling — When a twin (or other infant product of a multiple-birth gestation) is diagnosed with GBS disease, the sibling of the index case should be observed carefully [1]. If any signs of illness occur, the twin should undergo laboratory evaluation (as described for the index patient above) and empiric antibiotic therapy should be initiated. (See 'Tests to perform in the index patient' above and 'Empiric therapy' below.)

Although the unaffected twin is likely to be colonized with GBS (given the common contacts), evidence is lacking that prophylactic antibiotic treatment decreases the risk of subsequent invasive infection.

DIAGNOSIS

Isolation of GBS from a normally sterile body site (eg, blood, cerebrospinal fluid [CSF], pleural fluid, bone, joint) confirms the diagnosis of GBS infection. GBS antigen may be detected in CSF, which occasionally can assist in the diagnosis of infection [1]. However, antigen testing of other body fluids is not recommended, because of poor specificity.

MANAGEMENT

Antimicrobial therapy and supportive care combined with drainage of purulent collections as necessary are the cornerstones of treatment of GBS disease in neonates and young infants.

Supportive care — Supportive care for neonates and young infants with GBS infection may include:

- Ventilatory support (including endotracheal intubation and mechanical ventilation) (see "Overview of mechanical ventilation in neonates")
- Prompt recognition and treatment of shock (see "Neonatal shock: Etiology, clinical manifestations, and evaluation")

- Careful maintenance of fluid and electrolyte balance (see "Fluid and electrolyte therapy in newborns")
- Treatment of anemia (see "Anemia of prematurity (AOP)" and "Red blood cell (RBC) transfusions in the neonate")
- Management of seizures (see "Treatment of neonatal seizures")

Management in an intensive care unit is often required.

Antimicrobial therapy

Empiric therapy — Initial empiric antibiotic therapy of suspected sepsis includes broad coverage for organisms known to cause early- and late-onset disease in neonates and infants younger than three months of age (eg, GBS and other streptococci, gram-negative enteric organisms, and, rarely, *Listeria monocytogenes*) (table 2) [47]. Appropriate regimens for empiric therapy vary depending upon the focus of infection and whether the infection is of early onset or late onset. Local antibiotic resistance patterns should also be considered. Empiric therapy for suspected neonatal sepsis is summarized in the table (table 3) and discussed in greater detail separately. (See "Management and outcome of sepsis in term and late preterm neonates", section on 'Initial empiric therapy'.)

Definitive therapy — Once GBS is identified as the sole causative organism and the patient has improved clinically, we recommend that antimicrobial therapy be changed to penicillin G alone (table 4) [1]. Monotherapy with ampicillin is an acceptable alternative. The advantage of penicillin G over ampicillin is that it has less effect in altering the microbiome.

For infants with GBS meningitis, we suggest repeat lumbar puncture (LP) at 24 to 48 hours of therapy to document sterilization of CSF before narrowing antimicrobial therapy. (See "Bacterial meningitis in the neonate: Treatment and outcome", section on 'Repeat lumbar puncture'.)

GBS continues to be susceptible to penicillin G, ampicillin, extended-spectrum penicillins, cephalosporins, and, less so, vancomycin [6,77-80]. Penicillin G is preferred because it is the most narrow-spectrum and active agent in vitro. Approximately 50 to 55 percent of GBS isolates are resistant to erythromycin and 40 to 43 percent to clindamycin, rates that appear to be increasing [6,77,81-83]. Although most isolates show in vitro resistance to gentamicin, gentamicin provides synergy with ampicillin during initial therapy at achievable serum levels [78].

Additional aspects of the management related to the source of infection are discussed separately:

- In neonates with meningitis, neuroimaging studies may be warranted (see "Bacterial meningitis in the neonate: Treatment and outcome", section on 'Neuroimaging')

- Bone and joint infections uncommonly may require surgical drainage in addition to antimicrobial therapy (see "Hematogenous osteomyelitis in children: Management", section on 'Indications for surgery' and "Bacterial arthritis: Treatment and outcome in infants and children", section on 'Drainage')

- In neonates with urinary tract infection, radiographic evaluation of the kidneys and urinary tract may be warranted (see "Urinary tract infections in neonates", section on 'Radiographic evaluation')

Duration — The suggested duration of therapy is as follows (table 4) [1]:

- Bacteremia without a focus – 10 days
- Meningitis – 14 days is sufficient for uncomplicated meningitis; complicated central nervous system (CNS) infections require longer treatment
- Cellulitis-adenitis – 10 to 14 days
- Septic arthritis – 14 to 21 days
- Osteomyelitis – 21 to 28 days
- Urinary tract infection – 10 days

The optimal duration of intravenous (IV) therapy for neonates and young infants with late-onset GBS bacteremia without a focus is uncertain. Randomized controlled clinical trials are lacking. Some experts have suggested that a course shorter than 10 days may be reasonable in carefully selected infants [84,85]. This question was explored in a retrospective study using information abstracted from a large administrative database that included 775 infants diagnosed with uncomplicated late-onset GBS bacteremia [84]. "Uncomplicated" was defined as no concomitant non-GBS infection; no focal GBS infection such as meningitis, osteomyelitis, or septic arthritis; no history of prematurity or low birth weight; no intensive care required; and no prolonged hospitalization required. Rates of GBS disease recurrence were low overall and were similar between infants who received ≤8 days of IV antibiotic therapy and those who received 10 days of IV therapy (1.8 versus 2.3 percent, respectively). Other outcomes were not assessed. These data should be interpreted with caution due to the inherent limitations of an administrative dataset that does not include robust clinical details. Most notably, the investigators could not ascertain whether infants who received a shortened course of IV antibiotics received oral or intramuscular antibiotics in the outpatient setting after discontinuing inpatient IV therapy. In addition, given the low rate of GBS disease recurrence in this study, the sample size may not have been adequate to assure that the outcome was not different between the two groups.

Additional prospective clinical studies are needed to confirm this study's findings before we can endorse the practice of using a shorter duration of IV therapy. Given the established risk of recurrence in neonates and young infants with GBS bacteremia and the potential for serious sequelae (eg, meningitis), we continue to suggest a 10-day course of IV antibiotic therapy for all neonates with GBS bacteremia, including those with an uncomplicated course.

RECURRENT INFECTION

- **Epidemiology and risk factors** – Recurrent GBS infections are infrequent, occurring in 1 to 6 percent of cases [1,15,47,63,86]. Vulnerability to recurrent infection probably is caused by a combination of host and pathogen factors.

  - Prematurity – Prematurity is an important contributing factor, although recurrent infections can also occur in term infants.

  - Persistent mucosal colonization – Recurrent GBS infections usually represent persistent mucosal colonization, but, occasionally, reinfection occurs. This is illustrated by two studies in which GBS isolates from infants with recurrent infection were analyzed with serotyping and pulsed-field gel electrophoresis [86,87]. Paired isolates from 13 of 15 patients were serotypically and genetically identical; two infants had infections with newly acquired, genetically distinct strains of GBS.

  - Immature immune response – Infants with neonatal GBS infection often do not demonstrate a specific antibody response after the infection [47]. However, since only a small proportion of infected infants experience recurrence after completing treatment, there is no role for routinely prolonging antibiotic treatment (beyond the clinically indicated duration) or administering prophylactic antibiotics or intravenous immune globulin (IVIG) to prevent recurrent infection.

  - Breast milk as a potential source of recurrent infection – Cases of recurrent neonatal GBS infection associated with infected breast milk have been reported [88-92]. In each case report, it is unclear whether breast milk caused the recurrent infection or if exposure to the infected infant caused maternal GBS breast duct colonization.

- **Evaluation** – The evaluation of suspected disease recurrence involves the same tests as detailed above. (See 'Tests to perform in the index patient' above.)

  Evaluation for extremely rare causes of recurrent GBS infection (eg, endocarditis or brain abscess) is not routinely necessary unless there are specific clinical concerns for

these entities [86].

- **Treatment** – When treating recurrent GBS infection, susceptibility testing of the GBS isolate to penicillin is recommended. All GBS isolates to date are susceptible in vitro to penicillin, ampicillin, and cephalosporins. We suggest treating recurrent infections for one week longer than the usual recommended duration, though there are few data to support this [93]. (See 'Duration' above.)

  Rifampin, which eliminates colonization in other infections, such as meningococcal disease, does not reliably eradicate mucous membrane colonization with GBS, and it is not recommended for routine use [94].

OUTCOME

Outcomes of GBS infection in neonates and young infants vary depending on the gestational age of the infant, timing of disease onset (early versus late onset), and severity of infection.

- **Mortality** – Among term infants with GBS infection, case fatality rates are approximately 2 to 3 percent for early-onset disease and 1 to 3 percent for late-onset invasive disease [6,15,47]. Mortality is considerably higher among preterm infants (20 to 30 percent for early-onset, and 5 to 8 percent for late-onset) [6,15,47]. Among infants who survive to hospital discharge, the risk of mortality remains elevated throughout the first decade of life. In one study, the risk of mortality was approximately threefold higher in GBS-infected children compared with uninfected children [15].

  Factors associated with increased risk of mortality in early-onset GBS infection include [17,95-97]:

  - Preterm birth
  - Low birth weight (<2500 g)
  - Hypotension and shock
  - Apnea
  - Seizures
  - Neutropenia and/or thrombocytopenia

- **Long-term morbidity** – Infants with GBS infection are at risk for serious long-term sequelae, including cerebral palsy, intellectual disability, seizures, hearing loss, and visual impairment. Survivors of neonatal GBS infection have a higher likelihood of requiring hospitalization during the first five years of life compared with uninfected children [15]. In a population-based study from 2000 to 2011, common reasons for

hospital admission during early childhood among survivors of neonatal GBS disease included infections, respiratory conditions, genitourinary problems, and neurologic disorders (eg, epilepsy and cerebral palsy) [15].

Infants with GBS meningitis are at greatest risk for permanent neurologic sequelae [56,98-103]. In a study of 53 term and near-term (36 weeks gestation) infants with neonatal GBS meningitis occurring from 1998 to 2006, 22 percent of survivors had neurologic impairment at hospital discharge [96]. In multivariate analysis, seizure activity at the time of presentation was a strong independent predictor of poor neurologic outcome. In another study of 43 survivors of GBS meningitis occurring from 1998 to 2006 who underwent follow-up examination at age 3 to 12 years, 56 percent demonstrated age-appropriate development, 19 percent had severe impairment (eg, cognitive skills >2 standard deviations below the mean in at least one domain, severe neurologic or functional impairment), and 25 percent had mild to moderate impairment (eg, cognitive skills within 1 to 2 standard deviations below the mean in any domain, academic underachievement, or evidence of mild neurologic or functional impairment) [104]. (See "Bacterial meningitis in the neonate: Treatment and outcome", section on 'Outcome' and "Bacterial meningitis in the neonate: Neurologic complications".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Sepsis in neonates" and "Society guideline links: Group B streptococcal infection in pregnant women and neonates".)

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

SUMMARY AND RECOMMENDATIONS

- **Pathogenesis and epidemiology** – Group B *Streptococcus* (GBS; or *Streptococcus agalactiae*) infection in neonates and young infants is classified by age at onset into early-onset (<7 days of age); late-onset (7 to 89 days); and late, late-onset infection (≥90 days). (See 'Terminology' above.)
  - Early-onset neonatal GBS infection is acquired in utero or during passage through the vagina. Late-onset GBS infection is acquired vertically at birth or horizontally in household and community settings. (See 'Microbiology and pathogenesis' above.)
  - Universal antenatal screening of pregnant women for GBS colonization and widespread use of intrapartum chemoprophylaxis have resulted in a decline in early-onset GBS disease in the United States but have not reduced late-onset GBS infections (figure 1). (See 'Incidence' above and "Prevention of early-onset group B streptococcal disease in neonates".)
  - Important clinical risk factors for early-onset neonatal GBS infection include (see 'Risk factors' above):
    - Maternal GBS colonization (ie, positive GBS vaginal or rectal screening culture or GBS bacteriuria during pregnancy)
    - Prior delivery of an infant with GBS disease
    - Delivery at <37 weeks of gestation
    - Prolonged rupture of membranes (≥18 hours) before delivery
    - Preterm prelabor rupture of membranes
    - Intraamniotic infection (also called chorioamnionitis)
    - Intrapartum fever (temperature ≥38°C [100.4°F])
- **Clinical manifestations** – The clinical manifestations of GBS disease vary somewhat according to the timing of onset (see 'Clinical manifestations' above):
  - Early-onset – Early-onset GBS infection most commonly manifests as generalized sepsis and, less commonly, as pneumonia or meningitis. Clinical signs usually are

apparent in the first 24 hours after birth. Signs of sepsis are nonspecific and include irritability, lethargy, respiratory symptoms (eg, tachypnea, grunting, hypoxia), temperature instability, poor perfusion, and hypotension (table 1). (See 'Early-onset disease' above.)

- Late-onset – Late-onset GBS disease most often presents as bacteremia without a focus or meningitis. Less common but well-described late-onset GBS focal infections include septic arthritis, osteomyelitis, and cellulitis-adenitis. (See 'Late-onset disease' above.)

- **Evaluation** – Evaluation for clinically suspected GBS infection includes (see 'Evaluation' above and "Clinical features, evaluation, and diagnosis of sepsis in term and late preterm neonates", section on 'Laboratory tests'):

  - Complete blood count (CBC) with differential
  - Blood culture
  - Chest radiograph (if respiratory signs are present)
  - Lumbar puncture (LP) for cerebrospinal fluid (CSF) cell count and differential, protein and glucose concentration, Gram stain and culture
  - Urine culture (if the infant is >6 days of age)

  If osteoarticular infection is suspected, additional evaluation may include radiographs of affected extremities, magnetic resonance imaging, and bone biopsy or joint aspiration (see "Hematogenous osteomyelitis in children: Evaluation and diagnosis", section on 'Diagnostic approach' and "Bacterial arthritis: Clinical features and diagnosis in infants and children", section on 'Evaluation')

- **Diagnosis** – Isolation of GBS from a normally sterile body site confirms the diagnosis of GBS infection. (See 'Diagnosis' above.)

- **Management** – Antimicrobial therapy and supportive care combined with drainage of purulent collections as necessary are the cornerstones of treatment of GBS disease in neonates and young infants. (See 'Management' above.)

  - **Empiric therapy** – Appropriate regimens for initial empiric antibiotic therapy for neonates with suspected bacterial infection are summarized in the table (table 3) and discussed in greater detail separately. (See "Management and outcome of sepsis in term and late preterm neonates", section on 'Initial empiric therapy' and "The febrile infant (29 to 90 days of age): Management".)

  - **Definitive therapy** – Once GBS is identified as the sole causative organism and the patient has improved clinically, we suggest changing the antimicrobial therapy to monotherapy with penicillin G or ampicillin (**Grade 2C**). For infants with GBS

meningitis, a repeat LP should be performed at 24 to 48 hours of therapy to document sterilization of CSF before changing antimicrobial therapy. Duration of therapy depends on the source of infection, as summarized in the table (table 4). (See 'Definitive therapy' above and "Bacterial meningitis in the neonate: Treatment and outcome", section on 'Repeat lumbar puncture'.)

- **Outcome** – Outcomes of neonatal GBS infection vary depending on the gestational age of the infant, timing of disease onset (early versus late onset), and severity of infection. Among term infants with GBS infection, mortality rates are approximately 2 to 3 percent for early-onset infection and 1 to 3 percent for late-onset infection; mortality is considerably higher among preterm infants (20 to 30 percent for early-onset, and 5 to 8 percent for late-onset). Morbidity from GBS meningitis is substantial. (See 'Outcome' above.)

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