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Group B Streptococcus (*Streptococcus agalactiae)*

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Summary

Invasive disease due to group B *Streptococcus* infection (*Streptococcus agalactiae)* results in a wide spectrum of clinical disease. In North America, serotypes Ia, Ib, II, III, and V are most frequently associated with invasive disease. Group B *Streptococcus* remains a continued source of morbidity and mortality in high-risk populations, including pregnant women, neonates, and the elderly; an increasing incidence of invasive disease has been observed in non-pregnant adults. Group B *Streptococcus* remains the most common culture-confirmed neonatal bacterial infection in the United States and is a significant source of neonatal morbidity globally. Intrapartum antibiotic prophylaxis has reduced the incidence of early onset neonatal disease without a notable impact on the incidence of late onset neonatal disease. Penicillin G remains the mainstay of therapy although reduced penicillin susceptibility has been observed in select isolates. Increased frequency of resistance to non-beta-lactam antibiotics, including clindamycin, erythromycin, and fluoroquinolones, has been observed with some isolates demonstrating resistance to vancomycin. The development and implementation of strategies to identify hosts, treat judiciously with antimicrobials with the narrowest spectra, and prevent invasive disease, with vaccines, are essential to reduce the burden of group B *Streptococcus* disease.

Introduction

Streptococcus agalactiae, also known as Group B *Streptococcus* (GBS), was first differentiated from other streptococci by Rebecca Lancefield in the 1930s after it was isolated from milk and cows with bovine mastitis (1). Lancefield described GBS colonization of the vaginal tract of asymptomatic women, however, human pathogenicity was not described until 1938 when three reports of fatal post-partum infection were published (2, 3). Invasive GBS disease was rarely identified in humans until the 1960s, when increasing reports of adult and neonatal invasive infections were published (4–7). The incidence of invasive GBS disease continues to increase and it remains a significant pathogen among both infants and adults.

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Streptococcus agalactiae has historically been divided into nine serotypes (Ia, Ib, II, III, IV, V, VI, VII, VIII) based on the capsular polysaccharide (8). A tenth serotype (IX) was described in 2007 (9). Among non-pregnant adults in the United States (US), the most common serotype causing invasive disease is group V (29% in 2005–2006) followed by serotypes Ia, II, and III (10). A study from Alberta, Canada of serotypes associated with invasive GBS disease from 2003–2013, demonstrated that the most frequent serotype was III (20%) followed closely by serotypes V (19%), Ia (19%), Ib (13%), and II (11%) (11). The dominant serotypes causing disease varies regionally and differs by invasive and colonizing isolates.

Clinical Manifestations

The most common syndromes due to invasive GBS disease in adults are bacteremia without a focus and skin/soft tissue infections (10, 12–16). The former often presents with altered mental status, chills, and fevers (8). Bacteremia may also occur secondary to a focal source of infection; polymicrobial bacteremia, most commonly with *Staphylococcus aureus*, may be seen in 26%−45% of cases (17, 18).

Streptococcus agalactiae bacteremia may lead to seeding of the cardiac valves and endocarditis. Vegetations in GBS endocarditis may become extremely large with a high risk of embolization (19, 20). In the 1930s and 1940s, GBS endocarditis predominantly was associated with acute disease in pregnant and post-partum women, primarily involving the mitral valve (20). Rheumatic heart disease was a common predisposing factor and embolization of vegetations from valves to the kidneys and spleen was common (20). The epidemiology of GBS endocarditis has evolved over time; a case series beginning in the 1960s described endocarditis affecting men and non-pregnant women, involvement of the aortic and tricuspid valves, and subacute presentations (20). The percentage of GBS infections manifesting as endocarditis is in decline, from 9% in one study conducted in Atlanta in 1982–1983 to approximately 3% of GBS infections in the US in a study from 1990–2007 (10, 15). Although cardiac valve abnormalities are a risk factor for GBS endocarditis, cases have been described in structurally normal hearts (21–23). Mortality from GBS endocarditis may be quite high despite medical and surgical interventions with 41% mortality in one case series of 27 patients from 1984–2004 (21).

Skin and soft tissue infections attributed to GBS may manifest as cellulitis, abscesses, foot infection, or decubitus ulcers (24, 25). Diabetes mellitus is a common underlying condition in patients with GBS skin and soft tissue infections (13). Necrotizing fasciitis and pyomyositis attributed to GBS has occasionally been described (18, 25, 26).

Acute and chronic osteomyelitis attributed to GBS has been reported in neonates, children, and adults (27). Infection may arise from direct inoculation from overlying skin/soft tissue infection, skin breakdown such as a decubitus ulcer, or via hematogenous seeding (27). Septic arthritis due to GBS is usually monoarticular although multiple joints may be involved (28, 29). Any joint may be infected but commonly affected joints include the knee, ankle, and shoulder (28, 29). Septic arthritis may involve prosthetic joints with the majority of infections occurring at least 3 months after prosthesis placement; some GBS prosthetic

joint infections require prosthesis removal in addition to antimicrobial therapy for successful treatment (30–32).

Group B *Streptococci*, concurrently with other organisms, have been implicated in pneumonia (33). Imaging findings may include unilateral or bilateral lobar infiltrates (33). Dementia, neurological disease, and tracheoesophageal fistulae are associated with GBS pneumonia, likely due to increased risk of aspiration of the organism (34). Colonization of the airway with GBS is infrequent in patients with cystic fibrosis and when it occurs, does not appear to be associated with worse clinical outcomes (35).

Meningitis is a severe manifestation of invasive GBS disease. It is uncommon in adults but a common manifestation of late onset GBS infection in neonates (36). Infants who survive the acute phase of the GBS meningitis infection may have significant cognitive or neurological sequelae; 32–44% of infants who survived GBS meningitis have neurodevelopmental impairment with severe impairment occurring in up to 19% of surviving infants (37, 38).

Another manifestation of GBS disease includes urinary tract infections. Urinary tract infections may be associated with a variety of GBS serotypes (39). In men, GBS may be associated with prostatitis (34, 40). Group B *Streptococcus* bacteriuria in women during pregnancy is a risk factor for late gestational maternal colonization with GBS and early onset neonatal GBS infection (41, 42).

Pregnant Women

Pregnancy has been associated with a high incidence of invasive GBS disease. In a multi-state evaluation from 2007–2009, the incidence of invasive disease due to GBS was twice as high in pregnant women (0.04/1,000 woman-years) compared to non-pregnant women (0.02/1,000 woman-years) (43). Although the majority of GBS infections are detected during labor and delivery, women in the post-partum period are also at an increased risk for invasive GBS disease even in the absence of other predisposing conditions (43, 44). Rates of maternal invasive GBS disease in the US vary by state, with rates ranging from 0.1 per 1,000 deliveries to 0.8 per 1,000 deliveries (45). Worldwide, the incidence of systemic invasive GBS disease in pregnant women is estimated at 0.38 cases per 1,000 pregnancies with a case fatality rate of 0.2% (44). Serotypes Ia and III account for over half of maternal systemic GBS disease, followed by serotypes V, Ib, and II (44). Systemic maternal GBS disease is associated with increased odds of premature delivery and sepsis in infants (44).

Asymptomatic rectovaginal colonization rates among pregnant women vary widely worldwide [Figure 1] although the majority of estimates fall between 5%−30% (46–97). A recent review estimated that 18% of pregnant women worldwide are colonized with GBS, of which 98% of isolates were serotypes I-V, although there was regional variability in serotype distribution (98). In this study, colonization rates were highest among pregnant Caribbean women at 35% and lowest among pregnant East Asian women at 11% (98). Estimates of GBS colonization in the US range from 10–30% (94–96). Colonization of GBS may be intermittent throughout pregnancy although 17%−28% of women with vaginal or rectal colonization in the first trimester have sustained colonization in the second and third

trimesters or at term (99). Women with GBS colonization during pregnancy were recently found to have a 1.21 risk ratio for preterm birth compared to women without GBS colonization, with a higher risk ratio (1.98) observed in women with GBS bacteriuria (100).

Risk factors associated with maternal GBS intrapartum vaginal or rectal colonization include GBS bacteriuria during the current pregnancy and a history of GBS colonization in a previous pregnancy (41, 101, 102). In one study of pregnant women, no significant difference in the composition of the vaginal and rectal flora between GBS colonized and non-colonized women was observed (99). Studies examining the role of the composition of the vaginal microbiota in relation to colonization with GBS in non-pregnant women have similarly demonstrated no differences in the α-diversity or carriage of *Lactobacillus* between women with and without GBS colonization (103). The role of probiotic administration on GBS carriage remains unclear. One study of *Lactobacillus rhamnosus* and *Lactobacillus reuteri* administered to 110 women with detectable GBS colonization at 35–37 weeks gestation until delivery demonstrated 43% of women in the probiotic group subsequently had negative GBS screens at delivery compared to 18% in the placebo group; however, there were no significant differences in fetal outcome (104). However, in another placebo-controlled study of 34 women who received a 21 day course of *Lactobacillus rhamnosus* and *Lactobacillus fermentum/reuteri* probiotics after a positive GBS screen at 36 weeks, no difference in GBS colonization rates after completion of the supplementation and no difference in infant health was observed between the placebo and probiotic recipients (105).

Non-Pregnant Adults

Although traditionally thought of as a disease afflicting infants and pregnant women, the incidence of GBS disease is increasing among non-pregnant adults, from 3.6 cases/100,000 persons in 1990 to 7.3 cases/100,000 persons in 2007, based on surveillance in 10 US states (10). Comorbidities are present in most adults with invasive GBS disease, including heart disease, neurological disorders, kidney disease, liver disease, alcohol abuse, smoking, obesity, malignancy, and immunocompromising conditions (10, 13, 106). Diabetes mellitus is a common comorbidity, particularly among patients with skin/soft tissue infections (10, 13, 106). Higher incidences and mortality rates from GBS disease have been observed among those of black race compared to white race, although the reason for this difference remains unclear (10, 13, 106).

Older Adults

Higher incidence rates of invasive GBS disease among persons ages 65 or older compared to the general non-pregnant adult population have been described in the US [Figure 2], Canada, the United Kingdom, and multiple European countries (13, 107–111). Annually, invasive GBS affects 25/100,000 adults ages 65 and older in the US (94). The elderly population accounted for approximately 40% of invasive cases reported in the US in 2015 with a higher incidence of deaths (1.08–4.73 deaths/100,000 persons) compared to the incidence of death in the general population (0.54 deaths/100,000 persons) (111). An estimated 12% of elderly long-term care facility residents are colonized with GBS while an estimated 22% of community-dwelling elderly adults in Houston in one study were colonized with GBS, with

47% carrying serotype V (24, 112). Invasive GBS disease in the elderly commonly presents as bacteremia, urinary tract infection, skin infection, and pneumonia (110, 113). Risk factors for GBS disease in the elderly population include being bedridden and residence in a long-term care facility (107, 110).

Neonatal Disease

Group B *Streptococcus* is the leading etiology of culture-confirmed neonatal bacterial infection in the US and resulting in significant mortality (114). Invasive neonatal GBS disease may be divided into early onset disease (EOD), occurring <7 days of life, and late onset disease (LOD) occurring between 7–90 days of life. Serotypes I-V account for 97% of invasive neonatal GBS disease with serotype III accounting for nearly half (43%) of EOD and 73% of LOD (36). Estimates of the incidence of EOD GBS disease range from 0.7 cases/1,000 live births in 1997 to 0.21–0.25 cases/1,000 live births in 2014 and 2015 [Figure 3] (111, 114–133). Disease due to *Streptococcus agalactiae* has been found in 0.58% of neonates with encephalopathy who meet criteria for cooling, a more than 10-fold increase compared to all live term infants. These infants have double the risk of fatality compared to those with neonatal encephalopathy without associated GBS disease (134). Worldwide, estimates of EOD cases per 1,000 live births vary from country to country with incidences of 0.09 in Japan, 0.58 in Panama, 0.76 in Hong Kong, 0 to 1.5 in South Africa, and 2.35 in the Dominican Republic, with a pooled estimate of 0.49 cases per 1,000 live births (36, 135–137). Overall, rates of EOD are highest in Africa and lowest in Asia (36). Worldwide, the pooled incidence of LOD is 0.26 cases per 1,000 live births (36). In the US, the estimated incidence of LOD is 0.32 cases per 1,000 live births [Figure 3] (111, 138).

Risk factors for EOD disease include maternal vaginal or rectal GBS colonization, GBS bacteriuria during pregnancy, prolonged labor, prolonged rupture of membranes, low birth weight, prematurity, intrapartum fever, and systemic maternal GBS disease (42, 44, 139). HIV-exposure does not appear to be a risk factor for EOD (140). The risk factors for the development of LOD are not as well understood as for EOD; known risk factors include prematurity and black race (138, 141). While maternal GBS colonization increases the risk of developing LOD, it does not appear to be as strong of a risk factor as for EOD (138, 141). Infants exposed to HIV have a 4.43 higher likelihood for developing LOD compared to unexposed infants (140).

Early onset disease is attributed to vertical transmission of GBS from the mother either due to ascending infection from the genital tract or infection obtained during passage through the vaginal canal at delivery; symptom onset may be subtle and may be noted within 24 hours after birth (36, 142). Typical manifestations of EOD include bacteremia and pneumonia while meningitis, bone and joint, and soft tissue infections are less common (114). Late onset disease is attributed to horizontal acquisition of GBS. Rarely, LOD has been attributed to GBS transmission from human milk among women with GBS mastitis (143). Meningitis is more common in LOD than EOD although LOD may present as bacteremia, urinary tract infection, bone/joint infection, pneumonia, or soft tissue infections (36, 138, 139).

Neonatal invasive GBS disease overall results in case fatality rates ranging from 1%−8.4% in term infants to 5%−20% in preterm infants (36, 139). For EOD, mortality rates vary worldwide; in resource-endowed countries the estimated case fatality risk is 5% but a much higher risk, up to 27%, has been observed in Africa (36). Mortality from EOD in the US is estimated at 7% with higher mortality in low birth weight and premature infants (114). Worldwide, LOD is estimated to have a case fatality rate of 7% (36). In addition to early and late onset invasive disease, *in utero* GBS disease accounts for approximately 1% of stillbirths worldwide and up to 4% of stillbirths in Africa (144).

Prevention of Neonatal Disease

Due to the burden of GBS disease in neonates, preventative measures have been developed to minimize invasive disease. Maternal antibiotic administration to prevent neonatal GBS disease began in 1973 (145). Intrapartum antibiotic prophylaxis reduces EOD among infants born to GBS colonized women by 86%−89% (42). In countries without a national policy on intrapartum antibiotic prophylaxis, approximately 1.1% of infants born to GBS colonized mothers develop EOD due to GBS, whereas in countries with intrapartum antibiotic prophylaxis recommendations, the risk decreases to 0.03% (146). Penicillin or ampicillin administered at least 4 hours prior to delivery is first-line therapy for intrapartum antibiotic prophylaxis while cefazolin may be used for penicillin-allergic patients without anaphylaxis, angioedema, respiratory distress, or urticaria (42). In patients with severe penicillin allergy, testing of the GBS isolate for clindamycin susceptibility is recommended with use of intrapartum clindamycin for susceptible isolates and vancomycin for resistant isolates (42). Both risk-based and culture-based models for determining when to administer intrapartum antibiotic prophylaxis are currently in use worldwide and vary by geographic region (147). The United Kingdom does not recommend routine GBS screening cultures for pregnant women and uses risk-based algorithms to guide intrapartum antibiotic prophylaxis, whereas the US, Canada, Australia, and New Zealand recommend universal screening with GBS cultures (42, 97, 148, 149). Adherence to intrapartum antibiotic prophylaxis guidelines varies widely; estimates of adherence in countries with universal culture-based screening programs range from 20% to 95% while adherence estimates range from 10% to 50% in countries with risk-based screening programs (147).

Guidelines regarding intrapartum antibiotic prophylaxis for GBS in the US were first issued in 1996 and have undergone several updates, most recently in 2010. The current 2010 guidelines recommend a screening culture from the vagina and rectum of all pregnant women between 35–37 weeks gestation to assess for colonization with GBS and offering intrapartum prophylaxis to all women with positive screening cultures, a history of GBS bacteriuria during the current pregnancy, or a previous infant affected by EOD GBS disease (42). Since the institution of intrapartum antibiotic prophylaxis in the US, the incidence of invasive early onset GBS disease has declined from 1.7 cases/1,000 live births in 1993 to 0.76–0.77 cases/1,000 live birth in 2005–2008 and an estimated 0.21 cases/1,000 live births in 2015 (111, 150, 151). However, while 87% of women in the US undergo cultures to screen for GBS, only an estimated 36% of women in 2016 received care fully compliant to intrapartum antibiotic prophylaxis guidelines; challenges relate to appropriate timing of

screening culture collection, appropriateness of intrapartum antibiotic selection, and appropriateness of intrapartum antibiotic dosages and timing (152).

An alternative strategy, undergoing development, to prevent neonatal and maternal GBS disease is vaccination of mothers in the third trimester against GBS. Lower levels of capsular polysaccharide antibodies in mothers of children with invasive GBS disease compared to mothers of healthy infants have been noted since the 1970s (153, 154). Initial trials of unconjugated GBS polysaccharide vaccines elicited moderate serotype antibody responses (155). More robust antibody responses have been elicited with protein-polysaccharide conjugate vaccines using tetanus toxoid and additional surface protein target vaccines, such as Rib and Alpha C (156–158). Multiple GBS vaccine candidates, including a trivalent protein-polysaccharide conjugate vaccine targeting serotypes Ia, Ib, and III, are currently in development (159). Modeling based on 2015 worldwide disease estimates predict that a GBS vaccine with 80% efficacy and 90% maternal coverage could prevent 107,000 infant deaths and stillbirths (160).

Treatment

Streptococcus agalactiae are uniformly regarded to be susceptible *in vitro* to penicillin, although reduced penicillin susceptibility has been detected in isolates from Japan, thought to be secondary to reduced Penicillin-Binding-Protein (PBP) 2X expression (161, 162). However, penicillin G remains the mainstay of treatment for invasive disease. Generally, GBS is susceptible to other beta-lactam antibiotics, including ampicillin, first-, second-, and third-generation cephalosporins, and carbapenems, although the level of activity varies among different agents. In patients who demonstrate an anaphylactic or severe allergy to beta-lactam antimicrobials, alternative therapies include clindamycin, erythromycin, fluoroquinolones, and vancomycin. However, rising levels of GBS resistance to clindamycin, erythromycin, and fluoroquinolones have been noted in a multitude of studies and these antibiotics should only be used for treatment if a penicillin or cephalosporin is not appropriate and susceptibility of the organism has been determined (162–166). Resistance to vancomycin in clinical isolates has been reported (167).

Penicillin G is the first-line treatment for invasive GBS disease in adults (8). The duration of therapy depends on the clinical presentation. Ten days of therapy is generally acceptable for bacteremia, pneumonia, pyelonephritis, and skin/soft tissue infections. Longer durations of treatment are recommended for meningitis (minimum 14 days), and for osteomyelitis, endocarditis and ventriculitis (minimum 4 weeks) (8). The addition of gentamicin for the initial 2 weeks of therapy is recommended for endocarditis (168).

In neonates with presumptive early onset disease, empiric therapy with ampicillin combined with an aminoglycoside is the standard of care (139). Once GBS is isolated, penicillin G monotherapy is recommended for treatment of invasive GBS infection in infants. Recommended dosing of penicillin G for infants up to 7 days of age is 250,000–450,000 units/kg/day and 450,000–500,000 units/kg/day for infants older than 7 days (139). Ten days of appropriate therapy is recommended for uncomplicated bacteremia and 14 days for uncomplicated meningitis, while complicated infections may require a longer duration of

antimicrobial therapy. Septic arthritis or osteomyelitis is treated for 3–4 weeks and at least 4 weeks of therapy is recommended for endocarditis or ventriculitis (139).

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Figure 1:

Streptococcus agalactiae colonization rates in pregnant women.

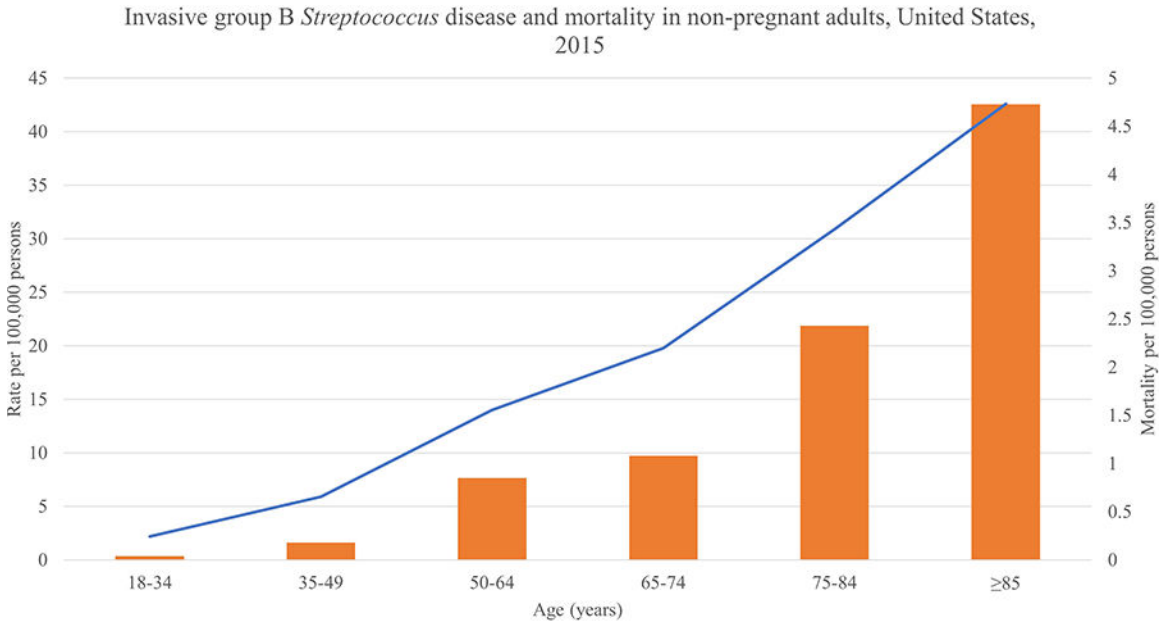

Figure 2:

Invasive group B *Streptococcus* disease and mortality in non-pregnant adults, United States, 2015. Incidence shown in blue; mortality shown in orange.

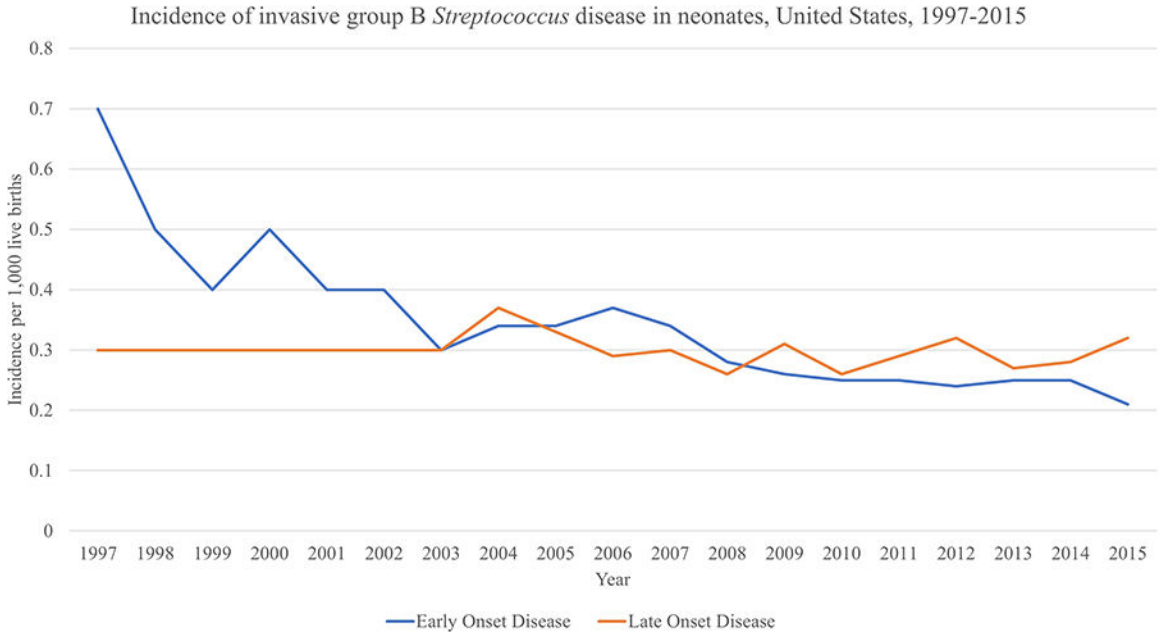

Figure 3:
Incidence of invasive group B *Streptococcus* disease in neonates, United States, 1997–2015.