Neonatal & maternal group B streptococcal infections: A comprehensive review

Anita Shet & Patricia Ferrieri*

Departments of Paediatrics & *Laboratory Medicine & Pathology, University of Minnesota Medical School, Minnesota, USA

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Group B Streptococcus is an important cause of maternal and neonatal morbidity and mortality in many parts of the world. The last two decades have seen intensified efforts in the Western hemisphere in the prevention of this devastating infection by identifying and treating pregnant women who carry group B streptococci or who are at highest risk of transmitting the organism to newborns. The intrapartum use of antibiotics in these women has led unequivocally to a decrease in the rate of neonatal group B streptococcal disease. Although studies in India show a predominance of Gram negative bacterial sepsis among infants, contributing to infant mortality, it is possible that the role of group B Streptococcus has been underestimated. This review discusses its epidemiology in India, and summarizes current concepts of microbiology, pathogenesis, clinical management and preventative issues regarding group B streptococcal disease.

Key words Chemoprophylaxis - colonization - group B Streptococcus - neonatal mortality - perinatal transmission - sepsis

Group B Streptococcus (GBS) is a leading cause of neonatal infection in the Western hemisphere. The recognition that maternal colonization with the organism is a key factor in the occurrence of GBS-associated neonatal morbidity and mortality was a milestone in the history of perinatal health. A nationwide change in health practices helped diminish mortality and morbidity associated with the disease. In India, however, the spectrum of group B streptococcal disease remains a largely under-recognized problem.

Puerperal sepsis has been described for centuries, and ancient Indian texts in 1500 BC have recorded that good hygiene leads to a reduction in perinatal disease. In 1879 Louis Pasteur identified the streptococcus as the causative organism for puerperal sepsis. Since the early 1930s when Rebecca Lancefield reported her grouping system for haemolytic streptococci, group A Streptococcus (Streptococcus pyogenes) was widely acknowledged as the major pathogen associated with puerperal sepsis. GBS was initially thought to be a commensal, until 1937, when Fry reported seven cases of GBS-associated puerperal fever with 3 deaths.

Epidemiology and transmission

During the 1970s and 1980s, GBS emerged as a significant neonatal and maternal pathogen in the United States (US) and Western Europe with reported mortality rates of 15 to 50 per cent. In the US, 10
to 35 per cent of pregnant women are asymptomatic carriers of GBS in the genital and gastrointestinal tract at the time of delivery\(^8\). The prevalence of GBS colonization during pregnancy is variable; in one study, among women who had positive GBS cultures between 26 and 28 wk gestation, only 65 per cent remained colonized at term, while 8 per cent of those with negative prenatal cultures were positive for GBS at term\(^9\). Treatment of these colonized mothers succeeded in temporarily eradicating the organism, but most of the women were re-colonized within 6 wk. At birth, 50 to 65 per cent of infants who are born to colonized mothers have positive GBS cultures from mucus membranes and skin (external ear canal, throat, umbilicus, anorectal sites)\(^1,10\). Approximately 98 per cent of colonized newborns remain healthy, but 1 to 2 per cent develop invasive GBS infection\(^7\). The overall incidence of neonatal GBS infection was approximately 2 per 1000 live births in the United States prior to the introduction of intrapartum prophylaxis\(^7\).

Epidemiological studies in India have shown lower colonization and infection rates in general\(^11-15\). However on closer analysis, taking into consideration use of adequate culture techniques and microbiological media, some of the GBS colonization rates reported from India and other developing countries are similar to those reported in the United States\(^11\). In a study done in 507 pregnant Indian women, 12 per cent were reported to have GBS isolated from the throat and vagina, and 10 per cent had positive vaginal cultures alone\(^12\). Similarly, another study showed the overall carriage rate in pregnant women to be 16 per cent\(^13\). Although both these studies used selective broth media, culture sites did not include the anorectum and this might have lowered the yield of positive cultures. Other studies have reported colonization rates of 5 to 6 per cent, but no selective broth media were used in these cases\(^14,15\). Colonization rates in infants born to asymptomatic maternal carriers of GBS are 53 to 56 per cent and are consistent with rates reported in other parts of the world\(^13,15\). Despite significant GBS colonization rates, reports of invasive neonatal GBS disease in India are infrequent. During a 10-yr study between 1988 and 1997 in Vellore, only 10 cases of neonatal GBS infection were identified, giving an incidence of 0.17 per 1000 live births\(^16\). However, this number represents only the cases occurring among deliveries in a tertiary care hospital located in a predominantly rural community. In India where 65 per cent of women give birth at home, the true incidence of invasive GBS disease in the newborn is largely unknown\(^17\). In addition, blood cultures from ill neonates are not always done in many rural primary health care centres, which may contribute to the underestimation of the number of GBS cases. Preterm births and stillbirths are also usually not investigated, and thus the total burden of perinatal GBS disease remains unrecognized.

The estimated incidence of neonatal GBS infection in India can be calculated from Indian epidemiological data reporting maternal and infant GBS colonization rates as 10 and 50 per cent respectively\(^12,13,15\). Since about 2 per cent of colonized neonates develop true infection\(^6\), the attack rate of neonatal GBS infection in India may be calculated as approximately 1 per 1000 live births. Bearing in mind the above estimated attack rate and current Indian demographic data (midyear population count in the year 2001 was approximately 1027 million, and birth rate was 26 births per 1000 population per year)\(^18\), the projected total number of GBS infection in newborn infants in India may be as high as 26,700 cases per year.

**GBS serotypes and pathogenesis**

Group B streptococci (also called *Streptococcus agalactiae*) are Gram positive cocci belonging to Lancefield group B. There are 9 antigenically distinct serotypes based on their capsular polysaccharide structure (types Ia, Ib, II - VIII) identified to date. In the US and Western Europe, types Ia, II, and III accounted for 85 per cent of the isolates from infants\(^19,20\). Recent studies in the US have demonstrated that serotypes Ia, III, and V (in descending frequency) accounted for 78-87 per cent of early-onset (less than seven days after birth) invasive disease in newborn infants and parturient women\(^21,22\). Late-onset GBS disease in infants 7-90 days of age is dominated by serotype III, followed by
serotypes Ia and V\textsuperscript{22}. Studies from the state of Minnesota from January 1993 through December 1999 reflected the above national trends in the US (Fig.), (Ferrieri P, unpublished data). Studies from India show a variable distribution of serotypes, but the most common isolates belong to types III, II and Ib\textsuperscript{13-15,23}.

The most important risk factor for early-onset GBS infection in the neonate is the presence of the organism in the maternal genitourinary tract at delivery. Ascending bacteria from the maternal genital tract reach the amniotic fluid, usually after rupture of the amniotic membranes\textsuperscript{1,24}. Alternatively, the newborn can come into contact with GBS during passage through the birth canal. When the foetus aspirates contaminated amniotic fluid, group B streptococci reach the lower respiratory tract and damage pulmonary epithelial cells, resulting in pneumonia and respiratory distress usually within the first few hours after birth. Severe GBS sepsis occurs with intravascular invasion of bacteria and failure of the host to eliminate the pathogen\textsuperscript{25,26}. Ascending infection can also occur through intact chorioamniotic membranes, with subsequent events occurring \textit{in utero}, resulting in stillbirths or death within hours after birth\textsuperscript{24}. The pathogenesis in late-onset disease is less clear. Horizontal transmission plays a major role: close contact with the mother, breast-feeding and nosocomial transmission.

The polysaccharide capsule is the most important virulence factor\textsuperscript{26}. However, the role of surface

\begin{figure}[h]
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\includegraphics[width=\textwidth]{gbserotypes.png}
\caption{Distribution of GBS serotypes isolated from patients with invasive disease from January, 1993 through December, 1999. The pie diagrams are for early-onset (left), late-onset (right) and non pregnant adults (center). (Ferrieri P, unpublished data).}
\end{figure}
localized GBS proteins in pathogenesis and protection is under intensive investigation\textsuperscript{26}. The presence of maternal serum antibodies to specific capsular polysaccharides of GBS serotypes appears to be protective against acquisition of neonatal GBS disease as colonized pregnant women with high levels of serum antibodies were less likely to have neonates with invasive GBS infection\textsuperscript{27,28}. Moreover, infected infants had low levels of specific antibody to the infective serotype\textsuperscript{29}. In a recent cross-sectional study, it was shown that pregnant women colonized with GBS serotypes Ia, II, III or V (the most common serotypes) had significantly higher serum concentrations at delivery of IgG specific for the capsular polysaccharide of their colonizing GBS strain than did noncolonized women\textsuperscript{30}.

Clinical features

Neonatal morbidity: Two distinct clinical syndromes are recognized, early-and late-onset disease. Early-onset GBS disease occurs within the first 7 days of life, although most cases are evident in the first 24 h after birth. As can be demonstrated by serotyping GBS isolates from colonized mothers and infants, transmission of early-onset disease is vertical\textsuperscript{9}. Infection may be acquired by the intraamniotic route, or directly during passage through the birth canal. The initial presentation is respiratory distress in more than 80 per cent of neonates. Pneumonia and septicaemia are the most common manifestations, and 5 to 10 per cent neonates will also have meningitis. The incidence of early-onset disease is about 10 times higher in premature than in term neonates\textsuperscript{31}. Late-onset disease develops in infants after 7 days and up to 3 months of age, the median age of onset being 1 month. Transmission can be either horizontal (from other infected infants or health care workers) or vertical (from the mother due to close proximity). These infants almost always have an unremarkable early neonatal history, and later present with meningitis or sepsis. Osteoarticular infections and cellulitis can also occur. The initial signs usually are fever, lethargy, irritability, poor feeding and tachypnoea. Respiratory distress as a presenting feature is less common. Over 20 per cent of survivors of GBS meningitis have permanent neurological sequelae, including sensorineuronal hearing loss, mental retardation, cortical blindness and seizures\textsuperscript{32}. Table I lists the differences between early-onset and late-onset GBS disease. The case fatality ratio in the US has dramatically decreased over the last 3 decades: from up to 50 per cent in the 1970s to 6 per cent in the early 1990s\textsuperscript{33}. The most recent report from the CDC reports a 2002 mortality rate of 0.7 per 100,000 population\textsuperscript{34}. Case series from India also report high mortality: in a 1975 study where 8 cases of neonatal GBS infection were followed over a period of 18 months, 6 infants died within the first week of diagnosis\textsuperscript{35}. A more recent study published in 1999 reported 10 infants with GBS infection, of which 1 died on the second day of life\textsuperscript{16}. However, there is insufficient data presented to calculate the actual mortality rate.

<table>
<thead>
<tr>
<th>Table I. Neonatal manifestations of group B streptococcal disease</th>
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<tr>
<td><strong>Early-onset disease</strong></td>
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<td>Onset</td>
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<td>(usually within the first 24 h)</td>
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<td>Clinical presentation</td>
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<tr>
<td>Incidence of prematurity</td>
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<td>Maternal obstetrical complications</td>
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<td>Transmission</td>
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<td>Predominant serotypes*</td>
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<td>Mortality (%)</td>
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*In descending order of prevalence
**Perinatal morbidity:** Group B streptococcal genital colonization has been considered a possible cause of premature deliveries and premature rupture of membranes\(^36\), although definite evidence of a causal relationship is still lacking. Several prospective studies have also suggested that GBS colonization may play a causal role in the occurrence of intrauterine deaths, late abortions and low birth weight infants\(^{19,37,38}\). In a prospective study of 325 pregnant women in Vellore, 31 per cent of the GBS-colonized mothers reported a history of foetal losses and neonatal deaths as opposed to only 18 per cent of non-colonized mothers\(^{16}\). Among the group of colonized mothers, the birth weights of their infants were lower, the duration of rupture membranes before delivery was longer and the incidence of peripartum fever was more frequent. Although these numbers were too small for statistical significance, there were clear trends of the association between GBS colonization and perinatal morbidity.

**Maternal infections:** GBS can cause significant morbidity in pregnant women. Manifestations of symptomatic maternal infection include chorioamnionitis, endometritis, cystitis, pyelonephritis and febrile GBS bacteraemia\(^{19}\). Caesarian delivery appears to be a prominent risk factor for postpartum endomyometritis\(^{39}\). GBS is also a common cause of fever in postpartum patients. Colonization with GBS was significantly associated with prolonged labour, premature rupture of membranes and preterm delivery\(^{36}\). Less commonly, GBS is isolated in cases of post-operative wound infection, pelvic abscess, septic pelvic thrombophlebitis and osteomyelitis. The current maternal mortality rate in India is 408 per 100,000 live births\(^{17}\). Maternal sepsis is responsible for over 35 per cent of these deaths\(^{40}\). The aetiological agents have been inadequately studied, but group B Streptococcus likely plays a major role in maternal mortality as well as morbidity\(^{41}\).

**Infection in nonpregnant adults:** Invasive group B streptococcal infections cause substantial morbidity and mortality in nonpregnant adults, especially those who are elderly and those who have serious underlying diseases\(^{42-44}\). Skin or soft tissue infections, osteomyelitis, bacteraemia, urosepsis, pneumonia and peritonitis were the most common clinical manifestations. The GBS serotype distribution is quite different in non-pregnant adults, compared to mothers and infants (Fig.). Serotype V accounts for 30-45 per cent of invasive infections in this nonpregnant population\(^{22}\).

**Detection of GBS colonization and diagnosis of infection**

Important factors that influence the accuracy of detecting GBS maternal colonization are the choice of bacteriological media, the body sites sampled, and the timing of the sampling. GBS resides in the genitourinary and gastrointestinal tracts where large numbers of Gram negative bacteria are also present. The use of a selective broth medium that inhibits the growth of Gram negative enteric bacilli and other normal flora can increase culture sensitivity for GBS to over 90 per cent\(^{12,45}\). The most widely used selective medium is Todd-Hewitt broth with gentamicin (8 µg/ml) or colistin (10 µg/ml) and nalidixic acid (15 µg/ml)\(^{46}\). The highest culture yield is obtained when both the lower vaginal area and anorectal sites are sampled\(^{47}\). The optimal time for performing antenatal cultures is between 35 to 37 wks’ gestation\(^{46}\).

Rapid diagnostic tests are based on identification of the GBS group-specific polysaccharide antigen from swab specimens and use latex agglutination or enzyme linked immunosorbent (ELISA) technology\(^{48}\). Although they have good specificities (95%), they tend to have low sensitivities (33 - 65%) which increase only with heavy colonization; hence a negative test cannot rule out GBS colonization. Some investigators have attempted to increase sensitivity by using enrichment media prior to antigen testing\(^{49}\). Recent development of real-time PCR and fluorescence labeling technologies has provided new detection platforms for bacterial identification\(^{50}\), however cost-effectiveness of these new methods is yet to be established.

The diagnosis of invasive GBS infection is established by isolation of the organism from culture of blood, cerebrospinal fluid, amniotic fluid or specific site of infection (i.e., bone or joint fluid)\(^7\). Further identification of GBS may be done by the CAMP test and specific group B antigen detection tests. (i.e., commercial latex agglutination kits)\(^7\). The only limitation of culture as a method is the time (24 to 48 h) required for a result, making it less useful when the patient presents in labour.
Chemoprophylaxis and therapeutic considerations

Prevention is of paramount importance in areas of high incidence of invasive group B streptococcal disease. It is impractical to administer chemoprophylaxis to all parturient mothers and neonates. The challenge therefore is to identify correctly high risk infants before they are born. The most effective way to prevent neonatal early-onset infection is maternal antibiotic administration during labour. However, there is no evidence that chemoprophylaxis prevents late-onset disease. The Centers for Disease Control and Prevention (CDC) consensus recommendations for group B streptococcal prophylaxis were originally put into practice in 1996, and by the end of 1999, the incidence of early-onset GBS disease in the US decreased by a remarkable 70 per cent51. Two preventative approaches were used: a culture screening-based and a risk-based approach. The first approach involved universal screening for GBS colonization of all pregnant women between 35 and 37 wk gestation using vaginal and rectal cultures to detect GBS colonization. As discussed earlier, properly obtained and processed antenatal cultures correctly identified most women colonized at the time of labour. Intrapartum antibiotics are administered to all those with a positive GBS culture regardless of risk factors. The risk-based approach involved administration of antibiotics based solely on the presence of antenatal or intrapartum risk factors. Maternal risk factors for group B streptococcal neonatal sepsis are as follows46: preterm labour or premature rupture of membranes (< 37 wks’ gestation); prolonged rupture of membranes (≥18 h); intrapartum fever ≥ 100.4° F (≥ 38.0° C); history of a previous newborn with GBS disease; and group B Streptococcus bacteruria during pregnancy. Revised guidelines from CDC were published in 200246.

In India, universal culture screening for GBS may be difficult to implement, from a logistic as well as cost-effective viewpoint. However, a strategy based on identifying maternal risk factors could potentially be used, which, according to one source, would require intrapartum antimicrobial prophylaxis in 18 per cent of deliveries and would hypothetically prevent 70 per cent of the cases of early-onset GBS disease52.

Intrapartum antibiotics

Group B Streptococcus remains exquisitely sensitive to penicillin. Penicillin G is preferred because of its narrow spectrum, and is expected to diminish induction of resistant organisms and maternal yeast infections. Clindamycin and erythromycin are acceptable alternatives for women with an allergy to penicillin, although there is increased resistance of GBS to these two antibiotics46. Table II outlines the most recent guidelines for intrapartum chemoprophylaxis, in non-penicillin allergic women and in penicillin-allergic women with a high or low risk for anaphylaxis. These guidelines for maternal prophylaxis will undoubtedly increase the use of antibiotics during labour. There are many concerns about adverse effects of increased use of antibiotics,

| Table II. Regimens for intrapartum chemoprophylaxis of group B streptococcal infection |
|-----------------------------------------------|-----------------------------|-----------------------------|
| No known allergy | Penicillin G: 5 million units as an iv load, then 2.5 million units every 4 h until delivery | Ampicillin: 2 g given in an iv load, then 1 g every 4 h until delivery |
| Penicillin allergy (not high risk for anaphylaxis) | First generation cephalosporin, i.e., cefazolin, 2 g iv initial dose, then 1 g every 8 h until delivery |
| Penicillin allergy (at high risk for anaphylaxis)* | Clindamycin: 900 mg iv every 8 h until delivery | Erythromycin: 500 mg iv every 6 h until delivery |

*If GBS resistance to clindamycin or erythromycin is known or suspected, the recommended treatment is vancomycin 1 g iv every 12 h until delivery. Source - Ref. 7
such as severe anaphylactic reactions or possible emergence of antimicrobial resistance in either group B streptococci or other perinatal pathogens. Although no penicillin-resistant isolates of GBS have been detected so far, resistance to erythromycin and clindamycin has been increasing.53

Clinical management of the infant

A term asymptomatic infant whose mother has received appropriate chemoprophylaxis (delivery four or more hours after intrapartum antibiotics, administered according to recommended dosage intervals) may be observed during the immediate neonatal period. For an asymptomatic infant less than 35 wk gestation, or an infant whose mother has received inadequate chemoprophylaxis, a limited diagnostic evaluation (white cell count with differential, blood culture, and chest radiograph if respiratory abnormalities are present) may be performed, and the infant observed without antimicrobial therapy for at least 48 h. If signs of sepsis develop, a complete diagnostic evaluation (including a lumbar puncture) and empiric antibiotic therapy should be initiated. An alternative approach is to consider those asymptomatic infants born to mothers with chorioamnionitis and/or fever to be at higher risk for infection than those born to asymptomatic mothers with only a positive GBS culture. This approach recommended performing a full diagnostic evaluation and empiric antibiotic initiation on these high-risk infants. Ampicillin plus an aminoglycoside (e.g., gentamicin) is the initial treatment of choice for a newborn infant with presumptive, invasive GBS infection as these agents are also active against other organisms that might cause neonatal sepsis.7,46 Such a combination is also synergistic for killing GBS and may be continued empirically for 48 to 72 h until definite infection with GBS is ruled out. When GBS sepsis with or without meningitis has been firmly established, the combination may be continued for 10 to 14 days, or the course of treatment can be completed with penicillin G or ampicillin alone. The suggested doses and duration of therapy for different infection types are summarized in Table III.

<p>| Table III. Antibiotic therapy for neonatal GBS infection |
|-----------------|-----------------|-----------------|</p>
<table>
<thead>
<tr>
<th>Infection type</th>
<th>Antibiotic</th>
<th>Dose per day (iv)*</th>
<th>Duration</th>
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</thead>
<tbody>
<tr>
<td>Suspected bacteraemia or meningitis</td>
<td>Ampicillin</td>
<td>300 mg/kg in 3-6 divided doses</td>
<td>Until blood and cerebrospinal fluid cultures are sterile (48 to 72 h)</td>
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<tr>
<td></td>
<td>plus Gentamicin</td>
<td>+ 7.5 mg/kg in 3 divided doses</td>
<td></td>
</tr>
<tr>
<td>Bacteraemia without meningitis</td>
<td>Ampicillin or</td>
<td>150-200 mg/kg in 4 div. doses</td>
<td>10 days (range: 10 to 14 days)</td>
</tr>
<tr>
<td></td>
<td>Penicillin G</td>
<td>200,000 units/kg in 4 div. doses</td>
<td></td>
</tr>
<tr>
<td>Meningitis</td>
<td>Ampicillin** or</td>
<td>300 mg/kg in 4-6 divided doses</td>
<td>Minimum 14 days (range: 2 to 3 wk)</td>
</tr>
<tr>
<td></td>
<td>Penicillin G</td>
<td>500,000 units/kg in 4-6 divided doses</td>
<td></td>
</tr>
<tr>
<td></td>
<td>plus Gentamicin</td>
<td>+ 7.5 mg/kg in 3 divided doses</td>
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</tbody>
</table>

* Antibiotic dosages should be adjusted for preterm infants and postnatal age.

**For infants 7 days of age or younger, recommended dosages are: Ampicillin: 200-300 mg/kg/day in 3 divided doses; Gentamicin: 5 mg/kg/day in 2 divided doses; Penicillin G: 250,000-450,000 units/kg/day in 3 divided doses. Source-Ref. 46
The future

While the different strategies for identification of high risk mothers and infants and provision of intrapartum prophylaxis may reduce the rate of neonatal sepsis, they are unlikely to eliminate the problem. Maternal immunization against GBS appears to be a promising and potentially lasting approach for preventing neonatal sepsis, preterm deliveries and low birth weight infants. GBS capsular polysaccharide-protein conjugate vaccines for types Ia, Ib and III have been shown to be safe and efficient in inducing type-specific antibody levels in healthy vaccinated individuals\textsuperscript{56,57}. Epidemiological surveillance of serotype distribution in the population is critical for vaccine studies. A vaccine formulation of GBS types Ia, II, III and V would be expected to provide protection against over 90 per cent of infections\textsuperscript{21}. The cost of developing and implementing a vaccine strategy, although formidable, is considerably less than that of treating these infections, and for some infants, their life-long sequelae\textsuperscript{58}. Among experts in this field, there is considerable discussion regarding whom to immunize - nonpregnant adolescents or pregnant women in the first trimester. In addition, an argument can be made for vaccinating 'at risk' nonpregnant adults. Clinical trials are in progress and research is ongoing for developing a safe and efficacious vaccine against GBS disease.

Conclusion

The infant mortality rate in India has decreased steadily over the last decade and is currently 68 per 1000 live births according to the 2001 census\textsuperscript{18}. This is mainly due to improvements in primary health care, immunization coverage, health education and other factors. Despite the progress, a large proportion of deaths occur in the neonatal period. Sepsis is a major cause of death in up to 30 to 45 per cent of cases\textsuperscript{59}. Microbial colonization of the maternal genital tract is a key factor in most cases of sepsis. Although prevalence studies report a predominance of Gram negative bacilli among female genital colonizers and Gram negative sepsis among their infants, it is possible that the role of group B \textit{Streptococcus} has been underestimated due to inadequate culture techniques and microbiological methods\textsuperscript{11,60}. In addition, it must be recognized that the spectrum of GBS disease in infants is a continuum that includes intrauterine death, preterm delivery, early-onset and late-onset disease. Currently reported low rates of invasive GBS disease in India may be due to one or several factors, such as prevalence of less virulent strains, or high levels of transplacentally acquired protective antibody in serum, and unrecognized causes of early neonatal or premature deaths and stillbirths. Maternal colonization rates are comparable with those in other parts of the world; hence neonatal exposure to the organism is similar. A genetic difference in susceptibility to disease is less likely to be responsible as seen in a study of families of Indian origin living in South Africa where the incidence of neonatal GBS sepsis was high at 2.6 per 1000 live births\textsuperscript{61}. Continued surveillance and more detailed studies are essential in the understanding of the epidemiology and spectrum of disease caused by the group B \textit{Streptococcus}.

The dramatic decrease of the burden of group B streptococcal disease in the United States did not result from a major scientific breakthrough; it resulted from provision of a simple well known antibiotic to a select group of women during labour and to high risk infants. An invaluable lesson that can be learned from the story of the group B \textit{Streptococcus} in the Western hemisphere is that often what is needed to make a major difference in public health is a decision that the problem must be addressed.

References


Reprint requests: Dr Patricia Ferrieri, MMC 134, 420 Delaware Street, S.E., Minneapolis, Minnesota 55455, USA

e-mail: ferri002@umn.edu