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GROUP B STREPTOCOCCAL SEPSIS IN INFANTS

THE NATURAL JOY of some Oregon parents is tempered each year by a sudden interest in learning about group B streptococci (GBS; *Streptococcus agalactiae).* GBS emerged as a leading cause of neonatal sepsis in the 1970's[1,2] following an increased use of antimicrobials and the decline of other perinatal villains such as group A streptococci and *Staphylococcus aureus*. Based on a 1990 multi-site study, 7,600 cases of infant GBS disease (1.8/1000 live births) and 940 deaths were projected to occur in the U.S. annually.[4] In this article we present Oregon GBS data and summarize recommendations for neonatal disease prevention.

GBS are Gram-positive cocci that appear as diplococci or in chains; the human gastrointestinal tract is their primary reservoir. Secondary spread is most commonly from the genitourinary tract. Culture surveys show that 10%-30% of asymptomatic pregnant women may have GBS in the vaginal or rectal area.[4-6] Infant colonization most commonly occurs at parturition.

Most neonatal infections are asymptomatic. For 1%-2% of newborns, however, exposure is the prelude to sepsis—usually manifested as bacteremia, pneumonia, or meningitis. Less common manifestations include cellulitis and septic arthritis. GBS disease is typically classified as early-onset (<7 days of age) or late-onset (>7 days to several months of age). Risk factors for early-onset disease include a heavily colonized or infected mother, premature or prolonged ROM, premature onset of labor, and intrapartum fever. Other maternal risk factors include GBS bacteriuria, a previous newborn with GBS disease, black race, or age <20 years.[7] Late-onset disease may appear in infants colonized at birth or afterwards from nosocomial or community sources.[8] Decreasing case-fatality rates over the past two decades is attributed primarily to prompt recognition and management of early-onset disease. Penicillin G is the treatment of choice, although penicillin-tolerant disease has been reported.[9] Resistance to erythromycin and clindamycin have also been reported.[10]

OREGON SURVEILLANCE DATA

In 1995, OHD's Emerging Infection Program undertook special surveillance for invasive GBS disease in the Portland area. This surveillance is done in collaboration with CDC and health departments in California, Connecticut, Minnesota, Georgia, Maryland, New York, Tennessee, and Toronto, Canada.[11] The Oregon surveillance area, chosen for logistical considerations, comprises Clackamas, Multnomah, and Washington counties (combined 1997 population, 1,341,700). Invasive infections were defined as isolation of GBS from a normally sterile site (blood, pleural fluid, cerebrospinal fluid, peritoneal fluid, pericardial fluid, or joint fluid and specimens collected during sterile procedures) in a resident of the Tri-County area. Microbiologists from area hospitals identify and report cases, and forward isolates to the public health lab. Medical records are reviewed for demographic and clinical information.

In 1996 and 1997, 42 cases of invasive GBS disease in infants were reported from 13 Tri-County hospitals with maternity services—an incidence of 1.1/1000 live births. The median age of these youngsters was 2 days (range, 0 days to 9 months); 22 (52%) were male. Thirty-five cases (83%) had primary bacteremia, 4 (10%) meningitis, 2 (5%) pneumonia and 1 (2%) presented with septic arthritis. For the 37 infants with race identified, 28 (76%) were white, 1 (3%) was black, 1 (3%) was Asian and 7 (19%) were of other races. Twenty-seven (64%) of the 42 cases had early-onset disease. Gestational age at birth was known for 19 cases, and of those, 9 (47%) were born at ≤37 weeks. One infant with early-onset disease had a recurrence at 80 days of age. Patients with late-onset disease were significantly older than cases with early-onset disease at the time of symptom onset (P <0.001). Remarkably, no deaths were identified among any of the 42 cases.

PREVENTION STRATEGIES

Although most experts agree that neonatal GBS disease is largely preventable, a consensus on strategies to address the problem have proven elusive. The American College of Obstetricians and Gynecologists (ACOG) and the American Academy of Pediatrics (AAP) independently published guidelines in 1992.[12,13] ACOG advocated intrapartum antibiotic prophylaxis for all women with certain clinical risk factors, while the AAP focused on prenatal screening (at 26-28 weeks), followed by intrapartum antibiotic prophylaxis for culture-positive women at high risk, viz., those with preterm labor and rupture of membranes (ROM); ROM >18 hours; fever; or a history of GBS disease.

Enthusiasm for these guidelines was underwhelming in a 1993 survey of physicians providing obstetric care, however. Even if screening was performed, specimens were often collected from sites other than those recommended; those who didn't screen cited unclear guidelines and lack of proof of the cost effectiveness of screening.[14] Things got so confusing that in 1994, 19(!) different strategies were compared: those authors concluded that the best options were to: 1) prophylax everyone, 2) prophylax based on risk factors, or 3) prophylax based on cultures obtained at 36 weeks.[15] In 1996, CDC, ACOG, AAP, family practice physicians, nurse midwives, et al. pulled together the latest data and settled on two prevention options.[16]

Plan A ("Screening Approach")

The first option calls for identifying GBS carriers through prenatal screening cultures collected at 35-37 weeks' gestation, and offering intrapartum antibiotic

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prophylaxis to those colonized, to women with premature onset of labor or ROM, fever ≥38˚C, or ROM ≥18 hours before delivery. The specimen site of choice is the vaginal introitus and anorectum—no cervical cultures—and don't use a speculum to get the sample. Transport the swabs in "transport" media. Labs should report screening results to the provider making the prophylaxis decision and to the facility where delivery is anticipated. The timing of cultures should generate fewer false-negative results and simplifies the answer of who gets prophylactic treatment. Also, prophylaxis can be started earlier—before complications such as fever or prolonged ROM develop.

Even if the patient is culture-negative for GBS, prophylaxis is recommended during labor or after ROM if the delivery is preterm or if mom has had a previous child with GBS disease. This approach addresses many of the issues of concern with previous guidelines. Estimates are that this strategy would prevent as many as 65% of early-onset GBS cases and that 11% of moms would receive prophylaxis.[15]

Plan B ("Risk Factor Approach")

What about women without prenatal screening? Intrapartum antibiotics are appropriate for those with risk factors for GBS disease in their infants, e.g., <37 weeks gestation, ROM ≥18 hours, temperature ≥38˚C. Using this approach, up to 50% of cases may be prevented.[15,17]

The first-line prophylactic drug is penicillin G: 5 million U IV loading dose, then 2.5 million U IV every 4 hours until delivery. Alternately, ampicillin can be given in a 2 g IV loading dose, followed by 1 g IV every 4 hours for the same duration. Clindamycin (900 mg IV q 8 hours) or erythromycin (500 mg IV q 6 hours) until delivery can be used for penicillin-allergic women, although efficacy trials are lacking for these. It goes without saying (we said) that women receiving treatment for amnionitis with agents effective against streptococci are already receiving prophylaxis. Routine antibiotics for infants born to these women is not recommended unless, of course, sepsis is suspected.

RECOMMENDATIONS

Obstetrical care should include a strategy for the prevention of GBS disease in infants. Both of the strategies outlined above have been shown to prevent disease and be cost-effective.[15,17-19]

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Influenza: Pulling the Plug

THE ANNUAL INFLUENZA testing program at the Oregon State Public Health Laboratory is hereby suspended until further notice (expected next autumn). We thank the 517 contestants this season; there were 82 lucky winners. All isolates are type A. Only 14 have been subtyped to date, and all so far are H3N2—including the one isolated from an exchange student from Hong Kong who flew in at the height of the H5N1 panic.