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Brief Report

The Prevalence and Adverse Effects of Group B Streptococcal Colonization during Pregnancy

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This study was done to evaluate the prevalence of rectovaginal colonization with group B streptococci among pregnant women who delivered in our center. Also maternal and neonatal complications were compared between colonized and noncolonized groups.

Rectovaginal cultures were obtained from 1197 pregnant women with gestational ages greater than 24 weeks who were admitted to the labor room of Zeinabieh Hospital affiliated to Shiraz University of Medical Sciences from April to September 2003. All of the neonates had surface cultures after birth. The B streptococci carrier and noncarrier groups were compared for maternal and neonatal complications that occurred in the first week after delivery.

Out of the 1197 pregnant women who were evaluated for B streptococci, 110 (9.1%) had rectovaginal colonization (group 1) and 1087 women were not colonized (group 2). Sixty-six neonates had positive B streptococci cultures after birth with a transmission rate of 60%. One neonate developed early-onset B streptococci sepsis. Out of the 110 women who had positive B streptococci culture, 40 (36.3%) developed preterm labor as compared with 155 (14.3%) out of the 1087 women in group 2 (*P*=0.001). The mean gestational age of newborns in group 1 was 32.8 ± 11 weeks compared with 36.2 ± 7.9 weeks for group 2 (*P*=0.001). Eighteen women (16.3%) in group 1 developed preterm rupture of membranes compared with 65 (6.0%) women in group 2 (*P*=0.001). Prolonged rupture of membranes was observed in 6.3% of women with B streptococci carrier states as compared with 0.5% in the second group (*P*=0.001). Intrapartum antibiotics were initiated primarily on the risk based strategy for 34 (30.9%) women in group 1 as compared with 12 (1.1%) in group 2 (*P*=0.001). There was one neonate with early-onset B streptococci sepsis born from a B streptococci carrier mother without any risk factor. Maternal complications were not different between the two groups.

In this study 9.1% of the women had positive rectovaginal B streptococci cultures with a 60% transmission rate to their neonates. Also preterm birth, prolonged rupture of membranes, and preterm premature rupture of membranes had a higher incidence among B streptococci colonized mothers.

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Introduction

Lower gastrointestinal tract is considered as the most likely human reservoir of group B streptococci (GBS), or *Streptococcus agalactiae* with secondary spread to the genitourinary tract. Pregnant women who are GBS carriers have the potential to transmit the organism to their newborn infants. There is a spectrum of maternal and fetal GBS infections ranging from asymptomatic colonization to sepsis. GBS has been implicated in adverse pregnancy outcomes, including premature rupture of membranes (ROM), preterm labor, and clinical and subclinical chorioamnionitis.[1]

Regarding the fact that colonization rates can differ among ethnic groups and geographic locations,[2] this study was designed to evaluate the incidence of rectovaginal colonization of GBS and

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its transmission rate to newborns among pregnant women in Zeinabieh hospital affiliated to Shiraz University of Medical Sciences in Shiraz, south of Iran. Maternal and neonatal complications were compared between the GBS carrier and noncarrier groups.

Materials and Methods

In this study, 2,394 individuals were evaluated for GBS colonization, which included 1,197 pairs of mothers and newborns. Rectovaginal cultures were taken from every singleton pregnant woman with a gestational age greater than 24 weeks who was admitted to the labor room of Zeinabieh Hospital from April to September 2003. Just after the delivery, superficial cultures were taken from the skin, ears, mouth, and throat of every neonate who was born in the study period. The swabs were incubated in Todd-Hewitt broth media and sub-cultured to blood agar plates after 18 hours.[2] Presence of GBS was confirmed by standard microbiological studies using catalase, bacitracin, and sodium hippurate hydrolysis tests.[3] Maternal and neonatal complications were compared between the GBS carrier and noncarrier groups.

The parturients with known GBS colonization received intrapartum antibiotic prophylaxis (intravenous ampicillin two grams initially, then one gram every 4 hours until delivery). For those mothers whose culture results were not ready yet, intrapartum chemoprophylaxis was started on the basis of clinical risk factors including: preterm labor (labor starting earlier than 37 weeks of gestation), prolonged ROM (ROM longer than 18 hours), preterm premature ROM (ROM in a gestational age of less than 37 weeks before labor), having previous infant with GBS disease, GBS bacteriuria, or intrapartum fever ≥38°C.[1]

All of the neonates in this study were observed and evaluated according to the recommendations of Centers for Disease Control and Prevention (CDC).[4] All symptomatic infants had a full diagnostic evaluation, including complete blood count and differential white cell count, blood culture, chest radiography, and lumbar puncture. Then, empiric therapy was initiated with ampicillin and gentamicin according to the standard protocols.[5]

The neonates who had a positive blood or cerebrospinal fluid culture were classified as septic.[6] Each case was reviewed by a neonatologist to confirm the diagnosis.

For statistical analysis, continuous variables were compared by Student *t*-test and categorical variables were compared by Chi-square test and Fisher's exact test. Level of significance was set at $P<0.05$.

Results

Among the 1197 pregnant women who were screened for GBS, 110 (9.1%) women had rectovaginal colonization. The mean age of the women in this study was 24.4±5.4 years ranging from 14 to 45 years. The mean age of GBS carrier women (group 1), was 24.6±5.2 and for GBS non-carriers (group 2), it was 24.3±5.4 years ($P=0.5$). The mean gestational age of neonates at birth, in group 1 was 32.8±11 weeks compared with 36.2±7.9 in group 2 ($P=0.001$). As shown in Table 1 the incidences of preterm labor, prolonged ROM, and preterm premature ROM were significantly higher among the first group (P=0.001).

Sixty-six neonates had positive GBS culture, which showed 60% transmission rate. There was only one out of the 66 neonates with a positive superficial culture, who was born from a culture negative mother (0.09%), while the other neonates were born from mothers in group 1 ($P=0.001$). Forty-six (3.8%) out of the 1197 women received intrapartum chemoprophylaxis. Of them 34 women (30.9%) were from group 1 and 12 (1.1%) from group 2 ($P=0.001$).

Table 1. Comparison of perinatal complications between the GBS carrier (group 1) and non-carrier (group 2) women.

Variable	Group 1 (*n*=110) *n* (%)	Group 2 (*n*=1087) *n* (%)	RR (95% CI)	*P* value
Preterm labor	40 (36.3%)	155 (14.3%)	2.94 (2.05 – 4.2)	0.001
Preterm premature ROM (<37 weeks)	18 (16.3%)	65 (6.0%)	2.63 (1.67 – 4.13)	0.001
Prolonged ROM (>18 hours)	7 (6.3%)	6 (0.5%)	6.19 (3.62 – 10.58)	0.001
Intrapartum prophylactic antibiotics used	34 (30.9%)	12 (1.1%)	11.19 (8.49 – 14.77)	0.001
Cesarean delivery	28 (25.45%)	242 (22.26%)	1.17 (0.76 – 1.76)	0.260

ROM=rupture of membranes

Intrapartum antibiotics were started for six women in group 1 due to positive rectovaginal culture and 40 mothers were treated following the risk-based strategy.

Seventeen patients from group 1 and 169 women from group 2 left the study after delivery. So, 93 women in group 1, 918 women in group 2 and their neonates remained for further evaluations. Early GBS sepsis developed in one neonate (1.07%) in group 1 whose mother had no risk factor and did not receive intrapartum chemoprophylaxis. Early-neonatal sepsis developed in four neonates (0.43%) in group 2. The rate of early-neonatal sepsis was not statistically different between the two groups (P=0.3).

Postpartum fever developed in two (2.15%) and eight (0.87%) of the mothers in group 1 and 2, respectively (P=0.2). No mother with postpartum wound infection was seen in group 1. However, there were seven cases (0.76%) of postpartum wound infection in group 2 (P=0.5).

Discussion

This study showed that 9.1% of pregnant women who delivered in this center had rectovaginal colonization with GBS. In most population based studies, a rate of 10 – 30% has been estimated for GBS carriage during pregnancy.[4] The effect of race and geographical area on the incidence of GBS may be related to the differences in nutrition, socioeconomic status, sexually transmitted diseases, or host immunity.[5]

Transmission of organism to the neonates can occur vertically or, rarely, by hematogenous dissemination. A neonatal transmission rate of 60% was calculated in this study. More than 98% of the cases of early-onset GBS sepsis are the consequence of vertical transmission from the genital tract of the mother to the infant.[7]

With an increase in prevention implementation measures since the mid-1990s, a 70% decline in the rate of early-onset neonatal GBS disease has been achieved. The screening approach is stated to be 50% more effective than the risk based approach,[8] because, about 30 – 50% of the cases with early-onset GBS sepsis develop in infants born to women without risk factors. In this study, there was one neonate who developed early GBS sepsis and was born from a risk-free mother. These data confirm the advantages of universal antenatal culture-based screening.

Microbial pathogens have been recovered from the amniotic cavity in 10 – 15% of the cases with spontaneous preterm labor and in 32 – 35% of women with preterm premature ROM.[9,10] This study showed a strong association between maternal GBS colonization with preterm birth, prolonged ROM, and preterm premature ROM.

Although universal culture-based screening at 35 to 37 weeks of gestation and intrapartum antibiotic prescriptions have decreased the rate of early-neonatal GBS sepsis, we believe that prevention of preterm birth and preterm premature ROM, which are important clinical issues, necessitate the promotion of new strategies for eradication of GBS carrier states earlier during pregnancy. Ideally, establishing immunization strategies against GBS would enable us to prevent the adverse maternal and neonatal effects of GBS colonization and antimicrobial resistance.

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