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EDITORIAL COMMENTARY

Maternal Colonization of Group B Streptococcus and Neonatal Sepsis

Bethou Adhisivam[1]

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Early-onset sepsis (EOS) is apparent within the first 72 h of birth and is usually caused by transmission of pathogens from the mother's genitourinary system to the newborn or fetus. Neonates can also acquire the infection in utero or during delivery as they pass through the vaginal canal. According to the Delhi Neonatal Infection Study (DeNIS), EOS accounted for 83% of the total sepsis, and the most common bacteria isolated included acinetobacter (22%), klebsiella (17%), and *Escherichia coli* (14%) [1]. In the recently completed Global Neonatal Sepsis Observational Study (NeoOBS), among infants with a significant pathogen, 62.9% were gram-negative bacteria. The common microbes isolated were *Klebsiella pneumoniae* and *Acinetobacter spp*., which were resistant to WHO-recommended antibiotic regimens and had carbapenem resistance in 32.6% and 71.4% of cases, respectively. GBS was isolated in only 19 neonates (3.4%) [2]. It would be worthwhile to develop local data on microbiological isolates to devise appropriate empiric antibiotic treatment regimes.

In a South Indian tertiary care hospital, Warrier et al. have conducted an interesting prospective cohort study to estimate the prevalence of maternal GBS colonization and the associated neonatal sepsis. They collected rectovaginal swabs from pregnant mothers (36–38-wk gestation) and rectal and throat swabs from the corresponding newborns at 48 h of age. GBS was isolated using a broth-enrichment step and conventional microbiological techniques. Antibiotic sensitivity patterns of the isolated organisms were also analyzed. Intrapartum antibiotic prophylaxis was administered to all mothers. Neonatal systemic illness was considered in cases of clinical sepsis, culture-positive sepsis, meningitis, pneumonia, or urinary tract infection. Neonates of mothers with GBS colonization were followed up at 3 mo for late-onset sepsis. Among the 310 mothers, 40 had GBS colonization (prevalence: 12.9%; 95% CI - 9.2%, 17.6%). GBS was not isolated from any neonate. As anticipated, maternal GBS colonization was significantly associated with premature rupture of membrane (RR - 2.93, 95% CI - 1.66–5.16) and neonatal systemic illness (RR - 2.78, 95% CI - 1.39–5.54). There was a positive correlation between the duration of intrapartum antibiotic prophylaxis ≤ 4 h and neonatal illness and between maternal GBS colonization and Apgar at 1 min ≤ 4. Twenty percent of the isolates had clindamycin resistance. All neonates remained healthy at 3 mo of follow-up. The authors have alerted regarding the increased maternal GBS colonization and clindamycin resistance, and have stressed the need for GBS screening [3].

It is indeed a good attempt to assess the prevalence of maternal GBS colonization. A total of 2278 mothers delivered during the study period; of which, 2175 deliveries were at ≥ 36 wk gestation. However, rectovaginal swabs were done only in 310 mothers, and 270 (87%) were GBS negative. The prevalence of maternal GBS colonization mentioned as 12.9% is just a percentage of positive isolation among those tested and not the true prevalence. Hence, with this small sample size, it would be inappropriate to conclude high maternal GBS colonization. There is no information provided for the organisms isolated from the GBS-negative mother–baby dyads. There was double the number of symptomatic neonates in the GBS-negative group compared to the GBS-positive group. Hence, focus should be on gram-negative organisms too. As most mothers with GBS colonization are usually asymptomatic, screening is required to identify these women. However, among the mothers in labor who have GBS colonization, only a few are likely to deliver GBS-infected neonates. Hence, administering intravenous antibiotics to all mothers in labor with GBS colonization may cause unnecessary adverse effects in several women and their babies [4, 5]. In the context of the increasing trend of preterm births and the alarming rise of antimicrobial resistance, there is an urgent need to review the existing treatment guidelines for neonatal sepsis and explore new strategies for prevention of infection among neonates.

✉ Bethou Adhisivam
adhisivam1975@yahoo.co.uk

[1] Department of Neonatology, Jawaharlal Institute of Postgraduate Medical Education & Research (JIPMER), Puducherry, India

Declarations

Conflict of Interest None.

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