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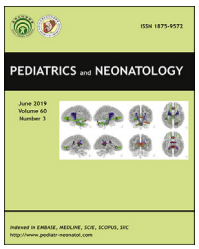

Editorial

Prevention of Group B streptococcus infection

Group B streptococcus (GBS), also known as *Streptococcus agalactiae* (*S. agalactiae*), is a bacterium surrounded by a polysaccharide capsule. Based on the serological reaction against the polysaccharide capsule or as determined by a multiplex polymerase chain reaction assay, GBS strains can be divided into 10 distinct serotypes (Ia, Ib, and II to IX). Multilocus sequence typing is also used to classify GBS strains. More than 750 sequence types (STs) have been identified, and most human isolates are clustered into six major clonal complexes. Some serotypes and/or STs are associated with specific disease phenotypes; for example, some strains are related to neonatal early-onset disease (EOD) and some to late-onset disease (LOD).[1]

Although GBS may be asymptomatically colonized in the gastrointestinal and genitourinary tracts of healthy human adults, it can sometimes cause serious illness especially in newborns, the elderly, and immunocompromized persons. In mothers, GBS can sometimes cause chorioamnionitis and postpartum infections, and maternal urinary tract infections may induce labor and cause premature delivery and miscarriage.

GBS is one of the pathogens causing the highest mortality in neonatal sepsis.[2] Colonization of GBS during labor is the primary risk factor for the development of EOD. Vertical transmission of GBS can occur either *in utero* or during birth through the vagina of a colonized woman. About half of infants born to GBS-colonized mothers are colonized with GBS during birth, and 1–2% of these newborns will develop EOD if proper management is not provided. GBS-LOD is not acquired through vertical transmission during delivery. It is acquired 7 days to 3 months after birth from breast milk or from environmental and community sources. While prevention of GBS-EOD has significantly decreased infection rate, LOD has not benefited much from the prevention.

In Taiwan, the maternal colonization rate of GBS was around 20% from January 2004 to June 2005 based on a study in six hospitals, and the incidence of neonatal GBS infection was one per thousand live births.[3] The data are similar from 0.7‰ to 3.7‰ live births in the United States and from 0.2‰ to 3.25‰ in Europe before the use of universal GBS antenatal screening and intrapartum antibiotic prophylaxis (IAP) was implemented. After the implantation was initiated in 2012, the GBS prevalence in pregnant women remained near 20%, but the rate of GBS-EOD declined from the original 1‰–0.2‰, a decrease of as high as 80%.[4]

Hsu et al.[5] enrolled 500 healthy newborns to detect their GBS carriage rate. The GBS colonization rate of healthy neonates' mothers was 23.7%, similar to the previous decade. While the babies of GBS-negative mothers had a GBS carriage rate of 1.6%, the carriage rate was up to 5% for mothers who were screened positive for GBS and even received IAP. The results point out that the threat of GBS infection still exists. The distribution of GBS serotypes in these healthy newborns was somewhat different from patients with invasive diseases.[6]

IAP is currently the only reliable way to prevent GBS-EOD and has been proven to be effective. However, it is considered to be associated with the emergence of resistant bacterial strains and with an increased incidence of EOD caused by other pathogens, including *Escherichia coli*. Nevertheless, most studies have not found an increased rate of non-GBS early-onset sepsis related to the widespread use of IAP.

Because IAP has no effect in preventing either GBS-LOD in infants or GBS infections in adults, GBS vaccination would be a good way to control not only GBS-EOD and GBS-LOD in infants but also infections in adults at risk. Maternal immunization as an adjunctive strategy to IAP in unimmunized pregnant women would prevent more GBS cases. Transplacental maternal-specific antibodies may be able to provide protection to babies against GBS infection. The capsular polysaccharide of GBS, as an important virulence factor, is also an excellent candidate for the development of an effective vaccine. Choosing the most suitable serotypes for vaccine production is of great importance. At present, there is no licensed GBS vaccine in the market. Despite the promising results from clinical trials, the phenomenon of serotype switching and replacement has been noted worldwide.[7] Ongoing surveillance to monitor GBS serotype distribution will be necessary to guide the development and use of GBS conjugate vaccines.

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Declarations of interest

None.

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