—Case Reports—

Neonatal Case of Late-onset Sepsis Involving Group B *Streptococcus* Type Ib

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Abstract

Group B *Streptococcus* (GBS) is an important pathogen that causes neonatal sepsis and meningitis, which have high mortality and morbidity. Most cases of infection are early onset, with late onset infections being less common. Moreover, many cases of infection are caused by type III GBS, while type Ib GBS infections are rare. We report a case of late-onset infection by type Ib GBS. A female neonate weighing 574 g was delivered at 27 weeks' gestation. An endotracheal tube was inserted shortly after birth because of respiratory distress syndrome, and ampicillin was administered by the age of 3 days. At the age of 54 days after cardiopulmonary adaptation had been achieved, the patient presented with tachycardia following refractory apnea and bradycardia, and her skin became pale. She was suspected of having sepsis, and intensive treatment, including intubation and administration of catecholamines, was started. Despite these measures, the patient died after 5 hours after the onset of sepsis. Type Ib GBS infection may be more frequent in Japanese infants because of the low concentration of IgG antibodies against type Ib in pregnant Japanese women.
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Key words: group B *Streptococcus*, neonate, sepsis

Introduction

Group B *Streptococcus* (GBS) is an important pathogen that causes neonatal sepsis and meningitis, which have high mortality and morbidity. Most cases of GBS infection occur in the first week of life (early-onset disease; age at onset, 0–6 days) and are related to maternal vaginal carriage. Conversely, late-onset infection (age at onset, 7–90 days) is less common[1]. GBS is classified into 9 serotypes (Ia, Ib, and II–VIII) on the basis of specific capsular polysaccharides[2], and type III GBS is the most common in early- and late-onset GBS infection. However, type Ib GBS is less frequently reported, particularly in late-onset disease[1,2]. We report a case of late-onset infection due to type Ib GBS and discuss the prevalence of the GBS serotypes in Japan.

Case Report

A female infant weighing 574 g was delivered by cesarean section at 27 weeks' gestation due to nonreassuring fetal status. The Apgar scores were 6 and 9 at 1 and 5 minutes, respectively. An

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Table 1

Period	Serotype	Total	Ia	Ib	II	III	IV	V	VI	VII	VIII
1999–2005	n	12	1	1	1	2	0	1	4	0	2
	%	100	8.3	8.3	8.3	16.7	0.0	8.3	33.3	0.0	16.7
2006–2009	n	43	7	15	1	6	0	3	7	0	4
	%	100	16.3	34.9	2.3	14.0	0.0	7.0	16.3	0.0	9.3

Serotype distribution of group B streptococcus isolated from colonized strains of Japanese neonates[5]. Type Ib was detected in 8.3% of asymptomatic neonates from 1999 through 2005 but in to 34.9% of neonates from 2006 through 2009.

endotracheal tube was inserted shortly after birth because of respiratory distress syndrome, and surfactant was subsequently administered. Because the results of the maternal vaginal GBS culture were pending, ampicillin was administered until the age of 3 days due to negative C-reactive protein and to negative gastric and blood cultures. At the age of 3 days, the endotracheal tube was removed, and both nasal continuous positive airway pressure therapy and enteral nutrition with the mother's expressed milk were started.

At the age of 54 days, tachycardia suddenly developed following refractory apnea and bradycardia, and the patient's skin became pale. The white blood cell count was 2,700/mm^3, and the platelet count was 125,000/mm^3. The C-reactive protein level in the serum was 0.37 mg/dL. Sepsis was suspected because of the rapid change in symptoms, and the patient required ventilatory support, antibiotic therapy, and administration of dobutamine, dopamine, dexamethasone, and bicarbonate through a central venous line. Despite these measures, the patient died 5 hours after the onset of sepsis.

Blood and cerebrospinal fluid cultures were subsequently found to be positive for type Ib GBS. During the same period no asymptomatic infants in our neonatal intensive care unit were found to be infected with GBS.

Discussion

Infections with GBS are relatively rare in Japan, with an incidence of 0.10 per 1,000 live births for early-onset type and 0 for late-onset type[4], as compared with the United States, with an incidence of 0.40 and 0.30, respectively per 1,000 live births[3]. However, the prognosis of neonatal GBS infection in Japan is poor, with an incidence of 22.6% for death and sequelae in early-onset infection, and 38.7% in late-onset infection[1].

In a nationwide Japanese survey of neonatal GBS infection from 1993 through 1997, type III was the most common, being detected in 38.4% of early-onset cases and 77.6% of late-onset cases[1]. In contrast, type Ib GBS infection was less frequent, with an incidence of 11.2% in early-onset cases and 12.1% in late-onset cases[1].

However, statistics in Japan show that asymptomatic infections by type Ib GBS in pregnant women and infants are becoming more common. Type Ib GBS was detected in 14.0% of pregnant women from 1999 through 2005, but in 19.8% of pregnant from 2006 through 2009. Type Ib GBS was also detected in 8.3% of asymptomatic neonates from 1999 through 2005, but the rate increased to 34.9% from 2006 through 2009 (**Table 1**)[5].

The concentration in pregnant Japanese women of IgG antibody against type Ib GBS is lower than that against other GBS types. In a comparison of concentration vs. prevalence of IgG antibody against GBS types Ia, Ib, II, III and VIII, the percentage of women with antibodies (>1.0 μg/mL) against serotype Ib was only 6.9%, which was significantly lower than the percentages of women with antibodies against serotype Ia (25.0%), III (23.7%), or VIII (58.0%)[6]. High prevalence of antibodies against particular serotypes and excellent placental transfer of antibodies may explain the better protection against neonatal invasive infection by this serotype. Inadequate transplacental antibody transfer and underdeveloped immune systems may further

predispose neonates to late-onset GBS infection. This immunological environment strongly suggests that type Ib GBS infection is increasing in Japan.

Although early-onset GBS infection is typically related to the presence of GBS in the mother's vagina and is the result of vertical transmission during birth, late-onset GBS infection is transmitted both vertically and horizontally from nosocomial and community sources[7]. Kobayashi et al. have found that GBS, particularly type III GBS, remains in the organs[8]. This finding supports the notion that newborns become carriers of GBS after vertical infection, as a result of a poor balance between host and bacteria. Although we could not confirm horizontal transmission in the present case, prophylactic measures for GBS-positive mothers may prevent both early-onset and late-onset GBS infection.

Increased colonization by type Ib GBS may alter the prevalence of this serotype, thereby increasing the prevalence of symptomatic disease caused by this serotype in Japan. We would like to stress that investigating the prevalence of asymptomatic colonization of GBS in Japanese newborns is important for preventing horizontal infection, and these data will lead to useful precautions for late-onset sepsis by type Ib GBS.

Conflict of Interest: None of the authors have any conflicts of interest associated with this paper.

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