Neonatal Group B Streptococcal Infection: A 7-Year Experience

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- **Background:** This retrospective study was designed to determine the trend of neonatal group B streptococcal (GBS) infection during the past 7 years at the Chang Gung Memorial Hospital of Kaohsiung, as well as to assess the risk factors, clinical features and patient outcomes.
- **Methods:** Medical records of infants with neonatal GBS infection identified by positive results of cultures of sterile body fluid in our hospital from January 1996 through December 2002 were reviewed for demographic and clinical data.
- **Results:** There were 33 infants with neonatal GBS infections during the past 7 years in our hospital. The number of patients increased from 1996 to 2001. Sixteen infants had early onset infections and 17 infants had late onset infections. Of the nine patients with maternal risk factors in the early onset group, prolonged rupture of membranes (7, 44%) was most frequently encountered. Distressed respiratory sign (8, 50%) was the most common clinical presentation in early onset group, while fever >38°C (17, 100%) was the most common presentation in late-onset group. The mortality rates were 13% and 6% in early and late onset groups, respectively. Gestational age (p=0.05) and pneumonia (p=0.015) were two most important factors influencing the mortality rate.
- **Conclusions:** The number of GBS-infected infants seemed to have increased during the past 7 years in our hospital. Because the incidence of neonatal GBS infection and maternal colonization in Taiwan has not been collected, we could not determine the necessity of intrapartum chemoprophylaxis. Setting a comprehensive surveillance in Taiwan should be considered. (*Chang Gung Med J 2004;27:501-8*)

Key words: Group B streptococcal infection, intrapartum chemoprophylaxis.

Group B streptococcus (GBS) has been the most common gram positive organism that causes sepsis and meningitis during the first month of life in neonates in the United States since the 1970s. Ten to thirty percent of pregnant women are colonized with GBS in the vagina and rectum,^(1,2) which may cause urinary tract infection, amnionitis, endometritis, sepsis or meningitis.⁽³⁻⁵⁾ Fifty percent of infants with col-

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Received: Nov. 5, 2003; Accepted: May 21, 2004

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onized mothers are asymptomatically colonized at birth. Only 1% of the colonized infants develop invasive diseases, such as septicemia, pneumonia, meningitis, osteomyelitis, septic arthritis, otitis media, and cellulitis. Baker et al. classified the neonatal GBS infection into two types: the early onset type occurs within 7 days after birth and the late onset type occurs 7 days or later after birth. The incidence of early onset GBS infection in the United States decreased from 1993 to 1998 to an incidence of 0.6 per 1000 live births due to the recommendation of intrapartum chemoprophylaxis.^(6,7) Intrapartum chemoprophylaxis has not been performed routinely in Taiwan. This retrospective study was performed to determine the trend and complications of the neonatal group B streptococcal infection during the past 7 years at the Chang Gung Memorial Hospital of Kaohsiung in order to evaluate the necessity of comprehensive surveillance of maternal colonization and neonatal GBS infection in Taiwan.

METHODS

Patients with neonatal GBS infection were identified by positive culture results of sterile body fluid from computerized medical records during the 7-year period from January 1996 through December 2002 at the Chang Gung Memorial Hospital of Kaohsiung. The patients' medical charts were reviewed. The following data were recorded: year; gestational age; birth body weight; gender; age of onset; delivery place; delivery method. The well-known obstetric risk factors including maternal age <18 y/o, maternal urinary tract infection, maternal chorioamnionitis, pre-term delivery, and prolonged rupture of membranes >18 hours were checked. In addition, clinical symptoms and signs including Apgar scores (1 and 5 minute), fever > 38°C, irritability, poor activity, poor feeding, seizure, respiratory signs, shock, persistent pulmonary hypertension of neonate and clinical diagnoses including bacteremia, meningitis, pneumonia, septic arthritis, osteomyelitis, cellulitis, adenitis, and urinary tract infection were used in analysis. Menigitis was defined by positive culture results of cerebral spinal fluid and urinary tract infection was defined by culture of bag urine more than 10⁵ colony counts per cubic centimeter. Laboratory examination results including white blood cell count, thrombocyte count, urine GBS antigen detection and complications including ventriculomegaly, hydrocephalus, subdural effusion, hearing impairment, cortical blindness, neurodevelopmental delay as well as death were also recorded. The number of deliveries every year in our hospital was checked by the records of the obstetric department to count the rate of neonatal early-onset GBS infection in our hospital.

The demographic data, obstetric risk factors, clinical symptoms and signs, clinical manifestations, laboratory test results and complications of cases of early onset GBS infection were compared with those of cases of late onset GBS infection using the chi square test. However, for the tables with more than 20% of expected cell sizes less than 5, the Fisher's exact tests were used instead. P < 0.05 was considered statistically significant difference. The same analyses were performed on the factors influencing the mortality rate.

RESULTS

During the 7-year period from January 1, 1996 through December 31, 2002, 33 cases of neonatal GBS infection were identified at our hospital. Sixteen (48%) cases were early onset type and 17 (52%) cases were late onset type. Eighteen (55%) infants were male and 15 (45%) infants were female. Twenty-eight (85%) were full-term babies and five (15%) were preterm babies. Only one was a preterm infant with gestational age less than 34 weeks. Thirty-one (94%) infants had a birth weight larger than 2500 g and two (6%) infants had a birth weight less than 2500 g. Twenty-four (73%) infants were delivered by spontaneous delivery. The comparisons between genders, birth weights, and delivery modes of patients in the early and late onset groups had no significant differences (Table 1). The number of premature births in early onset group seemed to be more than that in late onset group, but the sample size was too small to show any significant difference. If the sample size had been slightly bigger, the difference may have been significant. The rate of neonatal early onset GBS infection in the neonates born at our hospital ranged from 0 to 1.83 per 1000 live births (Fig. 1).

Changes in number of neonatal GBS infection

From the records at our hospital, the number of

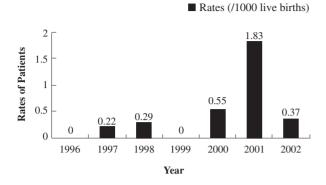


Fig. 1 The proportion of neonatal early onset GBS infection in neonates born at our hospital from 1996 through 2002. The proportion increased from 1996 to 2001 and slightly decreased in 2002.

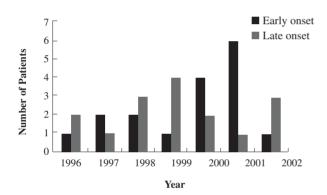


Fig. 2 The number of cases of neonatal early onset GBS infection and late onset GBS infection from 1996 through 2002.

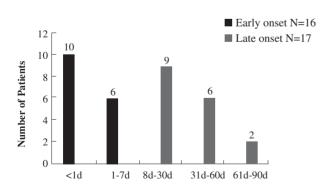


Fig. 3 Number of days to onset in those with neonatal GBS infection.

	Early onset	Late onset	р
	N=16	N=17	
Male	7 (44%)	11 (65%)	0.225
Premature	4 (25%)	1 (6%)	0.175*
Birth weight<2500 gm	1 (6%)	1 (6%)	1*
Spontaneous delivery	13 (81%)	11 (65%)	0.438
Maternal UTI	1 (6%)	0	0.485*
Maternal age <18 y/o	0	0	
Amnionitis	2 (13%)	1 (6%)	0.601*
PROM >18 hr	7 (44%)	1 (6%)	0.017†
Preterm delivery	4 (25%)	1 (6%)	0.175*

Abbreviations: UTI: urinary tract infection; PROM: prolonged rupture of membranes.

*: by Fisher exact test; †: difference was statistically significant

early onset GBS infection increased from 1996 to 2001, then slightly decreased in 2002 (Fig. 2). In addition, the number of late onset GBS infection increased from 1996 to 1999, then decreased in 2000 and 2001 (Fig. 2).

Number of days to onset

The numbers of days to onset were recorded in all 33 cases. Ten (63%) infants in the early onset group were found to have the disease within 24 hours after birth. Nine (50%) infants in the late onset group were found to have the disease within 7 days to 1 month. No infant was found to have the disease after 3 months (Fig. 3). No recurrent infections occurred.

Obstetric risk factors

Table 1 summarizes the obstetric risk factors in the early onset and late onset groups. No mother less than 18 years old was found. In early onset group, prolonged rupture of membranes of 18 hours or more (7, 44%) was the most common risk factor, followed by preterm delivery (4, 25%). Seven (44%) infants in the early onset group did not have any identifiable maternal risk factors.

Clinical symptoms and signs

Table 2 summarizes the clinical symptoms and signs in the early onset and late onset groups. In the early onset group, respiratory signs (8, 50%) was the

most common clinical feature, followed by poor feeding (7, 44%) and poor activity (7, 44%). In the late onset group, fever (17, 100%) was the most common clinical feature, followed by irritability (11, 65%), poor feeding (9, 53%), and poor activity (8, 47%). Fever and irritability in the late onset group were significantly more than those in the early onset group. As the same situation in the premature infants, the numbers of shock and persistent pulmonary hypertension were too small to show any significant difference.

Clinical diagnosis

In the early onset group, sepsis (12, 75%) was the most common clinical diagnosis, followed by urinary tract infection (7, 44%). Pneumonia was found in five (31%) infants and meningitis was found in two (13%) infants. In the late onset group, sepsis (15, 88%) was the most common clinical diagnosis, followed by meningitis (6, 35%). Pneumonia was found in five (29%) infants and urinary tract infection was found in three (18%) infants. Some patients had multiple diagnoses. The distributions of infant numbers in every diagnosis are showed in Figure 4.

Laboratory tests

Table 2 summarizes the laboratory tests in the early onset and late onset groups.

The leukopenia was found in seven (44%) infants in the early onset group. No laboratory tests could indicate the presence of GBS infection except for culture. The urine GBS antigen detection was positive in all infants.

Sequelae and prognosis

Table 3 summarizes the neurologic sequelae and mortality rates in the early onset and late onset groups. Although the mortality rate (13%) was higher in the early onset group, the neurologic sequelae (1, 7%) in the survivors of the early onset group were few. The neurologic sequelae in survivors of all neonatal group B streptococcal infection in our review was only 13%, which was much lower than that reported in previous reports. Because the incidence of sequelae in survivors was low, the comparison between these two groups became difficult. Premature birth (p = 0.05) and pneumonia (p = 0.015) were two most important factors influencing the mortality rate.

Table 2. Clinical Features a	nd Laboratory tests of Early Onset
Group and Late Onset Group	(Chi-square Test)

	Early onset N=16	Late onset N=17	р
Apgar score 1 min < 6	1 (6%)	0	0.485*
Apgar score 5 min < 6	1 (6%)	0	0.485*
Fever $> 38^{\circ}C$	5 (31%)	17 (100%)	< 0.0001†
Irritability	2 (13%)	11 (65%)	0.039†
Poor activity	7 (44%)	8 (47%)	0.849
Poor feeding	7 (44%)	9 (53%)	0.732
Seizure	1 (6%)	2 (12%)	1*
Respiratory distress	8 (50%)	4 (24%)	0.157
Shock	3 (19%)	1 (6%)	0.335*
PPHN	3 (19%)	0	0.103*
Asymptomatic	1 (6%)	0	0.485*
Leukopenia	7 (44%)	5 (29%)	0.391
Thrombocytopenia	3 (19%)	2 (12%)	0.656*

Abbreviations: PPHN : persistent pulmonary hypertension of neonate.

*: by Fisher's exact test; †: difference was statistically significant.

Table 3. Neurologic Sequelae and Mortality of Early Onset

 Group and Late Onset Group (Fisher's Exact Test)

	Early onset N=16	Late onset N=17	р
AHydrocephalus	0	2 (12%)	0.485
Ventriculomegaly	1 (6%)	0	0.485
Subdural effusion	0	3 (18%)	0.227
Hearing impairment	0	1 (6%)	1
Cortical blindness	0	0	
Neurodevelopment delay	1 (6%)	1 (6%)	1
Mortality	2 (13%)	1 (6%)	0.601

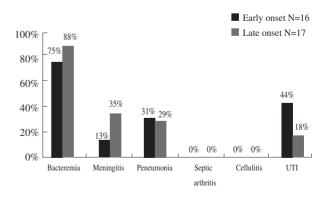


Fig. 4 Clinical diagnoses of the early onset group and late onset group

DISCUSSION

Group B streptococcal infection is an important cause of morbidity and mortality in newborn infants. In 1992, the American Academy of Pediatrics (AAP) published the first preventive measures.⁽⁸⁾ In 1996, the Centers for Disease Control and Prevention (CDC), the American College of Obstetricians and Gynecologists (ACOG), and the American Academy of Pediatrics recommended that obstetric providers adopt either a culture-based or a risk-based approach for the prevention of early-onset GBS infection.⁽⁹⁾ In the United States, after the introduction of preventive measures, the incidence of culture-confirmed cases of neonatal early onset GBS disease decreased from 1.7 per 1000 live births in 1993 to 0.6 per 1000 live births in 1998.^(6,7) The incidence of early onset GBS infection decreased during those years in Japan⁽¹⁰⁾ and European countries⁽¹¹⁻¹⁵⁾ because some preventive measures were taken even if they differed from CDC-recommended preventive measures. In Some European countries, the incidence of culture-confirmed cases of early onset GBS disease varied from 0.5 to 1.15 per 1000 live births.⁽¹¹⁻¹⁵⁾ In Taiwan, there was no formal intrapartum chemoprophylaxis although some preventive measures were taken at some institutions. At our hospital, preventive measures were not routinely used. The rate of early onset GBS infection in the neonates born at our hospital increased from 0 per 1000 live births in 1996 to 1.8 per 1000 live births in 2001. Since the increase in cases of early onset GBS infection in 2001, the obstetricians and neonatologists in our hospital have improved perinatal care including encouraging mothers to receive GBS screen and aggressive intrapartum chemoprophylaxis for those with maternal colonization with GBS and mothers with preterm labor who were suggested to be infected. These strategies led to a decrease in the rate of early onset GBS infection in 2002. No published data were available on the incidence of maternal colonization with GBS and neonatal GBS infection in Taiwan although some data were available from some institutions.⁽¹⁶⁻¹⁹⁾ Ho et al.⁽¹⁶⁾ reported that 66 infants with early onset GBS infection and 23 infants with late onset GBS infection were treated at Mackay Hospital from 1985 through 1995. The occurrence rate of early onset disease was 3.26-10.08/1000 admissions. The incidence of the disease was 0.11-1.39/1000 live births. Of the 66

infants with early onset disease, 24 (36%) were preterm infants. The mortality rate was 14% in the early onset group and 4% in the late onset group. The incidence and mortality rate were similar to our data. In comparison with the data from the United States, European countries, and Japan, the rates of early onset GBS infection in our hospitals were higher these years and in accordance with the reported incidence in the United States before the introduction of intrapartum chemoprophylaxis. Furthermore, the rates of early onset GBS infection in our hospital could have been underestimated because of the number of unrecognized cases that were treated by intrapartum antibiotics for other reasons. The growth of GBS in culture would be more difficult in patients under intrapartum antibiotic treatment. Otherwise, some inborn babies with GBS infection might present with symptoms after discharge and be admitted to other hospitals for treatment. Therefore, comprehensive surveillance of maternal colonization with GBS and neonatal GBS infection in Taiwan to access the necessity of intrapartum chemoprophylaxis may be necessary.

In our study, four (25%) infants in the early onset group were premature babies. Liao et al.⁽¹⁷⁾ reported that nine infants with early onset GBS infection and 16 infants with late onset GBS infection were treated at the National Taiwan University Hospital from January 1980 through March 2000. Eight of the nine (89%) infants with early onset disease manifested symptoms during the first day of life and three (33%) were premature births. Yang et al.⁽¹⁸⁾ reported that three infants with early onset GBS infection and 30 infants with late onset GBS infection were treated at the National Cheng Kung University Hospital from January 1988 through December 1996. All of the infants with early onset disease were detected during the first day of life and two of them were premature births. As mentioned in the above two studies, premature babies face high risk of GBS infection. According to the culturebased preventive measure recommended by the CDC, premature babies may be born without the mothers' culture data. Mothers with preterm labor should receive vaginal culture and intrapartum chemoprophylaxis to protect their babies from GBS infection.

In accordance with some previous reports,^(15,20-22) over 40% of the neonates who developed early onset

GBS infection in our hospital did not have any identifiable maternal risk factor. This made us consider the safety of the risk-based approach. Some previous reports in the literature (14,23) also suggested that the culture-based strategy was better than the risk-based strategy. Fortunately, the majority of these infants without maternal risk factors were observed in a hospital and received antibiotic treatment for other causes. On the other hand, the majority of early onset GBS diseases in our hospital occurred within the first 72 hours. According to the studies by Liao et al.⁽¹⁷⁾ and Yang et al.⁽¹⁸⁾, 89 to 100% of infants with early onset GBS infection were detected during the first 24 hours of life. During the first 72 hours, newborn infants still admitted in the baby room and were observed by physicians and nurses. The symptoms and signs of infection would be easily recognized. No early onset cases without maternal risk factors died or had neurologic sequelae after treatment in our hospital. This indicates that the risk-based approach and discharge plan of newborn after 3 days of observation in the baby room in Taiwan may be sufficient to avoid missing early onset cases and delaying treatment. The majority of early onset cases occurred within the first 24 hours, in whom the infection might be acquired by vertical transmission. It also highlights the importance of intrapartum chemoprophylaxis in this group of early onset cases.

Among the maternal risk factors, prolonged rupture of membranes and preterm delivery are the most frequent and can be used as indicators for risk of early onset GBS infection. Among the clinical features, the most common symptom in the early onset cases was respiratory sign, followed by poor activity and poor feeding. All late onset cases had fever. The rates of fever and irritability were significantly higher in the late onset group. All of the symptoms and signs in neonatal GBS infection were nonspecific. One (6%) of the infants with early onset infection was asymptomatic. In addition, no laboratory tests are available that indicate the presence of GBS infection except for culture. We suggest that no specific symptoms can indicate the presence of neonatal GBS infection and it is important to keep greater awareness as well as take blood culture routinely when caring for neonates who are not doing well.

Neonatal GBS infection may induce severe diseases such as sepsis, pneumonia, and meningitis. The sepsis was the most common manifestation in both early and late onset groups. According to previous reports in the literature, sepsis, pneumonia, and meningitis were the most common three manifestations of the early onset disease, but in our hospital, urinary tract infection was the second most common diagnosis in the early onset group. This phenomenon seems to be associated with routine urine analysis and culture in admitted neonates in our hospital. In late onset GBS infection, meningitis was commonly found (35%), but seizure was observed in two cases. It might be due to the difficulty of finding subtle seizure in neonates. Neurologic sequelae such as subdural effusion, hydrocephalus, hearing impairment, and neurodevelopment delay were found in some infants with meningitis.

Three patients died during our study. Two were in the early onset group and one was in the late onset group. The two deaths in the early onset group were both premature babies with maternal risk factors such as chorioamnionitis and prolonged rupture of membranes. One premature infant with a gestational age less than 34 weeks died due to perinatal asphyxia complicated with persistent pulmonary hypertension. All of the infants that died had sepsis, shock, and respiratory distress. Prematurity and pneumonia were the two most important factors influencing the mortality rate in our study. There were four survivors with neurologic morbidity. One had early onset infection and three had late onset infection. The infant with morbidity in the early onset group was a full-term infant with prolonged rupture of membrane. Pneumonia and sepsis complicated with persistent pulmonary hypertension occurred. Ventriculomegaly was found by brain ultrasonography and neurodevelopment delay was noted during a follow-up examination. All three infants with morbidity in the late onset group had sepsis, meningitis. The morbidity rate in survivors was 13%, which was far lower than that reported in the literature.

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高雄長庚醫院新生兒B型鏈球菌感染的回溯分析

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- **背 景**: 本文回溯分析過去7年來高雄長庚醫院新生兒B型鏈球菌感染的趨勢、危險因子、臨 床表現及預後。
- **方法**: 先以培養報告找出從1996年1月到2002年12月被B型鏈球菌感染的嬰兒,並且回顧 分析他們的病歷資料。
- 結果:在過去7年中本院共有33名被B型鏈球菌感染的嬰兒。病例數從1996年到2001年有 逐漸攀升的趨勢。16名為早發型,17名為晚發型。9名有產科危險因子的早發型病嬰 當中,破水超過18小時者占最多。呼吸症狀是早發型最常見的臨床表現,而發燒則 是晚發型最常見的臨床表現。死亡率於早發型為13%,於晚發型為6%。早產和肺炎 是造成死亡的重要因子。
- 結論: B型鏈球菌感染的新生兒近年來似乎有增加的趨勢。因為台灣沒有產婦帶原及新生兒 感染的全面報告,是以無法評估產程抗生素預防的必要性。我們或許該慎重考慮施 行產婦帶原及新生兒感染的全國性研究。 (長庚醫誌 2004;27:501-8)

關鍵字:B型鏈球菌感染,產程預防性抗生素。