

Neonatal Group B Streptococcal Infection : Incidence and Clinical Manifestation in Siriraj Hospital

PANITAN YOSSUCK, M.D.*,
KANCHANA PREEDISRIPIPAT, M.D.*

Abstract

From 1996 to 2001, nineteen episodes of bacteremia due to group B Streptococci (GBS) were diagnosed in Siriraj Hospital, Mahidol University. The incidence of early onset group B streptococcal disease (EOD) was 0.27 cases/1,000 live births in 1996, and decreased to 0.10 cases/1,000 live births in 2001. The incidence of the late onset disease (LOD) was 0.05 cases/1,000 in 1996, and there has been none since 1998. All of the infants were inborn. Low birth weight was found in 53 per cent of the infants. Fifty-eight per cent of infants were male. Forty-seven per cent of the infants were born prematurely. None of the mothers had antenatal GBS screening. Only one mother received one dose of intrapartum antibiotic prophylaxis. No risk factor could be identified in 72 per cent of the mothers. EOD accounted for 79 per cent of all infants with GBS infections, with a mortality rate of 40 per cent. All of them died within the first 72 hours of life. Most EOD infants developed disease manifestations within 12 hours of life. Most common clinical manifestations were respiratory distress (74%), temperature instability (68%), cyanosis (63%), hypotension (42%) and lethargy (42%). Only one infant with EOD had meningitis. There were two infants in the LOD group; one of whom had cellulitis, and the other had meningitis. Neutropenia was noted in 42 per cent of all infants. Radiographic studies suggested a diffuse reticulogranular pattern or ground glass appearance in 38 per cent. The chest X-ray was interpreted as normal in 25 per cent of the infants. In conclusion, the incidence of GBS infection in newborn infants in Thailand is still very low but with a very high mortality. Prematurity accounts for almost half of the cases. Even though antepartum screening with intrapartum antibiotic chemoprophylaxis has been recommended in developed countries, its benefit and cost needs to be further investigated in Thailand.

Key word : GBS, Group B Streptococci, Sepsis, Incidence

YOSSUCK P & PREEDISRIPIPAT K
J Med Assoc Thai 2002; 85 (Suppl 2): S479-S487

* Division of Neonatology, Department of Pediatrics, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok 10700, Thailand.

Group B Streptococcal (GBS, *Streptococcal agalactiae*) infection was first recognized as a cause of bovine mastitis. It was in the 1960's that GBS came to the attention of clinical medicine^(1,2). Since then, it has been the predominant cause of early onset neonatal infection in almost all developed countries^(3,4). GBS colonization has been identified in 2.8 per cent to 31 per cent of pregnant women⁽⁵⁾, depending on the site(s) and microbiological techniques used by various investigators. The colonization in an individual mother can also vary from trimester to trimester with the results obtained at 35 to 37 weeks' gestation having the best correlation with culture status at birth⁽⁶⁾. Recent reports have indicated that GBS colonization rates in pregnant women have remained constant (25%-30%) in developed countries. Transmission rates were previously reported to be between 29 per cent to 72 per cent (mean 51%). Approximately 1 per cent to 2 per cent of infants born to colonized mothers developed early-onset invasive disease⁽⁷⁾. In recent years, the mortality rate ranged from 4 per cent to 6 per cent of cases. High mortality rate, was observed in premature infants⁽⁸⁾. Major risks factors for early-onset neonatal GBS disease include maternal GBS colonization, longer duration of membrane rupture, intrapartum fever, less than 37 weeks of gestation, GBS bacteriuria during pregnancy, and previous delivery of an infant who had GBS disease⁽⁹⁾.

Several strategies and trials have been designed to reduce the neonatal colonization and early-onset invasive disease. In early 1996, the Center for Disease Control (CDC), with the support of the American Academy of Pediatrics (AAP) and the American Academy of Obstetrics and Gynecology, proposed the intrapartum chemoprophylaxis guidelines for GBS infection⁽⁹⁾. The risk factors approach would prevent 60 per cent to 70 per cent of early-onset GBS cases with treatment given to up to 20 per cent of all mothers in the United States. The combined risk factor-screening approach would reduce the amount of neonatal GBS disease by over 85 per cent while treating only 25 per cent to 30 per cent of mothers.

There have been limited data regarding neonatal GBS disease in Thailand even though neonatal sepsis is the major cause of infant mortality in this country. The incidence of neonatal sepsis was reported to be 2.57/1,000 live births in one university hospital in the early 1980's⁽¹⁰⁾. No case was identified to be causes by GBS infection from

that study. Whether GBS is the major cause of neonatal sepsis in Thailand is unclear. The authors studied the incidence of invasive GBS infection in newborn infants in Siriraj Hospital, Mahidol University, one of the largest teaching hospitals in Bangkok, Thailand during a six year period. The clinical manifestations, laboratory and radiographic findings are also described.

METHOD

Retrospectively documented GBS cases were reviewed from January 1996 to December 2001. Cases were defined by positive blood or CSF culture for GBS infection in infants under three months of age. Maternal charts reviewed for obstetrics data included: gravidity, parity, number of fetuses, number of prenatal visits, history of previous deliveries of siblings with invasive GBS disease, GBS colonization, history of diabetes and maternal GBS bacteriuria. Intrapartum information was collected which included: presence of chorioamnionitis, maternal fever, antenatal corticosteroid therapy, duration of rupture of membrane, mode of delivery, and use of intrapartum antibiotic chemoprophylaxis. Chorioamnionitis was diagnosed when one or more of the following data presented: maternal fever ($> 38^{\circ}\text{C}$), positive amniotic fluid culture, uterine tenderness, foul-smelling/purulent amniotic fluid.

Data from the infant's chart include: gestational age, birth weight, sex, delivery room resuscitation, Apgar scores, clinical manifestations, onset of disease, ventilator support, length of stay, and survival. Also the laboratory and radiographic data were reviewed. Early onset disease (EOD) defined as the onset of symptoms occurring in an infant younger than 7 days of age and/or positive sterile body fluid or exudate culture obtained before the 7th day of life. Late onset disease (LOD) defined when the onset of disease occurred in infants between seven days to three months old and/or positive sterile body fluid or exudate culture. Asymptomatic bacteremia was defined as a blood culture positive for GBS without symptoms and signs of sepsis.

RESULTS

Maternal data

There were 18 mothers who delivered infants with GBS infections. One mother had triplets with two babies infected. The median age of these mothers was 27.5 years (19-44), with a median

gravidity of 2 (1-5) and parity of 1.5 (0-4). Thirteen women (76%) had antenatal care with the median number visits 6 times during pregnancy. Spontaneous vaginal deliveries occurred in 78 per cent, leaving 22 per cent for cesarean sections. Prolonged rupture of membranes, more than 18 hours, was found in only 22 per cent of the mothers. Two mothers were diagnosed as having chorioamnionitis, one of these also had GBS bacteriuria during her pregnancy. None of the mothers had GBS screening antenatally and only one mother received one dose of ampicillin three hours prior to delivery.

Corticosteroid was prescribed in three of the mothers who had preterm delivery. No postpartum complication was noted. Maternal information is summarized in Table 1.

Infant data

During the study period, there were 19 documented GBS infections; all were inborn. There were 18,761 live births in 1996 with 6 invasive GBS disease in that year, so the incidence of neonatal GBS disease in 1996 was 0.32 cases/1000 live births. Since 1998 the delivery rate has decreased to 11,000

Table 1. Maternal data (n = 18).

	%	
Age (years) (median)	27.5 (19 - 44) range	
Age < 20 year	1	5.56
Gravidity (median)	2 (1 - 5) range	
Parity (median)	1.5 (0 - 4) range	
Multiple pregnancy	2	
twin	1	5.56
triplet	1	5.56
Antenatal care (ANC)	13	72.22
No antenatal care	3	16.67
Number of ANC (median)	6 (1 - 8) range	
Maternal Diabetes	1	5.56
Mode of delivery		
- Spontaneous vaginal delivery	15	77.78
normal labour	12	66.67
breech assisting	2	11.11
BBA 1 (NL)		
- Assisted vaginal delivery (Forups/vacuum)	0	0
- Caesarean section	4	22.22
Chorioamnionitis	2	11.11
Maternal fever	2	11.11
Temperature > 38 °C	2	11.11
Leukoerytosis (wbc > 15,000)	2	11.11
Duration ruptured membranes	Artificial ruptured membranes	Spontaneous ruptured membranes
	%	%
< 12 hours	4	22.22
12 to < 18 hours	1	5.56
≥ 18 hours	0	0
Previous delivery of sibling with invasive GBS disease	0	0
Maternal GBS colonization	0	0 (not done)
Maternal GBS urinary tract infection	1	5.56
Intrapartum antibiotic chemoprophylaxis	1	5.56
Received antibiotics > 4 h before delivery	0	0
Antenatal corticosteroid	3	16.67
Maternal postpartum complication	0	0

deliveries a year. The incidence of neonatal GBS was, therefore, decreased to 0.10 cases/1,000 live births in the year 2001.

Of all nineteen cases, 9 infants (47%) were premature. Ten of nineteen (53%) were low birth weight infants. Median birth weight was 2,180 grams (1,080-3,940). Male sex accounted for 58 per cent of the infants. Delivery room resuscitation was performed in 3 infants. Median Apgar score at 1 and 5 minutes was 9 and 10 respectively. Only 2 infants had Apgar scores less than 6 at 5 minutes.

Fifteen infants (79%) were categorized as having EOD, with two infants (10.5%) having LOD. Two infants (10.5%) had asymptomatic GBS disease. Respiratory distress was the most common manifestation, found in 74 per cent of the infants with the median onset of 1.5 hours. Other common manifestations included cyanosis (63%), temperature instability (68%), lethargy (42%) and hypotension (42%) (Table 3). Most of the clinical manifestations in EOD occurred within the first 12 hours of life. Meningitis was found in one case of EOD and one case of LOD. Cellulitis was noted in one LOD case who was one of the triplets and presented at 37 days of age. Two of 19 were asymptomatic, whose

blood culture was sent before partial exchange transfusion from one infant and because of maternal fever from the other. Both patients did not receive antibiotics and were sent home after one week of observation.

Thirteen infants (68%) survived. All of those who died had EOD, with a median age of 16 h. All died within 72 h. Four of the six infants (67%) who died were premature, making the mortality rate in premature infants 44 per cent (4 out of 9 infants).

Laboratory data

The median total white count was 10,080 cell/mm³. Six of 19 infants (32%) had an initial white blood cell count less than 5,000 cell/mm³. One infant presented with an initial white blood cell count more than 30,000 cell/mm³. Forty two per cent of infants had absolute neutrophil count less than 1,750 cell/mm³. Thrombocytopenia was noticed in only one infant. (Table 4)

There were 6 infants (32%) whose initial blood gas showed pH less than 7.25. Nine of the infants had evidence of metabolic acidosis from the blood gas.

Table 2. Infant data (n = 19).

		%
Gestational age (weeks) median	37 (range 27 - 43)	
Male	11	58
Female	8	42
Premature	9	47.37
Birth weight (g) median	2,180 (range 1,080 - 3,940)	
1,000 - 1,500 (g)	5	26.32
1,501 - 2,000 (g)	3	15.79
2,001 - 2,500 (g)	2	10.53
> 2,500 g	9	47.37
low birth weight	10	52.63
Apgar 1' (median)	9 (range 0 - 10)	
Apgar 5' (median)	10 (range 3 - 10)	
Apgar score < 6 at 5 minutes	2	11.11
Birth before arrival	1	5.26
AGA	15	78.95
SGA	3	15.79
LGA	1	5.26
Delivery room resuscitation	3	16.67

AGA = appropriate for gestational age

SGA = small for gestational age

LGA = large for gestational age

Table 3. Clinical manifestation.

		%
Hypoglycemia	4	21.05
Hyperglycemia	1	5.26
Fever T > 37.5 °C	8	42.10
Hypothermia T < 36.5 °C	5	26.32
Hypotension	8	42.10 onset median 9.5 hours (range 0 - 22 hour)
Poor perfusion	3	15.79
Cyanosis	12	63.16
Respiratory distress	14	73.68 onset median 1.5 hours (range 0 - 40 hour)
Lethargy	8	42.10
Apnea	7	36.84 onset median 7 hours (range 3 - 432 hour)
Tachycardia (> 160)	3	15.79
Persistent pulmonary hypertension of the newborn (PPHN)	4	21.05
Seizure	1	5.26
Cellulitis	1	5.26
Meningitis	2	10.53
EOD (1 wk)	15	78.94
LOD (1 wk - 3 m)	2	10.53
Asymptomatic	2	10.53

Chest radiographic finding

Radiographic features were characterized by diffuse reticulogranular or "ground glass" haziness in 37.5 per cent, transient tachypnea of the newborn (TTNB) 6.25 per cent, localized infiltrate (pneumonia) 12.5 per cent. Twenty-five per cent of the chest X-rays were reported to be normal. Chest X-rays were not done in three infants who had respiratory symptoms. Of all the infants who died, 50 per cent presented with chest radiographs suggesting diffuse reticulogranular (ground glass) pattern.

DISCUSSION

Group B Streptococcal (GBS) continues to cause neonatal disease ranging in severity from asymptomatic bacteremia to fulminating, fatal infection. It is the most important bacterial pathogen associated with early-onset neonatal sepsis in several developed countries⁽³⁾. It does not, however, seem to be responsible for more than 2 per cent of diagnosed neonatal sepsis in Africa, India, the Middle East, or South East Asia⁽³⁾. In the United States, although the mortality rate has decreased during the last decade, recent studies showed that fatal outcome occurs in 4 per cent to 6 per cent of cases (8,11). The mortality rate is much higher in premature infants, approximately 20 per cent in developed countries.

The present data showed a very low and decreasing incidence of invasive GBS disease in

newborn infants, 0.32 cases per 1,000 live birth in 1996 and 0.10 cases per 1,000 live births in 2001. A decreasing incidence in this hospital can be speculated from the use of intrapartum chemoprophylaxis adopted in early 1996⁽⁹⁾. However, the present data do not support such hypothesis since only one mother in the present study received intrapartum prophylaxis. Also, there are no guidelines in our hospital for management regarding GBS infection in mothers and newborn infants. A multi-state, active-surveillance program encompassing a population of 10 million in the United States showed the attack rate of 1.8 cases per 1,000 live births⁽¹²⁾. One of the reasons that the present study has a lower incidence rate could be due to the microbiological techniques used in the lab. The authors did not use the selective media specific for GBS culture. It has been shown that using the selective media could increase the yield by 50 per cent^(13,14). It is possible that the incidence could have been higher with the selective media culture technique.

Even though there is a lower incidence in the present study, the mortality rate is much higher than that of developed countries. Six of the 19 (32%) infants died. Premature infants had a much higher fatal outcome, (44%). The fatality rate of EOD in the United States and Great Britain have been reported to be between 0.8 to 0.9 death per 10,000 live births⁽⁴⁾. The present data indicated a fatality

Table 4. Laboratory data.

		%
Arterial pH < 7.25	6	31.58
HCO ₃ < 18 mEq/L	9	47.37
PCO ₂ > 40 torr	5	26.32
Total white blood cell (median)	10,080 (range 1,500 - 59,270)	
wbc < 5,000	6	31.58
wbc > 30,000	1	5.26
Absolute neutrophil count < 1,750/mm ³	8	42.10
Absolute neutrophil count (median)	2,520 (range 0 - 45,045.2)	
Thrombocytopenia < 150,000	1	5.26

rate of EOD to be 0.8 death per 10,000 live births. Interestingly, the present data is from an academic institute with level III NICU and full time neonatologists and thus the mortality could have been higher in different settings in this country. Prenatal care and intrapartum management could play an important role. In the present study, 72 per cent of the mothers had antenatal care with median ANC visits of 6 per pregnancy. The hospital is located in the heart of the largest city and cover a mostly lower income population. The higher death rate could have been from the fact that almost half of the infants who had GBS disease in this study were premature or low birth weight infants. Prematurity has been known to be a major risk factor for invasive GBS disease with fatal outcome(15). Other risk factors such as chorioamnionitis, maternal fever or prolonged rupture of the membrane (>18 h) accounted for 28 per cent of deliveries. There was no risk factor identified in the majority of the mothers, 72 per cent. Only one mother had GBS bacteriuria, with chorioamnionitis. None of the mothers were reported to have a previous child with GBS disease. Previous data have shown that younger mothers had an increased risk of GBS infection in the newborn(7,16). Only one mother in the present study was less than 20 years old.

None of the mothers had antenatal screening for GBS in the present study. A previous study from this institute using the selective media resulted in the colonization rate of 6.4 per cent in 559 full term pregnancy; 18.6 per cent in 118 normal women and 14.8 per cent in 400 women with various causes of vaginitis(17). A report from another academic institute in the northeastern part of the country showed a similar colonization rate of 5.1

per cent in pregnant women at the time of delivery (18). From that study, only 24 per cent of the neonates were colonized and none of the infants had invasive GBS disease. The lower colonization rate may explain the lower incidence of invasive GBS in the present study. It has been postulated that approximately 1 to 2 per cent of infants colonized with GBS via perinatal transmission will have invasive GBS(19).

Early-onset disease accounted for 79 per cent of all infants in the present study. A previous study demonstrated the rate of EOD to be 60 per cent to 80 per cent of all GBS infections(8). Meningitis was found in one patient with EOD and in another with LOD. Meningitis occurred in an estimated 5 per cent to 10 per cent of EOD and 30 per cent of LOD cases in developed countries(8). Like other reports(16,20,21), most of the symptoms and signs in the EOD of the present study occurred within the first 12 hours of life. Respiratory distress was the most common clinical presentation, followed by temperature instability, cyanosis, lethargy and hypotension. Three infants required resuscitation in the delivery room and only two had an Apgar score less than 6 at 5 minutes. One of the infants presented late at 37 days old with cellulitis. Howard et al(22) reported one case of an infant who presented with facial cellulitis which was diagnosed at 7 h of life. Rand(23) reported five cases of GBS cellulitis at age 4 to 11 weeks. His data suggested that hospitalization and/or parenteral antibiotic therapy was a risk factor for development of GBS cellulitis.

Two of the infants in the present study had positive blood cultures for GBS without any symptoms or signs. One was born just before arriving in

the emergency room and subsequently developed polycythemia requiring partial exchange transfusion. Blood culture was reported to be positive while the infant was free of clinical sepsis. No treatment was offered and the infant was discharged after a week of observation. The other case was a term infant born to a mother with intrapartum fever. The blood culture was obtained as routine work up and no antibiotic was given. The infant was discharged home within one week without any symptom and sign of GBS infection. There are reports describing infants with asymptomatic GBS bacteremia. Garcia et al(24) reviewed occult bacteremia with GBS in an out-patient clinic. From this study, younger infants with occult bacteremia did not have characteristic clinical syndrome. Howard et al(22) reported one case of asymptomatic GBS bacteremia whose culture was also obtained due to maternal fever and prolonged ruptured of membrane. This could be explained in the situation that there was enough protective antibody to GBS that had passed from the mother to these infants.

It has been postulated that the initial absolute neutrophil count was one of the predictive factors for fatal outcome(25). In the present study, 42 per cent of infants who had invasive GBS had absolute neutrophil count less than 1,750 cell/mm³. Christensen et al(26) reported that a normal CBC might actually be expected during the first several hours of early-onset neonatal sepsis. A report by Manroe et al(27) demonstrated that the differential white cell count appeared to be a useful tool for screening infants presenting with respiratory distress in the first 48 hours of life and for separating early-onset GBS disease from other causes of neonatal respiratory distress. Even though thrombocytopenia has been reported in 25 per cent of neonatal sepsis

by the time of diagnosis(28), the authors found only one infant who had a platelet count of less than 150,000 cell/mm³.

Several studies demonstrated the difficulty of differentiating chest X-ray findings of GBS infection from other newborn respiratory disorders. Lilian et al(29) reviewed 73 cases of neonates with EOD and found those who weighted less than 1,500 grams had HMD as a predominant finding from the chest X-ray (80% had reticulogranular or ground glass appearance). In larger premature and full-term infants from his study, radiographic findings were not specific and also were not helpful in distinguishing GBS infections from other respiratory disorders. Mortality was reported to be higher in the infants who presented with clinical RDS with a diffuse reticulogranular pattern. The authors found ground glass appearance in 37.5 per cent of the infants. Half of the fatal cases in the present study had a diffuse reticulogranular pattern in their chest X-rays. There were 4 cases (25%) who presented with respiratory distress but had normal chest X-ray finding. Vollman et al(30) previously reported three infants (total of 20) who had GBS presenting with clinical RDS and normal chest X-ray finding.

In summary, the authors found a lower incidence of invasive GBS in neonates during the six year period. Even though the incidence was very low, the mortality on the other hand is still very high, and with a high fatal outcome, the death rate is similar to those of developed countries. Clinical manifestations were also similar to those in developed countries. Premature infants accounted for almost half of the patients, again, with a higher mortality rate. Whether the recommendation of antenatal screening and intrapartum chemoprophylaxis is appropriate for this country, needs to be further investigated.

REFERENCES

- Hood M, Janney A, Dameron G. Beta hemolytic *Streptococcus* group B associated with problems of the perinatal period. *Am J Obstet Gynecol* 1961; 82: 809-1.
- Eickhoff TC, Klein JO, Daly AK, Ingall D, Finland M. Neonatal sepsis and other infections due to group B beta-hemolytic streptococci. *N Eng J Med* 1964; 271: 1221-8.
- Stoll BJ. The global impact of neonatal infection. *Clin Perinatol* 1997; 24: 1-21.
- Embleton N, Wariyar U, Hey E. Mortality from early onset group B streptococcal infection in the United Kingdom. *Arch Dis Child Fetal Neonatal Ed* 1999; 80: F139-41.
- Baker CJ, Edwards MS. Group B streptococcal infections. In: Remington JS, Klein JO, eds. *Infectious diseases of the fetus and newborn infants*. Philadelphia: WB Saunders, 1990: 742-811.
- Boyer KM, Gadzala CA, Kelly PD, et al. Selective intrapartum chemoprophylaxis of neonatal group B streptococcal early-onset disease. II. Predictive value of prenatal cultures. *J Infect Dis* 1983; 148: 802-9.
- Schuchat A, Oxtoby M, Cochi S, et al. Population-based risk factors for neonatal group B streptococcal disease: Results of a cohort study in metropolitan Atlanta. *J Infect Dis* 1990; 13: 672-7.
- Zangwill KM, Schuchat A, Wenger JD. Group B streptococcal disease in the United States, 1990: report from a multistate active surveillance system. In: CDC surveillance summaries (November 20). *MMWR* 1992; 41 (No. SS-6): 25-32.
- Schuchat A, Whitney C, Zangwill K. Prevention of perinatal group B streptococcal disease: A public health prospective. *MMWR* 1996; 45 (No. RR-7): 1-24.
- Thairasana P, Siripong P, Tejavej A. Neonatal septicemia in Ramathibodi Hospital. *Ramathibodi Med J* 1979; 2: 140-6.
- Decreasing incidence of perinatal group B streptococcal disease-United States, 1993-1995. *MMWR* 1997; 46: 473-7.
- Minkoff H, Mead P. An obstetric approach to prevention of early-onset group B streptococcal sepsis. *Am J Obstet Gynecol* 1986; 54: 973-7.
- Baker CJ, Goroff DK, Alpert S, et al. Vaginal colonization with group B streptococcus: A study of college women. *J Infect Dis* 1997; 135: 392-7.
- Ferrieri P, Blair LL. Pharyngeal carriage of group B streptococcus: Detection by three methods. *J Clin Microbiol* 1977; 6: 136-9.
- Boyer KM, Gotoff SP. Strategies for chemoprophylaxis of GBS early-onset infections. *Antibiot Chemother* 1985; 35: 267-80.
- Schuchat A, Denver-Robinson K, Plikaytis BD, Zangwill K, Mohle-Boetani J, Wenger JD. Multi-state case-control study of maternal risk factors for neonatal group B streptococcal disease. *Pediatr Infect Dis J* 1994; 13: 623-9.
- Nilakul C, Thaidhanaiswanya V, Sukroongreung S. Vaginal and perianal carriage of group B streptococci. *J Infect Dis Antimicrob Agents* 1984; 1: 17-9.
- Pengsa K, Puapermpoonsiri S, Taksaphan S, Seejorn K, Sirisalee S. Group B streptococcal colonization in mothers and their neonates. *Ramathibodi Med J* 1984; 7: 83-90.
- Baker CL, Edwards MS. Group B streptococcal infection. In: Remington J, Klein JO, eds. *Infectious disease of the fetus and newborn infant*. 4th ed. Philadelphia: WB Saunders, 1995: 980-1054.
- Carol JB. Group B streptococcal infection. *Clinic in Perinatol* 1997; 24: 59-70.
- Mercer BM, Griggs RG. Group B streptococcus and pregnancy. *Pediatr Ann* 1996; 25: 206-14.
- Howard JB, McCracken G Jr. The spectrum of group B streptococcal infections. *Am J Dis Child* 1974; 128: 815-8.
- Rand T. Group B streptococcal cellulitis in infants: A disease modified by prior antibiotic therapy or hospitalization. *Pediatrics* 1988; 81: 63-5.
- Garcia Pena BM, Harper MB, Fleisher GR. Occult bacteremia with group B streptococci in an outpatient setting. *Pediatrics* 1998; 102: 67-72.
- Payne NR, Burke BA, Day DL, et al. Prospective studies of group B streptococcal infections in infants. *J Pediatr* 1979; 95: 437.
- Christensen RD, Rothstein G, Hill HR, Hall RT. Fatal early onset group B streptococcal sepsis with normal leukocyte counts. *Pediatr Infect Dis* 1985; 4: 242-5.
- Manroe BL, Rosenfeld CR, Weinberg AG, Browne R. The differential leukocyte count in the assessment and outcome of early-onset neonatal group B streptococcal disease. *J Pediatr* 1977; 91: 632-7.
- Zipursky A, Jaber HM. The haematology of bacterial infection. *Clin Haematol* 1978; 7: 175.
- Lilian LD, Harris VJ, Pildes RS. Significance of radiographic findings in early-onset group B streptococcal infection. *Pediatrics* 1977; 60: 360-3.
- Vollman JH, Smith WL, Ballard ET, Light JI. Early onset group B streptococcal disease: Clinical, roentgenographic, and pathologic features. *J Pediatr* 1976; 89: 199-202.

การติดเชื้อสเตรปโตค็อกคัส กรุ๊ปบีในการกรากเกิดของโรงพยาบาลศิริราช

ปณิธาน ยศสุข, พ.บ.*, กาญจนा ปรีดิศรีพิพัฒน์, พ.บ.*

ภาวะการติดเชื้อสเตรปโตค็อกคัส กรุ๊ปบีในกระแสเลือด (GBS bacteremia) ในโรงพยาบาลศิริราช ระหว่างปี พ.ศ. 2538 ถึงปี พ.ศ. 2544 มีจำนวนทั้งสิ้น 19 ราย โดยพบอุบัติการณ์ของการติดเชื้อสเตรปโตค็อกคัส กรุ๊ปบีในระยะแรก (early onset group B Streptococcal disease; EOD) 0.27 รายต่อห้ารကคลอดมีชีวิต 1,000 รายในปี พ.ศ. 2538 อุบัติการณ์ ตั้งกล่าวลดลงเหลือเพียง 0.10 รายต่อห้ารคคลอดมีชีวิต 1,000 รายในปี พ.ศ. 2544 อุบัติการณ์ของการติดเชื้อในระยะหลัง (late onset disease; LOD) พบเพียง 0.05 รายต่อห้ารคคลอดมีชีวิต 1,000 รายในปี พ.ศ. 2538 และไม่พบการติดเชื้อในระยะหลัง (LOD) เลยตั้งแต่ปี พ.ศ. 2539 ทางกรหั้นทด 19 คน ที่พบว่ามีการติดเชื้อเป็นทางกรหั้นทดในโรงพยาบาลศิริราช ร้อยละ 53 ของทางกรมีน้ำหนักตัวแรกคลอดน้อย (low birth weight infant) ร้อยละ 47 เป็นเด็กการเพศชาย มากกว่าเด็กการเพศหญิง ไม่ได้รับการตรวจกรองทางเชื้อสเตรปโตค็อกคัสในระหว่างการตั้งครรภ์ มีมารดาเพียง 1 คนที่ได้รับยาปฏิชีวนะเพื่อป้องกันการติดเชื้อสู่ทางในระหว่างการคลอด ร้อยละ 72 ของมารดาไม่พบว่ามีภาวะเสี่ยงต่อการเชื้อสเตรปโตค็อกคัส กรุ๊ปบีในทางกร การติดเชื้อในระยะแรก (EOD) พบเป็นอัตราส่วนถึงร้อยละ 79 โดยมีอัตราการตายสูงถึง 40% ทางกรหั้นทดที่เสียชีวิตเสียชีวิตภายใน 72 ชั่วโมงหลังคลอด และพบว่าเกือบหั้นทดของการติดเชื้อระยะแรกมีอาการและอาการแสดงภายใน 12 ชั่วโมงหลังคลอด โดยอาการที่พบบ่อยตามลำดับได้แก่ ภาวะหายใจลำบาก ร้อยละ 74 ความผิดปกติในการควบคุมอุณหภูมิกาย ร้อยละ 68 ภาวะดัวเขียว ร้อยละ 63 ภาวะความดันโลหิตต่ำ ร้อยละ 42 และพบอาการเชื่องซึมถึงร้อยละ 42 ในกลุ่มที่มีการติดเชื้อในระยะแรก (EOD) พบภาวะเยื่อหุ้มสมองอักเสบร่วมด้วยเพียง 1 ราย พบทางกรเพียง 2 รายที่มีการติดเชื้อในระยะหลัง (LOD) รายหนึ่งพบมีภาวะเนื้อเยื่ออักเสบ ส่วนอีกรายมีภาวะเยื่อหุ้มสมองอักเสบ ภาวะเม็ดเลือดขาวตัวพับได้ถึงร้อยละ 42 ของทางกรหั้นทด ภาพรังสีเอกซ์เรย์ปอดพบลักษณะขุ่นผ้าคล้ายกระจาดฝ้าถึงร้อยละ 38 ร้อยละ 25 ในผู้พบความผิดปกติของเอกซ์เรย์ปอด โดยสรุปการติดเชื้อสเตรปโตค็อกคัส กรุ๊ปบีในการกรากเกิดในโรงพยาบาลศิริราชมีอุบัติการณ์ที่ต่ำมาก แต่กลับมีอัตราการตายที่สูงมาก โดยครึ่งหนึ่งเป็นทางกรากเกิดคลอดก่อนกำหนด แม้ว่าการตรวจกรองทางเชื้อสเตรปโตค็อกคัส กรุ๊ปบีจะนิยมตั้งครรภ์และให้ยาป้องกันการติดเชื้อสู่ทางในช่วงระยะการคลอดจะเป็นที่แนะนำและปฏิบัติตามในประเทศไทยที่พัฒนาแล้ว การจะนำมาระบุกต์ให้ในประเทศไทยคงต้องการการศึกษาค้นคว้าเพิ่มเติม

คำสำคัญ : GBS, สเตรปโตค็อกคัส กรุ๊ปบี, การติดเชื้อในกระแสเลือด, อุบัติการณ์

ปณิธาน ยศสุข, กาญจนा ปรีดิศรีพิพัฒน์

จุฬามหานครแพทย์ ๔ ๒๕๔๕; ๘๕ (ฉบับพิเศษ ๒): S479-S487

* หน่วยทางกรากเกิด, ภาควิชาภูมิการเวชศาสตร์, คณะแพทยศาสตร์ศิริราชพยาบาล, มหาวิทยาลัยมหิดล, กรุงเทพ ๔ ๑๐๗๐๐