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Neonatal Group B Streptococcal Infection: A Two and a Half-year Retrospective Study in a Local Regional Hospital

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Abstract We performed a retrospective review of those neonates suffering from Group B streptococcal (GBS) infection during the period of 1 January 1996 to 30 June 1998 when there was no implementation of intrapartum chemoprophylaxis. 12 patients with early onset GBS infection and 4 patients with late onset GBS infection were identified. The incidence of early onset and late onset GBS infection are calculated as 0.96 per 1000 live birth and 0.32 per 1000 live birth respectively. The overall incidence of invasive GBS diseases is 1.3 per 1000 live birth with 75% belongs to the early onset group. This incidence is of significance and is comparable to the western studies.

Key words Neonates; Group B Streptococcal infection; Retrospective review

Introduction

Neonatal Group B streptococcal infection and its best preventive strategies have been extensively studied in the western world. Multiple studies confirmed that intrapartum antimicrobial prophylaxis to colonized mothers or those with certain risk factors, is the most cost-effective way of prevention. In May 1996 the Centers for Disease Control and Prevention (CDC), in collaboration with American Academy of Pediatrics (AAP), American College of Obstetricians and Gynecologists (ACOG) and American Academy of Family Physicians, has published a consensus guideline on prevention strategies.[1] As the decision to use preventive measures depends on the incidence of GBS disease in the community, we believe that our estimate of local information concerning incidence, mortality and morbidity rate of the early onset GBS infection is useful. In fact we need to have more local information or data concerning neonatal GBS infection before we can decide on our own local preventive strategy.

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Methods

We have reviewed all the hospital notes and outpatient records of all our patients who have positive GBS cultures (blood or cerebrospinal fluid) recorded on the computer data, during the period of 1 January 1996 to 30 June 1998. They were discharged from our neonatal or paediatrics wards and followed-up in our outpatient clinics. We limited our search after January 1996 because before that time computerization of patients' records was not yet fully set up. We ended our search by June 1998 because after that time there was implementation of intrapartum antibiotic prophylaxis by some of the obstetricians that may make interpretation difficult. The incidences of early onset and late onset GBS disease were calculated with respect to our hospital live births rate during the study period. Only those patients who were born in our hospital were included in the calculation of the incidence.

Results

12 patients with early onset GBS infection and 4 patients with late onset GBS infection were identified. They were all born in our hospital. Their detail presentation is discussed as follows.

I) Early Onset Neonatal GBS Infection

Among the 12 patients with early onset diseases (Table 1), 10 of them have no neurological complication upon 1-year follow-up, one patient with extreme prematurity died while the other patient suffered from cerebral palsy and

Table 1 12 neonates with early onset group B streptococcal infection

Patient	GA wks	BW kg	Birth	Sex	Apgar score 1 & 5 min		AN HVS	Cultures before delivery	Mothers' age yrs	Antenatal/Perinatal risk factors
1*	38 6/7	3.4	NSD	F	8	8	-ve	6m	30	Maternal gestational diabetes mellitus on Insulin
2*	39 6/7	3.3	NSD	M	8	8	-ve	8m	24	---
3*	41 4/7	3.6	NSD	M	8	8	-ve	2m	18	LMSL
4*	39 2/7	3.4	VE	M	9	9	ND		24	---
5*	39 1/7	2.7	LSCS (failed induction, TMSL)	F	6	8	-ve	4.5m	26	TMSL
6*	38 3/7	3.8	VE	M	7	9	ND		25	Prolonged leaking
7*	39 3/7	4.1	NSD	F	7	9	H	3wk	25	MMSL
8*	38 6/7	3.5	LSCS (suspected fetal distress)	M	7	8	H	5.5m	34	Suspected fetal distress
9*	37 6/7	2.7	NSD	F	9	9	-ve	4m	28	Prolonged leaking
10*	37 2/7	2.8	VE	F	8	8	S	5m	24	Twin
11#	36	2.86	NSD	F	8	8	ND		28	---
12^	23 1/7	0.6	NSD	F	5	5	M	5m	29	Premature rupture of membrane

GA = gestational age. BW = birth weight. ANHVS = antenatal high vaginal swab. H = heavy growth. M = moderate growth. S = scanty growth. ND = not done. T/M/LMSL = thick/moderate/light meconium stained liquor. GDM = gestational diabetes. NSD = normal vaginal delivery. VE = vacuum extraction. LSCS = lower segment caesarean section.
*No long-term complication. #Long-term complication. ⊥Died

psychomotor delay.

The 10 patients (patients 1 to 10 in Tables 1 & 2) that have no long-term complication were exclusively full term babies with birth weight > 2.5 kg and Apgar score above 8 at 5 minutes. Their mothers were all above 18 years old. There was equal male to female ratio. Perinatal risk factors, including prolonged rupture of membrane, premature labor, twin pregnancy and suspected fetal distress were present in 6 patients. All the 10 patients had positive blood cultures but no evidence of meningitis (Table 2). Eight patients developed symptoms within the first day of life. The symptoms included respiratory distress, abdominal distension, milk intolerance, delayed crying and early onset neonatal jaundice. None of them suffered from high fever or hypotension. Ear swabs were performed in 9 of them with 8 positive cultures. Gastric lavage cultures were performed in all 10 patients with only 50% positive rate. Postnatal high vaginal swabs were performed in 9 mothers and confirmed maternal colonization in 7 mothers. Most of the positive superficial ear swabs, placental swabs and gastric lavage cultures confirmed moderate to heavy colonization. 2 of the 10 patients had leucocytosis with raised band forms. 1 patient had neutropenia. 5 of the patients received antibiotics soon after birth because of meconium-stained liquor, prolonged leaking or known maternal GBS colonization but 2 of them developed symptoms despite on antibiotics. Chest x-rays finding in those symptomatic neonates revealed unilateral or bilateral streakiness, or pneumonic changes. All the 10 patients were treated with Penicillin and aminoglycosides for 7 to 10 days. Upon follow-up, none of them developed neurological complication.

Among these 12 patients, one survived with quadriplegic cerebral palsy, psychomotor delay and epilepsy (patient 11 in Tables 1 & 2). He was discharged from post-natal ward on D2 and was readmitted on D4 presenting with poor oral feeding, irritability and persistent crying. Examination revealed jitteriness and respiratory distress. No antenatal vaginal culture was done. He developed recurrent cyclical upper limb movement and fever with unstable temperature. Sepsis workup confirmed meningitis without septicaemia. CT scan brain showed meningitis with ischaemic changes while electroencephalogram revealed epileptic activities. On day 10, pseudoparalysis of the left hip was noted with tenderness and limited passive range of movement. However the bone scan and X-ray findings were normal. She was treated with Penicillin and Gentamicin

Table 2 12 neonates with early onset group B streptococcal infection

P	Symptoms	Cell Count						Culture					
		WBC x 10^9/L					x10^9/		Swabs				
		TOTAL	N	L	BF	TG	Plt	Bld	E	P	GL	CSF	PNHVS
1*	Respiratory distress at 5 hrs of life. Milk intolerance & abdominal distension since day 1	34↑	30.9↑	2.4	<5%	-ve	109	+	M		H	-	S
2*	Respiratory distress at 5 hrs of life	26	23	2.5	<5%	-ve	138	+	S		-	-	H
3*	Respiratory distress since 8 hrs of life. One kick of 38C on day 1	6.7↓	4.7↓	1.6	8%↑	-ve	239	+	M		S	-	S
4*	Respiratory distress at 5 hrs of life	11.7	9.3	1.7	NA		169	+	M		-	-	H
5*	Asymptomatic	18	11.3	4.3	<5%	-ve	331	+			-		
6*	Delayed crying. Early onset neonatal jaundice	8.4	6.7	1.4	10%↑	-ve	167	+	H	H	H	-	H
7*	Abdominal distension & milk intolerance since D2	28.8	25.6	2.9	<5%	-ve	255	+	H	H	-	-	-
8*	Delayed crying	16.8	9.9	4.7	<5%	-ve	211	+	H		H	-	M
9*	Respiratory distress at 8 hrs of life	31↑	NA				283	+	M	S	-	-	-
10*	Asymptomatic	NA						+	-		M	-	M
11#	Discharged on D2. Readmitted on D4: Poor feeding, jitteriness, fever, respiratory distress	NA						-				+	
12^	Extreme prematurity. Multiple prematurity complications (RDS, NEC, IVH)	5.3					172	+	M		H	-	-

P = patient. N = neutrophil. L = lymphocyte. BF = band form. TG = toxic granulation. Plt = planelet. Bld = blood. E = ear swab. P = placental swab. GL = gastric lavage. CSF = cerebrospinal fluid. PNHVS = post-natal high vaginal swab. H = heavy growth. M=moderate growth. S = scanty growth. RDS = respiratory distress syndrome. NEC = necrotizing enterocolitis. IVH = intraventricular haemorrhage.
*No long-term complication. #Long-term complication. ⊥Died

for a total of 4 weeks and, Luminal and Phenytoin for 3 weeks.

The last patient (patient 12 in Tables 1 & 2) was an extremely premature baby with birth weight of 0.6 kg. Antenatal high vaginal swab taken 5 weeks before delivery yielded moderate growth. After birth, superficial swabs confirmed baby's colonization (moderate growth in ear and umbilicus swabs; heavy growth in throat, nasal swabs and gastric lavage). Blood culture confirmed septicemia. Cerebrospinal fluid culture was negative. She was put on Penicillin and Azactem. However she suffered from multiple complications of prematurity and developed multiple end-organs failure despite aggressive resuscitation. She finally succumbed on day 52.

II. Late Onset GBS Infection

Unlike the early onset group, all the 4 patients with late onset diseases had symptomatic course (Table 3). 1 patient developed meningitis and 1 patient had septicaemia with soft tissue infection (patient 1 in Table 3). 2 patients had both septicaemia and meningitis and one of them (patient 4 in Table 3) who suffered from status epilepticus and SIADH developed cerebral palsy, global delay and cortical blindness on subsequent follow-

Table 3 4 babies with late onset GBS infection

P	GA wk	BW kg	Birth	Sex	AS 1&5 min	AN HVS	Mo yrs	Antenatal/ Perinatal risk factors	Presentation	Bld	CSF	Treatment	Neurological Outcome
1	32	1.5	CS	F	7 8	ND	18	PPROM Triplet pregnancy	Prophylactic anti-biotics after birth. (All cultures -ve) Presented on **D34** with repeated apnea, bradycardia, dullness, marked erythema at suprapubic region.	+	-	Penicillin Netromycin IPPV	Normal
2	39 3/7	3.0	NSD	M	8 8	ND	29	---	Uneventful delivery. Stayed in hospital for social reason. **D13** developed high fever, twitching & hyponatraemia.	+	+	Penicillin Claforan Luminal	Normal
3	38	3.3	NSD	F	8 9	ND	29	---	Uneventful delivery. Readmitted on **D22**, presented with high fever, vomiting, abdominal distension & repeated desaturation	-	+	Penicillin Claforan IPPV	Normal
4	35 2/7	2.7	NSD	M	8 9	-ve 4m before birth	28	Maternal postpartum fever	Prophylactic Penicillin & Netromycin for 3 days for maternal postpartum fever & cyanosis after birth. (Cultures -ve) Readmitted on **D26** with tachypnoea, fever, SIADH, status epilepticus with recurrent limbs twitching, Rt. eye deviation, Rt. ptosis & horizonal nystagmus.	+	+	Penicillin Netromycin IPPV Luminal Fluid restriction	Spastic quadreplegia. Global delay. Cortical blindness. Cerebral atrophy.

P = patients. GA = gestational age. BW = birth weight. ANHVS = antenatal high vaginal swab. AS = apgar score. Mo = mother's age. Bld = blood culture. CSF = cerebrospinal fluid culture. ND = not done. CS = caesarean section. NSD = normal spontaneous delivery. PPROM = premature prolonged rupture of membrane.

up. Two of the 4 patients are premature babies. Antenatal and perinatal risk factors were present in these 2 premature babies. Two of the patients were readmitted to the hospital after uneventful discharge while the other 2 patients stayed in the hospital for gradual weight gain or social reason.

Discussion

Since 1970, Group B streptococcus or Streptocococcus agalactiae emerged as the predominant organism causing bacteremia and meningitis in neonates. Since then the incidence of neonatal infection has remained stable, with reported attack rates ranging from 0.2 to 5.4 per 1000 live births.[2] Early onset diseases account for 80% of cases.

In our retrospective study we have identified 12 cases of early onset GBS diseases from January 1996 to June 1998. There were no late, late onset diseases identified. During that period the total live births in our hospital were 12455. The incidence of early onset GBS infection was calculated as 0.96 per 1000 live birth. When we computed the incidence for each year, the results revealed a mild decreasing trend (Table 4).

Compared with the overseas data, our incidence of early onset GBS infection is not that high. However, our calculated incidence is higher when compared to a locally reported incidence of 0.58 cases per 1000 live birth in 1985.[3] This suggests no decrease or even an increase in incidence of early onset GBS infection in our locality, within the last 15 years. In our hospital, we routinely gave post-natal prophylactic Penicillin to those asymptomatic babies, and with aminoglycosides to those symptomatic babies, whose mothers were confirmed GBS carriers. This suggests no significant improvement concerning prevention using post-natal chemoprophylaxis. Compared with the western figures, the early onset GBS infection rate among Chinese babies is not that high, as demonstrated by the local 1985 study and our study.[3] This may be related to our lower maternal colonization rate among Asian population.[4-6] In a local prospective study (between 1983 and 1984) of 186 women in third trimester pregnancy, the maternal carriage rate of GBS from multiple site (endocervix, rectum and perineum) was 19% with a genital carriage of 7.4%.[4] In another local prospective study of 367 women between 16 to 24 weeks gestation, GBS were only found in 1.1% of low vaginal swabs and 0.8% of endocervical swabs.[5] In a western study Asian women had a significantly lower rate of GBS colonization of 14% as compare with the 20 to 40 % in the other ethnic groups.[6] Since the first two local prospective studies were carried out in different time and using different culture medium, one cannot simply postulate that there is an increase in maternal GBS colonization (using endocervical swab) from the 0.8% in the second trimester to 7.4% in the third trimester. In the study by Yim et al,[5] solid agar media without the antimicrobial agents were used, but in that of Liang et al[4] cultures were taken from multiple sites and both broth and solid agar with antimicrobial agents were used. The recommended culture techniques for optimal identification of carriers include the use of broth media (not solid agar) that contain antimicrobial agents (selective broth) to inhibit competing organisms present in the normal vaginal and rectal flora. The use of selective media may increase the yield of GBS screening cultures by as much as 50%.[7] Therefore the reported 0.8% GBS carriage rate is actually much underestimated because of the culture media used and single site chosen. The difference in design of measuring maternal GBS colonization in these two prospective studies, nicely reflect our current variable practice on routine antenatal screening among many of our hospitals. Most of the routine antenatal swabs are usually taken more than 2 months before delivery, from the vagina mainly. This may make correlation of the antenatal maternal colonization and early onset GBS infection difficult. Studies have shown that although colonization rates are similar by trimester, cultures obtained at 35 and 37 weeks' gestation have the greatest predictive value for colonization at delivery.[8] To increase the yield of GBS isolation from the pregnant women, the specific body site sampled is also important. Studies showed that collection of specimens from two sites, the lower vagina and rectum, increase the likelihood of GBS isolation by 5% to 27% when compared to sampling of only the lower vagina.[8] Collection of cervical specimens rather than lower vaginal specimens also decreases the yield of group B streptococcus. Therefore, the reported 7.4% maternal carrier rate in Liang's 1986 study may be underestimated, as endocervix was cultured instead of lower vagina.

Comparison of early onset GBS infection between our patients and the overseas data was performed (Table 5). Prematurity was not that common among our population compared with overseas data. In fact prematurity did not

Table 4 Incidence of early onset GBS infection per 1000 live birth in Queen Elizabeth Hospital (QEH)

Year	No. of live birth in QEH	No. of early onset GBS infection	Incidence of early onset GBS per 1000 live birth
1996	5802	7	1.2 per 1000 live birth
1997	4835	4	0.8 per 1000 live birth

Table 5 Comparison of the clinical features of early and late onset GBS infection of our patients with the overseas data. (Modified from Baker and Edwards.[5])

	Early onset GBS infection		Late onset GBS infection	
Features	**QEH patients**	**Dr. Baker's review[5]**	**QEH patients**	**DR. Baker's review[5]**
Median age at onset	5 hrs	1 hr	24 days	27 days
Incidence of prematurity	20%	70%	50%	Uncommon
Maternal obstetric complications	50%	Common	25%	Uncommon
Usual clinical presentations	Septicemia (100%) Respiratory disease (42%) Meningitis (8.3%) Hip infection (8.3%)	Septicemia (25-40%) Respiratory disease (35-55%) Meningitis (5-15%)	Septicemia without focus (50%) Meningitis (75%) Cellulitis /adenitis (25%)	Septicemia without focus (40-50%) Meningitis (30-40%) Cellulitis /adenitis (2%) Osteoarhritis (5-10%)
Common serotypes	Not tested	I (Ia, Ib, Ia/c), II, III, V	Not tested	III (90%)
Mortality rate	8.3%	5-20%	0%	2-6%

seem to have an important influence on the occurrence of invasive disease in the 1986 local study as well.[3] Part of the explanation lies in the relatively low incidence of prematurity in our locality, especially those with very and extreme low birth weight, who have the highest attack rate. A survey carried out in the Chinese population in Hong Kong in 1980-1981 showed that while the incidence of low birth weight (LBW) was comparable to that in the West (7.45%), there was significantly fewer very low birth weight (VLBW) (0.69%) and extreme low birth weight (ELBW) infants (0.14%).[9] Another possibility may be due to the more liberal use of post-natal prophylactic antibiotics in our prematurity population. All our premature babies less that 34 weeks gestation were given prophylactic antibiotic for 48 to 72 hours until blood cultures revealed negative growth. This may help to prevent those early onset infections due to early nosocomial transmission.

Comparing with Dr. Baker series[10] we have a much higher rate of septicemia without major complication, a similar rate of respiratory symptoms and a lower rate of meningitic involvement. Majorities of our patients (80%) with early onset GBS infection are full term babies, instead of prematurity, with satisfactory birth weight and Apgar score. Two of our patients delivered by ceacerean section developed early onset disease suggesting in-utero infection rather than vertical transmission. Superficial ear swab cultures are the most sensitive and simplest test to detect babies colonization. 83% of our patients had positive superficial cultures that confirmed babies' colonization. 80% of these positive superficial cultures showed moderate and heavy growth. Post-natal high vaginal swabs were taken in 83% of our cases with 70% of positive rate. This suggested that the maternal colonization rate is high among this group of patients with early onset GBS infection. 75% of those mothers with positive high vaginal swab cultures had either moderate or heavy colonization. Majority of our babies also has moderate to heavy colonization.

50% of our 12 mothers have maternal obstetrics complication. These included prolonged leaking (2 patients), prematurity (1 patient), twin pregnancy (1 patient), gestational diabetes (1 patient) and suspected fetal distress (1 patient). 67% of our patients with early onset GBS infection developed symptoms within the first 8 hours of life. The symptoms presented among our patients included respiratory distress and cyanosis (5 babies), milk intolerance and abdominal distension (2 babies), delay crying (1 baby), fever (1 baby), early onset jaundice (1 baby) and rarely the hip infection (1 baby). Those babies with full term delivery, satisfactory birth weight and Apgar score, and septicemia alone with early treatment had favorable outcome. The one who suffered from fulminant infection and cerebral palsy may be related to the delay attention after initial discharge and the meningitic involvement. He suffered from meningitis without septicemia and the rare Lt. hip infection, which rarely occurred in early onset disease. This patient in contrast with other uneventful babies reflects the importance of early treatment that improves prognosis significantly. There is no specific association between cell counts, symptomatology and the degree of colonization noted.

We have 4 patients with late onset GBS infection. The incidence is calculated as 0.32 per 1000 live birth. Half of our patients are premature babies with their mothers having obstetrics complications. 3 of them have meningitis (75%). There is no fatal outcome. Since the case number is too small. A meaningful comparison in terms of percentage with that of Dr. Baker's review is difficult (Table 5).

The major drawback of this review is the small number of patients being studied. This can be overcome by combining data from several hospitals for more reliable

analysis. However our study result may give some information and ideas about our local practice as well as the incidence of early and late onset GBS disease before the implementation of intrapartum chemoprophylaxis.

Further studies on the local colonization rate (cultures from lower vagina and anorectum after 35 weeks gestation with selective media), the infective rate of colonized mother and babies, the significance of dosage of colonization on infectivity and the incidence of GBS disease (early, late, late-late onset) are suggested. Studies involving collaboration of several hospitals will definitely increase the number of patients being analyzed and therefore the power and reliability of the results.

In summary, early onset GBS disease is still a common infection among our locality. Our calculated incidence of GBS invasive disease is 1.3 per 1,000 live birth with 75% belonging to the early onset group. The calculated incidence of early onset GBS disease is 0.96 per 1,000 live birth. As there is a high possibility that some of the babies who were born in our hospital and suffering from GBS infection were being admitted to other hospital for management, the true incidence of GBS infection is likely to be underestimated. Compared with the local 1986 study, there is an increase in attack rate. Apparently post-natal chemoprophylaxis does not prevent GBS infection effectively. The designation of our local preventive strategy involves the cooperation of both the obstetricians and pediatricians. Is an expensive preventative measure justified? Is intrapartum antibiotic chemoprophylaxis better than post-natal chemoprophylaxis? Is the universal culture-screening approach better than the risk factor-based non-screening approach, for intrapartum antibiotic chemoprophylaxis? In order to answer the above questions, further local controlled studies or multi-hospitals retrospective reviews are needed before we can decide on our own optimal preventive strategy and the most cost-effective way to prevent neonatal GBS diseases with the minimal risks.

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