

NEONATAL GROUP B B-HAEMOLYTIC STREPTOCOCCAL INFECTION IN KUALA LUMPUR

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SUMMARY

First six cases of neonatal group B B - haemolytic streptococcal sepsis in GHKL & Maternity Hospital K.L. were reported and in one third, it was fatal. Five of the cases were 'early - onset' type and one was 'late - onset' type. While maternal infant transmission of the disease is important in the 'early - onset' type, environmental sources of infection are also significant. No gestational age or birthweight is spared from the disease. Finally, there are cases of 'early - onset' GBS sepsis presenting like hyaline membrane disease of the newborn and it is important to find ways to distinguish them which so far has not been satisfactory.

INTRODUCTION

The changing pattern of neonatal bacterial infections is wellknown to Paediatricians for many years. In the late 1940's B- haemolytic streptococcus was replaced by Coliform organisms as a major cause of neonatal infection. And in the 1950's, epidemic staphylococcal problems became apparent in neonatal nurseries. But with the introduction of Hexachlorophene, staphylococcal infection became uncommon and once again infection with coliform organisms

predominated from the 1960's. However, Group - B - Streptococcus (now known as GBS) began to show as an important cause of infection in the neonatal period since early 1970.

In Malaysia, GBS infection in the neonates apparently has not been much of a problem. The purpose of the present report is to draw attention to the fact that GBS infection is not uncommon in the newborns here and to discuss its modes of presentation and clinical significance.

MATERIALS AND METHODS

From January 1979 we started to look actively for GBS infection among the neonates. During the next year we identified six cases. Records of confirmed GBS infections in the neonates in the General Hospital Kuala Lumpur and the Maternity Hospital Kuala Lumpur were reviewed. The cases were confirmed from positive cerebrospinal fluid or blood cultures done antemortum or postmortum. Bacterial isolation was done by the Microbiology Department of the University Kebangsaan Medical Faculty. Isolated GBS were identified but no serotyping of the organism was done due to inavailability of the facility.

RESULTS

Out of six cases, one was diagnosed from postmortum blood culture (case III), and one was associated with meningitis (case II). Two of the affected infants died.

a) Neonatal Characteristics: [Table I]

There were 3 males and 3 females and 5 were Malays. Three cases were preterm. Five cases

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TABLE I
NEONATAL GROUP-B-STREPTOCOCCUS INFECTION
NEONATAL CHARACTERISTICS

Case	Place of Delivery	Race	Sex	Gestation in Weeks	Birth Weight in Gm	Age of Presentation	Clinical Presentations
I	Home	Malay	Male	39	2525	28 hrs.	Fever, refusing feeds tachypnoea, lethargy, mild dehydration
II	MHKL	Malay	Female	40	3015	15 days	Fever, refusing feeds tachypnoea, lethargy, tense ant. fontanelle septic skin.
III	MHKL	Malay	Female	28	900	2 hrs.	Respiratory distress syndrome, Apnoeic spells at 9 hrs.
IV	MHKL	Malay	Female	34	1530	96 hrs.	Fever, lethargy, jaundice.
V	MHKL	Malay	Male	40	2535	At birth	Asphyxia neonatorum, muconium aspiration, respiratory distress syndrome.
VI	Private Hospital	Indian	Male	35	1735	4 hrs.	Respiratory distress syndrome.

presented within 10 days age i.e. early-onset type, and one was at 15 days of age i.e. late-onset type. Clinically, presentation of three of the babies (Case I, II, IV) were no different from the usual presentation of neonatal septicaemia and one (case V), following muconium aspiration. However, two cases presented with features indistinguishable from hyaline membrane disease (referred to as HMD), with one being diagnosed only after death.

b) Laboratory findings: [Table II]

Blood cultures from all the cases grew GBS

and in case II, the organism was also isolated from the cerebrospinal fluid. Chest X - rays of cases II & VI (which presented with Respiratory Distress Syndrome) were reported by the radiologists as consistent with features of HMD, while that of case V showed consolidation of (R) upper zone. Total white cell and neutrophil counts were increased in two cases.

Maternal findings: [Table III]

Antenatal warning of the infection was absent in all except one. All mothers had membranes ruptured of less than twenty four hours. Only one case had a history of traumatic delivery i.e. foetal

TABLE II
NEONATAL GROUP-B-STREPTOCOCCUS
INFECTION LAB. FINDINGS

Case	Chest X-ray findings	Blood Culture	CSF Culture	Total WBC [10 /L]	Neutrophils %
I	Normal	+ve	—ve	19.8	90
II	Normal	+ve	+ve	8.6	63
III	Ground glass appearance	+ve	—ve	9.7	58
IV	Normal	+ve	—ve	8.0	60
V	Pneumonitis	+ve	not done	16.4	80
VI	Ground glass appearance	+ve	not done	2.7	40

TABLE III
NEONATAL GROUP-B-STREPTOCOCCUS INFECTION
MATERNAL OBSTETRIC FEATURES, TREATMENT
AND OUTCOME

Case	Maternal Obstetric Features		Treatment and Outcome			
	Amniotic membrane rupture in hrs.	Maternal vaginal culture of GBS	Initial treatment	Subsequent treatment to blood culture result	Duration of treatment	Outcome of treatment
I	4	+ve	Ampicillin + Gentamicin	Penicillin + Gentamicin	2 weeks	Discharge well
II	7	—ve	Ampicillin + Gentamicin	—	—	Died at 2 hrs after admission
III	1	—ve	Nil	Nil	—	Died at 36 hrs.
IV	2	—ve	Penicillin + Gentamicin + Exchange transfusion	Penicillin + Gentamicin	2 weeks	Discharge alive
V	20	not done	Ampicillin + Gentamicin	Penicillin + Gentamicin	2 weeks	Discharge alive
VI	5	not done	Ampicillin + Gentamicin	Penicillin + Gentamicin	2 weeks	Discharge alive

distress with birth asphyxia.

One of the four mothers subsequently investigated showed a positive vaginal GBS colonization.

Management and outcome: [Table III]

As a routine, all cases of suspected neonatal septicaemia were started on either ampicillin (200-300 mg/kg/day) or crystalline penicillin (100,000 Units 3 x per day) in combination with gentamicin (5 mg/kg/day) initially. Ampicillin was changed to crystalline penicillin (except in one case) when positive blood culture results came back.

One case (case III) received no antibiotic at all because there was no suspicion of septicaemia. This infant was treated as a case of HMD and died at 36 hours of age. Another case (case II) died two hours after admission i.e. half an hour after administering the antibiotics.

Four cases survived and were discharged well after completion of a two week course of antibiotics. Case IV, in addition had an exchange transfusion as part of treatment of septicaemia.

DISCUSSION

For the first time cases of neonatal GBS septicaemia were being recognised in the General and Maternity Hospital Kuala Lumpur. This organism, which has been playing a significant role in neonatal infection in other parts of the world since early 1970, has created great interest among paediatricians because of its high mortality especially among the 'early-onset type'.

Baker *et al* (1973) reported a mortality of 58% for the 'early-onset type' and 14% for the 'late-onset type', while Hey (1973) reported a 100% mortality in the 'early-onset type' and 50% mortality in the 'late-onset type'. The overall mortality of neonatal GBS infection is in the region of 50%. Early clinical recognition and institution of treatment of this condition are of prognostic importance. This report showed a

mortality of 2 out of 5 cases.

The manner by which the infant acquires early-onset GBS septicaemia has been a subject of considerable study. Most authors stress the importance of mother - to - infant route of transmission. Prolonged rupture of membranes (more than 24 hours), maternal fever, positive maternal vaginal culture and foetal distress are reported to be important factors associated with this mode of transmission. In this report, only one of the four mothers of the affected infants was found to have GBS grown from the vaginal swabs. One infant had a history of foetal distress during labour. These would imply that while maternal - infant transmission of the disease is important, environmental factors like delivery room, resuscitative instruments and attendants are also important as sources of infection.

Farida *et al.* 1979 reported a vaginal GBS colonization of 21% from 83 women studied in the same hospitals i.e. General and Maternity Hospital Kuala Lumpur, which was consistent with most reports. However, no attempt was made to correlate this neonatal infection. No case of neonatal GBS sepsis was reported from the same hospitals during that study period i.e. 1978. Most probably it was because it was not specifically looked for and not because the true absence of the infection. Since then cases of neonatal GBS are being recognised here.

Quirante *et al.* (1974) reported that all cases of 'early onset' GBS sepsis were confined to low birth weight and preterm infants. However, this report together with reports by other workers like Menke *et al.* (1979), showed that no gestational age or birth weight is spared from the disease.

Finally, the presentation of some of 'early - onset' GBS infection simulating that of HMD is of clinical importance. Cases III and VI presented with clinical features indistinguishable from HMD. Since the prognosis of GBS sepsis depends greatly on early diagnosis and antibiotic treatment, it is therefore important to find ways to distinguish cases of 'early-onset' GBS sepsis from HMD early.

Ablow *et al.* (1979) and Menke *et al.* (1979) have suggested some criteria to distinguish 'early onset' GBS sepsis presenting with respiratory distress from HMD. However, these criteria still require further confirmation and more research on this is obviously required. While waiting for this problem of differentiating early onset GBS septicaemia from idiopathic respiratory distress syndrome to be resolved, we in the Maternity Hospital decided to start all babies with respiratory distress syndrome with antibiotics (ampicillin and gentamicin) after taking blood culture. The antibiotics are then reviewed when results of the blood culture comes back, and discontinued if they are negative.

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