

# Case Report

## Group B Streptococcal Sepsis in a Newborn: a Case Report

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### ABSTRACT

Newborn's bacterial infections due to group B *Streptococcus* (GBS) happen in two forms including early-onset disease or late-onset disease. In this paper, we report a case of early-onset GBS infection in a male infant. A 22-year-old primigravid woman delivers a term normal looking male infant. Nasal flaring, grunting, and poor feeding presented soon after birth. An empiric treatment with intravenous ampicillin and amikacin initiated. On the second day, he was transferred to Newborn Intensive Care Unit (NICU). The intravenous antibiotics were changed to tazocin and vancomycin in NICU. The blood culture (BC) was positive for GBS. After 48 hours, respiratory distress symptoms disappeared, BC was negative, and ABG and CBC became normal. Finally, the infant was discharged after 15 days. GBS is a normal flora of women's gastrointestinal and genitourinary tracts. Infants with early-onset GBS sepsis need very close observation including repeated vital signs evaluation.

**Keywords:** Group B Streptococcal, Sepsis, Newborn

### Introduction

Since 1970, the major cause of newborns' bacterial infections has been Group B *Streptococcus* (GBS). GBS happens in two forms including early-onset disease (seven or fewer days of life) or late-onset disease (more than 7 days of life) (1).

The ascending transmission of organism into the chorioamniotic space before delivery may occur in 5% to 45% of pregnancies (2-4).

Mother-related risk factors for early-onset GBS sepsis consist of membranes ruptured 18 hours or more prior to labor, positive recto-vaginal culture, fever of 38°C or higher in mother, delivery before week 37, GBS bacteriuria in preg-

nancy and a past history of infant with a GBS infection (2).

In most cases of early-onset GBS infection symptoms present in the first 24 hours of life and frequently appear in the first hour (2, 3).

Respiratory distress is the most frequent sign of early-onset GBS infection in newborns. Other signs of early-onset GBS infection are lethargy, temperature instability, poor feeding, and glucose intolerance. Hypotension, fetal asphyxia, rapidly worsening respiratory distress, and persistent pulmonary hypertension are generally present in severe invasive infection (1). Early-onset GBS infection has the mortality rate of 4.5% to 15% (2,5-7).

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Late-onset GBS has unclear symptoms including lethargy, poor feeding, or irritability. Meningitis may present in about 30% to 40% of infants with late-onset GBS (2, 8). Its mortality rate is about 2% to 6%, and the intrapartum chemoprophylaxis could not change the outcome (5, 9).

The expansion of either early- or late-onset GBS infection has no relationship with gender (2).

Despite reducing the general rate of GBS infection, it is still the major reason of neonatal morbidity and mortality. In this paper, we report a case of early-onset GBS infection in a male infant.

### Case Report

A 22-year-old primigravid woman delivers a term male infant weighing 3,320 g through vaginal delivery. His height was 50 cm, and the head circumference was 37 cm. The infant's first vital signs and physical examinations were normal. The mother had no history of HTN, DM and rupture of membranes. Apgar scores were 9 and 10 at 1 and 5 minutes, respectively. Except grasping, he had weak reflexes. Nasal flaring and grunting presented soon after birth but he had no retraction sign. Poor feeding appeared two hours after the delivery. An empiric treatment with intravenous (IV) ampicillin and amikacin initiated. The chest radiography was normal. On the second day, he suffered from grunting and respiratory distress and was transferred to Newborn Intensive Care Unit (NICU).

The infant was intubated in NICU and received surfactant. His ABG results showed severe metabolic acidosis in NICU. The intravenous antibiotics were changed to tazocin and vancomycin in NICU.

Complete blood count revealed leukopenia and neutropenia with normal hemoglobin (Hb). He had positive CRP and the blood culture (BC) was positive for gram-positive coccus, which was later diagnosed as GBS. The cerebrospinal fluid (CSF) culture was negative. An echocardiography revealed a pulmonary hypertension. In order to prevent pulmonary bleeding, he took one dose of intravenous Mg-sulfate 50% (3mg/kg/h). At this time, the chest X-ray showed patchy infiltrates related to pneumonia. The brain sonography was normal. The abdominal sonography demonstrated hepatomegaly and a little cholestasis; the kidneys were a little bigger than normal with the enhancement of cortical echo. He had no renal stone, hydronephrosis or mass lesions. After 48 hours, respiratory distress symptoms disappeared, BC was negative, and ABG and CBC became normal. Although hypocalcemia and low blood pressure (BP) were detected, they were completely controlled with IV-Ca gluconate and dopamine, respectively.

After 22 hours of intubation, the infant was extubated and was put under respirator hoods. His general condition was better at 4 days of age and finally, the infant was discharged after 15 days.

Table 1 shows the serial changes of CBC, ABG and other laboratory tests lab exams.

**Table 1-** Complete Blood Count (CBC) and Arterial Blood Gas (ABG)

Case	First	Second	Third
WBC (1000/ml)	2.9	14.68	10.7
Hb (mg/dl)	17.7	13.6	15
RBC (Mil/ul)	4.97	3.79	4.33
HCT (%)	50.4	38.8	44.2
Plt (/ml)	173000	129000	370000
Neutrophil	38%	70%	42%
Lymphocyte	57%	6%	43%
Monocyte	1%	5%	9%
Basophile	3%	1%	5%
pH	7.45	7.3	7.23
PO2(mmHg)	54.8	54.1	189.1
PCO2(mmHg)	28.2	43.1	26.4
HCO3(mmol/lit)	19.5	20.4	10.9
O <sub>2</sub> Sat	89.6%	83.5%	99.2%

## Discussion

GBS is a normal flora of women's gastrointestinal and genitourinary tracts. It is usually colonized without any symptoms. Intrapartum positive cultures may be detected in 20% of women (10). Age less than 20 years; diabetes and African American race are maternal GBS carrier risk factors (11). In our case, we had none of these risk factors. However, recent reports indicate that there are no particular maternal risk factors in early-onset GBS (2).

Hypotension with a rate of over 25% of all cases is a warning sign of severe invasive infection, as seen in our case (2).

Rench *et al* at 1993 reported two cases of GBS infection with common presentations, which had a good response to general GBS treatment. Their first patient was a male term infant with early-onset GBS that presented apnea and respiratory failure and the second case was a premature girl with late-onset GBS infection (12).

In 2009, Sabnis *et al* reported a term female infant weighing 3,112 g delivered by caesarean section. She had normal physical examinations, but tachypneas, and grunting appeared in the fourth hour of life. The chest X-Ray was normal.

Leukopenia, neutropenia and thrombocytopenia were seen in her complete blood count and the blood culture was positive. She also had a metabolic acidosis and hypotension. Using intravenous ampicillin and cefotaxime made the infant better and she was extubated on postnatal day 15 and was discharged at 18 days of age (13).

Infants with early-onset GBS sepsis need very close observation including repeated vital signs evaluation (at least every 2–4 hours). Any temperature instability, appearance of poor feeding, respiratory distress, apnea, abdominal distension, frequent emeses, lethargy, or seizure must instantly be a notice to the doctors (14).

Waiting for laboratory results, ampicillin with the addition of an aminoglycoside is useful for treating GBS infection based on the recent suggestions (2).

There is a significant reduction in early-onset GBS and meningitis rates in newborns via the extensive achievement of intrapartum antibiotic prophylaxis (15).

As Centers for Disease Control and Prevention (CDC) in 1998 suggests, GBS infection must have perinatal prevention strategies like pre-

venting neural tube defects and transmission of hepatitis B infections by using folic acid and vaccination, respectively (9).

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