Group B Streptococcal Bacteremia Associated with Acute Hemorrhagic Gastritis in a Healthy Full-Term Neonate

Phisek Yimyaem, M.D.*
Porntep Suandork, M.D.**
Voranush Chongsrisawat, M.D.**
Boosba Vivatvakin, M.D.**
Sasithorn Likitnukul, M.D.**
Naruemon Wisedopas, M.D.***

ABSTRACT

Group B Streptococcus has been identified as a major cause of severe systemic and focal infections in the neonatal period including septicemia, meningitis, pneumonia, osteomyelitis, arthritis and cellulitis. Acute hemorrhagic gastritis associated with group B streptococcal infection is extremely rare. We report a 2,950-gram female neonate who presented with profuse upper gastrointestinal bleeding and hypotension without coagulopathy on the second day of life. She was delivered by the vacuum extraction. She was treated with blood transfusion, intravenous octreotide and ranitidine. Upper gastrointestinal endoscopy revealed diffuse hemorrhagic gastritis and blood culture subsequently grew group B Streptococcus. She was treated with intravenous ampicillin and gentamicin for 14 days. Pneumonia and meningitis were not noted during this course. The infant has been continued with oral ranitidine for 8 weeks and remains in good health at 3 months of age. (J Infect Dis Antimicrob Agents 2004;21:21-4.)

INTRODUCTION

Group B Streptococcus (GBS) is well recognized to cause a variety of infections including primary bacteremia, meningitis, and nonmeningeal infections during the first 2 to 3 months of life.1-3 However, GBS bacteremia is usually associated with pneumonia, meningitis or less commonly with osteomyelitis, arthritis and cellulitis.4-7 GBS bacteremia associated with phlegmonous (suppurative) duodenitis in adult is extremely rare. Only one case has been reported.8 In the neonatal period, GBS bacteremia associated with severe upper gastrointestinal bleeding and gastro-duodenitis is uncommon, and very few cases have been reported in healthy full-term infants. We report a case of severe upper gastrointestinal hemorrhage due to diffuse hemorrhagic gastritis in the first 48 hours of life.
in an apparently healthy full-term newborn, who had occult GBS bacteremia.

**CASE REPORT**

A 2,950-gram full-term, Thai female infant was born by vacuum delivery, due to maternal exhaustion, to a 29-year-old primigravida in another hospital. Apgar scores were 9 and 10, at 1 and 5 min, respectively. The mother’s antenatal history was uncomplicated. There was no family history of peptic ulcer disease. The baby received cow’s milk-based formula at 12 hours of age. She was well until 14 hours of age, when she vomited fresh blood. Soon after, the infant vomited a large amount of bloody gastric contents and passed several melanotic stools, then she became hypotensive. She was noted to be afebrile, pale but alert without petechiae or ecchymosis. The rest of physical examination was unremarkable. Nasogastric aspiration revealed a large amount of fresh blood. She was initially treated with nasogastric lavage, intravenous fluid replacement, intravenous ranitidine (1 mg/kg every 8 hours) and a continuous infusion of octreotide (1 µg/kg/hour). She was transfused with 40 ml of packed red blood cells and 20 ml of fresh frozen plasma before being transferred to King Chulalongkorn Memorial Hospital for further management.

At King Chulalongkorn Memorial Hospital, she was still alert, afebrile, mildly pale, with a heart rate of 115/min, respiratory rate of 64/min and blood pressure of 84/60 mmHg. Physical examination revealed a normal appearance of infant with cutis marmorata telangiectatica congenita at her right lower leg. The rest of physical examination was unremarkable. Intravenous ranitidine and octreotide infusion were continued. An Apt-Downey alkali denaturation test of gastric aspirate revealed the baby’s own blood. Blood cultures were done. The cerebrospinal fluid analysis was normal. A complete blood analysis showed a hematocrit of 49 percent, white blood cell counts of 13,770/mm³ (66% neutrophils, 24% lymphocytes, 5% monocytes, and 5% eosinophils), and platelet counts of 85,000/mm³. Prothrombin and partial thromboplastin times were 14.4 (control 10.6 sec, INR 1.2) and 32.1 sec (control 29.7 sec), respectively. Chest radiography was unremarkable. Upper gastroduodenal endoscopy was performed and revealed diffuse gastric erosions without evidence of gastric hemangioma. The esophagus and duodenum appeared normal. Biopsy of stomach revealed acute erosive hemorrhagic gastritis with scattered neutrophilic infiltration in the gastric mucosa (Figure).

On the third day of admission, blood cultures grew GBS. The infant was treated with intravenous ampicillin plus gentamicin for 14 days. Her clinical status gradually improved without further bleeding, and protein hydrolysate formula was fed to avoid cow’s milk protein in case it might have induced hemorrhagic gastritis. The mother’s vaginal swab culture was negative for GBS. On the fifth day of admission, the stool was negative for occult blood. At 15 days of age, she was feeding very well and was discharged home. Oral ranitidine was continued for 8 weeks. At 3 months of age, she was growing well and admitted to the hospital for cow’s milk challenge. There was no reaction to cow’s milk protein, so she started on a regular cow’s milk diet. She has remained well and has shown no recurrence of symptoms during the following 6 months.

**DISCUSSION**

GBS is a major causative agent of perinatal bacterial infection. Invasive disease of neonates is classified into 2 entities based on time of onset after birth. Early-onset disease frequently occurs within the first 24 hours of life (range, 0 to 6 days) and the clinical manifestations are respiratory distress, pneumonia, apnea, shock and less often meningitis. Symptoms such as irritability and hyperthermia are often noted in term rather than preterm infants, and asymptomatic bacteremia occurs almost exclusively in term infants.
Neonates at risk of infection are preterm infants born at less than 37 weeks of gestation, infants born after the amniotic membranes have been ruptured for more than 18 hours and infants born to mother with a high GBS inoculum, intrapartum fever, GBS bacteriuria and chorioamnionitis. Payne et al identified 6 features predicting fatal outcome in early-onset disease including birth weight less than 2,500 grams, apnea, hypotension, absolute neutrophil counts less than 1,500/mm³, initial blood pH less than 7.25, and pleural effusion shown on the chest radiography. Late-onset disease, which typically occurs about 4 weeks of age (range, 1 week to 3 months), is characterized by occult bacteremia and meningitis.

In our report, the infant had profuse upper gastrointestinal bleeding with shock in the first 48 hours of life. Endoscopy revealed diffuse hemorrhagic gastritis. GBS bacteremia was present, and therefore it is possible that the stomach might be the primary site of GBS bacteremia probably due to aspiration or ingestion of contaminated amniotic fluid or vaginal secretions during delivery: because neutrophilic infiltration was observed in the gastric mcosa. It would be nice if Gram’s staining and bacterial culture of the gastric biopsy specimen were performed to show the presence of GBS. Weisman et al have shown GBS can cause systemic infection after oral inoculation of GBS in the suckling rats during the first four days of life with varying doses and strains of GBS. Autopsy confirmed sepsis, intestinal colonization, meningitis and pneumonia.

Cow’s milk protein allergy (CMPA) is not uncommon and may cause upper gastrointestinal bleeding in babies less than one year of age. It is less likely that the patient had CMPA because it is too early to develop gastritis within 24 hours of feeding of cow’s milk. However cow’s milk challenge test should be done to exclude CMPA in this patient.

In conclusion, we present a full-term female neonate who presented with profuse upper gastrointestinal bleeding due to acute hemorrhagic gastritis in the first 48 hours of life, and had occult GBS bacteremia.
ACKNOWLEDGEMENT

We extend special thanks to Associate Professor Pongpun Nunthapisud and National Streptococcal Reference Center of Thailand for providing of group B streptococcal data.

References