Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

updates may be found by visiting the CDC website (1).

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Case Report

Necrotizing Enterocolitis in an Infant with Group B Streptococcal Sepsis

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Necrotizing enterocolitis (NEC) is an entity that mainly affects premature infants but has been described occasionally in full-term neonates (1). The etiologic roles of the infectious agents in this condition are not yet well established, although NEC has been reported in neonates during the course of bacterial, viral, and mycotic infections (2). In this paper, we describe a full-term neonate who developed NEC in the course of sepsis caused by *Streptococcus agalactiae*. To our knowledge, NEC has not been previously reported in association with this microorganism.

Case Report

A female infant weighing 3,300 g was born by forceps delivery after an uneventful 36-week gestation. Amniorrhexis occurred intrapartum and the amniotic fluid was clear. Apgar scores were 6 and 7 at 1 and 5 min, respectively. The infant had respiratory distress that started at birth, and she was transferred to our hospital at 12 h of age. Examination on admission showed an ill-appearing, cyanotic, and poorly perfused newborn who had a blood pressure of 53/36 mm Hg, a pulse of 195 beats/min, and respiratory rate of 84/min. The rectal temperature was 38°C. The infant had respiratory distress and auscultation revealed bilateral hypoventilation and tachycardia. The liver was palpated 4 cm below the right costal margin. She was hypotonic and hypoactive. The anterior fontanelle was flat and there were no meningeal signs.

Laboratory investigation included leukocyte count of 3,100/mm^3 with 38% neutrophils, 6% band forms, and 56% lymphocytes. Hemoglobin was 16.7 g/dl; hematocrit, 47.8%; platelet count, 197,000/mm^3; Quick test, 44%; and partial thromboplastin time, 68 sec (control 30 sec). Blood urea was 43 mg/dl and creatinine, 1 mg/dl. Arterial blood gases showed severe hypoxemia, hypercapnia, and mixed acidosis. The urine antigen detection test was positive for group B *Streptococcus* antigen. A blood culture grew group B *Streptococcus*. The cerebrospinal fluid analysis was normal and its culture was sterile. Culture of a vaginal swab from the mother yielded group B *Streptococcus*. A chest radiograph revealed a micronodular bilateral alveolar pattern.

Bacterial sepsis was initially suspected and the patient was treated with ampicillin, gentamicin, dopamine, dobutamine, plasma therapy, and mechanical ventilation. Umbilical artery and vein catheterizations were performed. Her condition initially improved but deteriorated again on day 3, and she developed a marked abdominal distention with signs of paralytic ileus. An X ray examination of the abdomen at this time showed a large pneumoperitoneum. After her thrombocytopenia was corrected, a surgical intervention was carried out. At laparotomy, fecal material was found in the abdominal cavity and she had 40 cm of necrotic small bowel. Removal of this loop and a proximal end to distal side anastomosis were performed. The surgical pathology findings showed a congested small bowel with edema, hemorrhage, and ischemic necrosis and wall perforation, compatible with necrotizing enterocolitis. The patient had a complicated postoperative course with dehiscence of the sutures and outward fistulation. On day 22, she underwent surgery again and resection of another 20 cm of necrotic small bowel was required. After this procedure, the patient had a favorable course with mild symptoms of short bowel syndrome.

Comment

NEC mainly occurs in premature infants but has occasionally been reported in full-term neonates. In most of these cases, predisposing factors are usually present. Although our understanding of the pathogenesis of NEC remains incomplete, the disease seems to have a multifactorial basis (3-5). A role of infectious agents as factors or cofactors in the development of some cases of NEC has been proposed. The list of pathogens responsible for NEC has included bacteria (*Escherichia coli*, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, *Enterobacter*, *Salmonella*, and *Clostridium* species; coagulase-negative staphylococci, *Staphylococcus aureus*; *Bordetella pertussis*), viruses (coxsackie B2, coronavirus, rotavirus, echovirus type 22, cytomegalovirus), and fungi (*Candida* spp. including *albicans*, and *glabrata*) (2).

Although 20 to 30% of patients with NEC may have an associated bacteremia, most likely this represents bacterial translocation through a previously injured intestinal mucosa rather than the primary initiating event (1). The

speculation that a bacterial species can be the primary etiologic factor in the development of NEC, with secondary bacterial translocation from the bowel lumen resulting in septicemia, has not been proven.

The patient that we describe had sepsis and pneumonia caused by group B *Streptococcus* and had two main factors for the development of NEC: septic shock and umbilical catheterization. Both factors reproduce the ischemic-hypoxic state that decreases the mesenteric blood flow and ultimately causes a hypoxic damage to the enteric mucosa. We have not seen previous mention in the English literature of the association of *S. agalactiae* with NEC; therefore, this report adds a new infectious agent to the list of those associated with this devastating gastrointestinal entity.

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