A term gestation male infant presented at 48 hours of life with a history of delayed meconium passage and bilious emesis. He was delivered by spontaneous vaginal delivery following an uncomplicated pregnancy. A soft, systolic ejection murmur was noted during the initial physical examination. Chest radiograph, arterial blood gas levels, electrocardiogram, and 4 extremity blood pressures were normal, and the murmur resolved on the second day of life. The patient's medical history was otherwise unremarkable. Results of a physical examination disclosed a slightly distended abdomen and hyperactive bowel sounds. Rectal examination revealed a patent anus with normal sphincter tone. Serum electrolyte and glucose levels were normal. The infant’s white blood cell count was 18 x10⁹/L, with 0.03 bands, 0.67 segmented neutrophils, and 0.30 lymphocytes on differential cell count. Abdominal radiographs were obtained (Figure 1 and Figure 2). The patient was treated with intravenous fluids, broad-spectrum systemic antibiotics, and bowel decompression, and was discharged from the hospital at 2 weeks of age with no complications.

From the Department of Pediatrics, United States Naval Hospital, Guam.
Necrotizing Enterocolitis in a Term Infant

Vomiting in the initial days of life is suggestive of an obstruction of the gastrointestinal tract, but it also may represent a nonspecific symptom of sepsis. Bile-stained vomitus in the newborn is a surgical emergency and should be considered a sign of an intestinal obstruction beyond the second portion of the duodenum until proven otherwise. Intestinal obstruction occurs in approximately 1 of every 1000 newborns, with signs of vomiting, abdominal distension, and delayed passage of meconium. Abdominal radiographs (supine and cross-table lateral views) are obtained to detect air-fluid levels, distended loops of bowel, pneumoperitoneum, characteristic patterns of obstruction, or pneumatosis intestinalis, indicating necrotizing enterocolitis.

Necrotizing enterocolitis represents the most common gastrointestinal emergency seen in the neonatal intensive care unit, and affects almost 2000 neonates in the United States each year.1 The etiology for necrotizing enterocolitis is multifactorial, and no universal theory of pathogenesis has been accepted. Risk factors include birth asphyxia, shock, hyaline membrane disease, apnea, presence of a patent ductus arteriosus, and use of umbilical artery and vein catheters.2 However, there is no difference in the incidence of these risk factors between infants with necrotizing enterocolitis compared with infants of similar birth weight and gestation without necrotizing enterocolitis.3-5

The disease has diverse clinical manifestations, ranging from a relatively mild course characterized by feeding intolerance, abdominal distension, hematochezia, and vomiting to a more fulminant course characterized by intestinal necrosis, perforation, sepsis, and death. Newborns may initially display abdominal distension and marked gastric residual fluid or vomiting. Occultly positive for blood or grossly bloody stools may be present. Frequently, affected infants have nonspecific signs and symptoms of sepsis. The initial findings from the physical examination are nonspecific, but as the disease progresses, the abdomen becomes firm and discolored, and visible loops of bowel are seen.

Necrotizing enterocolitis usually affects premature infants. In a review of 123 affected infants, the mean gestational age was 31 weeks, with an average birth weight of 1460 g. Only 7.3% of the reported patients were term births.6 In another review, however, 13 (20%) of the 64 infants were term births. While the histopathologic observations do not differ between term and preterm infants, the classic radiographic features are encountered less frequently in term infants.7 Other investigators have suggested that infants with symptoms of necrotizing enterocolitis during the first 1 to 2 days of life are larger weight, more mature, and less frequently asphyxiated than infants with the onset of necrotizing enterocolitis presenting later.8

The treatment of necrotizing enterocolitis, in the absence of signs of intestinal necrosis or perforation, is medical and includes nasogastric decompression and institution of broad-spectrum systemic antibiotic therapy. Serial abdominal radiographs are obtained to document progression or resolution of the disease. Early surgical consultation should be obtained. Intestinal perforation or disease refractory to medical management requires surgical intervention.9

Necrotizing enterocolitis is a serious and potentially life-threatening disease that affects primarily preterm infants. The pathogenesis of this disorder appears to be multifactorial, and term infants may suffer from a histopathologically and etiologically similar disease, although term infants are more likely to present with these symptoms within the first several days of life. Term infants are less likely to exhibit the classic radiologic manifestations of this disease, and thus a high index of suspicion is required to ensure timely intervention.

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