

### **Necrotizing enterocolitis**

Clinical presentation & general epidemiology: Necrotizing enterocolitis is acute inflammatory injury of distal small and often proximal large intestine(1). It is the most common and most serious gastrointestinal emergency of neonates. It is characterized by ischemic necrosis of intestinal mucosa, associated with inflammation and invasion of enteric gas-forming organisms. The incidence is inversely proportional to gestational age, with 90% occurring in premature infants. NEC usually develops 2-3 days following birth, with 90% developing within first 10 days of life(2). It occurs between 1% and 5% of all NICU admissions, and 5% to 10 % of very low birth weight infants. NEC is responsible for 12% deaths in extreme premature infants and accounts for substantial long term morbidity in survivors of neonatal intensive care.

Both ischemic and infectious risk factors with contributory conditions like immature immune system have been implicated in pathogenesis of NEC. Low birth weight, prematurity, formula feeding and intestinal dysbiosis are primary risk factors. If bowel wall injury progresses, then mural congestion, edema, transmural necrosis, and perforation of bowel may occur. If healed, these may result in fibrosis, adhesions, and strictures. The terminal ileum is the most commonly involved location.

The infant presents with poor feeding, lethargy, and temperature instability. There may be signs of respiratory distress, apnea, bradycardia, hypotension, oliguria, and bleeding diathesis. There is gradual abdominal distension, tenderness, and features of ileus. Vomiting (of bile, blood or both) and hematochezia may occur. Abdominal wall may demonstrate erythema or induration, and there may be features of ascites.

Based on the systemic, abdominal, and radiographic signs, necrotizing enterocolitis has been staged under "Bell's Criteria"(3).

Imaging features: Supine abdominal radiograph is the modality of choice to assess bowel gas pattern(4). Additional cross-table lateral or left-lateral decubitus views also help in further investigation, particularly to assess for pneumoperitoneum. An abnormal gas pattern with dilated bowel loops, often asymmetric in distribution is seen early on in the disease course (Bell stages IA and IB). Normal polygonal intraluminal gas shapes may be lost. Bowel wall edema may be noted with "thumbprinting". Pneumatosis intestinalis, the hallmark of NEC, appears as bubbles of gas within bowel wall (Bell stages II and III). Portal venous gas may be noted, which may be a precursor for more advanced disease. Pneumoperitoneum appears when bowel perforation occurs (Bell stage IIIB). It may be seen as air on both sides of the bowel (Rigler sign) or air outlining falciform ligament (football sign).

Abdominal ultrasonography reveals bowel wall thickening. Central echogenic focus and hypoechoic rim (pseudo-kidney sign) may indicate necrotic bowel and imminent perforation. Bowel wall may be hypervascular (viable but engorged in early stage) or hypovascular (infarcted in a later stage). Abdominal ultrasound can also detect portal venous gas. Intra-abdominal free fluid, especially with echogenic debris, suggests perforation.

Prognosis, treatment or therapeutic options: Medical management consists of supportive care, antibiotic therapy and close monitoring. When NEC extends through the bowel wall resulting in perforation, exploratory laparotomy is performed with resection of affected section.