

Intensive Care Nursery House Staff Manual

Necrotizing Enterocolitis (NEC)

INTRODUCTION: NEC, the most common acquired acute gastro-intestinal illness in the neonatal period, affects about 5% of infants with birthweight $\leq 1,500$ g and typically is characterized by **abdominal distension, bloody stools** and **pneumatosis intestinalis**.

The actual spectrum of illness ranges from mild cases of feeding intolerance and abdominal distension to severe cases characterized by intestinal necrosis, perforation, and septic shock. Fulminant cases may progress from minimal symptoms to peritonitis and death within 12h. Although NEC usually presents in preterm infants with risk factors and who have been fed, it also occurs in term infants and infants who have never received enteral feedings. NEC most commonly involves terminal ileum and colon, although in severe cases the entire small and large bowel may be affected.

ETIOLOGY is not definitely known and is probably **multifactorial**. Suggested etiologies include **immaturity of intestinal mucosa, intestinal ischemia/reperfusion injury, infection, and immature immune response**. There is probably a common, final pathway involving endogenous production of inflammatory mediators (*e.g.*, PAF, TNF, cytokines) that precipitate intestinal injury.

RISK FACTORS include:

- Prematurity (>95% of cases)
- Aggressive advance (volume and strength) of enteral feedings in preterm infants
- Hyperosmolar formulas
- Bacterial colonization or overgrowth (predominantly with *E. coli*, *Klebsiella*, *Enterobacter*, *C. difficile*): may be inciting event or a permissive factor.
- Polycythemia
- Patent ductus arteriosus (PDA, decreased systemic output due to left→right shunt)
- Indomethacin (decreased intestinal perfusion through inhibition of cyclo-oxygenase)
- Steroids, when given in conjunction with indomethacin
- Umbilical arterial catheter (UAC) with tip at or above inferior mesenteric artery
- Umbilical venous catheter (UVC) with tip in portal system (especially with exchange transfusion)
- Cocaine exposure *in utero*
- Respiratory Distress Syndrome

PRESENTATION:

A. **Clinical Findings** include any of the following:

- Abdominal distension
- Abdominal tenderness or redness
- Feeding residuals, often bilious
- Absent bowel sounds
- Gross or occult blood in stool
- Bluish discoloration of abdominal wall
- Non-specific signs (temperature instability, glucose instability, lethargy, apnea/bradycardia, hypotension)

B. Radiographic Findings (in order of severity):

- Non-specific bowel dilatation
- Thickening of bowel wall
- Fixed, dilated loop (unchanged on >1 radiograph)
- Pneumatosis intestinalis (small gas bubbles in bowel wall, almost always associated with dilated bowel loops)
- Portal venous gas
- Free intraperitoneal gas (indicative of intestinal perforation)

C. Laboratory Findings include:

- Thrombocytopenia
- Abnormally ↑ or ↓ WBC
- Neutropenia
- Metabolic acidosis (poor prognostic sign)
- Left shift of WBC (toward immature precursors)
- Evidence of DIC

STAGING of NEC by Bell's Criteria (DIAGNOSIS):

Stage 1. Suspected NEC: gastric residuals, abdominal distension, occult or gross blood in stool, x-ray normal to mild distension, temperature instability, apnea, bradycardia

Stage 2. Definite NEC: mild to moderate systemic illness, absent bowel sounds, abdominal tenderness, pneumatosis intestinalis or portal venous gas, metabolic acidosis, ↓ platelets

Stage 3. Advanced NEC: severely ill, marked distension, signs of peritonitis, hypotension, metabolic & respiratory acidosis, DIC, pneumoperitoneum if bowel perforation present

MANAGEMENT: With any feeding intolerance, maintain high level of suspicion for NEC, especially with preterm infants.

A. Suspected NEC:

- Make patient NPO
- Obtain baseline KUB
- Test all stools for occult blood
- CBC, platelets & blood culture
- Culture urine & CSF if systemic signs
- R/O surgical cause of distension
- If improvement occurs, consider cautious feeding in 3d
- Start maintenance IV fluids
- Perform serial abdominal examinations
- Consider gastric decompression
- Consider starting antibiotics
- Consider stool culture
- Observe closely for worsening

B. Definite/Advanced NEC:

- Obtain consult with Pediatric Surgery.
- NPO for at least 7-10d
- IV fluids: Because of “**third spacing**,” patient may require fluid resuscitation to improve bowel perfusion (*e.g.*, D5-Lactated Ringer's at 150 mL/kg per 24 h).
- Follow urine output closely; renal failure is common due to hypoperfusion.
- Gastric decompression (Replogle tube to low, continuous suction)
- Abdominal radiographs (AP & cross table lateral q6-8h) to look for perforation
- Endotracheal intubation and assisted ventilation as needed
- Circulatory support: Monitor arterial blood pressure and maintain in normal range with volume expanders and dopamine (Dobutamine is less effective in infants and may actually cause hypotension).
- Blood culture and start antibiotics: ampicillin & gentamicin for 7-10d (Anaerobic

- coverage is usually not necessary unless infant is several weeks old).
- Follow CBC, platelets, PT, PTT, fibrinogen; replace clotting factors products prn.
- Frequent measurements of arterial pH and blood gas tensions
- Correct metabolic acidosis.
- Frequent measurements of electrolytes; watch for hyperkalemia.

C. Surgical Considerations:

- Operative intervention is indicated for bowel perforation, evidence of necrotic bowel (fixed loop, metabolic acidosis, DIC, shock), or progressively worsening clinical condition despite intensive medical management
- A peritoneal drain may be inserted in extremely ill infants to delay or avoid laparotomy.
- If NEC develops in a baby with PDA, begin medical management and **consider urgent operative closure of PDA. Do not give indomethacin to an infant with suspected or definite NEC.**

OUTCOME:

- Mortality rate is 20-30% depending on severity of illness and amount of bowel removed.
- Complications include:
 - Intestinal stricture with bowel obstruction
 - Short bowel syndrome
 - Cholestasis, if prolonged dependence on TPN

PREVENTIVE MEASURES:

- Intestinal priming (gut stimulation feedings): dilute, low volume feedings to stimulate GI mucosal development
- Advance feedings slowly in small preterm infants (see section on Feeding of Preterm Infants, P. 50).
- Do not advance feedings if there are gastric residuals, **especially if bile stained**
- Fresh human milk appears to be protective against NEC.
- Do not feed infants with PDA, UAC, or UVC.
- Do not give enteral feedings during and for 48-72h after indomethacin.
- Minimize antibiotic use, as they alter intestinal flora and select for resistant species.
- Epidemiologic controls: cohorting of multiple cases (possible infectious cause)
- Antenatal glucocorticoids for lung maturation also accelerate intestinal maturation.
- Suggested possible future approaches: enteral IgG/IgA, formula acidification, anaerobic bacterial supplementation (bifidobacteria)