Case Report

Necrotizing enterocolitis: a case report

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ABSTRACT

Necrotizing enterocolitis (NEC), a disease predominant in the premature formula fed infants, is a major cause of morbidity and mortality in NICU survivors. The symptoms may vary from apnea, fever, lethargy to abdominal distension, bloody stools, poor feeding and vomiting. The mainstay of treatment is the IV feeds, discontinuation of oral feeds, nasogastric (NG) decompression, possible breathing support and surgery. The objective of this case report is to discuss the presentation, treatment, prognosis and proposed preventative measures of NEC, which can help raise awareness and henceforth improve the management and subsequent prognosis of this disease. Authors present to you the case report of a VLBW (Very Low Birth Weight) premature infant with NEC.

Keywords: Complications, Management, Necrotizing enterocolitis, Prognosis, Symptoms

INTRODUCTION

Necrotizing enterocolitis (NEC), which typically occurs in the 2nd-3rd week of life in premature, formula fed infants is characterized by variable damage to the intestinal tract, ranging from mucosal injury to full thickness necrosis and perforation. NEC affects close to 10% infants who weigh less than 1500 grams, with mortality rates of 50% or more depending on the severity, but may also occur in term and near term babies.1

Infectious agents, hypoxia and ischemia have an important role in the etiology of the disease with lesions of the intestinal mucosa triggering inflammatory reaction and necrosis, with enteral nutrition being an extremely relevant factor for the contribution of disease development.2

NEC is categorised with the help of Bell Stages Stage I (Suspected NEC)- Includes non specific systemic signs of mild intensity, discrete intestinal signs such as mild abdominal distension and an elevation of stomach residue. Stage II (Defined NEC) Signs and symptoms of Stage I along with abdominal discomfort and absence of airborne noises. Intestinal pneumatosis and total intestinal obstruction may be present.

Stage III (Advanced, severe NEC) Signs and symptoms of Stage I and II and associated bradycardia, hypotension, severe metabolic acidosis, coagulopathy, respiratory failure, neutropenia. Perforation and signs of generalised peritonitis may be present.2

Early recognition, bowel rest, antimicrobial therapy, periodic radiologic evaluation, supportive therapy of fluids, electrolytes and nutrition are the mainstays of the management.3

CASE REPORT

A preterm infant, born at 28 6/7 weeks of gestation and weighing 1200 grams (VLBW), was brought to the paediatric emergency on the 2nd day of her life with the chief complaints of respiratory distress and intermittent grunting. Enteral feed was initiated with the breast milk on the 1st day. The mother reported the leakage of clear
fluid per vagina for 2 days prior to the onset of labour (likely Prolonged preterm rupture of membranes) without any symptoms consistent with chorioamnionitis.

On physical examination, the child appeared sick, with visible mild subcostal and intercostal retractions. The abdomen was distended and tense on inspection and palpation respectively (Figure 1). On neurologic evaluation, the tone was normal, activity: decreased, moro reflex: incomplete and rooting and sucking reflexes: poor. Anterior fontanel was open and anus was patent. The genitalia appeared normal and no gross congenital malformations were seen.

The symptoms often include swelling/bloating/distension of abdomen, bloody stool, diarrhea, poor feeding and vomiting. Other symptoms of an infection such as apnea, fever, lethargy may be present. NEC occurs in 1-5% of all neonatal intensive care admissions and 5-10% of all VLBW (<1500 grams) infants. Historically it was believed that NEC arose predominantly from ischemic injury to the immature GI tract, yet other contributing factors such as introduction of enteric feeding, alterations in the normal bacterial colonization of the GI tract, bacterial translocation and activation of the cytokine cascade, decreased epidermal growth factors, increased platelet activating factors, and mucosal damage from free radical production, have been implicated. 

Radiographic signs may include ileus, dilated or fixed intestinal loops, air in the intestinal wall or free air in the abdomen. Complications of NEC may include peritonitis, sepsis, intestinal perforation, intestinal strictures, liver problems (from prolonged inability to tolerate enteral feeds and needs for parenteral nutrition), short bowel syndrome (if a large amount of intestine is lost). Early, aggressive treatment can help improve the outcome. The treatment of NEC mainly includes antibiotics, checking bowel movements, stopping regular feedings by mouth and instead feeding through an IV, regular X Rays to monitor the progression of disease, NG decompression, regular blood work to monitor for infection, possible breathing support (depending upon severity), and in severe cases, a surgery to remove the diseased part of the intestine.

**CONCLUSION**

Babies at higher risk for NEC include premature infants, formula fed infants or who have received blood exchange transfusion.
Several recommendations for preventative strategies based on the proposed mechanisms of NEC. There have been in vitro and animal studies as well as human clinical trials investigating the benefits of probiotics, however larger trials are necessary prior to recommending routine use in preterm infants.³

There are recommendations for early initiation of enteral feeds and advancement of enteral feeds in accordance with evidence based feeding protocols, promoting adequate intestinal motility and prevention of carbohydrate malabsorption. The use of human milk is preferred over the use of formula, allowing for passage of maternal immunoglobulins to the infant and promotion of healthy intestinal flora. There is support for the judicious use of antimicrobial therapy to minimize unnecessary exposure, risk of necrotizing enterocolitis, increased bacterial antibiotic resistance and potential alteration of intestinal flora with selection for alternative bacterial strains including fast fermentors.³

NEC continues to be a significant cause of mortality and morbidity in NICU survivors; avoidance of predisposing factors, early recognition, aggressive treatment and further investigations into potential therapies and preventative measures are needed.³

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REFERENCES


