Necrotising Enterocolitis and Nutrition

Abstract

The incidence of Necrotising enterocolitis (NEC) varies widely but occurs in approximately 1 in 1000 live births and up to 10% of Extremely Low Birth Weight Infants. Mortality is high around 20-30% but highest in preterm infants and in those requiring surgery. There is an association with feeds and bacteria in the pathogenesis as well as bacterial toxins. Formula milk is associated with a higher incidence of NEC than those infants receiving human milk or a mixture of both. There is no benefit in delaying the introduction of enteral feeds with breast milk but the rate of increase remains an area for research and discussion. An adopted standardized regime does appear to be protective. Further improvements in the prevention, diagnosis and treatment of NEC are still required.

Keywords: Necrotising enterocolitis; Prebiotics; Probiotics; Breast milk

Case Report

The incidence of Necrotising enterocolitis (NEC) varies widely but occurs in approximately 1 in 1000 live births and up to 10% of Extremely Low Birth Weight Infants [1]. Mortality is high around 20-30% but highest in preterm infants and in those requiring surgery. The common site for NEC to occur is the terminal ileum, caecum and ascending colon but it can occur anywhere in gastrointestinal tract. The process is essentially inflammatory becoming transmural associated with ulceration, necrosis, oedema, haemorrhage and potential perforation. There is an association with feeds and bacteria in the pathogenesis as well as bacterial toxins but no single consistent organism has been implicated.

Clinical presentation

Presentation varies from feed intolerance, increased aspirates, apnoea and bradycardia to abdominal distention, rectal bleeding and shock. X-ray findings can include fixed loops, pneumatosis intestinalis, portal gas and of course, perforation. Infants progressing to shock often need inotropic and ventilator support. There have been a number of classifications for NEC but ‘Bell’s Staging’ is commonly used to classify this disease (Table 1).

Milk and its constituents

Formula milk is associated with a higher incidence of NEC than those infants receiving human milk or a mixture of both [2]. There is no benefit in delaying the introduction of enteral feeds with breast milk but the rate of increase remains an area for research and discussion [3]. An adopted standardized regime does appear to be protective. Recommendations include starting feeds at any gestation within the first 1-2 days of life and advancing around 30mls/kg/day in infants <$1000g [4].

Prebiotics and Probiotics are advocated as being protective against NEC. Prebiotics are oligosaccharides promoting the growth of normal bacteria found in the colon but despite this, there is little evidence that they prevent or protect against NEC. Probiotics however can help colonise the gut with normal gut bacteria normalising the microbiome. Despite meta-analyses demonstrating a reduction in NEC there remain divisions over which strain or strains of bacteria to use or whether we should await further studies before universally recommending the routine use of prob. Many authors do, however, recommend their routine use and offer guides as to how to introduce them into practice [5].

The idea of supplementing feeds with protein and energy during illness seems plausible. Although some artificial milks and intravenous feeds have been modified to achieve a higher supplementation, long-chain polysaturated fatty acids (LCPUFAs), Glutamine and Arginine have failed to show any significant benefits with respect to NEC prevention.

Treatment

Traditional treatment includes resting the bowel, antibiotics specific to each individual units policy intensive care support and surgery when required. Fluid restriction is often undertaken but calorie content should not be reduced aiming for over 100kcal/kg/day. Parenteral intake includes proteins, carbohydrate and lipid itself comprising 30-40% of non-protein calories to allow healing and repair.

The length of time to rest the bowel remains controversial but ordinarily 7-14 days is usual with a guiding reduction in C reactive protein often seen in the biochemistry albeit not a very sensitive marker. The timing of surgery also remains uncertain and also whether to drain or undergo laparotomy. If perforation has occurred or necrotic bowel removed both requiring ileostomy formation then re-feeding may be even slower post-operatively as the inflammation subsides. Human milk is the ideal choice for re-feeding containing growth and immune factors not present in formula.

Strategies to reduce NEC

Preventing NEC is likely to be a better strategy than improving the treatment modalities. Clearly reducing the number of preterm infants, standardizing feeding guidelines and using breast milk exclusively has impacted on the incidence of NEC [6]. The components of Breast Milk include, Oligosaccharides, Probiotics, Lactoferrin, Alpha-Lactalbumin, Lysozyme, Fatty Acids and other factors. This has led to many studies suggesting the regular use of probiotics to help reduce NEC although each study differs greatly and uses many different bacterial species [7,8].
Table 1: Bells staging.

| Stage          | Systemic Signs                      | Intestinal Signs                                          | Radiology                      |
|----------------|------------------------------------|-----------------------------------------------------------|--------------------------------|
| 1A Suspected   | Apnoea, Bradycardia, Temp instability | Gastric residuals, mild distention, occult blood          | Normal or ileus                |
| 1B Suspected   | As Above                           | As Above plus fresh blood per rectum                     | Normal or ileus                |
| 11A NEC Mild   | As Above                           | Above plus absent / decreased bowel sounds, +/- tender    | Dilatation, ileus, pneumatosis |
| 11B NEC Moderate | As above mild metabolic acidosis and thrombocytopenia | Above plus abdominal tenderness, absent bowel sounds, +/- cellulitis | As 11A +/- portal vein gas, +/- ascites |
| 11A NEC Advanced | As 11B plus hypotension, severe apnoea, mixed acidosis, DIC, anuria, neutropoenia | Above plus signs of peritonitis, abdominal distention and abdominal wall erythema | As 11A with Ascites |
| 11A NEC Perforation | As 11A                          | As 11A                                                   | 11A with Ascites plus pneumoperitoneum |

Conclusion

Further improvements in the prevention, diagnosis and treatment of NEC are still required and hopefully will aid the reduction of this potentially catastrophic condition for the newborn infant at risk.

References

1. Berman L, Moss RL (2011) Necrotising enterocolitis: an update. Semin Fetal Neonatal Med 16(3): 145-150.
2. Sullivan S, Schaner RJ, Kim JH, Patel AL, Travoger R, et al. (2010) An exclusively human milk-based diet is associated with a lower rate of necrotizing enterocolitis than a diet of human milk and bovine milk-based products. J Pediatr 156(4): 562-567.
3. Morgan J, Young L, McGuire W (2014) Slow advancement of enteral feed volumes to prevent necrotizing enterocolitis in very low birth weight infants. Cochrane Database Syst Rev 12: CD001241.
4. Fallon EM, Nehra D, Potemkin AK, Gura KM, Simpser E, et al. (2012) ASPEN. Clinical Guidelines: nutritional support of neonatal patients at risk for necrotizing enterocolitis. J Parent Nutr 36(5): 506-523.
5. Deshpande G, Rao SC, Keil AD, Patole SK (2011) Evidence based guidelines for use of probiotics in preterm neonates. BMC Medicine 9: 92.
6. Pietz J, Babu A Lawrence L, Erin CS, Sudhir KM (2007) Prevention of necrotizing enterocolitis in preterm infants: a 20 year experience. Pediatrics 119(1): 164-170.
7. Srikanth V, Rao S, Patole S (2013) Prebiotic supplementation in preterm neonates: updated systematic review and meta-analysis of randomized controlled trials. Clin Nutr 32(6): 958-965.
8. Yang Y, Guo Y, Kan Q, Zhou XG, Zhou XY, et al. (2014) A meta-analysis of probiotics for preventing necrotizing enterocolitis in preterm neonates. Braz J Med Biol Res 47(9): 804-810.