Necrotizing Enterocolitis with Perforation in an Extreme Preterm, Extremely Low Birth Weight Neonate

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Abstract

Necrotizing enterocolitis (NEC) remains the most common and serious surgical condition among preterm, very low birth weight neonates with a mortality of 40% to 50% in those requiring surgery. We report perforation secondary to NEC in an extreme preterm, extremely low birth weight neonate with a good outcome due to aggressive surgical management and intensive care.

Key words: Necrotizing enterocolitis; Pneumoperitoneum; feeding; Surgical management

Introduction

Necrotizing enterocolitis (NEC) is a potential surgical emergency with the majority of cases occurring in very preterm (<32 wks), very low birth weight (< 1500 g) neonates¹. The presentation can be insidious over a few days with abdominal distension, altered gastric aspirates, feed intolerance along with systemic symptoms or it can present as a catastrophic deterioration with perforation and multiorgan dysfunction². The age at onset is around 11 – 12 days and is inversely related to the postmenstrual age at birth³. Current research is focussed on prevention and early recognition of NEC and the most promising approach seems to be the role of probiotics⁴. We report a case of NEC with perforation in an extreme preterm, extremely low birth weight (ELBW) neonate on day five after birth that was successfully managed with surgical resection and had a good outcome.

The Case

An extreme preterm 27 weeks, ELBW 880 g, female neonate was born by spontaneous vaginal delivery to a 23-year-old primigravida mother who had premature prolonged rupture of membranes (pPROM) for eight days and presented with antepartum haemorrhage three days prior to delivery. An antenatal ultrasound four weeks ago had shown severe oligohydramnios and placenta previa. She had not received any antenatal steroids (ANS) and delivered en-route from a peripheral hospital in the ambulance. The neonate was born severely asphyxiated and received bag and mask ventilation for three min and shifted to the neonatal intensive care unit. In view of early onset respiratory distress, nasal continuous positive airway pressure (CPAP) was begun and early rescue surfactant was administered. Intravenous (IV) 10% dextrose, 10% Aminoven and IV antibiotics

(Cefotaxime and Amikacin) were begun in view of maternal pPROM.

The neonate remained stable and minimal enteral nutrition (MEN) with expressed breastmilk was started on day two after birth. The respiratory distress settled and CPAP was weaned off by day fourafter birth. However abdominal distension with altered/bilious nasogastric aspirateswas noted by day five after birth. Over the next 24 hrs there were recurrent episodes of apnoea and increasing oxygen requirement and the neonate required mechanical ventilation. The neonate was kept nil per oral on continuous nasogastric aspiration and antibiotics were upgraded to Meropenem and Metronidazole. X ray abdomen revealed evidence of intestinal perforation (Figure 1) and the neonate was taken up for exploratory laparotomy. Per operatively, there was a jejunal perforation and a resection anastomosis was done. The neonate required mechanical ventilation for three days and was successfully extubated to nasal CPAP and weaned to room air by day 12 after birth. The postoperative course was complicated by culture positive late onset sepsis (E Coli and coagulase negative staphylococcus(CoNS) in blood culture, no meningitis) which was treated with two weeks of IV antibiotics and feed intolerance requiring prolonged parenteral nutrition and gradual increase in feed volume along with antireflux medications. At discharge, the infant weighed 1600 g and was on expressed breast milk along with human milk fortifier/vitamin D3/ Iron supplements and had normal retinopathy of prematurity (ROP), ultrasound skull and hearing screens.



Fig 1: X-ray abdomen erect view: Air under diaphragm suggestive of intestinal perforation

Discussion

Necrotizing enterocolitis, despite advances in neonatal care is still associated with a high incidence of morbidity and mortality around 20-40%⁵. The indications for surgery include evidence of perforation, as was seen in our index case and presence of necrotic bowel as evidenced by severe and persistent metabolic acidosis and/or thrombocytopenia and a lack of improvement with medical management⁵.

In our index case, the mother had not received ANS and the neonate had severe perinatal asphyxia at birth. There is still controversy with regard to initiation of feeds in such extremely preterm, ELBW neonates with the current evidence favouring MEN with human milk at the earliest sign of clinical stability^{6,7}. We had initiated expressed breast milk at 10 ml/kg after 24 hours as the neonate had passed meconium and was clinically and haemodynamically stable. The feeds were increased to 20 ml/kg after two days and the neonate became symptomatic by day five after birth. The neonate was managed aggressively with IV Fluids/parenteral nutrition, IV antibiotics and mechanical ventilation. As the x ray showed evidence of air under the diaphragm. an exploratory laparotomy was done immediately after stabilization. The neonate was successfully operated with resection of jejunal segment and primary reanastomosis after confirming the site of perforation in the jejunum. The post-operative period was marked by late onset sepsis, feed intolerance and the neonate required prolonged parenteral nutrition. There was however no neonatal cholestasis. The overall mortality for ELBW neonates that undergo surgery for NEC has been reported to be 40% to 50% despite intensive care^{8,9,10}.

Conclusion

This case highlights the need for close clinical monitoring for signs of NEC and early recognition of the need for surgery in such an extreme preterm, ELBW neonate. The prompt and successful surgical management and aggressive neonatal intensive care resulted in a favourable outcome. Early recognition and prompt surgical management of perforation in NEC along with neonatal intensive care is crucial for a good outcome.

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