Necrotising enterocolitis in older infants

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SUMMARY Thirteen children, ranging in age from 45 days to 2 years, had severe gastrointestinal illness with the features characteristic of neonatal necrotising enterocolitis. All 13 children had preceding gastroenteritis leading to hypovolaemia. Necrotising enterocolitis can occur in children beyond the neonatal age group and it may occur as a sequel to gastroenteritis.

Necrotising enterocolitis (NEC) is described as occurring in the stressed premature neonate or in one of low birthweight.¹ Bilious vomiting, abdominal distension, constipation followed by diarrhoea, and the passage of blood per rectum are the presenting features. X-ray film of the abdomen shows multiple fluid levels, intramural gas, pneumoperitoneum, or portal vein gas. Pathologically, dilated friable necrotic bowel can be seen and air bubbles may be visible in the bowel wall. The purpose of this paper is to show that this condition can occur in older infants and children.

Patients

During the last 2 years, in addition to the neonatal cases of NEC, 13 children aged between 45 days and 2 years have presented at this hospital with clinical, radiological, and pathological evidence of NEC (Table). Only 6 had a fully documented maternal history and these infants were not breast fed. The children initially presented with gastroenteritis—that is vomiting and diarrhoea for 3 to 14 days—and

all failed to respond to treatment for gastroenteritis. In 10 the vomiting persisted or recurred. One child had 'coffee ground' vomitus. After severe diarrhoea, 7 children had no bowel action for 24 to 48 hours. They then developed sudden abdominal distention and passed blood per rectum. Ten of the children suffered from severe hypovolaemic shock. On radiological examination, all the patients had pneumonitis intestinalis. Eight infants were treated conservatively, and 4 survived. Five patients were treated surgically, and only 1 survived.

Results of stool cultures from 6 children were: 2 negative, 2 *Escherichia coli*, 1 *Salmonella* sp., and 1 *Klebsiella* sp. Three of the 5 children who survived had disaccharide intolerance and the other 8 died before they could be examined. Blood culture was done in only one infant and this was negative.

Overall mortality was 62%. Two of them had other congenital anomalies: hypothyroidism (Case 10) and Down's syndrome (Case 3). The progress of the NEC was rapid and the duration between onset and death ranged from 6 hours to 10 days.

Retrospectively, it was clear that the patients

Table Analyses of cases

Case	Age (months)	Sex	Breast fed	Vomit	Duration of gastroenteritis (days)	Preceding constipation	Hypovolaemic shock	Blood per rectum	Laparotomy	Duration of NEC (days)	Outcome
1	1½	M	?	Yes*	4	?	+	+	—	?	Died
2	3	F	?	?	?	?	+	+	—	1	Died
3	24	F	—	Yes†	8	+	+	+	—	<1	Died
4	2	M	?	Yes	14	+	+	+	+2	10	Died
5	4	M	?	Yes	7	—	+	+	+2	4	Died
6	2	M	?	?	9	—	+	—	—	?	Died
7	2	M	—	Yes	7	+	+	—	+	<1	Died
8	3	F	?	?	7	—	+	+	—	3	Alive
9	5	F	—	Yes	4	+2	—	—	—	2	Alive
10	3	F	—	Yes†	3	+	—	—	+2	10	Alive
11	1½	F	—	Yes	10	+2	—	—	—	2	Alive
12	5	M	—	Yes	3	?	+	+	+	<1	Died
13	5	F	—	Yes	7	+	—	+	—	9	Alive

*Blood stained, †bile stained.

without severe hypovolaemic shock on admission had a good prognosis and infants with a good general condition survived either conservative or surgical treatment. The outcome of Case 10 exemplifies this.

Case 10. A 3-month-old girl was admitted with symptoms of intestinal obstruction after an attack of gastroenteritis. She was noted to have the features of cretinism, and later hypothyroidism was confirmed. Initial laparotomy showed a dilated terminal ileum which had a thinned out wall but no perforation. On the fifth postoperative day, she was started on oral feeding and found to suffer from lactose intolerance. X-ray film of the abdomen showed pneumonitis intestinalis on the eighth postoperative day (Fig. 1). Gentamicin was given intravenously with nasogastric aspiration and intravenous fluid. Two days later she suffered a wound dehiscence. At operation there were multiple perforations of the ileum. Resection and end-to-end anastomosis was done. After recovery, she was put on a lactose-free diet. Subsequent progress has been satisfactory.

Fig. 1 (Case 10.) Abdominal radiograph showing dilated intestinal loops with pneumonitis intestinalis.

Fig. 2 (Case 7.) Abdominal radiograph showing pronounced pneumonitis intestinalis with dilated loops of bowel and fluid levels.

In contrast, children with hypovolaemia and endotoxic shock, and admitted in a moribund condition due to prolonged severe gastroenteritis, had a sudden onset of NEC with a rapid progression of the disease process and a poor prognosis, as Case 7 shows.

Case 7. This 2-month-old boy had had two previous admissions for jaundice and failure to thrive, but there was no history of exchange transfusion or surgery. He developed very sudden and rapid abdominal distension after 7 days of gastroenteritis, and a rectal prolapse was noticed. An x-ray film of the abdomen showed a single hugely dilated loop of the bowel and minimal pneumonitis intestinalis. Fluid between the bowel loops was apparent (Fig. 2). The child developed severe respiratory distress because of his gross abdominal distension. Respiratory arrest occurred on the way to the operating

Fig. 3 (Case 7.) Low power view of resected terminal ileum after H and E stain showing ghost-like villi, the mucosa is necrotic and has partly sloughed off. There is extensive haemorrhagic infiltration and loss of glandular epithelium. Several submucosal gas-filled cysts can be seen.

theatre. After resuscitation, at laparotomy, several areas of the ileum had a yellowish, thinned-out intestinal wall. There was patchy necrosis present. Air bubbles could be seen inside the vessels and on dividing these, the air was seen to escape. Macroscopically, the mucosa showed patchy necrosis and haemorrhage, but no perforation. The features of the early stages of NEC were present. Histology showed coagulation necrosis, haemorrhage, and intramural gas. Intravascular gas was demonstrated (Fig. 3).

Discussion

There are only a few reports of NEC in older infants.¹⁻⁵ Polin classified term infants with NEC into 2 categories. One group developed the disease within the first 7 days of life and died, and the other group developed NEC at a mean age of 42 days. In the latter all had diarrhoea for at least 11 days before the development of NEC, or before death due to a complication of NEC.

In our series, there is a pronounced clustering of cases in the winter (November to March). All the children had a previous history of gastroenteritis of 3-14 days' duration. According to a report of the Ministry of Preventive Health in Kuwait, gastroenteritis is more prevalent in the winter season, and half of affected children are under 1 year of age. In our children with apparently normal bowel length, severe gastroenteritis induced hypovolaemic shock. Stein *et al.*⁶ reported 11 preterm babies who developed NEC in an epidemic of gastroenteritis and *Salmonella* sp. infection which occurred in a preterm baby ward. Our series lends support to the aetiological role of bacteria in the pathogenesis of NEC.

There appear to be three essential components in the development of neonatal NEC: (1) injury to the intestinal mucosa, (2) the presence of bacteria, and (3) the availability of a metabolic substrate—that is feedings.¹ These factors appear to apply to infants also.

Gastroenteritis resulting in hypovolaemia,^{7,8} and gastroenteritis causing lactose malabsorption with production of excess hydrogen^{9,10} appeared to influence the occurrence of NEC in our cases. Intramural gas in NEC shows a high hydrogen content.¹¹

Abdominal distension, vomiting, and bleeding per rectum were the three main symptoms. Onset was sudden with hypovolaemic shock, and all showed radiological signs characteristic of NEC: pneumatosis intestinalis, separation of dilated intestinal loops, and air in the radicles of the portal vein.¹²

In our series, 3 infants (Cases 4, 5, and 10) progressed to perforation of the bowel perhaps because of premature attempts at oral feeding and the use of suppositories.

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References

- Polin R A, Pollack P F, Barlow B, *et al.* Necrotizing enterocolitis in term infants. *J Pediatr* 1976; **89**: 460-2.
- Wilson S E, Woolley M M. Primary necrotizing enterocolitis in infants. *Arch Surg* 1969; **99**: 563-6.

- ³ Babiaba G Y. Ulcerative necrotising enterocolitis in a one-month ten day old infant (in Russian). *Vopr Okhr Materin Det* 1977; **22**: No 8, 86.
- ⁴ Ramenofsky M L. Necrotizing enterocolitis occurring in an infant three months of age. *J Pediatr Surg* 1977; **12**: 597-9.
- ⁵ Hofmann S, Baumann H, Emmrich P, Schreiber E. Surgical problems in necrotizing enterocolitis in childhood. *Prog Pediatr Surg* 1978; **11**: 87-96.
- ⁶ Stein H, Beck J, Solomon A, Schmaman A. Gastro-enteritis with necrotising enterocolitis in premature babies. *Br Med J* 1972; **ii**: 616-9.
- ⁷ Lloyd J R. The etiology of gastrointestinal perforations in the newborn. *J Pediatr Surg* 1969; **4**: 77-84.
- ⁸ Avey G S, Villacivencio O, Lilly J R, Randolph J G. Intractable diarrhea in early infancy. *Pediatrics* 1968; **41**: 712-22.
- ⁹ Ingelfinger F J. Malabsorption, the clinical background. *Fed Proc* 1967; **26**: 1388-90.
- ¹⁰ Douwes A C, Fernandes J, Degenhart H J. Improved accuracy of lactose tolerance test in children, using expired H₂ measurement. *Arch Dis Child* 1978; **53**: 939-42.
- ¹¹ Engel R R, Vernig N L, Hunt C E, Levitt M D. Origin of mural gas in necrotizing enterocolitis (abstract). *Pediatr Res* 1973; **7**: 292.
- ¹² Leonidas J C, Krasna I H, Fox H A, Broder M S. Peritoneal fluid in necrotizing enterocolitis: a radiologic sign of clinical deterioration. *J Pediatr* 1973; **82**: 672-5.

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