Necrotising enterocolitis in older infants

KAZUE TAKAYANAGI AND LEELA KAPILA
Paediatric Surgical Unit, Al-Sabah Hospital, Kuwait

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. 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 distension 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 2 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

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (months)</th>
<th>Sex</th>
<th>Breast fed</th>
<th>Vomit</th>
<th>Duration of gastroenteritis (days)</th>
<th>Preceding constipation</th>
<th>Hypovolaemic shock</th>
<th>Blood per rectum</th>
<th>Laparotomy</th>
<th>Duration of NEC (days)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1½</td>
<td>M</td>
<td>?</td>
<td>Yes*</td>
<td>4</td>
<td>?</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>Died</td>
</tr>
<tr>
<td>3</td>
<td>2 1/4</td>
<td>F</td>
<td>?</td>
<td>Yes†</td>
<td>8</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>&lt;1</td>
<td>Died</td>
</tr>
<tr>
<td>4</td>
<td>2</td>
<td>M</td>
<td>?</td>
<td>Yes</td>
<td>14</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+2</td>
<td>10</td>
<td>Died</td>
</tr>
<tr>
<td>5</td>
<td>4</td>
<td>M</td>
<td>?</td>
<td>Yes</td>
<td>7</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>4</td>
<td>Died</td>
</tr>
<tr>
<td>6</td>
<td>2</td>
<td>M</td>
<td>?</td>
<td>?</td>
<td>9</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Died</td>
</tr>
<tr>
<td>7</td>
<td>2</td>
<td>M</td>
<td>-</td>
<td>Yes</td>
<td>7</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>&lt;1</td>
<td>Died</td>
</tr>
<tr>
<td>8</td>
<td>3</td>
<td>F</td>
<td>?</td>
<td>?</td>
<td>7</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>3</td>
<td>Alive</td>
</tr>
<tr>
<td>9</td>
<td>3</td>
<td>F</td>
<td>-</td>
<td>Yes</td>
<td>4</td>
<td>+2</td>
<td>-</td>
<td>-</td>
<td>-2</td>
<td>10</td>
<td>Alive</td>
</tr>
<tr>
<td>10</td>
<td>3</td>
<td>F</td>
<td>-</td>
<td>Yes†</td>
<td>4</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+2</td>
<td>2</td>
<td>Alive</td>
</tr>
<tr>
<td>11</td>
<td>1½</td>
<td>F</td>
<td>-</td>
<td>Yes</td>
<td>10</td>
<td>+2</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>2</td>
<td>Alive</td>
</tr>
<tr>
<td>12</td>
<td>5</td>
<td>M</td>
<td>-</td>
<td>Yes</td>
<td>3</td>
<td>?</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>&lt;1</td>
<td>Died</td>
</tr>
<tr>
<td>13</td>
<td>5</td>
<td>F</td>
<td>-</td>
<td>Yes</td>
<td>7</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>9</td>
<td>Alive</td>
</tr>
</tbody>
</table>

*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.

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
necrotic and has partly sloughed off. There is extensive haemorrhagic infiltration and loss of glandular epithelium. Several submucosal gas-filled cysts can be seen.

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 hydrogen9,10 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: pneumonitis 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 prematurity attempts at oral feeding and the use of suppositories.

We thank Dr M Al Bader, Dr M Issa, and Dr N Fernando for allowing us to study their patients, Dr Judith Mohachi for assistance with the histological diagnosis and slides, Mr N V Freeman for the photographs, and Mrs A Bailey for secretarial assistance.

References

Necrotising enterocolitis in older infants


Correspondence to Dr Kazue Takayanagi, PO Box 2464, Safat, Kuwait, Arabian Gulf.

Received 2 April 1980

The following articles will appear in future issues of this journal:

Steroid-responsive nephrotic syndrome and allergy S R Meadow, J K Sarsfield, D G Scott, and S M Rajah

Haemoglobin and prognosis in childhood acute lymphoblastic leukaemia I M Hann, J H Scarffe, M K Palmer, D I K Evans, and P H Morris Jones

Unilateral proptosis A Oakhill, H Willshaw, and J R Mann

Nesidioblastosis of the pancreas S Aynsley-Green, J M Polak, S R Bloom, M H Gough, J Keeling, S J H Ashcroft, R C Turner, and J D Baum

Origin of handicap in young children C E M Jones and M Radford

Adolescents with cystic fibrosis E M Bywater