CASE REPORT

Necrotising enterocolitis complicated by gastrocolic and jejunocolic fistulas

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A premature infant had been treated conservatively elsewhere for necrotising enterocolitis (NEC). The infant was referred to Chris Hani Baragwanath Hospital on day 33 of life with severe vomiting and sepsis. Abdominal radiographic findings were nonspecific, demonstrating no direct features of NEC or pneumoperitoneum (Fig. 1). A water-soluble contrast enema demonstrated early filling of the stomach and small-bowel loops from the splenic flexure of the colon (Fig. 2). The filling of the stomach directly from the colon was significant, causing severe gastro-oesophageal reflux till the level of the thoracic inlet (Fig. 3). Surgery confirmed gastrocolic and jejunocolic fistulas requiring intestinal resection and diversion. The patient did well postoperatively.

Discussion

NEC is a major cause of abdominal emergencies in premature newborns.1 While NEC is commonly associated with stricture formation, which occurs in approximately 25% of cases, enteric fistulisation is a rare complication, with only isolated reports in the literature.2,3

NEC usually presents in premature infants from days 7 to 22 of life, while the complications of NEC, including enteric fistulas, occur from day 35 to 18 weeks.1-3

Clinically, infants with enteric fistulas may present with symptoms of incomplete intestinal obstruction, vomiting, diarrhoea, abdominal distention, a palpable inflammatory mass or recurrent sepsis.1,3

The plain film findings are nonspecific and may demonstrate multiple dilated bowel loops, pneumatisos intestinalis, portal venous gas or pneumoperitoneum.1,3

The investigation of choice for diagnosing enteric fistulas is a water-soluble contrast enema or distal loopogram. Upper gastro-intestinal tract contrast studies may be required to exclude proximal pathology.2,3
There are several mechanisms implicated in the development of intestinal fistulas. A key cause is the occurrence of a distal stricture with proximal bowel obstruction. The severe colonic ischaemia and associated transmural bowel necrosis occurring in NEC incite a local inflammatory response instead of a generalised perforation. This continuous inflammation results in adherence of affected segments of colon to adjacent bowel loops, and with time a fistula develops. The second theory involves a localised, subacute perforation which may be walled off by adjacent bowel leading to fistula formation. The treatment is surgical, with the timing of the surgery depending on the infant’s condition, including sepsis, electrolyte balance and nutrition. Surgical complications include short-bowel syndrome.

Conclusion
As the survival rate of infants with severe NEC improves, the incidence of complications such as enteric fistulas will increase. Symptoms in an infant following resolution of NEC, with a nonspecific bowel gas pattern on a plain radiograph, should raise the possibility of a fistula and prompt appropriate investigation and treatment of this significant complication.

References

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