

# NEONATAL GASTROINTESTINAL PERFORATIONS

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## ABSTRACT

Fourteen neonates presented with clinical and radiological evidence of gastrointestinal perforation, of which 13 were treated by operative intervention. No infant was formula fed, had rectal bleeding or radiological evidence of pneumatosis intestinalis. Operative findings revealed localized perforation in ten of which three were gastric, two distal ileal, three cecal and two in sigmoid colon. Gangrene with extensive perforation of jejunum, ascending colon and transverse colon were seen in one each. Blood cultures were positive in four, one grew *E. coli* and three *Klebsiella* while peritoneal cultures were positive in six, one grew *E. coli* and five *Klebsiella*. Ten neonates survived and are being followed up. Three of the four cases who died had white blood cell count  $>25 \times 10^9/L$  and grew *Klebsiella* on peritoneal culture.

Factors predisposing to gastrointestinal perforations in neonates are discussed, emphasis is made on the cautious use of umbilical, gastric and rectal catheters, and the need for early surgical intervention.

**Key words:** Neonates, Gastrointestinal perforations.

Neonatal gastrointestinal perforation which commonly occurs as a result of necrotizing enterocolitis (NEC) or in association with anatomic intestinal obstruction is an uncommon yet potentially fatal complication, particularly in stressed neonates. In this series, we report our experience of 14 such cases, without intestinal obstruction, highlighting their causes and management.

## Material and Methods

During an 18 month period from December 1989 to May 1991, 14 neonates, ten males and four females, with mean gestation of 38 weeks, and mean weight of 2300 g, presented with gastrointestinal perforation. Their salient features are summarized in *Table I*. The mean age at presentation was eight days. Nine were full-term of which one was small for gestational age (SGA) and five were preterm of which two were SGA. Predisposing risk factors noted were birth asphyxia in four, sepsis in four, rectal enema in two, patent ductus arteriosus (PDA), exchange transfusion (ET) and persistent fetal circulation (PFC) in one each. In five cases prematurity was among the other risk factors identified. Moderate to severe abdominal distension was noted in all, and bilious aspirates in three. All neonates had venous packed cell volume less than 0.65. All neonates except three who were on total intravenous fluids (Cases 5, 11, 13), received breast milk feeds and

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TABLE I—Summary of Relevant Information of 14 Cases

Case No.	Birth weight (g)	Gestation (weeks)	Age at presentation (days)	Risk factors	Operative findings	Surgical procedure and outcome
1	1750	36	6	Birth asphyxia, sepsis	Gastric perforation near lesser curvature 2 × 2 mm	Primary suturing Survived
2	3000	40	3	Nil	Cecal perforation	Primary suturing Survived
3	2500	40	6	Exchange transfusion	Gangrene transverse colon	Transverse colectomy with colostomy Survived
4	1700	35	6	PDA	Gangrene ascending colon	Ascending colectomy ileostomy and colostomy Survived
5	1400	39	15	Sepsis	Cecal perforation	Primary suturing Survived
6	3600	36	3	Severe birth asphyxia, PFC MAS	Gangrene 23 cm jejunum	Partial jejunectomy with end to end anastomosis Expired
7	1500	40	3	Birth asphyxia	Gastric perforation lesser curvature 3 × 3 mm	Primary suturing Survived
8	2500	40	14	Nil	Cecal perforation, massive dilatation sigmoid colon	Primary suturing, transverse colostomy Survived
9	3000	40	5	Birth asphyxia	Gastric perforation, greater curvature 5 × 5 mm	Primary suturing Expired
10	2800	40	25	Sepsis	Sealed ileal perforation	Survived

*Contd.*

Case No.	Birth weight (g)	Gestation (weeks)	Age at presentation (days)	Risk factors	Operative findings	Surgical procedure and outcome
11	1300	34	14	Rectal enema	Sigmoid colon perforation 2 × 1 cm	Sigmoid colectomy, colostomy of both ends Survived
12	1100	32	3	Sepsis	Distal ileal perforation 1 × 1 cm	Partial ilectomy with ileocolic anastomosis Expired
13	2300	40	3	Nil	Nil	Survived
14	2700	40	5	Barium enema	Linear recto sigmoid perforation 2 cm in length	Ileostomy and colostomy Expired

WBC = White blood cell.

PDA = Patent Ductus Arteriosus.

PFC = Persistent fetal circulation.

MAS = Meconium aspiration syndrome.

none had frank bleeding per rectum. X-ray abdomen revealed pneumoperitoneum in all except in Case 13 with meconium peritonitis and Case 14 with barium peritonitis. Dilated loops of intestine were seen in seven while pneumatosis intestinalis was not observed in any.

## Results

Of the 14 cases, blood cultures were positive in four, one grew *Escherichia coli* and the others *Klebsiella*, while peritoneal cultures were positive in six with *Klebsiella* grown in five and *E. coli* in one. White blood cell (WBC) counts ranged from 5.9 to  $32.3 \times 10^9/L$  and all four cases with sepsis had WBC  $>18.5 \times 10^9/L$ .

Operative findings revealed localized perforation in ten, of which three cecal and two in sigmoid colon. Gangrene of transverse colon, ascending colon and jejunum

were seen in each of the other three. The patient with jejunal gangrene had areas of congestion and edema over a large extent of small bowel, following severe birth asphyxia and PFC. Histopathology of cases 3 to 6, 9 and 10 revealed ganglionated nerve plexuses in rectum and colon, without pneumatosis intestinalis, but with copious cellular exudates. Histology of the patient with microcolon (Case 14) revealed normal ganglionated nerve plexuses, without NEC. Biopsies were not done in seven cases since 5 had small localized perforations not associated with diffuse bowel inflammation, one was a healed ileal perforation, and the case with meconium peritonitis did not require surgical intervention.

Various surgical procedures were performed. The necrotic bowel was resected and proximal end brought out as enterostomy while the distal end was kept as a

mucous fistula in cases 3, 4 and 11. Case 6 who was subjected to resection of 23 cm of jejunum with end to end anastomosis, expired soon after surgery. The perforations, when small and localized, were sutured and no enterostomy performed as in Cases 1, 2, 5, 7, and 9. The patient with meconium peritonitis (Case 13) was treated conservatively. In Case 12 who had distal ileal perforation, the diseased ileum was resected and an ileocolic anastomosis done.

In Case 10, the perforation appeared to have sealed spontaneously, a through peritoneal lavage was performed and flakes of pus and debris removed. In Case 14, who developed a rectosigmoid perforation while undergoing barium enema, the perforation was sutured and ileostomy and colostomy performed.

Of the four cases who died, one was a preterm with sepsis, one had severe birth asphyxia with PFC, one had catheter induced rectosigmoid perforation with barium peritonitis and one had a large gastric perforation with birth asphyxia and sepsis.

## Discussion

Apart from prematurity which is the single most significant risk factor causing NEC(1,2) important amongst other predisposing variables are intestinal ischemia, infection and neonatal feeding practices(3,4).

*Neonatal ischemic bowel injury* may follow acute asphyxia as a result of the 'diving reflex'(5) as seen in four cases in this series, or following umbilical venous exchange transfusion (ET)(6,7). The histologic features of the affected gut in such cases have striking similarities to those observed in preterms with classic NEC and presumably have a vascular basis(7,8). The histologic features of our cases wherein the disease was in the jejunum, transverse

colon, and ascending colon were consistent with those of an ischemic insult.

Suspicion exists that placement of catheter tip during umbilical ET in the portal system, rather than in the inferior vena cava with rapid volumetric and pressure changes alters hemodynamics in the portal circulation. This results in venospasm, hypoperfusion and hypoxia leading to venous infarcts, necrosis and gut perforation. Anatomic reviews suggest that hemodynamic alterations in the inferior mesenteric vein leads to colonic perforation as in Case 3, while changes in superior mesenteric vein causes small bowel perforation(8). In Case 6, PFC may have resulted in even greater hypoxic gut injury resulting in jejunal perforation.

*Necrotizing enterocolitis* classically occurs in preterms of <32 weeks gestation and only 7-10% are full terms(3,9,10). In contrast, in the present series the mean gestation age was 38 weeks, with no infant being less than 32 weeks. While the protective effect of breast milk in prevention of NEC has been well documented(11) in this series, eleven neonates were exclusively fed breast milk, while three were totally on intravenous fluids.

*Sepsis* was noted in four cases in this series. Bowel injury may be the direct effect of bacterial products causing intestinal ischemia(12). However, no consistent intestinal pathogen has been identified from such patients, although there is enough evidence that the disease has a microbial etiology.

*Meconium peritonitis* which occurs in 1 in 35,000 neonates(13) can result in multiple adhesive bands that produce intestinal obstructions either during neonatal period or later in infancy. This condition is usually associated with widespread intraperitoneal flakes of calcium(14). This calcified variety

of meconium peritonitis as observed in Case 13 is the result of intrauterine bowel perforation with sterile meconium leakage into the peritoneal cavity. The appearance of small amounts of calcium as an incidental radiologic finding unrelated to signs of obstruction or inflammation is an innocuous finding with no therapeutic significance(15). In our case this neonate responded to conservative line of therapy and did not require surgical intervention.

*Catheter trauma* can be amongst the causes of perforation in the gastroduodenal and rectosigmoid areas (16). In one particular review(17), catheter trauma accounted for 11 out of 144 cases of gastric perforation. Attempts to lavage and or decompress the distended stomach using nasogastric tube in at risk patients may have apparently contributed to perforations in Cases 1 and 9. Insertion of catheters for bowel enema can lead to similar disastrous results as seen in Cases 11 and 14. This can be avoided by undertaking proper care during such procedures. We emphasize that great caution should be exercised when using infant feeding tubes or catheters for gastric lavage or enemas especially in stressed neonates.

Except for critically ill patients (Cases 6, 9, 12 and 14) all survived the initial insult and were discharged. Cases 3, 4, 8 and 11 are awaiting reconstructive stomal anastomoses. None of the patients in our series had polycythemia which is a known factor responsible for necrotizing enterocolitis(18).

Since bowel perforation is uncommon in newborns, the diagnosis may be overlooked if sufficient attention is not paid to early evidences of abdominal distension and bilious vomitus or aspirate. Pediatricians should be aware of the hazard of bowel perforation following rectal

catheterization, gastric intubation and umbilical catheterization, particularly in neonates with predisposing factors causing intestinal ischemia or infection(12). Early diagnosis, prompt surgical intervention and intensive care has lowered the mortality to less than 25% at most centres(19,20). Good outcome (71% survival) in our patients is due to the fact that most were term or near term and had localised disease. Problems related to short bowel syndrome(20) have not yet been observed in our cases probably because of resection of small segments of the bowel.

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