INTESTINAL OBSTRUCTION IN THE NEWBORN ASSOCIATED WITH PERITONITIS*

BY

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The purpose of this paper is to report six cases of neonatal intestinal obstruction caused by acute inflammatory lesions in the intestinal tract. In none of these cases was there any mechanical cause for the obstruction, such as atresia, volvulus or aganglionosis.

Case Reports

Case 1. A male, aged 3 days, was a full-term normal delivery with a birth weight of 7 lb. 4 oz. (3.3 kg.). He passed a blood-stained stool at 56 hours, later developed abdominal distension and vomited green material. Physical examination showed poor general condition with jaundice, rapid pulse and respiration. Abdomen was distended and 'doughy' on palpation. Plain radiograph of the abdomen revealed a pneumoperitoneum. At laparotomy there was a generalized peritonitis, with a fibrinous exudate covering the loops of bowel. A perforation 2 cm. in diameter was found in the left end of the transverse colon. In addition, there were, in this area, three patches where the colonic wall was necrotic and thin and about to perforate. The thin patches were oversewn and the perforation exteriorized as a colostomy.

A swab of the peritoneal cavity produced a culture of coliform organisms insensitive to all antibiotics. A similar type of organism was found in the urine, though this was sensitive to nitrofurantoin.

Surprisingly, the baby made a satisfactory post-operative recovery, and one month later the colostomy was closed after resection of the scarred transverse and descending colon. Histological examination of the excised bowel revealed no abnormality apart from evidence of past inflammation.

Case 2. A male, aged 36 hours, born eight weeks prematurely after a normal pregnancy and delivery (birth weight 3 lb. 8 oz. (1.56 kg.)), began vomiting green material and developed abdominal distension at 24 hours. He passed a small quantity of meconium. Plain radiograph of the abdomen revealed a large pneumoperitoneum. Laparotomy showed a localized peritonitis in the left lower quadrant of the abdomen with a well-organized fibrinous exudate covering the loops of bowel. A perforation was found in the terminal ileum 5 cm. from the ileocaecal junction. The bowel wall around

the perforation appeared acutely inflamed. There was no mechanical obstruction present. The segment of ileum containing the perforation was resected and an end-to-end anastomosis performed. Post-operatively, the baby developed generalized oedema and jaundice with increasingly severe apnoeic attacks and died on the sixth post-operative day. Antibiotic therapy with tetracycline and streptomycin had been used.

A swab from the peritoneal cavity, taken at operation, produced a culture of coliform organisms sensitive to all antibiotics except penicillin. Histological examination of the resected ileum revealed 'a perforation 2.5 cm. from one end of the specimen and a blackened and necrotic area 1.5 cm. from the other end. The small intestine is oedematous and congested, and shows in the sub-mucosa areas of haemorrhage'. There was no autopsy.

Case 3. A male, aged 4 days, born after a normal pregnancy and delivery, weighing 4 lb. 12 oz. (2.1 kg.), commenced vomiting on the second day and gradually developed abdominal distension. A small amount of meconium was passed. Plain radiograph of the abdomen revealed a large pneumoperitoneum. At laparotomy, there was a generalized peritonitis with an organizing fibrinous exudate covering the surface of the bowel. A perforation, 2 cm. in diameter, was found in the terminal ileum, 4 cm. from the ileocaecal junction. There was nothing present in the intestinal tract anywhere that could have produced mechanical obstruction of any kind. The segment of ileum containing the perforation was resected and an end-to-end anastomosis performed. The baby's post-operative progress was poor, and there were several cyanotic attacks during the first 24 hours. Resuscitative measures were of no avail and the baby died.

Histological examination of the operative specimen showed 'a segment of ileum, a small portion of which has undergone complete necrosis with perforation and secondary peritonitis. The cause for the necrosis is not apparent from the sections at this level'. At autopsy the following relevant features were noted. The intestinal mucosa was inflamed and studded with ulcerative lesions in many segments. There were petechial haemorrhages on the cut surface of both adrenals and on the surfaces of the lungs and the heart. The cerebral hemispheres, cerebellum and the base of the brain were also covered in haemorrhages.

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Case 4. A male, aged 4 days, was born at term after a normal pregnancy and delivery. His birth weight was in the region of 6 lb. (2.7 kg.). Respiratory distress and cyanosis occurred within a few hours of birth, but responded to resuscitative measures and antibiotic therapy. On the fourth day, the baby commenced vomiting green material and developed abdominal distension. Meconium had been passed before this. Plain radiograph of the abdomen revealed distended loops of intestinal tract with fluid levels. At laparotomy, there was free purulent fluid in the peritoneal cavity and marked distension of the upper small intestinal tract. When traced distally, the bowel was found to enter a mass of fibrinous adhesions, in which several loops were matted together. When these loops were separated, black gangrenous areas were seen in the bowel wall over a distance of 4 cm. None of these areas had actually perforated and the mesenteric vessels were patent and pulsating. The remainder of the small and large intestinal tracts appeared normal. The segment of bowel containing the gangrenous areas was resected and an end-to-end anastomosis performed. On the fourth post-operative day, the abdomen again became distended and a radiograph showed a pneumoperitoneum. A second laparotomy revealed a leaking anastomosis. The bowel was discoloured for a distance of 0.5 cm. on either side of the suture line, and it seemed as though insufficient bowel had been resected at the first operation. A further resection and anastomosis was performed. Four days after this operation, the abdominal wound burst and had to be resutured. Subsequently, intestinal activity returned and oral feeding was commenced.

Culture of a peritoneal swab taken at the first operation produced a growth of coliform organisms sensitive only to neomycin and kanamycin. The baby was, therefore, given these drugs intramuscularly. Histological examination of the operative specimen showed 'acutely inflamed small intestine with a patchy fibrinous exudate on its surface and purulent contents. There is acute suppurative inflammation of the bowel wall affecting the mucosa most severely. The mucosal epithelium is largely destroyed and what remains is an unspecialized reparative type of epithelium'.

In the succeeding two weeks, the baby developed a series of pyogenic lesions, from each of which a coliform organism sensitive only to neomycin and kanamycin was cultured. These lesions included a wound infection, a generalized pustular eruption, osteitis of both ends of the left femur and the upper end of the left humerus. The femoral lesions were treated by aspiration and gradually resolved though there was obvious permanent damage to the hip joint. The humeral lesion subsided spontaneously. Eventually, the baby made a complete recovery.

Case 5. A male, aged 10 days, was born by caesarian section for pre-eclamptic toxæmia. The liquor was noted to be stained with meconium. Birth weight 3 lb. 1 oz. (1.4 kg.). On the fourth day of life, blood-stained meconium was passed, vomiting commenced, and the abdomen became distended. Oral feeding,

however, was continued for a further week, at which time a radiograph of the abdomen was taken and a pneumoperitoneum was revealed. At laparotomy there was purulent free fluid in the peritoneal cavity and a mature fibrinous exudate on the surface of the bowel. On separating the intestinal loops, an abscess was found in the left paracolic gutter. Closer inspection of this area revealed that the entire descending colon from splenic flexure to sigmoid had undergone complete necrosis. The colonic wall had disintegrated and consisted only of black shreds. The vessels in the mesentery of the colon were patent and pulsating. The necrotic material was removed and an end-to-end anastomosis performed between the splenic flexure and sigmoid colon. The baby made good post-operative progress until the fourth day, when it passed some urine that was blood-stained. This was followed almost immediately by a fit with respiratory arrest, which failed to respond to resuscitative measures.

A swab taken from the peritoneal cavity at operation produced a culture of *Staph. albus* and coliform organisms. Histological examination of the excised bowel revealed, 'necrosis and ulceration of the colon as well as focal suppuration. Indeed, there are several tiny abscesses in the bowel wall. The venous thrombosis present here and there is probably secondary. Large numbers of Gram positive cocci mark some of the lesions'. At autopsy, the only additional finding of significance was a left renal vein thrombosis.

Case 6. A male, aged 13 days, who had been born after a normal delivery, with a birth weight of approximately 3 lb. (1.4 kg.), commenced vomiting on the third day and developed progressive abdominal distension. Oral feeding was, however, continued until the thirteenth day, when the abdomen was radiographed, revealing distended loops of intestinal tract with fluid levels. At laparotomy, there was purulent free fluid in the peritoneal cavity and a fibrinous exudate covering the surface of the bowel. After mobilizing the intestinal tract, it could be seen that the whole transverse colon had undergone complete necrosis. All that remained were a few black strands. The middle colic vessels were seen in the remains of the mesentery and were clearly patent. After peritoneal toilet, the ends of the colon were brought out at either end of the transverse abdominal incision. The baby's post-operative progress was good for three days, but then apnoeic attacks occurred, which did not respond to treatment.

A swab taken of the peritoneal fluid at operation produced a culture of *Candida albicans*. Histological examination of the pieces of colon excised revealed the following: 'A section from the disintegrated bowel shows an acute inflammatory response with numerous foreign body giant cells and P.A.S. positive structures which are not unlike fungi'. The only additional findings at autopsy were a massive left adrenal haemorrhage and pulmonary congestion and oedema.

Having noted the fungal-like structures in the colon of Case 6, the pathologist re-examined material from Case 5 and reported the presence of similar bodies in this case.

The cases are summarized in the Table.

TABLE
SUMMARY OF CASES

Case	Age (days)	Sex	Maturity	Pre-operative Diagnosis	Operative Findings	Results	Pathology
1	3	M	Full-term	Obstruction and perforation	Peritonitis; gangrene transverse colon with perforation	Alive	<i>E. coli</i> ; peritonitis
2	1½	M	Premature	Obstruction and perforation	Peritonitis; perforation of terminal ileum	Dead	Acute inflammation of ileal wall
3	4	M	Premature	Obstruction and perforation	Peritonitis; perforation of terminal ileum	Dead	Acute inflammation of ileal wall and necrosis with perforation
4	4	M	Full-term	Obstruction	Peritonitis; gangrene of jejunum	Alive	Acute inflammation of jejunal wall with necrosis of mucosa; developed multiple coliform osteitic lesions later
5	10	M	Premature	Obstruction and perforation	Peritonitis; gangrene of descending colon with perforation	Dead	Acute suppurative necrosis of colon
6	13	M	Premature	Obstruction	Peritonitis; gangrene of transverse colon with perforation	Dead	Acute inflammatory response; ? invasion by fungi

Discussion

That acute inflammatory lesions of the intestinal tract in the newborn may give rise to a picture simulating mechanical obstruction is not well documented in the literature. Intestinal obstruction in these cases is caused primarily by the peritonitis and not by mechanical factors. The peritonitis is present whether or not perforation has occurred. In the absence of lesions that produce mechanical obstruction and intestinal gangrene from distension, and in the presence of obviously patent mesenteric vessels, it is difficult to elucidate the train of events that has taken place. In all the cases, scattered gangrenous lesions have been present in the intestinal wall in either small intestine or colon, and some of these lesions have proceeded to complete necrosis and perforation. Since the lesions do not seem to have a vascular origin, they are presumed to be bacterial. They could arise either as a result of a perinatal infection which caused septicaemia and blood-borne bacterial invasion of the bowel wall or, alternatively, from the ingestion of virulent organisms that invaded the intestinal mucosa. There can be little doubt that a coliform septicaemia occurred in Case 4. Levin and Isaacson (1960) reported two cases of spontaneous perforation of the colon in newborn babies, in one of which fungal hyphae were found in the colon at the site of the perforation. Their cases are similar to Cases 5 and 6 in the present series. The fungal infection must have occurred during or soon after birth, though why it should produce lesions localized in the colon is not explained. Emery (1957) and Zachary (1957) suggested that hard meconium might cause stercoral ulceration and perforation of the colon in the newborn, particularly if premature.

None of the babies in this series passed hard meconium on any occasion, and neither were pieces of such meconium found in their peritoneal cavities. Thelander (1939), in a review of the world literature, found 83 cases of spontaneous perforation of the intestinal tract in the newborn, 14 being in the small intestine and 22 in the large intestine. The remainder were in the stomach, duodenum or Meckel's diverticula. He concluded that the perforations were associated with septicaemia and stated that, although blood culture was carried out in only three cases, it was positive in all of them. He recommended looking for and treating all possible forms of perinatal sepsis.

What little evidence there is, therefore, suggests that the babies reported in this series suffered from an overwhelming invasion by bacterial or fungal organisms immediately before, during or after birth. The invaders may have gained access either by ingestion or by such routes as the umbilicus. The intestinal lesions may, therefore, have arisen from direct invasion of the mucosa or via the blood stream. It seems that, whichever is the case, blood-stream invasion eventually occurs, giving rise to metastatic lesions such as osteitis, if the baby lives long enough.

Summary

Six newborn babies who presented with the clinical picture of intestinal obstruction are described.

No mechanical cause for the obstruction was found in any of the cases. The obstruction was associated with peritonitis and perforation of the intestinal tract in five cases and localized gangrenous

enteritis in one. In two cases of colonic perforation, the responsible organism was thought to be a fungus.

It is thought that perinatal sepsis is the underlying cause for the intestinal gangrene.

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