Idiopathic Perforation Of The Colon In Neonates: A Clinical Review Of Seven Cases
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Citation

Abstract
The aetiology of idiopathic colon perforation in neonates remains unclear. We reviewed seven patients with idiopathic colon perforation from January 1996 to October 2001. Clinical data included age, sex, weight, associated problems, perforation-operation interval, the location of colon perforation, treatment, morbidity, and mortality. All patients were boys. The median age was 34.8 days. Six of the newborn infants were full-term, while one was premature. The aetiology was established in none of the patients. The side of the perforation was transverse colon in three patients, left colonic flexure in two, and sigmoid colon in two patients. After resection of the perforation area, proximal colostomy was performed in four patients and sigmoid colostomy in three. Two patients died. Postoperative wound dehiscence developed in all five patients who survived. Ganglion cells were present in all colonic specimens.

In conclusions, many different factors may play a role in developing colon perforation in neonates. However, because of limited number in our series the relative significance of each factor is difficult to assess.

INTRODUCTION
Idiopathic colon perforation in neonates is extremely rare, and early diagnosis and prompt surgical treatment is life saving [1,2,3]. Such perforations have been explained on the basis of trauma or mechanical obstruction. Traumatic causes include trauma from rectal thermometer, barium enemas or other rectal instrumentation. Mechanical obstruction causes include blow out proximal to obstruction by colonic atresia, imperforate anus, meconium plug, small left colon syndrome, Hirschsprung's disease or obstructive bands [4,5,6,7]. Colonic perforation of unknown causation also occurs in the newborn infant. We present our experience on seven cases of idiopathic colon perforation.

PATIENTS AND METHODS
We reviewed seven cases of idiopathic colon perforation retrospectively from January 1996 to October 2001. All infants whose perforations were due to necrotizing enterocolitis, appendicitis, Hirschsprun's disease, meconium ileus, intestinal atresia, or drug therapy were excluded from the series. None of the infants had associated anomalies. Clinical data included age, sex, weight, associated problems, perforation-operation interval, the location of colon perforation, treatment, maternal complication, fetal complication, usage of medication, morbidity, and mortality.

The erect abdominal x-ray demonstrated a pneumoperitoneum and viscous organ perforation, and the surgery made the diagnosis of colon perforation in all cases.

RESULTS
All 7 patients were boys. The median age was 34.8 days (range, 9 to 90 days). Three of newborn infants were transferred from our university hospital neonatal intensive care unit and four babies were transferred from another hospital. Table 1 outlines the main findings of all patients. Six of the newborn infants were full-term, while one was premature and had low birth weight. The aetiology was established in none of the newborn infants. Microorganism was not present in any of the faeces specimens. Additionally, in case 1 trauma was suspected due to anal thermometer usage.
All of the patients received intravenous fluids, nasogastric decompression, and antibiotics preoperatively. There was fecal and purulent matter in all patients abdominal cavity. Specimen from abdominal purulent material was obtained for culture antibiogram; no microorganism was present in any of the specimens. Colostomy was performed in all patients. Before closure of the abdomen, irrigation of the abdominal cavity with normal saline solution was carried. The biopsy of the colon wall adjacent to the perforation was removed in all patients. The histopathological changes were non-specific; oedema, inflammation, haemorrhage and necrosis were present. Bacteria or microthrombi were not present in any of the biopsy specimens. All of the biopsy specimens of the colon wall adjacent to the perforation and distal were present ganglion cells in all patients.

Postoperatively, wound dehiscence developed in 5 patients (71%). Escherichia coli was detected in wound of only one patient with wound dehiscence. Patients with wound dehiscence underwent operation and improvement was observed. Colostomy closure was performed at postoperative 6 to 8 weeks. Two patients died due to sepsis at 2nd and 1st postoperative days respectively. The average duration between perforation and operation was 4 days.

There is no doubt that the treatment of idiopathic colon perforation is operative. A primary closure or a resection and anastomosis may be performed in hemodynamically stable patients with well-localised and single perforation. On the other hand, the exteriorisation of the perforated area of the colon may be safer in unstable patients associated with severe peritoneal contamination and ischemic bowel [1].

Four of patients in our series were unstable and had severe and perforating by meconium plugs, and perforation of colon by necrotizing bacterial enterocolitis were reported as aetiology [8,9,10]. Touloukian et al [8] suggested that anoxia or hypoxia can cause mucosal ischemia and subsequent necrotizing enterocolitis in piglets, and this etiology may certainly be related to hypoxic neonates. Weinberg et al [11] suggested that the etiology of colon perforation may be ischemic necrosis secondary to a very localized vascular accident in the wall of the affected bowel. In their series, 6 of the perforations were on the antimesenteric wall of the intestine, the terminal point of the vascular supply of the area, and they explained it by a vascular theory. The previously existing clinical and experimental experiences indicate that the ischaemic component plays an important part in the aetiology of this disease. Although, we observed sepsis in two patients, diarrhea in three, and history of rectal trauma in one patient, there was no certain evidence of those theories mentioned above regarding the etiological factors of colon perforations in our series. In addition, we found no ganglionic abnormality in any of 6 presenting cases. Zamir et al [12] recently presented a series that was similar to our findings. In a majority of their cases, the aetiology of the perforations was also unclear.

Idiopathic colon perforation in the newborn is often difficult to diagnose clinically. The clinical symptoms and signs in such cases include vomiting, sepsis, cyanosis, tachypnea, respiratory distress, and significant abdominal and scrotal distension; however, none of these is pathognomonic for idiopathic colon perforation. Radiographic evaluation may allow earlier diagnosis and prompt surgical treatment. In the presence of a massive pneumoperitoneum in a neonate, both gastric and colonic perforation should be considered [11,13]. The abdominal upright film will show the “saddle” or “football” sign due to massive pneumoperitoneum [4]. All of our infant's abdominal roentgenograms demonstrated a pneumoperitoneum and viscus organ perforation, and the surgery made the diagnosis of colon perforation in all cases. The average duration between perforation and operation was about 4 days.
peritonitis. Therefore, we exteriorized the two ends of bowel. Wound dehiscence was developed in 5 patients postoperatively (71%). Two patients died; one was premature and had low birth weight, while the other had sepsis. The average duration between perforation and operation was 5 days for those died patients.

**CONCLUSIONS**

In conclusions, many different factors such as trauma, ARDS, prematurity, sepsis, anaemia, and diarrhea may play a role in developing colon perforation in neonates. However, because of limited number in our series the relative significance of each factor is difficult to assess. Early recognition of perforation contributes to successful treatment in such cases. In addition, the exteriorisation of the colon may be safer for hemodynamically unstable cases, and patients with large perforations and severe peritonitis.

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