SPONTANEOUS GASTROINTESTINAL PERFORATION IN THE NEONATE

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ABSTRACT

Twenty seven cases of spontaneous gastrointestinal perforation were treated from 1981 to 1990. Four perforations were in the stomach, 17 in the small bowel and seven in the large bowel. One of them had dual perforation, one in the stomach and another in the duodenum. The exact etiology remained obscure. Various factors observed were maternal obstetric complications, prematurity and perinatal asphyxia. Stress should be laid on early diagnosis by following up 'at risk neonates' thus, giving a better overall survival rate.

Key words: *Neonate, Gastrointestinal perforation.*

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Spontaneous gastrointestinal perforation in a neonate was first reported by Siebold in 1825. The first successful repair was reported only in 1943(1). In 1969, Lloyd(2) complied 400 cases of spontaneous gastrointestinal perforation by reviewing the literature. In this group of patients, isolated perforations occurred without any evidence of mechanical intestinal obstruction or necrotizing enterocolitis. The underlying etiology of this entity still remains obscure although various factors like maternal obstetric complications, perinatal stress, hypoxia and shock have been implicated. Our experience with 27 neonates over a period of 10 years is presented.

Material and Methods

Twenty seven cases of spontaneous gastrointestinal perforation were treated in the Department of Pediatric Surgery, SPM Child Health Institute of S.M.S. Medical College, Jaipur from 1981 to 1990. All perforations secondary to necrotizing enterocolitis, atresia or other mechanical obstruction were not included in the present series. This article includes the retrospective study of all the cases of spontaneous gastrointestinal perforations treated in the Department during the last 10 years. There were 24 males and 3 females. The routine investigations included hemogram, serum electrolytes, urea and creatinine estimation, routine urine analysis and skiagram of the chest and abdomen in upright position. After resuscitation, an exploratory laparotomy was performed in all the cases under general anesthesia. The gastric, duodenal and ileal perforations were primarily closed in two layers. Local resection was done in multiple jejunal perforations. Most of the large bowel perforations were exteriorized.

Results

In these twenty seven cases, perforations usually measured less than 2 cm and were single except in 2 cases in whom multiple perforations were found. Thirteen cases had perforation in the ileum, three in the stomach, two in the duodenum and three each in the cecum and colon. There was a single case of rectal perforation. One patient had multiple jejunal perforations and another had two perforations, one in stomach and another in the duodenum (*Table I*). Most of the patients (eighteen) were delivered at home and detailed obstetric history was not available. Out of nine hospital deliveries, seven had Cesarean section and the other two had normal vaginal deliveries. Among those who were delivered in the hospital, a significant proportion (six) had maternal obstetric complication of some kind which included premature rupture of membranes in three, toxemia of pregnancy in two and amnionitis in one case. Out of twenty seven mothers, twenty had a history of multiple births and a history of previous abortions could be elicited in nine cases. Most of the children (twenty two) were of low birth weight and three were born preterm. Three patients among the hospital deliveries suffered from asphyxia and were given respiratory assistance of some kind. One case had exchange transfusion for jaundice. In eighteen patients, perforation occurred in the first week and in seven perforation occurred in the second week. In the remaining two cases it occurred in the third and fourth weeks. The commonest presenting features were abdominal distension of sudden onset, persistent vomiting, respiratory distress and occasionally hematemesis and melena. In all the cases, pneumoperitoneum could be demonstrated radiographically.

The overall survival rate in the present series was 67%. Of the 9 deaths, 6 died in the immediate postoperative period due to toxemia and shock. Two died of pulmonary complications and one of generalized peritonitis secondary to leakage of repair (*Table II*). Other complications included wound infection in three cases and burst abdomen in one case which was managed successfully. All the exteriorized large bowel perforations were subsequently closed without any complication. Ten cases were available for follow up with the age

TABLE I–*Distribution of Cases in Relation to Site of Perforation*

Site of perforation	No.
Jejuno-ileal	14
Gastric	3
Cecal	3
Colonic	3
Duodenal	2
Rectal	1
Gastric & duodenal	1
Total	27

TABLE II–*Complications and Outcome*

Outcome	No.
A. *Survived*	18
No complications	14
Wound sepsis	3
Burst abdomen	1
B. *Died*	9
Toxemia and shock	6
Pulmonary complications	2
Leakage of repair	1

ranging from three months to eight years. All were free of any symptoms related to their previous illness.

Discussion

Perforation in the neonatal age group may occur secondary to mechanical causes, *e.g.*, atresias, meconeum ileus and Hirschsprung's disease, but there are a few patients in whom perforations occur with no evidence of intestinal obstruction. The exact etiology of spontaneous gastrointestinal perforation is unknown. Few cases previously listed as unknown causes may have been due to necrotizing enterocolitis since both entities occur predominantly in premature infants and in those who suffered from perinatal stress. Also the age of onset of both entities is usually between the third and tenth day of life. But prodromal gastrointestinal symptoms suggesting necrotizing enterocolitis are lacking and radiological features suggesting necrotizing enterocolitis are absent in spontaneous perforations(3,4). Moreover, the pattern of involvement of the gut is also different. In necrotizing enterocolitis, terminal ileum and ascending colon are usually affected whereas spontaneous perforations occurred in our series mostly in proximal ileum, stomach and duodenum which are usually spared in necrotizing enterocolitis. Lloyd(2) proposed that gastrointestinal perforation in the neonate is the result of asphyxial defence mechanism. According to this theory babies react to hypoxia and shock by shunting of blood to the heart and brain at the expense of mesenteric as well as peripheral and renal blood flow. Local hyperactivity of this mechanism may cause ischemia of the stomach or intestine resulting in perforation. An increased incidence of perinatal stress and anoxia was observed in the present series which supports this theory. But the stomach has a generous blood supply and is usually spared in situations attributed to newborn intestinal ischemia. Hence, ischemic changes secondary to hypoxemia and hypovolemia do not seem to be responsible for causing perforation of such a vascular organ like stomach.

It was the presence of normal microscopic mucosa and submucosa along with a gap in the muscularis mucosa that led Herbut(5) to conclude that a congenital absence of muscle was the etiology of the perforation. The histopathological examination was carried out in the present series. Holgerson(6) has given evidence in support of a mechanical disruption as the etiology of spontaneous neonatal gastric perforation. Spontaneous disruption may occur secondary to gastric dilatation resulting from aerophagia, intraoral oxygen administration, overfeeding, trauma due to nasogastric tube(7). No such case of perforation was recorded in the present series. Therapeutic measures like indomethacin administration(8) has also been implicated in spontaneous gastric perforation. Therefore, we have a multifactorial etiology for spontaneous gastrointestinal perforation in neonates.

Early recognition of perforation contributes to successful treatment. A newborn infant delivered by cesarean section and whose mother's pregnancy is complicated by placenta previa, amnionitis, abruption placentae is at increased risk of developing gastrointestinal perforation and therefore, should be carefully observed. Prematurity, neonatal asphyxia, hyaline membrane disease, the use of exchange transfusion and indomethacin therapy should also be considered as risk factors. Exploration is undertaken after resuscitation and correction of fluid and electrolyte

imbalances. Perforations of stomach and duodenum are simply closed in two layers. Localized lesions of ileum may also be repaired or respected. Perforations of the colon are generally associated with fecal contamination, hence exteriorization of the perforation is a safe procedure than closure. Systemic antibiotics effective against Gram-negative and anaerobes are given routinely and occasionally hyperalimentation is needed.

Spontaneous perforations have a better prognosis than those secondary to necrotizing enterocolitis. In our series, we could save 67% of patients. This series carries a high mortality rate because of the late presentation. The patients were often brought in a moribund state from distant rural areas thus leading to a high mortality rate.

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