INDEFINITE CAUSES OF GASROINTESTINAL PERFORATION IN NEONATES

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SUMMARY : Between the years 1986 and 1990 22 newborns with gastrointestinal [GI] perforation were treated at the Ankara SSK Children's and Gazi Hospitals. Fourteen [63.6 %] of the neonates were male and 8 [36.4 %] were female. The perforations were distributed along the GI tract from the greater curvature of the stomach to the transverse colon. There were 1 gastric, 2 duodenal, 3 jejunal, 5 ileal and 11 colonic lesions, respectively. We were able to find the cause of the perforation in only 9 patients [40.9 %]. Overall survival was 54.5 %, regardless of the perforation site and the surgical procedure performed.

Key Words : *Gastro - Intestinal Perforation, Pneumoperitoneum.*

INTRODUCTION

Perforation of the gastro - intestinal [GI] tract may occur in the neonate anywhere from the stomach to the rectum. Aside from neonatal necrotizing enterocolitis [NEC]; intestinal atresia, meconium ileus, vascular accidents, mid-gut volvulus or Hirschsprung's disease are the most frequent causes of GI perforations. [Bell, 1985; Lloyd 1969; Martin and Perrin, 1967; Raffensperger et al. 1970; Raffensperger, 1990; Soper and Opitz 1963; Welch et al. 1986]. However, in most of the cases no obvious cause is found.

During the last four years, we have operated on 22 newborns with GI perforation at the Ankara SSK Children's and Gazi Hospitals. The purpose of this study is to discuss the aetiological causes, pathology, surgical treatment and prognosis of neonatal GI perforations.

MATERIALS AND METHODS

A retrospective 4-year review from 1986 to 1990 was made of 22 neonates admitted to the Ankara SSK Children's and Gazi hospitals with GI perforation. The patient files were examined to identify age, sex, site of perforation, aetiological causes, clinical and radiological findings and eventual outcome.

RESULTS

Fourteen [63.6 %] of the neonates were male and 8 [36.4 %] were female. Twelve [54.6 %] babies were admitted in the first week of life, five [22.7 %] in the second week and another five [22.7 %] between the age of 15 to 28 days.

Common complaints of the babies were failure to pass meconium or faeces, abdominal distention and vomiting bile stained fecaloid. Physical findings of the 22 neonates with GI perforation are listed in Table 1.

Upright plain x-rays of the abdomen demonstrated free air under the diaphragm in 14 [63.6 %] patients. Radiological signs of intestinal obstruction were present in 8 [36.4 %].

Physical Findings	No. of Patients		% Percent
Abdominal distention			
Severe	17	77.3 %	100.0 %
Moderate	4	22.7 %	
Absence of peristaltic sounds	22		100.0 %
Tachycardia	22		100.0 %
Tachypnea	21		95.5 %
Tympanitic abdominal percussion	14		63.6 %
Sclerema	12		54.5 %
Periumblical echimosis	4		18.2 %

Table - 1 : Physical findings of the 22 Neonates with GI perforation.

Site of Perforation	No. of Patients	%	Percent
Stomach	1	4.6 %	13.7 %
Duodenum	2	9.1 %	
Jejunum	3	13.6 %	36.3 %
Ileum	5	22.7 %	
Colon			
Caecum	7	31.8 %	50.0 %
Transverse Colon	4	18.2 %	
Total	22	100.0 %	100.0 %

Table - 2 : Site of perforations.

The perforations were distributed along the GI tract from the greater curvature of the stomach to the transverse colon [Table 2].

We were able to explain the cause of the perforation in only 9 [40.9 %] patients [Table 3]. The babies with gastric and duodenal perforations were otherwise healthy and older than 15 days. No explanation on the aetiology could be made in this group. There were 3 jejunal and 5 ileal perforations. In two of the jejunal perforations, the cause was jejunal atresia and malrotation with mid - gut volvulus, respectively. No obvious cause was found in the third case. Ileal perforations were due to ileal atresia in three babies. Aetiology remained unidentified in two patients of this group. We have operated upon 7 babies with caecal perforation. Five of them appeared to have Hirschsprung's disease with the typical narrowed sigmoids, but two turned out to have normal ganglion cells in their rectum. In one baby, the cause was congenital fibrotic bands crossing over the hepatic flexura with subsequent obstruction. In three perforations, the cause of the perforation remained unknown. In 13 [59.1 %] of the babies, the aetiology also remained unknown [Table 3].

In the baby with gastric perforation, the perforation site was sutured and a gastrostomy tube was inserted for post - operative decompression. Duodenal perforations were also sutured and covered with a tag of omentum.

Two of the ileal perforations in which no aetiological factor was found, were treated by resection and end - to - end anastomosis. Bishop - Koop exteriorization was performed in three patients who had ileal atresia.

Seven infants with caecal perforation were treated by appendicectomy and exteriorization of the caecum. Loop colostomy was done in all of the transverse colon perforations.

All of the patients had intra - abdominal sepsis at the time of operation. Overall survival was 54.5 % regardless of the perforation site and the surgical procedure performed [Table 4].

DISCUSSION

The most common perforation of the stomach is a laceration of the greater curvature [Holgerson, 1981; Raffensperger, 1990 Rosser et al. 1982;

Site of Perforation	No.of Patients	Cause of Perforation	
Stomach	1	Unidentified	[1]
Duodenum	2	Unidentified	[2]
Jejunum	3	Malrotation + midgut volvulus	[1]
		Jejunal atresia	[1]
		Unidentified	[1]
Ileum	5	Ileal atresia	[3]
		Unidentified	[2]
Colon			
Caecum	7	Hirschsprung's disease	[3]
		Congenital bands	[1]
		Unidentified	[3]
Transverse Colon	4	Unidentified	[4]

Table - 3 : Causes of the GI Perforations in 22 newborns.

Site of Perforation	No	Surgical Therapy	Survivors	%
Stomach	1	Pr.Closure + Gastrostomy	None	0.0 %
Duodenum	2	Primary Closure	1	50.0 %
Jejunum	3	Resection + Anastomosis	None	0.0 %
Ileum	2	Resection + Anastomosis	2	100.0 %
	3	Bishop - Koop Enterostomy	1	33.3 %
Colon				
Caecum	7	Exteriorization	[4] 57.1 %	72.7 %
Transverse Colon	4	Exteriorization	[4] 100.0 %	
Total	22		12	54.5 %

Table - 4 : Surgical procedures and survival rate.

Welch et al. 1986]. Insertion of stiff nasogastric tubes, compression of a fluid - filled stomach during difficult deliveries, overdistention of the stomach during vigorous resuscitation with the ambu mask, congenital muscular weakness and increased hyperacidity had all been considered as causes of gastric perforation in neonates. However, such explanations are now discarded. Recent studies suggest that perinatal hypoxia and / or hypovolemia plays the major role in neonatal gastric perforations [Bell, 1985; Grund and Dzieniszewski, 1981; Holgerson, 1981; Rosser et al. 1982; Touloukian, 1973; Welch et al. 1986].

The aetiology of the duodenal perforation seen in healthy full - term newborns is still unknown. Follow-up roentgenograms as late as four years failed to demonstrate further ulcers in this group of babies [Raffensperger, 1970]. We have operated upon two babies with duodenal perforation. Each had the perforation on the anterior wall of the first portion of the duodenum. The etiology in these patients remained unknown.

Small bowel perforations are usually secondary to mechanical obstructions, such as atresia, meconium ileus or gangreneous volvulus. If no obstruction is present, then perinatal hypoxia or NEC should be considered.

Perforation of the caecum or appendix in a neonate should be attributed to distal Hirschsprung's

disease until proven otherwise [Ghory and Sheldon, 1985; Martin and Perrin, 1967; Raffensperger, 1970; Raffensperger, 1990; Soper and Opitz, 1963; Welch et al. 1986]. The small left colon syndrome or umblical vein catheterization resulting in retrograde portal vein thrombosis may also cause caecal perforation [Friedman et al. 1970].

Although ischemia may be the common denominator, a multifactorial aetiology is likely when no obvious cause is found. The actual mechanisms by which these ischemic changes are induced still remain uncertain. Lloyd [1969] called attention to the fact that these lesions were the result of ischemic necrosis, regardless of location. Lloyd suggests that the newborns have the "diving reflex" like the amphibious animals. It is believed that blood is shunted to the heart and brain at the expense of the peripheral, renal and mesenteric vascular beds by means of this reflex mechanism. Newborns protect themselves with selective circulatory ischemia during the time they pass through the birth canal. Redistribution of blood flow during hypoxia and / or hypovolemia or other stress states, with shunting away from mesenteric vascular beds, is thought to result in microvascular thrombosis, and subsequent loss of mucosal integrity. Persistance of the ischemic insult, allowing extention of microvascular thrombosis, leads to the transmural necrosis and perforation. For this reason, infants born of pregnancies characterized by abruptio placentae, placenta previa, amnionitis and infants delivered by emergency cesarean section are at increased risk and should be carefully observed for 4-5 days in the hospital [Welch et al. 1986].

An infant who is born with a distended abdomen that is dull to percussion has either a large intra - abdominal tumor or meconium peritonitis. In contrast, an infant who is doing well and suddenly develops abdominal distention that is tympanitic to percussion most likely has an intestinal perforation. In other infants, the signs of perforation either are masked by or are secondary to the primary intestinal obstruction. Abdominal distention in a baby with GI perforation is frequently abrupt and rapidly progressive. Signs of hypovolemia and decreased perfusion are usually present. Respiratory difficulty from massive pneumoperitoneum may be the first sign of the perforation.

Any infant with abdominal distention must have abdominal roentgenograms obtained. Upright or lateral decubitis plain x- ray films will demonstrate free air under the diaphragm or the lateral wall of the abdomen. As little as 3 mililitres of free air in the peritoneal cavity can be detected by an upright abdominal roentgenogram [Raffensperger, 1970; Raffensperger, 1990]. Large amounts of free air in the peritoneal cavity gives the abdomen a rugby ball shape [Figure 1].

The infant with GI perforation and subsequent peritonitis deteriorates rapidly and early recognition and prompt treatment are necessary for a favorable outcome. However, there is no need to rush the patient to the operation theatre. The loss of pati-

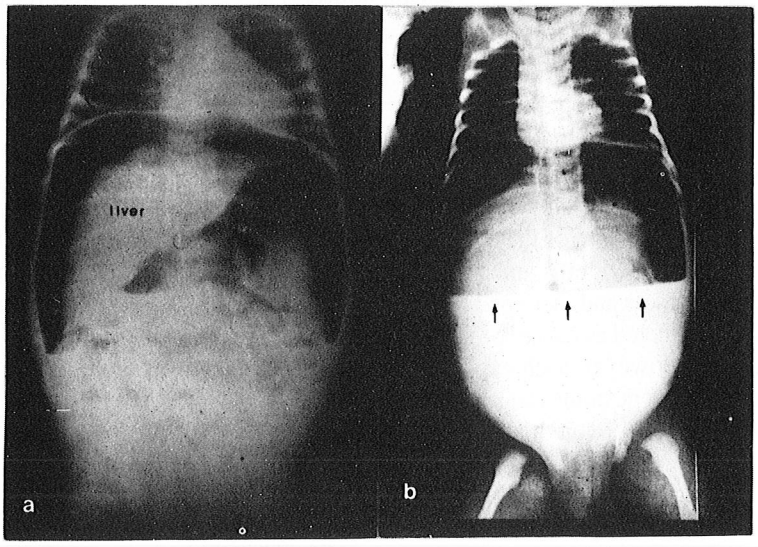

Fig . 1 : a : Large amount of free air under the diaphram "football sign".

b : Note the giant air - fluid level caused by the intraperitoneal free air and gastric contents.

ents is due to the sepsis, electrolyte imbalance or circulation problems rather than the surgical procedure itself and related complications.

Perforation of the stomach and duodenum are primarily closed. Vagotomy or other definitive procedures for ulcer are unnecessary.

Treatment of proximal small intestine perforation involves resection and an end-to-end anastomosis [Raffensperger, 1970; Raffensperger, 1990; Welch et al. 1986].

Perforation of the terminal ileum, caecum and colon should be exteriorized because of the great likelihood of a distal obstruction. However, in proximal ileal lesions, simple suturing or resection and primary anastomosis could also be advised.

The prognosis is determined by the maturity of the patient, associated conditions, the duration between the perforation and ressuscitation, the exactness of the surgical procedure, and the intensive post - operative care. Approximately 30 % survival rate was reported in early series [Raffensperger, 1970]. More recent series suggest 60 % [Raffensperger, 1990; Welch et al. 1986]. However, the survival rate for the isolated gastric perforation still continues to be discouraging. In the present series, overall survival has been found to be 54.5 %. The ways to increase the survival rate are; the elimination of the pre - and postnatal predisposing factors, early diagnosis and judicious pre - and - postoperative care.

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