

## Neonatal Pneumoperitoneum and Pattern of Gastrointestinal Perforations and its Outcome - Single Center Study

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### Abstract

**Objective:** To look for pattern and pathological causes of Gastrointestinal perforations in neonates and evaluation of current modalities of surgical management and their short term outcome.

**Design:** Retrospective study.

**Place and Duration of study:** Department of Pediatric Surgery, The Children's Hospital and the institute of child health, Lahore Pakistan.

**Patients and Methods:** Between January 2011 to January 2013, a total of forty eight neonates with GI perforations were managed. Birth weight, age, clinical presentation, etiology, site of perforation, surgical management performed and complications encountered are analyzed.

**Results:** 60% of patients (28/48) had appropriate birth weight where as 31% (14/48) had low birth weight. Most of the patients (35/48) were males. Exclusive number (83%) i.e. 38 out of total of 48 presented in first week of life. Most of the patients presented with abdominal distension, vomiting, constipation and septicemia. Apart from usual investigations, exploration was performed on the basis of pneumoperitoneum on abdominal x-rays. There were 28 cases of NEC, 10 of complicated meconium ileus, 03 with gastric perforation and 04 of anorectal malformations. 2 cases of colonic atresia and 01 of Hirschsprung's disease were included in the study. The complications encountered in these cases included septicemia, abdominal wound infection, leakage of anastomosis and stoma related complications like bleeding, necrosis and retraction. The mortality in study was 16 patients comprising 09 cases of NEC, 5 cases of complicated meconium ileus, 01 case of imperforate anus and 01 case of colonic atresia.

**Conclusion:** Gastrointestinal perforation in the neonatal age is a common manifestation of various disease entities and management of these neonates requires understanding of these pathological entities. Septicemia and late presentation of patients to hospital is responsible for high morbidity and mortality in these tiny sick neonates in our setup.

**Keywords:** Gastrointestinal perforation; Neonate; Pneumoperitoneum

### Introduction

Spontaneous perforations of the gastrointestinal tract of the newborn were reported in the literature since early 18<sup>th</sup> century. Siebold stern., *et al.* in 1929 described one of the earliest attempts at operative intervention but it was not until 1943 that a neonatal GI perfora-

tion was closed successfully [23]. In 1964, Lloyd, *et al.* reported 61 cases of neonatal GI perforations and proposed that these lesions were the result of ischemic necrosis regardless of location [24]. Many authors credit Generic with publishing the first case of NEC in 1891. In 1939, Thielander collected 16 cases of perforations of stomach, 30 of duodenum and 39 of small and large intestine. In 1944, Von Willi recognized that infants suffering from infectious enteritis could develop bowel perforation, peritonitis [41] and even death [25].

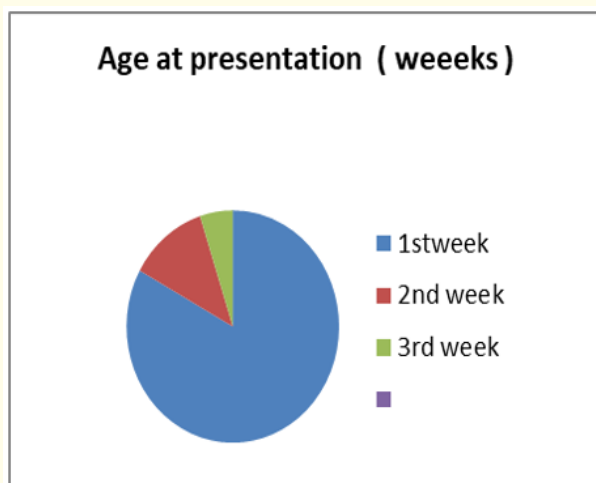
**Materials and Methods**

Over [1] a period of two years from January 2011 to January 2013, forty-eight neonates were treated for gastrointestinal perforations at the Department of pediatric surgery The children’s hospital and the institute of child health, Lahore, Pakistan.

A retrospective review of all the neonatal cases of gastrointestinal perforations presenting to the department of pediatric surgery from January 2011 to January 2013 was done. Pertinent information included mode of delivery, birth weight, gestational age, and mode of presentation, timing, etiology, site of perforation, investigations performed and surgical method of management of the perforation in these neonates. The complications encountered in these cases with varying underlying etiologies are also analyzed.

**Results**

Most of the patients (83%) presented in first week of life with mean age at presentation of 6 days. 10 patients (30%) were weighing less than 2500 grams (LBW) whereas 3 patients (9%) weighing less than 1500 grams were VLBW.



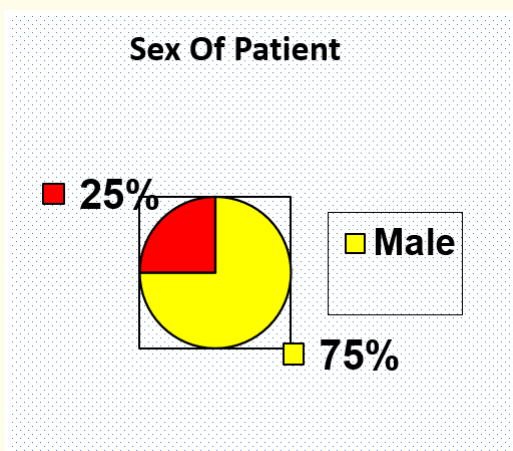
*Figure 1*

Age (Days)	No. of Patients
0	1
1	1
2	4
3	5
4	10
5	8
6	10
7	0
7-15	7
>15	2

**Table 1:** Number of patients according to age (days).

Weight (in grams)	No. of Patients
< 1500	03
< 2500	10
> 2500	19

**Table 2:** Birth weight and number of patients.



**Figure 2**

Most of the cases presented with abdominal distension, vomiting, constipation and failure to pass meconium in descending order of frequency [42].

A good number of these neonates were referred from neonatology unit of pediatrics facility of the hospital whereas remaining cases were initially seen and managed by GPs or pediatrician of the area and later referred to surgical emergency of The childrens hospital and the institute of child health, Lahore.

**Clinical presentation**

<b>Abdominal distension</b>	<b>95%</b>
Vomiting	90%
Constipation	71%
Failure to pass meconium.	60%
Septicemia	55%
DIC	30%

**Table 3:** Clinical presentation.

Disease entity	No. of Patients
NEC	28
Meconium ileus (complicated)	10
Spontaneous Gastric perforation	03
Anorectal Malformation	04
Colonic atresia	02
Hirschsprung’s disease	01

**Table 4:** Pathological entities encountered.

Etiologies included Necrotizing Enterocolitis 28 patients (58%) [37,42], Complicated meconium ileus 10 patients (20%), spontaneous gastric perforation [36] 03 patients (6%), Bowel atresias 02 patients (6%), Hirschsprung’s disease 01 patient (3%) and anorectal malformations 04 patients (8%) [39,43].

A total of forty-six neonates were managed surgically, whereas two were considered high risk patients and managed with intraperitoneal drainage under local anesthesia [44]. There were 16 (33%) deaths. Most of the mortality occurred in patients with complicated meconium ileus and Necrotizing enterocolitis, whereas all neonates with idiopathic gastric perforations survived. The site of perforation and underlying etiology has an important bearing on the ultimate outcome and this is an important prognostic consideration.

**Investigations**

Routine investigations as performed in these cases are shown in the following table.

CBC
CRP
HBsAg, Anti HCV
PT/APTT
LFTs / RFTs
X- ray (s) Abdomen, supine, erect, decubitus views
Abdominal Ultrasonography/Paracentesis or Drain

**Table 5:** Investigations.

Abdominal sonography and serum Profiles (Liver/Renal) were performed in all cases. Abdominal paracentesis was not performed as a diagnostic modality in our study and abdominal drain under local anesthesia was instituted whereas sweat Chloride and other specific tests for cystic fibrosis are not available in our set up.



**Figure 3**



Figure 4

**Management**

Operative intervention was decided on the basis of abdominal distension, erythema/edema of abdominal wall, abdominal mass, and pneumoperitoneum on radiography along with presence of fluid collections/sludge on sonography in selected cases.

Repair of intestinal perforations	13 (total)
Gastric perforations	05
Jejunal/Ileal perforations	06
Colonic perforations	02
End to end anastomosis	05 (total)
Small intestine	04
Ileocolic	01
Stoma formation	19 (total)
Ileostomy	11
Colostomy	08

**Table 6:** Operative procedures performed.

There were 28 cases of NEC (08 cases of isolated small intestinal perforations -repaired primarily, and in 20 cases with multiple perforations stomas were created- 11 loop ileostomies and 3 colostomies. whereas 05 end to end anastomosis were done including 03 illeo-ileal and two ileo-colic anastomosis [37,38,42].

All cases of Gastric perforations were managed with primary closure of perforation (4 perforations on anterior wall and one on the posterior wall without any gastrostomy) [36,40,43,51].

There were 10 cases of meconium ileus [41] (06 complicated with ileal atresia, 02 with ileal atresia and volvulus and 02 with meconium peritonitis) In all of these cases ileostomy was created as primary procedure 4 cases of anorectal malformations complicated by bowel

perforations were managed (2 perforations were exteriorized as colostomy and 2 were repaired primarily with proximal colostomy) Two cases of colonic atresias were managed (2 cases of left colonic atresia one with perforation distally and other with caecal perforation -exteriorization of perforation performed in both).

One case of Hirschsprung’s Disease presenting with Caecal perforation was managed by proximal loop ileostomy, repair of perforation and distal extra mucosal muscle biopsies. Mortality was 16ncases including 09 cases of NEC, 05 cases of complicated meconium ileus, and one case of Imperforate anus and 01 case of colonic atresia.

12 cases were complicated by septicemia/DIC including 5 cases of complicated meconium ileus, 4 of NEC, 3 of imperforate anus All cases of septicemia were given broad spectrum antibiotics, fresh frozen plasma, platelet transfusions and fresh blood transfusions as needed in individual case.

Leakage of anastomosis occurred in two cases of NEC complicated with septicemia and major abdominal wound dehiscence.

Septicemia	12 (total)
Abdominal wound infection	12
Minor	10
Major (wound dehiscence)	02
Leakage of anastomosis	02
Stoma complications	
Bleeding	06
Necrosis	03
Retraction	02

**Table 7:** Complications encountered.

Stoma complications occurred in 11 (58%) out of 19 cases and include bleeding managed with FFP, Platelet transfusions. Vit K and stoma care. In one case bleeding point was suture ligated. Stoma necrosis was seen in 03 cases of NEC.

2 cases of NEC were complicated by stoma retraction managed by revision of stomas.

**Discussion**

First indication of perforation usually occurs at 4 - 5 days of life. Abdominal distension is frequently abrupt and progressive signs of hypovolemia and low perfusion are also present in these cases of intestinal perforation. The respiratory difficulty from pneumoperitoneum may be first indication. Early on radiographs may not reveal any pneumoperitoneum [10] which becomes obvious with the passage of time [19]. Infants born of pregnancies complicated by abruption placentae, placenta previa, amnionitis and infants delivered by emergency cesarean section are at increased risk and should be carefully observed for 4 - 5 days.

NEC is one of the most common serious acquired gastrointestinal conditions to occur in the neonatal period, accounting for 1% to 5% of all nursery admissions with high mortality rate [16,47,48]. The pathological features of NEC include intestinal mucosal hemorrhage, ulceration and in advanced cases, full thickness necrosis with perforation. Several risk factors for NEC has been proposed and include prematurity, the presence of bacteria within the lumen of GIT, and enteral feeding. These components probably act in concert to upset an already immune and delicate gastrointestinal mucosal barrier [26,27]. Epidermal growth factor produced primarily in the saliva, duodenum, kidney and breast milk is trophic to GIT [28]. Exogenous administration of EGF has been reported to salvage a critically ill neonate with NEC [29,31]. NEC results in variable degrees of ischemic necrosis of small and large intestine of neonate. Simultaneous involvement

of small intestine and colon in 44%, colon alone in 26% and small intestine alone in 30%. Guaiac positive gastric aspirates are a common clinical presentation of NEC. The most common histopathological microscopic finding in NEC is coagulation necrosis and other features include acute and chronic inflammation, bacterial overgrowth and pneumatosis intestinalis. Blood cultures, peritoneal cultures and intestinal cultures grow common enteric organisms such as *Klebsiella*, *E. coli* and *Clostridial* species. There are reports of association of NEC with specific viruses such as rotavirus, corona virus and coxsackie B2 [47,48].

The various surgical procedures for management of patients with NEC include: peritoneal drainage, laparotomy and defunctioning enterostomy, resection of the necrotic segment and proximal enterostomy and with creation of multiple stomas/multiple anastomosis in distal small bowel creating mucous fistula [9,37,38,42,47].

In our series there were 28 cases of NEC, all presenting with pneumoperitoneum consequent upon perforation with abdominal distension. Laparotomy was performed in all cases with repair of isolated small intestinal perforation in 6 cases, proximal enterostomy in 15 with multiple perforations /pan necrosis In 5 cases end to end bowel anastomosis was performed using single layer interrupted coated vicryl sutures.

Complications encountered in cases of NEC include necrosis of stoma in 5 cases, problematic bleeding in 3 cases, Leakage of anastomosis in 2 cases followed by reexploration in 1 case where proximal enterostomy and mucous fistula was fashioned. 8 patients died post operatively including 4 cases of ileostomy, 2 cases of end to end anastomosis that leaked and 1 case where primary closure was performed. Two sick patients having VLBW with NEC were managed with peritoneal drainage procedures developed septicemia followed by death in both cases [22,44,48,50].

The survival of neonates operated for NEC at our Hospital was 53% (18 of 28 cases) which is comparable to figures reported from University of New Mexico Hospital [9].

Various pathophysiological explanations for gastric perforations [43] are congenital muscular defects increased gastric acidity and compression of fluid filled stomach during birth [21]. Kieswetter suggested that a hypothalamic pituitary adrenal mechanism activated by perinatal stress including anoxia could result in gastromalacia and subsequent perforation of the stomach. Asphyxia at birth in infants with low Apgar score is especially likely to lead to intestinal perforation. Redistribution of blood flow during hypoxia and hypovolemia or other stress states with shunting of blood away from mesenteric vascular bed is thought to result in microvascular injury and subsequent loss of mucosal integrity [24,25]. Persistence of ischemic insult allowing extension of microvascular thrombosis leads to transmural necrosis seen in intestinal perforation. Indomethacin therapy in low birth weight infants has been implicated in gastrointestinal perforations.

There were 3 cases of spontaneous gastric perforations, all presented with massive pneumoperitoneum on radiography, followed by laparotomy, repair of perforation with coated vicryl sutures and nasogastric aspiration postoperatively. All of these neonates survived having excellent postoperative course without any significant complications [49]. Excellent results in these cases of gastric perforations are probably due to differences in pathogenesis, excellent blood supply of the involved segment of intestine, use of broad spectrum antibiotics and of course, good birth weights seen in these neonates.

Meconium contamination of peritoneum in utero or in immediate perinatal period causes sever peritonitis usually with calcification [45]. The probable cause of meconium soliage of the peritoneum is in utero perforation of the obstructed bowel as a result of obstruction and distension of involved segment of intestine. The presentation of meconium peritonitis in the newborn is classified into four types which are meconium pseudocyst, adhesive meconium peritonitis, meconium ascites and infected meconium peritonitis [34,36]. The clinical presentation of the newborn with meconium peritonitis includes abdominal distension, a palpable abdominal mass and as erythema-

tous edematous abdominal wall and occasionally respiratory compromise from abdominal distension. In these cases, third space fluid losses may be severe [35].

There were 10 cases of complicated meconium ileus operated in our series, all presenting with pneumoperitoneum. Laparotomy was performed in all of these patients followed by formation of ileostomy and mucous fistula. Post operatively irrigation of stomas was done using N-Acetylcystine and through NG tube. Broad spectrum antibiotics were instituted in all of these patients. There were 5 deaths in these cases of complicated meconium ileus and in all cases post op ileus/obstruction did not resolve and patients developed septicemia/sclerema followed by death.

Perforation of the colon in the newborn is the most serious and rare complication and review of the literature reveals a few case reports. At least in two third of cases colonic perforation have been found secondary to atresias, Hirschsprung's disease and necrotizing enterocolitis and in remaining cases as no cause could be ascertained, hence termed idiopathic. Efforts to explain the etiology has given rise to various speculations such as trauma, infection, Schwatzmann's phenomena or hypoxia induced ischemia leading to colonic wall weakness. The perforation associated with GI Malformations can be explained on the basis of closed loop obstruction with diastasis rupture [33].

4 newborns with imperforate anus complicated with pneumoperitoneum were managed by Laparotomy and exteriorization of perforation in 2 cases and repair of perforation and proximal colostomy in other [2,32]. 3 of these neonates survived while 4<sup>th</sup> succumbed to death because of septicemia 2 cases of colonic atresia were managed with exteriorization of perforation in both cases and both survived without any significant morbidity.

1 case of Hirschsprung's disease complicated by caecal perforation was managed with proximal colostomy and repair of perforation.

Delay in diagnosis/ delayed referral to pediatric surgical facility, low birth weight and septicemia consequent upon fecal peritonitis were among most significant contributory factors for high mortality in these tiny sick neonates.

### Conclusion

- Pneumoperitoneum in neonates is a common presentation of various pathological processes as described in discussion section
- NEC is a major cause of morbidity and mortality in these patients presenting with Pneumoperitoneum.
- NEC is a continuously evolving disease and can lead to stricture formation in healed segments of intestine and can lead to death in sick neonates.
- Pneumoperitoneum may be a lethal complication of late presentation in cases of imperforate anus and Hirschsprung's disease.
- All those health professionals caring for Sick newborn and neonates presenting with Pneumoperitoneum should have a thorough knowledge and understanding of these pathological processes that lead to intestinal rupture / perforation and management options.

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