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NEONATAL GASTROINTESTINAL PERFORATIONS

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Purpose: *Neonatal gastrointestinal perforations continue to be associated with high morbidity and mortality rates. The aim of this study was to define causes, risk factors, and management of neonatal gastrointestinal perforations; and to analyze factors relevant to outcome.*

Methods: *The medical records of 45 neonates treated at Tanta University Hospital for gastrointestinal perforations during the last 6 years were retrospectively reviewed.*

Results: *Forty-five infants (25 males and 20 females) were analyzed. Their birth weight ranged from 1100 to 3500 grams (mean 2200 ± 750 grams). Perforations occurred from birth to 26 days (mean 10.5 days). Main causes of perforations included necrotizing enterocolitis (NEC) (21, 46.7%), spontaneous gastroduodenal perforation (5, 11%), iatrogenic colorectal perforations (4, 8.8%), spontaneous intestinal perforations (3, 6.6%), Hirschsprung's disease (2, 4.4%), and meconium ileus (2, 4.4%). The terminal ileum and cecum were the most frequent sites of perforation. All patients underwent laparotmy except one patient, who died prior to surgical intervention. There were 19 deaths (42.2%). Nearly half of deaths (9, 47.4%) occurred in infants with NEC.*

Conclusions: *1: Septicemia, low birth weight, prematurity and delayed recognition of perforation were responsible for the majority of deaths in our series; 2: Although the overall mortality in this series compares favorably with similar other large series in the literature, there is still a room for improvement should iatrogenic perforations and delay in diagnosis be avoided: and 3: Simple closure for isolated perforations or resection and primary anastomosis is the treatment of choice, however, creation of stoma in some instances may be warranted.*

Keywords: Gastrointestinal perforations, necrotizing enterocolitis, Neonatal peritonitis.

INTRODUCTION

Gastrointestinal perforation is a life threatening complication in neonates and continues to be associated with mortality rates of 40% to 70% [1-8]. Infants with gastrointestinal perforations comprise a heterogeneous group of patients ranging from very sick prematures [1,8], to otherwise healthy full–term babies [9]. The causes of such perforations are diverse and knowledge of such causes is of great importance for successful management[3]. The purpose of the present study is to outline causes of perforations, results of various surgical procedures used in our unit and to analyze factors relevant to outcome.

PATIENTS AND METHODS

A retrospective review of the medical records of all neonates with gastrointestinal perforations treated at NICU of Tanta University Hospital from November 1993 to November 1999 was done. The records were reviewed for birth weight, gestational age, sex, age at presentation, site, extent and cause of perforation, associated anomalies, predisposing factors, causes of delayed diagnosis, surgical procedures and outcome.

RESULTS

There were 45 infants (25 males and 20 females) with

an average gestational age of 36 ± 4.2 weeks. Their birth weight ranged from 1100 to 3500 grams (mean 2200 ± 750 grams). Perforations occurred from birth to 26 days (mean 10.5 days). The causes of perforations are summarized in (Table 1). Main causes of perforations included NEC (21, 46.7%), spontaneous gastroduodenal perforations (5, 11%), iatrogenic colorectal perforation (4, 8.8%), spontaneous intestinal perforations (3, 6.6%), Hirschsprung's disease (2, 4.4%), and meconium ileus (2, 4.4%).

Table(1): ***Causes of perforations and mortality***

Cause of GI perforations	*No of patients*	*Mortality (%)**
NEC	21	9 (47.4%)
Spontaneous gastrodudenal Perforation	5	3 (15.8%)
Hirschsprung's disease	2	0 (0%)
Iatrogenic perforations	4	2 (10.5 %)
Meconium ileus	2	1 (5.3%)
Intestinal atresia	2	0 (0 %)
Volvulus	1	1 (5.3%)
Spontaneous intestinal perforation	3	0 (0%)
Unknown	5	3 (15.8%)
Total	45	19 (100%)

* % of total deaths

The sites of perforations are summarized in (Table 2) Sixteen patients (35.6%) had perforations in the small intestine, 14 (31.1%) in the large bowel, 4 (8.9%) in the stomach and 1 in the duodenum. Four patients (8.9%) had extensive type of NEC involving large areas of small and large intestine with multiple sites of perforations, all of these 4 patients died.

Table(2): ***Sites of perforations and relationship to associated NEC***

	NEC	*Non - NEC*	*Total - %*
Stomach-duodenum	0	5	5 (11.1%)
Small bowel	10	6	16 (35.6%)
Colorectum	6	8	14 (31.1)
Small & large bowel	4	0	4 (8.9%)
Unknown	1	5	6 (13.3%)
Total	21 (46.7%)	24 (53.3%)	45 (100%)

The site of perforation was unknown in 6 patients. All of them had well documented pneumoperitoneum. One premature with NEC and fulminating sepsis died before laparotomy. The sites of perforations were not detected during laparotomy despite intra-operative meticulous search in the other 5 patients. Three of the 5 infants were preterm. Mild to moderate respiratory distress was noted in 3, but non-of them was on mechanical ventilation. Mild clear intraperitoneal fluid was noted in 2. The fluid was turbid in 2, and absent in one. An intra-peritoneal tube drain was inserted. No intestinal contents were drained through this tube in postoperative period. Three infants survived while 2 died.

NEC was the cause of perforation in 21 patients (46.7%) and the terminal ileum was the most common site of perforation. Ten of the 16 (62.5%) patients with small bowel perforations and 6 of the 14 with colorectal perforations (42.9%) had NEC.

Five infants (4 males and 1 female) presented with gastroduodenal perforations. Their ages ranged from 7 to 16 days (mean 10.5 days). All were very sick with severe abdominal distension causing respiratory distress (Fig.1&2). One baby had esophageal atresia and tracheoesophageal fistula type 3. Preoperative percutaneous decompression of pneumoperitoneum was urgently required for stabilization in 3 of them. Perforations were located at posterior wall near greater curvature in 3 cases (Fig. 3), at pyloric canal in one (Fig. 4), and at first part of duodenum in one. Only 2 of these 5 infants survived. The main causes of death were respiratory failure and fulminating sepsis.

Localized perforations without significant evidence of NEC in other parts of bowel were noted in 3 cases, 1 at terminal ileum and 2 at cecum. All were managed successfully with peritoneal lavage and simple closure of the defect. No deaths were encountered in those 3 patients.

Two children with Hirschsprung's disease presented with perforations, which were located proximal to the narrow aganglionic segments. Perforations occurred at sigmoid colon in one patient and at cecum in the other patient. Closure of perforation and colostomy proximal to transition zone was done. Both patients survived and undergone pull-through later. Two infants with delayed diagnosis of ileal atresia, and another 2 infants with meconium ileus presented with perforation at dilated proximal part. The perforation occurred most probably in uetro in both cases of meconium ileus, a significant intraperitoneal adhesions and calcifications were noted in both of them (Fig. 5).

Iatrogenic colorectal perforations occurred in 4 infants. Perforation occurred during performing contrast enema in 2 (Fig. 6A&B), and during colonic wash for suspected Hirschsprung's disease and meconium ileus in 2. The first 2 babies survived but the later 2 infants died.

Abdominal distension was the most consistent clinical finding. The distension was severe enough to cause

respiratory distress in 10 patients (22.2%). All cases with gastric perforations were included in this category. Other clinical findings included poor suckling and increased gastric residual (25, 55.5%), vomiting (16, 35.6%) lethargy (15, 33.3%) and bleeding per rectum (13, 28.9%).

Diagnosis of perforations was established by the presence of pneumoperitoneum in 40 (88.9%) patients. In 5 (11.1%) patients, the perforation was detected during laparotmy performed for intestinal obstruction and peritonitis secondary to suspected gangrenous intestinal loops in babies with NEC. Delayed recognition of perforation was documented in 10 cases. The delay ranged from 8 hours to 5 days, with a mean of 36 hours. All of these 10 infants had variable degrees of pneumoperitoneum, which was missed initially at plain abdominal radiograph done in supine position (Fig.7&8). Seven of these 10 patients died, 5 of the 7 infants had delay in diagnosis more than 2 days.

The various surgical procedures performed are shown in (Table 3) The type of surgery performed was dictated by the primary cause of perforation, the general condition of the baby and the local condition of bowel both at and around site of perforation. Closure of perforation or resection and primary anastomosis were the preferred methods whenever possible.

Table (3): ***Surgical procedures***

Surgical Procedures	*No of Patients*
Simple closure of the defect	12
Closure of perforation and stoma	6
Resction and primary end to end anastomosis	16
Resection and stoma	5
Drain only	5
No laparotomy	1

There were 19 deaths in our series (42.2%). Nearly half of deaths (9, 47.4%) occurred in infants with necrotizing enterocolitis. Four patients died because of extensive involvement of the bowel with NEC. Two patients died due to delayed diagnosis of iatrogenic perforation. Patients who had gestational age more than 33 weeks and those with a birth weight more than 2500 grams had lower mortality rate (Table 4). Delay in diagnosis, low birth weight, prematurity and septicemia were other contributing factors to mortality.

Table (4): ***Relationship mortality, gestational age and birth weight***

	Mortality/ Total (%)
Gestational age	
<33 weeks	5/ 10 (50 %)
> 33 weeks	14/ 35 (40 %)
Birth weight	
< 2000 g	7/12 (58.3%)
>2000 g	12/ 33 (36.4%)

There were 20 significant complications in the 26-surviving patients. Wound infection[7], stomal complications[6], anastomotic leak[3], incisional hernia[3] ands wound dehiscence[1] were the most significant complications.

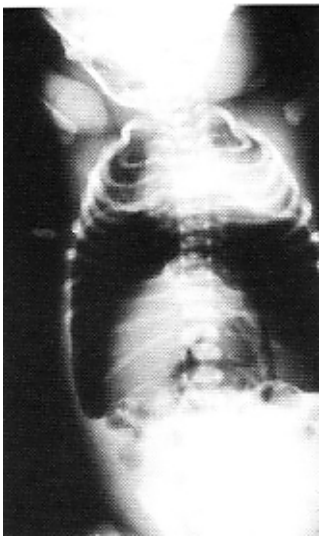


Fig. (1): Plain x-ray babygram, erect position, in 7 day- old pre-term with spontaneous gastric perforation. A massive pneumoperitonium pushing diaphragm upwards, liver and spleen downwards (saddle shape sign).

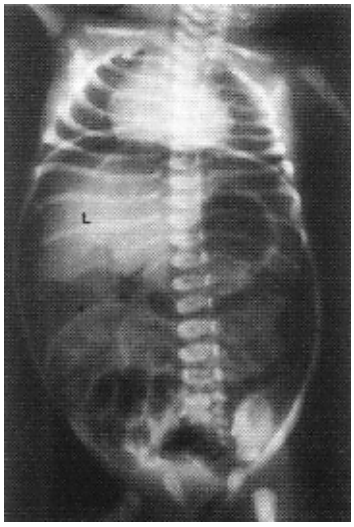


Fig. (2): Plain x-ray abdomen (supine position) in 10-day-old infant with duodenal perforation showing Rigler sign (well-visualized intestinal wall), arrow head, well-visualized liver (L) edge silhouette, air triangles at flank (arrows).

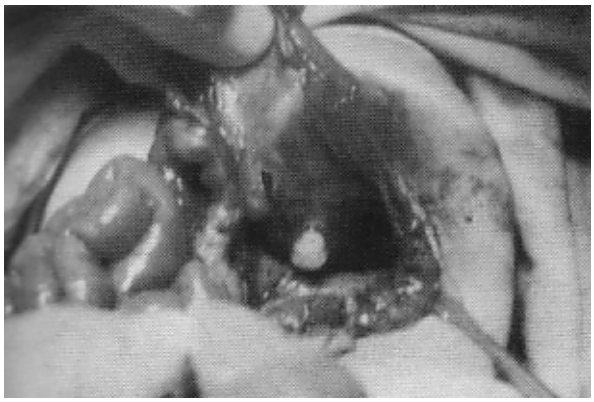


Fig. (3): Operative photograph of 13 day- old baby with spontaneous gastric perforation showing a 2 cm circular perforation at posterior wall of stomach near greater curvature

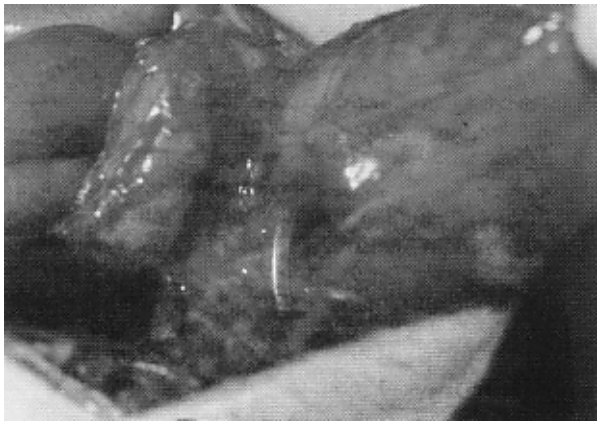


Fig. (4): Operative photograph showing gastric perforation at pyloric canal. The nasogastric tube is passing through the rent.

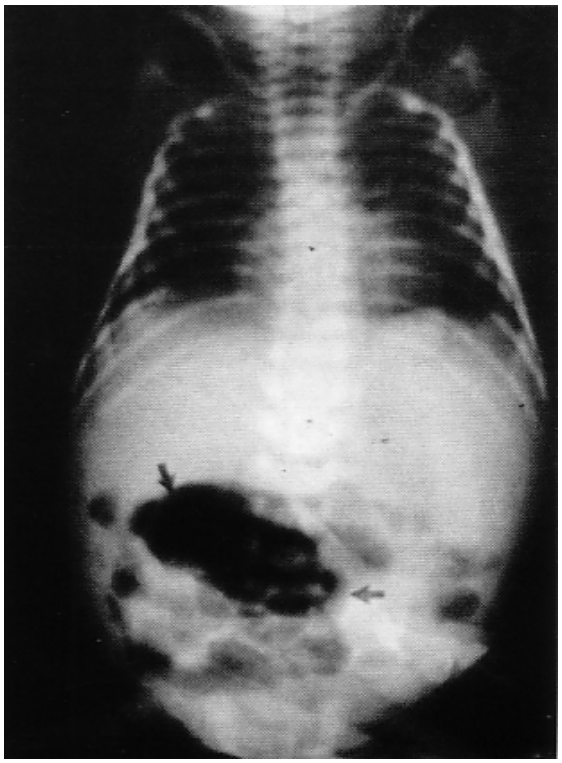
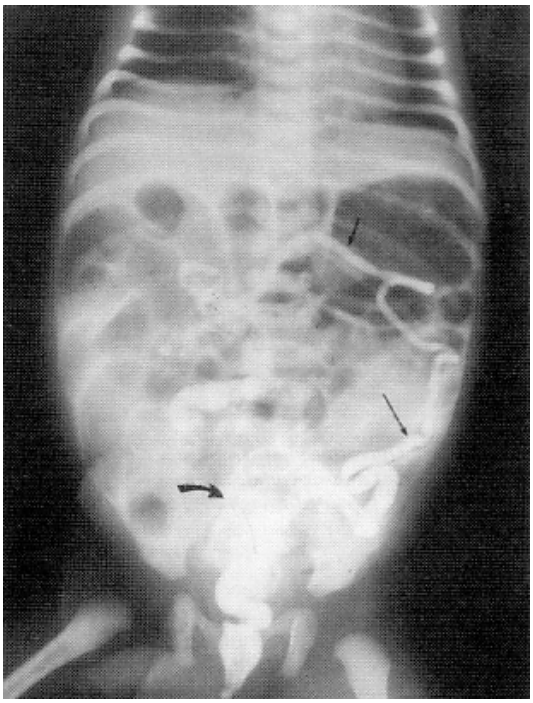


Fig. (5): Plain x-ray babygram, supine position, in 3-day-old baby with complicated mechonium ileus. A well-localized globular air (arrows) and fine calcifications is noted.



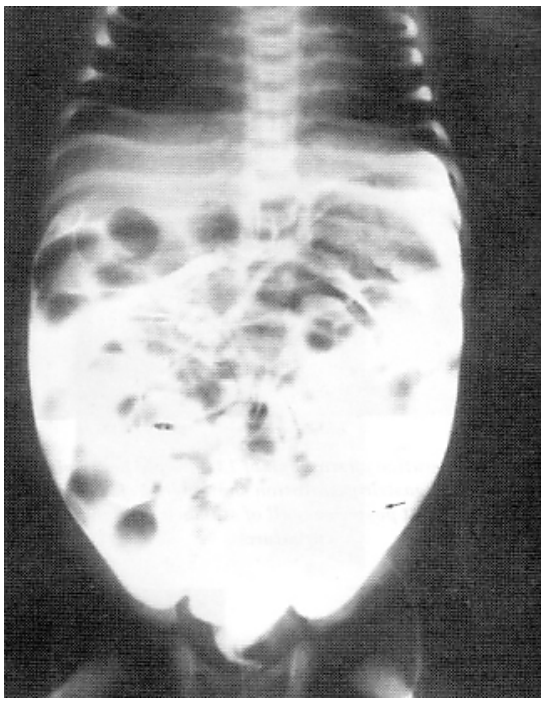


Fig. (6 A & B): Contrast enema in 2 day-old baby with ileal atresia showing an unused colon (arrows) and site of iatrogenic Perforation at the rectosigmoid (curved arrow).

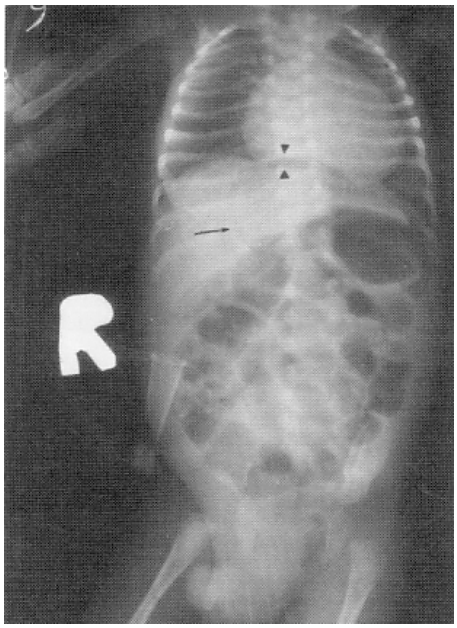


Fig. (7): Plain x-ray babygram, supine position, shows minimal free air " anterosuperior oval" which was missed mitially.

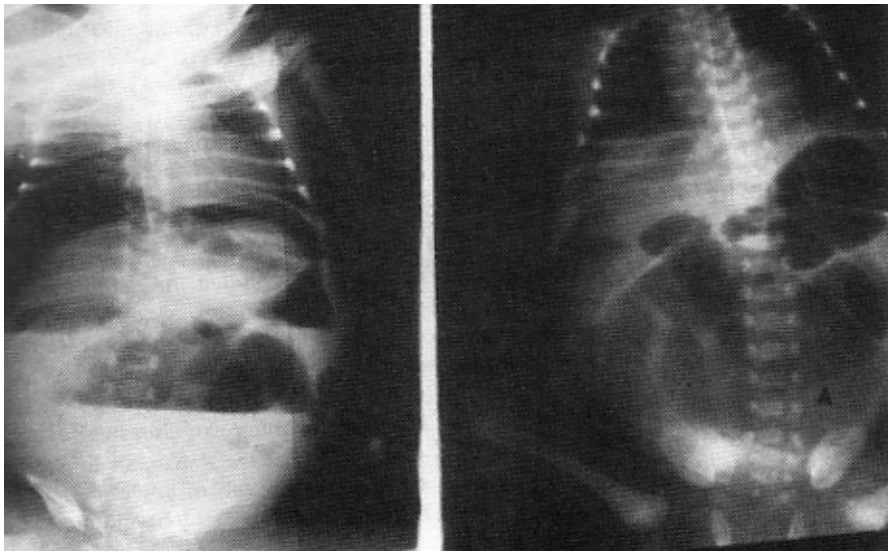


Fig. (8): Plain x- ray at supine position (right) and erect position (left) in the same patient, demonstrating various radiological signs of pneumopritoneum.

DISCUSSION

Despite advances in perinatal care, gastrointestinal perforation continues to carry 40% to 70% mortality[1-9]. NEC has been ranked as the leading cause in many large series[4,10-12]. As a result of improved perinatal care, the incidence of NEC is continuously rising as more low birth weight babies survive[13]. Consequently, the overall incidence of perforation of the bowel is on rise also in most large neonatal surgical units[10]. Perforations was the indication of laparotmy in over half of infants with NEC who undergone surgery in some series [11]. Although NEC was the predominant cause of perforation in our series, its percentage (46.7%) was significantly less than the 62.5% and 68% reported by others [3,10].

Spontaneous gastric perforation was the second most common entity in our series as well as in some other large series[3.10]. Similar to many other series, our infants with spontaneous gastric perforations showed some characteristic features such as: male preponderance, the location of perforation at greater curvature of upper part of stomach in the majority of cases, and the timing of perforation within first 2 weeks of life [14,15]. The etiology of spontaneous neonatal gastric perforation is still unknown. gastric tissue ischemia secondary to hypoxia[16], Congenital muscular defect[17,18], mechanical disruptions, trauma during vigorous resuscitation, have all been incriminated as possible etiologic factors[14,15]. Nasogastric tubes seemed to have played a rule at least in one of our cases where the tube was found through the rent. A similar observation was reported previously [19].

Although our 5 infants with gastroduodenal perforations were very sick, 2 of them survived. The mortality rates among infants with spontaneous gasroduodenal perforations differ considerably from one to another series. Rosser et al [14] reported 16 cases of spontaneous neonatal gastric perforation over a period of 10 years with an overall mortality rate of 25%. Holgersen[15] reported 28 infants with nine deaths (32%). Tan et al[4] reported *5* cases with a 60% mortality. This is in contrast to St-Vil et al[10] series in which a 100% survival was noted among 7 infants with gastroduodenal perforations.

Three infants in our series developed localized bowel perforations with no evidence of antecedent disease of the bowel. In 1987, Aschner et al[20] described a syndrome of idiopathic spontaneous intestinal perforation (SIP) in very low birth weight infants. Cases of SIP were defined as the presence at laparotomtv or autopsy of isolated intestinal perforations surrounded by normal-appearing bowel and by the absence of characteristic gross or microscopic features of NEC[8,20,21]. SIP typically presents in the first 2 weeks of life of very premature infants. The characteristic features of SIP consist of a relatively well looking infant with a prominent blue abdominal discoloration, Ieukocytosis, and a gasless abdominal radiograph[8,20,21]. The pathogenesis of SIP is not clear. Some investigators have hypothesized congenital muscular defects predisposing to perforation[22,23]. Others suggested that focal perforation may result from or are

predisposed to by intestinal ischemia.[21]

Although the operative findings in our 3 cases with SIP were very similar to those very low birth weight infants with standard SIP syndrome, yet their gestational ages as well as body weight were quite different. If our cases may, in fact, represent a very localized form of NEC or specific subtype of SIP, is not quite clear.

One of the alarming findings in our series was the relative high frequency of iatrogenic colorectal perforations (4, 8.8%). Unfortunately, these four cases of perforations were potentially preventable. In 2 cases, perforation occurred during performing contrast enema without fluoroscopy guidance. Colonic perforation caused by Nelton catheter inserted by inexperienced nursing staff during colonic lavage. Since that time, a strict policy for colonic wash out and contrast enema was adopted at our NICU with no further similar tragic accident encountered.

One of the unique subgroups in our series is those 5 newborns with pneumoperitoneum of obscure source, which have been reported previously by us[24]. Pneumopenitoneum does not necessarily mean gastrointestinal perforation[24-30]. Among the entities causing pneumopenitoneum without gastrointestinal perforations are tension pneumothorax and pneumomediastinurn, rupture pneumatosis intestinalis cystoides, and idiopathic pneumoperitoneum [25-29]. Zerela et al [25] reported 10 infants with pneumoperitoneum without gastrointestinal perforations. These infants had respiratory failure and were on mechanical ventilation. Few similar reports clearly documented this entity [25-29]. In contrary to the previous reported patients with pneumoperitoneum of chest origin, none of our 5 patients was on mechanical ventilation. These 5 infants, most probably, had spontaneous sealed minor perforations rather than pneumoperitoneum of chest origin. Whether the cause of perforation was a very localized form of NEC or a specific variant of SIP, is not clear.

Recently, the mortality rates of infants with neonatal gastrointestinal perforations have showed a decreasing trend and are currently around 30-36% [10]. This is considered a high mortality rate despite recent advances in perinatal care. Perhaps one of the reasons for the plateau in overall survival rates is increasing proportion of NEC related perforations which reached 73% in some series [10].

The prognosis of infants with perforated viscus depends on several factors. These include birth weight, gestational age, extent and type of underlying pathology and the severity of associated anomalies [30]. Tan et al [4] found in their series that survival was better in female patients with colorectal perforations, while St-Vil et al [10] found birth weight to be of more prognostic significance when compared to gestational age. Both birth weight and gestational age proved to be of prognostic significance in our series as in some other series[3].

The delay in diagnosis of perforation carries a very important prognostic value. The two infants, who had immediate recognition of iatrogenic perforation during performing contrast enema, were saved following urgent laparotmy. On contrast, delayed recognition of this complication resulted in death of 2 infants with iatrogenic bowel perforation during colonic wash out. The main causes of delay in diagnosis of perforation were infrequent taking abdominal radiograph and lack of recognition of various radiological signs of pneumoperitoneum in supine position such as the "anterosuperior oval", "air dome" or "football" sign, free air at the flanks, air in the scrotum, and Rigler's sign (apparent wall of bowel due to presence of air both inside their lumen and free pnumoperitoneum) [31, 32].

The surgical approach must be tailored to the situation. Recent trends in the management of gastrointestinal perforation are moving toward simple closure or resection and primary anastomosis[33,34]. This recent trend has its impact on decreasing length of hospital stay, the period of total parental nutrition, the time to full feeds and the time on ventilator [35]. Our data is in agreement with others[3,33-37], who advocate immediate reconstruction of bowel continuity as the best form of treatment and reserve creation of stoma in specific instances when circumstances may warrant that.

CONCLUSIONS

The frequency of gastrointestinal perforations is on rise in our NICU. Based on our results we may conclude that: 1. Septicemia, low birth weight, prematurity and delayed recognition of perforation were responsible for the majority of deaths in our series; 2. The relative high number of iatrogenic perforations in this series dictates a strict policy for colonic wash and rectal manipulations; 3. Plain abdominal radiographs should be repeated more frequently in every sick newborn with abdominal distension; 4. Awareness with different radiological signs of pneumoperitoneum in supine plain x-ray film is crucial to avoid delay in diagnosis of perforation; and 5. Although simple closure for isolated perforations or resection and primary anastomosis is the treatment of choice, creation of stoma in some instances may be preferred.

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