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Research Article

Neonatal Perforated Gut: Etiology and Risk Factors

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Abstract

Despite the recent improvement of neonatal management, gastrointestinal perforation in the neonatal period is still a major challenge for neonatologists and pediatric surgeons. Neonatal bowel perforation may be due to necrotizing enterocolitis (NEC), mechanical obstruction or idiopathic. The prognosis of infants with perforated viscus depends on several factors. These include birth weight, gestational age, extent and type of underlying pathology.

Keywords: *Neonatal Bowel Perforation; Necrotizing Enterocolitis; Spontaneous Intestinal Perforation; Pneumoperitoneum; Peritoneal Drainage*

Abbreviations

NEC: Neonatal Bowel Perforation; SIP: Spontaneous Intestinal Perforation, Pneumoperitoneum, Peritoneal Drainage; CBC: Complete Blood Count; CRP: C-Reactive Protein; ABG: Arterial Blood Gases; PD: Peritoneal Drainage

Introduction

Although there had been a prolific improvement of neonatal management, gastrointestinal perforation during the neonatal period is still a major problem for neonatologists and pediatric surgeons [1]. It may result from NEC, mechanical obstruction or idiopathic. Perforations due to mechanical intestinal obstruction as in Hirschsprung's disease (HD), intestinal atresia, imperforate anus, and meconium ileus are uncommon nowadays due to earlier diagnosis and treatment [2]. However, NEC remains the major cause of bowel perforation especially in premature babies [3].

Aim of the Study

The aim of this study is to highlight the possible causes, clinical presentation, operative procedures and the outcome of neonatal gut perforation admitted to Alexandria University Hospital.

Materials and Methods

The type of our study was a prospective cohort one, carried on neonates suffering from gastrointestinal perforation who were admitted to Alexandria University Hospital in the period between January to December 2018. It included all neonates presenting clinically or radiologically with perforated gut; however, cases that had major cardiac or neurological associated anomalies that may affect the outcome were excluded.

All cases were subjected to meticulous history with emphasis on antenatal history, postnatal history including gestational age, birth weight and Apgar score. Enquiry about the type of feeding, whether breast feeding or bottle feeding, was also done. Then, the cases

were examined thoroughly to check for any associated anomalies as well as features of shock and sepsis. Local examination focused on searching for abdominal distension, abdominal wall edema, tenderness, ecchymosis and visible loops. Digital rectal examination was done to check for empty rectum or bloody stools.

Investigations were done including complete blood count (CBC), with special attention to white blood cells and platelet count, serum electrolytes, coagulation profile, C-reactive protein (CRP) and arterial blood gases (ABG). Plain X-ray abdomen (anteroposterior) in standing position was done for all cases to detect air under diaphragm (Figure 1), air-fluid level, pneumatosis intestinalis, portal vein gas or fixed loop sign.

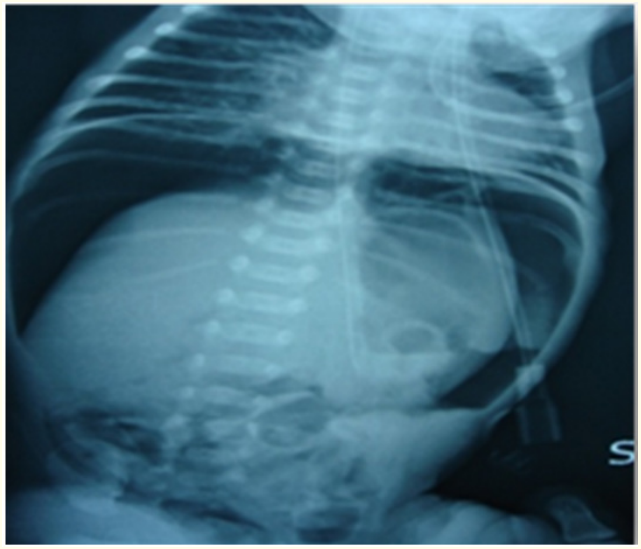

Figure 1: *Erect plain x-ray abdomen (PA view) showing pneumoperitoneum.*

All patients were resuscitated as per standard protocol. Nothing per os (NPO), a nasogastric tube (NGT) was placed to decompress the stomach, volume resuscitation with isotonic fluids together with antibiotic administration. Close clinical observation for these patients in the form of frequent physical examination, abdominal radiography, platelet and leukocytic counts and blood gas determination.

Peritoneal drainage (PD) was ideally performed on very low birth weight (VLBW) infants who are unstable and demonstrate either pneumoperitoneum or meconium staining of the abdominal wall. If no improvement, a laparotomy was performed. Laparotomy was done and according to the operative finding either: Resection with enterostomy or resection and primary anastomosis. Postoperatively, IV fluids, antibiotics coverage for anaerobic and aerobic bacteria were continued for 7 - 10 days, together with gastric suction, inotropic medications if needed, serial CBC, CRP, ABGs, plain erect X-ray. Oral feedings were resumed slowly using elemental formula after 10 - 14 days.

Results

During 2018, 50 cases were admitted to our hospital with perforated gut. The details of those cases are summarized in table 1. There were 36 males and 14 females. About two-fifth of the cases were presented after the 3rd week. Weight range was 1500 - 4700g (mean of 3100g), with more than a half were below 2500 g. About 40% of the cases were operated within 24 hours of admission; however, about one-third of the cases required more than 72 hours before operation took place.

	No. (%)
Age (Days)	
Less than 10 days	15 (30%)
10 - 20 days	16 (32%)
More than 20 days	19 (38%)
Sex	
Male	36 (72%)
Female	14 (28%)
Presenting weight	
Less than 2500 gram	26 (52%)
2500 - 3500 gram	10 (20%)
More than 3500	14 (28%)
Site of pathology	
Jejunum	6 (12%)
Ileum	18 (36%)
Meckel's	3 (6 %)
Colon	18 (36%)
Unidentified	5 (10%)
Cause	
Spontaneous intestinal Perforation	18 (36%)
NEC	13 (26%)
Hirschsprung's disease	8 (16%)
Meconium ileus	4 (8%)
Meckel's diverticulum	3 (6%)
Anorectal malformation	1 (2%)
Unknown	3 (6%)
Duration between admission and operation	
Less than 24 hours	20 (40%)
24 – 27 hours	13 (26%)
More than 72 hours	17 (34%)
Surgical intervention	
Drain	13 (26%)
Resection and anastomosis	10 (20%)
Resection and stoma	27 (54%)

Table 1: *Distribution of the studied cases according to different parameters (n= 50).*

Qualitative data was expressed using number and percent.

At surgery, ileum and colon were the commonest sites of perforation, 36% each. Others sites were jejunum (6 cases) and Meckel's diverticulum in a 3 case. The site of perforation was unidentified in 5 cases due to the presence of sealed perforation. The highest mortality was noted in jejunal ones. Necrotizing enterocolitis was the most common cause of perforation as well as the highest mortality in our study. It comprised 18 cases, 15 of which were premature, and all had been fed infant formula feeds prior to onset of symptoms. Spontaneous intestinal perforation was next in frequency. Other obstructive pathologies causing perforation were also implicated.

All the babies underwent exploratory laparotomy. Thirteen cases very sick preterm babies, so they underwent an initial PD and later delayed laparotomy due to non-improvement of their clinical state. Corrective surgical procedures done include resection and enterostomy (n = 27), resection and anastomosis (n = 10). Another two had repeat surgeries (enterostomies) for anastomotic failures. With regards to outcome, 21 babies died giving an overall mortality of 42% (Figure 2). Mortality was associated with young ager age of presentation, delayed surgery, low birth weight, perforations at the jejunum and perforation due to NEC, with the last 3 showing statistical significance.

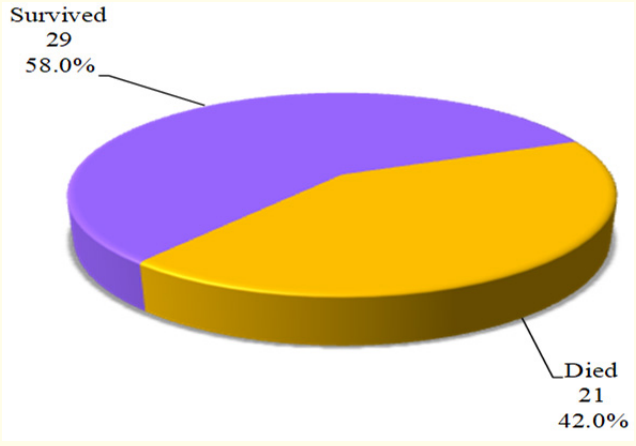

Figure 2: *Distribution of the studied cases according to outcome (n = 50).*

Discussion

Necrotizing enterocolitis (NEC) represented the major cause of neonatal gut perforation in our series. Worldwide, necrotizing enterocolitis (NEC) is the leading cause of neonatal gut perforation exceeding other causes like spontaneous intestinal perforation (SIP) and mechanical obstruction [4]. Owing to the increased survival of premature and critical ill neonates, NEC incidence is expectedly rising. Up to 90% of NEC occurs in preterms [5]. Intestinal perforation occurs in about one fifth of those babies who develop NEC [6]. Perforation is often multiple. Exposure of neonates especially preterms to artificial milk with its high substrate load increases the risk of NEC and perforation [7]. In our series all the babies with perforation from NEC were fed formula milk.

Pneumoperitoneum in clinically suspicious patients is often considered as an indicator of bowel perforation and the need for a surgical intervention. However, pneumoperitoneum can occur without perforation as seen in children on mechanical ventilation or pneumomediastinum, or may be idiopathic [8]. Pneumoperitoneum can be found in about 63% of infants with perforated gut [9]. In our study, only 36 (72%) had intraperitoneal free gas. The proposed reasons for absence of pneumoperitoneum in x-ray films are may be due to gas reabsorption. Moreover, it had been postulated that decompression of the extra-luminal gas especially in wide perforations involving the proximal bowel loops can be triggered by nasogastric decompression [10].

Turning to the treatment, PD may be effective in the initial phase of resuscitation for perforated gut by reducing abdominal distention. This helps in reducing mean airway pressure, and consequently, improves oxygenation index in those critically ill neonates [11]. Moreover, a significant proportion of infants will not require any subsequent operative procedure, therefore, will avoid laparotomy and enterostomy related morbidity and mortality. However, primary peritoneal drainage carries many drawbacks. First and foremost, it does not allow for confirmation of the location or extent of perforation. Secondly, drainage alone will eventually fail in infants with intestinal perforation secondary to circumferential necrosis of the intestine; these infants will either develop a fecal fistula to the drain site or complete bowel obstruction. In this condition, formal laparotomy enables the complete identification of the site, extent, and severity of the disease, as well as direct control of both bleeding and contamination [12,13].

As for the site of pathology, 4 out of 6 cases with jejunal involvement didn't survive, survival rate was only 33.3%. Better survival was noted in ileal lesions (11/18, i.e. 61.1%), and colonic lesions (14/18, i.e. 77.8%). Similar findings were noted by De Souza., *et al.* [14] who, by bivariate analysis, revealed that involvement of the jejunum and ileum was associated with high mortality rates (Table 2) and whenever the jejunum was involved with the disease mortality rate was 85.1%, hence poorer prognosis. This was attributed to the important role of jejunum in the process of absorption.

	Outcome		P
	Survived (n = 29)	Died (n = 21)	
Age (Days)			
Less than 10 days	7 (24.1%)	8 (38.1%)	0.568
10 - 20 days	10 (34.5%)	6 (28.6%)	
More than 20 days	12 (41.4%)	7 (33.3%)	
Presenting weight			
Less than 2500 gram	11 (37.9%)	15 (71.4%)	0.029*
2500 - 3500 gram	6 (20.7%)	4 (19.0%)	
More than 3500	12 (41.4%)	2 (9.5%)	
Site of pathology			
Jejunum	2 (6.9%)	4 (19.0%)	0.012*
Ileum	11 (37.9%)	7 (33.3%)	
Meckel's	2 (6.9%)	1 (4.8%)	
Colon	14 (48.3%)	4 (19.0%)	
Undefined	0 (0.0%)	5 (23.8%)	
Cause			
Necrotizing enterocolitis	6 (20.7%)	12 (57.1%)	0.028*
Spontaneous intestinal perforation	12 (41.4%)	1 (4.8%)	
Hirschsprung's disease	5 (17.2%)	3 (14.3%)	
Meconium ileus	2 (6.9%)	2 (9.5%)	
Meckel's diverticulum	2 (6.9%)	1 (4.8%)	
Anorectal malformation	1 (3.4%)	0 (0.0%)	
Unknown	1 (3.4%)	2 (9.5%)	
Duration between admission and operation			
Less than 24 hours	14 (48.3%)	6 (28.6%)	0.199
24 - 27 hours	8 (27.6%)	5 (23.8%)	
More than 72 hours	7 (24.1%)	10 (47.6%)	
Surgical intervention			
Drain	5 (17.2%)	8 (38.1%)	0.236
Resection and anastomosis	6 (20.7%)	4 (19.0%)	
Resection and stoma	18 (62.1%)	9 (42.9%)	

Table 2: *Relation between outcome and different parameters.*

Qualitative data was expressed using number and percent and was compared using Chi square or Monte Carlo test.

**: Statistically significant at p ≤ 0.05.*

The possibility of spontaneous healing of gut perforations in neonates has been described [15] and some authors now consider initial conservative management for intestinal perforation [16]. In 5 of our patients with clinical evidence of perforation was clinically and radiologically evident; however, on exploration, careful search failed to reveal any perforation points; but instead only evidence of healed perforation were seen. Being not very common, we eventually explore all neonates with clinical and radiologic evidence of gut perforation as preoperative identification of infants with sealed perforation diagnosis on preoperative basis is not easy, and clinical features may be confusing.

Mortality from neonatal intestinal perforation is obviously high (Table 2), ranging from 40 - 70% [17]. This persistent high mortality is attributed to recent advancements in neonatal intensive care and anesthesia that resulted in increasing survival of extreme premature babies [18]. Unlike NEC which accounts for a greater proportion of neonatal gastrointestinal perforations mortality, SIP and other mechanical obstructions carry a better prognosis [10].

Conclusion

To sum up, it is evident from our study that NEC, low birth weight, location of the perforation are identifiable mortality risk factors of neonatal gut perforation.

Conflict of Interest

Authors have nothing to declare.

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