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ABSTRACT

Background: **Gastro-intestinal tract (GIT) perforation in neonates is a serious problem associated with high mortality due to resulting sepsis. Co-morbid factors, eg. prematurity, respiratory problems, low birth weight, and nutritional factors, negatively affect the outcome.**

Objectives: **To review the management outcome of gastro-intestinal tract perforation in neonates in KwaZulu-Natal and identify factors that require attention for better survival of neonates with GIT perforation.**

Design: **Retrospective study of consecutive complete data sets of patients presenting with a diagnosis of GIT perforation.**

Setting: **Department of Paediatric Surgery, Nelson R. Mandela School of Medicine, University of Natal, Durban, South Africa.**

Subjects: **Fifty four neonates treated for gastro-intestinal tract perforation between January 1998 and January 2003.**

Main outcome measures: **Morbidity as determined by complications and mortality.**

Results: **More males (69%) were affected than females (31%). The median birth weight was 2.3 kg and median age at presentation was four days. Eighty nine percent were referred from peripheral hospitals. Abdominal distension was the leading symptom and sign (74%). Co-morbid factors were present in 89%, with prematurity as the leading factor (52%). Necrotising enterocolitis (NEC) was the main cause of perforation (33%) and the terminal ileum was the most common site. Most (56%) were treated by excision and primary repair of perforations. Sepsis was the leading complication (44%) and major cause of death (72%). Mortality was highest (56%) in perforations due to other primary pathology followed by NEC (53%). Overall mortality was 46%.**

Conclusion: **It is essential to prevent secondary perforations by early recognition and management of primary pathology. Management of pneumoperitoneum in neonates with respiratory difficulties should be included in resuscitation before transfer. Rectal temperature monitoring and herbal enemas should be strongly discouraged.**

INTRODUCTION

Neonatal gastro-intestinal (GI) tract perforation has a significant morbidity and mortality (1-11), and management poses a major challenge to the paediatric surgical team. Necrotizing enterocolitis (NEC) has been shown to be the predominant cause (4,6,7,9,10). Improvement in perinatal care is followed by an increase in the incidence of neonatal GI tract perforations due to an increase in the survival of neonates at risk (4,9).

Air and fluid shift into the peritoneal cavity following perforation resulting in abdominal distension, peritonitis and hypotension (5,6). Distension compromises respiration by splinting the diaphragm, the primary muscle of breathing in neonates. Clinical diagnosis of even advanced peritonitis may be difficult in neonates, yet delay in diagnosis results in rapid deterioration and sepsis (5,6).

The aim of the study was to review the clinical presentation, cause, management and outcome of neonatal GI tract perforations in KwaZulu-Natal in order to identify factors that require attention for better survival of neonates with perforation.

MATERIALS AND METHODS

Hospital computer records of neonates admitted with gastro-intestinal tract perforations and managed by the Department of Paediatric Surgery, Nelson R. Mandela School of Medicine, in KwaZulu-Natal between January 1998 and January 2003 were retrieved and analysed. The data analysed included patient demographics, mode of delivery, predominant symptoms and signs, diagnostic investigations, co-morbid factors, operative details, post operative complications and outcome.

RESULTS

Of the 54 neonates, 37 were males (69%) and 17 females (31%). Birth weight was available for 42 neonates ranging from 0.875 kg to 4.31 kg (Figure 1). Median birth weight was 2.3 kg. Thirty-two (59%) were delivered per vaginam and 22 (41%) were born by Caesarian section, indications being cephalo-pelvic disproportion, poor progress, abruptio placentae, eclampsia, pre-eclampsia, multiple pregnancy, premature rupture of membranes and foetal distress. The median age at presentation was four days with a range of 1-30 days. Forty-eight (89%) were referred from peripheral hospitals while six (11%) were born at the central obstetric unit. Perinatal problems encountered were human immune deficiency syndrome virus antibody (HIV) positive in four mothers, (eclampsia/pre-eclampsia in three, premature rupture of membranes in one and multiple pregnancy in three.

Abdominal distension was the presenting feature in 36 (74%) of the neonates, excluding five with an anterior abdominal wall defect. Fifteen (28%) had features of sepsis, namely fever, tachycardia, low platelet count and abnormal white blood cell count at presentation. Two (4%) patients who had meconium peritonitis presented with a mass in the right iliac fossa at birth. Herbal enemas had been given prior to perforation in two patients, while one developed abdominal distension in the nursery following rectally taken temperature thermometer readings. Other features are shown in Table 1. The duration of symptoms was a mean of two days (range of 1-14 days).

Forty-six patients had a plain abdominal X-ray which showed evidence of perforation in 42 (86%), either free intra-peritoneal air or calcification. Calcification was seen in six of nine neonates who had meconium peritonitis. The anatomical distribution of the perforations is shown in Table 2.

Gastric perforations occurred in seven (13%) of the neonates. Secondary perforations due to underlying pathology occurred in five, idiopathic perforations in one, and NEC was the cause in one. The primary pathologies in secondary perforations were duodenal atresia, gastroschisis, malrotation complicated by midgut volvulus, and tracheo-oesophageal atresia with tracheo-oesophageal fistula.

The small bowel was involved in 20 (37%) the most common site of perforation being terminal ileum (n=11). The jejunum was involved in two neonates while seven perforations were not specified. Secondary perforations occurred in four, idiopathic in nine, and NEC was the cause in seven. Primary pathology in secondary perforations included: gastroschisis, incarcerated hernias, and exomphalos.

Table 1

Co-morbid factors

Co-morbid factor	No. of patients
Prematurity	28
Cardiac anomaly	1
Respiratory problems (Respiratory distress syndrome hyaline membrane disease, asphyxia, pneumonia)	11 9
Down's Syndrome	2
Anorectal anomaly	2
Malrotation	1
Incarcerated inguinal hernia	3
CMV enterocolitis	2
Duodenal atresia	2
Tracheo-oesophageal fistula/atresia	2
Hirschsprung's disease	1
Gastroschisis	3
Exomphalos	2
Hypoglycaemia	1
Trauma	3

Table 2

Distribution of perforations

Site of perforation	No. of patients	No. of deaths	% mortality
Stomach	7	6	86
Duodenum	1	1	100
Small bowel	20	7	35
Colon	17	6	35
No perforation found	2	0	0
Multiple sites	3	3	100
Died before operation	2	2	100
Managed conservatively with drains	2	0	0

Table 3

Post-operative complications

Complication	No. of patients	%
Septicaemia	24	44.4
Wound sepsis	1	1.9
Respiratory failure	2	3.7
Renal failure	1	1.9
Haemorrhage	4	7.4
Anastomotic leak	2	3.7
Short bowel syndrome	1	1.9
Intra-ventricular haemorrhage	1	1.9

Table 4

Relationship between diagnosis and mortality

Diagnosis	No. of patients	Mortality	%
Necrotizing enterocolitis	19	10	53
Idiopathic	17	5	30
Perforations secondary to other pathology	18	10	56

The colon was involved in 17 out of 54 (31%) of the neonates, with the caecum being most common in eight cases. Two patients had a sigmoid colon perforation, two had a rectal perforation, while the transverse colon was involved in four neonates and one colonic perforation was not specified. Secondary perforations occurred in five, idiopathic perforations in seven, and NEC was the cause in five. Underlying pathology in secondary perforations included: herbal enema, rectal thermometer, high anorectal malformation and Hirschsprung's disease.

Multiple perforations due to extensive CMV enterocolitis were found in two patients who were also HIV antibody positive. Extensive intestinal necrosis was found in one neonate who had an incarcerated right inguinal hernia and caecal perforation. At operation, perforations were not found in two neonates despite a pre-operative X-ray showing free intra-peritoneal air.

Perforations were primarily repaired in 30 (debridement and repair or limited resection and primary anastomosis) and stomas were performed in 15 cases. Peritoneal lavage was done in two cases where perforations were not found, procedure was abandoned in three with extensive necrosis and two were managed conservatively with percutaneous drain decompression and survived. Two died before operation.

Figure 1

Birth weight distribution in 42 neonates

Figure 2

Mortality in relation to birth weight of 42 neonates

Initial antimicrobial cover in all patients consisted of a combination of penicillin, metronidazole and gentamicin or cefoxitin and metronidazole, with fluconazole added in gastric perforations. Culture and sensitivity results of peritoneal fluid taken at operation or pre-operative cultures guided subsequent antimicrobial treatment. A multiplicity of organisms were cultured; multiresistant *acinetobacter* and *pseudomonas* being commonly isolated.

Re-laparotomy was necessary in 11 (22%) patients. Of these, one had intra-peritoneal haemorrhage, two had a leaking anastomosis and were converted to enterostomies, four with NEC continued to deteriorate despite the primary procedure but no new perforations found, two with gastroschisis as part of staged anterior abdominal wall repair in one and for a new perforation of small bowel in the other, and for early closure of stomas in two. Table 3 shows the complications in the post-operative period and their frequency.

Twenty nine (54%) of the neonates survived while 25 (46%) died. Of those who died, 13 (52%) were premature. Sepsis was the cause of death in 18 (72%). Table 4 relates the mortality to the diagnosis. Mortality in relation to site of perforation is shown in Table 2 above. Mortality in relation to birth weight is shown in Figure 2.

DISCUSSION

Eighty nine per-cent of the neonates with GI tract perforation managed by the Department of Paediatric Surgery were referred from peripheral hospitals within KwaZulu-Natal and parts of the Eastern Cape. Hadley *et al.* (12) found that transfer of neonates with surgical emergencies in this region to the tertiary paediatric surgical unit was inefficient and preparation was poor. As this review falls within the time of Hadley's study, his findings are relevant. At the time of admission to the paediatric surgical unit, the mean duration of known perforation was two days with a range of 1-14 days. Two neonates arrived in poor condition and died before operation. The long duration between perforation and definitive treatment as well as inadequate pre-operative resuscitation both have a negative effect on the outcome of treatment (6,9).

Abdominal distension was the most common feature of perforation (74%). Distension causes respiratory distress by splinting the diaphragm. Decompression of pneumoperitoneum by percutaneous drainage relieves respiratory distress and also decreases the progression of peritoneal contamination and subsequent sepsis (6,9). Drainage may be definitive as seen in two of our patients (4,6). Advice on placement of peritoneal drains and on preparation for transport is given to all referring hospitals prior to transfer. Only 8% had drains placed.

In 19 (35%) neonates, the cause of perforations was NEC, and in 18 (33%) another pathology was identified. These were incarcerated inguinal hernias,

anterior abdominal wall defects, Hirschsprung's disease, high anorectal malformations, atresias of gastrointestinal tract, trauma and malrotation. All these patients were referred from peripheral hospitals. These patients had the highest mortality (56%), in contrast to studies which have shown NEC to be responsible for most deaths (4,6,7,9,10). The primary pathologies were associated with mortality in 70% of neonates with birth weight of 3 kg and over (second peak) (Figure 2). Early recognition which incorporates antenatal diagnosis for some of the lesions ensures early referral and transportation of the baby in its "natural incubator", the uterus, for delivery in tertiary centers where early treatment can be instituted (13). Simple interventions like nasogastric intubation (to decompress the GIT reducing intraluminal hydrostatic pressure in obstructive lesions) and reduction of hernias under sedation can be achieved before transfer, reducing rates of perforation.

Treatment and investigative procedures performed on the neonate can cause perforation of the gastrointestinal tract (2) as in three of our patients. Herbal enemas were responsible for sigmoid colon and rectal perforations in two neonates, while a thermometer was responsible for rectal perforation in one. In developing countries rectal temperature monitoring and herbal medicinal insertions are common. Avoidance of this practice in the developed world has led to a decline in rectal perforations (2).

The emergence of human immune deficiency syndrome (HIV) infection and its associated complications raises new challenges which will increase unless mother-to-child transmission is checked. Two of the three neonates with multiple perforations were HIV antibody positive and the perforations were due to CMV enterocolitis. The patients have poor long-term survival (14) and gancyclovir is not available in our practice.

To improve this grim experience prevention of secondary perforations is essential. In addition to early recognition and management of perforation, resuscitation should include percutaneous decompression of pneumoperitoneum in children with respiratory difficulties. This should be within the compass of all medical officers and senior nurses yet frequently is omitted in the erroneous belief that it is a surgical procedure which can only be performed by a surgeon.

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