Neonatal Gastrointestinal Perforations: the 10-Year Experience of a Reference Hospital

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Received: 30 June 2016 / Accepted: 20 October 2016 / Published online: 27 October 2016
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Abstract The aim of this study was to present our experiences with, as well as the factors that affect, the treatment and outcome of patients with neonatal gastrointestinal perforations (GIPs). Thirty-eight newborn cases that were operated on for GIP in our hospital's tertiary newborn intensive care unit between January 2005 and December 2015 were retrospectively evaluated. The patients were divided into the two following groups: group 1, perforations related to necrotizing enterocolitis (NEC), and group 2, non-NEC perforations. In total, 38 patients (16 males, 22 females) participated in this study. The perforations were related to NEC in 12 patients (group 1; 31.6 %), and the other 26 patients (group 2; 68.4 %) were classified as non-NEC perforation cases. The incidence of neonatal GIP was 0.53 % in all newborn patients, while the incidence of perforation in NEC cases was 20 %. Of all patients, 25 (65.7 %) were premature. Non-NEC pathologies were the most common cause of GIP (68.4 %) and included stomach perforation related to a nasogastric catheter ($n = 5$), volvulus ($n = 4$), intestinal atresia ($n = 3$), esophageal

atresia and tracheoesophageal fistula ($n = 2$), cystic fibrosis ($n = 2$), Hirschsprung's disease ($n = 2$), appendicitis ($n = 2$), congenital stomach anterior wall weakness ($n = 1$), duplication cyst ($n = 1$), invagination ($n = 1$), incarcerated inguinal hernia ($n = 1$), and idiopathic causes ($n = 2$). Primary surgical repair was performed in all cases without a conservative approach. The mortality rate related to GIP in newborn cases was 47.3 %. While the mortality rate in group 1 was 66.6 %, it was statistically insignificantly lower in group 2 (38.4 %) ($p > 0.05$). In group 1, the mortality rate of those with intestinal and colorectal perforations was 45.6 and 20 %, respectively ($p > 0.05$). Non-NEC pathologies are the most frequent causes of GIP in newborns, and primary surgical repair is the primary treatment choice for neonatal GIP. However, GIP remains one of the most significant causes of mortality in newborns. While the prognosis for neonatal colon perforation is good, that for stomach and jejunoileal perforations is worse.

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Keywords Newborn infant · Gastrointestinal perforation · Necrotizing enterocolitis · Mortality

Introduction

Neonatal gastrointestinal perforations (GIPs) presently have high mortality rates (40–70 %) [1]. Despite advancements in mechanical ventilation, antibiotherapy, surgical techniques, and anesthesia, as well as developments in intensive care units (ICUs), morbidity and mortality rates associated with GIP have yet to decrease to the desired level [2]. Neonatal GIP occurs in a large heterogenous group, ranging from premature and low-birth-weight infants to healthy term infants [3]. The most frequent causes of GIP include necrotizing enterocolitis (NEC), sepsis, intestinal obstructions, iatrogenic traumas secondary to irritations due to orogastric (OG) or nasogastric (NG) catheters, Hirschprung's disease, mechanical irritation, and spontaneous intestinal perforations (SIPs) [4].

The aim of this study was to evaluate the etiologies, clinical findings, and factors affecting the outcomes of GIP patients operated on in our hospital.

Material and Methods

Study Design The clinical data of 38 neonatal cases that were operated on for GIP and followed up at the neonatal ICU (NICU) of our tertiary-care health institute between January 2005 and December 2015 were retrospectively evaluated. Our hospital is a reference center with a 46-bed capacity that serves the Northern Anatolian region of Turkey. The diagnosis of GIP was based on history, findings on physical examination, and radiological evidence of free air in the abdomen. Diagnosis of GIP based on clinical and radiological findings was confirmed intraoperatively. Similarly, diagnosis of NEC was also based on clinical and radiological findings in consideration of the modified Bell criteria [5]. The cases were divided into two groups as follows: group 1, GIP related to NEC, and group 2, GIP unrelated to NEC. Approval from the Ethics Committee of our hospital was obtained for this study (protocol no. 03.01; date May 13, 2016).

Outcome Parameters The patients' demographic characteristics, including birth weight, gestational age, gender, type of delivery, and postnatal age during the perforation event, as well as clinical data, including whole blood count, C-reactive protein (CRP) levels, etiology, area of perforation, concomitant anomalies, intraoperative findings, surgical complications, and clinical outcomes, were evaluated.

Statistical Analysis Data were analyzed using SPSS for Windows software (version 22; SPSS Inc., Chicago, IL, USA).

Parametric tests were applied to data with a normal distribution, and non-parametric tests were applied to non-normally distributed data. The distribution of categorical variables in both groups was compared using the Pearson's chi-squared test. Data are expressed as the mean $\pm$ standard deviation (SD) or the median and interquartile range, as appropriate. A p value <0.05 was considered statistically significant.

Results

Demographic and Clinical Characteristics of the Patients

In total, 38 patients (16 males and 22 females) were included in this study. The incidence of neonatal GIP was 0.53 % among all newborns treated in the NICU, while it was higher (20 %) among NEC cases. The median gestational age was 34 ± 4.1 weeks (range 24–38 weeks), and the median body weight was 2150 ± 97 g (range 670–4050 g). The median postnatal age at the time of surgery was 7.86 ± 6.2 days (range 1–30 days). In 12 (31.6 %) cases, the perforations were related to NEC, while in 26 (68.4 %) cases, the perforations were unrelated to NEC. In our study series, GIP occurred from the prenatal period to postnatal day 30 (median, 7.8 ± 6.2 days). The areas of perforation described during surgical exploration are presented in Table 1. Perforations were observed in the small intestine ($n = 22$), the stomach ($n = 9$), the colon ($n = 5$), and the appendix ($n = 2$).

Non-NEC perforations were divided into idiopathic (2 cases, 5.2 %) and secondary perforation (24 cases, 63.1 %) subgroups. Non-NEC pathologies that resulted in secondary perforation included gastric perforation due to irritation of OG and NG catheters ($n = 5$), volvulus ($n = 4$), intestinal atresia ($n = 3$), esophageal atresia and tracheoesophageal fistula (EA + TEF) ($n = 2$), Hirschprung's disease ($n = 2$), cystic fibrosis ($n = 2$), appendicitis ($n = 2$), congenital weakness of the anterior wall of the stomach ($n = 1$), duplication cysts ($n = 1$), invagination ($n = 1$), and incarcerated inguinal hernia ($n = 1$).

Table 1
Intraoperatively detected areas of perforation

Area of perforation	Number (%)
Stomach	9 (23.6 %)
Small bowel	22 (57.8 %)
Jejunum	4
Ileum	18
Appendix	2 (5.2 %)
Colon	5 (13.1 %)
Cecum	3
Descending colon	1
Sigmoid colon	1
Total	38 (100 %)

Table 2 Concomitant pathologies seen in patients with gastrointestinal perforation

Concomitant pathologies	NEC cases, <i>n</i> (%)	Non-NEC cases, <i>n</i> (%)
Respiratory distress syndrome	11 (91.6 %)	11 (42.3 %)
Sepsis	5 (41.6 %)	13 (50 %)
Cardiac pathology	5 (41.6 %)	3 (11.5 %)
Esophageal atresia	–	2 (7.6 %)
Intrauterine perforation secondary to intestinal atresia	–	3 (11.5 %)
Volvulus secondary to malrotation	–	4 (15.3 %)
Weakness of the anterior wall of the stomach	–	1 (3.8 %)
Hirschprung's disease	–	2 (7.6 %)
Cystic fibrosis	–	2 (7.6 %)
Catheter-related trauma	–	5 (19.2 %)
Rectal trauma	–	1 (3.8 %)
Meningomyelocele	1 (8.3 %)	–
Prenatal history	5 (41.6 %)	6 (23 %)

NEC necrotizing enterocolitis

The causes of intestinal perforation included NEC ($n = 12$), volvulus ($n = 4$), intestinal atresia ($n = 3$), invagination ($n = 1$), incarcerated inguinal hernia ($n = 1$), and SIP ($n = 1$). Gastric perforations developed because of iatrogenic causes related to OG and NG catheter insertion ($n = 5$), EA + TEF ($n = 2$), and congenital weakness of the anterior wall of the stomach. However, in one patient, no cause of perforation could be detected. In cases with suspected iatrogenic perforations due to irritation by OG or NG catheters, congestion and hyperemia around the area of perforation were observed.

Appendiceal perforation was observed in two cases and cecal perforation developed due to cystic fibrosis in two cases, while it developed secondary to duplication cysts in a further

two cases. In one case, it was concluded that perforation of the descending colon secondary to Hirschprung's disease had developed iatrogenically during rectal irrigation in an external center. A statistically significant correlation was not detected between the area of perforation and mortality rates.

Comparison of the Patients in Both Groups The average birth weight was 1.363 ± 505 g in group 1 and 2.514 ± 922 g in group 2; the difference was significant ($p < 0.05$). The median gestational age was 30.9 ± 4.03 weeks in group 1 and 35.4 ± 3.4 weeks in group 2; the difference was not significant ($p > 0.05$).

Table 3 Clinical characteristics of patients with necrotizing enterocolitis

Case no.	Gestational age (weeks)	Body weight (g)	Type of delivery	Gender	Maternal problems	Additional anomalies	RDS
1	27	650	Cesarean	F	–	ASA, PFO, PDA	Present
2	37	1300	Cesarean	F	–	ASA, PDA, VSD	Present
3	28	1160	NSVR	F	PMR	ASA, PFO, MF	Present
4	32	1920	Cesarean	M	–	PFO, PDA, subdural hematoma	Present
5	37	1900	Cesarean	M	–	ASA, ASD, TI, MF, hydrocephalus, scoliosis	Present
6	33	1840	Cesarean	F	Chorioamnionitis	ASA, PDA, PFO, ovarian teratoma	Present
7	24	645	Cesarean	F	PMR		Present
8	28	1140	Cesarean	F	Preeclampsia	ASA, aortic coarctation	Present
9	34	2200	Cesarean	M	–		Present
10	31	1450	Cesarean	F	Preeclampsia, steroid use		Present
11	28	1135	Cesarean	M	PMR, preeclampsia	PFO, PDA	Present
12	32	990	Cesarean	F	PMR		Absent

NSVR normal spontaneous vaginal route, *M* male, *F* female, *RDS* respiratory distress syndrome, *PMR* premature membrane rupture, *ASA* atrial septal aneurysm, *PFO* patent foramen ovale, *PDA* patent ductus arteriosus, *ASD* atrial septal defect, *VSD* ventricular septal defect, *MF* mitral failure, *TI* tricuspid valve failure

Moreover, concomitant clinical factors including maternal age, prenatal history, associated anomalies, sepsis, white blood cell count and/or CRP levels, surgical findings, and mortality rates were not significantly different between the two groups ($p > 0.05$). In group 1, 11 (91.6 %) of 12 patients had concomitant respiratory distress syndrome (RDS), and in group 2, 11 (42.3 %) of 26 patients had concomitant RDS ($p < 0.05$). Concomitant pathologies of the patients in both groups are summarized in Table 2.

On abdominal examination, abdominal distention and abdominal hyperemia were detected in all (100 %), and in 9 (75 %), group 1 patients, respectively; they were also detected in all (100 %) and 9 (34.6 %) group 2 patients, respectively. Radiological examination revealed pneumatosis intestinalis in all cases with NEC. Moreover, subdiaphragmatic free air was detected in 11 (91.6 %) patients in group 1 and in 22 (84.1 %) patients in group 2 ($p > 0.05$).

Eight (66.6 %) patients in group 1, and 10 (38.8 %) patients in group 2, died ($p < 0.05$). The clinical, surgical, and radiological characteristics of the patients with NEC are listed in Tables 3 and 4. No statistically significant differences were found between the two groups with regard to these characteristics. Birth weights and gestational ages of the deceased patients with NEC were lower than those of the patients who survived. The correlation between birth weight and mortality

is shown in Table 5. In seven (87.5 %) of the eight deceased patients in group 1, additional associated cardiac anomalies and/or RDS manifestations were found.

Discussion

Although the incidence of GIP in newborns is not known exactly, its incidence in NICUs has been reported as 0.6 % [6]. Similarly, in our study, the incidence of neonatal GIP was 0.53 %. The etiopathogenesis of GIP most frequently involves NEC, followed by iatrogenic traumas caused by placement of OG or NG catheters, mechanical ventilation, sepsis, distal gastrointestinal obstructions, Hirschsprung's disease, and SIP [7, 8]. In the series presented herein, intestinal ($n = 22$), gastric ($n = 9$), colonic ($n = 7$), and appendiceal ($n = 2$) perforations were detected. NEC was described in 12 of 22 cases (55 %).

The prognosis of neonatal GIP is dependent on the underlying pathology, the presence of associated anomalies, the preoperative and postoperative levels of health care, the degree of prematurity, and the birth weight. In a previous study, the mortality rate ranged from 30 to 50 % [3]. Overall, in this study, the mortality rate for all cases of perforation was 47 %. It was previously reported that the incidence of perforation was nearly 20 % among NEC patients [9]. Similarly, in our

Table 4 Radiological and surgical findings of the cases with necrotizing enterocolitis

Case no.	Age at surgery (days)	Symptoms	Radiological findings	Area of perforation	Type of surgery	Follow-up
1	4	Abdominal distension, abdominal wall hyperemia	Pneumoperitoneum	Ileum	Ileostomy	Exitus on postoperative day 7
2	16	Abdominal distension, abdominal wall hyperemia	Pneumoperitoneum	Ileum	Ileostomy	Exitus on postoperative day 15
3	13	Abdominal distension, abdominal wall hyperemia	Pneumoperitoneum	Ileum, cecum, colon	Ileostomy, colostomy	Exitus on postoperative day 4
4	7	Abdominal distension	Pneumoperitoneum	Ileum, cecum	Ileostomy colostomy	Discharge on postoperative day 30
5	4	Abdominal distension, abdominal wall hyperemia	Pneumoperitoneum	Jejunum, ileum, colon	Jejunostomy, colostomy	Exitus on the postoperative day 1
6	13	Abdominal distension, abdominal wall hyperemia	Pneumatosis intestinalis	Ileum	Ileostomy	Discharge on postoperative day 30
7	15	Abdominal distension	Pneumoperitoneum	Jejunum, ileum	Jejunostomy	Exitus on postoperative day 5
8	7	Abdominal distension	Pneumoperitoneum	Ileum	Ileostomy	Discharge on postoperative day 120
9	30	Abdominal distension	Pneumoperitoneum	Jejunum, ileum	Jejunostomy	Discharge on postoperative day 15
10	7	Abdominal distension, abdominal wall hyperemia	Pneumoperitoneum	Ileum	Ileostomy	Exitus on postoperative day 1
11	9	Abdominal distension, abdominal wall hyperemia	Pneumoperitoneum	Ileum, cecum, colon	Ileostomy colostomy	Exitus on postoperative day 17
12	4	Abdominal distension, abdominal wall hyperemia	Pneumoperitoneum	Ileum	Ileostomy	Discharge on postoperative day 5

Table 5 Correlation between birth weights, gestational age, and mortality

		NEC cases	Non-NEC cases	Mortality exitus/total (%)
Gestational age	≤30 weeks	4	2	6/7 (85.7 %)
	>30 weeks	4	8	12/31 (37.5 %)
Birth weight	≤2000 g	8	2	10/18 (55.5 %)
	>2000 g	0	8	8/20 (40 %)

NEC necrotizing enterocolitis

study, the incidence of perforation in NEC cases was 20 %. Moreover, the mortality rate reported in the literature for NEC patients with perforations was 50 % (1), while that in our study was high, at 66 %. In our series, all patients who died due to NEC-related perforations were premature and/or were low-birth-weight infants and had concomitant cardiac anomalies or clinical manifestations of RDS.

In parallel with the favorable developments that have occurred in the NICU, the rate of survival of very low birth weight babies has increased, which has led to an increase in the incidence of NEC [9]. Worldwide, the incidence of NEC-related perforations is higher than that of perforations due to other causes [8]. Contrary to the literature, in our study, NEC-related perforations constituted 31.5 % of all perforations. We attributed this phenomenon to the fully equipped tertiary level NICU of our hospital, as well as to advancements in health care services.

In agreement with the literature, the incidence of prematurity in cases with NEC-related perforations was higher than that in cases with non-NEC-related perforations in our study [10], and mortality rates were higher in patients with NEC-related perforations compared with patients with non-NEC-related perforations. Nakamura et al. [11] reported a mortality rate of 84.6 % in infants born at <30 gestational weeks with NEC-related GIP. However, in our study, the mortality rate of these patients was 66.6 %. Moreover, the association between the manifestations of RDS and NEC is a very well-described clinical phenomenon [9]. In our study, 91.6 % of the cases with NEC had associated RDS manifestations. Infants with NEC are generally exposed to perinatal asphyxia and/or fed with hyperosmolar formulas. Therefore, areas of perforation are generally seen in various parts of their small bowels [12]. In our series, multiple perforations were detected in 6 of 12 cases with NEC and in 7 cases with NEC; maternal problems that may have caused perinatal asphyxia were determined.

Hakan et al. [3] reported average mortality rates of 30.7 and 22 % for intestinal and colonic perforations, respectively, while Chung et al. [13] reported an average mortality rate of 58 % for gastric perforations. However, in our study, the average mortality rates in cases with gastric and intestinal perforations were 66 and 45 %, respectively, while only one case with colonic perforation died.

In a previous study, pneumoperitoneum was described in 63 % of neonatal GIP cases [14], and in our series, 87 % of the

patients had pneumoperitoneum. Neonatal gastric perforation is a life-threatening condition seen frequently in premature infants. The etiopathogenesis of GIP includes congenital weakness of gastric muscle layers, gastric ulcer due to gastric secretions with high acidity, increased intragastric pressure due to distal obstruction, iatrogenic traumas secondary to OG and NG catheter irritation, and SIP [15, 16]. In our series, gastric perforations were related to OG and NG catheter placement ($n = 5$), abdominal distension due to EA + TEF ($n = 2$), and weakness of the muscles of the anterior wall of the stomach ($n = 1$); in one case, the cause of gastric perforation could not be determined. Irrespective of its etiology, gastric perforation in newborns generally occurs within 2–7 days of birth [17], and in our study, gastric perforation occurred within an average of 3.8 days after birth. Early diagnosis of GIP is very important; however, because of its non-specific symptoms, it is usually difficult to make an early diagnosis. An important indicator of gastric perforation on upright abdominal radiograms is massive subdiaphragmatic free air despite the absence of gastric gas [18]. In all of our patients with gastric perforation, massive abdominal distension and subdiaphragmatic free air were found.

In five of nine patients, the cause of gastric perforation was iatrogenic, due to OG or NG catheter irritation, which is higher than the incidence currently reported in the literature [12]. We attribute this higher rate of perforation to the inexperience of our novice physicians and nursing staff. Gastric perforation has been reported frequently in premature and low-birth-weight infants (18); 78 % of our patients who developed gastric perforation were premature, while the other two babies were term infants with birth weights of ≥ 3000 g. The mean gestational age of the premature babies with gastric perforation was 33 weeks, and their average birth weight was 1860 g. In two recent studies published in Turkey, the mortality rate due to gastric perforation was reported to be 60 % [19, 20]; in our series, the mortality rate was also 60 %. We believe that the presence of concomitant life-threatening anomalies and the higher rates of prematurity are responsible for the increased rates of mortality.

In conclusion, multiple factors play a role in the etiology of neonatal GIP, and causes unrelated to NEC are frequently observed. In premature babies with concomitant manifestations of RDS, the development of NEC is relatively probable. The mortality rate of newborns with GIP remains high, and the prognosis of NEC-related perforations is dependent upon

the presence of prematurity, low birth weight, and any additional associated anomalies. Gastric and intestinal perforations are associated with high mortality rates, while prognosis of cecal and colonic perforations is relatively more favorable.

Compliance with Ethical Standards

Research Involving Human Participants and/or Animals This is a retrospective study.

Informed Consent Formal and written informed consents were obtained from parents.

Conflict of Interest The authors declare that they have no conflict of interest.

References

- Venkatesh MA, Vijay K, Rashmi VA, Mahesh M, Anil BH (2013) Gastrointestinal perforation in neonates. *Karnataka Paediatric Journal* 28:140–146
- Sakellaris G, Partalis N, Dede O, Alegakis A, Seremeti C, Korakaki, Giannakopoulou C (2012) Gastrointestinal perforations in neonatal period: experience over 10 years. *Pediatr Emerg Care* 28:886–888
- Hakan N, Aydin M, Erdogan D, Cavusoglu YH, Dursun A, Zenciroglu A, Okumus N, Ozguner IF, Karaman A, Karaman I (2013) Neonatal gastrointestinal perforations: a 7-year single center experience at a tertiary neonatal intensive care unit in Turkey. *CIBTech Journal of Surgery* 3:1–7
- Attridge JT, Herman AC, Gurka MJ, Griffin MP, McGahren ED, Gordon PV (2006) Discharge outcomes of extremely low birth weight infants with spontaneous intestinal perforations. *J Perinatol* 26:49–54
- Walsh MC, Kliegman RM (1986) Necrotizing enterocolitis: treatment based on staging criteria. *Pediatr Clin N Am* 33:179–201
- do Lee K, Shim SY, Cho SJ, Park EA, Lee SW (2015) Comparison of gastric and other bowel perforations in preterm infants: a review of 20 years' experience in a single institution. *Korean J Pediatr* 58:288–293
- Kazem A, Yekeler H, Turgut M, Apak S, Parmaksız E (1996) Idiopathic focal intestinal perforation in low birth weight neonates. *Pediatric Cerrahi Dergisi* 10:21–24
- Emil S, Davis K, Ahmad I, Strauss A (2008) Factors associated with definitive peritoneal drainage for spontaneous intestinal perforation in extremely low birth weight neonates. *Eur J Pediatr Surg* 18:80–85
- Boston VE (2006) Necrotizing enterocolitis and localized intestinal perforation: different diseases or ends of a spectrum of pathology. *Pediatr Surg Int* 22:477–484
- St-Vil D, LeBouthillier G, Luks FI, Bensoussan AL, Blanchard H, Youssef S (1992) Neonatal gastrointestinal perforations. *J Pediatr Surg* 27:1340–1342
- Nakamura H, Hirano K, Watanabe K (2003) Gastrointestinal perforation in 31 neonates with surgical treatment: statistical analysis by quantification theory type in. *Journal of the Japanese Society of Pediatric Surgeons* 39:748–757
- Hyginus EO, Jideoffor U, Victor M, N OA (2013) Gastrointestinal perforation in neonates: aetiology and risk factors. *J Neonatal Surg* 2:30
- Chung MT, Kuo CY, Wang JW, Hsieh WS, Huang CB, Lin JN (1994) Gastric perforation in the neonate: clinical analysis of 12 cases. *Zhonghua Min Guo Xiao Er Ke Yi Xue Hui Za Zhi* 35:460–465
- Singh M, Owen A, Gull S, Morabito A, Bianchi A (2006) Surgery for intestinal perforation in preterm neonates: anastomosis vs. stoma. *J Pediatr Surg* 41:725–729
- Ohshiro K, Yamataka A, Kobayashi H, Hirai S, Miyahara K, Sueyoshi N, Suda K, Miyano T (2000) Idiopathic gastric perforation in neonates and abnormal distribution of intestinal pacemaker cells. *J Pediatr Surg* 35:673–676
- Yamataka A, Yamataka T, Kobayashi H, Sueyoshi N, Miyano T (1999) Lack of C-KIT + mast cells and the development of idiopathic gastric perforation in neonates. *J Pediatr Surg* 34:34–38
- Calisti A, Perrelli L, Nanni L, Vallasciani S, D'Urzo C, Molle P, Briganti V, Assumma M, De Carolis MP, Maragliano G (2004) Surgical approach to neonatal intestinal perforation. An analysis on 85 cases (1991–2001). *Minerva Pediatr* 56:335–339
- Fenton TR (2003) A new growth chart for preterm babies: Babson and Benda's chart updated with recent data and a new format. *BMC Pediatr* 3:13
- Duran R, Inan M, Vatansever U, Aladağ N, Acunaş B (2007) Etiology of neonatal gastric perforations: review of 10 years' experience. *Pediatr Int* 49:626–630
- Oztürk H, Onen A, Otçu S, Dokucu AI, Gedik S (2003) Gastric perforation in neonates: analysis of five cases. *Acta Gastroenterol Belg* 66:271–273