Stallard Scientific Editing
your trusted partner in English-language excellence

11 June 2014

To whom it may concern,

This letter certifies that the manuscript by Jeik Byun entitled

Neonatal gastric perforation: a single center experience

has been edited by a native-English-speaking scientific editor. The edited text is shown in the following pages of this document.

Yours sincerely,

Aaron Stallard

Managing Editor
Stallard Scientific Editing
56 Brougham St
Nelson 7010
NEW ZEALAND

www.stallardediting.com
aaron@stallardediting.com

Tel: +64 3 5489108
Fax: +64 3 5489106

Name of journal: World Journal of Gastrointestinal Surgery
ESPS Manuscript NO: 9604
Columns:[1] **[It is unclear what is required here. Do you need to specify one of the categories provided in the "Columns Scope Note" file?]**

Neonatal gastric perforation: a single center experience

Jeik Byun, Hyun-Young Kim, Seung-Yeon Noh, Soo-Hong Kim, Sung-Eun Jung, Seong-Cheol Lee, and Kwi-Won Park

Department of Pediatric Surgery,
Seoul National University Children's Hospital,
Seoul, Republic of Korea

Running Title: Etiology and prognosis of NGP
Grant support: None
Author Disclosure Statement: The authors have no financial or conflicts of interest to declare.
Corresponding Author
Sung-Eun Jung
Department of Pediatric Surgery, Seoul National University Children's Hospital, 101 Daehang-ro, Yeongeon-dong, Jongro-gu, Seoul, Korea 110-744
Telephone: +82-02-2072-2478
Fax: +82-02-740-5130
E-mail: sejung@snu.ac.kr

ABSTRACT

Aim: To determine the etiology and prognostic factors for neonatal gastric perforation (NGP), a rare but life-threatening disease.[2]

Methods: Between 1980 and 2011, nine patients underwent surgical intervention for NGP at Seoul National University Children's Hospital. The characteristics and prognosis of the patients were retrospectively analyzed.

Results: Among the nine patients, three (33.3%) were preterm babies and five (55.5%) had associated anomalies, which included diaphragmatic eventration (n = 2), congenital diaphragmatic hernia, esophageal atresia with tracheoesophageal fistula, and antral web. Three (33.3%) patients were born before 1990 and three (33.3%) had a birth weight of <2500 g. Pneumoperitoneum was found on preoperative images in six (66.7%) patients, and incidentally in the other three (33.3%) patients. Surgery was performed within 24 h after the onset of symptoms in seven (77.8%) patients. The overall mortality rate was 22.2% (2/9). The time between symptoms and surgical intervention was the only prognostic factor for survival, whereas premature birth and birth weight were not.

Conclusion: Early detection and advances in neonatal intensive care may improve the prognosis of NGP.

Key words: Neonate, gastric perforation, etiology, prognosis, surgical intervention

CORE TIP

Neonatal gastric perforation (NGP) is extremely rare and very few cases have been reported to date. We determined the etiologies and prognostic factors for NGP in nine cases that were treated at a single center. Early detection and prompt surgical intervention is essential to improve the outcomes of NGP.

INTRODUCTION

Neonatal gastric perforation (NGP) accounts for approximately 7% of all gastrointestinal perforations in neonates, and has a poor prognosis with a high motility rate[1-2]. Factors associated with NGP include prematurity, asphyxia, congenital anomalies, stress at birth, vigorous respiratory resuscitative measures, increased intragastric pressure caused by distal obstruction, and anatomic abnormalities of the stomach[3-7]. Male gender, metabolic acidosis, premature birth, and low birth weight are associated with worse outcomes[8,9]. However, the etiology and prognostic factors of NGP are still widely debated. Here, we describe our experience of treating nine patients with NGP at a single center. The aim of this study was to review patients with NGP and discuss its etiology and prognosis, in order to improve patient outcomes.

METHODS

Data collection

Between 1983 and 2011, nine neonates (five males and four females) who underwent surgical treatment for NGP at a single center were identified using written and electronic medical records.

Variables

We focused on preoperative and intraoperative characteristics that are known or thought to be prognostic factors for NGP. The characteristics retrieved from medical records included gender, year of birth, gestational age, birth weight, method of delivery, maternal gestational problems, maternal age at delivery, Apgar scores, initial symptoms, time from birth to initial symptoms after birth, time between symptom onset and surgery, serum pH, serum pCO_2, use of a nasogastric tube, ventilator therapy, O_2 therapy, diagnostic method, associated anomalies, site of perforation, length of perforation, type of surgical procedure, and postoperative complications.

Statistical analysis

Statistical analyses were performed using SPSS 19.0 software for Windows (IBM Inc., Armonk, NY, USA). Descriptive data are reported as the percentage of patients or as the mean (range). The χ^2 test was used to identify possible prognostic factors.

RESULTS

The clinical features of the nine patients are described in Table 1. There were five boys and four girls; three were born before 1990 and six were born after 1991. The mean gestational age was 38^{+0} weeks (range, 24^{+0}–40^{+2} weeks) and the mean birth weight was 2950 g (range, 730–4040 g). Three patients were preterm and six were full term. Two patients had a low birth weight (LBW; <2500 g) and one had an extremely low birth weight (ELBW; <1000 g). Seven were born via natural delivery and two were born via cesarean section. Two patients were born after premature rupture of the membranes, of which one was a twin. The mean maternal age at delivery was 32 years (range, 25–32 years). In one patient, Apgar score was 1 and 4 at 1 and 5 min, respectively. In another patient, Apgar score was 2 and 7 at 1 and 5 min, respectively. Preoperative conditions are also described in Table 1. Preoperative serum pH was <7.30 in five patients and >7.30 in two patients, and was not determined in the other two patients. A nasogastric tube was used preoperatively in seven patients. Six were on a specialist diet, four were on ventilators, and six received supplemental O_2.

All nine patients presented with mild to severe abdominal distension. Patient A initially presented with high fever, vomiting, and dyspnea. Congenital diaphragmatic hernia was initially suspected, but the final diagnosis was diaphragmatic eventration. Gastric perforation was found during surgery. Patient B initially presented with vomiting and dyspnea. Abdominal exploration was performed because of suspected congenital diaphragmatic hernia, which was ultimately diagnosed as diaphragmatic eventration. Gastric perforation was also found intraoperatively. Patient C initially presented with dyspnea and was intubated at birth. At 3 days of age, the patient exhibited hematemesis and severe abdominal distension. Paracentesis revealed bloody ascites, and explorative surgery was performed 2 days after the onset of symptoms. All of the patients, except for Patients B and C, underwent surgery within 24 h of the onset of symptoms. The mean age of symptom onset was 3 days (range, 0–5 days). Five patients had associated anomalies, which included diaphragmatic eventration, congenital

diaphragmatic hernia, esophageal atresia with a tracheoesophageal fistula, and antral web.

The intraoperative and postoperative findings are summarized in Table 2. The mean size of the perforation was 4.5 cm (0.5–10 cm). The body (n = 5, 55.5%) of the stomach was the most common site of perforation. The perforation was located in the greater curvature in four patients and in the lesser curvature in three patients. Primary repair was performed in six patients, while resection and anastomosis was performed in three patients. Postoperative complications occurred in five patients (55.6%), which included wound problems in two patients and recurrence in one patient. The other four patients were discharged without any complications.

Patient H, who had recurrence, was male and was born at a gestational age of 24 weeks with a birth weight of 730 g. He presented with pneumoperitoneum on infantogram and underwent surgery at 5 days old. A 5 cm long laceration on the lesser curvature and pyloric thickening were found during explorative surgery. Therefore, primary repair was done. However, the postoperative infantogram and sonogram revealed an increase in free air. Small bowel series suggested obstruction of the gastric outlet. Three days after initial surgery, the patient underwent a second operation that revealed another perforation of the upper body of the anterior wall and prepyloric antral web. Primary repair and Heineke–Mikulicz pyloroplasty were performed, and an ileostomy was formed because of enteritis of the entire small bowel. Ileostomy repair was done 3 months after surgery.

The overall mortality rate was 22.2% (2/9). Patient B was born at a gestational age of 40 weeks and the birth weight was 2950 g. The patient was diagnosed with Bochdalek hernia at another hospital and was transferred to our institute for surgery. On surgical exploration, the anterolateral portion of the diaphragm was eventrated, and a 2.5 cm long laceration was found in the posterior wall of the stomach. Diaphragm repair and primary suturing of the stomach were done. The patient died 5 days after surgery because of septic shock. Patient C was born at a gestational age of 35 weeks and birth weight of 2190 g. Hematemesis and hematochezia were found at 3 days of age. The patient received conservative therapies, including transfusion for 24 h. Diagnostic paracentesis performed at 4 days of age revealed bloody ascites.

Explorative laparotomy was done to evaluate the patient's hemoperitoneum and gastrointestinal bleeding. On exploration, a 10 cm long laceration with a necrotic margin was found on the greater curvature of the stomach, and primary repair was performed. Fifteen days later, the patient suffered from abrupt onset of abdominal distension and vomiting, and an erythematous discoloration was found on the left flank. Necrotizing enterocolitis was suspected based on infantogram, and the patient underwent surgery to repair multiple small bowel perforations. Gross fecal spillage into the abdominal cavity and multiple perforations of the small bowel were found, and approximately 20 cm of the ileum was resected. Despite intensive postoperative care, the patient's septic condition, hepatic dysfunction, and renal dysfunction resulted in death 29 days after the second surgery.

When we performed analyses to identify factors associated with survival, the time between symptoms and surgical intervention was the only prognostic factor for survival ($P < 0.05$) (Table 3). However, factors that appeared to show some association with survival included the presence of pneumoperitoneum on preoperative imaging ($P = 0.083$) and the year of birth ($P = 0.083$). Prematurity and birth weight were not associated with survival (both, $P = 1.000$).

DISCUSSION

Since Herbut first suggested that the congenital absence of muscular structures of the stomach may result in perforation[10], multiple theories have been proposed to describe the etiology of NGP. High gastric acid production and stress ulceration[11], abdominal trauma[12], ischemia of the stomach wall due to asphyxia[13] or vascular shunting[14], lack of intestinal pacemaker cell[15], and lack of C-KIT mast cells[16] have all been proposed as possible causes of NGP. NGP was historically thought to occur spontaneously[10,18,19] without any association with distal obstruction or other gastrointestinal conditions. However, since Shaw et al.[20] reported perforation of the stomach after tying both ends of the stomach and insufflating it with air, mechanical pneumatic rupture has been proposed as a possible etiologic factor[6,7,14]. Gryboski investigated the mechanism involved in neonatal swallowing, and reported that esophageal peristalsis was not coordinated until 3 days after birth[21]. Jones et al. suggested that neonatal immaturity of the vomiting mechanism made it possible to increase the intragastric pressure to its limit[22].

Irrespective of the etiology, NGP mostly occurs between 2 and 7 days of age[25]. Indeed, all of the patients in the present series presented with symptoms by 7 days of age. Some authors have noted that premature birth is a common finding in patients with NGP[23,24]. In our study, 33.3% (3/9) of patients were preterm, which is higher than the normal rate. O_2 supplementation or hypoxic stress were also reported as etiologic factors for NGP[26], and 55.6% (5/9) received supplemental O_2 in our study and the initial symptom was dyspnea in 22.2% (2/9). None of the patients in our series had trauma, but intragastric acidity was not assessed. Leone et al. suggested that NGP is not spontaneous and most patients have accompanying anomalies, including tracheoesophageal fistula or duodenal strictures, which may lead to intestinal obstruction and increased intragastric pressure. In fact, 55.6% (5/9) of patients in our series had an associated anomaly and one patient with NGP and accompanying antral web experienced disease recurrence. This finding supports the theory that distal obstruction is a common cause of NGP. Thus, in patients with suspected NGP, the consultant should consider the likelihood of accompanying

disorders, especially of disorders that may increase intragastric pressure. Although the greater curvature is thought to be the most common site of perforation[6,20], the distribution of perforation sites was fairly even.

Factors predicting the survival of NGP have not been extensively examined. Lin et al. reported that the mortality rate was significantly higher in premature infants and in those with a low birth weight[9]. Chung et al. reported that male gender and metabolic acidosis (pH <7.3) were associated with poor prognosis. In the present patients, prematurity was not associated with survival; of three premature patients, only one died (because of septic shock) and the χ^2 test yielded a *P*-value of 1.000. Likewise, low birth weight was not associated with survival. There were two LBW and one ELBW patients, and only one LBW patient died. Furthermore, male gender was not associated with survival. There were five boys and four girls, and gender was not associated with survival. Five patients had preoperative metabolic acidosis, of which two died because of postoperative septic shock. In both of these patients, the preoperative serum pH was <7.30, but the association between preoperative pH and survival was not significant.

The time between symptoms and surgical intervention was the only prognostic factor for survival, with a *P*-value of <0.05. However, because the study group was small, involving just nine patients, there is the possibility of type II error. We considered the *P*-value as a factor of relativity and extended its interpretation criteria. Even though several other factors were not statistically significant, they may be clinically relevant in terms of survival outcomes. The factor with the lowest *P*-value was the year of birth. Of note, two of three patients born before 1990 died. There is a great difference between the clinical and mechanical environments of the neonatal intensive care unit in the 1980s compared with those today. It is likely that clinical and technical developments in pre- and postoperative intensive care have improved the survival outcome of NGP patients. Another factor with a low *P*-value was preoperative pneumoperitoneum on plain X-ray. Notably, two of three patients who did not undergo preoperative infantography died. Had intestinal perforation been detected or suspected based on preoperative radiographs, earlier intervention may have been possible, increasing the likelihood of survival. Interestingly, all three

patients who did not undergo infantography were treated before 1990. Thus, the lack of a diagnostic protocol and diagnostic tools probably contributed to the poor prognosis before 1990 in particular.

Limitations of our paper are that it was performed retrospectively and the number of patients was too small to achieve statistical significance. However, NGP is extremely rare and very few cases have been reported to date. Therefore, our findings should help clinicians and surgeons with their decisions.

In conclusion, early detection and prompt surgical intervention is essential to improve the outcomes of infants with NGP. The survival outcomes of preterm infants or LBW infants were not inferior to those of other patients. NGP can accompany other significant anomalies. Therefore, careful examination of the patient, together with imaging studies, may lead to early detection and improve the outcomes of NGP.

COMMENTS

Background

Neonatal gastric perforation (NGP) is a very rare but life-threatening disease.

Research frontiers

Because of its rarity, the etiology and prognostic factors for NGP are debated.

Innovations and breakthroughs

The time between symptoms and surgical intervention was the only prognostic factor for survival; premature birth and birth weight were not associated with the survival of patients with NGP.

Applications

Early detection and advances in neonatal intensive care may improve the prognosis of NGP.

Terminology

NGP: Neonatal gastric perforation

LBW: Low birth weight

ELBW: Extremely low birth weight

Peer review

This is an important case series to publish as it deals with a rare but important neonatal emergent disorder. The abstract, introduction, method and results are all well written.

REFERENCES

1. **St-Vil D**, LeBouthillier G, Luks FI, Bensoussan AL, Blanchard H, Youssef S. Neonatal gastrointestinal perforations. *J Pediatr Surg* 1992; **27**: 1340-1342 [PMID: 1403517 DOI: 10.1016/0022-3468(92)90292-f]

2. **Rosser SB**, Clark CH, Elechi EN. Spontaneous neonatal gastric perforation. *J Pediatric Surg* 1982; **17**: 390-394 [PMID: 7120006 DOI: 10.1016/s0022-3468(82)80496-x]

3. **Holgersen LO**. The etiology of spontaneous gastric perforation of the newborn: a reevaluation. *J Pediatr Surg* 1981; **16**: 608–613 [PMID: 7277163 DOI: 10.1016/0022-3468(81)90014-2]

4. **Tan CE**, Kiely EM, Agrawal M, Brereton RJ, Spitz L. Neonatal gastrointestinal perforation. *J Pediatr Surg* 1989; **24**: 888–892 [PMID: 2674391 DOI: 10.1016/s0022-3468(89)80589-5]

5. **Kara CS**, Ilçe Z, Celayir S, Sarimurat N, Erdogan E, Yeker D. Neonatal gastric perforation: review of 23 years' experience. *Surg Today* 2004; **34**(3): 243-245 [PMID: 14999537 DOI: 10.1007/s00595-003-2675-3]

6. **Leone RJ Jr**, Krashna IH. 'Spontaneous' neonatal gastric perforation: is it really spontaneous? *J Pediatr Surg* 2000; **35**: 1066-9 [PMID: 10917298 DOI: 10.1053/jpsu.2000.7773]

7. **Jawad AJ**, Al-Rabie A, Hadi A, Al-Sowailem A, Al-Rawaf A, Abu-Touk B, Al-Karfi T, Al-Sammarai A. Spontaneous neonatal gastric perforation. *Pediatr Surg Int* 2002; **18**: 396-399 [PMID: 12415364 DOI: 10.1007/s00383-002-0749-8]

8. **Chung MT**, Kuo CY, Wang JW, Hsieh WS, Huang CB, Lin JN. Gastric perforation in the neonate: clinical analysis of 12 cases. *Acta Paediatr Sin* 1994; **35**: 460-465 [PMID: 7942035 DOI: 10.1002/ccd.20116]

9. **Lin CM**, Lee HC, Kao HA, Hung HY, Hsu CH, Yeung CY, Sheu JC, Wang NL. Neonatal gastric perforation: report of 15 cases and review of the literature. *Pediatr Neonatol* 2008; **49**(3): 65-70 [PMID: 18947001 DOI: 10.1016/s1875-9572(08)60015-7]

10. **Herbut PA**. Congenital defect in musculature of stomach with rupture in newborn Infant. *Arch Pathol* 1943; **36**: 91

11. **Kiesewetter WB**. Spontaneous rupture of the stomach in the newborn. *AMA J Dis Child* 1956; **91**: 162–167 [PMID: 13282628 DOI: 10.1001/archpedi.1956.02060020164013]

12. **Arnold GG**. Perforation of the stomach in the neonatal period; report of a survival in a premature infant. *J Pediatr* 1955; **46**: 276–279 [PMID: 14354581 DOI: 10.1016/s0022-3476(55)80281-8]

13. **Touloukian RJ**, Posch JN, Spencer R. The pathogenesis of ischemic gastroenterocolitis of the neonate selective gut mucosal ischemia in asphyxiated piglets. *J Pediatr Surg* 1972; **7**: 194-205 [PMID: 5023196 DOI: 10.1016/0022-3468(72)90496-4]

14. **Houck WS**, Griffin JA. Spontaneous linear tear of the stomach in the newborn infant. *Ann Surg* 1981; **193**: 763–768 [PMID: 7247521 DOI: 10.1097/00000658-198106000-00012]

15. **Ohshiro K**, Yamataka A, Kobayashi H, Hirai S, Miyahara K, Sueyoshi N, Suda K, Miyano T. Idiopathic gastric perforation in neonates and abnormal distribution of intestinal pacemaker cells. *J Pediatr Surg* 2000; **35**: 673–676 [PMID: 10813320 DOI: 10.1053/jpsu.2000.5940]

16. **Yamataka A**, Yamataka T, Kobayashi H, Sueyoshi N, Miyano T. Lack of C-KIT+ mast cells and the development of idiopathic gastric perforation in neonates. *J Pediatr Surg* 1999; **34**: 34–37 [PMID: 10022139 DOI: 10.1016/s0022-3468(99)90224-5]

17. **Touloukian RJ**. Gastric ischemia: the primary factor in neonatal gastric perforation. *Clin Pediatr (Phila)* 1973; **12**: 219□225 [PMID: 4701092]

18. **Meyer JL 2nd**. Congenital defect in the musculature of the stomach resulting in spontaneous gastric perforation in the neonatal period. *J Pediatr* 1957; **51**: 416-421 [PMID: 13476323 DOI: 10.1016/s0022-3476(57)80126-7]

19. **Amadeo JH**, Ashmore HW, Aponte GE. Neonatal gastric perforation caused by congenital defects of the gastric musculature. *Surgery* 1960; **47**: 1010-1017 [PMID: 13793066]

20. **Shaw A**, BlancWA, Santulli TV, Santulli TV, Kaiser G. Spontaneous rupture of the stomach in the newborn: A clinical and experimental study. *Surgery* 1965; **58**: 561-571 [PMID:14338551]

21. **Gryboski JD**. The swallowing mechanism of the neonate: esophageal and gastric motility. *Pediatrics* 1965; **35**: 445-452 [PMID: 14260304]

22. **Jones TB**, Kirchner SG, Lee FA, Heller RM. Stomach rupture associated with esophageal atresia, tracheoesophageal fistula and ventilatory assistance. *AJR Am J Roentgenol* 1980; **134**: 675-677 [PMID: 6767350 DOI: 10.2214/ajr.134.4.675]

23. **Bruce J**, Bianchi A, Doig CM. Gastric perforation in the neonates. *Pediatr Surg Int* 1993; **8**: 17-19 [DOI: 10.1007/bf02352993]

24. **Young AE**, Sury MR. Spontaneous neonatal gastric perforation. *Pediatr Anesthesiol* 1996; **6**: 143-145 [PMID: 8846280 DOI: 10.1111/j.1460-9592.1996.tb00378.x]

25. **Saracli T**, Mann M, French DM, Booker CR, Scott RB. Rupture of the stomach in the newborn infant: report of three cases and review of the world literature. *Clin Pediatr (Phila)* 1967; **6**: 583-588 [PMID: 6077501 DOI: 10.1177/000992286700601014]

26. **Hood JH**. Clinical consideration on intestinal gas. *Ann Surg* 1966; **163**: 359–366 [PMID: 5907560 DOI: 10.1097/00000658-196603000-00006]

Table 1. Patient characteristics

Patient	Gender	Date of birth	GA (wks)	Term	BW (g)	Delivery	Symptom onset (day)	Time from symptoms to surgery (day)	pH	NG tube
A	F	1983-02-24	40+2	Full term	3500	Natural	5	1	N.D.	Yes
B	F	1987-07-09	40	Full term	2950	Natural	2	2	7.13	Yes
C	M	1990-09-05	35	Preterm	2190	Natural	2	2	7.03	Yes
D	M	1993-06-03	38+4	Full term	2950	C-sec	4	0	7.19	No
E	M	1999-05-13	36+3	Full term	2860	Natural	2	0	7.43	No
F	F	2003-05-10	32+6	Preterm	1960	C-sec	2	0	N.D.	Yes
G	M	2009-02-03	38	Full term	3620	Natural	0	1	7.086	Yes
H	M	2011-08-03	24	Preterm	730	Natural	4	1	7.058	Yes
I	F	2011-12-27	39+3	Full term	4040	Natural	2	0	7.391	Yes

Initial symptom	Diet	Ventilator	O_2 therapy	Pneumoperitoneum on X-ray	Associated anomaly	Maternal problem
High fever,	Yes	Yes	Yes	No	Diaphragmat	None

vomiting, dyspnea					ic eventration	
Vomiting dyspnea	Yes	Yes	Yes	No	Diaphragmatic eventration	None
Hematemesis	Yes	No	Yes	No		PROM
Abd. dist, vomiting, fever	Yes	No	No	Yes		None
Abd. dist	Yes	No	No	Yes		None
Abd. dist	No	No	No	Yes	TEF	None
Abd. dist	No	Yes	Yes	Yes	CDH	None
Metabolic acidosis, abd dist,	No	Yes	Yes	Yes	Antral web	PROM
Abd dist.	Yes	No	No	Yes		None

GA: gestational age; BW: birth weight; F: female; M: male; PROM: premature rupture of membranes; C-sec: cesarean section

Table 2. Intraoperative and postoperative outcomes

Patient	Surgical procedure	Perforated site	Length (cm)	NEC	Complications	Hospital stay (days)	Survival
A	R&A	Body, LC, PW	4	Yes	Wound problem	18	Alive
B	R&A	Body, PW	2.5	No	Sepsis	4	Deceased
C	Primary repair	Whole, GC	10	Yes	Sepsis	46	Deceased
D	Primary repair	Whole, GC	10	No		11	Alive
E	R&A	Body, GC	5	Yes		28	Alive
F	Primary repair	LC	3	No		40	Alive
G	Primary repair	Body, GC	1	No		24	Alive
H	Primary repair	(1) LC (2) body, AW	5	No		131	Alive
I	Primary repair	Antrum, AW	0.5	No	Wound problem	11	Alive

NEC: necrotizing enterocolitis; R&A: resection and anastomosis; LC: lesser curvature; PW: posterior wall; GC: greater curvature; AW: anterior wall

Table 3. Prognostic factor analysis

	Survival (n = 7)	Deceased (n = 2)	P-value
Male	4	1	1.000
Birth before 1990	1	2	0.083
Birth before 2000	3	2	0.444
Preterm	2	1	1.000
BW <2500 g	2	1	1.000
pH <7.30	3	2	1.000
NG tube	5	2	1.000
Diet	4	2	0.500
Ventilator	3	1	1.000
O_2 therapy	3	2	0.464
Pneumoperitoneum	6	0	0.083
Associated anomaly	5	1	1.000
Time from symptom onset to surgery >24 h	0	2	< 0.001
Length >2 cm	5	2	1.000
Primary repair	5	1	1.000
NEC	2	1	1.000

BW: body weight; NG: nasogastric; NEC: necrotizing enterocolitis