Neonatal Gastrik Perforasyon: 2002'den 2015'e Tek Merkezde Bir Deneyim

Neonatal Gastric Perforation: A Single-Center Experience From 2002 to 2015

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ÖZ

GİRİŞ ve AMAÇ: *Mide perforasyonu yenidoğan döneminde daha çok prematüre bebeklerde görülen ciddi ve hayatı tehdit eden bir sorundur. Etiyolojide çok çeşitli nedenler rol oynamaktadır. Bu retrospektif çalışmada, yenidoğan yoğun bakım ünitemizde mide perforasyonu tanısı alan yenidoğanlarda etyolojiyi,temel risk faktörlerini, klinik özellikleri, prognozu ve mortaliteyi belirlemeyi amaçladık.*

YÖNTEM ve GEREÇLER: *Bu çalışmaya 2002-2015 yılları arasında mide perforasyonu tanısı alan yenidoğanlar dahil edildi.Doğum ağırlığı, gebelik yaşı, cinsiyet, risk faktörleri, mide delinme zamanı, perforasyon yeri ve prognozu içeren veriler kaydedildi.*

BULGULAR: *Mide perforasyonu tanısı konulan 11 yenidoğan çalışmaya dahil edildi. Yenidoğanların medyan gestasyonel doğum ağırlığı 2.014 gr idi. Vakaların 2' si term, 9'u preterm idi. Hastaların 3'ü kız, 8 'i erkekti. Ana klinik bulgu abdominal distansiyondu ve tüm hastalarda pnömoperitoneum saptandı. Ortalama tanı yaşı 5,6 gündü.Hastaların 6'sında spontan mide perforasyonu, 5'inde ise sekonder mide perforasyonu saptandı.İki hastada tekrarlayan mide perforasyonu tespit edildi. Mide perforasyonun boyutu 0,5-2,5 cm arasında idi ve daha çok midenin büyük kurvaturunda saptandı.Bir hastadan alınan mide kası biyopsisinde interstisyel Cajal hücresinin olmadığı gösterildi.Ölüm oranı%45 idi.*

TARTIŞMA ve SONUÇ: *Mide perforasyonu, yeni doğan bebeklerin hayatını tehdit eden acil bir durumdur. Çoğunlukla distal obstrüksiyon veya interstisyel Cajal hücrelerinin yokluğuna bağlı olarak gelişebilir. Tekrarlayan mide perforasyonunu izlemek için mide kası biyopsi materyalinde interstisyel Cajal hücrelerini aramak önemlidir.*

Anahtar Kelimeler: *mide delinmesi, yenidoğan, interstisyel cajal hücresi*

ABSTRACT

INTRODUCTION: *Gastric perforation is a serious and life-threatening problem seen mostly in premature newborns in the neonatal period.Various causes play a role in the etiology.This retrospective study aimed to determine the etiology, basic risk factors, clinical features, prognosis, and mortality in newborns diagnosed with gastric perforation in our neonatal intensive care unit.*

METHODS: *Newborns diagnosed with gastric perforation between 2002 and 2015 were included in this study.Data including birth weight, gestational age,sex,risk factors,gastric perforation time,perforation location, and prognosis were recorded.*

RESULTS: *A total of eleven newborns diagnosed with gastric perforation were included in the study.The median gestational birth weight of the newborns was 2.014 g. Two of the newborns were term, and nine were preterm.Three of the newborns were female, and eight were male.The main clinical finding was abdominal distention, and pneumoperitoneum was detected in all newborns.The average age at diagnosis was 5.6 days.Spontaneous gastric perforation was detected in 6 newborns, and secondary gastric perforation was found in 5 newborns.Recurrent gastric perforation was detected in two newborns.The gastric perforation size was between 0.5 and 2.5 cm, and it was primarily detected in the greater curvature of the stomach.The absence of ınterstitial cells of Cajal (ICC) was shown in a gastric muscle biopsy taken from a newborn.The mortality rate was 45%.*

DISCUSSION AND CONCLUSION: *Gastric perforation is an emergency that threatens the life of newborns.It can often develop due to distal obstruction or the absence of ICC cells.It is essential to look for ICC cells in the stomach muscle biopsy material to monitor for recurrent gastric perforation.*

Keywords: *gastric perforation, newborn,iInterstitial cells of Cajal*

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INTRODUCTION

Gastric perforation (GP) is a severe and life-threatening problem often seen in premature newborns in the neonatal period. It was first described by Siebold in 1825 (1,2). In 1943, Herbut reported the first case of neonatal gastric perforation with a congenital defect in the muscles of the perforated gastric wall (3). Most previous reports on neonatal gastric perforation include limited case series and case studies. GP incidence accounts for 7.8% of all gastrointestinal perforations (GIP), the mortality rate is lower at 9.5% (4).

Much of the discussion about this rare entity centers on the causes and etiology (1-4). However, causes and etiologies remain controversial and difficult to determine. Recently, studies have focused on the study of potential prognosis factors for clinical outcomes. However, the relative significance of these possible factors to neonatal gastric perforation is uncertain and controversial (3).

A wide variety of mechanisms have been suggested in pathogenesis. Recent studies have shown the absence of interstitial cells of Cajal (ICC) as the reason for spontaneous gastric perforation (SGP), although the facilitating factors are prematurity, mechanical ventilation, or nasal ventilation (2,4). Although many theories have been suggested, the exact mechanism and pathogenesis of have not been fully clarified. Neonatal GP may be categorized as spontaneous or traumatic. Prematurity, sepsis, necrotizing enterocolitis (NEC), ischemia-asphyxia and steroid use have been reported as the most critical risk factors for SGP. It has been stated that traumatic gastric perforations developed after gastric tube placement, intubation attempt, and aggressive use of bag-mask ventilation (5,6). According to a recent study, in the neonatal period of focal intestinal perforation (FIP) (28%), NEC (24%), meconium peritonitis (MP) (14%), and meconium-related ileus (MRI) (10%) have been reported with high mortality rates in cases with FIP (28%) and NEC (20%) (4).

The aim of this study was to determine the etiology, basic risk factors, clinical features, prognosis, and mortality in newborns with gastric perforation in our neonatal intensive care unit.

MATERIAL and METHOD:

A retrospective analysis was made of the newborns diagnosed with GP in our neonatal intensive care unit between 2002 and 2015. During this period, 600 newborns were followed up annually in the unit. The preliminary diagnosis of GP in the newborn was made by pneumoperitoneum on radiography after clinical suspicion. The diagnosis was confirmed during surgery. Intraoperatively, a straight, sharply demarcated area with no hyperemia-hemorrhage focus was defined as spontaneous gastric perforation (SGP). Only newborns diagnosed with SGP were included in the study group. Newborns diagnosed with non-SGP intestinal perforation such as traumatic perforations or related with NEC were excluded from the study.

Parameters retrieved were gestational age, birth weight, gender, APGAR score, transfontanel ultrasonography, echocardiography, GP time, mode of delivery, main symptoms and signs, preoperative abdominal radiographic findings, time between the development of main symptom and surgical intervention (surgical intervention time), site of perforation, size of perforation, associated gastrointestinal anomalies and conditions, pathologic findings, and survival status.

The pathologic features of neonatal gastric perforation were well defined and can be classified into the absence or presence of musculature (7). Microscopic examination of the perforated gastric tissue showed that, at a distance away from the perforation, there was an absence of gastric musculature. The stomach wall in such areas was usually composed only of mucosa, muscularis mucosa, and the loose connective tissue of the submucosa and subserosa (8). In contrast, the absence of musculature was confirmed in only one patient by pathology reports documenting gastric musculature in perforated gastric tissue.

The diagnosis of Respiratory Distress Syndrome (RDS) was defined according to the European consensus criteria, and the diagnosis of NEC was defined according to the modified Bell criteria (9,10)

Ethical approval of the study was obtained from Sivas Cumhuriyet University Clinical Research Ethics Committee (2020-12/17).

RESULTS

Evaluation was made of a total of 16 gastric perforations, which were recurrent in 2 of the 11 newborns included in the study. Newborns with diagnosis confirmed with the demonstration of GP in the operation were included in the study. The demographic characteristics of the newborns are shown in Table 1. The average birth weight was 2014 g (range, 990-3600 g). There were three females and eight males, two full-term infants, and nine preterm infants. Of the latter, one was born at 23 weeks, two at 31 weeks, one at 32 weeks, two at 34 weeks, two at 35 weeks, and one at 36 weeks of gestation. One of the term newborns presented from home at the age of 8 days with the complaint of upper gastrointestinal bleeding and was diagnosed with gastric perforation (patient no. 7). In six newborns included in the study, a total of eleven gastric perforations were defined spontaneously during the operation. These GPs, defined as SGP, occurred four times in one case and three times in one case at different regions at different times, and all were in the greater curvature. The absence of ICC in the gastric biopsy taken from the perforation site was reported in one newborn as a result of histopathological examination. No information was obtained regarding pathological examination in this regard in previous cases.

The mean time of GP in all newborn groups was 5.6 days after birth and ranged from 2 to 17 days. Perforation was detected in the greater curvature in 9/11 (80%) cases, in the lesser curvature in one newborn, and in the esophageal cardiac junction in one newborn.

The perforation width varied between 0.5-2 cm. The mortality rate of the newborns was 45% (5/11). Four of five newborns with secondary gastric perforation had gastrointestinal system anomalies. Two of these newborns had anal atresia and esophageal atresia; one had esophageal atresia and choanal atresia; one had annular pancreas. One newborn had a diagnosis of Down's Syndrome. Six newborns with esophageal atresia and RDS were intubated and received mechanical ventilation therapy. Two newborns were diagnosed with gastric perforation while receiving non-invasive ventilation, and two while receiving hood oxygen therapy.

Table.1 Characteristic of our newborns with gastric perforation

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8	Case 9	Case 10	Case 11
Gestation Age	23	39	31	34	35	36	39	31	35	32	34
Weight (gr)	990	1110	1510	2250	2310	3000	3600	1530	2350	2160	1350
Gender	Female	Female	Male	Male	Female	Male	Male	Male	Male	Male	Male
Apgar	8	8	10	8	8	8	8	7	8	8	7
TFUS	Normal	Normal	Grade 3-4 Hemorrhage	Normal	Grade 3-4 Hemorrhage	Normal	-----	Normal	Normal	Normal	------
ECHO	PDA 2,3 mm	Normal	----	Normal	-----	----	-----	-------	-----	VSD+ASD, PDA(2,6 mm)	VSD; Intermittent Aortic Arch, PH
GP Time (Day)	5	2	3,6,7,9	4,5,9	3	2	7	10	17	3	2
Type of Birth	C/S	C/S	C/S	C/S	C/S	C/S	C/S	C/S	NVD	C/S	C/S
Perforation Region	Greater Curvature	Esophagus-cardia junction	Greater Curvature	Greater Curvature	Greater Curvature	Greater Curvature	Greater Curvature	Greater Curvature	Greater Curvature	Lesser Curvature	Greater Curvature
Death	Yes	No	No	No	Yes	No	Yes	Yes	No	No	Yes
Surgical Diagnosis	Spontaneous	Secondary	Spontaneous	Spontaneous	Spontaneous	Spontaneous	Spontaneous	Secondary	Secondary	Secondary	Secondary
Congenital Anomaly	No	Annular Pancreas	No	No	No	No	No	Anal and Esophageal Atresia	Anal and Esophageal Atresia	Down Syndrome	Esophageal Atresia, Choanal Atresia
NEC	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO

Abbreviations: C/S: Cesarean; NVD: Normal vaginal delivery; GP: Gastric perforation; ECHO: Echocardiography; TFUS:Transfontanelle ultrasonography; NEC: Necrotizing enterocolitis;

VSD: Ventricular septal defect; ASD: Atrial septal defect; PH: Pulmoner hypertension; PDA:Patent ductus arteriosus

DISCUSSION

This study is a systematic review of the literature to try to identify pathogenetic and prognostic factors in neonates with gastric perforation.

The causes and etiology of neonatal gastric perforations remain controversial and unclear. Perforation occurs in the stomach due to the increase in intragastric pressure due to insufficient development of pneumatic or GI systems during mechanical ventilation and insufficient vomiting reflex (11). Touloukian et al. suggested the vascular theory that ischemia occurring in the neonatal period may be the etiological cause (12). NEC, stress ulcer, gastric intubation, corticosteroids, or non-steroidal anti-inflammatory drugs are among the other etiological causes thought to be responsible (13). Although there are generally none of the risk factors, GP in newborns is called "spontaneous neonatal gastric perforation" (13,14). In the current study, six newborns were defined as SGP because of the absence of histopathological data to cause SGP, and also because the perforated lesion was a flat, sharply demarcated area without a focus of hyperemia-hemorrhage during the operation.

GP usually occurs in the greater curvature or fundus region. In the current case series, higher rates of perforation were determined in the greater curvature, consistent with the above-mentioned literature (Table 1). Leone et al. also found a perforation in the greater curvature in 5/7 (71%) cases with gastric perforation (5). Kara et al. reported perforation in the lesser curvature at the

rate of 60% in their study (15). Perforation was found in the small curvature in 3/5 (60%) cases in a study by Duran et al. (16), and in the greater curvature in 4/9 (44%) cases in a study by Byun et al. (17). Although the GP is in the greater curvature in most of the cases in literature, series have also shown perforation in the lesser curvature, as reported in the above-mentioned studies (13). Lacusso et al reported GP in the greater curvature in 50% of 207 cases and Babayiğit et al similarly found GP at a higher rate in the greater curvature (18, 19).

Newborns with GP were defined as SGP in the studies conducted in the 2000s (20). Distal obstruction, tracheoesophageal fistula, nasal ventilation, steroid use, prematurity, and chorioamnionitis have been shown to be risk factors. Starting to cry too quickly is also counted among the iatrogenic causes (21). In the current study, there was no chorioamnionitis, asphyxia, or steroid use. Secondary causes (anal-esophageal atresia, Down Syndrome, esophagus, and choanal atresia) were found in five cases. Inouye and Evans detected gastric obstruction as the cause in 10 (7%) of 143 cases with GP in 1964 and the cause of distal obstruction in these cases was reported to be duodenal atresia in 3 cases, malrotation in 2 patients, pyloric stenosis or atresia in 2 patients, ileal stenosis in 1 case, a superior mesenteric vessel in 1 case, and meconium ileus in 1 case (20). Case series with GP and distal obstruction are shown in Table 2 (1,5,6,11,14-16,20,22-25).

Recent studies have shown the absence of ICC as a reason for SGP (2). ICCs are responsible for gastric motility and play a role in signal transduction with voltage-independent calcium channels, ensuring contraction of the gastric muscles through this signal. Five subtypes have been identified, and these function as pacemakers by settling in the myenteric plexus (26). Hypomotility due to the decrease in ICC has been shown as a cause of non-iatrogenic GP in previous studies (26). Jactel et al. reported that six infants with GP and no primary cause did not have ICC (26). In the current study, there was seen to be no ICC in the gastric material taken from case 2 when a gastric biopsy was taken for pathology as a result of having been reported as spontaneous by the surgeon (Table 1).

Mortality due to GP ranges from 27 to 83% (1-10). In the current study it was approximately 45%. Surgical results are excellent when diagnosis is made early, and metabolic stability is achieved. However, when there is a delay in diagnosis and metabolic imbalance, the cases become irreversible, and the risk of mortality increases (11,27).

It is crucial to identify the absence of ICCs by looking at the Cajal cells pathologically, especially in patients with no additional problems who are considered to have SGP. It is a warning that perforation may recur in these patients, and monitoring should be done accordingly.

This research had some limitations, primarily that because of the retrospective design, it was not possible to randomize newborns with a GP diagnosis. The second limitation of the study was that the medical records of all the newborns could not be fully accessed and gastric biopsy could not be obtained from newborns who had previously been operated on.

In conclusion, although GP is not a common diagnosis, it is a life-threatening emergency for newborns. GP develops due mainly to distal obstruction or the absence of ICC cells, as seen in the present and previous studies. Therefore, early diagnosis, appropriate fluid, and early surgical treatment are essential. If spontaneous gastric perforation is considered during the operation, the

presence of ICC cells must be investigated in the biopsy material for the follow-up of recurrent gastric perforation. In terms of this issue, cohort studies and long-term prospective studies will contribute to the literature.

Table 2. Characteristics of newborns in the literature with gastric perforation

Authors	Year	Male/ Female	Preterm/Term	Gestational Age (Week)	Body Weight (gram)	PN Age (Day)	Spontaneous	Etiology	Mortality Rate (%)
Inouye WY[22] (n:143)	1964	74/51 Unknown n:18	44/69 Unknown n:30	---	--	5	40/143 (%27)	10 DO, 5 KA,4 D 3 M,3 IKB,3 CR,2 DH,2 TEF,1 O, 1P,1 MIC, 1AC,1 PDA,1 Hemangioma	72
Shashikumar VL[1](n:19)	1975	9/10	2/17	38	2478	3,3	19	0	45
HolgersenLO[24] (n:28)	1981	16/12	16/12	-	-	2-7	27	1- Duodenal atresia	23
Rosser SB[25] (n:16)	1982	13/3	10/6	37,5	2528	3,1	16	0	25
Chung MT[23](n:12)	1994	9/3	-	-	-	-	10	1-PROM 1-TEF	58
Leone RJ [5] (n:7)	2000	3/4	3/4	33	1942	8	2	2 –NEC 1-TEF 1-Nasal CPAP 1- Meconium plug	57
Jawad AJ[20] (n:5)	2002	2/3	-	33	1830	6	1	2- NEC 1-RDS-MV 1- Asphyxia -MV	0
Öztürk H [14] (n:5)	2003	4/1	2/3	-	-	-	3	1-TEF 1- Dexamethasone	60
Kara SC[15] (n:12)	2004	11/12	4/13	29-35	2375	3,2	11	1-Intestinal volvulus	54
Duran R[16] (n:5)	2007	3/2	3/2	32	1650	10	1	1-RDS-MV 1-RDS-nSIMV 1-EA, TEF, RDS-MV,1-Asphyxia, Dexamethasone, MV	60
Terui K[11] (n:11)	2012	-	0/11	38	2800	3,5	6	3 volvulus, 1 jejunal stenosis, 1 diaphragm eventration	53
Yang CY[6] (n:13)	2014	9/4	5/8	28-39	3200	3	4	2 malrotations, 5 NEC, 1 gastroschisis, 1 duodenal web, 1 Meckel diverticulum,	46
Our patients (n:11)	2002-2015	3/8	9/2	23-39	2014	5,6	6	1 Annular pancreas, 3 esophageal atresia, 1 Down syndrome	45

Abbreviations:CPAP: Continuous positive airway pressure; EA: Esophageal atresia; PROM: Premature rupture of membranes; RDS Respiratory distress syndrome; SIMV: Synchronized intermittent mandatory ventilation; TEF: Tracheoesophageal fistula; ICC: Interstitial cells of Cajal; DO: Distal obstruction; KA: Kidney anomaly; M: Mongolism; IKB: Intracranial bleeding; CR: Cryptorchidism; DH: Diaphragmatic hernia; O: Omphalocele; P: Polyhydramnios; MIC:Microphthalmia; AC: Aortic coarctation; PDA: Patent ductus arteriosus; MV: Mechanical ventilation

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