Ripped from The Cradle: Neonatal Gastric Perforation - A 10-Year Review of a Devastating Condition

Waseem Ashraf [1*], Shyam Bihari Sharma[2], Samarth Sinha[3], Shubham Gupta[4], Umesh Kumar[5], Anurag Kumar[6], Ankit Sachan[7]

[1] Assistant professor, Department of Urology, Kanti Devi Medical College Hospital and Research Center, Mathura, Uttar Pradesh.

[2] Professor, Department of Pediatric Surgery, Kanti Devi Medical College Hospital and Research Center, Mathura, Uttar Pradesh.

[3] Post Graduate Scholar in General Surgery, Kanti Devi Medical College Hospital and Research Center, Mathura, Uttar Pradesh.

[4] Post Graduate Scholar in General Surgery, Kanti Devi Medical College Hospital and Research Center, Mathura, Uttar Pradesh.

[5] Senior Resident, Kanti Devi Medical College Hospital and Research Center, Mathura, Uttar Pradesh.

[6] Post Graduate Scholar in General Surgery, Kanti Devi Medical College Hospital and Research Center, Mathura, Uttar Pradesh.

[7] Post Graduate Scholar in General Surgery, Kanti Devi Medical College Hospital and Research Center, Mathura, Uttar Pradesh.

***Address for Correspondence:** Dr Waseem Ashraf, Assistant professor, Department of Urology, Kanti Devi Medical College Hospital and Research Center, Mathura, Uttar Pradesh. (email: Waseemashraf1279@gmail.com)

How to cite this article:

Ashraf W, Bihari Sharma Sh, Sinha S, Gupta Sh, Kumar U, Kumar A, Sachan A. Ripped from the Cradle: Neonatal Gastric Perforation - A 10-Year Review of a Devastating Condition. Iranian Journal of Pediatric Surgery 2025; 11(2): 150 – 178.

DOI: https://doi.org/10.22037/irjps.v11i2.47153

received: 31 December 2024
accepted: 25 August 2025
Published online: 26 October 2025

Abstract

Introduction: Neonatal gastric perforation (NGP) is a rare but life-threatening condition affecting neonates. The exact mechanism of NGP is poorly understood, but it's often associated with preterm birth, hypoxia, and sepsis. Spontaneous cases of NGP are unpredictable, resulting in complicated diagnosis and management. In the present study, we elucidate the risk factors, clinical findings, management strategies, and outcomes in patients retrospectively over a period of 10 years.

Materials and Methods: A retrospective study of all patients presenting with NGP was conducted from March 2013 to March 2023 in the Department of Pediatric Surgery, KD Medical College and Research Centre, Mathura, and complete data of 87 of these patients were accrued and analyzed. Complete demographic data, preoperative characteristics, clinical presentation, intraoperative characteristics, clinical outcomes, and postoperative complications were recorded and analyzed. A comparison was made between the surviving and non-surviving neonates to elucidate the risk factors associated with mortality.

Results: A total of 87 cases of neonatal gastric perforation were included in our study. The majority of NGP patients were male (73.56%, M: F = 2.78:1), preterm (59.77%), and of low birth weight (51.72%). Common clinical presentations included abdominal distension (88.5%), vomiting (71.26%), and respiratory distress (57.47%). Key laboratory findings included a mean hemoglobin of 15.84 ± 1.2 g/dl, a leukocyte count of 18.71 ± 4.12 × 10^3/µl, and a serum pH of 7.26 ± 0.14.

Radiological findings prominently showed the "football sign" (93.1%) and gas under the diaphragm (74.71%). Gastric perforations predominantly involved the greater curvature (36.78%) and were mostly single (82.75%) and small (<5 cm, 65%). Spontaneous perforation was the most common cause (65%), followed by trauma (21%) and necrotizing enterocolitis (NEC, 10%). Surgical management included gastric repair with peritoneal lavage and venting gastrostomy. Postoperative complications included respiratory issues (74 cases), sepsis (54 cases), and wound infections (25 cases). Neonatal mortality was 42.53%, with significant predictors including male sex (p value = 0.004), prematurity (p value = 0.002), low birth weight (p < 0.001), thrombocytopenia (p < 0.001), high CRP levels (p < 0.001), acidosis (p < 0.001), and elevated lactate levels (p < 0.001).

Conclusion: Neonatal gastric perforation presents a substantial risk of mortality, particularly among male, preterm, and low birth weight infants. Early recognition of clinical signs, prompt surgical intervention, and vigilant postoperative care are crucial to improving survival rates. Monitoring laboratory indicators such as platelet counts, CRP levels, pH, and lactate levels can aid in assessing prognosis and guiding treatment strategies.

Keywords

- Neonatal gastric perforation
- Pneumoperitoneum in neonates
- Neonatal abdominal emergencies
- Venting gastrostomy
- Exploratory laparotomy

Introduction

Neonatal gastric perforation (NGP) is a rare but life-threatening condition with significant mortality in newborns[1-3]. It represents around 7% of neonatal gastrointestinal perforations and occurs at a rate of 1 in every 2900 live births[4-5].

Historically, NGP carried a 100% mortality rate, particularly in low birth weight neonates, but with improvements in neonatal care, the mortality rate has significantly decreased[6]. The first reported case of neonatal gastric perforation was by

Siebold et al. in 1825, whereas the first reported survival of NGP was reported by Legar et al. in 1945[7].

Despite the significant improvement in mortality from NGP due to improved neonatal care, neonatal gastric perforation (NGP) is considered a poorly understood condition[8]. NGP is often associated with preterm birth, hypoxia, and sepsis, but spontaneous or idiopathic cases lack a definitive explanation, which complicates diagnosis and management[9]. The present study on NGP patients was conducted from March 2013 to March 2023 at a single tertiary care center. This study presents our experience with neonates diagnosed with NGP to elucidate the risk factors, clinical findings, management strategies, and outcomes observed in these patients during this period.

Materials and Methods

This study was a retrospective design conducted from March 2013 to March 2023. Between March 2013 and March 2023, 1,145 neonates (aged ≤30 days) presented to the neonatal emergency department of KD Medical College with features of pneumoperitoneum. Among them, 87 cases were identified as Neonatal Gastric Perforation (NGP) on exploratory laparotomy, accounting for 7.6% of all intestinal perforations in neonates.

Comprehensive data of these 87 patients were collected, including demographic details preoperatively such as gender, age at presentation, gestational age, birth weight, mode of delivery, presence of neonatal asphyxia, key clinical signs and symptoms, gastric tube placement, mechanical ventilation before the onset of perforation, and the perinatal risk factors, associated congenital anomalies (both gastrointestinal and extra-intestinal, including congenital heart disease), preoperative radiological findings. Intraoperatively, the surgical intervention done, the site and size of the perforation, clinical outcomes, and postoperative complications were recorded. Data analysis was performed using SPSS version 26, with qualitative variables presented as mean ± standard deviation and quantitative variables reported as frequencies and percentages.

Result

A total of 1,145 patients were admitted to the neonatal surgery department as cases of

neonatal acute abdomen meriting an exploratory laparotomy between March 2013 to March 2023. Of these, 87 patients had gastric perforation on exploratory laparotomy, accounting for 7.6% of all gastrointestinal perforations in neonates. **Table 1** shows the preoperative characteristics of the study population. Of the NGP patients, 73.56% (n=64) were males, making the M:F ratio of NGP as 2.78:1. The majority of the patients were born by normal vaginal delivery (77.01%, n=67). 59.77% (n=52) of patients were preterm neonates, while 51.72% (n=45) of patients were low birth weight (<2500 gms) and 1.15% (n=1) were very low birth weight (<1500 gms) neonates. The mean ± SD birth weight of these patients was 2396.09 ± 386.43gms. Of the 87 patients studied, 40 patients (45.97%) had respiratory distress in the neonatal period and needed respiratory support in the neonatal ICU. While 28 (32.18%) patients had associated Necrotizing enterocolitis (NEC) at laparotomy. Other associated congenital anomalies in these patients included duodenal atresia (16.09%, n=14), intestinal malrotation (12.64%, n=11), and congenital heart disease (8.04%, n=7). The average presentation of symptoms of these patients was 2.11 ± 0.64 days (1-4 days). The most frequent presentations at admission **(Figure 1)** were abdominal distention (88.5%, n=77), followed by vomiting (71.26%, n=62), feeding intolerance (55.17%, n=48), respiratory distress (57.47%, n=50), lethargy (45.98%, n=40), melena (34.48%, n=30), and hemodynamic instability (25.28%, n=22). On examination, the findings on physical examination were the absence of bowel sounds (n=23, 26.44%), abdominal erythema (n=15, 17.24%), prominent abdominal veins (n=19, 21.84%), and cyanosis (n=56, 64.37%).

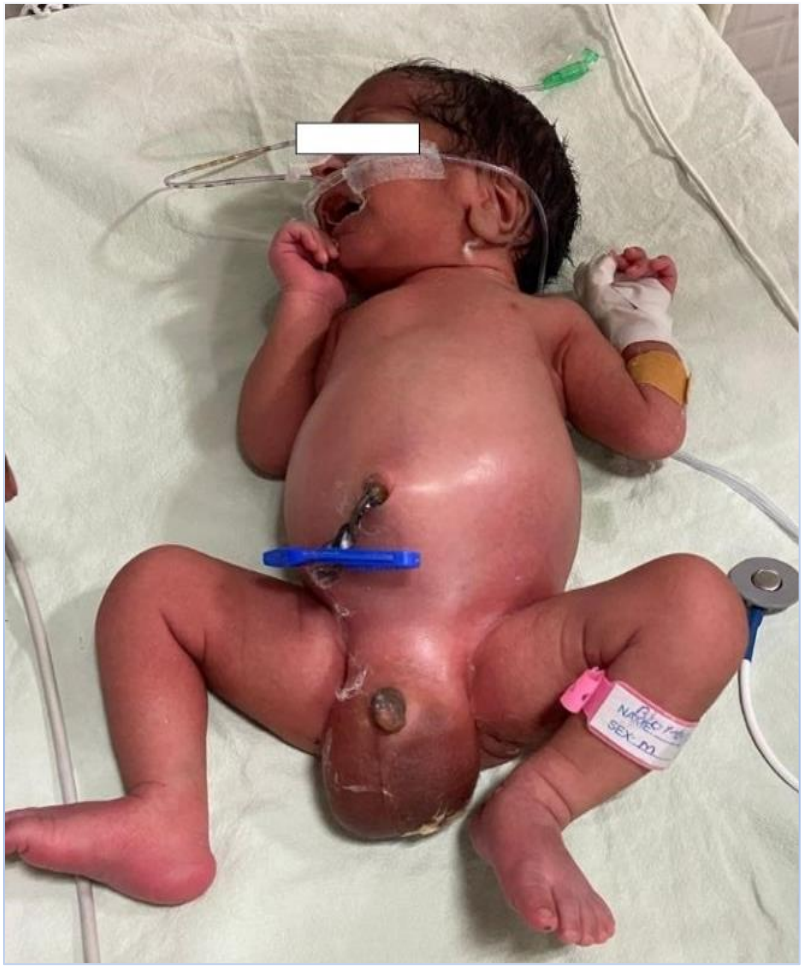

Figure 1: Presentation of NGP with abdominal distention and pneumoscrotum

On laboratory analysis, the mean hemoglobin was 15.84 ± 1.2 g/dl, mean total leukocyte count (x1000/µl) was 18.71 ± 4.12, and the mean platelet count (x1000/µl) was 194.44 ± 50.91. The mean serum pH was 7.26 ± 0.14, and the serum lactate level was 2.57 ± 0.96 **(Table 1)**.

Table 1: Preoperative characteristics of the study population of NGP

Parameter	Value
Gender	
Male	64(73.56%)
Female	23(26.44%)
Gestational Age(weeks)	
35.39 ± 2.19	
Gestational Age	
Term pregnancy	35(40.23%)
Preterm pregnancy	52(59.77%)
Mode of delivery	
Normal	67(77.01%)
C section	20(22.99%)
Birth weight(gms)	
2396.09 ± 386.43	
Birth Weight	
normal	41(47.13%)
low birth weight	45(51.72%)
very low birth weight	1(1.15%)
Perinatal complications	
neonatal asphyxia	31(35.63%)
Mechanical ventilation	21.58(24.8%)
Nasogastric tube insertion	69(79.31%)

Associated anomalies	
Congenital heart disease	7(8.04%)
duodenal atresia	14(16.09%)
Intestinal malrotation	11(12.64%)
Respiratory distress	40(45.97%)
NEC	28(32.18%)
Age of presentation for surgery(days)	2.11 ± 0.64
Clinical features	
Abdominal distention	77(88.5%)
Vomiting	62(71.26%)
Feeding intolerance	48(55.17%)
Respiratory distress	50(57.47%)
Lethargy	40(45.98%)
melena	30(34.48%)
Haemodynamic instability	22(25.28%)
Signs	
absence of bowel sounds	23(26.44%)
abdominal erythema	15(17.24%)
prominent abdominal veins	19(21.84%)
cyanosis	56(64.37%)
laboratory features	
HB(g/dl)	15.84 ± 1.2
TLC(x1000/µl)	18.71 ± 4.12
platelet count(x1000/µl)	194.44 ± 50.91
pH	7.26 ± 0.14
Sr lactate	2.57 ± 0.96
Radiological features	
Football sign	81(93.1%)
Cupula sign	76(87.36%)
Gas under diaphragm	65(74.71%)

The radiological features of these Neonatal gastric perforation patients included the "football sign" with or without pneumoscrotum (n=81, 93.1%) **(Figure 2 and 3)**, cupula sign (n=76, 87.36%), and gas under the diaphragm (n=65, 74.71%).

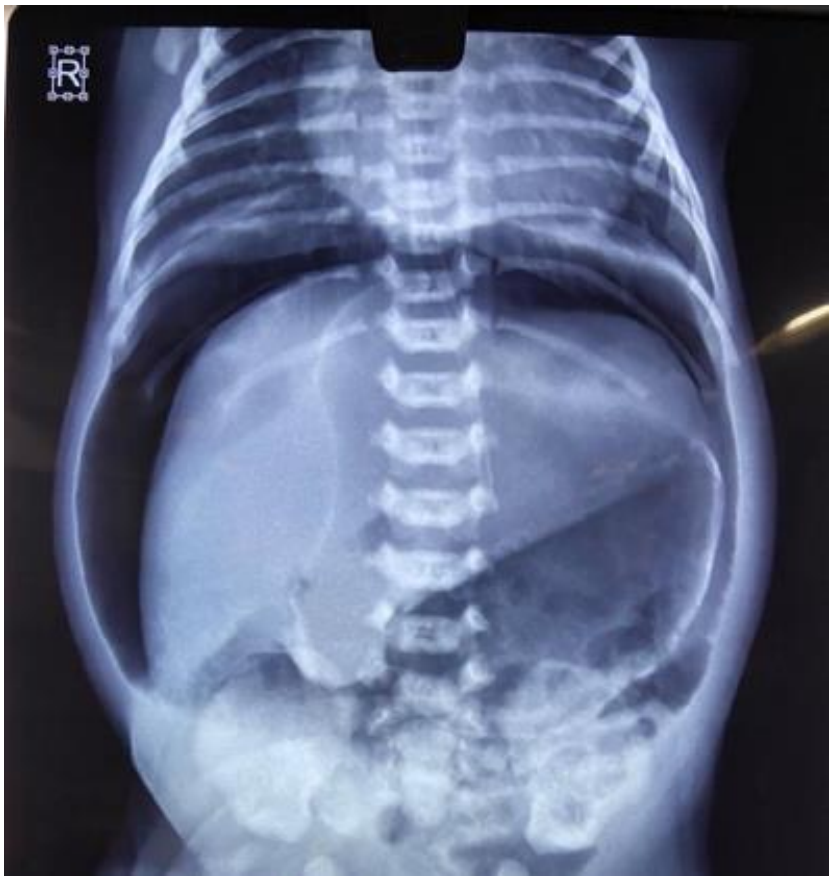

Figure 2: X-ray Film showing “Football Sign”

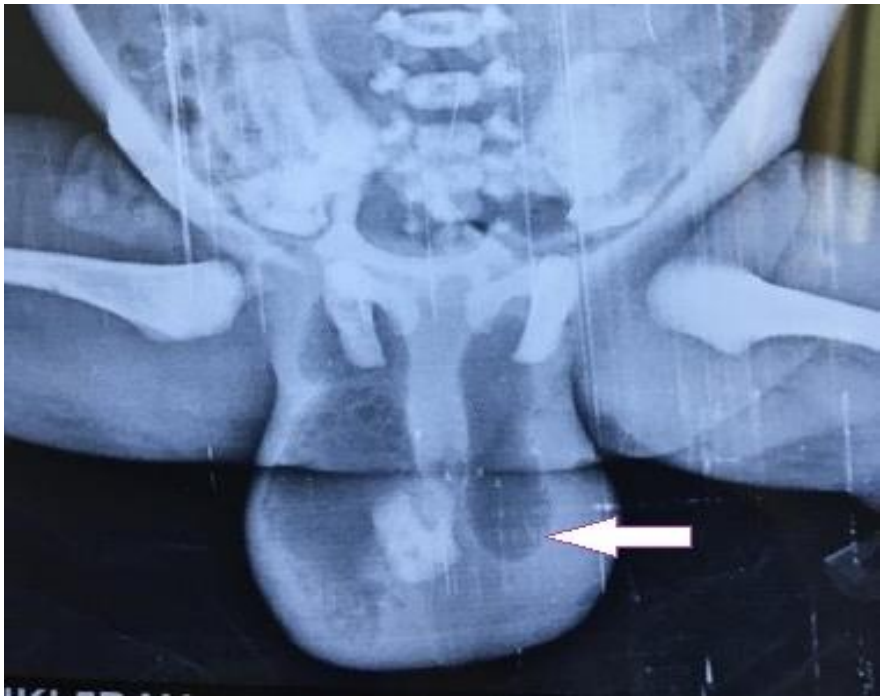

Figure 3: X-ray Film focussed on pneumoscrotum. (arrow)

Table 2 shows the intraoperative characteristics of the patients with Neonatal gastric perforation. The average timing of surgical intervention (in days) in NGP was 5.23 ± 1.14 (range 3-9), while the operative time (in hours) was 1.82 ± 0.61 (range 1-3.2). Exploratory laparotomy was attempted in these patients with repair of gastric perforation and lavage of the peritoneal cavity. On exploratory laparotomy, it was found that the greater curvature (n=32, 36.78%) was involved in most of the cases of gastric perforation **(Figure 4)**, followed by the lesser curvature (n=28, 32.18%), anterior wall (n=23, 26.44%), and the posterior wall of the stomach (n=17, 19.54%).

Majority of the patients had single perforation (82.75%) and small-sized perforation of size less than 5cm (65%). In the majority of the cases, the possible cause of perforation appeared spontaneous (65%) as no etiology on exploratory laparotomy was found and hence was labeled as spontaneous. Traumatic gastric perforation

(21%) was due to traumatic nasogastric tube placement before surgery or because of positive pressure ventilation. In 10% of cases, gastric perforation was associated with NEC as necrotic patches were found in some portion of the small gut, and also associated multiple perforations were found in portions of the small gut.

In addition to NGP, 17.2% of patients (n=15) had associated gastrointestinal anomalies, with intestinal malrotation being the most common associated gastrointestinal anomaly (n=11, 12.64%), followed by duodenal atresia (3.4%) and Meckel's diverticulum (2.3%) intraoperatively **(Table 2)**.

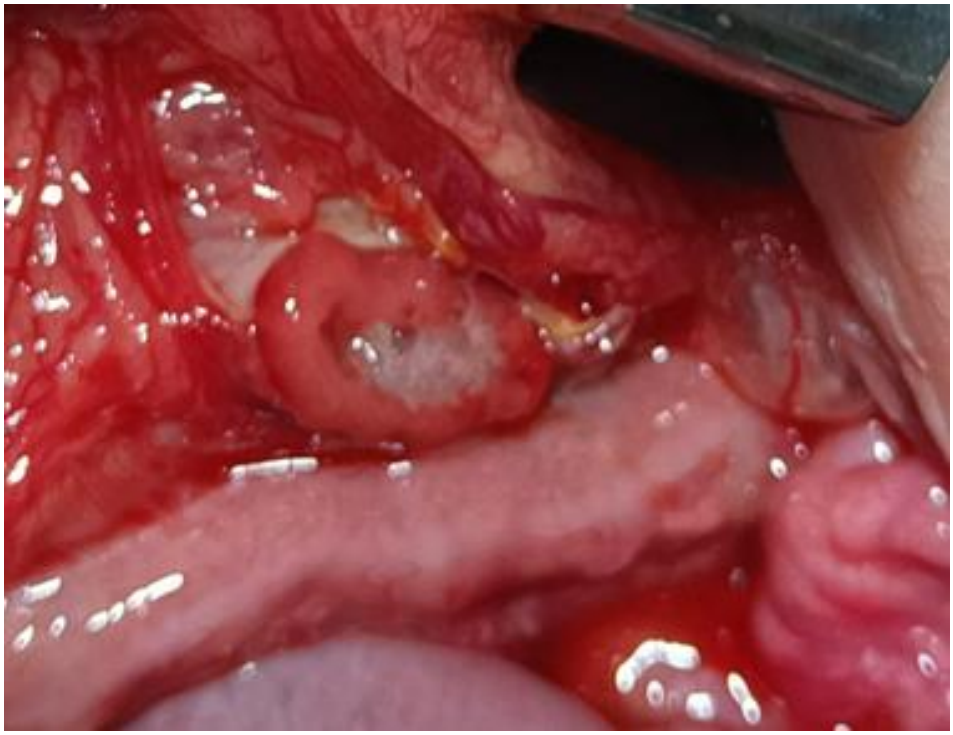

Figure 4: Intraoperative picture showing perforation along greater curvature with healthy margins

Table 2: Intraoperative characteristics of the patients with NGP

Intraoperative features	Value
Time of surgical intervention(days)	5.23 ± 1.14
Operative time(hours)	1.82 ± 0.61
Site of perforation	
Greater curvature	32(36.78%)
Lesser curvature	28(32.18%)
anterior wall	23(26.44%)
Posterior wall	17(19.54%)
Number of perforations	
Single	83%
Multiple	17%
Size of perforation(cms)	
1-5 cm	65%
5-10 cm	23%
>10cm	12%
possible causes of perforation	
Spontaneous	65%
Traumatic	21%
NEC	10%
ischaemic	4%
Associated anomalies	15(17.2%)
intestinal malrotation	11(12.64%)
duodenal atresia	3(3.4%)
Meckels diverticulum	2(2.3%)
Surgical procedure	
Gastroraphy+venting gastrostomy	70(80.5%)
Debridement+Gastroraphy+venting gastrostomy	17(19.5%)

Multiple perforations were seen in 17% of cases. Only 2 cases had ischemic perforation that needed debridement in addition followed by primary closure as these patients had devitalized tissue in the perforated area that needed debridement **(Figure 5)**. Repair of the perforation was done in a single layer using vicryl 4/0 suture **(Figure 6)**.

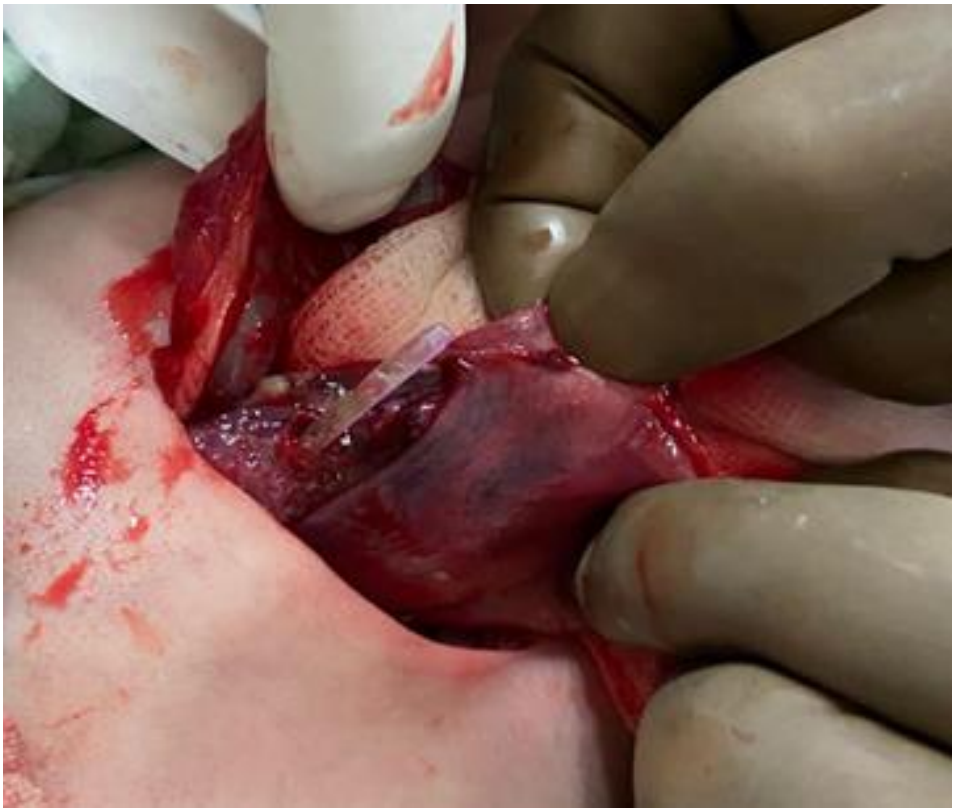

Figure 5: Protrusion of feeding tube through the perforation, with sloughing of gastric mucosa, surrounded by necrotic area. (extensive involvement)

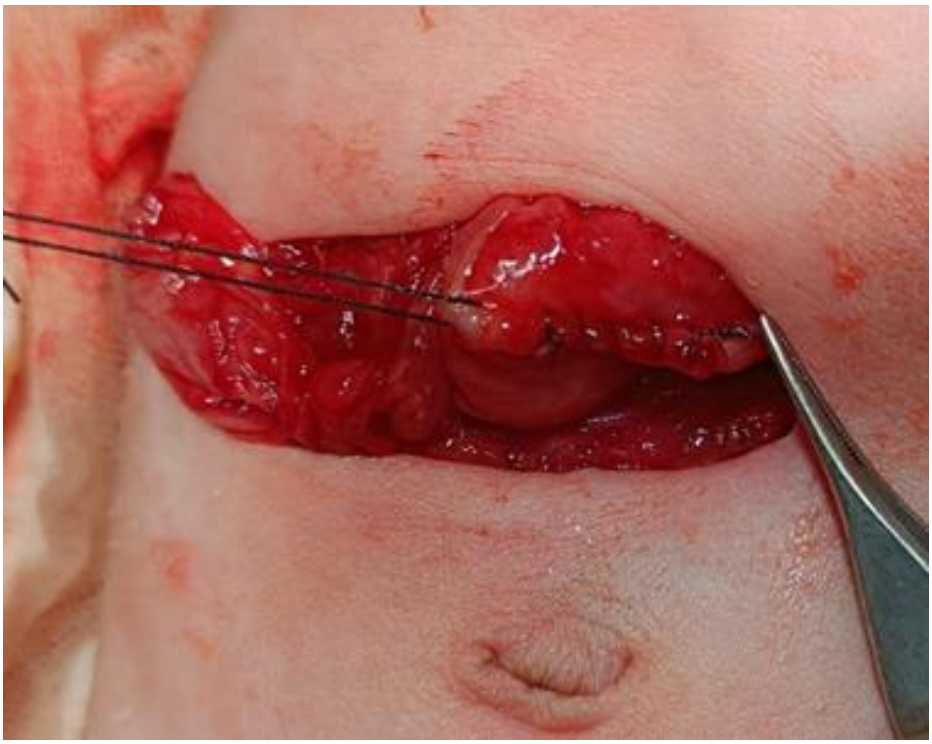

Figure 6: Repair of Gastric perforation

Gastrorrhaphy with venting gastrostomy, that is, the repair of gastric perforation with the placement of a proximal gastrostomy tube for decompression, was done in the majority of cases, though some patients needed debridement of devitalized tissue prior to repair **(Figure 7)**.

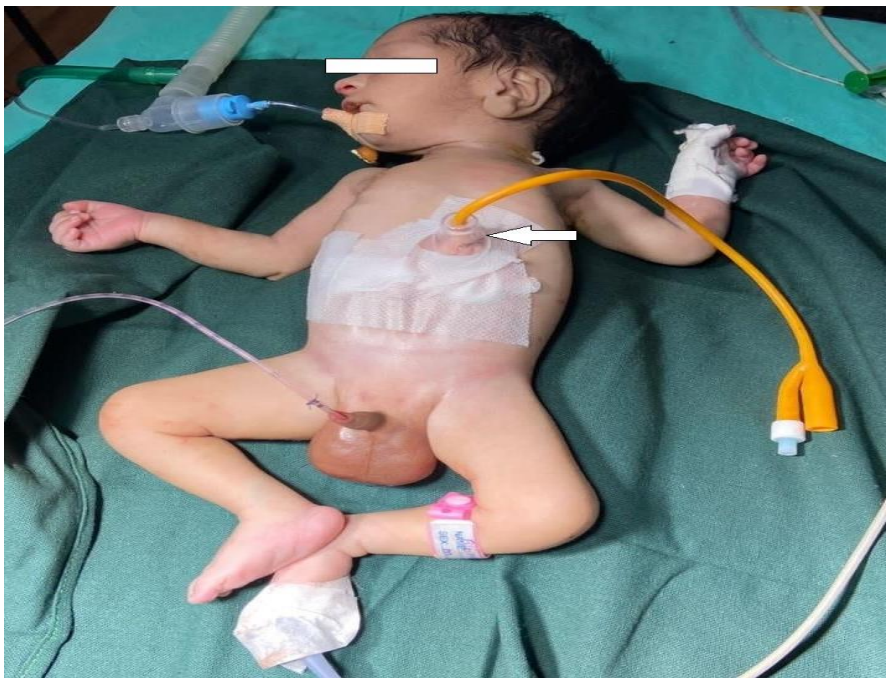

Figure 7: Venting Gastrostomy, post perforation repair. (arrow)

The post-operative complications are shown in **Table 3**. Postoperatively, a total of 74 neonates developed respiratory complications, out of which 28 neonates needed ventilator support in the post-operative period. 54 patients developed postoperative sepsis. 25 patients developed wound infection, out of which 11 patients developed burst abdomen. 37 patients did not survive the neonatal period, making the neonatal mortality from NGP 42.53%.

Table 3: Postoperative complications of the study population

Post-operative complications	N (%)
Post-operative sepsis	54(62.07%)
Respiratory complications	74(85.06%)
Wound infection	25(28.74%)
Burst abdomen	11(12.64%)
Mortality	37(42.53%)

The comparison of risk factors for survival in NGP is shown in **Table 4**. Subgroup classification of all cases of neonatal gastric perforation was done into two groups based on their survival to identify the risk factors for NGP-based mortality. The major clinical predictors of mortality for NGP were male gender (p value = 0.004), preterm delivery of less than 37 weeks (p = 0.002), low birth weight (p < 0.001). Among the laboratory predictors of mortality, thrombocytopenia (p < 0.001), high CRP levels (p < 0.001), acidosis (p < 0.001), and high lactate levels (p < 0.001) were significant predictors of mortality in neonatal gastric perforation patients.

Table 4: Mortality comparison in the patients of Neonatal gastric perforation

Parameter	Total	Alive (50)	Dead (37)	P value
Male Gender	64	31(38.75%)	33(89.19%)	0.004
Preterm delivery	52	23(46%)	29(78.38%)	0.002
Birth weight(grams)	2396.09 ± 386.43	2524.56 ± 345.92	2222.49 ± 374.12	<0.001
Birth weight Ordianal				
normal	41	31	10	

low	45	19	26	0.004
very low	1	0	1	
Mode of delivery				
normal	67	43	24	
caseraian	20	7	13	0.021
Gestational age(weeks)	35.39 ± 2.19	36.12 ± 2.07	34.41 ± 1.98	<0.001
Term/preterm delivery				
Term (>37 weeks)	35	27	8	
Preterm (<37 weeks)	52	23	29	0.002
Day of surgery	5.23 ± 1.14	5.3 ± 1.22	5.14 ± 1.03	0.547
Age of admission	2.11 ± 0.64	2.04 ± 0.64	2.22 ± 0.63	0.204
HB (gm/dl)	15.84 ± 1.2	15.72 ± 1.28	16 ± 1.08	0.284
TLC count (x1000/µl)	18.71 ± 4.12	19.26 ± 3.73	17.98 ± 4.55	0.153
platlet count (x1000/µl)	194.44 ± 50.91	223.72 ± 38.56	154.86 ± 36.99	<0.001
CRP levels(mg/dl)	4.37 ± 2.01	3.04 ± 0.75	6.16 ± 1.76	<0.001
pH	7.26 ± 0.14	7.34 ± 0.09	7.15 ± 0.11	<0.001
Lactate (mmol/l)	2.57 ± 0.96	2.27 ± 0.69	2.98 ± 1.11	<0.001

Discussion

Neonatal gastric perforation is a rare but life-threatening condition in neonates, representing a small subset of acute abdomen cases with high morbidity and mortality. In our ten-year analysis, we found neonatal gastric perforation comprising 7.6 percent of all cases of intestinal perforation. The percentage of neonatal gastric perforation mostly reported in the literature is 7%[10-15] which is slightly less than reported in our study.

The etiopathogenesis of neonatal gastric perforation remains poorly understood, although a number of theories have been put forward to explain the initial stages of the disease. Congenital muscular defect theory has been put forward by Herbut et

al. [16], attributing NGP to a congenital defect in the musculature of the stomach, suggesting it to develop from the improperly developed circular muscle layer of the stomach. Another theory known as the neurogenic theory proposed by Harvey Cushing et al. [17] attributed NGP to aberrations in central nervous system development leading to gastric erosion, ulcers, and perforation. Meconium plug theory or the hydrostatic pressure theory by Russel hypothesized that the causative effect of gastric perforation might be the hydrostatic pressure that is being applied to the intestine during delivery.[18]. The main accent of this hypothesis was that the pressure that is building up is not distributed along the length of the gastrointestinal tract. One of the major explanations clarifying this concept was the presence of a meconium plug, which is generating excess physical forces on certain areas of the intestine. Increased gastric acid production as a possible cause of gastric perforation has been proposed as well[19]. Furthermore, experimental studies have been undertaken providing evidence against the concept of a congenital basis for gastric perforation[20]. Shaw et al. examined gastric distensibility in a canine model in 1965 by insufflating the stomach until gastric rupture occurred and ligating the esophagus and duodenum. Every puncture was found on the larger curvature, and the pathology that followed showed that the rupture site lacked muscle. The scientists deduced from these results that the lack of muscle fibers could be a generic outcome of the perforation itself rather than its clinical cause. All these theories explain the cause and possible pathogenesis of neonatal gastric perforation in neonates.

The male: female ratio of NGP in our study is 2.78:1. The male-to-female ratio of neonatal gastric perforation (NGP) varies across studies, but males are more commonly affected than females in all studies[21]. A study with 13 neonates by Yang CY et al.[22]. reported a male-to-female ratio of 9:4, indicating males constituted the majority of cases. A similar study by Sharafeddin F et al[23]. on 15 patients found a male-to-female ratio of 3:1 in patients admitted with neonatal gastric perforation, which is slightly more than that found in our study. The mean gestational age of neonates of NGP in our study was 35.39 ± 2.19 weeks, and a significant proportion, 59.77% (n=52), of these neonates were preterm at birth. This finding underscores the association between prematurity and the risk of developing NGP. Preterm

neonates are more vulnerable due to immature gastrointestinal systems, decreased perfusion, and an increased susceptibility to stress-related conditions, which may contribute to the development of gastric perforation[24]. The predominance of cases in neonates born via normal vaginal delivery (77.01%) is noteworthy. Vaginal delivery, while generally safe, may contribute to gastric perforation in neonates due to increased stress and intra-abdominal pressure during labor and delivery. This observation aligns with previous reports emphasizing the vulnerability of neonates to such complications during delivery processes, although the reason for this association is not available in the literature.

The high proportion of preterm neonates (59.77%) and low-birth-weight infants (51.72%) among the affected cases further underlines the significant association of NGP with prematurity and restricted fetal growth. Preterm infants often have underdeveloped gastric and intestinal walls, making them more susceptible to perforation under stress conditions, such as hypoxia or mechanical ventilation[25]. Additionally, the data on very low birth weight neonates (1.15%) aligns with literature indicating that extremely low birth weight significantly increases mortality risk in NGP cases[26].

The mean birth weight of 2396.09 ± 386.43 grams provides context for the population studied and aligns with findings that NGP disproportionately affects neonates at the lower spectrum of normal or reduced birth weight[27]. These observations suggest that early identification of at-risk neonates, particularly those born preterm or with low birth weight, is essential for timely intervention and improved outcomes.

Neonatal gastric perforation (NGP) has significant associations with respiratory distress and the interventions used to manage it. In a study of 87 patients, 40 (45.97%) experienced respiratory distress requiring support in the neonatal ICU. This highlights the interplay between respiratory compromise and gastrointestinal complications in neonates. Ventilators with leak compensation and high-pressure settings have been linked to an increased incidence of NGP. The pressure exerted during these interventions can elevate intragastric pressure, particularly in fragile neonates, leading to perforation. Used commonly in neonatal resuscitation, bag-mask ventilation also increases the risk of gastric overdistension and rupture. Positive pressure ventilation,

while critical for resuscitation in neonates with respiratory distress syndrome, can inadvertently cause excessive air entry into the stomach, predisposing the neonate to perforation. [28] Premature infants with underdeveloped respiratory systems and increased susceptibility to respiratory distress syndrome are particularly vulnerable. They often require prolonged ventilatory support, further increasing the risk of complications like NGP[29]. In total, up to 20.6% of neonatal gastric perforation cases exhibit gastrointestinal anomalies[30]. Among these, intestinal malrotation and Meckel's diverticulum are the most common anomalies. Both malrotation and Meckel's diverticulum may remain asymptomatic and can also occur in otherwise healthy individuals. [31]-[32]

The average duration of symptoms before presentation (2.11 ± 0.64 days) suggests a relatively acute onset, aligning with common clinical patterns in abdominal emergencies, where early intervention is critical[33]. Neonatal gastric perforation often presents with abdominal distention, which is the most common sign, reported in up to 88.5% of cases. This symptom aligns with previous findings highlighting abdominal distention's prevalence in neonatal surgical emergencies. Other common presentations include vomiting (71.26%), feeding intolerance (55.17%), and respiratory distress (57.47%), indicating significant gastrointestinal and systemic involvement, which are hallmark signs of perforation[34]. As the condition progresses, more severe signs like cyanosis (64.37%) and absence of bowel sounds (26.44%) are frequently observed, suggesting advanced stages of the disease. These symptoms are often indicative of complications such as peritonitis or shock. Additionally, abdominal erythema (17.24%) and prominent abdominal veins (21.84%) point toward severe inflammation, ischemia, or necrosis. Pneumoperitoneum, which indicates the presence of free air in the abdomen, and lethargy are also key clinical signs of gastric perforation.

In cases of neonatal gastric perforation (NGP), laboratory analysis typically reveals significant abnormalities. The mean hemoglobin level is notably high at 15.84 ± 1.2 g/dL, which may reflect an underlying issue like dehydration or compensatory mechanisms for acidosis. An elevated total leukocyte count, such as 18.71 ± 4.12 x 1000/μL, is indicative of an inflammatory or infectious process and is commonly observed in peritonitis,

particularly following a perforation. Increased leukocyte levels often align with diagnostic markers of peritonitis, where inflammation is typically accompanied by elevated white blood cell (WBC) counts in peritoneal or systemic samples[35]. The platelet count, averaging 194.44 ± 50.91 x1000/µL, is generally within normal limits, though it may be altered depending on associated coagulopathies or other systemic effects.

The mean serum pH is slightly acidotic at 7.26 ± 0.14, suggesting metabolic acidosis, which is often seen in neonates with septic conditions. Additionally, the serum lactate level is elevated (2.57 ± 0.96), which points to tissue hypoxia and anaerobic metabolism, both indicative of significant infection or shock.

Typically, surgery is performed around 5.23 ± 1.14 days after symptom onset, with the procedure lasting an average of 1.82 ± 0.61 hours. The standard surgical approach is exploratory laparotomy, aimed at repairing the perforation and performing lavage of the peritoneal cavity. During these surgeries, the greater curvature of the stomach is the most commonly affected area, followed by the lesser curvature, anterior wall, and posterior wall. These findings align with previous studies which show that perforation sites in the stomach can vary, but certain areas, such as the greater curvature, tend to be more frequently involved[36]. In the majority of cases (82.75%), NGP involves a single perforation, and 65% of these perforations are small, under 5 cm in size. The most common cause of NGP is spontaneous perforation (65%), where no clear etiology is found during surgery. However, traumatic perforations, often resulting from nasogastric tube placement or positive pressure ventilation, account for 21% of cases. In some instances, particularly with premature neonates, necrotizing enterocolitis (NEC) contributes to 10% of NGP cases[37].

In our study, we found 17.2% of patients (n=15) with NGP had associated gastrointestinal anomalies, with intestinal malrotation being the most common associated gastrointestinal anomaly (n=11, 12.64%), followed by duodenal atresia (3.4%) and Meckel's diverticulum (2.3%). These findings echo the existing literature where gastrointestinal anomalies are present in 17.2% of NGP patients, with intestinal malrotation being the most common anomaly. Other gastrointestinal issues like duodenal atresia and Meckel's diverticulum associated with NGP can also

contribute to the development of NGP[38]. We performed gastric repair through gastrorrhaphy, and a venting gastrostomy is often performed as part of the management strategy in all cases. Decompression of the stomach after repair by venting gastrostomy is the key surgical management as it protects the suture line from disruption. In our cases, we did it by using a silicone nipple through which a Foley's catheter (14F) is passed, and a Stamm gastrostomy is done. Venting gastrostomy is a preferred decompression method compared to nasogastric tube disruption as it often gets blocked by a mucous plug besides patient comfort and long-term use[39].

Respiratory complications are prevalent in neonates following surgery, especially among preterm infants with underdeveloped lungs. The necessity for ventilator support in 28 out of 74 cases underscores the severity of respiratory insufficiency in this population. Preterm infants are particularly susceptible due to immature lung development, leading to conditions like respiratory distress syndrome (RDS) and bronchopulmonary dysplasia (BPD)[40]. The occurrence of postoperative sepsis in 54 patients highlights the vulnerability of neonates to infections after surgical interventions. Neonatal sepsis is associated with significant morbidity and mortality, often resulting from factors like immature immune systems and prolonged hospital stays. Wound infections were noted in 25 patients, with 11 developing burst abdomens—a severe complication involving the reopening of a surgical incision. Risk factors for abdominal wound dehiscence in children include young age, wound infection, and emergency surgery[41]. The mortality rate of 42.53% among neonates with NGP is alarmingly high. This is due to the challenges faced on account of prematurity and low birth weight of these infants contributing to increasing mortality. Traditional literature reports a high mortality in these patients despite adequate perinatal care, with the mortality range of 62-70 percent and higher; however, over time, because of improvements in perinatal care, the mortality from NGP dropped to 17% in western countries[42-43]. in their study on 15 patients reported a mortality rate of 47%, slightly more than reported in our study.

In our study, the major clinical predictors of mortality for NGP were male gender (p value = 0.004), preterm delivery of less than 37 weeks (p = 0.002), low birth weight

($p < 0.001$). The male predominance in NGP could be attributed to differences in neonatal immune responses and hormonal influences that may predispose males to higher rates of systemic infections or slower recovery. The possible reasons for this male preponderance may include Immune System Immaturity Klein, S. L., & Flanagan, K. L. (2016) et al. 44, hormonal influence (vom Steeg & Klein, 2016 et al. 45, Fischer et al. 46), delayed lung maturation in male infants[47], Genetic and Epigenetic Factors[48], increased male susceptibility to peritonitis and sepsis[49], sex difference in neonatal stress activity[50] contribute to increased male mortality in neonatal gastric perforation. Besides this, in our study, we also found prematurity and low birth weight have also been considered as the major factors contributing to male mortality in neonatal gastric perforation. A number of reasons are in the literature for this association, one being the necessity of mechanical ventilation in preterm and low birth weight infants leading to increased intragastric pressure and the consequent gastric perforation[51]. Besides this, preterm infants have immature immune systems, increasing their susceptibility to infections, and severe infections with sepsis are significant risk factors for gastrointestinal perforation in extremely low birth weight infants[52].

Among the laboratory predictors of mortality, thrombocytopenia ($p < 0.001$), high CRP levels ($p < 0.001$), acidosis ($p < 0.001$), and high lactate levels ($p < 0.001$) were significant predictors of mortality in neonatal gastric perforation patients. Several laboratory parameters have been identified as significant predictors of mortality in these patients. Thrombocytopenia, defined as a platelet count below 150×10^9/L, has been independently associated with increased mortality in NGP patients[53]. Elevated serum lactate levels, particularly those exceeding 2.5 mmol/L, are also linked to higher mortality rates, indicating tissue hypoperfusion and metabolic distress[54]. Metabolic acidosis, characterized by a blood pH less than 7.3, reflects systemic acid-base imbalance and has been correlated with adverse outcomes in neonates. Additionally, high C-reactive protein (CRP) levels, a marker of systemic inflammation, have been associated with increased mortality in neonatal gastrointestinal perforations[55]. Early identification and management of these laboratory abnormalities are crucial for

improving survival rates in neonates with gastric perforation.

Conclusion

Neonatal gastric perforation presents a substantial risk of mortality, particularly among male, preterm, and low birth weight infants. Early recognition of clinical signs, prompt surgical intervention, and vigilant postoperative care are crucial to improving survival rates. Monitoring laboratory indicators such as platelet counts, CRP levels, pH, and lactate levels can aid in assessing prognosis and guiding treatment strategies.

Ethical Consideration

Approval was obtained from the Ethics Committee of the Kanti Devi Medical College Hospital and Research Center, Mathura, Uttar Pradesh. (Number: KDMCHRC/IEC/2023/08)

Acknowledgment

Not applicable

Funding/Support

Not applicable

Conflict of interests

There is no conflict of interest

References

1. Pulzer F, Bennek J, Robel-Tillig E, Knüpfer M, Vogtmann C. Gastric perforation in a newborn. Lancet. 2004 Feb 28;363(9410):703.
2. Huang Y, Lu Q, Peng N, Wang L, Song Y, Zhong Q, Yuan P. Risk Factors for Mortality in Neonatal Gastric Perforation: A Retrospective Cohort Study. Front Pediatr. 2021 May 13; 9:652139.
3. Iacusso C, Boscarelli A, Fusaro F, Bagolan P, Morini F. Pathogenetic and Prognostic Factors for Neonatal Gastric Perforation: Personal Experience and Systematic Review of the Literature. Front Pediatr. 2018 Apr 4; 6:61.
4. Lone, Y. A., Singh, S. K., Naaz, A., Chetan, C., & Kashyap, S. V. (2024). Tiny Tummies, Big Challenges: A Case Series of Neonatal Gastric Perforations. Cureus, 16(4), e58149.
5. Cardiel-Marmolejo LE, Peña A, Urrutia-Moya L, Crespo-Smith D, Morales-Vivas CA, Camacho-Juárez KV, Roque-Ibanez C. Neonatal gastric perforation: a case report. Revista Médica del Hospital General de México. 2018 Apr 1; 81:36-40.
6. HOUCK, WILLIAM S. Jr. M.D.; GRIFFIN, JOE A. III M.D. Spontaneous Linear Tears of the Stomach in the Newborn Infant. Annals of Surgery 193(6): p 763-768, June 1981.
7. Linkner, Laurence M. M.D.; Benson, Clifford D. M.D., F.A.C.S. Spontaneous Perforation of the Stomach in the Newborn: Analysis of Thirteen Cases. Annals of Surgery 149(4): p 525-533, April 1959.
8. Azirar A, Ech-Chebab M, Ayyad A, et al. (July 14, 2025) Spontaneous Neonatal Gastric Perforation: A Case Report. Cureus 17(7): e87877. DOI 10.7759/cureus.87877
9. Yang, T., Huang, Y., Li, J., Zhong, W., Tan, T., Yu, J., Li, L., Pan, J., Hu, C., Yang, J., & Zou, Y. (2018). Neonatal Gastric Perforation: Case Series and Literature Review. World Journal of Surgery, 42(8), 2668-2673.
10. St-Vil D, LeBouthillier G, Luks FI, Bensoussan AL, Blanchard H, Youssef S. Neonatal gastrointestinal perforations. J Pediatr Surg. 1992 Oct;27(10):1340-2.
11. Rosser SB, Clark CH, Elechi EN. Spontaneous neonatal gastric perforation. J Pediatr Surg. 1982 Aug;17(4):390-4.

12. Duran R, Inan M, Vatansever U, Aladağ N, Acunaş B. Etiology of neonatal gastric perforations: review of 10 years' experience. Pediatr Int. 2007;49(5):626-30.
13. Sakaria RP, Zaveri PG. Neonatal gastric perforation: 14-year experience from a tertiary neonatal intensive care unit. Am J Perinatol. 2021.
14. Aydin M, Deveci U, Taskin E, Bakal U, Kilic M. Percutaneous peritoneal drainage in isolated neonatal gastric perforation. World J Gastroenterol. 2015;21(45):12987-8.
15. Terui K, Iwai J, Yamada S, Takenouchi A, Nakata M, Komatsu S, et al. Etiology of neonatal gastric perforation: a review of 20 years' experience. Pediatr Surg Int. 2012;28(1):9-14.
16. Herbut, P. A. (1943). Congenital defect in the musculature of the stomach with rupture in a newborn infant. Arch Pathol, 36(9), l-94.
17. Cushing, H. (1932). Peptic ulcers and the interbrain. Surg Gynec Obstet, 55, 1-34.
18. Holgersen, Leif O.The etiology of spontaneous gastric perforation of the newborn: A reevaluation Journal of Pediatric Surgery, Volume 16, Issue 4, 608 – 613
19. Vacaru, A., Sharafeddin, F., Maidan, A., Moores, D. C., Raymond, S. L., Mladenov, G. D., & Radulescu, A. (2023). Neonatal gastric perforation: Case report. Journal of Pediatric Surgery Case Reports, 95, 102675.
20. Shaw a, blanc wa, santulli tv, kaiser g. Spontaneous rupture of the stomach in the newborn: a clinical and experimental study. Surgery. 1965 sep; 58:561-71.
21. Kiesewetter wb. Spontaneous rupture of the stomach in the newborn. Ama am j dis child. 1956;91(2):162–167.
22. Yang CY, Lien R, Fu RH, Chu SM, Hsu JF, Lai JY, Minoo P, Chiang MC. Prognostic factors and concomitant anomalies in neonatal gastric perforation. J Pediatr Surg. 2015 Aug;50(8):1278-82.
23. Sharafeddin, F., Edelbach, B.., Vacaru, A. ., Mladenov, G. ., Moores, D. ., Singh, Y. ., & Radulescu, A. (2023). Gastric perforation in neonates: Our experience. Journal of Neonatal Surgery, 13, 2
24. Lee, D. K., Shim, S. Y., Cho, S. J., Park, E. A., & Lee, S. W. (2015). Comparison of gastric and other bowel perforations in preterm infants: A review of 20 years' experience in a single institution. Korean Journal of Pediatrics, 58(8), 288.

25. Rogulska, J., Fenton, T. R., & Szczapa, T. (2024). Association of Neonatal Morbidities and Postnatal Growth Faltering in Preterm Neonates. Healthcare, 13(3), 235. https://doi.org/10.3390/healthcare13030235
26. Manuck TA, Rice MM, Bailit JL, Grobman WA, Reddy UM, Wapner RJ, Thorp JM, Caritis SN, Prasad M, Tita AT, Saade GR, Sorokin Y, Rouse DJ, Blackwell SC, Tolosa JE; Eunice Kennedy Shriver National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. Preterm neonatal morbidity and mortality by gestational age: a contemporary cohort. Am J Obstet Gynecol. 2016 Jul;215(1): 103.e1-103.e14.
27. Tana M, Tirone C, Aurilia C, Lio A, Paladini A, Fattore S, Esposito A, De Tomaso D, Vento G. Respiratory Management of the Preterm Infant: Supporting Evidence-Based Practice at the Bedside. Children (Basel). 2023 Mar 10;10(3):535.
28. Boughaba, N. (2022). Spontaneous Newborn Idiopathic Gastric Perforation. Journal of Pediatric Surgery Case Reports, 78, 102187.
29. Güney, C., & Tunç, G. (2021). Evaluation of Gastrointestinal Perforations in Newborns: A Single Center Experience. Haydarpasa Numune Med J, 61(4), 426-430.
30. Huerta, C., & Perez, E. (2022). Diagnosis and management of neonatal gastric perforation: a narrative review. Digestive Medicine Research, 5. doi:10.21037/dmr-21-105
31. Yang, C., Lien, R., Fu, R., Chu, S., Hsu, J., Lai, J., Minoo, P., & Chiang, M. (2015). Prognostic factors and concomitant anomalies in neonatal gastric perforation. Journal of Pediatric Surgery, 50(8), 1278-1282.
32. Kim, E. (2019). Gastric perforation in a newborn. Journal of Pediatric Surgery Case Reports, 43, 87-89.
33. Mushtaq N, Elwood E, Westwood E, Macdonald A, Saxena AK, Bretherton J. Intestinal malrotation and Meckel's diverticulitis in a 19-month-old boy. BJR Case Rep. 2021 Oct 28;8(1):20210127.
34. Basak A, Dey NS, Dhar S. Successful management of neonatal gastric perforation in Tripura medical college and dr. BRAM teaching hospital-a rare case report. Int J Contemp Pediatr 2023; 10:1749-52.

35. Staniland JR, Ditchburn J, De Dombal FT. Clinical presentation of acute abdomen: study of 600 patients. Br Med J. 1972 Aug 12;3(5823):393-8.
36. Abdelgawad, A. E., Darwish, A. A., Hughes, E., & Cusick, E. (2019). Spontaneous gastric perforation in neonates: a Tertiary Pediatric Surgical Center experience. Journal of Neonatal Surgery, 8(3), 20.
37. Gephart SM, McGrath JM, Effken JA, Halpern MD. Necrotizing enterocolitis risk: state of the science. Adv Neonatal Care. 2012 Apr;12(2):77-87; quiz 88-9.
38. Byun J, Kim HY, Noh SY, Kim SH, Jung SE, Lee SC, Park KW. Neonatal gastric perforation: A single center experience. World J Gastrointest Surg. 2014 Aug 27;6(8):151-5.
39. Blumenstein I, Shastri YM, Stein J. Gastroenteric tube feeding: techniques, problems and solutions. World J Gastroenterol. 2014 Jul 14;20(26):8505-24.
40. Chen, Tsung-Yen MDa; Liu, Hsien-Kuan MDa; Yang, Ming-Chun MDa,b; Yang, Yung-Ning MDa; Ko, Po-Jui MDc; Su, Yu-Tsun MDa; Huang, Ru-Yi MDd; Tsai, Ching-Chung MD, PhDa,b,*. Neonatal gastric perforation: a report of two cases and a systematic review. Medicine 97(17): p e0369, April 2018.
41. Leone RJ Jr, Krasna IH. 'Spontaneous' neonatal gastric perforation: is it really spontaneous? J Pediatr Surg. 2000 Jul;35(7):1066-9.
42. Babayigit A, Ozaydın S, Cetinkaya M, et al. Neonatal gastric perforations in very low birth weight infants: a single center experience and review of the literature. Pediatr Surg Int 2018; 34:79-84.
43. Lin, C., Lee, H., Kao, H., Hung, H., Hsu, C., Yeung, C., Sheu, J., & Wang, N. (2008). Neonatal Gastric Perforation: Report of 15 Cases and Review of the Literature. Pediatrics & Neonatology, 49(3), 65-70.
44. Klein, S. L., & Flanagan, K. L. (2016). Sex differences in immune responses. Nature Reviews Immunology, 16(10), 626-638.
45. vom Steeg LG, Klein SL (2016) SeXX Matters in Infectious Disease Pathogenesis. PLoS Pathog 12(2): e1005374.
46. Fischer, J., et al. (2015). "Hormonal influences on neonatal immunity: Estrogen and testosterone effects." Journal of Neonatal Medicine.

47. McCoy, D. M., Salome, R. G., Kusner, D. J., Iyar, S. S., & Mallampalli, R. K. (1999). Identification of Sex-Specific Differences in Surfactant Synthesis in Rat Lung. Pediatric Research, 46(6), 722.
48. Ober, C., Loisel, D. A., & Gilad, Y. (2008). Sex-specific genetic architecture of human disease. Nature Reviews Genetics, 9(12), 911-922.
49. Davis, M., & Emory, E. (1995). Sex Differences in Neonatal Stress Reactivity. Child Development, 66(1), 14-27.
50. Schröder J, Kahlke V, Staubach K, Zabel P, Stüber F. Gender Differences in Human Sepsis. Arch Surg. 1998;133(11):1200–1205. doi:10.1001/archsurg.133.11.1200
51. Huerta CT, Perez EA. Diagnosis and management of neonatal gastric perforation: a narrative review. Dig Med Res 2022; 5:27.
52. Feng, Wei et al. Gastrointestinal perforation in extremely low birth weight infants: A single center retrospective study in China Pediatrics & Neonatology, Volume 65, Issue 2, 111 – 116
53. C Ree, I. M., Fustolo-Gunnink, S. F., Bekker, V., Fijnvandraat, K. J., Steggerda, S. J., & Lopriore, E. (2017). Thrombocytopenia in neonatal sepsis: Incidence, severity and risk factors. PLoS ONE, 12(10), e0185581.
54. Matsushita FY, Krebs VLJ, De Carvalho WB. Association between Serum Lactate and Morbidity and Mortality in Neonates: A Systematic Review and Meta-Analysis. Children. 2023; 10(11):1796.
55. Peng Yuan, Yao Huang, Yi Wang et al. Prognostic Factors in Preterm Infants with Neonatal Gastrointestinal Perforation: A Retrospective Cohort Study, 15 March 2022, PREPRINT (Version 1) available at Research Square.