Aim: Neonatal gastric perforation (NGP) is a rare, perplexing, life-threatening entity affecting neonates. We share our experience of operating upon cases of NGPs and highlight important points observed which may aid in further improving care of neonates, diagnosed with this entity.

Materials and Methods: A retrospective analysis of all consecutive patients with NGP operated by the author, at various centers between January 2015 and December 2018, was carried out. We analyzed different variables for these and reached logical conclusions.

Results: Between January 2015 and December 2018, we treated ten patients with gastric perforation. All the neonates were preterm, except one. Mean birth weight in our series was 1745 g (range 1300–2400 g). Deterioration in activity, worsening of sepsis, metabolic acidosis, increased ventilator requirements, and abdominal distension were prominent clinical features identified in all patients. All patients subsequently had massive pneumoperitoneum before surgery. Six patients had perforation along the greater curvature, two had perforation at the posterior wall, and two had near total gastric necrosis. We had four mortalities out of ten patients operated.

Conclusion: NGP is associated with high mortality, especially in premature and low birth weight neonates. Severity of contributing factors in a premature predisposed neonate determines the severity of gastric necrosis, which in turn is an important prognostic factor. Certain preoperative signs can be useful and can aid in initiating preventive measures to curtail severity of the pathology.

Keywords: Gastric necrosis, gastorrhaphy, neonatal gastric perforation

INTRODUCTION

Neonatal gastric perforation (NGP) is a unique entity with predisposition in premature neonates resulting in perforation of a part of the gastrointestinal tract with richest blood supply. Gastric perforations are also unique as they are less frequent, more life-threatening with higher mortality rates in preterms, especially in very low birth weight (LBW) infants. Multiple causes have been described previously in literature, of which prematurity-related predispositions worsened by multiple contributing factors (such as hypoxia, sepsis, external trauma, and hypotension) seem to be the most pertinent. Increased intragastric pressure due to various causes is the most important variable. Usual site of perforation is at the greater curvature. However, in all of the studies, mortality was unrelated to the site of perforation. Very few studies mentioned the size of perforation and none the extent to which gastric necrosis occurred. In most of the studies, the

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commonly performed procedure was gatstrorrhaphy with or without gastrostomy with very few cases requiring gastrectomies.\cite{1-6}

We share our experience of operating upon cases of NGPs and highlight important points observed, which may aid in further improving care of neonates diagnosed with this entity.

**MATERIALS AND METHODS**

A retrospective analysis of all consecutive patients with NGP operated by the author at various centers, from January 2015 to December 2018, was carried out. Variables considered included: gestational age at birth, BW, mode of delivery, prenatal maternal or fetal complications/ treatments, clinical presentations, age at presentation, possible contributing factors, site of perforation, intraoperative findings, associated sepsis, laboratory studies, and outcome in terms of mortality. We also calculated the percentage of gastric necrosis and mortality based on same.

**RESULTS**

Between January 2015 and December 2018, we treated ten patients with gastric perforation [Table 1]. All the neonates were preterm (range 32–42 weeks, mean = 34.7 weeks), except one (Case 7). All were delivered through lower segment caesarian section except one (Case 7). Mean birth weight in our series was 1745 g (range 1300–2400 g). Mean age at presentation with perforation was 5.9 days (range = 2–12 days). Four patients (Cases 5, 6, 8, and 9) were referred to us from peripheral hospitals after perforation was diagnosed, while six (Cases 1–4, 7, 10) developed gastric perforation in the course of their admission. All had nasogastric tube/orogastric tube insertion done preperforation. Five of our patients\cite{1,3,5,7,8} were on continuous positive airway pressure (CPAP) support due to various respiratory concerns, while four were ventilated (Cases 2, 5, 6, and 9). All had septic profile positive and were on higher antibiotics preoperatively. Three out of ten were started on feeds which had to be stopped due to nontolerance to feeds.

Deterioration in activity, worsening of sepsis, metabolic acidosis, increased ventilator requirements, and abdominal distension were prominent clinical features identified in all patients. Initial Abdominal X-rays showed gastric pneumatisos in two patients (Cases 4, and 7), gastric distension despite nasogastric decompression in four patients (Cases 1, 2, 3, and 10), and massive pneumoperitoneum in four (Cases 5, 6, 8, and 9) referred from outside. All patients subsequently had massive pneumoperitoneum before surgery [Figure 1].

Six patients had perforation along the greater curvature (Cases 1, 2, 5, 6, 9, and 10), two had perforation at the posterior wall (Cases 3 and 8) and two had near total gastric necrosis (Cases 4 and 7). During surgery, two patients with gastric pneumatisos had massive necrosis of stomach and required near total gastrectomy, with only a sliver of tissue remaining along the lesser curvature (Cases 4 and 7) [Figure 2]. We used small bowel for repair in one (Case 7) and did a feeding jejunostomy and esophagostomy in other (Case 4). In four patients (Cases 1, 2, 5, and 6), partial gastrectomy was done as nearly 50% of gastric tissue had to be sacrificed, before viable reparable gastric tissue could be identified [Figure 2]. We repaired three primarily (Cases 1, 5, and 6) and used jejunal loop interposition in one(Case2),with poor gastric viability despite adequate debridement.. In two patients (Cases 3 and 8), the perforation was in the posterior wall of stomach around 1 cm size, which was primarily repaired. In two patients (Cases 9, 10), perforation was at the greater curvature (1, 1.2 cm size), which was again primarily repaired. We also observed that in most patients the omentum was unhealthy and singed and very small amount of viable omentum was found in cases with large perforations. We used gastrostomy in four patients (Cases 3, 8, 9, and 10) where good amount of viable gastric epithelium was available and good capacity stomach was reconstructed.

We had four mortalities (Cases 4, 5, 6, and 7) out of ten patients operated. These patients were two were with total gastric necrosis and two with nearly 50% gastric tissue necrosed. One patient with 50% necrosis of gastric tissue with jejunal interposition survived and one with primary repair survived. All others with smaller greater curvature perforations (Cases 9 and 10) and perforation at posterior wall of stomach (Cases 3 and 8) survived.

**DISCUSSION**

There are various reasons and predisposing factors described for NGP.\cite{1-6} Prematurity and low BW are
| BW (g) | Gestational age (weeks) | Mode of delivery | Age at perforation (days) | Associated problems | CPAP | Ventilation | Sepsis | X-ray | Intraoperative findings | Surgery performed | Survival |
|--------|-------------------------|------------------|---------------------------|---------------------|------|-------------|--------|-------|-------------------------|------------------|----------|
| 1900   | 32                      | LSCS             | 3                         | HMD                 | Yes  | No          | Yes    | Yes   | Gastric distension with NG tube in situ | Greater curvature 50% necrosis | Primary repair without gastrostomy | Survived |
| 2100   | 36                      | LSCS             | 8                         | EONS, HMD           | No   | Yes         | Yes    | Yes   | Gastric distension with NG tube in situ | Greater curvature 50% necrosis | Jejunal loop interposition | Survived |
| 1500   | 34                      | LSCS             | 5                         | PDA, Sepsis         | Yes  | No          | Yes    | Yes   | Gastric distension with NG tube in situ | Posterior wall | Primary repair with gastrostomy | Survived |
| 1300   | 32                      | LSCS             | 2                         | Sepsis, HMD, PDA   | Yes  | No          | Yes    | Yes   | Gastric pneumatosis | Massive total gastric necrosis | Feeding jejunostomy, esophagostomy | Death   |
| 1450   | 32                      | LSCS             | 4                         | HMD                 | No   | Yes         | Yes    | Yes   | Massive gas under diaphragm | Greater curvature 50% necrosis | Primary repair without gastrostomy | Death   |
| 1600   | 34                      | LSCS             | 7                         | MAS                 | No   | Yes         | Yes    | Yes   | Massive gas under diaphragm | Greater curvature 50% necrosis | Primary repair without gastrostomy | Death   |
| 2400   | 42                      | Normal           | 12                        | Delayed cry         | Yes  | No          | Yes    | Yes   | Gastric pneumatosis | Greater curvature 50% necrosis | Jejunal loop interposition | Death   |
| 2100   | 36                      | LSCS             | 4                         | TTN                 | Yes  | No          | Yes    | Yes   | Massive gas under diaphragm | Greater curvature, 1.2 cm size | Primary repair with gastrostomy | Survived |
| 1600   | 35                      | LSCS             | 8                         | MAS                 | No   | Yes         | Yes    | Yes   | Massive gas under diaphragm | Greater curvature, 1.0 cm size | Primary repair with gastrostomy | Survived |
| 1500   | 34                      | LSCS             | 6                         | TTN                 | No   | No          | Yes    | Yes   | Gastric Distension with NG tube in situ | Greater curvature, 1.0 cm size | Primary repair with gastrostomy | Survived |

HMD: Hyaline membrane disease, TTN: Transient tachypnea of newborn, MAS: Meconium aspiration syndrome, PDA: Patent ductus arteriosus, CPAP: Continuous positive airway pressure, NG: Neonatal gastric, EONS: Early onset neonatal sepsis, LSCS: Lower segment cesarian section, BW: Birth weight
among the most frequently and consistently associated predisposing factors.[1-6] The prematurity leads to decreased maturity of gastric tissue documented by deficiency in interstitial cells of Cajal which leads to impaired motility and hence an increased intragastric pressure.[1,2] Premature neonates circulatory regulation too is not fully established which further increases chances of ischemia in premature neonates.[1-6] Prematurity further exposes the neonate to sepsis with fungal and bacterial infections related ischemia similar to necrotizing enterocolitis (NEC) also suggested as another reason.[1-6] The premature neonatal gastric milieu also is susceptible to the use of various medications like nonsteroidal anti-inflammatory drugs.[1-6] Also in a premature neonate, incoordinated and immature esophagogastric motility, typical of premature neonates, is suggested as a possible mechanism causing increased intragastric pressure, thereby predisposing to gastric rupture.[2,4,5] Another significant variable encountered in most studies in cases of NGP was hypoxia and sepsis.[1-6]

An increase in intragastric pressure is usually considered as a prerequisite for NGP to occur. Raised intra-gastric pressure can be primary related to prematurity or secondary related to associated anatomical factors such as malrotation and duodenal web. Further contributing factors such as hypoxia, sepsis, intravenous drugs, nasogastric tube placement, inadvertent traumas, excessive feeding, and nasal CPAPs need to be looked for too.[1-6]

An experiment by Shaw et al.[7] and later by Holgersen[8] showed that only raised intragastric pressure usually resulted in a perforation along the greater curvature. Chen et al.[1] found in their review that highest incidence of perforation was at the greater curvature, followed by lesser curvature, posterior wall, and anterior wall. Chen et al.[1] found no statistical significance between the site of perforation and the mortality. All the series and review articles mention the site of perforation, with only two mentions the size of perforation and none mentioned the percentage of gastric necrosis.[1-6] We found that the ischemia and perforation in NGP are too widespread to mention just the site of perforation and more important is the extent of involvement of the necrotic process. The mortality as shown in our series is more related to the extent of involvement. Only Yang et al.[3] and Babayigit et al.[4] had mentioned the size of perforation. Similar to our results, Babayigit et al.[4] found mortality related to the size of perforation and mentioned that perforation >1.5 cm was associated with poor outcome. However, we propose that percentage of gastric necrosis is more important than the size of perforation. Babayigit et al.[4] also found that those with posterior wall perforation had improved survival.

Most of the authors have mentioned primary repair as the most frequently done procedure followed by partial gastrectomy and anastomosis.[1-6] We saw in many of our cases that after freshening the margins very less viable stomach was available. Furthermore, in many cases, the bleeding coming from freshened margins was still very dark and further freshening of margins resulted in a very small amount of available tissue to be repaired. We feel that the pathological process resulting in NGP, similar to NEC is a continuous one and hence the prognosis depends on other contributing factors which primarily resulted in the perforation.[1,4] We also found that the omental vessels along the greater curvature which are responsible for the robust blood supply of stomach were seinged probably because of the harsh acid content (which is more in premature infants) and phlogistic response. Thus, we propose that the amount of gastric necrosis and condition of the available repairable viable stomach also dictates survival. In few of our cases, we interposed small bowel with better viability in our repairs and found better response than primary repairs. There have been only one report describing colonic interposition in a case of near total gastrectomy after NGP.[9]

Clinical presentation usually is in the form of abdominal distention with sepsis and respiratory failure.[1-6] In all cases, in our series, preoperative X-rays showed massive gastric distension despite nasogastric decompression. Altered aspirates were found in few patients too. In few patients, this was followed by pneumatosis restricted to the stomach and later gastric perforation thereafter. Thus, the above finding is found consistent with all cases of gastric perforation and can be used for prognostication and measures to decrease intragastric pressures can be vigorously started.[10] Furthermore, control of
contributing factors at this stage can be preventive in some cases (such as decreasing positive end-expiratory pressure of nasal CPAP or ventilator, usage of wider bore feeding tube for drainage, increasing inotropes to improve circulation, stepping up on antibiotics). An early expectation based on above sign can ascertain early intervention and can also help in prognostication.

We felt that the severity of contributing factors such as sepsis, hypoxia, and hypotension which led to the NGP was reflected in the intraoperative picture and the amount of gastric necrosis encountered. This is reflected in the survival rates in our study and in previous studies where neonates with smaller perforations and lesser necrosis had better survival than others.[3,4]

**CONCLUSION**

NGP is a unique entity with predisposition in premature neonates resulting in perforation of a part of the gastrointestinal tract with richest blood supply. We felt that survival depends on early diagnosis and intervention. Sudden abdominal distension in a preterm LBW infant in first 7 days and an abdominal X-ray showing persistent gastric distension despite adequate drainage can be taken as preoperative danger signs for NGPs. We also felt that success of surgery depends on the contributing factor for gastric perforation and the amount of viable stomach available. We advocate that in cases of massive necrosis remaining gastric mucosa which is routinely primarily repaired is still not well vascularized and interposition of small bowel or colonic mucosa with a more dependable blood supply would improve surgical success and improve prognosis.

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**Conflicts of interest**
There are no conflicts of interest.

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