Original Research Article

Neonatal gastric perforation: is prematurity the only cause?

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ABSTRACT

Background: Neonatal Gastric Perforation (NGP) is a serious and life-threatening emergency and challenging in terms of diagnosis and management. The precise aetiology remains obscure in most cases. Published literatures provide theories of gastric ischemia, aeroaphagia & trauma with or without definite inciting pathology. Owing to its high mortality rate, it requires early detection & urgent intervention to bring out the newborn from this catastrophe.

Methods: We report five cases of neonatal gastric perforation over period of 2013-2019 admitted to a tertiary paediatric surgical care hospital managed by early detection, prompt resuscitation along with exploration and primary repair of the defect in gastric wall.

Results: The common feature in these five cases were preterm age, low birth weight, hypoxic event and marked abdominal distension causing respiratory distress. Features of imminent or complete septic shock were present in all babies. Four babies out of five survived except in one case of jejunoileal Artesia that developed gastric perforation in post operative period.

Conclusions: Neonatal gastric perforation should be suspected in cases of rapidly deteriorating premature newborn with gross abdominal distension and pneumoperitoneum. In our opinion, etiology of NGP is multifactorial. Prematurity, low APGAR score with vigorous resuscitation could be a clue for early diagnosis. Good outcome in our series was due to early diagnosis and prompt pre and postoperative measures. Additionally, sterile gastric content in peritoneum might lead to survival from this catastrophic event.

Keywords: Asphyxia, Gastric perforation, Gastrointestinal anomalies, Neonate, Pneumoperitoneum, Resuscitation complication

INTRODUCTION

Neonatal hollow viscous perforations have wide spectrum of causes. Incidence of Neonatal Gastric Perforation (NGP) is approximately 7% of them.1 This process evolves so rapidly that accurate diagnosis is necessary to improve the survival rate as it has been reported having poor outcome and a high mortality rate. Premature babies with poor APGAR score are more vulnerable. Categorically, it may be a Primary (spontaneous) gastric perforation or a secondary gastric perforation. Searched literature could be analyzed only by retrospective isolated case reports and case series.

This case series is a single centre retrospective study of NGP to find out the probable aetiology behind this catastrophe and to outline successful management plan. To our luck, four cases out of five survived with uneventful long term follow-up.

METHODS

Retrospective data were collected from hospital records of a tertiary level care centre for the period of 2013-2019. All cases which were finally diagnosed as gastric perforation and being operated in emergency wing were sorted out. Total five newborns who presented with acute...
onset of abdominal distension and bloody gastric aspirate with features of shock were managed. Four of 5 cases were preterm and all of them presented to us between 3 to 6 days of life. Majority had a history of birth asphyxia and poor APGAR score (mean <6) and were attempted bag & mask ventilation for resuscitation (Table 1).

**Table 1: Clinical profile of cases with relevant history.**

| Antenatal history | Days of life | Other GI anomaly | Birth weight in gr | *Mode of delivery | APGAR score @ 1min. | Sex | BAG and mask ventilation | Abdominal distension | Hemorrhagic aspirate | Hospital stay | Recovery |
|-------------------|--------------|------------------|-------------------|-------------------|---------------------|-----|-------------------------|---------------------|---------------------|------------|---------|
| Absent            | 3            | N                | 2100              | NVD               | 6                   | Female | Y                       | Y                   | y                   | 11         | Y       |
| Absent            | 4            | N                | 1850              | NVD               | 5                   | Male   | Y                       | Y                   | Y                   | 16         | Y       |
| Absent            | 7            | Jejunoileal atresia | 2650            | LSCS              | 10                  | Male   | N                       | Y                   | Y                   | -          | N       |
| Absent            | 2            | N                | 1600              | LSCS              | 5                   | Male   | N                       | Y                   | y                   | 18         | Y       |
| Absent            | 6            | N                | 1700              | NVD               | 7                   | Female | Y                       | Y                   | y                   | 14         | Y       |

Mean Birth weight of neonates were 1980±280 grams. Lethargy, progressive abdominal distension, poor cry with or without bloody gastric aspirate were criteria for referral to us. Adequate resuscitation with inotrope agents, when required was administered. Arterial blood gas revealed metabolic acidosis in all cases of pH < 7.30 demanding generous fluid resuscitation and inotrope support. Preoperative X-ray abdomen in supine / lateral view was done showing free gas in abdomen which warranted exploration (Figure 1).

**Figure 1: X-ray abdomen of two cases showing pneumperitoneum with paucity of gas in small bowel.**

After taking complete obstetrics history and written informed consent from parents, we proceeded for exploration under general anaesthesia with strict vitals monitoring. On table there was sero-purulent peritoneal fluid with fibrinous flakes, which was sucked out. As we were expecting hollow viscous perforation of large or small intestine, whole length of bowel was inspected and emptied without any definitive pathology. On inspecting upper abdomen, anterior gastric wall was seen stuck with yellowish exudates in two of the cases with unhealthy margin of perforation; another two cases had gastric perforation with bleeding margin. All perforation was at greater curvature with size of 1 to 2 cm. We could correlate their time of presentation to us by gastric perforation character. Author preferred to close the perforation by trimming margins and closing in two layers using 5-0 Polyglactin (910) suture with proper placement of 8 French feeding tube. Thorough peritoneal lavage was done in all cases with warm normal saline ensuring no flakes were left inside. Intraperitoneal drain was given with closure of abdomen done in layers. Post operative period was stormy which required continuous monitoring and intensive care including ventilator support in all cases. Strict gastric aspiration at frequent intervals were done via nasogastric tube. Weaning from ventilator support and inotrope was done as per NICU team protocol. Feed was started on 5th to 8th post operative day and drain was removed on day 6th to 9th post operatively. We encountered superficial wound infection in all cases which was managed by drainage and dressings. All neonates got discharged by 11th to 18th day postoperatively with follow-up advice after seven days.

**Figure 2: Follow up after seven days for stitch removal in two post-operative cases.**

Follow-up was uneventful and all four babies were doing well (Figure 2). Preterm screening as per department of paediatrics was also followed.
Neonatal gastric perforation was first described by Siebold in 1825. The reported incidence is 1:5,000 live births. Majority of published case series pointed out gastric perforations were due to iatrogenic trauma followed by ischaemic or spontaneous mode of insult to stomach mural layers. If other additional factors leading to luminal compromise such as malrotation, oesophageal atresia with distal fistula, amniotic gastritis or left diaphragmatic hernia, term ‘secondary gastric perforation’ could be used. Perforation was usually along the greater curvature that might be related to anatomical dependent greater curvature and appeared as a puncture wound or a short linear laceration. In all of our cases, sites along greater curvature were affected locations. In 1964, an article explained lesser curvature perforation was more common, if inciting factor was solely over-distension. In contrast, use of orogastric tube and related trauma due to it might cause perforation on lesser curvature. Traumatic gastric perforation might develop during the course of positive pressure ventilation following rigorous resuscitation attempt. More commonly, preterm had greater chance of asphyxia and weaker gastric musculature making them prone for transmural necrosis and stress induced ulcers. Similar observation was found in our cases. Spontaneous NGP had been reported in otherwise term neonate in one of our case which could be due to inadvertent oesophageal ventilation or over enthusiastic resuscitation. One published theory was spontaneous perforations were due to the congenital defects in the muscular wall of the stomach. As discussed, this case was a term neonate weighing 2650grams operated initially for atresia of bowel suggesting association of this defect with atresia of foregut (esophageal atresia, pyloric atresia, duodenal atresia) or midgut atresia. Gastroduodenal perforation has been associated with postnatal steroid therapy but there was no history of steroid or NSAID for ductal dependency in any of our cases. X-ray abdomen finding of gastric perforation was pneumoperitoneum in all of our cases with typical single rounded gas shadow with paucity of gas in small bowel. Because of the large size and proximal nature of perforation, these babies may had developed pneumoperitoneum in acute manner leading to deteriorating cardiopulmonary status. Early surgical intervention with repair of most perforation by debridement and two layer closure of stomach was fruitful. Thorough lavage of peritoneal spaces and drainage, with insertion of a feeding tube for free drainage and decompression of the stomach as protection for the gastric lining was mandated. In our case series, sepsis was the only variable significantly associated with morbidity. Therefore, in a neonate with suspected NGP, signs of hypovolemic or septic shock must be actively checked, as timely treatment of this complication appeared to be the single most important factor.
influencing survival.13 In very sick VLBW babies, bedside peritoneal drainage might be only intervention required followed by surgical exploration once the baby’s condition stabilized. Persistence of pneumoperitoneum on X-ray, deranged ABG analysis and features of peritonitis should mandate surgical exploration.14 Improvement of neonatal intensive protocol in postoperative period had drastically reduced mortality by ten folds in NGP cases. Even in our case series, dedicated post operative care had lead to better results.13 Although we have 80% survival in our series, but a larger sample size is warranted for clarity of clinical course and survival.

CONCLUSION

Neonatal gastric perforation (NGP) is a dreaded condition and may progress rapidly to a point of no return and should be addressed with vigorous supportive therapy coupled with the use of broad spectrum intravenous antibiotics and early diagnosis. A rapidly deteriorating preterm with history of poor APGAR score, a trial of overenthusiastic resuscitation, with evidence of pneumoperitoneum is a clue to diagnose NGP. Although prematurity is common among the cases of gastric perforation, the most important factors affecting survival appear to be the interval between the onset of symptoms and prompt diagnosis, extent of peritoneal contamination to urgent surgical exploration. Even term babies are not spared from wrath of gastric perforation or its complications. Therefore, authors opine that there could be multi-factorial etiology for neonatal gastric perforation. Finally a dedicated postoperative intensive care is pre requisite for better survival rate.

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