Neonatal Gastric Perforation: A case series

Aishwarya Venkataraman, Anastasia Vareli, Devesh Misra, Bhupinder Reel, Kalaimaran Sadasivam

ABSTRACT

Introduction: Gastric perforation in neonates is a rare surgical emergency of uncertain etiology. Case series: We report a case series of three neonates diagnosed with gastric perforations in the first week of life and successfully managed by urgent surgical intervention. All three patients had associated gastrointestinal anomalies contributing to the gastric perforation. Conclusion: Neonatal gastric perforation is extremely rare and is associated with high mortality. Early diagnosis and prompt surgical intervention are essential to improve the outcomes.
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Keywords: Gastric perforation, Pneumoperitoneum, Neonate, Gastrointestinal anomalies

INTRODUCTION

Neonatal gastric perforation is a rare and catastrophic condition with high mortality. Various factors and theories have been proposed as a possible cause [1–7] but the etiology still remains obscure. Gastric perforations are often large and associated with necrosis of a significant portion of the stomach wall. Early identification and treatment is essential and may improve the outcome. It is, therefore, imperative to highlight the need for increased awareness of NGP so as to optimize outcome through urgent surgical intervention in a timely manner. We hereby describe three cases of NGP, secondary to increased gastric pressure due to distal obstruction, managed successfully with prompt surgical intervention.

CASE SERIES

Case 1

A two-day-old term male neonate, born by elective c-section for breech, presented with a history of vomiting, abdominal distension and grunting. Antenatal period and scans were uneventful apart from polyhydramnios noted at 37 weeks gestation. Feeding was commenced immediately after birth and was well tolerated until 40 hr of life when he developed abdominal distension, vomiting, grunting and remained unsettled. Abdominal X-ray revealed free air in the abdomen (Figure 1). On exploration, undigested milk was found all throughout the peritoneal cavity. A gastric perforation of 5 cm was seen on the anterior wall along the greater curvature higher up. This area was excised coming up to 2 cm of the gastroesophageal junction. No other obvious
gastrointestinal abnormality was seen. Primary repair was done in three layers and abdomen was closed. Histopathology revealed extensive hemorrhagic necrosis of the mucosa and submucosa associated with severe congestion. A contrast study performed postoperatively to evaluate the gastrointestinal tract revealed narrowing at pylorus suggesting pyloric atresia. Hence, a second laparotomy with pyloroplasty was performed to excise the pyloric membrane. Enteral feeding was commenced seven days after the 2nd surgery. He was discharged home after four weeks with anti-reflux medications and full enteral feeds.

Case 2

Twin female neonates (twin 1 and twin 2) were born by elective c-section at 36+3 weeks in good condition. The antenatal period was uneventful and they were monochorionic diamniotic (MCDA) twins. Both of them were discharged at 36 hr of age after enteral feeding was well established. However, they presented on day-4 of life with vomiting, lethargy, distended abdomen and metabolic acidosis. Abdominal X-ray of both babies revealed free air in the abdomen and Abdominal X-ray of twin 2 showed classical 'football' signs of perforation (Figure 2). On exploratory laparotomy of twin 1, a large volume of a grossly contaminated peritoneal fluid containing undigested foul smelling milk was found. A 5-mm gastric perforation on the greater curvature near the esophagogastric junction and a malrotated bowel with severe narrowing of the root of mesentery was noted. Ladd’s procedure and appendectomy were performed. Primary repair was done in three layers and abdomen was closed. Laparotomy findings of twin 2 were similar to twin 1 but she had two gastric perforations on the greater curvature along with severe malrotation. Ladd’s procedure along with appendectomy was performed. Primary repair was done in three layers and abdomen was closed. There was no associated volvulus in both the babies and the postoperative period was uneventful. Histology of debrided tissues showed mucosal necrosis and hemorrhage. They received TPN for three weeks and triple antibiotics (metronidazole, amikacin and co-amoxiclav) for five days. Enteral feed was started 14 days after the surgery and slowly increased over one week.

DISCUSSION

Neonatal gastric perforation is extremely rare and associated with poor prognosis [1, 2, 4]. Various theories have been proposed describing the etiology and prognosis but it still remains obscure [1–3, 5]. Congenital absence of gastric musculature [6, 8], high gastric acid production [9], abdominal trauma [7, 10], and other associated gastrointestinal conditions like ischemic bowel, necrotising enterocolitis (NEC), intestinal malrotation, duodenal web, hiatus hernia, Meckel’s diverticulum, and gastroschisis have all been proposed as possible causes of NGP [3, 11–14]. Few authors have reported that gastric perforation was seen in the setting of a distal mechanical obstruction [3, 5, 15]. Shaw et al. [15] through their experiments suggested that gastric perforation was caused by a mechanical rupture of the stomach secondary...
to increased gastric pressure. In our series, the presence of pyloric web in one neonate and intestinal malrotation in the twins further supports this hypothesis. All three neonates in this study, as well as those previously reported [1, 3, 16] initially tolerated the feeds well and presented with gastric perforation after few days supporting the theory that raised intragastric pressure may contribute to perforation. Irrespective of the cause, neonatal gastric perforation most commonly occurs in the first week of life [3, 17–20] consistent to that observed in our series. Although predominantly seen in preterm and low birth weight newborns [2, 4, 21], neonatal gastric perforation can occur in healthy term infants [1–3] as seen in our series.

Early diagnosis of neonatal gastric perforation is often difficult due to the fact that the presentation and symptoms are non-specific and can mimic sepsis, respiratory distress, poor feeding, NEC, intestinal obstruction, and pneumoperitoneum without gastrointestinal perforation [3, 22]. The majority of neonates are normal at birth, feeding and passing stools normally until rupture occurs when the baby deteriorates rapidly [15, 22]. Abdominal distension can be striking and infants may also develop rapidly progressive pneumoperitoneum with associated cardiopulmonary compromise. Most of the infants are critically unwell on presentation needing intensive care support both pre and post operatively.

Neonatal gastric perforation is a serious and life-threatening condition, hence prompt and urgent surgical exploration is crucial. The time between symptoms and surgery is also a prognostic factor for survival [17]. Broad-spectrum antibiotics are essential to prevent mortality due to peritonitis and sepsis. Parenteral nutrition during the initial postoperative period is usually required. Despite the availability of the advanced neonatal intensive care facilities and parenteral nutrition, the mortality rate remains high (30–83%) [3, 16, 22–24]. Studies have reported that male sex [1, 16], hyponatremia (serum sodium < 130 mEq/L)[16], metabolic acidosis (pH < 7.3) [16], persistent leucopenia and thrombocytopenia [3], prematurity and low birth weight [1, 2, 4] are associated with poor prognosis. Fortunately, there was no mortality in our study. This could be attributed to early diagnosis and prompt surgical intervention.

CONCLUSION

In summary, neonatal gastric perforation is extremely rare and is associated with high mortality. Early diagnosis and prompt surgical intervention are essential to improve the outcome. Although the etiology of neonatal gastric perforation remains unclear, distal mechanical obstruction leading to increased gastric pressure appears to be a contributing factor as seen in our study.

REFERENCES

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