



NEONATAL GASTRIC PERFORATION: A REPORT OF 3 CASES AND LITERATURE REVIEW

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ABSTRACT

Neonatal gastric perforation (NGP) is an uncommon disease. Historical reports regard the aetiology as spontaneous while the mortality remains high. We present the report of the cases of neonatal gastric perforations who presented to our facility, its diverse aetiology and a review of the literature. Their clinical data including age, sex, weight, maternal complications, aetiology, location of perforation and the operation performed are summarized. NGP was complicated by anaemia, leukocytosis and thrombocytopenia in all cases. The overall mortality rate was 66.7% and this was attributed to late presentation. We recommend early recognition and prompt surgical intervention as a way to salvage the condition. Also, passing a nasogastric tube and ensuring free drainage of gastric air and fluid should form part of neonatal resuscitation procedures as a way to prevent gastric over distension and subsequent perforation.

INTRODUCTION

Neonatal gastric perforation (NGP) is an uncommon disease. When it does occur it could be fatal. It could happen spontaneously but other causes should also be sought for. Most gastric perforations are diagnosed out of serendipity. Thus, a high index of suspicion is necessary to make the diagnosis. More often, the clinical diagnosis is perforated necrotizing enterocolitis (NEC). Prompt resuscitation and early surgical intervention is key to the survival

of these neonates. Despite its rarity and paucity of reports in our environment, we present the report of 3 cases managed in our facility all of whom presented within 3 months in 2015.

CASE REPORTS

The case histories of 3 patients who presented to the hospital between August and October 2015 with neonatal gastric perforation were reviewed. Two of the patients were referred to the paediatric surgical unit from the paediatric neonatal unit on account of x-ray features of

pneumoperitonium while the other was previously managed as a case of tracheoesophageal fistula. On admission, they were resuscitated with intravenous infusions to correct hypovolemia and electrolyte imbalance, nasogastric tube was passed and all of them had broad-spectrum antibiotics. Whole blood transfusions were given to correct anaemia and coagulopathies. Gastric perforation was diagnosed at operation, with the preoperative diagnosis being perforated NEC in two of the cases and tracheoesophageal fistula in the other.

Parameters	Case 1	Case 2	Case 3
Age at presentation	10days	6days	13days
Sex	Male	female	Female
Gestational age at birth	Term	term	Term
Birth weight	2.3kg	3.8kg	2.2kg
Mode of delivery	Vaginal delivery	Caesarian delivery	Vaginal delivery
Maternal complication	Nil	Retroviral disease	Nil
Associated anomalies	Nil	Nil	Tracheo -esophageal fistula
Location of perforation	Greater curvature	Lesser curvature	Greater curvature
Surgical treatment	PPD, Gastrorrhaphy	Gastrorrhaphy	Gastrorrhaphy, feeding gastrostomy, lower esophageal banding
Outcome	Died	Alive	Died

RESULTS

There were two females and a male, all of them term neonates. The average birth weight was 2.8kg (range 2.2 to 3.8kg). Median age at presentation was 9.7days (range 6 to 13days) and they had a diverse aetiology. All patients presented with abdominal distension as well as

pneumoperitoneum on x-rays. Case three had tracheo-esophageal fistula while the others had no associated anomalies. The site of perforation was on the body of stomach- (2), and the lesser curvature of the stomach – (1)). Two patients had gastrorrhaphy while the patient with TEF had gastrorrhaphy, feeding gastrostomy, and lower esophageal banding. Two patients died while 1 survived.



Figure 1 - plain abdominal xray- erect view – showing air under the right hemidiaphragm, pneumoperitoneum pushing the bowel loops inferiorly.

DISCUSSION

Neonatal gastric perforation is an uncommon disease with a very high mortality. This high mortality may be attributed to delayed recognition and presentation as seen in 2 of our cases. These delays have been attributed to misdiagnosis and delay at the initial hospital of first contact.

The first case of spontaneous neonatal gastric perforation was reported by Siebold in 1825 while the first successful surgical repair was reported by Leger et al in 1950.² Its reported



Figure 2 - showing 2 cm ragged perforation on the anterior wall of the proximal stomach(lesser curvature), the perforation was tamponaded by the left lobe of the liver

incidence is 1 in 5000 live births, and it constitutes 7% of all gastrointestinal perforations.³ The incidence in black population is 1/2900 with a male to female ratio of 4:1.⁴

It predominantly affects premature⁵ and low birth weight⁶ infants but it can also occur in term infants as observed in our case series. It usually presents within two to seven days of life.^{5,6} This may be a distinguishing feature between it and perforated NEC. While several case reports have suggested an increase in the prevalence in full term neonates^{7,8}, prematurity is still among the most common cause of mortality⁹. The mortality among premature infants was 62% and among term infants 26%. Prematurity was the only factor that was significantly associated with death.⁸

The precise aetiology and pathogenesis of gastric perforation in the neonatal period are still unknown. Mechanisms that have been proposed include: traumatic, ischemic and spontaneous.⁹ Traumatic perforations are due to vigorous nasogastric or orogastric tube placement. This may also result from severe gastric distention during the course of positive pressure ventilation during bagmask resuscitation or mechanical ventilation for respiratory failure. This is presumed to be cause in case 1.

Ischaemic perforations are due to conditions causing severe physiologic stress such as extreme prematurity, sepsis and neonatal asphyxia. Also, gastric hyperacidity has been reported in the first few days of life coupled with the stress ulcers from these conditions, it has been proposed that perforation can result from the transmural necrosis of the ulcers.

Neonatal gastric perforation could occur spontaneously without any associated gastrointestinal conditions and it was originally thought to be caused by a congenital absence of gastric musculature. The development of spontaneous neonatal gastric perforation is associated with the decreased quantity of interstitial cell of Cajal and damaged gap junction structure of the stomach wall.¹⁰

Before now, most gastric perforations have been described as spontaneous despite plausible reasons to explain how it could occur. Subsequently, over the years, the “spontaneous etiology” of gastric perforation has been contested and several associated factors have

been described, such as vigorous respiratory resuscitative measures, prematurity, neonatal asphyxia, tracheoesophageal fistula, increased intragastric pressure caused by distal obstruction such as antral web, duodenal atresia or obstruction, necrotizing enterocolitis, HIV¹¹ and , anatomic abnormalities of the stomach.^{8,12} The challenges posed by HIV and formula feeds, (as seen in case 2) , a known risk factor for NEC remains to be studied.¹¹ Again, marked gastric distension and perforation have been reported in patients with tracheoesophageal fistula as was the case in case 3.

Other theories that have been proposed to explain these events include hypoxic stress leading to gastric ischemia by selective shunting of blood away from the splanchnic vascular bed, sepsis, treatment with steroid therapy for bronchopulmonary dysplasia and the use of positive pressure ventilation or nasal continuous positive airway pressure without decompression of the stomach.^{8,12,13} Thus, protection of the stomach against over-distension is necessary in neonates at risk and those requiring ventilation.

Anatomic defects of the gastric muscular wall have been suggested to potentiate gastric perforation among neonates, especially in prematurity. The circular muscle layer of the newborn stomach normally contains several gaps, most prominently in the fundus, near the greater curvature. These gaps are more common in premature infants. Under normal circumstances, such gaps may have little clinical significance, but they are potential weak points in the stomach wall that might be susceptible to rupture if intragastric pressure increases.⁸

The most common site of perforation is on the anterior wall at or near the greater curvature of the fundus.⁸ The most common time for the perforation is within the first week of life particularly in between the first 2 and 7 days of life.⁹ Mechanical disruptions were reported to have predilection for the greater curvature like spontaneous gastric perforation.

Clinical features include vomiting, abdominal distention, and respiratory distress. Signs of hypovolemic shock and sepsis may supervene particularly when they present late. The diagnosis of gastric perforation could be suggested by plain radiograph of the abdomen. The upright film will show the “saddle” or “football” sign due to massive pneumoperitoneum.¹⁴ These infants may have rapidly progressive pneumoperitoneum with associated cardiopulmonary compromise because of the large size and proximal nature of the perforation. Prior to definitive surgical intervention, during the evaluation and resuscitation of the infant, percutaneous peritoneal drainage or needle decompression of the abdomen with large intravenous catheter may be required.¹⁵ This may even suffice as a method of treatment without the need for a primary surgical repair.¹⁴ Differential diagnoses include necrotizing enterocolitis, septicemia, intestinal obstruction, spontaneous pneumoperitoneum without gastrointestinal perforation, and others.

The optimal treatment involves debridement of the necrotic edges and a two-layer closure as was done in our patients. However, the extent

of necrosis sometimes may necessitate a partial or total gastrectomy.¹⁶ A thorough examination of the stomach for a second perforation and the entire gastrointestinal tract for other unsuspected perforations or abnormalities should be carried out. Poor prognostic factors are acidosis, prematurity, low birth weight, and delayed surgical intervention.⁷ The time between symptoms and surgical intervention has been reported as the only prognostic factor for survival in these children. This correlated with the only survivor in our reports too. Sepsis and surgical site infection was the most common complication amongst survivors.⁷ All 3 of our patients had anaemia, thrombocytopenia and leukocytosis.

CONCLUSION

Neonatal gastric perforation is a surgical emergency with a very high mortality despite the advances in neonatal care. It requires a high index of suspicion to make the diagnosis and to differentiate it from NEC, which is more common. It is hoped that early diagnosis and early intervention will improve the prognosis. We also propose that passing a nasogastric tube and ensuring free drainage of gastric air and fluid should form part of any resuscitation effort.

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