

An Uncommon Cause of Neonatal Gastric Perforation: A Case Report

PMY TANG, JWS HUNG, CSW LIU, MWY LEUNG

Abstract

Background: Neonatal gastric perforation is an uncommon surgical emergency. It is often associated with high mortality and morbidity in the past. **Methods:** We present a case of a full term neonate with an uncommon cause of gastric perforation. A full term baby girl with a body weight of 2.9 kg presented with pneumo-peritoneum on abdominal X-ray on day two of life. Emergency laparotomy revealed gastric fundal perforation, primary gastric repair and drainage gastrostomy were done. **Results:** The baby's post operative recovery was complicated with prolonged feeding intolerance with non-bilious vomiting. Repeated contrast gastrograms and follow-through revealed satisfactory contrast passage into duodenum and small bowel. Failing to respond to conservative management for gastro-oesophageal reflux disease and gastric dysmotility, a second exploratory laparotomy was performed. A pre-ampullary fenestrated duodenal web causing incomplete duodenal obstruction was found. A side to side duodeno-duodenostomy was done and the baby finally tolerated normal feeding postoperatively before discharge. **Discussion:** We discuss the different etiologies for neonatal gastric perforation with review of literature, and also the diagnostic difficulties in infants with incomplete duodenal obstruction due to pre-ampullary fenestrated duodenal web.

Key words Neonatal gastric perforation

Introduction

Neonatal gastric perforation is a surgical emergency. It usually occurs without apparent precipitating events and it can often give rise to an alarming degree of pneumo-peritoneum within a short period of time. Traditionally, neonatal gastric perforation is often considered as idiopathic or spontaneous without a specific known cause. We report a case of a full term neonate with an uncommon cause of gastric perforation.

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Clinical Presentation

A full term baby girl with a body weight of 2.9 kg was referred to our hospital for abdominal distention on day two of life. Her antenatal, birth and postnatal history were unremarkable, her Apgar scores were >8 at 1 and 5 minutes. No positive pressure ventilation was given after birth and there was no insertion of oral gastric tube prior to this admission. She had passed meconium and started milk feeding within 24 hours of life. Upon admission, she was haemodynamically stable and had no respiratory distress in room air. Physical examination showed grossly distended abdomen with no inguinal hernia. Oral gastric tube was inserted yielding only small amount of clear non-bile stained gastric aspirate. Urgent abdominal X-ray confirmed gross pneumo-peritoneum. Emergency laparotomy was carried out and complete inspection of the gastro-intestinal tract from stomach to rectum was done. Intra-operative findings showed heavily soiled peritoneal cavity, normal duodeno-jejunal junction position and a 3 cm full thickness

perforation at the fundus of the stomach (Figure 1a). Other than this, the gastric axis was normal and there was no gastric volvulus, the remaining small and large bowel appear grossly normal and viable. A 5 French infant feeding tube was inserted into the stomach via the perforation site, and antegrade normal saline injection confirmed distal bowel patency. As there was smooth flow of normal saline into the duodenum and the perforation site was quite far away from the pylorus, the infant feeding tube was never passed trans-pylorically.

The fundal perforation site was trimmed and primarily repaired with 4-0 Vicryl interrupted. An open Stamm gastrostomy with 8 French Foley catheter was also performed to ensure adequate gastric drainage.

The baby had uneventful early post operative recovery

and the histology of the stomach specimen came back to be normal stomach mucosa with absence of muscle musculature. After discussion with the pathologist, while the resected fundal specimen was of adequate size, it could neither diagnosis nor exclude the possibility of congenital muscle disorder.

An elective contrast gastrogram and follow-through on day seven post operation (Figure 2a) confirmed healed gastric fundal repair and unobstructed passage of contrast material from stomach to distal small bowel. Oral milk feeding was then gradually resumed. Although she was able to tolerate oral feeding initially when only a small volume of milk was fed, intermittent post prandial non-bilious vomiting ensued when more than 70 ml of milk was fed at a single feeding.

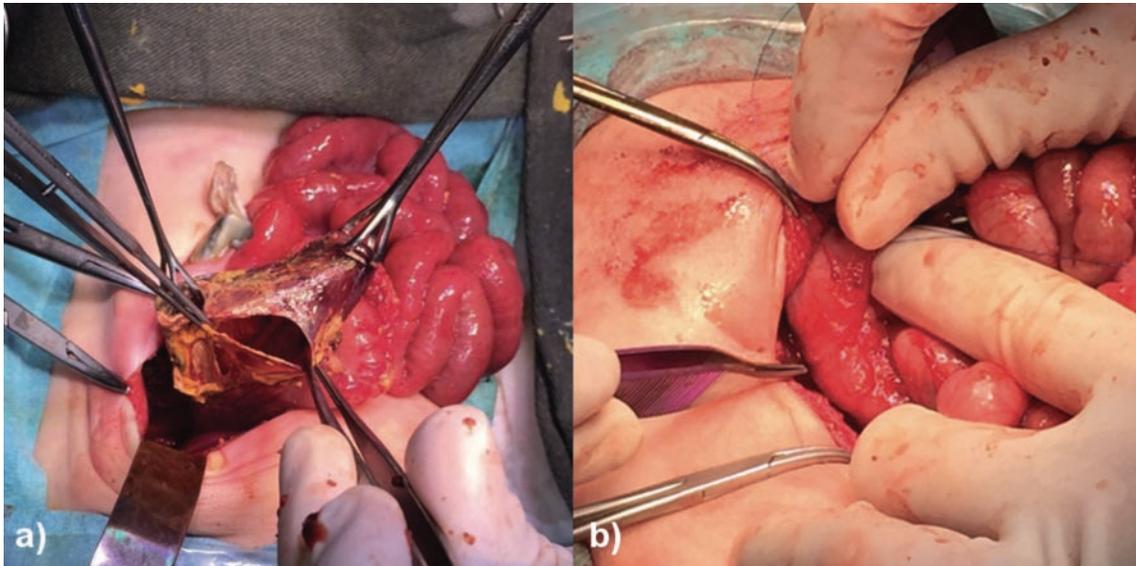


Figure 1 Intra-operative photo showing (a) stomach perforation; (b) subtle thickening at second part of duodenum.

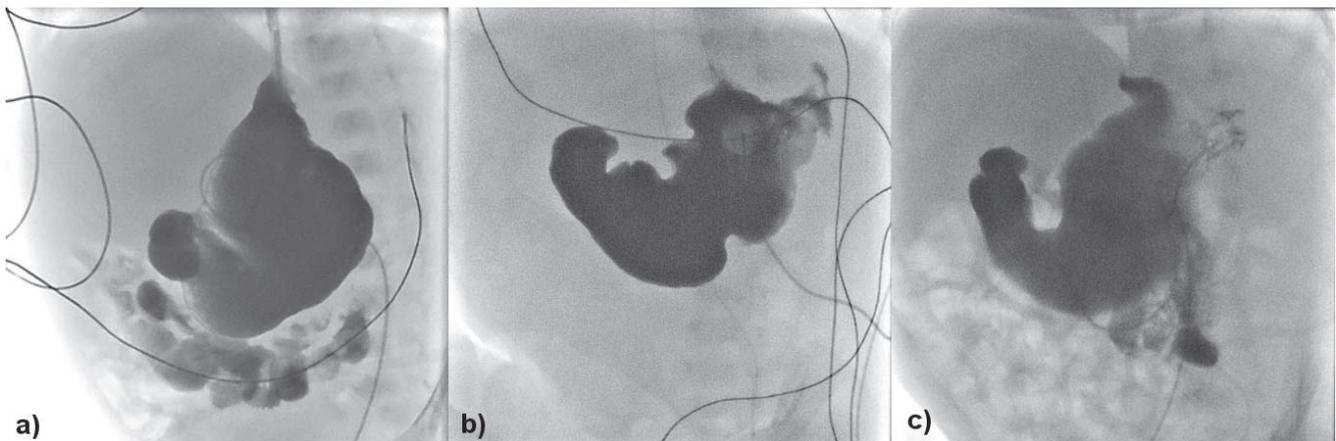


Figure 2 Contrast gastrogram done on (a) day 7 post-operation; (b) day 20 post-operation; (c) day 41 post-operation.

Given the risk factor for reflux due to the distorted stomach anatomy after operation and the presence of gastrostomy, the clinical diagnosis of gastro-oesophageal reflux was made and the baby was changed to an anti-reflux formula. Nonetheless, she continued to have feeding intolerance with large volume of pre-feed gastric aspirate despite even after switching to milk drip feeding. A second contrast gastrogram and follow-through was arranged on day 20 post operation (Figure 2b) and it showed hold up of contrast material in the stomach with a significant delayed passage into the small bowel. In view of the baby's age and the radiological findings of a suspected partial gastric outlet obstruction, an abdominal ultrasound was done to rule out pyloric stenosis.

The baby continued to suffer from impaired feeding tolerance and at this juncture, gastric dysmotility was suspected. Initially we adopted a conservative approach with watchful waiting as we believed her gastric motility problem might resolve with time as her gut matured. However, her feeding tolerance failed to improve despite multiple non-invasive measures such as frequent small meals, prop up positioning of the baby after meal, and the use of oral pro-kinetic agent (oral erythromycin at 10 mg/kg/dose every 6 hours). Full oral feeding was not achieved even at the sixth week after the first laparotomy. A third contrast gastrogram and follow-through was arranged on day 41 post operation (Figure 2c) and it showed partial passage of contrast material to the small bowel.

Progress

In view of the uncertain cause of feeding intolerance and the lack of clinical progress, an exploratory laparotomy with the possibility of a surgical drainage procedure such as pyloroplasty was arranged. The second laparotomy confirmed no significant bowel adhesion, no transition zone and no obvious discrepancy in the bowel caliber. On careful palpation, there was subtle thickening at the second part of duodenum (Figure 1b), raising the suspicion of a type 1 duodenal atresia. Upon exploration of the duodenum by longitudinal incision on the first part of duodenum, a 5 French infant feeding tube failed to pass through the duodenum to jejunum, confirming the diagnosis of a pre-ampulla fenestrated duodenal web resulting in incomplete duodenal obstruction. A side to side duodeno-duodenostomy with 4-O vicryl interrupted was performed.

The baby had uneventful post operative recovery and full oral feeding was tolerated. The Foley gastrostomy tube

was removed in the ward two weeks after the second laparotomy and it healed up completely two days later. The baby was then discharged home uneventfully. Subsequent follow up elective ultrasound did not reveal any associated congenital cardiac or urinary anomalies.

Discussion

Neonatal gastric perforation is a rare, serious and life-threatening condition.¹ While there are many theories regarding the pathogenesis of gastric perforation, the aetiology remains varied.² Historically, it was postulated that neonatal gastric perforation could be a result of a congenital absence of gastric musculature.³ However, this had been disputed by Shaw et al,⁴ his experiments had demonstrated that gastric perforation was caused by a mechanical rupture of the stomach secondary to increased gastric pressure, rather than a congenital agenesis of the gastric muscle. This theory was further supported by a more recent study by Yang et al,⁵ which showed association of concomitant gastro-intestinal tract anomalies with neonatal gastric perforation. Their study also showed that the morbidity and mortality associated with neonatal gastric perforation are often directly related to the causes, when GI anomaly related mortality rate is usually very low.

Another theory proposed by Gryboski⁶ suggested that neonates have uncoordinated oesophageal peristalsis until the third day of life and normal gastric motility does not occur until the third month of life. These anatomic immaturity and impaired motility would also potentially explain the peak incidence of neonatal gastric perforation being reported to be from the second to the seventh day of life.⁷ In our patient, failing to further advance enteral tolerance despite the use of pro-kinetic agents six weeks after the initial operation prompted us the second exploratory laparotomy, from which a missed duodenal web was found. While there is no single gold standard test to check the patency of the distal bowel during laparotomy when an intestinal perforation is encountered, it is not uncommon for surgeons to utilise the saline injection test. By observing for any abrupt change in the calibre of the distal bowel upon saline injection, the saline test would provide a quick and reliable way to ascertain distal bowel anatomical patency before closure of any intestinal perforation. Although it may not have the advantage of an enteroscopic direct visualisation of the intra-luminal condition, it is by far a much less invasive assessment of the distal bowel anatomy, especially in a neonate. However,

unfortunately in our patient, we have missed the fenestrated pre-ampulla duodenal web in the first operation and in the subsequent contrast gastrograms and follow-through.

Type 1 duodenal atresia is believed to be an early embryonic event. It is classically described to be associated with polyhydramnios antenatally and postnatally, it would give rise to a double bubble appearance on the plain radiograph with paucity of distal bowel gas. Upper gastrointestinal contrast study may also delineate a windsock deformity in cases with duodenal obstruction.⁸ During direct inspection at laparotomy, an external indentation may mark the site of attachment of the diaphragm. The presence of a mega-duodenum or an abrupt change in the calibre between different parts of the duodenum would also often prompt the surgeon the presence of an intra-luminal duodenal obstruction. These duodenal obstructions are most commonly sited just at the ampulla, and they are relatively uncommon to be found proximal to the ampulla of Vater.⁹ For a pre-ampulla fenestrated duodenal web giving rise to incomplete duodenal obstruction, non-bilious vomiting might be the only apparent clinical symptom, such as in our patient's presentation.

Moreover, the clinical course of our patient was further complicated by an early gastric perforation and the formation of the drainage gastrostomy. With the added consideration of the distorted anatomy at the gastro-oesophageal junction and potential underlying gastric dysmotility, we have mistaken the non-bilious vomiting to be part of the presentation of a more commonly seen condition such as gastro-oesophageal reflux. When the second contrast gastrogram and follow-through showed hold up of contrast in the stomach, we were again distracted by the potential cause of gastric dysmotility, partially supported by the histological finding of the lack of muscle musculature in the resected stomach. Another differential diagnosis such as pyloric stenosis was also considered, and it was ruled out

easily by an abdominal ultrasound.

We believe an incomplete pre-ampullary duodenal web could present as a diagnostic challenge, especially when its clinical presentation is complicated by an early gastric perforation. Clinicians should maintain a high index of suspicion and a thorough examination of the gastrointestinal tract is essential when a neonatal gastric perforation is encountered.

Declaration of Conflict of Interest

None

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