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Total gastrectomy with delayed Hunt-Lawrence pouch reconstruction for neonatal gastric perforation presenting with hematemesis

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Abstract

The differential for neonatal hematemesis ranges from benign etiologies to life-threatening emergencies. Neonatal gastric perforation is a rare cause of neonatal hematoma sis but is a deadly condition, requiring prompt diagnosis and treatment. The etiology is usually related to conditions predisposing to over distension of the stomach, such as positive pressure ventilation or distal obstruction, but in some cases cannot be determined. Patients generally present with abdominal distension and respiratory distress. We present a case of a 1-day old term baby girl who developed sudden onset hematoma sis and clinical deterioration, who was found to have a large proximal gastric perforation requiring emergent total gastrectomy with delayed reconstruction.

1. Introduction

Neonatal gastric perforation (NGP) has been described in multiple case reports and case series over the past century. Although previously thought to be a spontaneously developing condition, there are now thought to be multiple risk factors for the development of NGP including prematurity [1,2], positive pressure ventilation [2,3], injury from suction catheter

[4,5], and distal gastrointestinal obstruction [5]. In the majority of cases, symptoms include abdominal distension with or without emesis [6]. Nearly all are found to have pneumoperitoneum on abdominal X-Ray [7,8]. All but one reported case was managed with exploratory laparotomy and gastrectomy or primary repair [9], with mortality rates ranging from 0 to 75% [10]. Short-term complications include sepsis and anastomotic leak [4,6]. Long-term sequelae include poor feeding tolerance and vitamin B12 deficiency in the case of gastrectomy [11].

We present a case of a previously well, full term neonate who developed large-volume hematemesis at 12 hours of life and underwent emergent total gastrectomy with delayed reconstruction.

2. Case report

The patient was a full-term but small for gestational age (birth weight 2.835 kg) female neonate born at 41 weeks and 2 days to a 33-year-old prim gravid healthy mother by vaginal delivery. Prenatal care was uncomplicated except for a low-speed motor vehicle collision that the mother was involved in 3 weeks prior, with no injuries.

The infant had a weak cry and poor tone at birth and was started on CPAP at 6.5 minutes of life. She was transferred to the nursery on CPAP of 5 cm of pressure. An or gastric tube (OGT) was briefly placed for venting of gastric air. Her Apgar scores were 6 and 8 at 1 and 10 minutes respectively. Her initial hemoglobin was 14.7 g/dL. Chest X-ray was unremarkable. She was weaned to 2 L of oxygen by nasal cannula by 12 hours of life. She tried breastfeeding but had an episode of bloody emesis after her feed. As she was clinically improving and voiding and stooling normally, it was thought that this represented swallowed maternal blood so routine neonatal care was continued. At 21 hours of life, she had an episode of large volume bright red bloody emesis. Transfer to a tertiary care neonatal unit was arranged. An OGT was placed and 5 ml of bright red blood was aspirated. Repeat labs showed a hemoglobin of 13.2 g/dL and platelets of 398,000. Hematemesis continued, with a decrease in hemoglobin value to 13.2 g/dL 1.5 hours later. Coagulation studies showed an elevated INR of 1.7, and D-Dimer of 811 ng/ml.

She received a transfusion of 15 ml/kg of packed red blood cells (PRBCs) during transport due to ongoing blood loss. Her vitals were normal for her gestational age and she was not pale or mottled, however she was less reactive than a typical term neonate. She continued to have good oxygen saturation and was breathing comfortably with good pulses and perfusion. Her abdomen was soft, non-distended, non-tender, and did not appear discolored. She had produced meconium with very small streaks of blood. She was given intravenous vitamin K and a proton pump inhibitor in case of a possible bleeding ulcer. Pediatric Gastroenterology and Pediatric Surgery were consulted.

At this time, the OGT was placed to continuous low wall suction and drained 20 ml of bright red blood in 10 minutes. Despite the OGT, she continued to have episodes of hematemesis with large clots every 5 minutes, with repeated clogging of the OGT necessitating

replacement. To protect her airway, she was intubated. Preoxygenation was performed with blow-by oxygen and nasal cannula to avoid gastric dis-tension prior to intubation.

A supine and left lateral decubitus abdominal X-ray showed no free intraperitoneal air. An abdominal ultrasound also did not show any intraperitoneal air or fluid. At this time, her hemoglobin had decreased further to 8.6 g/dL despite the previous transfusion, so an additional 20ml/kg of pRBCs was given. Platelets were still reassuring at 163,000. Following transfusion, her hemoglobin had stabilized at 8.6 g/dL but platelets had decreased to 82,000. Due to concern for upper gastrointestinal bleeding, Pediatric Gastroenterology performed an urgent diagnostic endoscopy with plans for possible hemostasis.

Initial endoscopy showed normal appearing esophageal mucosa through the mid-esophagus but visualization of the distal esophagus and proximal stomach was obstructed by fresh and clotted blood. With insufflation, the patient's abdomen became distended and firm and she began having difficulty with oxygenation and ventilation. In addition, her lower extremities became pale and mottled with slow capillary refill. The endoscope was removed and a portable supine and cross-table lateral abdominal X-ray showed massive pneumoperitoneum (Fig. 1). The abdomen was decompressedby insertion of an 18-gauge angiocath at Palmer's point in the left upper quadrant, resulting in a rush of air with immediate improvement in oxygenation, ventilation, and lower extremity perfusion.

Exploratory laparotomy was performed via a supraumbilical trans-verse incision. Large volume hemoperitoneum was immediately encountered and the source of bleeding was localized to the left upper quadrant. The small bowel and colon appeared normal and uninjured with no congenital anomalies such as malrotation or intestinal atresia appreciated. The stomach was then inspected and found to be necrotic along the entire anterior surface up to the gastroesophageal junction, with a 6-cm perforation at the greater curvature of the stomach extending up to the gastroesophageal junction. Despite packing and near constant transfusion of various blood products, hemostasis was unable to be achieved and she continued to have massive bleeding. Due to thenecrotic and liquefied state of the stomach, there was no viable option for primary gastric repair. A total gastrectomy was performed, with proximal division at the gastroesophageal junction and distal ligation at the pylorus. Following gastrectomy, hemostasis was obtained. A Rouxen-Y feeding jejunostomy was created to allow for long-term enteral access, and the abdomen was closed. Total transfusion volume was 94 ml/kg of pRBCs, 31 ml/kg of platelets, 51 ml/kg of fresh frozen plasma, and 9 ml/kg of cryoprecipitate for a total of 185 ml/kg of blood products, or approximately 2.3 times her total blood volume.

The patient was extubated on post-operative day (POD) 3. She received 5 days of post-operative antibiotics and was started on jejunostomy feedings on POD 5. She tolerated advancement of jejunostomy feedings to her goal rate, however she developed vitamin B12 deficiency and required intermittent supplementation.

Six weeks after the initial surgery she underwent reconstruction of her gastrointestinal tract to restore continuity. The prior incision was opened and extensive adhesiolysis was performed. The esophageal pouch and all bowel were viable. A 6-cm Hunt-Lawrence pouch

was created to serve as a gastric replacement. This was performed by folding the proximal end of the transected jejunum onto itself and using the gastrointestinal anastomosis stapler to create a common lumen (Fig. 2). The jejunum was brought in an antecolic fashion to meet the distal end of the esophagus. The apex of the Hunt-Lawrence pouch as well as the esophageal stump were then opened, and a two-layer anastomosis was performed. A leak test was performed by instilling methylene blue via the nasojejunal tube and no leak was evident. The biliary limb of the reconstruction was then created by performing a jejunojejunostomy 30cm distal to the Hunt-Lawrence pouch. Finally, a feeding jejunostomy tube was placed 15 cm distal to the jejunojejunostomy and the abdomen was closed.

Esophagram performed POD 5 after re-anastomosis showed no evidence of leak at the anastomosis. She was started on jejunostomy feeds on POD 7 and on oral feedings on POD 11. Her post-operative course was complicated by peri-incisional cellulitis and superficial skin dehiscence which resolved with antibiotics and local wound care. She was discharged on POD 45 and is being followed by pediatric gastroenterology and pediatric surgery as an outpatient.

Surgical pathology of revealed diffuse hemorrhage within the mucosal, muscular, and serosal layers of the stomach. Extensive edema was present within the submucosal and muscular layers, resulting in accentuated spacing between muscle bundles; however, there was no convincing evidence of muscular agenesis. In addition, extensive patchy coagulative necrosis was present throughout the specimen.

3. Discussion

We present a case of catastrophic gastric hemorrhage with gastric wall necrosis and resultant perforation in a day-old term infant. Neonatal gastric perforation is a rare disease with high morbidity and mortality rates. Several risk factors are identified in this patient. It is possible that positive airway pressure resulted in overdistension of the gastric lumen, causing mucosal ischemia and subsequent hemorrhage. There may have also been trauma as a result of orogastric tube placement. The distension and ischemia may have been exacerbated by insufflation during the endoscopy.

Several risk factors of neonatal gastric perforation have been identified in the literature. The most commonly cited factor is gastric distension due to positive pressure ventilation via orotracheal intubation or CPAP [2,12]. Other proposed causes include perinatal hypoxia resulting in ischemia to the stomach [13], immature esophageal and gastric peristaltic coordination resulting in increased gastric pressures [1], vasculopathy or thrombosis within the blood vessels supplying the stomach [7], and the use of indomethacin for congenital heart disease [8] or steroids for neonatal respiratory distress syndrome [4]. There have also been reports of congenital muscular agenesis within the wall of the stomach [2], but this etiology has been called into question. It is possible that mucosal edema mimics muscular agenesis by causing separation of muscle bundles [12], as in this case. Finally, traumatic etiologies must be considered, including perforation from orogastric tube placement or accidental esophageal intubation with the endotracheal tube [14]. Prematurity and low birth weight have also been identified as associated risk factors [1,13].

The most common presenting symptom is acute abdominal distension with or without associated emesis. When emesis is present, it has been described as both nonbilious [11] and bilious [15]. Only rarely is hematemesis the primary presenting symptom as in our case [10]. Most neonates present within the first week of life, with days 3–6 being most commonly reported [16]. Almost uniformly, abdominal radiographs show pneumoperitoneum [2].

Both premature and term infants have been found to have neonatal gastric perforation. Mortality rates are consistently higher in premature infants [1]. When diagnosed early and treated promptly, many neonates have been reported to survive this condition, but mortality rates range as high as 75% [10]. Several congenital abnormalities have been seen in association with neonatal gastric perforation, including necrotizing enterocolitis [3], distal intestinal obstruction such as intestinal atresia or stenosis [6], and midgut volvulus [2]. None of these anomalies were identified in our patient, but the surgeon must be aware of the potential for associated anomalies and perform a thorough evaluation of the remainder of the gastrointestinal tract at the time of surgery.

The location of gastric perforation varies, but in most reports is located along the greater curvature of the stomach. Rarely, some perfo-rations have been found to occur along the lessercurvature [3]. In most cases the perforation is along the anterior wall of the stomach but posterior perforations have been reported as well [4]. In most cases, the perforation is relatively large in size, up to 10 cm [6,17].

In contrast to most reported cases where primary repair of the perforation was performed, the patient in this case underwent total gastrectomy due to the necrotic and liquefied nature of the gastric wall at presentation. She then subsequently required delayed reconstruction with creation of a neo-stomach by performing a Hunt-Lawrence pouch. This has been reported only one time previously in the literature for neonatal gastric perforation [13], although there have been other reports of total gastrectomy followed by immediate esophagojejunostomy reconstruction [11]. In these rare cases in which all or most of the stomach is removed, providers must be aware of the potential for vita-min B12 deficiency due to the loss of intrinsic factor secretion [11]. Interestingly, in one case with gastric perforation diagnosed by pneumoperitoneum on abdominal radiograph, the patient was successfully managed with placement of a drainage catheter and did not subsequently undergo surgery. However, this patient likely had a spontaneous gastric perforation, rather than extensive gastric necrosis, and non-operative management is not supported by the literature at this time.

4. Conclusion

Neonatal gastric perforation is rare but can result in cardiovascular collapse with high mortality. Presenting symptoms include abdominal distension and emesis which may be bilious, nonbilious, or bloody in nature. Abdominal radiographs will show pneumoperitoneum, and diagnosis is confirmed at the time of surgical exploration. If possible, gastric perforation may be repaired primarily, but in some cases may require total gastrectomy with delayed reconstruction.

Patient consent

Parental consent was obtained to report this case.

Funding

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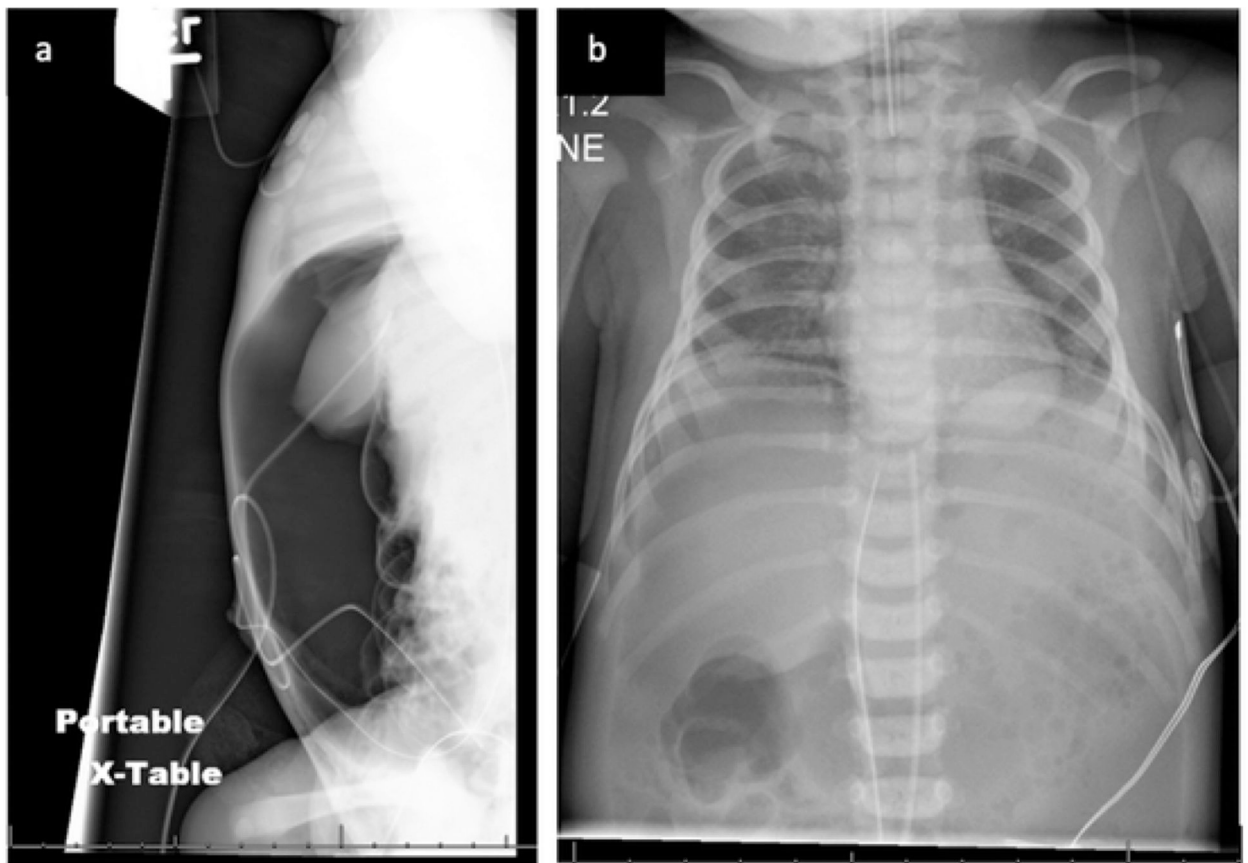
Fig. 1.
Cross-table (a) and supine (b) abdominal radiographs demonstrating massive pneu moperi toneum with diaphrag matic elevation.
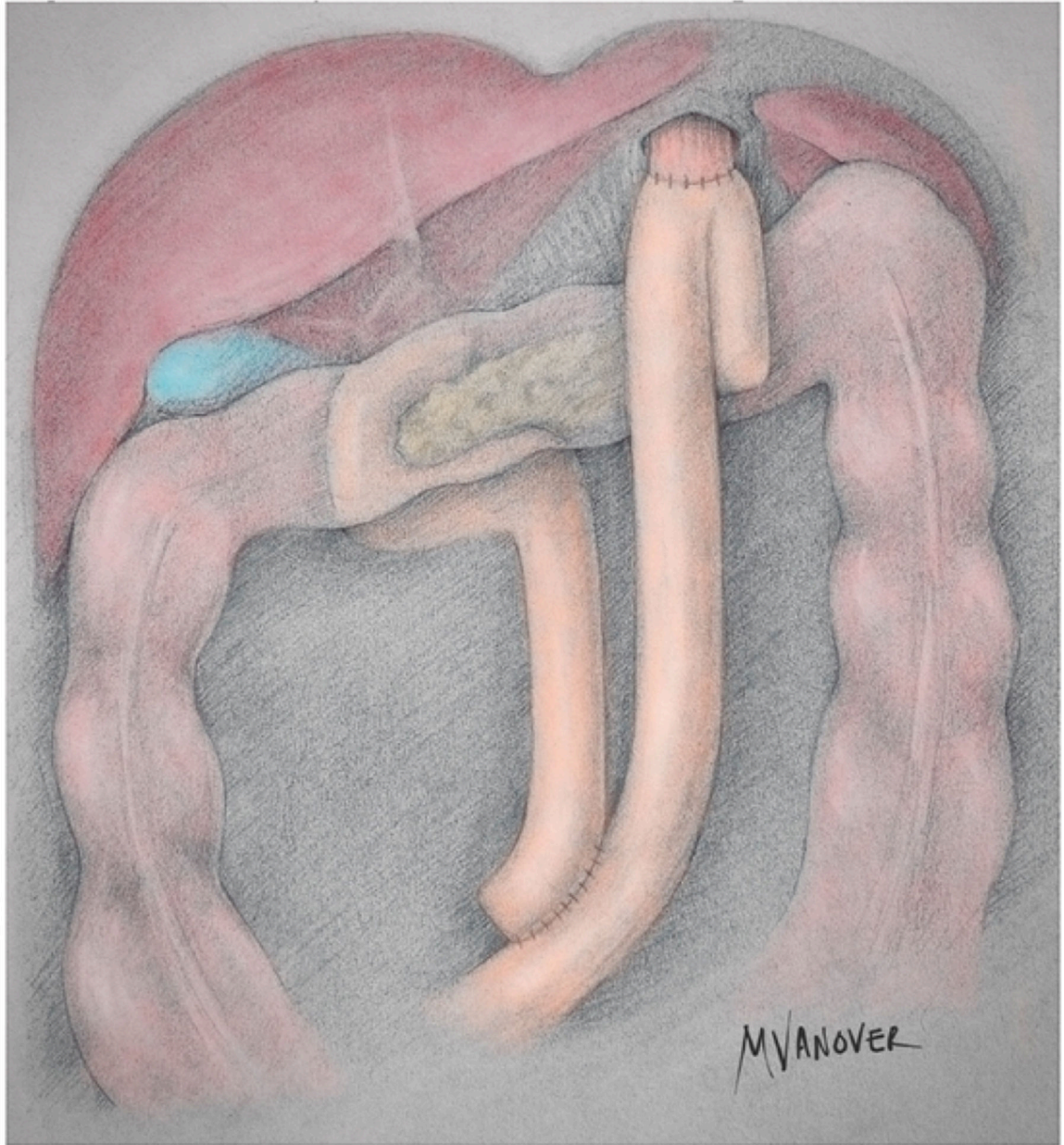
Fig. 2.
Anatomy of the Hunt-Lawrence pouch reconstruction.