Received: 2016.04.08
Accepted: 2016.05.17
Published: 2016.09.30

ISSN 1941-5923
© Am J Case Rep, 2016; 17: 694-698
DOI: 10.12659/AJCR.898939

Spontaneous Gastric Perforation in Two Adolescents

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Case series

Patients: Female, 11 • Male, 15
Final Diagnosis: Spontaneous gastric perforation
Symptoms: Abdominal pain • distention • vomiting • leukocytosis
Medication: —
Clinical Procedure: Both patients had surgery
Specialty: Gastroenterology

Objective: Rare etiology
Background: Spontaneous gastric perforation is a rare clinical disorder. The majority of the available data have been reported in the neonatal age group. There are a few cases of spontaneous gastric perforation in preschool children. To our knowledge, there is no published information on spontaneous gastric perforation in older children and adolescents.
Case Report: We describe the presentation and clinical course of two adolescent children who presented with spontaneous gastric perforation. Both children presented with acute onset abdominal pain, which progressively worsened. In both cases, the patient were taken urgently to the operating room after imaging studies had shown pneumoperitoneum. In both cases, surgery revealed gastric perforation with no obvious etiology, specifically no ulcer, inflammation, or other pathology.
Conclusions: These two cases highlight the importance of including spontaneous gastric perforation, not just the typical duodenal/gastric ulcer, in the differential of a patient with severe abdominal pain and distension, who has imaging showing pneumoperitoneum.

MeSH Keywords: Adolescent • Pneumoperitoneum • Rupture, Spontaneous

Full-text PDF: <http://www.amjcaserep.com/abstract/index/idArt/898939>

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Background

Spontaneous gastric perforation in children is a serious condition that could be potentially fatal [1]. There are several reports of gastric perforation in neonates, which although rare is associated with a poor prognosis [2]. The etiology of spontaneous gastric perforations remain unclear but certain variables, such as prematurity and nasal hypoventilation, have been implicated as contributing factors [2]. In older children, spontaneous gastric perforation remains very rare. Though gastric perforation occurs secondary to causal events such as trauma, peptic ulceration, or drug administration, cases of spontaneous gastric perforation have rarely been reported in children. There are a few reports of spontaneous gastric perforation in the adult population.

We present our experience at our institution from October 2013 and August 2015 that included four pediatric patients with gastric perforation. Two of the patients were excluded from this case study because their gastric perforations were attributed to gastric ulcers. Therefore, only two patients (11 years of age and 15 years of age) were included, as these two patients had no etiology for perforation. The clinical, imaging, and surgical findings in both children were reviewed. The images were interpreted by a pediatric radiologist at our institution. Application to the Institutional Review Board (IRB) for this case study was done and approved as per our hospital policy.

Case Report

Case Report 1

An 11-year-old female presented with abdominal pain of three days duration. Abdominal pain was associated with one episode of non-bilious non-bloody emesis. Due to worsening abdominal pain, she was taken to an outside hospital where laboratory studies were significant for elevated WBC of 18,000/UL with a neutrophil predominance and 25% bandemia. CT of the abdomen demonstrated pneumoperitoneum and ascites (Figure 1). The patient was transferred to our institution for definitive pediatric surgery care. Upon arrival at our hospital, she was noted to be in significant discomfort. Physical examination showed tachycardia and a tense and distended abdomen with both voluntary and involuntary guarding noted. She was taken emergently to the operating room where she underwent laparoscopic repair of a gastric perforation which measured 1×1 cm and was noted to be in the anterior distal stomach. The perforation was repaired using an omental patch. No biopsies were obtained since the gastric mucosa appeared normal. She was transferred to the PICU for close monitoring. She was transferred to the pediatric floor two days later. On post-operative day four, she was advanced to a clear

liquid diet and later a regular diet. Stool *Helicobacter pylori* enzyme linked immunosorbent assay (EIA) and antibody (Ab) panel were negative. She was discharged home on omeprazole 40 mg daily. She had a repeat *H. pylori* screen done as an outpatient six weeks later, which was negative. She underwent an upper endoscopy five months later, which was normal. Histopathology of endoscopic biopsies were negative for any pathology. She was weaned off omeprazole after eight months without any issues. She is being followed at the gastroenterology clinic at our hospital. She did well initially but recently developed epigastric abdominal pain one year after omeprazole wean. She was restarted on omeprazole 40 mg. She underwent a repeat upper endoscopy, which was significant for nodular mucosa in the antrum but was otherwise normal. A CLO (campylobacter-like organism) test done from gastric biopsy was negative. Histopathology was negative of any pathologic diagnosis.

Case Report 2

A 15-year-old male presented with acute onset periumbilical abdominal pain and left shoulder pain of one day duration. The shoulder pain preceded the abdominal pain, which he described as severe, constant, sharp, and stabbing in nature. In the emergency department, physical examination was significant for right lower quadrant tenderness and guarding. Preliminary labs were significant for elevated WBC of 16,000/UL. Chest x-ray demonstrated free air beneath his diaphragm (Figure 2). Surgery was consulted and he was urgently taken to the operating room. Laparoscopy showed a 3 mm perforation near the lesser curvature about midway between the pylorus and the gastroesophageal junction. Gastric perforation was repaired with a Graham patch. No biopsies were obtained since the gastric mucosa appeared normal. Post-operatively, the patient was started on Protonix infusion and given antibiotics prophylactically. *H. pylori* Ig A was positive but his stool *H. pylori* EIA was negative. On post-operative day three, he had an upper GI which was negative for a gastric leak. He was advanced to a clear liquid diet and eventually to a soft mechanical diet. He was discharged home on lansoprazole 30 mg twice daily. He was seen in follow-up one month later and was doing well. An upper endoscopy was done two months later demonstrating post-surgical changes at the perforation site. A CLO test was negative. The patient was not treated for *H. pylori* since his stool *H. pylori* and CLO test (which are more specific tests for evaluation of *H. pylori*) were negative. Histopathology results of his endoscopic stomach biopsies were negative for any pathologic diagnosis. He was eventually weaned off lansoprazole. He has been lost to follow-up since his last clinic visit in November 2013.

Figure 1. Radiographs from Patient A. Abdominal CT scan with intravenous and oral contrast. (A) Axial abdominal CT scan demonstrating intraluminal gastric contrast (white arrow) and free intraperitoneal air (asterisk). (B) Axial abdominal CT scan demonstrating extravasation of oral contrast from the gastric lumen (white arrows) and free intraperitoneal air (asterisk). (C) Sagittal abdominal CT scan demonstrating free intraperitoneal air (asterisk). (D) Coronal abdominal CT scan demonstrating extravasation of oral contrast (white arrows) and free pelvic fluid (star).

Figure 2. Radiographs from Patient B. (A) Frontal upright chest radiograph demonstrating air beneath the diaphragms, consistent with free intraperitoneal air (asterisk). (B) Left lateral decubitus radiograph demonstrating air overlying the right hemi-abdomen, consistent with free intraperitoneal air (asterisk).

Discussion

Spontaneous gastric perforation is rare. The etiology remains unclear.

Spontaneous gastric perforation in neonates has been reported in the literature. It is rare beyond the neonatal period [2]. The etiology of spontaneous gastric perforation in neonates still remains unclear. Several possibilities include congenital defects of the gastric wall, mechanical disruption, stress ulceration secondary to neurogenic disorders, and ischemia of the gastric wall secondary to vascular shunting. Other postulations include diving reflex and intestinal obstruction [1,2]. Some risk factors reported to be associated with the development of neonatal gastric perforation include prematurity and nasal ventilation [1]. The majority of neonatal gastric perforations occur in the anterior side of the greater curvature [3–6].

Reports of gastric perforation in 3- to 5-year-olds are uncommon. When it does occur, it is usually due to a casual event such as peptic ulceration, trauma, aerophagia, or drug administration [2,3]. It has been reported in conjunction with Rett

syndrome and Burkitt lymphoma [6]. Iatrogenic causes of gastric perforation include blunt trauma due to nasogastric tube placement or over-distention of the stomach due to positive pressure ventilation. There have been suggestions in the literature that elevated intraluminal pressure may play a role in the mechanism underlying this condition in preschool children [3,6]. Some authors propose that ischemia of the gastric wall is instrumental in the pathophysiology of this condition [3].

Spontaneous gastric perforation is occasionally seen in adults. In the adult population, the majority of gastric perforations are caused by carcinoma or peptic ulcer [2,3]. In adults, a female preponderance has been noted, with the majority of the ruptures occurring around the lesser curvature [2,3]. It has been proposed by several authors that the lesser curvature is more often affected because it has a decreased coefficient of elasticity due to the absence of a mucosal fold. In addition, the lesser curvature of the stomach undergoes greater stretching than other parts of the stomach when it is distended. Spontaneous gastric rupture has been reported in pregnancy in association with an abrupt rise in abdominal pressure in an already distended stomach [6]. The proposed mechanism in

these patients is that impaired perfusion and/or obstruction in venous drainage due to high intra-luminal pressure led to necrosis of the gastric wall [6]. Clinical features described in the adult population which suggest gastric perforation include abdominal distention, abdominal rigidity, subcutaneous emphysema, and evidence of shock [3,6]. In neonates, common clinical features were poor activity, abrupt onset abdominal distention, and respiratory distress [1].

Imaging features of spontaneous gastric perforation include pneumoperitoneum on radiograph and oral contrast extravasation with gastric wall irregularity or disruption [1]. Treatment of spontaneous gastric perforation is surgical. A review of the literature showed that the gastric defect is usually repaired using simple closure or by partial gastrectomy [1,2]. In children, repair of a gastric rupture by simple closure rather than a sleeve gastrectomy is preferred because of associated feeding difficulties with a sleeve gastrectomy [2].

In our experience, we had two pediatric patients with spontaneous gastric perforation. In our patients, there was an equal distribution between both sexes, one female and one male. Both patients were adolescents (11 years of age and 15 years of age). Our patients had perforations near the lesser curvature.

Both patients were negative for *H. pylori* which has been noted to cause ulcerations and if untreated could lead to perforation.

Conclusions

Gastric perforation is a surgical emergency. It should be included in the differential diagnosis of any patient who presents with sudden severe abdominal pain, distention, and peritoneal signs. The four features described in the adult population that suggest gastric perforation include: abdominal distention, abdominal rigidity, subcutaneous emphysema, and evidence of shock [3,6]. In neonates and children, clinical manifestations include: abdominal distention, vomiting, poor activity, and respiratory distress. Radiologic imaging with evidence of pneumoperitoneum is a common radiologic finding in these cases, and treatment is surgical repair of the gastric defect. Both of our patients presented with significant abdominal pain and distention with radiological evidence of pneumoperitoneum. Our patients underwent emergent repair of their perforations using a Graham patch (simple closure). We treated them with acid suppression for several months and weaned them off these medications without any difficulty. The causes of these perforations were deemed as spontaneous.

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