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Research Article

Perforated Gastric Ulcer in a 2-Year-Old Child: A Case Report and Literature Review

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Abstract

Perforated Gastric Ulcer (PGU) is an exceptionally rare diagnosis in paediatric population, particularly in the absence of identifiable pharmacologic, infectious, or neoplastic aetiologies. We report a case of a 2-year-5-month-old Malay boy with Global Developmental Delay (GDD) and failure to thrive, who presented with nonspecific gastrointestinal symptoms and was ultimately diagnosed with a perforated gastric ulcer at the pylorus-D1 junction. He underwent laparotomy and primary repair of the gastric ulcer, without omental patch. Histopathology was negative for *Helicobacter pylori*, fungal elements, and malignancy. This case underscores the diagnostic challenges in neurodevelopmentally impaired children and highlights the importance of early surgical intervention. We discuss the clinical presentation, operative findings, and review current literature on paediatric PGU.

Keywords: Perforated gastric ulcer; Case report; Toddler; Pneumoperitoneum; Omentopexy; Omentum patch; Primary repair

Introduction

Peptic Ulcer Disease (PUD) is relatively uncommon in paediatric population. It is characterized by mucosal injury to the stomach or duodenum as a result of an imbalance between protective and aggravating factors within the gastrointestinal tract [1,2]. The aetiology of peptic ulcers in children is multifactorial, and most commonly associated with *Helicobacter pylori* infection, the use of Non-Steroidal Anti-Inflammatory Drugs (NSAIDs), physiological stress, and systemic illness [3-5]. Perforation represents a rare but life-threatening complication of PUD. Perforated gastric or duodenal ulcer among children poses significant diagnostic challenges and may lead to delay in diagnosis due to their atypical presentation. Perforated gastric ulcers in toddlers, especially those with Global Developmental Delay (GDD), is exceptionally rare and poorly understood. This case highlights the diagnostic and therapeutic challenges of PGU in a neurologically impaired child. This case report was prepared following the CARE guidelines.

Case Presentation

A 2-year-5-month-old Malay boy with a background of GDD and failure to thrive (current weight: 8.6 kg) presented with a five-day history of reduced oral intake, intermittent fever, vomiting, and progressive lethargy. There was no history of diarrhea, respiratory symptoms, urinary complaints, or recent use of supplements or over-the-counter analgesics.

On Day 3 of illness, he was evaluated at a paediatric specialist clinic and treated presumptively for acute gastroenteritis. However, his condition deteriorated the following day, with increasing lethargy and clinical signs of dehydration, including sunken eyes. He was initially admitted with a diagnosis of acute gastroenteritis and 10% dehydration, but was subsequently referred to paediatric surgery for acute abdomen, where abdominal X-ray and ultrasound was done prior to referral. Upon assessment, the child exhibited persistent abdominal discomfort, abdominal distension, and a single episode of coffee-ground vomiting (Figure 1). He appeared lethargic with sunken eyes but maintained good peripheral perfusion. Vital signs revealed tachycardia (heart rate: 140 bpm), temperature of 37°C, supported on 3L nasal prong oxygen. His abdomen was distended but soft, without guarding or skin discoloration (Figure 2 and 3).

Laboratory investigations showed normochromic normocytic anaemia (Hb 8.9 g/dL), normal leukocyte count (7.3 $\times 10^9$/L), and platelet count of 150 $\times 10^9$/L. Arterial blood gas analysis revealed compensated metabolic acidosis: pH 7.38, pCO_2 23 mmHg, pO_2 189 mmHg, bicarbonate 13.6 mmol/L, base excess −9.9, and lactate 0.9 mmol/L. Abdominal radiography demonstrated pneumoperitoneum with Rigler's sign, suggestive of gastrointestinal perforation. Ultrasonography of the abdomen revealed a complex fluid collection in the lower abdomen and confirmed the presence of free intraperitoneal air (Figure 4-6).

The patient underwent emergency exploratory laparotomy. Intraoperatively, there is presence of 300 cc purulent peritoneal fluid and a 3×3 cm perforation at the anterior pylorus–D1 junction. Adhesions were noted between the gallbladder, transverse colon, and terminal ileum at the perforation site. The ulcer edges were debrided, and primary repair was performed using 4/0 Monoplus sutures. No omental patch applied. The peritoneal cavity was thoroughly irrigated with warm saline, and a subdiaphragmatic drain was placed (Figure 7 and 8).

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Figure 1: Coffee ground gastric aspirate.

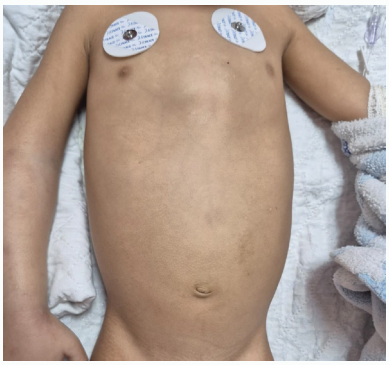

Figure 2: Patient's distended abdomen.

Figure 3: Patient's distended abdomen.

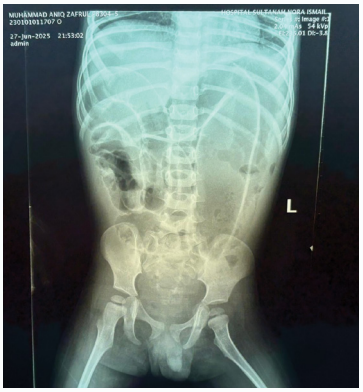

Figure 4: Abdominal x-ray shows double wall sign indicating pneumoperitoneum.

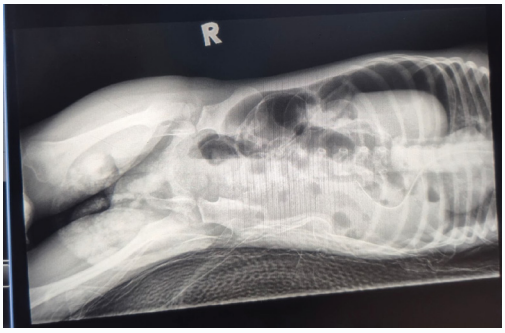

Figure 5: Lateral decubitus x-ray shows air under diaphragm.

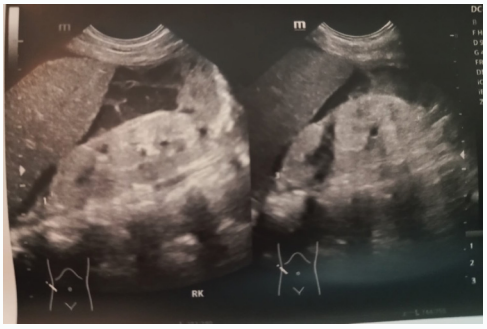

Figure 6: Abdominal ultrasound shows loculated free fluid at the Morrison's pouch.

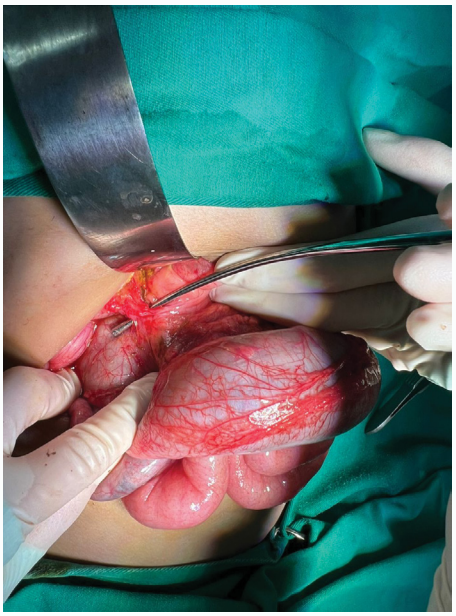

Figure 7: Omental adhesion at the duodenal ulcer.

Postoperatively, the child underwent further evaluation to exclude central nervous system infection. MRI of the brain revealed hydrocephalus, while cerebrospinal fluid analysis and cultures were negative for bacterial or fungal pathogens. Peritoneal fluid culture grew Saccharomyces cerevisiae, a rare finding in paediatric peritonitis. Histopathological examination of the ulcer margins revealed acute-on-chronic inflammation, with no evidence of *H. pylori*, fungal elements, or malignancy.

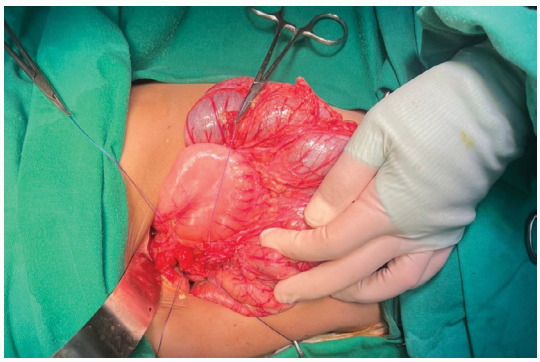

Figure 8: Perforated pylorus-D1 junction with sutures applied at the edge of perforation site.

He remained nil by mouth for seven days and received total parenteral nutrition during this period. He was hospitalized for two weeks for nutritional rehabilitation. He was treated with a week-course of IV cefotaxime and flagyl, and empirical eradication therapy for 2 weeks before HPE result was available. He was discharged well with no recurrent admission, weight gaining upon discharge.

Discussion

Paediatric Perforated Peptic Ulcer (PPU) is a rare entity, especially among children under the age of five. Although Peptic Ulcer Disease (PUD) is traditionally considered an adult condition, its incidence in children has been increasing over recent decades. This trend, coupled with the potential for severe complications like perforation, highlights the importance of maintaining a high level of clinical suspicion, even in very young patients.

Generally, the incidence of perforated peptic ulcer in children is more common among males than females [1]. The patient's initial presentation with nonspecific gastrointestinal symptoms: vomiting, lethargy, and abdominal distension, which was at first, misattributed to acute gastroenteritis. Such diagnostic delays are not uncommon in paediatric PGU, especially among children with neurodevelopmental delay. This case highlights the diagnostic and therapeutic challenges of PPU in a child with neurodevelopmental delay, where atypical symptoms and limited communication hinder early recognition.

Radiographic evidence of pneumoperitoneum ultimately led to the diagnosis of a perforated peptic ulcer. In this case, the patient also underwent an abdominal ultrasound, which revealed multiloculated free fluid in Morrison's pouch. While radiographic detection of pneumoperitoneum remains a crucial diagnostic indicator, its absence does not exclude the possibility of PPU. The optimal imaging modality for diagnosing PPU in children with non-specific clinical presentations and a negative abdominal X-ray (AXR) remains a subject of ongoing debate. Computed Tomography (CT) is particularly valuable due to its high sensitivity for detecting free air and its objective imaging capabilities. CT not only confirms the presence of pneumoperitoneum but also accurately identifies the site of pathology, potentially allowing children to avoid unnecessary laparotomy and surgical exploration [2]. Given the concern for radiation exposure in paediatric patients, many clinicians advocate for the use of ultrasound as the initial imaging modality. When performed by an experienced operator, ultrasound can be a reliable and effective tool in diagnosing Perforated Peptic Ulcer (PPU), particularly duodenal ulcers [6]. Characteristic sonographic findings include the presence of free intraperitoneal air, gas locules, and discontinuity of the duodenal wall, which may indicate a site of perforation. As in this case, ultrasound and abdominal X-ray are able to detect pneumoperitoneum, hence avoided the need for CT and its radiation side effect.

The case here is unusual not only due to the patient's age but also due to the fact that pyloric gastric perforations are far less common than perforations in the duodenum among children. According to a meta-analysis by Vidović et al. [1], duodenal perforations accounted for 73% of PPU cases, while only 27% were gastric perforations. The ratio of duodenal ulcers incidence compared to gastric ulcers is 18:4 worldwide [2]. Most paediatric patients with PPU are older children or adolescents, with a median age of 11 years (range 3.2–16.5) [1], making cases in younger children extremely rare. Similarly, Wang et al. [3] reported a series of 20 children with gastric perforation, in which only a minority were under the age of five.

Surgery remains the cornerstone of treatment for PPU. In this case, a sizeable 3×3 cm perforation at the pylorus–D1 junction was repaired primarily without an omental patch. While the Graham patch is traditionally favoured for its mechanical and vascular support, primary closure may be appropriate in select cases where tissue viability is adequate and omental mobilization is challenging. A recent systematic review found that simple suturing, with or without omentopexy, was the most commonly utilized technique in paediatric Perforated Peptic Ulcers (PPU), which was associated with a relatively low postoperative complication rate of 14.5% and a mortality rate of 3.8% and favourable outcomes. In larger perforations with friable edges which approximation of the perforation edges is not possible, pedicled omentopexy can be done as suggested by Wang et al. [4]. For giant perforations, primary repair with or without gastrostomy yield favourable outcomes [4]. However, gastrectomy is generally avoided in children as it causes feeding problems post-operatively, likely due to reduction of gastric volume for reservoir and decrease in ghrelin hormone production responsible for appetite [7,8]. Laparoscopic surgery is only recommended if patient is stable for operation. As in our case, child has labile blood pressure with transient response to fluid resuscitation, which we decided for laparotomy.

The aetiology of PPU in children is multifactorial. *H. pylori* infection remains a well-known risk factor in adult population, but its prevalence in paediatric PPU varies between studies. In many cases like ours, no identifiable infectious or pharmacologic trigger is found. In fact, Diaconescu et al. [5] describes a case that also lacked clear etiological factors, highlighting the idiopathic nature of some paediatric ulcers. Other proposed aetiologies include congenital anomalies like VACTERL, stress- related mucosal damage, and systemic infections [7]. Our case similarly lacked evidence of *H. pylori* infection, NSAID use, or systemic illness, but his presentation of PPU could be attributed to his hydrocephalus leading to stress-related ulcer.

The microbiological profile of this case is particularly noteworthy. Histopathology was negative for *H. pylori*, fungal elements, and malignancy, aligning with emerging data suggesting a lower prevalence of H. pylori in paediatric PGU compared to adults. Interestingly, peritoneal fluid culture yielded Saccharomyces cerevisiae, a rare finding in paediatric peritonitis. While typically considered a benign commensal or contaminant, its presence may reflect translocation in the context of severe mucosal disruption. Empiric antifungal therapy was not initiated, consistent with current guidelines that discourage routine antifungal use in the absence of immunosuppression or

clinical deterioration [9].

The patient's postoperative course was complicated by underlying hydrocephalus, which was unrelated to the ulcer pathology but highlights the importance of comprehensive evaluation in children with GDD. Nutritional rehabilitation and parenteral support were essential components of recovery, given the child's baseline failure to thrive.

Conclusion

This case reinforces several key principles such as the need for vigilance in atypical presentations, the feasibility of primary repair in selected PGU cases, and the importance of individualized antimicrobial strategies. It also contributes to the limited literature on PGU in toddlers, advocating for greater awareness and timely surgical intervention in this rare but serious condition.

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