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Peptic Ulcer Perforation in A Pediatric Patient: A Common Disease with an Uncommon Presentation

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ABSTRACT:

A 14-year-old boy from a remote locality of Palpa district, Nepal, presented to the emergency room with chief complaints of pain in the upper central abdomen for three hours associated with one episode of non-bilious vomiting. In view of raised neutrophil count and pneumoperitoneum on chest x-ray, a diagnosis of peptic ulcer perforation was reached. On exploratory laparotomy, a perforation of about 0.4x0.4 cm at the prepyloric region, near the lesser curvature of the stomach, was noted with flakes on stomach surface and small intestine. Minimal dirty fluid was present in the Morrison's pouch. Peritoneal lavage followed by primary repair of the perforation was performed by applying modified Grahm's patch technique. The postoperative period was uneventful, and subsequently the patient was discharged on the seventh postoperative day. Poorly understood mechanisms behind pediatric peptic ulcer perforations suggest possible roles for idiopathic ulceration, congenital muscular defects, ischemia, or stress-related mucosal injury.

Keywords: Exploratory laparotomy; Pneumoperitoneum; Pediatric patient; Peptic ulcer perforation.

INTRODUCTION:

In children, the foregut is the part of the digestive tract that is least likely to perforate.[1] The diagnosis of pediatric Peptic Ulcer Diseases (PUD) has increased since the advancement in endoscopy. Reports of PUD in adults make up the majority of the literature on the condition; however, studies on PUD in pediatric patients are less common. Despite being a recognized cause of pediatric stomach discomfort, PUD is still a rare condition in children. Over 50% of PUD cases are diagnosed due to complications. Adults who have a perforated peptic ulcer have an annual incidence of 3.8 to 14.0 cases per 100,000 people.[2] Here, we present an uncommon case of peptic ulcer perforation in a pediatric patient.

CASE REPORT:

A 14-year-old male patient from a remote locality of Palpa district, Nepal, presented to the emergency department with the complaints of pain in the right upper and central abdomen for three hours which was associated with one episode of non-bilious vomiting. The patient had passed stool and flatus regularly before presenting to the emergency department.

The patient denied prolonged intake of steroids or non-steroidal anti-inflammatory drugs (NSAIDS), consumption of alcohol or spicy food, prolonged fasting or any drug abuse. The patient had fasted for eight hours. There was no history of trauma or stress factors in recent past. He belonged to a middle-class family.

On general examination, the patient was looking ill with tachycardia and low volume pulse. There were signs of dehydration with the blood pressure of 90/60 mmHg. The abdomen was flat with tenderness over the epigastric and right upper quadrant. There was no organomegaly and per rectal examination was normal. With the initial diagnosis of peptic ulcer disease, the patient was investigated accordingly.

With the findings of raised leucocyte counts (18,000 cells/μL) and neutrophilia (80%) with pneumoperitoneum on chest x-ray(Fig.1), the final diagnosis was peritonitis due to peptic ulcer perforation. The patient was resuscitated with intravenous fluid, antibiotics and immediately planned for ex-

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Fig. 1. Chest X-ray showing gas under bilateral domes of diaphragm.

ploratory laparotomy in order to save patient critical conditions. Under general anesthesia, upper midline laparotomy was done which revealed flakes on the surface of the stomach and small intestine with minimal dirty fluid in the Morrison's pouch (Fig. 2). There was a perforation of about 0.4 cm x 0.4 cm at the prepyloric region, near the lesser curvature of the stomach. The perforation margin was refreshed, biopsy taken and closed in single layer with absorbable suture and reinforced with omentum (Modified Grahm's patch). After adequate peritoneal lavage and placement of a drain in the pelvis, the abdomen was closed. The post-operative period was uneventful, tubes and drains were removed subsequently and the patient was put on H. pylori eradication triple therapy.

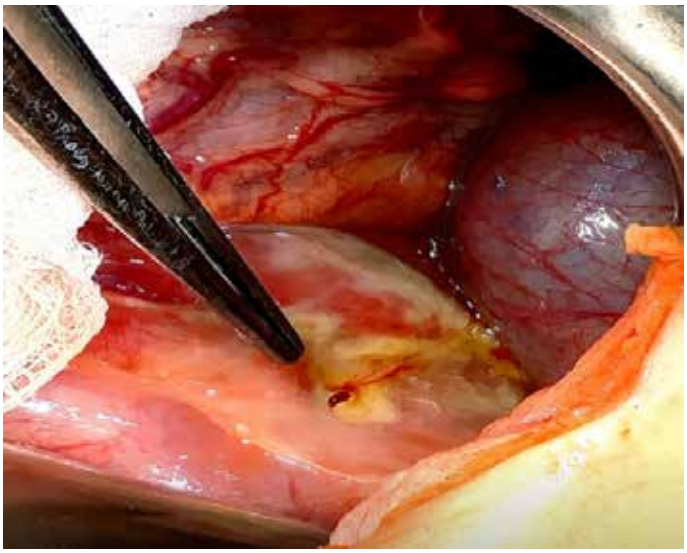

Fig. 2. Perforation at prepyloric region of stomach.

The patient was discharged on the seventh post-operative day in order to follow up in surgical out-patient department with histo-pathological and Helicobacter pylori (H. pylori) reports. He was also advised to undergo upper gastrointestinal endoscopy to look for healing of previous ulcer and any other ulcers at other sites.

On the twenty-first day of follow-up, histopathology report showed an ulcer from the antrum of stomach, acute suppurative lesion and no dysplasia/atypia and H. Pylori report negative.

DISCUSSION:

Gastric perforation is an uncommon but serious condition in both neonates and children. Among the most recognized causes in newborns are congenital defects in the stomach's muscular wall, which can result in high mortality rates ranging from 30% to 70%.[3] There is limited literature describing how gastric perforation presents in pediatric patients.

Several factors have been linked to poor outcomes in these cases. These include metabolic acidosis (pH <7.3), male gender, and hyponatremia (serum sodium <130 mEq/L).[1] In older children, H. pylori infection is frequently associated with peptic ulcers. However, the use of nonsteroidal anti-inflammatory drugs (NSAIDs) is also a significant contributor. Other medications such as steroids, chemotherapy agents, and immunosuppressants can weaken the gastric lining, increasing the risk of ulcers. Moreover, critically ill children exposed to stressful physiological states—including sepsis, shock, burns, major trauma, brain injury, surgery, or chronic illnesses—may also develop acute gastric ulcers.

In our patient, gastric perforation occurred spontaneously, with no clear cause identified. While H. pylori is a well-known culprit in many cases of spontaneous perforation, both Giemsa staining and H. pylori antigen testing were negative in this case, suggesting a noninfectious etiology.

A study by Xueqiang Yan et al., which reviewed 20 pediatric gastroduodenal perforation cases found that dexamethasone increased the risk for duodenal perforation, while H. pylori infection was a notable factor in gastric perforation. They also observed that the duodenal bulb and pyloric region were the most frequently affected areas.[2] In a 10-year review, Janet Abadir et al. noted that among children with foregut perforations, 50% were spontaneous, 25% were iatrogenic, and the remaining 25% were linked to underlying medical conditions.[4]

In neonates, the exact cause often remains unknown. Possible explanations include congenital muscle defects, mechanical injury, neurogenic stress ulcers, or ischemia due to disrupted blood supply.[5] One widely accepted theory involves pneumatic rupture from increased stomach pressure. In neonates with immature neurological control, poor coordination during vomiting can cause gastric over distension, leading to perforation.[6] Over a 20-year span, Man-Chin Hua et al. observed that nearly 81% of children with perforated peptic ul-

cers were boys.[7] These ulcers often appear as solitary lesions, typically located along the lesser curvature or near the antrum, and are believed to result from excessive gastric acid and pepsin production.[3]

In this case spontaneous gastric perforation near the lesser curvature which is similar to other publications, which similarly reports male predominance, typical presentation with acute abdominal pain, pneumoperitoneum, and common perforation sites in the prepyloric region.[2,7]

Unlike many cases linked to H. pylori infection or NSAID use, our patient was negative for H. pylori and had no identifiable risk factors, reflecting the less common idiopathic etiology documented in the literature.[3,4] The successful surgical repair using Modified Grahm's patch and uneventful recovery are consistent with favorable outcomes reported in older children, contrasting the higher mortality seen in neonates.[1,7] This study aligns with established knowledge and provides valuable data from a distinct geographic region.

CONCLUSION:

This case of spontaneous peptic ulcer perforation in a 14-year-old boy underscores the complexity and ambiguity of its etiopathology in pediatric patients. The absence of clear causative factors highlights the poorly understood mechanisms behind pediatric peptic ulcer perforations, suggesting possible roles for idiopathic ulceration, congenital muscular defects, ischemia, or stress-related mucosal injury. The case emphasizes the need for further investigation into noninfectious etiologies and the pathogenesis of spontaneous peptic ulcer perforations in children.

Conflict of Interest: None

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