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CASE REPORT | PEDIATRICS

Helicobacter pylori–Induced Gastroduodenal Stricture in a Child

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ABSTRACT

Helicobacter pylori is a known cause of peptic ulcers, but it has not been reported to cause strictures in children. We present the case of a previously healthy 12-year-old boy with sudden onset of abdominal pain and vomiting, positive stool *H. pylori* antigen testing, and esophagogastroduodenoscopy revealing a gastroduodenal stricture causing gastric outlet obstruction. Because of medically refractory disease, he ultimately required laparoscopic truncal vagotomy with open pyloroplasty. This is an unusually severe presentation and may warrant *H. pylori* being on the differential of pediatric gastrointestinal strictures as well as further discussion on other long-term implications.

KEYWORDS: *H. pylori*; gastroduodenal stricture; pediatric

INTRODUCTION

Helicobacter pylori, a Gram-negative bacterium, is often asymptomatic, but can directly and indirectly leads to host tissue damage.[1] Although *H. pylori* is known to cause gastritis, ulcers, and stomach cancer, strictures secondary to *H. pylori* have not been previously reported in children.[2]

Most reported strictures in both adults and children are esophageal. In adults, it is often due to gastroesophageal reflux disease.[3] In children, strictures are often due to caustic ingestion. Other etiologies include inflammatory bowel disease (IBD), eosinophilic esophagitis, or other infections including cytomegalovirus, herpes simplex, and human immunodeficiency virus.[3] Gastroduodenal strictures are far less reported, but the potential etiologies are similar.

Many children acquire *H. pylori* in childhood, and although the majority are asymptomatic, infection can result in gastroduodenal erosions and ulcers.[4] In adults, stricture formation has been only seen in recurrent ulcer disease, particularly from the pyloric and bulbar region; however, strictures typically resolve with resolution of the inflammation.[5] We present a pediatric case of a refractory *H. pylori*–induced gastroduodenal stricture, which ultimately required surgical correction.

CASE REPORT

Patient is a 12-year-old previously healthy boy who presented to an outside clinic with a 3-month history of abdominal pain, vomiting, and 15-pound weight loss. Initial stool *H. pylori* antigen (Ag) test was positive (specific test unknown). Patient was prescribed triple therapy (clarithromycin, amoxicillin, and a proton pump inhibitor) for 14 days but only completed 7 days because of worsening nausea. Subsequent repeat stool *H. pylori* monoclonal antigen testing was positive, and esophagogastroduodenoscopy showed nodular gastritis with severe antroduodenal mucosal edema without definite ulcer causing gastric outlet obstruction (Figures 1 and 2). Biopsies using immunohistochemistry stains showed peptic gastritis and duodenitis with no *H. pylori*, viral inclusions, dysplasia, or malignancy (Table 1). Colonoscopy was normal (Figures 3–5). Patient was retreated with quadruple therapy and started on postpyloric nasojejunal feedings for several months. Imaging studies (computed tomography, magnetic resonance enterography, and upper gastrointestinal series) were unremarkable. Ultimately, despite disease eradication by repeat

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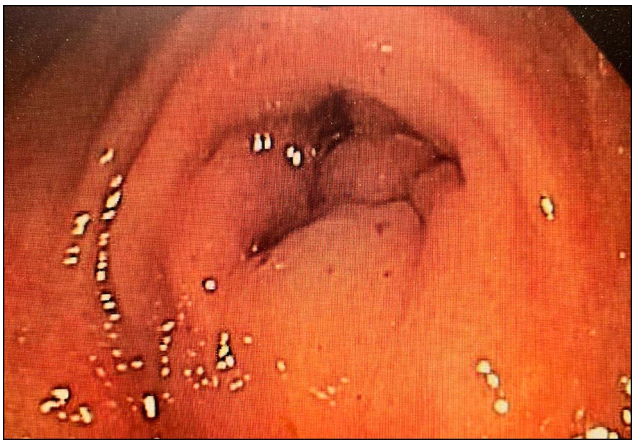

Figure 1. Significant gastroduodenal edema and inflammation causing near complete gastric outlet obstruction.

stool *H. pylori* monoclonal antigen testing, a laparoscopic truncal vagotomy with open pyloroplasty was required to relieve the gastric outlet obstruction (Figure 6). Since surgery, he has returned to baseline. Interestingly, patient's father had a similar history of sudden gastric outlet obstruction 10 years ago, which ultimately also required partial gastric resection in Mexico to resolve the issue.

DISCUSSION

H. pylori is a disease that is associated with gastric ulcers and cancers; however, it is rarely reported to cause strictures, and even in those rare adult cases, strictures tend to resolve with medication. As *H. pylori* infects up to 50% of the population worldwide, it is important to understand all possible complications, its potential pediatric risk, as well as speculate why some patient develop much more complicated disease.[1]

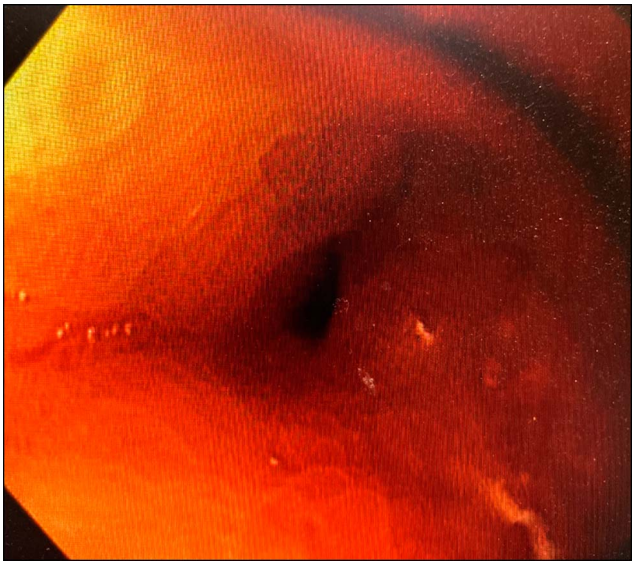

Figure 2. Close up of gastroduodenal stricture.

Table 1: Endoscopic biopsy results

A. Duodenum, bulb (biopsy)
• Peptic duodenitis
• No *Helicobacter pylori* (immunostain negative), dysplasia, or malignancy
B. Stomach, prepyloric tissue (Biopsy)
• Antral gastric mucosa/pyloric enteric tissue with mild chronic inflammation
• No *H. pylori*
C. Stomach, stomach body (biopsy)
• Oxyntic/antral gastric mucosa with minimal lamina propria chronic inflammation
• No *H. pylori*
D. Esophagus, distal esophagus (biopsy)
• Squamous mucosa with mild reactive changes and rare intraepithelial eosinophils (focally up to 3 eosinophils/high power field)
E. Esophagus, mid esophagus (biopsy)
• Squamous mucosa with rare intraepithelial eosinophils (focally up to 5 eosinophils/high power field)
F. Esophagus, proximal esophagus (biopsy)
• Squamous mucosa with no significant histopathologic changes
G. Ileum, terminal (biopsy)
• No significant abnormality
• No active ileitis or features of chronicity
H. Colon, right (biopsy)
• No significant abnormality
• No active colitis or features of chronicity
I. Colon, left (biopsy)
• No significant abnormality
• No active colitis or features of chronicity
J. Rectum (biopsy)
• No significant abnormality
• No active colitis or features of chronicity
Immunohistochemical stains
Cytomegalovirus–negative
Helicobacter-negative

Although the differential for strictures includes congenital lesions, IBD, other infections, and caustic injury, our patient was previously healthy with no previous gastrointestinal symptoms to suggest a congenital stricture. All tests for infection including cytomegalovirus, herpes simplex virus, and HIV were negative. There was no history of injury or caustic ingestion. Biopsies did not show evidence of IBD or eosinophilic esophagitis (Table 1). Thus, his stricture is consistent with *H. pylori*, which can be asymptomatic for years and only have symptoms once peptic ulcer disease has set in.[1] In addition, because *H. pylori* attacks the region between the stomach and the small intestines, the gastroduodenal location is characteristic.[6] Stool *H. pylori* monoclonal antibody testing in children is highly reliable with

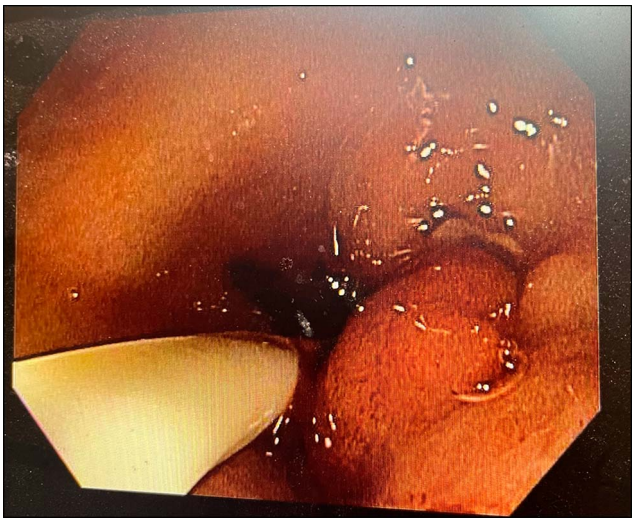

Figure 3. Persistent gastroduodenal inflammation several months later, despite eradication of *H. pylori* infection.

a specificity and sensitivity of 97%, which would make false positives unlikely.[7] As for the negative biopsies, we suspect it was due to a combination of partially treated disease as well as current proton pump inhibitor therapy, which has been associated with higher rates of false-negative testing.[8,9,11]

As there have not been previous reports of *H. pylori* strictures in children, it behooves us to question why our patient developed such severe complications. Similarly, father's seemingly identical history points to either both patients acquiring a more virulent strain of bacterium or potentially a shared genetic host predisposition to developing more severe disease. Host immune gene polymorphisms and gastric acid secretions play a huge role in determining the bacteria's ability to colonize a host's gastric

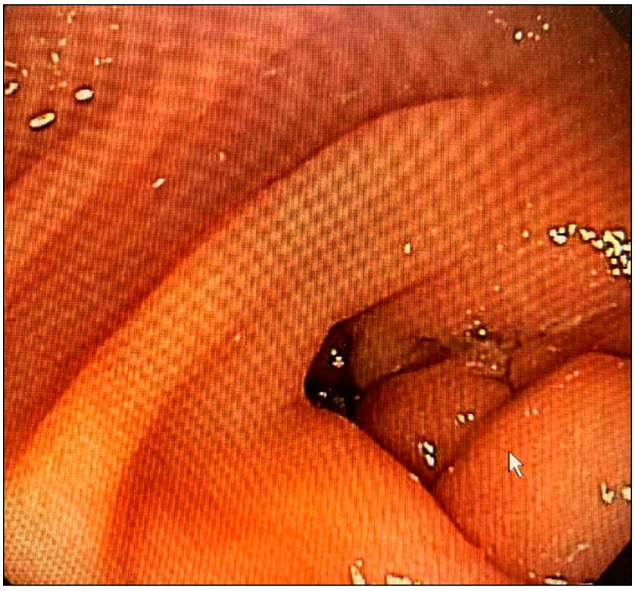

Figure 4. Persistent gastroduodenal inflammation several months later, despite eradication of *H. pylori* infection.

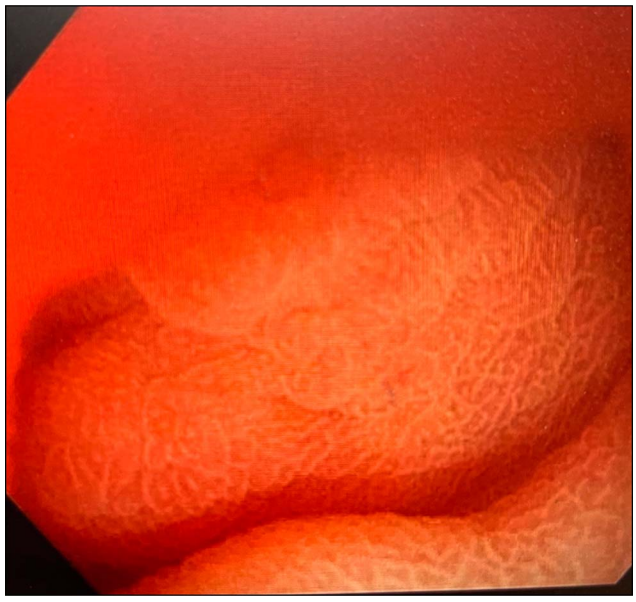

Figure 5. Close up of persistent gastroduodenal stricture several months later, despite eradication of *H. pylori* infection.

cells. Also, polymorphisms can affect the expression of various inflammatory mediators, which can then affect *H. pylori* infection risk. For example, mutations in CD14*-159T result in increased expression of CD14, which is associated with higher *H. pylori* infection rates.[5] In addition, bacterial virulence factors including cytotoxin-associated gene pathogenicity island-encoded proton CagA helps some strains invade and colonize gastric mucosa.[5] Thus, it is possible that a combination of the patient's genetic predisposition along with potentially a more virulent strain of *H. pylori* could have predisposed both patients to develop more severe stricturing disease. Unfortunately, genetic testing could not be completed for our patient because of financial constraints.

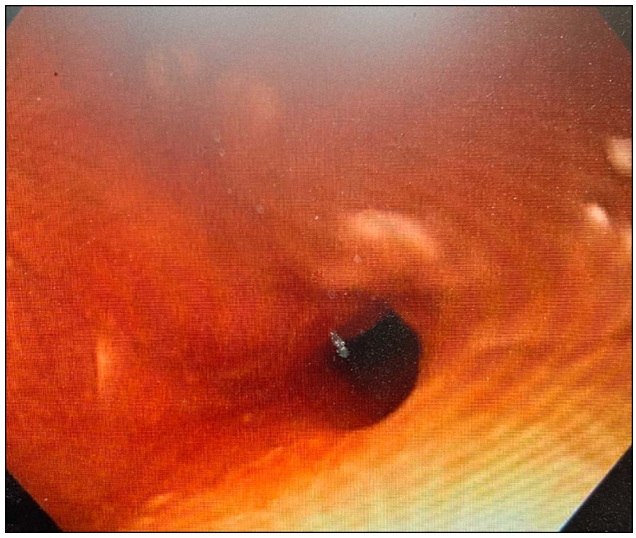

Figure 6. Nasojejunal tube endoscopically placed to relieve gastric outlet obstruction and for nutritional support.

This case offers an important presentation of stricturing *H. pylori* disease in a pediatric patient and shows the importance of not only the need for close follow-up but also allows us to further question our current standard of practice. In particular, with a family history of severe disease, should all family members be tested? If positive, is there a role for genetic testing as some polymorphisms have been linked to more severe disease?[5] Alternatively, should more testing be performed routinely on the bacterium itself as different strains of *H. pylori* have been associated with higher virulence patterns?[5] Last, in patients with severe disease, we may need to consider earlier cancer screening because *H. pylori* has been associated with the development of gastric carcinoma. In adults, 1%–3% of adults develop gastric adenocarcinoma with patients developing cancer up to 13 years after the infection has been cured.[10] This is believed to be due to *H. pylori*'s strains having different virulence factors which when combined with a patient's polymorphisms, lead to more severe disease and increase the likelihood of developing cancer.[2] Ultimately, if severe stricturing disease becomes more commonplace, we may need to consider readjusting our current standard of practice to include developing and performing more extensive host genetic and bacterial virulence testing as well as developing potentially earlier cancer screening protocols.

DISCLOSURES

Author contributions: All authors made substantial contributions to the design, interpretation, drafting and revising the work for important intellectual content, final approval of the version to be published, and agreement to be accountable in ensuring that questions related to the accuracy or integrity of the work are investigated and resolved. T. Truong is the article guarantor.

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