

# Pediatric Gastritis, Gastropathy, and Peptic Ulcer Disease

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## Education Gap

Providers should be able to identify the signs and symptoms of peptic ulcer disease in children, select the proper diagnostic tests needed to confirm the diagnosis, and describe adequate treatment regimens to start once the diagnosis is confirmed.

## Objectives After completing this article, readers should be able to:

1. Understand the mechanism and pathogenesis of gastritis, gastropathy, and peptic ulcer formation.
2. Recognize the symptoms of and risk factors for gastritis, gastropathy, and peptic ulcer disease.
3. Understand which patients warrant referral to a gastroenterologist for possible endoscopy.
4. Review the role of *Helicobacter pylori* in peptic ulcer disease.
5. Review the treatment of peptic ulcer disease with and without the presence of *H pylori*.

## INTRODUCTION

Gastritis, gastropathy, and peptic ulcer disease (PUD), collectively known as acid peptic disease, are often described as a spectrum of the same disease. Although these conditions are more common in adults, their incidence in the pediatric population is clinically significant. Left untreated, gastritis can progress to PUD, which can result in serious complications such as perforation, bleeding, bowel strictures, and obstruction. Studies describing the role of *Helicobacter pylori*, nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, and other causes have helped to further expand our understanding of gastritis and PUD in children. Direct visualization and the ability to biopsy with endoscopy have revolutionized the diagnosis and treatment of these diseases.

## EPIDEMIOLOGY

The incidence of PUD in children is lower than that in adults. Based on several international studies, the incidence in children varies from 2% to 8%. (1)(2) The

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## ABBREVIATIONS

H2RA	histamine 2 receptor antagonist
IBD	inflammatory bowel disease
NSAID	nonsteroidal anti-inflammatory drug
PPI	proton pump inhibitor
PUD	peptic ulcer disease

incidence of bleeding ulcers is lower and is estimated to be between 0.5 and 4.4 of 100,000 individuals. (3) These rates are increased with the use of NSAIDs, corticosteroids, and immunosuppressive medications. The prevalence is similar between both sexes, with the incidence of PUD increasing after age 10 years. (4) Racial and ethnic differences have been noted in the pediatric population, with non-Hispanic black and Mexican American populations being more affected than white populations. (5)

## PATHOGENESIS

The intestinal mucosa is composed of 3 layers: epithelium, lamina propria, and muscularis mucosa. Gastritis is described as the presence of inflammatory cells. Gastropathy occurs when there is gastric mucosal damage with no inflammatory cells. (2) Peptic ulcers occur when gastric or duodenal inflammation leads to defects of the muscularis mucosa. The acidic gastric contents, which normally aid in digestion, become corrosive when there is an increase in acid production or a disruption of protective factors. Parietal cells of the stomach produce gastric acid via proton pumps ( $H^+/K^+$  ATPase) in response to acetylcholine from vagal efferents, histamine from enterochromaffin cells, and gastrin from G cells.

There are several mechanisms to protect the gastric mucosa, including a mucus layer, a pH-neutral buffer zone, an epithelial layer, and a rich gastric blood supply. The mucus layer is composed of mucin secreted by surface foveolar cells. This mucus layer acts as a diffusion barrier and overlies a pH-neutral buffer zone composed of bicarbonate secreted by epithelial cells. Prostaglandin release mediates mucin secretion from surface foveolar cells and bicarbonate release from epithelial cells. Epithelial cells have tight junctions that act as an additional barrier of protection. A rich gastric blood supply redistributes excess protons that reach the lamina propria. Ultimately, peptic ulcers are formed when the damaging factors overcome the protective mechanisms. (6)

## PRESENTATION AND INITIAL EVALUATION

Symptoms of acid peptic disease vary by the age of the patient. Younger patients can present with irritability, poor feeding, regurgitation, vomiting, gastrointestinal bleeding, or poor weight gain. Older patients can present with dull or diffuse abdominal pain, gas, bloating, nausea, or nocturnal awakenings from abdominal discomfort. It is nearly impossible to differentiate between gastric and duodenal ulcers based on history alone. However, gastric ulcers classically

present with epigastric gnawing or burning immediately after meals and duodenal ulcers present with pain 2 to 3 hours after meals. Severe presentations of PUD can include intestinal perforation, severe gastrointestinal bleeding with erosion of vasculature, or pancreatitis with posterior intestinal erosion. The differential diagnosis for epigastric and periumbilical abdominal pain extends beyond acid peptic disease. Clinicians must also consider and rule out pancreatitis, appendicitis, celiac disease, lactose intolerance, inflammatory bowel disease (IBD), and parasitic infections. (6)

The initial evaluation of a patient with suspected acid peptic disease should begin with a thorough history. Special attention must be given to the onset and duration of symptoms and alleviating and exacerbating factors. Evaluation of medical history for conditions that predispose to PUD should be noted, including an assessment of medication use. Social and dietary history will also provide clues to predisposing factors. Family history, especially that of *H pylori* or gastritis, is crucial to ascertain.

## ETIOLOGIES

The common causes of peptic ulcers include *H pylori*, medication use, and stress-related gastric injury. Less common causes include ingestion of corrosive substances, hypersecretory states (Zollinger-Ellison syndrome), IBD, systemic mastocytosis, chronic renal failure, and hyperparathyroidism. (6)

### Infectious

There are many infectious causes for acid peptic disease. The most common bacterial cause is *H pylori*. Less common infectious etiologies include viruses (cytomegalovirus, Epstein-Barr virus), fungi (*Candida albicans*, histoplasmosis, and *Cryptosporidium*), and parasites (*Giardia lamblia*, ascariasis).

### Medications

High-dose corticosteroids and NSAIDs disrupt prostaglandin production, leading to decreased bicarbonate and mucin production, compromising mucosal protection. Less commonly, gastritis has been identified with the use of valproic acid, chemotherapeutic agents, and iron supplementation.

### Stress and Trauma

Stress-related gastric injury occurs in patients with severe physiologic stress due to trauma, burns, intracranial disease, major surgery, or serious medical disease. The pathogenesis of stress-related gastric injury is most likely related to local ischemia from splanchnic vasoconstriction or systemic hypotension. Local ischemia disrupts the redistribution of protons, leading to a more acidic milieu that is more

prone to ulcer formation. Intracranial disease can cause direct stimulation of vagal nuclei, resulting in acid hypersecretion. Physical trauma from compression of gastric mucosa at the level of a hiatal hernia is the cause of a specific type of ulcer known as Cameron ulceration. Other forms of physical trauma include ingestion of corrosive substances, such as strong acids and bases, that directly damage mucosal cells.

### Endocrine

Excess gastrin and histamine lead to acid hypersecretion and consequent ulcer formation. (6) Zollinger-Ellison syndrome results from the uncontrolled release of gastrin by a tumor. Inflammatory bowel disease results in increased levels of gastrin and histamine. (7)(8) In systemic mastocytosis, there is an excessive production of histamine. In chronic renal disease and hyperparathyroidism, hypercalcemia stimulates gastrin production.

### PHYSICAL EXAMINATION

The initial step of examination includes evaluation of vital signs, including weight and height. It is important to determine appropriate growth trajectory. The Z-scores, standard deviations from the mean, for weight-for-height and/or BMI should be calculated. If the patient has a poor growth trajectory or Z-scores less than  $-1$ , this might indicate the presence of chronic disease or poor nutrition. There are no focal findings specific for acid peptic disease. Examination of the oropharynx should include evaluation of dentition for caries and eroded enamel that may indicate frequent vomiting or reflux. Pale conjunctiva, tachycardia, or flow murmur may indicate anemia associated with chronic disease or blood loss. Halitosis with regurgitation or dysphagia may indicate achalasia. Examination of the lungs may reveal wheezing, which can be seen from bronchospasm-associated chronic reflux.

Abdominal examination should focus on determining areas of tenderness. Hepatomegaly and splenomegaly should also be ruled out. Rectal examination should determine the presence of fissures or rectal skin tags, which may be seen in Crohn disease. Fecal occult blood may also be useful for determining the presence of a gastrointestinal bleed. Potential extraintestinal manifestations of IBD should be noted on physical examination, including joint swelling, skin rashes, oral lesions, and eye abnormalities.

### LABORATORY STUDIES

Initial laboratory examination should be tailored to the patient's presenting symptoms. A complete blood cell count

may show leukocytosis or thrombocytosis, which can provide information about systemic inflammation. The presence of anemia can indicate blood loss or chronic disease. An electrolyte panel may show hypercalcemia, hypophosphatemia, and hyperchloremic acidosis, which are characteristic of hyperparathyroidism. A low albumin level may indicate poor nutritional status or decreased production related to chronic inflammatory states. An elevation in transaminases may point to viral etiologies. Inflammatory markers (ie. erythrocyte sedimentation rate and C-reactive protein) are also helpful, but these may not be specific. Infectious sources of symptoms should be ruled out, especially in immunocompromised patients. For example, cytomegalovirus may be identified by use of serum or urinary polymerase chain reaction. Stool studies, including ova and parasites, cultures, and polymerase chain reaction, may identify other etiologies.

### ROLE OF UPPER ENDOSCOPY

Certain signs and symptoms associated with acid peptic disease are an indication for gastroenterology referral for possible endoscopic evaluation (Table 1). Upper endoscopy has been well described as a superior diagnostic study for PUD compared with radiography. (9) It is important to note that not all patients with abdominal pain require upper endoscopy. The decision to proceed with endoscopy should be made only if the outcome of the test will change current patient management. An assessment of risks and benefits of endoscopy should be discussed with the patient and family.

Endoscopy is the diagnostic test of choice when IBD is suspected. The gastroenterologist may be able to grossly identify aphthous ulcers and other luminal manifestations of Crohn disease, ulcerative colitis, and indeterminate colitis.

### TREATMENT

Initial treatment of acid peptic disease includes discontinuation of offending agents and treatment of underlying

TABLE 1. Indications for Upper Endoscopy

Refractory anemia	Upper gastrointestinal bleeding
Refusal to eat	Odynophagia
Dysphagia	Constitutional symptoms
Pain despite acid suppressant therapy	Intractable vomiting

etiologies. Acid suppression is an effective strategy to alleviate symptoms and promote ulcer healing. Available agents on the market include histamine 2 receptor antagonists (H<sub>2</sub>RAs) and proton pump inhibitors (PPIs). Commonly used drugs and their doses are listed in Table 2. The H<sub>2</sub>RAs are associated with tachyphylaxis and rebound symptoms. (10) For this reason, some clinicians use PPIs if prolonged therapy is needed. There is mixed evidence regarding ideal dosing of PPIs in children and adolescents.

The mechanism of action of H<sub>2</sub>RAs is reversible and competitive inhibition of H<sub>2</sub> receptors on gastric parietal cells. Two commonly used agents are famotidine and

ranitidine. A third agent, cimetidine, is less recommended because it can inhibit cytochrome P<sub>450</sub> enzymes, leading to decreased metabolism of other drugs. Minor adverse effects include diarrhea, headache, and fatigue. (11)

The PPIs are prodrugs that are activated in an acidic environment and subsequently bind irreversibly to the H<sup>+</sup>/K<sup>+</sup> ATPase. Available agents include omeprazole, esomeprazole, pantoprazole, and lansoprazole. The PPIs are preferred over the H<sub>2</sub>RAs for the treatment of PUD and NSAID-induced ulcers. Generally these drugs are well tolerated. Adverse effects include headache, nausea, abdominal pain, and altered bowel habits. (11) There are studies

TABLE 2. Acid Suppressant Drugs and Their Doses

DRUG	TYPE	GENERAL DOSING	DOSE FOR ULCERS	DOSAGE FORMS (ORAL)
Ranitidine	H <sub>2</sub> RA	5–10 mg/kg per day divided twice daily; if age >16 y can use 150 mg BID; maximum dose is 300 mg/d for GERD and 600 mg/d for erosive esophagitis	4–8 mg/kg per day divided twice daily; if age >16 y can use 150 mg BID; maximum 300 mg/d	15 mg/mL of syrup 75-mg tablet 150-mg tablet or capsule 300-mg tablet or capsule
Famotidine	H <sub>2</sub> RA	0.5–1 mg/kg per day divided twice daily (infants aged 1–3 mo use only daily); if age >17 y may use 20–40 mg BID (higher dose for worse symptoms)	0.5–1 mg/kg per day divided twice daily; if age >17 y can use 20 mg BID	40-mg/5-mL suspension 10-mg tablet 20-mg tablet 40-mg tablet
Omeprazole	PPI	5 to <10 kg: 5 mg once daily 10 to <20 kg: 10 mg once daily ≥20 kg: 20 mg once daily	No specific dosing guidelines for PUD in pediatric patients	2-mg/mL suspension 10-mg capsule 20-mg tablet or capsule 40-mg capsule 2.5-mg packet 10-mg packet
Lansoprazole	PPI	Infants: 1–2 mg/kg per day Children: 0.7–3.0 mg/kg per day Fixed dosing: Age ≥3 mo: 7.5 mg BID Age 1–11 y: 15 mg/d if ≤30 kg; 30 mg/d if >30 kg Age ≥12 y: 15 mg/d	No specific dosing guidelines for PUD in pediatric patients	3-mg/mL suspension 15-mg capsule or disintegrating tablet 30-mg capsule or disintegrating tablet
Pantoprazole	PPI	Children aged 1–5 y: 0.3, 0.6, 1.2 mg/kg per day Children aged ≥5 y: ≥15 to <40 kg: 20 mg once daily for up to 8 wk ≥40 kg: 40 mg once daily for up to 8 wk	No specific dosing guidelines for PUD in pediatric patients	20-mg tablet 40-mg tablet or packet
Esomeprazole	PPI	Infants to children aged <12 y: 3–5 kg: 2.5 mg once daily >5–7.5 kg: 5 mg once daily >7.5–20 kg: 10 mg once daily >20 kg: 10 or 20 mg once daily Patients aged ≥12 y: 20–40 mg once daily	No specific dosing guidelines for PUD in pediatric patients	2.5-, 5-, 10-, 20-, or 40-mg oral packet 20- or 40-mg delayed-release capsule 20-mg delayed-release tablet

BID=twice daily, GERD=gastroesophageal reflux disease, H<sub>2</sub>RA=histamine 2 receptor antagonist, PPI=proton pump inhibitor, PUD=peptic ulcer disease.

that associate the use of PPIs with increased incidence of fractures, (12) *Clostridium difficile*, (13)(14) and hospital-acquired pneumonia in the adult population. The association with pneumonia, however, is thought to be in part due to confounding factors. (15)

Sucralfate is a sucrose sulfate and aluminum hydroxide salt that creates a gel over the mucosal surface that protects from acid injury. It has little systemic absorption and, therefore, few adverse effects. Sucralfate should be used with caution in patients with renal failure due to risk of aluminum toxicity. Another coating agent is bismuth. It is used frequently as part of *H pylori* infection eradication therapy.

A less used drug in the pediatric population is misoprostol, a prostaglandin E analogue that can be used to prevent and treat NSAID-induced ulcers. One common reported adverse effect is diarrhea. (11)

In the critically ill population, stress ulcer prophylaxis is widely used. Although the available evidence is rated low quality, the 2016 Surviving Sepsis campaign recommends use of either PPIs or H<sub>2</sub>RAs in patients with a high risk of bleeding, such as patients who require mechanical ventilation for more than 48 hours and those with coagulopathy. (16)

Due to advances in the understanding of the pathophysiology of PUD, medical therapy is now the mainstay of management. Today, surgical procedures to treat PUD (eg, selective vagotomy) are rarely performed. The role of surgical treatment is primarily in the management of complications from PUD, such as gastrointestinal bleeding and perforation.

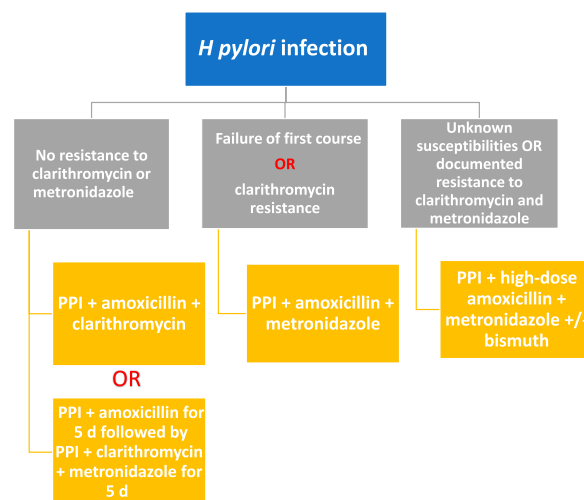
Some clinicians may recommend dietary restriction of irritant foods such as spicy foods, caffeine, and fried foods because it may allow the gastric and duodenal mucosa to heal faster.

## H PYLORI INFECTION

*H pylori* is a gram-negative bacillus described microscopically as spiral- or U-shaped. The prevalence increases with age. By age 18 years, 25% of patients had *H pylori*. (17) The bacteria is transmitted via the fecal-to-oral or oral-to-oral route. Most people are asymptomatic from infection, but the bacteria are also implicated as the cause for some gastric and duodenal ulcers as well as gastric adenocarcinoma and lymphomas. *H pylori* organisms have certain features that contribute to their virulence, including flagella, urease, adhesins, and toxin production. These features allow the organism to overcome the mucosal defenses and cause chronic gastritis, which can eventually lead to ulcer

formation. Recently updated guidelines from the 2016 joint European Society for Paediatric Gastroenterology, Hepatology, and Nutrition/North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition discuss testing for *H pylori*. Testing should be directed toward investigation of symptoms and elimination of other causes rather than specifically for diagnosing infection with *H pylori*. This is because patients with functional disease such as irritable bowel syndrome may be chronic carriers of *H pylori* and its treatment in these patients may not necessarily alleviate symptoms. The use of endoscopy for the diagnosis of *H pylori* remains the gold standard of care. At least 6 specimens from antrum and body should be collected for diagnosis. A positive culture alone is sufficient to confirm *H pylori* infection given that its specificity is 100%. Alternatively, a combination of *H pylori*-associated histopathologic findings plus a rapid urease test or a molecular-based test such as polymerase chain reaction or fluorescent in situ hybridization can be used. (18) Noninvasive testing such as urea breath testing and stool antigen testing are recommended for posttreatment testing only. Serology testing is not recommended as part of the evaluation. It is important to note that the use of PPIs and antibiotics can affect the results of *H pylori* testing. (18)

In patients with *H pylori*-associated PUD, eradication therapy should be selected based on susceptibility testing (Fig 1). The guidelines recommend 14 days of therapy and provide standard dosing per weight range (Fig 2). Standardization of dosing is important to ensure eradication.



**Figure 1.** Susceptibility-based treatment for *Helicobacter pylori*. (Adapted from Jones NL, Koletzko S, Goodman K, et al; ESPGHAN, NASPGHAN. Joint ESPGHAN/NASPGHAN guidelines for the management of *Helicobacter pylori* in children and adolescents (update 2016). *J Pediatr Gastroenterol Nutr.* 2017;64(6):991–1003.) PPI=proton pump inhibitor.

PPI	<ul style="list-style-type: none"> <li>• 15 – 24 kg give 20 mg BID</li> <li>• 25 – 34 kg give 30 mg BID</li> <li>• ≥35 kg give 40 mg BID</li> </ul>
Amoxicillin	<ul style="list-style-type: none"> <li>• 15 – 24 kg give 500 mg BID</li> <li>• 25 – 34 kg give 750 mg BID</li> <li>• ≥35 kg give 1,000 mg BID</li> </ul>
Clarithromycin	<ul style="list-style-type: none"> <li>• 15 – 24 kg give 250 mg BID</li> <li>• 25 – 34 kg give 500 mg in am and 250 mg in pm</li> <li>• ≥ 35 kg give 500 mg BID</li> </ul>
Metronidazole	<ul style="list-style-type: none"> <li>• 15 – 24 kg give 250 mg BID</li> <li>• 25 – 34 kg give 500 mg in am and 250 mg in pm</li> <li>• ≥35 kg give 500 mg BID</li> </ul>

Figure 2. Weight-based doses for *Helicobacter pylori* treatment. BID=twice daily, PPI=proton pump inhibitor.

Weight-based PPI dosing in younger children is higher than in adolescents and adults due to more rapid metabolism. (18) With appropriate treatment, approximately 80% of patients experience resolution of gastric and duodenal ulcers within 4 to 8 weeks. (6)

#### COMPLICATIONS AND PROGNOSIS

In the pediatric population, PUD could have rare complications such as perforation, hemorrhage, or gastric outlet obstruction. Many patients may present with hematemesis,

melena, or symptoms of anemia such as fatigue or syncope. Upper gastrointestinal bleeding is the most common cause of death and the most common indication for surgery. (19) Despite these complications, most patients receiving appropriate treatment will have resolution of gastric and duodenal ulcers within 4 to 8 weeks. (6)

#### Summary

- The pathophysiology of acid peptic disease relates to an imbalance of acid production and mucosal protective factors.
- The mainstay of treatment of peptic ulcer disease focuses on the use of histamine 2 receptor antagonists and proton pump inhibitors (PPIs).
- Based on some research evidence and consensus guidelines, *Helicobacter pylori* should be treated with a combination of a PPI and antibiotics.
- Based on grade A evidence, initial diagnosis of *H pylori* should be made with positive culture or histopathologic analysis of biopsy samples obtained via endoscopy.

References for this article are at <http://pedsinreview.aappublications.org/content/39/11/542>.

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1. A 9-year-old girl with persistent abdominal pain was seen multiple times in the clinic with no success in managing her symptoms. She is ultimately referred to a pediatric gastroenterologist for further evaluation. She undergoes an upper endoscopy. The pathology report notes the presence of inflammatory cells and defects in the muscularis mucosa. Based on the pathology report, which of the following is the most likely diagnosis in this patient?
  - A. Celiac disease.
  - B. Gastritis.
  - C. Gastropathy.
  - D. *Helicobacter pylori* infection.
  - E. Peptic ulcer.
2. A 10-year-old boy is seen in the clinic because of diffuse abdominal pain, bloating, and pain immediately after eating for the past 6 to 8 weeks. He wakes up from sleep several times per week with abdominal pain. His mother states that he holds his upper abdomen while he is helping clean up in the kitchen after the meal. He has no history of fever, vomiting, diarrhea, or weight loss. He takes no medications and eats a regular diet. Which of the following is the most likely diagnosis in this patient?
  - A. Appendicitis.
  - B. Gastric ulcer.
  - C. Gastroesophageal reflux.
  - D. Giardiasis.
  - E. Pancreatitis.
3. A 9-month-old boy is brought to the clinic by his parents for evaluation of poor feeding. The parents report that the patient has not been feeding well and seems to have lost some weight. He is very irritable and has been noted to have intermittent vomiting for the past several days. An electrolyte panel shows hypercalcemia, hypophosphatemia, and hyperchloremic acidosis. Which of the following conditions is this presentation most characteristic of?
  - A. Achalasia.
  - B. Hyperparathyroidism.
  - C. Inflammatory bowel disease.
  - D. Intestinal perforation.
  - E. Pyloric stenosis.
4. A 6-year-old girl with mild cerebral palsy was seen 3 months earlier in the clinic because of a new-onset abdominal pain associated with meals. At that time, the patient had a normal complete metabolic panel, complete blood cell count, and urinalysis. Urine culture was negative. She was started on a trial of omeprazole for presumed gastritis. She continued receiving occupational and physical therapy and is progressing nicely. Today she returns to the clinic for follow-up. The parents report that after a short period of improved symptoms, her symptoms recurred, and she complains of abdominal pain and is refusing to eat. Laboratory studies are within normal limits except for anemia on her complete blood count. There is no history of vomiting, diarrhea, or hematochezia. Which of the following tests is most likely to confirm the diagnosis in this patient?
  - A. Abdominal ultrasonography.
  - B. Serologic testing for *H pylori*.
  - C. Stool antigen testing for *Giardia*.
  - D. Upper endoscopy.
  - E. Urea breath testing.

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5. A 5-year-old girl is brought to the clinic with a history of diffuse abdominal pain that increases several hours after meals for the past few weeks. Her symptoms began around the time she started child care. She was previously diagnosed as having irritable bowel syndrome and was placed on a high-fiber diet. There is no history of vomiting, diarrhea, or constipation. Results of laboratory studies, including complete blood cell count, complete metabolic panel, and urinalysis, are normal. Urine culture is negative. The parents are concerned about peptic ulcer disease associated with *H pylori* because of a positive family history of *H pylori*. Which of the following diagnostic tests will most likely help confirm the diagnosis of *H pylori* in this patient?
- A. pH monitoring.
  - B. Serology testing for *H pylori*.
  - C. Stool antigen testing for *H pylori*.
  - D. Upper endoscopy with biopsies for *H pylori* culture.
  - E. Urea breath testing.



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