

# A 30-year-old Man with Syncope and Melena

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**A** 30-year-old man with a past medical history of asthma presented to the Emergency Department with a chief complaint of weakness. He described multiple episodes of light-headedness and diaphoresis, as well as several fainting spells over the previous 3 days. He reported a burning sensation in his epigastric region, a decrease in appetite, and recent development of loose bowel movements that were black. He denied vomiting or bright red blood in his stool. The patient had recently been hospitalized for an asthma exacerbation during which time he received a course of antibiotics and oral corticosteroids. He had had no prior episodes of weakness, abnormal bowel movements, weight loss, chronic abdominal pain or dyspepsia.

The patient had no other significant medical or surgical history. His medications included inhaled corticosteroids, a long-acting metered dose inhaled beta-agonist, an oral leukotriene inhibitor, and a short-acting beta-

agonist rescue inhaler. The patient reported an allergy to aspirin and non-steroidal anti-inflammatory drugs. His social history was significant for remote intranasal cocaine use prior to incarceration approximately 3 months earlier. He denied any tobacco or alcohol use.

Vital signs upon presentation to the Emergency Department included a temperature of 96.8°F, pulse of 121 beats per minute, respiratory rate of 24/minute, and a blood pressure of 117/60 mmHg. On physical examination, the patient appeared to be in mild discomfort. His conjunctiva and other mucus membranes were pale. There was no significant lymphadenopathy. Cardiovascular exam revealed a regular rapid rhythm without murmurs or gallops. The patient's lungs were clear to auscultation bilaterally with good airway movement. Abdominal exam was remarkable for mild tenderness with deep palpation in the epigastric region, but no guarding or rebound tenderness. There was no palpable mass or organomegaly, and normoactive bowel

### CME INFORMATION

#### TARGET AUDIENCE

The May/June Clinical Case of the Month is intended for primary care physicians, general internists, surgeons, and gastroenterologists.

#### EDUCATIONAL OBJECTIVES

After reading the article, the healthcare provider should be able to discuss the epidemiology, clinical manifestations, diagnosis, and treatment of *H.pylori* infection.

#### CREDIT

The LSMS Educational and Research Foundation designates this educational activity for a maximum of one (1) AMA PRA Category 1™ Credit. Physicians should only

claim credit commensurate with the extent of their participation in the activity.

#### DISCLOSURE

Dr. Fontenot has nothing to disclose.  
Dr. Salvatierra has nothing to disclose.  
Dr. Morris has nothing to disclose.  
Dr. Johnson has nothing to disclose.  
Dr. Lopez discloses that he is a member of the *Journal* Board of Trustees and the *Journal* Editorial Board.

ORIGINAL RELEASE DATE  
06/30/06

EXPIRATION DATE  
06/30/2007

Estimated time to complete this activity is 1 hour.

sounds were heard. Rectal exam was significant only for grossly heme-positive stool. The patient had palpable peripheral pulses that were equal in all extremities, and the neurologic exam revealed no abnormalities.

Initial laboratory evaluation demonstrated a hemoglobin of 7.0 gm/dL (normal range, 13.5-17.5), hematocrit of 21.2% (normal range, 40-51), platelet count of  $326 \times 10^3/\mu\text{L}$  (normal range,  $130-400 \times 10^3$ ), mean corpuscular volume of 94 FL (normal range, 80-100), and a red cell distribution width of 12.2 (normal range, 11.5-14.5). Serum chemistries revealed a sodium of 132 mmol/L (normal range, 135-146), potassium of 4.9 mmol/L (normal range, 3.6-5.2), chloride of 104 mmol/L (normal range, 96-110), bicarbonate of 24 mmol/L (normal range, 24-32), blood urea nitrogen of 34 mg/dL (normal range, 7-25), creatinine of 1.2 mg/dL (normal range, 0.8-1.5), calcium of 9.4 mg/dL (normal range, 8.4-10.3), and a non-fasting glucose of 163 mg/dL (normal less than 100). Based on the above, a diagnosis of bleeding duodenal ulcer secondary to *H. pylori* infection was made.

The patient was admitted to the Intensive Care Unit with a diagnosis of gastrointestinal bleeding presumed to be originating from the upper tract. The patient was started on an intravenous proton pump inhibitor by continuous infusion, and volume resuscitation was begun with normal saline and 2 units of packed red blood cells. The patient underwent urgent endoscopy. The findings on esophagogastroduodenoscopy were remarkable for 2 large ulcers in the first segment of the duodenum, one with a clean base, the second with an adherent clot, which was treated with clerothrapy and bipolar coagulation (Figure 1). A rapid urease test (i.e., CLO test) done in the endoscopy suite was positive. The remainder of the endoscopy exam was normal. A serum gastrin level was found to be at the upper limit of the normal range. The patient remained stable and food was started on day 3 after endoscopy. The patient was discharged after 7 days with outpatient follow-up in the gastroenterology clinic.

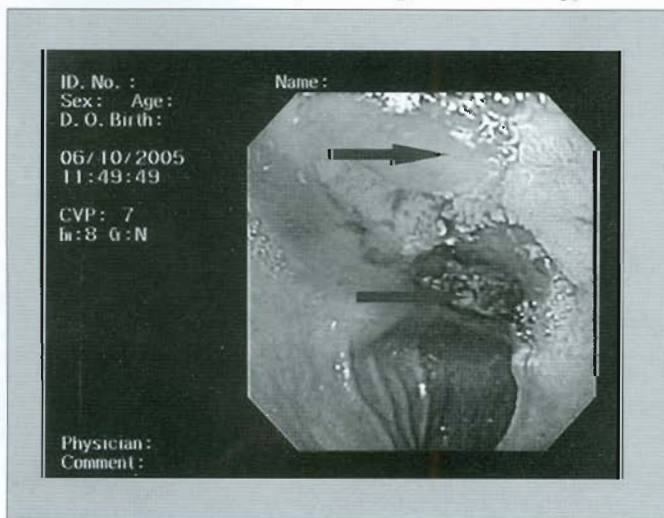


Figure 1. Endoscopic appearance of two duodenal ulcers with adherent clot on distal ulcer. Both ulcers are denoted by arrows.

## HELICOBACTER PYLORI AND PEPTIC ULCER DISEASE

### DISCUSSION

It has been known for over 100 years that microorganisms can exist in the stomach. However, it was not until the discovery of *H. pylori* in 1982 that the contributions of gastric organisms to pathologic states were appreciated. The organism was originally described by Marshall and Warren after biopsies and cultures were done on 100 consecutive patients presenting for gastroscopy.<sup>1</sup> They discovered a Gram-negative microaerophilic bacterium that was originally called *Campylobacter pyloridis*. The name was later changed to *Helicobacter pylori*. It is now known that *H. pylori* is associated with most cases of gastritis, peptic ulcer disease, and certain cases of gastric adenocarcinoma and lymphoma.

### BACTERIOLOGY AND EPIDEMIOLOGY

*Helicobacter pylori* possesses structural and functional characteristics that enable it to cause infection. The mode of transmission, although not fully understood, is believed to be through oral-oral and fecal-oral routes.<sup>2</sup> Once inside the gastric lumen, *H. pylori* is able to cause long-term infection. The organism initially creates an environment conducive to its survival through the use of an enzyme called urease. Urease hydrolyzes gastric luminal urea to form ammonia and bicarbonate which neutralize the environment and form a protective cloud around the organism.<sup>3</sup> Once in the mucus layer, the organism uses its flagella to move towards epithelial cells. Attachment to the epithelial cells is accomplished by adhesins found on the organism that are specific for certain receptors on the cell surface.<sup>4</sup>

*Helicobacter pylori* infection occurs worldwide and is the most common chronic bacterial infection in humans. In the United States, serologic testing for *H. pylori* rarely reveals infection before 10 years of age. However, by age 60 years, testing reveals infection in approximately 50% of the population. Risk factors for development of infection include African American and Hispanic ethnicity as well as low socioeconomic status. Living conditions such as overcrowding, lack of running water, and sharing a bed are also associated with an increased incidence of infection.<sup>2</sup>

### CLINICAL MANIFESTATIONS

Infection with *H. pylori* can cause acute and chronic gastritis. Acute gastritis has been demonstrated in healthy volunteers after ingesting the organism. The acute infection is characterized by neutrophil infiltration of the mucosa and underlying epithelium. The endoscopic appearance is variable and may resemble lymphoma or

carcinoma in severe cases. Acute infection evolves into chronic gastritis unless appropriate antibiotics are administered. Chronic gastritis is characterized by the presence of mononuclear cells, chiefly lymphocytes, plasma cells, macrophages, and scattered eosinophils.<sup>5</sup> Lymphoid follicles can be seen with chronic infection and are virtually pathognomonic.<sup>6</sup>

*Helicobacter pylori* infection is associated with greater than 60% of gastric ulcers and greater than 80% of duodenal ulcers (DU).<sup>7</sup> In two separate meta-analyses, eradication of *H. pylori* has been demonstrated to decrease the rate of ulcer recurrence to less than 20% compared to approximately 60% for controls.<sup>8,9</sup> Several mechanisms have been implicated in ulcer formation, including increased gastric acid secretion, gastric metaplasia, immune response, and abnormal mucosal defense mechanisms.

Increased acid secretion appears to be mediated by increased gastrin release and impaired somatostatin secretion. Gastrin leads to increased acid output by trophic effects on parietal cells and enterochromaffin-like cells which secrete histamine. This leads to enhanced stimulation of parietal cells and subsequent increased basal and maximal acid output. Eradication of *H. pylori* has been shown to decrease basal and stimulated acid output by 50% at one month and to normal levels by 1 year.<sup>10</sup>

Gastric metaplasia, which refers to the presence of gastric epithelium in the first part of the duodenum, likely occurs in response to increased acidity in the duodenum, usually with an intra-luminal pH of less than 2.5. In addition to acid hyper-secretion, impaired duodenal secretion of bicarbonate may also contribute to the decreased pH. The metaplasia provides a favorable environment for *H. pylori* colonization.<sup>11</sup> The metaplasia also weakens the mucosa and leads to increased susceptibility to acid-induced injury.<sup>12</sup>

Despite being a non-invasive organism, *H. pylori* induces a robust immune response that contributes to duodenitis and the development of ulcers. Several cytokines are released in response to *H. pylori*, most notably IL-8, a potent chemotactic factor that activates neutrophils. B-cells are also stimulated with subsequent release of IgG and IgA antibodies. This inflammatory response impairs local protective mechanisms and leads to increased susceptibility to ulcers.<sup>13</sup>

Local mucosal defense mechanisms such as the release of epidermal growth factor and transforming growth factor alpha are also inhibited by *H. pylori* infection. These mediators are potent inhibitors of gastric acid secretion and stimulants of mucosal growth and protection. In addition, local release of bicarbonate in the duodenum is impaired. *H. pylori* itself releases proteases that degrade normally protective mucus glycoproteins. All of these factors contribute to inflammation and duodenal ulceration.<sup>14</sup>

Other factors play a role in the development of *H. pylori*-associated peptic ulcers because only 10-15% of

patients with *H. pylori* develop ulcer disease. An important contributor to the development of DU is the concomitant use of NSAIDs. It has been found that both factors together increase the risk of DU by approximately 60-fold. *H. pylori* alone increases the risk by approximately 20-fold. In addition, the risk of ulcer bleeding is increased 6-fold with the concomitant use of NSAIDs.<sup>15</sup> No evidence exists currently regarding the combination of corticosteroids and *H. pylori* and the risk of ulcer formation.

Other important clinical associations of *H. pylori* infection are gastric adenocarcinoma and mucosa-associated lymphatic tissue lymphoma, i.e., MALT lymphoma or *MALToma*. Gastric adenocarcinoma has been hypothesized to progress through a stepwise sequence of events from normal mucosa to carcinoma: superficial gastritis, chronic atrophic gastritis, intestinal metaplasia, dysplasia, and finally carcinoma.<sup>16</sup> *H. pylori* has the ability to cause chronic gastritis and atrophic gastritis, early steps in the carcinogenesis sequence. The Eurogast study of 17 different populations from 13 different countries, including the United States, found a 6-fold increase in the risk of gastric adenocarcinoma in patients infected with *H. pylori*.<sup>17</sup> Two additional meta-analyses examining the relationship between *H. pylori* and gastric cancer have demonstrated a two-fold increased risk of gastric cancer with *H. pylori* infection.<sup>18,19</sup>

*H. pylori* has been associated with gastric MALT lymphoma, an extra-nodal marginal zone B-cell lymphoma. *H. pylori* infection results in T-cell activation and subsequent lymphoid follicle formation by T-helper activation of B cells.<sup>20</sup> Multiple studies have indicated a correlation between *H. pylori* and gastric MALT lymphoma. For example, one study demonstrated an odds ratio of 6.3 for serologic positivity in patients with MALT lymphoma compared to controls.<sup>21</sup> The most notable evidence supporting a role for *H. pylori* in gastric MALT lymphoma is remission induced by *H. pylori* eradication. Several studies have indicated a high rate of resolution of low grade lymphoma with eradication of infection.

## DIAGNOSIS OF *H. PYLORI*

Assessment of possible *H. pylori* infection is essential in all patients with peptic ulcers. Diagnostic tests for the detection of *H. pylori* infection are subdivided into invasive (endoscopic) and noninvasive (nonendoscopic) categories.<sup>22,23,25</sup>

Endoscopic tests include a rapid urease test, histopathology, and culture. Rapid urease testing is considered the endoscopic test of choice. The presence of *H. pylori* in gastric mucosal biopsy specimens is established by testing for bacterial urease. Test kits contain a combination of a urea substrate and a pH-sensitive indicator. One or more gastric biopsy specimens are placed in the rapid urease test kit. If *H. pylori* organisms are present,



the bacterial-associated urease converts urea to ammonia, resulting in a pH change that also produces a color change.<sup>22, 23</sup> Histopathology is used if the rapid urease test result is negative and a high suspicion for *H. pylori* persists (presence of a duodenal ulcer).<sup>23</sup> Histology is also used to evaluate the presence of gastritis, MALT lymphoma, and metaplasia. Culture is difficult and reserved for refractory disease to assess possible resistance to therapy.

Nonendoscopic diagnostic tests include *H. pylori* antibody detection, fecal antigen tests, and urea breath tests. Immunoglobulins (IgG) to *H. pylori* can be measured in serum, plasma, or whole blood. Antibody testing is the test of choice for the diagnosis of *H. pylori* in the untreated patient.<sup>26</sup> However, this test may remain positive for up to 3 years after bacterial eradication, limiting its role for documentation of eradication.<sup>22, 23</sup> Urea breath tests detect active *H. pylori* infection by testing for the enzymatic activity of bacterial urease. The patient ingests urea labeled with a carbon isotope ( $C^{13}$  or  $C^{14}$ ). Upon entering the stomach, the urease of *H. pylori* cleaves the urea and the isotope is absorbed into the bloodstream, diffused into the lungs, and exhaled. Fecal antigen testing identifies active *H. pylori* infection by detection of antigens in stool. This test is more accurate than antibody testing and less expensive than the urea breath test.<sup>28</sup>

The selection of the appropriate test depends on the clinical situation. If endoscopy is clinically indicated, urease tests are used first and histology samples are reserved for instances when the urease test is negative.<sup>23</sup> If endoscopy is not warranted then serologic tests are used for initial screening. However, serology is limited by the extended presence of antibodies after active infection. Therefore, the urea breath test or stool antigen test can document the presence of active infection both before and after treatment. It is recommended to discontinue antibiotics and bismuth compounds for at least four weeks and proton pump inhibitors at least one week prior to testing because these agents can decrease the sensitivity of the diagnostic tests.<sup>24, 26</sup>

## H. PYLORI TREATMENT

Treatment strategies for eradication of *H. pylori* should include effective eradication, ease of administration, minimal side effects, and a low cost. Eradication measures should achieve an 80% cure rate on intention to treat analysis. Antibiotics must be combined with other agents to achieve a cure rate of this degree. The American College of Gastroenterology (ACG) guidelines from 1998 recommend one of the following treatment regimens: 1) a proton pump inhibitor, clarithromycin, and either amoxicillin or metronidazole for 2 weeks, 2) ranitidine, bismuth citrate, clarithromycin, and either amoxicillin, metronidazole, or tetracycline for 2 weeks, or 3) a proton pump inhibitor, bismuth, metronidazole, and tetracycline

for 1 to 2 weeks.<sup>29, 30</sup> Gastric and duodenal luminal acidity significantly influences the antimicrobial effectiveness of these antibiotic regimens. These combination regimens maximize eradication of the organism and decrease the chance for development of antimicrobial resistance. The proton pump inhibitors aid in ulcer healing and reducing gastric inflammation.

Current U. S. Food and Drug Administration (FDA)-approved regimens consist of 10 to 14 day course of therapy (Table 1). Recent trials have investigated 7-day therapeutic durations based on recent European recommendations, although no current FDA indication exists for this length of therapy. In a multicenter, double-blind, randomized, parallel-group trial of 803 patients, eradication rates of 77% with a 7-day regimen and 78% with a 10-day regimen of rabeprazole, clarithromycin, and amoxicillin were reported.<sup>30</sup> The MACLOR trial found an 89% eradication rate with a regimen of lansoprazole, amoxicillin, clarithromycin, and metronidazole for 5 days and was not significantly different from a reference-control regimen of 10 days.<sup>31</sup>

Based on 3400 isolates that were tested in a survey of 17 U.S. clinical trials from 1993 to 1999, antimicrobial resistance appears to be developing against metronidazole (35%) and clarithromycin (11%), although resistance to amoxicillin and tetracycline appears to be rare.<sup>29</sup> Resistance to clarithromycin and metronidazole appeared to be higher in women than men with a gradual increase in resistance up to age 70 years, followed by a significant decline in resistance above this age.<sup>32</sup>

**Table 1.** FDA Approved Oral *Helicobacter pylori* treatment regimens.<sup>32</sup>

Omeprazole 40 mg QD + clarithromycin 500 mg TID x 2 wks, then omeprazole 20 mg QD x 2 wks
Ranitidine bismuth citrate (RBC) 400 mg BID + clarithromycin 500 mg TID x 2 wks, then RBC 400 mg BID x 2 wks
Bismuth subsalicylate (Pepto Bismol®) 525 mg QID + metronidazole 250 mg QID + tetracycline 500 mg QID* x 2 wks + H2 receptor antagonist therapy as directed x 4 wks
Lansoprazole 30 mg BID + amoxicillin 1 g BID + clarithromycin 500 mg TID x 10 days
Lansoprazole 30 mg TID + amoxicillin 1 g TID x 2 wks**
Ranitidine bismuth citrate 400 mg BID + clarithromycin 500 mg BID x 2 wks, then RBC 400 mg BID x 2 wks
Omeprazole 20 mg BID + clarithromycin 500 mg BID + amoxicillin 1 g BID x 10 days

(Table adopted from CDC website: [www.cdc.gov/ulcer](http://www.cdc.gov/ulcer))

Poor patient compliance or the development of resistance results in eradication failures.<sup>30</sup> Because culture data rarely exist for prior treatment failures, rationale exists to switch to an alternative antibiotic regimen (for example, clarithromycin to metronidazole) or to administer quadruple therapy (bismuth, proton pump inhibitor, and two antibiotics) for a longer duration (14 days). Pooled analysis of sixteen articles and twenty-four abstracts has revealed an eradication rate of 80.2% and 75.8% for ranitidine bismuth-based triple therapy and quadruple therapy, respectively.<sup>29</sup>

Side effects of individual drugs may be reported by patients. Metallic taste is noted with either metronidazole or clarithromycin. Metronidazole is known to cause a disulfuram-like reaction with concomitant use of alcohol. Metronidazole can also cause peripheral neuropathy and seizures. Amoxicillin may elicit an allergic reaction and quite frequently induces abdominal distress/diarrhea. Practitioners must be aware that tetracycline is contraindicated in the pregnant patient and that a photosensitivity reaction may occur in any individual. Histamine receptor 2 blockers may invoke cytochrome P450 system drug metabolism changes and are known in rare cases to cause central nervous system reactions or cytopenias. Side effects of bismuth are rare and the proton pump inhibitors are well tolerated with a negligible side-effect profile.

## REFERENCES

1. Marshall BJ, Warren JR. Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. *Lancet* 1984;1:1311-1315.
2. Cave DR. Transmission and epidemiology of *Helicobacter pylori*. *Am J Med* 1996;100(5A): 12S-17S.
3. Mobley HL. The role of *Helicobacter pylori* urease in the pathogenesis of gastritis and peptic ulceration. *Aliment Pharmacol Ther* 1996;10(Suppl 1):57-64.
4. Mobley HL. Defining *Helicobacter pylori* as a pathogen: Strain heterogeneity and virulence. *Am J Med* 1996; 100(5A): 9S-11S.
5. Morris A, Nicholson G. Ingestion of *Campylobacter pyloridis* causes gastritis and raised fasting gastric pH. *Am J Gastroenterol* 1987; 82:192-199.
6. Karttunen T, Niemela S, Lehtola J, et al. *Campylobacter*-like organisms and gastritis: histopathology, bile reflux, and gastric fluid composition. *Scand J Gastroenterol* 1987;22:478-486.
7. Tytgat G, Langenberg W, Rauws E, et al. *Campylobacter*-like organism in the human stomach. *Gastroenterology* 1985; 88:1620-1622.
8. Hopkins RJ, Girardi LS, Turney EA. Relationship between *Helicobacter pylori* eradication and reduced duodenal and gastric ulcer recurrence: a review *Gastroenterology* 1996;110:1244-1252.
9. Laine L, Hopkins RJ, Girardi LS. Has the impact of *Helicobacter pylori* therapy on ulcer recurrence in the United States been overstated? A meta-analysis of rigorously designed trials. *Am J Gastroenterol* 1998; 93:1409-1415.
10. el-Omar EM, Penman ID, Ardill JE, et al. *Helicobacter pylori* infection and abnormalities of acid secretion in patients with duodenal ulcer disease. *Gastroenterology* 1995;109:681-691.
11. Wyatt JJ, Rathbone BJ, Dixon MF, et al. *Campylobacter pyloridis* and acid induced gastric metaplasia in the pathogenesis of duodenitis. *J Clin Pathol* 1987;40:841-848.
12. Peura DA. Ulcerogenesis: integrating the roles of *Helicobacter pylori* and acid secretion in duodenal ulcer. *Am J Gastroenterol* 1997; 92(4 Supplement): 8S-13S.
13. Moss SF, Legon S, Davies J, et al. Cytokine gene expression in *Helicobacter pylori* associated antral gastritis. *Gut* 1994; 35:1567-1570.
14. Konturek, Ernst H, Bobrzynski AJ, et al. Mucosal expression and luminal release of epidermal and transforming growth factors in patients with duodenal ulcer before and after eradication of *Helicobacter pylori*. *Gut* 1997; 40: 463-469.
15. Huang JQ, Sridhar S, Hunt RH. Role of *Helicobacter pylori* infection and non-steroidal anti-inflammatory drugs in peptic ulcer disease: a meta-analysis. *Lancet* 2002; 359:14-22.
16. Siurala M, Varis K, Wiljasalo M. Studies of patients with atrophic gastritis: a 10-15 year follow-up. *Scand J Gastroenterol* 1966;1:40-48.
17. Eurogast Study Group. An international association between *Helicobacter pylori* infection and gastric cancer. *Lancet* 1993; 341:1359-1362.
18. Huan JQ, Sridhar S, Chen Y, et al. Meta-analysis of the relationship between *Helicobacter pylori* seropositivity and gastric cancer. *Gastroenterology* 1998; 114: 1169-1179.
19. Eslick GD, Lim LL, Byles JE, et al. Association of *Helicobacter pylori* infection with gastric carcinoma: a meta-analysis. *Am J Gastroenterol* 1999;94:2373-2379.
20. D'Elia MM, Amedei A, Manghetti M, et al. Impaired T-cell regulation of B-cell growth in *Helicobacter pylori*-related gastric MALT lymphoma. *Gastroenterology* 1999;117:1105-1112.
21. Parsonnet J, Hansen S, Rodriguez L, et al. *Helicobacter pylori* infection and gastric lymphoma. *N Engl J Med* 1994;330:1267-1271.
22. Guidelines for the Management of *Helicobacter pylori*. Ad Hoc Committee on Practice Parameters of the American College of Gastroenterology. *Am J Gastroenterol* 1998;93:2330-2338.
23. Feldman M, Friedman LS, Sleisenger MH. *Sleisenger and Fortran's Gastrointestinal and Liver Disease. Pathophysiology/ Diagnosis/ Management*, 7<sup>th</sup> edition. 2002:738-742.
24. Atta L, Vakli N, Ricci C, et al. Effect of Proton Pump Inhibitors and Antacid Therapy on C Urea Breath Tests and Stool Test for *Helicobacter Pylori* Infection. *Am J Gastroenterol* 2004;99:823-829.
25. Goldman L, Ausiello D. *Cecil's Textbook of Medicine*, 22<sup>nd</sup> edition. 2004 W.B. Saunders company. pp 830-831.
26. Laine L, Estrada R, Trujillo M, et al. Effect of proton-pump inhibitor therapy on diagnostic testing for *Helicobacter pylori*. *Ann Intern Med* 1998;129:547-550.
27. Malfertheiner P, Megraud F, O'Morain C, et al. Current concepts in the management of *Helicobacter pylori* infection—the Maastricht 2-2000 Consensus Report. *Aliment Pharmacol Ther* 2002;16:167-180.
28. Trevisani L, Sartori S, Galvani F, et al. Evaluation of a new enzyme immunoassay for detecting *Helicobacter pylori* in feces: A prospective pilot study. *Am J Gastroenterol* 1999; 94:1830.

29. Howden CW, Hunt RH. Guidelines for the management of helicobacter pylori treatment. *Am J Gastroenterol* 1998; 93: 2330-2338.
30. Vakil N, Lanza F, Schwartz H, et al. Seven-day therapy for helicobacter pylori in the United States. *Aliment Pharmacol Ther* 2004. 20:99-107.
31. Hojo M, Miwa H, Nagahara A, et al. Pooled analysis on the efficacy of the second-line treatment regimens for Helicobacter pylori infection. *Scand J Gastroenterol* 2001; 36:690-700.
32. <http://www.cdc.gov/ulcer/md.htm>

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## CME QUESTIONS

To earn CME credit, read the preceding CME article and complete the registration, evaluation, and answer form on page 159. Mail or fax the registration, evaluation, and answer form to the Educational and Research Foundation. Answers must be postmarked or faxed prior to June 30, 2007. Participants must attain a minimum score of 75% to receive credit.

For each question, choose the one answer that is most correct.

1. All of the following are true concerning *H. pylori* except:
  - a. The mode of transmission of *H. pylori* is believed to be oral-oral and fecal-oral in nature.
  - b. *H. pylori* infection is associated with greater than 80% of gastric ulcers and 100% of duodenal ulcers.
  - c. *H. pylori* is associated with gastric adenocarcinoma and MALT lymphoma.
  - d. Risk factors for development of *H. pylori* include low socioeconomic status, overcrowding, and the lack of running water.
2. True/False: Because of the strong association of *H. pylori* with peptic ulcers, it is recommended that all patients with peptic ulcer disease get tested for *H. pylori*.
3. All of the following are true concerning *H. pylori* treatment except:
  - a. Treatment strategies should involve both effective eradication with ease of administration, minimal side effects, and a low cost.
  - b. The most common reasons for treatment failure include poor compliance and development of resistance.
  - c. Proton pump inhibitors are well tolerated with a negligible side effect profile.
  - d. The FDA currently approves a 7-day course of therapy for *H. pylori* eradication.
4. True/False: *H. pylori* infection occurs worldwide and is the most common chronic bacterial infection in humans.

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