Acute and chronic gastritis due to Helicobacter pylori

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INTRODUCTION

Gastritis denotes inflammation associated with gastric mucosal injury. Epithelial cell damage and regeneration without associated inflammation is referred to as "gastropathy" [1,2]. Gastritis is usually caused by infectious agents (eg, *Helicobacter pylori*) or is immune mediated, although in many cases the cause of the gastritis is unknown.

This topic will review acute (active) and chronic gastritis due to *H. pylori* [3-5]. The other forms of gastritis and gastropathy and other issues related to *H. pylori* are discussed separately. (See "Gastritis: Etiology and diagnosis" and "Indications and diagnostic tests for Helicobacter pylori infection" and "Treatment regimens for Helicobacter pylori" and "Association between Helicobacter pylori infection and gastrointestinal malignancy" and "Association between Helicobacter pylori infection and duodenal ulcer" and "Helicobacter pylori and gastroesophageal reflux disease" and "Pathophysiology of and immune response to Helicobacter pylori infection".)

HELICOBACTER PYLORI GASTRITIS

H. pylori gastritis affects two-thirds of the world's population and is one of the most common chronic inflammatory disorders [6]. Most patients with *H. pylori* infection will show features of both acute and chronic gastritis (chronic active gastritis).

Pathophysiology — *H. pylori* resides primarily in the unstirred layer of gastric mucus, adjacent to epithelial cells at the mucosal surface and in gastric pits (picture 1A-B) [7]. Gastric glands are usually not involved. The epithelial localization reflects the affinity of *H. pylori* for gastric mucous

cells [8,9]. *H. pylori* does not attach to small intestinal or other gastric epithelial cell types. The organisms are uncommonly found in the lamina propria. (See "Pathophysiology of and immune response to Helicobacter pylori infection".)

The usual natural history of *H. pylori* gastritis is of an antral predominant early stage of infection with only minimal corpus involvement. This stage is associated with exaggerated gastrin release and reduced somatostatin release, often precipitating an increase in acid secretion, enough to cause duodenal ulcers in some patients [10].

With continued inflammation, gastrin producing (G) cells and acid producing parietal cells are gradually lost, precipitating a fall in acid secretion and the development of atrophy with intestinal metaplasia [11]. These changes facilitate the proximal migration of the bacteria, leading to corpus gastritis [12]. Thus, the natural history of *H. pylori* gastritis is of diffuse antral inflammation spreading to the corpus, resulting in an atrophic front of advancing corpus injury with concomitant reduction in acid secretion. This scenario is accelerated by hypochlorhydria such as that caused by chronic therapy with proton pump inhibitors (PPIs). However, this orad evolution is not inevitable since it can be modified by antibiotic treatment.

Patients in whom *H. pylori* colonization is heaviest in the gastric body may differ from those with antral predominant infection. Duodenal ulcers are typically associated with antral predominant gastritis, little or no atrophy, and high-normal or increased acid secretion. By contrast, gastric ulcers and gastric cancer are typically associated with more extensive gastritis, widespread intestinal metaplasia, and low-normal or reduced gastric acid secretion [13].

Acute gastritis

Clinical manifestations — Patients with acute *H. pylori* are asymptomatic or develop mild self-limited dyspeptic symptoms. Few examples of spontaneous acute infection have been recognized since the majority of patients who develop nonspecific dyspeptic complaints (which may signal acute infection) may not seek medical attention and thus are not immediately investigated [14-16].

The ability of *H. pylori* to cause acute gastritis was first demonstrated after healthy volunteers ingested the organisms and developed a mild illness (consisting of epigastric pain, nausea, and vomiting without fever) associated with acute inflammatory changes on gastric biopsy [17,18]. Acute infection was also demonstrated in volunteers undergoing gastric secretory studies who were inadvertently infected by contaminated equipment [19-21]. These cases also demonstrated that acute infection is associated with the development of transient hypochlorhydria, a phenomenon that was suspected to be caused by an infectious agent and

was referred to as "epidemic hypochlorhydria" [21]. (See "Pathophysiology of and immune response to Helicobacter pylori infection".)

Acute gastritis almost always evolves into active chronic gastritis unless the patient is treated with appropriate antibiotics. (See 'Chronic gastritis' below.)

Endoscopic and histopathologic features — The endoscopic appearance of acute *H. pylori* gastritis is variable and, in severe cases, can resemble lymphoma or gastric carcinoma [22]. In early infection, *H. pylori* gastritis preferentially involves the gastric antrum.

Histologic changes of acute *H. pylori* gastritis include intense neutrophilic infiltration of the mucous neck region and lamina propria. When severe, pit abscesses occur, along with mucin loss, erosion of the juxtaluminal cytoplasm, and desquamation of surface foveolar cells. Both neutrophils and bacteria are responsible for destruction of the epithelium. Lymphoid follicles appear within one week after the onset of acute *H. pylori* infection, and are uncommon in non-*H. pylori*-infected gastric mucosa [20,23]. In general, lymphoid follicles represent an immune response to the organism, and are composed of aggregates of lymphocytes and other lymphoid cells associated with a central germinal center made up of larger, paler mononuclear cells. The number of lymphoid follicles present correlates with the titer of serum IgG anti-*H. pylori* *a*ntibodies [24]. Lymphoid follicles accompanying *H. pylori* *g*astritis are involved in the genesis of primary gastric B cell lymphoma [25,26]. The pathogenesis may involve stimulation of B cells by activated T cells within the follicles. (See "Association between Helicobacter pylori infection and gastrointestinal malignancy".)

Chronic gastritis

Clinical presentation

Gastroduodenal manifestations — It is unclear if chronic H. *pylori* infection causes abdominal pain in the absence of peptic ulcer disease. However, it has been associated with functional dyspepsia. Patients with chronic *H. pylori* gastritis can present with complications of peptic ulcer disease or gastroduodenal complications of chronic infection including gastric atrophy, intestinal metaplasia, gastric cancer, and mucosa-associated lymphoid tissue (MALT) lymphoma. (See "Functional dyspepsia in adults", section on 'Epidemiology and pathophysiology' and "Peptic ulcer disease: Clinical manifestations and diagnosis", section on 'Clinical manifestations' and "Clinical features, diagnosis, and staging of gastric cancer", section on 'Clinical features' and "Clinical manifestations, pathologic features, and diagnosis of extranodal marginal zone lymphoma of mucosa associated lymphoid tissue (MALT)", section on 'Clinical features'.)

Extragastrointestinal manifestations — In some studies, *H. pylori* infection has been associated with other extragastrointestinal diseases including:

- **Iron deficiency anemia** – *H. pylori* gastritis has been associated with iron deficiency anemia [27,28]. The most plausible mechanism is decreased iron absorption due to *H. pylori*-associated gastric atrophy and hypochlorhydria [28]. (See "Indications and diagnostic tests for Helicobacter pylori infection", section on 'Indications for testing'.)

- **Idiopathic thrombocytopenic purpura (ITP)** – In some adults with ITP who were infected with *H. pylori*, platelet counts increase following eradication therapy. A proposed mechanism is molecular mimicry with cross-reactive antibodies. (See "Indications and diagnostic tests for Helicobacter pylori infection", section on 'Indications for testing'.)

- **Vitamin B12 deficiency** – *H. pylori* infection can lead to chronic (metaplastic) atrophic gastritis resulting in hypochlorhydria and vitamin B12 malabsorption. Also, the bacteria may elicit production of antibodies that cross-react with the gastric parietal H^+ K^+ ATPase. Eradication of chronic *H. pylori* infection has been associated with increases in vitamin B12 levels [28,29]. (See "Causes and pathophysiology of vitamin B12 and folate deficiencies", section on 'H. pylori infection' and "Metaplastic (chronic) atrophic gastritis", section on 'Helicobacter pylori'.)

Associations have been noted between *H. pylori* infection and a large number of other conditions, such as coronary artery disease, neurologic disorders, metabolic syndrome, cerebrovascular disease, nonalcoholic fatty liver disease, and diabetes mellitus. However, the data are controversial and insufficient to establish a causal link [28,30-32].

Endoscopic features — The endoscopic appearance is normal in as many as 50 percent of patients with chronic *H. pylori* gastritis [33,34]. Other patients may have nonspecific endoscopic features including mucosal erythema, friable gastric mucosa, and diffuse antral nodularity.

Histopathology — Histopathology plays a major role in diagnosing *H. pylori* infection, establishing the presence, severity, and extent of gastritis, and detecting associated complications of *H. pylori* infection (eg, intestinal metaplasia, MALT lymphoma, and gastric carcinoma). *H. pylori* is almost always accompanied by gastritis and the diagnosis of *H. pylori* should be suspect in its absence.

- **Identification of *H. pylori*** – A definitive histopathologic diagnosis of *H. pylori* infection depends upon the demonstration of the typical spiral shaped bacilli on a biopsy specimen. During antimicrobial treatment, *H. pylori* bacteria may lose their typical spiral shape and assume new forms, including U-shaped, rod-like, and coccoid forms. The coccoid forms

appear as round basophilic dots, 0.4 to 1.2 micrometer in diameter.

The organisms can be detected in both the antrum and the body of the stomach in the majority (80 percent) of chronically infected patients (image 1) [35]. *H. pylori* is localized to the antrum alone or the body alone in 8 and 10 percent of patients, respectively. Localization of *H. pylori* to the body alone is usually due to concurrent PPI use or marked gastric atrophy and intestinal metaplasia.

Given the variable distribution of *H. pylori* in the stomach, and the attenuated growth observed during treatment with PPIs, diagnostic accuracy can be increased when biopsies are taken from multiple sites in the stomach. The updated Sydney system [1] recommends that biopsy specimens be taken from five different sites for optimal assessment of both gastritis and *H. pylori* status: lesser and greater curvature of the antrum, lesser and greater curvature of the corpus, and the incisura angularis. (See "Indications and diagnostic tests for Helicobacter pylori infection", section on 'Patient undergoing upper endoscopy' and "Indications and diagnostic tests for Helicobacter pylori infection", section on 'Histology'.)

It is frequently possible to identify *H. pylori* in standard hematoxylin and eosin (H&E) preparations. However, when a low density of *H. pylori* and atrophic mucosal changes are both present, visualization of the organism becomes unreliable on H&E alone [36,37]. Most pathologists use H&E plus a second stain for *H. pylori* visualization. A variety of stains are available and can be divided into non-silver-based stains, silver-based stains, and immunohistochemical stains [38]:

- Immunohistochemical stains – Immunostaining techniques are highly sensitive, specific, and reliable (picture 2). They have a particular advantage in patients partially treated for *H. pylori* gastritis, a setting that can result in atypical (including coccoid) forms, which may mimic bacteria or cell debris on hematoxylin and eosin preparations. When PPIs and other hypochlorhydric states facilitate survival and overgrowth of non-*H. pylori* bacteria, immunohistochemical stains can confirm the absence of *H. pylori* [39].

- Non-silver-based stains – The quick Giemsa method (eg, Diff-Quik) is easy to use, inexpensive, and gives consistent results [40]. It is the preferred method in many laboratories, particularly for screening, and when immunohistochemistry is not readily available [41]. However, since it is a morphologic stain, it is not as specific as immunohistochemistry.

- Silver-based stains – Silver stains (such Warthin-Starry and Genta methods), which

were crucial to the original demonstration of *H. pylori*, are expensive and technologically complex. The results are not always reliable due to abundant background artifact [40]. This stain has largely been replaced by the simpler (Giemsa) and more specific (immunohistochemistry) stains.

- **Associated gastritis** – Acute and chronic inflammatory cells are concentrated in the upper part of the mucosa, beginning just below the surface epithelium and giving the appearance of superficial gastritis. This pattern is so characteristic that *H. pylori* gastritis may be suspected even at the lowest magnifications.

  The chronic inflammatory elements in *H. pylori* gastritis primarily consist of lymphocytes and plasma cells, scattered macrophages, and often eosinophils [42]. Lymphoid follicles are frequently present and represent an immune response to the bacteria; their presence provides a useful marker for *H. pylori* infection. Similarly, a prominence of plasma cells is a valuable clue to *H. pylori* infection.

  The acute (active) inflammatory component consists of neutrophilic infiltration of the surface and foveolar epithelium and the lamina propria, usually in scattered foci, often with small pit abscesses. The intensity of the inflammation varies among patients and sometimes from specimen to specimen in the same patient. Active inflammation is somewhat more common in antral than in oxyntic *H. pylori* infection [43]. Although casual observation reveals no obvious relation between the numbers of organisms and the severity of the acute or chronic inflammation, a correlation with bacterial density and active gastritis has been described [38]. Inflammation associated with chronic *H. pylori* gastritis improves dramatically after eradication of the organisms with appropriate antibiotics.

- Neutrophils disappear rapidly; the persistence of even small numbers of neutrophils may be predictive of relapse [44].

- Lymphocytes and eosinophils decrease more slowly, and some chronic inflammation can still be seen after one year. Lymphoid follicles are the slowest to disappear, and usually persist for more than one year [44].

- Studies have shown that intestinal metaplasia and atrophy (if present) usually do not resolve by one year but that significant improvement has been recognized after 10 years [45,46]. Organism eradication may also help prevent the development of further gastric atrophy and intestinal metaplasia [47].

- Fibrosis and architectural distortion, including foveolar hyperplasia, may persist long after *H. pylori* infection is eliminated and often resembles chemical gastropathy. (See "Acute

hemorrhagic erosive gastropathy and reactive gastropathy".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: H. pylori infection (The Basics)" and "Patient education: Gastritis (The Basics)" and "Patient education: Upper endoscopy (The Basics)")
- Beyond the Basics topics (see "Patient education: Helicobacter pylori infection and treatment (Beyond the Basics)" and "Patient education: Upper endoscopy (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- Gastritis denotes inflammation associated with gastric mucosal injury. Epithelial cell damage and regeneration without associated inflammation is referred to as "gastropathy." Gastritis is usually caused by infectious agents (eg, *H. pylori*) or is immune mediated, although in many cases the cause of the gastritis is unknown. (See 'Pathophysiology' above.)
- Patients with acute *H. pylori* are asymptomatic or develop mild self-limited dyspeptic symptoms. Acute gastritis almost always evolves into active chronic gastritis unless the patient is treated with appropriate antibiotics. (See 'Acute gastritis' above.)
- It is unclear if chronic *H. pylori* infection causes abdominal pain in the absence of peptic ulcer disease. However, it has been associated with functional dyspepsia. Patients with

chronic *H. pylori* gastritis can present with complications of peptic ulcer disease or gastroduodenal complications of chronic infection including gastric atrophy, intestinal metaplasia, gastric cancer, and mucosa-associated lymphoid tissue lymphoma. The endoscopic appearance is normal in as many as 50 percent of patients with chronic *H. pylori* gastritis. Other patients may have nonspecific endoscopic features including mucosal erythema, friable gastric mucosa, and diffuse antral nodularity. (See 'Chronic gastritis' above.)

- *H. pylori* gastritis typically begins as a diffuse antral gastritis, which subsequently spreads to the gastric corpus if untreated. Changes of chronic active gastritis may be associated with or result in intestinal metaplasia (atrophy). Chronic use of proton pump inhibitors (PPIs) may facilitate proximal migration of the organisms leading to corpus gastritis. Given the variable distribution of *H. pylori* in the stomach, and the attenuated growth observed during treatment with PPIs, we obtain one to two biopsies from five different sites within the stomach (greater and lesser curvature of antrum, greater and lesser curvature of the corpus, and the incisura angularis). (See 'Chronic gastritis' above.)

- A definitive histopathologic diagnosis of *H. pylori* infection depends upon the demonstration of the typical spiral shaped bacilli on a biopsy specimen. Immunohistochemistry may be necessary for the detection of *H. pylori* organisms in patients on an antibiotic, chronic PPI therapy, or with other hypochlorhydric states that predispose to gastric bacterial overgrowth. Acute inflammation disappears rapidly with treatment, but the chronic inflammation, including lymphoid follicles, can persist for years. (See 'Endoscopic and histopathologic features' above and 'Histopathology' above.)

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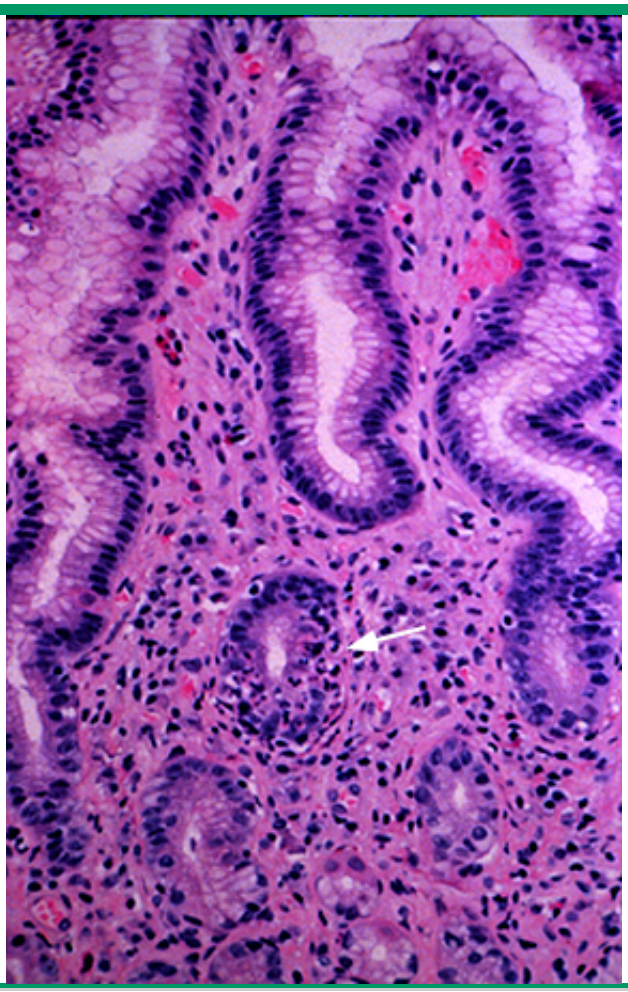
GRAPHICS

Helicobacter pylori gastritis

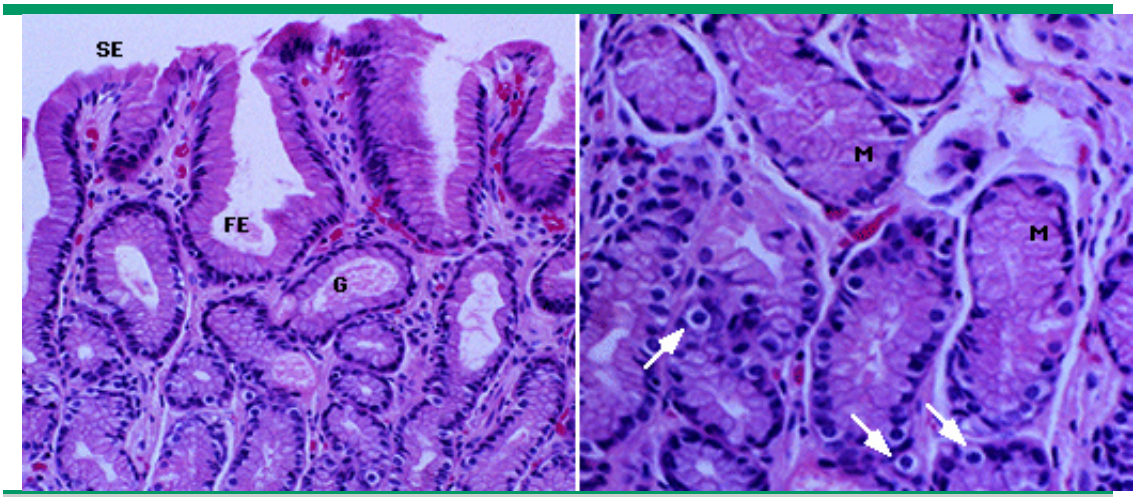


Medium power view of a gastric biopsy obtained at endoscopy shows infiltration of glands with neutrophils (arrow) and increased mononuclear cell infiltration typical of *Helicobacter pylori* gastritis.

Courtesy of Robert Odze, MD.

Graphic 64354 Version 2.0

Normal gastric antrum

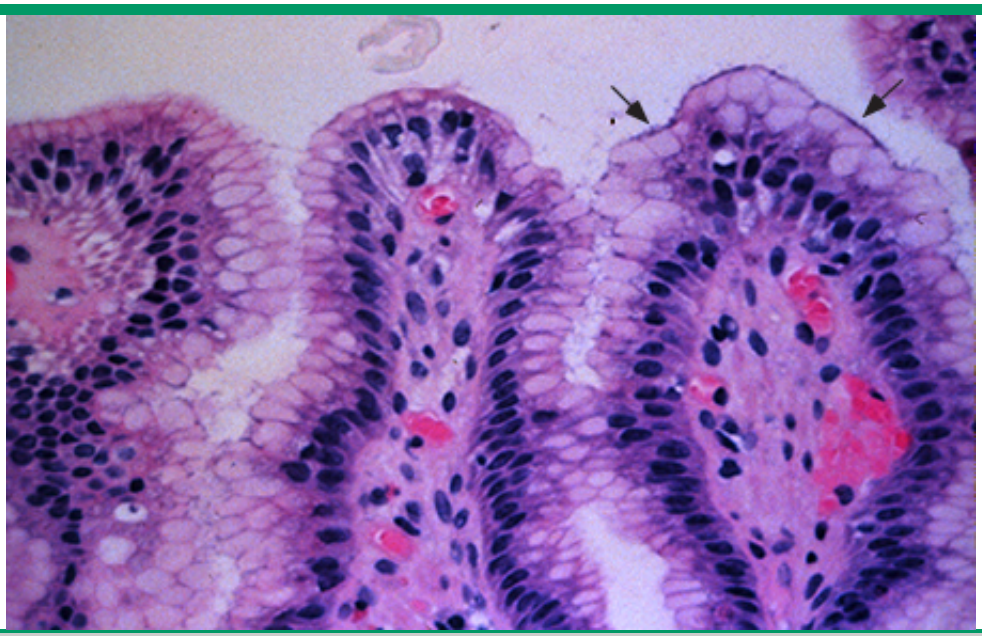


Left panel: Normal surface (SE) and foveolar epithelium (FE) and glands (G). Right panel: Higher power view of the glands shows mucous cells (M) and gastrin-secreting endocrine cells (arrows).

Courtesy of Robert Odze, MD

Graphic 79895 Version 1.0

Helicobacter pylori adherence on gastric surface cells

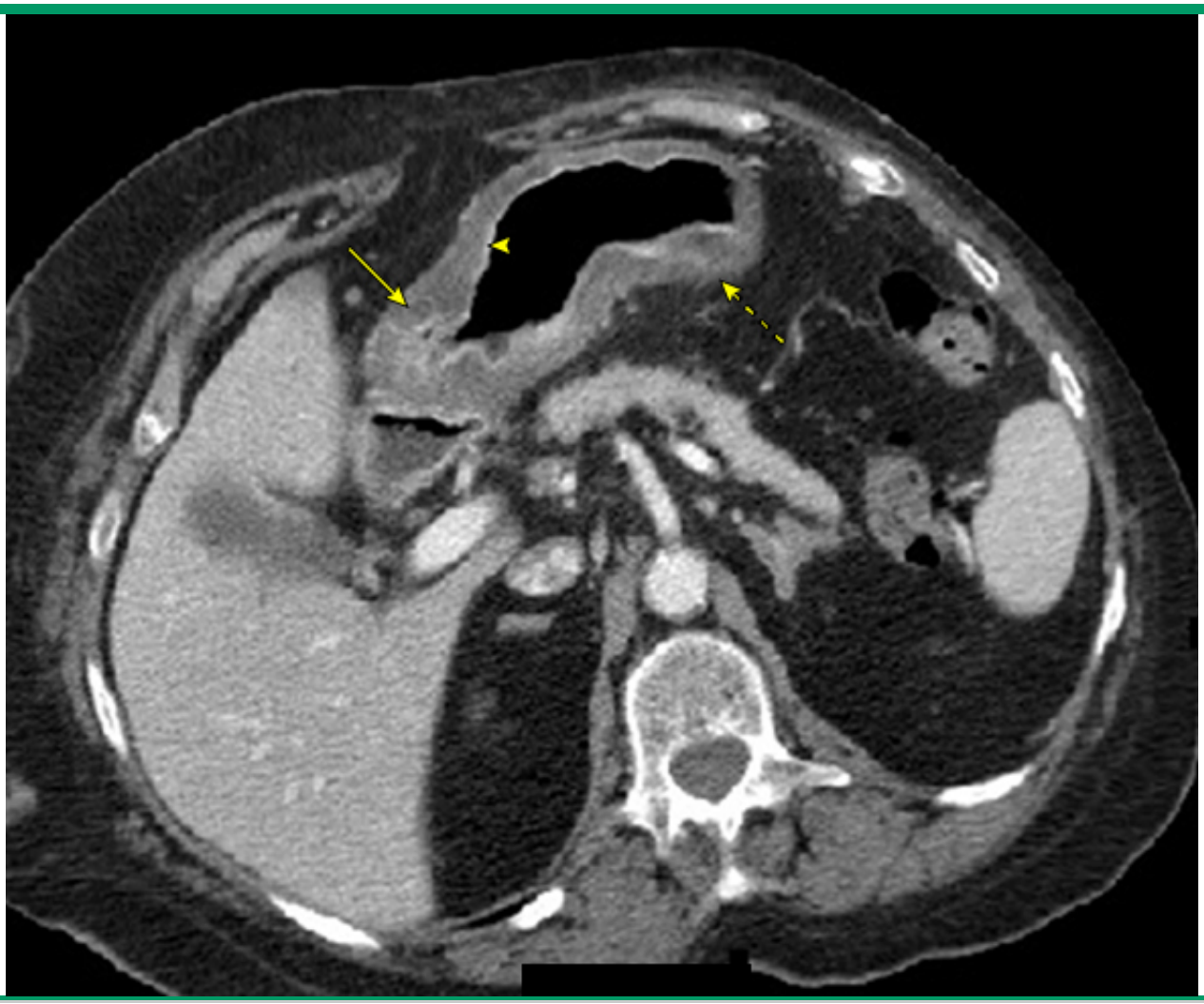


High power view of surface and foveolar epithelium shows numerous *Helicobacter pylori* organisms lining the surface of the cells (arrows).

Courtesy of Robert Odze, MD.

Graphic 63916 Version 2.0

Chronic active *Helicobacter pylori* gastritis on CT

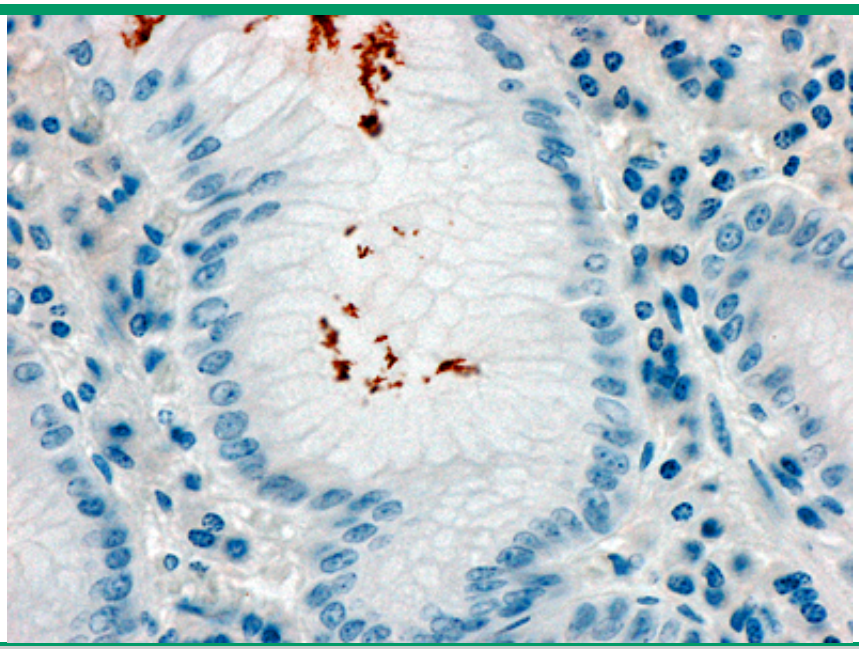


A CT scan through the upper abdomen shows thickened wall of the body of the stomach (dashed arrow) and antrum (arrow). The mucosa is hyperemic (arrowhead). *Helicobacter pylor*i was recovered from the gastric biopsy, which also showed chronic active gastritis.

CT: computed tomography.

Graphic 93360 Version 3.0

Helicobacter pylori **in gastric crypts**



A 600x magnification of a *Helicobacter pylori* immunostain with the luminal organisms shown in brown.

Courtesy of Pamela J Jensen, MD.

Graphic 73081 Version 2.0

Contributor Disclosures

Pamela J Jensen, MD Nothing to disclose **Mark Feldman, MD, MACP, AGAF, FACG** Nothing to disclose **J Thomas Lamont, MD** Nothing to disclose **Shilpa Grover, MD, MPH, AGAF** Nothing to disclose

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