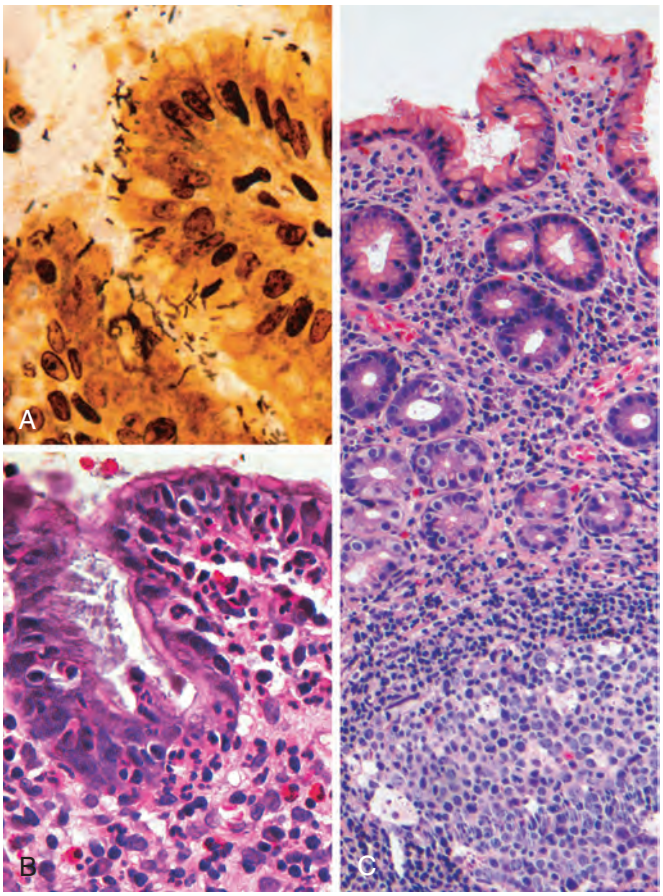

Figure 17-12 *Helicobacter pylori* gastritis. **A,** Spiral-shaped *H. pylori* are highlighted in this Warthin-Starry silver stain. Organisms are abundant within surface mucus. **B,** Intraepithelial and lamina propria neutrophils are prominent. **C,** Lymphoid aggregates with germinal centers and abundant subepithelial plasma cells within the superficial lamina propria are characteristic of *H. pylori* gastritis.

cardia occurs at somewhat lower rates. *H. pylori* are less common in oxyntic (acid-producing) mucosa of the fundus and body. Thus, an antral biopsy is preferred for evaluation of *H. pylori* gastritis. When viewed endoscopically, *H. pylori*-infected antral mucosa is usually erythematous and has a coarse or even nodular appearance. The inflammatory infiltrate generally includes variable numbers of neutrophils within the lamina propria, including some that cross the basement membrane to assume an intraepithelial location (Fig. 17-12*B*) and accumulate in the lumen of gastric pits to create pit abscesses. In addition, the superficial lamina propria contains large numbers of plasma cells, often in clusters or sheets, and increased numbers of lymphocytes and macrophages. Intraepithelial neutrophils and subepithelial plasma cells are characteristic of *H. pylori* gastritis. When intense, inflammatory infiltrates may create thickened rugal folds, mimicking the appearance of early cancers. Lymphoid aggregates, some with germinal centers, are frequently present (Fig. 17-12*C*) and represent an induced form of mucosa-associated lymphoid tissue, or MALT, that has the potential to transform into lymphoma.

Long-standing *H. pylori* gastritis may extend to involve the body and fundus, and the mucosa can become atrophic, with loss of parietal and chief cells. As a result, the oxyntic mucosa can take on the appearance of antral mucosa. In contrast to autoimmune gastritis, this is typically a patchy process, and biopsies of the gastric body can show intact oxyntic glands adjacent to antral-type glands. The development of atrophy is typically associated with the presence of intestinal metaplasia and increased risk of gastric adenocarcinoma.

Clinical Features. In addition to histologic identification of the organism, several diagnostic tests have been developed including a noninvasive serologic test for antibodies to *H. pylori*, fecal bacterial detection, and the urea breath test based on the generation of ammonia by the bacterial urease. Gastric biopsy specimens can also be analyzed by the rapid urease test, bacterial culture, or bacterial DNA detection by PCR.

Effective treatments for *H. pylori* infection include combinations of antibiotics and proton pump inhibitors. Individuals with *H. pylori* gastritis usually improve after treatment, although relapses can occur after incomplete eradication or reinfection, which is common in regions with high endemic colonization rates. Prophylactic and therapeutic vaccines are still at an early stage of development.

Autoimmune Gastritis

Autoimmune gastritis accounts for less than 10% of cases of chronic gastritis. In contrast to *H. pylori*–associated gastritis, autoimmune gastritis typically spares the antrum and is associated with hypergastrinemia (Table 17-2). Autoimmune gastritis is characterized by:

- Antibodies to parietal cells and intrinsic factor that can be detected in serum and gastric secretions
- Reduced serum pepsinogen I concentration
- Endocrine cell hyperplasia
- Vitamin B_{12} deficiency
- Defective gastric acid secretion (achlorhydria)

Table 17-2 Characteristics of *Helicobacter pylori*–Associated and Autoimmune Gastritis

	H. pylori–Associated	Autoimmune
Location	Antrum	Body
Inflammatory infiltrate	Neutrophils, subepithelial plasma cells	Lymphocytes, macrophages
Acid production	Increased to slightly decreased	Decreased
Gastrin	Normal to decreased	Increased
Other lesions	Hyperplastic/inflammatory polyps	Neuroendocrine hyperplasia
Serology	Antibodies to *H. pylori*	Antibodies to parietal cells (H^+,K^+-ATPase, intrinsic factor)
Sequelae	Peptic ulcer, adenocarcinoma, MALToma	Atrophy, pernicious anemia, adenocarcinoma, carcinoid tumor
Associations	Low socioeconomic status, poverty, residence in rural areas	Autoimmune disease; thyroiditis, diabetes mellitus, Graves disease