Stomach Duodenum Inflammatory Disease

26

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Introduction

Any injury to the gastric or duodenal mucosa results in epithelial damage and repair. The term "gastritis" or "duodenitis" reflects inflammation associated with mucosal injury. Gastritis and duodenitis may be caused by infectious agents (such as *Helicobacter pylori*), autoimmune disorders, or hypersensitivity reactions. It can also result from drugs or extreme stress reactions.

Epithelial damage and repair can occur without mucosal inflammation. This is referred to as "gastropathy" (Carpenter and Talley 1995; Dixon et al. 1996). Gastropathy may be caused by endogenous or exogenous irritants such as alcohol or refluxed bile, medications such as aspirin and NSAIDS, and conditions such as ischemia, stress, hypovolemia, and chronic congestion.

There is confusion between the terms "gastritis" and "gastropathy" because "gastritis" has been used by endoscopists and radiologists to describe their findings in both groups of diseases. The distinction between gastritis and gastropathy is of importance because the causes, natural history, and management differ. The variety of diseases can be represented on imaging of the stomach and duodenum in only one or a combination of only a few manifestations. A radiologist is faced with a considerable overlap of similar imaging appearances between diseases of differing etiology.

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Classification of Gastritis and Gastropathy

Gastritis and gastropathy may be classified by time course (acute vs chronic), etiology, histologic features, and proposed pathophysiology. However, no classification is universally accepted because there are gaps in knowledge of etiology and pathogenesis, variations in definitions, and patients may exhibit more than one type of gastritis or gastropathy.

Most classification systems distinguish these conditions clinically into acute, short-term disease from chronic, long-term disease. The terms "acute" and "chronic" can also be described by the type of inflammatory cell infiltrate. Acute inflammation is characterized by neutrophilic infiltration, while in chronic inflammation there are mononuclear cells, lymphocytes, plasma cells, and macrophages.

A radiological diagnostic approach could describe these conditions by etiology, as this may be made known to the radiologist, and by morphologic pattern as determined by imaging. Acute inflammatory disease is suggested by the presence of erosions and/or ulcers. Thickening of gastric or duodenal folds may be seen in inflammatory disease from the acute to a chronic stage. Progression of disease to a chronic "burnt out" stage usually leaves the involved segment scarred or featureless and narrowed.

Acute Erosive Gastropathy

Erosions are defined as epithelial defects that do not penetrate beyond the muscularis mucosae. Acute erosive lesions may occur when the gastric surface epithelium is damaged. This may occur with exposure

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to an agent causing mucosal injury including ingested medications such as aspirin and nonsteroidal anti-inflammatory drugs [NSAIDs], steroids and iron, alcohol, or bile acids. Reduced mucosal blood flow and resultant hypoxia such as in trauma, burns [Curling's ulcers], stress, cocaine use, or sepsis is another basis for the mucosal damage. Damage is due to a combination of multiple factors with antineoplastic chemotherapy including direct mucosal injury, vomiting-induced injury, and immunosuppression with infection. Gastric and duodenal ulceration occurring in patients with severe central nervous system damage (Cushing's ulcers) is often considered in this group (Marrone and Silen 1984; Silen 1987). Crohn's disease and viral or fungal infections are other identifiable causes. However, up to 50% of patients found to have gastric or duodenal erosions have no apparent predisposing factors (Levine 2000a).

The acute mucosal injury disrupts the normal protective barrier of epithelium, mucins, and bicarbonate. Acid, enzymes, and bile acids penetrate into the lamina propria, where they cause deeper injury (Davenport 1967; Oates and Hakkinen 1988). Inflammation is absent or only slight (consisting of only a few neutrophils). An additional factor in NSAID-induced acute gastropathy is the inhibition of prostaglandin production. Prostaglandins protect the mucosa by stimulating mucus and bicarbonate secretion, and maintaining mucosal blood flow (Robert et al. 1979).

Clinical Features

Acute gastropathy presents with abdominal discomfort or pain, heartburn, nausea, vomiting, and hematemesis. Bleeding occurs 3–7 days following the mucosal injury and can range from occult to massive hemorrhage. Conversely, patients may be asymptomatic.

Endoscopic Features

There will be multiple petechial hemorrhages and small red or black erosions on endoscopy (Sloan 1989; Sugawa et al. 1973). Gastropathy due to NSAIDs and alcohol involves the entire stomach, the lesions most evident in the antrum (Sugawa et al. 1973). Stress-related lesions (Curling's ulcers) usually occur in the proximal stomach (Sugawa et al. 1973). Hemorrhagic gastropathy may go on to develop gastric or duodenal ulceration, most commonly in shock-induced cases. The ulcers are usually multiple and shallow, usually in the body and fundus. The gastric ulcers commonly bleed when they extend beyond the muscularis mucosae (Lev et al. 1971; Marrone and Silen 1984; Silen 1987; Sugawa et al. 1973).

Radiology

Erosions are difficult to detect with single contrast barium studies but has become a frequent finding with double contrast studies, incidence up to 20% reported (Levine 2000a).

Two types of erosions are recognized. The more common is the *complete*, or *varioliform* erosion. These lesions appear as punctuate or slitlike barium collections surrounded by a small lucent mound of edema (Figs. 26.1 and 26.2a, b). The lesions are shallow and only a few millimeters in size each (Fig. 26.3). Lesions on the dependent or posterior surface may be better visualized with the use of barium flow techniques. They usually line up along the crests of thickened rugal folds (Fig. 26.4). The less common *incomplete*, or *flat* erosions account for only 5% of lesions detected. These appear a linear streaks or dots of barium collections without surrounding mucosal swelling (Levine 2000a).

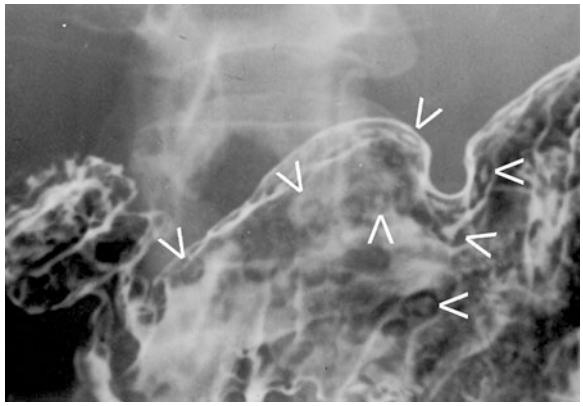

Fig. 26.1 Erosive gastritis. A 44-year-old woman with nausea and vomiting. Double contrast barium meal shows numerous gastric antral erosions as tiny shallow collections of barium, each surrounded by a halo of edema (*arrowheads*)

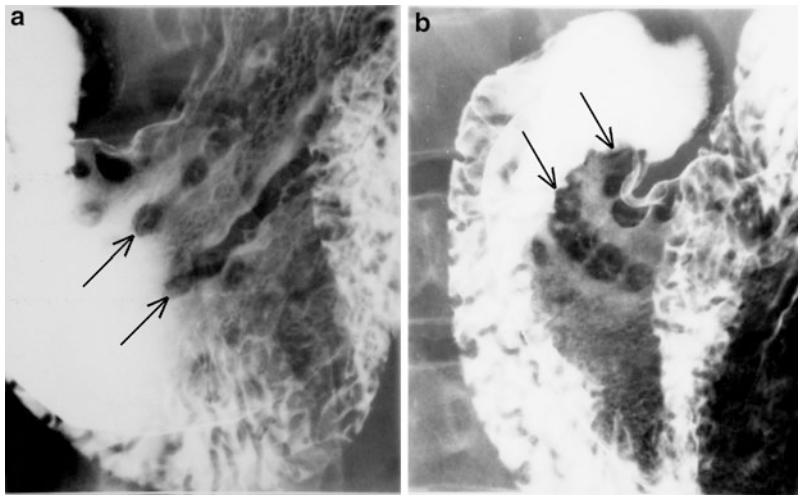

Fig. 26.2 (**a**, **b**) Erosive gastritis. A 66-year-old woman with epigastric discomfort, after commencing one Ecotrin every other day. Barium meal shows erosive gastritis with linear erosions surrounded by mounds of edema, lined up along rugal folds (*arrows*)

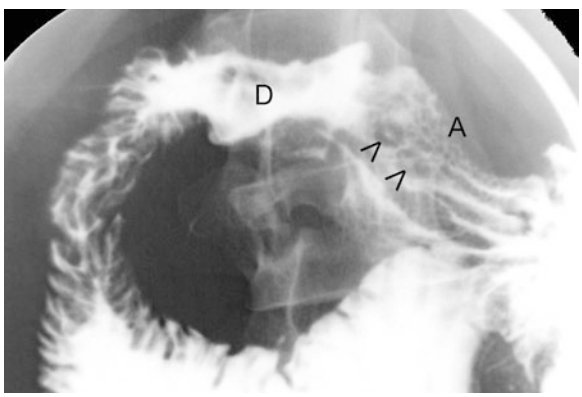

Fig. 26.3 Erosive gastritis. A 41-year-old woman with recurrent dyspepsia despite H2 blocker treatment. Barium meal shows inflammatory changes comprising coarse areae gastricae (A), antral erosions (*arrowheads*) and duodenal (D) fold thickening. *Helicobacter gastritis* identified endoscopically

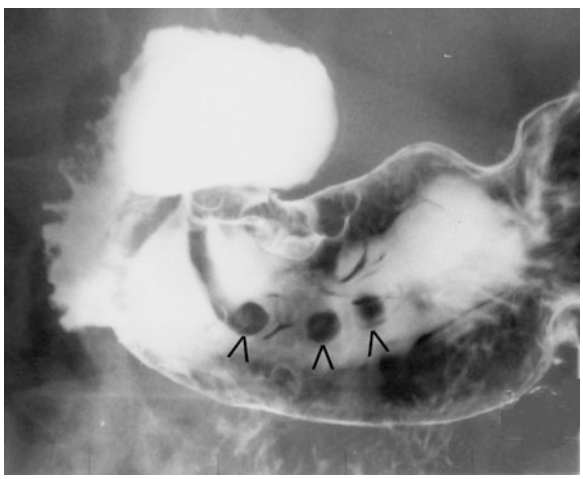

Fig. 26.4 Erosive antral gastritis. A 46-year-old woman with epigastric pain. A year ago she developed this pain following a course of Ibuprofen, with erosive gastritis then diagnosed. Follow-up barium meal shows 0.5 cm nodules (*arrowheads*) along the folds of the gastric antrum, almost polyp like in shape

Radiation Gastritis

In acute disease, the stomach suffers necrosis of chief and parietal cells and mucosal thinning, edema, and inflammatory infiltration (Fig. 26.5). There is an erosive or ulcerative gastritis, dysmotility with dilation, and gastroparesis (Goldgraber et al. 1954; Sell and Jensen 1966). Symptoms generally settle 1–2 weeks following completion of therapy. The effects are exacerbated by the concurrent administration of chemotherapy.

In chronic disease, 1–12 months after irradiation the mucosa becomes atrophic with intestinal metaplasia. Smoothened mucosal folds and mucosal atrophy are visible on endoscopy. Ulceration may occur on average 5 months after irradiation.

Months after irradiation scarring may result in radiographically evident antral stenosis and gastric outlet obstruction (Coia et al. 1995).

The threshold dose at which irradiation of the entire stomach results in later complications is estimated to be 50–60 Gy. The risk of late effects appears to be increased when larger dose fractions are used or when the patient has had prior abdominal surgery (Gunderson et al. 1983).

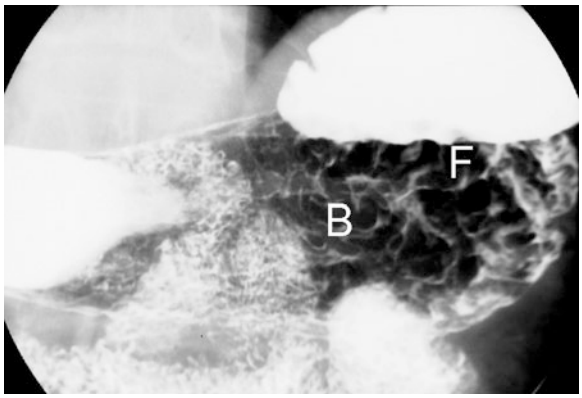

Fig. 26.5 Radiotherapy – thick folds. A 65-year-old man with gastric lymphoma in remission. Barium meal shows diffuse lobulated rugal fold thickening in the fundus (F) and proximal body (B). Biopsy found no evidence of lymphoma

AIDS-Related Disease

The HIV positive patient who develops an inflammatory disorder of the stomach may present with substernal and/or epigastric burning pain. Most patients are initially treated with a proton pump inhibitor for the symptoms of nonspecific dyspepsia. If there is no prompt response, further evaluation for possible mucosal lesions should then be undertaken. Unlike the general population, *H. pylori* has been infrequently found in HIV-infected patients with gastroduodenal ulcers (Varsky et al. 1998). If the patient's CD4 count is less than 50 cells, CMV disease should be considered. A definitive diagnosis is made by endoscopy with biopsy (Fig. 26.6).

Cytomegalovirus disease of the gastrointestinal tract is an uncommon but serious complication of AIDS. Five percent of patients with AIDS suffer GI involvement, particularly those with the worst degree of immunosuppression. The esophagus and colon are the most commonly involved sites. CMV gastritis presents with substernal and/or epigastric burning pain. Hemorrhage and perforation are recognized complications of CMV gastritis (Vanegas et al. 2000). On endoscopy, the disease is characterized by the presence of mucosal ulcers or erosions, and on biopsy histologic evidence of tissue destruction with presence of intranuclear or intracytoplasmic viral inclusion bodies with an inflammatory reaction at the edge of the ulcer (Goodgame 1993). Treatment requires a 6-week course of antiviral agents such as ganciclovir.

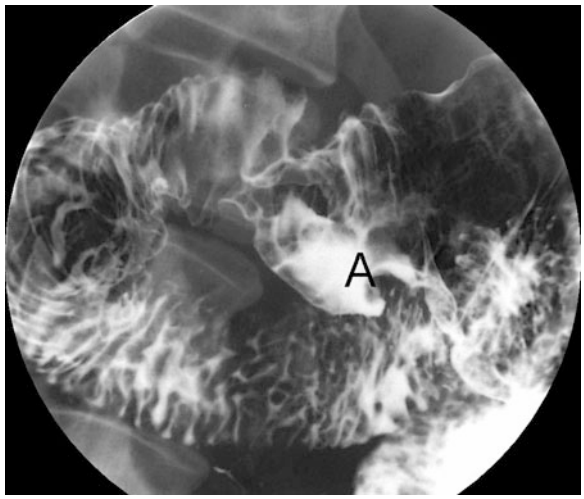

Fig. 26.6 CMV gastritis. A 45-year-old man with AIDS and 4 months epigastric pain. Barium meal shows thickened lobulated folds in the antrum (A). Suspicious for malignancy such as lymphoma. Endoscopic biopsy however revealed CMV gastritis

Gastritis in End-Stage Renal Disease

Dyspepsia, the nonspecific symptom that suggests upper gastrointestinal tract disease, occurs frequently in 10–20% of patients with end-stage renal disease (ESRD) (Hammer et al. 1998). In this study, however, the prevalence of dyspepsia in dialysis patients is not substantially different from the general population.

Gastritis (Fig. 26.7), gastric ulceration, and angiodysplasia are the most frequent causes of upper GI bleeding in patients with end-stage renal disease. Hemorrhagic gastritis in dialysis patients is due principally to the general effects of uremia (platelet dysfunction) and to therapy with nonsteroidal anti-inflammatory drugs (NSAIDs). The risk of hemorrhage is also increased due to therapy with heparin and aspirin (Chalasani et al. 1996; Gaspari et al. 1987).

Thickened Folds

The normal gastric mucosa is recognized by the presence of longitudinally aligned rugal folds usually less than 5 mm thick or high and of smooth symmetrical appearance. The normal duodenal fold pattern is transverse and thinner.

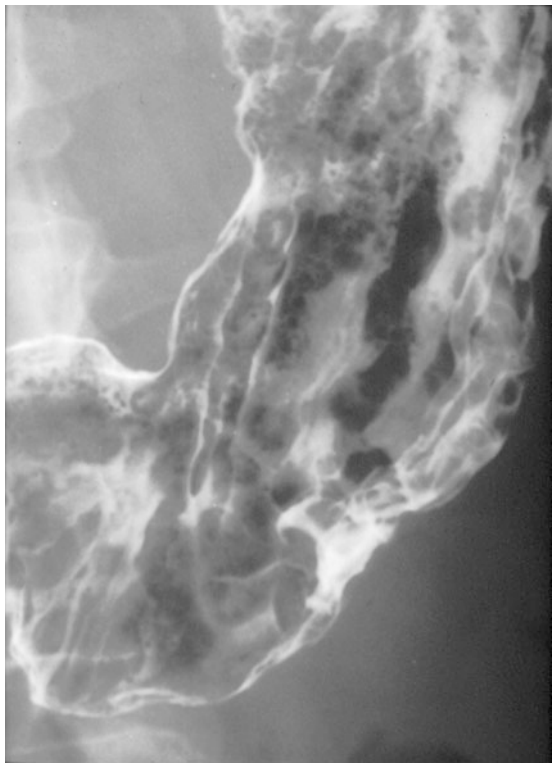

Fig. 26.7 Uremic gastritis. A 44-year-old woman with chronic glomerulonephritis and renal failure, nausea and vomiting. Barium meal shows thick nodular gastric folds

For a radiologist, the first diagnostic step in making an observation of thickened folds is to ensure that it is not an artifact of inadequate distension of the segment of interest. This holds true for both barium studies and for CT where such an observation may be made. Problem solving can be achieved with the administration of antispasmodics and positive or negative intraluminal contrast of sufficient volume to adequately distend the bowel.

Once the diagnosis of thickened folds is confirmed as a real finding, the underlying cause of the large mucosal folds cannot be determined from gross endoscopic or radiologic observation alone. A variety of proliferative, inflammatory, and infiltrative conditions are associated with enlarged mucosal folds in the stomach (Yardley and Hendrix 1995). These conditions have a similar appearance despite primary involvement of the mucosa, submucosa, or both in specific conditions. Of greatest concern is the possibility of infiltrative malignancies such as carcinoma or lymphoma as the etiology.

Gastritis due to *Helicobacter pylori*

Helicobacter pylori infection will cause both an acute and a chronic gastritis in most patients. This is the most common cause of gastric fold thickening (Levine 2000a).

Acute *Helicobacter pylori* Gastritis

Helicobacter pylori has been experimentally proven to cause an acute gastritis in healthy volunteers. Ingestion of the organisms resulted in a mild illness consisting of epigastric pain, nausea, and vomiting with gastric biopsy showing acute inflammatory changes (Marshall et al. 1985).

Histopathologic features of acute *Helicobacter 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. Acute gastritis almost always evolves into active chronic gastritis unless appropriate treatment is initiated.

H. pylori organisms are found primarily in the unstirred layer of gastric mucus, adjacent to epithelial cells at the mucosal surface, and in gastric pits. The organisms can be detected in both the antrum and the body of the stomach in the majority of infected patients (Paull and Yardley 1989). Biopsy will reveal the typical spiral shaped bacilli. During treatment, the bacteria may assume new forms, including U-shaped, rod-like, and coccoid forms.

Endoscopic Features

In the early stages of the disease, the gastric antrum is preferentially and diffusely involved with minimal involvement of the gastric body. This early stage is associated with an exaggerated gastrin response. This results in an increase in acid secretion which goes on to cause duodenal ulceration (Graham et al. 1991). The endoscopic appearance of acute *H. pylori* gastritis is variable. In severe cases, the thickened folds can resemble Menetrier's disease, lymphoma, or carcinoma (Herz et al. 1992; Stolte et al. 1993).

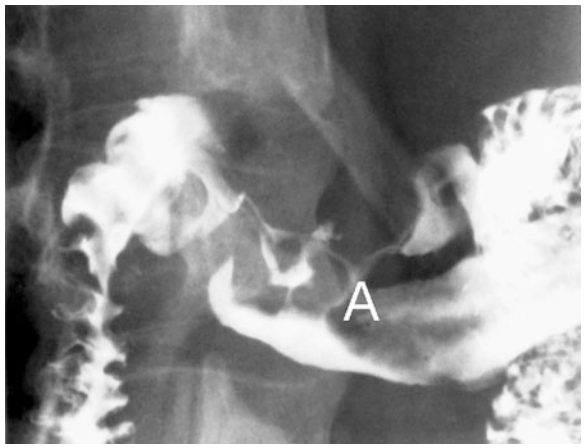

Fig. 26.8 Helicobacter gastritis – antral fold thickening with ulcers. A 41-year-old man with epigastric pain and reflux symptoms. Barium meal shows lobulated thickening gastric antral (A) rugal folds with ulceration

Radiology

The most common manifestation on double contrast barium studies is fold thickening. This is usually localized to the gastric antrum and distal body (Fig. 26.8). However, the distribution can be atypical in some patients, with either a proximal or a diffuse distribution (Fig. 26.9). The fold thickening can be quite marked, focal resembling a mass, or polypoid (Fig. 26.10). This appearance is indistinguishable from hypertrophic gastritides or infiltrative malignancy such as carcinoma or lymphoma. When fold thickening is marked, it is detectable on CT (Fig. 26.11). The other recognized manifestation of *H. pylori* gastritis is enlargement of the areae gastricae (greater than 3 mm in diameter) (Figs. 26.12 and 26.13). This had previously been observed in association with hypersecretory states and duodenal ulceration. It is likely that this association had been related to the presence of underlying *H. pylori* gastritis (Levine 2000a).

Chronic *Helicobacter pylori* Gastritis

In contrast to acute *Helicobacter gastritis* which is typically associated with duodenal ulceration, chronic *Helicobacter gastritis* is typically associated with extensive gastritis, intestinal metaplasia, and hypochlorhydria with risk of developing gastric ulcers and gastric cancer and MALT lymphoma (Correa 1992).

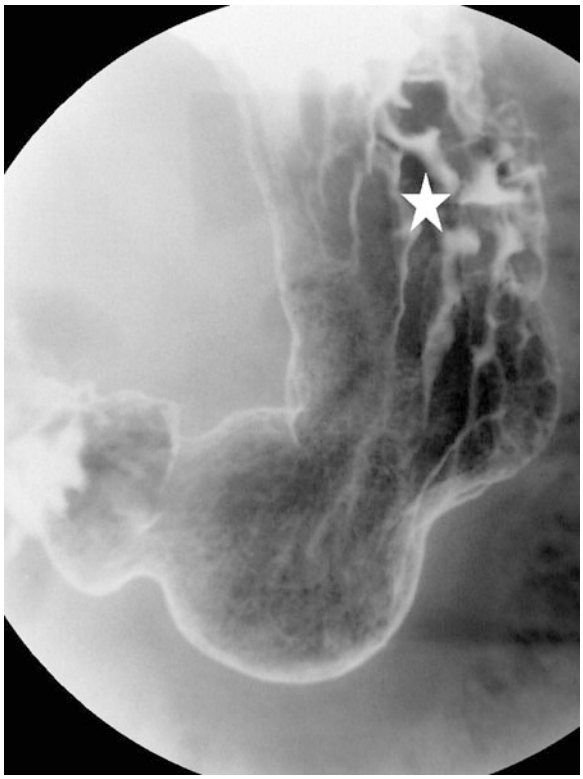

Fig. 26.9 *Helicobacter gastritis* – proximal fold thickening. A 43-year-old woman with epigastric dyspepsia. Barium meal shows diffuse nodular gastric fold thickening with a proximal distribution (*star*)

With chronic infection, involvement spreads to the gastric body. The mucosa becomes atrophic with the development of intestinal metaplasia (Vaananen et al. 2003). Gastrin producing cells are gradually lost, resulting in a fall in acid secretion.

Histopathologic features of chronic *Helicobacter pylori* gastritis consist of chronic inflammatory cells concentrated in the upper part of the mucosa. The inflammatory cells consist of lymphocytes, plasma cells, scattered macrophages, and often eosinophils (Karttunen et al. 1987). Lymphoid follicles are frequently present.

Iron deficiency anemia (without blood loss from the gastrointestinal tract or other sources) has been recognized in association with *H. pylori* gastritis. The pathogenesis is not well understood, but may relate to the organism's dependence upon iron as a growth factor or the presence of *H. pylori*-associated gastric atrophy. The anemia will be refractory to oral iron treatment and usually reverses with eradication of *H. pylori*

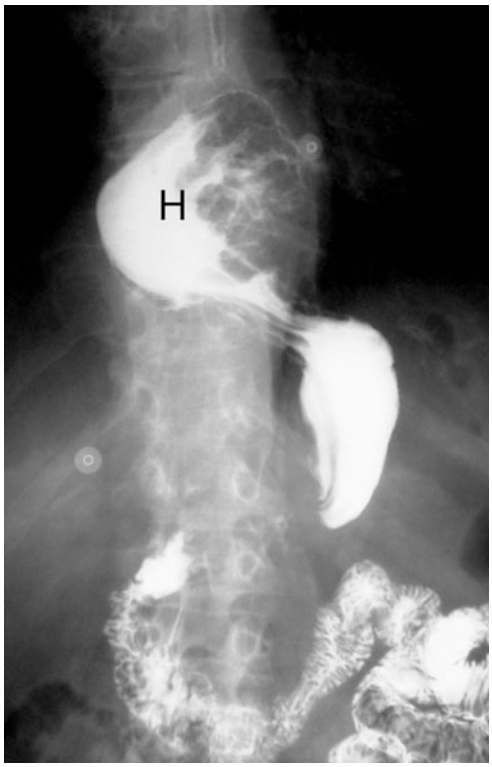

Fig. 26.10 *Helicobacter gastritis* – hiatal hernial pseudotumour. A 80-year-old woman with a pulmonary embolus and no identifiable source or cause for clot formation. Occult malignancy was a clinical concern. CXR had shown a retrocardiac mass. Barium meal shows a large sliding hiatal hernia (H) with half the stomach in the thorax, the cause of the retrocardiac mass. Within the hernia, there a 5 cm lobulated mass arising from the left posterior surface of the fundus just above the hernial neck. Although this could simply be a pseudotumor as a result of redundant mucosa due to the herniation, a true tumor must be considered. Endoscopy confirmed a mass of erythematous folds at this location. Biopsy revealed *Helicobacter gastritis*

infection (Annibale et al. 1999; Cardenas et al. 2006; Hershko et al. 2005; Yakoob et al. 2003).

Hypertrophic Gastritis

Hypertrophic gastritis, also known as hypertrophic hypersecretory gastropathy, is a condition of usually unknown etiology. It may be caused by pituitary, hypothalamic, or vagal stimuli. There is marked gastric glandular hyperplasia and increased acid secretion. Gastric folds are thickened by the glandular hyperplasia as well as edema and inflammation. Most patients

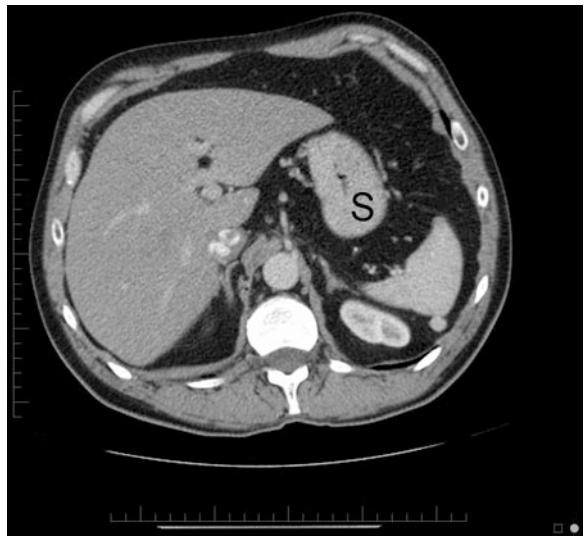

Fig. 26.11 *Helicobacter gastritis* – fold thickening visible on CT. A 48-year-old man with fatigue. On CT, the stomach (S) appears diffusely thick walled especially in its proximal half. The wall measures up to 25 mm in thickness

will have associated duodenal ulceration; some patients will also suffer gastric ulceration (Levine 2000a).

Radiology

On barium studies and on cross-sectional imaging, gastric folds are thickened and lobulated. This usually occurs in the fundus and body because this is the acid-secreting portion of the stomach. The diagnostic difficulty posed by this condition is the wide range of differential diagnoses that appear similar. *H. pylori* gastritis, Menetrier's disease, and lymphoma are the major considerations. Other considerations include gastric carcinoma, Zoliinger–Ellison syndrome, eosinophilic gastritis, and varices (Levine 2000b).

Menetrier's Disease

Menetrier's disease (protein-losing hypertrophic gastropathy) is a rare hyperplastic gastropathy. It is seen most commonly in middle-aged males. The pathogenesis is a proliferation of epithelial cells of the mucous cell compartment. The etiology is unknown although cytomegalovirus (CMV) is believed to play

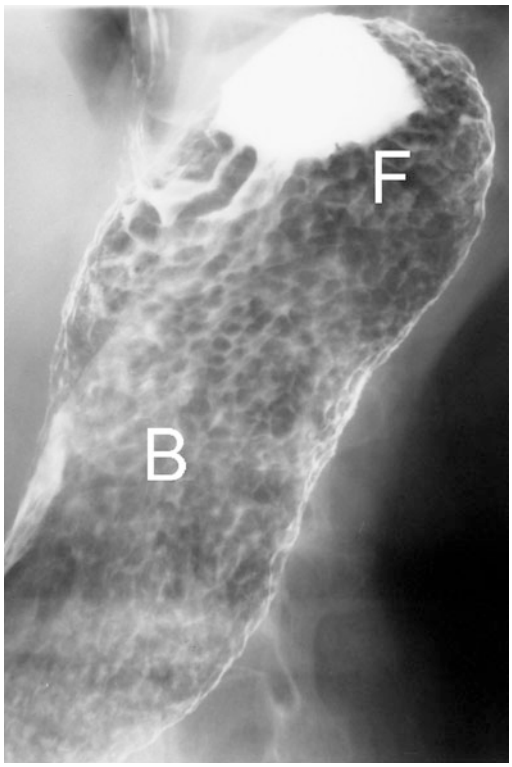

Fig. 26.12 *Helicobacter gastritis* – coarse areae gastricae. A 69-year-old woman with a history of mild reflux and more recently, colicky abdominal pain with some relief from antacids. Barium meal shows coarse areae gastricae involving the fundus (F) and upper body (B) of the stomach

a part in the pediatric form of this disease (Coffey et al. 2007; Toubia and Schubert 2008).

Patients with Menetrier's disease most commonly present with epigastric pain. However, a variety of presentations is recognized including weight loss, nausea, vomiting, diarrhea, gastrointestinal bleeding, protein loss (Meuwissen et al. 1992). Other features include edema, ascites, pleural, and pericardial effusions. There is a small increased risk of the patient developing gastric adenocarcinoma in the order of 2–15% (Scharschmidt 1977; Sundt et al. 1988).

Radiology

Radiology – shows striking thickening of gastric rugal folds. The enlarged folds are most commonly confined to the body and fundus with a symmetrical pattern of

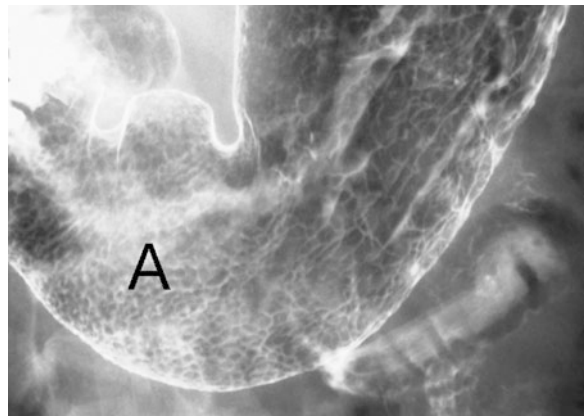

Fig. 26.13 *Helicobacter gastritis* – coarse areae gastricae. A 41-year-old woman with dyspepsia. Barium meal shows coarsening of antral (A) areae gastricae

enlargement. Occasionally the fold thickening may be asymmetrical and rarely a polypoid appearance may be encountered.

Diagnosis is made on biopsy of the thickened gastric folds. There is extreme foveolar hyperplasia with glandular atrophy. A full thickness biopsy is usually required (Sundt et al. 1988; Wolfsen et al. 1993).

Atrophic Gastritis

The term "atrophic gastritis" is used to describe a chronic gastritis that, in addition to inflammation, is associated with mucosal thinning, gland loss, and changes in epithelial cell types (i.e., metaplasia). Metaplasia is highly relevant to the pathogenesis of atrophic gastritis and to its complications (e.g., pernicious anemia, gastric ulcer, and gastric cancer). Two main subtypes, autoimmune and environmental are recognized. The autoimmune and environmental forms of metaplastic atrophic gastritis are pathologically, pathogenetically, and clinically distinct, but they share histologic features.

Chronic atrophic gastritis leads to a decline in Intrinsic Factor production and contributes to pernicious anemia. The gastritis is associated with autoantibodies directed against the gastric parietal cells (Toh et al. 1997). The chronic atrophic gastritis in pernicious anemia is associated with an increased risk of intestinal-type gastric cancer and of gastric carcinoid tumors.

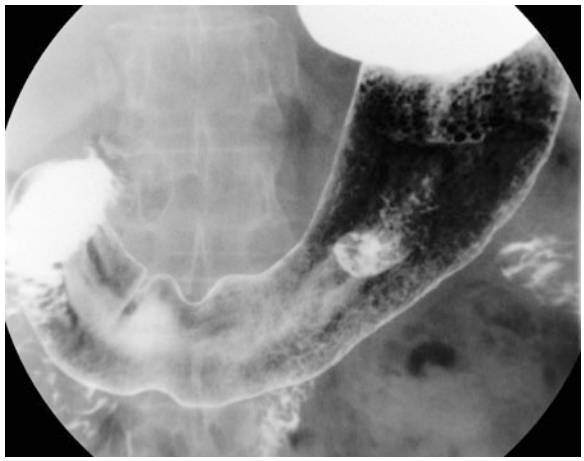

Fig. 26.14 Atrophic gastritis. A 57-year-old woman with reflux symptoms and previous peptic ulcer disease. Barium meal shows a stomach that is diffusely narrowed and tubular with decreased rugal folds and absent areae gastricae

The latter are presumably due to prolonged achlorhydria resulting from parietal cell loss, compensatory hypergastrinemia, and argyrophilic cell hyperplasia.

Radiology

Atrophic gastritis is suggested on barium studies by a stomach that is narrowed and tubular with decreased or absent mucosal folds and areae gastricae (Fig. 26.14). Changes are mainly seen in the body and fundus. Imaging is however neither sensitive nor specific. Changes are seen in only 50% of patients with pernicious anemia. Conversely, 10% of patients with these findings do not have this diagnosis, false-positives.

Autoimmune Metaplastic Atrophic Gastritis

Autoimmune metaplastic atrophic gastritis (AMAG) is an inherited form of metaplastic atrophic gastritis that is associated with an immune response in the oxyntic mucosa directed against parietal cells and intrinsic factor. An association with the major histocompatibility haplotypes HLA B-8 and DR-3 has been observed (Kekki et al. 1987). In a study of patients with iron deficiency anemia, refractoriness to oral iron treatment was noted in 70% of patients with autoimmune atrophic gastritis (Hershko et al. 2005).

Clinical Features

AMAG is inherited as an autosomal dominant disorder that commonly affects people of northern Europe or northern European extraction (Kekki et al. 1987). Women are more commonly affected with a ratio of 3:1 (Varis et al. 1979). Affected patients are at high risk for developing pernicious anemia (Siurala et al. 1980). The autoimmune origin of this disease is indicated by the presence of elevated serum antibodies to parietal cells and to intrinsic factor. The severity of the chronic inflammation, gland atrophy, and epithelial metaplasia seen correlates with the levels of these antibodies. The resultant in lack in intrinsic factor leads to vitamin B12 malabsorption and pernicious anemia.

Pathology

The changes of metaplasia, glandular atrophy, and inflammation occur in the gastric body and fundus in patients with AMAG. In the early stage of active disease, the gastric body and fundus may appear to contain multiple polyps. These are not true neoplasms, they are retained islands of uninvolved mucosa within the broad area of atrophy and metaplasia. In advanced or end-stage AMAG, examination of the stomach by endoscopy or contrast radiography demonstrates absent or inconspicuous rugae in the gastric body and fundus. Atrophy and metaplasia usually do not involve the antrum (Stemmermann and Hayashi 1968). These patients are at risk of developing gastric carcinoid tumors and adenocarcinoma.

Environmental Metaplastic Atrophic Gastritis

Environmental metaplastic atrophic gastritis (EMAG) is similar to AMAG but is due to environmental factors, such as diet and *H. pylori* infection, on the gastric mucosa. The mucosal changes occur in both the corpus and antrum in a multifocal distribution, but with heaviest involvement of the antrum. Furthermore unlike AMAG, gastric acid production does not disappear entirely, serum gastrin is not elevated and parietal cell and intrinsic factor autoantibodies and pernicious

anemia are absent. There is an increased risk for gastric ulcer and an increased risk for gastric cancer (Antonioli 1990).

Pathology

The pathology of EMAG are multiple, foci of atrophy, metaplasia, and inflammation. These changes are most heavily concentrated in the antrum, and more evident along the lesser curvature at the junction of the body and antrum (i.e., the transitional zone) in patients with mild or early disease. In severe or advanced disease, metaplastic epithelium can almost completely replace the normal antral mucosa (Stemmermann and Hayashi 1968).

Granulomatous Gastritides

A granulomatous gastritis is one of a diverse group of chronic gastritides characterized by the presence of granulomatous inflammation. A granuloma is an organized aggregation of combined histiocytic, lymphocytic, and plasma cell infiltrate. This group of conditions is usually subclassified into infectious, noninfectious, and idiopathic categories. Granulomatous gastritides are uncommon contributing less than 0.5% of gastritis cases (Ectors et al. 1993).

In developed countries, the most common causes are Crohn's disease (Fig. 26.15) and sarcoidosis, while in developing countries, infectious causes are the norm, particularly infection with *Mycobacterium tuberculosis* (Kamani et al. 2008).

Granulomas can occur as an inflammatory response to bacteria, fungi, and parasites. Other infections that have been recognized to cause granulomatous gastritis include *Helicobacter pylori*, tertiary syphilis (gumma), Whipple's disease, various fungal infections, anisakiasis, taeniasis, other parasitic worms, and schistosomes.

A variety of noninfectious causes of granulomatous gastritis have been described. These causes have included upper GI tract tumors (adenocarcinoma and lymphoma), vasculitides (e.g., Wegener's granulomatosis (Fig. 26.16)), drugs (e.g., cocaine and

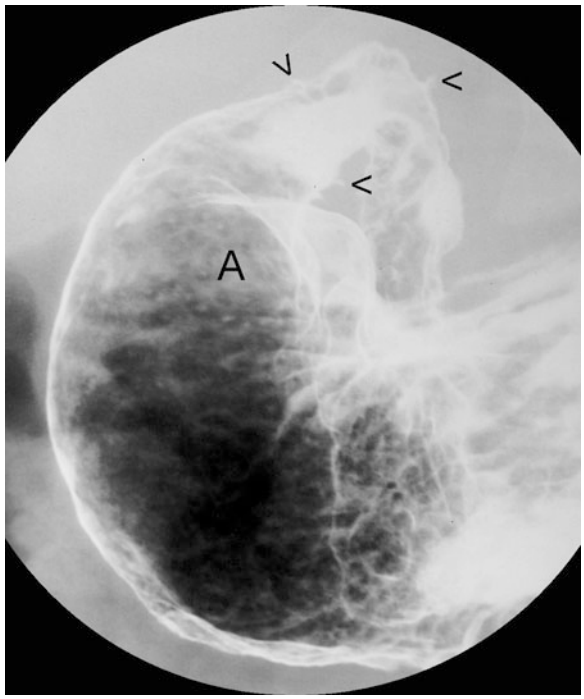

Fig. 26.15 Acute Crohn's disease. A 34-year-old man with crampy abdominal pain and diarrhea. Barium meal shows narrowing of the gastric antrum (A) with multiple punctuate collections of barium and surrounding halos of edema. The duodenal bulb is non-distended, with ulcerations (*arrowheads*) in the bulb and second part of the duodenum

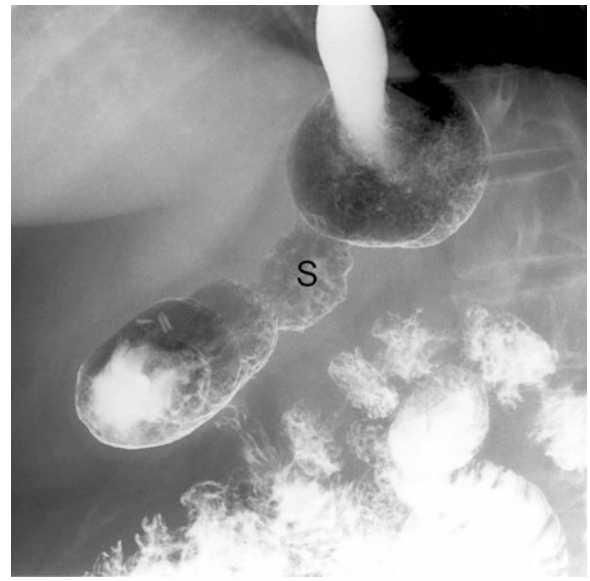

Fig. 26.16 Wegener's granulomatosis. A 51-year-old woman with known Wegener's Disease for several years presents with dysphagia and regurgitation of food. Barium meal shows that the gastric cardia and fundus has relatively featureless mucosa. There is a mid gastric body stricture (S). The stricture extends over a length of 5 cm with the lumen reduced to a caliber of 2.5 cm

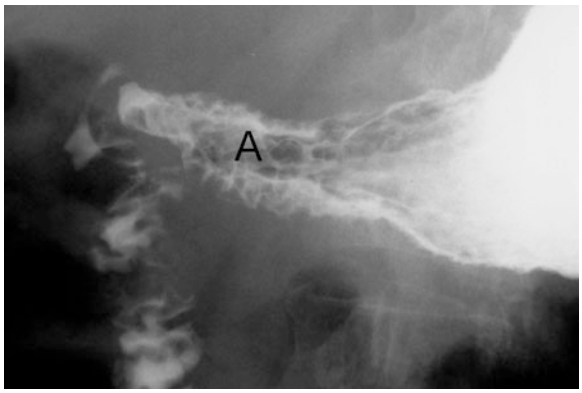

Fig. 26.17 Chronic Crohn's disease. A 84-year-old woman with several episodes of nausea, vomiting, and diarrhea. Barium meal shows abnormal gastric antrum (A) which is nondistensible with marked mucosal edema and intervening fissures. The process extends into the pylorus and duodenal bulb

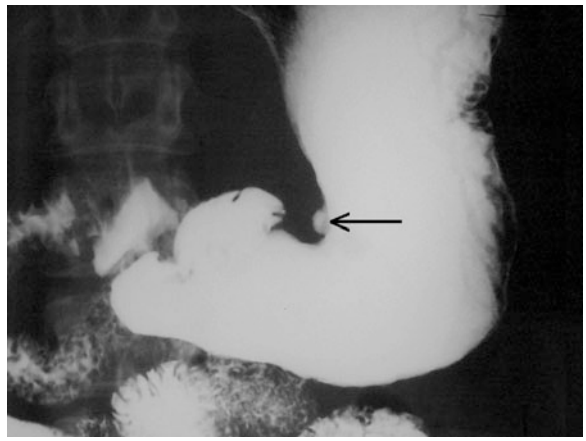

Fig. 26.18 Sarcoid ulceration. A 40-year-old woman with nausea and dyspepsia. Five year history of pulmonary sarcoid without progression with stable hilar and AP window adenopathy. Barium meal shows a small lesser curve antral ulcer (*arrow*) which appears excavated suggesting benignity. Endoscopic biopsy revealed sarcoid granulomas at the ulcer crater

carbimazole), xanthogranulomatous gastritis, foreign body reactions, and Langerhans cell histiocytosis.

Granulomas may be classified histologically into noncaseating, caseating, or necrotizing types. This is useful as it narrows the possibilities of etiology for the gastritis. Caseation necrosis is usually associated with an infectious etiology which may be fungal or bacterial (*Mycobacterium tuberculosis*, atypical mycobacteria, histoplasmosis). Well-defined epithelioid granulomas with a circumscribed solid appearance are commonly seen in sarcoidosis. Noncaseating granulomas are seen with *Helicobacter pylori* and Crohn's disease (Fig. 26.17). Necrotizing granulomas, especially if associated with vasculitis, suggest Wegener's granulomatosis.

Sarcoid Disease

Sarcoidosis is a systemic granulomatous disease of unknown etiology, characterized by the formation of noncaseating granulomas. Subclinical gastrointestinal tract involvement occurs in 5–10% of patients with systemic sarcoidosis while symptomatic disease has been reported in less than 1% (Vahid et al. 2007). The stomach is the most commonly involved segment of the GI tract. The disease results either in peptic ulceration or to narrowing of the gastric lumen due to granulomatous inflammation and associated fibrosis of the gastric wall. Ulceration may lead to potentially fatal hemorrhage.

Histological diagnosis from endoscopic biopsy is often difficult. The histologic finding of noncaseating granulomas will be similar to Crohn's disease, Whipple's disease, tuberculosis, fungal infections, syphilis, and foreign body reactions. The diagnosis is usually made by recognition of the clinical and pathologic evidence of multisystem involvement by sarcoidosis.

Radiology

The most common abnormality described on upper GI barium series is segmental mucosal thickening, poor peristalsis and nondistensibility that mimics the linitis plastica variety of gastric cancer. Deformity of the antrum and ulceration may also be seen (Fig. 26.18) (Farman et al. 1997; Low and Heyneman 1999).

Chronic Chemical Gastropathy

Long-term exposure to agents that damage the gastric mucosa may lead to a chronic chemical gastropathy. This is more appropriately described as

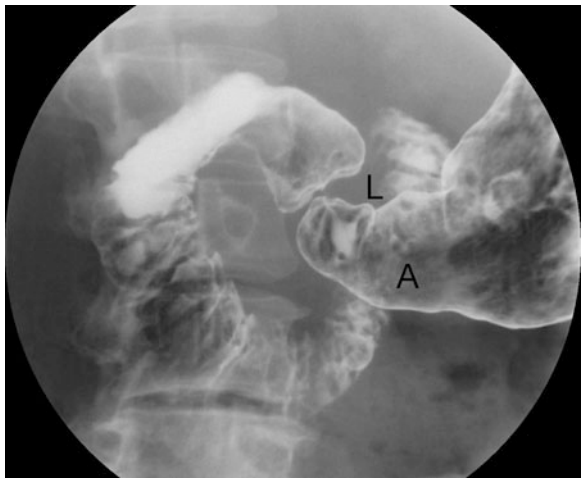

Fig. 26.19 Chronic NSAID gastropathy. A 45-year-old woman with recurrent episodes of epigastric pain, associated with NSAID ingestion. Barium meal shows deformed gastric antrum (A) with flattening of the lesser curvature (L) and inconspicuous areae gastricae

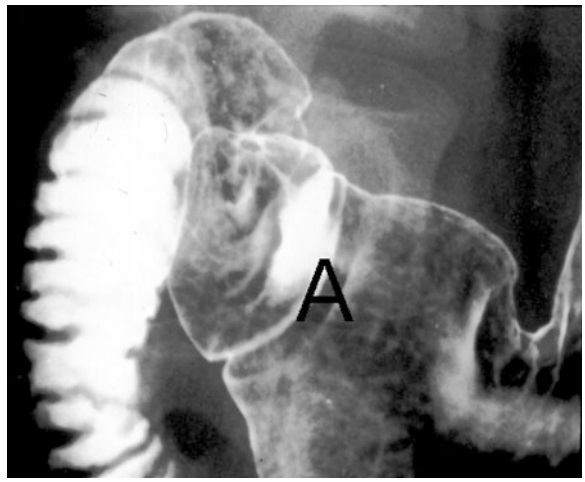

Fig. 26.21 Transverse antral fold. A 45-year-old woman on NSAIDs with several months of dyspepsia. Barium meal shows an antral (A) transverse fold indicating old antral gastritis and scarring

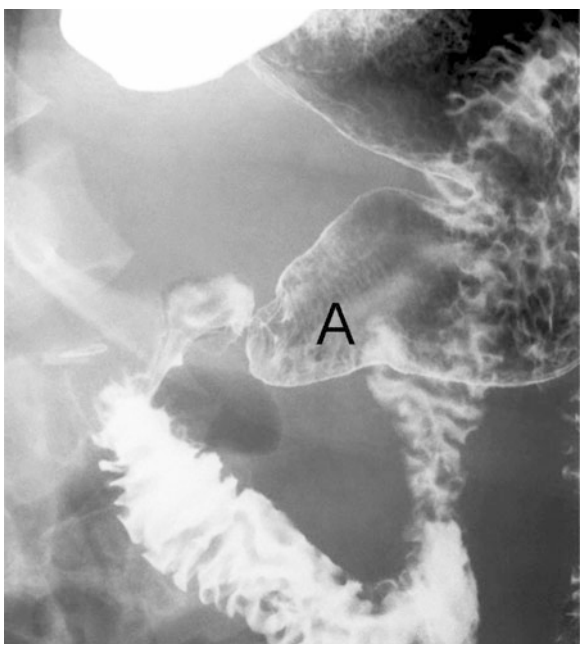

Fig. 26.20 Antral striae. A 40-year-old woman with chronic dyspepsia. Barium meal demonstrates gastric antral (A) striae due to chronic gastropathy and scarring

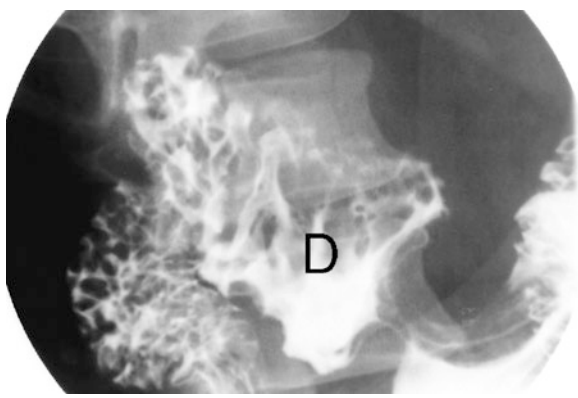

Fig. 26.22 Duodenitis – bulbar erosions and thickened folds. A 44-year-old woman with chronic RUQ pain. Barium meal shows nodular thickening of duodenal (D) bulbar folds. Punctate collections of barium are visible on some of the "nodules" due to erosions

a gastropathy rather than a gastritis because there is a lack of inflammatory changes. Instead there is foveolar hyperplasia, edema, proliferation of smooth muscle fibers in the lamina propria, and vascular dilatation, and congestion. This condition was first recognized in patients with severe bile reflux (Dixon et al. 1986). A variety of medications including NSAID, potassium, and iron, and most recently alendronate have been implicated (Abraham et al. 1999; Graham et al. 1997; Laine et al. 1988; McMahon et al. 1982; Sobala et al. 1990). Chronic

Fig. 26.23 Duodenitis – postbulbar thickened folds. A 38-year-old man with vomiting 5 minutes after eating. Barium meal shows duodenitis as thickened folds of the postbulbar duodenum (D)

exposure to alcohol (ethanol) correlates with presence of hemorrhagic and erosive lesions, (Bernersen et al. 1992; Laine and Weinstein 1988; Segawa et al. 1987). However, it is uncertain if alcohol cause chronic chemical gastropathy (Cheli et al. 1981; Oddsson et al. 1978; Uppal et al. 1991).

Clinical Features

There are no specific symptoms and no endoscopic findings that are diagnostic. Observed changes are usually located at the gastric antrum (Figs. 26.19, 26.20, and 26.21) and the diagnosis is made from the endoscopic biopsy.

Duodenitis

Duodenitis is usually considered to represent a component of peptic ulcer disease. Many patients have *H. pylori* infection contributing to the disease process. Duodenitis usually presents with upper gastrointestinal symptoms such as dyspepsia, epigastric pain, nausea, food intolerance, and early satiety. Occasionally bleeding occurs and there will be relevant signs and symptoms (Levine 2000a).

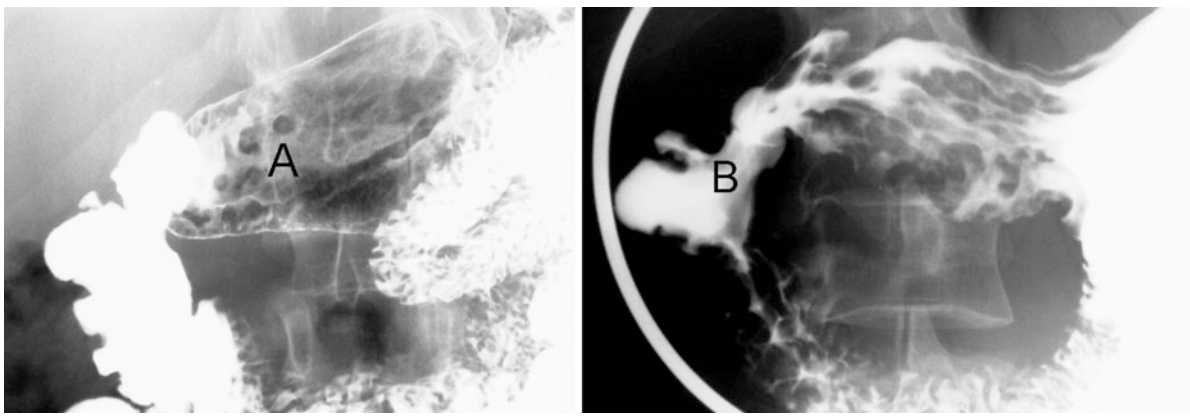

Fig. 26.24 (**a**, **b**) Erosive gastroduodenitis. A 35-year-old male with epigastric pain, nausea and vomiting. Barium meal shows gastric antral (A) disease with fold thickening where multiple round filling defects with central collections of barium are noted consistent with small (6–10 mm) erosions and surrounding edema. The duodenal bulb (B) never fully distends with sacculated contour indicating scarring

Radiology

Barium studies reveal changes mainly in the proximal duodenum. The involved segments are spastic and irritable with thickened nodular folds (Figs. 26.22 and 26.23). With double contrast technique, more subtle signs are detectable. Erosions may be seen in the duodenum bulb or less commonly in the descending segment. They are similar to that recognized in the stomach with the erosions seen as tiny flecks of barium surrounded by radiolucent halos of edematous mucosa (Levine 2000a) (Fig. 26.24).

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