

Gastritis: Classification, Pathology, and Radiology

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Abstract and Introduction

Abstracts

Gastritis has a broad pathologic spectrum and anatomic distribution, as well as an evolving etiology. The Sydney pathologic classification of gastritis, which appeared in 1990 and was revised in 1994, emphasized the importance of combining etiologic, topographic, and morphologic criteria for establishing clinically useful diagnoses. Using this revised Sydney pathologic classification as a guide, we report a simpler and more practical radiologic approach to gastritis. We emphasize those types of gastritis that may be detected on radiologic examination and illustrate typical findings.

Introduction

Gastritis remains a complex and often confusing topic for clinicians and radiologists. Pathologists, endoscopists, and radiologists have used various and often conflicting criteria for the diagnosis of gastritis. Problems include lack of histologic and endoscopic correlation of findings in "gastritis" and radiologic accuracy in evaluating the many confusing forms of this disease. The discovery of *Helicobacter pylori* has dramatically altered the concepts of etiology of a wide variety of gastric diseases, including peptic ulcer and nonimmune chronic gastritis. The updated Sydney System^[1] in this study used for the initial classification of gastritis combines these varied aspects of the disorder into a useful scheme for clinical diagnoses (Table 1).

Table 1. Revised Sydney Classification Combining Etiologic and Morphologic Criteria for Classifying Gastritis, recommends the following:

1. For antral and corpus biopsies to be assessed separately
2. For gastritis to be classified into:
 - a. Acute
 - b. Chronic
 - c. Special (e.g., lymphocytic, granulomatous)
3. For the following variables to be graded
 - a. *H. pylori*
 - b. Chronic inflammation
 - c. Neutrophils (as a sign of activity)
 - d. Atrophy
 - e. Intestinal metaplasia
4. A concluding summary is to be provided indicating the etiology (if known), topography (antrum corpus o pangastritis), and morphology (including all variables).

Gastritis can be classified into acute or chronic forms, and chronic types can be subclassified as nonatrophic, atrophic, and special types (chemical, radiation, lymphocytic, noninfectious, eosinophilic, and others).^[1,2] Etiologic and pathologic classification of gastritis should form the basis for developing a useful

radiologic approach to the evaluation and diagnosis of this disease and its many forms. The major goal of our review is to update the current classifications of gastritis, the impact of *H pylori* infection, and the potential role of radiology in the evaluation and diagnosis of some of these disorders.

Chronic Nonspecific Gastritis

Chronic nonspecific gastritis is not a single entity but a multifactorial disorder with ongoing injury to the gastric mucosa leading to chronic inflammation and gastric atrophy. Causes include hyperacidity, bile reflux, autoimmunity, and infection with *H pylori*. Chronic nonspecific gastritis is of two types, dependent on the gastric distribution. Type A gastritis involves the fundic region and may extend to the gastric body. Autoimmune atrophy related to antibodies against parietal cells,^[10] as in pernicious anemia, has been called type A disease, whereas the multifocal atrophic variety has been labeled type B. This type of gastritis involves the antral zone and is related to acid-peptic disease of *H pylori* origin.^[10] Chronic nonspecific gastritis may be further subclassified as superficial or atrophic gastritis, according to the depth of inflammation; however, these terms may cause more confusion than clarification.

Radiologic findings are often normal in this disorder. With more severe atrophic gastritis, a decrease in number or an absence of rugal folds ("bald fundus") occurs in the upper stomach^[14] (Fig 3). The contour of the stomach may become more tubular, with a distended fundus ("H-bomb sign"). Also, the folds along the greater curvature may become thin and crenated, and areae gastricae may be reduced in number and size^[14] (Fig 4). These changes may be seen in 81% of patients with atrophic gastritis but also have been reported in 11% of control group subjects.^[14]

Fig 2

Figure 2. *Helicobacter pylori* gastritis. Computed tomography in patient with dyspepsia shows (A) marked thickening of stomach (S) wall. Endoscopy verified fold thickening and *H pylori* infection. (B) Two months later after appropriate treatment, with gaseous and contrast distention of stomach, gastric wall appears normal. Other causes of gastric fold thickening, such as hyperplastic gastropathies and malignancies, must be considered on initial image (courtesy of Deborah Lucas, MD, Salisbury, NC).

Radiologic Evaluation

The radiologic evaluation and diagnosis of gastritis remains problematic because the pathologic classification is based on etiologic (eg, proof of *H pylori* infection) and histologic criteria that have no imaging correlation.^[3] Nonendoscopic gastric mucosal biopsy is a recent innovation used to augment the double-contrast upper gastrointestinal (UGI) tract examination and has improved radiologic accuracy in evaluating these disorders.^[4]

In general, the radiologic signs on a UGI study that suggest the diagnosis of gastritis have been nonspecific and often conflicting; these include (1) fold thickening; (2) loss of rugal folds; (3) contour and caliber changes; (4) antral alterations, such as narrowing; and (5) nodulation or erosions. A simplified classification for gastritis, based on current concepts and amenable to radiologic evaluation, includes erosive gastritis (acute and chronic); *H pylori* gastritis; chronic nonspecific gastritis; hyperplastic gastritis or gastropathy, including hypertrophic gastritis, Ménétrier's gastritis, and Zollinger-Ellison syndrome; and miscellaneous types, including granulomatous, phlegmonous, eosinophilic, corrosive, other infectious types, and rare types (Table 2).

Table 2. Simplified Gastritis Classification

Erosive gastritis (acute and chronic)

Helicobacter pylori gastritis

Chronic nonspecific gastritis

Hyperplastic gastritis or gastropathy

"Hypertrophic" gastritis

Ménétrier's disease

Zollinger-Ellison syndrome

Miscellaneous types

Granulomatous

Phlegmonous

Eosinophilic

Corrosive

Other infections

Rare types

Erosive Gastritis

Acute gastritis includes hemorrhagic or erosive gastritis, *H pylori* gastritis, and acute phlegmonous gastritis. Although erosive gastritis may result from various causes and may show acute or chronic changes pathologically, the radiologic hallmark is multiple erosions (Fig 1). The most common causes of erosions include ingestion of aspirin or other nonsteroidal anti-inflammatory drugs (NSAIDs), alcohol, stress, trauma, burns, viral or fungal infection, and Crohn's disease; however, up to half of patients with this disorder do not have apparent predisposing factors.^[5] *Helicobacter pylori* may cause an acute or chronic gastritis and is associated with peptic ulcers, but the relationship to erosions is uncertain.



Figure 1. Erosive gastritis. Double-contrast view of gastric antrum shows multiple nodules with central collections (arrowheads) of barium due to complete erosions. Erosions may also be observed on compression views.

The reported radiologic prevalence of erosive gastritis is 1.7% to 26%.^[6,7] In most patients, images show complete or varioliform erosions, which appear as punctate or linear barium collections surrounded by radiolucent halos of edematous mucosa. Double-contrast or compression radiologic views best reveal gastric erosions. These nodular erosions typically occur in the gastric antrum, often aligned on antral folds. Gastritis caused by aspirin and NSAIDs may appear as linear or serpiginous erosions in the body or near the greater curvature, because these are dependent portions of the stomach where the medication settles while dissolving.^[5,8] Erosions may be subtle; the primary finding may be thickening of the antral folds. If the shallow epithelial defects have healed, the only evidence is nodularity, which can mimic antral polyps from various causes. Most inflammatory nodules caused by gastritis are small and do not project clearly into the lumen. These nodules are usually less than 1 cm in diameter. The characteristic appearance of gastritis is that of inflammatory nodules lined up on the folds of the gastric antrum.

***Helicobacter pylori* Gastritis**

Helicobacter pylori was first isolated in 1983 and is now recognized as a major factor in the development of peptic ulcer disease, chronic gastritis, and, potentially, gastric malignancy.^[9] Infection with *H pylori* is ubiquitous. The prevalence of *H pylori* ranges from 60% to 80% in patients with gastric ulcer and 90% to 100% in those with duodenal ulcer.^[10] A common cause of gastric infection, *H pylori* is age-related; it is found in 24% of the population 20 to 39 years of age and in 82% of the population 60 years of age and older.^[11]

Most patients with peptic ulcers are infected with this organism. In one study, thickened folds were detected in 44% of patients with *H pylori*^[12] (Fig 2). The antrum was a common site affected, but the entire stomach may be involved.^[13] In addition to thickened antral mucosal folds, other possible signs include irregular contour of the lesser curvature, erosions, polypoid folds, ulcers (14%), nodularity, enlarged areae gastricae (16%), narrowing (9%), polyps (6%), and spasm.^[10,12] In a small percentage of patients without *H pylori* infection, findings may be similar. The role of radiographic imaging in evaluating *H pylori* gastritis warrants further study.

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Figure 3. Atrophic gastritis in patient with known gastric atrophy related to pernicious anemia. Absent rugal folds in upper stomach cause "bald fundus" sign.



Figure 4. Atrophic gastritis in patient with gastric atrophy with rugal fold paucity in fundus, thinned folds along greater curvature, and preserved areae gastricae.

Hyperplastic Gastropathy

Hyperplastic gastropathy is a pathologic term that includes hypertrophic gastritis, Ménétrier's disease, and Zollinger-Ellison syndrome. Several of these entities may mimic other forms of gastritis on radiologic examination and are included as differential considerations. The term "hypertrophic gastritis" is a misnomer, because rugal fold thickening due to hyperplasia and inflammation may be absent in these disorders. Thick folds can be defined as gastric folds more than 5 mm in caliber as measured on radiographs obtained with the stomach moderately distended.^[12]

The radiologic findings in these gastropathies are often similar. Marked thickening of gastric folds with a bizarre appearance is seen in the gastric fundus and along the greater curvature (Fig 5). Antral sparing may be a feature of Ménétrier's disease (Fig 6), but the validity of this finding has been debated. In

Zollinger-Ellison syndrome, peptic ulcers, hypersecretion, and enteric fold thickening may be additional radiographic features. Most importantly, hyperrugosity as a result of other causes, such as gastric lymphoma (Fig 7), carcinoma (Fig 8), and *H pylori* infection must also be considered in the differential possibilities.^[12]

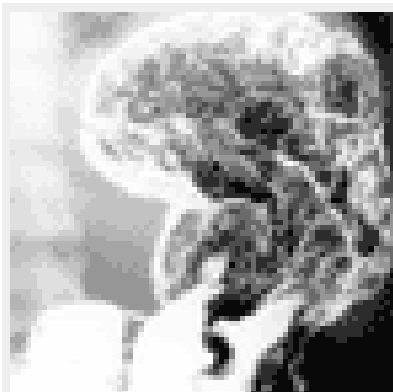


Figure 5. Thick, bizarre gastric folds in patient with endoscopic diagnosis of hypertrophic gastritis of uncertain cause.



Figure 6. Large folds along greater curvature of stomach in Ménétrier's disease.



Figure 7. Gastritis lymphoma. Diffuse fold thickening due to non-Hodgkin's lymphoma of stomach.



Figure 8. Gastric metastases. Polypoid fold thickening along greater curvature mimics Ménétrier's disease but is due to metastatic infiltration and peritoneal carcinomatosis from ovarian adenocarcinoma.

Miscellaneous Types

A heterogeneous assortment of other causes of gastritis includes infectious and noninfectious forms. Organisms other than *H pylori*, such as mycobacteria (tuberculosis, *Mycobacterium avium intracellulare*), *Treponema pallidum*, viruses (cytomegalovirus),^[15] fungi (candida), and parasites (*Cryptosporidium*, *Giardia lamblia*, *Anisakiasis*), can cause gastritis. Tuberculosis and syphilis were common in the early part of the last century; different organisms are implicated more recently, particularly in patients with the acquired immunodeficiency syndrome. Noninfectious causes are numerous and encompass granulomatous diseases (such as Crohn's disease), sarcoidosis, eosinophilic gastroenteritis, and radiation, chemical causes (bile reflux or corrosives), or injury.^[1,16,17]

The radiologic findings in these varied gastritides may be specific (eg, phlegmonous gastritis) or may include a broad differential list requiring careful clinical correlation. Antral narrowing is a common finding in a number of these disorders, such as Crohn's disease and syphilis, but other diseases causing similar appearances, such as scirrhous carcinoma, must be excluded.

Summary

Gastritis remains challenging for clinicians and radiologists because of an evolving pathogenesis and varied criteria used by pathologists and endoscopists that often do not correlate with imaging examinations. With the discovery of *H pylori* and consensus on pathologic classification, a clearer understanding of gastritis has emerged.

We have simplified the classification of gastritis for radiologic purposes. In erosive gastritis, radiologic findings are often specific but not sensitive. *Helicobacter pylori* gastritis is now recognized as the most common cause of a variety of gastric disorders; however, the role of radiology in the diagnosis of this infection needs further study. Chronic nonspecific gastritis can be suggested radiologically but only in the more severe forms. In hyperplastic gastropathy, fold abnormalities must be distinguished from those of other forms of gastritis and gastric disorders.

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