

Endoscopic, ultrasonographic, and pathologic correlation of lymphocytic gastritis

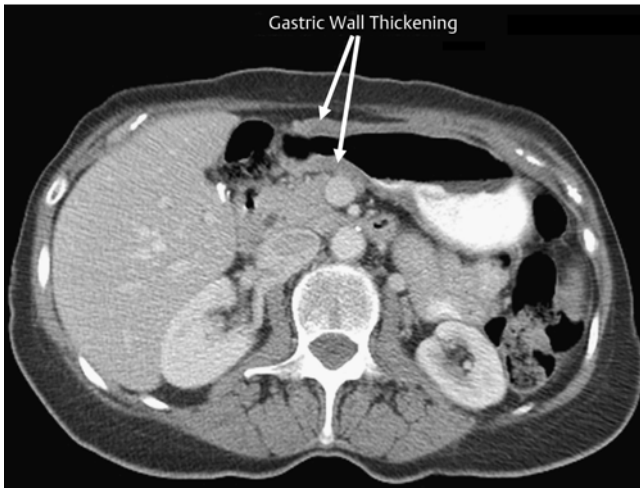


Fig. 1 Axial computed tomographic image demonstrating diffuse gastric wall inflammation in a 71-year-old woman with weight loss, postprandial epigastric pain, and mild chronic diarrhea.



Fig. 2 Endoscopic image demonstrating friability, edema, nodularity, and gastritis. Although difficult to visualize on imaging, the stomach was also difficult to insufflate.

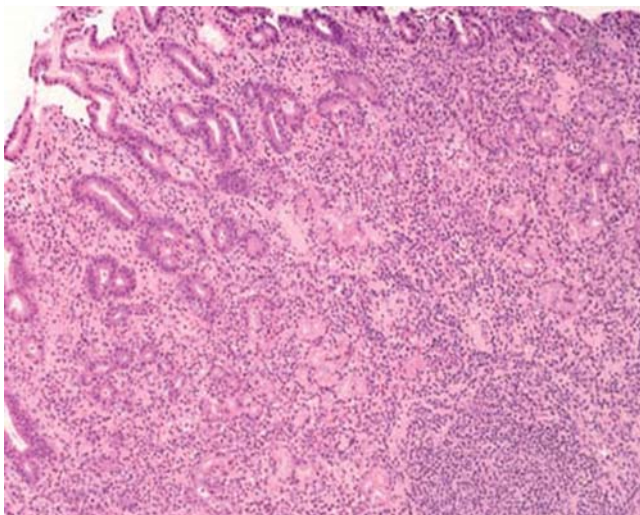


Fig. 3 A hematoxylin and eosin stained section of gastric mucosa demonstrating dense infiltration of lymphocytes and plasma cells in the lamina propria.

A 71-year-old woman presented with weight loss, postprandial epigastric pain, and mild chronic diarrhea. Computed tomography of the abdomen demonstrated mild antral wall thickening (► Fig. 1). Subsequent upper endoscopy showed diffuse inflammation, friability, and nondistensibility of the distal stomach (► Fig. 2), suspicious for linitis plastica or lymphoma. Histologic examination of biopsy samples of the stomach demonstrated dense lymphoplasmacytic infiltration of the lamina propria, as well as patchy neutrophilic infiltrate in an apparent ulcer bed (► Fig. 3). Immunostaining was not suggestive of a monoclonal process, and there was no evidence of *Helicobacter pylori* (*H. pylori*) colonization. Biopsies of a normal-appearing duodenum demon-

strated preserved villous architecture with prominent lymphocytic infiltrate. A 1-month follow-up esophagogastroduodenoscopy noted mild improvement, with jumbo forceps biopsies demonstrating prominent lymphoid follicles and negative immunostaining once again. The patient was referred to the university hospital for endoscopic ultrasound. Endoscopic evaluation showed edematous, erythematous, and friable mucosa throughout the stomach. The stomach distended normally with insufflation. The duodenum had a “scalloped” appearance suspicious for celiac disease. Sonographic imaging revealed diffuse wall thickening of the distal body and antrum of the stomach, which was free of folds (► Fig. 4). The gastric wall measured up to 9 mm. Duo-

denal biopsies showed villous blunting, crypt hyperplasia, and increase in intra-epithelial lymphocytes in the lamina propria, suspicious for celiac disease. Gastric biopsies demonstrated a dense infiltrate of lymphocytes in the lamina propria consistent with lymphocytic gastritis. The histology showed no evidence of *H. pylori* or lymphoma. The patient’s antiendomysial and antigliadin antibodies were negative, but she had a low-normal quantitative IgA level and she was HLA-DQ8 positive.

Lymphocytic gastritis is defined as >25 lymphocytes for every 100 epithelial cells in the gastric mucosa. The incidence of this disease in the setting of celiac disease has been widely debated and has been estimated to be as high as 46%. However, most data about the incidence of lymphocytic gastritis in celiac disease come from retrospective studies, which tend to overestimate the incidence. A prospective study showed the incidence of lymphocytic gastritis in celiac disease to be 3% [1]. Endoscopic findings of lymphocytic gastritis are quite variable; historically the findings of thick mucosal folds, nodularity, and aphthous erosions have been termed varioliform gastritis. However, many patients may present with normal endoscopies. In addition to celiac disease, lymphocytic gastritis is associated with *H. pylori*, Crohn’s disease, human immunodeficiency virus, inflammatory polyps, lymphoma, and esophageal carcinoma, and it can be idiopathic in up to 18% of patients [2]. Theories about the exact pathophysiology of lymphocytic gastritis

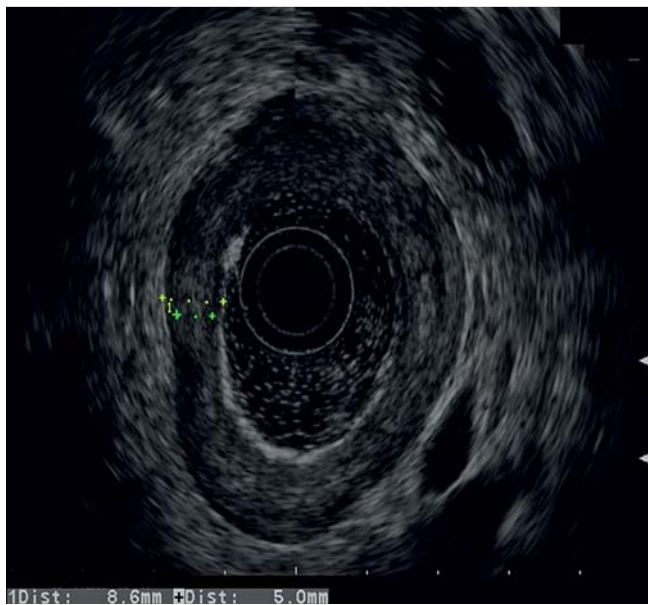


Fig. 4 Endoscopic ultrasonographic image of gastric antral wall showing diffuse thickening, most of which was in the submucosa (layer 3) at a maximum of 5 mm. The entire gastric wall measured >8 mm.

in celiac disease include a luminal response to an intolerable antigen (gliadin), an autoimmune phenomenon associated with celiac disease, and a contiguous spread of inflammation from the duodenum [3]. One study found a predominance of a monoclonal T-cell population throughout the gastric, duodenal, and colonic mucosa in patients with refractory celiac sprue, suggesting a dissemination of T cells from the duodenum into proximal and distal sites [3].

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Competing interests: None

**N. Aggarwal¹, S. F. Kuan²,
K. E. Fasanella³**

- ¹ Department of Medicine, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania, United States of America
- ² Department of Pathology, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania, United States of America

³ Division of Gastroenterology, Hepatology, and Nutrition, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania, United States of America

References

- 1 *Diamanti A, Maino C, Niveloni S et al.* Characterization of gastric mucosal lesions in patients with celiac disease: a prospective controlled study. *Am J Gastroenterol* 1999; 94 (Suppl. 05): 1313–1319
- 2 *Wu T, Hamilton SR.* Lymphocytic gastritis: association with etiology and topology. *Am J Surg Pathol* 1999; 23 (Suppl. 02): 153–158
- 3 *Verkarre V, Asnafi V, Lecomte T et al.* Refractory coeliac sprue is a diffuse gastrointestinal disease. *Gut* 2003; 52 (Suppl. 02): 205–211

Bibliography

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Corresponding author

N. Aggarwal
 Department of Internal Medicine
 University of Pittsburgh Medical Center
 200 Lothrop Street – N-713
 Pittsburgh
 PA 15232
 USA
 Fax: 412-648-4944
 aggarwaln@upmc.edu