Successful Treatment of Collagenous Gastritis in a Child With a Gluten-Free Diet

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ABSTRACT

Collagenous gastritis is a rarely encountered disease entity first described in 1989, and it is very rarely reported in children. We report the case of a 13-year-old boy with clinical, endoscopic, and histological findings of collagenous gastritis who reported rapid and sustained symptom resolution on a gluten-free diet.

INTRODUCTION

Collagenous gastritis (CG) is a rarely encountered disease entity first described by Colletti and Trainer in 1989.[1] Few of the subsequently published case reports involve children.[2,3] Although clinical symptoms and endoscopic findings are variable, diagnosis is based on standard histological criteria from an intestinal mucosal biopsy specimen.[3] The etiology, pathogenesis, and natural history of CG remain unclear. A variety of therapeutic interventions have been attempted without uniform improvement. We report a 13-year-old boy with clinical, endoscopic, and histological findings of CG who reported rapid and sustained symptom resolution on a gluten-free diet.

CASE PRESENTATION

A 13-year-old boy with no prior medical illness presented with a 6-month history of generalized abdominal pain; frequent loose, nonbloody stools; and a 5-pound weight loss. Outpatient laboratory evaluation consisting of complete blood count with differential, complete metabolic panel, *Helicobacter pylori* antibodies, free T4 and TSH (thyroid-stimulating hormone), inflammatory markers, and a celiac panel (serum immunoglobulin A [IgA] levels, endomysial, and tissue transglutaminase antibodies) was normal. Celiac genetics were performed due to a maternal history of "gluten sensitivity" with heterozygosity for HLA-DQ8/HLA-DQ2. Stools on several occasions were negative for bacterial and parasitic pathogens, and no occult blood was detected. A computed tomography scan of the abdomen and pelvis was unremarkable.

An esophagogastroduodenoscopy with unrestricted gluten exposure was notable for diffuse nodularity involving the gastric body and fundus with antral sparing (Figure 1). The gastric body showed gastritis, and the antrum showed mild chronic gastritis. The duodenum showed normal villi and normal disaccharidases. Colonoscopy was visually normal with normal histological appearance. Histology from the upper gastrointestinal tract revealed thickening of the collagen table (Figure 2).

Following the endoscopic evaluation, the patient was placed on a gluten-free diet with symptom abatement at 1 month and resolution at 6 weeks. Repeat esophagogastroduodenoscopy at 6 months showed a subjective normalization of the fundus, with unchanged gastric body nodularity and persistent antral sparing (Figure 3). The patient's abdominal pain resolved and his stools normalized, with resumption of appropriate weight gain. Histology showed improvement and continued normal linear growth velocity. He continues to do well on a gluten-free diet 3 years following diagnosis, with only transient diarrhea attributed to known gluten exposure.

DISCUSSION

Collagenous gastritis (CG) is an uncommon diagnosis, particularly in children. Clinical symptoms are variable and appear to have 2 age-related subgroups. Children and young adults present with upper abdominal pain and anemia, with disease limited to the gastric mucosa. Adults generally experience watery diarrhea and can manifest with associated collagenous colitis.[3-5] Significant overlap occurred in our patient, as in other case reports. Collagenous enterocolitides may represent an age-related spectrum. Endoscopic findings in published case reports consis-

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Figure 1. Diffuse Nodularity Involving the Gastric Body and Fundus on Initial Endoscopy

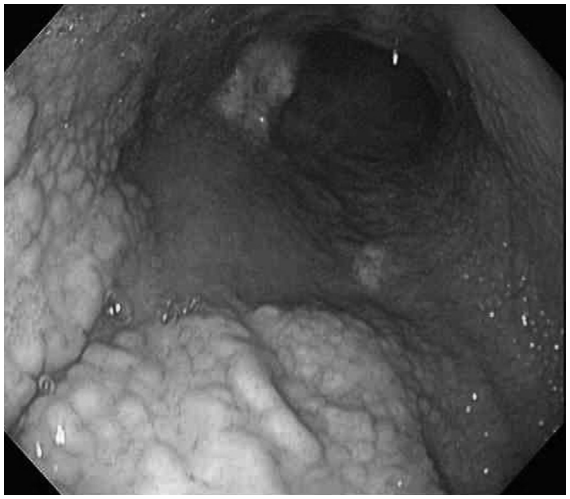
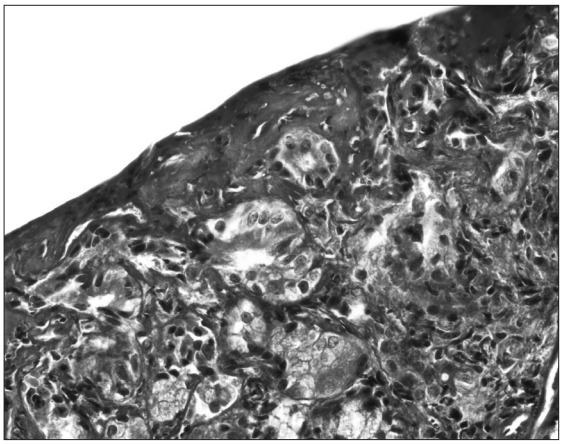
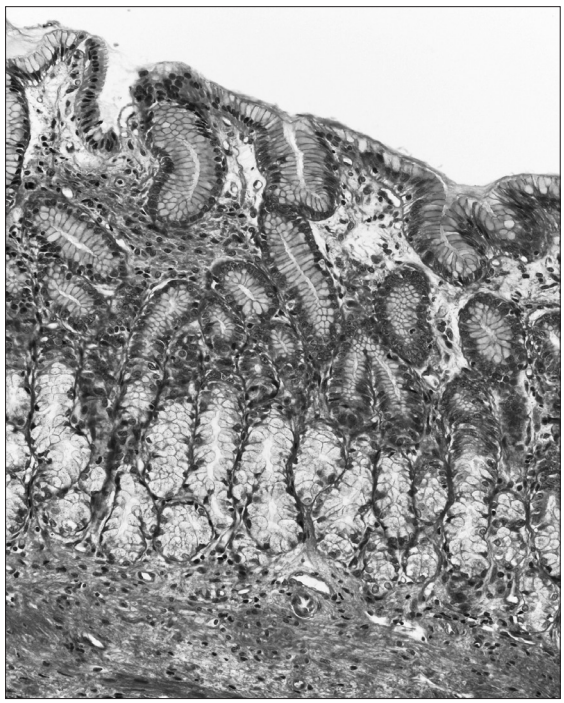


Figure 2. Gastric Mucosa With Thickening of Collagen Table Consistent With Collagenous Gastritis



Masson trichrome stain, original magnification x400.

Figure 3. Gastric Mucosa With Patchy Collagenous Involvement While on Gluten-free Diet



Masson trichrome stain, original magnification x400.

tently describe nodularity in the stomach along the greater curvature with variable antral sparing, and accompanying histological findings are consistent with subepithelial collagen deposition,[6] which also is seen in collagenous colitis and collagenous sprue. Several hypotheses have been proposed to explain the increased subepithilial collagen deposits including chronic inflammation and autoimmunity, abnormality of the pericryptal fibroblast sheath and plasma protein, and fibrinogen leakage with subsequent collagen replacement.[3] These mechanisms of subepithelial collagen deposition involve a reparative process in response to a prior inflammatory, infectious, or toxic insult. An intriguing possibility includes the pathogenetic role of an altered immune response to a luminal agent triggering an inflammatory response with subsequent collagen deposition.[6] It may be that CG is a feature of a diffuse disease process rather than a distinct disorder.[3,7] Celiac disease has been associated with CG in adults, although a causal relationship is unclear.[8] Although our patient did not have histologic evidence for celiac disease, removal of dietary gluten resulted in symptom resolution, which raises the question of the role of gluten in some individuals with CG.

There is marked variability in mucosal changes among patients with celiac disease, nonceliac gluten sensitivity, and collagenous gastritis. Celiac disease presents with an increased number of intraepithelial lymphocytes (>25 per 100 enterocytes), elongation of crypts, and partial to total villous atrophy.[9] In nonceliac, gluten-sensitive patients, the histological pictures show minor abnormalities with intraepithelial lymphocytes in their duodenal mucosa.[10] However, patients with CG have the defining feature of subepithelial collagen deposition. Arnason et al reported marked heterogeneity in associated inflammatory pattern.[11] In 40 patients with CG, there were increased intraepithelial lymphocytes in 5 patients, eosinophil-rich pattern was noted in 21 patients, and in 7 patients biopsy noted atrophic gastric mucosa.[11]

The natural course and treatment of CG is unknown. A variety of interventions including topical and systemic anti-inflammatory therapies, acid suppression, and gluten elimina-

tion have not resulted in consistent symptomatic or pathologic improvement.[3] In our patient, a gluten-free diet appeared to be an effective treatment for the management of his gastrointestinal symptoms. Although the precise pathogenic mechanism of CG is unknown, intraluminal gluten may be involved in the pathogenesis and/or symptoms of CG. Treatment with a gluten-free diet should be considered for pediatric patients diagnosed with CG. Further research may help uncover factors that can assist in determining whether a gluten-free diet may be efficacious for a given patient.

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