Collagenous gastritis presenting as chronic heartburn in a patient with psoriatic arthritis

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Abstract

Collagenous gastritis is a rare cause of heartburn in adults. Histopathological examination of gastric mucosal biopsy from the stomach shows submucosal collagen deposition. The pathophysiologic mechanism is unknown, and collagenous gastritis cases have been associated with certain drugs, such as olmesartan and non-steroidal anti-inflammatory drugs, and certain medical conditions, such as common variable immunodeficiency, primary IgM deficiency, autoimmune disorders, and psoriatic arthropathy. Here we report a case of collagenous gastritis in a 29-year-old woman with psoriatic arthropathy who presented with persistent heartburn. She was successfully treated with oral pantoprazole.

Keywords: heartburn, collagenous gastritis

Introduction

Collagenous gastritis (CG) is a rare gastrointestinal pathology often presenting with heartburn. Initially thought to be more common in the pediatric population, CG is increasingly reported in the adult population.[1–4] The diagnosis of this disease requires histopathologic examination showing submucosal collagen deposition. However, the underlying mechanism of the disease is not well understood. Here we report a case of CG diagnosed in a patient with symptoms of chronic heartburn and reflux and a medical history of psoriatic arthropathy.

Case

A 29-year-old woman with medical history of psoriatic arthritis, small intestinal bacterial overgrowth (SIBO), and Barrett's esophagus (BE) presented to the gastroenterology clinic with a persistent symptom of heartburn for several years that had been attributed to gastroesophageal reflux disease (GERD) in the past. At the time of presentation, her medications included pantoprazole and hydroxychloroquine.

Her initial symptoms included abdominal pain in the epigastric region, occasional burning sensation in the retrosternal area, and recurrent coughing spells. These symptoms increased when she was pregnant. A prior esophagogastroduodenoscopy (EGD) done at the time of her initial presentation 2 years ago showed salmon-coloured mucosa suspicious for Barrett's esophagus, a 2 cm hiatal hernia, and a Hill Grade III gastroesophageal flap valve. The histopathology examination of biopsied tissue confirmed Barrett's esophagus without dysplasia and concomitant evidence of moderate chronic and acute inflammation. She was treated with a course of proton pump inhibitor (PPI) at that time with partial improvement. A repeat EGD was done due to persistent intermittent generalized abdominal pain and abdominal bloating and was found to have SIBO and was treated with a course of rifaximin and sucralfate with minimal improvement. She also tried adopting a gluten-free diet without significant benefit. She continued taking PPI with intermittent symptoms of heartburn, nausea, and vomiting.

Due to her prior history of BE and persistence of symptoms of reflux, a repeat EGD was done that

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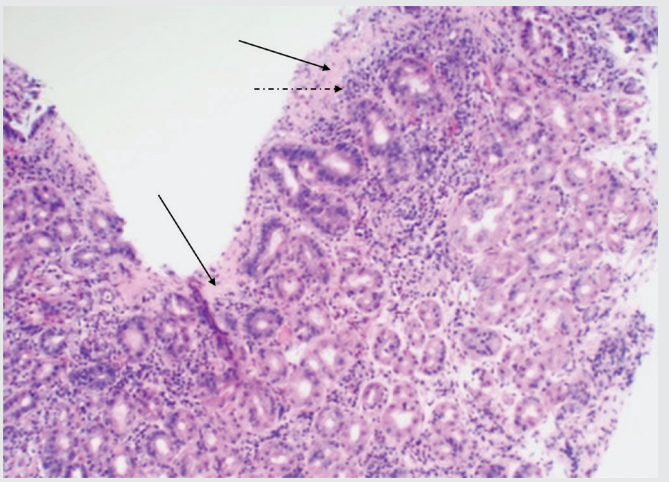

Figure 1. Histopathologic examination of biopsied tissue from stomach showing collagenous gastritis. H&E stain of stomach specimen shows deposition of collagen (solid arrows) and inflammatory cells (dashed arrow) in the lamina propria.

showed a normal esophagus and a few sessile polyps in the gastric fundus with abnormal vascularity. The polyps were removed by cold snare, and histopathologic examination of the biopsies showed polypoidal fragments of oxyntic mucosa with thickened collagen plate suggestive of chronic CG (Figure 1). Due to the relatively benign course of her disease, she was continued on pantoprazole once daily, with plan for a surveillance EGD in 1 year. Hydroxychloroquine for psoriatic arthritis was also continued.

Discussion

Collagenous gastritis is a relatively rare condition often masquerading as GERD. Although more often reported in the pediatric population, CG has also been reported in adults.[1–4] A recent case-control study of 168 patients with CG identified a bimodal age distribution with a peak in the pediatric population (age 11–19 years) and another peak in >60 years. This study also reported an estimated prevalence of 13 per 100,000 EGDs, with a female preponderance, especially in the adult age group.[5,6]

Collagenous colitis is a more commonly recognized clinical entity, with a similar pathophysiology of submucosal collagen deposition and microscopic evidence of chronic inflammation, which usually presents with watery diarrhea and weight loss.[7] Collagenous gastritis more commonly presents with symptoms mimicking GERD such as heartburn and chronic cough as was present in our patient, dysphagia, nausea, vomiting, dyspepsia, abdominal pain, diarrhea, and anemia.[5,8,9] Rarely weight loss, diffuse post-prandial abdominal pain and melena have also been reported.[8,10] Co-existing collagenous colitis has sometimes been reported.[6,11–13]

Although histopathologic features are quite characteristic, its exact pathophysiologic mechanism remains unknown. Use of certain medications such as olmesartan and non-steroidal anti-inflammatory drugs have often been associated with the development of chronic gastritis.[13–16] Several comorbid conditions have been concomitantly reported, such as common variable immunodeficiency, primary IgM deficiency, autoimmune disorders, and psoriatic arthropathy.[10,11,17–20] Our patient had a history of psoriatic arthropathy and was on treatment with hydroxychloroquine for this. One prior case of collagenous colitis has been reported in a patient with psoriatic arthropathy, which was attributed to the patient's treatment with secukinumab.[21] There has not been any previous report of prior hydroxychloroquine use in patients with CG, and therefore its role in the pathophysiology remains unclear.

The diagnosis of CG depends on biopsy and histopathological examination of the gastric mucosa obtained during an EGD. Some authors have reported characteristic endoscopic findings in CG, such as mucosal erythema, nodularity and ulceration.[8,22] However, the EGD findings in our patient were insignificant and the diagnosis was confirmed only with biopsy. The definitive diagnosis is done by detecting deposition of collagen >10 μm thickness in the lamina propria with entrapment of inflammatory cells and dilated capillaries.[10] Sometimes, enrichment of eosinophils with >30 eosinophils/high power field can also be observed.[23]

No specific therapy for CG has been established. Different treatment options that have been empirically tried in patients include steroids, ranitidine, PPI, misoprostol, sucralfate, 5-ASA and hypoallergenic diets.[6,24] A trial of PPI, H2 blocker, and oral iron has been

successfully used in some patients.[22,25] Treatment with oral budesonide for up to 2 months has led to resolution of symptoms in some cases refractory to acid suppression.[26] Our patient's symptoms were moderately controlled with PPI; it was decided not to start additional medication. A trial of sucralfate and use of a gluten-free diet done previously were not helpful for her. Since no definitive role of hydroxychloroquine has been reported in the pathogenesis of the condition, this was continued for her psoriatic arthropathy. She will not require frequent follow-ups for her symptoms, and if needed, corticosteroids can be added.

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