CASES

Patient 1: SB is a 63-year-old woman with chronic diarrhea for four years. She denied any association with dairy or wheat products, or any other symptoms including weight loss, abdominal pain, blood in stool or alternating constipation. This patient had no significant past medical or surgical history. No reported use of nonsteroidal anti-inflammatory medications. Her vitals and physical exam were normal. Stool cultures and serology for celiac disease were normal. Endoscopic exam revealed a prominent vascular pattern in the left colon (Figure 1A). No other abnormalities were noted during the examination. Biopsies of the mucosa revealed a thick subepithelial collagen band with epithelial detachment (Figure 1B).

Patient 2: LZ is a 38-year-old woman with an 18 year history of unexplained diarrhea. Standard work-up for chronic diarrhea was negative and included serologies, stool cultures, radiologic exams and endoscopic exams. During the fifth year of history, she had a colonic biopsy showing mild acute and chronic inflammation. However, this did not respond to medications such as azulfidine, prednisone, or metronidazole. During her ninth year of diarrhea, her colonoscopy revealed a marked hypervascular pattern in the entire colonic mucosa (Figure 2). Colonic biopsies revealed increased collagen bands in the subepithelial layer.

Questions
1. What is the diagnosis of these patients given their history and physical findings?
2. What is unusual about the endoscopic findings of these 2 patients given their diagnoses?

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DISCUSSION

The term “microscopic colitis” was first used in 1976 (1) to describe a patient with chronic diarrhea accompanied by normal appearing colonic mucosa, while rectal biopsy showed thick subepithelial collagenous deposit. Since then, there have been multiple reports of similar histologic finding in patients with unexplained chronic watery diarrhea (2–4). As the name implies, the diagnosis of microscopic colitis is made exclusively based on histologic findings. Usually, there are no gross findings on endoscopic examination. However, there have been reports of rare and nonspecific endoscopic changes including erythema, edema, (5,6), or even pseudomembranes (7,8).

We now report two cases of microscopic colitis that exhibited marked hypervascularity that was either localized to a portion of the colon or distributed throughout the colon. To the best of our knowledge, this has been described in one previous case report and briefly noted in another study (6,9).

We speculate the hypervascular appearance may be due to the underlying histologic architecture. Collagenous colitis, a subtype of microscopic colitis, is characterized by an abnormally thickened subepithelial collagen band. Other associated changes include entrapment of capillaries in the collagen and detachment of the epithelial layer overlying the collagen (10). It is conceivable that the combination of small vessels in the collagen band and the absence of epithelial cells on the surface of the mucosa may give the gross appearance of mucosal hypervascularity during an endoscopic exam.

The frequency of this endoscopic feature in microscopic colitis is unknown. Since it does not resemble other signs such as mucosal erythema, edema, ulcerations or exudates, it conceivably may be dismissed by the endoscopist. A prospective study examining and performing biopsies on all patients with this nonspecific hypervascular pattern during a colonoscopy may be worthwhile in order to document a correlation between microscopic colitis and this macroscopic finding.

In conclusion, we are reporting two cases of microscopic colitis exhibiting the gross finding of mucosal hypervascularity. We speculate that this may be due to a combination of loss of epithelial cells and the presence of microvasculature close to the surface of the colonic mucosa.

References