CLINICAL VIGNETTE

Segmental Colitis Associated with Diverticulosis

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Case Report

A 51-year-old man presented with two months of diarrhea with abdominal cramping, urgency, and rectal bleeding. He denied oral ulcers, joint pain, changes in vision, or skin changes. He took only occasional ibuprofen, about once a month, with no regular medications. Labs included normal complete blood count, comprehensive metabolic panel, and C-reactive protein, and an elevated fecal calprotectin (FC) at 3000 nl. Stool infectious studies were all negative. Colonoscopy was performed and revealed focal inflammation in the sigmoid colon surrounding diverticulosis with loss of vasculature, superficial ulceration, and friability with otherwise normal terminal ileum and colon. Biopsies of the sigmoid colon showed moderate chronic colitis without granulomas or dysplasia. Biopsies of terminal ileum, right colon, and rectum were all normal. Magnetic resonance elastography was performed and revealed a short segment of mild sigmoid colonic wall thickening correlating with the site of colitis noted on colonoscopy, diverticulosis, and no other areas with acute or chronic inflammation. He was diagnosed with segmental colitis associated with diverticulosis (SCAD) and treated with ciprofloxacin 500mg twice daily and metronidazole 500mg three times daily for 14 days. His symptoms improved and the elevated fecal calprotectin and inflammation on colonoscopy resolved.

Discussion

SCAD is defined by chronic inflammation in the interdiverticular mucosa without involvement of the diverticular orifices with normal rectum and right colon histology.[1,2] SCAD has been identified in 1.5% of the overall population with a mean age of 62-64 and a higher prevalence in men.[2,3] The pathogenesis of SCAD is uncertain, but may be multifactorial in the setting of prolapsed mucosa due to shear stress, changes in bacterial flora and enzyme activity due to fecal stasis in diverticulosis, changes in permeability, and focal mucosal ischemia.[4] There is a possible pathogenic relationship to inflammatory bowel disease (IBD), especially ulcerative colitis (UC), despite entirely normal rectum at the time of initial presentation.

Most patients with SCAD present with chronic diarrhea, left lower abdominal cramping, and hematochezia. About one third of patients have more than one symptom at the time of diagnosis.[2,3] Fecal calprotectin may also be elevated in the presence of increased intestinal inflammation, as in our patient.[5] There are four patterns of inflammation seen on colonoscopy which all spare the diverticular orifices. Type A or "crescentic fold disease" is characterized by swollen red patches confined to the mucosal folds.[2] Type B or "mild to moderate UC-like pattern" includes loss of vascular pattern, mucosa edema, and diffuse erosions, which was identified in our patient. Type C or "Crohn's colitis-like pattern" is characterized by scattered aphthous ulcers with normal intervening mucosa. Type D or "severe UC-like pattern" is characterized by diffuse loss of vascular pattern, marked edema with ulcerations, and friability, similar to a Mayo score of 3 for endoscopic disease activity in UC.[6]

Optimal treatment for SCAD is not well-defined and based on case reports and indirect evidence in patients with IBD. Multiple case reports and cases series utilized high fiber diet, antibiotics, 5-aminosalicylic acid (ASA) therapy, and/or steroids for treatment of SCAD. Only a small percentage of patients required surgery for complications, which included bleeding, strictures, or obstruction.[7–9] Two reports confirmed endoscopic mucosal healing after treatment with anti-tumor necrosis factor (TNF)-alpha inhibitors in 5-ASA, antibiotic, and steroid-refractory SCAD.[6,10] Antibiotics are recommended for initial treatment followed by 5-ASA and steroids if no response. Segmental resection and biologic therapy is reserved for steroid-refractory or steroid-dependent patients.[11,12]

Conclusion

Our case illustrates the importance of considering SCAD as a diagnosis in patients with bloody diarrhea, elevated fecal calprotectin, and inflammation in the left colon without involvement of the diverticular orifices or rectum. Differentiating between SCAD and IBD may be helpful in determining optimal treatment and prognosis.

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