



No Microscope Needed: A Macroscopic Presentation of Collagenous Colitis

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ABSTRACT

Microscopic colitis (MC) is a common cause of chronic secretory diarrhea with variable etiologies, including nonsteroidal anti-inflammatory drugs. As its name implies, the diagnosis requires consistent histopathologic findings that typically accompany normal-appearing mucosa. However, accumulating evidence suggests that the presence of distinct endoscopic features is associated with MC. We present a case of MC that highlights the importance of recognizing these macroscopic findings because they can aid in diagnosis and have significant management implications.

INTRODUCTION

A common cause of chronic secretory diarrhea, microscopic colitis (MC) has an incidence of 21.0 per 100,000 person-years.¹ Although the pathogenesis of MC is not entirely elucidated, several medications have been implicated in causing MC, including nonsteroidal anti-inflammatory drugs, proton pump inhibitors, and serotonin reuptake inhibitors.² The diagnosis of MC, as suggested by its name, is considered histopathologic and occurs in 2 subtypes, lymphocytic and collagenous colitis (CC), both typically presenting with symptoms of diarrhea. Treatment remains similar for all forms of MC.^{2,3} Therefore, obtaining the diagnosis is paramount. Although there is some guidance regarding the location and number of biopsies that should be obtained to confirm a diagnosis of MC, it most often presents with endoscopically normal-appearing colonic mucosa.⁴⁻⁹ However, macroscopic changes are being recognized with increased frequency. We present a rare case of MC that illuminates the importance of recognizing these macroscopic findings because they not only aid in diagnosis but also have significant management implications.

CASE REPORT

A 61-year-old woman with rheumatoid arthritis presented with 1 month of watery, nonbloody diarrhea. She reported lower quadrant, crampy abdominal pain and a 20-lb weight loss. Her medications included pantoprazole 20 mg, escitalopram 20 mg, celecoxib 200 mg, and ibuprofen, taken 3 times weekly. Her physical examination demonstrated diffuse abdominal tenderness, and the laboratory assessment revealed acute renal insufficiency with a Cr of 3.6 (baseline Cr 1), erythrocyte sedimentation rate of 28, and C-reactive protein of 0.4 mg/dl. A complete blood count was notable for a normocytic anemia of 9.3 down from a baseline around 12 and thrombocytopenia with a platelet count of 68. Infectious stool studies, including *Clostridium difficile*, stool culture, and ova and parasite testing, were negative. Fecal calprotectin was mildly elevated at 152, and fecal leukocytes were absent. Given progressive abdominal pain, a contrasted abdominal and pelvic computed tomography was performed that revealed diffuse colonic wall thickening and mild mucosal hyperenhancement consistent with pancolitis (Figure 1).

On colonoscopic examination, erythematous mucosa with congestion was found throughout the entire examined colon along with significant friability characterized by multiple shallow, linear mucosal rents that occurred with minimal insufflation of CO₂ (Figure 2). The colonoscope was advanced into the terminal ileum which revealed normal-appearing mucosa. Biopsies were taken separately from the right and left colon for histology, and the remainder of the procedure was uncomplicated. Pathology demonstrated abundant subepithelial collagen deposition in both sets of biopsies consistent with a diagnosis of severe CC (Figure 3).



Figure 1. Abdominal and pelvic computed tomography with diffuse colonic wall thickening and hyperenhancement. The arrow indicates to an area of wall thickening and hyperenhancement signifying pancolitis.

She was initiated on 9 mg of budesonide for 1 month, and both celecoxib and pantoprazole were discontinued on discharge. She was counseled to avoid nonsteroidal anti-inflammatory drugs. At a 1-month follow-up visit, she had complete resolution of her diarrhea. In addition, she denied any other red flag symptoms to include nausea, vomiting, fevers, abdominal pain, melena, or hematochezia. Her appetite improved, and her weight stabilized. Her budesonide was tapered to 6 mg for 2 weeks followed by 3 mg for 2 weeks. Normalization of her anemia and thrombocytopenia coincided with her clinical improvement. Repeat colonoscopy was not performed.

DISCUSSION

Despite the diagnosis of MC classically relying on histologic changes, macroscopic findings are increasingly recognized and present in up to 38.8% of cases of MC.⁹ These endoscopic findings are typically nonspecific, with the presence of mucosal erythema, edema, and vascular pattern changes, although discrete mucosal tears and ulcerations have been described.^{5,10,11} These mucosal

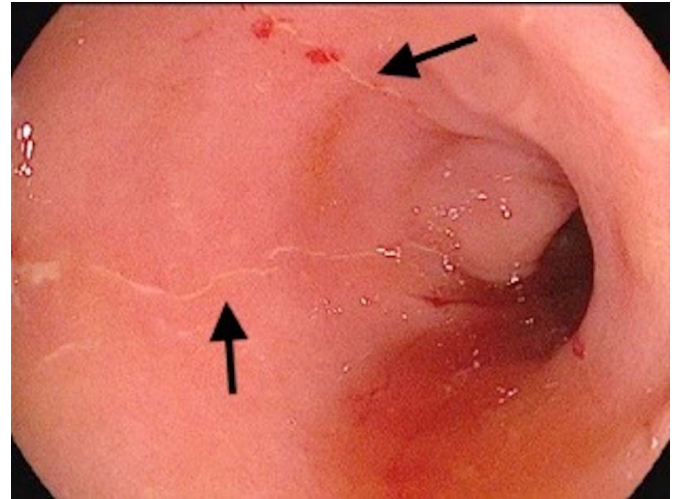


Figure 2. Longitudinal mucosal tears, erythema, loss of vascularity. The arrow indicates to the longitudinal tears that appeared with CO₂ insufflation. The importance of these are that they are high risk endoscopic features and may herald colonic perforation.

defects, while rare, have been associated with an increased risk of colonic perforation after colonoscopy and barium enemas.^{5,10,13} Radiographic studies are also typical, with only a handful of case reports describing nonspecific mucosal nodularity in the sigmoid colon and loss of haustral folds on double-contrast barium enema.¹² Association with transmural inflammation is even rarer, and to our knowledge, this is the first report of CC with both radiographic evidence of pancolitis and discrete mucosal ulcerations.

Patients with multiple symptoms beyond diarrhea, including abdominal pain and weight loss, are 3 times more likely to have macroscopic findings on endoscopy.¹¹ Although there is no evidence that macroscopic findings are more common in 1 variant over the other, previous studies have noted a relationship between mucosal ulceration and CC.^{5,10} In addition, a systematic

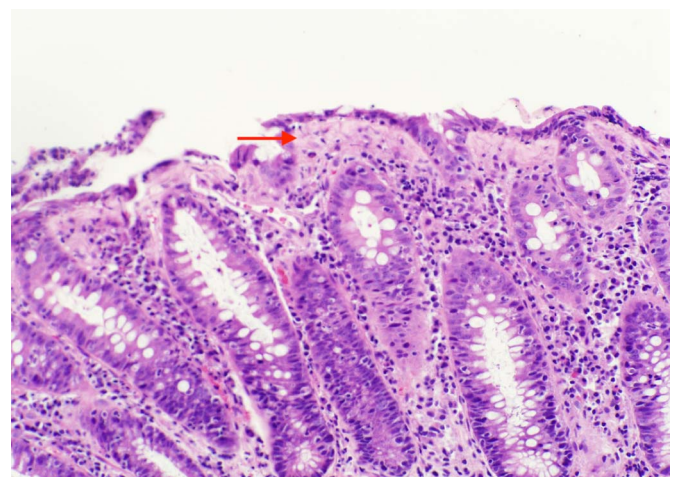


Figure 3. Colonic mucosa with increased intraepithelial lymphocytes and subepithelial collagen deposition. The arrow indicates to an area of collagen deposition.

review by Marlicz et al. found that 17 patients with colonic perforations all had underlying CC, supporting the hypothesis that collagen deposition may reduce colonic compliance, predisposing patients to mucosal tears that may herald colonic perforation.¹⁰

Although the treatment of MC remains similar in those with and without macroscopic findings, increased awareness and identification of macroscopic findings have the potential to aid in the attainment of an accurate diagnosis while mitigating risk to the patient during a diagnostic colonoscopy. Current American Society for Gastrointestinal Endoscopy guidelines recommend either at least 2 random biopsies be taken from the right, transverse, descending, and sigmoid colon during colonoscopy or 2 or more random biopsies from transverse, sigmoid, and descending colon on flexible sigmoidoscopy.⁴ Although colonoscopy with both left-sided and right-sided biopsies remains the preferred approach, previous studies have found similar diagnostic yields between biopsies taken exclusively from the left side, maintaining a sensitivity of >90%.⁵⁻⁷ Where our case is concerned, a complete colonoscopy including intubation of the terminal ileum was performed because inflammatory bowel disease was clinically compatible with the endoscopic findings. In hindsight, however, a flexible sigmoidoscopy with only left-sided biopsies may have been sufficient for a diagnosis in this case, which would have decreased risk to the patient.

This case highlights an atypical presentation of CC with pancolitis and discrete endoscopic lesions that should prompt endoscopists to alter management. Although future prospective studies are needed to determine whether flexible sigmoidoscopy alone is sufficient for diagnosis of MC, it should be considered with CO₂ insufflation or water emersion in those with high-risk endoscopic features. Education surrounding the association of deep mucosal tears and risk of perforation in CC is needed to not only increase recognition of these findings during endoscopy but also underscore the importance of aborting these cases to avoid potential complications when identified.

DISCLOSURES

Author contributions: A. L. Boyd wrote the article. D. A. Leiman and D. L. Dufault edited the article and revised the article for intellectual content. D. L. Dufault is the article guarantor.

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