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CASE REPORT | COLON

Segmental Colitis Associated With Diverticulosis Causing Hydroureteronephrosis

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ABSTRACT

Segmental colitis associated with diverticulosis (SCAD) is a rare inflammatory condition affecting segments of the colon with diverticular disease. We present an 85-year-old woman with flank pain, fevers, and chills found on imaging to have left colonic wall thickening and left-sided hydroureteronephrosis and workup confirming a diagnosis of SCAD. A detailed review of SCAD and discussion of the differential diagnosis are provided. This case emphasizes disease-specific clinical pearls and highlights hydroureteronephrosis as a rare complication seen in a patient with SCAD.

INTRODUCTION

Segmental colitis associated with diverticulosis (SCAD) is a rare but increasingly recognized inflammatory condition affecting segments of the large bowel with diverticular disease.[1,2] SCAD classically involves the sigmoid colon and often presents with rectal bleeding that may mimic malignancy or inflammatory bowel disease (IBD).[1–3] The natural history of SCAD is typically benign and is usually managed conservatively with antibiotics and/or 5-aminosalicylic acid (5-ASA) therapy. Complications are rare, especially when compared with IBD. In this case report, we see a patient presenting with flank pain, fevers, and chills found on imaging to have left colonic diverticulosis with wall thickening and left-sided hydroureteronephrosis and workup revealing a diagnosis of SCAD.

CASE REPORT

An 85-year-old woman with a history of uterine cancer status posthysterectomy 15 years prior, a recent diagnosis of deep vein thrombosis on anticoagulation, and a single previous episode of diverticulitis 5 years prior presented to our institution with fevers, chills, altered mental status, and flank pain. Her symptoms began 3 days before presentation with no associated abdominal pain, nausea, vomiting, hematochezia, or changes to her bowel movements.

Her most recent colonoscopy 5 years prior, following an episode of diverticulitis, revealed extensive sigmoid diverticulosis with the inability to traverse a segment of severe inflammation and stricturing approximately 30 cm from the anal verge. Biopsies were not taken at this time, and subsequent computed tomography (CT) colonography 2 months postcolonoscopy showed persistent inflammation, involving a 10-cm segment of the proximal sigmoid colon without a mass or polyp seen, linear soft tissue opacity connecting the sigmoid colon to the ileum suspicious for early fistulous tract, and mild ectasia of the left renal pelvis with minimal dilation of the proximal left ureter.

Upon arrival at our institution, her vital signs were within normal limits. Physical examination disclosed an elderly, frail woman in no acute distress with a benign abdominal examination, although left flank tenderness to palpation. Laboratory data revealed leukocytosis to 17 K/uL, normal carcinoembryonic antigen and cancer antigen 19-9 levels, unremarkable hematocrit, basic metabolic panel, and liver chemistries. Urinalysis demonstrated pyuria with positive nitrites and leukocytes. Abdominal and

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pelvic CT revealed a short segment of sigmoid colonic diverticulosis with wall thickening, adjacent lymphadenopathy, air-containing left psoas abscess, suspicion for sigmoid-ileal fistula, and moderate left-sided hydroureteronephrosis (Figure 1).

She was initially treated with bowel rest, broad-spectrum antibiotics, and a left-sided double-J ureteral stent. Interval CT scan demonstrated an increased volume of air within the psoas abscess. Therefore, colonoscopy was deferred because of the risk of perforation. Surgical consultation favored initial nonoperative therapy with antibiotics and interval resection following improvement of inflammation. She was discharged on oral antibiotics after resolution of her leukocytosis and pyuria with plans for repeat imaging and potential endoscopic evaluation to rule out neoplasm or IBD. Repeat imaging 4 weeks postdischarge revealed unchanged thickening of the midsigmoid colon extending into the left pelvic sidewall, fistulizing disease to the ileum, and interval increase in the left psoas collection with concern for fistulizing chronic diverticulitis, IBD, or potential malignancy. Colonoscopy was again deferred given the high risk of intraprocedural perforation, and surgical exploration with resection was offered.

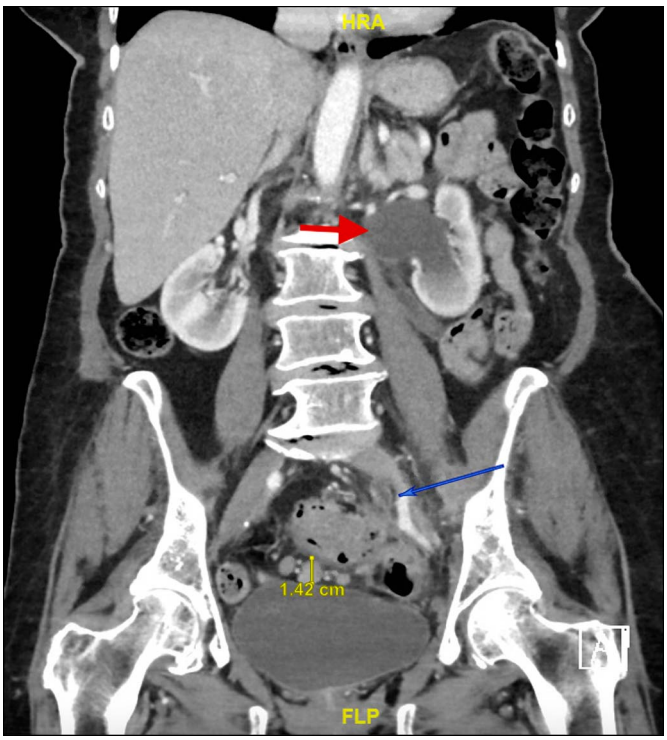

Figure 1. Abdominal and pelvic computed tomography showing a short segment of sigmoid colonic wall thickening and mild stranding in the adjacent fat and adjacent mesenteric lymphadenopathy. There is also evidence of left-sided moderate hydroureteronephrosis, without renal calculi noted, to the level of the colonic wall thickening and left psoas abscess concerning for fistulous tract. Midsigmoid colonic wall thickening (blue arrow) with upstream dilation of the ureter and renal calyx (red arrow).

The patient underwent exploratory laparotomy with low anterior resection and ileocolonic resection for the fistulizing disease to the ileum. Intraoperatively, a left lower quadrant phlegmon was identified and tethered to a thickened, inflamed sigmoid colon with innumerable diverticula. Pathology revealed diverticulitis with extensive old fibrosis and reactive lymphadenopathy without evidence of malignancy, granulomas, or histology suggestive of IBD (Figure 2). As such, the final diagnosis was SCAD with hydroureteronephrosis, and she recovered well without further complication.

DISCUSSION

Colonic diverticulosis is associated with numerous complications, including bleeding, perforation, inflammation, and abscess formation.[4] Recently, the taxonomical classification of symptomatic diverticular disease has evolved to encompass rare conditions such as SCAD and symptomatic uncomplicated diverticular disease.[5] Often mistaken for IBD or colorectal cancer, SCAD is an inflammatory condition that should be considered in patients with a history of diverticulosis and evidence of segmental colonic inflammation, especially in the absence of clinical, radiological, or biochemical data to suggest malignancy or IBD. SCAD conventionally involves the sigmoid colon, where diverticula commonly develop. A previous systematic review and meta-analysis revealed that SCAD appears to have a slight male predominance, with a mean age of diagnosis of 63.6 years, and occurs in approximately 0.3% to 1.3% of patients with diverticular disease.[6] Rectal bleeding, diarrhea, and abdominal pain were the most common presenting symptoms, whereas fever, leukocytosis, and rectal involvement were characteristically absent.[6]

Although SCAD typically follows a benign disease course with improvement following conservative treatment with or without antibiotics or 5-ASA therapy, ideal or preferred treatment remains undefined.[7] Part of this lack of ideal treatment relates to the underdiagnosis of SCAD and unclear rate of complications. It is important to note that no data currently are available to summarize the rate or severity of SCAD complications. Although hydronephrosis and colovesical fistulae associated with acute diverticulitis have been reported, this is the first case to these authors' knowledge to describe SCAD-induced hydroureteronephrosis.[8–13] Although the etiology of the hydroureteronephrosis cannot be definitively determined to be a result of SCAD, there remains a high clinical suspicion given the location of ureter narrowing and the known area of colonic involvement. We hypothesize that segmental colonic inflammation likely spread to the left ureter causing fibrosis with resultant ureteric narrowing. Other potential etiologies such as sequelae of previous hysterectomy or ureteral narrowing as a result of the phlegmon are unlikely given the temporal association with the patient's presentation, uncomplicated hysterectomy 15 years prior, and lack of findings during exploratory laparotomy to support phlegmon-associated mass effect.

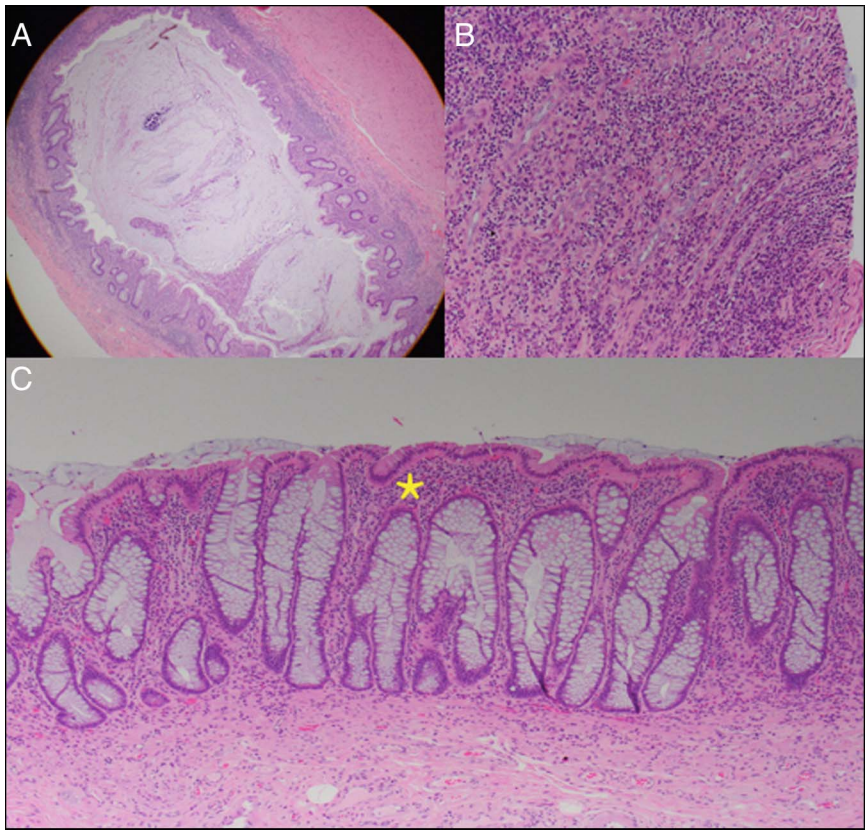

Figure 2. Low-power photomicrograph showing (A) dilated diverticulum with (B) peridiverticular granulation tissue formation. (C) The interdiverticular mucosa shows alteration of crypt architecture (branching) and expansion of the lamina propria, which contains predominantly chronic inflammatory cells (lymphocytes and eosinophils) (yellow asterisk).

Similar to other patients with SCAD, our patient's imaging was concerning for IBD and colonic malignancy. Given the recently diagnosed deep vein thrombosis, malignancy was of significant concern. Although biopsies may have been beneficial at the time of the previous colonoscopy, presentation, history, and repeat imaging findings were concerning for an alternative diagnosis. Tumor markers and high-resolution imaging may be helpful to narrow the differential, although should not exclude the need for pathologic diagnosis. The careful pathologic examination is key. On pathology, SCAD reveals involvement of the interdiverticular mucosa with sparing of the peridiverticular mucosa, whereas diverticulitis affects the diverticular orifices and peridiverticular mucosa.[3,4] This relative sparing of the peridiverticular mucosa and the lack of granulomas or other features of IBD help distinguish the disease. Stratification by the subtype of SCAD has been proposed, with types A–D described based upon histologic features (Table 1).[14] Despite some overlap with IBD, all of these subtypes are characterized by peridiverticular sparing, which is the main distinction between SCAD and IBD.[15] It is likely, based upon our patient's pathology, she would be classified as type B or D.

This case highlights the importance of recognizing atypical presentations of SCAD, as this patient lacked typical symptoms such as gastrointestinal bleeding but presented with urinary symptoms, altered mental status, and leukocytosis. Despite her benign abdominal examination, our patient exhibited severe inflammatory changes, including enterocolonic fistulae and hydroureteronephrosis, underscoring the possibility of severe

Table 1. Subgroup classification of segmental colitis associated with diverticulosis

Subgroup	Description
Type A	Crescentic fold pattern characterized by a lymphocytic or neutrophilic inflammatory infiltration with no alteration of the glandular architecture
Type B	Mild-to-moderate ulcerative colitis-like pattern characterized by loss of the submucosal vascular pattern, chronic changes of the lamina propria, crypt architecture distortion, crypt abscesses, and hemorrhage
Type C	Crohn's-like pattern characterized by transmural inflammation, fissures, epithelioid granulomas, isolated aphthous ulcers, and lymphohistiocytic vasculitis
Type D	Severe ulcerative colitis-like pattern characterized by chronic changes of the lamina propria, crypt architecture distortion, crypt abscesses, and hemorrhage

complications of SCAD and the importance of recognizing attenuated symptoms in the elderly. Furthermore, although colonoscopy is typically recommended for patients approximately at least 8 weeks after acute episode of diverticulitis episodes, no gastroenterology societal guidelines exist at present to address the timing of colorectal cancer surveillance with colonoscopy for patients with SCAD. However, colonoscopy can be helpful because this type of clinical picture can have a wide differential. In-depth understanding and recognition of SCAD as a potential etiology, creating a broad differential diagnosis, and a need for pathologic diagnosis underscore the importance of proper identification of SCAD as a clinical entity.

DISCLOSURES

Author contributions: All authors contributed equally to this manuscript. D. Homenko is the article guarantor.

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