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Segmental colitis associated with diverticulosis

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INTRODUCTION

Patients with colonic diverticulosis can develop a segmental colitis most commonly in the sigmoid colon. The endoscopic and histologic features of segmental colitis associated with diverticulosis (SCAD) or diverticular colitis can range from mild inflammatory changes with submucosal hemorrhages to florid, chronic active inflammation that resembles inflammatory bowel disease [1-9].

This topic review will focus on the pathogenesis, clinical manifestations, diagnosis, and management of SCAD. The epidemiology, risk factors, and the pathogenesis of diverticulosis and the clinical manifestations, diagnosis, and management of diverticulitis and diverticular bleeding are discussed in detail, separately. (See "Colonic diverticulosis and diverticular disease: Epidemiology, risk factors, and pathogenesis" and "Clinical manifestations and diagnosis of acute colonic diverticulitis in adults" and "Acute colonic diverticulitis: Triage and inpatient management" and "Colonic diverticular bleeding".)

DEFINITIONS

- A diverticulum is a sac-like protrusion of the colonic wall.
- Diverticulosis is defined by the presence of diverticula.
- Diverticular disease is a nonspecific term that is often defined as any clinically significant complication of diverticulosis, which includes diverticular bleeding, diverticulitis,

segmental colitis associated with diverticulosis, or symptomatic uncomplicated diverticular disease.

- SCAD or diverticular colitis is characterized by chronic mucosal inflammation in a segment of colon with diverticula.
- Diverticulitis is defined as inflammation in and adjacent to a diverticulum. Diverticulitis may be acute or chronic, uncomplicated or complicated by a diverticular abscess, fistula, stricture, bowel obstruction, or free perforation. (See "Clinical manifestations and diagnosis of acute colonic diverticulitis in adults" and "Acute colonic diverticulitis: Triage and inpatient management".)
- Diverticular bleeding is characterized by painless, large-volume hematochezia due to segmental weakness of the vasa recta associated with a diverticulum. (See "Colonic diverticular bleeding".)
- Symptomatic uncomplicated diverticular disease (SUDD) is characterized by persistent abdominal pain attributed to diverticula in the absence of macroscopically overt colitis or diverticulitis. This is a controversial diagnosis and should not be confused with chronic diverticulitis or ongoing gastrointestinal symptoms after resolution of acute diverticulitis [10-12].

EPIDEMIOLOGY

SCAD is a rare diagnosis, mostly described in case reports. In a prospective study to estimate the prevalence of SCAD, no patient was diagnosed with SCAD among 1383 patients who underwent colonoscopy [9]. In another prospective study of 6230 colonoscopies performed for evaluation of gastrointestinal symptoms, the prevalence of SCAD was 1.5 percent [13]. SCAD is more common in males and older adults. However, SCAD has also been reported in younger patients.

PATHOGENESIS

The pathogenesis of SCAD is unclear. Mucosal prolapse, fecal stasis, and localized ischemia have all been implicated [1]. It is hypothesized that prolapsed mucosa, by being exposed to shear stress, may cause chronic mucosal inflammation. Inflammation may also result from changes in bacterial flora and bacterial enzyme activity due to fecal stasis in diverticulosis, changes in

permeability to intraluminal antigens, or altered microcirculation leading to focal mucosal ischemia.

CLINICAL FEATURES

Clinical manifestations — Patients with SCAD typically present with chronic diarrhea, cramping abdominal pain primarily in the left lower quadrant, and in some cases intermittent hematochezia [3,4]. Approximately one-third of patients have more than one symptom at the time of diagnosis [13].

Imaging findings — On abdominal computed tomography (CT) scan, patients with SCAD may have evidence of colonic wall thickening in the segment of colon with pre-existing diverticulosis. Pericolonic fat stranding may occasionally be seen.

Laboratory findings — The white blood cell count is usually normal in patients with SCAD but may be abnormal in patients with severe inflammation. Fecal calprotectin or lactoferrin may be elevated in the presence of severe intestinal inflammation [14].

DIAGNOSIS

SCAD is usually diagnosed with endoscopy during evaluation of chronic diarrhea and/or abdominal pain. The diagnosis of SCAD is made by the presence of inflammation on endoscopy and chronic inflammatory changes on biopsy only in an area of the colon with diverticula and absence of inflammation in the rectum. A history of diverticulitis is not necessary to make a diagnosis of SCAD.

Endoscopy — On endoscopy, SCAD is characterized by inflammation of the interdiverticular mucosa without involvement of the diverticular orifices. The distribution of inflammation characteristically includes the interdiverticular mucosa of the sigmoid colon and may involve descending colon, but spares the rectum [15,16].

The endoscopic features of diverticular colitis can vary in severity and have been classified into four subtypes [13]:

- Type A (crescentic fold pattern) – Characterized by reddish round lesions ranging from 0.5 to 1.5 cm in diameter at the top of the colonic folds
- Type B (mild to moderate ulcerative colitis-like pattern) – Characterized by loss of the submucosal vascular pattern, edema of the mucosa, hyperemia, and diffuse erosions

- Type C (Crohn colitis-like pattern) – Characterized by isolated aphthous ulcers
- Type D (severe ulcerative colitis-like pattern) – Characterized by loss of submucosa vascular pattern, intense hyperemia, diffuse ulcerations, and reduced caliber of colonic lumen

Histology — SCAD is characterized by the presence of chronic inflammatory changes, the extent of which varies based on subtype. Patients with SCAD have characteristically normal histology on rectal biopsies.

- Patients with SCAD in a crescentic fold pattern (endoscopic type A) often have lymphocyte and neutrophilic inflammatory infiltration with no significant alteration of the glandular architecture.
- Patients with an ulcerative colitis-like pattern of SCAD (type B and D) have chronic changes in the lamina propria including basal plasmacytosis, diffuse mixed inflammation in the lamina propria, Paneth cell metaplasia, distortion of crypt architecture, cryptitis, crypt abscesses, and crypt hemorrhage [1,3].
- Patients with Crohn colitis-like pattern of SCAD (type C) have transmural inflammation, fissures, epithelioid granulomas, and lymphohistiocytic vasculitis.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of SCAD includes other causes of chronic diarrhea and lower abdominal pain with evidence of colitis on endoscopy. SCAD can usually be distinguished from most other causes based upon the clinical features, laboratory studies, imaging, and the distribution of colitis on endoscopy and biopsy.

- **Acute uncomplicated diverticulitis** – Patients with acute uncomplicated diverticulitis may present with abdominal pain and a small percentage of patients may have diarrhea. On imaging, patients with acute uncomplicated diverticulitis have colonic thickening and paracolic fat stranding in the area of the colon with diverticula. However, in contrast to patients with SCAD, the colonic wall thickening is mild and the degree of paracolic fat stranding is more severe than expected for the degree of colonic wall thickening [17]. The inflammatory process may result in accumulation of fluid in the root of the sigmoid mesentery and engorgement of mesenteric vessels. On colonoscopy, acute uncomplicated diverticulitis primarily affects the diverticular orifices and peridiverticular mucosa [18]. By contrast, in SCAD, the inflammation primarily involves the interdiverticular mucosa with

sparing of the peridiverticular mucosa. (See "Clinical manifestations and diagnosis of acute colonic diverticulitis in adults".)

- **Inflammatory bowel disease** – The clinical presentation of SCAD is indistinguishable from inflammatory bowel disease (IBD). However, the distribution of the colitis helps to differentiate it from IBD. SCAD does not involve the distal rectum, whereas the rectum is always involved in ulcerative colitis. Even in cases of ulcerative colitis where the mucosa of the rectum appears normal on endoscopy, there is evidence of chronic inactive colitis on biopsy [19].

  SCAD can be differentiated from Crohn disease by the finding of Crohn disease elsewhere in the gastrointestinal tract (eg, terminal ileum, stomach). (See "Clinical manifestations, diagnosis, and prognosis of Crohn disease in adults", section on 'Differential diagnosis'.)

- **Medication-associated colitis** – Nonsteroidal anti-inflammatory drugs (NSAIDs) can cause chronic diarrhea and bleeding [20]. Other drugs that may cause a similar clinical presentation include retinoic acid, ipilimumab, and gold. The diagnosis is established by a history of medication use and the presence of nonspecific mucosal inflammation or mucosal erosions on biopsy that resemble ischemic changes. (See "NSAIDs: Adverse effects on the distal small bowel and colon".)

- **Infectious colitis** – Infectious colitis may have similar symptoms and endoscopic appearance to SCAD (table 1). However, infectious colitis usually has an acute presentation and can be excluded with stool and tissue cultures, stool studies, and on biopsies of the colon. (See "Approach to the adult with chronic diarrhea in resource-abundant settings", section on 'Initial evaluation'.)

- **Solitary rectal ulcer syndrome** – Patients with solitary rectal ulcer syndrome may have bleeding, abdominal pain, and altered bowel habits. Mucosal ulceration may be seen on endoscopy similar to SCAD, but solitary rectal ulcer syndrome has a characteristic appearance on histology with a thickened mucosal layer and distortion of crypt architecture. The lamina propria is replaced with smooth muscle and collagen leading to hypertrophy and disorganization of the muscularis mucosa.

- **Radiation colitis** – Radiation colitis may be seen weeks to years after abdominal or pelvic irradiation. Radiation colitis involving the sigmoid colon may have a similar endoscopic appearance to SCAD. However, histologic findings of radiation colitis include eosinophilic infiltrates, epithelial atypia, fibrosis, and capillary telangiectasia. (See "Radiation proctitis: Clinical manifestations, diagnosis, and management", section on 'Endoscopy'.)

- **Ischemic colitis** – Ischemic colitis can present with abdominal pain, bloody diarrhea, and an endoscopic appearance similar to SCAD. The symptoms of SCAD are often more chronic, while ischemic colitis is usually more of an acute presentation. (See "Colonic ischemia", section on 'Clinical features'.)

DISEASE COURSE

The natural history of SCAD is poorly understood. In a seven-year follow-up study of 15 patients with SCAD, eight patients had no clinical recurrence and five patients had mild sporadic recurrences. Of the 15 patients, two patients were diagnosed with Crohn disease in the follow-up period [21]. Progression to inflammatory bowel disease has also been reported in case series [21-24]. In a five-year follow-up study of 27 patients with SCAD, patients with more severe endoscopic findings at diagnosis had a more severe disease course and more often required treatment with immunosuppressants [25]. No patients were diagnosed with inflammatory bowel disease in follow-up.

MANAGEMENT

The optimal treatment of SCAD has not been well defined and is based on case reports and indirect evidence in patients with inflammatory bowel disease and pouchitis [2,4,5]. (See "Medical management of low-risk adult patients with mild to moderate ulcerative colitis" and "Management of acute and chronic pouchitis".)

- We suggest initial treatment with ciprofloxacin 500 mg orally twice daily and metronidazole 10 mg/kg per day in two or three divided doses for 10 to 14 days.
- In patients who do not respond to oral antibiotics, we add oral mesalamine (800 mg orally three times daily for 7 to 10 days), and if the response is inadequate in two to four weeks, we increase the dose to 1600 mg orally three times daily.
- In patients who fail to respond to antibiotics and mesalamine, we use prednisone 40 mg daily for one week and then gradually reduce the dose and discontinue prednisone over six weeks. Treatment with budesonide can also be considered.
- We reserve segmental resection for patients with steroid-refractory or steroid-dependent SCAD (glucocorticoids cannot be tapered to less than 10 mg/day within three months of starting steroids, without recurrent disease, or if relapse occurs within three months of stopping glucocorticoids).

- There are limited data to support the use of other agents in patients with SCAD. Case reports suggest treatment with immunomodulators and/or biologics, similar to inflammatory bowel disease, may benefit patients with SCAD refractory to treatment with antibiotics and mesalamine [26,27].

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Colonic diverticular disease".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Diverticulitis (The Basics)")
- Beyond the Basics topics (see "Patient education: Diverticular disease (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- **Epidemiology and pathogenesis** – Segmental colitis associated with diverticulosis (SCAD) or diverticular colitis is characterized by chronic inflammation in a segment of colon with diverticula.

  SCAD is uncommon and the pathogenesis of SCAD has yet to be defined. Mucosal prolapse, fecal stasis, and localized ischemia have all been implicated. (See 'Epidemiology'

above and 'Pathogenesis' above.)

- **Clinical presentation** – Patients with SCAD typically present with chronic diarrhea, cramping abdominal pain primarily in the left lower quadrant, and in some cases intermittent hematochezia. Approximately one-third of patients have more than one symptom at the time of diagnosis. (See 'Clinical features' above.)

- **Diagnosis** – SCAD is usually diagnosed incidentally during the course of evaluation of chronic diarrhea and/or abdominal pain. The diagnosis of SCAD is made by the presence of inflammation on endoscopy and chronic inflammatory changes on biopsy only in an area of the colon with diverticula (usually the sigmoid colon and in some cases the descending colon) and absence of inflammation in the rectum. (See 'Diagnosis' above.)

- **Differential diagnosis** – The differential diagnosis of SCAD includes other causes of chronic diarrhea and lower abdominal pain with evidence of colitis on endoscopy (table 1). SCAD can usually be distinguished from most other causes based upon the clinical features, laboratory studies, imaging, and the distribution of colitis on endoscopy and biopsy. (See 'Differential diagnosis' above.)

- **Management**

  - **Initial treatment** – We suggest initial treatment with antibiotics (**Grade 2C**). We typically use ciprofloxacin 500 mg orally twice daily and metronidazole 10 mg/kg per day in two or three divided doses for 10 to 14 days.

  - **Persistent symptoms** – In patients who do not respond adequately, we add mesalamine starting at 800 mg orally three times daily for 7 to 10 days, and if symptoms persist in two to four weeks, we increase the dose to 1600 mg orally three times daily. In patients with continued symptoms despite antibiotics and mesalamine, we use prednisone 40 mg daily for one week and then gradually reduce the dose and discontinue prednisone over six weeks. (See 'Management' above.)

- **Disease course** – The majority of patients with SCAD appear to respond to medical therapy. Progression to inflammatory bowel disease has been reported in some case series. (See 'Disease course' above.)

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GRAPHICS

Infectious mimics of inflammatory bowel disease

Infectious agents causing colitis and/or ileitis
Bacteria
Shigella species
Enterohemorrhagic *Escherichia coli*
Enteroinvasive *E. coli*
Campylobacter jejuni
Salmonella species (gastroenteritis and typhoid fever)
Yersinia enterocolitica
Mycobacterium tuberculosis
Clostridioides (formerly *Clostridium*) *difficile*
Vibrio parahaemolyticus
Chlamydia trachomatis (lymphogranuloma venereum serotypes)
Parasites
Entamoeba histolytica
Schistosoma species
Balantidium coli
Trichinella spiralis
Viruses
Cytomegalovirus
Infectious agents causing proctitis
Neisseria gonorrhoeae
Herpes simplex virus
C. trachomatis
Treponema pallidum
Cytomegalovirus

Adapted from: Guerrant RL, Lima AA. Inflammtory enteritides. In: Principles and Practice of Infectious Diseases, 5th ed, Mandell GL, Bennett JE, Dolin R (Eds), Churchill Livingstone, Philadelphia 2000. p.1127.

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