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ORIGINAL RESEARCH—CLINICAL

Clinical and Radiographic Characteristics in Segmental Colitis Associated With Diverticulosis, Diverticulitis, and Crohn's Disease

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BACKGROUND AND AIMS: Segmental colitis associated with diverticulosis (SCAD) is an underrecognized disease characterized by chronic segmental inflammation surrounding colonic diverticula. SCAD is recognized as an autonomous entity, although shares similar pathogenic and therapeutic aspects to inflammatory bowel disease and may be considered a complication of diverticulitis. We aimed to characterize the clinical, endoscopic, and radiographic findings of SCAD and its potential overlap with diverticulitis and inflammatory bowel disease. **METHODS:** All patients with suspected diagnosis of SCAD were identified using a bioinformatics search tool from January 1996 to October 2021 at our institution. Relevant demographic, clinical, endoscopic, and radiographic data were abstracted. Disease-related outcomes and radiographic characteristics were determined. **RESULTS:** Seventy-five patients with SCAD were included (48.0% female) with average age at diagnosis 62.5 years. Thirty-seven (49.3%) had a prior episode of diverticulitis. The most common presenting symptoms were abdominal pain (33.3%) and hematochezia (22.7%). Antibiotics (42.7%) and mesalamine (36.0%) were most used as first-line treatment options. Twenty (26.7%) required surgical intervention. The most common initial endoscopic finding was isolated sigmoid inflammation (86.7%). Fifty-one patients with confirmed SCAD, 72 with diverticulitis, and 12 with Crohn's disease (CD) had imaging available for review. Penetrating disease was seen in 7 (13.7%) with SCAD compared to 7 (9.7%) and 2 (16.6%) with diverticulitis and CD, respectively ($P = .14$). Blinded radiologists diagnosed SCAD, CD, or diverticulitis correctly in 43.8%, 8.3%, and 27.1%, respectively. **CONCLUSION:** SCAD should be considered when isolated sigmoid colon inflammation is seen on cross-sectional imaging. Penetrating disease is not a specific radiologic feature for either SCAD or diverticulitis. Further prospective studies are needed to correlate imaging characteristics with endoscopic findings to better describe radiographic features in SCAD.

Keywords: Segmental Colitis Associated With Diverticulosis; SCAD; Diverticulitis; Inflammatory Bowel Disease; Crohn's Disease

Introduction

Diverticular disease is a common condition globally, especially in Western countries. Annually in the United States, there are more than 1.9 million outpatient visits and 208,000 inpatient admissions for diverticulosis at a cost of $5.5 billion.[1] The incidence of diverticulitis in the United States is 180/100,000 persons per year. Although diverticulitis is most common in older adults, the relative increase in diverticulitis in recent decades has been greatest in younger adults. The incidence of diverticulitis in individuals 40–49 year old increased by 132% from 1980 through 2007.[2,3] Although most patients with diverticula are asymptomatic, around 25% of patients will experience symptoms and 5% of patients may have an episode of acute diverticulitis.[4]

Diverticulitis, symptomatic uncomplicated diverticular disease, and segmental colitis associated with diverticulosis (SCAD) comprise the spectrum of diverticular disease. SCAD is a chronic inflammatory process that affects colonic mucosa in segments that are also affected by diverticulosis, typically the descending and sigmoid colon with sparing of the proximal colon and rectum.[5] Inflammation may be associated within and/or surrounding the diverticula themselves. SCAD 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 inflammatory bowel disease (IBD).[6] To confirm SCAD, correct

Abbreviations used in this paper: **5-ASA, 5-aminosalicylate; CD, Crohn's disease; CT, computed tomography; GI, gastrointestinal; IBD, inflammatory bowel disease; SCAD, Segmental colitis associated with diverticulosis; SCADD, segmental colitis associated with diverticulosis or diverticulitis; UC, ulcerative colitis.**

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biopsy sampling, including biopsies on the borders of diverticula, proximal to the diverticular area, and in the rectum, are required. The range of histological findings associated with SCAD is variable, including mild nonspecific chronic inflammation and IBD-like changes, making the diagnosis difficult to establish.[5]

Due to underrecognition of SCAD and its overlap with other conditions, the exact prevalence of SCAD is difficult to determine.[7] The reported prevalence varies between 0.25% and 1.4% in the general population and 2%–11% amongst those with diverticular disease.[8–10] Different histological criteria and lack of diagnostic standardized features contribute to this wide range of prevalence. Variability of biopsy sampling among available studies may further contribute to heterogeneity. Due to its association with diverticular disease, the mean age at SCAD diagnosis has been reported to be in the early to mid-60s.[5,8,11] There also seems to be a slightly higher male predominance, with one meta-analysis showing 58.7% of 486 diagnosed cases were men. The exact pathogenesis of SCAD remains unclear, but it is multifactorial. Various potential mechanisms have been described including mucosal prolapse, fecal stasis, and localized ischemia.[7]

Whether SCAD exists as a separate entity, or belongs on the spectrum of IBD, is still unclear. Not only is the clinical presentation of SCAD like IBD, but SCAD can closely mimic the histological changes of ulcerative colitis (UC) and Crohn's disease (CD).[10] Altered cytoarchitectural structure can be seen in both UC and SCAD.[9] In patients with SCAD refractory to conventional therapies, infliximab has been used with good success.[12] Due to the clinical, endoscopic, and histologic overlap between SCAD and IBD, there is little data available regarding use of radiographic imaging techniques to differentiate between diverticulitis, SCAD, and IBD, specifically colonic CD.

In this retrospective study, we sought to define the clinical, endoscopic, and radiographic characteristics of SCAD to better determine disease-related outcomes and assist in differentiating this entity from diverticulitis and IBD.

Methods

Patient Population

This retrospective study was approved by our center's Institutional Review Board. Using an institutional bioinformatics tool to search the electronic medical record and clinical notes at our large referral center, we identified all patients >18-year-old using the following search terms: "SCAD," "segmental colitis associated with diverticulosis or diverticulitis (SCADD)," "segmental colitis," or "diverticulitis" between January 1996 and October 2021. This allowed for identification of patients in which a gastroenterologist was considering the diagnosis of SCAD. The medical records of only patients with research authorization were included. This was followed by manual review of individual patient charts to confirm a suspected diagnosis of SCAD as determined by the treating gastroenterologist. In patients meeting inclusion criteria, clinical data was then abstracted for various demographic, clinical, endoscopic, and radiologic outcomes. Patients were excluded for the following reasons: <18-year-old at the time of last follow-up; segmental colitis was secondary to ischemia or active infection, unknown or uncertain diagnosis, or the term "SCAD" was unrelated to the gastrointestinal (GI) system, such as segmental coronary artery dissection. Patients were categorized into 1 of 3 groups: SCAD, diverticulitis, or colonic CD.

Patient Data

Patient data collected included age at diagnosis, sex, smoking history, personal history of colon cancer, Clostridioides difficile infection, or abdominal surgery, and family history of IBD and diverticular disease based on review of the medical record.

SCAD

SCAD was defined as the presence of endoscopic inflammation in the left colon involving interdiverticular mucosa with sparing of the peridiverticular mucosa, right colon, and rectum as determined by the performing endoscopist and review of the medical record, including presence of typical symptoms (abdominal pain, change in bowel habit, and/or hematochezia) and histologic findings (cryptitis and crypt abscesses with expansion of the lamina propria by mononuclear cells, prominent basal lymphoid aggregates, features of chronicity including basal lymphoplasmacytosis, crypt distortion, and Paneth cell metaplasia, and histologic sparing of the rectum).[13]

Diverticulitis

Diverticulitis was defined as presence of clinical symptoms including abdominal pain, fever/chills, nausea or vomiting, and/or hematochezia as well as imaging findings consistent with the diagnosis per the treating gastroenterologist, and review of the medical record. Prior episodes of diverticulitis were determined by review of the medical record.

Crohn's Disease

IBD, specifically colonic CD, was defined based on clinical diagnostic criteria as per the treating gastroenterologist and review of the medical record. Diagnosis was supported by characteristic endoscopic, radiographic, and/or histologic findings.

Symptom Assessment

Clinical symptoms at the time of SCAD diagnosis were determined based on review of the medical record, including history of abdominal pain, diarrhea, constipation, and/or hematochezia. Duration of symptoms prior to diagnosis were recorded in months.

Medical records were then reviewed to determine management and treatment of symptoms related to a diagnosis of SCAD, including observation, antibiotics, 5-aminosalicylate (5-ASA) agents, corticosteroids, or combination therapy which was defined as 2 or more of the previously listed pharmacotherapies. Following treatment, clinical symptoms were reassessed at time of follow-up to determine response as

documented in the medical record. Outcomes were defined as resolution (completion of treatment and absence of symptoms), recurrence (persistent symptoms after completing treatment), surgery (underwent sigmoidectomy for persistent symptoms), or lost to follow-up (received a diagnosis of SCAD and appropriate treatment, but no follow-up to reassess symptom response). Clinical symptoms at time of reassessment included chronic pain, chronic diarrhea, persistent hematochezia, perforation, or presence of 2 or more symptoms. Subsequent treatment options were collected if patients had recurrence of symptoms and/or additional investigations performed. Duration of symptoms following treatment initiation to symptom reassessment were recorded in months.

At the time of follow-up, patient charts were reviewed to determine if a change in diagnosis occurred from SCAD to IBD and vice versa as documented in the medical record by the treating gastroenterologist.

Endoscopic Evaluation

For patients who had prior endoscopic procedures available for review, findings were categorized as no active inflammation, sigmoid colon inflammation only, left-sided colon inflammation (including sigmoid colon, but not proximal to splenic flexure) with or without rectal involvement, and presence of sigmoid stricture prior to and following treatment. SCAD was then classified into 4 phenotypes (Type A–D) based on review of endoscopic images and reports according to Tursi et al.[14] Type A, a crescentic fold pattern, was defined as round reddish lesions ranging from 0.5 to 1.5 cm in diameter at the top of mucosal folds. Type B, a mild to moderate UC-like pattern, was defined as loss of the submucosal vascular pattern, edema of the mucosa, hyperemia, and pinpoint erosions. Type C, a CD-like pattern, was defined as isolated aphthous ulcers. Type D, a severe UC-like pattern, was defined as similar to Type B, but more severe with diffuse ulceration and reduced caliber of lumen.[14]

At the time of follow-up and symptom reassessment, patient charts were reviewed to determine if additional endoscopic procedures were performed to assess response to treatment. Findings were categorized according to the above schema.

Radiographic Data

Abdominopelvic computed tomography (CT), CT enterography, or magnetic resonance enterography were reviewed of patients with either a final diagnosis of SCAD, diverticulitis, or CD at the end of initial clinical evaluation and diagnosis as outlined in the medical record. The CT or magnetic resonance imaging exam closest to the date of initial diagnosis was used as the baseline study. Patient groups were randomized, and exams were interpreted by 1 of 3 abdominal radiologists. The radiologists were blinded to the diagnosis (SCAD, diverticulitis, or CD). Data collected included length of inflammation, presence of diverticula (with or without inflammation), presence of pericolonic inflammation, coexisting bowel wall muscular hypertrophy, presence of fistula or sinus tract (extramural, intramural, or both), and bowel wall thickness ($<$5 mm, 5–10 mm, and $>$10 mm). The final imaging diagnosis determined by the radiologist (blinded by the final diagnosis) was recorded as SCAD, diverticulitis, CD, or a combination of these conditions.

Statistical Analysis

Patient characteristics and clinical data were presented as mean with standard deviation, median with range, or frequency with percentages. Descriptive statistics were used to report findings from each cohort. Categorical variables were reported as a unique count and percentage of the sample. Statistical analysis included analysis of variance to compare variances across the 3 distinct groups. Quantitative variables were analyzed using Chi-squared test. A P value $<.05$ was considered significant.

Results

The initial data search identified 697 patients with suspected diagnoses of SCAD, diverticulitis, and colonic CD. After manual review, 198 patients were included in the final analysis and 499 were excluded due to diagnoses of segmental coronary artery dissection or other GI-related conditions that did not meet inclusion criteria (Figure 1).

SCAD Cohort

Demographics. Baseline characteristics of patients with SCAD including demographic data are summarized in Table 1. The mean age at diagnosis was 62.5 (standard deviation $\pm$10.7) years (48.0% female). Thirty-seven

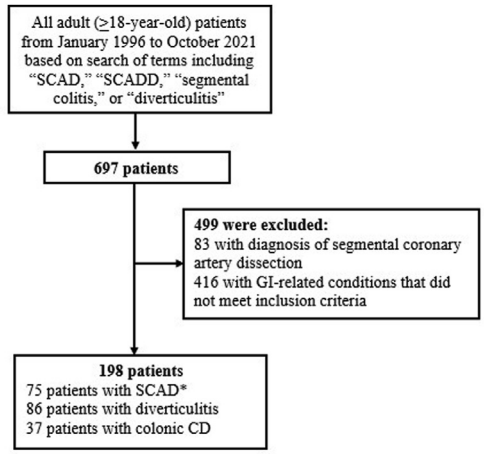


Figure 1. Screening of patients for study inclusion. Utilizing an institutional bioinformatics search tool, all adult (≥18-year-old) patients were identified using search terms including "SCAD," "SCADD," "segmental colitis," or "diverticulitis" who had research authorization. Medical records were reviewed to categorize patients into 1 of 3 groups: SCAD, diverticulitis, or Crohn's disease. A total of 499 patients were excluded. SCAD, Segmental colitis associated with diverticulosis; SCADD, segmental colitis associated with diverticulosis or diverticulitis.

Table 1. SCAD Demographics, Clinical Characteristics, and Clinical Outcomes

Clinical characteristic	SCAD (N = 75)
Age at diagnosis (y)	
Mean (SD) (range)	62.5 (±10.7)
Female sex, n (%)	36 (48.0)
Personal history of colorectal cancer, n (%)	1 (1.3)
Personal history of GI-related surgery, n (%)	2 (2.7)
Personal history of Clostridioides difficile infection	
Prior to SCAD diagnosis	5 (6.7)
After SCAD diagnosis	10 (13.3)
Prior episode of diverticulitis, n (%)	37 (49.3)
Family history of IBD, n (%)	4 (5.3)
Family history of diverticular disease, n (%)	1 (1.3)
Former/current tobacco use, n (%)	38 (50.7)
Duration of symptoms prior to SCAD diagnosis (mo)	
Median (range)	5 (1–48)
Primary symptom at time of SCAD diagnosis, n (%)	
None	3 (4.0)
Abdominal pain	25 (33.3)
Bowel disturbance (diarrhea)	11 (14.7)
Constipation	1 (1.3)
Hematochezia	17 (22.7)
Two or more symptoms	18 (24.0)
Initial endoscopic findings[a], n (%)	
No active inflammation	2 (2.7)
Sigmoid colon inflammation	65 (86.7)
Left-sided colon inflammation without rectal involvement	4 (5.3)
Sigmoid stricture	4 (5.3)
SCAD classification[b], n (%)	
No active inflammation	3 (4.0)
Type A	40 (53.3)
Type B	24 (32.0)
Type C	3 (4.0)
Type D	5 (6.7)
Initial choice of treatment, n (%)	
Observation	8 (10.7)
Medication	67 (89.3)
First medication prescribed, n (%)	
Antibiotics	32 (42.7)
Mesalamine	27 (36.0)
Corticosteroids	4 (5.3)
Two or more medications	4 (5.3)
Primary symptom following SCAD diagnosis, n (%)	
None	25 (33.3)
Chronic pain	20 (26.7)
Chronic bowel disturbance (diarrhea)	12 (16.0)
Persistent hematochezia	13 (17.3)
Bowel perforation	1 (1.3)
Subsequent interventions for recurrent or persistent symptoms, n (%)	
Observation	9 (12.0)
Antibiotics	6 (8.0)
Mesalamine	21 (28.0)
Corticosteroids	13 (17.3)
Biologic agents	2 (2.7)
Surgical intervention	9 (12.0)
IBD diagnosis changed to SCAD diagnosis on follow-up, n (%)	
Crohn's disease	2 (2.7)
Ulcerative colitis	10 (13.3)
SCAD diagnosis changed to IBD diagnosis on follow-up, n (%)	
Crohn's disease	4 (5.3)
Ulcerative colitis	12 (16.0)
Duration between SCAD and IBD diagnoses (mo)	n = 17
Median (range)	19 (10.5–36)
Final outcome following SCAD diagnosis[c], n (%)	
Resolved	18 (24.0)
Recurrence	14 (18.7)
Surgery	20 (26.7)
Lost to follow-up	19 (25.3)

Percentages were calculated on the basis of those with data available.
GI, gastrointestinal; IBD, inflammatory bowel disease; SCAD, segmental colitis associated with diverticulitis; SD, standard deviation.
[a]75 (100.0%) patients had an initial endoscopic procedure performed.
[b]SCAD, was classified into 4 phenotypes (Type A–D) based on review of endoscopic images and reports according to Schembri et al. Type A, a crescentic fold pattern, was defined as round reddish lesions ranging from 0.5 to 1.5 cm in diameter at the top of mucosal folds. Type B, a mild to moderate UC-like pattern, was defined as loss of the submucosal vascular pattern, edema of the mucosa, hyperemia, and pinpoint erosions. Type C, a CD-like pattern, was defined as isolated aphthous ulcers. Type D, a severe UC-like pattern, was defined as similar to Type B, but more severe with diffuse ulceration and reduced caliber of lumen.
[c]Outcomes were defined as resolution (completion of treatment and absence of symptoms), recurrence (persistent symptoms after completing treatment), surgery (underwent sigmoidectomy for persistent symptoms), or lost to follow-up (received a diagnosis of SCAD, and appropriate treatment, but no follow-up to reassess symptom response).

(49.3%) had a prior episode of diverticulitis. Thirty-eight (50.7%) had former or current tobacco use (Table 1).

Symptom assessment. Primary presenting symptoms included abdominal pain (33.3%) and hematochezia (22.7%). Antibiotics (42.7%) and 5-ASA agents (36.0%) were used as first-line treatment options. Recurrent or persistent symptoms occurred in most patients (61.3%). 5-ASA agents (28.0%) and oral corticosteroids (17.3%) were most used as a second treatment option if recurrent or persistent symptoms were noted. Twelve (16.0%) patients were given a diagnosis of IBD after the initial diagnosis of SCAD with a median of 19 (10.5–36) months between diagnoses. Most patients underwent surgical intervention

(26.7%) or were lost to follow-up (25.3%) following SCAD diagnosis (Table 1).

Endoscopic evaluation. Isolated sigmoid inflammation was the most common finding in 65 (86.7%) patients. Peridiverticular inflammation or "Type A" SCAD was most common in 40 (53.3%) patients. Subsequent endoscopic data was available in 39 (52.0%) patients with a second endoscopic procedure and 16 (21.3%) with a third endoscopic procedure. Mucosal inflammation involving the left colon and rectum was observed in 7 (9.3%) and 4 (5.3%) on the second and third endoscopy, respectively (Table 1 and Supplement 1).

Radiology Cohort – SCAD, Diverticulitis, and Colonic CD

Clinical and endoscopic characteristics. Fifty-one patients (49.0% female) with SCAD (mean age at diagnosis 61.3 years) had CT (n = 49) or magnetic resonance imaging (n = 2) exams available for review. Seventy-two with diverticulitis and 12 with colonic CD were also included for comparison. At the time of SCAD diagnosis, most (64.7%) had abdominal pain as the primary symptom with mean 8.8 (standard deviation ±10.2) months from the time of symptom onset to SCAD diagnosis. Most patients received 5-ASA agents (29.4%) and antibiotics (47.1%) following diagnosis of SCAD and developed persistent abdominal pain (37.3%) on follow-up (mean 15.2 ± 18.9 months). Most had evidence of isolated sigmoid inflammation (92.3%) endoscopically with "Type A" SCAD being most common (49.0%) (Table 2).

Radiographic characteristics. Forty-eight (94.1%) SCAD patients had segmental inflammation identified in the sigmoid colon on the reference scan with a mean length of 13.1 (±9.1) cm compared to 12.0 cm and 9.8 cm for CD and diverticulitis, respectively ($P = .05$). Forty-eight (94.1%) patients had diverticula visualized involving the sigmoid colon. Twenty (39.2%) had evidence of an inflamed diverticulum and 32 (62.7%) had pericolic stranding. Both characteristics were less common in patients with SCAD compared to diverticulitis ($P < .001$). Twenty-nine (56.9%) had sigmoid wall thickening present secondary to muscular hypertrophy. Penetrating disease (sinus tract or fistula) was seen in 7 (13.7%) patients with SCAD compared to 2 (16.6%) and 7 (9.7%) patients with CD and diverticulitis, respectively ($P = .14$) (Table 3).

On retrospective review, the radiologist felt the visualized sigmoid inflammation in patients with SCAD (n = 48) was secondary to SCAD in 21 (43.8%), diverticulitis in 13 (27.1%), SCAD ± diverticulitis in 8 (16.7%), CD in 4 (8.3%), CD and/or SCAD in 1 (2.1%), and CD and/or diverticulitis in 1 (2.1%) (Table 3).

Discussion

Our study shows that there is overlap between the clinical, endoscopic, and radiographic features of SCAD, diverticulitis, and colonic CD. Like prior studies, we identified older age, prior episodes of diverticulitis, and endoscopic inflammation isolated to the sigmoid colon as common features in patients with SCAD. However, on blinded radiology review of cross-sectional imaging studies in patients with SCAD, only 43.8% suspected SCAD as the cause of sigmoid inflammation. Therefore, radiographic information alone is not sufficient for diagnosis, but rather one must consider thorough review of clinical history and presentation as well as endoscopic evaluation to arrive at the correct diagnosis.

In recent decades, SCAD has been recognized as a distinctive clinical and pathological entity, although has significant overlap with IBD.[15] Age can be a key distinguishing feature as older individuals are more often affected by SCAD whereas IBD typically affects younger individuals. Endoscopically, older patients with CD are more likely to have distal colitis and proctitis rather than small and proximal large bowel involvement as seen in younger patients.[7] The rectum and proximal colon are spared from endoscopic and histologic inflammation in SCAD in contrast to IBD where UC most often affects the rectum and CD can affect any portion of the GI tract. CD can also preferentially affect areas of diverticulosis and in some patients, even giving rise to diverticulitis. SCAD often shows a benign course in comparison to IBD. Maintenance therapy is required in select patients with SCAD whereas maintenance treatment is mandatory in IBD to reduce risk of disease recurrence. At times, distinction between SCAD and IBD may be only possible at the time of surgery or on histological examination of the resected specimen. The extent to which SCAD may be a separate form of IBD or an early stage of colonic CD is unclear.[7,15–17]

Both diverticulitis and SCAD affect the same colonic distribution and share several similarities. Diverticulitis and SCAD most often affect older patient populations and exhibit a benign course. Surgery is required only for severe forms of these conditions. However, there are specific differences that can distinguish between diverticulitis and SCAD. The endoscopic appearance of SCAD typically shows inflammatory involvement of the interdiverticular mucosa with sparing of the peridiverticular mucosa which may be involved only in severe inflammation. In diverticulitis, inflammation affects peridiverticular mucosa and involvement of interdiverticular mucosa may occur only in severe disease. Moreover, the endoscopic and histologic appearance of SCAD is often like that of IBD.[16]

SCAD most often is seen in middle to older aged males presenting with rectal bleeding. The optimal treatment for SCAD is still undefined and is based on case series and treatment extrapolated from IBD. First line treatment typically includes use of antibiotics, 5-ASA agents, or a combination of the two together. In contrast to IBD, immunosuppressant agents, including corticosteroids, are rarely required in SCAD.[7] A prominent clinical feature evidenced in long-term studies is the frequency of spontaneous resolution and remission of symptoms with limited (oral 5-ASA agents) or no pharmacological therapy. Our study

Table 2. SCAD, Diverticulitis, and Colonic Crohn's Disease Demographics, Clinical Characteristics, and Endoscopic Outcomes

Clinical characteristic	SCAD (N = 51)[c]	Diverticulitis (N = 72)[d]	Crohn's disease (N = 12)[e]	*P* value
Age at diagnosis (y)				.01[a]
Mean (SD)	61.3 (±11.5)	58.6 (±13.8)	48.3 (±17.5)	
Female gender, n (%)	25 (49.0)	49 (68.1)	4 (33.3)	.02[b]
Prior episode of diverticulitis, n (%)	35 (68.6)	71 (98.6)	0 (0)	<.001[b]
Former/current tobacco use, n (%)	27 (52.9)	30 (41.7)	7 (58.3)	.43[b]
Clostridioides difficile history, n (%)				.55[b]
Prior to diagnosis	5 (9.8)	2 (2.8)	0 (0.0)	
After diagnosis	4 (7.8)	5 (6.9)	0 (0.0)	
>2 episodes	1 (2.0)	1 (1.4)	0 (0.0)	
Primary symptom at diagnosis, n (%)				<.001[b]
Abdominal pain	33 (64.7)	70 (97.2)	4 (33.3)	
Diarrhea	13 (25.5)	8 (11.1)	4 (33.3)	
Constipation	1 (2.0)	2 (2.8)	0 (0.0)	
Hematochezia	18 (35.3)	5 (6.9)	4 (33.3)	
Two or more symptoms	14 (27.5)	12 (16.7)	0 (0.0)	
Endoscopic findings, n (%)				<.001[b]
No active inflammation	2 (3.9)	21 (29.2)	0 (0.0)	
Sigmoid inflammation	47 (92.3)	12 (16.7)	12 (100.0)	
Left-sided colon inflammation without rectal involvement	2 (3.9)	0 (0.0)	0 (0.0)	
SCAD classification, n (%)[f]				
No active inflammation	3 (5.9)			
Type A	25 (49.0)			
Type B	15 (29.4)			
Type C	3 (5.9)			
Type D	5 (9.8)			
Duration of symptoms prior to diagnosis[g]				
Mean (SD)	8.8 (±10.2)	1.7 (±1.6)	8.1 (±5.9)	<.01[a]
SCAD treatment, n (%)				
Observation	5 (9.8)			
Antibiotics	24 (47.1)			
Mesalamine	15 (29.4)			
Corticosteroids	4 (7.8)			
Combination therapy[h]	3 (5.9)			
Primary symptom after initial management, n (%)				.02[b]
None	15 (29.4)	21 (29.2)	6 (50.0)	
Chronic pain	19 (37.3)	46 (63.9)	3 (25.0)	
Chronic diarrhea	9 (17.6)	8 (11.1)	3 (25.0)	
Persistent hematochezia	6 (11.8)	1 (1.4)	0 (0.0)	
Perforation	1 (2.0)	1 (1.4)	0 (0.0)	
Two or more symptoms	14 (27.5)	5 (6.9)	0 (0.0)	
Duration of symptoms after diagnosis[g]				<.01[a]
Median (range)	15.2 (±18.9)	2.9 (±1.7)	17.1 (±14.2)	

Percentages were calculated on the basis of those with data available.
SCAD, segmental colitis associated with diverticulitis; SD, standard deviation.
[a]Analysis of variance (ANOVA).
[b]Chi-squared test.
[c]51 (100.0%) patients had an endoscopic procedure performed.
[d]33 (45.8%) patients had an endoscopic procedure performed.
[e]12 (100.0%) patients had an endoscopic procedure performed.
[f]SCAD, was classified into 4 phenotypes (Type A–-D) based on review of endoscopic images and reports according to Schembri et al. Type A, a crescentic fold pattern, was defined as round reddish lesions ranging from 0.5 to 1.5 cm in diameter at the top of mucosal folds. Type B, a mild to moderate UC-like pattern, was defined as loss of the submucosal vascular pattern, edema of the mucosa, hyperemia, and pinpoint erosions. Type C, a CD-like pattern, was defined as isolated aphthous ulcers. Type D, a severe UC-like pattern, was defined as similar to Type B, but more severe with diffuse ulceration and reduced caliber of lumen.
[g]Duration of symptoms prior to diagnosis for SCAD, and Crohn's disease was measured in months. Duration of symptoms prior to diagnosis for diverticulitis was measured in weeks.
[h]Combination therapy was defined as at least 2 medications including antibiotics, mesalamine, and/or corticosteroids.

Table 3. Radiographic Features of SCAD, Diverticulitis, and Crohn's Disease

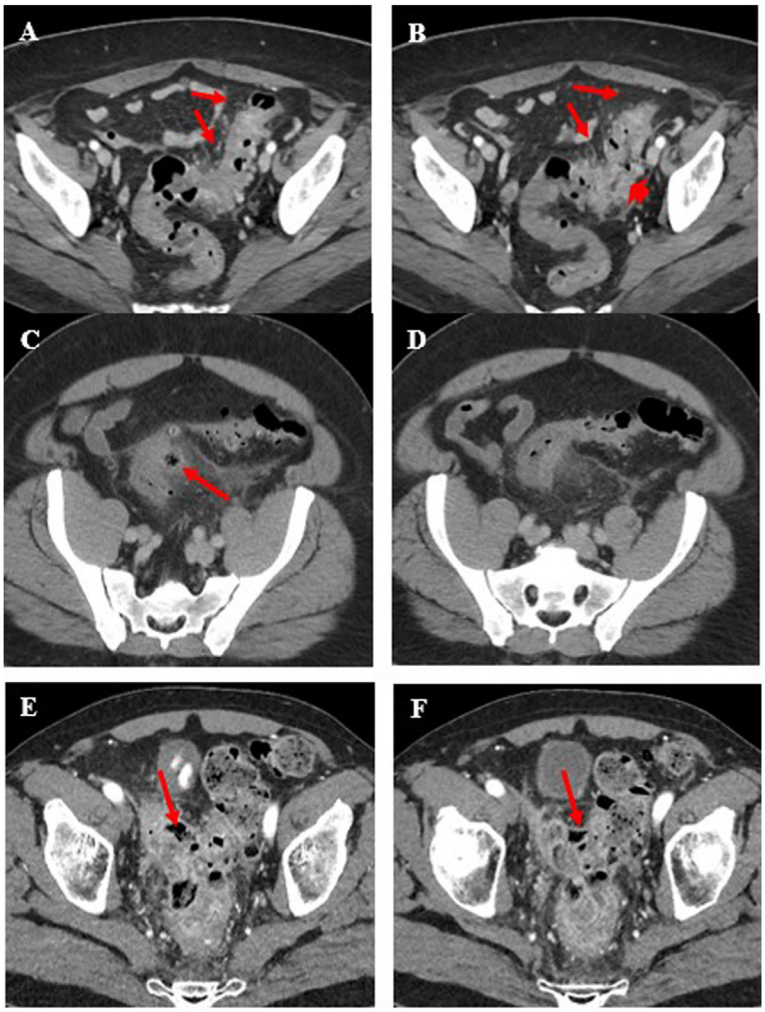
Radiographic characteristic	SCAD (N = 51)	Diverticulitis (N = 72)	Crohn's disease (N = 12)	*P* value
Presence of inflamed diverticula, n (%)	20 (39.2)	56 (77.8)	1 (8.3)	<.001[c]
Pericolic stranding, n (%)	32 (62.7)	66 (91.7)	8 (66.7)	<.001[c]
Peri-inflammatory free fluid, n (%)	6 (11.8)	18 (25.0)	2 (16.7)	.15[c]
Length of colitis (cm)				.05[b]
Mean (SD)	13.1 (±9.1)	9.8 (±4)	12 (±6.7)	
Presence of muscular hypertrophy, n (%)	29 (56.9)	36 (50.0)	7 (58.3)	.61[c]
Wall thickening				.43[c]
<5 mm	5 (9.8)	6 (8.3)	2 (16.7)	
5–10 mm	22 (43.1)	37 (51.4)	4 (33.3)	
>10 mm	24 (47.1)	29 (40.3)	6 (50.0)	
Presence of fistula or sinus tract, n (%)				.14[c]
Absent	44 (86.3)	65 (90.3)	10 (83.3)	
Extramural	0 (0.0)	5 (6.9)	1 (8.3)	
Intramural	5 (9.8)	2 (2.8)	1 (8.3)	
Both extramural and intramural	2 (3.9)	0 (0.0)	0 (0.0)	
Final imaging diagnosis after blinded imaging review[a], n (%)				
SCAD	21 (43.8)			
Diverticulitis	13 (27.1)			
SCAD and diverticulitis	8 (16.7)			
Crohn's disease	4 (8.3)			
Crohn's disease and/or SCAD	1 (2.1)			
Crohn's disease and/or diverticulitis	1 (2.1)			

Percentages were calculated on the basis of those with data available.
cm, centimeter; SCAD, segmental colitis associated with diverticulitis; SD, standard deviation.
[a]48 (94.1%) of the 51 SCAD, patients were included in the blinded retrospective imaging review.
[b]Analysis of variance (ANOVA).
[c]Chi-squared test.

showed 33.3% of patients had resolution of symptoms on follow-up, but a considerable proportion had persistent abdominal pain (26.7%). In these patients, the use of corticosteroids or other immunosuppressant medications may be beneficial. The percentage of patients that had ongoing symptoms in our study was greater than other reported studies.[18–21] However, most patients in this cohort were treated with antibiotics and 5-ASA agents, like prior studies. Surgery is usually reserved for cases refractory to medical treatment, although it is possible these cases may represent IBD initially misdiagnosed as SCAD.[7] Unlike other reported SCAD populations, 26.7% of our SCAD cohort underwent surgical intervention. Other studies in the literature report surgical resection is rare.[15,17,20,21] The results from this study may reflect referral bias due to the nature of our clinical practice.

Based on endoscopic features, SCAD has been classified into 4 different subtypes. Sparing of the diverticular orifices from inflammation is a unifying feature between all SCAD subtypes. This may help to distinguish from IBD, especially since all the histological parameters typically observed in IBD can be present in SCAD although to a milder extent. Type A or "crescentic fold disease" is the most common pattern of SCAD whereas the other descriptive endoscopic subtypes B, C, and D are thought to be similar to UC-like, CD-like, and severe UC-like, respectively.[7,22] Patients with relapse of SCAD may show the same or similar types of endoscopic lesions diagnosed at the onset of disease. Prior studies have demonstrated type B and D SCAD may fail to maintain long-term remission, often requiring immunosuppressive treatment.[22] Type A and C SCAD may have a more benign course; however, long-term treatment may guarantee longer periods of remission in those patients.[17] In our study, most patients with SCAD had isolated sigmoid colon inflammation (86.7%) with over half categorized as Type A based on review of endoscopic reports and images. In comparison to prior studies, we demonstrated that patients with SCAD continued to exhibit primarily isolated sigmoid inflammation on second (30.7%) and third (9.3%) endoscopic procedures when available.

There is a paucity of data regarding the radiographic features associated with SCAD, due to the underrecognized nature of SCAD and the substantial overlap between this condition, diverticulitis, and IBD. In this study, 48 (94.1%) SCAD patients had longer length of segmental inflammation identified in the sigmoid colon on the reference scan in comparison to patients with CD and diverticulitis, which was statistically significant ($P = .05$). Twenty (39.2%) had evidence of an inflamed diverticulum and 32 (62.7%) had pericolic stranding (Figure 2). Both characteristics were less common in patients with SCAD compared to diverticulitis ($P < .001$). Overall, imaging findings can overlap with diverticulitis and isolated Crohn's colitis, making diagnosis challenging. Penetrating disease can occur in SCAD.



Key: SCAD, segmental colitis associated with diverticulosis

Figure 2. Cross-sectional imaging features associated with SCAD. Panels A and B demonstrate CT imaging findings most suggestive of SCAD including sigmoid diverticulosis with long segment wall thickening and inflammation (arrows) and a small amount of surrounding fluid (arrowhead). Focally inflamed diverticulum not identified distinguishing it from acute diverticulitis. Panels C and D demonstrate CT images of inflamed sigmoid diverticulum (arrow) with surrounding fat stranding and short segment wall thickening. This patient had SCAD with superimposed acute diverticulitis, demonstrating the radiographic overlap that can occur. Panels E and F demonstrate CT images with marked wall thickening involving sigmoid colon and proximal rectum with diverticulosis. There is air and fluid filled intramural sinus tract (arrows) in the sigmoid colon. Penetrating disease is more commonly found in SCAD and Crohn's disease compared to diverticulitis but is a nonspecific imaging finding that can be seen in all 3 diseases. CT, computed tomography; SCAD, Segmental colitis associated with diverticulosis.

However, due to increased incidence of coexisting diverticulitis in SCAD, it is difficult to determine which exact etiology may cause penetrating features.

The limitations of this study include it being performed at a single large referral center which may limit the study's generalizability. This study also had limited follow-up of 19 months and a substantial proportion of patients were lost to follow-up (25.3%). Furthermore, SCAD endoscopic subtyping is not routinely utilized consistently in clinical practice. Subtype classification of SCAD required independent review of endoscopic reports and images as documented in the electronic medical record. This was also a retrospective study, and as a result, there was significant reliance on documentation within the electronic medical record. However, our study consisted of many patients with SCAD who had serial endoscopic evaluations, robust outcomes data, and blinded review of radiographic features by expert GI radiologists.

In conclusion, SCAD is an uncommon and poorly understood disease characterized by chronic segmental inflammation surrounding colonic diverticula. Like prior studies, we identified older age, prior episodes of diverticulitis, and endoscopic inflammation isolated to the sigmoid colon as common features in patients with SCAD. This entity should be considered in the differential diagnosis when inflammation is seen within the sigmoid colon on cross-sectional imaging. The imaging findings can overlap with diverticulitis and isolated Crohn's colitis, making diagnosis challenging. Penetrating disease can occur in SCAD. However, due to increased incidence of coexisting diverticulitis in SCAD, it is difficult to determine which may cause penetrating features. Further, more extensive studies are needed to correlate imaging characteristics with endoscopic findings to better describe the radiographic features in SCAD.

Supplementary Materials

Material associated with this article can be found, in the online version, at https://doi.org/10.1016/j.gastha.2024.06.002.

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The data that support the findings of this study are available from the corresponding author upon reasonable request.

Reporting Guidelines:
The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement and checklist was utilized for reporting this study.