

## Surgical Resection As Treatment For Segmental Colitis Associated With Diverticulosis (SCAD)

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# Surgical Resection As Treatment For Segmental Colitis Associated With Diverticulosis (SCAD)

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## Abstract

**Background:** Mucosal inflammation isolated to a segment of colon affected by diverticular disease with relative sparing of the rectum refers to Segmental Colitis Associated with Diverticulosis (SCAD, also known as Diverticular Disease-Associated Colitis (DDAC)). Although SCAD is relatively rare, its clinical and pathologic resemblance to inflammatory bowel disease (IBD) represents a diagnostic challenge for clinicians and misdiagnosis can lead to its mismanagement.

**Objective:** This study aimed to demonstrate that early elective colonic resection for symptomatic SCAD should be the treatment following a short course of conservative management with mesalamine derivatives.

**Design:** This was a single-institution, retrospective observational study combined with a systematic review of the literature.

**Data Sources:** Morristown Medical Center institutional database. Medline. EMBASE. Cochrane.

**Interventions:** Medical therapy included anti-inflammatory and immunosuppressive agents currently used for IBD. Surgical therapy included segmental colonic or more extensive resections with or without primary anastomosis.

**Outcome Measures:** Symptomatic or endoscopic recurrences at follow up

**Results:** Our literature review yielded seventeen studies with two hundred-forty patients. Of these, 169 patients were managed medically. Eighty-five patients either initially or eventually required surgery. Study duration ranged from 0 to 15 years. Recurrence rate in the medically managed cohort was 26.0% (44/169) compared to the surgical cohort 10.5% (9/85). In our database, we found a total of seven patients (5 males and 2 females; mean age 62.9) all with endoscopically diagnosed acute-on-chronic colitis associated with diverticular disease. Two patients underwent Hartmann's procedure, and five underwent segmental colectomy with primary anastomosis. Follow up ranged from 0-5 years, and four patients had surveillance colonoscopies that were negative for recurrence.

Limitations: The study was limited by its retrospective nature and small sample size.

Conclusions: Medical treatment does not alter the natural history of SCAD, and more aggressive subtypes will chronically recur or develop complications. In our experience, surgical intervention following a short treatment course of Mesalamine treatment will afford patients treatment with low morbidity and improved quality of life.

## INTRODUCTION

Segmental Colitis Associated with Diverticulosis (SCAD) refers to mucosal inflammation isolated to a segment of colon affected by diverticular disease with relative sparing of the rectum. The reported endoscopic prevalence of SCAD is between 0.25-1.48% in patients with diverticulosis<sup>1, 2, 3, 4</sup>. SCAD often presents with diarrhea, hematochezia, lower abdominal pain, and tenesmus<sup>1, 4</sup>. The progression of SCAD can lead to refractory anemia, colonic obstruction due to stricture, and visceral perforation. Interestingly, SCAD does not seem to increase cancer risk<sup>5</sup>.

Despite its clinical and pathologic resemblance to inflammatory bowel disease (IBD), SCAD is a distinct clinical entity. Unlike IBD, SCAD is often isolated to the sigmoid colon sparing the rectum and predominantly affects males and the elderly, with age of onset ranging from 61-67 years<sup>2</sup>. Although its endoscopic pattern is variable, the features of SCAD include patchy mucosal areas of erythema, granularity, and friability. Unique to SCAD, the inflammation is often confined to an area of diverticulosis and often spares the diverticular orifice<sup>3</sup>. Histologically, SCAD is characterized by chronic inflammatory infiltrate with lymphomononuclear invasion of the lamina propria, with no specific alteration in glandular architecture, and very rarely granulomas<sup>6</sup>.

There are currently several theories on the pathophysiology of SCAD. SCAD may be an atypical form of IBD secondary to diverticular disease<sup>7</sup>. Chronic mucosal inflammation could also be due to maximal shear forces on prolapsed mucosa. It is also possible that fecal stasis in diverticulae leads to changes in bacterial flora and bacterial enzyme activity. Lastly, the pathogenesis of SCAD has also been related to the increased mucosal permeability of intraluminal antigens and changes to microcirculation<sup>5</sup>.

Currently, acceptable treatment of SCAD is medical, surgical, or a combination of both. Medical treatment currently includes mesalamine derivatives, corticosteroids, probiotics, and biologic agents<sup>5, 8, 9, 10</sup>. Even with aggressive medical therapy, however, SCAD may persist, relapse, or progress. We propose that segmental colonic resection is an alternative to long-term anti-inflammatory and/or immunosuppressive therapy.

In this study we present our own experience in the treatment of SCAD patients at Morristown Medical Center as well as the experience reported in the literature utilizing several meta-analyses of outcomes following the treatment of SCAD patients.

## METHODS

### *Inclusion Criteria*

We conducted a search with Medline (January 1966 to March 2013), EMBASE, and Cochrane databases. Our search engines included PubMed, Ovid, and Google Scholar. The following medical subject heading (MeSH) terms were used with no language restriction: “inflammatory bowel disease,” “colitis,” “ulcerative colitis,” “Crohn’s disease,” “diverticulitis,” “diverticular associated colitis,” “diverticular disease associated colitis,” “segmental colitis,” and “segmental colitis associated with diverticular disease.” Additional studies cited within the literature were investigated. Based on the title and abstract of the publication, we downloaded full articles through our library. Included articles contained patient cohorts with a diagnosis of SCAD or DDAC (diverticular disease associated with colitis) based on accepted clinical, endoscopic, or histologic findings. Most articles contained reports with clinical data such as age of onset, treatment, recurrence of symptoms, and follow-up time.

A search was then performed on our institutional database for the following terms: “inflammatory bowel disease,” “colitis,” “ulcerative colitis,” “Crohn’s disease,” “diverticulitis,” “diverticular associated colitis,” “diverticular disease associated colitis,” “segmental colitis,” and “segmental colitis associated with diverticular disease.” We then proceeded to review the diagnostic and pathologic data to confirm a definitive diagnosis of segmental colitis associated with diverticular disease based on accepted clinical, endoscopic, or histologic findings.

### *Exclusion Criteria*

Based on our primary screening criteria, we excluded letters, comments, and reviews with insufficient details.

### *Data Extraction*

Data were appraised by two primary reviewers. Each article was comprehensively reviewed to determine whether it met the inclusion and exclusion criteria. The authors abstracted the following data from each report: first author, year of publication, journal, total number of cases, and population demographics.

Included in this systematic review were data from 7 patients treated at our institution (Section of Colon and Rectal Surgery; Morristown Medical Center, Morristown, NJ) over a 10-year period whom met the inclusion criteria. Patient data were retrieved following Institutional Review Board approval according to the medical center Institutional Review Board guidelines. Clinical data included

age, gender, symptoms, endoscopy results, and histology suggestive of concurrent diverticular disease and IBD, treatments instituted (medical and surgical), and recurrence of symptoms. Symptoms of SCAD included primarily left lower quadrant abdominal pain, rectal bleeding, tenesmus, and diarrhea. The clinical diagnosis of SCAD was made based on the results of endoscopic, radiologic, and pathologic reports. Endoscopic findings included inflamed friable, edematous mucosa, predominantly in an area of colon affected with diverticula. After initial diagnosis, a gastrointestinal pathologist with a special interest in IBD reviewed endoscopic biopsies or surgical specimens in all 7 patients. Histologic changes included mucosal inflammation, distorted crypt architecture, cryptitis, crypt abscesses, granulomas, pericolonic fibrosis, and acute on chronic colitis.

## RESULTS

A total of 17 studies met our predefined inclusion criteria yielding a total of 240 patients (Table 1). Of the 17 studies, 6 were from centers in the United States<sup>12, 13, 15-18, 24</sup>, 2 were from Canada<sup>5, 19</sup>, and 8 were from Europe<sup>4, 14, 20-23</sup>. The studies were conducted over a 37-year period from 1974 to 2011. Ten of the 17 studies were retrospective cohort studies, 6 were case reports, and 1 was a prospective cohort study. The number of participants in each study ranged from 1 to 34. In contrast to IBD, the average age of onset was older and ranged from 50-73. Male gender was predominant among the participants (60%). Study duration ranged from 0 to 15 years. Eleven studies described their entire cohort as having SCAD. The reported clinical and histologic presentation of SCAD patients varied amongst studies. Patients often presented with abdominal pain, diarrhea, tenesmus, and/or hematochezia. Histopathological features of surgical or biopsy specimens consistently had a diffuse pattern of inflammation with focal involvement containing mononuclear infiltrate into the lamina propria and cryptitis bordered by normal mucosa.

In our systematic review, most participants (70.4%, 169 of 240) were medically managed with a step-up approach including mesalamine derivatives, antibiotics, probiotics, corticosteroids and biologic agents. Surgical intervention was initially or eventually required in eighty-five patients (35.4%, 85 of 240). Indications for surgery included large bowel obstruction, perforation, or refractory rectal bleeding with anemia. The type of surgery varied from total abdominal colectomy, proctocolectomy, and segmental colectomy with or without primary anastomosis. The symptomatic or endoscopic recurrence rate of SCAD was 26.0% (44 of 169) following medical therapy in contrast to 10.5% (9 of 85) after surgical intervention.

In our case series, we report a total of seven SCAD patients requiring surgical intervention (Table 2). As shown, there were 5 males (71.4%) and 2 females (28.6%). The mean age of the entire group was 62.9 years (males 57.8 years; females 75.5 years). At initial presentation, 2 of the 7 patients presented with perforation (28.6%) and the other patients presented with abdominal pain, obstruction, and/or lower gastrointestinal bleeding. Histological examination of the specimens included diverticulosis and/or diverticulitis with pericolic and peridiverticular inflammation, abscesses, and fibrosis consistent with previously described histologic features of SCAD. Medical therapy consisting of 5-ASA derivatives and steroids had been initiated in 5 of 7 patients. Of the 5 patients on medical therapy, 2 progressed to develop visceral perforation requiring an emergent Hartmann's procedure, and one eventually developed large bowel obstruction requiring segmental colonic resection. After surgical intervention, follow up time ranged from 1 to 5 years. Postoperatively, all patients experienced resolution of their symptoms. Four of the 7 patients that had follow up colonoscopies required surgery, and 3 of 4 had patent anastomoses with healthy mucosa. One patient had a focal area of rectal ulceration and colonic stricture with pathology consistent with ischemic colitis.

## DISCUSSION

Although the pathogenesis of SCAD has yet to be elucidated, its clinical, endoscopic, and histologic similarities to inflammatory bowel disease represent a diagnostic challenge for clinicians, and misdiagnosis can lead to mismanagement. Due to its relative obscurity and subtle features, it is tempting to treat SCAD as IBD when deriving a diagnosis and formulating a treatment plan.

Currently, medical therapy for SCAD often includes the same anti-inflammatory/immunosuppressive regimens as IBD such as mesalamine derivatives, antibiotics, probiotic agents, corticosteroids, and TNF-alpha inhibitors<sup>6, 8, 10, 12, 22</sup>. Despite recent optimism in inducing remission with these agents, there are several problems with long-term immunosuppressive therapy in SCAD. First, the unnecessary side effects of these agents such as delayed wound healing, increased susceptibility to infections, etc. negatively impact patients' quality of life as well as their tolerance and adherence to these medications<sup>25, 26</sup>. Secondly, despite medical therapy with one or multiple agents, there is still an unacceptably high recurrence rate with 1 out of 4 SCAD patients experiencing symptomatic relapse or disease progression during their medication treatment course. Therefore, we believe that medical treatment does not alter the natural history of SCAD, and more aggressive subtypes can progress to colonic

stricturing or visceral perforation<sup>5, 27</sup>. In a previous study, Makapugay *et al* reported that SCAD patients were predisposed to surgical intervention compared to patients with non-colitic diverticular disease. Therefore, it was concluded that active chronic inflammation of the colonic mucosa might alter the clinical course of patients with diverticular disease and predispose them to requiring surgical intervention.

Current indications for surgical management in SCAD include: symptoms refractory to medical management, intractable rectal bleeding with anemia, colonic stricturing, and visceral perforation leading to abdominal catastrophe. Surgical options for SCAD include segmental resection of the diseased portion with primary anastomosis, Hartmann's procedure (especially for visceral perforation), proctocolectomy, and total abdominal colectomy for diffuse disease. Despite the different treatment modalities, the reported recurrence rate of symptoms after surgery is low<sup>5, 6</sup>. Koutroubakis *et al* reported a series of four SCAD cases unresponsive to medical treatment that subsequently underwent a sigmoid colectomy, and none of the four patients had symptomatic recurrence<sup>13</sup>.

Since SCAD is a localized inflammatory process associated with diverticulosis, we surmise that SCAD is a manifestation of complicated diverticular disease. With surgical resection of the diseased area, source control of the inflammation can be achieved and long-term remission can be induced without the untoward side effects of long-term immunosuppression. Consistent with previous studies, our experience demonstrates that elective early surgical intervention following only a short treatment course with mesalamine derivatives can provide definitive treatment and improved quality of life.

The weaknesses of this study include its retrospective data acquisition, small sample size of patients that underwent surgical intervention, heterogeneity of definitions and diagnosis of SCAD in the literature, and variable follow-up intervals. Due to the relative anonymity of this condition to the surgical community, we hope that this paper will shed light on SCAD and propel further investigation to better define, diagnose, and treat this disease. In the near future, we hope to initiate biomarker analysis of specimens to aid in the clinical differentiation of SCAD from IBD. In order to recognize more aggressive SCAD subtypes earlier, the authors hope to further define clinical, biochemical, endoscopic, and histologic features of through subgroup analysis and collaboration.



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Reference	Year	Design	N	DAC Cases	Age of Onset	Gender (n male)	Medical Treatment	Recurrence (n) and Percentage (%) after Medical Tx	Surgical treatment	Recurrence (n) and Percentage (%) after Surgical Tx	Follow up (years)
Sladen &Filipe	1984	CR	5	3	59	4	5	2, 40%	1 (SC)	0, 0%	0.7
Gore	1992	RC	34	8	60	24	17	0, 0%	2 (SC)	0, 0%	NR
Peppercorn	1992	RC	8	8	71	6	6	3, 50% %	0	0, 0%	4
Hart	1995	RC	14	12	61	9	14	4, 28.6%	0	0, 0%	2.25-7.75
Van Rosendaal & Anderson	1996	CR	2	2	73	2	0	0, 0%	2 (NR)	0, 0%	1
Gledhill & Dixon	1999	RC	11	9	64	3	0	0, 0%	11 (TC,SC)	1, 9%	0-15
Makapugay & Dean	1996	PC	23	23	69	12	23	5, 21.7%	5 (SC)	0, 0%	0.33-8
Pereira	1998	CR	3	3	62	3	0	0, 0%	3 (SC)	2, 66.7%	1.5
Goldstein	2000	RC	29	25	61	19	0	0, 0%	29 (SC)	2, 6.9%	6
Evans	2002	CR	4	4	65	1	4	2, 50%	2 (SC)	0, 0%	0.36

Jani	2002	CR	1	1	69	1	1	0, 0%	1 (SC)	0, 0%	0.75
Koutroubakis	2005	RC	23	4	62	17	23	0, 0%	4 (SC)	0, 0%	3.2
Imperali	2006	RC	15	15	63	9	15	5, 33.3%	2 (SC)	0, 0%	7
Freeman	2008	RC	24	24	50.2	14	21	6, 28.6%	4 (SC)	1, 25%	4.75
Mullhall	2009	RC	13	13	61	4	13	7, 53.8%	7(TC, SC)	3, 42.9%	3.5
Tursi	2012	RC	27	27	63.7	13	27	8, 29.6%	8 (NR)	0, 0%	5
Total			240	195		143	169, 70.4%	44, 26%	85, 35.4%	9, 10.5%	

**Table 1.** Summary of literature, treatment, and recurrence rates.

<b>Patient</b>	<b>Presentation</b>	<b>Preop Treatment</b>	<b>Surgery</b>	<b>Follow up Time</b>	<b>Outcome</b>
60 M	Perforation	Colazal and Prednisone	Hartmann (eventual reversal)	5 years	No Recurrence
82 F	Perforation	Apriso and Asacol	Hartmann	2 years	No Recurrence
64 M	Colon Obstruction	None	Segmental Resection	2 years	No Recurrence
69 F	Colon Obstruction	Mesalamine and Asacol	Segmental Resection	1 year	No Recurrence
71 M	Abdominal Pain	None	Segmental Resection	2 years	No Recurrence
37 M	Rectal Bleeding	Steroids	Segmental Resection	1 years	No Recurrence
57 M	Rectal Bleeding	Pentasa	Segmental Resection	4 years	No Recurrence

**Table 2.** Our SCAD patient experience and outcomes.