Chapter

Segmental Colitis Associated with Diverticulosis

Rafael Luís Luporini, Marcel Domeniconi, Ana Carolina Parra, André Rizzo, Daniela Freitas, Sthefânia Frizoland Antonio Tursi

Abstract

Segmental colitis associated with diverticulosis is a pathology of recent knowledge, whose pathogenesis is still poorly defined. Diagnosis is mainly based on colonoscopy and histopathological study. Clinical features are chronic diarrhea, abdominal cramps in the lower right quadrant, and intermittent hematochezia. The diagnosis is evidenced by colonoscopy showing inflammation in the colic mucosa between the diverticula, sparing the diverticular orifice associated with an anatomopathological condition showing chronic inflammation. The involvement is preferably sigmoid and may involve a descending colon, sparing the rectum. The treatment is similar to that of inflammatory bowel diseases in mild forms, but recent studies have presented new alternatives with good results. The treatment is not yet well defined, and antibiotics, mesalamine, and corticoid therapy can be used, and surgery can even be performed for refractory cases.

Keywords: segmental colitis associated with diverticulosis, diverticular disease, inflammatory bowel diseases

1. Introduction

Colonic diverticular disease occurs in over 60% of people over 60 in the West. It can present various forms of manifestation, from asymptomatic conditions detected in endoscopic or imaging exams, to cases of acute diverticulitis, with a pericolic inflammatory process originating in a diverticulum and spreading to adjacent tissues due to micro-perforations [1, 2].

Segmental colitis associated with diverticulosis (SCAD) is a chronic inflammatory change that occurs in colonic mucosal segments between colonic diverticula, limited to the sigmoid and left colon, generally not affecting the diverticular ostium [2–6]. Rectum and right colon are spared from any endoscopic and histological inflammation [5, 6]. It is a recently recognized pathology within the spectrum of manifestations of colonic diverticular disease [7].

The inflammation of the colonic mucosa may resemble other inflammatory bowel diseases (IBD) [Crohn's disease (CD) and ulcerative colitis (UC)] in both clinical and endoscopic aspects [3, 7, 8]. Inflammation is non-specific, non-granulomatous, and localized. It appears to be a self-limited inflammatory process [3, 9].

2. Terminology

Several terms have been used to refer to this pathology, including crescentic fold disease, colitis associated with diverticular disease, diverticular colitis, diverticular-associated colitis (DAC), or segmental colitis associated with diverticulosis (SCAD), this being the most currently accepted [3, 9].

3. Pathogenesis

The exact pathogenic mechanism is not known, but it appears to be multifactorial pathogenesis [10]. Several hypotheses have been raised, including mucosal prolapse leading to mucosal ischemia, fecal stasis within diverticula, and consequent dysbiosis that can trigger an immune response, and high exposure to intraluminal antigens and toxins [3–5, 11].

4. Epidemiology

It is a rare pathology, with a prevalence ranging from 0.3 to 1.3% of patients undergoing colonoscopy [7] and 1.15 to 11.4% of patients with colonic diverticula.

The mean age at diagnosis is 61.7 to 66.5 years, similar to the mean age of onset of diverticular disease of the colon [3, 4]. There is also a predominance of males (58.7%) [4, 9].

5. Associated factors

SCAD is believed to be associated with several factors, including changes in intestinal motility, bacterial stasis in the colon, bacterial overgrowth, and inflammation [3].

6. Clinical condition

Although SCAD is still considered a pathology among the spectrum of diverticular diseases, it differs from diverticulitis (which is the inflammation of a diverticulum). As consequence, also the clinical picture is different.

The clinical picture associated with SCAD is variable, with studies citing complaints of diarrhea, rectal bleeding (live or with other changes), abdominal pain, and tenesmus [3, 4, 10]. Fever and weight loss are rare [4]. More than a third of patients have at least two associated symptoms at diagnosis [12].

The clinical picture can be very similar to that of mild inflammatory bowel disease. Occasionally, the patient may be asymptomatic, and the diagnosis is made only at random during colonoscopy for another reason.

Symptoms may vary according to the subtype of SCAD presented, with diarrhea being more common in type A and rectal bleeding in types C and D [12]. Type B presents with more than one symptom 39% of the time, with diarrhea and rectal bleeding being the main ones. Abdominal pain is more frequent in types C and D [2].

7. Laboratory and radiological exams

Laboratory tests (blood count, white blood count, erythrocyte sedimentation rate, C-reactive protein, fecal inflammatory markers, fecal calprotectin) and radiological

tests do not show specific results, but help to exclude differential diagnoses [4, 13]. Computed tomography may show thickening of the colonic wall in a colon segment with pre-existing diverticulosis. Fat blurring can be seen occasionally [14].

8. Diagnosis

The diagnosis of SCAD must initially be performed excluding other pathologies that cause intestinal inflammation, among these differential diagnoses: ischemic colitis, colitis induced by anti-inflammatory drugs, infectious colitis (mainly cytomegalovirus and Clostridium difficile), and IBD [3]. SCAD can mimic clinical and endoscopic presentations of IBD [10], and some pictures are indistinguishable from IBD [7].

Colonoscopy is the mainstay for the diagnosis of SCAD [15]. By its use, inflammation restricted to the mucosa between the diverticula is detected, sparing the diverticular orifice [3, 4]. Inflammation basically affects the sigmoid colon and left colon with normal mucosa in the rest of the colon (**Figure 1**).

It is indicated to perform biopsies of affected areas and normal areas to accurately locate inflammatory changes [15]. It is also suggested to perform a rectal biopsy (whose result should be negative) to strengthen the diagnosis [3, 7]. The pathologist must be informed about the clinical suspicion for better elucidation of the case.

Histopathologically, SCAD presents an active inflammatory infiltrate, similar to ulcerative colitis, unlike colonic diverticular disease, which presents a nonspecific inflammatory infiltrate, sometimes active, but different from those of inflammatory bowel diseases [5, 16, 17].

Therefore, it is concluded that the diagnosis must include colonoscopy and histopathological examination, confirming the inflammatory process in the affected area, and ruling out inflammation in other areas, in addition to laboratory and imaging tests excluding other pathologies.

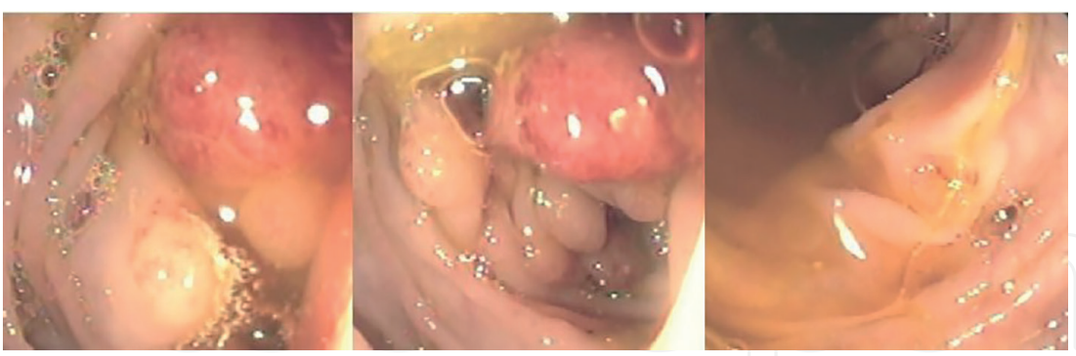

Figure 1.
Endoscopic appearance of a case of SCAD. A large, swollen, and red lesion on the top of the colonic fold can be seen: This lesion could be scored as type A SCAD.

9. Classification

SCAD can be classified endoscopically into four categories considering the absence of diverticulitis (**Table 1**) [3, 12, 14, 18, 19]:

The feature that unifies the four types of SCAD is the fact that it saves diverticular orifice. The main points used to classify SCAD in the different categories are how much it resembles or does not have mild-to-moderate UC, severe UC, or CD [12].

In the figures below, taken from Tursi, 2010, we observe endoscopic and histopathological images of SCAD type A (**Figure 2**), SCAD type B (**Figure 3**), SCAD type C (**Figure 4**), and SCAD type D (**Figure 5**) [2].

Type	Pattern	Endoscopic appearance	Histological appearance
A	**Crescentic fold**	Swollen red patches as of 0.5 to 1.5 cm in diameter.	Without architectural changes in the crypts. Neutrophil and lymphocyte infiltrate are limited to crypt epithelium.
B	**Mild to moderate UC-like**	Diffuse loss of vascular pattern, mucosal edema and hyperemia, and diffuse erosions.	Active inflammation with architectural changes in the crypt, crypt abscesses, and goblet cell depletion. Chronic changes of the lamina propria.
C	**Crohn's colitis-like**	Isolated aphthous ulcers.	Highest variability. Transmural inflammation with microfissures. Lymphoid follicles and non-specific infiltrates.
D	**Severe UC-like**	As type B but more severe with diffuse ulceration and reduced caliber of the lumen.	Crypt architectural changes, diffuse cryptitis, crypt abscesses, and goblet cell depletion. Chronic changes of the lamina propria.

Table 1.
SCAD classification (endoscopic and histological appearance).

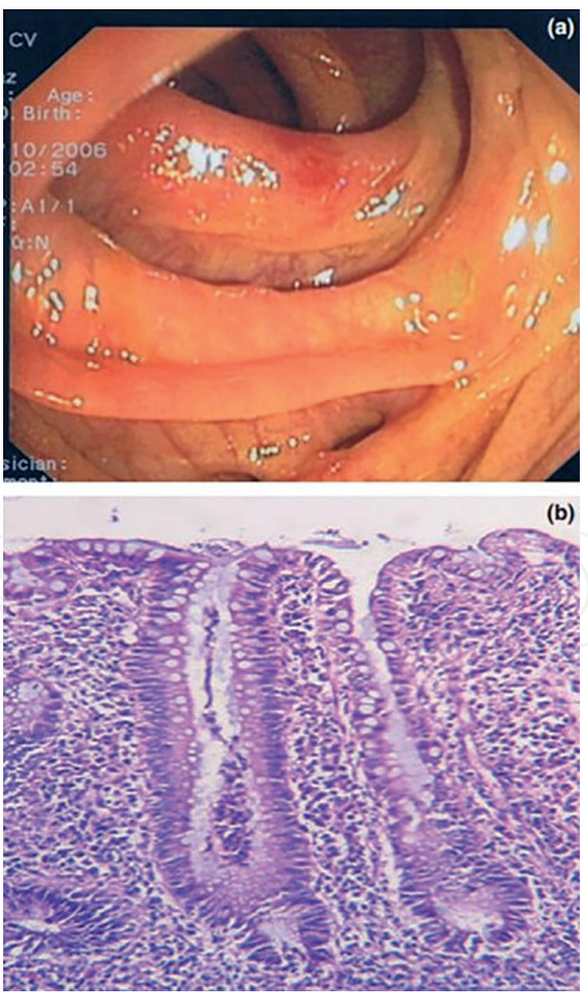

Figure 2.
Segmental colitis associated with diverticulosis' as "crescentic fold disease." The endoscopic pattern shows a swollen red patch about 1 cm in diameter, without hemorrhage or ulceration, confined to the crescentic mucosal fold. The diverticular orifices are spared, as well as the vascular pattern of the colonic mucosa is spared (a). The histopathological pattern shows an acute-on-chronic inflammation. Two crypt abscesses are evident in the center of the field (Hematoxylin & Eosin, magnification x20) (b).

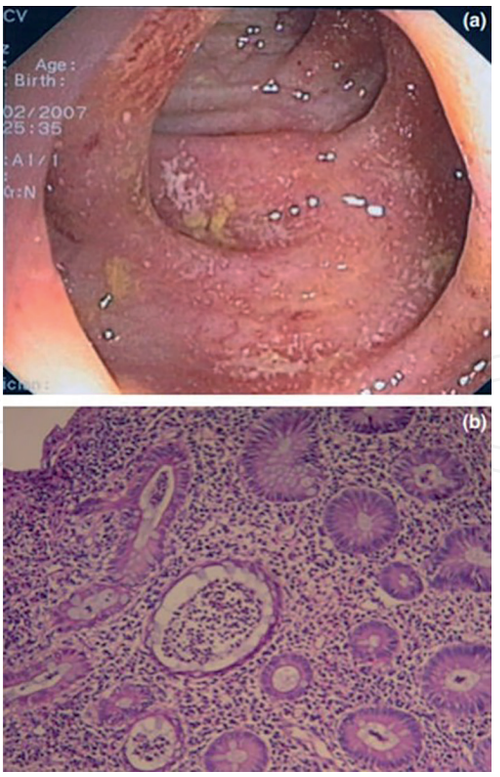

Figure 3.
Segmental colitis associated with diverticulosis as "Mild-to-moderate ulcerative colitis-like." the endoscopic pattern shows patchy loss of vascular pattern, edema, and hyperemia of the mucosa, and diffuse erosions. All diverticular orifices are spared (a). The histopathological pattern shows an acute-on-chronic inflammation. Two crypt abscesses are evident in the center of the field. There is also slight disarray and an architectural distortion with crypts irregularly arranged and varying in size and shape, patchy lymphoplasmacytic inflammation with some eosinophils (Hematoxylin & Eosin, magnification x100) (b).

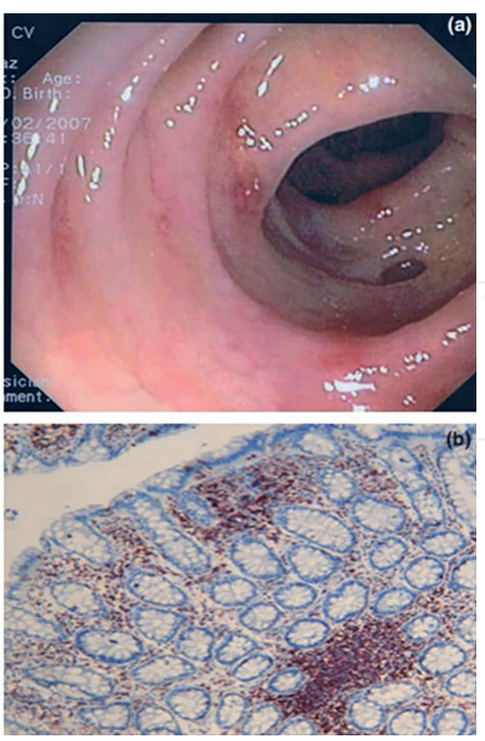

Figure 4.
Segmental colitis associated with diverticulosis as "Crohn's disease colitis-like." The endoscopic pattern shows scattered aphthous ulcers, some of them on the crescentic mucosal fold, within a normal colonic mucosa, with the normal vascular pattern. The diverticular orifices are always spared (a). The histopathological pattern shows a patchy lymphoplasmacytic inflammation in the upper part of the field, without significant cryptic architectural changes, and with a lymphoid follicle (count lymphocyte assay, magnification x20) (b).

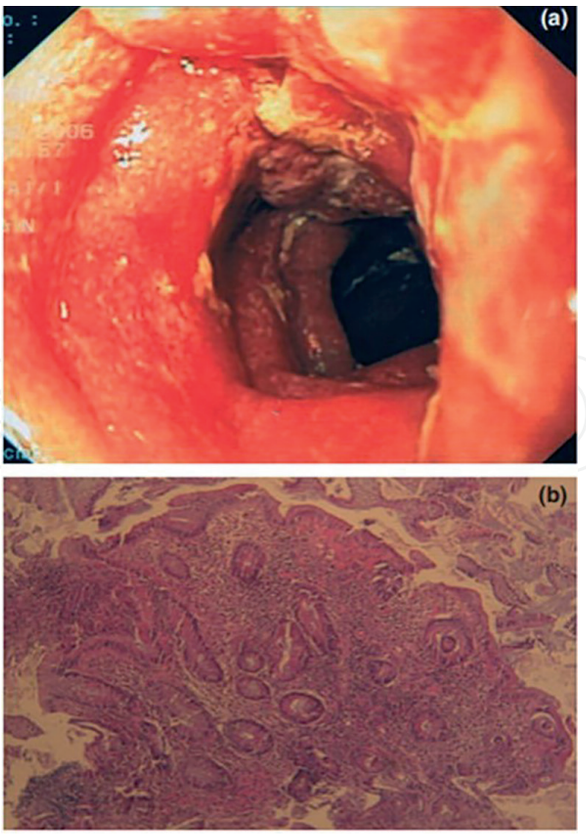

Figure 5.
Segmental colitis associated with diverticulosis as "Severe ulcerative colitis-like" SCAD pattern. The endoscopic pattern shows marked edema in the sigmoid mucosa with ulcerations, reduction of the lumen, diffuse loss of vascular pattern, diffuse hyperemia, and easy bleeding on contact with colonoscopy. The diverticular orifices are not easy to recognize, but they may be visible, spared by inflammation, at the maximal air inflation (a). Histology showed severe active ulcerative colitis (indistinguishable from classic inflammatory bowel disease), with severe diffuse inflammatory infiltrate, crypts irregularly arranged with architectural distortion, and cryptic abscesses (Hematoxylin & Eosin, magnification x40) (b).

10. Treatment

Since SCAD can resemble inflammatory diseases. It could be treated following the precepts of mild forms of IBD [3]. It is considered more benign than other IBD, with some patients evolving with full resolution of the condition without recurrences, even without any type of treatment being established [4, 10]. Types A and C SCAD seem to show a milder evolution, while types B and D have a high propensity to relapse, being highly indicated a more aggressive clinical and endoscopic follow-up and treatment for these two types [12, 20].

The standard treatment for SCAD is still not well defined, and it is possible to use a high fiber diet, use antibiotics (e.g., ciprofloxacin 500 mg twice a day associated with metronidazole 500 mg three times a day for 7 days) and aminosalicylates (mesalamine 2.4 to 3.2 g per day) in cases of unsatisfactory response to antibiotics or recurrent symptoms, which can be continued for 7 to 10 days or have its dose increased in case of therapeutic failure [4, 10, 14]. Immunosuppressants and steroids are used in severe third-line cases [4, 10].

In case of recurrence after having responded to the use of antibiotic therapy, we suggest repeating the same regimen for a long time.

Recent studies have demonstrated the possibility of combining beclomethasone dipropionate (BDP) (for 4 weeks) and the probiotic VSL#3 (for 15 days in a row) for the treatment of mild-to-moderate acute conditions, with the vast majority of patients reaching remission in week 4 [4, 10].

In patients' refractory to conventional therapy for SCAD, infliximab and adalimumab could be good therapeutic options [5, 21–23].

Table 2 shows the medicament advice, posology, and suggested time of use.

Surgery reserved for cases refractory to clinical treatment or that become dependent on corticosteroid therapy [4, 10].

It is always important to rethink whether the SCAD diagnosis is correct in case of failure of the instituted therapies, always bearing in mind the differential diagnoses, especially IBD [22].

The treatment algorithm for SCAD adapted from Kucej and Poggio 2018 is shown in **Figure 6** and divides the therapeutic indications according to the SCAD classification [3].

Medicament	Posology	Usage time
Ciprofloxacin	500 mg twice a day	7–14 days
metronidazole	500 mg 3 times a day	7–14 days
Mesalamine	2.4 a 3.2 g per day	Variable with the answer
Prednisone	40 mg + wean	7 days + weaning until week 6
Beclomethasone dipropionate	10 mg/day (1 month) + 5 mg/day (next month)	8 weeks
VSL#3	1 bag/day	15 days

Table 2.
Medicament, posology, and usage time for SCAD.

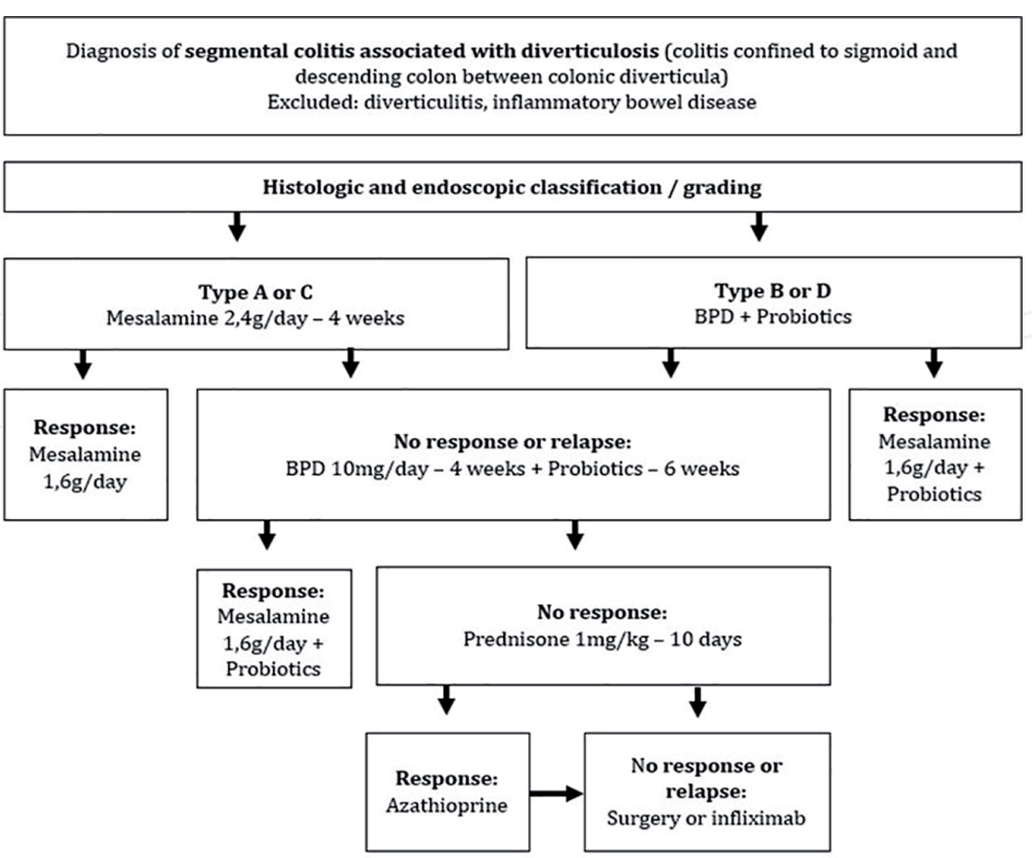

Figure 6.
Proposed algorithm for SCAD management (adapted by Tursi et al. [2]).

Conflict of interest

The authors declare no conflict of interest.

Abbreviations

SCAD	Segmental colitis associated with diverticulosis
IBD	Inflammatory bowel diseases
CD	Crohn's disease
UC	Ulcerative colitis

Author details

Rafael Luís Luporini[1,2*], Marcel Domeniconi[2], Ana Carolina Parra[2], André Rizzo[2], Daniela Freitas[2], Sthefânia Frizol[2] and Antonio Tursi[3]

1 Department of Medicine, Federal University of São Carlos (UFSCar), São Paulo, Brazil

2 General Surgery of Santa Casa de São Carlos, São Paulo, Brazil

3 Territorial Gastroenterology Service, Azienda Sanitaria Locale BAT, Andria, Italy

*Address all correspondence to: rafaelluporini@ufscar.br

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