

Solitary rectal ulcer syndrome: endoscopic spectrum and review of the literature

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First described by Cruveilhier¹ in 1830, when he reported 4 unusual cases of rectal ulcers, the term “solitary ulcers of the rectum” was coined by Lloyd-Davis in the late 1930s.² However, it was only in 1969 that the condition became widely recognized after a review of 68 cases by Madigan and Morson.³ A few years later, Rutter and Riddell² introduced a more comprehensive pathogenic concept of the disease and linked it to the effects of rectal mucosal prolapse. Solitary rectal ulcer syndrome (SRUS) is a chronic, benign disorder of young adults, affecting the rectum, often related to straining or abnormal defecation. SRUS is an infrequent or an underdiagnosed disorder, with an estimated prevalence of one in 100,000 persons per year.⁴ The term SRUS probably is a misnomer, because ulcers are only found in 40% of patients, while 20% of patients have a solitary ulcer, and the rest of the lesions vary in shape and size, from hyperemic mucosa to broad-based polypoid lesions.⁵ There is even a suggestion that the disease process also may involve the sigmoid colon.⁶

PATHOGENESIS

The pathogenesis of SRUS is not well established, but it is believed that multiple factors contribute to its development. The most accepted theories are related to direct trauma or local ischemia as causes. Parks et al,⁷ in 1966, proposed the “flap valve” theory whereby a flap is created upon contraction of the puborectalis along with increased intraabdominal pressure (as during straining), and the anterior rectal wall is pressed on the upper anal canal and lead to occlusion of the outlet.⁸ Furthermore, prolonged straining at stool and difficulty initiating defecation, along with digital evacuation in persons with constipation, may lead to direct trauma to the mucosa and the formation of an ulcer.⁹ However, many patients deny rectal digitation, and the ulcer often occurs in the mid rectum, which generally is not reached by digitation. Moreover, no

association between self-digitation and SRUS was found in a study of homosexuals with bowel complaints (only 6 patients were found to have rectal ulcers and, of those, only one had histologic criteria that fit SRUS).⁹ Mucosal prolapse, overt or occult, is the most common underlying pathogenetic mechanism in SRUS. Also, in such patients, the tone of external sphincter is elevated, generating a high intrarectal pressure.⁵ This, along with rectal prolapse during defecation, causes venous congestion, which leads to mucosal ischemia. The cause of ischemia may be related to traction on the submucosal vessels; fibroblasts replacing blood vessels; pressure by the anal sphincter, causing necrosis of prolapsed mucosa; and venous congestion and ulceration secondary to high intrarectal pressure generated by rectal prolapse.⁸ Moreover, rectal mucosal blood flow has been found to be reduced in SRUS to a level similar to that seen in normal transit constipation, suggesting similar impaired autonomic cholinergic gut-nerve activity.

CLINICAL FEATURES

SRUS is a disorder of young adults, occurring most commonly in the third decade in men and in the fourth decade in women. It, however, has been described in children and in the geriatric population. Men and women are affected equally, with a small predominance for women.^{5,10,11} Patients usually present with passage of mucus and blood per rectum on defecation. The amount of blood varies from slight fresh blood to severe hemorrhage that requires blood transfusion.¹²⁻¹⁴ Other complaints are tenesmus, straining, altered bowel habits, incontinence, and a sensation of incomplete evacuation.¹⁵ Up to 26% of patients can be asymptomatic,⁵ discovered incidentally while investigating other diseases. The average time from the onset of symptoms to diagnosis, in one study, ranged from 3 months to 30 years (mean, 5 years)¹⁵ with similar results in different series.^{16,17}

DIAGNOSIS

The diagnosis of SRUS is based on symptomatology in combination with the endoscopic and histologic findings.

TABLE 1. Differential diagnosis of the solitary rectal ulcer syndrome

Inflammatory bowel disease
Infectious (amebiasis, lymphogranuloma venereum, secondary syphilis)
Malignancy
Chronic vascular insufficiency (chronic ischemic colitis)
Endometriosis
Colitis cystica profunda
Drug-induced (e.g., ergotamine tartrate-containing suppositories)
Stercoral (pressure) ulcer
Trauma
Idiopathic

A thorough history taking is of utmost importance in the initial diagnosis of SRUS. It is essential to differentiate SRUS, which is a benign disease, from other chronic, devastating, and potentially lethal disorders (Table 1). The endoscopic spectrum of SRUS may vary from simple hyperemic mucosa to small or giant ulcers to broad-based polypoid lesions in different sizes and number (Figs. 1 and 2). Macroscopically, SRU typically appear as shallow ulcerating lesions on a hyperemic surrounding mucosa, most often located on the anterior wall of the rectum at 5 to 10 cm from the anal verge. Ulcers may range from 0.5 to 4 cm in diameter but usually are 1 to 1.5 cm in diameter.^{5,15} Twenty-five percent of SRUS may appear as a polypoid lesion, 18% may appear as patchy mucosal erythema, and 30% as multiple lesions.⁴ Because of the wide endoscopic spectrum of SRUS and the fact that the condition may go unrecognized or, more commonly, misdiagnosed, it is crucial to take biopsy specimens from the involved area to make a positive confirmation of the diagnosis and to exclude other diagnoses, including cancer. In a study of 98 patients with a final clinicopathologic diagnosis of SRUS, an incorrect diagnosis was made in more than 25% of patients with a median duration of incorrect diagnosis of 5 years, primarily the result of inadequate tissue specimens and failure to recognize the diagnostic histopathologic features of SRUS (see below).¹⁵

Defecography is very useful at identifying the presence of internal or external mucosal prolapse or intussusception in SRUS (Fig. 3). Defecography also can demonstrate a hidden prolapse, as well as a nonrelaxing puborectalis muscle and incomplete or delayed rectal emptying.¹⁷⁻¹⁹ In a study by Goei et al²⁰ involving 53 patients with histologically proven SRUS, evacuation proctography revealed delayed or incomplete emptying and/or both internal and external rectal prolapse in 75% of patients.²⁰ However, because of the wide availability of endoscopy and

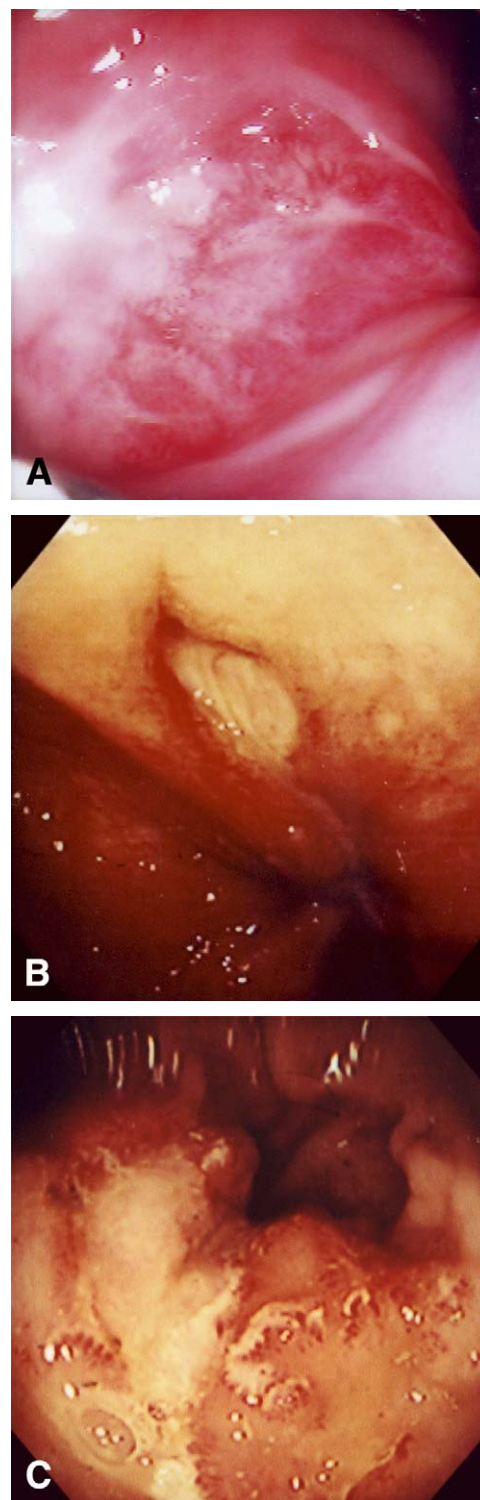


Figure 1. The ulcerated variant of the SRUS. **A**, Flat shallow ulcer with abundant mucus and hyperemic base. **B**, Punched-out isolated ulcer in the base of the rectum. **C**, Large shallow ulcer with multiple adjacent areas of erythema, edema, and superficial ulcerations.

biopsy, defecography usually is reserved for the investigation of the underlying pathophysiology and possibly for preoperative assessment.²¹ Other testing modalities of

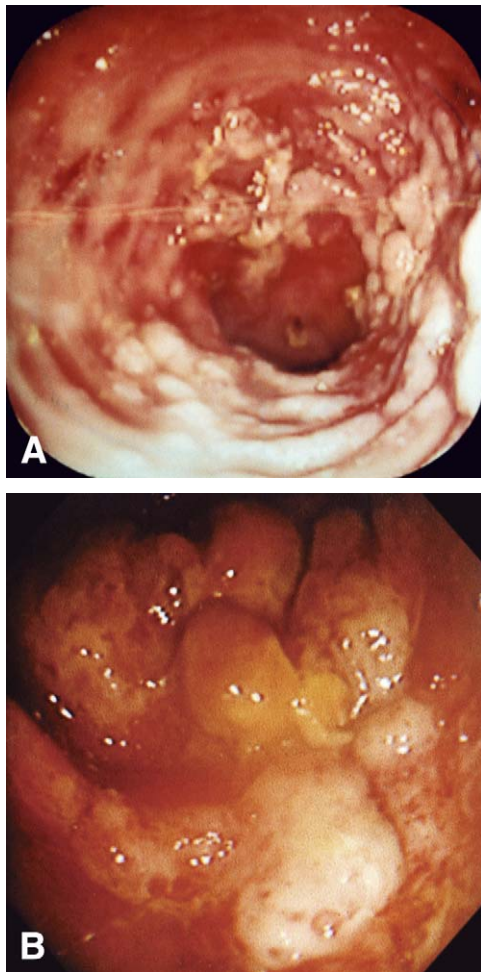


Figure 2. A and B, The polypoid variant of SRUS.

anorectal function have neither been useful in diagnosing SRUS nor in predicting response to therapy. Barium enema may show rectal stricture, granularity of the mucosa, and thickened rectal folds, all of which are nonspecific.^{22,23} Transrectal US is useful to distinguish SRUS from other conditions, e.g., invasive cancer.²⁴⁻²⁶ An inhomogeneous submucosa, a thickened muscularis propria, and a marked thickening of the internal anal sphincter have been described on EUS, although the exact mechanisms underlying these changes are unknown. Moreover, it has been suggested that sonographic evidence of a thick internal anal sphincter is highly predictive of high-grade rectal prolapse and intussusception in patients with SRUS.²⁵ Lastly, anorectal manometry does not appear to provide additional or clinically useful information in patients with SRUS except in identifying sphincteric defects.^{5,27}

HISTOLOGIC FEATURES

The characteristic histologic features include fibrous obliteration of the lamina propria; disorientation and thickening of the muscularis mucosa; and regenerative

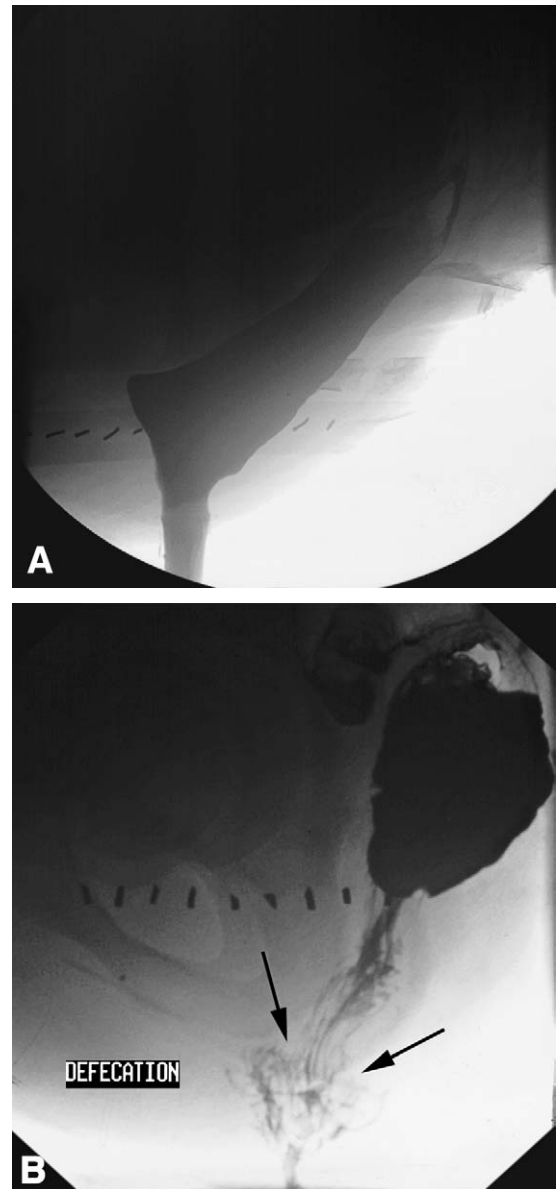


Figure 3. Lateral view of normal (A) and abnormal (B) evacuation proctography, showing circumferential folding of the rectal mucosa seen at 3.5 cm proximal to the anal margin (arrows), compatible with an annular type of rectal intussusception.

changes, with distortion of the crypt architecture (Figs. 4 and 5).²⁸ The lamina propria is replaced with smooth muscle and collagen, which leads to hypertrophy and disorganization of the muscularis mucosa, referred to as fibromuscular obliteration. Similar changes are seen in patients with colitis cystica profunda disease, a disorder characterized by the presence of mucus-filled cysts, usually affecting the rectum. The histologic findings of the polypoid variant of SRUS are similar to the ulcerative variant except for regenerative hyperplastic changes, such as a relatively high incidence of mucous-cell proliferation, dilatation of glands, and serrate change.²⁹ The presence of collagen infiltration of the lamina propria distinguishes

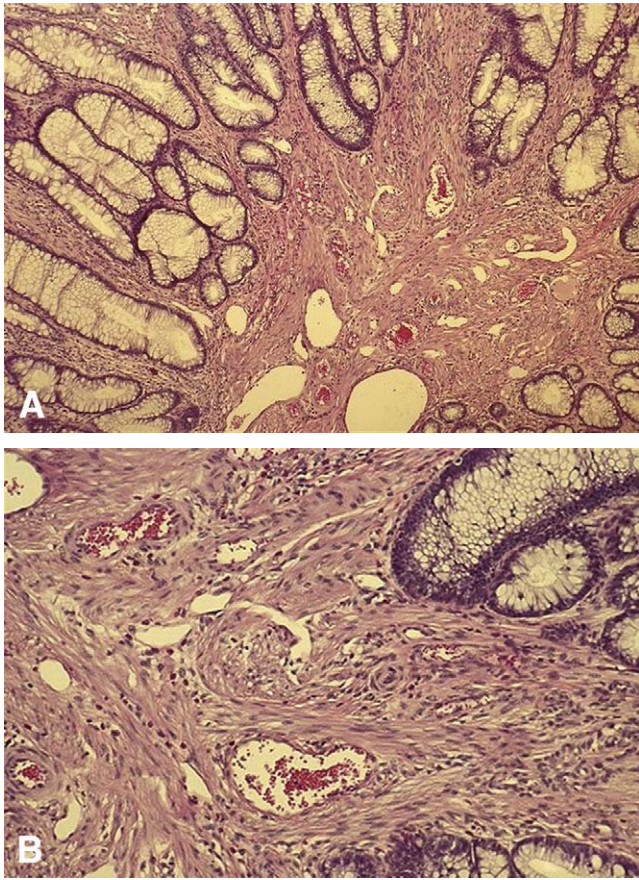


Figure 4. **A**, Low-power magnification of a biopsy specimen from a SRUS, showing smooth muscle hyperplasia within lamina propria between benign colonic glands (H&E, orig. mag. $\times 50$). **B**, High-power magnification of a biopsy specimen from a SRUS, featuring prominent smooth muscle hyperplasia within the lamina propria (H&E, orig. mag. $\times 125$).

SRUS from inflammatory bowel disease and chronic ischemic colitis. Biopsies are mandatory to exclude a malignancy that presents as SRUS. However, SRUS may be present in association with an underlying carcinoma.³⁰⁻³²

TREATMENT

Treatment of SRUS mostly depends on the severity of symptoms and whether there is an underlying rectal prolapse or not. Asymptomatic patients may not require any treatment other than behavioral modifications. Because of the limited number of controlled trials, there are no definitive treatment recommendations for SRUS. It is important to note that the primary end point remains the resolution or the improvement of symptoms and that patient education and a conservative, stepwise, individualized approach are most likely to succeed. Several treatment options have been used in the management of SRUS, ranging from behavioral modification to topical treatment, biofeedback, and surgery (Table 2).

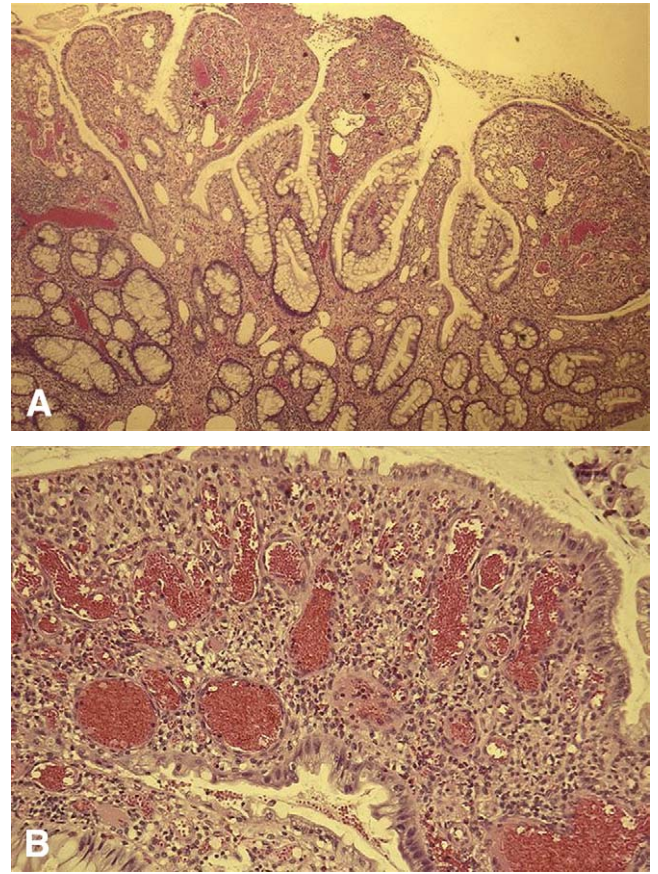


Figure 5. **A**, Low-power magnification of polypoid form of SRUS, featuring broad papillary formations with erosion of their tips; note branching colonic glands (H&E, orig. mag. $\times 30$). **B**, High-power magnification of SRUS, featuring granulation tissue formation within the lamina propria with inflammation (H&E, orig. mag. $\times 125$).

Patient education and behavior modification remain the cornerstone of treatment of SRUS. Once the diagnosis is established, patients should be instructed on a high-fiber diet and the use of bulk laxatives, and also to avoid straining and anal digitation. Time spent on the commode should be minimized and defecation training emphasized. These dietary and behavioral modifications are especially effective in patients with mild to moderate symptoms and in the absence of significant mucosal prolapse. In one study, behavior modification and dietary supplementation with fiber resulted in symptom improvement and complete ulcer healing in 15 of 21 patients with SRUS.³³

SRUS, however, may be resistant to such conservative treatment, and a significant proportion of patients fail to achieve remission. Several topical agents have been used with variable success rates, but none has been tested in prospective controlled trials. Topical medications, such as glucocorticoids and sulfasalazine enemas, are not effective,³⁴ although 5-aminosalicylate enemas have been anecdotally reported to be of some use.³⁵ Sucralfate

TABLE 2. Summary of published studies on the treatment of the solitary rectal ulcer syndrome

Treatment modality	Design	No. patients	Study details	Comments
Behavioral modification				
van den Brandt-Gradel et al ³³	Prospective	21	Mean F/U of 10.5 months	Improvement in symptoms; disappearance of ulcers in 15/21 patients
Topical therapy				
Kumar et al ³⁴ : sulfasalazine	Case report	1	N/A	
Zargar et al ³⁷ : sucralfate	Open label	6	2 g twice daily for 6 wk; mean F/U of 8 months	Macroscopic healing and improvement in all patients
Ederle et al ³⁸ : fibrin glue	Case series	12	Fiber + behavioral modification in 6 patients; topical human fibrin sealant in 6 patients	Complete healing after 14 d vs. no healing in the control group
Biofeedback				
Vaizey et al ⁴⁰	Prospective	13	Median F/U of 9 mo; symptomatic improvement in 50%-60% of patients	Ulcers did not heal completely in any patient
Jarrett et al ⁴²	Prospective	16 patients; 26 controls	Subjective improvement: 12/16 patients; ulcer resolution in 5/16 patients	Laser Doppler mucosal flowmetry was reduced in patients with SRUS and improved significantly with biofeedback
Binnie et al ⁴³	Retrospective	31	14 patients treated conservatively or with surgery 17 patients underwent biofeedback	Symptomatic cure similar between groups; 4 vs. 15 recurrences in favor of biofeedback
Malouf et al ⁴⁴	Case series	13	Median F/U of 36 mo; improvement wanes over time	Retraining may help regain effect in ~50% of initial responders
Surgery				
Marchal et al ⁴⁵	Retrospective	13	1 patient with simple ulcer resection; 1 patient with stoma as primary operation; 3 patients with rectopexy; 8 patients with modified Delorme's procedure	Mean F/U 57 mo; modified Delorme's procedure and rectopexy helpful in 7/11 patients
Nicholls et al ⁴⁶	Retrospective	14	Anteroposterior rectopexy	Symptomatic improvement in 12/14 patients
Sitzler et al ⁴⁷	Retrospective	66	2 patients with anterior resection; 4 patients with stoma as primary operation; 49 patients with rectopexy; 9 patients with Delorme's procedure	Median F/U=90 mo, 22/49 rectopexies and 4/9 Delorme's procedures failed Anterior resection disappointing as salvage; overall stoma rate, 30%

F/U, Follow-up; N/A, not available; SRUS, solitary rectal ulcer syndrome.

retention enemas have been reported to lead to clinical improvement in small, uncontrolled case series.^{36,37} Fibrin glue, in one report, resulted in complete healing after 14 days when applied topically, with no recurrence

after a 1-year follow-up.³⁸ The theory is that fibrin glue stimulates the proliferation of fibroblasts and vessel growth and leads to more rapid healing of the ulcer.

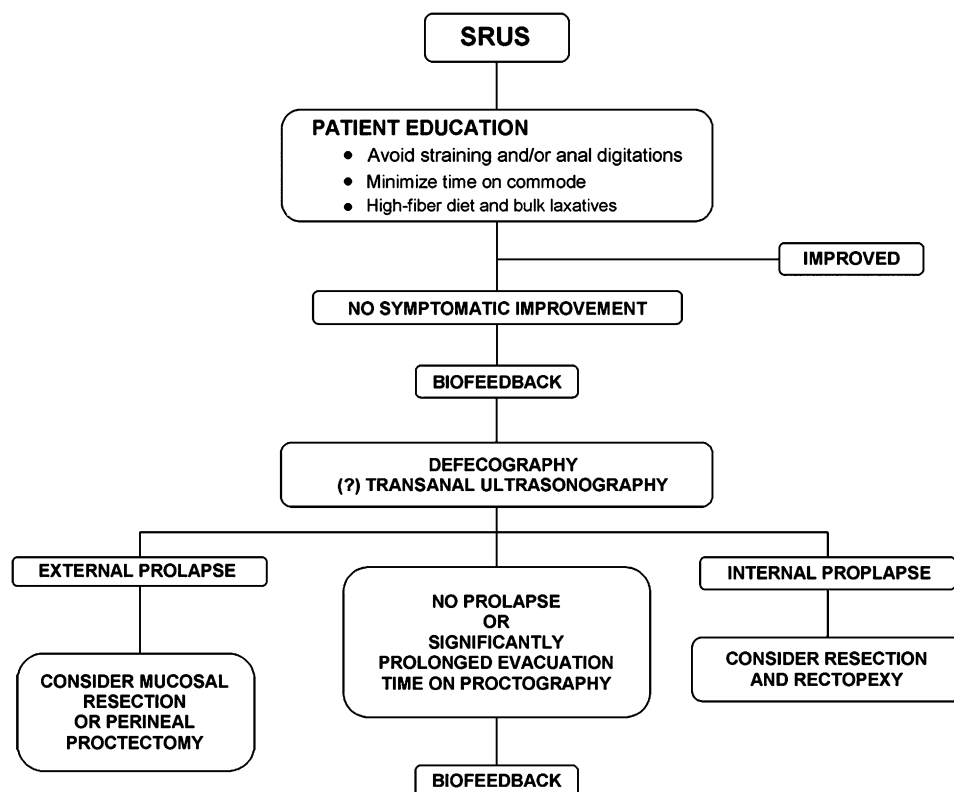


Figure 6. Proposed algorithm for the management of SRUS.

It has been suggested that, in selected patients, biofeedback improves symptoms by altering efferent autonomic pathways to the gut.³⁹ Biofeedback includes reducing excessive straining with defecation by correcting abnormal pelvic-floor behavior and by attempting to stop the aid of laxatives, suppositories, and enemas.⁴⁰ In one prospective study, biofeedback resulted in clinical improvement in 13 patients with SRUS, with a decrease in straining and a need for either manual rectal evacuation or the use of laxatives.⁴⁰ In one report, biofeedback was particularly helpful in patients with a nonrelaxing puborectalis muscle; straining, bleeding, and mucus decreased, and defecation improved.⁴¹ In one recent report by Jarrett et al,⁴² biofeedback was an effective behavioral treatment for the majority of patients with SRUS, and a favorable outcome was associated with increased rectal mucosal blood flow, suggesting that improved extrinsic innervation to the gut could be responsible for treatment response. Although biofeedback improves symptoms in the majority of patients with SRUS, this improvement may deteriorate with time in some patients.⁴³ However, more than half the patients with an early clinical response would be expected to have ongoing clinical benefit from biofeedback at a median of 3 years.⁴⁴

Surgical treatment is reserved for patients who are refractory to conservative treatment and biofeedback or in those with full-thickness or significant mucosal rectal

prolapse. Antiprolapse surgeries include local excision of the ulcer, rectopexy, perineal proctectomy, or diversion.^{45,46} Halligan et al²¹ demonstrated complete resolution of rectal prolapse after rectopexy in 18 of 19 patients with defecography-proven SRUS and prolapse. In another retrospective study, by Sitzler et al,⁴⁷ on the long-term outcome of antiprolapse surgery on patients with SRUS refractory to medical therapy, the investigators found significant improvement or complete resolution of symptoms in about 55% to 60% of cases. In a retrospective study of 21 patients by Tjandra et al,⁵ a third of patients with SRUS benefited from surgery, with complete healing of the ulcer occurring in 28% of patients after rectopexy and in 33% of patients after undergoing resection and rectopexy. Based on postoperative evacuation defecography studies, it has been shown that rectopexy alters rectal configuration and successfully treats rectal prolapse in SRUS and that a prolonged preoperative evacuation time is predictive of poor symptomatic outcome.²¹ For full-thickness prolapse, a mucosal resection (Delorme's procedure) or a perineal proctectomy (Altemeier's procedure) have been advocated.⁴⁸ When the above measures fail, consideration is given to mucosal-sleeve resection with coloanal pull-through or a diverting colostomy. Based on existing literature and experience, a therapeutic algorithm is presented (Fig. 6).

In conclusion, SRUS is a chronic, benign disorder of young adults, affecting the rectum, often related to

straining or abnormal defecation. Ulcers are only found in 40% of patients, while 20% of patients have a solitary ulcer and the rest of the lesions vary in shape and size, from hyperemic mucosa to broad-based polypoid lesions. The pathogenesis of SRUS is not well understood but probably is multifactorial. Usually patients present with straining, altered bowel habits, anorectal pain, incomplete passage of stools, and passage of mucus and blood. The diagnosis is made clinically, endoscopically, and histologically. Symptoms may resolve spontaneously or may require treatment. A variety of therapies has been tried. Several therapies thought to be beneficial include topical medications, behavior modification supplemented by fiber and biofeedback, and surgery. Patient education and a conservative, stepwise individualized approach remain paramount in the management of this syndrome.

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