

## Solitary rectal ulcer syndrome in children: A literature review

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tant problem as reflected by persistence of symptoms, especially rectal bleeding. In this review, we discuss current diagnosis and treatment for SRUS.

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### Abstract

Solitary rectal ulcer syndrome (SRUS) is a benign and chronic disorder well known in young adults and less in children. It is often related to prolonged excessive straining or abnormal defecation and clinically presents as rectal bleeding, copious mucus discharge, feeling of incomplete defecation, and rarely rectal prolapse. SRUS is diagnosed based on clinical symptoms and endoscopic and histological findings. The current treatments are suboptimal, and despite correct diagnosis, outcomes can be unsatisfactory. Some treatment protocols for SRUS include conservative management such as family reassurance, regulation of toilet habits, avoidance of straining, encouragement of a high-fiber diet, topical treatments with salicylate, sulfasalazine, steroids and sucralfate, and surgery. In children, SRUS is relatively uncommon but troublesome and easily misdiagnosed with other common diseases, however, it is being reported more than in the past. This condition in children is benign; however, morbidity is an impor-

### INVITED COMMENTARY ON HOT ARTICLES

Solitary rectal ulcer syndrome (SRUS) is an uncommon chronic and benign rectal disorder often related to abnormal defecation or straining. It was first described by Cruveilhier<sup>[1]</sup> in 1829, when he reported four unusual cases of rectal ulcers. The term "solitary ulcers of the rectum" was used by Lloyd-Davis in the late 1930s and in 1969 the disease became widely recognized after a review of 68 cases by Madigan *et al*<sup>[2]</sup> and few years later, a more detail pathogenic concept of the disease was reported by Rutter *et al*<sup>[3]</sup>. SRUS is an infrequent or unrecognized or misdiagnosed disorder, with an estimated prevalence of 1 in 100 000 persons per year<sup>[4]</sup>. Solitary rectal ulcer is a misnomer as ulcers are found in 40% of patients, while 20% of patients have a solitary ulcer, and the rest of the lesions are different in shape and size, including hyperemic mucosa to broad-based polypoid lesions<sup>[5]</sup>. The disease process also may involve the sig-

moid colon<sup>[6]</sup>.

Although it is uncommon, it is well recognized in adult populations<sup>[3]</sup>. SRUS seems to be rare in childhood<sup>[7-10]</sup> and may masquerade as other more common conditions, causing difficult-to-manage lower gastrointestinal symptoms. Opinion differs regarding the best treatment for this troublesome condition, varying from conservative management and enema preparations to more invasive surgical procedures such as rectopexy<sup>[11]</sup>.

In this review article, several aspects of this syndrome will be evaluated with an especial focus on the condition in children. Detailed risk factors, causes, and treatment methods will help guide future treatment and prevention strategies in children.

### **Pathophysiology and clinical presentation**

The pathophysiology of SRUS is incompletely understood; however, rectal hypersensitivity leading to the persistent desire to defecate and sensation of incomplete evacuation may have a role in SRUS<sup>[12]</sup>. Inappropriate contraction of the puborectalis muscle and rectal mucosal prolapse have been commonly implicated, although trauma and ischemia have been suspected in some children<sup>[7,13]</sup>. In children, secondary to chronic mechanical and ischemic trauma, inflammation by hard stools, and intussusception of the rectal mucosa, some histological features of SRUS can be seen such as fibromuscular obliteration of the lamina propria and disorientation of muscle fibers<sup>[14]</sup>.

In our previous study of 256 children who were evaluated endoscopically for recurrent lower gastrointestinal bleeding, 4.7% had this syndrome<sup>[15]</sup>. In adult patients, men and women are affected equally, with a small predominance for women<sup>[16]</sup>, but 75%-80% of children with SRUS are boys<sup>[15,17]</sup>. Suresh *et al*<sup>[18]</sup> have evaluated 325 children aged < 18 years during 8 years for various indications such as bleeding, polyps and anal fissure. Twenty-two (6.8%) children were diagnosed with SRUS and ranged in age from 18 months to 18 years (median: 10 years), and 18 (81.8%) of these were  $\geq$  8 years of age. The male to female ratio in this group was 1.4:1. To date, the youngest patient with SRUS was a child of 1.5 years<sup>[18]</sup>; Gabra *et al*<sup>[19]</sup> also have reported two boys with SRUS who were 2 and 3 years old.

The average time from the onset of symptoms to diagnosis is 5 years, ranging from 3 mo to 30 years in adults, which is longer than in pediatric patients (3.2 years, range: 1.2-5.5 years)<sup>[15,17,20-22]</sup>. This syndrome results from obstructed defecation secondary to internal rectal prolapse with a collection of symptoms including rectal bleeding, passage of mucus and straining on defecation, perineal and abdominal pain, tenesmus, feelings of incomplete defecation, constipation, and rectal prolapse<sup>[23-25]</sup>. The amount of blood varies from a little fresh blood to severe hemorrhage that requires blood transfusion<sup>[26-28]</sup>. Up to 26% of patients can be asymptomatic and may not show the bleeding that is discovered incidentally while investigating other diseases<sup>[5]</sup>. The use of

digital manipulation to assist with a bowel movement is variably reported in patients with SRUS<sup>[15,29]</sup>. Bright-red blood from the rectum or mucoid rectal discharge, tenesmus, proctalgia, and constipation are the major symptoms. Some children present with apparent diarrhea (because of prolonged visits to the bathroom) and the associated bleeding, abdominal pain, and tenesmus may suggest to clinicians the presence of inflammatory bowel disease<sup>[25]</sup>.

### **Diagnosis**

SRUS is a relatively uncommon but bothersome and easily misdiagnosed condition of childhood. Clinical suspicion and paraclinical evaluations are needed and diagnosis is via symptomatology in combination with endoscopic and histological findings<sup>[17]</sup>. A complete and thorough history is most important in the initial diagnosis of SRUS. It is essential to differentiate SRUS from other devastating, chronic, and potentially lethal disorders such as inflammatory bowel disease, amebiasis, lymphogranuloma venereum, chronic ischemic colitis, endometriosis, colitis cystica profunda, and malignancy. Obstructive symptoms (anismus) in children may be interpreted by parents as constipation. Concomitant haematochezia may be misinterpreted as originating from an anal fissure caused by constipation, or as other causes of rectal bleeding such as a juvenile polyp<sup>[30-32]</sup>.

Defecography is a useful method for determining the presence of intussusception or internal or external mucosal prolapse and can demonstrate a hidden prolapse, as well as a non-relaxing puborectalis muscle and incomplete or delayed rectal emptying<sup>[33]</sup>. Barium enema shows granularity of the mucosa, polypoid lesion, rectal stricture and ulceration, and thickened rectal folds; all of which are nonspecific findings<sup>[33,34]</sup>. Temiz *et al*<sup>[35]</sup> have recommended that defecography and anorectal manometry should be performed in all children with SRUS to define the primary pathophysiological abnormality and to select the most appropriate treatment protocol.

The endoscopic spectrum of SRUS varies from simple hyperemic mucosa to small or giant ulcers to broad-based polypoid lesions of different sizes. Macroscopically, SRUS typically appears as shallow ulcerating lesions on a hyperemic surrounding mucosa, most often located on the anterior wall of the rectum at 5-10 cm from the anal verge. Ulcers may range from 0.5 to 4 cm in diameter but usually are 1-1.5 cm in diameter<sup>[5,15,30]</sup>.

Histological examination of biopsy material is necessary to confirm a diagnosis of SRUS. The histological criteria for diagnosis are as follows: fibrous obliteration of the lamina propria, streaming of fibroblasts and muscle fibers between crypts, thickening of muscularis mucosa, branching and distorted glandular crypts and diffuse collagen infiltration of the lamina propria<sup>[15,36-38]</sup>.

Recent studies have shown the usefulness of anorectal ultrasound in assessing internal anal sphincter thickness, which is shown to be increased in patients with this syndrome<sup>[37,39]</sup>, and 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<sup>[39]</sup>.

There is a need for a high index of suspicion for the possibility of SRUS in young children with clinical picture of nonspecific proctitis.

### Treatment

The most frustrating aspect of SRUS is the difficulty in treatment; experiences have shown that most therapeutic regimens are inadequate. There are few data on treatment and its outcome in children with SRUS. In most reported pediatric case series, active intervention using enemas<sup>[15]</sup>, laxatives<sup>[40]</sup>, and surgical approaches have been used more frequently than behavioral modification, mainly biofeedback therapy in adults<sup>[11,41-45]</sup>.

Some suggestions for the treatment of SRUS include reassurance of the patient and parents that the lesion is benign, encouragement of a high-fiber diet<sup>[46]</sup>, avoidance of straining, regulation of toilet habits, and attempt to discuss any psychosocial factors<sup>[20,36,37,47,48]</sup>. The use of a high-fiber diet, in combination with stool softeners and bulking laxatives, and avoidance of straining have had varying responses<sup>[4,46]</sup>.

In children, primary medical treatment is proposed for most cases<sup>[49]</sup>. Topical application of sucralfate can be effective for treatment of SRUS in some patients<sup>[15,50]</sup>. Many medications that are useful in the treatment of patients with inflammatory bowel disease have been tried in those with SRUS, such as sulfasalazine and corticosteroids, with varying responses<sup>[16,51]</sup>. In one study, oral salicylate and other topical agents such as mesalamine and steroids were not effective<sup>[52]</sup>. Endoscopic application of human fibrin sealant<sup>[53]</sup>, laser therapy<sup>[54]</sup>, and biofeedback<sup>[45,47,48]</sup> are some of the effective treatment methods for SRUS.

A therapeutic role for botulinum toxin injection into the external anal sphincter for the treatment of SRUS and constipation associated with dyssynergia of defecation dynamics has been reported by Keshtgar *et al*<sup>[55]</sup>. The effect of botulinum toxin lasts approximately 3 mo, which may be more beneficial than biofeedback therapy<sup>[55]</sup>.

Surgical methods for treatment of SRUS are rectopexy<sup>[42,43,56]</sup>, excision<sup>[4,5,16]</sup> and Delorme's procedure<sup>[41,44,57,58]</sup>.

The choice of treatment protocol depends on acuteness of symptoms and whether there is an underlying rectal prolapse or not<sup>[25,31]</sup>. Maintaining compliance in children may prevent progression to the type of long-term morbidity and treatment resistance sometimes seen in adults with this condition<sup>[25]</sup>. Recommended treatment in children by Abbas *et al*<sup>[59]</sup> is initially conservative, but, if that fails, transrectal resection followed by a high-fiber diet. Conservative management including behavioral modification and reduction of time spent straining at defecation has been reported as a good method<sup>[25,46,60]</sup>.

Compliance with simple behavioral modification appears to produce a good outcome in childhood SRUS, probably because of the short disease duration compared with adults.

Early recognition and management of these patients may avoid some of the chronic long-term morbidity often associated with this condition; however, late relapse because of noncompliance is a substantial risk and children should be followed up long term.

SRUS is thought to be part of the bigger disease process known as mucosal prolapse syndrome, which incorporates inflammatory cloacogenic polyps, inflammatory cap polyps, and gastric antral vascular ectasia. In fact, all these syndromes have the same histological features<sup>[61]</sup>. As a result 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 malignancy<sup>[31,62]</sup>.

The exact etiology is unknown<sup>[30,63,64]</sup> but it has also been noted that this syndrome is often associated with trauma resulting in focal ischemia and ulceration, pelvic floor disorders<sup>[64]</sup>, mucosal prolapse<sup>[14,52,64,65]</sup> and/or a larger systemic process<sup>[66]</sup>. Also, it has been associated with perineal descent, nonrelaxing puborectalis syndrome, and rectal prolapse<sup>[21,52]</sup>. In children, paradoxical contraction of pelvic floor and external anal sphincter muscles contributes to constipation, rectal prolapse, ischemia and finally rectal ulcer.

Diversity of the clinical presentation of SRUS requires a high index of suspicion of both the clinician and the pathologist for the definite diagnosis<sup>[30]</sup>.

The clinicopathological similarities between SRUS and inflammatory bowel disease and the limited pediatric experience of these conditions may lead to difficulties in differentiating these conditions, and could result in under-reporting of SRUS in this age group.

It can present as more common childhood intestinal conditions such as inflammatory bowel disease or constipation, causing difficult-to-manage lower gastrointestinal symptoms. Also, it may present as polypoid mass lesions<sup>[67]</sup>. A biopsy is required for confirmation of diagnosis, because ulceration may not be apparent at the time of endoscopy. SRUS should be considered in children presenting with rectal bleeding, mucorrhea and excessive straining during defecation.

Biofeedback<sup>[44,45,47,48]</sup>, sucralfate enema<sup>[15,50]</sup> and surgery seem to be ideal strategies because they aim to correct the underlying processes<sup>[57,58]</sup>. Behavioral modification or biofeedback therapy improves both rectal blood flow and symptoms and includes bowel habit training, avoiding excessive straining, and normalization of pelvic floor coordination<sup>[46-48,68,69]</sup>. Surgery is indicated in children with persistent bleeding per rectum not amenable to medical therapy and includes rectopexy, excision of ulcer, and rarely colostomy<sup>[42-44,56]</sup>. In children, a multitude of procedures have been advocated for rectopexy and a cure rate of at least 90% has been reported for posterosagittal rectopexy<sup>[45]</sup>. Also, El-Hemaly *et al*<sup>[70]</sup> have reported that the results of surgery and biofeedback are satisfactory in comparison to conservative treatment.

Most patients with SRUS in childhood have a satisfactory outcome using a simple behavioral modification approach. Ongoing follow-up to reinforce behavioral modification is important and may avoid long-term, treatment-resistant disease into adulthood. Despite the previous reports about SRUS in children that indicate low prevalence of the disease in childhood, recently we have been faced with higher prevalence rates in this age group. It seems that detailed and effective diagnosis methods such as endoscopy and histological examinations, as well as more attention by clinicians to this syndrome in children, have improved the diagnosis rate of the disease. Despite this being a benign condition in children, morbidity remains a problem as reflected by persistence of symptoms especially bleeding per rectum. Therefore, we are faced with an important condition that needs more attention and attempts for prevention and treatment.

More studies are needed to evaluate all of the aspects of the syndrome in children and to recommend the best treatment protocol. Every child with SRUS must be assessed individually using all modalities of investigation to define clearly the underlying pathophysiology, and to select the appropriate treatment strategies.

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