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(CASE REPORT)

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SERRATED MISMATCH REPAIR-DEFICIENT ALTERATIONS IN RECTAL MUCOSAL PROLAPSE SYNDROME: A CASE REPORT AND LITERATURE REVIEW

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ABSTRACT

Solitary rectal ulcer syndrome/mucosal prolapse syndrome (SRUS/MPS) is an uncommon, often misdiagnosed benign disorder of the rectum arising from chronic mechanical mucosal trauma. Its co-occurrence with sessile serrated polyps is rare and incompletely characterized, with diagnostic and pathogenetic significance in colorectal medicine.

We report the case of a 36-year-old woman with a two-year history of rectal bleeding, mucus discharge, tenesmus, and alternating bowel habits that had failed dietary modification, laxatives, and topical steroids. Colonoscopy identified multiple ulcerated polypoid lesions approximately 6 cm from the anal margin, and pelvic magnetic resonance imaging (MRI) demonstrated focal rectal wall thickening with mucosal irregularity, raising concern for malignancy. Following multidisciplinary tumor board discussion, the patient underwent surgical resection. Histology revealed fibromuscular obliteration of the lamina propria characteristic of mucosal prolapse alongside diffusely serrated, saw-toothed crypts with basal dilation and mild adenomatous change considered for sessile serrated polyps. Immunohistochemistry (IHC) demonstrated combined loss of MLH1 and HES1 expression within the serrated crypts with an elevated Ki-67 proliferative index, while other mismatch repair proteins remained intact. At 18-month follow-up, the patient was asymptomatic with no endoscopic recurrence.

This case illustrates that chronic mucosal prolapse injury can generate serrated, mismatch-repair-deficient morphology mimicking neoplasia, underscoring the need for careful histopathological correlation before presuming malignancy in similarly presenting rectal lesions.

Keywords: Solitary rectal ulcer syndrome; Mucosal prolapse syndrome; Sessile serrated-type crypt changes; MLH1; Serrated neoplasia pathway

1 INTRODUCTION

SRUS/MPS is a rare, benign but frequently misdiagnosed disorder of the rectum characterized by a clinicopathological triad of symptoms, endoscopic appearance, and histology that reflects chronic mechanical injury to the rectal mucosa rather than a primary inflammatory or neoplastic process. [1,2] Despite its name, the lesion is neither invariably solitary nor invariably ulcerated. It may present as a flat, polypoid, or erythematous mucosal abnormality anywhere from the rectosigmoid junction to the anal verge, a spectrum that predisposes clinicians to conflate it with adenocarcinoma, inflammatory bowel disease, or infectious proctitis. [1,3,4,5]

Patients, typically young adults with a female predominance, present with rectal bleeding, mucus discharge, tenesmus, straining, and a sense of incomplete evacuation that may persist for years before diagnosis. [5,6,7] diagnosis requires endoscopic visualization with biopsy, often supplemented by pelvic imaging and anorectal physiology testing to characterize prolapse or dyssynergic defecation. [8,9] First-line management is conservative, combining dietary fiber, laxatives, and biofeedback, while surgical resection is reserved for refractory, diagnostically ambiguous, or malignancy-suspicious lesions. [10,11,12] Outcomes are generally favorable, though healing and recurrence rates vary widely across reported series. [10] We report a case in which SRUS/MPS coexisted with sessile serrated polyps, an association with important implications for diagnosis and long-term surveillance, and we review the relevant literature.

2 CASE PRESENTATION

2.1 Clinical Presentation

A 36-year-old female presented with a two-year history of rectal bleeding, passage of mucus, and a sensation of incomplete evacuation, accompanied by alternating diarrhea and constipation. Her symptoms had progressively worsened, prompting her to seek specialized care. She had previously tried a high-fiber diet, laxatives, and topical steroids prescribed at a local clinic, but these measures provided only minimal and temporary relief.

Her medical history was otherwise unremarkable, and she reported no significant family history of colorectal cancer or inflammatory bowel disease. A recent surveillance colonoscopy revealed multiple small sessile serrated polyps on the right side of the colon. These were completely removed during the procedure, and no further surgical intervention was planned. However, the persistence of her primary symptoms, particularly the rectal bleeding and tenesmus, motivated her referral for further evaluation and definitive management of the rectal lesion.

2.2 Physical Examination and Diagnostic Workup

Physical examination was unremarkable except for a palpable, non-tender, irregular mass in the anterior rectal wall on digital rectal examination. A complete blood count revealed mild anemia, likely secondary to chronic blood loss. Carcinoembryonic antigen (CEA) levels were within normal limits. Flexible sigmoidoscopy and colonoscopy were performed, revealing multiple ulcerated polypoid lesions approximately 6 cm from the anal margin, which were surrounded by otherwise normal-appearing mucosa.

MRI of the pelvis demonstrated focal thickening of the rectal wall with mucosal irregularities, raising initial concern for a neoplastic process. The differential diagnosis based on clinical and radiological findings included rectal adenocarcinoma, inflammatory bowel disease, infectious proctitis, and SRUS/MPS. The primary clinical suspicion remained SRUS, given the patient's age, the nature of the symptoms, and the presence of associated serrated polyps.

2.3 Multidisciplinary Tumor Board Discussion

The case was presented at the weekly multidisciplinary tumor board meeting attended by gastroenterologists, colorectal surgeons, radiologists, and pathologists. The central discussion focused on the diagnostic dilemma posed by the patient's clinical, endoscopic, and radiological findings. The board carefully weighed the possibility of a primary rectal malignancy against the more benign, but rare, diagnosis of solitary rectal ulcer syndrome complicating sessile serrated polyps. The final pathological findings were considered the key to guiding management.

The team ultimately recommended proceeding with a definitive resection of the rectal lesion to achieve both a complete histological diagnosis and a therapeutic cure. The surgical plan was favored over repeated biopsies due to the patient's persistent, debilitating symptoms and the risk of underlying malignancy not fully captured on endoscopic sampling.

2.4 Treatment, Special Studies and Pathology

Following the multidisciplinary discussion, the patient underwent surgical resection of the rectal lesion. Histological examination of the resected specimen revealed a superficial ulcer with characteristic fibromuscular obliteration of the lamina propria, a hallmark of mucosal prolapse syndrome. (**Figure 1A, B**) Notably, the surrounding crypt architecture showed diffuse serrations with a saw-toothed pattern and basal dilation, along with mild adenomatous change with enlarged, tall, hyperchromatic basal nuclei, features consistent with sessile serrated polyps. (**Figure 2A, B**)

IHC studies demonstrated loss of MLH1 and HES1 expression in the serrated crypts, whereas other mismatch repair proteins, such as MSH2, MSH6, and PMS2, retained nuclear positivity. An increased Ki-67 proliferative index was observed in the surface epithelium of the serrated areas. These findings confirmed the diagnosis of solitary rectal ulcer syndrome in association with sessile serrated polyps, highlighting the potential for neoplastic transformation in such cases and underscoring the importance of careful pathological assessment of serrated lesions in the setting of mucosal prolapse.

2.5 Outcome and Follow-up

The procedure was successful, with no immediate postoperative complications. At 18 months of follow-up, the patient reported complete resolution of her symptoms, including rectal bleeding, tenesmus, and the sensation of incomplete evacuation. Follow-up colonoscopy showed no evidence of recurrence of the rectal ulcer or the previously removed sessile serrated polyps. Her quality of life improved significantly, and she returned to normal daily activities. The favorable outcome supports that definitive resection, guided by a multidisciplinary approach, can be a safe and effective treatment for symptomatic and diagnostically challenging cases of solitary rectal ulcer syndrome and associated serrated lesions.

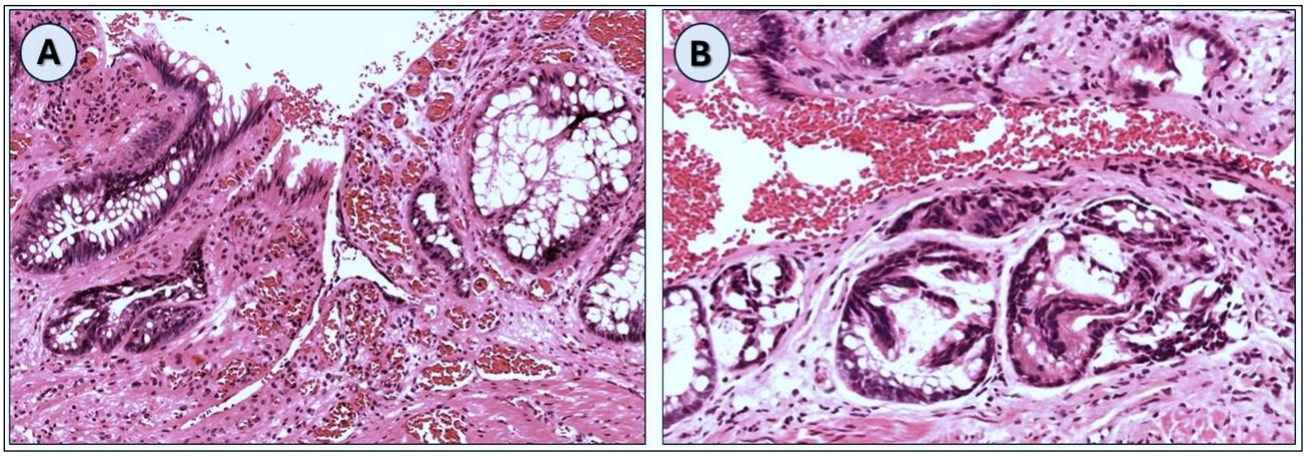

Figure 1: Microscopic features of mucosal prolapse syndrome

- 1A**:** Low-power view showing a superficial ulcer with characteristic fibromuscular obliteration of the lamina propria, a hallmark of mucosal prolapse syndrome (H&E X20)
- 1B**:** Intermediate-power view showing fibromuscular obliteration of the lamina propria and distorted diamond-shaped glands (H&E X40)

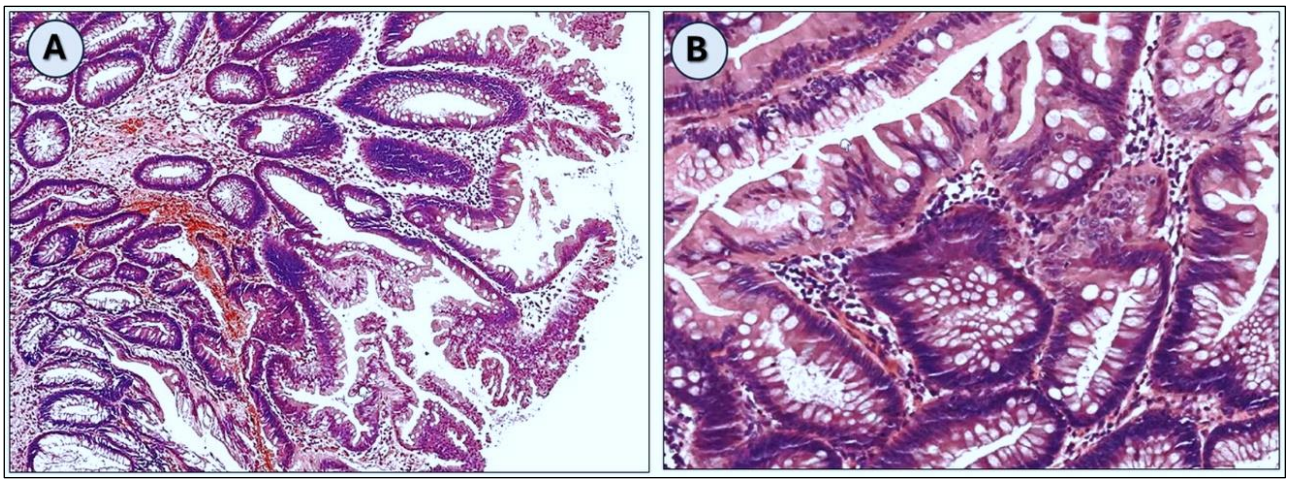

Figure 2: Microscopic features of sessile serrated-type crypt changes

- 2A**:** Low-power view showing surrounding crypt architecture with diffuse serration, a saw-toothed pattern, and basal dilation (H&E X20)

- 2B**:** Intermediate-power view showing mild adenomatous change with enlarged, tall, hyperchromatic basal nuclei, consistent with serrated adenomatous change (H&E X40)

3 DISCUSSION

3.1 Background

The entity now recognized as solitary rectal ulcer syndrome (SRUS) was first delineated as a distinct clinicopathological condition by Madigan and Morson in 1969, who described its characteristic symptom complex, endoscopic findings, and histology in a large series from St Mark's Hospital. [2] Rutter subsequently expanded this description in the mid-1970s [2], and in 1983 du Boulay and colleagues proposed "mucosal prolapse syndrome" as a unifying term after demonstrating closely overlapping histological and histochemical features between classical solitary ulcers and rectal mucosal prolapse, establishing the combined SRUS/MPS terminology now commonly used. [1]

The condition is rare, with an estimated incidence of approximately one per 100,000 per year and is widely considered underdiagnosed given its overlap with neoplastic, inflammatory, and infectious mimics. [4] It typically arises in the third and fourth decades of life with a female predominance, consistent with the patient described here, though pediatric and elderly presentations are recognized. [4, 5] Established risk factors include chronic straining at defecation, paradoxical puborectalis contraction, and internal or overt rectal prolapse/intussusception. [5, 13]

Separately, the World Health Organization classifies serrated colorectal lesions into hyperplastic polyps, sessile serrated lesions (synonymous with sessile serrated adenoma/polyp) with or without cytological dysplasia, and traditional serrated adenomas, a framework that identifies sessile serrated lesions as precursors of roughly one-third of colorectal cancers arising through the serrated neoplasia pathway. [14,15]

3.2 Pathogenesis, Pathophysiology

The mechanical pathogenesis of SRUS/MPS centers on chronic, repetitive trauma to the anterior rectal wall generated by internal mucosal prolapse or intussusception combined with paradoxical contraction of the puborectalis and external anal sphincter during straining. This produces a high transmural pressure gradient, localized ischemia, and a chronic injury-repair cycle. [9,13] This process yields the histological hallmark of fibromuscular obliteration of the lamina propria together with reactive crypt hyperplasia and distortion, findings shared by SRUS and complete rectal prolapse specimens alike. [16]

In parallel, sessile serrated polyps arise through a distinct molecular route, the serrated neoplasia pathway, initiated by activation of the BRAF V600E mutation in most lesions, which drives a CpG island methylator phenotype (CIMP) and, in a subset, MLH1 promoter hypermethylation with resultant mismatch repair deficiency. [17,18] Notably, Ball and colleagues demonstrated that repeated mucosal trauma in SRUS itself can generate serrated, hyperproliferative epithelium with focal MLH1 loss, suggesting that chronic prolapse injury may directly promote acquisition of serrated,

preneoplastic morphology and providing the most direct literature-supported mechanistic bridge between the two processes converging in this case. [19]

3.3 Comparative Analysis of Our Case with Existing Literatur

The present case aligns closely with Sun and colleagues' 2018 report of a 59-year-old man with an ulcerative nodular rectal lesion and MRI findings suspicious for neoplasia, whose histology showed hyperplastic lamina propria with diffusely serrated crypts and IHC loss of both HES1 and MLH1. Their report is essentially identical in morphological and immunophenotypic profile to that described here, differing chiefly in patient age and sex. [20]

Giardiello and colleagues described a patient whose multiple rectal hyperplastic and adenomatous-appearing polyps were ultimately reclassified as a polypoid variant of MPS after histopathological review, paralleling the multiple ulcerated polypoid lesions and mild adenomatous change encountered in the present patient and reinforcing how MPS can convincingly mimic true polyposis. [21]

Amaechi and Papagrigoriadis reported SRUS producing MRI findings indistinguishable from rectal carcinoma, directly mirroring the pelvic MRI findings that raised initial concern for malignancy in this case. [8]

Lambin and colleagues, and separately Nonaka and colleagues, each described adenocarcinoma arising within long-standing prolapsed or chronically ulcerated rectal mucosa, underscoring that, unlike the present case, which showed only mild adenomatous change without frank malignancy, chronic MPS can occasionally progress to invasive carcinoma and therefore warrants durable endoscopic surveillance even after apparently curative resection. [22,23]

4 WHAT HAVE WE LEARNED FROM THIS CASE?

This case underscores that SRUS/MPS should remain firmly on the differential diagnosis when rectal ulceration or polypoid change is accompanied by imaging findings suggestive of malignancy, since endoscopic and radiological mimicry of adenocarcinoma is well documented and can otherwise prompt overly aggressive or misdirected management. [8,20]

It reinforces that serrated, MLH1-deficient morphology arising within a background of chronic mucosal prolapse does not automatically equate to a sporadic sessile serrated polyp or an independent neoplastic process. That combined clinical, endoscopic, and IHC correlation, including Ki-67 and mismatch repair protein studies, is essential to reach the correct diagnosis. [18,19]

The favorable outcome achieved through definitive resection guided by multidisciplinary discussion, rather than repeated inconclusive biopsy, supports surgery as a reasonable strategy in diagnostically ambiguous, symptomatic, refractory cases. [10,20]

Finally, given the documented, if uncommon, potential for chronic MPS and serrated lesions to harbor or progress to dysplasia and carcinoma, sustained endoscopic surveillance beyond the immediate postoperative period is a prudent component of long-term management, even after symptom resolution. [20,22,23]

Abbreviations:

- Solitary rectal ulcer syndrome/mucosal prolapse syndrome (SRUS/MPS);
- Immunohistochemistry (IHC)

5 CONCLUSION

This case adds a rare, well-documented instance of solitary rectal ulcer syndrome/mucosal prolapse syndrome coexisting with sessile serrated polyps, corroborating the small existing body of literature suggesting that chronic mucosal prolapse injury can generate serrated, mismatch-repair-deficient epithelium that closely mimics both sporadic serrated neoplasia and invasive malignancy.

By pairing detailed clinical, endoscopic, radiological, and immunohistochemical documentation with a favorable 18-month outcome after multidisciplinary-guided resection, this report reinforces that recognition of this overlap syndrome is achievable without resorting to unnecessarily radical surgery, provided pathological assessment is rigorous.

We share this case to sharpen clinical suspicion for this entity among gastroenterologists, colorectal surgeons, and pathologists, and to recommend continued endoscopic surveillance in patients with this uncommon but clinically consequential association.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

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- Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organization that might be interested in the submitted work.

Statement of ethical approval

The present research work does not include any studies involving animal or human subjects by any of the authors. This study was conducted as a retrospective review of archival pathology material collected during routine clinical care. All data were fully de-identified prior to analysis.

Statement of informed consent

Informed consent was obtained from the patient for publication of this case.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

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