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ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2016, 33(1):135–136

# Surgery Quiz – Case 10

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An otherwise healthy 42-year-old man with family history of colorectal cancer meeting Amsterdam II criteria presented to the emergency department after observing the passage of bright red blood from the rectum. Abdominal examination was unremarkable and rectal examination demonstrated bright red blood. Laboratory investigations showed signs of iron-deficiency anemia. Colonoscopy revealed: (a) Two pedunculated sigmoid adenomas submitted to snare polypectomy. Histology demonstrated two tubulovillous adenomas with mild dysplasia; (b) erythematous appearance of the anterior rectal mucosa 6 cm from the anal verge (fig. 1). No biopsy of the affected area was taken and (c) first degree internal hemorrhoids. The cause of hematochezia was considered to be the sigmoid adenomatous polyps. One month after the initial endoscopy, the patient presented to the outpatient department complaining of continuation of rectal bleeding. Subsequent 60 cm flexible sigmoidoscopy was performed which demonstrated: (a) No post-polypectomy complications from the polypectomy sites at the sigmoid colon, and (b) persistence of the erythematous appearance of the anterior rectal mucosa. Biopsies of the involved rectal mucosa revealed fibromuscular obliteration of the lamina propria, hypertrophied muscularis mucosa with extension of muscle fibers upwards between the crypts and mild inflammation (fig. 2). The patient had early response to high-fiber diet, bulking laxatives and mesalazine enemas with remission of rectal bleeding.

What is the diagnosis?

## Comment

*Solitary rectal ulcer syndrome (SRUS) is a chronic, benign disorder. Although SRUS is characterized by a combination of well-described clinical, endoscopic and histopathological features, it remains an underdiagnosed entity. SRUS has an estimated annual prevalence of 1 per 100,000 people submitted to colonoscopy. This syndrome is usually presented in the third to fourth decades of life and tends to affect more commonly women than men. The pathogenetic mechanism of SRUS is considered to be mucosal ischemic injury of the rectum. It has been suggested that uncoordinated defecation with excessive straining may result in mucosal prolapse and local ischemia of the anterior upper anal canal wall. Indeed, dyssynergia due to paradoxical anal contractions and subsequent rectal prolapse are present in a significant percentage of patients with SRUS. However, rectal mucosal prolapse without SRUS is also a common finding in healthy subjects. Consequently, rectal prolapse and SRUS are considered to be two disparate conditions.*

*SRUS is a well-described syndrome. However, the term "solitary rectal ulcer" is a misnomer as only a quarter of the adults have a true rectal ulcer and the lesion is not necessarily solitary or ulcerated. Diagnosis is based on specific clinical, endoscopic and histopathological features. SRUS is characterized by hematochezia, mucous discharge, tenesmus and local perianal pain. Rectal bleeding in the absence of other anorectal disorder should raise high clinical*

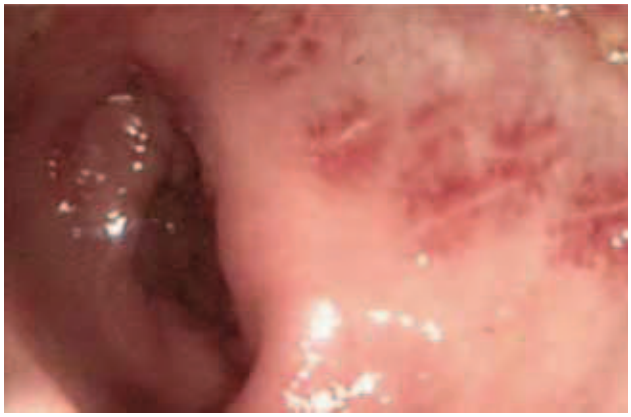

**Figure 1.** Colonoscopy revealed diffuse erythematous mucosa of the anterior rectal wall 6 cm from the anal verge.

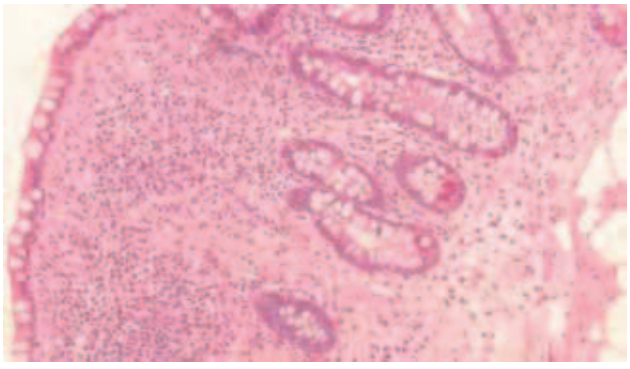

**Figure 2.** Histology showed fibromuscular obliteration of the lamina propria, hypertrophied muscularis mucosa with extension of muscle fibers upwards between the crypts and associated mild inflammation.

*suspicion of the syndrome and should dictate further evaluation. The appearance of SRUS on endoscopy may vary from pre-ulcer hyperemic changes of the rectal mucosa to established ulcers covered by a white, grey or yellowish slough. The ulceration is usually shallow and the adjacent mucosa may appear nodular, lumpy or granular. Multiple lesions are recognized in 30% of patients. As a result of the wide endoscopic spectrum, SRUS may be misdiagnosed and biopsy specimens of the involved rectal and colonic mucosa are needed to confirm the diagnosis. Key histological features include fibromuscular obliteration of the lamina propria, hypertrophied muscularis mucosa with extension of muscle fibers upwards between the crypts and glandular crypt abnormalities. Indeed, diffuse collagen deposition in the lamina propria and abnormal smooth muscle fiber extensions are sensitive markers for differentiating SRUS from other conditions. Other minor microscopic findings include surface erosion, mild inflammation and reactive epithelial atypia. Differential diagnosis includes inflammatory bowel disease, ischemic colitis, chronic radiation colitis/proctitis, vasculitides, arteriovenous malformations, varices and dielafoy lesion of the rectum.*

*First line treatment of SRUS includes patient's behavioral modification (avoidance of straining, regulation of toilet habits) and encouragement of a high-fiber diet in combination with stool softeners and bulking laxatives. When the symptoms persist, a more organized form of behavioral therapy (biofeedback) and topical treatments (sucralfate, corticosteroids, sulfasalazine, mesalazine, topical fibrin sealant) have been reported to be effective in improving the symptoms. Conservative approaches are less useful when SRUS is associated with advanced grade of inflammation, fibrosis and rectal mucosal prolapse. Surgery is indicated when conservative treatment fails to control severe symptoms. Surgical treatment includes ulcer excision, management of internal or overt rectal prolapse (open and laparoscopic rectopexy, mucosal resection, perineal proctectomy, mucosal sleeve resection with coloanal pull-through) and defunctioning colostomy.*

*In our patient's case, SRUS had an atypical presentantion. Rectal bleeding was the solitary clinical symptom. Initial endoscopy revealed two adenomatous polyps of the sigmoid colon submitted to polypectomy and multiple sites of pre-ulcer erythematous rectal mucosa instead of a "solitary rectal ulcer". The cause of hematochezia was considered to be the sigmoid adenomas. Continuation of rectal bleeding despite of the index polypectomy raised high clinical suspicion of SRUS. Subsequent sigmoidoscopy was performed for biopsy confirmation of the syndrome before initiation of treatment. Biopsies of the hyperemic rectal mucosa revealed fibromuscular obliteration of the lamina propria and hypertrophied muscularis mucosa with extension of muscle fibers upwards between the crypts, and established the diagnosis of SRUS. The patient had early response to high-fiber diet, bulking laxatives and mesalazine enemas and subsequent response to behavioral modification and high-fiber diet with remission of symptoms.*

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