

# Colonic Endometriosis Mimicking Colonic Carcinoma

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## Abstract

### INTRODUCTION

Endometriosis was first defined in 1860 by von Rokitansky as the presence of functioning endometrial glands and stroma outside the uterine cavity. While ectopic endometrial tissues are frequently observed on the surfaces of the uterus and adnexae, they might rarely be observed on the serosal surfaces of bowel and laparotomy incisions, in the lungs, bones and in the urinary tract [1,2,3]. Extra-pelvic endometriosis is most frequently seen in bowels, which is usually asymptomatic. However, bowel endometriosis might show non-specific symptoms such as abdominal colic-like pain, nausea, vomiting, constipation, diarrhea and rectal bleeding. From 0.7% - 2.5% of patients require bowel resection for symptomatic lesions [6].

We present a case of sigmoid endometriosis causing intestinal obstruction mimicking carcinoma of the sigmoid colon.

### CASE REPORT

This is a case of a 41-year-old lady who had endometriosis for 18 years. The patient was well till three months prior to her symptoms where she started to have bleeding per rectum, which was bright red coming during the days of menstrual period, 4-6 times per day during passing stool and in between, minimal in quantity and covering the stool sometimes. It was associating with mucous with painful defecation. She was complaining also of abdominal pain which was dull and generalized and was more severe during passing stool. It was severe enough to make her stay for half an hour in the washroom. There was no history of bleeding tendency, bleeding from other sites, melena, or hematemesis. She had regular menses. Systemic review was unremarkable.

On examination, she looked well, not in pain and she was not pale. She was not tachycardic and she was afebrile. Abdominal examination revealed soft abdomen and no

tenderness. Per rectal examination was normal and no blood was seen. Blood Chemistry, complete blood count, Coagulation profile, alfa-fetoprotein and carcinoembryonic antigen were within normal limits.

Colonoscopy showed stricture in the sigmoid colon which cannot be advanced to the sigmoid colon. Barium study showed area of constant narrowing at the sigmoid colon (Figure 1).

### Figure 1

Figure 1: shows constant narrowing (arrowed) at the sigmoid colon.

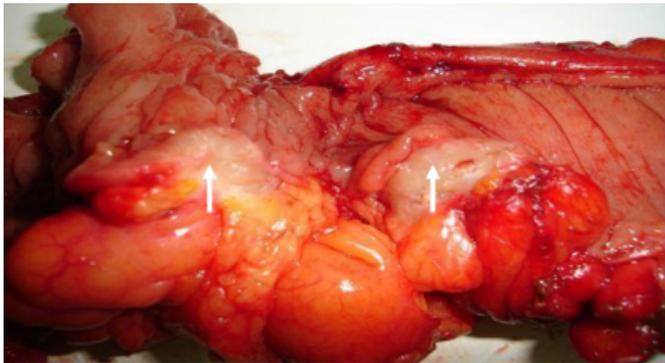


MRI showed cystic lesion in the liver at the right side of the recto-sigmoid compressing the lumen of the rectum. Another mass was found between the left colon and the left ovary, and a third one in the fundus of the uterus most likely a fibroid. The impression of this case was either endometriosis involving the recto- sigmoid colon or carcinoma of the mentioned area. On laparotomy, a free constricting mass, was

identified in the sigmoid and the upper part of the rectum. The sigmoidectomy was done and the frozen section was sent for pathology. Gross examination showed a 26 cm sigmoid colon. Upon opening there were two masses; one measured 4.5 cm, and the other measured 4 cm. The overlying mucosa appeared intact. The cut surface was white and firm with punctuate hemorrhage (Figure 2). The masses involve the serosal surface.

**Figure 2**

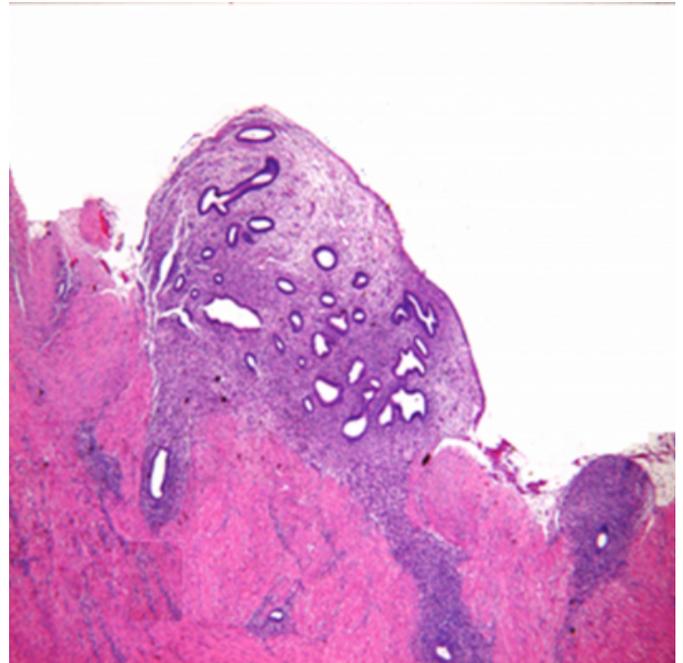
Figure 2: Two discrete firm white masses, with an overlying punctuate haemorrhage seen at the serosal surface of the sigmoid colon



Microscopic examination showed colonic wall with scattered endometrial-type glands surrounded by varying amounts of dense cellular endometrial-type stroma involving the mucosa, submucosa and muscularis propria (fig 3a, b, and c).

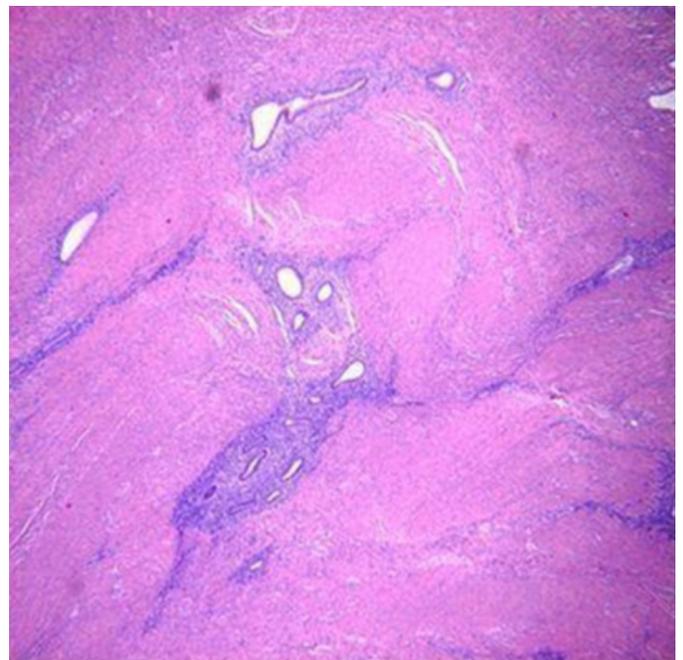
**Figure 3**

Figure 3 a : Shows colonic surface mucosa replaced by endometrial glands and stroma , some of which are protruding into the muscle layer X10



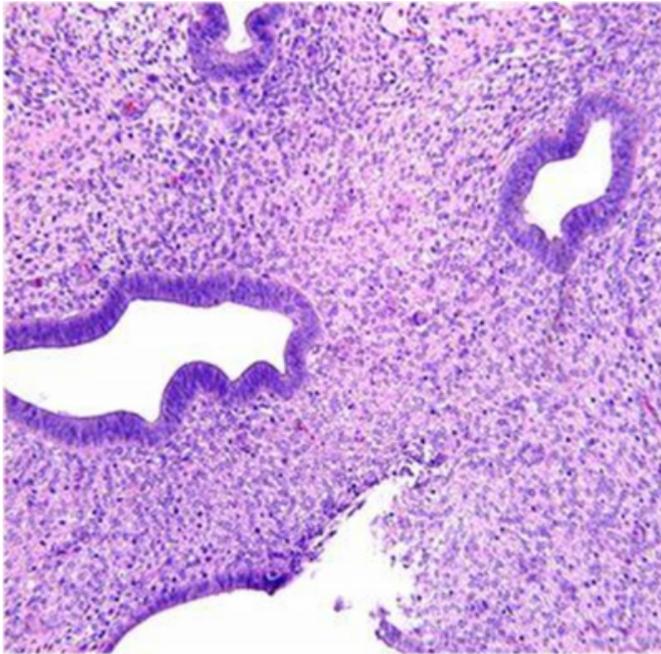
**Figure 4**

Figure 3 b : Endometrial glands and stroma scattered in a hypertrophied muscular layer of the colon. X4



### Figure 5

Figure 3 c: High power shows well developed endometrial glands and stroma. X20.



### DISCUSSION

The exact cause and pathogenesis of endometriosis is [are] unclear. Three main theories exist that attempt to explain this disease. Previous theories suggest that endometriosis results from the transport of viable endometrial cells through retrograde menstruation. Retrograde menstruation, however, is a common physiologic event during which viable endometrial cells are shed into the peritoneal cavity. Another theory suggests that transtubal dissemination is the most common route of spread, although other routes have been observed, including lymphatic and vascular channels. Finally, iatrogenic deposition of endometrial tissue has been found in some cases following gynaecologic procedures and caesarean sections. [1]. The endometrium and the peritoneum are derivatives of the same coelomic wall epithelium. Peritoneal mesothelium has been postulated to undergo metaplastic transformation to endometrial tissue. Such transformation may occur spontaneously or may be facilitated by exposure to chronic irritation of the retrograde menstrual fluid.

Recent researches have suggested involvement of the immune system in the pathogenesis of endometriosis. Women with this disorder appear to exhibit increased humoral immune responsiveness and macrophage activation while showing diminished cell-mediated immunity with decreased T-cell and natural killer cell responsiveness [1].

Although the exact prevalence of endometriosis among premenopausal women is not known, it is assumed to be between 1% and 7% [12]. Extra-genital endometriosis is seen mostly at the recto-sigmoid junction, with less frequent observation at the rectovaginal septum, small intestine, cecum and appendix [3456]. In a review by Weed and Ray of 3037 laparotomies performed for endometriosis, 163 (5.4%) cases of them were involving the bowel. The distribution in the bowel was 39% in the sigmoid colon (65 cases), 20% in the recto-sigmoid colon (33 cases), 19% in the appendix (32 cases), 10% in the rectum (17 cases), 7% in the terminal ileum (11 cases), 4.5% in the caecum (9 cases) and 0.6% in the transverse colon (1 case); sigmoidal and rectal endometriosis accounted for 69% of the cases [7]. In a 20 year review of bowel endometriosis at the Mayo Clinic, the incidence of small intestinal endometriosis was 0.53% (38/7200) [13].

Microscopic examination of the intestine involving endometriosis usually discloses an endometrial stroma and gland islands located between muscular fibers, subserosa and serosa. Normally, the mucosa is found to be intact, but in some cases, the endometrial tissue reaches it in the form of small islands, which cause rectal bleeding coincidental with the menstrual period [312]. In our case, there was mucosal involvement that caused the rectal bleeding. Endometrial glands formed of multifocally placed cuboidal epithelium and stroma spreading under the intestinal mucosa to the serosa were observed.

Out of 7200 cases of endometriosis presented by the Mayo Clinic, 38 cases of small intestine involvement were observed; however, small intestinal obstruction was reported in only 11 cases [13]. In the series presented by Prystowsky and colleagues, gastrointestinal involvement was observed in 85/1574 (5.4%) patients; intestinal resection was done in only 11 patients (0.7%) because of unremitting gastrointestinal symptoms, four cases with small bowel, and five cases with large intestine involvement [6]. Graham et al. reported colon obstruction in only two patients among 32 (6%) cases of endometriosis with colon or rectum involvement [10]. Similarly, Williams et al. noted only one intestinal obstruction case out of 178 patients with gastrointestinal involvement [4].

Intestinal endometriosis is usually asymptomatic, but gastrointestinal bleeding, nausea; vomiting, cramp-like abdominal pain, painful defecation, diarrhea, constipation, recto-vaginal colonic mass, intussusception, bowel

obstructions and intestinal perforation can be seen [31011121314]. Our patient was complaining of bleeding per rectum, associating with mucous with painful defecation. Intestinal endometriosis can also lead to massive exudative ascites and hemoperitoneum due to diffuse serosal and peritoneal involvement which may imitate carcinomatosis [15]. In some rare instances, endometrial foci might show malignant transformation [16]. The clinical differential diagnosis encompasses a wide range of diseases including primary bowel carcinoma, diverticulosis, chronic inflammatory bowel disease, carcinoid tumor, benign intra-mural tumors, metastases from occult intra-abdominal malignancies, pelvic and mesenteric tumors and cysts [9].

It is remarkably difficult to diagnose intestinal endometriosis by the pre-surgical radiological imaging methods. Recto-sigmoid endometriosis usually manifests on double contrast barium enema studies as an extrinsic mass, flattening, tethering with speculation of the anterior border of the Recto-sigmoid colon. Much less commonly, colonic disease may manifest as an annular lesion or as a polypoid intraluminal mass [9101718]. In our case, barium study showed area of constant narrowing at the sigmoid colon. Since endometriosis is located in the bowel wall or in muscle layer as implants, narrowing, spasm, discoloration, and hyperemia in the affected bowel wall can be seen through colonoscopy [1017]. As the biopsies from the suspected area do not contain submucosa and muscle layers, they do not allow for diagnosis [17]. Although it is possible with computerized tomography to determine thickening of the bowel wall, a discrete mass and intestinal obstruction, it gives no clues about the histopathologic diagnosis. Recently devised methods such as endoscopic US and CT (virtual) colonoscopy can also be used in the diagnosis of endometriosis seen in the recto-sigmoid region [1920].

There are various manifestations of endometriosis that may lead to stricture, kinking or angulations of the bowel producing partial or complete obstruction. Intestinal obstruction may be produced by a localized endometriosis pressing on the lumen or causing intussusceptions. More commonly, however, diffuse endometriosis is seen with marked smooth muscle hyperplasia or fibrosis and consequent stenosis of the bowel lumen. The muscle layer usually becomes hypertrophied when benign glandular tissue is found ectopically in the bowel wall. In addition, cyclical hemorrhage may occur in the bowel wall when the endometriosis is stimulated by cyclical hormonal changes leading to inflammation and subsequent fibrosis. Adhesions

secondary to endometriosis can also cause bowel obstruction [22].

The objective of the treatment in pelvic endometriosis is to cease the endometrial stimulus in order to ameliorate the symptoms. Thus, danazol, gonadotropin-releasing hormones, oral contraceptives, and prostaglandin inhibitors can be used. The conclusive treatment of endometriosis is total abdominal hysterectomy, bilateral salpingo-oophorectomy and removal of all endometrial foci. Because malignant transformation cannot be excluded preoperatively and medical treatment may cause fibrosis, the definitive treatment is surgical [2491017]. Also, in the case of intestinal obstruction and severe rectal and abdominal pain, surgery is indicated. The main objective of surgery is the resection of the affected bowel segment, enabling the histopathological examination of the resection material. Limited surgery, such as excision or cauterization of superficial lesions, following confirmation through frozen section analysis could be performed [91011].

## CONCLUSION

Intestinal endometriosis is a disease that may imitate various gastrointestinal system diseases. The diagnosis could only be confirmed by histopathologic examination, since there are no pathognomonic, radiological or colonoscopic findings. In female patients who have unexplained digestive complaints, endometriosis should also be considered in the differential diagnosis.

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