

# Endometriotic stricture of the sigmoid colon presenting with intestinal obstruction

M Jayant,<sup>1</sup> MS; R Kaushik,<sup>1</sup> MS, DNB; R S Punia,<sup>2</sup> MD

<sup>1</sup> Department of Surgery, Government Medical College and Hospital, Sector 32, Chandigarh, India

<sup>2</sup> Department of Pathology, Government Medical College and Hospital, Sector 32, Chandigarh, India

**Corresponding author:** R Kaushik (robinkaushik@yahoo.com)

Endometriosis, a relatively common condition, rarely involves the bowel; even more rarely does it present as a large-bowel stricture with intestinal obstruction. We report the case of a young woman who presented to an emergency department with intestinal obstruction secondary to an endometriotic stricture of the sigmoid colon, without evidence of disease elsewhere in the peritoneal cavity. Although large-bowel obstruction is usually caused by a malignant tumour, it can sometimes result from rare causes such as endometriosis. Symptoms of a cyclical nature may remind the clinician of this possibility.

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Endometriosis, first described by von Rokitansky in 1860, is an oestrogen-dependent inflammatory disease characterised by the presence of functional endometrial tissue outside the uterus.<sup>[1,2]</sup> This benign but painful condition affects up to 15% of women of childbearing age,<sup>[2]</sup> and nearly half of all infertile women, and 70% of women with chronic pelvic pain have endometriosis.<sup>[1,2]</sup> Together with dyspareunia, the above symptoms form the 'classic triad' of the disease.<sup>[3]</sup>

Endometriosis can be intra- or extraperitoneal, depending on the location of the endometrial tissue. Intraperitoneal disease is mainly pelvic or genital, and the most common sites of involvement are the ovaries, uterine ligaments, fallopian tubes, pelvic peritoneum and pouch of Douglas.<sup>[2,4]</sup> The most common site of extra-genital (extra-pelvic) endometriosis is the gastrointestinal tract (GIT),<sup>[3]</sup> but the omentum, peritoneum and other intra-abdominal organs can also occasionally be involved. Extraperitoneal disease is uncommon, and may be seen in the cervix, vagina, abdominal scars and hernia sacs. Rarely, endometriosis affects the urinary system, lungs, skin, diaphragm and central nervous system.<sup>[1,2,4]</sup>

The GIT is involved in 3 - 37% of women with endometriosis, and although it can affect any site, the rectosigmoid colon (72%), small intestine (7%), caecum (3.6%) and appendix (3%) are most commonly involved.<sup>[1-5]</sup> Usually the disease takes the form of small asymptomatic serosal implants, but these can progress and become symptomatic.<sup>[6]</sup> The symptoms are usually chronic and cyclical;<sup>[1-6]</sup> acute presentations such as intestinal obstruction, appendicitis, appendicular intussusception, rectal bleeding and bowel perforation are relatively uncommon.

We report the case of a young woman who presented with intestinal obstruction due to rectal stricture secondary to endometriosis. A search of available English language literature revealed that large-bowel obstruction due to colonic endometriosis is rare.

## Case report

A 23-year-old woman presented to our emergency department with abdominal pain, nausea, vomiting and abdominal distension of 1 week's duration. She had been having similar symptoms repeatedly over the past 6 months, and her local doctor had diagnosed intestinal tuberculosis and started her on antituberculosis therapy. On examination, she was dehydrated and sick-looking, with tachycardia, tachypnoea and palpable bowel loops. Abdominal radiographs revealed dilated ileal and large-bowel loops. In view of her condition, a provisional diagnosis of recurrent small-bowel obstruction (possibly secondary to tuberculosis) with the possibility of strangulation or perforation was made, and she was taken to the operating theatre after she had been adequately resuscitated.

Laparotomy revealed adhesions of the ileum in the pelvis, which were easily mobilised. On following the bowel, the sigmoid colon was found to be densely adherent to the left ovary and the left iliac vessels, all forming a jumbled-up mass that could not be separated safely. This was the point of bowel obstruction, and there appeared to be no way of mobilising or resecting it safely. A decision to perform a relook operation after further investigations was made, and a loop ileostomy was performed in the right lower abdomen. Postoperatively the patient underwent colonoscopy, which revealed a tight, non-negotiable stricture in the sigmoid colon. Multiple biopsies revealed nothing of note, and carcino-embryonic antigen levels were within normal limits. After discussing the situation with the patient and her family, it was decided to re-operate 4 - 6 weeks later.

A computed tomography scan after 6 weeks revealed a circumferential growth in the sigmoid colon, with obliteration of the lumen. There was no other abnormal finding. At surgery a tight stricture was seen in the sigmoid colon, with minimal adhesions. The ovaries were enlarged, but there was no other significant intra-abdominal finding. A sigmoid colectomy was performed, with primary colorectal anastomosis in a single layer. The postoperative

course was uneventful, and the ileostomy was taken down and bowel continuity restored 3 weeks later.

Histopathological examination of the resected specimen (Fig. 1) revealed endometriotic deposits in the muscularis and submucosa, without involvement of the mucosa (Fig. 2).

## Discussion

Although GIT endometriosis was first described by Sampson in 1922,<sup>[1]</sup> it is often not suspected or diagnosed as a cause of GIT symptoms – the aetiology is obscure, the presentation is variable, and there are no specific tests for making the diagnosis.<sup>[2,5]</sup>

A range of theories have been put forward to explain the pathogenesis of endometriosis, but none has yet been fully accepted. The most widely accepted is Sampson's theory of retrograde menstruation, according to which endometrial tissue refluxes through the fallopian tubes during menstruation and implants on the serosal surface of abdominal and pelvic organs.<sup>[2,7,8]</sup> Another theory (Minh's theory) suggests that extra-uterine growth of endometriotic tissue occurs as a result of metaplastic transformation of pluripotent peritoneal mesothelium.<sup>[2,8]</sup> Other hypotheses suggest migration of cells through the lymphatics or bloodstream, development of

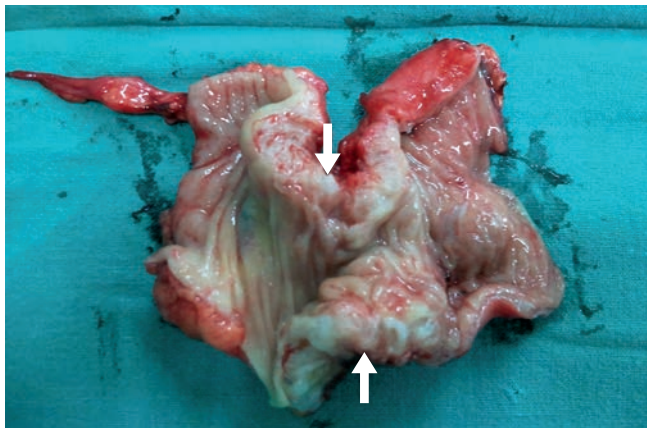


Fig. 1. Gross specimen of the resected sigmoid colon cut open to show a tight stricture (arrows).

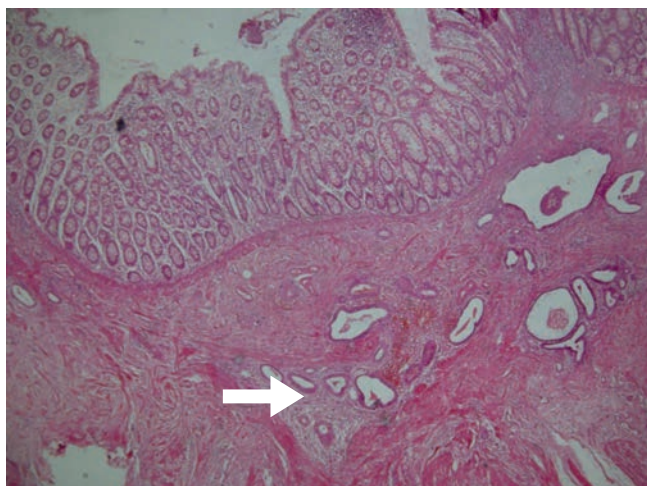


Fig. 2. Histopathology photomicrograph of the resected specimen showing endometriotic deposits in the muscularis and submucosa (arrow), without involvement of the mucosa (H&E  $\times 40$ ).

endometrial nodules by metaplasia of Müllerian remnants, iatrogenic implantation, infiltration along the nerves, or direct infiltration along structures such as the round ligament.<sup>[2,7-9]</sup> In all probability, endometriosis is a complicated, multifactorial disease involving a complex interplay of genetic, familial, immunological and other factors.

Although the rectosigmoid colon is the commonest site of involvement, any part of the GIT can be affected.<sup>[1-9]</sup> Bowel endometriosis occurs as an 'invasion phenomenon' from outside – implantation begins on the serosa and sometimes invades the muscularis propria, but the mucosa is rarely affected.<sup>[2,5,7]</sup> Lesions that do not infiltrate beyond the subserosa remain asymptomatic, symptoms appearing only when the disease infiltrates the muscular layer.<sup>[6,7]</sup> The symptoms and signs of bowel endometriosis vary depending on the site and extent of involvement,<sup>[1-6]</sup> although the mechanism by which bowel endometriosis causes intestinal symptoms remains unclear – large lesions may cause fibrosis and thickening of the bowel wall, resulting in stricturing and mechanical bowel obstruction, or they may infiltrate and damage the intestinal nervous plexus or the interstitial Cajal cells, or cause attrition of the intestinal sympathetic nerve fibres.<sup>[5]</sup>

It can be very difficult to distinguish GIT endometriosis from other GIT pathology, since there are no pathognomonic symptoms of the disease. Patients may sometimes be asymptomatic, but the majority present with nausea, constipation, diarrhoea, bloating, tenesmus, painful defaecation, lower abdominal or pelvic pain, bowel perforation or rectal bleeding. These symptoms are usually cyclical, occurring at the time of menstruation.<sup>[1-9]</sup> Endometriosis of the appendix can mimic acute appendicitis, and obstruction of the small or large bowel can occur when they are involved and needs to be differentiated from carcinoma, especially in the large intestine. Rarely, malignant change can occur in endometriotic foci.<sup>[2]</sup>

The diagnosis of large-bowel endometriosis remains difficult.<sup>[1-9]</sup> A variety of investigative techniques have been used, including transvaginal or transrectal ultrasound, colonoscopy, barium enema examination, computed tomography (CT) and magnetic resonance imaging (MRI), with variable results.<sup>[2,3,5-7]</sup> Colonoscopy is of limited value in the diagnosis, because the disease invades inwards from the serosa, and the mucosa remains uninvolved in the majority of cases. In addition, the endometriotic deposits may cause secondary mucosal changes that can mimic the appearance of other diseases such as inflammatory bowel disease, ischaemic colitis or even cancer.<sup>[3,5,7,10]</sup> Barium enema examination or a CT scan usually do not demonstrate anything beyond extrinsic bowel compression. MRI remains the most sensitive imaging technique, although recently multidetector CT enteroclysis has been reported to be useful.<sup>[5]</sup> There are no set guidelines for patient evaluation, and if endometriosis is clinically suspected, laparoscopic assessment along with excision of lesions (for histological examination) remains the gold standard for diagnosis.<sup>[3,5,6]</sup>

The treatment of large-bowel endometriosis depends upon the severity of disease and the patient's age and desire to maintain her fertility.<sup>[2,3,5,10]</sup> If it is detected before there are complications, hormonal therapy with danazol, gonadotrophin-releasing hormone analogues or progestins is indicated.<sup>[5,10]</sup> These cause ovarian suppression, leading to a state of pseudo-pregnancy, and may reduce the size of

the endometrial deposits and improve the symptoms, but it is not clear what effect hormonal therapy has on the natural long-term progression of intestinal endometriosis. Patients need to be informed that even if their pain improves the disease may progress, so they need to be carefully monitored.<sup>[5]</sup> When patients present with pain, bleeding and changes in bowel habits, and it not possible to rule out a malignant tumour and intestinal obstruction, surgery (laparoscopic or open, dictated by the level of expertise available) is indicated, and has resulted in a significant improvement in pain, quality of life and fertility.<sup>[3,5,10]</sup> The goal of surgery for bowel endometriosis is removal of the affected area and restoration of bowel continuity and function, in addition to providing tissue for definitive diagnosis.<sup>[2,3,7]</sup> Surgery is tailored to the extent of disease found at operation, and can range from simple nodule excision to segmental bowel resection.<sup>[1-3,5,7,10]</sup>

#### REFERENCES

1. Nezhat C, Hajhosseini B, King LP. Laparoscopic management of bowel endometriosis: Predictors of severe disease and recurrence. *JSLs* 2011;15(4):431-438.
2. De Ceglie A, Bilardi C, Bianchi S, et al. Acute small bowel obstruction caused by endometriosis: A case report and review of the literature. *World J Gastroenterol* 2008;14(21):3430-3434. [<http://dx.doi.org/10.3748/wjg.14.3430>]
3. Dimoulis P, Koutroubakis I, Tzardi M, et al. A case of sigmoid endometriosis difficult to differentiate from colon cancer. *BMC Gastroenterology* 2003;3(1):18. [<http://dx.doi.org/10.1186/1471-230X-3-18>]
4. Saleem A, Navarro P, Munson JL, Hall J. Endometriosis of the appendix: Report of three cases. *Int J Surg Case Rep* 2011;2(2):16-19. [<http://dx.doi.org/10.1016/j.ijscr.2010.11.001>]
5. Ferrero S, Camerini G, Maggiore ULR, et al. Bowel endometriosis: Recent insights and unsolved problems. *World J Gastrointest Surg* 2011;3(3):31-38. [<http://dx.doi.org/10.4240/wjgs.v3.i3.3>]
6. Lin Y, Kuo L, Chuang A, Cheng T, Hung C. Extrapelvic endometriosis complicated with colonic obstruction. *J Chin Med Assoc* 2006;69(1):47-50. [[http://dx.doi.org/10.1016/S1726-4901\(09\)70111-X](http://dx.doi.org/10.1016/S1726-4901(09)70111-X)]
7. Yildirim S, Nursal TZ, Tarim A, et al. Colonic obstruction due to rectal endometriosis: Report of a case. *Turk J Gastroenterol* 2005;16(1):48-51.
8. Liang CC, Tsai CC, Chen TC, Soong YK. Management of perineal endometriosis. *Int J Gynaecol Obstet* 1996;53(3):261-265. [[http://dx.doi.org/10.1016/0020-7292\(95\)02592-8](http://dx.doi.org/10.1016/0020-7292(95)02592-8)]
9. Khetan N, Torkington J, Watkin A, Jamison MH, Humphreys WV. Endometriosis: Presentation to general surgeons. *Ann R Coll Surg Engl* 1999;81(4):255-259.
10. Liao H, Hsu T. Endometriosis inducing complete colon obstruction managed by one stage resection and anastomosis. *Journal of Society of Colon and Rectal Surgeons (Taiwan)* 2010;21:43-47.