

# Bowel Endometriosis: An Overview

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#### Abstract

Endometriosis is defined as the presence of ectopic endometrial gland or stroma outside the endometrial cavity. When endometriosis involves organs outside the pelvis is characterized as extragenital endometriosis. Bowl involvement with endometriosis is not a common occurrence, but when it happens it stimulates various symptoms of inflammation and sometime local neoplastic conditions which are rare occasion and may lead to malignant changes that have been discussed in this literature. Though it is a benign & estrogen dependent gynecological condition it has certain gastroenterological manifestations that are commonly seen in the various gastrointestinal disorders which misleads or delays the proper diagnosis and lengthens the suffering of the patient.

#### Introduction

**Review Article** 

Though endometriosis predominantly affects the pelvic organs but the extra genital Endometriosis may involve the gastrointestinal tract at any site and can present with solitary or multiple areas of isolated satellite lesion or as nodule (Figure 1) with a strong association of pelvic endometriosis showing 15-35% cases of true multifocal involvement [1]. Though existence of independent bowl endometriosis has also been reported. Bowel endometriosis can be used to indicate the presence of endometrium like gland or stroma which infiltrate the intestinal wall involving the subserous fatty tissue along with the subserous part of the enteric plexus.



Figure 1: Endometriotic nodule is shown by the asterisk.

Presence of endometriotic foci in the serosa of the intestine without the evidence of involvement of subserous layer should be considered as peritoneal endometriosis [2]. Prevalence of bowl endometriosis in the general population is unknown but the highest frequency of involvement as endometriotic nodule is noted on the rectum including the rectosigmoid segment for about 70-80% of cases then followed by the sigmoid colon, ileum, the appendix and the cecum. The small intestine shows a less frequency of involvement [3]. Gastric and transverse colonic endometriotic involvement has also been reported. 0.53% of 7200 cases of small intestinal endometriosis have been presented in the Mayo Clinic over last 20 years [3]. According to autopsy studies incidence of appendeceal endometriosis has been estimated to be 0.054% of the general population [3]. Intestinal endometriosis is most commonly found within the subserosa as superficial serosal implants but may also be found infiltrating all the layer of the bowel wall. Under cyclical hormonal influences, these implants may proliferate and infiltrate the intestinal wall and cause a fibrotic reaction with formation of strictures and adhesions, which may lead to bowel obstruction and recurrent abdominal pain [4].

## Histology



**Figure 2:** Section of a bowel endometriotic nodule, hemeatoxylin eosin staining demonstrates the nodule infiltrate the mucosa.



Figure 3: (Hemeatoxylin and eosin stain, low power view) showing endometrial glands within the muscularis propria of the terminal ileum.

Bowel endometriosis shows variety in extent typically involving the serosa and the muscularis propria rarely shows involvement to the submucosa and mucosa (Figure 2). The implants of the endometrial stroma are usually found in the anti-mesenteric edge of the bowel. While microscopically the endometrial glands & stroma are seen to invade the bowel wall from serosa inwards. The extent varies from microscopic foci to large space occupying lesions invading the bowel wall causing significant narrowing of the lumen. Mural thickening and stenosis may be produced by smooth muscle hyperplasia and fibrosis surrounding the endometriotic nodule in case of larger lesions invading the muscular layer (Figure 3) [3,5,6].

## Symptomology

Bowel symptoms are extremely common in patients with endometriosis. While 60% or more patients may have at least one symptom referable to their gastrointestinal tract though the exact percentage is difficult to pin down [7]. It remains unclear how bowel endometriosis causes intestinal symptoms. Endometriosis can cause bowel syndrome even when does not occur directly on the bowel. Inflammatory mediators released by the tissues by the response to inflammation include prostaglandin, tumor necrosis factor (TNF), interleukins and cytokines can affect the bowl and contribute to them [7]. They cause change with in the intestinal tissue by new vascularization, chemo taxis and scarring [7]. Diarrhea and intestinal cramping occurs due to increase smooth muscle contractility results by the release of Prostaglandin and likely other mediators that are released by the endometriosis implants and the uterus during menstruation. Occasionally, deep implants in adjacent structures such as the uterosacral ligaments or rectovaginal septum can also cause bowel symptoms. Large endometriotic nodules may contain extensive fibrosis and cause thickening of the bowel wall, resulting in a stenosis of the intestinal lumen. In addition they infiltrate and disrupt intestinal nervous plexus, damage interstitial cajal cells, reduce the density of intestinal sympathetic nerve fibers and thus, cause an alteration in bowel physiology [4,2]. Intestinal endometriotic lesions may have variable size and depth of infiltration in the bowel wall,

therefore causes a wide range of symptoms [2]. Small endometriotic lesions affecting only the subserosal fatty tissue do not cause symptoms. A wide range of symptoms including dyschezia, constipation, diarrhea, abdominal bloating, painful bowel movements, passage of mucus in the stools and cyclical rectal bleeding can be produced by larger nodules infiltrating the intestinal muscular layer [2,6,8,9]. The symptoms associated with intestinal endometriosis often mimic irritable bowel syndrome and Inflammatory bowel disease Crohn's Disease and Ulcerative Colitis. A part of the patients of endometriosis presenting with bowel symptoms may initially diagnosed as inflammatory bowel disease. Colonoscopic examination is usually required to confirm the diagnosis.

The extent of bowel involvement by endometriosis varies widely with a wide range of presenting symptoms. Disease limited to the bowel serosa may be asymptomatic [5]. The main manifestations are primary or secondary dysmenorrhea, bleeding disturbances, infertility, dysuria, pain on defecation (dyschezia), cycle-dependent or (later) cycle-independent pelvic pain, nonspecific cycle-associated gastrointestinal or urogenital symptoms. Most common in young and premenopausal women. With the presence of such manifestations, endometriosis should be considered in the differential diagnosis, and evidence for it should be sought. Patients presenting with mild or moderate symptoms, these symptoms become even more common. During their periods or pre-menstrually often these symptoms are more problematic. Women with symptoms similar to those of IBD but without any abnormalities on colonoscopy are often diagnosed with Irritable Bowel Syndrome (IBS). However after a series of GI tests a negative colonoscopy may be reassuring, indicating that endometriosis has not penetrated through the bowel wall. Unfortunately, bowel endometriosis may be an unexpected finding or may simply be overlooked if the possibility of bowel endometriosis was not fully entertained.

Bowel endometriosis includes following specific symptomatology: [3,2,7]

Painful defecation and tenesmus.

Cyclic hematochezia.

Change in bowel habits (diarrhea, constipation, hyperperistalsis, flatulence);

Rectal bleeding.

Rectal pain and painful defecation

Colonic endometriosis should be considered in women with chronic pelvic pain and intestinal bleeding, even if not cyclical with menstrual periods [10].

## **Diagnosis:**

Diagnosis of bowel endometriosis is still a clinical challenge as the clinical examination and the evaluation of the symptoms are inadequate for the accurate diagnosis of intestinal endometriosis. Although there is no gold standard universally accepted method but Magnetic resonance imaging (MRI) is one of the most commonly used techniques. Though Multidetector computerized tomography enteroclysis (MDCT-e) has recently been suggested for the diagnosis of bowel endometriosis [11,12]. On the other hand though the diagnostic accuracy of ultrasonography depends on the experience of the operator, several studies have demonstrated that rectosigmoid endometriosis can not only be accurately diagnose by transvaginal

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ultrasonography it also may detect the depth of infiltration of the nodule in the intestinal wall [8,13-16]. During transvaginal ultrasonography after a proper bowel preparation by adding water contrast to the rectum may facilitate the identification and characteristic evaluation of the endometriotic nodular lesion [17-19]. Rectal endoscopic ultrasonography has been widely used for the diagnosis of intestinal endometriosis [20-22]. This permits to estimation of the precise depth of infiltration of endometriosis lesion in the intestinal wall, the maximum diameter of the lesions and the distance of the lesions from the anus [22].



Figure 4: MDCT-e, the arrow shows an endometriotic nodule infiltrating the ileum.

Multidetector computerized tomography enteroclysis (MDCT-e) can identify the presence of endometriosis foci and also evaluate the characteristic, depth and infiltration of the nodule, by the presence of nodule adjacent to the bowl loop resulting in irregular intestinal outline the infiltration of the endometriosis can be characterized (Figure 4). Multilayered stratification of the thickened bowel wall is observer when the muscularis propria is infiltrated (Figure 5). Presence of a hypodense layer between the lumen and the muscularis propria indicate the full thickness infiltration of the bowel wall involving the submucosa and the mucosa [11,12]. Use of iodinated contrast medium and ionizing radiations is the major disadvantage of MDCT-e [2]



Figure 5: MDCT-e, coronal reconstruction. Endometriotic nodule infiltrating the muscularis propria of the sigmoid (shown by the arrowheads); the mucosa is not infiltrated.



**Figure 6:** Colonoscopy showing focal mucosal lesion with areas of irregular thickening.

Endoscopic diagnosis of intestinal endometriosis is difficult because the lesion hardly reaches the intestinal mucosa, though it can be evaluated by the presence of localized pathological foci, growth, presence of mucosal lesion (Figure 6) [23,24,10]. 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. As the biopsies from the suspected area do not contain submucosa and muscle layers, they do not allow for diagnosis [25]. A case reported by A.G.Macedo on 27*th* Feb. 2008 revels a 5 cm long laterally spreading lesion in the recto-sigmoid junction occupying half of its lumen. The lesion was characteristically friable in nature slightly harden with irregular surface. The histopathology of the biopsy sample obtained from the lesion confirms intestinal endometriosis [10]. Another study on diagnostic colonoscopy shows detection of multifocal lesion in the colonic mucosa revealing authentic signs of colorectal endometriosis which include polypoid growth over the endometriotic foci, endometrioid heterotopias in the colonic mucosa presence of ulceration in the mucosal membrane in the endometriotic projection and geoidal mucosa [23].

# **Intestinal obstruction**

Partial of complete intestinal obstruction can be produced by endometriosis by various manifestations like angulation of the bowl or localized endometriosis causing pressure over the lumen interrupting the flow or causing intussusceptions. Commonly observed phenomena is stenosis of bowl lumen (Figure 7) caused by smooth muscle hyperplasia and fibrosis by benign ectopic glandular tissue and localized repeated inflammation of the bowl wall due to cyclical hemorrhage [26]. Case reported by Unalp HR shows acute intestinal obstruction to a 45 year old lady due to ileal endometriosis (Histology reveals endometriosis with annular stricture showed foci of ectopic glandular tissue) where laparotomy evaluated an ulcerated, edematous and fragile segmental lesion above the iliocecal valve [27]. Case reported by A Alhumidi describes two free constricting mass in the sigmoid colon involving the serosal surface of the lumen causing symptoms to a 41 year old lady with 18 years of known history of endometriosis



**Figure 7:** Barium study showing constant narrowing (arrow head) in the sigmoid colon.

Presented with cyclical history of fresh bright red bleeding per rectum with abdominal pain associated with dyschezia. Colonoscopy reveals stricture in the sigmoid colon and barium study showed area of constant narrowing at the sigmoid colon.

## **Intestinal perforation**

As mostly the affected cases of bowel endometriosis the intestinal mucosa remains intact, a transmural intestinal wall involvement causing perforation of the intestinal tract is a very rare complication. 42.8% among these reported cases are occurred in pregnancy. Sigmoid colon is the most common site of perforation among the published case. Although endometriosis improves during pregnancy under the effect of progesterone the ectopic endometrium becomes decidualized with a progressive reduction in size [28,29].

A case reported by Adolfo Pisanu [Which is believed to be the first reported case of spontaneous rectal perforation from endometriosis in pregnancy] shows perforation of rectum in the 30th week of pregnancy where symptoms were suggestive of pyelonephritis, later evaluated as a 3 cm wide perforation of rectal wall which shows Histopathological findings were consistent with decidualization of the rectal wall. Intestinal wall becomes weaken by the reduction of the transmural endometriotic lesion under the influence of progesterone during pregnancy, particularly in the third semester. Perforation may also facilitate by progressive traction of the enlarged uterus on the strictly adherent loops of intestine [29]. Haufler described a jejunal perforation due to rupture of an endometriotic cyst during the sixth month of pregnancy in a 30 years old woman. In 1955, Henriksen briefly mentioned a case of sigmoid perforation in his series of 1000 cases of endometriosis. Clement in 1977, Rud in 1979, and, most recently, Schweitzer also reported similar cases of sigmoid colonic perforation secondary to endometriosis during pregnancy [3,30-32]. Colonic perforation by endometriosis generally presents with asymptomatic or painful pelvic mass (Figure 8). A case reported by Neeraj Kumar Garg [30,31] shows a perforation of sigmoid colon by a large endometriotic mass arising from the left fallopian tube and ovary, which had adhered to the sigmoid colon in a previously fit 44years old patient with previous known history of mild endometriosis (Figure 9). Presented with pyrexia, 10 days history of worsening colicky pain in the left flank and iliac fossa with a mass.

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abdominal cavity; B: Pelvic abscess at the right side of the enlarged uterus

# Endometriosis involving the appendix





## **Inflammatory Bowl Disease**

A study report published by Tine Jess on unselected nationwide Danish cohort of 37,661 women hospitalized with endometriosis during 1977–2007 shows that the risk of IBD in women with endometriosis was increased even in the long term, hence suggesting a genuine association between the diseases. Women with endometriosis had a increased risk of Inflammatory Bowel Disease overall (Standardized Incidence Ratio=1.5; 95% CI 1.4 to 1.7) and of Ulcerative Colitis (Standardized Incidence Ratio=1.5; 95% CI 1.3 to 1.7) and Crohn's Disease (Standardized Incidence Ratio=1.6; 95% CI 1.3 to 2.0) separately, even 20 years after a diagnosis of endometriosis (Ulcerative Colitis: Standardized Incidence Ratio=1.5; 95% CI 1.1 to 2.1; Crohn's Disease: Standardized Incidence Ratio=1.8; 95% CI 1.1 to 3.2). Restricting analyses to women with surgically verified endometriosis suggested even stronger associations (Ulcerative Colitis: Standardized Incidence Ratio=1.8; 95% CI 1.2 to 2.3; Crohn's Disease: Standardized Incidence Ratio=1.7; 95% CI 1.4 to 2.3; Crohn's Disease: Standardized Incidence Ratio=1.7; 95% CI 1.2 to 2.5). [33]



Figure 10: Gross appearance of crohn's ilitis with multiple fistulas.

Zafer Teke reported a case of complicated Crohn's disease clinically mimicking ileal endometriosis in a 31 year old woman due to her premenstrual symptoms of mechanical subileus for 3 years; at first monthly, but later continuous, and gradually increasing in severity. A right hemicolectomy and partial distal ileum resection were performed but histopathology revealed Crohn's disease without endometrial tissue. The differential diagnosis of Crohn's disease with intestinal endometriosis is difficult as they share overlapping clinical, radiological and pathological features. They both are characterized grossly by patchy involvement of small intestine with intervening uninvolved skip areas. Moreover transmural process with chronic inflammatory changes results in formation of strictures, adhesion, mucosal thickening, mural fibrosis and obstruction and shares common manifestation such as perianal abscess or fistulas (Figure 10) and inflammatory pseudo tumor involving the cecum terminal ilium and the appendix. In rare case endometriosis may cause deep fissures even fistulous tracts, further mimicking regional enteritis [33].

## Malignant transformation

The phenomenon of malignant transformation of endometriosis was first described by Sampson in 1925. The gastrointestinal tract involvements were classified as endometriosis associated intestinal tumors (EAITs). Development of a malignancy is a relatively common complication of endometriosis and may occur in up to 5.5% of female patients with endometriosis and malignant transformation of

gastrointestinal endometriosis with out pelvic involvement is uncommon [34]. However malignant transformation of the intestinal tract not been reported as frequently [35]. An exogenous estrogen is the widely accepted factor for the development of premalignant or malignant transformation of endometriosis [36,37]. Endometrioid carcinoma is a common histologic type confuse with colorectal carcinoma. Colon carcinomas always involve the mucosa and in advanced case and extend through the bowl wall in to serosa or adjacent fat. While endometriosis shows an opposite extramural growth pattern invading in to the bowl mucosa from the serosa [38,39]. The colon carcinoma can be differentiated from the endometrioid adenocarcinoma by the presence of a background of benign endometriosis [40]. As practically accurate diagnosis is difficult so for confirmation of immunochemistry is the recommended technique for all cases of EAITs [41,42]. Nine cases of primary EAITs arising in the rectosigmoid colon in patients using unopposed estrogen for several years prior to the development of carcinoma was reported by Jones where they concluded that their findings supported a casual, cancer causing roll of unopposed estrogen [43].

Case reported by Joaquin Marchena-Gomez described a 45 year old lady with past medical history of ovarian endometriosis treated with bilateral oophorectomy received treatment with medroxyprogesterone and transdermal estrogens presented with paroxistic abdominal pain and vomiting. Intestinal obstruction was confirmed by an X-ray and laparotomy revealed an ileal mass protruding in to the lumen where morphologic and immunohistochemical features were typical of an endometrioid carcinoma. Several endometriosis foci were identified adjacent to the mass. The prognosis for estrogen-stimulated EAIT arising in any gastrointestinal site is highly favorable with a survival rate of 82 per cent at 5 years.

Another case reported in Red Orbit in Sept 2005 describes a primary endometrioid carcinoma arising in the rectal wall endometriosis in a 60 year old woman presented with hematochezia with history of extensive endometriosis and total abdominal hysterectomy with bilateral salpingo-oophorectomy 10 years earlier. She underwent colonoscopy which revealed a 3.5 to 4 cm sessile polyp in the anterior rectal wall which was partially excised. Pathologic specimens of hysterectomy and oophorectomy specimen was reexamined and found to contain benign endometriosis.

## Management

Management of patient with bowel endometriosis depends on the patient's symptoms, size of the nodule and the degree of luminal stenosis. Large endometriotic lesion causing symptoms can be treated by hormonal therapy or surgery or both. In case of uncomplicated intestinal endometriosis treatment depend patient's age and intention to conceive, If malignancy cannot be excluded with symptoms of obstruction or bleeding in case of child bearing age resection of the involved bowl followed by hormonal therapy is the choice otherwise hysterectomy with bilateral oophorectomy is indicated and with a past history of endometriosis or co-existent gynecological symptoms a laparoscopy prior to laparotomy should be considered [30,31]. Though in case of estimated bowl stenosis <60% hormonal therapy can be safely offered but because of ovulation inhibition, patients willing to conceive are not good candidates for this therapy [2]. Options for pharmacotherapy include Gestagens, Oral contraceptive drugs, GnRH analogues and analgesics [44]. Progestins are most commonly used which is in general well tolerated and effectively reduce the severity of symptoms caused by pelvic endometriosis. More

recently, aromatase inhibitors have been suggested for the treatment of endometriosis [2]. Colorectal endometriotic nodules infiltrating the intestinal muscularis propria can be excised by either by partial or full thickness nodulectomy or segmental bowel resection by laparoscopy or laparotomy, though women with colorectal endometriosis, segmental bowel resection improves pain, intestinal symptoms and quality of life [45,46]. However nodulectomy may have the advantage of a lower incidence of postoperative unpleasant functional digestive outcomes compares with the segmental bowl resection [47,48]. Several studies have showed that good pregnancy rates are achieved after surgical excision of intestinal endometriosis. A study of 288 women wishing to conceive demonstrated that nodulectomy is associated with an 84% pregnancy rate obtained either naturally or after assisted reproductive technologies, after a median follow-up of 3.1 years [2].

## Conclusion

A wide spectrum of disease may mimic clinically and pathologically with intestinal endometriosis including infectious etiology, ischemic enteritis/colitis, inflammatory bowel disease, and neoplasm's [48]. Postmenopausal patients may predominantly affect by EAIT though diagnostically they are challenging as they clinically and pathologically resemble common primary neoplasm's of the gastrointestinal tract. In summary a case of intestinal endometriosis should always suspect in a female patient suffering of chronic abdominal & bowl symptoms presenting in cyclical or non-cyclical pattern, in differential diagnosis before starting the treatment and where usual application of treatment has already failed to improve the symptoms. Strong consideration should be undertaken for the patients with treated history of endometriosis specially those received unopposed estrogen hormone replacement and currently presenting with abdominal symptoms or rectal bleeding.

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