Acute intestinal obstruction caused by endometriosis mimicking sigmoid carcinoma

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Abstract

Endometriosis is a relatively frequent disease in fertile women. The intestine is involved in 12-37% of cases. Intestinal endometriosis is usually asymptomatic and complete obstruction of the bowel lumen occurs in less than 1% of cases. We report a case of endometriosis of the sigmoid, which caused complete intestinal obstruction and mimicked carcinoma of the sigmoid colon. This case demonstrates the difficulty of establishing an accurate pre- and peroperative diagnosis and the propensity of intestinal endometriosis to mimic colon cancer. (Acta gastroenterol. belg., **1998**, 61, **376-378**).

Key words : endometriosis, colon carcinoma, intestinal obstruction.

Introduction

Endometriosis, the ectopic existence of endometrial tissue, is found in 4-17% of fertile women and is most often located in the ovaries, uterus and the pouch of Douglas (1,2). Less often, in 12-37% of cases, the gastrointestinal tract is involved (2,3). In most cases intestinal endometriosis is asymptomatic and an incidental finding during laparotomy for other diseases (2). Rarely, deeper and more extensive infiltration of the intestinal wall results in obstruction, perforation or bleeding and requires differentiation from malignancy. Complete intestinal obstruction is seen in less than 1% of cases of endometriosis of the bowel (2-4). We report a patient with an acute complete bowel obstruction by sigmoid endometriosis, mimicking sigmoid carcinoma.

Case report

A 45-year-old woman presented with a 3-day long history of diffuse abdominal pain and distension of the abdomen. For the last 17 days she had not passed stool or flatus. She gave no history of weight loss or rectal blood loss per rectum. Other gastrointestinal or gynecologic symptoms were absent. She had had her appendix removed 6 years previously. Physical examination revealed normal vital signs, abdominal distension, tinkly bowel sounds and diffuse abdominal tenderness without guarding. No abdominal mass was evident. Rectal and vaginal examination were normal. The rectal ampulla was empty. Blood examination showed leukocytosis and slightly elevated liver function tests. Plain abdominal x-rays showed the large bowel distended to the level of the sigmoid, suggestive of sigmoid obstruction. Endoscopy demonstrated a total obstruction of the sigmoid with severe oedema of the bowel wall. No biopsies were taken during endoscopy, since prolonged complete obstruction of the sigmoid had made urgent laparotomy necessary. A tumour of the sigmoid with complete obstruction of the bowel lumen and partial necrosis of the bowel wall was found, suggestive of sigmoid carcinoma, and a Hartmann-procedure was performed. At the origin of the inferior mesenteric artery two large lymph nodes were found and were removed with the specimen. Inspection of the peritoneal cavity did not reveal other abnormalities. The postoperative course was uneventful. Pathology reported the tumour as endometriosis of the sigmoid. In the area of the stenosis benign endometrial glands of different size and cellular stroma were found in submucosa, muscularis propria and serosa (fig. 1 and 2).

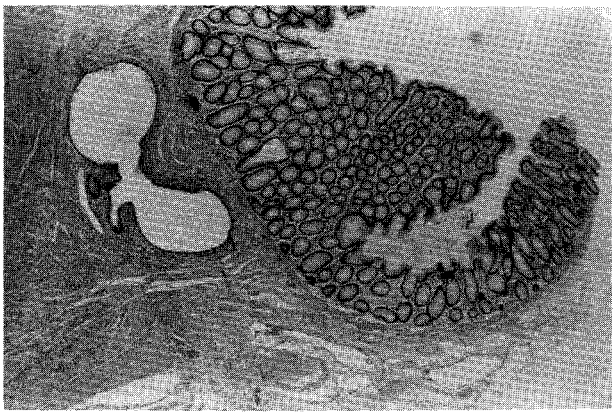

Fig. 1. — Endometriosis of the large bowel. Endometrial glands and stroma in the submucosa and the muscularis propria (H-E × 25).

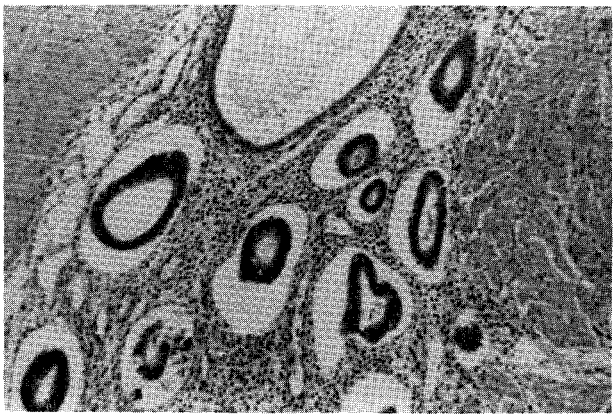

Fig. 2. — Endometriosis of the large bowel. Endometrial glands and stroma in the muscularis propria (H-E × 160).

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Locally the glands were cystic dilated and micro-hemorrhages, hemosiderine containing macrophages and fibrosis were seen in the stroma. The muscularis propria was hypertrophic and in the serosa a fibroplastic reaction with inflammatory infiltration was seen. The mucosa was normal.

Six months later, colonoscopy and endoscopy of the rectal stump were normal. The patient refused the advised hysterectomy with bilateral oophorectomy. The colostomy was closed and the intestinal continuity restored. Inspection of the peritoneal cavity revealed no macroscopic endometriosis. The postoperative course was uneventful. Two years after her second operation she remains in good condition, without any complaints.

Discussion

Various theories for the development of endometriosis exist, but the implantation of endometrial tissue in the peritoneal cavity after retrograde menstruation is the most probable explanation (1). The rectosigmoid is involved in 88-95% of cases of intestinal endometriosis and the ileum, cecum and appendix less frequently (2,4). It most often affects fertile women between 40 and 50 years of age (5-8). As usually only the serosa is involved, it is often asymptomatic and of no clinical importance. Cyclic hormonal influence results in deeper infiltration in the intestinal wall, but the mucosa is rarely reached. This explains why intestinal bleeding is a rare and late clinical feature (9). Fibrosis and marked smooth muscle hyperplasia characterize the response of the intestinal wall to the invading endometrial tissue and the cyclic bleeding, resulting in stenosis and obstruction of the intestinal lumen. Obstruction can also be caused by adhesions, volvulus and intussusception (5). Complete obstruction of the intestinal lumen occurs in less than 1% of cases (2-4). The invasive type of endometriosis is difficult to differentiate clinically from a malignant tumour (3,5,10-13). Malignant degeneration of ectopic endometrial tissue is a rare complication (14). Recurring crampy lower abdominal pain with cyclic frequency is the most common symptom of intestinal endometriosis. Other symptoms include constipation, diarrhea, rectal pain, rectal discomfort during defecation, tenesmus, stools of small calibre, abdominal distension and episodic bloody stools (2,4-7,15). The cyclic appearance or worsening of intestinal symptoms coincidental with menstruation is characteristic, but present in only 40% of cases (15). Since many women also have pelvic endometriosis, gynecologic symptoms, including dysmenorrhea, dyspareunia, menorrhagia, metrorrhagia and infertility, are often present (2,4-7,15).

Rectal and vaginal examination may demonstrate characteristic tender nodularity, induration and thickening of the uterosacral ligaments or of the Douglas pouch (5,15). Enteroclysis is the preferred method of investigating the small intestine. Double-contrast enema usually demonstrates an extrinsic mass with intact mucosa, but malignancy cannot conclusively be ruled out (5-7). Colonoscopy may demonstrate stenosis and sometimes a bluish submucosal discoloration. Usually, the mucosa is intact without ulcerations. Endoscopic biopsies are usually not helpful because the lesions rarely affect the mucosa (6-8). Endorectal and endovaginal sonography may be helpful in establishing the diagnosis, in evaluating its extent and demonstrating the infiltration depth of endometriomas, with a view to patients for superficial excision (13,16). Computed tomography, magnetic resonance imaging and sonography may give additional information about the exact location and extent of the disease. Diagnostic laparoscopy is very helpful, allowing evaluation of the extent of endometriosis and the taking of biopsies.

The diagnosis of intestinal endometriosis is often not established preoperatively (5,7,11,13). Intestinal endometriosis may mimic a wide variety of inflammatory, infectious and neoplastic digestive diseases (14). The presence of endometriosis elsewhere may be helpful, but endometriosis and colon carcinoma may coexist (3,6,7). Frozen section examination may avoid unnecessary radical surgery.

The treatment of patients with intestinal endometriosis depends on the extent of the disease and the patient's menstrual status, age and desire for future pregnancy. Asymptomatic patients and symptomatic patients with small lesions may be treated initially by hormonal therapy, if malignancy can be ruled out. Medical options for the treatment of gastrointestinal endometriosis mirror those available for pelvic endometriosis, and include danazol, progestational agents, and gonadotropin-releasing hormone (GnRH) analogues (17). Hormonal therapy with these drugs, inducing pseudomenopause, has been shown anecdotally to yield good responses in patients with small lesions, although discontinuation often leads to recurrent symptoms and this therapy has not been effective in intestinal endometriosis with extensive fibrosis (4,6-8,10,13,15,18). When asymptomatic patients are operated for another reason, excision of the lesions should be performed. In asymptomatic patients and symptomatic patients failing to respond to medical therapy, small superficial lesions may be excised and intestinal resection may be avoided if malignancy is ruled out (3). The treatment of choice for extensive intestinal endometriosis is resection of the involved bowel segment, with hysterectomy and bilateral oophorectomy (2,3,5,12,19). For women who desire to become pregnant, resection of the involved intestine along with excision of pelvic endometriosis may be appropriate treatment. Baily *et al.* (4) documented no instances of recurrent colorectal endometriosis in 68 patients undergoing fertility-preserving operations for intestinal endometriosis. However, the results of conservative surgery are clearly inferior to those achieved by a more aggressive surgical approach. When only endometriosis has been resected, subsequent operations for endometriosis will be necessary in approximately 20% of cases, while no such operation will be needed

when hysterectomy with bilateral oophorectomy has been performed additionally for endometriosis (2,20). Following ovary salvage procedures in premenopausal women with intestinal endometriosis only 54% remain free of symptoms, as compared to 78% when bilateral oophorectomy is performed (4). Recently, laparoscopic resection of intestinal endometriosis has been performed successfully (20,21). Postoperative medical treatment may be indicated only in patients with severe refractory pelvic pain and in whom disease has not been completely extirpated (18,20). Hormonal substitution therapy to avoid menopausal symptoms after bilateral oophorectomy is not advised, since this may stimulate any residual endometrial tissue to proliferate and has the risk of malignant transformation (3,12,13).

This case demonstrates the difficulty of establishing an accurate pre- and peroperative diagnosis and the propensity of intestinal endometriosis to mimic colon cancer. Although intestinal endometriosis is not uncommon, it is a very rare cause of complete obstruction of the colon.

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