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ntestinal ndometriosis

Constantine A. Samaras, M.D., M.Ph.
Robert B. Greenblatt, M.D.
Farr Nezhat, M.D.

Fig. 1 Endometriosis of the sigmoid colon, one of the most commonly involved sites.

INTRODUCTION

Endometriosis is the occurrence of functioning endometrial tissue in ectopic sites and remains an enigma to the gynecologist and pelvic surgeon. It has been regarded as a product of modern lifestyle, delayed child bearing, family limitation, and sexual excitation without orgasm[1]. Three current theories are extant as to its cause: 1) retrograde flow at menstruation,[2,3] 2) lymphatic or hematogenic spread from the uterus,[4] 3) the embryologic theory: aberrant endometrial tissue resulting from serosal metaplasia.[5] Ectopic endometrium is invasive to the surrounding tissues, shows a response to the cyclic hormonal variations of the menstrual cycle, and at menstruation may rupture and bleed. It also induces a desmoplastic reaction that compromises the blood supply and produces dense fibrous adhesions with surrounding structures. The frequency of endometriosis varies in from 4-17% of menstruating women,[6] and it appears that there is a hereditary tendency in certain families.[7] Endometriosis has been regarded as the most malignant of the benign gynecological diseases.

The most commonly involved sites are the pelvis (ovaries, uterus, Fallopian tubes, uterosacral ligaments, rectovaginal septum, and pelvic peritoneum), the urinary tract (bladder and ureters), and the gastrointestinal tract (appendix, small bowel, sigmoid colon, rectum) as shown in Fig. 1. Instances of endometriosis involving the umbilicus, inguinal region, abdominal and perineal scars, and pleura are on record. Occasionally the appendix is the site of endometriosis, without evidence of other pelvic lesions (Fig. 2).

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Dr. Constantine A. Samaras is senior clinical research fellow. Dr. Farr Nezhat is clinical research fellow. Dr. Robert B. Greenblatt is professor emeritus of endocrinology, Medical College of Georgia School of Medicine, Department of Endocrinology, Augusta, Georgia.

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FREQUENCY — SITES — APPEARANCE

Over the past 40 years it has become clear that 8-15% of all menstruating women will develop endometriosis,[8] and that 3%[9] to 35%[10] of these will have bowel involvement. The most commonly affected gastrointestinal sites are clearly illustrated in a recent study done by Williams et al[11] (Table I). In the majority of the studies, endometriosis was found to involve organs with close proximity to the uterus and adnexa, which explains the high frequency of involvement of the rectosigmoid, cecum, appendix, and terminal ileum. More recent reports have described jejunal involvement and anal canal lesions.[12] Colonic involvement of the rectosigmoid usually occurs in about 25% of females with endometriosis.[13] Appendiceal endometriosis found in 3% of the cases of pelvic endometriosis is usually associated with right adnexal disease and is often misdiagnosed as acute appendicitis. The macroscopic appearance of endometriotic lesions is that of serosal nodules or plaques, and stricture formation with intestinal obstruction or endometrioma formation.

Any endometriotic deposit, given a good blood supply, will cycle the same way the uterine endometrium does. Active endometriotic areas will provoke severe reaction from the neighboring tissues during monthly menses or form endometriotic cysts, which on rupture produce marked fibrosis. If the developed fibrosis greatly impedes the blood supply, the deposit will cease to cycle and will become dormant. Thus, the periodicity of the symptoms will disappear and obstructive symptoms will develop as a result of the fibrosis.

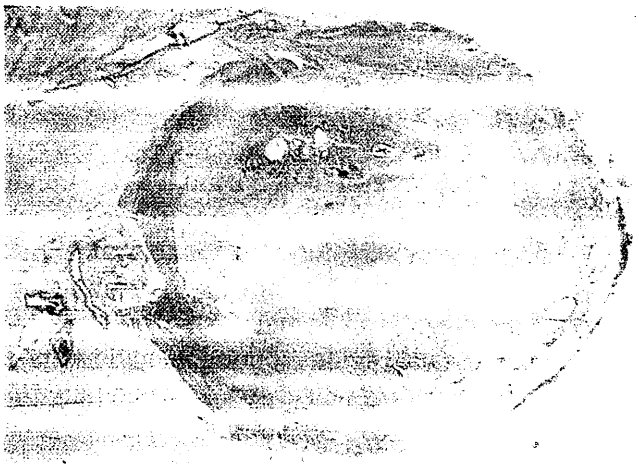

Fig. 2 Sagittal section of the appendix. Endometriotic glands can be clearly seen in the fibromuscular appendiceal wall.

SYMPTOMS

Intestinal endometriosis can present a wide variety of symptoms or may be symptomless and recognized only at laparotomy. However, symptoms do not always correspond to the severity of the disease. Mild endometriosis may cause more pain, dysmenorrhea, or deep dyspareunia than more advanced endometriosis that is insulated by surrounding scar tissue. Menstruation always aggravates the existing symptoms. Endometriosis has never been reported in premenarcheal girls or in men. Klug[14] reports that a correct preoperative diagnosis of endometriosis is reached in only 10% of cases. A common feature of intestinal endometriosis is the chronicity and periodicity of symptoms. Jenkinson et al[8] found an average duration of symptoms of 3.3 years in his case of incomplete bowel obstruction. Also bright rectal bleeding occurring simultaneously with menses is considered almost pathognomonic of endometriosis.[15] However, it should be kept in mind that sometimes rectal bleeding occurs in the absence of mucosal lesions.[16] Periodicity of any gastrointestinal symptom like constipation, diarrhea, or dyspareunia should alert the physician.

TABLE I

Sites of involvement of endometriosis in 485 women, 15-55 years old, undergoing laparotomy (1965-1969)

Site	No
Gastrointestinal tract	181+
Rectosigmoid	172
Ileum	9
Appendix	19
Segment of bowel resected	7
Colostomy	0

*Data from Williams, T.J., Pratt, J.H.: Endometriosis in 100 consecutive celiotomies: Incidence and management. Am Obstet Gynecol 129:245, 1977.

+Multiple areas may be affected in one patient.

diagnosis of endometriosis. Unfortunately, ch symptoms are present in only half of the ses. Infertility is said to be present in 50% of strointestinal endometriosis cases. Menstrual in and irregularities are also commonly present most cases but are not considered of great gnostic value. Tenderness and fixation of the dometriotic area is an excellent diagnostic sign is present in nearly all cases.

ABORATORY TESTS

e three commonly used tests for the diagnosis gastrointestinal endometriosis are endoscopy proctoscopy with biopsy and x-rays. In all the dies reviewed, endoscopy or proctoscopy rarely nfirmed the suspected diagnosis of en- metriosis; the only information obtained was at of the presence of an extramucosal mass. In any cases the procedure had to be discontinued ecause of discomfort to the patient. The same ap- ies to endoscopic biopsy; rarely can endometrial ands be seen microscopically in bowel mucosa.[17] arium enema, the most commonly employed x- y investigation, may demonstrate various grees of asymmetric constriction of filling defect the lumen due to an extramucosal lesion. owever, neither is a pathognomonic symptom of dometriosis but should cause suspicion if it oc- rs in a woman of childbearing age who has enstrual dysfunction.

REATMENT

rgical resection of the lesion, castration, and arious hormone regimes have been used in the eatment of colonic or generalized endometriosis, cording to the age, parity, and endocrine status the patient. Hormonal therapy with estrogen- ogestogen preparations was used by Kistner[18] with 80% symptom relief. Recently, Danazol, an itigonadotropic agent with mild androgenic operties, has been reported to produce signifi- nt improvement.[19,20] However, it seems man- tory that surgical excision of the involved co- should generally be performed for the follow- reasons: Radiologic findings of endometriosis rongly resemble carcinoma, thus, it seems ex- emely difficult to exclude malignancy operatively. Additionally, tissue obtained after doscopic biopsy could never establish a firm operative diagnosis. Bowel resection, or colec- y and excisional biopsy as described by Gray[21] ems to be the best therapeutic approach for iptomatic colonic lesions, accompanied by rgical castration, especially in women who do not need to preserve their fertility. However, in women who want to keep their reproductive potential intact, one or both ovaries are preserved. The hormonal agents mentioned previously are recommended as an additional treatment modality for intestinal endometriosis, but rarely as the primary one. Asymptomatic serosal lesions require only confirmation biopsy at laparotomy although Gray[21] reported excellent results from localized incision. Hormone replacement therapy in younger patients who have undergone surgical castration to eliminate menopausal symptoms should not be undertaken for at least three months following surgery.

CASE REPORT

In August 1971 a 44-year-old Caucasian female, gravida 1, para 1, visited the clinic complaining of migrainoid headaches. Physical examination and blood chemistry were normal. We had found that sustained levels of estrogen, with or without testosterone, often held such headaches in abeyance for five to six months. Accordingly, one estradiol and two testosterone pellets were implanted subcutaneously. A course of norethindrone 10mg was given for seven days each month to assure regular withdrawal menstrual periods. She remained on the same therapeutic regimen at six month intervals for the next nine years with satisfactory results.

In July 1981 the patient reported that she had experienced diarrhea for the last five months that occurred after the course of norethindrone. Physical examination revealed a mass 5x6cm in size in the right iliac fossa. She was admitted to the hospital a week later and underwent a complete preoperative investigation. An upper GI series was normal and a barium enema revealed a presacral colonic mass. An IVP showed a mass pressing on the bladder superiorly and a pelvic sonogram confirmed the presence of a lobulated mass, possibly of ovarian origin. A liver and spleen radioisotope scan was normal, but proctoscopy revealed a hard, firm extraluminar mass at the rectosigmoid junction with intact bowel mucosa. Three biopsies were taken during the procedure. The rest of the investigation was normal.

The patient then underwent exploratory laparotomy and the mass was visualized in the anterior rectal wall adherent to the uterosacral ligaments. On palpation it felt like a sclerotic carcinoma. A clinical diagnosis of carcinoma was made and the patient underwent resection of the

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tumor by Wertheim hysterectomy with node dissection, salpingo-oophorectomy with upper vaginectomy, and mobilization of the left colon and splenic flexure with lower anterior anastomosis.

Postoperatively the diagnosis of endometriosis was established (Fig. 3), and the patient placed on Danazol, one 200mg tablet daily for six months. She did well and no recurrence of endometriosis is apparent.

Fig. 3 Endometriotic glands of various size in the resected bowel specimen.

DISCUSSION

Gastrointestinal endometriosis is not as rare a disease as thought in the past. The recent advances in endoscopic procedures and the alertness of the gynecologist has raised the incidence of pelvic endometriosis to a level of 50% in women undergoing laparotomy for pelvic pain, etc.[9] The lower gastrointestinal tract is the second most commonly affected site because of its proximity to the reproductive system, and because it fulfills two mandatory conditions for successful endometrial implantation: an uninfected recipient site (that explains why endometriosis is rarely encountered in the vagina), and a high level of serum and tissue estrogens during early growth.[22] However, the most outstanding characteristics of intestinal endometriosis is its close resemblance symptomatically, radiographically, and microscopically to carcinoma. Nevertheless, some differences do exist. In carcinoma, the lesion usually originates in the epithelial surface and gradually grows into the adjacent fibromuscular wall while endometriosis grows from the opposite direction. It starts on the serosal surface and then grows inward into the adjacent fibromuscular structures, rarely reaching the mucosa. The commonly used laboratory tests seldom offer a clue to the diagnosis. In radiology, particularly the differential diagnosis of a filling defect or stricture includes: primary carcinoma, secondary metastasis, pelvic abscess, diverticulitis, inflammatory bowel disease, ischemic stricture, amebiasis, radiation colitis and benign polyps. When radiography demonstrates that the lesion is extramucosal, the possibility of endometriosis in a premenopausal woman with chronic menstrual complaints is great. Malignant transformation of colonic endometrial implants are nearly nonexistant; however, there is a case report in the literature by Reintoft et al[23] describing a rectal carcinoma arising from endometrial implants simultaneously with a granulosa cell tumor of the ovary.

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