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Endometriotic lesions mimicking advanced ovarian cancer - A case report and a review of the literature

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Summary

Background: Approximately 2-5% of women affected by endometriosis are postmenopausal. The disease may simulate various malignancies. A case of endometriosis in a postmenopausal woman, spreading similarly to ovarian cancer, is reported. A broad review of existing literature on postmenopausal endometriosis, endometriosis involving the urinary tract, and the links between endometriosis and cancer are also presented. *Case description:* A 51-year-old woman was diagnosed with a pelvic mass. She complained of pain in the lower abdomen that began three weeks prior, and had no history of dysmenorrhea, acyclic pelvic pain, or infertility. CT scan revealed a solid and cystic tumor in the region of the right adnexa, infiltrating the surrounding tissues, with possible infiltration of the urinary bladder, as well as soft-tissue lesions of the small intestinal mesentery. Bilateral hydronephrosis and distension of the ureters were also present. A malignant neoplasm of the ovary or the corpus uteri was suspected. Total abdominal hysterectomy and appendectomy were performed. A superficial infiltration of the urinary bladder was also excised. The pathology report revealed endometriotic foci in the tumor and in the bladder infiltration, as well as an endometriotic cyst in the right ovary. Two years postoperatively the patient is disease-free and in good condition. *Conclusions:* Clinicians should remain conscious of the possibility of endometriosis mimicking advanced ovarian cancer and infiltrating the peritoneum and internal organs of the abdominal cavity, including the urinary tract. Imaging techniques, including CT and MRI, are not always effective in establishing the correct diagnosis preoperatively.

Key words: Endometriosis; Postmenopause; Ovarian neoplasms.

Introduction

Endometriosis is a benign, estrogen-dependent disease, with an estimated incidence of 6-10% of women globally [1]. The vast majority of the affected patients are women of reproductive age but 2-5% are postmenopausal [2]. The currently available high-quality data on postmenopausal endometriosis are limited. However, many cases of endometriosis both, ovarian and localized in other regions, e.g. the intestines and the urinary tract, have been reported in postmenopausal women [3-6]. When disseminated, the disease may sometimes simulate malignancies, such as advanced ovarian cancer, colorectal cancer, or carcinomatous infiltration of the ureter. Diagnostic confusion is particularly probable when symptoms of bowel obstruction or hydronephrosis occur. Due to the advanced age of those patients, the possibility of a malignant lesion may seem even more likely. Differential diagnosis is especially difficult in cases with no history of dysmenorrhea and/or infertility.

The authors present a case of endometriosis in a postmenopausal woman, spreading in a manner similar to ovarian cancer and infiltrating the tissues adjacent to the uterus and adnexa, with ureteral involvement. The literature has been reviewed in order to present the existing data on postmenopausal endometriosis, endometriosis involving the urinary tract, and the links between endometriosis and cancer.

Case Report

A 51-year-old woman was admitted to the Gynecology Ward of the First Chair and Department of Obstetrics and Gynecology, Medical University of Warsaw, in May 2013 due to a tumor of the pelvis. Obstetric history included two vaginal deliveries and no miscarriages. Since 2000 the patient took combined oral contraceptives (ethinyl estradiol + levonorgestrel) for approximately seven years. Apart from the tumor, she had been diagnosed with gastric ulcers and depression. She had undergone no previous surgeries. She reached menarche at the age of 15 and her age at menopause was difficult to establish since she received hormone therapy (estradiol valerate + norgestrel) before she reached menopause, from 2007 to April 2013, when the tumor was diagnosed. Her BMI was 23.4. She did not have any history of dysmenorrhea, acyclic pelvic pain, dyschezia or infertility.

On admission, the patient was in good overall condition. She complained of intermittent, sharp pain in the lower abdomen, radiating to the spine, that began three weeks prior and became more severe with time. Previously, no symptoms were present. CA-125 level was 77 U/ml. The abnormalities revealed in the CT scan of the abdomen included a solid and cystic tumor localized in the region of the right adnexa, measuring 90×60×85 mm and infiltrating the surrounding fat tissue. Infiltration of the urinary bladder could not be excluded. Soft-tissue lesions of the small intestinal mesentery, measuring up to 10×7 mm, were found in the surroundings of the tumor (implants or lymph nodes). Free fluid was discovered in the peritoneal cavity, between the intestinal loops. The upper and medial portions of both ureters were distended up to 11 mm on the right and up to eight mm on the left side. Inflammatory lesions were present on the

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thickened walls of both ureters. Hydronephrosis was observed on both sides.

On pelvic examination, the following findings were found: normal vagina and vaginal walls. The cervix was normal, both in the speculum and on bimanual examination. The uterus was anteflexed and slightly enlarged. Behind the uterus, contiguous to the sacrum, an immobile mass, eight cm in diameter, was palpable. The tumor was also palpable on rectal examination, high in the right parametrium.

Patient blood count was within the normal range. Her creatinine and urea levels were 0.4 mg/dl and 12.0 mg/dl, respectively. Chest radiogram and electrocardiogram were normal.

A malignant neoplasm of the ovary or the corpus uteri was suspected and the patient was scheduled for surgery. Laparotomy revealed the following: corpus uteri was round and measured 8-9 cm; the left adnexa were changed into a cystic-shaped structure, 3-4 cm in diameter. On the right side of the uterus, there was a pathological tissue conglomerate (tumor), involving the right ovary, the right parametrium, the distal part of the appendix, and the right wall of the uterus. The right ureter was distended, 6-7 mm wide, from the level of the infiltration of the right parametrium. Pelvic and abdominal peritoneum, the liver and other internal organs were macroscopically normal and normal on palpation. Total abdominal hysterectomy and appendectomy were performed. The tumor was resected 'en bloc' with the uterus. An urologist joined the operators and decided against resection of the infiltrated part of the ureter. Small superficial infiltration of the urinary bladder was also excised. The result of the intraoperative histopathological examination was a benign lesion. Due to intraoperative bleeding, both iliac internal arteries were ligated. After surgery, anemia was diagnosed (hemoglobin level 8.0 mg/dl). The postoperative period was otherwise unremarkable and the patient was discharged seven days after surgery.

The pathology report included the following information: both, the tumor and the infiltration of the urinary bladder are composed of connective fibrous tissue and adipose tissue with endometriotic foci. The corpus uteri and the cervix are free from neoplasm and endometriosis. The right ovary: an endometriotic cyst two cm in diameter, corpora albicantia, and serous cysts. The fallopian tube is normal. The left ovary: inclusion cysts and corpora albicantia. Ectasia oviducti.

Results of serum creatinine level testing and urinalysis performed within a year after surgery were normal. Ultrasound and CT scans of the abdomen performed after the surgery (one and 1.5 years postoperatively, respectively) revealed neither recurrence of the tumor nor distention of the ureters. At the moment, two years postoperatively, the patient is in good condition and has no symptoms of disease recurrence.

Discussion

A case of extensive endometriosis, involving the ureter and mimicking disseminated ovarian cancer, in a postmenopausal patient is reported. Despite the fact that the disease is generally perceived as a premenopausal condition, whose clinical manifestations resolve after the last menstruation, it has been shown that 2-5% of women with proven endometriosis are postmenopausal [2]. In a recently published retrospective study performed by Haas *et al.*, 2.55% of all patients admitted to the hospital for treatment of endometriosis were postmenopausal [7].

The presented patient had no history suggesting that she had been suffering from endometriosis before menopause (no dysmenorrhea, chronic pelvic pain, dyschezia or infertility). However, Houston *et al.*, reported the rate of endometriosis to reach 0.5-5% among fertile women [8]. According to yet another report, endometriosis was diagnosed with the use of laparoscopy in 7% of multiparous, asymptomatic women [9]. In a study that investigated 72 patients with postmenopausal endometriosis, the disease had been diagnosed before menopause in only 15.3% of the women [10]. In case of the presented patient, undiagnosed endometriosis before menopause was also possible as she had never undergone laparoscopic inspection of the abdominal cavity, which is considered the gold standard for diagnosing endometriosis [11]. It is possible that the disease remained asymptomatic until it was stimulated by hormone therapy, since estrogen dependence is considered central to both initiation and progression of the disease [12]. Nevertheless, it should be stressed that the question whether and which type of hormone therapy increases the risk of endometriosis in postmenopausal women remains challenging. In a retrospective study, recurrent endometriosis was diagnosed in 2% and recurrent pain in 6% of the women treated with estrogen-only therapy after total abdominal hysterectomy and bilateral salpingo-oophorectomy performed for endometriosis. However, no recurrence was observed in women who received combined estrogen-progestogen therapy [13]. In the aforementioned report by Morotti *et al.*, only three (4.2%) out of 72 patients with postmenopausal endometriosis took hormone therapy at the time of admission to hospital and only four (5.6%) had received it in the past [10]. In another study, the risk of symptomatic endometriosis was similar in women who underwent castration for endometriosis and subsequently took estrogen-only therapy and those who underwent the same surgery and did not take any hormones afterwards [14]. According to a prospective randomized study, the risk of recurrence in women who underwent total abdominal hysterectomy with bilateral salpingo-oophorectomy and took combined estrogen-progestogen therapy was low: 0.9% per year [15]. On the basis of a broad review of the literature, Soliman *et al.* concluded that continuous combined hormone therapy seems a safe option for postmenopausal patients with a history of endometriosis [16]. This conclusion was confirmed by the position statement of the European Menopause and Andropause Society published in 2010 [17].

The pathomechanism of postmenopausal endometriosis remains to be fully elucidated. Several theories have been proposed. Assuming the trophic effect of estrogens on the ectopic endometriotic tissue, the decrease in serum estradiol that naturally accompanies menopause should result in resolution of the lesions. However, due to high local expression of aromatase, an enzyme that converts adrenal androstenedione into estrogen, these endometriotic foci appear to autonomically synthesize estrogens that could stimulate their own growth, as well as act as paracrine

growth stimulators for other ectopic foci, e.g. localized in close proximity in the peritoneal cavity [12, 18]. The positive feedback exerted by PGE2 further enhances estrogen production [19]. Moreover, endometriotic implants are deficient in 17-beta-hydroxysteroid dehydrogenase type 2, the enzyme that converts estradiol into the less potent estrone [20]. These mechanisms could explain the persistence or development of endometriosis in certain postmenopausal women in the absence of hormone therapy [3, 5]. Apart from local production in the endometrioid implants, estrogens in postmenopausal women can also be derived from adipose tissue, skin, and hormonally active ovarian tumors [18, 21]. In a study investigating cases of postmenopausal endometriosis, 70% of the patients were obese [2]. Median BMI of the postmenopausal patients with endometriosis in the study by Morotti *et al.*, was 25.0 [10]. Another mechanism that could explain at least some cases of postmenopausal endometriosis is the theory of coelomic metaplasia [12]. Silva *et al.*, speculated that this theory, as well as the effect of local aromatase steroidogenesis, could be responsible for their three cases of postmenopausal endometriosis in lean women without history of hormone therapy [3].

The potential stimulating effect of hormone therapy on endometriotic lesions has already been discussed. Combined estrogen-progestogen therapy does not appear to elevate the risk of endometriosis. The patient described above was lean (BMI 23.4) and no ovarian tumors, apart from endometriosis, were diagnosed. Therefore, local production of estrogens in endometriotic lesions that had remained silent or coelomic metaplasia seem to be the most probable pathomechanism causing endometriosis in the reported case.

Endometriosis after the menopause is usually less disseminated and less active than in premenopausal patients [21]. The most common site of postmenopausal endometriosis is the ovary [2, 10]. Ovarian endometriomas occurring postmenopause have been reported both in large retrospective studies and in case reports [2, 3]. Other possible localizations include intestines [5], broad ligament [6], abdominal wall [3], rectovaginal septum [3, 10], and peritoneum [10].

The urinary tract may also be involved [4, 22] but the incidence of urinary tract endometriosis is low (1-5% of all cases, including premenopausal women) [23, 24]. The first case report describing ureteral obstruction associated with endometriosis in a postmenopausal woman was published in the 1970s [25]. Moreover, cases of endometriosis occurring during hormone therapy in women after the menopause, involving the ureters, have also been reported [26, 4]. Studies show that the incidence of ureteric endometriosis oscillates between 0.3% and 1.5% of women with endometriosis [27, 24]. The distal third of the ureter is usually involved [28]. The symptoms of ureteric invasion are often nonspecific or even absent [24, 28]. Out of the 15 patients with urologic endometriosis confined to one of the ureters in the study conducted by Antonelli *et al.*, five women (33.3%) reported no pain complaints. Renal failure was diagnosed as the only symptom in three of them, whereas hypertension was observed in two women. The remaining of these patients suffered from renal pain (seven) and dyspareunia or dysmenorrhea (three) [24]. In a case report of a postmenopausal woman with ureteric endometriosis, the disease manifested with recurrent urinary tract infections, left iliac fossa pain, and labile hypertension [4]. Although the CT scan of the abdomen showed hydronephrosis on both sides and distention of the right ureter was observed on gross examination during surgery, the described patient presented no symptoms of ureteric obstruction. Her serum urea and creatinine levels, as well as blood pressure, were within the normal range. Pain in the lower abdomen, probably attributable to the mass in the right parametrium, was her sole complaint. The ureteric involvement presented by the patient was not typical, since ureteric endometriosis is usually unilateral and most commonly affects the left ureter [28].

Early-stage ovarian cancer is usually asymptomatic or the symptoms are non-specific and mild. When the neoplastic process becomes advanced, the disease may result in pain, distention of the abdomen due to the tumor and ascites, and weight loss. At this stage, imaging techniques usually reveal single or multiple pelvic masses and numerous implants spread in the abdomen.

The symptomatology of the described patient was confined to non-specific pain localized in the lower abdomen, that began three weeks before the surgery. The CT scan of the abdomen showed a giant mass in the region of the right adnexa, infiltrating the surrounding structures, as well as pathological implants of the small intestinal mesentery and bilateral hydronephrosis. Despite only mildly increased serum level of CA-125 (77 U/ml), advanced carcinoma of the ovary seemed the most probable diagnosis and laparotomy was scheduled.

Endometriosis mimicking advanced ovarian cancer has been reported in the literature. Bhat *et al.*, described a case of a 50-year-old patient with a history of total abdominal hysterectomy for fibroid uterus, bilateral salpingo-oophorectomy, and partial vaginectomy for deeply infiltrating endometriosis, who did not take hormone therapy, presenting with a pelvic mass, bilateral hydronephrosis, hemoperitoneum, and pulmonary embolism. Active bleeding from a branch of the inferior mesenteric artery was diagnosed and emergency embolization of the vessel had to be performed. Laparotomy revealed a mass adherent to the rectosigmoid colon and pelvic side walls that involved both ureters. The lesion eroded into the vagina, causing ulceration of the vaginal vault. Carcinoma of the recto-vaginal septum of clear cell type was initially suspected, but histologic examination proved the lesion to be endometriotic tissue, with no signs of malignancy [29].

In another report, a 51-year-old woman (of unknown menopausal status) was operated on due to umbilical hernia. Histologic examination of the resected tissue revealed abundant acellular mucin. Along with the results of the CT scan, in which a large cystic mass in the right adnexa was reported, a metastatic adenocarcinoma and pseudomyxoma peritonei were suspected and laparotomy was scheduled. Total abdominal hysterectomy and bilateral salpingo-oophorectomy were performed, as well as partial omentectomy and excision of sigmoid serosa. Histology evaluation revealed endometriosis of both ovaries and the cervix, as well as pools of acellular mucin in the omentum and bladder peritoneum [30].

In another case, reported by Goumenou *et al.*, a 46-year-old woman presented with large bilateral ovarian masses associated with significant ascites and pleural effusions. She had lost seven kg of weight over the previous five months and her serum CA-125 level was 3504 U/ml. The clinical picture was strongly suggestive of advanced ovarian cancer. The patient received two courses of neo-adjuvant chemotherapy and was operated two months later. Histologic examination revealed inflammation and endometriosis, without any evidence of malignancy [31]. Likewise, a younger women with endometriosis may also be misdiagnosed with advanced ovarian cancer. Recently, a case of a 29-year-old woman with polypoid endometriosis (a rare type of the disease), mimicking ovarian carcinoma with peritoneal dissemination, was described. Both in MRI and at laparotomy, the picture was strongly suggestive of disseminated cancer. A left ovarian cyst of approximately six cm in diameter was detected, and solid nodules were noted on the right ovary, omentum, and rectum. Owing to an intraoperative pathology consultation, endometriosis was detected and the surgery was confined to the left salpingo-oophorectomy only [32].

Endometriosis has also been reported to mimic advanced cervical cancer in MRI pictures performed as part of preoperative staging [33]. The issue of the links between endometriosis and cancer becomes even more complicated when we consider the possibility of malignancy occurring in endometriotic tissue. Such cases have also been reported [34].

In case of the presented patient, endometriosis was not suspected. However, in some cases, especially in women of reproductive age, clinical examination and imaging techniques suggest that both diagnoses are possible. Unfortunately, serum CA-125 levels cannot aid in differentiating endometriosis from ovarian carcinoma, since both conditions are associated with increased levels of that marker. Careful evaluation of the MRI pictures may, however, be of use in these cases, as was concluded in one of the reports mentioned before [32].

Endometriosis has also been shown to be able to mimic advanced malignancy originating from other organs than the ovary, e.g. cervical cancer, as in the aforementioned case, described by Selo-Ojeme *et al.* [33], or even the urinary bladder [35]. In both cases, neither the results of ultrasound scan nor MRI proved helpful in establishing the correct diagnosis before surgery.

Conclusions

Despite it being a benign disease, in most advanced cases of endometriosis, its pattern of spread resembles a neoplastic process. The diagnosis of advanced neoplastic process, particularly in postmenopausal women, is sometimes much more tempting than endometriosis, the occurrence of which is classically perceived as confined to women of reproductive age. However, clinicians should remain conscious of the possibility that endometriosis may mimic advanced ovarian cancer and infiltrate the peritoneum and internal organs of the abdominal cavity, including the urinary tract. In fact, these lesions may prove to be endometriotic, even in postmenopausal patients without history of the disease during premenopausal years. Imaging techniques, including CT and MRI, are not always effective in elucidating the correct diagnosis preoperatively.

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