Case Report

Surgical Management of Recurrent Ureteric Endometriosis Causing Recurrent Hypertension in a Postmenopausal Woman

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ABSTRACT

Endometriosis is a common condition that affects as many as 10% to 20% of women of reproductive age. Because of the subtle clinical signs and symptoms and limitations of imaging methods, the diagnosis is frequently delayed or missed, with serious consequences including hypertension, hydronephrosis, and loss of kidney function. We present an unusual case of recurrent ureteric endometriosis in a postmenopausal woman to highlight the challenges of screening for and management of endometriosis. Journal of Minimally Invasive Gynecology (2010) 17, 100–103 © 2010 AAGL. All rights reserved.

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Endometriosis is a common condition that affects as many as 10% to 20% of women of reproductive age [1]. The urinary tract is involved in about 2% of cases, with the ratio of bladder to ureter to urethra of 40:5:1 [1]. The true incidence of ureteric endometriosis remains unknown but has been estimated at 0.3% [1] to 1.5% [2] of all endometriotic cases. The typical woman with ureteric endometriosis is between age 30 and 35 years [3]. The lesion is usually unilateral, left-sided, and found in the distal third of the ureter [3].

Because of the subtle clinical signs and symptoms and limitations of imaging methods, the diagnosis is frequently delayed or missed, with serious consequences including hypertension, hydronephrosis, and loss of kidney function [4]. We report an unusual case of recurrent ureteric endometriosis in a postmenopausal woman to highlight the challenges of screening for and management of the disease.

Case Report

A 62-year-old postmenopausal woman had a 2-year history of recurrent urinary tract infections and left iliac fossa pain. At age 50 years, she had undergone a total abdominal hysterectomy and bilateral salpingo-oophorectomy for treatment of pelvic pain. Operative findings revealed an 18-week sized uterus with multiple myomas and a left cystic ovary that was densely adherent to the pelvic side wall. The surgeon commented that he could not be confident that all ovarian tissue had been excised. The right ovary and ureters were noted to be normal. Histologic analysis confirmed myomas, adenomyosis, and endometriosis in the left ovary. Estrogen patch therapy was initiated to treat menopausal symptoms.

The patient first came to our clinic in July 2002 at age 56 years with recurrent urinary tract infections, left iliac fossa pain, and labile hypertension. She had been receiving estrogen therapy since undergoing the total abdominal hysterectomy and bilateral salpingo-oophorectomy. Examination revealed tenderness in the left iliac fossa and left fornix. Ultrasonography and intravenous pyelography revealed an obstructed left ureter and hydronephrosis. Hormonal therapy was stopped at this stage because of suspicion that the underlying condition could be related to ureteric endometriosis.

In November 2002, with the assistance of a urologist, a severely obstructed left ureter was identified, ureterolysis was performed, and a chronically inflamed fibrotic mass was excised (Fig. 1) from the ureter at the level of the pelvic brim via laparoscopy for both excision of endometriosis and ureterolysis. Histologic analysis demonstrated complex endometrial hyperplasia with no atypia (Fig. 2). The patient made a good recovery with resolution of pain, hydroureter,
hypertension, and urinary tract infection. Hormone therapy was not resumed postoperatively.

The patient was followed up by her family physician on an annual basis and remained well until 2006, when she reported recurrent left iliac fossa pain. A computed tomography scan of the abdomen and pelvis showed no abnormality. Renal functions were normal. A year later, she again had recurrent left iliac fossa pain, recurrent urinary tract infections, and hypertension. Urologic assessments that included renal biochemistry, intravenous pyelography, cystoscopy, and retrograde ureterography revealed no abnormality. In November 2008, because of persistent symptoms that she described as similar to those she had experienced in 2002, a decision was made to perform exploratory laparoscopy. By that time, the patient was receiving multiple oral antihypertension medications (metoprolol, indapamide, and moxonidine). On the day of surgery, preoperative blood pressure was 150/100.

At laparoscopy, a small pigmented endometriotic lesion on the serosal layer of the rectosigmoid was found and excised. After mobilization of dense sigmoid adhesions from the pelvic brim, the left ureter was found to be mildly dilated. Following the course of the ureter by retroperitoneal dissection to the ureteric tunnel, a $2 \times 2 \times 2$-cm fibrotic mass encircling the ureter was identified (Fig. 3). The location of this lesion was different from that of the ureteric mass found in 2002, approximately 1 cm proximal from the vesicoureteric junction.

Intraoperative blood pressure was extremely labile. Immediately after induction of anesthesia and intubation, there was a notable decrease in systolic blood pressure (Fig. 4, left arrow), from 149 to 100 mm Hg. Once surgery progressed, and in particular in association with surgical handling of the pathologic areas, sustained elevation of systolic blood pressure, increasing to 200 mm Hg, became an issue (Fig. 4, middle arrow).

Numerous pharmacologic interventions were undertaken by the anesthetist to minimize the acute intraoperative hypertension including administration of a variety of drugs used to increase depth of anesthesia (propofol, sevoflurane, and opioid agents) and specific antihypertension agents (metoprolol and clonidine). The blood pressure lowering effect of these interventions was unimpressive in both degree and duration. Once the ureteric endometriotic lesion was completely excised, the acute intraoperative hypertension rapidly resolved, with systolic pressure decreasing to 90 to 100 mm Hg (Fig. 4, right arrow). The postoperative recovery was uneventful, and blood pressure remained normal with systolic pressure in the range of 90 to 110 mm Hg. At discharge, the patient was able to cease taking 3 antihypertension medications, and blood pressure has remained normal. The left iliac fossa pain and urinary tract infections have also resolved. Histologic analysis confirmed endometriosis with no evidence of hyperplastic changes.

Discussion

The primary goals in the management of ureteric endometriosis are to relieve symptoms and prevent recurrence, relieve any urinary tract obstruction, and preserve renal function [4]. Factors such as patient age, fertility desire, extent of disease, severity of urinary tract and gynecologic symptoms, and presence of other pelvic disease determine the choice of treatment [4].

Ureteric endometriosis can be managed medically and surgically. In patients with early disease in which renal function is normal with no evidence of obstructive uropathy, hormone therapy such as danazol, progestins, and gonadotropin-releasing hormone agonist may be considered. However, success rates have been variable. Some authors have reported success with hormone therapy in reversing ureteral obstruction [5,6], whereas others have shown no disease resolution but instead deterioration [7] during treatment. Close regular monitoring of renal function in patients receiving medical treatment is, therefore, essential. Clinicians should also be aware that drug compliance may be compromised because of adverse effects [3].
In patients with deep infiltrating endometriosis, medical treatment has proved to be of limited benefit, leading to temporary regression rather than eliminating the disease [3,4]. It is likely that the endometriotic lesions in the presence of dense adhesions and fibrosis are no longer responsive to hormonal suppression [8].

Debulking surgery is considered the criterion standard treatment but is complex and can be associated with serious complications. Ureterolysis is recommended if the endometriotic deposits have not invaded the ureteric wall and renal dysfunction is reversible. When there is ureteric invasion, a ureteroureterostomy or ureteroneocystomy with or without a psoas hitch or Boari flap is performed to restore continuity of the urinary tract [3,4]. Nephrectomy may be required in the presence of a nonfunctioning kidney.

Historically, total abdominal hysterectomy and bilateral salpingo-oophorectomy and resection of pelvic endometriosis had been the operation of choice to treat severe endometriosis. The objective was to eliminate endogenous estrogen production and, consequently, to inactivate endometriosis. Not only is this procedure limited to women who no longer desire fertility, it does not necessarily reverse the damaging effects on the ureter or kidney. Recurrence of endometriosis has been reported despite pelvic clearance surgery [9]. This may be due to incomplete resection of ovarian tissue because of technical difficulty in the presence of distorted pelvic anatomy. Alternatively, the activity of the disease may be sustained by estrogen production by an extraovarian endogenous source such as the adrenal gland or exogenously in the form of hormone therapy [10]. In rare cases, the tendency to infiltrate may become independent of hormonal control [1] and can even result in hyperplastic [10] and malignant changes [11].

Postoperatively, long-term hormone therapy to prevent disease recurrence has been reported, with inconsistent results [12]. Follow-up of patients with diagnosed ureteric endometriosis is challenging because there is no clear guidance for the clinician. The options include monitoring for signs and symptoms of recurrence such as worsening hypertension, regular imaging to screen for ureteric strictures or hydronephrosis, and repeat laparoscopy. There are limitations with each option.

Because of the nonspecificity or absence of symptoms, ureteric endometriosis may be silently progressive, resulting in irreversible renal damage. Hence, measurable factors such as blood pressure and urinalysis (for hematuria) should be monitored regularly. It is reasonable to perform repeat laparoscopy to assess for disease recurrence; however, this involves operative and anesthetic risks. Early or intrinsic ureteric endometriosis may be missed if it is out of the surgical field of view.

Various imaging techniques to assess ureteric and renal function have been described. Ultrasonography of the renal tract is considered a good screening tool because it is noninvasive and can demonstrate hydronephrosis while providing information about other pelvic pathologic conditions. However, hydronephrosis is a late sign of ureteric obstruction, and renal damage may already be irreversible. In contrast, intravenous pyelography can enable identification of ureteric obstruction and demonstrate renal function; however, this test is invasive, and the findings are not specific for
endometriosis. Magnetic resonance imaging is currently the recommended imaging method [3]. It has the ability to visualize all components of the urinary tract and pelvis and does not expose the patient to any radiation; however, it is expensive.

In theory, hormone therapy could be associated with endometriosis recurrence by stimulating new growth or reactivating residual lesions. However, the true risk of recurrence is still unknown because of scanty data in the literature. It seems to be safe and, hence, should not be withheld from a patient with menopausal symptoms [13]. Nevertheless, women with a history of endometriosis while receiving hormone therapy should be monitored closely for disease recurrence. There is currently no guidance on the best hormone therapy regimen; however, because endometriosis is estrogen responsive, perhaps unopposed estrogen therapy should be avoided and combined estrogen-progestagen preparations or tibolone be preferred.

The mechanism for the acute hypertension associated with ureteric manipulation remains unclear. Unilateral ureteral obstruction induces the genes that encode renin, angiotensin-converting enzyme, and angiotensin in rats [14]. It has been postulated that ureteral manipulation, possibly by stretch or nerve stimulation, differentially regulates the genes that encode the renin-angiotensin system in the kidney and as a result substantially affects renal hemodynamics and systemic vascular control. However, further research is required to confirm whether this pathophysiologic process applies to human beings.

Patients with ureteral endometriosis can experience a range of symptoms, many of which are rather vague. They do not necessarily have concurrent gynecologic symptoms suggestive of pelvic endometriosis [15]. Therefore, clinicians should always maintain a high index of suspicion. Endometriosis can occur even in postmenopausal women, as illustrated in this case. While uncommon, it may be prudent to consider ureteric endometriosis in women with hypertension [16] and a history of endometriosis [9,16].

References