Isolated Bladder Endometriosis: A Rare Case Report

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ABSTRACT

Endometriosis is defined as presence of functional endometrial glands and stroma beyond the normal confines of the uterus. Overall incidence is 10 to 20% of women in reproductive age, with peak incidence between 30 and 45 years. About 40% of women with infertility and 60% of those presenting with chronic pelvic pain have endometriosis. About 1% of women with endometriosis have urinary tract involvement, of which 84% involve the bladder. Urinary bladder endometriosis as a part of deep infiltrating pelvic endometriosis is known, but isolated bladder involvement is extremely rare. Patients present with vague and distressing urinary symptoms mimicking recurrent cystitis, hence strong clinical suspicion with prompt recognition of this entity is important to avoid prolonged morbidity. We report a case of isolated bladder endometriosis in a 28-year-old female with previous two cesarean sections. Open partial cystectomy was performed. Histopathology of the excised mass was diagnostic.

Keywords: Endometriosis, Isolated, Partial cystectomy, Urinary bladder, Urinary symptoms.


Source of support: MGMIHS
Conflict of interest: None

CASE REPORT

A 28-year-old, married woman presented with chief complaints of cyclical episodes of frequency, urgency, dysuria, and lower abdominal pain during premenstrual and menstrual period, for 15 days every month for last 4 years. There was no history of hematuria. Menstrual cycles were regular. The patient was married for last 10 years and had a history of three intrauterine deaths – all at full term, the 1st being normal delivery followed by two cesarean sections. The last cesarean section was performed 5 years back. Abdominal examination revealed healed scar of previous cesarean section. Pelvic examination was normal.

Ultrasonography (USG) suggested echogenic polypoidal mass projecting from posterior bladder wall (Fig. 1A). A pelvis magnetic resonance imaging (MRI) scan showed 3.7 x 3.3 x 2.7 cm heterointense lesion with smooth indentation along posterosuperior wall of urinary bladder with loss of fat planes between anterior myometrium and bladder serosa (Fig. 1B). Cystoscopic examination identified a sessile, irregular, nodular, bluish mass in the midline, at the junction of dome and posterior wall of bladder (Fig. 1C). Histopathology of cystoscopic biopsy suggested endometriosis of bladder.

Figs 1A to C: (A) Ultrasonography showing echogenic mass arising from posterior bladder wall, (B) magnetic resonance imaging showing heterointense mass involving posterior bladder wall, and (C) cystoscopy showing nodular bluish mass arising from posterior bladder wall.
On exploration, posterior bladder wall was separated by sharp dissection from adherent anterior wall of uterus. The mass (Fig. 2A) was completely resected and bladder wall was closed in two layers. Histopathology of excised specimen revealed transitional epithelium with intact basement membrane and plenty of round tubular and cystic dilated endometrial glands (Fig. 2B) in compact cellular stroma underneath. The glands were seen infiltrating into detrusor muscles, suggestive of endometriosis of urinary bladder.

DISCUSSION

Infiltration of endometrial glands into the uterine muscle is referred to as “adenomyosis” and outside the uterus it is called “endometriosis.” Theories for pathogenesis of endometriosis: (1) Transplantation theory – extension of an adenomyotic nodule of the anterior uterine wall; (2) Embryonal theory — developing from Müllerian duct remnants in the vesicouterine/vesicovaginal septum; and (3) Sampson’s migratory theory supported by Vercellini et al — the menstrual effluent containing viable endometrial cells can be transported to ectopic sites. This theory has been supported by the ability of endometrial tissue to engraft itself in other areas, and this is observed in many cases of urinary tract endometriosis in patients who have undergone previous uterine surgery. In our case, migratory theory with intraoperative dissemination of endometrial cells during previous caesarean section may be the etiology of vesical endometriosis. Urinary tract endometriosis predominantly affects the bladder, followed by the ureter and the kidney in the ratio of 40:5:1.

Bladder endometriosis could be primary or secondary. The primary form is spontaneous, commonly in association with deep infiltrating pelvic endometriosis (11%). The secondary manifestation results following pelvic surgery, such as cesarean section and hysterectomy. Up to 50% of patients with bladder endometriosis have a history of previous pelvic surgery. Bladder endometriosis can be intrinsic (full thickness) or extrinsic, involving serosa and peritoneal surface, generally found in the trigone, dorsal wall, or ureterovesical junction.

The symptoms of bladder endometriosis vary depending on the location and size of the lesion. About 30% patients are asymptomatic and 70% present with urinary symptoms at the time of diagnosis. Lower urinary irritative voiding symptoms, such as frequency, urgency and dysuria, suprapubic pain, and hematuria are common, mimicking recurrent cystitis without bacteriuria. Urinary symptoms are catamenial, occurring in temporal “cyclic” relationship to monthly menstruation in about 40% of the patients, while majority (60%) present with noncyclical symptoms.

Ultrasonography is the initial diagnostic modality of choice in suspected bladder endometriosis. Localized bladder wall thickening can be appreciated, leading to the differential diagnosis of bladder endometriosis, subserosal anterior leiomyoma, and bladder cancer. A pelvis MRI can accurately delineate the morphologic abnormalities of bladder endometriosis and also potentially identify other common sites, particularly at the uterosacral ligament. The diagnosis of vesical endometriosis is difficult, and it should be confirmed by cystoscopy with biopsy. The endometrioma may show marked congestion and edema with translucent bluish nodules on cystoscopy. Bladder endometriosis can be classified as superficial (< 5 mm) or deep (> 5 mm), with depth of lesion reflecting severity of symptoms and guiding therapeutic management. Urinary bladder endometriosis may be treated surgically or medically with hormone-suppressive therapy. Treatment needs to be individualized according to patient’s age, severity of symptoms, extent of disease, associated pelvic disease, and parity. Hormonal treatment involves oral
contraceptives, danazol, progestin, and gonadotropin-releasing hormone agonists. However, medical treatments usually are only palliative, and symptoms generally recur on discontinuation. Surgical treatment is therefore definitive treatment for endometriosis. Type of surgical treatment depends on size and location of lesion. Small, superficial symptomatic lesions can be cauterized or ablated with bipolar or CO₂ laser. Full thickness larger lesions, as in our case, require open or laparoscopic segmental resection of bladder.

CONCLUSION

Diagnosis of bladder endometriosis is often difficult to make because of its nonspecific symptoms. It requires high index of suspicion in premenopausal women complaining of catamenial bladder symptoms with negative urine cultures. The management is mainly surgical and resection should be complete. Partial cystectomy is the most effective treatment of choice for large full thickness lesions with high success rate and recurrence. Laparoscopic and open approaches have comparable results.

REFERENCES