Bladder Endometriosis: "The Hidden Culprit Behind Cyclical Urinary Symptoms"

Shreedhar Khadka K.C., MBBS, FCPS Urology Trainee; Rajesh Batajoo, MBBS, MS(TUTH), Fellowship in Urology (Singapore), Fellowship in Uro-Oncology (Tata, India)

Department of Urology, B&B Hospital, Gwarko, Lalitpur, Nepal

Address of Correspondence:

Shreedhar Khadka K.C., FCPS Urology Trainee

Department of Urology, B&B Hospital, Gwarko, Lalitpur

Email: khadkaeden@gmail.com

Phone: +977-9841035276

Bladder endometriosis is an uncommon manifestation of deep infiltrating endometriosis, often presenting with cyclical lower urinary tract symptoms that overlap with other urological conditions. We report the case of a 37-year-old woman with a 17-month history of pelvic discomfort, dysuria, urinary frequency, and intermittent hematuria, with symptoms intensifying around menstruation. Initial ultrasonography and transvaginal imaging were unremarkable, leading to a delay in diagnosis despite persistent clinical suspicion. Repeat imaging later revealed a non-mobile polypoidal lesion on the anterior bladder wall. Cystoscopy identified a multiloculated submucosal mass, and transurethral resection was performed. Histopathological evaluation confirmed bladder endometriosis accompanied by Von Brunn nests and cystitis cystica. Postoperative hormonal therapy with norethisterone and dienogest resulted in symptomatic relief. This case underscores the diagnostic challenges posed by bladder endometriosis, particularly when early imaging is inconclusive. It highlights the importance of maintaining a high index of suspicion in reproductive-age women with cyclical urinary symptoms and demonstrates the value of close collaboration between urology and gynecology in achieving timely diagnosis and optimal management.

Keywords: bladder endometriosis, deep infiltrating endometriosis, cyclical hematuria, caesarean section, transurethral resection.

Bladder endometriosis, defined as endometrial tissue infiltrating the detrusor muscle or deeper, affects approximately 1–2% of women with endometriosis but accounts for up to 70–85% of all urinary tract involvement.[1,2] Its incidence has risen dramatically in parallel with increasing caesarean section rates, with multiple series reporting previous pelvic surgery—particularly caesarean delivery—as the strongest independent risk factor (odds ratios 4–21).[3,4] The postulated mechanism involves direct implantation of viable endometrial cells into the vesicouterine pouch defect created during caesarean section, followed by progressive invasion through the bladder wall.[5] Despite classic teaching that urinary symptoms in endometriosis are rare, contemporary data reveal that up to 50–80% of women with DIE experience lower urinary tract symptoms (LUTS), with storage symptoms predominating when the bladder is involved.[6] The diagnostic delay averages 4–7 years, largely due to overlap with bladder pain syndrome/interstitial cystitis (the so-called "evil twin" syndrome), overactive bladder, recurrent UTI, and, most dangerously, transitional cell carcinoma.[7]

Case Details

A 37-year-old lady, G2P2, presented with a 17-month history of a sensation of heaviness and incomplete bladder emptying after micturition. Five months into her symptoms, she developed additional complaints of dysuria, urinary frequency, and severe pain in the left pelvic region and back. She also experienced intermittent hematuria. Over time, she noticed that these symptoms were cyclical, occurring predominantly around her menstrual cycle—from one day before menstruation up to 10 days into her period. The duration and intensity of discomfort progressively increased during menstruation.

She reported a history of recurrent urinary tract infections and occasional passage of chocolate-colored urine, as well as reduced menstrual bleeding following her second cesarean section two years prior. A gynecological consultation was sought five months after symptom onset. During this period, ultrasonography (USG) and transvaginal sonography (TVS) were performed and reported as normal; however, based on her clinical presentation, endometriosis was suspected by the gynecology team.

Her medical history was significant for a cesarean section seven years ago. She has been a smoker for nine years and consumes alcohol socially.

Upon evaluation, the patient had no pallor, and abdominal findings were unremarkable. Laboratory investigations, including urine routine, culture, and kidney

function tests, were within normal limits. Ultrasonography revealed a non-mobile polypoidal lesion at the mid-anterior wall of the bladder measuring 2.5×2.3 cm, raising the differential diagnosis of a neoplastic lesion as shown in **Figure 1**.

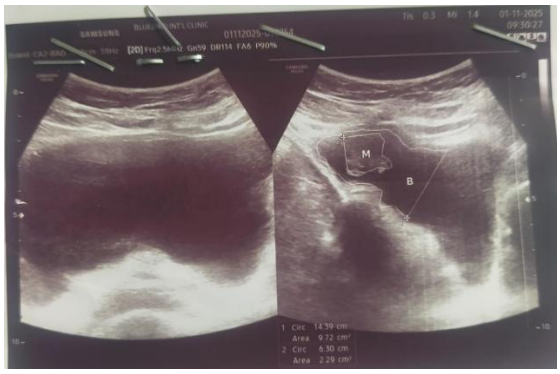

Figure 1. Transabdominal ultrasound showing a well-defined heterogeneous hypoechoic mass involving the anterior bladder wall, suggestive of deep infiltrating bladder endometriosis

Flexible cystoscopy revealed a large (≈3×3×4 cm), multiloculated submucosal mass with overlying bullous oedema, mucosal erythema, and multiple actively bleeding punctate vessels—features highly suggestive of endometriotic implantation during the menstrual phase as shown in **Figures 2 and 3**.

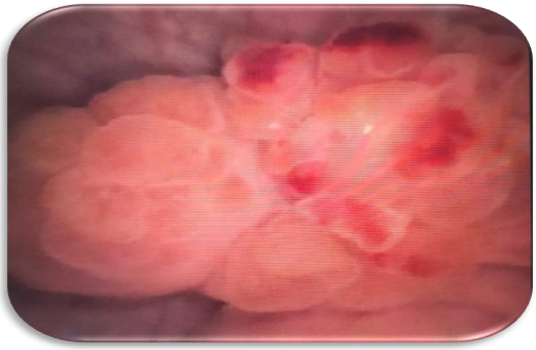

Figure 2. Cystoscopic appearance of the mass demonstrates classic bullous oedema and submucosal hemorrhage with active bleeding sites

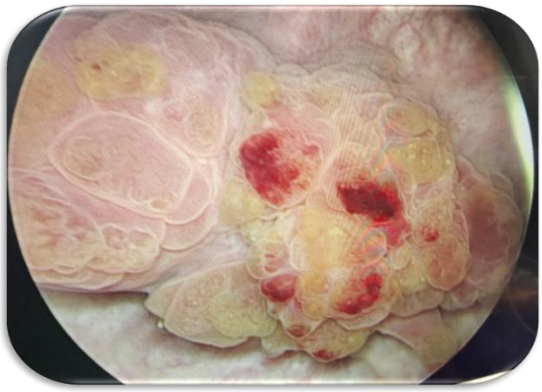

Figure 3. Higher magnification showing the grape-like, multiloculated submucosal tumor with fresh hemorrhage

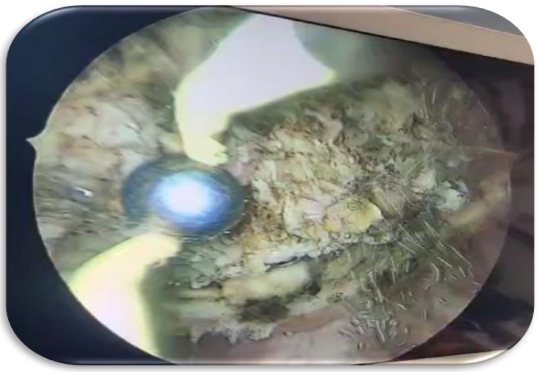

Figure 4. Intraoperative appearance after gentle dissection, confirming full-thickness involvement with serosal puckering and muscularis infiltration

Transurethral resection of the entire intravesical component was performed with minimal bleeding. **Figure 4** shows the lesion bed after transurethral resection. Intraoperatively lesion had full-thickness involvement where the serosal surface had noticeable puckering, and the underlying muscularis layer was infiltrated. Histopathological examination revealed

endometrial glands and stroma deeply infiltrating the detrusor muscle, surrounded by prominent von Brunn nest proliferation, cystitis cystica, and chronic inflammatory changes—confirming intrinsic bladder endometriosis with reactive urothelial hyperplasia

Postoperatively, the patient commenced on continuous combined hormonal suppression using dienogest 2 mg+norethisterone 5 mg twice daily, and the patient has been thoroughly counselled regarding the potential need for a partial cystectomy should her symptoms worsen.

Discussion

The temporal relationship between caesarean delivery and the onset of bladder endometriosis in this case strongly supports the iatrogenic implantation theory.[1–5] The second caesarean section, performed only two years prior, likely facilitated direct seeding of endometrial cells into the vesicouterine defect, with subsequent cyclical hormonal stimulation driving progressive transmural invasion. The striking menstrual synchronization of symptoms—including macroscopic hematuria with "chocolate" clots—represents pathognomonic evidence of active endometrial tissue within the bladder wall, as mucosal involvement allows direct menstrual shedding into the lumen.[8] This cyclical pattern, present in 70–90% of confirmed BE cases, remains the single most valuable clinical clue and should trigger immediate specialist referral even when initial imaging is negative.[1,9]

Diagnostic delay in this patient (17 months) reflects common pitfalls: mid-cycle ultrasound missing active lesions, empirical antibiotic overuse for presumed UTI, and failure to recognize the "evil twin" overlap with bladder pain syndrome.[7] The dramatic visualization during perimenstrual scanning underscores the critical importance of timing imaging to the early menstrual phase when lesions are maximally congested and vascularized.[10] The combination of specialized transvaginal ultrasound (sensitivity 78–92% in expert hands), cystoscopy showing bullous oedema with active bleeding, and laparoscopy demonstrating serosal implants with intraoperative menstruation provided near-pathognomonic confirmation and obviated the need for more invasive staging[11,12]

The coexistence of florid von Brunn nest proliferation and cystitis cystica, while previously considered rare, is increasingly recognized as a reactive phenomenon secondary to chronic endometriotic inflammation and hormonal stimulation of urothelium.[13] These changes can mimic urothelial carcinoma both radiologically and histologically, emphasizing the necessity of biopsy in all suspicious bladder

masses in reproductive-age women.[14]

Management remains controversial. Although partial cystectomy with bilateral ureteric reimplantation achieves the lowest recurrence rate (0–8%), it carries significant morbidity and is reserved for extensive or recurrent disease.[15,16] Transurethral resection combined with long-term hormonal suppression, as employed here, offers excellent outcomes (complete remission rates 75–95%) with minimal morbidity and preserved fertility—making it the preferred first-line surgical approach for lesions <3–4 cm without ureteric involvement.[17–19] The addition of norethisterone to dienogest in this case likely enhanced progestogenic downregulation, achieving rapid lesion regression and symptom control.

This case reinforces that bladder endometriosis should be considered in every reproductive-age woman with new-onset LUTS following caesarean delivery, particularly when symptoms are cyclical. High clinical suspicion, appropriately timed multimodal imaging, and bladder-sparing surgery with prolonged hormonal therapy represent the contemporary gold standard for achieving a cure while preserving quality of life.

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