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Eisenberg V[1], Arbib N[1], Achiron R[1], Schiff E[1], Goldenberg M[1], Soriano D[1]
1. *Sheba Medical Center*

BLADDER DETRUSOR ENDOMETRIOSIS – WHERE DOES IT COME FROM?

Hypothesis / aims of study:
Bladder pain syndrome is a chronic inflammatory condition of the bladder, which is the cause of pain in more than 30% of women with chronic pelvic pain. Endometriosis and bladder pain syndrome may coexist in up to 65% of women. Bladder endometriosis is defined as endometriosis infiltrating the detrusor muscle, and occurs in approximately 1% of women with pelvic endometriosis. A diagnostic delay may ensue due to non-specific symptoms mimicking recurrent cystitis. The pathogenesis of bladder endometriosis is not fully understood and several major theories have been proposed; implantation of regurgitated endometrium, extension of adenomyosis from the anterior uterine wall to the bladder, Mullerian metaplasia in the vesicovaginal septum, and iatrogenic bladder endometriosis after cesarean delivery (1, 2). The aim of our study was to evaluate the appearance of bladder detrusor endometriosis on ultrasound in order to try to elucidate its pathogenesis and any association with uterine adenomyosis.

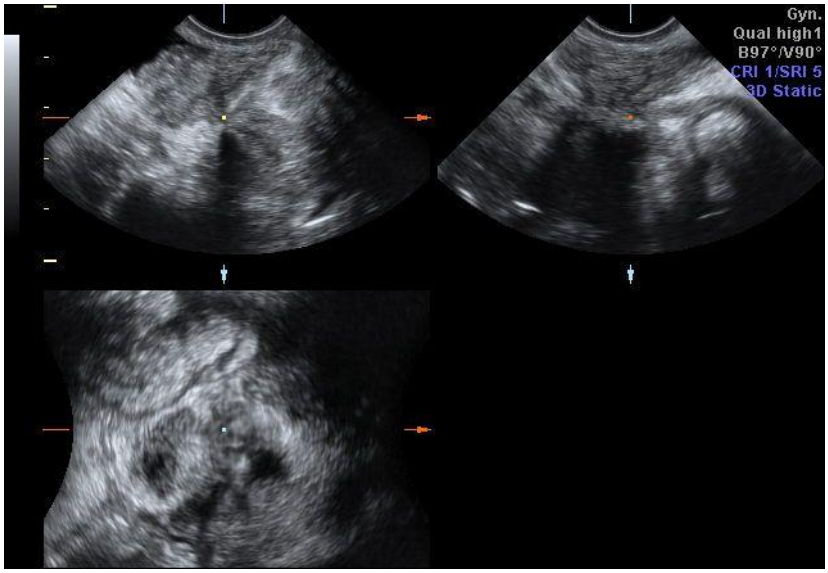
Study design, materials and methods:
Clinical records were retrieved for consecutive women who were evaluated at a tertiary referral centre for endometriosis from May 2011 to December 2014. Only women with ultrasound diagnosis and histological confirmation of bladder detrusor endometriosis were included in the study. The preoperative workup included a detailed history, clinical examination, urinalysis, cystoscopy, and an ultrasound scan. All ultrasound scans were performed by one operator both transabdominally and transvaginally using a Voluson 730 machine (GE Kretz). All laparoscopies were performed by a multidisciplinary dedicated team of surgeons, and partial bladder cystectomy was performed for all cases infiltrating the bladder detrusor. The diagnosis of bladder endometriosis was confirmed at histopathology when endometrial glands and stroma were found infiltrating the bladder muscularis propria. Statistical analysis was performed with SPSS version 21 with $P < 0.05$ for significance.

Results:
Out of 288 endometriosis surgeries, eleven women (3.8%) with bladder detrusor endometriosis were included in the study. The mean age was 35.7 (29-47) years, BMI 21.5 (18.4-27.7), 3 (27.3%) were parous all by vaginal deliveries, and 3 (27.3%) had undergone previous surgery for endometriosis. Symptoms at presentation included: dysmenorrhea – all (100%), dyspareunia – 8 (72.7%), urinary complaints – 9 (81.8%): frequency and urgency – 7 (63.6%), dysuria - 9 (81.8%), pain on urination – 7 (63.6%), and hematuria – 1 (9.1%), gastrointestinal complaints – 5 (45.5%), and infertility – 6 (54.5%).
On physical examination the urinary finding was suspected in all but one patient. The bladder nodule was visualized on cystoscopy in all patients.
On ultrasound all had an anterior uterus and all had signs of adenomyosis including: microcysts, hyperechoic nodules and heterogeneous myometrium (100%), asymmetric dimensions favouring the anterior wall (90.9%), parallel striations (63.6%), poorly defined endometrial myometrial junction (90.9%). Nine women (81.8%) had an adenomyoma in the anterior uterine wall. Additional endometriosis involvement included: ovarian (4 - 36.4%), uterosacral ligaments (4 - 36.4%), posterior compartment lesion (4 – 36.4%), ureteral (2 – 18.2%). Seven patients had free movement of the bladder against the vesicouterine pouch, and four had an absent sliding sign in the posterior compartment suggesting pouch of Douglas obliteration. The appearance of the vesical nodule on ultrasound was: comma-shaped – 4 (36.4%), spherical – 5 (45.5%). The mean maximal nodule diameter in the bladder was 32.7 (20-52) mm. Ten nodules were seen penetrating the bladder detrusor, nine of these originated from the anterior uterine wall with a mean nodule size in the anterior wall of 28.4 (15-43) mm. Two nodules were located in the bladder base. The appearance of the nodule penetrating the uterine wall was like an hourglass (Figure 1). There was no ureteral constriction inside the bladder. The sensitivity, specificity, PPV, NPV, and accuracy for diagnosing bladder endometriosis on ultrasound were 100%.
AT surgery the mean ASRM score was 47.2 (6-148) and endometriosis stage was: 2 – 2 (18.2%), 3 – 3 (27.3%), and 4 – 6 (54.5%). Two women had isolated bladder endometriosis. Five women had obliteration of the vesicouterine pouch with retraction of the round ligaments. All of the women underwent partial bladder cystectomy with complete resection of the nodule. Concomitant ureteral involvement was found in 3 women who also underwent ureterolysis.
Histological analysis confirmed infiltration of the bladder muscularis by endometrial glands and stroma without mucosal penetration in all women.
Mean follow-up interval was 12.5 (2-30) months. All were free of symptoms. Three had conceived, 2 have delivered and one is 29 weeks pregnant at this time. One patient has recurrent dysmenorrhea but no bladder symptoms.

Interpretation of results:
Our findings suggest that the pathogenesis of bladder endometriosis is more often by penetration of adenomyosis from the anterior uterine wall into the bladder muscularis. This is in contrast with previous observations by other groups (3). The implication of this finding in endometriosis management is to obviate preoperative accurate diagnosis which will consequently guide the surgical approach and particularly the extent of nodule resection.

Concluding message:
Bladder endometriosis seems to originate from adenomyosis penetrating from the anterior uterine wall. Accurate preoperative diagnosis is imperative in order to guide successful management and to prevent recurrence.

Figure: Bladder detrusor endometriosis penetrating from anterior uterine wall. See hourglass appearance



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2. Fertil Steril 1998; 69: 972-75
3. Am J Obstet Gynecol 2002; 187: 538-42

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