

Chronic Voiding Dysfunction Due to Symphysis Pubis Mass in a Female- Case Report

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Abstract

Voiding dysfunction due to Bladder outlet obstruction in females is one of poorly understood conditions and is much rarer as compared to males. More difficult is the objective diagnosis of this condition. Here in, we present our case of a chronic voiding dysfunction in a female caused by symphysis bone tumor, which was managed surgically.

Keywords: Voiding dysfunction, bladder outlet obstruction in female, symphysis bone Tumore.

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INTRODUCTION

Voiding dysfunction (VD) in women is a common health problem. 6% of >40 aged women suffer from (VD). It can be related to either an abnormality in detrusor muscle activity and/or bladder outlet Obstruction (BOO) [1]. (BOO) Is a rare condition in females compared to males. There are no standard Urodynamic criteria to diagnose it. Many factors might contribute to developing voiding disorder in female-like anatomic, neurogenic, pharmacologic, endocrine, pharmacological and other causes [2].

The non-standard urodynamic criteria of VD and the pathophysiology of urination mechanism in females prolong reaching a correct diagnosis.

We present an interesting case of an elderly woman who presented with chronic voiding disorder secondary to outlet obstructive caused by a degenerative connective tissue mass under the symphysis pubis.

CASE PRESENTATION

A 79 years old woman presented to our outpatient clinic with chronic high residual urine and recurrent lower urinary tract infection (LUTI) in the last few years. She reported experiencing difficulty in passing urine for approximately six months. Before few months the patient was admitted in another hospital through the emergency department because of hemorrhagic cystitis. Since then a suprapubic catheter was inserted. A medical history of total abdominal hysterectomy due to endometrial cancer in 2009 and

two times insertion of retropubic slings was revealed. On examination she was a febrile and her vital signs within reasonable limits. Vaginal examination revealed a rigid pelvis with high-suspended, non-mobile urethra. There was a smooth, confined and hard mass on the symphysis pubis bone. Transvaginal ultrasound revealed round with central hyperechoic mass placed between the urethra and Symphysis pubic bone. The sling was placed correctly. An MRI was undertaken to investigate the mass further. This revealed a hypointense round lesion in the lower surface of Symphysis pubic, which expands to the urethra and displace it. The MRT showed no signs of metastasis by the history of endometrium cancer. The case was discussed in the Pelvic floor conference with the presence of an orthopedic college. A decision of surgical exploration was made. In Operation Theater, the suprapubic catheter was removed. A lower abdominal transverse incision was done. A Conversion to vaginal access was done, because of the severe adhesions in Cavum retzii. A transurethral catheter was inserted. A dissection between the urethra and Symphysis done carefully and a fragile mass was excised. The histopathological results showed degenerative changes with necrosis of the connective tissue around the symphysis pubis. The patient had an uneventful post-operative period. On the day after the surgery, she was able to obtain a normal voiding ability without any difficulty. The course of antibiotics was completed and the patient was discharged on the 4th postoperative day. At a clinic review in 3 months later, the patient exhibited no complain and was urinary continent.

DISCUSSION

Voiding dysfunction in female lacks a standard definition. It is defined by ICS as abnormal slow urine flow rates and abnormally high postvoid residuals.

VD in a female can be presented with both storage and voiding symptoms as well as those associated with urinary incontinence [3].

As explained in a large cross-sectional Internet survey including 15,861 women, the Presentation of VD varies between terminal dribbling 38.3%, feeling of incomplete bladder emptying in 27.4% and a weak stream in 20.1% [4].

VD might be due to either detrusor underactivity (DU), and/or BOO. Detrusor underactivity could be related to neurological, myogenic or mixed factors. Causes of BOO are either anatomical or functional [5]. BOO estimated to be 2.7-8% in females who have LUTS [6].

Genital prolapse is one of the anatomical causes, which related to VD in females. In a prospective study including 60 women with mean age of 52 years who had cystocele grade 1-2 in 35 women (58%) and grade 3-4 in 25 women (42%). In Urodynamics, two patients (4%) with grade 1 or 2 cystocele and 18 (58%) with grade 3 or 4 cystoceles ($p < 0.001$) showed a BOO. After placement of vaginal pessary, 17 women (94%) with grade 3 or 4 cystocele had normal free flow [7].

Iatrogenic BOO post-urinary incontinence surgeries are also reported. Post-operative VD after tension-free vaginal tape (TVT) insertion is reported in 3-15% of women [8].

Other anatomical causes of BOO like urethral stricture in females are infrequent and need a high degree of suspicion for the diagnosis [9]. Grotz A *et al.*, reported in their study an incidence of urethral stricture (13%), primary bladder neck obstruction (8%), learned voiding dysfunction (5%), and detrusor external sphincter dyssynergia (5%) [10].

Dysfunctional voiding and detrusor sphincter dyssynergia are considered the most common causes of functional BOO. Other rare causes like Fowler's syndrome and a primary bladder neck obstruction are reported [11].

VD is diagnosed by symptoms and urodynamic investigations. Coexisting of storage and voiding symptoms in female makes the diagnosis of VD challenging. The pathophysiology of urination in females is multifactorial as mobility of bladder neck, urethra and pelvic floor movements plus the presence of pelvic organ prolapse all play a role [12].

In Grotz A *et al.*, study, Bladder outlet obstruction was defined as a persistent, low, maximum "free" flow rate of < 12 mL/s in repeated non-invasive uroflow studies, combined with high detrusor pressure at a maximum flow ($p(\text{det.Q})(\text{max}) > 20$ cm H₂O) during detrusor pressure-uroflow studies. From the urodynamics database of 587 women 38(6.5%) diagnosed with BOO. The mean $p(\text{det. Q})(\text{max})$ was 37.2 ± 19.2 cm H₂O [10].

Kuo HC had another video urodynamic criteria to diagnose BOO. He defined BOO as the radiologic evidence of bladder outlet narrowing plus a voiding pressure greater than 35 cm H₂O and a maximum flow rate less than 15 mL/s or a voiding pressure greater than 40 cm H₂O [13].

Other studies reported other values of $p(\text{det. Qmax})$ and $Qmax$ between 35-60 and 9-15 respectively [14, 10].

Relieving the symptoms and avoiding developing the complication must be the crucial goal of a physician. As reaching the diagnosis of VD due to BOO mostly prolongs, an intermittent self-catheterization education or suprapubic catheter must be considered.

CONCLUSION

VD due to BOO is uncommon in women. Although the diagnosis is challenging but taking a good clinical history and applying a proper physical examination plus an accurate urodynamic test, are all crucial to reach the right diagnosis and proceed with the appropriate management.

Abbreviations

VD: voiding disorders
BOO: bladder outlet obstruction
DU: detrusor underactivity
LUTS: Lower urinary tract symptoms
ICS: International continence association

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