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ORIGINAL ARTICLE

Obstructive patterns in videourodynamic studies predict responses of female dysfunctional voiding treated with or without urethral botulinum toxin injection: a long-term follow-up study

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Abstract

Introduction and hypothesis We report long-term changes in VUDS profiles of women with dysfunctional voiding and investigate potential predictors for treatment response.

Methods Women with dysfunctional voiding and available VUDS data between November 1997 and June 2018 were enrolled for retrospective analysis. The patients were all treated with medication first. In refractory patients, urethral botulinum toxin was provided as an additional option. The primary outcome was the change of VUDS parameters between baseline and follow-up studies. The secondary outcomes were baseline parameters and clinical factors that were associated with the BOOI response (> 10 points of BOOI reduction).

Results A total of 195 women with DV were included in this study. The mean age was 54.5 years old. Sixty patients received urethral botulinum toxin injection. For all patients, Pdet decreased from 47.2 to 36.8 cm H_2O ($p < 0.0001$), and BOOI decreased from 26.4 to 17.7 ($p = 0.0001$). Patients with urethral injection had significantly smaller Qmax, voided volume, and voiding efficiency (VE) and significantly larger PVR and BOOI at baseline, indicating a severer obstruction in this group. The overall BOOI response rate was 44% (85/195). A higher baseline BOOI was associated with the BOOI response in multivariate analysis.

Conclusions In this long-term study of women with dysfunctional voiding, medical treatment with or without urethral botulinum toxin injection both resulted in reduction of Pdet and BOOI. A more prominent obstructive profile at baseline VUDS study was associated with a higher rate of BOOI response at follow-up study.

Keywords Videourodynamics · Dysfunctional voiding · Botulinum toxin

Introduction

Female voiding dysfunction is a common but poorly understood disorder. The prevalence of female voiding dysfunction ranged from 2.7% to 23% in previous reports [1–3]. A more recent prospective study reported that the prevalence of voiding dysfunction (defined by a flow rate < 15 ml/s) was 12.8% among 792 women with lower urinary tract symptoms (LUTS), and 87.2% of these voiding dysfunctions resulted from bladder outlet obstruction (BOO) [4]. Female BOO can be classified into anatomical, functional, and neurogenic BOO by the underlying etiology. Anatomical BOO can result from pelvic organ prolapse, anti-incontinence surgery, and urethral stricture. Functional BOO includes primary bladder neck dysfunction, dysfunctional voiding (DV), and Fowler's syndrome. Neurogenic BOO results from known neurological diseases damaging the brain stem or the spinal cord [5].

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Among all types of BOO in women, DV consists of approximately 17–32% of the cases [2, 4, 6]. DV is defined as an intermittent and/or fluctuating flow rate due to involuntary intermittent contractions of the peri-urethral striated muscle during voiding, in neurologically normal individuals [7]. It is characterized by increased external sphincter activity during the voiding phase, resulting in a typical spinning-down shape in voiding cystourethrography or videourodynamic studies (VUDS) (Fig. 1). Because there are no standard criteria for the diagnosis of female BOO [8, 9], the diagnosis of DV in women is even more difficult, requiring VUDS to determine the accurate level of obstruction.

There are several treatment options for female DV. Observation, pelvic floor muscle training, and medical therapy are common noninvasive managements. Medical therapy often includes α-blockers or baclofen to relieve bladder outlet obstruction, with antimuscarinics or β3 agonists for those with storage symptoms [5]. For those for whom medical therapy failed, botulinum toxin can be injected into the external sphincter to lower the urethral resistance further [10]. The rationale of these options has been based largely on the experience of treating male LUTS. Nonetheless, clinical evidence for the treatment of female BOO is scarce, and data from previous reports were inconsistent [11]. Due to the lack of standard care for the disease, treatment and follow-up are often incomplete in these patients.

Whereas VUDS characteristics of female DV have been described in the past [12, 13], long-term follow-up results of bladder function in these patients were seldom reported. The aims of our study are to report long-term changes in VUDS profiles of women with DV and to investigate potential predictors of treatment response.

Materials and methods

Ethical approval: This study was approved by the local institutional review board. Patients' informed consent was waived because of the retrospective nature of this study.

Patient enrollment

Between November 1997 and June 2018, all women with DV visiting a medical center for treatment were reviewed. The diagnosis of dysfunctional voiding was made by VUDS under fluoroscopy and multichannel urodynamic equipment. DV was defined as evident radiographic obstruction at the middle urethra with an open bladder neck. Only women with available baseline and follow-up VUDS data were enrolled. The current study includes the long-term follow-up result of the patients from a previous clinical trial [14].

VUDS definitions

The VUDS parameters were defined as the following: Bladder volume was recorded at first sensation of filling (FSF), full sensation (FS), and urge sensation (US). Bladder capacity was calculated by voided volume and post-voiding residual (PVR). Bladder compliance was calculated at full sensation (FS). Voiding efficiency (VE) was defined as voided volume divided by bladder capacity. Maximal flow rate (Qmax) and detrusor pressure at maximum flow rate (Pdet) were recorded, and the bladder outlet obstruction index (BOOI) was calculated by (Pdet − 2 × Qmax). Detrusor overactivity (DO) was defined as any detrusor contraction which has been understood by the patient during the filling phase of cystometry.

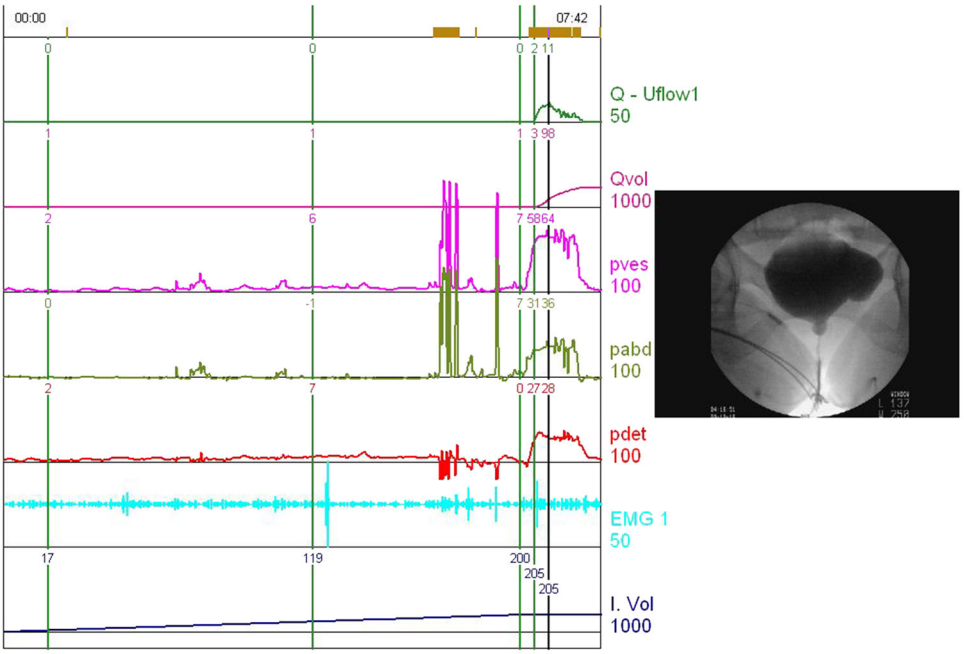

Fig. 1 A VUDS of a woman with dysfunctional voiding. The VUDS showed an elevated detrusor pressure during voiding phase with a low, fluctuating flow rate. Increased external sphincter activity during the voiding phase was noted on EMG. The voiding cystourethrography showed a typical spinning-down shape with a narrowed middle urethra

Medical treatment and urethral botulinum toxin injection

The patients were first treated medically. Medication included α-blockers, baclofen, antimuscarinics, and β3 agonists. Women with a 3-month history of medical refractory dysfunctional voiding were provided with the option of urethral botulinum injection. The use of botulinum toxin for an external sphincter is off-label. There were no pre-defined criteria based on symptom scores or UDS parameters. The techniques for urethral injection were described previously [14]. Briefly, each vial of onabotulinumtoxinA (100 U) was diluted to 5 ml with normal saline, yielding the concentration of 20 U/ml. In the lithotomy position and under general anesthesia, patients received injections of onabotulinumtoxinA solution in the urethral sphincter at five sites around the urethra except at the 6 o'clock position. Patients were discharged the following day if there were no complications. An oral antibiotic agent (cephalexin 500 mg every 6 h) was administered for 7 days.

Outcome assessment

The primary outcome was the change of VUDS parameters between baseline and follow-up studies. The secondary outcomes were baseline parameters and clinical factors that were associated with BOOI response, which was defined as more than 10 points of reduction in BOOI. For statistical analysis, the Student's *t* test was performed to compare numerical data, and the chi-square test was performed to compare categorical data. A generalized linear model was used to calculate the odds ratio and *p* value for each factor associated with BOOI response. Statistical analyses were performed using free software (R version 3.1.2). All statistical tests were two-tailed, with $p < 0.05$ indicating significance.

Results

A total of 195 women with DV were included in this study. The mean age was 54.5 ± 19.5 years old. The median interval between baseline and follow-up VUDS was 33.6 (interquartile range [IQR]: 12.2–88.6) months. The prevalence rates of comorbidities are shown in Table 1. Sixty patients received urethral botulinum. In the 60 patients who received urethral botulinum toxin injection, 31 had baclofen, 48 had α-blockers, 11 had β3 agonist, and 30 had antimuscarinics. In the 135 patients without urethral injection, 15 had baclofen, 57 had α-blockers, 30 had β3 agonist, and 61 had antimuscarinics. There were higher percentages of patients using baclofen in the urethral injection group (51.6% vs. 11.1%, $p < 0.0001$) as well as α-blockers (80% vs. 42.2%, $p < 0.0001$). The use of β3 agonist and antimuscarinics was similar between groups. The details of medications are described in Supplementary Table S1.

Table 1 Clinical characteristics of women with dysfunctional voiding

Comorbidity	Total = 195	%
HTN	83	43%
DM	66	34%
CAD	12	6%
CKD	10	5%
COPD	2	1%
PD	5	3%
CVA	14	7%
Dementia	4	2%

CAD: coronary artery disease, CKD: chronic kidney disease, COPD: chronic obstructive pulmonary disease, DM: diabetes mellitus HTN: hypertension, PD: Parkinson's disease

Overall, the most prevalent symptoms at baseline were frequency (60%), urgency (61%), difficult urination (36%), and retention (10%). Most symptoms decreased substantially (frequency 41%, urgency 43%, retention 4%) at the time of follow-up (Table 2). When comparing the overall changes of the VUDS parameters in all patients, significant decreases in the Pdet and BOOI were found between the baseline and follow-up visits. Pdet decreased from 47.2 to 36.8 cmH_2O ($p < 0.0001$), and BOOI decreased from 26.4 to 17.7 ($p = 0.0001$). The mean of BOOI change was 8.56 (SD = 30.7), and the median was 7 (IQR = 34). However, the values of the other VUDS parameters did not change significantly between the baseline and follow-up studies (Table 2). The prevalence rates of DO were similar (57.4% vs. 62.5%).

The baseline, follow-up, and interval changes of VUDS are presented in Table 3. The patients were stratified according to urethral botulinum injection. First, the baseline bladder volumes at different stages of sensation (FSF, FS, and US) were all larger in patients with urethral injection, indicating a less sensitive bladder in this group of patients. These volumes decreased significantly in the follow-up studies, resulting in similar bladder sensitivities in both groups. The bladder compliances were similar between groups and remained unchanged during the follow-up period. Second, patients with urethral injection had significantly smaller Qmax, voided volume, and voiding efficiency (VE) and significantly larger PVR and BOOI at baseline, indicating more severe obstruction in this group. Pdet was marginally higher but did not reach statistical significance. Among these voiding parameters, only Pdet and BOOI decreased significantly at follow-up studies. Although BOOI decreased in both groups, it remained significantly higher in the urethral injection group at the follow-up studies.

To investigate the factors associated with BOOI response after treatment, the patients were further divided into two groups based on their BOOI response (Table 4). The overall BOOI response rate was 44% (85/195). The baseline Pdet and

Table 2 Symptoms and VUDS parameters at baseline and follow-up: all patients

	Baseline		Follow-up		Change	*p* value
	N	%	N	%		
Symptoms						
Frequency	117	60%	80	41%	-19%	0.0002 *
Urgency	118	61%	83	43%	−18%	0.0004 *
Urge incontinence	36	18%	17	9%	−10%	0.0050 *
Nocturia	2	1%	2	1%	0%	1.0000
Difficult urination	71	36%	66	34%	−3%	0.5959
Retention	20	10%	8	4%	−6%	0.0186 *
Residual sensation	2	1%	3	2%	1%	0.6526
Bladder pain	10	5%	12	6%	1%	0.6607
Micturition pain	2	1%	2	1%	0%	1.0000
VUDS parameters						
FSF	125.0	75.0	128.7	76.9	3.66	0.5588
FS	190.5	100.6	189.2	104.9	−1.34	0.8651
US	226.0	118.0	215.5	118.4	−10.40	0.2252
Pdet	47.2	24.5	36.8	22.8	−10.38	0.0000 *
Compliance	60.7	61.5	57.8	59.9	−2.94	0.5955
Qmax	10.5	7.0	9.5	6.6	−0.91	0.0689
Volume	180.8	115.7	175.8	124.0	−4.97	0.5786
PVR	108.4	131.2	110.4	155.2	2.03	0.8575
Capacity	290.0	143.8	286.2	158.0	−3.71	0.7564
VE	0.66	0.32	0.65	0.32	−0.01	0.8051
BOOI	26.4	29.6	17.7	27.6	−8.56	0.0001 *

BOOI: bladder outlet obstruction index, FS: full sensation, FSF: first sensation of filling, Pdet: detrusor pressure at maximum flow rate, PVR: post-voiding residual, Qmax: maximal flow rate, SD: standard deviation, US: urge sensation, VE: voiding efficiency, *$p < 0.05$

BOOI were higher in the BOOI responsive group, whereas the Qmax and voided volume were smaller. Other VUDS parameters did not differ between responsive and non-responsive patients. To account for the effect of different treatments, multivariate analysis was performed to evaluate the possible predictive factors. After controlling urethral injection and other medications, a higher baseline BOOI remained significant for BOOI response (Table 5).

Discussion

In this long-term VUDS follow-up study of female DV patients, we had several important findings. First, the baseline VUDS profiles were different between the urethral injection group and medical treatment group. The urethral injection group had lower Qmax, larger PVR, and higher BOOI, indicating that the selection of treatment modality was influenced by disease severity (Table 3). As a result, caution should be taken when comparing the results between different treatments. Second, we found that there were significant decreases in Pdet and BOOI from baseline to follow-up VUDS in both groups. However, there were no significant improvements in Qmax, PVR, and VE after treatment. These results suggest that the effectiveness of treatment for DV can only be properly evaluated by pressure-flow study, instead of basic uroflowmetry (Table 2). Third, key voiding parameters, including Qmax, PVR, VE, and BOOI, were significantly different between the two groups, in both baseline and follow-up studies. The absolute difference between baseline and follow-up studies was similar for both treatment groups. These results indicated that the treatment outcome was largely determined by the baseline conditions of the patients (Table 3). Finally, some factors associated with treatment response were identified, including higher Pdet, lower Qmax, smaller voided volume, and higher BOOI (Table 4). Whether BOOI is associated with treatment response or is just related to treatment modalities needs to be addressed, so we conducted a multivariate analysis, controlling some important treatments. The result showed that baseline BOOI was associated with treatment response regardless of urethral injection, baclofen, and orα-blockers (Table 5). In summary, a more prominent obstructive VUDS profile predicts a higher rate of BOOI response.

Table 3 Comparison of VUDS parameters from baseline to follow-up in patients with or without urethral botulinum

Urethral botulinum		Yes ($N = 60$)		No ($N = 135$)		p value
		Mean	SD	Mean	SD	
FSF	Baseline	145.3	75.8	116.1	73.2	0.0135 *
	Follow-up	129.2	81.1	128.5	75.3	0.9601
	Δ	−16.1	85.0	12.4	87.2	0.0336 *
	p value (Δ)	0.1469		0.0991		
FS	Baseline	221.6	107.0	176.8	94.8	0.0062 *
	Follow-up	187.5	111.3	190.0	102.4	0.8823
	Δ	−34.1	109.2	13.2	108.6	0.0061 *
	p value (Δ)	0.0187	*	0.1596		
US	Baseline	253.9	120.1	213.6	115.4	0.0306 *
	Follow-up	206.3	118.3	219.6	118.6	0.4717
	Δ	−47.6	117.1	6.0	118.4	0.0040 *
	p value (Δ)	0.0026	*	0.5562		
Pdet	Baseline	53	34.3	44.7	18.2	0.0835
	Follow-up	40.2	28.0	35.4	20.0	0.2353
	Δ	−12.8	28.7	−9.3	21.8	0.4091
	p value (Δ)	0.0010	*	0.0000	*	
Compliance	Baseline	58.2	59.6	61.9	62.6	0.6897
	Follow-up	53.6	56.4	59.7	61.5	0.5000
	Δ	−4.5	61.6	−2.2	83.6	0.8282
	p value (Δ)	0.5689		0.7572		
Qmax	Baseline	8.48	6.5	11.4	7.0	0.0063 *
	Follow-up	7.57	6.6	10.4	6.5	0.0060 *
	Δ	−0.8	8.5	−1	6.1	0.8752
	p value (Δ)	0.4824		0.0699		
Volume	Baseline	147	108.1	195.8	116.2	0.0053 *
	Follow-up	129.9	110.0	196.2	124.8	0.0003 *
	Δ	−17	122.7	0.4	125.8	0.3654
	p value (Δ)	0.2864		0.9711		
PVR	Baseline	177.5	128.1	77.6	120.9	0.0000 *
	Follow-up	165.7	188.8	85.8	131.1	0.0039 *
	Δ	−11.8	174.9	8.2	149.7	0.4420
	p value (Δ)	0.6013		0.5252		
Capacity	Baseline	327.1	152.9	273.5	137.0	0.0218 *
	Follow-up	295.6	180.4	282.1	147.5	0.6106
	Δ	−31.4	157.8	8.6	170.2	0.1133
	p value (Δ)	0.1282		0.5583		
VE	Baseline	0.45	0.3	0.75	0.2	0.0000 *
	Follow-up	0.5	0.3	0.7	0.3	0.0000 *
	Δ	0.05	0.4	−0.02	0.3	0.1488
	p value (Δ)	0.3137		0.2285		
BOOI	Baseline	36.2	39.1	21.9	23.0	0.0101 *
	Follow-up	25	31.4	14.5	25.2	0.0246 *
	Δ	−11.2	36.7	−7.4	27.8	0.4734
	p value (Δ)	0.0213	*	0.0024	*	

BOOI: bladder outlet obstruction index, FS: full sensation, FSF: first sensation of filling, Pdet: detrusor pressure at maximum flow rate, PVR: post-voiding residual, Qmax: maximal flow rate, US: urge sensation, VE: voiding efficiency, Δ: change from baseline to follow-up, *$p < 0.05$

Table 4 Baseline VUDS characteristics and treatments received according to BOOI response

BOOI response	Yes ($N = 85$)		No ($N = 110$)		
Baseline	Mean	SD	Mean	SD	*p* value
Age	54.3	18.8	57.7	20	0.2362
FSF	126.1	84.7	124.2	66.9	0.8708
FS	191.5	110.4	189.7	92.7	0.9053
US	223.9	125.9	227.6	112.1	0.8322
Pdet	53.9	29.1	42.1	18.7	0.0014 *
Compliance	65.8	73.4	56.8	50.5	0.3357
Qmax	8.19	5.3	12.2	7.62	0.0000 *
Vol	161.5	117.1	195.7	112.9	0.0412 *
PVR	126.1	153.6	94.7	109.7	0.1120
Capacity	287.6	159.1	290.4	133.3	0.8968
VE	0.619	0.35	0.695	0.29	0.1115
BOOI	37.7	32.5	17.5	23.7	0.0000 *
DO	49	58%	63	57%	0.9582
Treatments	N	%	N	%	p value
Urethral botulinum	28	33%	32	29%	0.5635
α-Blockers	47	55%	58	53%	0.7214
Baclofen	25	29%	21	19%	0.0923
Antimuscarinic	39	46%	52	47%	0.8470
β3 Agonist	15	18%	26	24%	0.3088

BOOI: bladder outlet obstruction index, DO: detrusor overactivity, FS: full sensation, FSF: first sensation of filling, Pdet: detrusor pressure at maximum flow rate, PVR: post-voiding residual, Qmax: maximal flow rate, SD: standard deviation, US: urge sensation, VE: voiding efficiency, *$p < 0.05$

The diagnosis of DV is as difficult as other functional BOO in women; thus, the characteristics of DV in urodynamic studies (UDS) were rarely reported. Unlike male BOO, which has been clearly defined by UDS parameters, no standard in the diagnosis of female BOO has been established. Most studies used both Qmax and Pdet as basic diagnostic criteria. However, the cutoff point could range from 11 to 15 ml/s for Qmax [9, 15] and from 20 to 50 cmH_2O for Pdet [1, 9]. Additional requirements may include an index calculated from Qmax and Pdet [3] or radiological features [6]. Nomograms have been developed to account for Qmax and voided volume [16]. Although DV can be suspected from clinical presentation and the typically intermittent uroflowmetry pattern, the definitive diagnosis can only be made by electromyography (EMG) [17] or VUDS.

Table 5 Multivariate analysis for BOOI response

	OR	Lower 95%CI	Upper 95%CI	*p* value
BOOI	1.03	1.02	1.05	0.0000*
Urethral botulinum	0.81	0.37	1.73	0.5945
α-Blockers	0.80	0.37	1.73	0.5718
Baclofen	1.31	0.53	3.27	0.5579
Antimuscarinic	1.58	0.80	3.21	0.1960
β3 Agonist	0.89	0.39	2.01	0.7813

BOOI: bladder outlet obstruction index, *$p < 0.05$

The baseline VUDS parameters in our cohort revealed some characteristics of female DV patients. The mean Pdet was 47.2 cmH_2O, which was higher than the normal range by most criteria. The mean Qmax was 10.5, which was lower than most standards for female uroflowmetry. The high-pressure, low-flow pattern indicated a typically obstructed bladder outlet. In addition, the lower compliance and increased bladder sensitivity suggested the effects of chronic BOO, which could be associated with the high prevalence of DO (57.4%) in our cohort. These findings are consistent with previous reports [12, 18] and could be explained by the current consensus that DV is considered a learned behavior disorder in pelvic muscles [13].

In our study, Pdet reduced significantly from 47 to 36 cmH_2O. Whether the reduction of Pdet is clinically relevant is still unclear. The reduction of Pdet has always been an important indicator of the relief of BOO. For example, Pdet reduced from 71 to 59 cmH_2O in men receiving dutasteride for BPO [19]. Pdet reduced from 77 to 36 cmH_2O in men receiving laser enuleation of prostate [20]. As treatment response was rarely evaluated with pressure flow study in female BOO, further study is required to determine how much this reduction correlates with symptom improvement.

In the current study, we use BOOI reduction to represent the relief of obstruction. Although BOOI is extensively used in men, it is less used in women. The use of BOOI provides some advantages. First, it includes two of the most important elements in pressure flow studies, Pdet and Qmax, and both are widely used in various clinical trials to define female BOO. Second, it is simple to calculate, easy to use, and familiar to urologists. The diagnostic power of BOOI for female BOO was shown to be superior to Pdet alone by Gravina et al. in 2007 [21], followed by some other studies. Since there is no widely accepted change in BOOI for women that would denote clinical improvement, we defined the BOOI response as having a reduction of > 10 points. Considering the mean of BOOI reduction was 8.56 (SD = 30.7) and the median was 7 (IQR = 34), we think > 10 points of BOOI reduction could be considered responsive.

The efficacy of medical treatment specifically for female DV has rarely been reported. In a few randomized trials using ether α-blockers for female BOO, the responses varied between studies, and no subgroup analysis has been done for different types of BOO. In a randomized double-blind trial, using tamsulosin vs. placebo for women with LUTS, Pummangura et al. reported a significant improvement of the International Prostate Symptom Score (IPSS) in the tamsulosin group vs. placebo group (−5.6 vs. –2.6), but the change in Qmax was no different from the placebo group's

[22]. In an open-label, randomized trial using tamsulosin with or without tolterodine, the Qmax increased from 10 ml/s to 13 ml/s in both groups [23]. In one retrospective study for female bladder neck dysfunction, Pdet decreased from 79 to 59 in 1 month of tamsulosin treatment [24]. Low et al. reported superior outcomes of terazosin vs. placebo in a double-blind randomized trial; however, the baseline Qmax was >20 ml/s, and only IPSS, but not Qmax and PVR, was significantly better [25]. In a randomized control trial using alfuzosin for 8 weeks, symptom scores, voiding diaries, Qmax, and PVR were not significantly different between the alfuzosin and placebo group [26]. Two studies investigated the effect of baclofen in female BOO. Xu et al. reported superior outcomes with baclofen vs. placebo in a double-blind randomized trial in EMG-confirmed female patients with DV and reported improvements in voiding diary and EMG scores [27]. In an observational study, Chen reported an increase of Qmax from 10.3 to 11.6 after 12 weeks of baclofen treatment for female BOO [28]. In most placebo-controlled studies, α-blockers or baclofen improved IPSS but not Qmax or PVR in female DV or BOO. These findings are consistent with our results, in which treatment response could be detected better with Pdet and BOOI, but not Qmax or PVR.

Botulinum toxin has been used in various neurogenic lower urinary tract disorders, and the efficacy is well established in neurogenic detrusor overactivity [10]. In 1988, Dykstra et al. reported the first results of urethral sphincter injection with botulinum toxin to treat detrusor-sphincter dyssynergia in patients with spinal cord injury [29]. In 1997, Steinhardt et al. reported its first application for dysfunctional voiding [30]. After urethral injection, successful self-voiding could be achieved in various cases of neurogenic or non-neurogenic urinary retention [31], and the Pdet and urethral resistance could be reduced significantly [32]. There has been only one randomized controlled trial for non-neurogenic voiding dysfunction in men and women. Whereas the subjective and objective parameters improved after urethral injection, the efficacy was similar between botulinum toxin and placebo [14].

The advantages of our study include a large cohort of women with dysfunctional voiding diagnosed with VUDS, a long-term follow-up period up to 10 years, and the use of multivariate analysis to control possible confounding factors. There are two major limitations in our study. First, the diversity in medical treatments was a great limitation. As these patients often have complex symptoms, it is quite uncommon to provide monotherapy outside of clinical trials. In addition to α-blockers and baclofen, antimuscarinics are commonly used for female BOO [5]. The main reason for the use of antimuscarinics was not only to relieve BOO-related storage symptoms, but also to reduce the urethral sphincter hyperactivity by decreasing the detrusor overactivity. It is postulated that increased detrusor overactivity can enhance dysfunctional voiding by the guarding effect. In our cohort, there were about 60% with storage symptoms and 41% with DO at baseline in the medical treatment group, indicating high prevalence of concomitant storage dysfunction. Second, due to the retrospective nature of the study design, the differences between the two groups resulted largely from patient selection. This study should not be viewed as a head-to-head comparison. A randomized control trial with specific inclusion criteria and pre-defined sub-group analysis is required to confirm the efficacy and select the best candidates for urethral injection.

Conclusions

In this long-term study of women with dysfunctional voiding, medical treatment with or without urethral botulinum toxin injection both resulted in reduction of Pdet and BOOI. A more prominent obstructive profile, including higher Pdet, lower Qmax, smaller voided volume, and higher BOOI at baseline VUDS, was associated with a higher rate of BOOI response at follow-up study.

Authors' contribution PM Chow: Manuscript writing, Data management, Data analysis.
SM Hsiao: Data analysis.
HC Kuo: Project development.

Compliance with ethical standards

Conflict of interest None.

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