Clinical and urodynamic characteristics of underactive bladder

Data analysis of 1726 cases from a single center

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Abstract
There have been no universally accepted criteria and have been established for classification of underactive bladder (UAB) at present. Thus, the study described the comprehensive clinical and urodynamic characteristics of UAB in patients with lower urinary tract symptoms.

A total of 1726 patients (1259 men and 467 women; 6–88 years old) who were admitted to our center with a diagnosis of UAB were included in this retrospective study. It was due to the type of rehabilitation hospital, so higher percentage of neurological patients were included. The demographics, clinical characteristics, and urodynamic recordings were reviewed. The clinical characteristics and urodynamic findings of UAB were further classified.

For the etiologic analysis, UAB with aging and without clear causes accounted for 11.5% of cases (199/1726), UAB with bladder outflow obstruction accounted for 2.6% (45/1726), and UAB acting on the nerve pathway of the voiding reflex accounted for 84.6% (1460/1726). There were a number of cases (1.3% [22/1726]) which had > 2 factors assigned. For studies involving urodynamic findings and clinical symptoms, the percentage of patients with detrusor hyperreflexia with impaired contractility (DHIC), detrusor underactivity (DU), and acontractile detrusor (AcD) was 0.7%, 5.6%, and 93.7%, respectively.

UAB can be classified into 4 types based on possible etiologic mechanisms (idiopathic, myogenic, neurogenic, and integrative). Based on urodynamic findings and symptoms, UAB can be classified into 3 types (DU, AcD, and DHIC). The classification of UAB can provide a reasonable basis for the future research.

Abbreviations: AcD = acontractile detrusor, BOO = bladder outflow obstruction, DO = detrusor overactivity, DU = detrusor underactivity, DHIC = detrusor hyperreflexia with impaired contractility, ICS = International Continence Society, LUTD = lower urinary tract dysfunction, LUTS = lower urinary tract symptoms, OAB = overactive bladder, PFS = pressure-flow study, Pdet = detrusor pressure, SCI = spinal cord injury, UAB = underactive bladder.

Keywords: detrusor underactivity, etiology, underactive bladder, urodynamics

1. Introduction
Detrusor underactivity (DU) is a form of common lower urinary tract dysfunction (LUTD) that occurs in approximately 20% to 25% of all patients with lower urinary tract symptoms (LUTS).[1] Although the International Continence Society (ICS) standardization report defines DU as “a contraction of reduced strength and/or duration, resulting in prolonged bladder emptying and/or failure to achieve complete bladder emptying within a normal time span,”[2] it is a urodynamic definition not based on symptoms. Underactive bladder (UAB) focuses on symptoms that patients find bothersome and raises the profile of the important clinical condition.[3] Chapple et al proposed that a definition of a symptom complex for UAB analogous to the ICS definition of overactive bladder (OAB) syndrome would be of potential clinical value: UAB is a symptom complex suggestive of DU and is usually characterized by a prolonged urination time with or without a sensation of incomplete bladder emptying, usually with hesitancy, reduced sensation on filling, and a slow stream.[4]

Because the underlying pathophysiology of UAB is multifactorial, a single medication that focuses on a single mechanism could not be effective for others. Therefore, it is necessary to be clear on its classification based on possible etiologic mechanisms induced by various risk factors; however, one question is that no universally accepted criteria have been established for classification of UAB at present. Urodynamic studies of UAB can help us better understand the nature of UAB, another question is whether or not UAB presents invariable urodynamic modality is unknown. Recently a review described the possible etiology and made an initial classification of UAB.[5] Based on this, the purpose of this study was to analyze the etiology, urodynamic findings, and clinical symptoms in patients with UAB from our database as detailed as possible, make a reasonable classification of UAB, and clarify the aforementioned questions.
2. Methods

2.1. Patient selection

We retrospectively reviewed the medical records of 4,538 consecutive patients who had LUTS and underwent video-urodynamic examinations between December 2004 and January 2016 at our institution. The study was approved by the Institutional Review Board at the China Rehabilitation Research Centre (No. 2015-K-066). All of the patients underwent detailed physical examinations, comprehensive history taking, and invasive video-urodynamic examinations. Clinical urodynamic practice was performed according to the ICS Good Urodynamic Practice standards.[6] All measurements were obtained using a Triton urodynamic analyzer (LABORIE, Mississauga, ON, Canada). A double-lumen transurethral catheter (7F, Cook, Bloomington, IN) was used for filling and to record vesical pressure. Abdominal pressure was measured with a balloon catheter (12F, Cook). The residual urine in the bladder was drained before cystometry. Saline with contrast medium at room temperature was infused at 10 to 30 mL/min. The type of detrusor dysfunction was evaluated during the urodynamic examination and X-ray images of the bladder were also obtained. A pressure-flow study (PFS) was performed if the patient could void. Each patient underwent the urodynamic test once.

2.2. Patient evaluation

UAB was diagnosed according to symptoms.[14] The inclusion criteria for this study were as follows: prolonged urination time with or without a sensation of incomplete bladder emptying, hesitancy, reduced sensation on filling, and a slow stream; decreased sensation of the need to urinate, impaired or absent bladder sensation, or high postvoid residual volume; and emptying bladder by catheterization or voiding with straining or increased abdominal pressure. The exclusion criteria were as follows: acute urinary tract infection; bladder tumor or stone before operations; spinal cord injury (SCI) in spinal shock period; urethral stricture; therapies for UAB in order to artiﬁcially increase bladder capacity including augmentation cystoplasty, recent (in 6 months) botulinum toxin injection, sacral neuromodulation, and so on; and pure bladder outﬂow obstruction (BOO).

In this study, the Schaefer nomogram was used to grade detrusor contractility.[17] Detrusor contraction strength below W+ according to the Schaefer nomogram is regarded as weak detrusor contractility if the patient has urine flow during the PFS. If there is no urine flow during the PFS, DU is defined as detrusor pressure (Pdet) ≤ 40 cm H₂O.[18,19] BOO combined with DU was diagnosed by PFS, urinary pressure proﬁle, and cystoscopy. We reviewed and analyzed the urodynamic findings for the included patients according to the above-mentioned standards. We also reviewed the medical histories of the included patients in detail and recorded the possible etiologies leading to UAB. UAB was classiﬁed according to diverse etiologies with different pathogenic mechanisms. LUTD resulting in UAB was clariﬁed and classiﬁed by urodynamic ﬁndings.

2.3. Statistical analysis

Data are expressed as percentage (%).

3. Results

During the period from December 2004 to January 2016, there were 4,538 consecutive patients with LUTS who sought evaluation at the urodynamic center in our department. A total of 1,726 patients with UAB (1,259 men and 467 women; 6–88 years old) were included into the study cohort. By analyzing the related etiology, many risk factors causing UAB were found (Table 1). In some patients, the causes of UAB were not been identiﬁed, thus UAB may be classiﬁed as idiopathic due to the lack of an apparent etiology. It accounted for 11.3% (199/1726) of the cases. Patients with BOO leading to UAB had myogenic UAB, which accounted for 2.6% (45/1726) of cases. Patients in whom risk factors leading to UAB acted on the neurologic etiology had neurogenic UAB, which accounted for 84.6% (1460/1726) of cases. This type of UAB was further divided into 3 subtypes, as follows: nervous system factors (87.4% [1276/1460]); infectious factors involving the nervous system (0.5% [7/1460]); and iatrogenic factors (12.1% [177/1460]) (Table 2). The nervous system factors contributed to the majority of UAB cases. UAB patients with ≥2 factors were referred to as integrative, which occurred 1.3% (22/1726) of cases.

The urodynamic ﬁndings included reduced detrusor contractility (with or without detrusor overactivity [DO]) during the ﬁlling phase) or absent detrusor contraction. The patients might have urine ﬂow or complete urinary retention (Table 3). Among 1,726 UAB patients, the percentage of detrusor hyperreﬂexia with impaired contractility (DHIC), DU, and acontractile detrusor (AcD) was 0.7%, 5.6%, and 93.7%, respectively. Among the 3 groups of patients, the symptom presentations were different, as follows: DU with typical symptoms of the above-mentioned UAB; DHIC with UAB and OAB symptoms; and AcD with urinary retention and emptying bladder by catheterization or voiding with straining or increased abdominal pressure.

4. Discussion

Few studies have focused on the etiologic classiﬁcation of UAB. Osman et al proposed a classiﬁcation based on etiologic factors and classiﬁed UAB into 4 types (idiopathic, neurogenic, myogenic, and iatrogenic).[10] But iatrogenic factors often resulted in injury to the peripheral pelvic plexus and it should be classiﬁed as neurogenic. Meanwhile, some patients could have 2 or more etiologies. This might be labeled as integrative. Tyagi et al[11] classiﬁed UAB into 3 types based on the mechanisms underlying UAB (neurogenic, myogenic, and integrative). But they neglected many aged patients with the symptoms of UAB who had no apparent causes. This might be labeled as idiopathic. We proposed a similar but distinct classiﬁcation based on the possible causes (idiopathic, myogenic, neurogenic, and integrative) (Table 1). van Koeveringe et al[12] deﬁned idiopathic UAB as an age-related decrease in detrusor contractility or other inapparent causes, but BOO with age was not excluded. Kuo[13] deﬁned idiopathic UAB as no evident neuropathy, no functional or anatomic bladder outlet obstruction, low or no Pdet combined
patients were idiopathic in the current study. The characteristics of UAB might be labeled as idiopathic. Cucchi et al.[14] thought that neurogenic factors or a combination of 2 or more neurogenic factors. Indeed, integrative UAB could be the combination of myogenic and neurogenic factors or a combination of 2 or more neurogenic factors. Because the etiologies leading to UAB are not understood completely at present, we also propose a classification based on urodynamic findings and symptoms and classify UAB into 3 types (DU, AcD, and DHIC) (Table 3). DU means that detrusor contraction function is impaired. Patients with DU are able to void (emptying completely or incompletely) or no urine flow (retention) during the urodynamic examination. AcD indicates that there are no detrusor contractions during urodynamic studies. The logical assumption is that AcD is the extreme of DU during progression.[13] Patients with AcD have urinary retention, and the emptying bladder is performed by straining, increased abdominal pressure, or catheterization. Most patients (93.7%) in this study had characteristics of AcD. The possible reasons may be the following: our center is affiliated with a rehabilitation hospital and most of the patients have different degrees of nervous system lesions, thus the neurogenic bladder population is considerable; and our center is a well-known center in neuro-urology, and most of the patients have serious conditions and visit us in an advanced stage. DHIC is defined as DO during the filling phase and underactive detrusor contractions during the voiding phase with or without urine flow.

### Table 2

| Factors leading to neurogenic underactive bladder in 1460 patients. |
|---------------------------------------------------------------|
| **Subtype**                                       | **Possible factors** | **N (%)** |
| Nervous system factors                                      |                    |          |
| Spinal cord abnormality                                     | Spinal cord injury  | 992 (67.9) |
|                                                           | Myelitis            | 25 (1.7)  |
|                                                           | Syringomyelia       | 2 (0.1)   |
|                                                           | Myelocyst           | 1 (0.07)  |
| Congenital dysplasia                                        | Tethered cord syndrome | 25 (1.7) |
|                                                           | Spina bifida       | 25 (1.7)  |
|                                                           | Spinal meningioma   | 91 (6.2)  |
|                                                           | Recessive hiatus sacralis | 21 (1.4) |
| Cerebrovascular accident                                    | Cerebral hemorhaghe | 18 (1.2)  |
|                                                           | Cerebral infarction | 15 (1.0)  |
| Others                                                      | Herniated disk      | 17 (1.2)  |
|                                                           | Spinal canal stenosis | 3 (0.2) |
|                                                           | Brain trauma        | 9 (0.6)   |
|                                                           | Cerebral palcy      | 5 (0.3)   |
|                                                           | Multiple sclerosis  | 2 (0.1)   |
|                                                           | Multiple system atrophy | 2 (0.1) |
|                                                           | Diabetes mellitus   | 6 (0.4)   |
|                                                           | Pelvic fracture     | 17 (1.2)  |
| Infectious factors                                          | Herpes zoster       | 2 (0.1)   |
|                                                           | Meningitis          | 5 (0.3)   |
|                                                           | Poliomyelitis       | 2 (0.1)   |
| Iatrogenic factors                                          | Brain and spinal surgery | 48 (3.3) |
|                                                           | Herniated disk surgery | 48 (3.3) |
|                                                           | Centrum surgery     | 46 (3.2)  |
|                                                           | Spinal canal surgery | 20 (1.4)  |
|                                                           | Spinal cord surgery | 6 (0.4)   |
|                                                           | Craniocelebral surgery | 8 (0.5) |
| Pelvic surgery                                              | Prostatectomy       | 9 (0.6)   |
|                                                           | Incision of bladder cancer | 3 (0.2) |
|                                                           | Cervical cancer surgery | 6 (0.4) |
|                                                           | Hysteromyectomy      | 3 (0.2)   |
|                                                           | Hysterectomy        | 11 (0.7)  |
|                                                           | Colporrhaphy        | 2 (0.1)   |
|                                                           | Rectal cancer surgery | 3 (0.2) |
|                                                           | Intraspinal anesthesia | 8 (0.5) |
|                                                           | General anesthesia  | 2 (0.1)   |

### Table 3

| Urodynamic classification of underactive bladder in 1726 patients. |
|---------------------------------------------------------------|
| **Type**                                              | **Urodynamic findings and symptoms** | **N (%)** |
| Detrusor hyperreflexia with impaired contractility         | Detrusor overactivity during the filling phase and reduced detrusor contractions during the voiding phase | 13 (0.7) |
| Detrusor underactivity                                    | Reduced detrusor contraction strength with or without urine flow | 96 (5.6) |
| Acontractile detrusor                                      | No detrusor contractions during micturition with urine retention | 1617 (93.7) |
voiding phase, thus a weak detrusor.\textsuperscript{[18]} DHIC presents as a combination of DU and DO (UAB and OAB symptoms). We found that 0.7\% (13/1726) of patients with UAB demonstrated urodynamic manifestations of DHIC. It is important to identify DHIC with urodynamic testing because treating patients with anticholinergic drugs may worsen bladder emptying leading to urine retention and/or urinary tract infection.\textsuperscript{[19]}

There have been no studies on the relevant issues with large patient numbers. We propose classification based on etiology, urodynamic findings, and symptoms. This study had some limitations. The study was a single center, retrospective data analysis. The data were derived from the rehabilitation hospital and had bias. Factors, including patient position, unfamiliar environment, and intraurethral catheter, can influence the urodynamic results and may result in more AcD. A multicenter, community-dwelling population, prospective study should be performed to validate our classification of UAB.

5. Conclusion

UAB has raised considerable controversy in many areas at present. Due to its complexity of clinical categorization, we face a new challenge in the field of functional urology. Based on the large sample data, we classified UAB into 4 types based on etiology, and into 3 types based on symptoms and urodynamic findings in this retrospective study. The classification can provide a reasonable basis for future research.

References

[1] Seki N, Kai N, Seguchi H, et al. Predictive regarding outcome after transurethral resection for prostatic adenoma associated with detrusor underactivity. Urology 2006;67:306–10.
[2] Abrams P, Cardozo L, Fall M, et al. The standardisation of terminology of lower urinary tract function: report from the Standardisation Subcommittee of the International Continence Society. Am J Obstet Gynecol 2002;187:116–26.
[3] Chancellor MB, Kaufman J. Case for pharmacotherapy development for underactive bladder. Urology 2008;72:966–7.
[4] Chapple CR, Osman NI, Birder L, et al. The underactive bladder: a new clinical concept? Eur Urol 2015;68:351–3.
[5] Li X, Liao L. Updates of underactive bladder: a review of the recent literature. Int Urol Nephrol 2016;48:919–30.
[6] Schäfer W, Abrams P, Liao L, et al. Good urodynamic practices: uroflowmetry, filling cystometry, and pressure-flow studies. Neurourol Urodyn 2002;21:261–74.
[7] Schaefer W. Basic principles and clinical application of advanced analysis of bladder voiding function. Urol Clin N Am 1990;17:553–66.
[8] Jeong SJ, Kim HJ, Lee YJ, et al. Prevalence and clinical features of detrusor underactivity among elderly with lower urinary tract symptoms: a comparison between men and women. Korean J Urol 2012;53:342–8.
[9] Thomas AW, Cannon A, Bartlett E, et al. The natural history of lower urinary tract dysfunction in men: the influence of detrusor underactivity on the outcome after transurethral resection of the prostate with a minimum 10-year urodynamic follow-up. BJU Int 2004;93:745–50.
[10] Osman NI, Chapple CR, Abrams P, et al. Detrusor underactivity and the underactive bladder: a new clinical entity? A review of current terminology, definitions, epidemiology, aetiology, and diagnosis. Eur Urol 2014;65:389–98.
[11] Tyagi P, Smith PP, Kuchel GA, et al. Pathophysiology and animal modeling of underactive bladder. Int Urol Nephrol 2014;46(suppl 1): S11–21.
[12] van Koeveringe GA, Vahabi B, Andersson KE, et al. Detrusor underactivity: a plea for new approaches to a common bladder dysfunction. Neurourol Urodyn 2011;30:723–8.
[13] Kuo HC. Recovery of detrusor function after urethral botulinum A toxin injection in patients with idiopathic low detrusor contractility and voiding dysfunction. Urology 2007;69:57–61.
[14] Cucchi A, Quaglini S, Rovereto B. Development of idiopathic detrusor underactivity in women: from isolated decrease in contraction velocity to obvious impairment of voiding function. Urology 2008;71:844–8.
[15] Belenky A, Azerbadian Y, Cohen M, et al. Detrusor resistive index evaluated by Doppler ultrasonography as a potential indicator of bladder outlet obstruction. Urology 2003;62:647–50.
[16] Lee JG, Shim KS, Koh SK. Incidence of detrusor underactivity in men with prostatum older than 50 years. Korean J Urol 1999;40:347–52.
[17] Miyazato M, Yoshimura N, Chancellor MB. The other bladder syndrome: underactive bladder. Rev Urol 2013;15:11–22.
[18] Stav K, Shilo Y, Zisman A, et al. Comparison of lower urinary tract symptoms between women with detrusor overactivity and impaired contractility, and detrusor overactivity and preserved contractility. J Urol 2013;189:2175–8.
[19] Kitta T, Mitsui T, Kanno Y, et al. Postoperative detrusor contractility temporarily decreases in patients undergoing pelvic organ prolapse surgery. Int J Urol 2015;22:201–5.