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Review Article

Clinical presentation and underlying pathophysiology of an underactive bladder

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ABSTRACT

Detrusor underactivity (DU) is frequently encountered in elderly patients with chronic medical or neurological diseases. DU causes chronic urinary retention or large postvoid residual urine that is usually difficult to manage. The pathophysiology of DU may involve neurogenic, myogenic, and bladder outlet pathologies. Recent studies also reveal that urothelial dysfunction of the urinary bladder may be associated with impaired bladder sensation as well as impaired detrusor contractility. This article reviews recent research on the prevalence, pathophysiology, and clinical management of DU. Comprehensive clinical investigations and basic research may provide a better understanding and effective treatment for this common but difficult bladder disorder.

1. Introduction

Chronic urinary retention is a debilitating bladder disorder that negatively impacts quality of life and also threatens health. Chronic urinary retention is frequently encountered and is difficult to manage in elderly patients with chronic medical diseases, such as diabetes mellitus (DM), and chronic heart failure or neurological diseases, such as cerebrovascular accident, Parkinson's disease, and dementia. Urinary tract infection (UTI) and renal function deterioration will develop if the bladder condition is not properly managed with clean intermittent catheterization or an indwelling Foley catheter. The pathophysiology of chronic urinary retention may involve neurogenic, myogenic, and bladder outlet pathologies [1]. Recent studies have also revealed that urothelial dysfunction of the urinary bladder may be associated with impaired bladder sensation as well as impaired detrusor contractility [2]. Furthermore, urethral mucosal dysfunction and smooth muscle hyperactivity of the bladder neck and the urethra might also play important roles in the initiation of micturition. Research is needed to explore the pathophysiology of detrusor underactivity (DU) and underactive bladder (UAB).

2. Definition of underactive bladder

The International Continence Society 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 reasonable time [3]. Patients with UAB/DU usually have a diminished sensation of bladder fullness or urgency and cannot contract the detrusor sufficiently to complete bladder emptying. Urodynamic study of UAB/DU may be characterized by a noncontractile detrusor, low pressure, or poorly sustained detrusor contraction in association with a poor flow rate with or without a large post-void residual (PVR) volume [4]. Patients with UAB/DU usually void with abdominal straining and an intermittent flow pattern is noted. The bladder sensation may be normal or reduced in sensing a first or urge sensation [5]. Some patients with UAB/DU may have both detrusor hyperactivity and inadequate contractility (DHIC), resulting in urgency incontinence and a large PVR [6]. In patients with UAB/DU, the intrinsic detrusor contraction speed is more compromised than intrinsic strength. Patients with UAB/DU can be divided into the following three groups according to the urodynamic findings: (1) low maximum detrusor contraction velocity, low isovolumetric detrusor pressure, and bladder emptying efficiency of <67%; (2) low maximum detrusor contraction velocity, low isovolumetric detrusor pressure, and bladder emptying efficiency of ≥67%; and (3) low maximum contraction velocity, normal isovolumetric detrusor pressure, and bladder emptying efficiency of ≥67% [7]. These urodynamic findings are also found in women

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with idiopathic UAB/DU [8]. The underlying pathophysiology for each group might be different and can be attributed to varying detrusor muscle contractility and bladder outlet resistance in individual patients.

3. Prevalence of UAB/DU

UAB/DU is a common urological problem in elderly patients presenting with urinary retention and lower urinary tract symptoms (LUTS). DU has been found in nearly two-thirds of incontinent institutionalized elderly people [9]. The incidence and prevalence of UAB/DU is highly dependent on the definition and the availability of diagnostic tests. In a retrospective study, 40.2% of men and 13.3% of women undergoing urodynamic study for LUTS were classified as having DU [10]. In urodynamic evaluation of patients with non-neurogenic LUTS, DU was found in 9–48% of men and 12–45% of older women [11]. One urodynamic pressure flow study revealed that 41% of elderly men with symptoms of difficult bladder emptying had an obstructive high pressure low flow pattern, 28.2% had an underactive detrusor contractility pattern, 20.5% had a mixed obstructive and underactive detrusor type, and 10.3% had a normal pattern [12]. The prevalence of bladder outlet obstruction (BOO) voiding difficulty and DU in female urology patients who visited several urology clinics in nine hospitals was 87.2% and 12.8%, respectively [13]. Gotoh et al [14] found impaired detrusor contraction in 81.9% and BOO in 14.8% of women with impaired bladder emptying.

UAB/DU usually occurs in patients with spinal cord injury (central neuropathy) or post pelvic surgery (peripheral neuropathy). In 100 women with urinary retention, Sakakibara et al [15] found the underlying diseases included multiple system atrophy, multiple sclerosis, cervical/thoracic tumors, and lumbar spondylosis. DU is also common in older patients, in those with general weakness and medical diseases such as DM, debilitating disease, and cancer in the terminal stages, and after major surgery [16]. Women with diabetic voiding dysfunction were found to have a longer duration of DM than those with an overactive bladder. Ageing and UTI are two independent factors contributing to impaired voiding function and diabetic bladder dysfunction [17]. A large proportion of patients with diabetic cystopathy were found to have electrophysiological (EP) evidence of neuropathy, which can moderately predict the presence of cystopathy [18]. DM can affect the bladder, presumably via peripheral pathogenetic mechanisms that induce DHIC. Patients with DU showed impaired emptying function and decreased sensation on cystometry and intravesical current perception threshold testing [19].

UAB/DU and DHIC were also common (15% and 1%, respectively) in patients with recent ischemic stroke [20]. Although UAB/DU may occur in any age group, both conditions have an age-associated prevalence. The actual contribution of ageing on detrusor contractility, however, has not been conclusively demonstrated. In one study, UAB/DU was observed in 41% of patients presenting with urinary incontinence or LUTS following radical prostatectomy [21]. UAB/DU may be chronic or temporary. In clinical practice, we have observed patients with BOO and normal detrusor contractility who have developed transient UAB/DU after transurethral resection of the prostate or immediately after a minor stroke. These patients may regain spontaneous voiding within 1–3 months. However, some patients might develop chronic DU and spontaneous voiding may not return in the short term. There must be some underlying pathogenesis for the development of transient UAB/DU, such as detrusor muscle damage or neurological inhibition, which interferes with the integration of musculomucosal mechanoreceptors, mucosal mechanoreceptors, and chemoreceptors [22].

We have previously investigated videourodynamic characteristics in men and women with LUTS refractory to conventional medication. DU was noted in 146 (5.2%) of 2,831 men with LUTS and DHIC in 150 (5.3%) of 2,831 men with LUTS. The incidence of DHIC increased with ageing; however, the incidence of DU with age was not significantly different from that in other vesicourethral dysfunctions such as detrusor overactivity, bladder neck dysfunction and benign prostatic obstruction. DU was noted in 108 (36.1%) of 299 patients with a PVR >250 mL and DHIC in 44 (14.7%) of 299 patients with a PVR >250 mL. The bladder sensation of filling and fullness were significantly reduced compared with that in patients with BOO. The incidence of DU in 1,333 female patients with LUTS was 11.4% and DHIC was noted in 4.3% (Table 1). The medical comorbidity in 118 women presenting with chronic urinary retention or a large PVR included DM (43, 30.7%), hypertension (52, 37.1%), coronary arterial disease (17, 12.1%), chronic obstructive pulmonary disease (3, 2.1%) and chronic kidney disease (25, 17.9%) [23].

4. Pathophysiology of UAB/DU

The pathogenesis of UAB/DU is likely to be multifactorial [1]. UAB/DU may be classified into myogenic and neurogenic mechanisms [24]. The causes of UAB/DU include DM, BOO, ageing, neurological diseases, spinal cord lesions, and pelvic plexus and infectious neurological problems [25]. UAB/DU can result from damage to the bladder afferent pathways, bladder efferent pathways, or lumbosacral spinal cord or be due to pure detrusor failure [26]. There is a need for longitudinal patient data to define risk factors, develop screening tools, and establish an animal model for translational research into UAB/DU [27].

4.1. UAB/DU in patients with chronic urinary retention may be caused by latent neuropathy

In videourodynamic study (VUDS), patients with chronic urinary retention usually have low voiding pressure or DU without significant BOO, defined as non-obstructive voiding dysfunction. We retrospectively collected 60 patients, who were diagnosed with nonobstructive voiding dysfunction on VUDS and had received lower urinary tract EP studies. EP studies included examination of the bulbocavernous reflex by electrical stimulation, electromyography of the external urethral sphincter (EUS), and nerve conduction velocity (NCV) study of the internal pudendal nerve. In electromyography study, denervation, reinnervation changes, and reduced recruitment of the EUS was observed in 21.7%, 71.7%, and 86.7% of patients, respectively. Decreased amplitude of the internal pudendal nerve was noted in NCV study in 73.3% of the patients. Significant sacral neuropathy (e.g., saddle anesthesia) was present on neurological examination in 19 out of 60 patients (31.7%).

Table 1
Prevalence of detrusor underactivity in women with lower urinary tract symptoms.

Normal findings	272 (16.9)
Sensory disorders (459)	
• Bladder oversensitivity	289 (18.0)
• Suggestive of interstitial cystitis/painful bladder syndrome	170 (10.6)
Motor disorders (560)	
• Idiopathic detrusor overactivity	308 (19.2)
• Detrusor hyperactivity and inadequate contractility	69 (4.3)
• Detrusor underactivity	183 (11.4)
Bladder outlet disorders (314)	
• Bladder neck dysfunction	27 (1.7)
• Dysfunctional voiding	168 (10.5)
• Urethral stricture	17 (1.1)
• Poor relaxation of pelvic floor muscles	102 (6.4)

Data are presented as *n* (%).