Practical urodynamics

Urodynamic characteristics of lower urinary tract dysfunction in patients with Parkinson's disease

Chia-Hao Kuei, Chun-Hou Liao, Hann-Chorng Kuo

1. Introduction

Parkinson's disease (PD) is the second most common neurodegenerative disease, after Alzheimer's disease. The prevalence of PD is rising, currently affecting 1 million people in the United States, and approximately 50,000 new cases are reported there annually. In Taiwan, the prevalence was 340 per 105 people, and the incidence rate was 345.46 per 105 people in 2004. PD is a multisystem disorder involving the dopaminergic, noradrenergic, serotonergic, and cholinergic systems and is characterized by motor and nonmotor symptoms. A large proportion of patients with PD present with lower urinary tract dysfunction (LUTD) including urgency, increased frequency, and incontinence as predominant symptoms. LUTD is a prominent autonomic feature of PD and is both troublesome and a cause of morbidity in patients with the disease. Recent advances in investigative neurology provided insights into the underlying pathophysiology and appropriate management of urinary dysfunction. Urinary symptoms were shown to be statistically correlated with the severity of PD, but not with the duration of illness or in sexual differences. A videourodynamic study (VUDS) provides useful information according to our clinical practice, including details of the urethral sphincter and detrusor activity. Urethral sphincter pseudodyssynergia is a phenomenon in which the external urethral sphincter contracts at the initiation of urination in patients without spinal cord injury. Detrusor overactivity (DO) is frequently noted in these patients. Most patients have subsequent urethral sphincter relaxation and initiation of urination. Intermittent urethral sphincter activities can also occur during urination and result in considerable postvoiding residual (PVR) urine. In some aspects, pseudodyssynergia is similar to detrusor sphincter dyssynergia type 1; the main difference between them is the neurological deficit seen in detrusor sphincter dyssynergia that occurs in patients with spinal cord injury.

2. Urodynamic characteristics and interpretation

Patients with PD may present with normal voiding, DO, and dyskinesia of the urethral sphincter.

In the first case, a 72-year-old man was diagnosed with PD 5 years earlier; however, lower urinary tract symptoms (LUTSs) became exacerbated during the year before presentation. A digital rectal examination revealed a moderately enlarged prostate without a hard nodule. Transrectal sonography of the prostate showed a total prostatic volume of 36 mL with a transition zone index of 0.3. A VUDS revealed DO (Fig. 1). The urethral sphincter showed increased activity, and voiding detrusor pressure \( (P_{\text{det}}) \) was high and poorly sustained when the patient had the urge to void. Although the detrusor contracted and urine began to flow, the patient's sphincter did not relax for approximately 2 minutes. Voiding cystourethrography showed a narrow urethral sphincter during voiding. DO and dyskinetic urethral sphincter activity constitute urethral sphincter pseudodyssynergia during voiding, seen clinically as urgency and dysuria.

In a second case, a 75-year-old woman with a 3-year history of PD presented with progressive frequency, urgency, and urgency urinary incontinence for 1 year. A trial with an antimuscarinic agent was unsuccessful. She did not have dysuria or miction pain; however, she complained of constipation after antimuscarinic therapy. Urinalysis and cystoscopy revealed no abnormalities.
Urethral sphincter electromyographic (EMG) activity increased when the bladder was almost full and further increased when an involuntary detrusor contraction occurred (Fig. 2). When the patient relaxed the urethral sphincter, uroflow began, and the maximum flow rate ($Q_{\text{max}}$) usually occurred at a $P_{\text{det}}$ lower than the maximal voiding pressure.

In a final case, a 63-year-old woman with a 5-year history of PD complained of urinary incontinence, difficult urination, and residual urine sensation. Symptoms had worsened over several months before presentation. Cystoscopy revealed a normal-appearing bladder and urethra. Urinalysis showed no abnormal findings. Transrectal sonography showed no bladder base hypermobility during coughing and straining. A VUDS showed uninhibited detrusor contractions, and urethral sphincter EMG activity increased with increasing bladder volume until the maximal voiding pressure was reached when the urethral sphincter relaxed. During the voiding phase, the bladder neck and proximal urethra were widely opened, but the middle urethra was

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**Fig. 1.** Videourodynamic study revealed detrusor overactivity elicited at a volume of 108 mL (arrow 1). When the patient had the urge to void, the sphincter showed a further increase in activity, and the detrusor pressure ($P_{\text{det}}$) was high and poorly sustained (arrowheads 2). After 2 minutes of detrusor contraction, the sphincter began to relax and urine started to flow at a $P_{\text{det}}$ lower than the maximal $P_{\text{det}}$ (arrows 3). $P_{\text{det}}$ at the maximum flow rate ($Q_{\text{max}}$) was 32 cmH₂O. $Q_{\text{max}}$ was 14 mL/s, and the postvoiding residual was minimal. Voiding cystourethrography showed the bladder neck, a patent prostatic urethra, and a narrow urethral sphincter during voiding (arrows 4).

**Fig. 2.** The first sensation of bladder filling was perceived at a volume of 120 mL, and urge sensation began at 180 mL. Increased urethral sphincter electromyography (EMG) activity was noted when the bladder was almost full (arrowheads). The sphincter EMG activities further increased when detrusor contraction began (arrows 1). The maximal voiding pressure was 35 cmH₂O, but the detrusor pressure ($P_{\text{det}}$) at the maximum flow rate ($Q_{\text{max}}$) was only 20 cmH₂O. The $Q_{\text{max}}$ was 9 mL/s, and the voided volume was 202 mL. During voiding, the bladder neck and urethra were open, and the bladder wall was smooth (arrows 2).
relatively narrowed. Intermittent urethral sphincter activities were also noted during the voiding phase (Fig. 3). Urethral sphincter pseudodyssynergia was characterized by a high $P_{\text{det}}$, low $Q_{\text{max}}$, and increased urethral sphincter activity during the voiding phase; the patient usually had a large PVR after interruption of urination.

3. Discussion

The presence of voiding dysfunction in PD patients was reported in several studies with an incidence of 37–71%.6 Voiding dysfunction occurs when the extrapyramidal system is impaired. A previous investigation showed that 40–70% of patients with PD have some degree of voiding dysfunction, and nearly 70% have irritative bladder symptoms.7 DO was the predominant urodynamic finding, which may explain the LUTSs in patients with PD. Pseudodyssynergia existed in 25–60% of patients with PD.8 All three cases reported here presented with pseudodyssynergia and were found to have high-pressure voiding followed by sphincter relaxation.

Most patients with pseudodyssynergia have frequency, urgency, and dysuria; however, urgency and urinary incontinence without dysuria are also observed, as in the second case presented here. The key findings of pseudodyssynergia in the VUDS are increased voiding pressure and sphincter activity with initiation of urination followed by sphincter relaxation and the start of urine flow. During urination, sphincter activity can be silent or intermittent. Skeletal muscle tone is regulated by a spinal reflex arc (gamma loop), which may be exaggerated with an extrapyramidal neurological lesion.6 Impaired sphincter coordination or poor relaxation of the pelvic-floor muscle can be the result of increased excitability of gamma neurons in PD. It was postulated that this bradykinetic phenomenon may be related to disease severity in patients with PD.

Patients with PD who have a hyperactive urethral sphincter may be unable to adequately relax the urethral sphincter during volitional or reflex micturition, resulting in inadequate detrusor contractility and an increased PVR volume.9 In consideration of this factor, treatment with medication to relax the urethral sphincter might provide relief to patients with LUTSs. Detrusor instability and bladder outlet obstruction are common in men with LUTSs.10 Treatment targeting benign prostatic hyperplasia is usually unsuccessful in relieving LUTSs in patients with PD, especially those who have not undergone a urodynamic study. Treatment of urodynamic symptoms should be aimed at relaxing the urethral sphincter. Medication with antimuscarinics for DO along with skeletal muscle relaxants can be helpful. If medications fail or if the patient cannot tolerate the adverse effects of antimuscarinics, such as constipation, blurred vision, dry mouth, and impaired cognitive function, treatment with a urethral sphincter botulinum toxin A injection is the next therapeutic step and can provide a good therapeutic effect.11,12 Surgical intervention for benign prostatic hyperplasia is contraindicated in patients without a definite evidence of bladder outlet obstruction.

4. Conclusions

DO is the main urodynamic finding in PD patients with LUTSs. A hyperactive urethral sphincter with pseudodyssynergia causes an increased PVR volume in some patients. A VUDS can aid in distinguishing the pathophysiology of voiding dysfunction in patients with PD. Treatment of PD and LUTSs should be based on underlying bladder and urethral sphincter dysfunctions.

Conflicts of interest statement

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References


