Parkinson's Disease and Its Effect on the Lower Urinary Tract Evaluation of Complications and Treatment Strategies



Benjamin M. Brucker, MD^{a,b,*}, Sidhartha Kalra, MD^a

KEYWORDS

• Parkinson's disease • LUTS • Complications • Management

KEY POINTS

- Neurogenic lower urinary tract dysfunction is prevalent in patients with Parkinson's disease (PD) and has a great impact on quality of life, resulting in potentially debilitating sequelae.
- The key to diagnostic and therapeutic effectiveness lies in distinguishing centrally mediated lower urinary symptoms (LUTS) secondary to PD from LUTS that are merely coincidental.
- PD-associated bladder dysfunction is not significantly responsive to levodopa; add-on therapy for ameliorating lower urinary tract problems is often necessary; associated risk should be taken into consideration.
- Prostate reduction surgery for comorbid benign prostatic hyperplasia is no longer contraindicated in this population, assuming multiple system atrophy is excluded.
- We recommend a multidisciplinary approach, with health care providers focusing on motor and nonmotor symptoms and quality of life issues to maximize bladder-specific quality of life.

INTRODUCTION

Parkinsonian syndromes include Parkinson's disease (PD) and atypical Parkinsonism (multiple system atrophy [MSA], progressive supranuclear palsy, corticobasal degeneration, and Lewy body dementia). Differentiating these 2 clinical entities is challenging given the considerable overlap in their clinical profiles, especially early in the disease course. However, careful understanding of the progression of symptomatology can help clinicians in establishing the correct diagnosis. This is an integral aspect for adequate management of motor and nonmotor symptoms, estimating prognosis, and providing information to patients and their

caregivers. Differentiating among syndromes, however, is particularly important for lower urinary tract symptoms (LUTS), which evolve differently and with variable impact and prognoses. Although PD is the most common type of Parkinson's syndrome, 15% to 20% patients presenting with these conditions have an underlying atypical disease process progressing to primary atypical Parkinsonism syndrome or symptomatic Parkinsonism.³

PARKINSON'S DISEASE

PD is a progressive degenerative neurologic movement disorder, and motor symptoms are characterized by resting tremor, bradykinesia, (cogwheel)

E-mail address: Benjamin.Brucker@nyumc.org

^a Department of Urology, New York University Langone Medical Center, 150 East 32nd street second floor, New York, NY 10016, USA; ^b Department of Obstetrics and Gynecology, New York University Langone Medical Center, 550 First Avenue, New York, NY 10016, USA

^{*} Corresponding author. Department of Obstetrics and Gynecology, New York University Langone Medical Center, New York, NY.

rigidity, and postural instability. PD is estimated to affect 100 to 180 per 100,000 of the population with an annual incidence of 4 to 20 per 100,000.4 PD is associated with the degeneration of dopamine-producing cells in the substantia nigra of the midbrain and Lewy body formation. Braak and colleagues^{5,6} proposed that the formation of intraneuronal Lewy bodies and Lewy neuritis begins at 2 sites and continues in 6 stages, during which components of other systems become progressively involved. In stages 1 to 2, the Lewy body pathology is confined to the medulla oblongata/ pontinetegmentum and anterior olfactory structures. In stages 3 to 4, the substantia nigra, other nuclei of the basal midbrain and forebrain, and the mesocortex are affected; the illness usually becomes clinically manifest during this phase. Finally, lesions appear in the neocortex in stages 5 to 6. Lewy bodies and dopaminergic neuron degeneration are also observed in peripheral nerves innervating the gastrointestinal tract, even before the onset of motor symptoms (Table 1).5 Over the last decade, there has been increasing interest in and understanding of nonmotor aspects of PD, which include dysphagia (30%-82% of patients), constipation (>50%), orthostatic hypotension (20%-58%), depression (>16%), cognitive decline and dementia (>6 times higher than healthy individuals), sexual dysfunction (43%-81%), and LUTS.3

Prevalence of Urinary Symptoms in Parkinson's Disease

Using validated questionnaires and that analyses that have included subtypes such as atypical

Table 1 Clinicopathologic correlation in Parkinson's disease		
Lewy Body Neuritis Stage	Area of Brain Affected	Clinical Progression
1–2	Medulla oblongata/ pontine tegmentum Anterior olfactory structures	Asymptomatic
3–4	Substantia nigra Nuclei of the mid and forebrain Mesocortex	Symptomatic
5–6	Mature neocortex	Advanced disease

Data from Braak H, Ghebremedhin E, Rub U, et al. Stages in the development of Parkinson's disease related pathology. Cell Tissue Res 2004;318:121–34. Parkinsonism, the prevalence of LUTS has been reported as urinary disturbances in 27% to 64% of PD patients.7 Campos-Sousa and colleagues7 studied 61 patients with PD and 74 control individuals using the International Prostate Symptoms Score. Thirty-nine percent of PD patients reported urinary symptoms on this questionnaire, whereas only 10.8% of control individuals had such symptoms. Nocturia was reported by 64% of PD patients and in 32% of control individuals; urgency was reported by 32% of PD patients and 9% of control individuals. Despite these findings, there is no clear consensus on the nature, severity, or temporal occurrence of LUTS among PD patients, although more data are becoming available as newer studies are using validated questionnaires to follow urinary symptoms. In a review on PD and urinary symptoms, Winge³ noted that 2 study used the International Prostate Symptoms Score for assessing bladder symptoms and reported that urinary symptoms do not increase with disease severity, whereas another study using the Danish Prostate Symptoms Score questionnaire reported a positive correlation between urinary dysfunction and stage of PD. This seems to indicate that other symptoms, both motor and nonmotor, may affect the overall severity of urinary symptoms. Impaired mobility, tremor, gait, and balance deficits as well as attention span difficulties are thus postulated to exacerbate urinary urgency and other bladder symptoms.

PATHOPHYSIOLOGY OF BLADDER DYSFUNCTION IN PARKINSON'S DISEASE Normal Neural Control of Micturition

The neurologic control of the bladder is a coordinated action between the somatic and autonomic nervous systems. During the urinary storage phase, the efferent sympathetic nervous system via hypogastric nerves originating in the lumbar spinal cord acts to relax the bladder muscle, as well as maintaining closure of the internal urethral sphincter, thereby helping in storage of urine. Efferent parasympathetic innervation to the bladder, originating in S2-S3-S4 segments of the spinal cord acting via pelvic splanchnic nerves, has the opposite effect of contraction of the detrusor muscle and facilitation of voiding. The central nervous system ensures that micturition occurs under voluntary control, at a time and place that is socially acceptable. Two micturition centers, namely the pontine micturition center and the pontine storage center, have been shown to control the micturition pathway centrally. The former is the more important of the 2 areas and

Download English Version:

https://daneshyari.com/en/article/5730554

Download Persian Version:

https://daneshyari.com/article/5730554

<u>Daneshyari.com</u>