**Lower urinary tract dysfunction in Parkinsonian syndromes**

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Ekawat Vichayanrat, MD PhD[1,2], Claire Hentzen, MD [1,4], Amit Batla, MD DM [3], Sara Simeoni[1,3], MD, Valeria Iodice, MD, PhD[2,3], Jalesh N. Panicker, MD, DM, FRCP[1,3]

[1]Department of Uro-Neurology, The National Hospital for Neurology and Neurosurgery, Queen Square, London, UK

[2]Autonomic Unit, National Hospital for Neurology and Neurosurgery, Queen Square, London, UK

[3]UCL Queen Square Institute of Neurology, Faculty of Brain Sciences, University College London, London, UK

[4]Sorbonne Université, GRC 01, GREEN Groupe de Recherche Clinique en Neuro-Urologie, AP-HP, Hôpital Tenon 4 rue de la Chine, F-75020 Paris, France

**Correspondence to:**

Jalesh N. Panicker

Department of Uro-Neurology, The National Hospital for Neurology and Neurosurgery

UCL Queen Square Institute of Neurology, Faculty of Brain Sciences, University College

London, London, United Kingdom

j.panicker@ucl.ac.uk

Orcid 0000-0001-5190-3259

## Abstract

**Purpose of review:** The aim of this review is to outline the clinical presentation, pathophysiology and evaluation of lower urinary tract (LUT) dysfunction in Parkinson's disease and other parkinsonian syndromes including multiple system atrophy, dementia with Lewy bodies, progressive supranuclear palsy and corticobasal degeneration.

**Recent findings:** LUT dysfunction commonly occurs in neurological disorders, including patients with parkinsonian syndromes. The pattern of LUT dysfunction and its severity are variable, depending upon the site of lesion within the neural pathways. Parkinsonian syndromes are broadly divided into Parkinson's disease (PD) and atypical parkinsonian syndromes such as multiple system atrophy (MSA), dementia with Lewy bodies (DLB), progressive supranuclear palsy (PSP) and corticobasal degeneration (CBD). Different parkinsonian syndromes have distinct clinical features (e.g., dysautonomia, early dementia, supranuclear gaze palsy, higher corticol signs), and the pattern of LUT dysfunction and its severity can differ.

**Conclusions:** LUT dysfunction is a common feature in patients with parkinsonian syndromes. Recognising the pattern of LUT dysfunction during the assessment of these patients can help management and possibly facilitate an earlier diagnosis.

## Introduction

Lower urinary tract (LUT) symptoms are a prominent presentation of autonomic dysfunction in different parkinsonian syndromes. In Parkinson's disease (PD), LUT symptoms are the most prevalent of non-motor symptoms [1], whereas urogenital symptoms can precede other neurological features in multiple system atrophy (MSA)[2]. LUT symptoms not only have an impact on quality of life, but are also associated with an increased risk of falls and worsening of neurogenic orthostatic hypotension in the presence of urinary tract infection (UTI) [3,4], and early institutionalisation[5].

The aim of this review is to outline the clinical presentation, pathophysiology and evaluation of LUT dysfunction in Parkinson's disease and other parkinsonian syndromes including multiple system atrophy, dementia with Lewy bodies, progressive supranuclear palsy and corticobasal degeneration.

## Lower urinary tract in health and disease

The lower urinary tract (LUT) consists of the bladder and urethra (including the internal and external sphincters). The detrusor muscle, a smooth muscle located within the bladder wall, and urethral sphincters are highly regulated by a complex innervation contributed by the somatic and autonomic (parasympathetic and sympathetic) nervous systems.

Normal LUT function includes storage and voiding phases and requires coordination between detrusor and urethral sphincters, which are ultimately under the control of a highly distributed neural pathway within the central (brain and spinal cord) and peripheral nervous system. The LUT is in the storage phase more than 99% of the time due to detrusor relaxation and urethral sphincter contraction. Storage function primarily is mediated by central control from the pontine storage centre, hypothalamus, cerebellum, basal ganglia and frontal cortex. The voiding phase is mediated by a spinobulbospinal reflex pathway, whereby the periaqueductal grey (PAG) no longer exerting tonic inhibition on the pontine micturition centre (PMC), resulting in relaxation of pelvic floor muscles and urethral sphincters, and detrusor contraction[6]. LUT functions are susceptible to changes following neurological injury, and dysfunction can affect the storage phase and/or the voiding phase. Urinary frequency, urgency and urgency incontinence - overactive bladder (OAB) symptoms are suggestive of storage

dysfunction. In contrast, urinary symptoms that suggest voiding dysfunction include hesitancy, slow/interrupted urine stream, straining and sensation of incomplete bladder emptying.

The different sites of the neurological lesions result in predictable patterns of LUT dysfunction. Suprapontine or suprasacral spinal cord lesions result in storage dysfunction, leading to reduced bladder capacity and involuntary detrusor muscle contraction: detrusor overactivity. Lesions in the spinal cord may result in simultaneous contractions of the urethral sphincter and detrusor muscle, called 'detrusor-sphincter dyssynergia-DSD', and patients can present with incomplete bladder emptying alongside abnormally high pressures in the bladder.

In contrast, sacral or infrasacral lesions cause denervation of sphincters and/or bladder and result in incompetent sphincter and poorly sustained/absent detrusor contractions, causing voiding dysfunction. Patients with voiding dysfunction can present with urinary hesitancy, straining during micturition, interrupted and/or slow stream, incomplete bladder emptying and urinary retention.

## Lower urinary tract dysfunction in parkinsonian syndromes

### *Parkinson's disease*

LUT symptoms are the most common presentation of autonomic dysfunction [7], present in 38-71% of patients with Parkinson's disease (PD) [8] [9], and increase in severity with progression of Parkinson's[10]. The LUT symptoms add a significant burden of care in PD. Bladder problems fall within the top three reasons for non-elective hospital admission of patients with PD. The most common complication might be urinary tract infection (UTI), and this can lead to frequent hospitalisation and morbidity. Data from Hospital Episode Statistics (HES) in UK suggests that the estimated cost of patients with PD being admitted for genitourinary causes exceeds £68,200,000 over four years [11,12].

Reduced bladder capacity and detrusor overactivity, reported in 45–93% of PD patients, result from degenerative changes in the striatum and other subcortical and cortical regions. It has been demonstrated that Overactive Bladder Symptom Score (OABSS) in PD relates to poor performance of frontal lobe executive function, REM sleep behavior disorder and a higher Hoehn & Yahr score [13].

The mechanisms of LUT symptoms in PD are complex. However, the disruption of the dopamine D1-GABAergic direct pathway and its GABAergic collateral to the micturition circuit, resulting in loss of inhibition of the micturition reflex and play an important role on LUT symptoms[14,15]. LUT symptoms correlate in severity with urodynamic abnormalities and dopaminergic deficit on dopamine transporter scans[14].

Nocturia (56.7%) is the most common LUT symptom, followed by urinary urgency and these together are the commonest nonmotor symptoms in PD. Nocturia has been shown to be associated with sleep disturbances, greater risk for falls and hip fractures, and higher mortality in older individuals[16-19]. Primary sleep disturbances such as obstructive sleep apnoea, loss of circadian pattern in blood pressure control (night-time dipping followed by morning surge coinciding with arousal), autonomic dysfunction resulting in orthostatic hypotension and neurogenic supine hypertension, concomitant medical disorders such as diabetes mellitus and congestive heart failure, and medication use are cited as causes for nocturia in the literature, though not specifically in Parkinson's[20].

Although an overactive bladder (OAB) is likely to be the primary cause for urological symptoms in PD, nocturnal polyuria (NP) may also significantly contribute to nocturia in PD. NP is defined as voiding more than 33% of the entire 24-hour urine output at night, and is often missed during the assessment of LUT symptoms. NP is associated with excessive production of urine at night, possibly related to a loss of circadian rhythm in PD[21,22] and is not expected to improve with antimuscarinic agents used to ameliorate OAB symptoms, but may be amenable to some other approaches[23,22].

Subclinical detrusor weakness during voiding can be found on urodynamics, and the severity of PD was the only predictive factor for voiding dysfunction [24]. However, detrusor overactivity (DO) with minimal PVR remain the most common urodynamics findings in patients with PD.

There is evidence for mild urethral obstruction in approximately half of patients with PD on urodynamics findings. These findings were thought to arise due to some form of impaired external urethral sphincter relaxation. Historical studies described bradykinesia of pelvic floor muscles when initiating voiding causing "pseudo-dyssynergia", however this condition is now thought to be rare.

Other urological conditions can also contribute to LUT dysfunction in patients with PD. In particular, men over 50 years old may have concomitant benign prostate enlargement, whilst there may be pelvic floor weakness or stress incontinence in women. Performing transurethral resection of the prostate gland in patients with PD is not contraindicated, however meticulous patient selection involving neurological and urological input to secure the diagnosis of PD rather than mimics such as MSA, and assessing the degree of bladder outflow obstruction, are essential before proceeding with surgery [25].

***Multiple system atrophy***

Autonomic dysfunction is an essential diagnostic criterion for possible and probable Multiple System Atrophy (MSA) according to the second consensus statement for the diagnosis of MSA [26], and often predates neurological signs of parkinsonism and cerebellar ataxia and pyramidal signs. Orthostatic hypotension with supine hypertension, urogenital dysfunction characterized by urinary storage, voiding dysfunction and erectile dysfunction in male patients are common manifestations of autonomic dysfunction. Autonomic dysfunction can occur early in the course of disease in MSA and previous studies have demonstrated that the onset of urogenital dysfunction often precedes, or coincides, with the onset of orthostatic hypotension[27,28]. Other non-motor features including respiratory, gastrointestinal, sudomotor dysfunction and REM sleep behaviour disorder (RBD), occur in MSA and can be presenting symptoms preceding motor symptoms [2].

LUT symptoms, including urinary urgency with or without incontinence, nocturia, hesitancy, urinary retention, and erectile dysfunction were the most common presenting complaint in MSA patients from the European Multiple System Atrophy (EMSA) registry [29]. Urinary incontinence and frequency occur even in the early stages of disease due to detrusor overactivity, but urinary retention and/or incomplete bladder emptying develop as the disease progresses [6]. Urinary retention requiring catheterization occurs less commonly at disease onset, but the prevalence increased to 14% over a 5-year period[30]. However, MSA patients may present with chronic urinary retention requiring urinary catheterisation, sexual and bowel dysfunction, and abnormal anal sphincter electromyography (EAS-EMG) before developing motor signs[31]. In this series, EAS-EMG was abnormal thereby suggesting involvement of the Onuf's nucleus, a group of neurons located in the anterior horn of the sacral cord that innervate the sphincters. This nucleus is particularly susceptible to degeneration in MSA. These findings suggest that pelvic organ dysfunction can predate other motor signs/symptoms in some

patients and raises the possibility that the pathological substrate for non-motor onset patients may start from the spinal cord before involving other regions of the central nervous system[31].

MSA patients presenting with non-motor features are often misdiagnosed with other conditions. A previous study has shown that more than 40% of MSA patients who presented with LUTS were misdiagnosed with prostatic hypertrophy or bladder dysfunction and underwent urological surgery, predictably with poor outcome[32].

A recent prospective study also demonstrated that more than 18% of MSA patients initially presented with LUT symptoms, with a mean of 2.8 years, prior to any motor features [33]. LUT symptoms were reported in all patients at the first visit and bladder dysfunction was demonstrated in 96% of the patients. Among these patients, 74% had elevated post-void residual (PVR). There was evidence of detrusor overactivity and detrusor sphincter dyssynergia in 56% and 45%, respectively. Therefore, voiding dysfunction can occur in the early stage of MSA and a large residual volume may help to distinguish MSA and PD patients [34], since the latter do not usually have significant urinary retention.

Urodynamic study is useful to understand the cause for these urinary symptoms. In the storge phase, detrusor overactivity and uninhibited external sphincter relaxation are reported both in patients with MSA and PD, whereas an open bladder neck at the start of filling, suggesting denervation of the internal sphincter, is seen in MSA. During the voiding phase, detrusor underactivity and detrusor sphincter dyssynergia is more commonly reported in MSA compared to PD[35]. Therefore, detrusor underactivity, detrusor sphincter dyssynergia and evidence of open bladder neck on urodynamic testing are useful findings in distinguishing MSA from PD.

Because of the early neuronal loss of Onuf's nucleus in MSA, pelvic neurophysiology testing including EAS-EMG and bulbocavernosus reflex (BCR) often reveals abnormalities. EAS-EMG has been proposed as a diagnostic tool for distinguishing between PD and MSA.[36-38], and the neurogenic changes demonstrated by EAS-EMG were reported to be abnormal in 75-100% of MSA patients [39]. Although the abnormalities on EAS-EMG can be present in other conditions such as PD[40,41], progressive supranuclear palsy (PSP) [42] and DLB [43]and its usefulness in distinguishing MSA from PD may be lower in patients with longer disease duration >5 years [36], this test remains a valuable diagnostic tool in distinguishing MSA from other parkinsonian symptoms when being used in conjunction of other investigations in patients with suspected MSA. A delay in BCR latencies has also been shown in parkinsonian

disorders compared to controls, and prolonged BCR latencies were found in MSA patients compared to PD[44-46].

Pure autonomic failure (PAF) is a sporadic alpha-synucleinopathy disorder characterized by autonomic failure without neurological symptoms and signs [47]. A recent natural history study in PAF demonstrated that half of patients report LUT symptoms and up to 65% erectile dysfunction at 5 years after disease onset. More importantly, severe bladder dysfunction in patients with PAF, especially urinary retention, is a predictor of phenoconversion to MSA[48].

***Dementia with Lewy Bodies***

Dementia with Lewy Bodies (DLB) is characterised by early onset progressive memory impairment and deficits on different tests of cognitive functions such as executive function, attention and visuoperceptual skills. Cognitive impairment and parkinsonism usually occur within a one-year interval of the disease, and autonomic dysfunction including LUT dysfunction and orthostatic hypotension usually manifests subsequently [49]. Overactive bladder is common [43] and one study suggests that symptoms of urgency and urge incontinence suggesting detrusor overactivity are more prevalent in DLB compared with other cognitive disorders such as Parkinson disease with dementia and Alzheimer disease [50]. Urinary retention is reported in DLB, however this is uncommon [51].

***Progressive supranuclear palsy***

Urinary symptoms are present in more than 80% of patients with PSP [52,53]. According to a recent study, 57% of patients report storage symptoms and 56% voiding symptoms; voiding dysfunction in PSP appears to be similar to MSA, but more severe than in Parkinson's disease. Urgency incontinence has prognostic value as it is a predictor of more rapid disease progress and reduced survival[53-55].

Sakakibara et al. performed urodynamics in 6 patients with PSP; findings included detrusor overactivity (n=4), post-void residual over 100 ml (n=1), low compliance (n=1) and detrusor-sphincter dyssynergia (n=1)[52]. More recent studies have compared urodynamics findings between PSP, PD and MSA and suggest that storage dysfunction was similar in the three groups (detrusor overactivity in PSP was found in 70-81% of patients), however patients with PSP demonstrate significantly higher post-void residual volumes and more impaired detrusor contraction compared to patients with PD. The degree of voiding dysfunction was comparable to changes seen in MSA [56,57].

Urinary dysfunction in PSP is likely to be due to the involvement of different neural structures that have a role in the control of micturition. In PSP, there was evidence of neuronal and glial cytoskeletal pathology affecting the rostral brainstem tegmentum and basal ganglia that have a central role in the regulation of micturition [58,59]. Moreover, involvement of different regions of the frontal lobe[60], which is involved in the control of LUT function [61], has been demonstrated in patients with PSP.

A clinico-pathological study of 3 patients with a definite diagnosis of PSP demonstrated that Onuf 's nucleus is affected in PSP as well as in MSA. The morphological and morphometric assessment showed severe cell loss, the presence of neurofibrillary tangles, neuropil threads and glial inclusions. Amongst the aforementioned patients, 2 out of the 3 had abnormal anal sphincter electromyography [62]. This confirms the findings of previous studies which showed abnormal sphincter EMG with neurogenic changes in up to 50% of PSP patients [52,42].

***Corticobasal degeneration***

Urinary dysfunction is a common feature in patients with corticobasal degeneration (CBD). Wenning et al. reported that frequency of urinary incontinence was 62% in a group of patients with pathologically confirmed CBD; the median latency of onset was 70 months and the median duration of urinary incontinence from onset to death was 39 months[63]. Sakakibara et al found that the prevalence of LUT symptoms was 80%; the latency of onset was 1-3 years; duration of disease greater than 5 years and the presence of forced grasp reflex were all more commonly associated with urinary dysfunction [64]. Patients reported primarily storage symptoms (nocturia, urgency incontinence, urgency, frequency) and occasionally voiding symptoms, but none of them had urinary retention. The common urodynamics findings were detrusor overactivity and reduced capacity[64]. Urinary dysfunction is thought to be related to the involvement of the anteromedial frontal cortex and basal ganglia [64].

**Management of patients with parkinsonian syndromes reporting LUT symptoms**

Patient management aims to identify possible associated factors contributing to LUT symptoms, reduce patient discomfort and improve quality of life impaired by overactive bladder and urinary incontinence, and prevent complications such as UTIs in specific cases.

## Evaluation

The preliminary evaluation of the neurological patients reporting LUT symptoms consists of history taking, a bladder diary/questionnaires and may be supplemented by investigations such as uroflowmetry, post-void residual measurement, renal ultrasound, (video-)urodynamics, neurophysiology, and urethrocystoscopy, depending on the clinical indications[65]. The clinical assessment should also evaluate for other urological and gynaecological conditions that may contribute to LUT symptoms such as prostate enlargement, stress incontinence, and pelvic organ prolapse.

*History taking*

History taking is the cornerstone of the assessment. The timing of onset of the symptoms, particularly the chronology with neurological symptoms, must be considered as it may help to differentiate among the parkinsonian syndromes, and to explore non-neurological contributors for LUT symptoms. Both storage symptoms (e.g., urgency, frequency, urgency urinary incontinence, and nocturia) and voiding dysfunction (e.g., hesitancy, low stream and intermittent stream) should be assessed. However, voiding symptoms are not always reliable as more than half of patients may not be aware of incomplete bladder emptying. Bowel and sexual symptoms should be assessed as they are concomitantly affected due to pathology affecting shared neural pathways, and also exploring the impact of lower bowel dysfunction on bladder symptoms.

General and perineal examinations should look out for non-neurological causes such as an enlarged prostate gland in men, pelvic floor prolapses, post-menopausal vaginal atrophy in women, or medication, as they are common causes of LUTS in this age group.

The common factors that contribute to LUT symptoms include degenerative spinal disease and myelopathy, diabetes mellitus, congestive cardiac failure or pedal edema, and use of diuretic drugs or significant cerebrovascular disease. Recurrent urinary tract infections and haematuria are red flags requiring urgent investigation.

*Bladder diary*

A 3-day bladder diary completes the history taking, providing key information concerning fluid intakes, voids volume, frequency, and leakages. It is also the only way to objectively detect nocturnal polyuria, one of the possible mechanisms responsible for nocturia.

*Questionnaires*

Questionnaires of symptoms and quality of life may be useful to quantify the severity of the discomfort and its impact on daily life. It also may help the physician to assess treatment efficacy. Their use is recommended by the European Association of Urology, but none is specifically validated in patients with PD. A recent systemic review from the Movement Disorder Society (MDS) task force reports the use of generic questionnaires in this population and recommended certain questionnaires such as the Overactive Bladder Symptom Score (OABSS) or the OverActivity of the Bladder Questionnaire (OAB-q), with caveats. The use of some questionnaires is limited due to the absence of assessment of all LUT symptoms, and the lack of specifical analysis for nocturnal polyuria[66].

*Investigations*

The general recommendations for neuro-urological patients include performing urinalysis to exclude urinary tract infection (UTI), blood biochemistry, ultrasonography, residual volume measurement and free flowmetry[67]. It is important to establish whether the UTIs relate to incomplete bladder emptying (e.g., bladder outflow obstruction, reduced detrusor contractility, etc.) or a structural abnormality (foreign body in bladder, bladder stone, tumour, etc.), and for this reason the input of a Urologist would be valuable.

*Free flowmetry* along with the assessment of post-void residual (PVR) volume with a bladder scan is a non-invasive essential test and provides useful information about the voiding phase, particularly in patients with voiding dysfunction or MSA. These tests should be repeated as the disease course progresses, and in case of paradoxical worsening of storage symptoms after the introduction of an antimuscarinic agent. Furthermore, the volume should be measured more than once to improve accuracy.

*Bladder and upper urinary tract ultrasonography* help to exclude upper urinary tract dilatation or apparent causes for OAB such as bladder stone.

*Cystometry* is not necessary before initiating treatment [68] but can be useful to understand the mechanism underlying LUT symptoms, especially in the absence of improvement with initial management. Cystometry evaluates the pressure-volume relationship during a non-physiological bladder filling and may demonstrate detrusor overactivity, the most frequent finding in patients with PD. The pressure-flow study improves understanding of bladder function during the voiding phase and may help to diagnose associated components such as

bladder outlet obstruction secondary to prostate enlargement. In a recent retrospective study, MSA patients were more likely to have a higher rate of large post-void residual volume, impaired contractility, and low bladder compliance compared to individuals with PD[69]. If available, video-urodynamic brings complementary information concerning ureteral reflux during filling, and eventual bladder obstruction or lack of sphincteric relaxation.

*Cystoscopy* is useful in select cases to detect a local irritating factor responsible for OAB symptoms and can help in the assessment of bladder obstruction. The timeline to perform it is not specific and will depend on patient history.

*Pelvic neurophysiology testing* plays a role in select cases of parkinsonism. It is well established that the prevalence of EAS-EMG abnormalities increases with the onset of parkinsonian disease. Thus, higher motor unit potential (MUP) mean duration and a larger number of polyphasic MUP are observed in patients with parkinsonian disorders compared to healthy control[36]. In the first 5 years of the disease onset, MUP abnormalities in EAS-EMG could help to distinguish MSA from PD[70,71]. However, contradictory results exist concerning the specificity of this test since a prospective study failed to demonstrate any difference between patients with PD at the early stage and MSA[72].

**Treatment**

The management of LUT symptoms must be individualized. Conservative measures such as ensuring an adequate fluid intake (1.5-2 litres), avoiding caffeine and alcohol, bladder training or pelvic floor muscle training may improve LUT symptoms, particularly in patients reporting mild discomfort [73]. Restriction of fluid intake a few hours before bedtime may also improve nocturia.

The effect of dopaminergic medications on LUT symptoms remains unclear. The acute effect of levodopa could worsen symptoms inducing a reduced bladder capacity and earlier detrusor overactivity[74], whereas chronic medication might slightly improve symptoms, especially for patients with bladder complaints[75]. However, the level of evidence is low with a small number of subjects, and the clinical significance of the difference showed is uncertain. Recently, an observational study on 106 patients suggests a benefit of extended-release levodopa at bedtime on nocturia[76].

Antimuscarinic medications are the mainstay of pharmacological treatment for managing overactive bladder symptoms. Solifenacin has been found to be beneficial in a randomized-control trial in this situation but larger studies with other antimuscarinics are limited[77]. This must, however, be balanced against impairment of cognition and consciousness in susceptible individuals[78]. Percutaneous tibial nerve stimulation (PTNS) is an effective option for managing overactive bladder symptoms and does not exacerbate the voiding dysfunction or increase PVR[79].

Intradetrusor injections of botulinum toxin have been shown to be effective for detrusor overactivity in PD[80], however, are associated with the risk of urinary retention[81]. Neuromodulation is a promising, minimally invasive treatment for PD-related OAB symptoms[78,81].

An elevated PVR/urinary retention in patients with parkinsonian syndromes should be managed with intermittent self-catheterisation (ISC). The evaluation of suitability for ISC should include a review of neurological deficits that hinder catheterization such as poor manual dexterity, tremor, truncal imbalance and cognitive dysfunction. An indwelling urinary catheter is an alternative option; however, a suprapubic catheter would be a preferred option if the ISC is not suitable.

Desmopressin is effective for the management of nocturnal polyuria which has been reported to be common in PD[23]. More recently, an open label study demonstrated beneficial effects of melatonin with significant improvements in problems related to nocturia, specifically number of episodes of nocturia per night and average urine volume voided at night[21].

## Conclusion

LUT dysfunction is a frequently occurring feature in patients with parkinsonian syndromes. The disorders share motor signs, however the prevalence, onset, severity and pattern of LUT dysfunction can vary. In this review, we have discussed the clinical presentation, pathophysiology and evaluation of patients with LUT symptoms in different parkinsonian syndromes.

**Table 1: Assessment of patients with Parkinsonian syndrome reporting lower urinary tract symptoms, *(Adapted from Panicker (2015)[82])***

| | Essential | Desirable | Required in Specific Situations |
|---|---|---|---|
| Bedside | History taking<br>Bladder diary<br>Physical examination to eliminate a non-neurological etiology | LUT specific symptoms and quality of life questionnaires | |
| Noninvasive tests | Urinalysis<br>PVR measurement*<br>(bladder scan if available or in-out catheterization or bladder ultrasonography)<br>Ultrasound kidneys, ureters and bladder | Uroflowmetry<br>Blood biochemistry | urine culture<br>urine cytology |
| Minimally-invasive tests | | | (video) urodynamics*<br>cystoscopy<br>pelvic neurophysiology* |

* Might be useful in distinguishing diagnosis between PD and MSA

**Table 2 Comparison between different parkinsonian syndromes**

| | PD | MSA | DLB | PSP | CBD |
|---|---|---|---|---|---|
| Onset of LUT symptoms | Rarely an initial presentation | At onset or often precedes motor symptoms | Early (within 3 years after onset) | Early (within 2 years after onset) | Early (1-3 years after onset) |
| Prevalence of LUT | 53-71% | 90-100% | 84-97% | 56-80% | 80% |
| Storage LUT symptoms | +/++ | +++ | +++ | +++ | ++ |
| Voiding LUT symptoms | +/- | +++ | + | ++ | +/- |
| Elevated PVR | - | +++ | + | ++/+++ | - |
| Urodynamics findings | DO, mild BOO, delayed striated sphincter relaxation | DO and external sphincter weakness (early) DU, DSD and open bladder neck during filling | DO | DO, DU, DSD | DO Reduced capacity |
| Abnormal anal sphincter EMG | +/- | +++ (83%) | ++(50%) | ++ | - |
| Reference | [83] | [83] | [43] | [56] | [64] |

*PD* Parkinson's disease, *MSA* multiple system atrophy, *DLB* dementia with Lewy bodies, *PSP* progressive supranuclear palsy, *CBD* Corticobasal degeneration, *PVR* Post-void residual urine, DO detrusor overactivity, DU detrusor underactivity, DSD detrusor-sphincter dyssynergia

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