Review

Lower urinary tract symptoms in Parkinson's disease: A review

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Abstract

Background: Parkinson's disease (PD) manifests not only through its characteristic motor symptoms but also through a wide range of non-motor symptoms. Of these, lower urinary tract symptoms (LUTS) are among the most frequent non-motor manifestations, with their prevalence increasing as the disease progresses. LUTS significantly impair patients' quality of life and cause considerable distress, particularly as symptom severity increases. LUTS represent a clinical challenge for physicians treating patients with PD and often require an interdisciplinary approach to achieve accurate diagnosis and effective management. **Objective:** Based on the scientific literature and current guidelines, we briefly discuss the pathophysiology, epidemiology, clinical manifestations, and therapeutic options related to LUTS. To improve understanding of the underlying mechanisms of LUTS in PD, the influence of the basal ganglia and, in particular, dopamine deficiency is discussed based on currently hypothesized models. Therapeutic strategies for the management of LUTS are outlined, along with their limitations and key considerations in determining the most appropriate treatment approach. Furthermore, differential diagnoses and potential comorbidities accompanying PD, as well as correlations between LUTS and other neurological disorders and their shared therapeutic aspects, are discussed. **Conclusion:** Given the high prevalence and substantial clinical impact of urinary tract symptoms in PD, as well as the limited availability of comprehensive data, further studies are warranted to refine therapeutic strategies and improve patients' quality of life.

Keywords: Parkinson's disease, Lower urinary tract symptoms, Post-void residual volume

1. Introduction

In addition to the typical motor symptoms, patients with Parkinson's disease (PD) experience a wide range of non-motor symptoms (NMS), including pain and neuropsychiatric, gastrointestinal, cardiovascular, and urogenital disturbances.[1]

NMS in PD are common and significantly reduce the quality of life (QoL) of patients.[2,3] Therefore, a 30-item scale for assessing NMS in PD—the NMS Scale—was developed, comprising nine dimensions, including urinary symptoms represented by three items.[3] The prevalence of all NMS domains increases with the disease stage.[1,4] The relative occurrence of specific NMS domains varies with disease progression: urinary disturbances rank fifth in frequence at Hoehn–Yahr stage 1 and become the most frequent at stages 4–5, reaching an overall prevalence of approximately 60% in the PRIAMO study.[4] The high prevalence of urogenital NMS has been confirmed by subsequent studies.[5,6] NMS frequency and severity are closely associated with health-related QoL (HRQoL) in PD, with HRQoL worsening as NMS scale scores increase.[2] Moreover, patients with PD are twice as likely to be hospitalized for urinary tract infection (UTI)[7] compared to non-PD controls, further compounding the burden on their HRQoL.[7]

Patients with PD and urogenital disturbances mainly complain of urinary urgency, pollakiuria, nocturia, and urinary incontinence, although some remain asymptomatic, as early urinary disorders might remain unnoticed.[8,9] The most noticeable and disturbing symptom among the reported urinary NMS in PD is incontinence. Urinary incontinence has been widely researched, while nocturia, pollakiuria, urinary urgency without incontinence, and post-void residual urine are relatively underrepresented in the scientific literature.

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In this article, we provide an overview of the physiology of lower urinary tract control, as well as the pathophysiology, clinical manifestations, diagnosis, and treatment options for urinary NMS in PD.

2. Physiology and pathophysiology of the lower urinary tract control

The lower urinary tract stores and periodically eliminates urine. This function is regulated by a neural control system that maintains a reciprocal relationship between the bladder and urethral outlet, similar to a switching circuit.

The micturition control system comprises afferent and efferent neuronal pathways, subcortical nuclei, and cortical regions that operate at various levels and interact with each other. To facilitate a clearer understanding of the topic at hand, we will focus on and simplify the current knowledge regarding the regulation of micturition with an emphasis on the supraspinal control centers.

Afferent pathways, which convey information from receptors in the bladder concerning the degree of bladder filling, are initially relayed to the thalamus and the periaqueductal gray (PAG). The projections to the thalamus convey information relevant to the conscious perception of bladder fullness, which is subsequently used for the voluntary control of micturition. In contrast, the pathways projecting to the PAG are involved in unconscious mechanisms, specifically the micturition reflex.[10]

Afferent signals transmitted to the thalamus are further processed in the insula and the cingulate cortex, particularly the anterior cingulate cortex (ACC). The insula is thought to contribute to the subjective awareness of bladder status, while the ACC is involved in generating basic emotional responses that influence goal-directed behavior. The ACC projects to the prefrontal cortex, which in turn sends descending signals to the dorsal pons, where two key centers are located: the pontine micturition center (PMC) and the pontine storage center (PSC). These centers, through intermediate relay pathways, coordinate the activity of the lower urinary tract. Activation of the PMC leads to detrusor muscle contraction and external urethral sphincter relaxation, facilitating micturition. In contrast, the PSC exerts an inhibitory effect, promoting urine storage.[10]

In addition, cerebellar activation is frequently reported during bladder filling in functional neuroimaging; therefore, the cerebellum may play a role in the aforementioned network.[11] The PAG interacts directly with the PMC, forming the PAG–PMC switching circuit, which plays a central role in the reflexive control of micturition.

The neural control is also modulated by several neurotransmitter systems, including acetylcholine, norepinephrine, and nitric oxide, making it sensitive to various drugs and neurologic diseases.[12] Acetylcholine promotes detrusor contraction and inhibits the release of norepinephrine. Norepinephrine inhibits the detrusor muscle through beta receptors and contracts the bladder neck through alpha receptors, while nitric oxide relaxes the urethral smooth muscle.[13]

Neurological diseases cause different neuro-urological symptoms depending on the topography of the lesion.[14] The most frequently proposed hypothesis for the pathophysiology of urinary disturbances in PD is cell loss in the substantia nigra pars compacta.

In their review, Sakakibara *et al*.[10] summarized the existing data on the role of the basal ganglia in the control of micturition.[10] In this model (Figure 1), the overall function of the basal ganglia was described as inhibitory to micturition. It is assumed that dopamine exerts an inhibitory effect on the micturition reflex through D1 receptors and facilitates it through D2 receptors. Furthermore, interposed neurons releasing gamma-aminobutyric acid (GABA) are thought to exert an inhibitory influence.

Firing in the substantia nigra pars compacta and the subsequent release of dopamine activate the D1-mediated GABAergic direct pathway and modulate the micturition circuit not only through the output nuclei of the basal ganglia but also through GABAergic collaterals, primarily through the PAG. Dysfunction of this pathway in PD may lead to detrusor overactivity and the resulting lower urinary tract symptoms (LUTS).

In addition, it has been proposed that, besides the nigrostriatal pathway, the dopaminergic fibers of the ventral tegmental area–mesolimbic pathway are also involved in the control of micturition.

Patients with PD and bladder symptoms exhibit reduced uptake of [123I]-2β-carbomethoxy-3β-(4-iodophenyl) tropane in the striatum compared to PD patients without bladder symptoms, suggesting a correlation between urinary dysfunction and nigrostriatal dopaminergic cell degeneration.[15,16] In another study, bladder dysfunction was associated with a decrease in the total number of striatal dopaminergic neurons, and the relative degeneration of the caudate nucleus correlated with the severity of urinary symptoms.[17]

All of these findings provide strong evidence that LUTS in PD should be regarded as a consequence of dopamine deficiency.

3. Clinical symptoms and epidemiology

LUTS of any type, observed in neurological diseases or non-neurological conditions, have a high prevalence of 63.2%

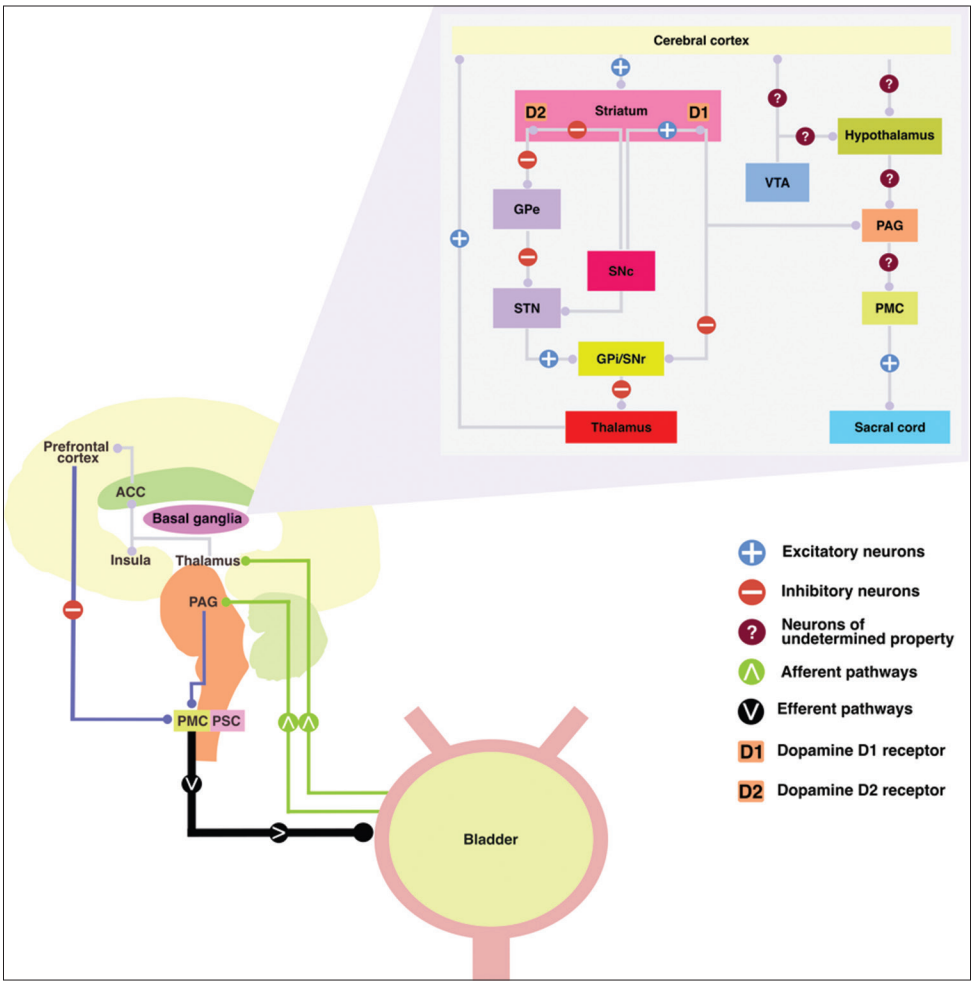

Figure 1. The physiological and pathophysiological mechanisms of the lower urinary tract control. Image modified from Sakakibara *et al*.[10] and Müller and Kaufmann[18].
Abbreviations: ACC: Anterior cingulate cortex, GPe: Globus pallidus externus, GPi: Globus pallidus internus, PAG: Periaqueductal gray, PMC: Pontine micturition center, PSC: Pontine storage center, SNc: Substantia nigra pars compacta, STN: Subthalamic nucleus, VTA: Ventral tegmental area.

in the overall population.[19] In the case of male patients, disorders of the bladder, prostate, and urethra (such as over- or underactive bladder, neurogenic bladder dysfunction, bladder tumor, benign prostatic obstruction, chronic pelvic pain syndrome, distal ureteric stone, urethral stricture, UTIs, and nocturnal polyuria[20]) collectively fall under the term LUTS.[19] However, LUTS in men are primarily associated with benign prostatic hyperplasia (BPH).[21] Notably, LUTS secondary to BPH have been identified as an independent risk factor for erectile dysfunction,[22] and LUTS, in particular nocturia, might be a subsequent predictor of erectile dysfunction.[22] In female patients, LUTS are often broadly classified into clinical entities such as over- or underactive bladder, urinary incontinence, nocturia, dysfunctional voiding, or genitourinary fistulae.[23] LUTS are also common in women, particularly symptoms of urinary incontinence, with a prevalence rate ranging from 17% to 38% and increasing with age.[19] Independent of age, urinary incontinence is 2–4 times more prevalent in women than in men.[19] Among women, stress urinary incontinence caused by pelvic floor dysfunction is common,[24] with symptomatic pelvic organ prolapse occurring in 14% of the female population.[25]

Furthermore, LUTS are commonly observed in patients with neurological disorders, being one of the most common causes of unplanned hospital admissions in multiple sclerosis, PD, and spinal cord injury.[26] Other neurological conditions in which neurogenic bladder can develop are normal-pressure hydrocephalus, cerebral palsy, traumatic brain injury, stroke, meningomyelocele, spina bifida, dementia, Guillain–Barré syndrome, and tumors involving the central nervous system.[27] The urinary symptoms typically differ according to the anatomical location of neurological lesions[26,28,29] (Table 1).

The occurrence of different LUTS also varies considerably depending on the underlying neurological condition and the stage of disease progression.[29] Detrusor overactivity is observed in approximately 27–70% of patients with PD, 50–90% of those with multiple sclerosis, 10–90% of

Table 1. Lower urinary tract symptoms according to the topography of neurological conditions

Topography of lesions	Urinary storage problem	Voiding symptoms	Post-void residual volume	Detrusor activity	Urethral tone	Other possible symptoms
Suprapontine	+	-	Low	Overactive	Normal	-
Spinal lesions (infrapontine-suprasacral)	+	+	Increased	Overactive	Overactive	Detrusor sphincter dyssynergia/areflexia; Autonomic dysreflexia if the lesion is at T-6 or higher
Sacral and infrasacral	-	+		Hypoactive/atonic	Normal/hypoactive	Cauda equina syndrome

Note: + indicates present.

individuals with dementia, and 20–50% of patients with cerebrovascular disease. Detrusor dyssynergia occurs in up to 50% of patients with spinal cord lesions, 6–30% of those with multiple sclerosis, and in approximately 50% of individuals with congenital neurological disorders. In contrast, detrusor underactivity is reported in 20–40% of patients with polyneuropathy, up to 20% in MS or chronic inflammatory demyelinating polyneuropathy, 6–18% in disc prolapses, around 30% in Guillain–Barré syndrome, and 10–60% in iatrogenic (post-operative) cases.

In neurological conditions, which may include LUTS as an early feature, an examination by the urologist precedes the diagnosis of the relevant neurological condition, such as multiple sclerosis, normal pressure hydrocephalus, early dementia, PD (including early PD), atypical Parkinson syndromes (e.g., multiple system atrophy), and spinal cord disorders (including spina bifida occulta with tethered cord and spinal stenosis). Therefore, atypical features should be identified through the new onset of severe LUTS (excluding infection), such as unusual aspects (e.g., enuresis without chronic retention) or neurological symptoms (e.g., numbness, weakness, speech disturbance, gait disturbance, cognitive impairment, and autonomic symptoms).[30]

LUTS are common yet often overlooked among the NMS of PD. LUTS subtypes in PD are typically grouped into storage symptoms, such as urinary incontinence, urinary urgency, high urinary frequency, nocturia, pollakiuria, and voiding symptoms, such as retention, dysuria, hesitancy, slow or weak urinary stream, and intermittency.[6]

According to a recent meta-analysis[6] (27 studies; n = 5,179), the pooled prevalence of LUTS in PD was 61% (95% confidence interval: 53–69), while that of storage and voiding symptoms was 59% and 24%, respectively. The frequency of LUTS by subtypes in PD is presented in Table 2.

The considerable heterogeneity of the studies should be considered a limitation of the meta-analysis, which found a higher prevalence of urinary retention in PD than previously recognized.[6] This discrepancy may stem from diagnostic inaccuracies (e.g., multiple system atrophy [MSA] vs. PD) or incomplete exclusion of comorbidities that can lead to urinary retention (e.g., diabetic neuropathy or lumbar spondylosis).

Table 2. Prevalence of storage and voiding symptoms

Type of symptoms	Frequency (%)
Storage symptoms	
Urinary incontinence	30
Urinary urgency	46
High urinary frequency	52
Nocturia	59
Pollakiuria	65
Voiding symptoms	
Retention	27
Dysuria	22
Hesitancy	20
Slow stream/prolongation	41
Weak stream (spraying)	22
Intermittency	30

The prevalence of LUTS depends on the Hoehn and Yahr stage[6,31] and the presence of dementia.[32] Higher prevalence of incontinence was associated with higher age (≥65 years), female gender, and higher Hoehn and Yahr stage.[6,31] Cognitive impairment in patients with PD is more strongly related to the severity of urinary incontinence than to motor symptoms.[33]

While pollakiuria, nocturia, urgency, and prolongation occur more frequently,[6] incontinence and dysuria are usually more concerning for the patients, based on our clinical experience. Another point of interest is the post-void residual volume (PVR), a quantifiable measure of voiding function, proposed as a screening method.[29] PVR has the lowest prevalence rate (4–6%) according to previous studies.[5,6] Notably, depending on the patient selection criteria, the characteristics of a urodynamic study cohort might differ from a patient cohort in a neurological clinical setting.

However, in a study involving 196 patients with *de novo* PD without urological and gynecological disorders, the occurrence of high PVR (>100 mL) showed a significant difference between the PD, progressive supranuclear palsy, and MSA subgroups—6%, 5%, and 43%, respectively. These findings suggest that elevated PVR may serve as a supportive marker in the differential diagnosis of MSA.[5]

4. Diagnostic procedure

Because diagnostic approaches for the various subtypes of neuro-urological dysfunction differ little, the following diagnostic cascade is proposed according to the relevant guidelines:[34]

(i) Medical history of the patient. For example, the exclusion of internal medical causes, such as heart failure, diabetes, chronic venous insufficiency, or sleep disorders, that is, sleep apnea, in the background of nocturia.[29,34]
(ii) Neurological, urological, and, if necessary, gynecological physical examination.
(iii) Bladder diary for more than 3 days.
(iv) Urine sediment
(v) PVR.[35]
(vi) Uroflow
(vii) Video-urodynamics. Urodynamics showed patterns specific to PD in previous research that investigated the changes in urodynamic parameters (maximum urinary flow rate, bladder volume at the first desire to void, maximum bladder volume, detrusor pressure at maximum flow rate, PVR, and bladder compliance) as well as the treatment efficacy in male PD patients with voiding dysfunction.[36] According to the voiding symptoms and the results of the urodynamic examination, an overactive detrusor and detrusor dysfunction, with or without bladder outlet obstruction, can be distinguished, with different therapeutic responses.
(viii) Urethrocystoscopy.
(ix) Renal sonography.
(x) Biochemical tests (creatinine, urea, and possibly 24-h creatinine clearance).

In addition, electrophysiological examinations such as electromyography of the pelvic floor muscles, urethral sphincter, and/or anal sphincter, nerve conduction studies of the pudendal nerve, reflex latency measurements of the bulbocavernosus and anal reflex arcs, evoked responses from the clitoris or glans penis, and sensory testing on the bladder and urethra may be considered.[37] Notably, NMS in PD can fluctuate. According to a cross-sectional study, bladder urgency was reported to be more frequent in the "off" state compared to the "on" state.[38]

5. Differential diagnosis

5.1. Non-neurological conditions

In differential diagnosis, it is important to exclude non-neurogenic causes of bladder dysfunction, such as pelvic floor prolapse in women,[24] prostatic hyperplasia in men, or bladder tumours.[34] Prostatic hyperplasia may present as a reduced urinary stream and/or elevated PVR, while tumors often present with hematuria. Psychiatric and psychosomatic illnesses must also be differentiated.[34] Treatment of nocturnal enuresis might require the involvement of sleep medicine experts.[29] In addition, UTIs should be ruled out. The risk of UTI increases with age, and it generally affects women more; however, similar to the LUTS, PD-associated UTI occurrence is relatively identical in older men and women.[7] Fowler's syndrome (isolated urinary retention) due to impaired sphincter relaxation in women is often observed alongside polycystic ovaries.[34]

6. Treatment options

Management of LUTS in PD is complex and aims to protect the upper urinary tract,[29] improve urinary continence and QoL, and restore lower-urinary-tract function.[34,37]

Non-neurological causes of LUTS (e.g., BPH) should be excluded or treated before treating LUTS associated with neurological conditions.[29] Therapeutic options include non-invasive approaches such as bladder training and pharmacotherapy, and invasive interventions such as catheterization, the sacral neuromodulation of S3 or sometimes S4, botulinum neurotoxin (BoNT) injections into the detrusor,[29,39] and surgical methods.

Bladder training and behavioral therapy, including pelvic floor muscle exercises, may improve the QoL of PD patients with LUTS[40-42] without significantly reducing urgency episodes, according to a previous study.[42]

As dopamine selectively inhibits or activates the PMC (Figure 1), optimizing dopaminergic treatment may help in the management of urinary urgency secondary to detrusor overactivity in PD.[43] However, the effects of levodopa on LUTS vary[44] and can be unpredictable.[45] There may be an initial period of worsening in bladder symptoms after starting levodopa treatment; however, improvement in bladder function has been observed in the same patients after 2 months of continuous use.[46] In one study, 3 months of levodopa treatment in PD patients was associated with modest improvements in storage urodynamic parameters,[44] whereas another study reported worsening detrusor overactivity with levodopa in some patients.

The mainstay of pharmacotherapy is the use of anticholinergics operating on the muscarinic acetylcholine receptors (i.e., antimuscarinics) and β3 adrenergic receptor agonist drugs in detrusor overactivity and detrusor sphincter dyssynergia, and the use of alpha blockers in detrusor underactivity.[34] Antimuscarinics and the vasopressin analog desmopressin are proposed for treating nocturia.[34] Before administering desmopressin, underlying internal medical causes should be ruled out, and nocturia should be differentiated from pollakiuria. Notably, the use of desmopressin is contraindicated in patients with polydipsia,

cardiac impairment, or other conditions requiring treatment with diuretic therapy, as well as in those with moderate or severe renal impairment or established hyponatremia. Pollakiuria is also recognized as a common side effect.[47] Propiverine, tolterodine (also in extended-release form),[48] fesoterodine, solifenacin, darifenacin, and trospium chloride fall under the category of antimuscarinics. Typical adverse effects of this pharmacotherapeutic group are residual urine formation,[49] constipation, accommodation disorder, dry mouth,[50] tachycardia, arrhythmia, increase in intraocular pressure, fatigue, concentration disorders, and occasionally psychosis in patients with PD.[34] Due to their anticholinergic effect, antimuscarinics might worsen the symptoms of PD. A therapy with antimuscarinics alone might be insufficient to prevent high bladder pressure in cases of detrusor sphincter dyssynergia; however, detrusor sphincter dyssynergia in PD is rare,[31] and in this case, it could be supported by intermittent catheterization.[29] Trospium chloride is well-suited for PD, as it does not cross the blood–brain barrier.[34] Likewise, the active metabolite of fesoterodine has limited ability to cross the blood–brain barrier and is actively transported from the central nervous system. Based on its pharmacokinetics, this prolonged-release drug is well tolerated by the elderly. Fesoterodine does not undergo first-pass hepatic activation and has a balanced affinity for M2 and M3 receptors.[51]

Mirabegron is a representative of the β3 adrenergic receptor agonist group. Mirabegron is an effective therapeutic agent in managing neurogenic bladder[52] unresponsive to antimuscarinics, particularly in patients with storage symptoms.[53] Vibegron is the second active substance of this pharmacological group.[54] Caution is recommended for combination therapy with anticholinergics in the elderly due to the increased risk of dementia.[29] According to a meta-analysis, anticholinergic use for more than 3 months increased the risk of dementia on average by an estimated 46% compared to those without usage. This relationship was consistent in studies assessing overactive bladder medications.[55]

The combination treatment of mirabegron and the antimuscarinic solifenacin was more effective than mirabegron alone; however, the treatment presented with anticholinergic side effects,[56] such as dry mouth and constipation.[49]

It should be noted that both antimuscarinics and β3 agonists are approved for the symptomatic treatment of urge incontinence and/or increased urinary frequency and urgency associated with overactive bladder syndrome in adults. However, they may also be used off-label to manage neurogenic bladder dysfunction in patients with PD.

Although the antimuscarinics and β3 agonists have been shown to be effective in managing LUTS, they have high treatment discontinuation rates due to their adverse effects.[57] In cases where anticholinergic treatment has an insufficient effect and/or serious side effects, a therapy with BoNT[58,59] can be considered. However, existing residual urine is a relative contraindication. The BoNT therapy is typically performed by a urologist: 100–300 units of BoNT-A are injected into the detrusor muscle under endoscopic control.[59] There are also clinical studies and regulatory approvals supporting the use of aboBoNT-A. Expected advantages of the therapy include reductions in urgency, reductions in incontinence, and an improvement in QoL as well as in urodynamics.[59] The possible side effects include an increased risk of high PVR, hematuria, or UTI.[59] Similarly to antimuscarinics and β3 agonists, BoNT is approved to treat overactive bladder, but its use in managing neurogenic bladder dysfunction in PD remains off-label.

Urinary retention can also be treated with intermittent self-catheterization,[34] where the frequency depends on the urodynamic bladder capacity.[34]

If the above-mentioned procedures are unsuccessful in treating a detrusor overactivity or detrusor–sphincter dyssynergia, invasive measures such as suprapubic catheterization (Foley), sacral neuromodulation, or surgical methods are proposed, such as urinary bladder augmentation, urinary diversion, sphincterotomy, or transurethral resection of the prostate in patients with coexistent BPH.[34,37] In addition, when assessing the appropriateness of urological procedures in PD patients, cognitive functions should be considered in addition to motor function.[33]

The management of individuals with neurological conditions, including PD—particularly those at increased risk of developing upper urinary tract damage (such as elevated PVR volumes, requiring catheterization), with suspected comorbid primary urological conditions, or with urinary symptoms unresponsive to first-line treatment—should be conducted collaboratively between neurologists and neuro-urologists.[26]

7. Conclusion

Urinary symptoms associated with PD are NMS that adversely affect the overall QoL of patients. The pathomechanism of LUTS in PD is complex, and not all the aspects are fully understood. Beyond the clinically evident storage and voiding symptoms, increased PVR has been proposed primarily as a screening tool for neuro-urological dysfunctions in PD. Available study data suggest that clinically significant elevations in PVR are relatively uncommon and differ significantly between PD and MSA, suggesting that the significance of PVR might be greater than that of a LUTS screening tool. It is very important to consider the relationship between PD and bladder dysfunction and not to classify these symptoms independently. This integrated perspective is equally important when planning therapy. Further studies

are needed to clarify the pathogenesis of LUTS in PD, which potentially leads to new treatment options.

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Conflict of interest

The authors declare that they have no competing interests.

Author contributions

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Ethics approval and consent to participate

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