Review Article

Bladder dysfunction following stroke: An updated review on diagnosis and management

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Abstract

Bladder dysfunction represents a frequent and important clinical problem in stroke patients. The aim of this narrative review was to explore the currently available information regarding the diagnosis and management of bladder dysfunction following stroke. The most common symptoms of bladder dysfunction following stroke are urinary incontinence, urgency, increased frequency, and difficulty voiding. Medical history, including voiding diary, physical examination, and urodynamic studies are useful in establishing diagnosis. Bladder pressure in stroke patients with detrusor overactivity is rarely high enough to damage the upper urinary tract. In neurogenic bladder, however, there is always a risk for transmission of intravesical pressure to the upper tract. In incontinent patients, urodynamic studies can reveal bladder hyper- or hyporeflexia, detrusor overactivity with impaired contractility or detrusor-sphincter dyssynergia, or even no abnormalities at all. With stroke patients with urinary dysfunction, establishing a proper diagnosis is of paramount importance to start appropriate treatment, prevent upper tract damage, maintain continence, and ensure complete emptying. After diagnosis, an individually tailored treatment plan is mandatory, including behavioral techniques, lifestyle interventions, and anticholinergic medication. Other therapeutic choices include alternative drugs, intradetrusor injection of botulinum toxin, and spinal neuromodulation. A bladder rehabilitation program is essential for improving post-stroke lower urinary symptoms and depends on the patient's awareness, cooperation, and independence. Bladder dysfunction after stroke, as a strong prognostic factor of disability, exerts an enormous impact on health and economy. Therefore, every single effort toward a proper diagnosis and effective rehabilitation is crucial.

Keywords: Bladder dysfunction, Lower urinary tract dysfunction, Stroke, Urinary incontinence, Urodynamic studies

1. INTRODUCTION

Cerebral stroke is among the most common causes of death and disability worldwide [1-3]. Bladder dysfunction is a well-known complication in stroke patients [4-6] that severely affects quality of life (QoL) [2,7,8]. Urinary symptoms are reported in the majority of patients [9] and primarily include urinary incontinence (UI) and voiding difficulty [2]. UI is more frequent [4], affecting approximately 33% of patients with acute stroke [4,9-11] and persisting for up to 3 months after stroke [11-14]. Its frequency ranges between 28% and 79% [12,13,15-17], and several factors contribute to this issue [16].

The European Association of Urologists (EAU) has not formulated any specific guidelines for urinary dysfunction after a stroke. According to EAU, the pattern of urinary symptoms depends on the anatomical site of neurological lesions, namely supra-pontine, spinal, and sacral/infrasacral ones. Anatomical lesions in the brain are not so clearly classified (EAU Guidelines. Edn. presented

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at the EAU Annual Congress, Paris 2024, ISBN 978-94-92671-23-3) [18].

Clinically, post-stroke symptoms mainly involve nocturia, overactive bladder (OAB), urgency UI, neurogenic detrusor overactivity, and other less frequent patterns: (a) 57 – 83% of neuro-urological symptoms at 1-month post-stroke, (b) 71 – 80% spontaneous recovery at 6 months, and (c) persistence of UI that is correlated with poor prognosis.

Spasticity is a well-studied stroke complication, which can severely impair bladder control [19]. Neurogenic bladder [14,20] may coexist with the so-called "functional bladder," which results from associated deficits (immobility, cognitive dysfunction, etc.) [14]. Pre-existing urological problems, such as benign prostatic hyperplasia (BPH), may worsen urinary symptoms in many cases. Stroke has a higher prevalence in the elderly and, as a result, comorbidities are common. BPH should always be kept in mind in dealing with aging men with stroke and need to be excluded to manage urinary symptoms properly. Specifically, the presence of detrusor–sphincter dyssynergia in elderly males with stroke should warn clinicians of possible prostatic urethra obstruction. The differential diagnosis is difficult and the presence of BPH needs to be confirmed by integrating clinical, imaging, and urodynamic assessment. Another example is diabetic patients in whom urinary problems may coexist due to peripheral neuropathy [21].

Bladder dysfunction, which is of prognostic importance, exerts social impact, and incurs a remarkable economic burden [22], is unfortunately often overlooked in these patients. Relevant literature data are also relatively scarce. In light of the impact of bladder problems on victims of cerebrovascular events, the primary purpose of this review article was to look into currently available data regarding the diagnosis and management of bladder dysfunction following stroke.

2. METHODOLOGY

Methodologically, a literature search was conducted in PubMed using the terms "bladder dysfunction" and "stroke," and the search retrieved 272 related articles. The authors examined all articles published from the inception until March 2021 for potential eligibility. Publications in the English language, including those in foreign language publications with English abstracts, were included in this non-systematic review. Articles concerning bladder dysfunction after stroke, its diagnosis, and management were further analyzed. Our exclusion criteria were (1) articles irrelevant to our topic or them, (2) lesions not affecting the brain, and (3) non-stroke brain lesions. Eventually, a total of 62 articles remained and were used for this narrative review. Data were collected by five independent investigators and then synthesized in a narrative format for a thorough review.

3. CLINICAL FINDINGS

3.1. UI

UI is the predominant urinary symptom and may last for up to 1 year after stroke. Incontinence is correlated with large infarcts [9-11,16], aphasia, cognitive impairment, and functional disability [10,11]. Different types of UI have been described, including urge incontinence from detrusor overactivity, due to disruption of the neuro-micturition pathways (reduced supra-pontine micturition control) [23], overflow incontinence from detrusor underactivity, impaired awareness of incontinence [10,11,13], functional incontinence, exacerbation of pre-existing stress incontinence [11,13], incoordination/weakness of sphincter muscles, impaired bladder's sensation [24], and transient incontinence due to potentially reversible causes, such as fecal impaction or medications [11]. Risk factors for UI include advanced age, female sex, depression, limited activity, large lesions [9,25], aphasia [25], and underlying medical conditions, such as diabetes or pelvic prolapse in women [9,11,13,25,26]. UI has been found in 73% of patients with hemorrhagic and 64% with ischemic stroke and urinary retention in 13% and 52%, respectively [27].

3.2. Other urinary symptoms

Apart from incontinence [4,9-11], other common post-stroke urinary complaints involve urgency [14,25,28], high frequency [14,25,28], nocturia [25,28], leakage of urine [28], and difficulty voiding [14]. Urgency and frequency of micturition along with urge UI consist of the so-called "Overactive Bladder" syndrome [14]. According to another study, nocturnal urinary frequency is the most common urinary problem, which is followed by incontinence [14]. The prevalence of both urgency and UI is closely associated with the infarction size [7]. Retention is much rarer [9,14,26] and has been associated with cortical strokes, diabetes, aphasia, and cognitive disorders [9]. Other bladder issues in chronic stroke patients include infections (10 – 27%) [4,26,29], asymptomatic (12%), and significant bacteriuria (39%) [29]. In one study, the patients were divided into two groups: those who had an infarction and those who had a hemorrhagic stroke with lower urinary tract dysfunction. Patients with ischemic stroke presented larger bladder capacity (250 mL) and larger post-void residual urine (PVR) volume (136 mL) than their counterparts with hemorrhagic stroke (195 mL and 30 mL, respectively), which may indicate a more severe bladder dysfunction [14].

The bladder-emptying method and residual (post-void) urine volume >50 mL are important risk factors for

urinary infections and significant bacteriuria [26,29]. Lower urinary tract manifestations after stroke are sometimes more complicated to address. For instance, during urinary catheterization, which is often used to count urinary output and estimate fluid balance [30], trauma, or infection can result, in further deteriorating such symptoms [31]. Table 1 summarizes the most important locations of lesions and their relevant patterns of bladder dysfunction.

3.3. Diagnostic evaluation

Bladder dysfunction after stroke is very common and should be primarily evaluated to better formulate treatment and follow-up plans. A thorough history taking should include past and present urinary symptoms, and also medications received and other concomitant diseases. Information about types of dysfunction, such as voiding or storage problems, that include frequency, voided volume, stress or urgency incontinence, and incontinence episodes, should also be collected or obtained.

Bladder diaries are considered a valuable diagnostic tool for the initial/preliminary assessment of neurogenic lower urinary tract dysfunction. They provide data on the number of voids (spontaneous or intermittent catheter), voided volume, stress/urgency/mixed UI episodes and facilitate the interpretation of urodynamic testing. Preferably, bladder diaries should be completed for 3 consecutive days [42].

Along with the voiding diary, a clinical examination is also necessary before any additional diagnostic investigation [41]. In early assessment of stroke severity, the NIH Stroke Scale/Score is the most well-established tool. However, it does not include any field concerning lower urinary symptoms. Moreover, in the acute phase after stroke, patients usually need to wear an indwelling catheter, depending on the level of consciousness, the clinical status of the patient, and his or her ability to urinate. In those with indwelling catheters, ideally, an urodynamic study should be performed a few days after decatheterization. In those without a catheter, residual urine should be measured after voiding with a bladder ultrasound repeatedly [43]. The bladder's pathophysiology and functional status can evolve over time [13]. Consequently, urodynamic findings vary, depending on post-stroke phases [44]. Urinary obstruction due to non-neurologic causes also exists [45]. Urodynamic studies (cystometry and pelvic floor electromyography) are very useful in determining pathophysiological mechanisms of bladder dysfunction [11,13,16,19,20,43,46,47] and investigating the cause of incontinence. A standard urodynamic study involves non-invasive uroflowmetry, followed by invasive cystometry and a pressure-flow study, and may reveal a great deal about urinary flow, intravesical and abdominal pressure, urethral pressure profile, and PVR. Urodynamic testing in stroke patients can vary but can reveal uninhibited detrusor overactivity in up to 90% of patients [13]. The following patterns have been described in stroke patients: normal study meaning that there is no abnormal detrusor activity, normal bladder sensation, and normal voiding with complete bladder emptying, detrusor overactivity with involuntary detrusor contractions during the filling phase, detrusor overactivity with impaired contractility during filling phase accompanied by urine loss and terminal overactivity which results in incomplete emptying with large PVR, and detrusor underactivity where contractions are either weak or of short duration leading to prolonged and incomplete emptying [44].

Video-urodynamics, which is considered the gold-standard investigation for patients with neurogenic bladder, combines standard urodynamic testing with fluoroscopic imaging, with radiographic contrast used in bladder filling [10,43].

Bladder pressure in stroke patients with detrusor overactivity is rarely high enough to damage the upper urinary tract [46]. With neurogenic bladder, however, there is always a risk for the transmission of intravesical pressure

Table 1. The most usual locations of lesions related to bladder dysfunction, their pathophysiological pathways, and clinical consequences

Location of lesion	Pathophysiology	Clinical effect	References
Frontal lobe	Disruption of frontal connections: overdrive of renal angiotensin system	Detrusor overactivity leads to urinary incontinence (predominantly) and retention	[14,17,26,32-34]
Parietal lobe	Disruption of afferent pathways to the micturition center (reflex)	Reduced or absent urine sensation: leakage	[32,35]
Insula	Unclear	Urinary retention	[36]
Basal ganglia	Unclear	Detrusor areflexia	[37]
Cerebellum	Unclear	Detrusor overactivity	[14,38,39]
Pons	Disruption of the descending inhibitory fibers of the midbrain tegmentum Damage to the pontine micturition center The lesion in the central pons	Increased bladder residual volume Detrusor dyssynergia Detrusor underactivity leads to urinary retention	[33,34,40]
Medulla oblongata	Disruption of the descending stimulatory fibers of the pontine micturition center	Detrusor overactivity	[41]

to the upper tract [43]. In incontinent patients, urodynamic studies can reveal bladder hyper- or hyporeflexia [10,44], detrusor overactivity with impaired contractility [44], or detrusor-sphincter dyssynergia [10], or even no abnormalities at all (37%) [10,44]. Patients with hyporeflexic bladders are usually either diabetic or on anticholinergic medication [10]. Furthermore, urodynamic findings in patients with a disturbed perception of the need to void include terminal detrusor overactivity, incompetent urethral closure, or no abnormalities [23]. In possible neurogenic bladder cases, PVR should be measured since its volume is associated with infection, bladder stone formation, and impaired renal function [46].

3.4. Management and rehabilitation

In stroke patients with urinary dysfunction, an accurate diagnosis is of paramount importance to start an appropriate treatment, prevent upper tract damage, maintain continence, and ensure complete emptying. Treatment strategies should be individually tailored and structured [15]. Nursing care during the acute stroke phase is also important for early detection of bladder dysfunction [48].

Behavioral techniques should be applied as the first step. These include urination on a schedule with or without physical assistance (tapping over the bladder), bladder training, pelvic floor muscle training [11-13,15,16,26,46,49,50], "double voiding" (waiting and attempting to void again after voiding once) [26], and fluid management [11,12,26], along with other lifestyle interventions, such as reducing medications that exacerbate incontinence [11,13]. In fact, scheduled voiding or timed voiding has been shown to be superior to pharmacological treatment in many incontinence cases [10]. Some evidence proved that a voiding program starting at 12-week post-stroke is beneficial, mainly in patients with urge and stress incontinence [50]. Patients' training that focuses on the bladder's sensation, inhibition of its contractions, and improvement of the sphincter's voluntary control, can also treat incontinence effectively [24]. Patients with disturbed awareness of their voiding need may benefit from early training and additional medication for bladder overactivity if needed [35].

The bladder rehabilitation program is essential in improving post-stroke lower urinary tract symptoms and depends on the patient's awareness, cooperation, and independence. For cooperative patients with independent ambulation, but unaware of the status of their bladder experiencing either urinary retention or UI, timed voiding is a rational choice. For those with the same symptoms but aware of the bladder situation, prompted voiding is more appropriate. For independent patients with urge symptoms who are motivated to participate in a rehabilitation program, bladder training with urge suppression is a good choice. The best approach to improve post-stroke symptoms of the lower urinary tract is a combination of remedial approach and functionally oriented approach. The first uses neurodevelopmental techniques to improve impairment and the second aims to optimize function [51].

If these measures are not efficacious, then medication, primarily anticholinergics, should be considered [11,13,16,43,46,49,52]. Antimuscarinic drugs are considered the first-line medication choice for patients with neurogenic lower urinary tract dysfunction. Antimuscarinic agents reduce urge, stabilize detrusor contractility, and increase bladder capacity. Oxybutynin chloride, trospium chloride, tolterodine tartrate, and propiverine are established and effective antimuscarinic agents. Solifenacin is a new oral anti-cholinergic drug, with a high affinity for the M3 muscarinic receptor in the bladder, but data for its efficacy in neurogenic bladder are limited. Antimuscarinic drugs are generally well tolerated and safe, but adverse effects limit their use. The most common adverse reactions are dry mouth, blurred vision, and constipation. Less frequent are somnolence and impaired cognitive function [52]. The gold standard for patients with bladder overactivity after stroke is the combination of intermittent catheterization with anti-muscarinic drugs [42]. Pilot studies have shown that phosphodiesterase-5 inhibitors have worked effectively on detrusor overactivity and in the future, they may be used as adjuncts to antimuscarinic drugs [43]. Adverse events and limited efficacy, however, make alternative therapies desirable [13,52].

Other less-used drugs include adrenergic alpha-blockers [12,53] and beta-3 agonists [13]. Most alternative drugs act peripherally, primarily on neurotransmission or directly on the detrusor muscle. Just a few drugs with a well-defined central nervous system action can be used for treating micturition disorders, such as baclofen and duloxetine [52]. These treatment strategies are good choices for patients with overactive detrusor [13,16,46,49]. Some clinical data also mentioned the potential benefits of electroacupuncture in detrusor overactivity [54]. Patients with underactive detrusor muscles should be managed with intermittent catheterizations or an indwelling catheter to minimize PVR and prevent upper tract damage [13,16,46]. If these measures fail, intra-detrusor injection of botulinum toxin has shown promising results [12,46]. Most usual applied medications are summarized in Table 2. Of course, if UI persists, containment devices may be required [11].

Non-invasive neuromodulation may improve urinary dysfunction in selected patients [12,15,55,56]. Transcutaneous electrical nerve stimulation in combination with an exercise program has shown significant improvement in spasticity

Table 2. Medications for stroke-related bladder dysfunction, their action, and main adverse effects

Frequency of use	Medical agents	Actions	Adverse effects	References
Frequently used	Antimuscarinic agents: oxybutynin chloride, trospium chloride, tolterodine tartrate and propiverine	Reduce urge, stabilize detrusor contractility, and increase bladder capacity	Frequent: dry mouth, blurred vision, and constipation. Less frequent: somnolence and cognitive impairment	[19,52]
Less frequently used	Phosphodiesterase-5 Inhibitors	Effective on detrusor overactivity	Headache, flushing, nasal congestion, nasopharyngitis, and dyspepsia	[13,43,52]
	Adrenergic alpha-blockers and beta3 agonists	Effective on detrusor overactivity	Hypertension, nasopharyngitis, urinary tract infections, dry mouth, back pain, upper respiratory tract infections, sinusitis, headache and dizziness	[12,13,53]
	Intra-detrusor injection of botulinum toxin	Effectiveness on detrusor underactivity	Pain at the injection site, urinary tract infections, hematuria, and an increase in post-void residual volume	[12,46]

and bladder function [3]. Sacral nerve stimulation is a surgical treatment (invasive neuromodulation) of lower urinary dysfunction of non-neurogenic origin approved by the Food and Drug Administration and can benefit patients with neurogenic bladder as well [57]. In addition, electrical stimulation of the posterior tibialis nerve may improve OAB symptoms after stroke [58]. Another non-invasive treatment approach for the UI may be the combination of pelvic floor muscle exercise with biofeedback or/and pelvic floor electric stimulation. A meta-analysis and systematic review regarding this combination approach was beneficial to the recovery of UI after radical prostatectomy. Hence, this treatment option may also, to some extent, work on stroke patients [59].

3.5. Prognosis and impact

In stroke survivors, UI usually improves with time [7,12,13,49,60], with a frequency of 32 – 79% in the acute phase of stroke, 27% at the time of discharge, and 16% several months following stroke [7], reflecting some recovery of bladder control [60]. Other studies have shown an incontinence frequency of 19% at 3 months, 15% at 1 year, and 1% at 2 years [13]. Half of patients with urgency incontinence may become continent after 1 year, a common improvement for patients with disturbed perceptions of their need to void [35].

Although post-stroke recovery of urinary symptoms is possible, it remains a lingering problem for many patients. Problems associated with urinary dysfunction can significantly affect QoL [15,16,25]. Urgency incontinence and nocturia probably have the strongest impact on QoL [24]. UI is a strong predictor of long-term disability [9,13,55], depression [9,13,14,55], and prolonged institutionalization [4,9,11,13,16,25,55]. Moreover, incontinence is associated with stroke severity [9,11,24], poor functional [9,11,14,15] and cognitive status [9,14], urinary tract infection [9,16,25,61], skin breakdown [16,25], falls [14,25], and mortality [11,14,16]. Activities of daily living can also be affected by anxiety [4], low self-esteem, embarrassment, and a sense of fear, which may lead to decreased participation in the rehabilitation program [25]. As a result, bladder dysfunction can interfere with the rehabilitation process and delay the social integration of stroke patients [14,16,61].

Mobility independence after stroke accompanies independence of continence in most cases. This parallel recovery may be explained by common cortical/subcortical pathways of continence and motor function. Rehabilitation interventions also help urinary recovery in patients with functional or mixed UI through improvement of mobility independence [49]. Furthermore, the absence of severe bladder dysfunction is associated with home return from a rehabilitation center [62-64]. Thus, evaluation of urinary dysfunction and its consequences, as well as their treatment, is vital, and rehabilitation efforts should aim to promote recovery of urinary dysfunction [9,13,25,49].

4. CONCLUSION

The prevalence of stroke nowadays has been on the rise and bladder function after stroke is frequently impaired, affecting the QoL of stroke survivors. Despite rehabilitation efforts and the fact that bladder dysfunction usually improves over time, it remains a major issue substantially impacting both the patient and society. However, this problem is often overlooked and poorly managed. A thorough evaluation of the severity of lower urinary tract symptoms and an individualized treatment plan is crucial. The latter is associated with better prognosis and outcomes and should be included in the primary goals of all physicians treating stroke sufferers.

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CONFLICT OF INTEREST

The authors declare no competing interests.

AUTHOR CONTRIBUTIONS

Conceptualization: Eleni Agapiou, Efstratios-Stylianos Pyrgelis
Writing – original draft: All authors
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ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Not applicable.

CONSENT FOR PUBLICATION

Not applicable.

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