Contemporary Evaluation and Treatment of Poststroke Lower Urinary Tract Dysfunction

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KEYWORDS

• Stroke • Overactive bladder (OAB) • Urinary incontinence • Underactive bladder (UAB) • Lower urinary tract symptoms (LUTS) • Geriatrics

KEY POINTS

- A detailed clinical history and physical examination are key components to understanding lower urinary tract dysfunction in patients who have experienced stroke.
- The physiology and clinical function of the bladder depend on the location and severity of stroke, and can evolve over time.
- Urodynamic evaluation can be useful cases to evaluate bladder function and guide therapy, but should generally be delayed until the patient has reached a stable point.
- Many therapies can be used to treat poststroke lower urinary tract dysfunction; therapy should be tailored to each individual patient's unique clinical situation and goals of care.

INTRODUCTION

Strokes, sometimes referred to as cerebral vascular accidents, continue to be a leading cause of morbidity and mortality in the United States. Of the 800,000 strokes that occur annually in the United States, approximately 140,000 people die, leaving a substantial patient population with poststroke sequelae, such as urinary incontinence.[1] The prevalence of patients affected by urinary incontinence after a stroke ranges from 28% to 79%.[2] The odds of increased mortality are worse in patients with sustained incontinence at 1 year than those who regain normal function in the same time frame.[3] Poststroke urinary incontinence is a strong predictor of increased disability, greater institutionalization, and mortality.[4] These data suggest that poststroke urinary incontinence, when successfully treated, can improve the quality of life for patients. Additionally, when clinicians address and manage the specific urologic needs of stroke patients, such as urinary incontinence, evidence suggests better stroke outcomes.[5]

The literature for the specific management of lower urinary tract symptoms in stroke survivors is limited. The goal of this review is to evaluate the prevalence, risk factors, types, causes, and contemporary management of voiding dysfunction in stroke survivors. Studies were identified by performing an electronic database search of PubMed, Medline, and Cochrane Library using the following keywords: Stroke, Overactive Bladder (OAB),

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Urinary Incontinence, Underactive Bladder (UAB), Lower Urinary Tract Symptoms (LUTS), and Geriatrics. Articles reviewed included papers published before 2017 and in English or English translation.

PREVALENCE OF POSTSTROKE INCONTINENCE

Urinary incontinence is a well-described acute poststroke sequela, with reports of incidence at the time of initial hospitalization ranging from 28% to 79%.[6] Patel and colleagues[7] found that 40% of patients were incontinent 1 week after admission to the hospital after a cerebral vascular accident in a population-based study from 2001. Similarly, a community-based study by Kolominsky-Rabas and colleagues[8] spanning a 4-year period that included 699 patients reported incontinence rates of 35% 7 days after stroke in previously continent patients. Those patients with persistent incontinence at 12 months had worse morbidity from their stroke and a greater risk of being institutionalized (45% vs 5%). More recent data from 2012 by Williams and colleagues[4] found that nearly 44% of stroke survivors were incontinent at 3 months and 38% at 12 months with urge urinary incontinence being the most prevalent. If a patient has persistent urinary incontinence at 1 year, this is a predictor of greater mortality, poorer functional recovery, and institutionalization.[9] Importantly, many patients will show improvement in their voiding dysfunction within 1 year after cerebral vascular insult. This finding suggests the need for a dynamic and needs-specific treatment plan.

CAUSES OF POSTSTROKE URINARY INCONTINENCE

The causes of poststroke urinary incontinence seem to be multifactorial and distressing to both patients and their caregivers. It often negatively impacts a patient's well-being by creating social stigma, decreasing quality of life, and creating physical and emotional discomfort.[7] Jørgensen and colleagues[10] found an increased prevalence of urinary incontinence in stroke patients with impaired cognition, poor lower extremity motor function, and signs of depression. Tibaek and colleagues[11] demonstrated through questionnaires an association of poorer sense of well-being in stroke patients with lower urinary tract symptoms compared with stroke patients who did not develop urinary incontinence. Earlier data from Gelber and colleagues[12] prospectively studied 51 patients after a unilateral hemispheric stroke, of which 19 had urinary incontinence. Voiding dysfunction was associated with large infarcts, aphasia, cognitive impairment, and functional disability.

In many stroke patients, urinary incontinence is transient. Patel and colleagues[7] followed 235 patients over a 2-year period and found the prevalence rates of incontinence decreased with time: 19% at 3 months, 15% at 1 year, and 1% at 2 years. Rotar and colleagues[3] found that more than one-half of patients with first-ever stroke had urinary symptoms after vascular insult. Those patients who regained continence quickly after stroke, within 1 week, had a similar prognosis to those who did not have poststroke urinary incontinence. Sustained voiding dysfunction after stroke is a predictor of greater mortality and a poorer functional outcome.[7]

The relationship between infarct size and location with poststroke urinary incontinence remains controversial. An earlier study by Reding and colleagues[13] reported no correlation between infarct size and poststroke urinary incontinence. However, Feder and colleagues[14] did find a significant association between infarct size (>40 mm in diameter) and onset of urinary incontinence. Gelber and colleagues[12] elucidated that infarct size does matter, because it likely disrupts the neuromicturition pathway resulting in detrusor overactivity. More recent data from Patel and colleagues[5] found that patients who experienced total anterior circulatory infarctions were less likely to regain continence compared with patients with lacunar infarctions with an odds ratio of 3.65 (95% confidence interval, 1.1–12.2) at 3 months.

To date, these studies have linked poststroke urinary incontinence with depression, age, hemiparesis, motor weakness, and impaired cognition. There remains controversy about the relationship between infarct size and location, and their roles in poststroke urinary incontinence. This continues to be a fertile area for future research. It is clear, however, that those patients who have sustained incontinence do more poorly than their counterparts who regain continence quickly after a vascular insult.

NORMAL NEUROMICTURITION PATHWAY

Normal neural control of storage and voiding involves complex coordinated communication between the peripheral ganglia, spinal cord, and brain.[15] These pathways control smooth and striated muscles of the bladder, bladder neck, urethra, and sphincters that act in a coordinated manner to allow for bladder filling, or urine storage, and voiding at a socially acceptable time. The