LETTERS TO THE EDITOR

Dermatologist and urologist? A neglected relationship in neurogenic voiding dysfunctions

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Neurogenic bladder (NB), also known as neurogenic lower urinary tract dysfunction, due to central and/or peripheral nervous system diseases, is one of the most challenging problems in urology. Various disorders or injuries (e.g., stroke, Parkinson's disease, multiple sclerosis, spinal cord injury) affecting the nervous system may cause chronic bladder dysfunction, whose type depends on central or peripheral nervous system damage level and intensity. The bladder can become underactive (not emptying completely) or overactive (emptying too frequently/quickly), with urethral sphincter deficiency (evoking incontinence) or overactivity (leading to dyssynergia with partial or complete urinary retention).

The pattern of specific dysfunction following neurological disorder is determined by the site and the nature of the lesion [1]. Whereas brain lesions and those above the pontine micturition center typically lead to detrusor overactivity, those below the pons and above the sacral spinal cord may additionally result in detrusor-sphincter dyssynergia. Lesions located below the sacral spinal cord (infrasacral lesions and those involving peripheral innervation) typically present with detrusor underactivity, often with concomitant sphincter deficiency.

In the day-to-day clinical practice of dermatology, physicians often consult patients with skin lesions caused by urogenital infections that may further involve the nervous system or are signs of disorders with neural involvement. The infection mechanism includes autoimmune reaction to peripheral nerves, roots and/or central neural pathways or spreading of the infection from cutaneous nerve endings to the corresponding dorsal root ganglia. This leads to alteration of bladder sensation, detrusor contractility, and detrusor-sphincter synergy [2]. The most common disorders that may result in neurogenic voiding dysfunctions with concomitant dermatological lesions include lumbosacral herpes zoster (varicella-zoster virus – VZV), genitourinary herpes simplex (herpes simplex virus – HSV), tabes dorsalis (Treponema pallidum), HTLV-1-associated infective dermatitis (human T cell lymphotropic virus type 1 – HTLV-1), and acquired immunodeficiency syndrome (AIDS, human immunodeficiency virus – HIV).

It has been shown that up to one-quarter of VZV-infected individuals who present with involvement of lumbosacral dermatomes may have concomitant neurogenic lower urinary tract dysfunction [3, 4]. Those patients typically complain of urine retention (due to detrusor underactivity or detrusor-sphincter dyssynergia). Retention appears concurrently with or within a few days following the onset of the rash presented in a sacral dermatomal distribution, typically affecting one or more of adjacent S2, S3, or S4 areas [5]. The lesions can be either unilateral or bilateral. Similarly, urine retention is the most common urological finding in patients with genitourinary infection of HSV [6]. Nevertheless, clinicians should keep in mind that because of the specific rash distribution of HSV type 2 in the genital area, most cases of retention are due to severe dysuria caused by direct contact of urine with the blistering urethral mucosa. True neurogenic urinary retention in anogenital HSV infection is a rare finding and occurs in less than 1% of infected patients [6]. The onset of neurogenic dysfunction occurs 1–2 weeks after the onset of a vesicular and painful rash in the anogenital region. Fortunately, neurogenic voiding dysfunctions in both HSV- and VZV-infected patients are transient and fully reversible, often within 4–8 weeks, even without dermatology-specific treatment [7, 8].

Micturition disturbances related to neurosyphilis had a high prevalence in the pre-penicillin era but improvements in medical care have made neurosyphilis a rare entity with neurological sequelae even less prevalent. However, in cases of neural involvement (particularly in immunodeficient patients) neurogenic bladder dysfunctions might occur and

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patients most often present with retention resulting from neurogenic detrusor underactivity.

Virtually all patients with the most severe skin manifestations of HTLV-1 infection (T-cell leukemia/lymphoma and infective dermatitis associated with HTLV-1) have a wide spectrum of neurogenic voiding dysfunctions ranging between incontinence and retention [9]. Urological symptoms in HTLV-1-infected individuals usually begin simultaneously with severe skin presentation. In some cases, urological symptoms can persist and progress with a tendency for urinary dysfunction to become worse with further disease progression [10].

It has been estimated that AIDS-related voiding dysfunctions affect 16–45% of patients with neurological complications of this disease [11]. As AIDS predisposes to opportunistic infections, skin conditions commonly seen in those patients (e.g., molluscum contagiosum, herpes simplex infections) often coexist with voiding problems. At the time of seroconversion, detrusor-sphincter dyssynergia is the most common finding [12, 13]. Advanced stages of AIDS may also be associated with detrusor under- or overactivity (up to 45% and 25% of patients, respectively) [11, 13]. Impaired micturition becomes more common with disease progression and neurogenic voiding dysfunction in AIDS patients portends poor prognosis [14, 15].

Although the presented conditions are rare, dermatologists need to be familiar with the discussed voiding dysfunctions, as they can substantially affect bladder function and lead to serious complications including severe renal impairment. Treatment depends on clinical presentation and urodynamic findings. Patients with retention should be catheterized as soon as possible and clean intermittent catheterization may sometimes be necessary to implement. Those with incontinence owing to detrusor overactivity usually require specialized pharmacotherapy. Therefore, strong concerted efforts of dermatologists and urologists are needed in these cases in order to change the prognosis for affected patients and to improve their quality of life.

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