# Transient Acute Urinary Retention Following Herpes Zoster Infection

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**Herpes zoster** can lead to **transient urinary retention**, particularly when sacral dermatomes are involved. Clinicians should be alert to this complication, especially in elderly patients, and perform timely assessment including bladder scanning and urodynamic evaluation.

Treatment should include **antivirals**, **pain management**, and **temporary decompression via intermittent catheterization**.

This case series of seven patients demonstrates that with appropriate management, **complete recovery of bladder function can be expected within two months.**

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**Hypothesis / aims of study**

Herpes zoster (HZ), the reactivation of latent varicella-zoster virus (VZV), typically presents with a painful vesicular rash distributed along one or two dermatomes. While its cutaneous and sensory complications are well known, acute urinary retention (AUR) is a rare and often overlooked manifestation. We report a series of seven patients who developed transient AUR shortly after herpes zoster infection, with a focus on their clinical presentation, diagnostic work-up, and functional recovery.

**Study design, materials and methods**

Seven patients—six women and one man, with a mean age of 75.1 years (range: 68–83)—presented with new-onset voiding dysfunction shortly after herpes zoster eruptions. Dermatomal involvement included the right buttock (n=2), left buttock (n=1), right-sided T10–S4 dermatomes (n=3), and bilateral S2–S4 (n=1). In each case, the onset of urinary symptoms followed the appearance of the rash by a few days to two weeks.

None of the patients had prior history of lower urinary tract dysfunction, neurological disease, or relevant comorbidities. Genital examinations were normal, and there were no signs of herpes zoster in the external genitalia. Routine blood tests and renal function were within normal limits. Urinalysis and cultures were negative for infection. All patients had post-void residual (PVR) volumes exceeding 400 mL and required indwelling catheterization. Multichannel urodynamic studies were performed once acute infection subsided.

**Interpretation of results**

Urinary retention following herpes zoster is likely related to viral neurotropism and its impact on the innervation of the lower urinary tract. One proposed mechanism involves direct viral invasion of the bladder wall through autonomic pathways, potentially leading to herpetic cystitis. Another likely explanation is viral-induced neuritis, with retrograde spread from dorsal root ganglia affecting the sacral motor neurons and disrupting the detrusor reflex arc. In some cases, involvement of spinal cord segments has been implicated, resulting in functional abnormalities of detrusor-sphincter coordination. Lastly, infection affecting the thoraco-lumbar sympathetic chain may contribute to sphincter dysfunction and urinary retention due to increased urethral outlet resistance.

In our cohort, all patients exhibited features consistent with lower motor neuron-type bladder dysfunction, most plausibly caused by sacral neuritis. The absence of central nervous system symptoms and the transient nature of the urinary retention reinforce the peripheral neurogenic origin. Importantly, full recovery occurred in all patients, supporting the notion that herpes zoster–related AUR, while alarming, is generally self-limiting when managed appropriately.