



Inflammation and infection

Herpes-zoster associated urinary retention in a 57-year-old immunocompromised male

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ABSTRACT

Herpes zoster-associated urinary retention is a rare but acknowledged phenomenon. It is usually a bladder areflexia resulting from a viral infection in the sacral dermatomes. We describe a 57-year-old immunosuppressed male patient with delayed urinary retention, following an outbreak of shingles in the setting of supratherapeutic immunosuppressive medications and poorly-controlled diabetes mellitus. The patient received a foley catheter, failed a trial of void in the acute treatment setting, completed zoster treatment with antivirals and corticosteroids, and fully recovered bladder function 6 weeks later.

1. Introduction

Varicella-zoster virus (VZV) is a DNA virus within the Herpesvirus family that clinically manifests as varicella (chickenpox) and herpes-zoster (shingles). During the initial varicella infection, VZV gains access to sensory ganglia and can remain latent for years. Herpes-zoster can then result from the reactivation of latent VZV, which leads to inflammatory lesions in the sensory root ganglia, the meninges, and occasionally the spinal cord. Patients present with a painful, unilateral vesicular eruption in a dermatomal distribution. Reactivation can be due to immunosuppression or increasing age, and most commonly occurs in the thoracolumbar dermatomes.¹ Although rare, involvement of the lumbosacral sensory ganglia can cause bladder dysfunction and urinary retention. We report an unusual case of an immunocompromised male with acute urinary retention likely due to herpes-zoster infection within the T10-S4 dermatome.

1.1. Case presentation

A 57-year-old man with a past medical history significant for hypertension, insulin-dependent diabetes mellitus, and renal transplantation in 2016 (on immunosuppressives), presented to the emergency department with 2 weeks of left lower back pain, difficulty urinating, and decreased oral intake. The patient was found to be in diabetic ketoacidosis (DKA) and urinary retention. A Foley catheter drained a residual vesical volume of 2000 ml. The patient reported no prior instrumentation for acute urinary retention, lower urinary tract

symptoms, or benign prostatic hyperplasia. Of note, supratherapeutic levels of anti-rejection medications were also found in the patient, which were then adjusted appropriately. Two weeks prior to this presentation, the patient was seen in the emergency room for left lower leg pain, was diagnosed with Herpes Zoster infection at the time, and was discharged with oral valacyclovir and gabapentin.

The physical exam was significant for left-sided T10-S4 dermatomal rash with nontender scabbing lesions (Fig. 1). Genitourinary exam was normal. Labs were significant for pH of 7.23, lactate of 5.4 with an anion gap of 31, serum glucose of 895, hyperkalemia at 6.8 and elevated levels of creatinine. The creatinine levels returned to baseline after Foley catheter placement and fluid resuscitation in the ED. Two weeks after the catheter placement, the patient failed a trial of void and required catheter reinsertion. The patient was subsequently started on tamsulosin, discharged home with the catheter, failed two trial of voids but passed on his third void trial as an outpatient 8 weeks from the initial Foley insertion.

2. Discussion

This case is an example of acute urinary retention from neuritis in T10-S4 dermatome with concurrent diabetic ketoacidosis in a patient on immunosuppressive therapeutics. Due to the immunocompromised status of this patient, the dermatomal involvement of the vesicular rash was significantly more extensive than that in immunocompetent patients, and resulted in non-sacral and sacral herpes-zoster infection. The efferent innervation of the bladder consists of the sympathetic lumbar

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Fig. 1. Herpes-zoster rash extending through sacral dermatomes.

segments 1 and 2, and the parasympathetic sacral segments 2 and 3. Infection of L1 and L2, as seen in our patient, can damage the sympathetic motor fibers of the bladder trigone and result in urinary retention.^{2,3} Zoster-associated urinary retention typically occurs four to nineteen days after the eruption of the dermatomal vesicular rash, and can persist for four to eight weeks, as evidenced by our patient's multiple failed trial of voids.² In addition to the sympathetic nerves, VZV can also infect the sacral dorsal root ganglion and posterior roots. Sacral involvement interrupts the detrusor reflex and results in bladder atonia^{4,5}; both of which are commonly evaluated with urodynamic studies. As mentioned in previous case reports, herpes zoster-associated voiding dysfunction can also be attributed to ipsilateral hemicystitis and myelitis-associated spastic bladder.⁴ Ipsilateral hemicystitis is a result of direct invasion and replication of VZV in the bladder wall, and

commonly presents with dysuria and hematuria. However, neither of which were complaints preceding the urinary findings in our patient.

Due to the patient's insulin-dependent diabetes mellitus, high serum glucose, and concurrent DKA, it is important to consider whether the urinary retention also has some component of diabetic autonomic neuropathy or underlying bladder outlet obstruction. However, our patient reported no history of diabetic neuropathy, lower urinary tract symptoms, urinary tract infections, or poor urinary stream; all of which are associated with diabetic autonomic neuropathy of the bladder.⁵

3. Conclusion

Urinary retention is one of the rare complications of reactivation of latent Varicella-zoster virus. Elderly and immunocompromised patients are particularly prone to this complication, and require prompt treatment. Typical management includes antiviral therapy, catheterization in an acute setting and symptomatic relief. The urinary retention can persist up to eight weeks, and patients usually return to full bladder function upon resolution.

Consent was obtained by all participants in the study.

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Authors CRediT

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Declaration of competing interest

None.

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