Herpes zoster-induced urologic and gastrointestinal dysfunction with residual neurogenic detrusor underactivity

Bianca Biglione, BS, Stella X. Chen, MD, Bethany Cucka, BS, and Daniela Kroshinsky, MD, MPH
Boston, Massachusetts

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INTRODUCTION

Herpes zoster, or shingles, is a common infectious disease, occurring in approximately 1 million people in the United States annually.[1] It is characterized by a painful, unilateral vesicular eruption in a restricted dermatomal distribution. Postherpetic neuralgia and ocular involvement are common complications. Rarely, other neurologic dysfunction may occur and is important to recognize given potential severe sequelae. Here, we present a case of herpes zoster complicated by urologic and gastrointestinal dysfunction.

Abbreviation used:

VZV: varicella-zoster virus

CASE REPORT

A 75-year-old man with no significant past medical history and minimal engagement with the healthcare system presented to the emergency department with 5 days of genital and gluteal vesicular lesions (Fig 1). The patient was in his usual state of health, when he developed a painful, vesicular rash involving his scrotum and buttock. He did not have hematuria, fevers, chills, or nausea/vomiting. He denied receiving the shingles vaccine. Upon further questioning by the dermatology consult team, the patient endorsed mild dysuria, difficulty voiding, and decreased bowel movements, with last bowel movement 3 days prior to presentation. He denied any previous history of these urologic symptoms.

On physical examination, the patient had multiple erythematous-to-violaceous clustered papules and vesicles extending from the left glans penis, ventral shaft, and scrotum to the left medial buttock along the S3-S4 dermatome. Lesions did not cross the midline and were mildly tender to palpation. Laboratory studies, including a complete blood count and a basic metabolic panel, were within the normal limits. Urinalysis did not indicate cystitis. Ultrasound of the scrotum demonstrated a moderate right-sided varicocele and small right epididymal cysts. A postvoid residual urine test revealed an elevated residual volume of 425 milliliters. Swabs of skin lesions were obtained and subject to polymerase chain reaction testing for herpes simplex virus and varicella-zoster virus (VZV), with a positive result for VZV. The patient was diagnosed with herpes zoster involving the left S3-S4 dermatome and complicated by acute neuropathy, bladder paralysis, and constipation.

The patient was treated with 5 days of intravenous acyclovir 10 mg/kg every 8 hours and gabapentin 300 mg nightly. He had a foley catheter placed with simultaneous initiation of tamsulosin on arrival to the hospital. For zoster-associated constipation, the patient was given polyethylene glycol, senna, and a bisacodyl suppository. Skin lesions began to resolve after 4 days. However, he failed to produce an adequate bowel movement after 5 days and was then given a soap suds enema. He failed a voiding trial and was discharged home with a foley catheter. At the 3-week follow-up with dermatology and

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Correspondence to: Daniela Kroshinsky, MD, MPH, Department of Dermatology, Massachusetts General Hospital, 50 Staniford St, Ste 200, Boston, MA 02114. E-mail: dkroshinsky@mgh.harvard.edu.
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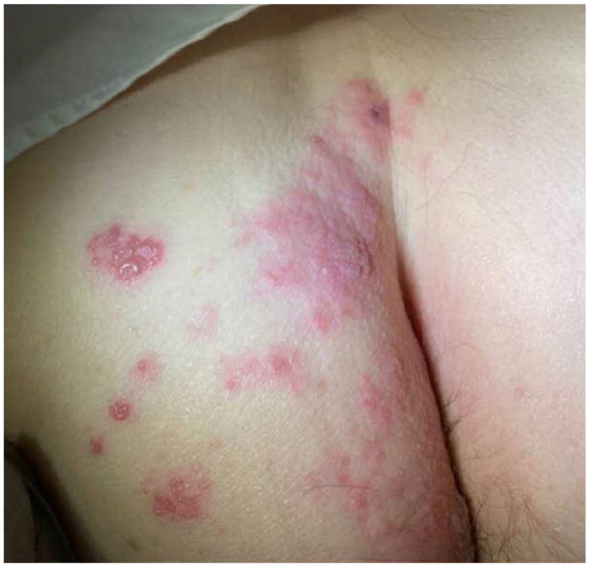

Fig 1. Vesicular rash in a patient with herpes zoster at initial presentation. Left scrotum and left buttock with clustered, erythematous-to-violaceous papules and vesicles along the left S3-S4 dermatome that did not cross the midline.

urology, the patient had resolution of lesions and constipation, but persistent urinary retention. At the 8-week follow-up, a urodynamic study confirmed the diagnosis of hypotonic neurogenic bladder. The patient was started on lifelong therapy with alfuzosin for autonomic nerve damage. He was able to be tapered off gabapentin after 15 weeks.

DISCUSSION

Reactivation of VZV is influenced by immune senescence, and most commonly affects patients > 50 years of age and the immunocompromised.[2] VZV lies dormant in neural ganglia after primary varicella infection or "chicken pox" and reactivates and tracks along nerves. Most often, VZV reactivates along sensory nerves. However, it can remain latent in cranial nerve ganglia, dorsal root ganglia, and autonomic ganglia, so herpes zoster may be observed anywhere on the body.[3] When VZV spreads from the dorsal root ganglia to the anterior horn of the spinal cord, motor manifestations such as ptosis and foot drop occur, typically 3-20 days after the cutaneous findings.[4] Although muscle atrophy may be a consequence, approximately 75% of the patients gradually recover muscle strength with the incidence of paralysis ranging from 0.5% to 31%.[4,5] When VZV involves sacral or lumbar dermatomes (most commonly S2-S4, followed by L4-L5), acute urinary retention, and more rarely, gastrointestinal complications may occur.[6] VZV reactivation leads to inflammation and subsequent demyelination of the related nerve roots. Sacral involvement may result in interruption of the detrusor reflex, leading to voiding dysfunction. Gastrointestinal dysfunction, including abdominal distension and constipation, are thought to result from anal sphincter dysfunction and are rarer than urologic associations. The prognosis for acute urinary retention and constipation secondary to herpes zoster is generally regarded as benign and selflimiting.[7] Previous reports have shown resolution of voiding abnormalities after 4-6 weeks. In contrast, our patient demonstrated continued neurogenic detrusor underactivity as evidenced by a maximum cystometric capacity of 738 mL and a postvoid residual of 154 mL. α-1a receptor-targeting therapy showed symptomatic benefit in this case.

VZV is managed with antiviral agents such as acyclovir, valacyclovir, and famciclovir. VZV-associated urinary retention can be managed with urethral catheterization in the acute setting and intermittent self-catheterization in the outpatient setting. An α-adrenergic blocker specific to α-1a in the smooth muscle of the genitourinary tract, bladder neck, and various regions of the pelvic floor may be trialed to shorten the duration. Urodynamic investigations are recommended, if symptoms fail to improve within 6-8 weeks from onset. Since antiviral therapy has no ability to prevent long-term sequelae, two doses of the recombinant zoster vaccine are recommended to prevent disease and related complications in adults' $\geq$ 50 years of age, whether or not a previous episode is reported. There is no formal recommendation on the wait period, though the consensus is to wait for the acute episode and symptoms to resolve. Fortunately, shingles vaccination among adults $\geq$ 60 years of age has increased from 6.7% in 2008 to 34.5% in 2018.[8] Our elderly patient had not received the shingles vaccine due to lack of regular medical care prior to this presentation. Although dermatologists have not played a central role in the recommendation and administration of vaccines historically, a more active role has been encouraged to improve patient education and access to herpes zoster vaccination.[9]

In conclusion, this case highlights rare and notable herpes zoster-related urologic and gastrointestinal complications. Sacral dermatome involvement should raise suspicion for these potential complications with a thorough assessment of gastrointestinal/genitourinary systems and prompt treatment to prevent long-term sequelae.

Conflicts of interest

None disclosed.

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