Case report

Disseminated herpes zoster with cauda equina symptoms

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A B S T R A C T

Herpes zoster is a common infection resulting from the reactivation of dormant varicella zoster virus (VZV) in a posterior dorsal root ganglion [1]. Its incidence is approximately 4 per 1000 person-years in the United States (US) population [2]. An increased risk is seen with increased age and immunosuppression [2]. The typical dermatomal involvement includes the thoracic region more than 50% of the time, followed by facial, cervical and lumbosacral regions, with 1% of patients presenting with disseminated disease [3]. The most common complications include: post-herpetic neuralgia (PHN), occurring in 10% of patients; ocular complications, 4% of patients; and motor neuropathies occurring in 3% of patients [3]. Here we present a case involving disseminated varicella zoster virus (dVZV) with lumbosacral plexopathy in an immunocompetent patient manifesting as cauda equina syndrome. Diagnosis was confirmed on magnetic resonance imaging (MRI) and by polymerase chain reaction (PCR). Risk factors, diagnosis, complications and treatment of dVZV are also discussed with the intention of raising awareness of primary care physicians (PCP) regarding varying presentations and therapy for herpes zoster infections.

Case report

An 85-year-old male with a past medical history of hypertension, hyperlipidemia, sciatica, prostate cancer (status post radiation therapy, on leuprolide), and pulmonary fibrosis was admitted to the hospital by his PCP after developed bladder and bowel incontinence in the setting of weakness and unsteadiness. Ten days prior to his admission he reported a mechanical fall with head trauma during a racquetball match without loss of consciousness. Computed tomography (CT) of his head and magnetic resonance imaging (MRI) of his hips were negative for acute injuries.

He subsequently developed a prodrone of sharp right leg pain and numbness, followed by a zosteriform, linear, vesiculopustular eruption initially distal to the knee and progressed to involve the medial and posterior region of his right lower extremity and groin area (Fig. 1). Additionally, he developed discrete lesions of the face, trunk, and remaining extremities. Polymerase chain reaction (PCR) test was positive for VZV, confirming herpes zoster. Valacyclovir at 1 g three times a day (TID) was started for treatment of disseminated shingles. Patient at that time was also noted to be up to date on immunizations, with the exception of live zoster vaccine.

A few days later, he developed bladder and bowel incontinence in the setting of weakness and unsteadiness. Review of systems
Fig. 1. Zosteriform rash of right lower extremity with L4-S1 dermatomal involvement.

was otherwise negative. No history of immunosuppressive pharmaceutical drug use. He was seen by his PCP, who directly admitted him at the hospital.

Physical exam was significant for a vesiculopustular rash on the patient’s face, trunk, and extremities, worse on the right lower extremity. More than 20 lesions were appreciated beyond the initial dermatome. Neurologic exam was significant for diminished right hip flexion and abduction strength, decreased pinprick and proprioception sensation on the right lower leg medially, without saddle involvement; and diminished right patellar and Achilles reflexes.

Initial labs were significant for a leukocytosis of 13,400/µL, with left shift. Imaging studies included a CT of the abdomen/pelvis that was significant for a dilated bladder and a MRI of the lumbar spine that was significant for diffuse abnormal enhancement of the cauda equina nerve root and right lumbosacral plexus.

The patient was diagnosed with disseminated herpes zoster, predominantly along the L3-S2 dermatomes with lumbosacral plexopathy, manifesting as cauda equina syndrome. He was started on intravenous (IV) acyclovir 10 mg/kg TID for 14 days, followed by valacyclovir 1000 mg for 7 days as well as gabapentin for associated neuropathic pain. He continued to have bowel incontinence and was found to have a coinfection with Clostridioides difficile. Leukocytosis and frequency of bowel movement improved after initiation of metronidazole 500 mg TID. An indwelling Foley catheter was placed for overflow incontinence.

After the lesions crust over, the patient was discharged from the hospital. He continued to have bladder and bowel incontinence; however, he had marked improvement in motor function. He was followed closely by the Departments of Urology and Neurology. At a 10-week follow-up phone interview patient reported significant improvements in ambulating, resolution of neuropathic pain, and complete resolution of prior incontinence.

Discussion

Herpes zoster is extremely common in the US population with the rate of incidence continuing to rise –from 2.5 in 1000 cases in 1993 to 7.2 in 1000 cases in 2016 [4] - despite the introduction of both chicken pox and herpes zoster vaccines [2]. A clinical condition, herpes zoster or shingles is caused by reactivation of a “varicella zoster virus” or VZV infection; as such, the three terms may be used interchangeably [2].

Approximately 1 in 3 people in the US population will develop the disease over their lifetime, and with an incidence of 4 per 1000 person-years annually. This incidence increases to 1 per 100 person-years annually among people greater than 60 years. Additionally, 1–4% of patients diagnosed with herpes zoster will be hospitalized, with approximately 96 deaths per year; and 10–18% develops post herpetic neuralgia (PHN) [2].

In May 2006, the Food and Drug Administration (FDA) approved zoster vaccine live (ZVL, Zostavax) for use in populations greater than 60 years old. In October 2017, a new genetically engineered recombinant vaccine (RZV, Shingrix) was approved by the FDA for use in healthy adults age 50 and older, and it was indicated by the Advisory Committee on Immunization Practices (ACIP) as the preferred herpes zoster vaccine over ZVL, except in cases of allergic reaction to RZV [5]. RZV showed an efficacy of 97.2% in reducing the risk of herpes zoster in adults 50 years of age or older when compared to placebo, including patients ≥70 years old, which differs from the live vaccine that showed a lower efficacy in this age group [6].

Despite the availability of a vaccine for herpes zoster, the incidence of dVZV is reported as 2% in general population and 15–30% in immunocompromised patients [7]. The diagnosis of dVZV occurs when 20 or more vesicles develop outside of the initial dermatome [8]. Our patient was admitted with more than 20 vesicles and had a positive PCR for VZV, therefore the diagnosis of dVZV was established. Once confirmed, concerns regarding possible complications should be raised [7].

PHN is the most common complication, however dVZV can also affect lungs, liver and brain [9]. Other rare and important complication that should be considered is VZV plexopathy, which is more common in the brachial than the lumbosacral plexus [10,11]. VZV plexopathy usually presents as an asymmetrical limb weakness, as in our patient, and may be mistaken for degenerative diseases causing radiculopathy. Different diagnosis for plexopathy include idiopathic, diabetic neuropathy, CNS malignancy, radiation therapy, trauma, autoimmune and inflammatory [10].

However, in a patient with a vesiculopustular dermatomal rash and plexopathy, an infection cause including VZV plexopathy must be immediately considered.

Several cases reported dVZV in immunocompetent patients, mostly aged greater than 60 years old, that were treated with IV therapy [12]. Advanced age is an important risk factor, due to the senescence of the immune system [9], causing a reduction in the T cell immunity to VZV. The treatment of choice is IV acyclovir [12,13]; minimal data has assessed oral therapy. If antiviral treatment is delayed there is an increased risk of development of disseminated VZV [9]. Therefore, when treating herpes zoster, it is imperative to initiate antiviral therapy within 72 h of onset of rash. There may be some benefit in the additional corticosteroids however there is controversial evidence [13].

In this case, the immunosenescence likely contributed to the development of dVZV. Early diagnosis and treatment with IV acyclovir in the setting of his complications including cauda equina syndrome led to decreased morbidity and mortality.

Conclusion

In this case we demonstrated the importance of physical examination and awareness of the complications of herpes zoster in the immunocompetent patient. The majority of complications are seen in immunocompromised patients however awareness and diagnosis of complications should be made early also in immunocompetent patients, especially the elderly. This will also
for early treatment and decreased morbidity and mortality. Simple cases of herpes zoster can be managed in the outpatient setting with oral antivirals and appropriate follow up for resolution and complications. Patients who do not respond to oral therapy may require hospitalization for IV therapy as seen in this case. When a patient is diagnosed with herpes zoster the variations in presentation and complications should be considered. Appropriate diagnosis and early treatment initiation led to complete resolution of the neurologic symptoms. The hallmarks of herpes zoster treatment are rest, antivirals, and pain control.

Consent
Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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Author contribution
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Declaration of Competing Interest
None.

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