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Acute Herpes Zoster Radiculopathy of the Lower Extremity With Dermatomal Rash and Lumbar Nerve Enhancement on MRI

Samay Bhushan, MD; Lisette Dominguez, DO; Ehsan Shirazi, MD; and Vivek Gupta, MD

Abstract

Herpes zoster is a frequent cause of neuralgia and dermatomal vesicular rash secondary to reactivation of latent varicella zoster virus. However, it rarely presents with acute lumbar radiculopathy and the diagnosis can be quite challenging in such cases. Nerve signal abnormalities on magnetic resonance imaging are well recognized in herpes zoster neuropathy or plexopathy affecting the extremities, although gadolinium enhancement is characteristically absent. In this article, we describe a case of acute herpes zoster lumbosacral radiculopathy with characteristic vesicular dermatomal rash and second ever reported finding of gadolinium enhancement of the lumbar nerve on magnetic resonance imaging.

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From the Department of Diagnostic Radiology (S.B., V.G.), Department of Neurology (L.D.), and Department of Emergency Medicine (E.S.), Mayo Clinic, Jacksonville, FL.

Herpes zoster, or shingles, is neurotropic reactivation of latent varicella zoster virus (VZV) in the sensory ganglion.[1] Currently, there are more than 1 million cases of herpes zoster in the United States every year.[2] Typical clinical manifestations include a vesicular erythematous rash in a dermatomal distribution and neuralgic pain.[1] Although the vesicular erythematous skin rash of shingles and associated neuralgia are well recognized and relatively easily identified in the head, neck, and trunk, herpes zoster as the underlying cause of acute lumbosacral radiculopathy is rare and can be easily overlooked despite dermatomal rash in the lower extremity. We present a case of a herpes zoster radiculopathy in a middle-aged woman presenting with acute onset sciatica, lower extremity rash, and gadolinium enhancement of the L5 nerve on magnetic resonance imaging (MRI) of the lumbar spine.

CASE REPORT

A 55-year-old woman presented to the emergency department with 1-week history of acute onset left foot paresthesia rapidly progressing to radiating left buttock and leg pain. Three days later, she developed a skin rash on her left lateral calf upon "contact with poison ivy" in her backyard. Her medical history was remarkable for a similar "poison ivy" rash preceding year treated with topical corticosteroids by her primary care provider and in situ melanoma of the contralateral great toe treated with local excision, also a year ago. Patient did not report taking any prescription medications, either currently or in the past. The clinical examination revealed 5/5 motor strength in both the upper and lower extremities. The straight leg test result was negative. A vesicular erythematous rash (Figure A) was present in the left lateral lower thigh and upper calf along the L5/S1 dermatome. Routine testing results were normal except for mild anemia with a hemoglobin level of 1.78 mmol/L and leukopenia with a white blood cell count of 3.3×10^9/L. Magnetic resonance imaging of the lumbar spine revealed abnormal enhancement of the left L5 dorsal root ganglion and descending nerve root (Figure B and C) in addition to enlargement and increased T2 signal, suggestive of acute neuritis. Minimal protrusion of the L4-5 and L5-S1 disks was also noted without nerve impingement or stenosis. No evidence of metastatic disease was noted. Given the physical examination and MRI findings, a clinical diagnosis of herpes zoster radiculopathy was made and she was discharged with oral

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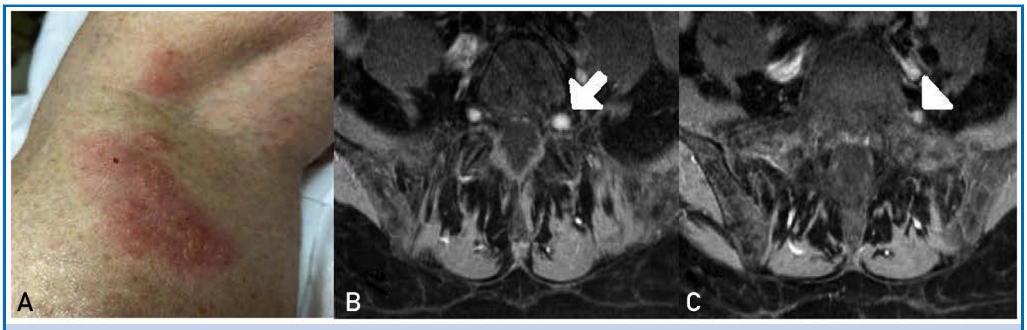

FIGURE. A, Four centimeter vesicular erythematous rash and another 1 cm erythematous rash in the L5/S1 dermatome of the left calf. B and C, Enhanced axial T1-weighted magnetic resonance imaging displaying enlargement of the left L5 dorsal root ganglion (arrow) and abnormal enhancement of the descending left L5 nerve (arrowhead).

valacyclovir and gabapentin. The polymerase chain reaction assay of the vesicular swab later confirmed VZV infection.

DISCUSSION

Shingles results from reactivation of latent VZV infection in the sensory ganglia; the incidence increases with age and immunosuppression. Upon reactivation, the retrograde virus spread results in neuritis and a vesicular erythematous rash in the corresponding dermatome.[3,4]

Diagnosis of herpes zoster radiculopathy of the lower extremity can be challenging. The lower extremity involvement is rare in shingles, and the skin rash is easily mistaken for various other forms of vesicular rash and infections. The MRI findings of lumbosacral neuritis can easily be overlooked in the absence of a high index of suspicion. Therefore, herpes zoster should always be included in the differential diagnosis of a skin rash accompanied by recent onset radiculopathy symptoms such as dermatomal paresthesia and radiating pain. A detailed history of symptom progression and thorough search for skin rash is necessary in all patients with acute lumbosacral radiculopathy, particularly when MRI is reported as negative for nerve compression. In contrast, careful evaluation of lumbosacral nerve roots and dorsal root ganglia for intrinsic abnormality must routinely be performed when a more common explanation of radiculopathy such as herniated disk or stenosis is not found. Development of dermatomal rash after the onset of lower extremity pain or paresthesia was highly suggestive of herpes zoster in our patient, though the diagnosis was initially suggested by MRI. Even in the absence of the cutaneous rash, zoster needs to be included in the differential diagnosis of any acute or subacute cervical or lumbar mononeuritis manifesting as abnormal signal on T2-weighted MRI and/or enhancement of these nerves. Contrary to a previous case series by Zubair et al[5] reporting the absence of nerve enhancement on MRI in zoster-associated plexopathy, we found abnormal enhancement of the involved nerve and dorsal root ganglion in addition to enlargement and increased T2 signal. A comprehensive literature search revealed only 2 other cases of abnormal nerve and/or lumbosacral plexus enhancement in a patient with confirmed zoster lumbosacral plexopathy.[6,7] Nerve enhancement is, therefore, likely a transient finding in the acute phase of herpes zoster neuropathy.

Although the diagnosis of herpes zoster can often be established on clinical grounds alone, detection of VZV DNA by polymerase chain reaction on swabs taken from vesicular lesions is a highly accurate test, with a sensitivity and specificity of 95% and 100%, respectively.[8] Other laboratory tests such as Tzanck test and viral DNA in blood or cerebrospinal fluid are not useful in excluding the diagnosis because of lower sensitivity.[9]

Herpes zoster reactivation can also involve the motor neurons, leading to paresis in 1% to 5% of cases known as *segmental zoster paresis*.[10] Radicular pain combined with motor weakness can mimic other spinal pathologies and make the diagnosis difficult, especially in the absence of classic cutaneous

manifestations. Commonly affected spinal nerves include C5-7 and L2-4.[11]

CONCLUSION

This case report describes the classic presentation of acute lumbar VZV radiculopathy and highlights the importance of including it in the differential diagnosis of acute lumbar radiculopathy with or without skin rash in the absence of a compressive etiology such as herniated lumbar disk.

Abbreviations and Acronyms: **MRI** = magnetic resonance imaging; **VZV** = varicella zoster virus

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Correspondence: Address to Vivek Gupta, MD, Department of Diagnostic Radiology, Mayo Clinic, 4500 San Pablo Rd S, Jacksonville, FL 32224 (Gupta.Vivek@mayo.edu; Twitter: @MayoRadiology).

ORCID

Samay Bhushan: https://orcid.org/0000-0003-2112-9869

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