Polyradiculopathy and herpes zoster

Brandon Claflin, DO, Milton Thomas, MD, and Amy J. Wilson, MD

Herpes zoster causing skin eruptions and neuropathic pain is routinely seen in the clinical setting. We present an interesting case of weakness involving more than one nerve root level secondary to shingles. The differential diagnosis of lower extremity weakness is long. While a polyradiculopathy secondary to herpes zoster infection is rare, it must be considered in a patient with dermatomal involvement. A thorough history and physical examination with appropriate diagnostic testing is essential to establish the correct diagnosis and thus the appropriate treatment. We present a retrospective review of a case of polyradiculopathy secondary to herpes zoster infection.

Table 1. Results of initial sensory and motor nerve conduction studies

Nerve	Site	Lat (ms)	Amp	Pk-Pk amp	Distance (cm)	Velocity (m/s)
Left sural (antidromic)	Calf (lateral malleolus)	4.00 (peak)	12.6 μ V	5.5	14	43.1
	Ankle	3.90	2.5 mV		8	
Left common peroneal—EDB	Fibular head	10.35	1.9 mV		29	45.0
	Ankle	3.65	9.0 mV		8	
Left tibial—AH	Knee	11.60	7.7 mV		35	44.0

Lat indicates latency; Amp, amplitude; Pk-Pk, peak to peak; EDB, extensor digitorum brevis; AH, abductor hallucis.

CASE PRESENTATION

An 80-year-old woman was admitted for complaints of altered mental status and severe left lower extremity pain. Her symptoms began several days prior to medical evaluation. The pain was progressive and was accompanied by mental status changes, including disorientation and aggression. She was diagnosed with shingles upon presentation to the emergency department.

The patient had a past medical history of anemia (secondary to a bleeding duodenal ulcer), hypertension, hyperlipidemia, total knee replacement, shoulder replacement, hysterectomy, and laminectomy. There was no previous history of lower extremity weakness. Physical examination upon presentation to the emergency department was unremarkable except for clear-cut grouped vesicular lesions and mental status changes. Some of the lesions were ulcerated on an erythematous base in a dermatomal distribution on the left lower extremity (specific dermatomes not mentioned). No lower extremity weakness was noted bilaterally upon initial examination. On the admission history and physical, there was no documentation of a sensory or motor examination. The patient was admitted to Baylor University Medical Center from the emergency department for further evaluation.

Lumbar puncture with cerebral spinal fluid analysis disclosed an elevated white cell count of $79 \times 10^3/\mu\text{L}$, with 27% monocytes and 10% plasma cells, a glucose of 60 mg/dL, and

total protein of 174 mg/dL. Cerebral spinal fluid gram stain, culture, and fungal culture were all negative. Routine laboratory results disclosed a white blood cell count of $7.9 \times 10^3/\mu\text{L}$, hemoglobin of 16.7 g/dL, hematocrit of 47.1%, platelet count of $250 \times 10^3/\mu\text{L}$, sodium of 121 mEq/L, potassium of 3.4 mEq/L, glucose of 121 mg/dL, blood urea nitrogen of 13 mg/dL, creatinine of 0.7 mg/dL, aspartate aminotransferase of 25 U/L, and alanine aminotransferase of 22 U/L. Neither herpes simplex virus DNA in cerebral spinal fluid nor varicella zoster virus DNA in blood was detected by polymerase chain reaction. In addition, tests for West Nile virus, cryptococcal antigen, syphilis, and acid-fast bacilli were negative.

An electroencephalogram (EEG) showed significant slowing and disorganization of EEG background activities that remained reactive; this was described as consistent with moderately severe global cerebral dysfunction. Two brain magnetic resonance imaging (MRI) scans were unremarkable except for notation of a previous right lacunar infarct. The thoracic spine MRI showed small disc bulges at T1–T2 and T8–T9, and the lumbar spine

From the Department of Physical Medicine and Rehabilitation, Baylor University Medical Center and Baylor Institute of Rehabilitation, Dallas, Texas.

Corresponding author: Brandon Claflin, DO, Resident, Department of Physical Medicine and Rehabilitation, Baylor University Medical Center, 3500 Gaston Avenue, Dallas, Texas 75246 (e-mail: brandocl@BaylorHealth.edu).

Table 2. Initial electromyographic results

Site	Spontaneous					Motor unit action potential			Recruitment pattern
	IA	Fib	PSW	Fasc	HF	Amp	Dur	PPP	N
Tibialis anterior	0	0	0	0	0	0	0	0	0
Gastrocnemius (medial)	0	0	0	0	0	0	0	0	0
Vastus medialis	1+	2+	2+	0	0	0	1+	1+	0
Adductor longus	0	0	0	0	0	0	0	0	0
Rectus femoris	2+	0	2+	0	0	1+	0	1+	—
Gluteus medius	0	0	0	0	0	0	0	0	0
Biceps femoris (long head)	0	0	0	0	0	0	0	0	0
Biceps femoris (short head)	0	0	0	0	0	0	0	0	0
Semimembranosus	0	0	0	0	0	0	0	0	0
L4 paraspinal	0	0	0	0	0	0	0	0	0
L2 paraspinal	0	0	0	0	0	0	0	0	0

IA indicates insertional activity; Fib, fibrillation potential; PSW, positive sharp wave; Fasc, fasciculation; HF, high frequency; Amp, amplitude; Dur, duration; PPP, polyphasic potentials.

Table 3. Electromyographic results at week 6

Site	Spontaneous					Motor unit action potential			Recruitment pattern
	IA	Fib	PSW	Fasc	HF	Amp	Dur	PPP	N
Tibialis anterior	0	0	0	0	0	0	0	0	0
Gastrocnemius (medial)	0	0	0	0	0	0	0	0	0
Rectus femoris	1+	0	2+	0	0	0	0	2+	0
Gluteus medius	2+	0	2+	0	0	0	0	2+	0
Adductor longus	1+	0	1+	0	0	0	0	1+	0
Adductor magnus	0	0	0	0	0	0	0	0	0
Biceps femoris (long head)	1+	0	1+	0	0	0	0	2+	0
L4 paraspinal	1+	0	1+	0	0	0	0	0	0
L2 paraspinal	2+	0	2+	0	0	0	0	2+	0

IA indicates insertional activity; Fib, fibrillation potential; PSW, positive sharp wave; Fasc, fasciculation; HF, high frequency; Amp, amplitude; Dur, duration; PPP, polyphasic potentials.

MRI showed evidence of previous bilateral posterior fixation of L3–L4 and L4–L5 with bilateral laminectomies. There was moderate bilateral lateral recess narrowing at L2–L3 due to degenerative changes at that level. There was no encroachment on the neural foramen or spinal canal.

The patient was believed to have varicella encephalitis and was treated with intravenous acyclovir. She initially improved but then became delirious. It was thought that the acyclovir was making her more confused. After 10 days, the acyclovir treatment was stopped, and her mental status improved to baseline.

She was discharged to home after 17 days and started on gabapentin for continued left lower extremity neuropathic pain. During the course of her acute hospitalization at Baylor University Medical Center, there was no documentation of left lower extremity weakness. The patient was seen by her primary care physician approximately 2 weeks after hospital discharge. At this time, the patient's family reported multiple near falls and trips, which they attributed to the "giving way" of the left lower leg and dragging of the left foot. The patient continued to have excruciating left lower extremity pain. The patient was referred to the outpatient physical medicine and rehabilitation clinic at the Baylor Institute for Rehabilitation.

On referral to the Baylor Institute of Rehabilitation outpatient clinic, approximately 26 days after acute hospital admission, the patient was found to have strength in the right lower leg that was normal for age; however, she was weak in her left lower leg. Left hip flexors were 2/5, knee extensors 2/5, ankle plantarflexion and dorsiflexion 3/5, and extensor hallucis longus 3/5. In the left lower extremity, sensation was described as decreased, and reflexes were 0/4. Four days later she was admitted to Baylor Institute for Rehabilitation for inpatient rehabilitation and electrodiagnostic evaluation (*Table 1*). An initial electrodiagnostic evaluation, performed 33 days after initial hospital admission for her left lower extremity, showed monoparesis consistent with a left femoral neuropathy (*Table 2*). A second electrodiagnostic study was performed at an outpatient appointment approximately 6 weeks later. At that time, the findings of abnormal spontaneous activity were more widespread, involving multiple nerve roots (*Table 3*). The left L2, L3, L4 nerve roots (femoral, obturator, and sciatic peripheral nerves) were all involved. Clinically, the knee extensor muscles were still the most affected. The writers of this case study were not present for the initial or subsequent electrodiagnostic studies.

DISCUSSION

Herpes zoster (shingles) is one of the two clinical forms of varicella zoster virus disease, the other being varicella (chickenpox). Chickenpox is a childhood infection characterized by an exanthematous vesicular rash. The latent virus, located in the dorsal root ganglion, can be reactivated later in life, most commonly in the sixth decade. There are approximately 1.2 million new cases in the USA every year (1).

Clinically, herpes zoster predominantly affects the T3–L3 dermatomes. The disease is described as a unilateral vesicular

eruption within a dermatome. Pain is the usual presenting symptom. Cutaneous symptoms then follow in 48 to 72 hours, beginning as an erythematous maculopapular rash that changes into vesicular lesions. Lesions may continue to form for 3 to 5 days, with a disease duration of 7 to 10 days. It may take 2 to 4 weeks for the skin to return to normal. Complications include changes in sensation in the affected dermatome, central nervous system involvement, and paresis (1).

While rare, there is evidence of herpes zoster causing neuritis in the brachial plexus. Fabian et al stated that "brachial plexus inflammation was a distal extension of a dorsal ganglionitis," as seen in a postmortem examination of a patient with clinical herpes zoster paresis who died as a result of a myocardial infarction (2).

Motor nerve involvement has been explained by the fact that the virus can cause a local neuritis that then affects the spinal nerve with eventual involvement of motor axons (3). In this patient, the cutaneous manifestation was known to be in the left lumbosacral plexus nerve distribution, as demonstrated by the vesicular eruption in the left lower extremity. (The specific dermatomal/peripheral nerve distribution was not mentioned on initial presentation to the emergency department.) Segmental muscle paralysis can occur in overlapping myotomes and may be seen in up to 7% of patients with vesicular lesions. This motor involvement typically occurs 3 to 20 days after the cutaneous findings, with the incidence of paralysis varying from 0.5% to 31% (4).

This patient initially demonstrated most significant clinical involvement in the femoral nerve (L2, L3, L4) distribution. She also had weakness in the tibial nerve (L5, S1) distribution, as evidenced by weakness in ankle plantarflexion, and weakness in the common peroneal nerve (L5, S1), as evidenced by weakness in dorsiflexion and evident on subsequent EMG. The length of time it takes to show changes on needle EMG varies by time after injury and length of the affected nerve. Clinically, one can appreciate weakness immediately; however, 3 to 6 weeks is needed to see abnormal spontaneous muscle membrane activity on needle EMG. These changes in needle EMG were noted in our patient. The abnormal spontaneous findings in the femoral nerve were noted first, followed by similar findings on subsequent EMG in more caudal nerves (3). Admittedly, nerve conduction studies were not performed during the second electrodiagnostic examination. In reviewing this case, nerve conduction studies may have proved beneficial to further rule out a peripheral neuropathy.

Herpes zoster virus can affect the motor and/or sensory peripheral nerves. Although this seems to be rare clinically, peripheral motor and/or sensory nerves are affected in 5% to 30% of patients with herpes zoster. Although the time frame of motor and sensory nerve involvement varies, the most common time frame is 2 weeks after the skin eruption. The myotomal distribution of the cervical and lumbosacral spine is significantly affected (26% each), as well as cranial nerves, with the facial nerve most affected (4). This patient initially exhibited a femoral neuropathy, noted both clinically and then confirmed with electrodiagnostic studies.

Electrodiagnostic findings of a peripheral neuropathy, or neuritis, include both sensory and motor nerve findings. Sensory nerve action potential amplitude is typically reduced or, depending on severity, may be absent. Compound motor action potentials are also frequently affected with the disease. One may see decreased amplitude, as well as conduction velocity (not performed on the femoral nerve in this case). Needle sampling of the involved muscles can show abnormal insertional activity, including positive sharp waves and fibrillation potentials (5). This patient's initial nerve conduction studies revealed no evidence of sensory abnormalities, but needle examination exhibited 2+ insertional activity and positive sharp waves in the rectus femoris, indicating injury to at least one nerve root comprising the femoral nerve. The second needle examination showed a polyradiculopathy involving the L2, L3, and L4 nerve root levels represented in multiple peripheral nerve innervated muscles. Without the availability of pathologic evidence, the patient's zoster infection is the most reasonable explanation for these findings.

1. Whitley RJ. Varicella-zoster virus infections. In Fauci A, Braunwald E, Kasper D, Hauser S, Longo D, Jameson J, Loscalzo J, eds. *Harrison's Principles of Internal Medicine*, 17th ed. New York: McGraw Hill, 2008:1102–1105.
2. Fabian VA, Wood B, Crowley P, Kakulas BA. Herpes zoster brachial plexus neuritis. *Clin Neuropathol* 1997;16(2):61–64.
3. Preston DC, Shapiro BE. *Electromyography and Neuromuscular Disorders*. Philadelphia: Elsevier, 2005:233–235.
4. Dumitru D. *Electrodiagnostic Medicine*. Philadelphia: Hanley & Belfus, 1995:803–804.
5. Gilden DH, Tyler KL. Herpesvirus infection and peripheral neuropathy. In Dyck PJ, Thomas PK, eds. *Peripheral Neuropathy*, vol 2, 4th ed. Philadelphia: Elsevier Saunders, 2005:2117–2123.