

Case Report

Herpes Zoster Radiculopathy Treated with Fluoroscopically-Guided Selective Nerve Root Injection

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Background: Varicella-zoster virus, a member of the herpes virus family, is a neurotrophic virus that primarily affects afferent sensory neurons. Reactivation of latent virus within the dorsal root ganglion and axoplasmic transport to epithelial nerve terminals causes the segmental cutaneous rash and neuralgic pain characteristic of herpes zoster.

Setting: Outpatient orthopedic practice.

Case Description: A 75-year-old male developed a herpetic rash followed by burning pain in the right L5 distribution. The pain was exacerbated by standing or walking. Six weeks later, the rash had improved, but the patient developed a right foot drop requiring use of a molded ankle-foot orthosis. MRI of the lumbar spine revealed mild degenerative changes without evidence of significant spinal stenosis or disc disease. Electrodiagnostic studies confirmed the diagnosis of right L5 radiculopathy.

Results: The patient had dramatic improvement of pain and weakness after undergoing a fluoroscopically guided right L5 selective nerve root block with Depo-Medrol and Lidocaine. He then began a course of physical therapy and, 6 weeks later, had only trace weakness of the ankle dorsiflexor group on the right side. The patient has continued without significant weakness or pain since the procedure and has returned to normal functioning.

Discussion: This case demonstrates apparent treatment of a relatively uncommon phenomenon, herpes zoster radiculopathy, using selective nerve root block.

Limitations: There is a limited amount of data regarding this disorder presently available regarding Herpes Zoster Radiculopathy. A second limitation would be an inability to exclude spinal pathology as an alternative etiology of this patient's condition.

Conclusion: Cases of herpes zoster-induced radiculopathy may become more frequent, as evidenced by the increasing number of cases of herpes zoster in the United States noted epidemiologically.

Key words: Herpes zoster, shingles, lumbar radiculopathy, lumbar stenosis, dermatome, electrodiagnosis, selective nerve, root injection

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A 75-year-old male with a past medical history of osteoarthritis, chronic lymphocytic leukemia, and gout was referred by Neurology to our Orthopedic clinic with a complaint of right foot drop which began 6 weeks after development of a herpetic rash over the lumbosacral spine. The patient also had pain which extended down the leg in an L5 distribution

to the dorsum of the great toe. At the time of his initial evaluation, the rash had resolved, but he complained of burning pain, numbness, and paresthesias in the lateral aspect of the right leg distal to the knee. The pain was worse with walking or standing. His pain score by visual analog scale remained a constant 8/10, despite treatment with 4 tablets daily of hydrocodone

7.5/750, 2 lidocaine patches, and 1800mg gabapentin in 3 divided doses. He also complained of milder, intermittent low back pain as well as urinary urgency and intermittent urinary incontinence. On physical examination, he was a well-developed, well-nourished male in no apparent distress with normal vital signs. There were no skin rashes. There was trace edema of the right ankle. There was no allodynia, no hyperalgesia nor hyperesthesia on examination of the legs. The peripheral pulses were intact. Straight leg raise test was negative bilaterally. Lumbar range of motion was mildly painful at end extension. Sensation was diminished to pinprick distal to the right knee. Although historically the patient stated never having prior weakness, motor examination revealed obvious foot drop with 1/5 strength of the right ankle dorsiflexors and 3/5 strength of the right extensor hallucis longus. Strength was otherwise 5/5 in the upper and lower limbs. Deep tendon reflexes were diminished in both lower limbs and the toes were downgoing bilaterally. An MRI of the lumbar spine was performed 3 months after symptom onset and showed mild spondylotic central canal narrowing at all levels except at L2-L3, where a concentric disc-osteophyte complex and facet hypertrophy caused moderate to severe, left greater than right tricompartamental stenosis. Electrodiagnostic studies were performed 6 months after symptom onset and revealed absent sural, tibial motor, and peroneal motor responses. Needle electromyography revealed small amplitude fibrillation potentials and decreased recruitment in the right peroneus longus, tibialis anterior, and medial gastrocnemius muscles. Polyphasic motor unit potentials were noted in the lumbar paraspinal, gluteus maximus, peroneus longus, tibialis anterior, and medial gastrocnemius muscles.

Treatment course

The patient was prescribed a molded ankle-foot orthosis to assist with ambulation and enrolled in a course of physical therapy. Three weeks into physical therapy the patient continued to have severe pain and weakness. Therefore we performed a right L5 transforaminal epidural injection. The VAS was 7/10 and improved to 3/10 after the first injection. A second transforaminal epidural injection was performed 2 weeks later at the same level. The patient reported excellent relief of his pain to a score of 1/10 and improvement of his weakness. On motor examination, the strength of his right ankle dorsiflexors had improved to 3/5 and the extensor hallucis longus remained 3/5.

DISCUSSION

Varicella-zoster virus, a member of the herpes virus family, is a neurotrophic virus that primarily affects afferent sensory neurons. Reactivation of latent virus within the dorsal root ganglion and axoplasmic transport to epithelial nerve terminals causes the segmental cutaneous rash and neuralgic pain characteristic of herpes zoster. Motor neuron involvement, referred to as segmental zoster motor paresis, can occur in 0.5%–31% of cases of herpes zoster and is caused by the extension of inflammation first from the dorsal root ganglion proximally into the dorsal root and posterior horn of the spinal cord, and then into the adjacent anterior horn and ventral root. Inflammation can also extend distally into the spinal nerve (1). This inflammation causes degeneration of the motor neurons of the ventral root or spinal nerve and results in weakness in a radicular distribution (1). The majority of cases of zoster motor paresis involve the cranial nerves, most frequently the facial nerve. Upper limb motor nerves may be more commonly affected than lower limb motor nerves. Motor paresis almost always follows the appearance of the typical vesicular rash by an average of 2–3 weeks and, in greater than 90% of cases, occurs in the same segmental distribution as the rash (2,3).

Urinary bladder involvement causing incontinence or retention has been reported. Interestingly, as illustrated in this case, malignancy is found 3 times more frequently in patients with zoster motor paresis than in patients with purely cutaneous zoster (4).

The typical electrodiagnostic findings in segmental herpes zoster paresis are similar to those seen with radiculopathy due to root compression (reduced recruitment, denervation potentials, and/or polyphasic motor unit potentials in the muscles supplied by the affected root, including the paraspinal muscles) with one notable exception: sensory conduction studies in the affected limb are nearly always abnormal; the majority of patients will have absent or reduced amplitude sensory nerve action potentials. Compound motor action potentials may, less frequently, also be absent or of reduced amplitude. Rarely, motor nerve conduction slowing may be seen. Isolated peripheral nerve involvement (median neuropathy proximal to the elbow, for example) has also been reported. This case illustrates a potential application of transforaminal epidural injections: the treatment of neuralgic, dermatomal pain associated with cutaneous herpes

zoster as well as zoster motor paresis (5-7). Presumably, zoster motor paresis is caused by extension of inflammation from the dorsal root ganglion to the spinal nerve or ventral root. Therefore, it can be inferred that installation, via epidural injection/selective nerve root block, of a steroid along the spinal root and within the epidural space might result in diminished pain and weakness by decreasing this inflammation (6). Shakir et al (6) reported in 2007 successful treatment of post herpetic neuralgia with C5 distribution symptomology with a cervical transforaminal epidural steroid injection.

CONCLUSION

Herpes zoster is most commonly found in middle-aged to elderly patients, and as our population ages, the incidence of segmental zoster motor paresis can be expected to increase. Practitioners should be aware that zoster motor paresis can mimic a compressive root lesion in its presentation. Electrodiagnostic studies, which typically show abnormal sensory responses in herpes motor paresis, can be helpful in distinguishing the 2 disorders. Transforaminal epidural injection or selective nerve root block may be an effective treatment for zoster motor paresis.

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