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Post-herpetic Paresis of the Lower Extremity: An Unusual Complication of Shingles

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Abstract:

Herpes zoster, or shingles, is a disease process caused by re-activation of varicella zoster virus in the dorsal root ganglia. Most commonly, it presents with burning pain and a characteristic vesicular rash. However neurologic complications may occur, most often including neuralgia, and less commonly paresis. We present a case of post-herpetic paresis in an 86 year old female.

Keywords: Herpes Zoster, Shingles, Paresis

INTRODUCTION:

Herpes zoster, or shingles, is a disease process caused by re-activation of varicella zoster virus in the dorsal root ganglia. By traveling from the dorsal root ganglia to peripheral sensory nerves, the virus causes burning pain and a characteristic vesicular rash. The rash follows a dermatomal distribution, most commonly on the trunk. Herpes zoster is frequently seen in elderly patients or those with compromised immune systems. Despite the advent of the zoster vaccine, an estimated 50% of those living to the age of 85 years will experience an episode of the disease.^{1,2} Diagnosis of the condition is made clinically upon observation of the characteristic rash, and treatment includes antiviral therapy combined with, in some cases, corticosteroids and pain medications.³ Skin lesions usually resolve over a period of two weeks although complete healing may take up to one month. Neurological complications may be observed, most commonly post-herpetic neuralgia. Less commonly, muscle paresis may be seen. This occurs in 3-5% of cases.^{1,4,5} We report a case of herpes zoster complicated by motor weakness in the leg.

CASE REPORT:

An 86-year-old Caucasian female presented to our clinic with complaints of left leg pain and weakness, necessitating the use of a cane to complete activities of daily living. Four weeks prior to presentation, she was diagnosed with shingles localized to the left leg. She was treated with a course of valacyclovir. However, she had persistent left leg pain that was not relieved with tramadol, hydrocodone, or gabapentin. Just prior to this office visit, she developed weakness in her left leg that caused her to fall and resulted in increased pain in her left hip and lower back. Aside from left leg weakness, she denied any other neurological complaints.

Her medical history was significant for hypertension, atrial fibrillation, and transient ischemic attacks. Her hypertension and atrial fibrillation were well controlled with medication, including warfarin. At the time of this visit, her international normalized ratio was therapeutic. There was no recent history of drug therapy that may cause immune suppression.

On examination, her vital signs were within normal limits. She was awake, oriented, and had a limping gait. Skin examination was significant only for hyperpigmented macular lesions on the left anterior thigh and left lower leg. Neuromuscular examination revealed decreased strength in the left leg, measuring 3/5, accompanied by decreased muscle tone. Her left patellar reflex was decreased, and hypesthesia was present over the left anterior thigh. Sensation to pinprick on the

left leg was otherwise intact. Complete cranial nerve examination was within normal limits. Examination of the upper extremities and right leg revealed no motor or sensory deficits.

To rule out injury associated with her fall, the patient underwent x-ray of the lumbar spine, sacroiliac joint and left hip. She was unable to undergo magnetic resonance imaging (MRI) due to the presence of a pacemaker, but computed tomography (CT) of her spine was completed. No fractures were seen on plain films, and CT was only significant for posterior disc protrusion at the L5-S1 and mild degenerative changes at the levels of L3-S1. Although there was no evidence of nerve impingement, the patient was started on a trial of oral steroid medication and her weakness significantly improved within one week. She showed full improvement after completing a two month course of physical therapy.

DISCUSSION:

The most common complication of herpes zoster is post-herpetic neuralgia, or pain lasting more than three months after presentation. However, as seen in our patient, complications may also include motor weakness. It is believed that motor weakness occurs when virus reactivation or inflammation involves extension to the anterior horn motor cells of the spinal cord.⁴ Of the 500,000 to 1,000,000 annual shingles cases in the United States, muscle paresis is seen in only 3-5% of cases.^{1,4,5}

On physical exam, muscle paresis most commonly presents in dermatomes corresponding to cranial nerves, although any muscle group may be involved. Muscle weakness does not always follow the same dermatomal distribution as skin eruptions associated with herpes zoster.⁶ Typically, weakness presents within two weeks of herpes zoster infection. However, it may occur simultaneously with lesions or up to one month following healing of lesions.^{6,7} Nerve conduction studies are generally within normal limits during this condition, but electromyography (EMG) may be abnormal confirming muscle denervation with fibrillation and positive sharp waves.⁷ The severity of EMG findings may predict time needed for return of normal muscle function.⁶

Motor weakness becomes apparent in patients with cutaneous zoster who are usually over the age of sixty and weak proximally.⁸ While more conclusive research is needed to determine the most beneficial treatment of this complication, a combination of corticosteroids with antiviral therapy and physical therapy was effective in our patient as well as others in the literature.⁹ Of the patients who suffered from segmental paralysis, 75% obtained relief within 1-2 years.⁸

CONCLUSION:

Because 3-5% of herpes zoster patients suffer from motor weakness, it is important that physicians be aware of its occurrence when generating a differential diagnosis in the case of paresis. After more serious causes of paresis have been ruled out, treatment with corticosteroids and possible physical rehabilitation should be considered for treatment.

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