



AN ATYPICAL CAUSE OF FOOT DROP: HERPES ZOSTER - CASE REPORT

Orthopaedics

Dr Sidhant Rana Civil Hospital Kangra, Himachal Pradesh

Dr Vipul Singh* Primary Health Centre Bagli, Himachal Pradesh. *Corresponding Author

ABSTRACT

We report on a 56-year-old female who presented with acute, atraumatic right foot drop 4 weeks after developing herpes zoster in the ipsilateral L4 and L5 dermatomes treated with acyclovir. Lumbar MRI revealed no explanatory abnormality, and the aetiology of her right foot drop was presumed to be due to an outbreak of herpes zoster. After three months of physical therapy and bracing, her right lower extremity weakness greatly improved. We present a comprehensive literature review of atraumatic foot drop etiopathogenesis, with a focus on herpes zoster as a potential causative agent.

KEYWORDS

Peroneal neuropathy, Herpes zoster, Foot drop.

INTRODUCTION

Herpes zoster, commonly known as shingles, is a viral infection that produces a painful, blistering skin rash. It is caused by reactivation of the dormant varicella zoster virus that resides in the dorsal root ganglia of the central nervous system. The lifetime risk of developing herpes zoster is 20 to 30%, with elderly and immunosuppressed patients being most affected¹. Timely initiation of antiviral medication may shorten its course, but occasionally neurological complications can persist². Although the nociceptive, sensory, and dermatomal sequelae of active herpes zoster are well known, a relatively uncommon neurological complication is motor paresis of the peroneal nerve which can present clinically as acute foot drop alongside numbness along the dorsum of the foot^{3,4}. This report describes an immunologically normal 56-year-old female who developed sudden onset of atraumatic foot drop four weeks after an outbreak of herpes zoster. We provide a comprehensive literature review of the various etiopathologies of atraumatic foot drop, with a special focus on herpes zoster as a potential causal agent that should be considered in this differential.

CASE REPORT

A 56-year-old female with a medical history of hyperlipidaemia and hypertension, presented to our orthopaedic outpatient clinic with right foot drop which had progressively developed roughly four weeks prior to her initial evaluation.

She originally presented to her primary care physician eight weeks prior with burning pain throughout her right lower back and thigh and with a vesicular rash to her proximal lateral right thigh spanning the L4/L5 dermatome. She was diagnosed with herpes zoster and prescribed a one-week course of acyclovir. One month after completion of the antiviral treatment, the patient reported progressive weakness and inability to dorsiflex her right foot and presented to our office for evaluation roughly one week later. She denied any lower extremity motor weakness prior to the herpes zoster outbreak, nor had her pre-existing low back pain ever included radiating symptoms.

On physical examination, she was a well appearing female with normal lower extremity alignment. The previously described vesicular rash to the low back and lateral thigh had resolved. She had normal passive motion but weakness with active motion of the ankle: dorsiflexion 0/5; plantarflexion 3/5; inversion 2/5; eversion 2/5. She was prescribed an ankle-foot orthosis, physical therapy, in addition to a neurology referral.

Neurologic assessment confirmed the diagnosis of right common peroneal neuropathy alongside a less significantly involved tibial nerve. A lumbar spine MRI, low back and right hip radiographs obtained did not reveal any structural abnormalities or polyradiculopathy that could explain the right foot drop. Based on the delayed neurologic symptoms that appeared weeks after the vesicular rash, it was determined that her foot drop was a likely sequela of the preceding herpes zoster infection. Neither nerve conduction studies nor EMG testing were performed.

DISCUSSION

Postherpetic motor paralysis is rare. When it does occur, however, it will generally correlate with the dermatomal distribution of the associated herpes zoster rash^{5,6}. It is estimated that such segmental

zoster paralysis occurs in 0.5-5% of all herpes zoster cases⁵. Approximately half of such cases involve the cranial nerves, with patients typically demonstrating facial and optic nerve impairment⁷. Peripheral motor involvement affecting distal muscle groups is less common and, when present, is twice as likely to affect the upper extremities as compared to the lower extremities³.

After varicella exposure, the herpes virus remains dormant in the dorsal root ganglion, a primarily sensory nerve body. The mechanism by which the herpes zoster virus subsequently invades motor neurons is unknown, but it is hypothesized that inflammation produced by the virus can extend from the dorsal root ganglion into the anterior horn (ventral root). The resultant hypervascularity in the perineural tissue, alongside potential disruption of the blood-nerve barrier, may lead to the motor deficits seen clinically^{3,8,9}.

A literature review revealed numerous causes of isolated atraumatic foot drop, including ganglion cysts of the peroneal nerve, brain lesions, spinal lesions, herniated discs, osteochondromas of the tibia or fibula, autoimmune diseases, and viral infections^{5,10-15}.

CONCLUSION

In rare cases, herpes zoster infection can lead to motor deficit in the form of an atypical foot drop-most commonly affecting elderly patients. In the absence of pre-existent trauma, the presence of foot drop along with concomitant complaints of zoster associated pain and rash in corresponding lumbar dermatomes should suggest the possibility that viral inflammation of the nervous system could account for these findings. This index of suspicion would be helpful in facilitating prompt diagnosis and appropriate non-operative treatment of this patient population.

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