

Herpes zoster in the T1 dermatome presenting with Horner's syndrome, radicular weakness, and postherpetic neuralgia

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Abstract: Herpes zoster is caused by varicella zoster virus infection involving the dorsal root ganglia of spinal nerve roots. This common problem can be associated with multiple neurological abnormalities. We present a case of Herpes zoster affecting the T1 dermatome associated with ipsilateral Horner's syndrome and radicular weakness.

Keywords: Herpes zoster, Horner's syndrome, spinal ganglia, postherpetic neuralgia, spinal nerve roots

Case presentation

An 85-year-old right-handed female presented with a 3-day history of right hand weakness and right ptosis. Her past history was significant for left internal capsule cerebrovascular infarction one year previous with no residual deficit, type II diabetes mellitus, pacemaker, migraine with aura and Herpes zoster infection diagnosed two weeks previously.

Physical examination revealed right sided ptosis and meiosis. Other cranial nerves were normal. Abductor pollicis brevis, flexor pollicis brevis, opponens pollicis, lumbricals, hypothenar muscles, adductor pollicis, flexor pollicis brevis, and palmar and dorsal interossei muscles of the right hand were weak. A vesicular eruption was noted in the right T1–T2 distribution (Figure 1).

Computed tomography revealed no acute intracranial or intrathoracic processes. Iodinated contrast administration did not alter the physical signs. Electromyography showed a low amplitude right median motor compound action potential with slow ulnar and median motor conduction velocities. The right medial ante brachial cutaneous sensory response was absent. Needle examination demonstrated fibrillation potentials and mildly large motor unit potentials with reduced recruitment in C8 to T1 innervated muscles. Pharmacologic testing for Horner's syndrome was not performed.

A diagnosis of Herpes zoster virus associated with Horner's syndrome and principally C8 to T1 radicular weakness was made.

Follow-up at 10 weeks revealed neuropathic pain affecting dermatomes C8 to T3, without sensory loss. This suggests postherpetic neuralgia.¹ Persistent weakness involved C8 and T1 innervated muscles. There was no change in her meiosis or ptosis.

Discussion

Herpes zoster (HZ) infection has an annual incidence of 3.2–4.2 per 1000 population per year in the general population,² and 5.42 per thousand person years in adults over 60 years of age.³ The most frequent neurological manifestation is sensory nerve involvement resulting in a vesicular eruption of the skin and mucous membranes in the relevant dermatome. We present an unusual presentation of this HZ with Horner's syndrome and postherpetic neuralgia.

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Figure 1 Photograph of patient showing Herpes zoster rash in T1-T2 distribution with associated right sided ptosis and meiosis.

Varicella zoster infection is pathologically associated with inflammation of the dorsal root ganglion of the involved segment of the spinal cord⁴ and has been shown to be present in the anterior horns causing a unilateral segmental poliomyelitis.⁵ The majority of injury occurs in the posterior root and its ganglion, contrasting with poliovirus,⁵ which occurs in the anterior root ganglion. This can result in pain, paralysis and urinary retention.² Radicular paralysis always follows the dermatome of sensory involvement.⁶

The occurrence of Horner's syndrome is presumably due to involvement of the sympathetic pupillodilator pathway. Varicella zoster viral reactivation and inflammation at the C8-T1 region of the spinal cord may interrupt the exiting sympathetic fibers.

Zoster paralysis, is associated with a good prognosis for recovery,⁷ however the prognosis of ptosis associated with Herpes zoster reactivation is less clear.

Disclosure

The authors report no conflicts of interest in this work.

References

1. Rowbotham MC, Petersen KL. Zoster-associated pain and neural dysfunction. *Pain*. 2001;93:1–5.
2. Harpaz R, Ortega-Sanchez IR, Seward JF. Prevention of herpes zoster: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Recomm Rep*. 2008;57(RR-5):1–30.
3. Oxman MN, Levin MJ, Johnson GR, et al. A vaccine to prevent Herpes zoster and postherpetic neuralgia in older adults. *N Engl J Med*. 2005;352:2271–84.
4. Tilki HE, Mutluer N, Selcuki D, et al. Zoster paresis. *Electromyogr Clin Neurophysiol*. 2003;43:231–4.
5. Denny-Brown D, Fitzgerald PJ, Adams DR. Pathological features of herpes zoster. *Arch Neurol Psychiatry*. 1944;51:216–31.
6. Rosenfeld T, Price MA. Paralysis in herpes zoster. *Aust N Z J Med*. 1985;15:712–6.
7. Gabriel CM, Gale AN, Rossor MN. Zoster paresis with Horner's syndrome. *J Neurol*. 2003;250:1492–3.