Postherpetic Neuralgia After Herpes Zoster Ophthalmicus

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CLINICAL HISTORY

A 77-year-old woman presented to the emergency department with severe right eye pain, periorbital swelling and blurry vision. Five days prior to presentation, she developed itching around her right eye followed by a vesicular rash on the right side of her face. She was prescribed antibiotics but had progressive worsening of her symptoms. The patient reported a history of chickenpox as a child and shingles vaccination as an adult.

On presentation she was febrile with physical examination notable for a vesicular rash with honey-colored crusting over the right eye, forehead and upper nose. Skin swab from a lesion on her forehead was positive for varicella zoster virus (VZV). CT (**Figure 1**) and MR imaging of the brain and orbits revealed diffuse right periorbital and facial soft tissue edema with a periorbital rim-enhancing fluid collection.

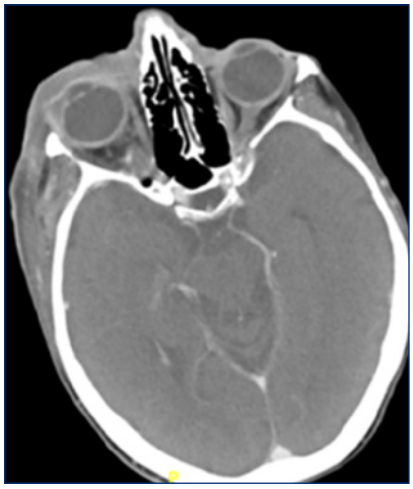


Figure 1. Herpes Ophthalmicus. Axial contrast-enhanced CT demonstrates right periorbital and lateral facial soft tissue edema.

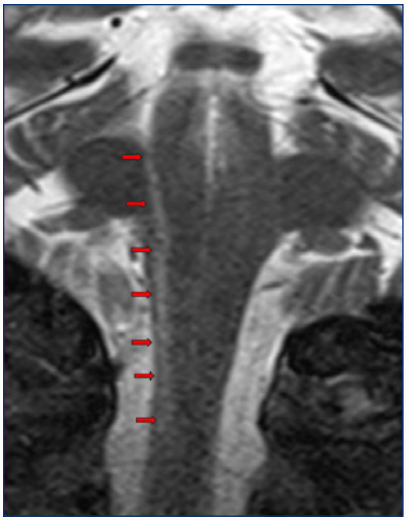
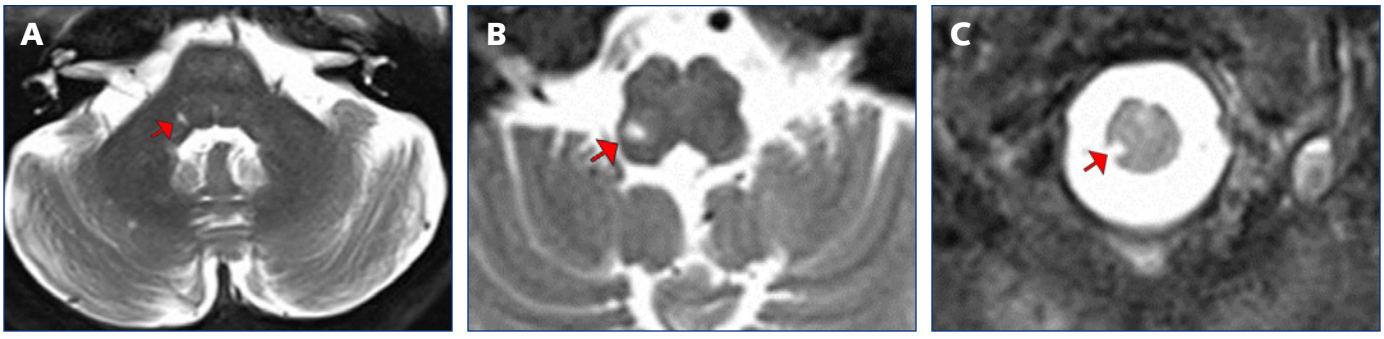


Figure 3. Post-herpetic neuralgia manifesting as hyperintense signal on coronal T2-weighted MRI in the right trigeminal spinal nucleus. Coronal T2-weighted MR magnified inset shows linear hyperintense signal corresponding to trigeminal spinal nucleus (red arrow) from pons to upper cervical cord.

The patient was admitted and initially treated with intravenous acyclovir as well as ampicillin/sulbactam, vancomycin, and linezolid. On the day of discharge, repeat MRI demonstrated linear T2-hyperintense signal within the right dorsolateral medulla extending into the cervical spinal cord (**Figures 2,3**). The following week she was seen in clinic, where she reported intermittent pain around her eye. Residual crusting was observed around her eye on physical examination. She completed her final course of antibiotics two weeks after discharge with a plan for continued follow-up with ophthalmology.

Figure 2. Post-herpetic neuralgia manifesting as hyperintense signal on serial transverse axial T2-weighted MR images in the right trigeminal spinal nucleus. [**A**] Transverse axial MR imaging from mid-pons, [**B**] through medulla, and [**C**] upper cervical cord show focal hyperintense signal along course of right trigeminal spinal nucleus (red arrow).



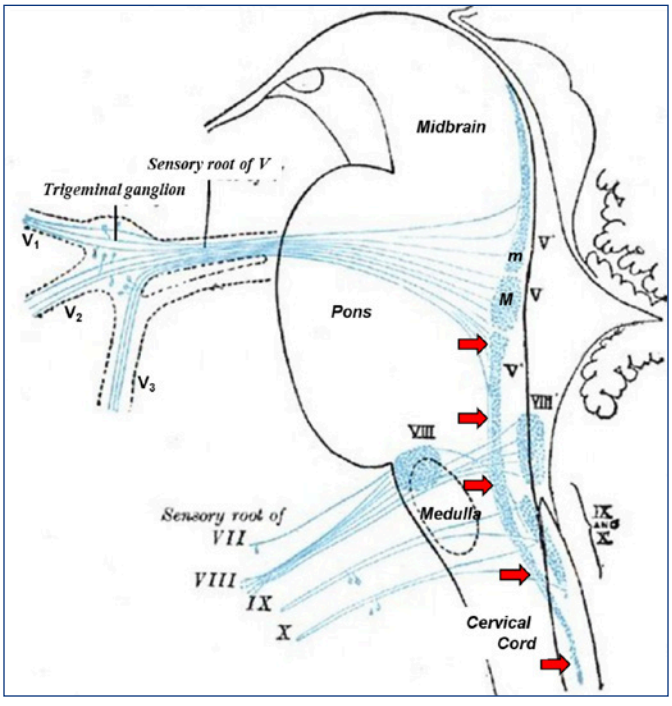
DISCUSSION

Herpes zoster is a viral infection caused by reactivation of the varicella-zoster virus (VZV). The virus remains latent in the dorsal root ganglion after primary varicella infection until reactivation later in life. Herpes zoster can damage peripheral nerves and result in a neuropathic pain condition called postherpetic neuralgia (PHN). Wilcox et al studied the association of chronic neuropathic pain with anatomic changes. Compared with pain-free controls, patients with chronic trigeminal neuropathy had decreased volume of the spinal trigeminal nucleus as well as decreased mean diffusivity and increased fractional anisotropy on diffusion tensor imaging.[1] Haanpaa et al found an association between MRI abnormalities and the subsequent development of PHN: 56% of patients who had positive MRI findings of herpes zoster reported pain three months after infection, whereas all patients who had a normal initial MRI were pain-free.[2] MRI may therefore be a useful predictor of which patients will suffer long-term pain.

Herpes zoster ophthalmicus specifically refers to infection in the distribution of the ophthalmic branch of the trigeminal nerve (V_1), which provides sensory information from the upper face and scalp including the eye. Patients have involvement of the spinal nucleus of the trigeminal nerve, which receives sensory information from the ipsilateral face with input from the trigeminal, facial, glossopharyngeal and vagus nerves. The trigeminal nucleus extends from the dorsolateral pons and medulla to the level of the C3 vertebral body (**Figure 4**). This corresponds to the site of abnormal signal intensity that we report in this patient. Case reports have suggested the possibility of trans-axonal migration of VZV, including spread of the virus to the spinal trigeminal nucleus from the trigeminal ganglion in Meckel's cave,[3] geniculate ganglion,[4] and glossopharyngeal ganglion.[5]

While there have been case reports of patients with herpes zoster ophthalmicus with abnormalities of the spinal trigeminal nucleus and tract on MRI,[6-8] these findings are thought to be rare. Our case of postherpetic neuralgia after herpes zoster ophthalmicus with corresponding increased T2/FLAIR signal intensity in the ipsilateral trigeminal spinal nucleus is consistent with trans-axonal migration of VZV from the ophthalmic nerve (V_1) to the spinal nucleus of the trigeminal nerve.

Figure 4. Spinal Nucleus of the Trigeminal Nerve. The trigeminal nuclei are depicted on this parasagittal figure of the brainstem: mesencephalic (m), main (M) and spinal. Spinal trigeminal nucleus (red arrow) receives information about deep and crude touch, pain and temperature from the ipsilateral face and extends from pons through the medulla and to level of upper cervical cord, approximately C2 to C3, where it becomes continuous with dorsal horn of cervical cord. V, trigeminal cranial nerve; V1, ophthalmic nerve; V2, maxillary nerve; V3, mandibular nerve.



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