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Zoster Paresis: Asymptomatic MRI lesions far beyond the site of rash and focal weakness

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Abstract

We describe a patient with zoster paresis and an MRI that revealed extensive spinal cord lesions from the upper cervical to the lower thoracic spinal cord. Importantly, the patient reported considerable spontaneous improvement in strength 2–3 weeks after zoster. This report reveals a previously undescribed remarkable preponderance of MRI lesions far beyond the site of zoster rash and focal lower motor neuron weakness.

Keywords

zoster; paresis; MRI

1. Case report

A 65-year-old woman developed left C4–7 distribution zoster with arm weakness and was treated with valacyclovir, 1 gram 3 times daily for 3 days. When zoster developed, she was not taking immunosuppressive medications. One month later, EMG and NCV abnormalities were consistent with severe left C5 radiculopathy, and MRI revealed increased signal in nerve roots of the left brachial plexus (Fig. 1, A) as well as in the upper cervical cord that extended to the lower thoracic spinal cord and which enhanced with gadolinium (Fig. 1, B–E). Eight years earlier, she had total hysterectomy for uterine sarcoma. One year earlier, right thigh leiomyosarcoma was treated by resection and local irradiation. At neurological consultation one month after zoster, pain had disappeared, and the patient reported that her strength was almost back to normal. A healing zoster rash was apparent on the posterior aspect of the left arm and forearm, extending to the index finger. Despite extensive MRI abnormalities described above, zoster paresis was limited to minimal weakness of abduction, external rotation and supination in the left arm and a trace left biceps reflex. CSF contained 61 cells/mm³, all mononuclear; CSF protein was 121 mg/dL and normal glucose. Cytology

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Conflict of interest statement

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was normal. A family physician prescribed intravenous methylprednisolone, 1 gram divided over 5 days followed by prednisone taper. Three months after acute zoster paresis, there was minimal weakness of left arm external rotation, forearm flexion and supination. Repeat imaging of the brachial plexus, cervical and thoracic spinal cord revealed near-complete resolution of all abnormalities (Fig. 1, F-J).

2. Discussion

We describe a case of mild cervical zoster paresis in an otherwise healthy woman in whom MRI remarkably revealed increased signal in the brachial plexus, along with unexpected enhancing lesions in the upper cervical spinal cord that extended to the lower thoracic spinal cord, all in the absence of any symptoms or signs of myelopathy. The features of zoster paresis could have been due to viral involvement of the brachial plexus or the cervical spinal cord or both. The extent of disease indicated on MRI was particularly impressive since the patient was immunocompetent. Normally, MRI scanning is not performed in patients with uncomplicated zoster. A PubMed search of patients with zoster paresis (lower motor neuron weakness corresponding to the dermatome where rash was present) revealed that MRI scans are normal [1-4] or that spinal cord lesions are present 1-2 levels adjacent to the area of segmental weakness [5-7]. Thus, to our knowledge, this is the first report of widespread subclinical imaging abnormalities far beyond the site of zoster and focal zoster paresis. Although there is no apparent reason why enhancing spinal cord lesions were asymptomatic, enhancement only indicates capillary permeability and not necessarily neuronal injury. Likewise, increase T2 signal can reflect edema from the same process and also be asymptomatic. Since zoster involves blood vessels along Virchow-Robin spaces, enhancement without neuronal injury is quite conceivable. Importantly, such asymptomatic lesions in our patient's spinal cord are comparable to extensive asymptomatic lesions commonly seen on brain and spinal cord MRI in patients with clinically isolated syndromes and clinically definite multiple sclerosis.

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Fig. 1.

Increased T2 signal is seen in nerve roots of the left brachial plexus (A, arrows). Sagittal STIR images of the cervical spinal cord show increased signal (B, arrows) with avid post-contrast enhancement on the surface and within the cervical spinal cord (C, arrows). Contrast enhancement was confluent and bilateral across the anterior and lateral cord from C2 to C5-6. Additional focal lesions were seen in the right posterolateral cord at C4, bilaterally in the dorsal columns at C4-5, midline central to posterior at C6 and lateral and posterolateral at C7-T1. Similarly, sagittal STIR images show extensive increased signal throughout the thoracic spinal cord (D, arrows) and post-contrast T1-weighted fat saturated image shows multiple punctate foci of enhancement both on the periphery and within the thoracic spinal cord (E, arrows). Two months later, follow-up images show near complete resolution of increased signal in the brachial plexus (F, arrows) and in the cervical spinal cord before (G, arrow) and after (H, arrow) contrast enhancement. Similarly, there is near complete resolution of increased T2 signal in the thoracic spinal cord (I, arrow) with only a single residual lesion after contrast enhancement (J, arrow).