
Herpes Zoster Myelitis: MR Appearance

David P. Friedman

Summary: The author describes a 71-year-old woman in whom cutaneous cervical herpes zoster was complicated by the development of cervical myelitis. T2-weighted MR showed two focal areas of hyperintensity in the cervical cord and suggested a slight enlargement at C2-C3 and C7.

Index terms: Spinal cord, magnetic resonance; Herpes zoster

Myelitis is a relatively rare complication of herpes zoster infection (1-8). Prior to the advent of magnetic resonance (MR) imaging, the diagnosis could not be confirmed by radiographic evaluation; rather, imaging studies were obtained to exclude other structural lesions. This report describes a case of cutaneous cervical herpes zoster complicated by the development of cervical myelitis. The MR scan revealed two intramedullary lesions in the cervical cord; the larger lesion was situated at a level corresponding to the dermatomal distribution of the patient's rash. The importance of correlating the clinical and radiographic findings in this case is emphasized.

Case Report

A previously healthy 71-year-old woman developed a vesicular rash in the right C2-C3 dermatomal distribution. A diagnosis of herpes zoster was made. She developed mild right facial weakness 2 weeks later, and progressive bilateral lower extremity weakness 1 month thereafter. Several episodes of falling prompted admission. Examination again demonstrated a vesicular rash, with lesions in various stages of evolution, in the right C2-C3 distribution. Neurologic evaluation revealed a spastic quadriparesis; strength in the lower extremities was 0/5 and in the upper extremities was 3-4/5. Dorsal column and spinothalamic sensory modalities were diminished. There was also a right peripheral seventh nerve palsy.

The cerebrospinal fluid showed 55 white blood cells (98% lymphocytes) and 35 red blood cells; the protein and glucose were normal. Oligoclonal bands were absent. Cerebrospinal fluid viral culture was negative. Lyme titers (IgG but not IgM) were weakly positive, suggesting exposure in

the past. A noncontrast cranial MR scan showed changes of ischemic white matter disease. T2-weighted MR scans of the cervical spine demonstrated focal areas of hyperintensity at C2-C3 and C7 with possible swelling; the lesion at C2-C3 was eccentric to the right (Figs. 1A-1C). T1-weighted images obtained before and after gadolinium administration revealed vague hypointensity at these levels with little or no enhancement (Figs. 1D-1F). A diagnosis of herpes zoster myelitis was made based upon the clinical, laboratory, and radiographic findings. The patient was treated with corticosteroids and a 10-day course of intravenous vidarabine with resultant significant improvement. Upon discharge 5 weeks after admission, strength was 4-5/5 in the upper extremities and 4/5 in the lower extremities; sensory modalities were virtually normal.

Discussion

Myelitis is a distinctly unusual manifestation of herpes zoster infection. In most of the cases reported, spinal cord dysfunction began after the appearance of the vesicular rash (3-6). However, a variety of zoster-associated neurologic diseases have been described in the absence of skin lesions (7, 8). The appearance of the rash and the subsequent onset of neurologic symptoms days to weeks later is helpful in making the diagnosis. As in this case, the presence of signal abnormality in the spinal cord at a level that corresponds to the dermatomal distribution of the vesicular rash is even more suggestive. Most patients with zoster myelitis develop a sensory deficit up to a thoracic cord level (3), which is consistent with the high frequency of thoracic dermatomal involvement. However, there may be more than one spinal cord segment affected, and the level of dysfunction may ascend during the course of the disease. Myelitis frequently occurs in association with encephalitis; in the current case, the only other manifestation of zoster was a right peripheral seventh nerve palsy. The full spectrum of clinical outcomes in zoster myelitis has been reported,

Received October 28, 1991; accepted and revision requested December 12; revision received January 15.

Department of Radiology, Jefferson Medical College and Thomas Jefferson University Hospital, 10th and Sansom Street, Room #1069-Main Building, Philadelphia, PA 19107.

AJNR 13:1404-1406, Sep/Oct 1992 0195-6108/92/1305-1404 © American Society of Neuroradiology

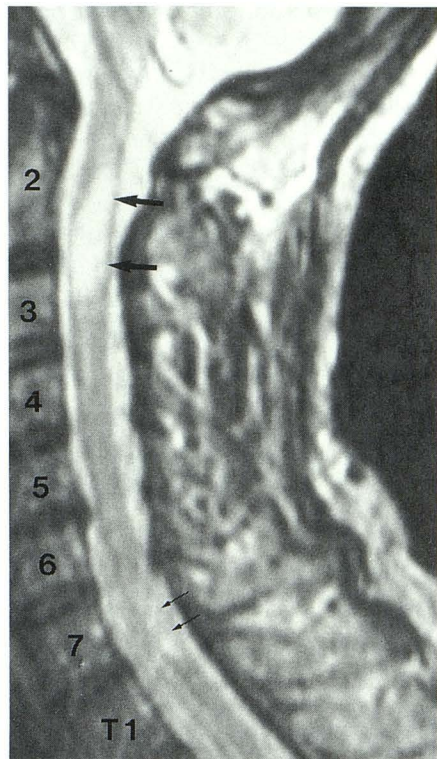


Fig. 1. A, T2-weighted (2000/80) sagittal scan reveals focal areas of hyperintensity and possible enlargement at C2-C3 and C7. The lesion at C2-3 (*large arrows*) is significantly larger than the one at C7 (*small arrows*).

B, T2-weighted (2000/80) axial image at the level of C2-C3 shows that the signal abnormality is eccentric to the right (*arrow*) and occupies the majority of the cross-sectional area of the cord. The patient's herpetic rash was in the right C2-C3 dermatomal distribution.

C, T2-weighted (2000/80) axial scan at the level of C7 confirms the presence of a large intramedullary lesion (*arrow*).

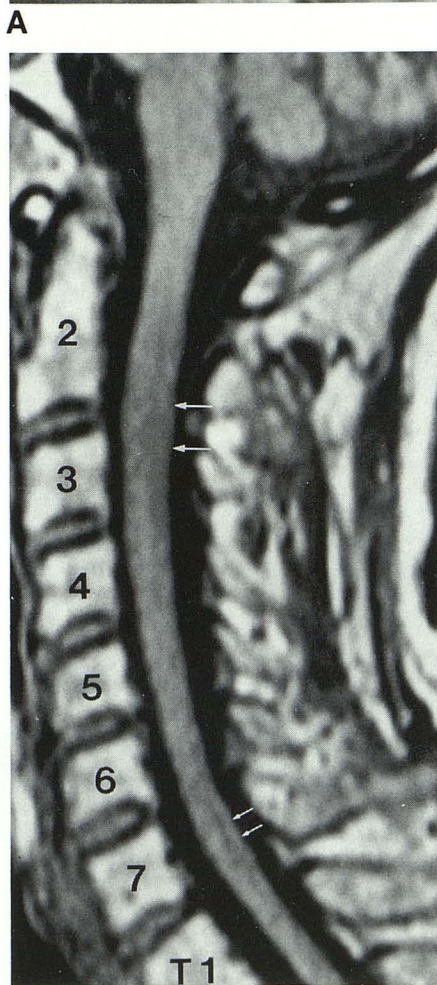
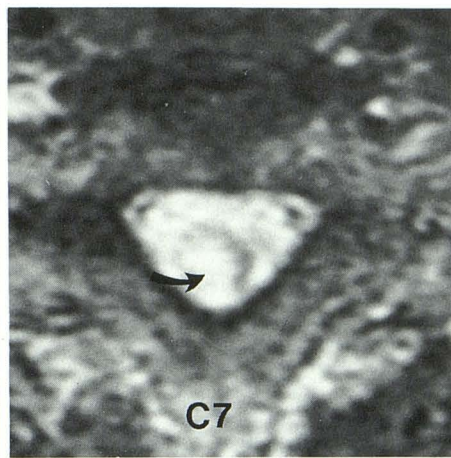
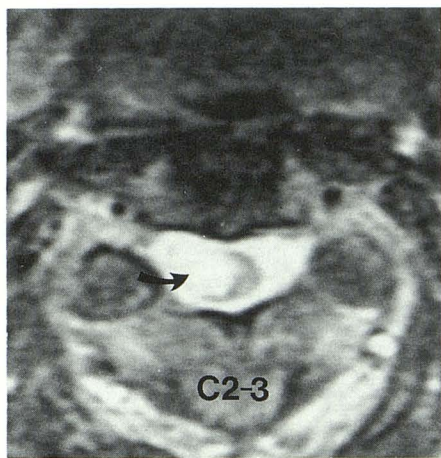
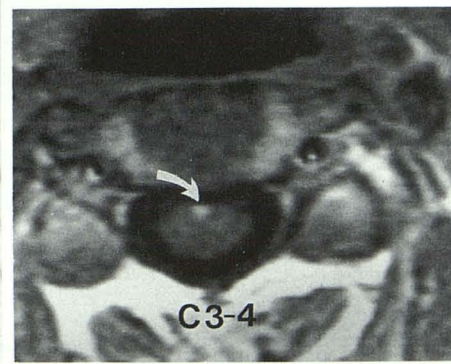


Fig. 1. —Continued. D, T1-weighted (500/11) sagittal image demonstrates vague hypointensity and possible cord enlargement at C2-C3 (*large arrows*) and C7 (*small arrows*).

E, Postcontrast T1-weighted (500/11) sagittal image shows little or no enhancement at the affected levels (*thin arrows*). Enhancement of the ventral aspect of the cord at C3-C4 (*thick arrow*) is of uncertain significance.

F, Postcontrast T1-weighted (517/12) axial scan at the level of C3-C4 confirms the presence of enhancement (*arrow*).



D

E

F

ranging from spontaneous recovery to relentless ascending progression and death (2–6).

The spinal fluid may show elevated protein, hypoglycorrhacia, and a lymphocytic pleocytosis (1, 3). The virus is rarely cultured from spinal fluid, although antiviral antibody titers may be elevated (2, 3). The pathologic lesion is necrosis of cord tissue. Hemorrhage, microscopic areas of perivascular inflammation, and disruption of normal neural architecture are seen (2–4). Although the etiology of zoster myelitis is not fully established, an allergic response, autoimmune vasculitis, demyelination, and direct viral invasion have been postulated (1–6). In this case, two mechanisms may be implicated: direct viral invasion at C2–C3 (the level of the rash), as well as an autoimmune, vasculitic, or demyelinating process resulting in additional lesions of C7 and the right facial nerve.

MR is the first imaging technique capable of directly visualizing the spinal cord. The scans in this patient demonstrated two focal areas of hyperintensity in the cervical cord on T2-weighted images; there was also possible slight enlargement at these levels (Figs. 1A–1C). There were no macroscopic foci of hemorrhage detected, and the lesions showed little or no enhancement (Figs. 1E and 1F). Additional cases would be required to determine if more prominent enhancement or hemorrhage can occur in this setting. Moreover,

a normal MR scan does not exclude the diagnosis of herpes myelitis (3). Unfortunately, the radiographic findings are nonspecific. The differential diagnosis of multiple foci of intramedullary high signal in a cord of normal caliber includes multiple sclerosis, lyme disease, infarction, contusion, myelopathy, and other causes of demyelination. Neoplasm generally causes more cord enlargement and abnormal enhancement. Clearly, correlation with the clinical findings, especially the temporal sequence of events and the distribution of the rash, was essential in reaching the correct diagnosis.

References

1. Barnes DW, Whitley RJ. CNS disease associated with varicella zoster virus and herpes simplex virus infection. *Neurol Clin* 1986;4:265–283
2. McCormick WF, Rodnitzky RL, Schochet SS, McKee AP. Varicella-zoster encephalomyelitis. *Arch Neurol* 1969;21:559–570
3. Baethge BA, King JW, Husain F, Embree LJ. Case report: herpes zoster myelitis occurring during treatment for systemic lupus erythematosus. *Am J Med Sci* 1989;298:264–266
4. Hogan EL, Krigman MR. Herpes zoster myelitis. *Arch Neurol* 1973;29:309–313
5. Muder RR, Lumish RM, Corsello GR. Myelopathy after herpes zoster. *Arch Neurol* 1983;40:445–446
6. Gardner-Thorpe C, Foster JB, Borwich DD. Unusual manifestations of herpes zoster. *J Neurol Sci* 1976;28:427–447
7. Mayo DR, Booss J. Varicella zoster-associated neurologic disease without skin lesions. *Arch Neurol* 1989;46:313–315
8. Heller HM, Carnevale NT, Steigbigel RT. Varicella zoster virus transverse myelitis without cutaneous rash. *Am J Med* 1990;88:550–551