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MR of the Spinal Cord in a Patient with Herpes Zoster

Michael B. Esposito,[1] John A. Arrington,[1] F. Reed Murtaugh,[1] John M. Coleman,[1] and Stephen M. Sergay[1]

Summary: Increased signal intensity on initial magnetic resonance images of the spinal cord in a patient with herpes zoster demonstrated that this virus caused inflammation of the cervical spinal cord. This pathology corresponded well with neurologic deficits seen clinically, but the extent of the neurologic deficits ultimately could not be determined by magnetic resonance of the spinal cord alone because the nerve roots were also affected.

Index terms: Spinal cord, magnetic resonance; Herpes zoster

Magnetic resonance (MR) of the spinal cord in a patient with herpes zoster revealed cervical cord pathology that corresponded with neurologic deficits seen clinically.

Case Report

A 65-year-old nonimmunocompromised man presented with pain and weakness in his left arm that was followed later by a typical herpes zoster rash. The rash resolved but impairment of movement of the limb worsened over the ensuing few weeks. Examination revealed generalized atrophy of the left upper extremity with motor and sensory impairment as well as decreased deep tendon reflexes. Electromyogram revealed denervation of the left deltoid and biceps muscle indicating involvement of the C5–C6 roots. Spinal tap was deferred because the patient was improving at the time of presentation.

MR imaging of the cervical spine showed enlargement of the cervical cord with noncontiguous areas of intramedullary increased signal on the gradient-echo and gadolinium-enhanced images from C2 level through C4–C5 in the posterior-lateral and dorsal aspect of the cord (Fig. 1). Etiology was uncertain, but because of the clinical presentation and diffuse neurologic deficits it was believed to represent a posttherapeutic myelopathy.

Fifteen months later, some weakness and sensory deficits persisted. Report electromyogram revealed only minor slowing of median nerve conduction velocity. Two years postinfection, the patient was reexamined because of complaints of worsening function in his left arm.

MR imaging at this time revealed the cervical cord to have returned to normal size, but there continued to be noncontiguous areas of intramedullary increased signal from C3–C4 level to C5–C6 primarily on the left, which was felt to represent a glial scar secondary to the viral infectious process. A clinical exam showed that the patient's neurologic function had not worsened.

Discussion

The clinical presentation in our patient is typical of herpes zoster and included the initial symptoms of pain in the affected dermatomal segments, followed by a characteristic rash and occasional motor and sensory neurologic deficits (1–4). Zoster infection may cause paralysis by extension of viral infection from the dorsal root ganglion to the anterior horn cells of the involved segment (5). The spinal cord pathology is secondary to inflammation, mononuclear cell infiltration, perivascular demylination, or localized small vessel vasculitis (1, 5, 6). The vasculitis is histologically similar to granulomatous angiitis (6).

MR imaging has been used to detect herpetic lesions in the cerebral white matter in children and the cranial nerves of patients with Ramsay Hunt syndrome. In this case, the increased signal intensity seen on initial gradient-echo and gadolinium-enhanced images demonstrated that the herpes zoster virus caused inflammation of the cervical spinal cord (7).

Clinically, the patient had features of both a radiculopathy and a myelopathy that combined to cause the global upper extremity weakness seen. Initial nerve conduction studies revealed damage to the C5 and C6 nerve fibers, correlating well with the location of the areas of enhancement in the initial MR images (Fig. 1A). Follow-up MR revealed glial scars in the same cord locations with resolution of the cord and root inflammation. The small residual deficits in the patient's intrinsic

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[1] Department of Radiology, University of South Florida College of Medicine, 12901 N. 30th Street, Box 17, Tampa, FL 33612. Address reprint requests to F. Reed Murtaugh, MD.

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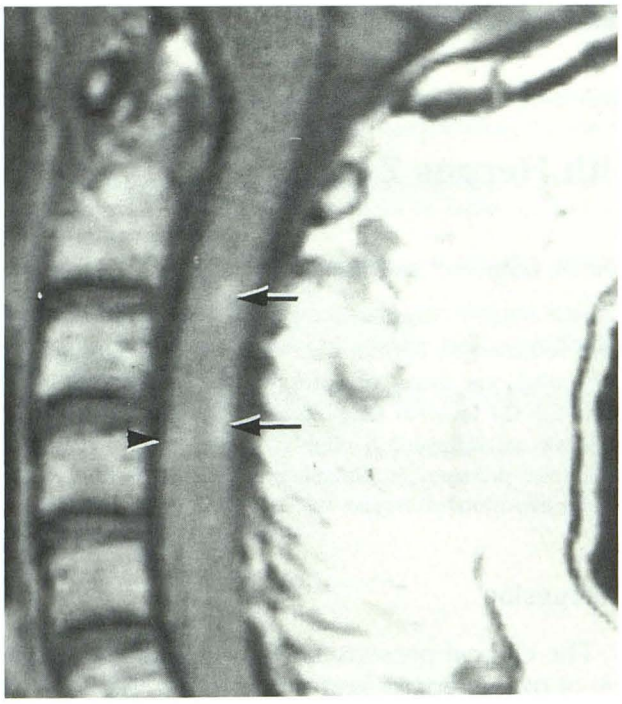

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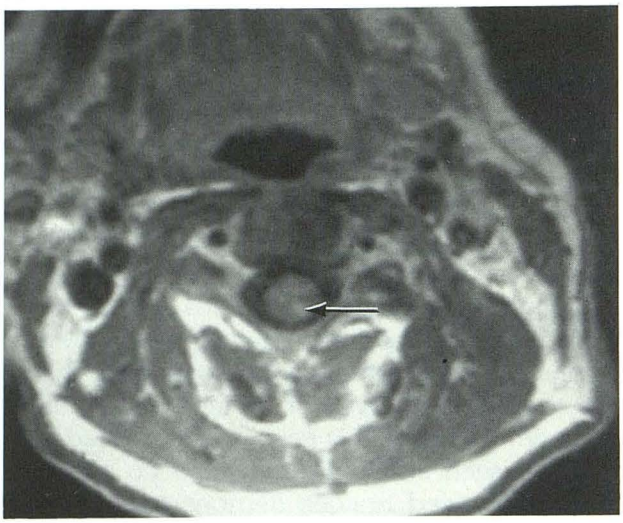

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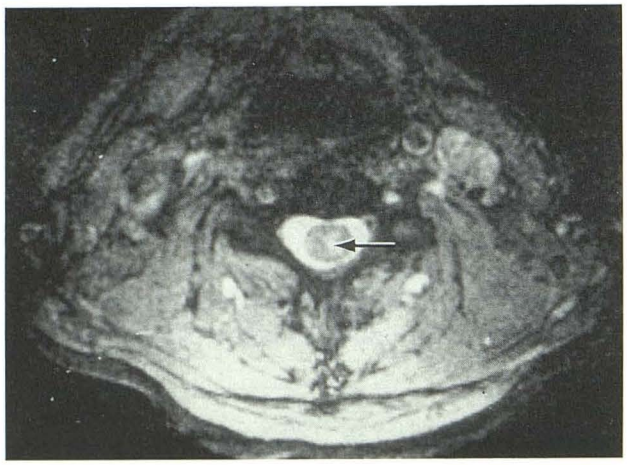

C

Fig. 1. *A*, Sagittal gadolinium-enhanced, T1-weighted spin-echo image, 600/20/4 (TR/TE/excitations) demonstrates enhancement in the cervical cord due to inflammation caused by the herpes zoster infection (*arrows*). The *arrowhead* indicates the involvement of the anterior portion of the cord.

B, Axial section through the cervical cord seen in *A* at the C3–C4 disk space. The *arrow* points to the area of enhancement from the dorsal root entry zone to the dorsal lateral aspect of the cord on the left.

C, Axial gradient-echo image (T2*-weighted image) (75/15/4) at the C5 level. The *arrow* indicates the region of diffuse increased intramedullary signal intensity on the left caused by the spread of a herpes zoster infection from the dorsal root ganglion into the spinal cord itself.

hand muscles remained, for the motor neurons to these muscles are well-localized in the spinal cord and were damaged by the infection (3). Although the weakness could be attributed to nerve root damage, the MR findings in the cervical cord suggest spinal cord involvement. The relatively minor residual functional loss in the remainder of the upper extremity can be attributed to the diffuse pattern of innervation to the muscles in this area.

The sparing of the contralateral upper extremity and the lower extremities is common in zoster-induced paresis (2). MR images revealed marked swelling and diffuse focal lesions that were not clinically significant. There areas can be seen posteriorly to the C4 vertebral body in Figure 1A. The extent of the neurologic deficits could not be determined by examining the MR images of the spinal cord alone because the nerve roots were also affected.

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