Computed Tomography of Spinal Cord Necrosis from Multiple Sclerosis

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Despite frequent involvement of the spinal cord by multiple sclerosis clinically and pathologically, to our knowledge the literature concerning computed tomography (CT) of these lesions has been limited to a single case report [1]. Our case demonstrates the sequential CT imaging of a necrotic multiple sclerosis lesion involving the cervical spinal cord.

Case Report

A 55-year-old woman was admitted to a local hospital in May of 1982 with rapidly developing weakness and numbness in all extremities. The weakness was greater on the right and the numbness greater on the left. Significant medical history included a diagnosis of left optic neuritis in 1978 and right optic neuritis in 1980.

On admission, CT of the cervical spine after intravenous administration of contrast material demonstrated an area of increased density in the center and to the right of the center of the cervical spinal canal that was believed to be pathologic (fig. 1A). Reformatted coronal and sagittal images showed that the lesion extended from C2 to the C4–C5 level (figs. 1B and 1C). Repeat CT without contrast material 2 weeks later demonstrated no abnormalities (fig. 2A). A CT scan at the same time after intravenous contrast administration showed marked decrease in the extent and intensity of contrast enhancement, with the enhancement now located to the left of the center of the spinal canal and limited to the C3–C4 disk space level (figs. 2B–2D). Four weeks after hospital admission, the patient was transferred to the University of Michigan Hospitals, where cervical myelography and CT were done after the lumbar introduction of metrizamide. The myelogram showed cervical spondylosis and a prominent but not

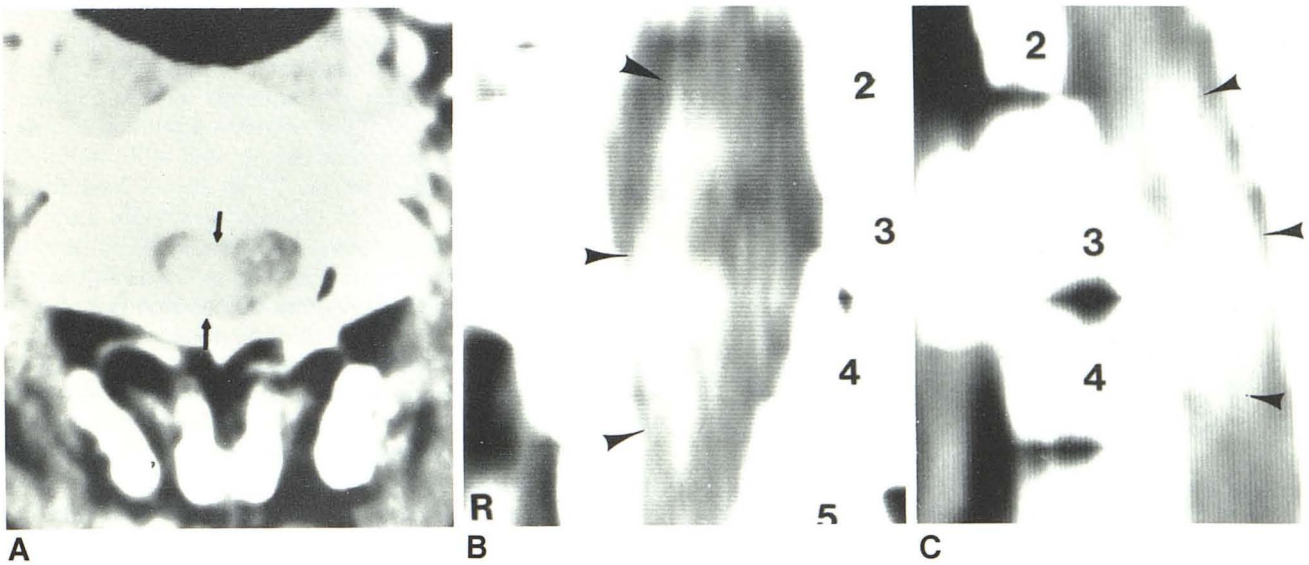

Fig. 1.—**A**, CT scan at C3 level after intravenous administration of contrast. Area of increased density in center and to right of center of cervical spinal canal (*arrows*). **B**, Reformatted CT scan from C2 to C5 in coronal plane through center of spinal canal. Increased-density lesion from C2 to C4–C5 level with involvement primarily to right of center (*arrowheads*). **C**, Reformatted CT in sagittal plane to right of center of spinal canal. High-density lesion again demonstrated (*arrowheads*).

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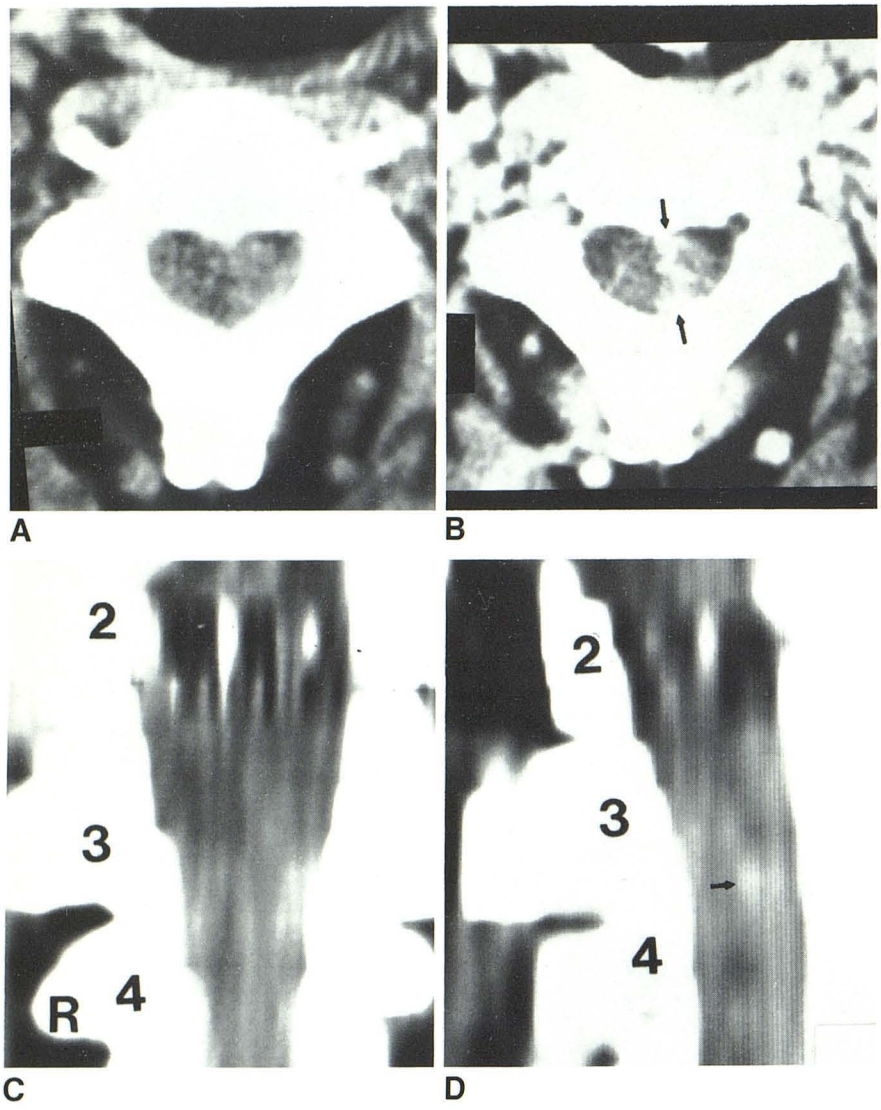

Fig. 2.—**A**, CT scan at C3 level without intravenous contrast 2 weeks after fig. 1. **B**, After intravenous contrast administration. Marked decrease in amount of pathologic contrast enhancement, now located to left of center of spinal canal (*arrows*). **C**, Reformatted CT in same coronal plane as fig. 1B is unremarkable. **D**, Reformatted CT in same sagittal plane as fig. 1C shows only suggestion of slight enhancement at C3 (*arrow*).

definitely enlarged cervical spinal cord. CT suggested mild, diffuse enlargement of the entire cervical spinal cord (fig. 3A). The next day CT with intravenous contrast administration demonstrated no evidence of contrast enhancement (fig. 3B). Cerebrospinal fluid analysis was negative for oligoclonal bands, myelin basic protein, and infectious agents. Because of the changing character of the contrast enhancement and history of optic neuritis, a presumptive diagnosis of multiple sclerosis was made. The patient was transferred to the Mayo Clinic where, despite steroid treatment, her condition deteriorated. The cervical spinal cord lesion led to respiratory failure, pneumonitis, and subsequently septic shock, of which she died 6 weeks after her initial hospital admission.

At autopsy, gross inspection revealed necrosis of the medulla oblongata and mid–cervical spinal cord. At the C3 level, the cord was involved bilaterally but more so on the right. Microscopic examination of the cervical spinal cord disclosed an acute demyelinating process surrounding an area of resolving and active necrosis compatible with multiple sclerosis (fig. 4). Demyelinating plaques consistent with old multiple sclerosis were also found in the optic chiasm and both optic nerves.

Discussion

The contrast enhancement of the cervical spinal cord on CT correlated with the areas of necrosis seen at autopsy. The diagnostic possibilities considered initially were intramedullary neoplasm, such as hemangioblastoma [2], astrocytoma [3], or ependymoma [4]; extramedullary, intradural neoplasm, such as meningioma and neurofibroma [4]; vascular malformation [5]; and multiple sclerosis [1]. Other demyelinating processes due to a postinfectious or postimmunization myelopathy, traumatic contusion, and ischemia could also possibly cause an enhancing lesion of the spinal cord. The subtle enlargement of the spinal cord as documented on metrizamide CT could be seen in many of the lesions listed above, but in our case was thought probably to represent an edematous spinal cord, which can occur in acute multiple sclerosis [6]. The decreased contrast enhancement 2 weeks after initial CT and lack of enhancement 4 weeks after initial CT would

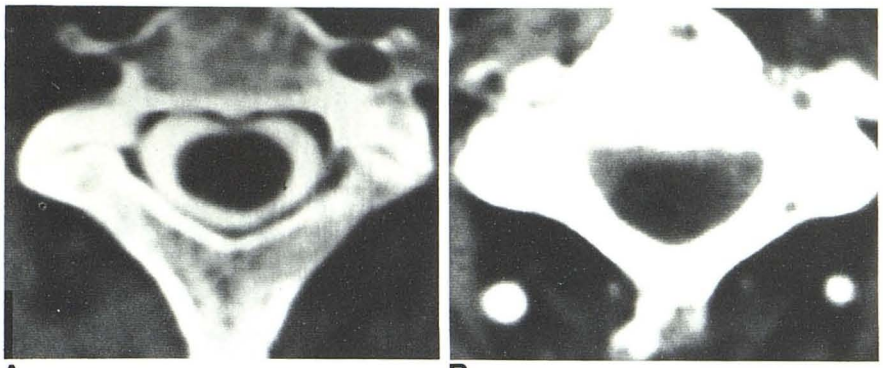

Fig. 3.—**A**, CT scan with intrathecal metrizamide but no intravenous contrast administration at C3 level 4 weeks after fig. 1. Midline bony spur and subtle cord enlargement. **B**, 1 day later with intravenous contrast administration. No pathologic contrast enhancement.

Fig. 4.—Spinal cord C3 level. Extensive demyelination and acute necrosis of right side of spinal cord (*arrows*) corresponding to area of increased density seen in fig. 1. Less marked involvement of posterior and anterior columns on left side corresponds to region of contrast enhancement seen in fig. 2. (Luxol fast blue/PAS/hematoxylin stains.)

be expected as the acute multiple sclerosis or other nonneoplastic process subsided. The rapidly changing character of the lesion made a surgically correctable lesion such as neoplasm or vascular malformation an unlikely possibility. The sequence of CT findings combined with the history of optic neuritis prompted a presumptive diagnosis of multiple sclerosis, which was substantiated at autopsy.

Multiple sclerosis is a disease characterized by demyelination of the central nervous system. In one series, 80% of the patients manifested clinical evidence of multiple sclerosis involving the spinal cord [7]. Although the spinal cord, optic nerves, brainstem, and cerebellum all are involved more frequently than the cerebrum [8], most efforts to image the plaques of multiple sclerosis by CT have been directed to the cerebrum. The lack of demonstration of multiple sclerosis spinal cord lesions by CT can be explained by the inherent technical difficulties involved in the imaging of enhancing lesions of the spinal cord within its irregular dense bony structures as well as the much more subtle findings expected in a typical nonnecrotic demyelinated plaque of smaller size than that demonstrated in our case.

A clinical syndrome of transverse myelitis with optic neuritis has been described and referred to as Devic disease or neuromyelitis optica. Sensory and motor changes secondary to spinal cord involvement as well as the visual symptoms of optic neuritis are commonly seen in multiple sclerosis. Controversy exists as to whether Devic disease is just a multiple sclerosis variant or actually a separate disease. As in our case, Devic disease often assumes an acute fulminant course leading to spinal cord necrosis, a rare finding in other forms of multiple sclerosis [9].

An awareness that multiple sclerosis of the spinal cord may present as an expanding process with pathologic contrast enhancement on CT, combined with a clinical history consistent with multiple sclerosis, should help to establish a correct diagnosis of multiple sclerosis and prevent a misdiagnosis of neoplasm and unwarranted operation.

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