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Radiation Myelopathy of the Cervical Spinal Cord: MR Findings

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Radiation myelopathy is one of the most serious complications of radiation therapy, with over 300 cases reported since its discovery in the 1930s [1]. Diffuse cord enlargement on myelography has been described as an occasional and nonspecific finding [2], although the spinal cord may be normal in size. We describe the MR findings in a case of radiation myelitis in which the diagnosis was suggested preoperatively by the combination of MR and clinical findings even though myelographic and T1-weighted MR imaging results were normal (cord size and contour were normal). To our knowledge, this is the first MR demonstration of a histologically confirmed case of radiation myelopathy, and the first demonstrating contrast enhancement.

Case Report

A 44-year-old man presented with a 6-week history of progressive right-sided weakness and paresthesias beginning in the lower extremity and progressing to involve the upper extremity. Twenty months earlier the patient had undergone resection of a medulloblastoma of the left cerebellar hemisphere, followed by a 6-week course of craniospinal radiation therapy, given by a double-junction technique [3]. At that time, 2700 cGy were delivered to the posterior fossa in 180 cGy fractions through parallel opposed lateral portals measuring 9.5×9.5 cm. During subsequent craniospinal irradiation, the whole brain received an additional 2880 cGy in 180 cGy fractions, and the cord received 2520 cGy in 180 cGy fractions. Hence, the posterior fossa received a total dose of 5580 cGy. The spine was treated in two separate parts. The cervical and thoracic spine was encompassed in a port measuring 6×32 cm, and a dose of 2520 cGy was delivered at 180 cGy/fraction. Appropriate gaps were calculated between treatment portals. All treatments were given with 4 MeV X-rays.

On examination, the patient had a mild (4/5) right hemiparesis and a mild left T4 sensory deficit. Deep tendon reflexes were hyperactive on the right with spontaneous ankle clonus and a positive Babinski sign. His gait was wide-based with circumduction of the right leg. A cervical myelogram at this time was normal, as were CSF chemistries and cytology.

An MR examination at this time revealed extensive abnormal signal within the cervical spinal cord. On T2-weighted images (Fig. 1)

abnormally high signal was observed within the posterior medulla, extending inferiorly through the C4–C5 level. Pre- and postinfusion (gadopentetate dimeglumine) T1-weighted images (Fig. 2) revealed a more focal ovoid area of contrast enhancement within the central and right lateral cord at the C1–C2 level. The differential diagnosis included metastatic medulloblastoma and radiation myelitis.

In view of the progressive nature of the patient's signs and symptoms, as well as the failure of steroid therapy, a C1–C2 laminectomy and a biopsy of the cervical cord lesion were performed. Upon opening the dura, the right lateral aspect of the spinal cord was noted to be soft and yellow in color. Through a small myelotomy, two biopsies were taken. On pathologic examination, the intramedullary spinal cord specimen demonstrated fragments of white matter with mild vacuolation and minimal gliosis, with fibrinoid damage of small vessels and capillaries; findings consistent with radiation change.

Discussion

Four distinct clinical syndromes of radiation myelopathy have been described, of which chronic progressive radiation myelitis [4] is the only form for which pathologic confirmation in humans exists. The patient typically presents 9 to 15 months after the completion of radiotherapy (a latency range of 1 to 72 months has been reported) [5], with paresthesias and inability to perceive pain and temperature. All cord systems tend to become involved over the next 6 months.

Three criteria for the diagnosis of radiation myelopathy have been established [6]:

1. The spinal cord must have been included in the radiation field.
2. The neurologic deficit must correspond to the cord segment that was irradiated.
3. Metastases or other primary spinal cord lesions must be ruled out.

Extensive research has been done to quantitatively define the risk factors for the induction of radiation myelopathy. Although there is disagreement concerning the limits of cord tolerance, certain qualitative criteria are well accepted. Most importantly, there is an increased risk associated with a higher fraction size to attain a given dose, a shorter treatment time,

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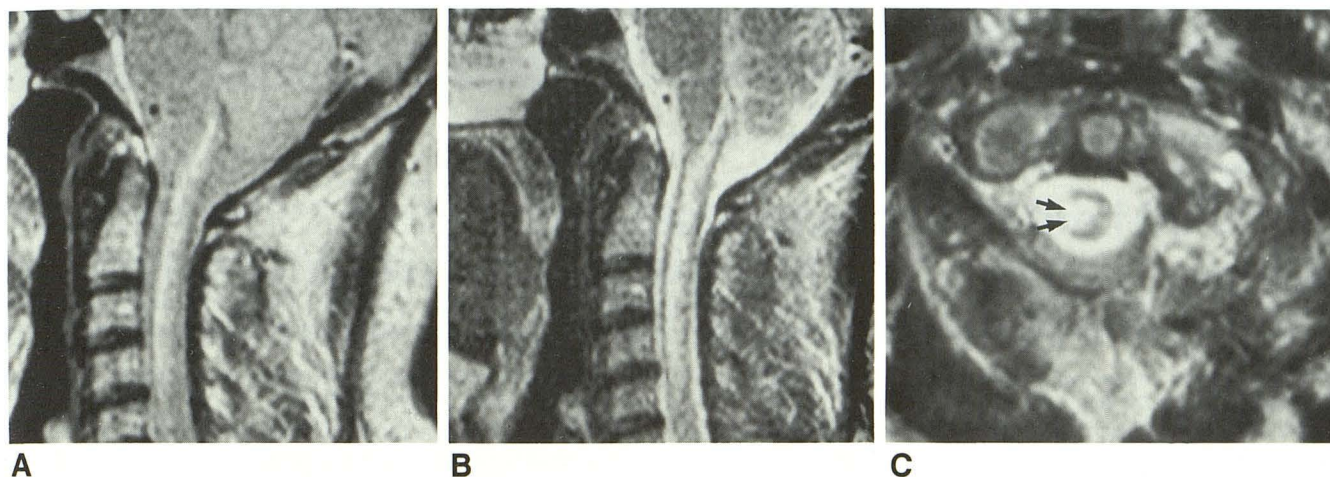


Fig. 1.—43-year-old man with biopsy proved radiation myelitis. MR images of the cervical spinal cord obtained at 0.5 T. A and B, Sagittal proton density and T2-weighted images, 2000/50 (A) and 2000/100 (B), show abnormally increased signal within spinal cord extending from posterior medulla to C4–C5 level. C, Transverse axial T2-weighted SE image, 2000/100/2, at C1–C2 level shows eccentric high-signal lesion within cord, extending toward the right (arrows).

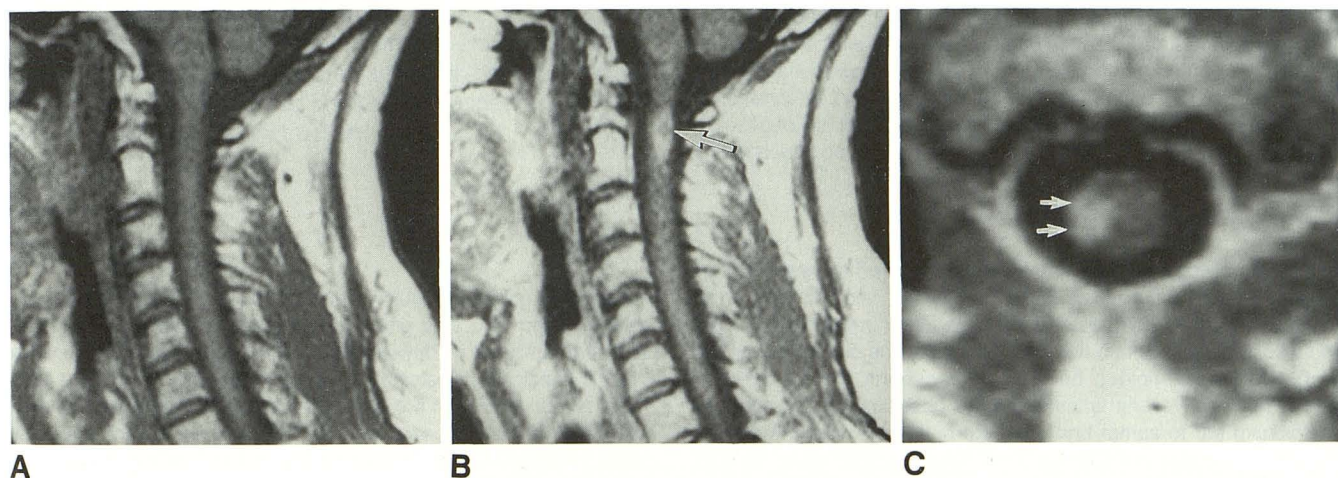


Fig. 2.—Same patient as in Fig. 1. T1-weighted SE images, 700/30/2, obtained before (A) and after (B and C) administration of 0.1 mmol/kg gadopentetate dimeglumine.

A, Preinfusion sagittal image is normal. There is no evidence of hemorrhage (no T1 shortening). B and C, Postinfusion sagittal (B) and axial (C) images show focal enhancement at C1–C2 (arrows). This represents a subsegment of the area that exhibited high signal on T2-weighted images (Fig. 1). The enhancement is eccentric toward the right (C).

and a higher total dose. According to the research of Wara et al. [7], a dose of 4174 cGy in 25 fractions was considered safe in 99% of irradiated patients. Similarly, 95% of patients would tolerate 4500 cGy and 50% would tolerate 6069 cGy in 25 fractions. The patient in the present case received 5580 cGy in 31 fractions.

There is also evidence to suggest that increasing the length of cord treated and concomitant administration of certain chemotherapeutic agents [8] are additional risk factors in the development of radiation myelopathy.

Radiologic imaging in cases of suspected myelopathy is performed primarily to exclude other abnormalities such as intramedullary or extramedullary tumor, degenerative spinal stenosis, and other potentially treatable conditions that may

affect spinal cord function. Myelography is typically normal although diffuse cord enlargement [2] or mild cord atrophy [9] have been observed.

Our case illustrates the advantage of MR over myelography in detecting localized spinal cord disease when the external dimensions of the cord are normal. The MR findings of an intramedullary lesion that exhibits T1 and T2 prolongation are, however, nonspecific. Neoplasm, cord contusion, demyelinating disease, cord ischemia, or infarction may present this way. Any of these lesions may also show enhancement with gadopentetate dimeglumine. In the case described, these nonspecific findings were present; however, the classic history and pathologic findings support the diagnosis of radiation myelopathy. As only the region showing enhancement was

biopsied, it is unclear whether any histologic differences would have been found in areas of cord exhibiting T2 prolongation without contrast enhancement.

In conclusion, radiation myelitis may be added to the differential list of conditions exhibiting prolonged T2 relaxation and contrast enhancement on MR imaging studies.

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