Pitfall of MR in a patient with spinal ependymoma and drop metastasis.

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LETTERS

Pitfall of MR in a Patient with Spinal Ependymoma and Drop Metastasis

False-negative magnetic resonance (MR) scans have been reported, before the use of gadolinium-based contrast agents, in patients with ependymoma associated with significant neurologic findings (1). We report a case of an ependymoma in a patient with minimal neurologic findings whose primary lesion was demonstrated by MR while a substantial drop metastasis was not detected, even after administration of contrast.

A 28-year-old man was admitted for severe, acute lower lumbar pain. Physical examination revealed left-sided L-5 and S-1 radiculopathies. Lumbar puncture yielded blood-tinged cerebrospinal fluid, moderate pleocytosis, and a protein concentration elevated to 160 mg/dL (normal, 15 to 45 mg/dL).

An MR scan at 1.5 T of the lower thoracic and lumbar spine was performed. The T1-weighted images revealed an abnormal, small focus of increased signal just inferior to the conus medullaris, at the L-1 level, which enhanced after intravenous administration of gadopentetate dimeglumine (0.1 mmol/kg) (Fig 1). A corresponding focus of abnormal, heterogeneous signal intensity was seen on the proton-density and T2-weighted images (Fig 2). No other lesions were appreciated. A computed tomographic myelogram confirmed the abnormality just inferior to the conus medullaris at the L-1 level. An additional, well-circumscribed, oval 2.0 × 1.0 × 1.0-cm intradural mass was demonstrated at the L-4/L-5 level (Fig 3). There was no evidence to suggest a vascular malformation.

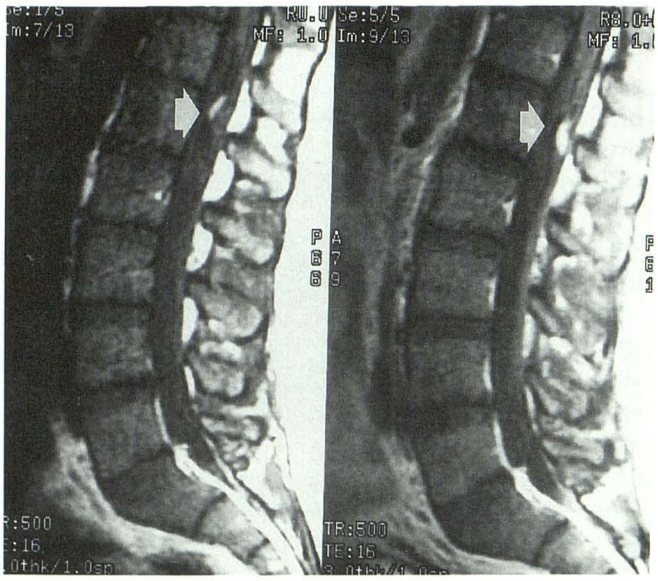

Fig 1. Sagittal spin-echo T1-weighted images (500/16/1.0 [repetition time/echo time/excitations]; 256 × 192 matrix size; 3-mm section thickness; 1-mm interspacing) obtained before (left image) and after (right image) administration of gadopentetate dimeglumine. There is a small focus of abnormal increased signal (left image, *white arrow*) inferior to the conus medullaris, at the L-1 level. This lesion demonstrates homogeneous enhancement (right image, *white arrow*).

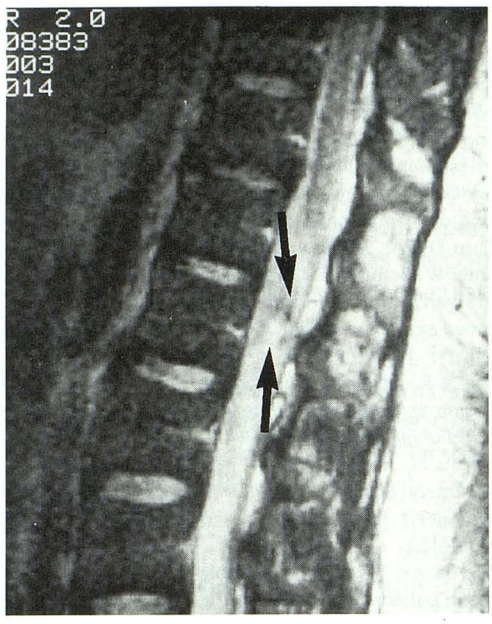

Fig 2. Sagittal fast spin-echo T2-weighted image (3000/96 effective/2.0; 256 × 256 matrix size; 3-mm section thickness; 1-mm interspacing) demonstrating abnormal heterogeneous signal within the lesion at the L-1 level (*black arrows*).

Separate laminectomies at L-1 and L-4 were performed. The superior lesion, which involved the filum terminale, was resected. The inferior lesion, surrounded by the cauda equina, was removed en block. This lesion had no vascular pedicle or attachment to nerve roots. Histologic evaluation of the surgical specimens showed the superior lesion to be a myxopapillary ependymoma. The inferior lesion was composed of a very thin rim (approximately 1 mm) of ependymoma cells surrounding the bulk of the lesion, which was filled with coagulative necrosis.

The coexistence of spinal ependymoma, sciatica, and blood on lumbar puncture is known as the Fincher syndrome. Although our case had these features, this association of findings is felt to be rare.

The rostral lesion displayed classic MR features of myxopapillary ependymoma. However, the inferior ependymoma was not detected with a high-quality MR study. We hypothesize that the inferior lesion was not appreciated because it comprised a very thin layer of tumor cells, which MR was not able to resolve. The remainder of the lesion was necrotic and had signal characteristics similar to the surrounding cerebrospinal fluid. Because there was no vascular pedicle, it is not surprising that enhancement was not seen.

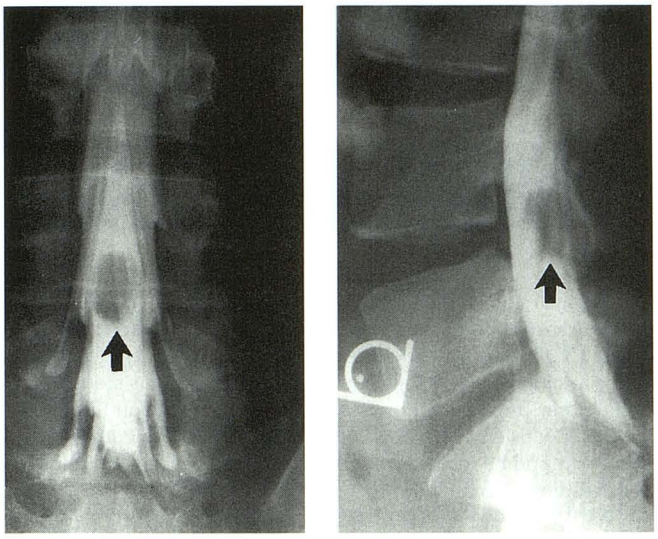

Fig 3. Anteroposterior and lateral radiographs from the myelogram demonstrating an intradural lesion at the L-4/L-5 level (*black arrows*).

The failure of MR to clearly detect the second lesion was significant for two reasons. Had this drop metastasis not been discovered, adjuvant radiation therapy may not have been given; and because the lesion was not seen on MR, it is unclear what imaging strategy should be used to follow the patient.

We agree with Epstein et al (1) that in a patient with a neurologic deficit and a negative or equivocal MR, a myelogram may be of value. Further, this case demonstrates that the drop metastases of myxopapillary ependymoma can have an MR appearance similar to cerebrospinal fluid, making them difficult to resolve.

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Total Intravenous Anesthesia with Propofol in Pediatric Patients for MR Examination

There is still no agreement among people dealing with sedation of pediatric patients undergoing a magnetic resonance (MR) examination, and the "ideal" drug that is safe, fast in onset, has a short recovery time, and has no collateral effects, is still not on the market. We have given propofol (Diprivan) intravenously to induce general anesthesia with spontaneous ventilation in 118 pediatric patients 14 days to 11 years of age who are either inpatients or outpatient undergoing MR study of the central nervous system.

Previous reports have described the use of intravenous propofol for anesthesia in minor surgical procedures (1, 2), for pediatric sedation during radiologic imaging studies (3), and for neurosurgical procedures. This drug is capable of reducing the cerebral metabolic rate of carbon dioxide and reducing intracranial pressure (4). Reported properties of propofol such as rapidity of action and speed of emergence from anesthesia justify the use of this drug for anesthesia in neuropediatric patients undergoing MR. Because in Italy the use of propofol is "inadvisable in children under 3 years of age" and it is "not licensed" in intensive care sedation of children, we prospectively recruited a group of 118 patients for our study after obtaining parental consent and approval of the hospital ethics committee. Anesthesia was induced by intravenous bolus or, more frequently, by fast drip of propofol in 68 patients, by propofol associated with neuroleptanalgesic intravenous drugs in 16 patients, by propofol and halothane in 14 patients, and by halothane with supplemental oxygen in 20 patients. Halothane alone was used for induction in younger children or when an intravenous access, necessary to inject propofol, was difficult to establish. Anesthesia was maintained by intravenous continuous infusion of propofol controlled through the use of an infusion pump in all patients who continued with spontaneous ventilation receiving supplemental oxygen (4 L/h) through a small tube. Variable individual mean doses of the drug were identified adapting the dosage from data reported in the literature. The sedation regimen we developed allowed anesthesia of adequate depth with spontaneous ventilation and complete immobility of all children during MR, so that all MR examinations were successfully completed.

No side effects occurred. However, we would like to stress that propofol is an "intravenous anesthetic," and its infusion requires the constant supervision of an anesthesiologist or a trained person and monitoring the patient's vital signs, including electrocardiogram, end-tidal CO_2, pulse oximetry, respiratory frequence, pulse rate, and blood pressure, recorded from induction to complete recovery according to recommendations of the American Society of Anesthesiologists (ASA) and the Section of Anesthesiology of the American Academy of Pediatrics (AAP). As opposed to the Bloomfield study (3), in which 10% of the patients receiving propofol had sufficient disruption of ventilation to develop a pulse oximetry under 90%, in our experience the depressant effect on ventilation