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False-Positive Spinal MR Findings for Subarachnoid Spread of Primary CNS Tumor in Postoperative Pediatric Patients

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We describe three children who exhibited increased CSF signal intensity (T1 shortening) on gadopentetate-dimeglumine-enhanced T1-weighted spinal MR scans. Two patients were being evaluated for spinal leptomeningeal metastases 3 days after surgical resection of posterior fossa neoplasms (juvenile pilocytic astrocytoma and medulloblastoma). The CSF hyperintensity on contrast-enhanced images was initially thought to represent diffuse subarachnoid spread of tumor. However, the similarity of findings in these two cases led us to speculate that the apparent CSF enhancement (T1 shortening) was a false-positive finding caused by the presence of methemoglobin and/or contrast enhancement of leptomeninges irritated by occult postoperative subarachnoid blood.

Evaluation of CSF from both patients within 24 hr of the MR scans revealed xanthochromia and numerous red blood cells without cytologic evidence of malignant cells. Repeat contrast-enhanced MR scans in both patients 13 days after surgery documented resolution of the shortened T1 of the CSF. Repeat CSF analysis and CT myelography 13 or 17 days after surgery were negative for malignancy in both patients.

A third patient had dependently layering CSF hyperintensity on T1-weighted contrast-enhanced MR images 3 days after resection of a fourth ventricular choroid plexus papilloma, a benign tumor with no known incidence of subarachnoid drop metastases [1].

Our observations represent an important potential pitfall in the postoperative MR evaluation of spinal subarachnoid metastases in pediatric patients with primary CNS neoplasms. Gadopentetate-dimeglumine-enhanced MR imaging to assess subarachnoid metastases in this population should be performed at least 2 weeks after surgery.

Materials and Methods

All three patients in our series had enhanced MR scans of the spine immediately after routine postoperative brain imaging, which

included IV-administered gadopentetate dimeglumine in a dose of 0.2 ml/kg (0.1 mosm/kg). Axial and sagittal T1-weighted spin-echo pulse sequences with TR/TE of 500/20 were obtained of the entire spine with a 14-in. rectangular surface coil. Slice thickness was 3 mm with a 1.5-mm interslice gap for the sagittal images and 5 mm with a 2.5-mm interslice gap for the axial images using a 16-cm field of view, 128 × 256 matrix, and four excitations. All studies were done on a GE 1.5-T Signa scanner. Follow-up spine MR examinations in two patients after IV administration of gadopentetate dimeglumine were performed using similar imaging parameters. CSF was examined for cell count and the presence of malignant cells within 24 hr of the initial MR scans and again at the time of water-soluble contrast CT myelography in the two patients with malignant tumors. CT scans were performed on a GE 9800 scanner.

Case Reports

Case 1

A 10-year-old girl presented with a 2-month history of early morning headache and vomiting. CT showed an enhancing midline cerebellar mass. Surgical resection via a suboccipital craniotomy in the prone position yielded a pathologic diagnosis of juvenile pilocytic cerebellar astrocytoma. Initial contrast-enhanced MR imaging of the spine 3 days after surgery revealed diffuse CSF hyperintensity within the thoracic and lumbar regions (Figs. 1A and 1B). An atraumatic lumbar puncture on the following day revealed xanthochromic CSF with 3300 red blood cells and 18 white blood cells per cubic mm, glucose of 120 mg/dl, and total protein of 67 mg/dl. Cytology was negative for malignant cells. Repeat contrast-enhanced MR imaging 13 days after surgery documented resolution of the CSF hyperintensity (Figs. 1C and 1D). The CT myelogram performed on the same day was normal. CSF obtained at that time was no longer xanthochromic and was again negative for malignant cells.

Case 2

A 10-year-old boy presented with a 2-week history of nausea, vomiting, headache, and ataxia. CT demonstrated a homogeneously enhancing midline posterior fossa mass. Surgical resection via sub-

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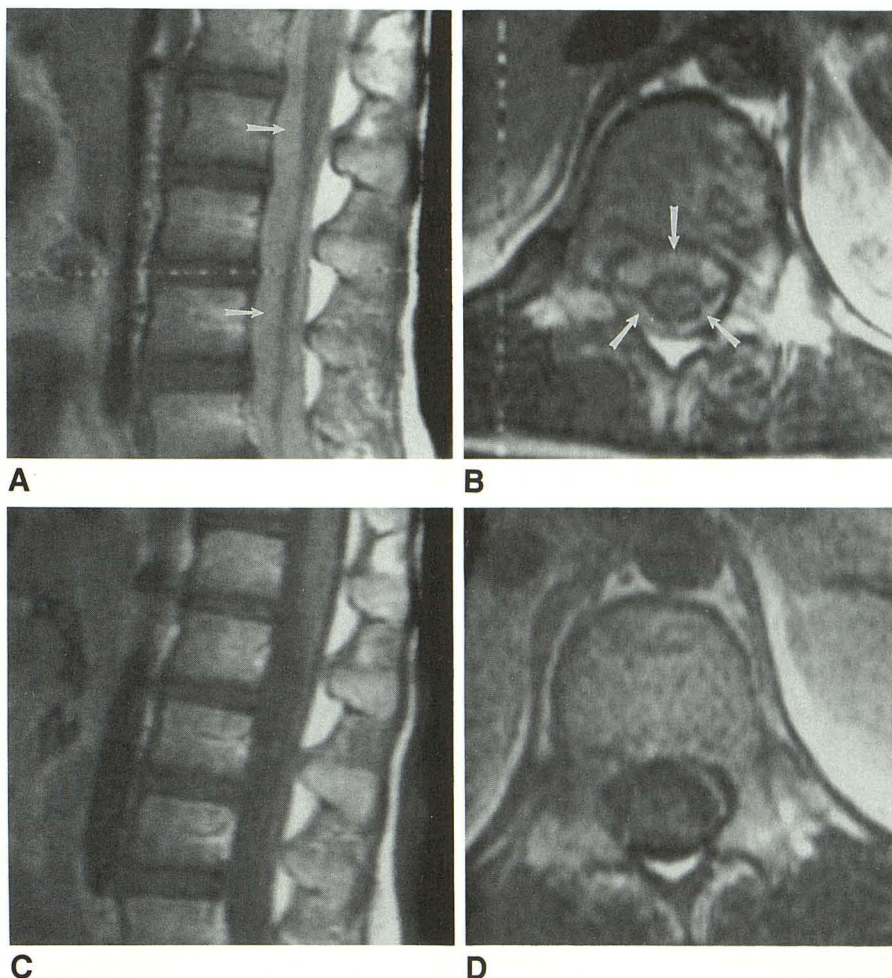
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Fig. 1.—Case 1: 10-year-old girl after resection of juvenile pilocytic cerebellar astrocytoma. **A and B,** Sagittal (**A**) and axial (**B**) contrast-enhanced T1-weighted SE images (20/500) of the lumbar spine 3 days after surgery show diffuse CSF hyperintensity (*arrows*).

C and D, Sagittal (**C**) and axial (**D**) contrast-enhanced T1-weighted SE images (20/500) 13 days after surgery reveal resolution of the CSF hyperintensity.



occipital craniotomy in the prone position yielded a pathologic diagnosis of medulloblastoma. Initial contrast-enhanced MR imaging of the spine 3 days after surgery revealed diffuse CSF hyperintensity in the thoracic and lumbar regions (Figs. 2A and 2B). An atraumatic lumbar puncture on the following day revealed xanthochromic CSF with 80,000 red blood cells and 500 white blood cells per cubic mm, glucose of 105 mg/dl, and total protein of 225 mg/dl. Cytology was negative for malignant cells. Repeat contrast-enhanced MR imaging 13 days after surgery documented resolution of the abnormal CSF signal (Figs. 2C and 2D). The CT myelogram performed 4 days later was normal. CSF obtained at that time was no longer xanthochromic and was again negative for malignant cells.

Case 3

A 13-year-old girl presented with a 1-year history of headaches. CT demonstrated a homogeneously enhancing midline posterior fossa mass. Surgical resection via a suboccipital craniotomy in the prone position yielded a pathologic diagnosis of choroid plexus papilloma. Contrast-enhanced MR imaging of the spine 3 days after surgery revealed dependent subarachnoid increased T1 signal intensity in the dorsal thecal sac, only within the thoracic kyphosis (Fig. 3). No lumbar puncture or myelogram was performed owing to the benign histology of the primary tumor.

Discussion

While initial reports of unenhanced MR imaging of cranial and spinal meningeal carcinomatosis were disappointing [2, 3], more recent reports on gadopentetate-dimeglumine enhancement in human and animal models offer greater promise [4–10]. Some researchers believe that enhanced MR scans will make a strong bid to replace CT myelography in the evaluation of spinal leptomeningeal tumor seeding. On the basis of these preliminary reports and the desire to eventually replace the invasive myelographic examination (which often requires general anesthesia in our pediatric population) with the noninvasive MR examination, we began a prospective comparison of contrast-enhanced MR imaging with CT myelography for assessment of subarachnoid drop metastases in pediatric patients with primary CNS neoplasms. It was with our initial two patients that we recognized the described important potential pitfall in postoperative MR imaging of this population.

The presence of red blood cells in the xanthochromic CSF and the absence of cytologic evidence of malignant cells support the hypothesis that the MR findings of diffuse CSF

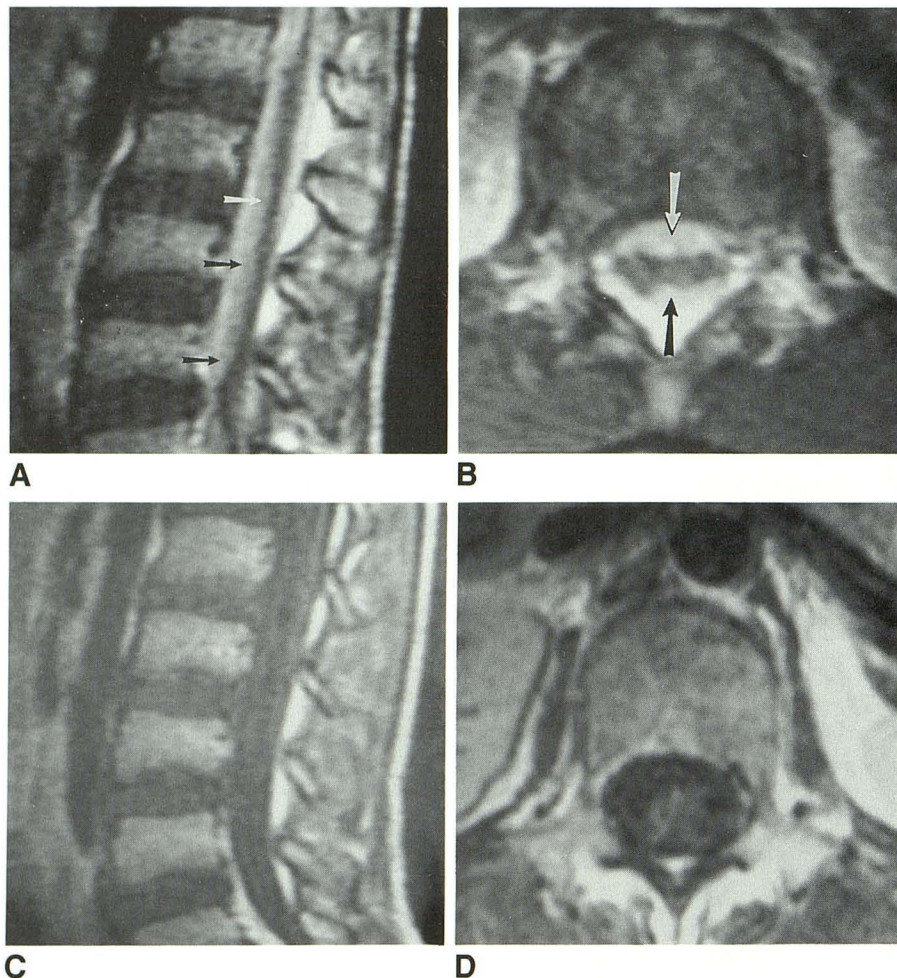


Fig. 2.—Case 2: 10-year-old boy after resection of posterior fossa medulloblastoma.

A and B, Sagittal (A) and axial (B) contrast-enhanced T1-weighted SE images (20/500) of the lumbar spine 3 days after surgery show diffuse CSF hyperintensity (arrows), similar to Figs. 1A and 1B.

C and D, Sagittal (C) and axial (D) contrast-enhanced T1-weighted SE images (20/500) 13 days after surgery reveal resolution of the CSF hyperintensity, similar to Figs. 1C and 1D.

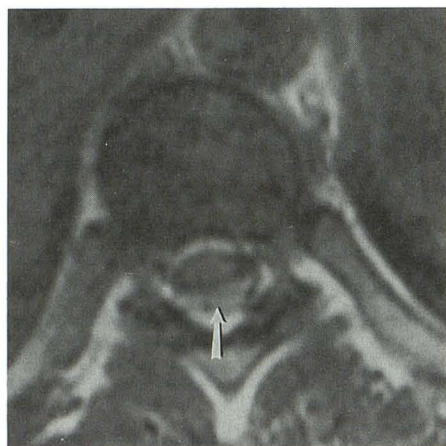


Fig. 3.—Case 3: 13-year-old girl after resection of fourth ventricular choroid plexus papilloma. Axial contrast-enhanced T1-weighted SE image (20/500) at level of thoracic kyphosis 3 days after surgery shows dependent CSF hyperintensity (arrow).

hyperintensity on contrast-enhanced images were caused by occult subarachnoid blood introduced during recent surgery. This hypothesis is further supported by the resolution of the abnormal MR findings on contrast-enhanced scans performed

10 days later using identical imaging parameters and the fact that both patients had normal CT myelograms.

Blood is a known leptomeningeal irritant and has been implicated in the pathogenesis of arachnoiditis [11]. Leptomeningeal irritation from subarachnoid blood, in our experience, commonly enhances on both CT and MR examinations of the brain following surgery, trauma, or spontaneous hemorrhage. Recently, postoperative cranial leptomeningeal contrast enhancement on MR imaging has been well documented [12–15]. Sze and colleagues [12] described diffuse cranial leptomeningeal contrast enhancement on MR imaging in a 60-year-old patient 1 week after removal of a brain metastasis that resolved 4½ weeks after surgery. They speculated that the transient enhancement “could result from meningeal irritation caused by blood in the subarachnoid space as a result of the craniotomy.” Burke and coworkers [15], in their review of 20 postcraniotomy patients, believed that “postcraniotomy meningeal enhancement is most likely the result of a local inflammatory process or a diffuse chemical arachnoiditis caused by bleeding into the subarachnoid space at the time of surgery.” As to the time course of leptomeningeal contrast enhancement, Elster and DiPersio [14] noted dural enhancement in two patients within 18 and 72 hr, respectively, after surgery; however, the initial appearance of brain/pial enhancement was less precisely documented in four patients “within

1 month" of surgery. There is no reason to believe that the contrast enhancement characteristics would differ with spinal leptomeninges.

MR imaging has been reported to be insensitive for detecting acute intracranial subarachnoid hemorrhage [16–18]. Methemoglobin formation has, however, been shown to contribute to T1 shortening of CSF in subacute subarachnoid hemorrhage [18]. Mixtures of 20% human whole blood in CSF have been shown in vitro to have progressively shorter T1 relaxation times over the initial 90 hr followed by a plateau to 160 hr [18]. Also, methemoglobin was demonstrated within bloody CSF stored hypoxically for 3 days.

Because the initial contrast-enhanced MR scans of the spine in our patients were obtained after routine contrast-enhanced brain imaging in order to shorten the examination time, we did not have precontrast images. Plausible explanations for the MR finding of CSF hyperintensity on T1-weighted contrast-enhanced images include T1 shortening due to the presence of methemoglobin alone and/or diffuse leptomeningeal enhancement as a result of meningeal irritation caused by subarachnoid blood. The apparent dependent layering of CSF hyperintensity in case 3 (Fig. 3) supports the contention that the methemoglobin or blood itself is the cause of T1 shortening. However, we have observed diffuse CSF hyperintensity on contrast-enhanced images similar to that shown in cases 1 and 2 (Figs. 1 and 2) in patients with carcinomatous meningitis and no history of surgery. Therefore, further work is needed, particularly with nonenhanced images and CSF methemoglobin measurements, before the explanation is clarified.

Attempts to duplicate the described findings in two additional children who had had surgery for primary CNS neoplasms were unsuccessful. One child had a parietal craniotomy for resection of a supratentorial primitive neuroectodermal tumor and the other had a suboccipital craniotomy for biopsy of a brainstem glioma. However, the amount of blood lost during both these procedures was not nearly as great as that lost during surgery for the three intra- and periventricular lesions described. Therefore, false-positive MR findings for spinal subarachnoid metastases may only be a clinical problem with certain tumors and/or surgical procedures associated with greater degrees of blood loss.

On the basis of our preliminary experience, we conclude that to avoid false-positive diagnosis of spinal subarachnoid metastases on contrast-enhanced MR images in pediatric

patients with primary CNS neoplasms, scans should be performed at least 2 weeks after surgery or repeated at this interval if earlier scans reveal diffuse CSF hyperintensity.

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