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Spinal Subdural Enhancement after Suboccipital Craniectomy

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PURPOSE: To characterize transient intraspinal subdural enhancement (potentially mimicking the subarachnoid spread of tumor) seen on MR images in some children after suboccipital craniectomy for posterior fossa tumor resection. **METHODS:** Radiologic and medical records of 10 consecutive children who had MR imaging for spinal staging after resection of posterior fossa tumor during a 9-month period were reviewed retrospectively. In addition, one case with similar findings of intraspinal enhancement on spinal staging MR images obtained at another institution was included in the review. **RESULTS:** Intraspinal enhancement thought to be subdural was seen in four of 10 patients undergoing spinal staging MR imaging 6 to 12 days after surgery. In these four patients, MR studies 5 to 18 days later, without intervening treatment, showed resolution of the abnormal enhancement. A fifth patient (from another institution) with similar intraspinal enhancement underwent CT myelography 4 days later, which showed no subarachnoid lesions. No metastases have developed in any of these five patients during the 2.5- to 3.5-year follow-up period. **CONCLUSION:** From analysis of the MR appearance and on the basis of prior myelographic experience, we suggest an extraarachnoid, probably subdural, location of this enhancement. Awareness of this phenomenon will reduce the rate of false-positive diagnoses of metastatic disease. Preoperative spinal staging should be considered for patients undergoing suboccipital craniectomy.

Index terms: Spine, magnetic resonance; Surgery, resective

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In the past, we occasionally encountered large subdural fluid collections during staging myelography after resection of posterior fossa tumors in children. As this generally occurred only when myelography was performed less than 2 weeks after the suboccipital craniectomy, we instituted a policy whereby myelography was delayed until after this 2-week period. With magnetic resonance (MR) imaging replacing myelography in the evaluation of drop metastases, we continued to stage patients after resections. Since then, we have encountered cases in which extensive intraspinal enhancement was present on MR images that we suspected was subdural in location. This experience prompted our retrospective evaluation of all cases of spinal staging MR examinations performed after posterior fossa tumor resection during a 9-month period to estimate the frequency of this finding and to characterize this enhancement pattern in order to distinguish it from subarachnoid spread of tumor.

Materials and Methods

The tumor registry was used to identify all patients (seven with medulloblastoma, five with cerebellar astrocytoma, three with ependymoma, and one with brain stem glioma) treated at Children's Hospital for posterior fossa tumors who underwent suboccipital craniectomies from November 1991 to August 1992. Radiologic studies and medical records of the 10 consecutive patients with either medulloblastoma or ependymoma were reviewed retrospectively. Routine spinal staging was not performed in the patients with cerebellar astrocytomas or brain stem gliomas; hence, these patients were not included in the review. An additional patient with a medulloblastoma treated at another institution was included in the analysis of imaging appearance.

Imaging was performed on 1.5-T MR systems. Sagittal noncontrast T1-weighted images were obtained, and both

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sagittal and axial images were obtained after administration of contrast material (0.2 mL/kg). In one patient, noncontrast sagittal T2-weighted images were also obtained.

Results

Of the 10 patients (age range, 1 to 12 years) who had undergone spinal MR imaging 4 to 18 days after posterior fossa tumor resection, one had evidence of spinal subarachnoid spread of tumor. Enhancement thought to be subdural in location was seen in four patients imaged 6 to 12 days after suboccipital craniectomy. In these four patients, and in an additional patient with presumed subdural enhancement (not treated at this institution, imaged 6 days after surgery), repeat MR imaging (n = 4) or computed tomographic (CT) myelography (n = 1) was performed 5 to 18 days after the initial MR study.

All five patients with subdural enhancement had variable areas of moderate to marked abnormal intraspinal enhancement after administration of contrast material (Figs 1 and 2). Follow-up MR studies in four of the patients 5 to 18 days later showed resolution of this abnormal enhancement, without intervening treatment. The fifth patient, who underwent myelography 4 days after the initial spinal MR study, had smaller subdural defects and no subarachnoid defects.

One of the patients with subdural enhancement did have a lumbar puncture before the initial spinal staging MR examination. The rest of the patients had lumbar puncture after spinal staging MR imaging. None of the patients had positive cytologic findings (including the one patient with metastatic disease, who did have arachnoid spread on intraoperative biopsy specimens). Cerebrospinal fluid analysis was otherwise remarkable for mildly elevated white blood cell counts (7 to 10×10^6/L; normal, 0 to 5×10^6/L) in three patients; two of these conditions could probably be accounted for by bloody taps, since numerous red blood cells were present (one of these patients had subdural enhancement, the other did not). None of the samples was reported as xanthochromic.

The most extensive abnormalities were seen in a patient whose spinal staging MR study was performed 6 days after surgery, with enhancement beginning in the upper thoracic spine and extending through the lumbar spine (Fig 1). Enhancement up to 5 mm in thickness was seen both anteriorly and posteriorly within the spinal canal. These areas were hypointense relative to cord on noncontrast T1-weighted images and were difficult to distinguish from cerebrospinal fluid. Sagittal T2-weighted images showed hyperintensity, indistinguishable from cerebrospinal fluid. The configuration on enhanced axial images suggested these collections were extraarachnoid, surrounding a partially collapsed subarachnoid cerebrospinal fluid space. A repeat MR examination 16 days later revealed resolution of the enhancement.

A second patient had moderate enhancement in the lower thoracic and lumbar spine on an MR study 12 days after surgery. This patient underwent CT myelography 4 days after the initial MR examination, at which time focal defects consistent with subdural collections were evident (although less extensive than on the initial MR study) and no subarachnoid abnormality was present.

Three additional patients had areas of variable, moderate enhancement in the thoracic and lumbar spinal canal on MR examinations 11 to 12 days after surgery (Fig 2). Follow-up MR studies 6 to 18 days later were unremarkable, with resolution of abnormal enhancement. All 10 patients in the series had at least some postoperative pseudomeningocele over the craniectomy sites. Surgical reports revealed all patients had suboccipital craniectomies with removal of the posterior ring of C-1. Dura was closed with a periosteal patch in 8 of the 10 patients.

All five patients with subdural enhancement subsequently received craniospinal irradiation with 3240 to 3600 cGy, as well as chemotherapy. None of these five patients has had evidence of metastatic disease at 2.5 to 3.5 years follow-up.

Discussion

Weiner et al (1) described three patients who had increased signal intensity within the spinal canal on contrast-enhanced MR images of the spine obtained 3 days after resection of posterior fossa tumors. The authors hypothesized that the abnormality of diffuse cerebrospinal fluid hyperintensity was due to the presence of occult subarachnoid blood and/or diffuse leptomeningeal enhancement as a result of meningeal irritation caused by the subarachnoid blood. They did not have noncontrast images to determine whether the T1 hyperintensity represented enhancement or T1 shortening as a re-

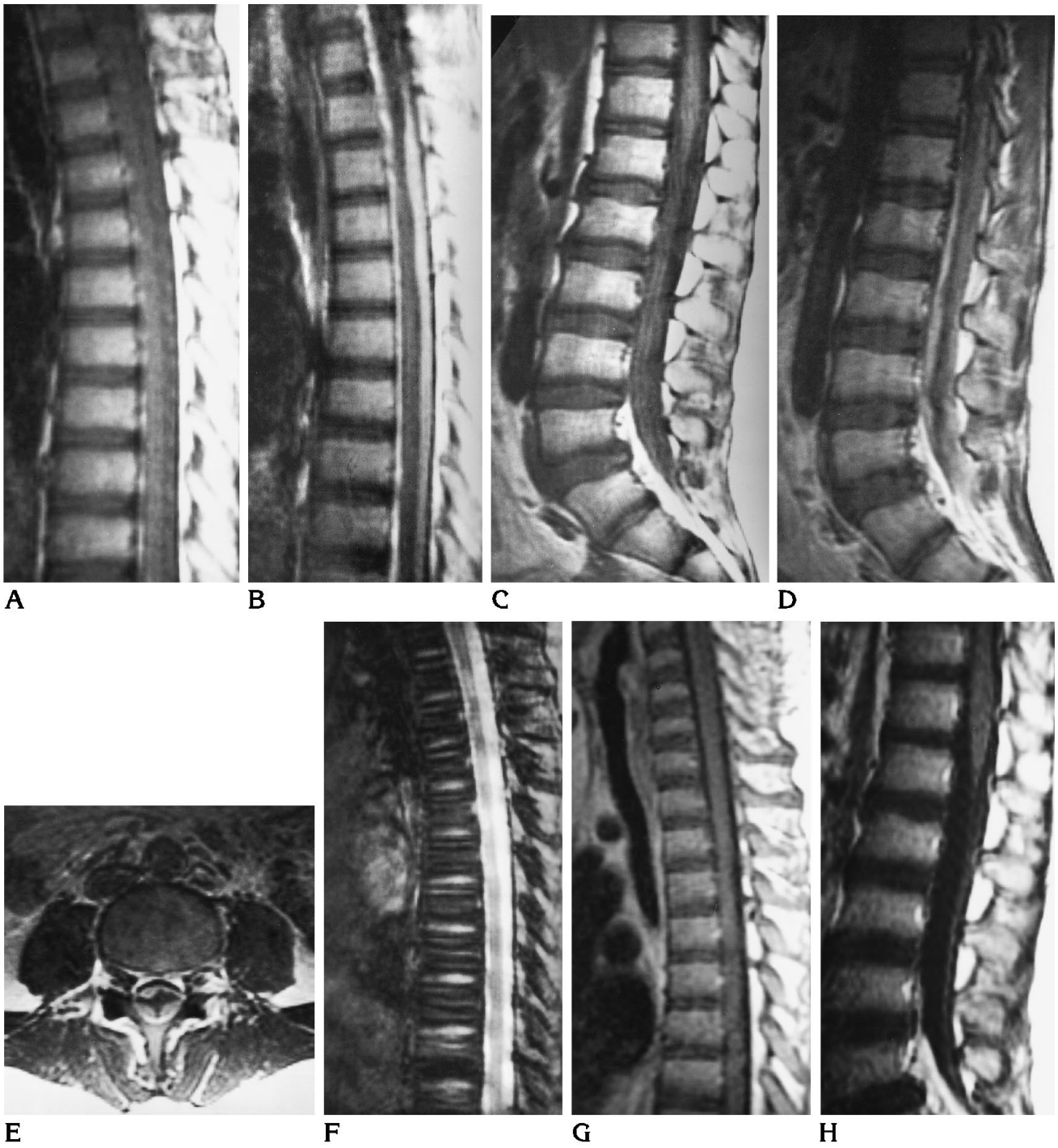

Fig 1. MR study obtained 6 days after resection of a medulloblastoma.

Sagittal T1-weighted images of the thoracic spine before (*A*) and after (*B*) contrast administration, and of the lumbar spine before (*C*) and after (*D*) contrast administration show extensive enhancement posterior and anterior to the spinal cord.

An axial contrast-enhanced T1-weighted image through the midlumbar spine (*E*) shows enhancement around the collapsed residual subarachnoid space. A sagittal T2-weighted image of the thoracic spine at this time (*F*) shows no obvious filling defect.

Sixteen days after the initial MR study, sagittal contrast-enhanced T1-weighted images of the thoracic (*G*) and lumbar (*H*) spine show resolution of the abnormal enhancement.

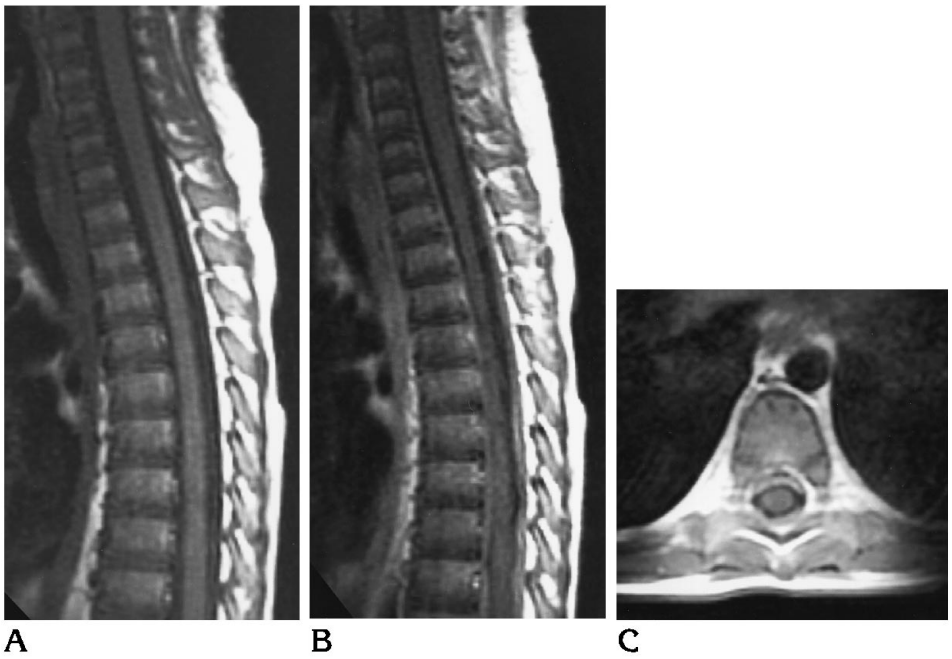

Fig 2. MR examination 11 days after resection of a medulloblastoma.

Sagittal noncontrast T1-weighted image (*A*) and sagittal (*B*) and axial (*C*) contrast-enhanced T1-weighted images of the thoracic spine show moderately intense enhancement anteriorly and posteriorly within the spinal canal.

sult of the blood. The cases in the present series clearly represent enhancement, as there was no hyperintensity on noncontrast T1-weighted images. Review of cerebrospinal fluid analysis does not provide any clear differentiation between those who did and those who did not have subdural enhancement.

Although myelography itself can result in subdural injection, it had been our impression that occasional large subdural fluid collections encountered with staging myelography were related to the preceding posterior fossa surgery. This opinion had led us to recommend delaying myelography until at least 2 weeks after suboccipital craniectomy. In the one patient who had a lumbar puncture before the spinal MR study, it is possible that a subdural collection was initiated by this procedure. The association with suboccipital craniectomies raises the possibility that disruption of the normal posterior dural attachments to the foramen magnum allows fluid to track into the potential subdural space. We were unable, however, to identify any significant difference in surgical approach between those patients who had postoperative subdural enhancement and those who did not. All patients had the posterior ring of C-1 resected and most had periosteal patches used to close the dura. The five patients with subdural enhancement were treated by three neurosurgeons, so that an unusual technique seems unlikely. In previous myelographic experience, collections generally had resolved when a repeat study was done, usually after about 2 weeks' delay. Concordantly, the abnormal enhancement in our series resolved in patients who had repeat MR imaging of the spine within a similar time period.

The frequency of this phenomenon is difficult to know, because it will be detected only in patients who are imaged after surgery. Furthermore, as it apparently resolves with time, detection will occur only if imaging is performed soon after surgery. During the period covered in this series, the subdural enhancement was seen in four of 10 patients undergoing surgery and spinal imaging. In the period of time in which these cases were accumulated, no preoperative spinal imaging was performed to prove the abnormalities were not preexisting. More recently, we have begun doing preoperative spinal staging with MR imaging in patients who are thought to have an ependymoma or medulloblastoma. During a 2-year period (encompassing 17 patients) none of these abnormalities has been seen before surgery.

Although a subarachnoid location is possible, configuration of abnormalities at least in some areas in the present cases suggests that they are extraarachnoid. The most likely potential area available would be within the subdural space. At several points there appears to be near circumferential abnormality around a compressed subarachnoid space. A subdural location would explain the posterior and ante-

rior (dependent and nondependent) locations of the enhancement at the crescentic configuration seen (2). Myelography performed in one patient showed defects consistent with residual subdural collections. All five of the present instances of enhancement occurred less than 2 weeks after surgery, similar to the previous myelographic experience. A review of images from the Wiener series suggests, at least in their first two cases, the possibility of some circumferential mass effect surrounding subarachnoid cerebrospinal fluid, which could then be subdural in location.

Other investigators have reported apparent septa, perhaps caused by intrathecal blood, on staging myelograms obtained after resection of posterior fossa tumors (3). These septa, however, were reported to have been seen in the thoracic and not the lumbar spine, where many of the present abnormalities were seen. Furthermore, no precontrast hyperintensity was seen in the present series to suggest subacute blood collections, although if sufficiently diluted this could go undetected. Finally, no subarachnoid septa were encountered in the one patient in our series who underwent myelography.

The mechanism of the enhancement in our cases is not known. Meningeal enhancement on MR images is associated with cranial subdural collections as well as with those subdural collections present after surgery, especially in children (4). Enhancement presumably is related to neovascularization, which has been seen histologically (5, 6), perhaps provoked by inflammatory components of the fluid that undoubtedly would contain some blood products. This neovasculature has open endothelial junctions with numerous fenestrations; exudation from these vessels is one mechanism proposed for the growth of chronic subdural collections. Contrast material leaking from similar neovasculature could accumulate in the subdural collection itself, thus explaining the findings in our cases. An analogous mechanism has been used to explain delayed accumulation of contrast agent in pineal cysts, where there is probably passive diffusion of contrast material from the surrounding pineal tissue, which similarly lacks a blood-brain barrier (7).

Resolution of these abnormalities without treatment is consistent with the presumption that they do not represent metastatic disease. This would also be consistent with previous myelographic experience in which subdural collections resolve.

On the basis of our experience, we conclude that to avoid possible misdiagnosis of spinal subarachnoid metastasis in children with posterior fossa tumors, consideration should be given to performing preoperative spinal staging MR imaging or to delaying the examination until at least 2 weeks after suboccipital craniectomy.

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