AJNR

Gd-DTPA-enhanced MR imaging of leptomeningeal spread of primary intracranial CNS tumor in children.

D J Rippe, O B Boyko, H S Friedman, W J Oakes, S C Schold, Jr, G R DeLong and W J Meisler

AJNR Am J Neuroradiol 1990, 11 (2) 329-332
http://www.ajnr.org/content/11/2/329

Gd-DTPA–Enhanced MR Imaging of Leptomeningeal Spread of Primary Intracranial CNS Tumor in Children

David J. Rippe[1]
Orest B. Boyko[1]
Henry S. Friedman[2]
W. Jerry Oakes[3]
S. Clifford Schold, Jr.[4,5]
G. Robert DeLong[4]
William J. Meisler[1]

Three children with known primary brain neoplasms and leptomeningeal disease were evaluated with MR imaging. Two of the patients had medulloblastoma and one had pineoblastoma. The presence of leptomeningeal tumor spread was established by positive CSF cytopathology in conjunction with compatible contrast-enhanced CT findings. Contrast-enhanced CT, nonenhanced MR, and Gd-DTPA–enhanced MR studies were then compared. In two cases, leptomeningeal lesions were seen better with Gd-DTPA–enhanced MR than with contrast-enhanced CT. In all three cases, Gd-DTPA MR imaging revealed lesions that were not identified on noncontrast MR.

Gd-DTPA–enhanced MR imaging is useful when searching for intracranial leptomeningeal tumor deposits in pediatric patients at risk for this condition.

***AJNR* 11:329–332, March/April 1990**

Leptomeningeal metastasis consists of diffuse seeding of the leptomeninges by metastatic tumor. Commonly, the disorder results from carcinoma that has originated in the breast or lung or from other systemic neoplasms such as lymphoma [1]. Spread of primary brain neoplasms also represents a significant cause of tumor infiltration of the meninges. Primary CNS lesions associated with this condition include medulloblastoma, pineoblastoma, retinoblastoma, ependymoma, lymphoma, and others [2, 3].

Clinically, patients with meningeal tumor may present with protean signs and symptoms [4]. Definitive diagnosis of this condition relies heavily on the demonstration of malignant cells in the CSF. Unfortunately, patients with leptomeningeal metastasis may have negative CSF cytologic examinations [4]. In order to minimize false negative results, repeated CSF sampling is required. Additionally, false positives can result if inflammatory cells are mistaken for tumor cells [4].

Radiologic methods for the detection of spinal and intracranial subarachnoid tumor deposits have traditionally included myelography and CT [4–7]. Recently, the efficacy of MR imaging has been evaluated in patients with meningeal tumor spread. However, leptomeningeal metastatic disease may not be apparent on routine T1- and T2-weighted spin-echo sequences [7]. In this article we demonstrate the efficacy of Gd-DTPA–enhanced MR imaging in evaluating three patients with leptomeningeal disease from primary intracranial lesions.

Subjects and Methods

From August 1988 to January 1989, three children with pathologically proved primary CNS neoplasms were evaluated with contrast-enhanced CT and with MR with and without IV Gd-DTPA. Two patients (a 6-year-old girl, case 1; and a 6-year-old boy, case 3) had medulloblastoma and one patient (a 4-year-old girl, case 2) had pineoblastoma. The presence of leptomeningeal neoplasm was established by positive CSF cytopathology. MR imaging was performed to better define known lesions or to identify additional lesions.

In all patients, axial CT scans were obtained with a GE 9800 scanner and a slice thickness of 5 mm. Contrast enhancement was obtained by using a split bolus-drip IV injection of

Received May 2, 1989; revision requested July 25, 1989; revision received September 7, 1989; accepted September 21, 1989.

[1] Department of Radiology, Box 3808, Duke University Medical Center, Durham, NC 27710. Address reprint requests to D. J. Rippe.

[2] Department of Pediatrics, Duke University Medical Center, Durham, NC 27710.

[3] Department of Surgery (Neurosurgery), Duke University Medical Center, Durham, NC 27710.

[4] Department of Medicine (Neurology), Duke University Medical Center, Durham, NC 27710.

[5] Department of Pathology, Duke University Medical Center, Durham, NC 27710.

0195–6108/90/1102–0329

Isovue-300 (Squibb) at a dosage of 2 ml/kg (150 ml maximum). To better compare CT and MR studies, examinations performed closest in time were selected.

All MR imaging was performed with a superconducting magnet (GE Signa) operating at 1.5 T. For each patient, axial spin-echo T1-weighted images, 500/20/1 (TR/TE/excitations), and multiecho cardiac-gated T2-weighted images, 2400-2667/30,80/0.75, were acquired. Additional images in the coronal or sagittal planes were obtained as needed. Slice thickness ranged from 5 to 7 mm. For Gd-enhanced imaging, an IV dosage of 0.1 mmol/kg (0.2 ml/kg) gadopentetate dimeglumine (Berlex) was used. T1-weighted images were acquired immediately after the IV Gd-DTPA dose. When patients required sedation for MR, IV Nembutol (4–6 mg/kg) was used.

A retrospective comparison between nonenhanced MR, Gd-MR, and contrast-enhanced CT was performed. In particular, the appearance of the subarachnoid spaces including sulci and basilar cisterns was noted.

Results

Contrast-enhanced CT, MR, and Gd-MR were obtained in all patients. CT and Gd-MR examinations correlated well in patients 2 and 3. Patient 2 had evidence of cisternal disease on both contrast-enhanced CT (Fig. 1A) and Gd-DTPA–enhanced MR (Fig. 1B). Widespread cisternal and sulcal leptomeningeal involvement was documented in patient 3 on both studies (Fig. 2). Posterior fossa enhancement was more apparent with Gd-MR owing to the lack of beam-hardening artifact seen on CT. Specifically, leptomeningeal involvement within the internal auditory canal bilaterally was apparent on Gd-MR but not appreciated with contrast-enhanced CT (Figs. 2A and 2B).

There were disparate results between contrast-enhanced CT and Gd-MR examinations in patient 1. In this case, equivocal cisternal involvement on contrast CT was confirmed by definite abnormal enhancement on the Gd-MR examination (Figs. 3A and 3B). Coronal images were especially useful. In addition, cerebellar hemispheric sulcal enhancement present on Gd-MR was not seen on contrast-enhanced CT (Fig. 3C).

In no case did noncontrast MR reveal evidence of subarachnoid disease. CSF spaces involved with tumor were indistinguishable from uninvolved areas on all spin-echo sequences. All patients had previously placed ventriculoperitoneal shunts, and ventriculomegaly was not a prominent feature in our patients.

Discussion

Leptomeningeal tumor spread has been reported with a variety of primary intracranial lesions. The most common are medulloblastoma, pinealoma, retinoblastoma, lymphoma, and

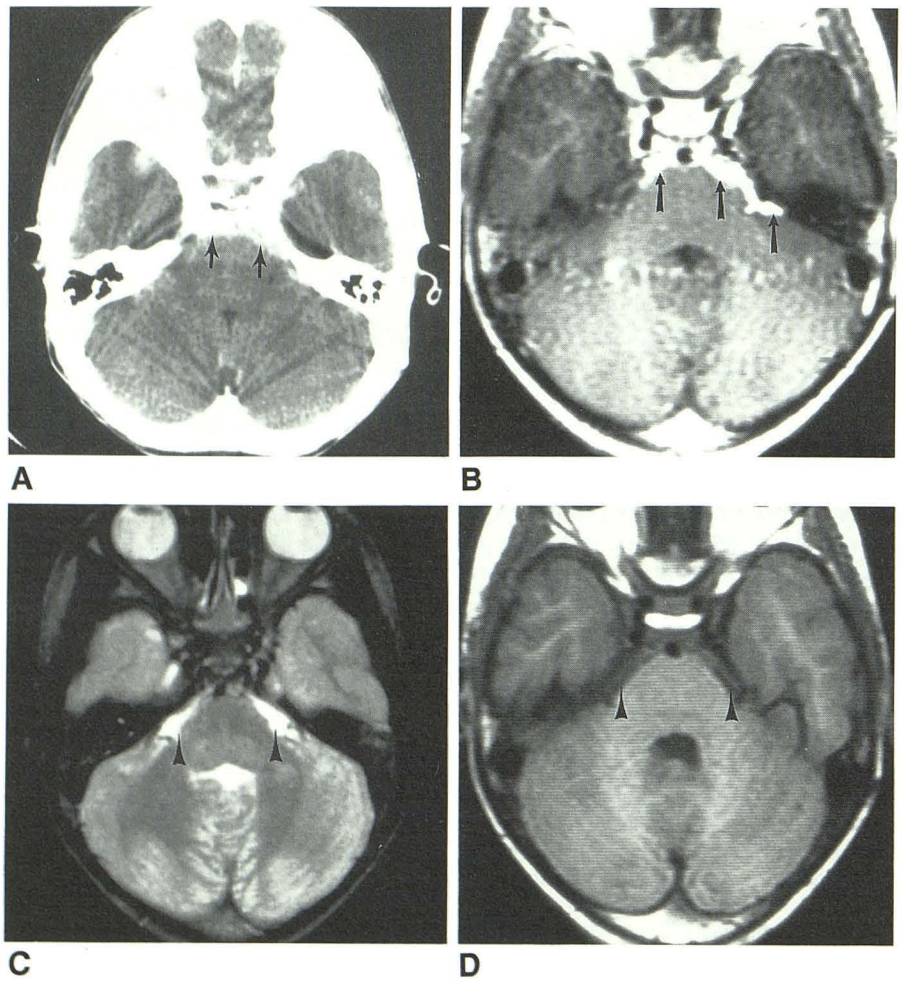

Fig. 1.—Case 2: 4-year-old girl with pineoblastoma.

***A*, Axial contrast-enhanced CT scan shows abnormal density anterior to pons (*arrows*) consistent with leptomeningeal disease. Proximity to bone partly obscures region of interest.**

***B*, Axial Gd-DTPA–enhanced T1-weighted (500/20) MR image demonstrates abnormally enhancing tissue (*arrows*) anterior to pons, extending to left cerebellopontine angle. Lesion is more apparent here than on contrast-enhanced CT.**

***C*, Axial T2-weighted (2000/80) MR image shows leptomeningeal metastasis anterior to pons, blending imperceptibly with adjacent CSF (*arrowheads*).**

***D*, Axial nonenhanced T1-weighted (500/20) MR image shows that leptomeningeal metastases (*arrowheads*) are not appreciated on nonenhanced MR image.**

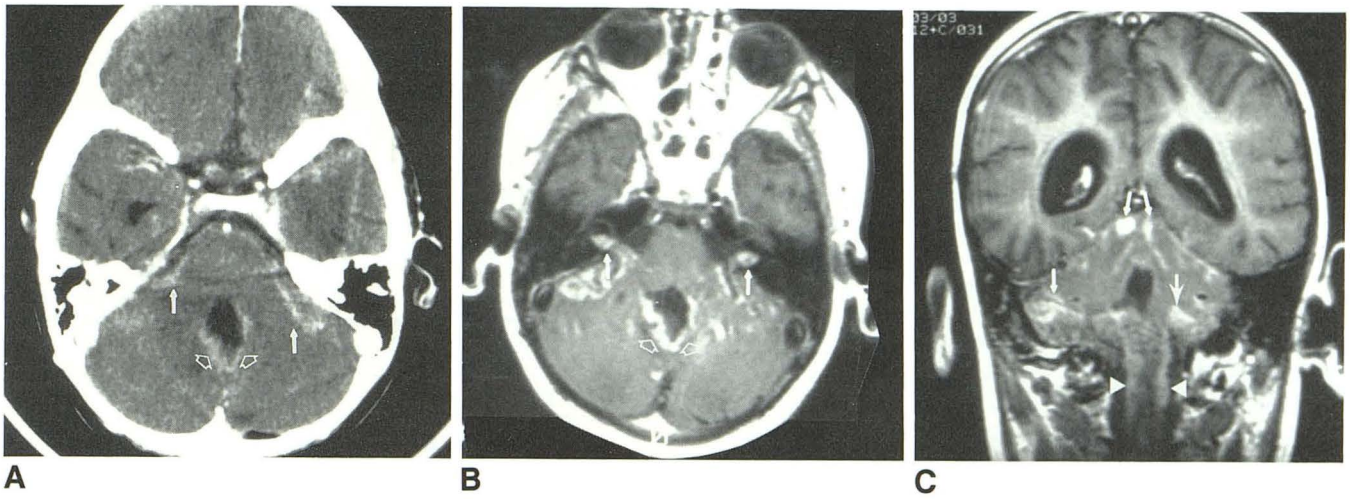

Fig. 2.—Case 3: 6-year-old boy with medulloblastoma.
A, Axial contrast-enhanced CT scan shows abnormal enhancement within fourth ventricle and over pontine surface (*arrows*).
B, Axial Gd-DTPA–enhanced MR image reveals corresponding enhancement within fourth ventricle (*open arrows*). Involvement of internal auditory canals is apparent (*solid arrows*).
C, Coronal Gd-DTPA–enhanced T1-weighted (500/20) MR image shows abnormal sulcal and cisternal enhancement (*arrows*). Note extension of leptomeningeal disease into spinal canal (*arrowheads*).

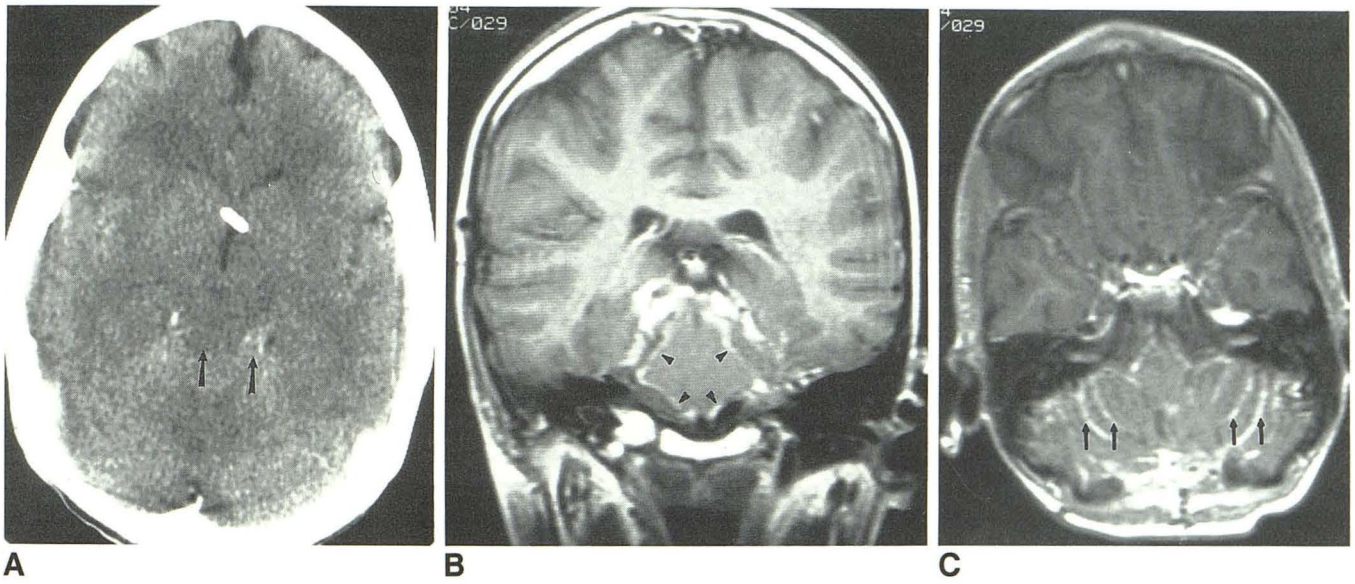

Fig. 3.—Case 1: 6-year-old girl with medulloblastoma.
A, Axial contrast-enhanced CT scan shows indistinct quadrigeminal plate cistern (*arrows*). No definite abnormal enhancement was seen. Shunt catheter is near foramen of Monro.
B, Coronal Gd-DTPA–enhanced T1-weighted (500/20) MR image shows abnormally enhancing tissue along surface of brainstem (*arrowheads*).
C, Axial Gd-DTPA–enhanced T1-weighted (500/20) MR image shows abnormal enhancement of cerebellar folia (*arrows*) representing leptomeningeal spread of tumor. No enhancement of cerebellar folia was detected on CT.

ependymoma [2, 3]. Primary gliomas of the optic chiasm, corpus callosum, and brainstem, as well as oligodendrogliomas, can also produce metastatic leptomeningeal involvement [3]. Tumor spread may occur as a result of CSF seeding from a primary intraventricular neoplasm or from direct extension of parenchymal tumor through the pia or ependyma. Interestingly, the gross appearance of brains involved with diffuse leptomeningeal disease can be normal because there may be only a very thin layer of neoplastic cells lining the subarachnoid space [3, 8].

The diagnosis of meningeal spread of tumor is made by positive CSF cytopathology. Unfortunately, only 15 to 60 percent of patients with leptomeningeal metastases have positive cytologic findings [3, 9]. Failure to find malignant cells in the CSF does not exclude the diagnosis of leptomeningeal disease when this is suspected on clinical grounds [4]. Other

CSF abnormalities, including elevated protein, mononuclear pleocytosis, and hypoglycorrhacia, are nonspecific [4].

Previously, the radiologic demonstration of intracranial leptomeningeal tumor has relied heavily upon CT [2, 3, 5, 9, 10]. Characteristic CT findings include sulcal and basilar cistern obliteration with enhancement, ependymal-subependymal enhancement, gyral enhancement without subcortical edema, and ventricular dilatation [2, 3, 5]. A single CT scan is, however, relatively insensitive in detecting meningeal spread [5, 6]. Some reports suggest improved results with high-resolution CT (5-mm-thick cuts) through the posterior fossa [9], or with serial CT scanning [10]. It should be noted that sulcal or cisternal enhancement is nonspecific. These findings may be seen in cases with prior subarachnoid hemorrhage or in granulomatous meningeal disease [2, 9].

MR has been able to identify spinal subarachnoid tumor deposition although noncontrast MR is rather insensitive in detecting this process [7, 11]. Relaxation characteristics of leptomeningeal tumor are near those of surrounding CSF. Recently, the value of Gd-DTPA–enhanced MR imaging in detecting spinal leptomeningeal seeding has been described [8, 12]. Additionally, it has been suggested that Gd-MR may have a role in the detection of intracranial leptomeningeal tumor spread from primary CNS neoplasms [13].

Our limited study further substantiates the value of Gd-MR in the detection of intracranial meningeal tumor spread. Findings correlated well with those on contrast-enhanced CT. In two cases (patients 1 and 3) Gd-MR imaging resulted in improved visualization of subarachnoid disease. This was particularly so within the posterior fossa—a favored site of intracranial involvement. In all three cases, Gd-MR revealed lesions that were not identified on noncontrast MR. Although further evaluation is necessary, we support the use of Gd-MR when searching for intracranial leptomeningeal deposits in pediatric patients at risk for this condition.

ACKNOWLEDGMENT

The authors gratefully acknowledge Helen Bass for her assistance with manuscript preparation.

REFERENCES

1. Sorensen S, Eagan RT, Scott M. Meningeal carcinomatosis in patients with primary breast or lung cancer. *Mayo Clin Proc* **1984**;59:91–94
2. Lee Y, Glass JP, Geoffray A, Wallace S. Cranial computed tomographic abnormalities in leptomeningeal metastasis. *AJNR* **1984**;5:559–563
3. Ascherl GF, Sadek HK, Brisman R. Computed tomography of disseminated meningeal and ependymal malignant neoplasms. *Neuroradiology* **1981**;31:567–574
4. Twijnstra A, Ongerboer de Visser BW, van Zanten AP. Diagnosis of leptomeningeal metastasis. *Clin Neurol Neurosurg* **1987**;89:79–84
5. Umeo I, Tomita H, Yamazaki S, Takada Y, Inaba Y. CT findings of leptomeningeal and periventricular dissemination of tumors. *Clin Neurol Neurosurg* **1986**;88:115–120
6. Enzmann DR, Krikorian J, Yorke C, Hayward R. Computed tomography in leptomeningeal spread of tumor. *J Comput Assist Tomogr* **1978**;2:448–455
7. Krol G, Sze G, Malkin M, Walker R. MR of cranial and spinal meningeal carcinomatosis: comparison with CT and myelography. *AJNR* **1988**;9:709–714
8. Sze G, Abramson A, Krol G, et al. Gadolinium-DTPA in the evaluation of intradural extramedullary spinal disease. *AJNR* **1988**;9:153–163, *AJR* **1988**;150:911–921
9. North C, Segall HD, Stanley P, Zee CS, Ahmadi J, McComb JG. Early CT detection of intracranial seeding from medulloblastoma. *AJNR* **1985**;6:11–13
10. Jaeckle KA, Krol G, Posner JB. Evolution of computed tomographic abnormalities in leptomeningeal metastases. *Ann Neurol* **1985**;17:85–89
11. Barloon TJ, Yuh WT, Yang CC, Shultz DH. Spinal subarachnoid tumor seeding from intracranial metastasis: MR findings. *J Comput Assist Tomogr* **1987**;11:242–244
12. Berns DH, Blaser S, Ross JS, Masaryk TJ, Modic MT. MR imaging with Gd-DTPA in leptomeningeal spread of lymphoma. *J Comput Assist Tomogr* **1988**;12:499–500
13. Powers TA, Partain CL, Kessler RM, et al. Central nervous system lesions in pediatric patients: Gd-DTPA–enhanced MR imaging. *Radiology* **1988**;169:723–726