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Gadopentetate Dimeglumine-Enhanced MR Imaging in Children Following Surgery for Brain Tumor: Spectrum of Meningeal Findings

Gadopentetate dimeglumine-enhanced MR imaging was performed in 51 consecutive postoperative pediatric neurosurgical patients with a diagnosis of brain tumor. These studies were examined retrospectively to determine the spectrum of meningeal findings in this patient population. Patterns of enhancement were correlated with type of surgery, interval since surgery, clinical and CSF findings, and the use of radiation and steroid therapies. Normal postoperative meningeal findings include no meningeal enhancement or mild focal or diffuse dural enhancement. More moderate dural or subdural enhancement may be seen in clinically well children who have postsurgical subdural collections, or who have a remote history of serious meningeal disease (meningitis or subarachnoid hemorrhage). In all six cases in which nodular dural, leptomeningeal, or ependymal enhancement was seen, recurrent local tumor, leptomeningeal metastases, or infection were present. Leptomeningeal tumor or infection should be suspected if such patterns of enhancement are noted. Parameters that did not appear to affect the pattern of meningeal enhancement included type of surgery, interval since surgery, or therapeutic radiation.

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MR is essential for initial and follow-up imaging of pediatric patients with intracranial lesions. Advantages of MR include superior soft-tissue contrast, multiplanar capability, and absence of beam-hardening artifacts in the posterior fossa [1-4]. Gadopentetate dimeglumine has proved to be a useful adjunct for demonstration of lesions that disrupt the blood-brain barrier, for extraaxial lesions, and for leptomeningeal diseases [5-12].

Recent reports describe meningeal enhancement with gadopentetate dimeglumine following a wide variety of CNS surgical procedures, primarily in adults [10, 13-15]. In children, the expected postoperative enhancement patterns, the time course of this enhancement, and the relationship of this enhancement to radiation therapy have not been reported. Children with brain tumors are a unique population and are different from adults: they more commonly have lesions located in the posterior fossa, neurosurgical intervention frequently involves both tumor biopsy or resection and placement of ventricular shunts, and leptomeningeal spread of tumor is a common complication.

In this series, patterns of meningeal enhancement on MR in 31 children with known brain tumors and prior cranial surgical procedures were correlated with the interval and type of surgery, clinical and CSF evaluations, and other therapeutic treatments used (radiation and steroid therapy). From these data we attempted to identify the spectrum of findings associated with normal patterns of postoperative meningeal enhancement. Analysis of nine cases of documented meningeal disease revealed pattern differences that suggest tumor or infection in the postoperative child.
Materials and Methods

Over a 10-month period, from September 1988 to July 1989, 58 consecutive contrast-enhanced brain MR examinations were performed in a total of 38 pediatric patients with CNS neoplasia. Seven patients were excluded from the study owing to absence of recognizable enhancement of normal structures on immediate postenhancement sequences, indicating that these patients did not receive contrast medium. The study group then consisted of 31 patients who underwent 51 postoperative enhanced MR examinations. The population comprised 18 boys and 13 girls aged 11 months to 18 years (mean, 9 years 5 months). Nineteen patients had one postoperative enhanced MR study and 12 had two or more follow-up enhanced MR studies. Twenty scans were obtained after ventricular shunt placement and craniotomy, 12 after CT-guided biopsy and shunt placement, 10 after cranioectomy only, six after ventricular shunt placement, and three after CT-guided biopsy. Enhanced MR was performed to define a known lesion or to evaluate the efficacy of surgical intervention. Clinical findings (neurologic status; interval since surgery, radiation, and chemotherapy; and other significant CNS illnesses including meningitis, hemorrhage, and shunt malfunctions) and CSF analyses (29/31) were studied by chart review.

Thirty of the 31 patients had histologic or cytologic confirmation of a CNS neoplasm. One child without histologic proof had a presumed germ cell tumor of the pineal region on the basis of MR intensities suggesting fat. Neoplasms are listed in Table 1.

MR was performed with a 1.5-T superconducting magnet (Phillips Medical Systems, Shelton, CT) operating at 0.5 T (17 scans) or 1.5 T (34 scans). All studies were performed with a head coil and a spin-echo technique. All patients had an initial localizing sagittal sequence, 400–800/20, 30/1 (TR/TE/excitations), at 5-mm slice thickness, followed by axial T2-weighted images, 1900–2200/50–100. Additional unenhanced short TR sequences (400–800/20, 30/1) in either the coronal or axial plane were completed in 28 of 31 patients. After obtaining written informed consent, gadopentetate dimeglumin (Magnevist, Berlex Laboratories, Wayne, NJ) was given in a dose of 0.1 mmol/kg IV. At least one immediate (beginning within 5 min of contrast administration) short TR sequence was completed in all patients, with additional enhanced sequences in 20 of 31 patients. Imaging planes were chosen for optimal demonstration of the primary lesion.

The unenhanced and enhanced short TR sequences were compared retrospectively by two experienced neuroradiologists. Five meningeal characteristics were evaluated: (1) degree of enhancement (mild, moderate, or marked); (2) distribution of the enhancement (diffuse or focal); (3) if focal, the proximity of the enhancement to the surgical site; (4) location of enhancement (dural, pia-arachnoid, or ependymal); and (5) nodularity of enhancement.

Results

The distribution and location of meningeal enhancement patterns, mean interval since surgery, child’s clinical status, existence of significant leptomeningeal disease, and history of radiation therapy are noted in Table 2. Thirty-eight of 51 scans revealed either no meningeal enhancement or only mild dural enhancement, either diffuse or focal at the surgical site (Fig. 1). Detailed findings on the 13 of 51 scans with more extensive enhancement are given in Table 3. Four of the 13 scans (cases 1, 2 [scan 1], 3, and 4) showed bilateral postoperative collections in the subdural space (Fig. 2). These children were clinically well. Two of the 13 with more extensive enhancement (case 2 [scans 2 and 3]) were seen in a patient with chemical meningitis based on clinical and CSF analysis (Fig. 3). This child had a cystic craniopharyngioma and the meningitis was attributed to subarachnoid contamination by intracystic contents. This patient was scanned during acute illness and again 3 months later when the illness had resolved. Both scans showed moderate, smooth, diffuse dural enhancement. One patient (case 5) who had a craniotomy and radia-
A 14-year-old child 4 years after craniotomy and shunt placement for craniopharyngioma, now clinically well.

A, Coronal unenhanced short-TR MR image (600/30) shows ferromagnetic artifacts at craniotomy site (arrows).

B, Enhanced short-TR MR image (600/30) reveals thin, symmetric dural enhancement over both convexities and along interhemispheric fissure (white arrows). If unenhanced study had not been performed, ferromagnetic artifacts at craniotomy site (black arrows) might have been mistaken for meningeal enhancement.

**TABLE 3: Abnormal Patterns of Meningeal Enhancement**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Tumor Detected</th>
<th>Scan No. (Interval Since Previous Study)</th>
<th>Pattern of Enhancement</th>
<th>Reason for Enhancement Pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Craniopharyngioma</td>
<td>1</td>
<td>Moderate: dural, biconvexity</td>
<td>Postcraniotomy SDH; clinically well</td>
</tr>
<tr>
<td>2</td>
<td>Craniopharyngioma</td>
<td>1</td>
<td>Moderate: dural, biconvexity</td>
<td>Postcraniotomy SDH; clinically well</td>
</tr>
<tr>
<td>3</td>
<td>Hypothalamic astrocytoma</td>
<td>1</td>
<td>Moderate: dural, biconvexity</td>
<td>Chemical meningitis based on CSF/clinical findings</td>
</tr>
<tr>
<td>4</td>
<td>Pineal region</td>
<td>1</td>
<td>Moderate: dural, tentorial</td>
<td>Postshunt SDH; clinically well</td>
</tr>
<tr>
<td>5</td>
<td>Ependymoma</td>
<td>1</td>
<td>Moderate: dural, preoptic area</td>
<td>Known subtotal resection (presumed residual tumor); clinically well</td>
</tr>
<tr>
<td>6</td>
<td>Brainstem astrocytoma</td>
<td>1</td>
<td>Nodular: dural, CP angle</td>
<td>S/P hyperfractionation therapy; CSF normal</td>
</tr>
<tr>
<td>7</td>
<td>Medulloblastoma (CP angle)</td>
<td>1</td>
<td>Nodular: dural, CP angle</td>
<td>Subtotal resection (presumed residual tumor)</td>
</tr>
<tr>
<td>8</td>
<td>Pineoblastoma</td>
<td>1</td>
<td>Marked: leptomeningeal; nodular: suprasellar, preoptic, pineal region</td>
<td>Clinically well</td>
</tr>
<tr>
<td>9</td>
<td>Cerebellar astrocytoma</td>
<td>1</td>
<td>Intraventricular</td>
<td>Aspergillus ventriculitis &amp; mycotic aneurysm</td>
</tr>
</tbody>
</table>

Note.—mo = month(s); yr = year(s); SDH = subdural hematoma; S/P = status post (after); SAH = subarachnoid hemorrhage; CP = cerebellopontine.

ton therapy for an ependymoma and had undergone multiple shunt revisions experienced a subarachnoid hemorrhage of uncertain origin. The MR examination showed moderate dural enhancement, although the patient was clinically well at that time. One patient (case 6 [scan 1]) had undergone subtotal resection of a brainstem glioma; enhanced MR showed a residual tumor nodule in the cerebellopontine angle, although the patient was clinically well. The patient then underwent hyperfractionation therapy, and a sequential enhanced MR study 2 months later (scan 2) revealed progression of enhancement. This focal enhancement was in the preoptic cistern, and precise localization to a specific meningeal layer was not possible. The patient’s only symptom was recurrent fever; he was otherwise clinically well and CSF was negative. In case 7, two scans showed focal nodular enhancement in the cerebellopontine angle; this represented residual tumor (Fig. 4). The patient had undergone subtotal resection of a medulloblastoma and was otherwise clinically well. One patient (case 8) had marked nodular leptomeningeal enhancement remote from the primary tumor site consistent with leptomeningeal metastases; CSF cytology at that time revealed metastatic pineoblastoma (Fig. 5). One patient (case
Fig. 2.—6-year-old child 2 weeks after biventricular shunt placement for hydrocephalus resulting from hypothalamic astrocytoma.
A, Coronal unenhanced short-TR MR image (600/30) shows large bilateral low-intensity subdural collections that developed after shunt placement.
B, Enhanced short-TR MR image (600/30) shows symmetric thin rim of dural enhancement along periphery of subdural effusions. The child is well, without meningeal symptoms.

Fig. 3.—3-year-old girl with cystic craniopharyngioma.
A, Enhanced short-TR MR image (600/30) 1 month after biventricular shunt placement and frontal craniotomy reveals moderate bifrontal and interhemispheric dural enhancement. CT scan confirmed small bilateral subdural effusions. The child was clinically well.
B, Enhanced short-TR MR image (800/20) 4 months later reveals more extensive and prominent dural enhancement. CSF and clinical findings confirm chemical meningitis, probably resulting from spill of cystic tumor contents.

Fig. 4.—14-year-old boy 3 months after craniectomy for subtotal resection of medulloblastoma. Enhanced short-TR MR image (800/20). Nodular enhancement in right cerebellopontine angle represents known residual tumor.

Fig. 5.—4-year-old girl after ventricular shunt placement and CT-guided biopsy of pineal region tumor (pineoblastoma). Sagittal enhanced short-TR MR image (500/20) shows abnormal enhancement along ventral medulla. At this time, CSF was positive for tumor cells.
A 4-year-old boy 2 months after suboccipital craniectomy for gross total resection of cerebellar astrocytoma. 

A, Axial unenhanced short-TR MR image (550/30) shows mild asymmetry of frontal horns; it is otherwise normal.

B, Enhanced short-TR MR image (550/30) reveals ependymal enhancement in frontal horn of left lateral ventricle. CSF culture revealed Aspergillus; the patient subsequently died of complications of a mycotic aneurysm.
of ionizing radiation, recent reports suggest that contrast-
enhanced MR is more sensitive than contrast-enhanced CT
in revealing and characterizing meningeal lesions [10]. Particu-
larly because of beam-hardening artifacts, CT is poor in
evaluating meningeal abnormalities at the base of the skull
and inner table of the calvaria, regions visualized discreetly
on MR. Furthermore, while breakdown of the blood-brain
barrier is a requirement for abnormal enhancement on both
CT and MR, it has been suggested that enhanced MR may
be more sensitive than enhanced CT and may reveal the
breakdown at an earlier stage [8].

The blood-brain barrier is a physiologically complex
structure. Simplistically, it comprises cerebral capillaries with tight
junctions, the function of which is to prevent the free passage
of molecules into the brain tissue [16]. The meninges, com-
prising the dura, the arachnoid, and the pia, are a significant
site of blood-brain barrier. The dura itself is devoid of blood-
brain barrier; capillary junctions within the dura are not tight
and, therefore, form no barrier to prevent the free passage
of molecules. One would anticipate that since it is devoid of a
blood-brain barrier, the dura would enhance intensely and
predictably following the administration of IV contrast mate-
rial. However, it is relatively avascular, and the second require-
ment for contrast enhancement, an adequate delivery of
contrast material via arteries and capillaries, is not met. This
is the explanation for the variable enhancement pattern of the
dura on both CT and MR. Both layers of the arachnoid and
the pia have tight junctions within the capillaries, and, there-
fore, have a functional blood-brain barrier. For this reason,
and despite the relative vascularity of the arachnoid and pia,
enhancement within the arachnoid and the pia is not seen
normally following contrast administration for either MR or
CT. If arachnoidial or pial enhancement is seen, leptomeninge-
al disease should be suspected.

A wide variation in gadopentetate dimeglumine enhance-
ment patterns of the dura in postoperative patients has been
described [10, 13, 14]. As the enhancement pattern in the
intact dura is variable [17], it is not surprising that the traum-
atized dura might show a variety of enhancement patterns.
If this spectrum of normal postoperative enhancement pat-
terns is familiar, recognition of pathologic meningeal enhance-
ment is facilitated.

The ability to detect dural and leptomeningeal lesions is of
particular importance in children with intracranial neoplasms,
as a number of the common pediatric tumors (medulloblas-
toma, ependymoma, pineal region tumors, and anaplastic
astrocytomas) may metastasize to the leptomeninges via the
CSF. While enhanced MR is more sensitive than enhanced
CT in detecting leptomeningeal enhancement, it is no more
specific. Criteria for differentiation between insignificant (i.e.,
postoperative) and significant (i.e., neoplastic, inflammatory)
meningeal enhancement have not been established for the
pediatric population. The goal of this article is to describe
the normal appearances of the dura and leptomeninges in the
postoperative neurosurgical pediatric patient with an intracra-
nial neoplasm.

Common postoperative findings in clinically well patients
varied from no dural enhancement to smooth, thin dural
enhancement, either immediately at the operative site or more
diffusely over the convexities. These findings may persist for
prolonged periods (e.g., 10 years) postoperatively. A history
of prior therapeutic radiation appeared to have no recogniz-
able relationship to the presence or degree of enhancement.
Similarly, the specific type of neurosurgical procedure per-
formed had no relationship to the presence or degree of
enhancement. In all cases in this study, therefore, the pres-
ence of mild, smooth dural enhancement, either focal or
diffuse, implied benign postoperative meningeal changes.

A common cause of moderate dural or subdural enhance-
ment is a subdural postoperative collection, usually nonhem-
orrhagic. Our experience was similar to that of other inves-
tigators in that the enhancement occurred in the peripheral
portion of the collection [10]. During the organization phase
of a subdural collection, numerous capillaries permeate the
outer aspect of the hematoma or hygroma, forming a mem-
brane that is relatively vascular, especially when compared
with the inner avascular membrane on the arachnoidal side
of the clot [18]. This explains the different degree of enhance-
ment within a subdural collection.

Other causes of moderate dural enhancement in our series
included remote subarachnoid hemorrhage and acute or re-
13, 14]. As the enhancement pattern in the

clude remote subarachnoid hemorrhage and acute or re-

current meningitis. Interestingly, enhancement in one

mote enhancement was demon-

strated. Although we noted only two patients with patterns
of ependymal or leptomeningeal enhancement, both had sig-
nificant disease. Even after intraventricular shunt placement,
no ependymal enhancement was seen on 38 scans obtained
after the procedure. On the basis of this limited experience,
we believe that leptomeningeal or ependymal enhancement
should suggest recurrent tumor or infection.

Nodular dural or subdural enhancement was not a pattern
seen on scans of clinically well children. Four scans in our
series showed nodular dural enhancement; in all cases this
represented recurrent or residual exophytic tumor.

Radiation therapy may result in blood-brain barrier break-
down, but at clinical doses this effect may be delayed [19].
This may explain why no increase in meningeal enhancement
was apparent in the subset of patients scanned soon after
receiving radiation therapy. Additionally, steroid therapy sta-
bilizes the blood-brain barrier [16, 20–23] and is often admin-
istered during part or all of the radiation course. This may be
a second explanation for the lack of an increased prevalence
of enhancement following radiation. Although in theory radia-
tion may result in delayed blood-brain barrier breakdown, 14
of 29 patients scanned 1 year or more after radiation therapy
had no evidence of blood-brain barrier breakdown. Thus,
radiation-induced disruption of the blood-brain barrier in the
relatively avascular meninges is probably subclinical and be-
low the threshold of detection with enhanced MR. Abnormal
meningeal enhancement should not be ascribed to radiation therapy until other causes have been excluded.

No patient in this series with normal dura, pia, arachnoid, or ependyma on enhanced MR had evidence of leptomeningeal or ependymal disease based on clinical or laboratory data. However, we do not propose that a normal enhanced MR examination absolutely excludes meningeal disease. Other investigators have described normal enhanced MR scans of the brain and spine in patients with cytologically proved leptomeningeal disease; presumably this could also occur intracranially with either neoplasia or infection [10] (Yousem DM, Grossman RI, presented at the annual meeting of the Radiological Society of North America, November 1989). Rather, the appearance of the dura, leptomeninges, and ependyma on enhanced MR scans complements the clinical examination and CSF findings.

Our current technique in this patient population includes a T2-weighted axial sequence and an unenhanced T1-weighted sequence through the surgical site and prior tumor bed. We then repeat the T1-weighted sequence immediately after the administration of contrast material. Most patients undergo at least one additional T1-weighted sequence in another plane. If a patient has a tumor with a known propensity for metastasis to the leptomeninges, the entire brain is scanned after enhancement.

Conclusions

Mild focal or diffuse dural enhancement is a normal finding on enhanced MR in the pediatric patient who has undergone biopsy, craniotomy, or intraventricular shunt placement for an intracranial neoplasm or associated hydrocephalus. Benign appearing meningeal enhancement was noted on some scans obtained 8 or more years after surgery. Prior radiation therapy did not appear to increase the prevalence or degree of meningeal enhancement. Moderate dural enhancement may occur in clinically well patients with postsurgical subdural collections or when there is a history of remote serious meningeal disease such as a subarachnoid hemorrhage or meningitis. Nodular dural enhancement, leptomeningeal enhancement, or ependymal enhancement were patterns not seen in our clinically well postsurgical group of patients. On the basis of our small population of patients who exhibited such enhancement, these patterns of enhancement may suggest serious disease, such as leptomeningeal tumor, residual tumor, or meningitis. Finally, enhanced T1-weighted sequences and careful contrast administration techniques are essential for optimal use of enhanced MR in the postoperative pediatric patient.

REFERENCES

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