CLINICAL STUDIES

Surgical Implications of Magnetic Resonance-enhanced Dura

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THE PURPOSE OF this study was to assess tissue changes responsible for dural enhancement on magnetic resonance imaging (MRI) and its clinical implications. A prospective surgical, histopathological, and MRI study was performed in 73 patients with various types of disease, including meningiomas (n = 29), craniofacial tumors with possible direct intracranial extension (n = 21), gliomas and brain metastasis in close proximity to the dura mater (n = 9), and a variety of nonneoplastic processes (n = 14). Contrast-enhanced MRI was obtained within 5 days before surgery and in some cases within 3 days after surgery as well. Histopathological examination of the dural specimens was performed in all 59 patients with neoplasia and in selected patients with nonneoplastic processes. Dural invasion was noted in 18 of 29 meningiomas, 15 of 21 craniofacial neoplasms, 3 of 5 gliomas, and 3 of 4 brain metastases. In these patients invasion was focal and in direct continuity with the tumors. MRI disclosed that dura invaded by the tumor had a break in the continuity of enhancement, or that there was no discernible enhancement. Association between patterns of dural enhancement and tumor invasion of dura was statistically significant ($P < 0.001$). The thickened-enhanced portion of the dura represented reactive changes. Postoperative enhancement was seen as early as 24 hours after surgery and was shown histologically to be associated with vasodilation and reactive changes. Conclusions from this study are: 1) dural enhancement is a nonspecific reaction and may be seen in association with many pathological conditions; 2) a fairly uniform "enhanced dura" adjacent to a tumor correlated with a dural inflammatory reaction, whereas discontinuous enhancing dura indicated dural invasion; 3) a few false-negative cases of dural invasion (one extracranial and four intracranial neoplasms) underscore that there are some limitations of contrast-enhanced MRI in predicting dural invasion by adjacent neoplasms. (Neurosurgery 35:370–377, 1994)

Key words: Gadolinium, Head and neck neoplasms, Intracranial neoplasms, Magnetic resonance, Meninges, Neoplasms

Controversy exists as to whether a "thickened-enhanced dura" adjacent to an intracranial neoplasm represents inflammation (2, 21) or invasion of dura (8, 25). Correctly recognizing dural invasion from that of reactive changes has important surgical implications, in that a decision must be made whether to resect (along with tumor itself) the "enhanced dura" that is contiguous with the neoplasm to prevent or reduce local recurrence (5, 9, 13, 16). To address this issue, we have prospectively studied three groups of patients that included a wide range of diseases.

PATIENTS AND METHODS

We prospectively studied a total of 73 newly diagnosed adult patients 29 to 81 years of age with possible abnormal dural enhancement from a wide variety of causes (*Table 1*). These patients were diagnosed from a total of approximately 4000 contrast-enhanced magnetic resonance (MR) images of brain performed during a 3-year period ending September 1993. Fifty-nine consecutive patients with neoplasia, who underwent surgery, consisted of 21 with craniofacial and calvarial neoplasms with possible intracranial extensions, 29 with intracranial meningiomas, and with 9 gliomas and metastases in close proximity to the dura mater. We also included 14 patients with dural enhancement caused by nonneoplastic processes. These nontumor cases were selected at random. In this group of patients, there was no evidence of associated acute or chronic meningitis, either by MR imaging (MRI) or analysis of cerebrospinal fluids.

Seventy-one patients were imaged with 1.5-T MR imagers (S15 HP, Phillips Medical Systems North America, Shelton, CT,

TABLE 1. Patients (n = 73)

Craniofacial/calvarial neoplasms[a]	21
Squamous cell carcinoma	5
Nasopharyngeal carcinoma	4
Sinonasal sarcoma or carcinoma	3
Esthesioneuroblastoma	2
Metastasis	2
Meningioma (extracranial)	3
Hemangiopericytoma	1
Craniopharyngioma (intrasphenoid)	1
Intracranial neoplasms	38
Meningioma	29
Glioma	5
Metastasis	4
Nonneoplastic processes	14
Postoperative	4
Granulomatous infection	3
Sarcoidosis	2
Superior saggital thrombosis	1
Lymphocytic adenohypophysitis	2
Chronic subdural hematoma	1
Epidural empyema	1

[a] Some of these patients have been previously reported (1).

TABLE 2. Histological Diagnosis in 59 Cases of Neoplasms Assessed for Dural-Arachnoid Invasion

Type of Tumor	No. of Cases	Cases of Dural Invasion
Craniofacial		
Scalp squamous carcinoma	5	3
Nasopharyngeal carcinoma	4	2
Sinonasal sarcomas or carcinoma	3	2
Esthesioneuroblastoma	2	2
Calvarial meningiomas	3	3
Intrasphenoid craniopharyngioma	1	1
Hemangiopericytoma	1	0
Calvarial metastasis	2	2
Intracranial		
Meningioma	29	18
Glioma	5	3
Metastasis	4	3

or Signa Advantage, General Electric Medical Systems, Milwaukee, WI) and two patients with a 0.5-T MR imager (Vista, Picker International, Highland Heights, OH). Contrast-enhanced T1-weighted images (in addition to conventional noncontrast-enhanced spin-echo sequences) were obtained in two or three planes and within 5 days before surgery in all 73 patients included in this study. In addition, in four patients who had minor intracranial procedures through burr holes, additional postoperative MR studies were done within 3 days after surgery. Sections of 3- to 5-mm thickness and 1-mm intersection gaps were acquired within 2 to 18 minutes after intravenous administration of 0.1 mmol/kg of gadopentetate dimeglumine (Magnevist, Berlex Imaging, Wayne, NJ), using repetition times of 600 to 750 msec, echo times of 16 to 20 msec, two signal averages, and a 256 × 204 matrix.

TABLE 3. Correlation of Magnetic Resonance Imaging and Histological Examination of Dura-Arachnoid Adjacent to Neoplasms (n = 59)[a]

Magnetic Resonance Findings	Type of Tumor	Histology of Dura	
		Invaded	Not Invaded
Continuous enhancing dura	Craniofacial	0	4
	Meningioma	0	6
	Glioma	0	1
	Metastases	0	1
Discontinuous enhancing dura	Craniofacial	14	0
	Meningioma	16	0
	Glioma	2	0
	Metastasis	2	0
Nonenhancing dura	Craniofacial	1	2
	Meningioma	2	5
	Glioma	1	1
	Metastasis	1	0
Total		39	20

[a] **Association between tumor invasion and the patterns of dural-enhancement is statistically significant ($\chi^2 = 46.00$, $df = 2$, $P < 0.001$), whereas the association between tumor invasion and dural enhancement is not ($\chi^2 = 5.682$, $df = 2$, $P = 0.058$).**

FIGURE 1. Sinonasal carcinoma with reactive changes in the dura. A sagittal postcontrast T1-weighted MR image shows a long strip of fairly uniform enhancing dura (*arrows*) with no break in the continuity of the enhancing dural band. The neoplasm was abutting the dura (*curved arrow*) but not invading it.

Histopathological examination of dura in all 59 patients with neoplasms and in selected patients with nonneoplastic processes was performed. In 12 patients with neoplasia, the tumors and large segments of dura (3–5 cm beyond the tumor extends) were resected *en-bloc*, and thorough histological examinations of the surgical specimens were performed. In the other 47 patients with neoplasia, multiple dural biopsies were obtained for histopathological examination. For statistical analysis of data the χ^2 test (SigmaStat program, Jandel Scientific, San Rafael, CA) was used, and $P < 0.05$ was considered significant.

RESULTS

Contrast-enhanced MR images were evaluated for possible morphological changes along dura-arachnoid membranes in a wide variety of pathological processes. They included 59 patients with neoplasia and 14 patients with nonneoplastic processes. Prominent dural enhancement is defined as a linear strip of enhancing tissue along the dura mater originating from and/or extending outward from the tumor margin that is observed in at least two different imaging planes.

Neoplasm

From a total of 59 patients with neoplasia assessed for possible dural invasion (*Table 2*), dural enhancement was observed adjacent to and beyond the tumors in 46 patients, and no enhancement of the dura-arachnoids was visualized in 13 patients. Two patterns of abnormal dural-arachnoid enhancement (continuous and discontinuous) were identified (*Table 3*). In 12 patients, the dura was enhanced in continuity and was fairly uniform in thickness. Histopathological examination of the dura-arachnoids revealed only reactive changes without neoplastic invasion (*Fig. 1*). In the other 34 patients with abnormal dural enhancement, there were either areas where the enhancement of the tumors could not be separated from that of the dura or where the enhancing dura appeared discontinuous (*Figs. 2 and 3*). The dura mater in these 34 patients was focally invaded by the adjacent neoplasm. Histopathological examination of the dura from these patients confirmed that the dural invasion corresponded either to a discontinuous segment of the enhancing dura or to the area where the dura could not be distinguished from the adjacent enhancing tumor. Sta-

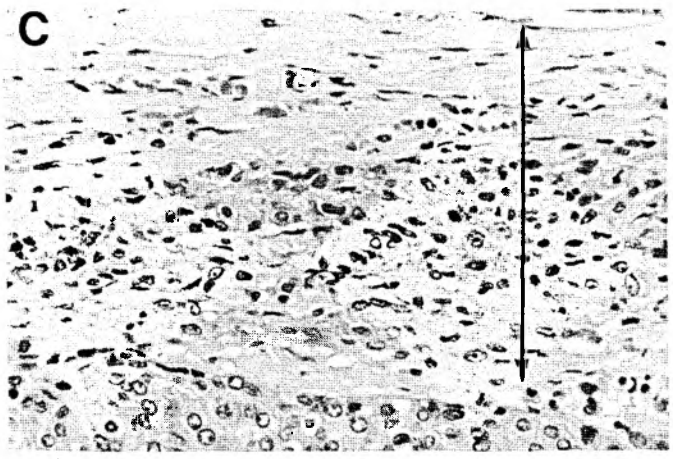

FIGURE 2. Convexity meningioma with focal dural invasion. *A*, postcontrast coronal T1-weighted MR image revealed a thickened enhanced strip of tissue adjacent to and emanating from the meningioma (*arrow*). Medially, a segment of the dura is, however, indistinguishable from the enhancing meningioma that abuts the inner table of the calvarium (*curved arrow*). *B and C*, photomicrographs (hematoxylin and eosin) of a sample (at the site where the dura was not distinguishable from the meningioma) shows an aggressive meningioma with a high proliferation rate and zones of focal necrosis. The tumor shows focal invasion (*arrow*) into the dura, seen at lower original magnification in *B* (×25) and at higher original magnification in *C* (×100). Note the lack of associated inflammation in the dura. The thickness of the dura is shown by the *double-headed arrows;* the infiltrating tumor in the dura is indicated by an *arrowhead. D*, Photomicrograph of the thickened enhancing tissue emanating from the meningioma (*solid straight arrow* in *Fig. 4A*) shows hypervascularization and arachnoidal proliferation but no tumor invasion (hematoxylin and eosin, original magnification ×25).

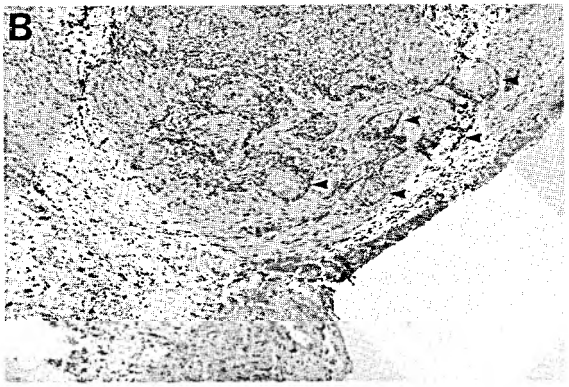

FIGURE 3. Frontal basal squamous carcinoma. *A,* axial postcontrast T1-weighted MR image shows that part of the dura in the left side is indistinguishable from the adjacent enhancing neoplasm (*straight arrows*). This part of the dura was invaded by the adjacent tumor (see *B*). A thickened enhancing strip of tissue is seen extending far away from the tumor along the dura in the opposite hemisphere (*curved arrows*). Histological examination of the latter dural sample revealed only reactive changes (see *C*). *B,* photomicrograph (hematoxylin and eosin, original magnification ×25) of dural specimen (at the site where the dura was indistinguishable from the adjacent neoplasm) demonstrates extensive dural invasion (*arrows*). *C,* photomicrograph of a specimen obtained from an enhancing strip of tissue along the dura (at the site of the *curved arrows* in *A*) shows arachnoidal proliferation (*arrows*) and mild chronic inflammation. No neoplastic cells were identified in this segment (hematoxylin and eosin, original magnification ×350). This meningothelial proliferation can, however, be easily distinguished from basal-squamous cell carcinoma in this case.

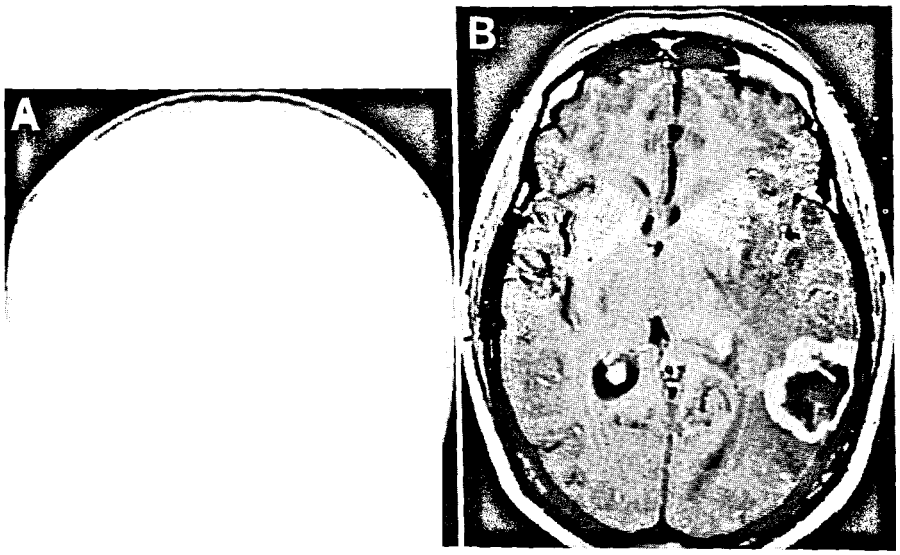

FIGURE 4. Metastatic adenocarcinoma with dural invasion. Coronal (*A*) and axial (*B*) postcontrast T1-weighted MR images show no distinguishable dural enhancement adjacent to or emanating from the tumor. *C,* photomicrograph (hematoxylin and eosin, original magnification ×40) of dural specimen adjacent to neoplasm showing the presence of metastatic adenocarcinoma, with its stroma adherent to the undersurface and infiltrating into the dura (*arrows*). The extent of the dural thickness is indicated by a *double-headed arrow,* and the infiltrating neoplasm is shown by an *arrowhead.*

tistical analysis of the data revealed that the patterns of dural enhancement were significantly associated with dural invasion by tumor ($P < 0.001$), whereas the association between tumor invasion of dura and merely presence or absence of dural enhancement was not statistically significant (P, 0.058).

In the remaining 13 patients contrast-enhanced MRI did not reveal any dural abnormalities, either prospectively or retrospectively. Histopathological examination of the dura, however, demonstrated dural invasion by the adjacent tumor in 5 (*Fig. 4*) and no dural invasion in 8 of these patients. Histopathological examination of the dura from these 13 patients (with no dural enhancement) also revealed minimal or no reactive changes.

Thirty-five of our patients with neoplasia underwent follow-up contrast-enhanced MRI. Thickened-enhanced dura-arachnoids were observed on all postoperative MRI scans, which were more extensive (compared with preoperative MRI scans) and covered much larger areas of the dura far beyond the sites of the craniotomies. Histopathological reexamination of postoperative dura was obtained in five patients who had second operations. Examination of the dura within 2 to 3 cm at the edges of the first operations showed marked reactive dural-arachnoid changes with no evidence of recurrence of tumors at the sites of the initial tumors (*Fig. 5*).

Nonneoplastic processes

Abnormal dural enhancement was observed in four patients, 1 to 3 days after minor surgical procedures such as shunt placements or computed tomography–guided stereotactic cyst punctures (*Fig. 6*). Histopathological examination revealed an acute inflammation of the dura. Dural enhancement was noted in the following cases: 1) in association with a single dural-based sarcoid lesion in two patients (*Fig. 7*); 2) adjacent to intracerebral granulomatous infections in three patients; 3) in association with thrombosis of the superior sagittal sinuses in one patient; 4) in association with lymphocytic adenohypophysitis in two patients; 5) in association with chronic subdural hematoma in one patient; and 6) in association with epidural empyema in one patient. In these groups of patients, there was no evidence of associated acute or chronic meningitis either by MRI or analysis of cerebrospinal fluids.

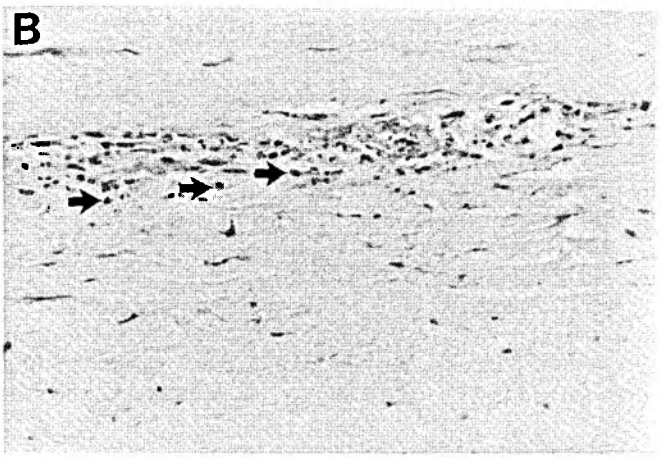

FIGURE 6. Acute postoperative inflammation of the dura. *A*, preoperative contrast-enhanced MR image shows a right ventricular epithelial cyst without dural enhancement (not shown). Computed tomography–guided stereotactic puncture of the cyst through a right parietal burr hole was unsuccessful. A contrast-enhanced MR image obtained 36 hours later shows dural enhancement (*arrows*). B, photomicrograph (hematoxylin and eosin, original magnification ×100) of dural samples obtained the next day at the time of craniotomy reveals presence of polymorphonuclear cells around small vessels extending into the adjacent connective tissue (*arrows*).

FIGURE 5. Biopsy of chronic postoperative enhancing tissue along the dura-arachnoid shows reactive changes consisting of chronic inflammation and neovascularization, which predominately involves the arachnoid membrane. There are no tumor cells present.

DISCUSSION

With the exception of the falx and tentorium, normal dura mater is invisible on routine contrast-enhanced T1-weighted spin-echo images. Enhancement of falx cerebri and tentorium, however, has been observed in 50% of healthy subjects using thin-section MRI (3, 10). A wide variety of pathological conditions may render dura visible on contrast-enhanced MRI (1,

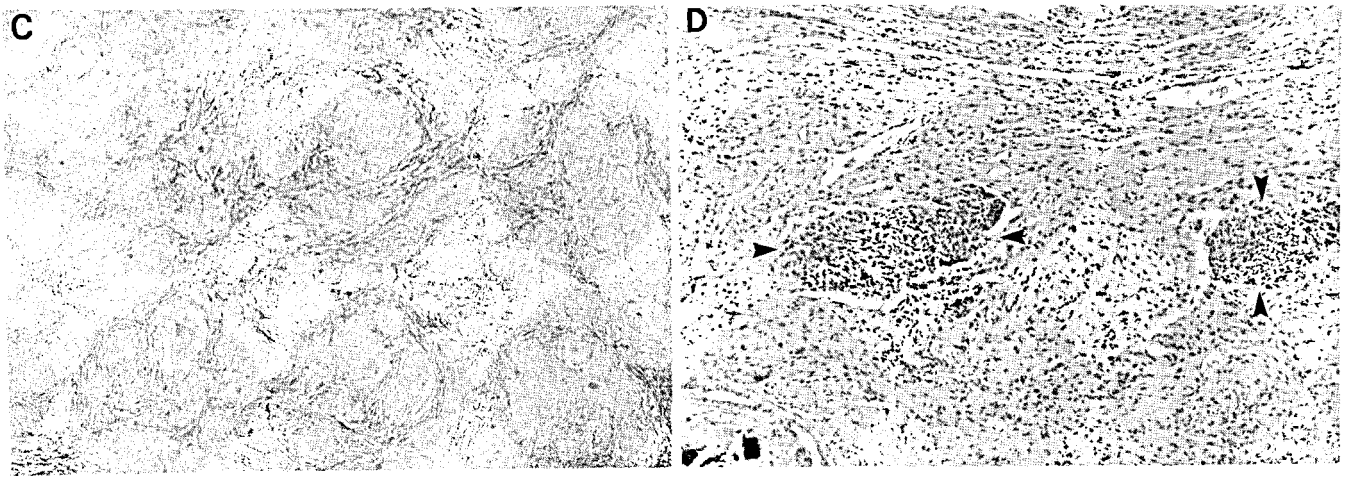

FIGURE 7. High-convexity single dural-based sarcoid lesion. *A*, precontrast coronal MR image shows a dural-based extraaxial soft tissue mass (*curved arrow*). *B*, postcontrast coronal MR image shows enhancement of the sarcoid plaque. In addition, there is a dense thickened enhancing dura-arachnoid seen emanating from the sarcoid plaque and extending over the convexity, as well as into interhemispheric fissure (*straight arrows*). C, photomicrograph of the subdural mass (*curved arrows in A and B*) shows the presence of numerous noncaseating sarcoid granulations septated by connective tissue septa (hematoxylin and eosin, original magnification ×10). D, photomicrograph of thickened enhancing dura-arachnoid membranes (*straight arrows in A and B*) shows arachnoidal proliferation (*arrowhead*) without involvement by the sarcoid (original magnification ×40).

2, 6, 8, 19, 21, 23–25). Any or all three membranes of the meninges (dura mater, arachnoid, and pia mater) may be affected. Absence of abnormal enhancement on MRI within the depth of the sulci and cisterna helps differentiate dural from pial involvement (1, 6, 19). It is important to emphasize that the so-called thickened-enhanced dura (8) is not merely limited to dura mater alone. Histopathological examination of our material revealed that tissue changes responsible for an enhanced band on MRI along the dura mater is composed of proliferation, inflammation, and hypervascularity, which predominately involves the arachnoid membrane rather than dura mater itself. Therefore, for practical purposes in interpretation of MRI, dura/arachnoid may be considered as one unit as opposed to pia/subarachnoid space. Diffuse meningeal enhancement after craniotomy is well known and has been regarded as a reactive change secondary to interruption of the dura (6, 12, 13). Reactive changes can occur acutely within a few days after even minor surgical procedures. Inclusion of several other examples of nonneoplastic pathological conditions in this study further underscores the fact that dural enhancement is a nonspecific reaction.

Neoplastic diseases can involve dura by several mechanisms: 1) craniofacial and calvarial neoplasms may grow intracranially and invade the dura directly; the tumor then may progress further inward, with possible brain parenchymal involvement (1, 15); 2) extracranial neoplasms may extend along foramina and fissures at the cranial base and involve the adjacent dura (7, 11, 13, 17, 20, 22, 23); 3) meningiomas originate from arachnoid cells and may then invade brain parenchyma, dura, or both (4, 5, 14, 15, 18); 4) a primary or metastatic brain neoplasm in close proximity to the surface can invade the adjacent dura (15, 25); and 5) a neoplasm may spread into the meninges (e.g., meningeal carcinomatosis) via a hematogenous route (19). The invasion of dura by a neoplasm is initially focal.

The invading neoplastic tissue may partially or totally replace the dural thickness. The invaded segment of dura may then enhance like the rest of the tumor. Thus, the invaded portion(s) of dura may become indistinguishable from the invading neoplasm (1). At the same time, the noninvaded segment of the dura may become thickened by secondary reactive vascular tissue and may display enhancement after administration of contrast material. Therefore, a dura that is invaded focally and is also associated with secondary reactive changes demonstrates a discontinuous pattern of dural enhancement (34 patients in this report). In contrast, in 12 of the patients in the present study in whom the dura adjacent to the tumors were not invaded at all, but there was histopathological evidence of inflammatory changes, the enhancing dura was continuous. In 13 of our patients the dura adjacent to neoplasms (whether the dura was invaded) did not enhance at all after the administration of contrast material. Interestingly, in these 13 patients there was minimal or no histopathological evidence of associated reactive changes in the dura mater. However, 5 of these 13 patients with nonenhancing dura had histopathological evidence of dural invasion. Patterns of dural enhancement in relationship to tumor invasion of dura was statistically significant. However, the presence or absence of dural enhancement in association with tumor invasion of dura was not statistically significant. These 5 false-negative cases illustrate potential limitations of the use of contrast-enhanced MRI. It had been pointed out in the past that dural enhancement adjacent to intracranial tumors need not imply neoplastic spread (2, 21). Tokumaro et al. (21) correlated MRI and histological examination of dura adjacent to four cases of convexity meningiomas. They have shown that the thickened enhancing dura associated with these meningiomas were caused by hypervascularity and proliferation of connective tissue without histopathological evidence of dural invasion by the tumors. Histopathological examination of postoperative dura in five patients in this study, who had second surgery for tumors at other sites, showed marked reactive dural-arachnoid changes with no evidence of recurrence of tumors at the sites of the initial tumors. Borovich and Doron have documented meningothelial cell aggregates in strips of dura far away from a solitary meningioma. They pointed out that the intradural location of these clusters and their independence from blood vessels negate seedings and dural metastases. The authors concluded that regional multicentricity of meningothelial clusters plays an important role in recurrence of intracranial meningiomas (4). Dural enhancement adjacent to calvarial metastases also have been described previously. However, because dural biopsy was not performed, the authors could not determine whether this dural enhancement was caused by meningeal tumor spread or by benign dural reaction (23).

Based on this study, if the dura adjacent to a neoplasm is seen as a fairly uniform enhancing band on contrast-enhanced MRI, this dura is most likely reactive and not invaded by the tumor. Conversely, the presence of areas of dura indistinguishable from the adjacent tumor, or a focal break in the continuity of enhancing dura adjacent to a neoplasm, is a likely indication of focal dural invasion by the tumor and thus warrants its resection along with the tumor.

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COMMENTS

This is a most interesting report by Ahmadi and colleagues on the diagnostic implications of dural enhancement when seen on preoperative and postoperative magnetic resonance imaging scans. The pattern of enhancement (i.e., continuous versus discontinuous) seems to be of greater import than its mere presence (*Table 3*). Discontinuous enhancement is associated with neoplastic invasion ($P < 0.001$), but the latter can occur in the absence of enhancement. These statistical conclusions are supported by the available histological material (*Figs. 4 and 5*).

Of further interest is the finding that meningeal enhancement in patients with brain tumors is apparently uncommon; 59 cases were seen during a 3-year period in which more than 4000 scans were performed. Unfortunately, we are not told the total number of patients with brain tumors who were scanned and who did not have meningeal enhancement. Additionally, because the presence of postoperative enhancement is especially confusing to the clinician and potentially ominous for the patient, it would have been important for the authors to make direct comparisons of the preoperative and postoperative scans for individual cases in which histologic analyses were also available. Does preoperative enhancement predict postoperative change? Is the incidence of dural invasion increased by surgery? These are just some of the many questions that remain unanswered by this fine contribution.

Michael Salcman
Baltimore, Maryland

This paper helps us understand better the phenomenon of dural enhancement seen so often in association with neoplasia near the dura.

Ahmadi et al. have nicely demonstrated here that dural enhancement with gadolinium is a nonspecific finding with brain tumors. They suggest but do not prove conclusively that "discontinuous enhancement" is a result of tumor, but other enhancement may be inflammation or neoplastic. It is an important contribution to clinical-radiological correlation.

Peter McL. Black
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