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Frequency of Unexplained Meningeal Enhancement in the Brain after Lumbar Puncture

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PURPOSE: To examine the hypothesis that lumbar puncture alone may cause meningeal enhancement in the brain. **METHODS:** We prospectively reviewed the brain MR examinations of all patients from a 6-month period who were studied within 1 month after lumbar puncture. We also retrospectively reviewed all cases of dural-arachnoidal enhancement in the brain from the preceding 18-month period. **RESULTS:** In the prospective group, only one case out of 97 enhanced brain MR examinations after lumbar puncture did not have a clear cause for dural-arachnoidal enhancement. In the retrospective group, only one case out of 11 with enhancement was not clearly explained. **CONCLUSIONS:** Dural-arachnoidal enhancement in the brain after lumbar puncture is uncommon, if it occurs at all, and lumbar puncture as a cause of enhancement should be considered a rare diagnosis of exclusion.

Index terms: Meninges, magnetic resonance; Brain, magnetic resonance; Lumbar puncture; Iatrogenic disease or disorder

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Many causes of diffuse and focal enhancement of the meninges have been reported. Diffuse enhancement may be caused by inflammatory processes such as infectious meningitis (1-3), neurosarcoidosis (4), or neoplastic processes such as carcinomatous meningitis or lymphomatous infiltration of the meninges (3, 5-7). Diffuse enhancement is also seen in many patients after craniotomy (8) and ventricular shunting (9). Other reported causes include venous sinus thrombosis (10-12), subarachnoid hemorrhage (3) (Brown E, DeLaPaz R, Intracranial Meningeal Enhancement with GdDTPA MR, presented at the 27th Annual Meeting of the American Society of Neuroradiology, Washington, DC, 1989), and vasculitis (13). Focal meningeal enhancement may be caused by meningiomas (14, 15), nonmeningiomatic extraaxial lesions, or parenchymal lesions such as

glioblastomas or metastases that are located superficially (16).

Several authors have stated that meningeal enhancement with gadopentetate dimeglumine on magnetic resonance (MR) examinations of the brain may be caused by lumbar puncture (17-19), and another raised the possibility that this may occur (20). If this observation were true, it would presumably be related to the observation that lumbar puncture may lead to intracranial hypotension, which is commonly manifested as postural headache (21-25). In rare reports, lumbar puncture has been associated with intracranial subdural collections (26-31). This has also been hypothesized to be secondary to intracranial hypotension and downward descent of the brain. Alternative hypotheses for the cause of subdural collections after lumbar puncture include vasodilatation caused by decreased cerebrospinal fluid (CSF) volume (32) or diffuse irritation caused by violation of the meninges after lumbar puncture similar to that seen after ventricular shunting. Similar mechanisms have been invoked as the causes of intracranial subdural collections reported after myelography (33, 34), pneumoencephalography (35), spinal anesthesia (36), and lumbar diskography (37).

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If lumbar puncture alone can cause intracranial meningeal enhancement, then the significance of meningeal enhancement as an indicator of meningeal disease would be reduced on MR examinations after lumbar puncture. We retrospectively and prospectively studied the frequency of unexplained enhancement of the dura-arachnoid around the brain after lumbar puncture.

Subjects and Methods

The retrospective analysis was performed using a computerized key word search of brain MR reports covering an 18-month period (November 1989 through April 1991). Eleven patients were retrospectively identified who were reported to demonstrate dural-arachnoidal enhancement that was not associated with prior craniotomy, CSF shunting procedure, or an adjacent parenchymal or extraaxial lesion, conditions that are well-described causes of meningeal enhancement. The medical records of the 11 patients were reviewed for a source of the enhancement and the results of the lumbar puncture.

During the subsequent six months, patients were prospectively identified who underwent MR imaging within 1 month after lumbar puncture. Patients were identified by interview and hospital chart review at the time of MR examination and by cross-referencing laboratory data of CSF examinations with MR scheduling records. Patients were excluded who had had prior craniotomy or CSF shunting procedures.

One hundred four patients met the criteria of an MR within 1 month of lumbar puncture. Ninety-seven patients underwent enhanced MR examination with gadopentate dimeglumine (0.1 mmol/kg). Eight of these patients had two MR studies after lumbar puncture, and one had three studies, for a total of 114 post-lumbar puncture studies. One patient with two studies received gadolinium for the first post-lumbar puncture study only, yielding a total of 106 enhanced post-lumbar puncture studies.

The patients' ages ranged from 19 to 84 years old (mean 45.4 years). The mean time to follow-up MR examination after lumbar puncture was 5.5 days (SD 6.8 days, range 1 to 30 days). Approximately half of the exams were performed within the first 72 hours of lumbar puncture (34 were obtained within 24 hours, 13 between 24 and 48 hours, and 11 between 48 and 72 hours). Thirteen patients underwent MR examination before and after lumbar puncture (the prelumbar puncture study was unenhanced in one patient).

MR imaging was performed on a 1.5-T system (Signa, GE Medical Systems, Milwaukee, Wis). All exams included sagittal short repetition time (TR)/short echo time (TE) (500–800/11–26/1 [TR/TE/excitations]) and axial long-TR (2700–3500/17–30, 70–96/1) spin-echo images. All patients who received contrast underwent axial short-TR/short-TE (500–800/11–26/1) spin-echo imaging immediately after contrast administration. Coronal short-TR/short-

TE (500–800/11–26/1) images were also obtained in all but 10 patients after contrast administration.

Imaging analysis was performed by a neuroradiologist who was blinded to the patients' histories and to the results of their CSF examinations. He was asked to record the presence and extent of dural-arachnoidal enhancement that was not isolated to an area adjacent to a parenchymal or dural-based mass. The presence of enhancement was rated as definite or equivocal. The extent of enhancement was rated as focal (involving an isolated segment of dura-arachnoid) or diffuse (extending continuously over the convexities). The presence of pial enhancement was recorded. The presence of subdural collections, as defined by inward displacement of cortical veins (38, 39), was also recorded.

Patient records were reviewed to determine the results of CSF examination, including cell count and differential, glucose, protein, culture and microbiologic stains, and cytologic and pathologic examination, when applicable. The histories were reviewed to determine the presumed cause of meningeal disease.

Results

Retrospective Group (Table 1)

Eleven patients with dural-arachnoidal enhancement were identified retrospectively from an 18-month period—nine with diffuse enhancement, and two with focal enhancement. The MR enhancement patterns and clinical information for these patients are presented in Table 1. Ten of these 11 patients had undergone lumbar puncture between 1 and 8 days before MR. One patient with central nervous system sarcoid underwent lumbar puncture approximately 10 months before MR examination.

Based on chart review and CSF results, only one patient in the retrospective group lacked a clear explanation for enhancement. This patient received epidural anesthesia for childbirth 4 days before MR (there was no record of unintentional dural puncture). She experienced hypertension, headaches, and seizures after delivery, which were attributed to eclampsia. A lumbar puncture was performed 3 hours before MR (4 days after epidural anesthesia) and was remarkable for 8000 red blood cells per microliter without xanthochromia and no white blood cells in the final tube.

Prospective Group (Table 2)

During the subsequent 6 months, 104 patients were prospectively identified who underwent lumbar puncture before MR examination of the brain. Ninety-seven patients received gadopentetate dimeglumine. None of the seven patients who

TABLE 1: Retrospective group

Patient	Dural-Arachnoidal Enhancement	Pial Enhancement	Collections	Diagnosis
1	+, Diffuse	—	—	Subarachnoid hemorrhage
2	+, Diffuse	+	+	Enteroviral meningitis, agammaglobulinemia
3	+, Diffuse	—	—	Carcinomatous meningitis, prostate cancer
4	+, Diffuse	—	+	Central nervous system leukemia by CSF cytology within two days of MR, had intrathecal methotrexate
5	+, Diffuse	—	+	Propionibacterium meningitis diagnosis by biopsy
6	+, Diffuse	—	—	Lymphomatous meningitis
7	+, Diffuse	—	—	Lymphomatous meningitis by CSF cytology
8	+, Diffuse	—	—	Aseptic meningitis
9	+, Focal	—	—	Central nervous system sarcoid
10	+, Focal	—	—	Tuberculous meningitis
11	+, Diffuse	+	—	Cause unknown Eclampsia, epidural anesthesia 4 days before MR

TABLE 2: Prospective group: definite enhancement

Patient	Dural-Arachnoidal Enhancement	Pial Enhancement	Collections	Diagnosis
1	+, Diffuse	—	+	Transverse sinus thrombosis
2	+, Diffuse	+	—	Central nervous system vasculitis, biopsy showed inflammatory infiltrate in dura-arachnoid
3	+, Diffuse	—	—	Aseptic meningitis
4	+, Diffuse	+	—	Carcinomatous meningitis on lumbar puncture, melanoma
5	+, Diffuse	+	+	Superior sagittal sinus thrombosis
6	+, Diffuse	—	—	Recently treated neurosyphilis
7	+, Diffuse	—	+	Unexplained

underwent only unenhanced examinations after lumbar puncture showed evidence of subdural collections.

Seven of the 97 patients who underwent contrast-enhanced exams demonstrated definite dural-arachnoidal enhancement. The findings were diffuse in all seven patients. The MR enhancement patterns and clinical information for these seven patients are presented in Table 2. None of the seven patients had enhanced MR examinations before lumbar puncture. The cause of enhancement was evident for six of the seven patients on review of the clinical data.

Only one patient in the prospective review with definite dural-arachnoidal enhancement demonstrated no clear cause. She was a 37-year-old woman who presented with abrupt onset of headache but unremarkable findings at lumbar puncture except for a low opening pressure (64 mm

H₂O). Her first brain MR was performed approximately 3 weeks after onset of symptoms (1 week after her first lumbar puncture) and demonstrated abnormal enhancement and small subdural collections. There was no evidence of brain descent as measured by the method of Reich et al (40). In view of the low opening pressure on initial lumbar puncture, the diagnosis of spontaneous intracranial hypotension was entertained, but attempted radionuclide cisternogram to confirm the diagnosis was unsuccessful because of unintentional epidural injection of the radioisotope.

Two patients had equivocal enhancement, diffuse in one case and focal in the other. One patient with sickle-cell disease demonstrated possible focal enhancement near a patent superior sagittal sinus which could not confidently be distinguished from a vessel. One patient with human T-cell lymphotropic virus type-I infection

and autopsy-proved supratentorial osmotic demyelination demonstrated possible diffuse dural-arachnoidal enhancement on postcontrast images. However, the images were suboptimal because of prominent motion artifact. The dura and arachnoid at autopsy were described as normal to visual inspection.

There was no evidence of a relationship between traumatic lumbar puncture and the presence of enhancement. Cell counts were available for 81 of the 97 patients in the prospective group, including all nine patients with enhancement (seven definite, two equivocal) and 72 out of the 88 without enhancement. Four of nine patients (44%) with enhancement and 30 of 72 (42%) of those without enhancement had more than 100 red blood cells per microliter in the first tube for which a count was performed. One of nine patients (11%) with enhancement and 10 of 72 (14%) of those without enhancement had more than 1000 red blood cells per microliter in the first tube.

Discussion

Several authors have recently stated or proposed that lumbar puncture alone may cause meningeal enhancement around the brain (17–20), possibly related to intracranial hypotension from continuous CSF leakage at the site of dural puncture. Based on rare case reports of intracranial subdural hematomas after lumbar puncture or spinal anesthesia (26–31), it has been proposed that decreased CSF pressure caused by leakage of CSF may cause downward shift of the brain, compensatory dilatation of cerebral veins, and rupture of small vessels within the meninges (21–25). Because MR is sensitive to subtle changes of the meninges intracranially, it would be capable of revealing enhancement or small extraaxial collections after lumbar puncture.

If meningeal enhancement caused solely by lumbar puncture were a common occurrence, it would make the significance of the finding of enhancement after lumbar puncture questionable. Farn and Mirowitz (Farn JW, Mirowitz SA, Contrast Enhancement of Normal Meninges on Gadolinium-Enhanced 3D Gradient-Echo MR Imaging, presented at the 78th Annual Meeting of the Radiologic Society of North America, Chicago, 1992) have shown that the overall incidence of diffuse dural-arachnoidal enhancement supratentorially in healthy patients is extremely low on spin-echo images, approximately 1%. Enhance-

ment was seen much more frequently, however, in the same patients evaluated with three-dimensional Fourier transform techniques, possibly related to the shorter echo times used. The number of patients who underwent lumbar puncture before MR examination was not reported.

Our analyses of the incidence of unexplained intracranial dural-arachnoidal enhancement on spin-echo imaging after lumbar puncture demonstrate that the rate of occurrence is very low. In our 6-month prospective study, we found only one case out of 97 enhanced exams in which dural-arachnoidal enhancement could not be explained by a condition that has been reported to cause meningeal enhancement. In our retrospective analysis for the 18-month period preceding the prospective study, only one case lacking a clear explanation for enhancement was found.

We considered any pathologic process that has been previously associated with diffuse meningeal enhancement to represent a likely cause of enhancement in our cases. These processes include bacterial or aseptic meningitis (1–3), carcinomatous or lymphomatous infiltration of the meninges (3, 5–7), sarcoidosis (4), and central nervous system vasculitis (13) (our case was also proved pathologically). Subarachnoid hemorrhage has been associated with enhancement in previous reports (3) (Brown E, DeLaPaz R, Intracranial Meningeal Enhancement with GdDTPA MR, presented at the 27th Annual Meeting of the American Society of Neuroradiology, Washington, DC, 1989), and this finding is compatible with the chemical inflammation of the meninges induced by blood which has been described pathologically (41). Dural sinus thrombosis has been associated with diffuse meningeal enhancement distant from the thrombosed sinus in previous MR and computed tomographic reports (10–12).

Other studies support the notion that most cases of meningeal enhancement are associated with disease processes that represent likely explanations for enhancement. Brown and DeLaPaz identified causes in 22 consecutive cases of meningeal enhancement (Brown E, DeLaPaz R, Intracranial Meningeal Enhancement with GdDTPA MR, presented at the 27th Annual Meeting of the American Society of Neuroradiology, Washington, DC, 1989). Phillips et al noted five cases out of 35 with dural-arachnoidal enhancement in which CSF or pathologic findings did not explain meningeal enhancement (3). However, four of their cases were patients with widely metastatic neoplasms, and clinical suspicion of meningeal

disease. The diagnosis of meningeal carcinomatosis could not be excluded because of the low sensitivity of only one or two CSF cytologic examinations (42).

There is no evidence in this study that traumatic lumbar punctures are a cause of unexplained dural-arachnoidal enhancement; there was no difference in the frequency of traumatic lumbar taps between the cases with and those without dural-arachnoidal enhancement. There was also no evidence that subdural hematomas were extending intracranially from the spine in any case.

One case in our retrospective group lacked a clear cause for enhancement. This patient received epidural anesthesia 4 days before MR. Although epidural anesthesia has been reported to cause abnormalities in the spine and meninges distant from the site of injection (43), there are no reports to our knowledge that it is associated with intracranial enhancement. Therefore, lumbar puncture, either unintentionally at the time of epidural anesthesia or when performed for diagnostic purposes 3 hours before MR, cannot be excluded as the cause of enhancement in this case.

Clinical features in the one unexplained case in the prospective group suggested the diagnosis of spontaneous intracranial hypotension, a syndrome characterized by postural headache and low CSF pressure (less than 70 mm H₂O) (25, 44) in which subdural fluid collections and dural-arachnoidal enhancement on MR have previously been reported (18, 19, 45). It has been proposed that many of these cases may be caused by leakage of CSF from defects in the meninges, often in the cervicothoracic spine, which have been hypothesized to occur at epidural or perineural cysts after minor trauma (25, 44). These defects have been documented in some cases by radionuclide cisternography or myelography (25, 46, 47). Subdural collections and meningeal enhancement are presumably caused by descent of the brain analogous to the mechanism proposed for the rare cases of large collections after lumbar puncture discussed above.

Lumbar puncture is complicated by headache in 11% to 58% of patients (48–52). This complication, which is benign and self-limited in the vast majority of cases, has been hypothesized to be caused by intracranial hypotension caused by leakage of CSF from the site of lumbar puncture and subsequent descent of the brain. This causes traction on pain-sensitive structures such as the

meninges producing a postural headache (21–25, 44, 51). Alternatively, it may relate to vasodilation in the meninges in response to lowered CSF volume (32). We did not assess the frequency of post-lumbar puncture headache or the degree of brain descent in our prospective study group. Clearly, however, the incidence of unexplained dural-arachnoidal enhancement seen in our prospective study group (one out of 97 patients) is much lower than the reported incidence of post-lumbar puncture headache (48–52).

We conclude from our data that unexplained dural-arachnoidal enhancement in the brain after lumbar puncture is uncommon, if it occurs at all. When seemingly unexplained meningeal enhancement is present, a thorough search, including repeat lumbar puncture and occasionally a follow-up MR, is warranted to exclude an underlying cause such as central nervous system malignancy, infection, or an inflammatory process. Flow-sensitive MR sequences should be considered to exclude sinus thrombosis. Rarely, meningeal biopsy may be required for difficult cases when clinical suspicion is high. Lumbar puncture alone is an unlikely cause of intracranial dural-arachnoidal enhancement.

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