Incidence of meningeal enhancement on brain MRI secondary to lumbar puncture

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Abstract

Background: Concern for reactive meningeal enhancement after lumbar puncture (LP) is a common reason for performing brain MRI prior to LP. We sought to determine actual incidence of unexplained meningeal enhancement after LP. **Methods:** We collected results from all contrasted brain MRIs in patients admitted to adult neurology at a New York City hospital over a 3-year period. We used electronic medical records to determine whether an LP had been done within 30 days prior to brain MRI. The control group comprised those brain MRIs not preceded by an LP within 30 days prior to imaging. Number of cases of unexplained meningeal enhancement was compared between groups using a Fisher exact test. We recorded variables such as number of LP attempts, needle size, amount of fluid removed, and days from LP to brain MRI. **Results:** From 2011 to 2013, there were 77 cases of LP prior to brain MRI and 707 controls ($n = 784$). Of the cases, 3 had meningeal enhancement, 1 (1.2%) of which was unexplained. Of the 707 controls, 36 had enhancement, and none was unexplained. The p value comparing unexplained enhancement in the cases vs controls was 0.098. **Conclusions:** Iatrogenic meningeal enhancement from prior LP that is not attributable to traumatic LP or intracranial hypotension is rare and not more common than in cases without a prior LP. Results suggest that the practice of delaying LP until after brain MRI might not be supported in cases where LP is necessary. **Neurol Clin Pract** 2016;6:315-320

There are many common causes of enhancement on brain MRI, including postoperative reactivity, neoplasm, infection, and intracranial hypotension.^{1,2} Of the potential iatrogenic causes, the least understood is the meningeal reactivity reportedly seen after uncomplicated lumbar punctures (LP) without associated CSF leak or trauma.

While this phenomenon is commonly cited in clinical practice, review of the literature reveals that there is only one study to date investigating the claim that uncomplicated LP

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manipulation can cause meningeal enhancement, and the proposed mechanisms are poorly understood. In 1994, Mittl and Yousem³ conducted a mixed retrospective-prospective study of patients who had had an LP performed within 30 days prior to a contrasted brain MRI, but they did not compare their cases to a control group that had not had LP prior to brain MRI. Moreover, the 2 cases of unexplained pachymeningeal enhancement identified in their study arguably were explained by a traumatic tap and intracranial hypotension.

In clinical practice, the concern for iatrogenic meningeal enhancement from LP is a common reason for performing brain MRI prior to LP. This practice, however, is often impractical and can lead to delayed diagnosis. Evidence-based medicine should be used either to support or refute this practice. We sought to determine the incidence of unexplained meningeal enhancement after LP and hypothesized that this phenomenon is rare and no more common than in cases without prior LP.

METHODS

Standard protocol approvals, registrations, and patient consents

Prior to beginning our data search, we obtained institutional review board approval through our institution.

We retrospectively compared the incidence of meningeal enhancement on brain MRI in cases of LP prior to imaging vs controls without LP prior to imaging. The study included all contrasted brain MRIs of patients ≥ 18 years old who were admitted to an adult neurology service at a New York City teaching hospital over the 3-year period 2011 through 2013. We used electronic medical records (EMR) to determine whether an LP had been done within 30 days prior to brain MRI. The control group comprised those brain MRIs not preceded by an LP within 30 days prior to imaging. If there was any meningeal enhancement in either a case or a control, we reviewed the EMR to determine if there was a known explanation for the enhancement. Of note, while post-LP meningeal reactivity is thought to be pachymeningeal, there is no established pattern that is evidence-based, and thus we decided to include any type of meningeal enhancement.

Exclusion criteria for cases and controls included patients with noncontrasted brain MRI and no available means of confirming or excluding that an LP had been done; for example, patients who had been transferred from another hospital but had a brain MRI upon arrival to our institution. Exclusion criteria for cases included patients in whom the LP had been attempted but no sample results were available.

Presence of meningeal enhancement was based on the final reports of multiple different in-house neuroradiology attendings over 3 years. Confirmation that an LP had been done was proven by the presence of CSF results in the EMR. At our institution, CSF results are dated for the day that the sample was collected.

The total number of cases of unexplained meningeal enhancement was compared between groups using a Fisher exact test; a p value of <0.05 was considered significant. We recorded age and sex for cases and controls, and we noted variables such as number of LP attempts, needle size, amount of fluid removed, and days from LP to imaging. For the number of days from LP to brain MRI, some cases had had more than one LP on different days leading up to

Figure 1 Brain MRI performed 5 days after lumbar puncture (LP)

Patchy diffuse pachymeningeal enhancement seen on contrasted brain MRI performed 5 days after LP; asymptomatic patient; no definite cause for enhancement found.

imaging. In these cases, the number of days was recorded based on the date closest to the brain MRI.

RESULTS

From 2011 to 2013, there were 77 cases of LP within 30 days prior to brain MRI, and there were 707 controls ($n = 784$). The average age in the case group was 47.3 years ($SD \pm 18.5$) and average age in the controls was 49.4 years ($SD \pm 18.4$). The ratio of women to men in the case group was 1.40 to 1, compared to 1.39 to 1 in the control group. In the case group, the average number of days from LP to brain MRI was 4.4. Only 7 cases (9.1%) had had an LP done more than 15 days and fewer than 30 days prior to brain MRI. The gauge of LP needle was available for 23 cases, and all but 5 of these had had a standard 20-G needle used for the procedure.

Of the 77 cases of LP prior to brain MRI, 3 cases had meningeal enhancement. Two of the 3 cases of enhancement were explained. One case was a meningioma of the right middle cranial fossa. The other case was an active herpes simplex encephalitis at the time of the LP and brain MRI. Of note, in the case of the latter infectious etiology, the enhancement was of the temporal leptomeninges, not of the pachymeninges.

Only one case of the 77 had meningeal enhancement that could not be fully explained by information in the EMR and thus was deemed to be idiopathic. The enhancement on brain MRI was pachymeningeal and diffuse (figure 1). The case was 5 days post-LP at the time of imaging. Of note, a brain MRI performed on the day prior to LP did not show meningeal enhancement. The gauge of the LP needle and the number of attempts was unknown. CSF studies showed 2 leukocytes, 1,365 erythrocytes, 37 protein, 61 glucose, and negative xanthochromia and CSF cultures. The total amount of fluid removed was 30 mL, and the opening pressure was elevated at 30.2 mm Hg. This patient had a known diagnosis of idiopathic intracranial hypertension.

Of the 707 controls, 36 had enhancement on the brain MRI. All incidents of enhancement were explained by known causes, such as dural metastases, meningioma, postoperative changes, infectious meningitis, cerebral amyloid angiopathy (figure 2), and intracranial hypotension from a spontaneous dural leak (table).

A limitation of our study was the inability to match cases and controls one-to-one with similar patient characteristics, such as age and sex.

Overall, of the 77 cases, 1 had unexplained enhancement as compared to no incident of unexplained enhancement among the 707 controls. The p value comparing unexplained enhancement in all cases to unexplained enhancement in all controls was 0.098. This finding supports our hypothesis that unexplained meningeal enhancement is rare and not more common after LP. The number needed to treat (NNT) for this study was 83.3.

DISCUSSION

The results support our hypothesis that, in the absence of trauma or CSF leak, meningeal enhancement after LP is rare and not more common as compared to a control group that had not had prior LP. While it has been common practice to perform LPs after a contrasted brain MRI to avoid iatrogenic meningeal reactivity, our findings suggest that reactive meninges without evidence of traumatic tap or CSF leak should be considered a diagnosis of exclusion and that other explanations should be sought before attributing the finding to a prior LP. From a clinical practice standpoint, these results suggest that the practice of delaying LP until after brain MRI might not be supported in cases where LP is necessary. An ideal study design, however, would require a randomized control trial and an even larger sample size.

Recognized causes of iatrogenic meningeal enhancement after LP include traumatic LPs and intracranial hypotension from CSF leak. Both such causes are easily discerned in clinical practice. Traumatic LPs are evidenced by a high CSF erythrocyte count. Intracranial hypotension is associated with symptomatic orthostatic headache and radiographic evidence of low-lying cerebellar tonsils and enlarged pituitary.¹ Intracranial hypotension is thought to cause dilation of the cerebral veins, which in turn allows for uptake of contrast in damaged meningeal vessels⁴ as well as interstitial edema in the dura from vasocongestion.¹

Figure 2 Coronal view of T1-weighted postgadolinium brain MRI

Coronal view of T1-weighted postgadolinium brain MRI shows right-sided focal meningeal enhancement from biopsy-proven cerebral amyloid angiopathy in a patient who had had prior lumbar puncture.

Table Breakdown of etiologies found for both pachymeningeal and leptomeningeal enhancement on contrasted brain MRI without prior lumbar puncture

Etiology for enhancement	No. of cases
Postoperative craniotomy	7
Metastatic cancer with dural involvement	7
Meningioma	6
Infectious meningitis	6
Autoimmune meningoencephalitis	1
Traumatic subarachnoid hemorrhage	1
Neurosarcoidosis	1
Secondary to ischemia	1
Primary CNS lymphoma	1
Sturge-Weber syndrome	1
Spontaneous intracranial hypotension	1
Amyloid angiopathy	1
Venous sinus thrombosis	1
Schwannoma in neurofibromatosis	1

While unlikely, we cannot fully rule out traumatic tap as a potential cause of our only unexplained case of pachymeningeal enhancement after LP. The CSF showed 1,365 erythrocytes, indicating a mildly traumatic tap. However, as the erythrocyte count was not highly elevated and as the brain MRI had been performed 5 full days after the tap, we determined that there was insufficient evidence to conclude that the traumatic tap and the enhancement were causally related.

Moreover, the authors note that the LP in our unexplained case was a large-volume tap for idiopathic intracranial hypertension. It is possible that transient intracranial hypotension periprocedurally could have accounted for the pachymeningeal enhancement after LP without the typical clinical or other radiographic features of intracranial hypotension from CSF leak.

A limitation of our study was the inability to match cases and controls one-to-one with similar patient characteristics, such as age and sex. The discrepancy between the number of cases and controls over this time likely was influenced by the practice of withholding LP prior to brain MRI. However, despite not having a one-to-one match, the average age and sex ratios of the patients in the case and control groups were remarkably similar. Data on ethnicity or racial background were not consistently available in the EMR.

Another study limitation was the smaller size of the case group, which was influenced in part by the relatively fewer number of LPs done prior to brain MRI. Given that idiopathic meningeal enhancement is a low event rate phenomenon per prior reports,³ a larger study would be helpful to reduce the potential for a type 2 error. Accordingly, the NNT might be better suited to aid clinical decision-making for patients who need both an LP and a contrasted brain MRI.

The ideal study design would be a large-scale randomized control trial that prospectively matches healthy cases and controls. However, such a study is limited by inherent impracticalities. Such a study would require an LP to be performed prior to a contrasted brain MRI in healthy patients who would not otherwise require an LP. Recruitment for a high-powered study that would require cases to undergo a painful procedure with known risks and no potential benefit would be challenging at best.

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AUTHOR CONTRIBUTIONS

S.F. Wesley: drafting/revising the manuscript, study concept or design, analysis or interpretation of data, acquisition of data, statistical analysis, and study supervision. R. Garcia-Santibanez: drafting/revising the manuscript, study concept or design, analysis or interpretation of data, acquisition of data, and statistical analysis. J. Liang: drafting/revising the manuscript and acquisition of data. D. Pyburn: analysis or interpretation of data and study supervision.

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