Frequency of meningeal enhancement caused by lumbar puncture.

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Frequency of Meningeal Enhancement Caused by Lumbar Puncture

A recent inquiry of whether a lumbar puncture can cause meningeal enhancement led us to the paper, “Frequency of Unexplained Meningeal Enhancement in the Brain After Lumbar Puncture,” by Mittl and Yousem (1). The conclusion that intracranial dural-arachnoidal enhancement after lumbar puncture is uncommon is not supported by the data that are presented. In their prospective study, they claim the occurrence of enhancement that may have been attributable to lumbar puncture was in 1 of 97 patients. A falsely low frequency of enhancement that may be attributable to lumbar puncture was obtained because 6 and possibly many more patients with another possible cause of meningeal enhancement were included. If any of these patients had demonstrated enhancement, the enhancement would have been attributed to the other cause and not to the lumbar puncture. To find the true frequency of enhancement caused by lumbar puncture, we need to know the number of patients who had no other possible cause for meningeal enhancement. The frequency would be the number of cases with unexplained enhancement divided by the number of patients with no other possible cause for the enhancement.

Additionally, given that a lumbar puncture was a possible explanation for enhancement in 2 of their 18 cases, the conclusion that lumbar puncture is an unlikely cause of intracranial meningeal enhancement is not supported.

Furthermore, because the entities that were used to explain the meningeal enhancement in the other 16 cases do not produce meningeal enhancement in 100% of affected patients, the authors have not proved that lumbar puncture did not contribute to or entirely cause the meningeal enhancement in these patients.

Despite our objections to the logic used in evaluating the data in this paper, we agree with the authors that meningeal enhancement after lumbar puncture should be entertained as a diagnosis only after more serious central nervous system conditions have been excluded.

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Reference

Reply

It is always refreshing to know that someone has read your article after it has been published. We thank Dr Rubenstein and Dr Cajade-Law for this inspiration. We also are encouraged that they agreed with our conclusion that meningeal enhancement after lumbar puncture should be a diagnosis of exclusion and that it occurs rarely.

Dr Rubenstein and Dr Cajade-Law point to a flaw in our method. Ideally, we would have liked to have healthy volunteers to perform lumbar puncture on and image with contrast-enhanced magnetic resonance (MR) to determine the frequency of meningeal enhancement. Because this is not possible in this era of institutional review board scrutiny of human experimentation, we chose to evaluate patients who had had MR within 1 month of lumbar puncture. These lumbar punctures were performed as part of a diagnostic workup for neurologic problems.

Dr Rubenstein and Dr Cajade-Law point out that if any of these patients had demonstrated meningeal enhancement, it would have been attributed to the cause for which the lumbar puncture was performed. In point of fact, we were hoping to see the absence of meningeal enhancement after lumbar puncture in this portion of the study. If none of these patients showed meningeal enhancement (regardless of the source of their neurologic symptoms), we would have had compelling evidence that a lumbar puncture is unlikely to cause meningeal enhancement.

Once we had 7 patients who did have meningeal enhancement, we could identify an upper limit of frequency of 7 of 97, or 7.2%. We then set out to define the lower limit by which meningeal enhancement might occur after lumbar puncture and were able to identify 6 of 7 patients who had a reasonable cause for meningeal enhancement beyond that of the lumbar puncture. This defined the lower limit at 1 of 97, or 1.0%. In truth, we were hoping to study a lot more patients who had diseases that have not been correlated with meningeal enhancement (such as multiple sclerosis), so that we could attribute the finding directly to the lumbar puncture. Therefore, though we agree that Dr Rubenstein and Dr Cajade-Law make an excellent point regarding enhancement in the 97 patients, we wanted to emphasize the 90 patients who had undergone lumbar puncture who did not show contrast enhancement. This is the important point in the analysis.

Dr Rubenstein and Dr Cajade-Law also note that a lumbar puncture was a possible explanation for enhancement in 2 of the 18 patients who showed meningeal enhancement over a 24-month period. We thought that we had made the point that the 1 patient in the prospective group with unexplained meningeal enhancement probably had the diagnosis of spontaneous intracranial hypotension and therefore should not be included in the lumbar puncture etiology group. This would reduce the frequency to 1 in 18 cases, or 5.6%. Taken another way, this would sug-
gest that in our practice (with 2½ clinical scanners working up to 16 hours per day), one might see meningeal enhancement attributable solely to a lumbar puncture once every 2 years. This indeed is a rare occurrence.

Recently presented data also support our conclusions (Frei DF Jr, Moran CJ, “Enhanced Magnetic Resonance of Infectious Meningitis: Correlation with CSF Laboratory Results,” presented at the 33rd Annual Meeting of the American Society of Neuroradiology, Chicago Ill, 1995). These authors performed a blinded retrospective review of enhanced MR examinations obtained after lumbar puncture in 89 patients with meningitis and 35 patients with normal cerebrospinal fluid. Fifty-seven percent of patients with meningitis demonstrated abnormal meningeal enhancement, whereas none of the patients with normal cerebrospinal fluid showed enhancement.

Even assuming the worst-case scenario that Dr Rubenstein and Dr Cajade-Law have proposed, the highest frequency of meningeal enhancement after lumbar puncture still would be somewhere between 7.2% and 11.1%. This friendly exchange notwithstanding, we can still agree that lumbar puncture should be entertained as a cause of exclusion of meningeal enhancement.

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Symmetric Lesions of the Subthalamic Nuclei in Mitochondrial Encephalopathies: An Almost Distinctive Mark of Leigh Disease with COX Deficiency

We read with interest the article on hemiballismus by Provenzale and Schwarzschild (1), and we congratulate the authors for their exquisite radiologic-clinical correlation. The correlation with the anatomic diagrams allows the recognition of the exact topographic location of the subthalamic nucleus.

To the list made by Provenzale and Schwarzschild, we would like to add other disorders that cause lesions neatly and symmetrically involving the subthalamic nuclei such as Leigh disease (2) and, rarely, other similar mitochondrial encephalopathies observed in children.

We observed bilateral, symmetric lesions in the subthalamic nuclei in seven children with mitochondrial disorders. Six had Leigh disease, all with cytochrome c oxidase (COX) deficiency (Fig 1); these patients also had lesions in the medulla oblongata, the pontine tegmentum, the periaqueductal area, and the cerebellar white matter. Four other children with Leigh disease attributable to COX deficiency (two of them with a mild form) and four with Leigh disease and different enzymatic defects did not have lesions in the subthalamic nuclei. Another patient with COX deficiency, not classified as Leigh disease because of a

Fig 1. Axial T2-weighted and coronal T1-weighted images of two patients with Leigh disease and COX deficiency show symmetric lesions in the subthalamic nuclei (arrows) (from Savoiardo et al [2]).

different clinical course, had diffuse abnormalities in the white matter but no lesions in the subthalamic nuclei. Finally, in an infant with a mitochondrial disorder attributable to NADH-CoQ-reductase deficiency, presenting with asymmetric swelling of the midbrain simulating a tumor, lesions developed in the subthalamic nuclei; these lesions disappeared, as did the other abnormalities, on dichloroacetate therapy.

Lesions in the subthalamic nuclei also were described by Medina et al (3) in one patient with Leigh disease attributable to COX deficiency. Their series included three patients with COX deficiency and four other patients with different enzymatic defects.

In our series we observed, therefore, a total of seven cases of mitochondrial encephalopathy with symmetric lesions in the subthalamic nuclei; six of them had Leigh disease with COX deficiency. Thus we would like to emphasize that bilateral involvement of the subthalamic nuclei should suggest a mitochondrial disorder, most of the time Leigh disease with COX deficiency. We have not observed such lesions in other metabolic disorders.

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References
I thank Dr Savoiardo and his coauthors for their comments regarding Leigh disease associated with COX deficiency. As the authors have shown, the subthalamic nucleus lesions in that disorder often are bilateral (unlike typical subthalamic nucleus lesions in other disease states), symmetric, and accompanied by multiple regions of abnormal signal within the brain stem on MR imaging (1). Foci of necrosis, capillary proliferation, and gliosis in the subthalamic nuclei have been documented at autopsy in a patient with Leigh disease (2). Such foci may be the cause of the MR lesions seen in Dr Savoiardo’s patients.

Dr Savoiardo’s letter illustrates one other interesting point. A lesion in the subthalamic nucleus is neither a necessary nor a sufficient condition for development of hemiballismus. It is well established that hemiballismus can occur as a result of lesions outside the subthalamic nucleus (3). The patients in Dr Savoiardo’s series, on the other hand, appear to illustrate the fact that a lesion in the subthalamic nucleus does not always produce hemiballismus, because none of his patients were reported to have a movement disorder. One possible explanation is involvement of the red nucleus or pyramidal tract, which must remain intact for hemiballismus to develop (4). It also is possible that slow development of a lesion over time (as in the setting of a metabolic disorder like COX deficiency) may not result in hemiballismus, unlike the acute lesions with which hemiballismus is usually associated.

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References

Erratum
In the May 1995 article “Giant Serpentine Aneurysms: A Review and Presentation of Five Cases” (Aletich et al., 16:1061–1072), parts E, H, and G of Figure 5 were reprinted with permission from an article in the Journal of Neurosurgery (Green KA, Anson JA, Spetzler RF. Giant serpentine middle cerebral artery aneurysm treated by extracranial-intracranial bypass. J Neurosurg 1993;78:974–978).