

unilateral facial numbness and weakness are occasionally the patient's chief complaints. Stupor is an unusual complication that we encountered in one patient because of a central herniation syndrome.

Although CSF pressure in SIH is usually low, this is not always the case. Mokri et al noted pressures between 70 and 130 mm in four of 26 patients (2), and we observed the same range in three patients with SIH. CSF lymphocytosis between 8 and 80 cells/mm³ is not uncommon, and can escalate as high as 226 cells/mm³ with a mild increase in CSF protein (3). These findings may lead to the erroneous diagnosis of "viral meningitis."

MR findings characteristic of SIH reflect an increase in venous volume throughout the brain and dural structures, including the spine. MR reveals diffuse dural enhancement (as if the dura were outlined with a felt-tip pen), prominent dural sinuses, an enlarged epidural venous plexus and pituitary gland, subdural collections from effusions or hematomas, and downward displacement of the chiasm, pons, and cerebellar tonsils. Although diffuse dural enhancement is almost invariably present, descent of the tonsils occurs in approximately 75% of SIH patients. Patients with elevated intracranial pressure may sometimes demonstrate descent of posterior fossa structures and chiasm on sagittal T1-weighted MR scans that appear similar to MR findings of SIH. With elevation of intracranial pressure, however, the venous dural sinuses are effaced, not enlarged, and usually diffuse enhancement of the dura is not present. In patients with dural metastases, infection, or inflammation, the enhancement of the dura is usually patchy, quite different from the appearance of SIH. Dural biopsy is not indicated in patients with clinical and radiologic features of SIH, and we have not recommended dural biopsy in over 40 cases.

Localization of a spinal fistula, usually secondary to a ruptured perineural (Tarlov) cyst, is *not* required unless the patient has failed to respond to at least two large-volume (20–30 mL) lumbar blood patches followed by a 10-minute, 30° lowering of the head for increased blood flow to the thoracic and cervical levels. If these fail, then localization of the CSF leak using *simultaneous* (single dural puncture) myelography and cisternography followed by CT at 3-mm increments are indicated. Consecutive decubitus cross table views obtained by positioning the patient on the left and then right side during myelography may facilitate the leakage of contrast medium from a laterally placed perineural cyst. Radiologic localization of the leak is necessary prior to direct instillation of an epidural blood patch at the precise location

of the fistula. If this fails, neurosurgical intervention is required. Epidural blood patching has been successful in the majority of our patients. Only three patients required surgical intervention, and surgery was successful in two.

The spinal CT and MR manifestations of SIH may also be misleading. Enlargement of the cervical epidural venous plexus may be misinterpreted as meningioma. Leakage of CSF may occur along a nerve root lateral to the spinal canal, resulting in the easy detection of the leak by isotope cisternography and CT myelography. Alternatively, the CSF leak may collect and extend within the "gutter" of the epidural space inside the spinal canal. In this instance, chronic leakage of CSF may become walled off within the epidural fat, creating a tubular pseudocyst which on cross-sectional imaging appears as a semicircular "dog ear" shaped collection, ventral or dorsal to the thecal sac. The wall of this "pseudocyst" may prevent an epidural blood patch from contacting the site of the leak, accounting for some treatment failures. In addition, the location of an epidural collection may not always reflect the actual site of the spinal fistula because CSF may extend to within the epidural space several levels away from the actual fistula site. Detection of the site of the CSF leak is usually not necessary since most patients are treated effectively by an epidural blood patch. Some patients, however, are refractory to this therapy. In our experience treatment is unsuccessful if the leak is too large, is positioned laterally along a nerve root sleeve, or enters a "pseudocyst" collection that is walled off from the epidural space.

The accurate localization of spinal fistulae and the treatment of these patients is rewarding. Most get better, and the radiologist plays an important role in the diagnosis and therapy of these patients.

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References

1. Rando TA, Fishman RA. Spontaneous intracranial hypotension: report of two cases and review of the literature. *Neurology* 1992;42:481–487
2. Mokri B, Piepgras DG, Miller GM. Syndrome of orthostatic headaches and diffuse pachymeningeal gadolinium enhancement. *Mayo Clin Proc* 1997;72:400–413
3. Fishman RA, Dillon WP. Intracranial hypotension. *J Neurosurg* 1997;86:165

Investigating Multiple Sclerosis with Spectroscopic Imaging: Harbinger of a New Paradigm

Multiple sclerosis (MS) is classically defined as a demyelinating disease (1). There is now evidence, however, suggesting that the difference in pathophysiology

between remitting-relapsing versus non-remitting-progressive MS is the degree of neuronal damage—damage that can be detected with neuroimaging.

In relapsing-remitting MS, a widely held belief is that neuronal function recovers from a conduction defect caused by myelin loss, loss resulting from up-regulated sodium channels in the neuron. This belief implies that myelin is damaged but neurons survive. Alternatively, a number of investigators have published MR spectroscopic evidence of neuronal death in MS lesions, pointing to decreased N-acetyl aspartate (NAA) in those lesions. Falini et al recently documented lower lesion NAA levels in cases of progressive MS than in benign MS (2). NAA reduction is present even in normal-appearing white matter in progressive MS, which has been interpreted as wallerian degeneration, axonal spread of damage, or both. In this issue of the *American Journal of Neuroradiology*, Heide et al (page 1047) report NAA decreases that were localized to visual pathways in patients with abnormal visual evoked potentials. These decreases were even found in areas where T2 imaging was normal, confirming and extending earlier work by correlating the NAA drop with abnormal neuronal function.

What causes these reduced NAA levels and neuronal death? Insight into this question was provided by a recent elegant study using 3-D confocal microscopy to examine active and chronic MS lesions (3). This study documented substantial damage to axons as well as myelin. Indeed, axonal transection was present in all lesions, with more damage in more active lesions. Such data indicate that neurons too are affected by MS—perhaps by inflammation, or perhaps by a “final common pathway” similar to ischemic damage. This latter possibility hints that neuroprotective agents may be effective in treating the lesions of MS patients, and slowing the neuronal damage that results from the demyelinating process. Many of the processes that escalate axonal injury and cell death may be delayed with the use of neuroprotective agents.

Radiology and neuroradiology in particular can take some of the credit for these advances in our understanding of MS as the authors of the confocal microscopy study attest (3). Such investigations suggest the role neuroradiology can play in furthering our understanding of disease and identifying new therapeutic targets. Heide et al's study is particularly exciting from a neuroradiologic perspective because of its application of spectroscopy.

Spectroscopic imaging (SI), or the generation of maps of metabolites such as NAA, lactate, and other markers such as pH, has the potential to revolutionize neuroradiologic diagnosis. Spectroscopy has, with the

use of single voxel techniques, demonstrated the ability to identify individual brain cell types, including specific malignancies and markers of early cerebral ischemia. The advent of routine SI, however, will likely shape a new paradigm for its use. Consider how the advent of imaging has aided T1-weighted, T2-weighted, and proton-density-based neurologic MR. Few neuroradiologists would try to make a diagnosis based on a single large T1-weighted or T2-weighted voxel over the brain. When the brain is imaged with 128×128 or even 64×64 voxels, however, diagnoses become more straightforward. When SI reaches the capability of providing 128×128 voxel images of the brain, the spatial distribution patterns of NAA and other metabolites will be much more clear. Such enhanced capability will make diagnoses of a host of new diseases as straightforward as establishing the distinction between MS plaques and metastases by T1 and T2 images. The article by Heide et al gives us a good first example of how analysis of the spatial distribution of metabolic markers can identify neurologic pathophysiology, even at a low resolution.

Spectroscopic imaging needs extensive additional refinement before it will be ready for routine clinical use. Studies demonstrating the utility of SI can only help encourage equipment vendors and researchers to push forward on various fronts to make this a standard clinical modality. Heide et al used echo-planar readout techniques and customized headcoils to boost signal-to-noise ratios, key methods needed for SI. Spectroscopic imaging would, of course, benefit from a higher field strength, a more expensive option than either of the two methods used by Heide et al. But if SI is to demonstrate its full potential, increases in signal-to-noise ratios resulting in acquisition time decreases and increased coverage are crucial. Clinical neuroradiologists—and their patients—eagerly await the technical advances needed to bring this tool into routine clinical practice.

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References

1. Waxman S. Demyelinating diseases—new pathological insights, new therapeutic targets (Editorial). *N Engl J Med* 1998;338:323–325
2. Falini A, Calabrese G, Filippi N, et al. Benign versus secondary-progressive multiple sclerosis: the potential role of proton MR spectroscopy in defining the nature of disability. *AJNR Am J Neuroradiol* 1998;19:223–229
3. Trapp B, Peterson J, Ransohoff R, Rudick R, Mork S, Bo L. Axonal transection in the lesions of multiple sclerosis. *N Engl J Med* 1998;338:278–285

Diffusion-Weighted Imaging of Stroke: A Brief Follow-up

In this issue of the *American Journal of Neuroradiology*, Lövblad et al (page 1061) nicely address issues I raised in a previous editorial on the subject of diffusion-weighted imaging (DWI) of acute cerebral ischemia (1). The main question I previously posed related to the pathophysiology of increased DWI signals, reflecting

decreased diffusion coefficients, as seen in the clinical setting of acute stroke. Does the signal reflect infarcted (dead) tissue or reversibly ischemic (live) tissue? Clearly treatment could depend on the underlying pathophysiology of the lesions identified.

The current article by Lövblad et al strongly supports