MR signal of mamillary bodies.

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LETTERS

MR Signal of Mamillary Bodies

I found the article "Gadolinium-Enhanced MR Findings in a Pediatric Case of Wernicke Encephalopathy" quite interesting and exquisitely illustrated (1). In the body of the paper and the legend of their Figure 1, however, the authors report that the signal intensity of the mamillary bodies appeared normal on the T2-weighted scans. To my view the mamillary bodies appeared of higher signal intensity than the adjacent brain structures. Based on the quality of the images and the generous size of these mamillary bodies, I think this unlikely because of volume averaging. In my experience the mamillary bodies are of uniform low signal intensity on T2-weighted images (Fig 1). Based on the T2 abnormalities apparent in other areas of the brain, it would seem reasonable that the mamillary bodies would also have abnormal signal, particularly in light of the corresponding enhancement demonstrated in their Figure 3.

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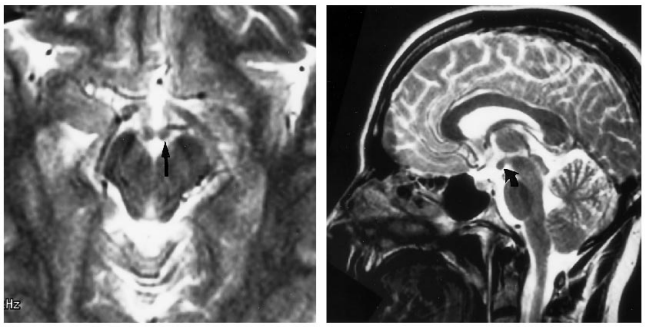
Reference

1. Harter SB, Nokes SR. Gadolinium-enhanced MR findings in a pediatric case of Wernicke encephalopathy. *AJNR Am J Neuroradiol* 1995;16:700–702

Reply

We appreciate Dr Mamourian's interest in our article. On the initial review, we felt that the slight increased signal in the mamillary bodies was probably explained by volume averaging. However, since receiving Dr Mamourian's letter, we have reviewed multiple other normal cases. Indeed, it appears that Dr Mamourian is correct. The mamillary bodies in healthy patients were uniformly of lower signal intensity than in our case of Wernicke encephalopathy. This is congruent with the pathologic involvement of the mamillary bodies demonstrated by intense enhancement.



A B

Fig 1. These axial (*A*) (3000/98/2 [repetition time/echo time/excitations], 6-mm section thickness) and sagittal (*B*) (3000/104/2, 5-mm section thickness) T2-weighted fast spin-echo MR images demonstrate the uniform low signal of the mamillary bodies (*arrows*).

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Widening of Virchow-Robin Spaces

We read with interest the recent *AJNR* article concerning the extreme widening of the Virchow-Robin spaces (1). We saw a similar case about a year ago. A 60-year-old man presented with headache, early recall disturbances, and hemifacial tics. The CT and MR findings were similar to those described by Ogawa et al. Only the left hemisphere was affected in our patient. The left temporal lobe was relatively spared. Most lesions were seen in the occipital and parietal lobes and several lesions were close to the midline and the corpus callosum (Fig 2). There was no enhancement after gadopentetate dimeglumine injection. These findings were unexpected and remain unexplained until now. An extensive diagnostic workup was normal and a follow-up examination five months later was identical. The discrepancy between the symptoms and the imaging findings was striking in our patient, and also was reported by Ogawa et al.

Although the pathogenesis remains a mystery, we believe that these imaging appearances, in the appropriate clinical setting, are pathognomonic of dilated perivascular spaces. An extensive differential diagnosis is superfluous.

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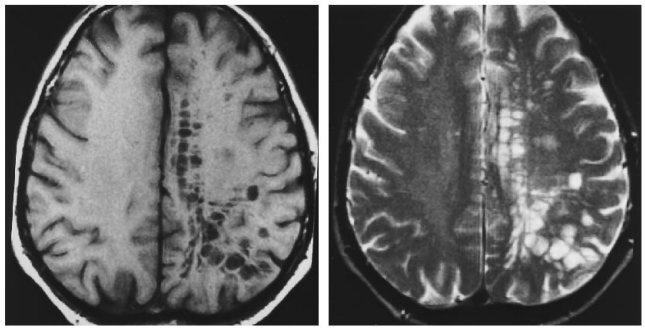

Fig 2. Axial T1-weighted (600/15) and T2-weighted (2500/90) spin-echo MR images show the dilated perivascular spaces in the left hemisphere.

Reference

1. Ogawa T, Okudera T, Fukusawa H, et al. Unusual widening of Virchow-Robin spaces: MR appearances. *AJNR Am J Neuroradiol* 1995;16:1238–1242

Editor's note.—*We forwarded the letter of Dr Demaerel et al to Drs Bacheschi, Magalhães, and Mathias, whose comments follow.*

Comment

The article by Ogawa et al and the letter by Demaerel et al are very interesting. Recently, we presented three patients with similar lesions detected with MR without corresponding neurologic dysfunction ("Multiple Cystic Lesions on White Matter without Clinical Manifestations [Unidentified Black Holes]," *Proceedings of the XV Symposium Neuroradiologicum, Neuroradiology* 1995;37[suppl]:246–247). Two patients had unilateral lesions similar to the case described by Ogawa et al, but the third patient had extensive bilateral lesions without neurologic disturbance (Fig 3). The histologic demonstration of dilated perivascular Virchow-Robin spaces by Ogawa et al shows that in similar cases, invasive methods of investigation are probably unnecessary.

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Is High Signal Intensity in the Corticospinal Tract a Sign of Degeneration?

I read with interest the cases reported by Mascalchi et al (1) in the April supplement issue of *AJNR*, describing the corticospinal tract degeneration in motor neuron disease. The authors noted that T2-weighted spin-echo MR images showed symmetric hyperintensity of the intracerebral corticospinal tracts in two patients with clinical and neurophysiologic diagnosis of primary lateral sclerosis and amyotrophic lateral sclerosis. They illustrated their findings with axial T2-weighted spin-echo MR images showing symmetric hyperintensity in the centrum semiovale and in the cerebral peduncles, in the first case, and in the cerebral peduncles and the internal capsules in the second case.

How did these lesions appear on T1-weighted and proton density–weighted images? In fact, Mirowitz et al (2) demonstrated that these findings could also correspond to the round area of high or low signal intensity that can be seen within the posterior limbs of the internal capsules on T2- and T1-weighted images, respectively. This normal feature probably reflects relatively lightly myelinated fibers, possibly a part of the parietopontine tract, passing through the internal capsule. Moreover, it is interesting that this normal feature, found in 50% of their patients on T2-weighted images at 0.5 T, was not identified in their control subjects.

Mascalchi et al did not indicate the appearance of the lesions on T1-weighted and proton density–weighted images. In the Mirowitz et al study, the normal areas are bilaterally symmetric and of low signal intensity on T1-weighted images and isointense or hypointense to surrounding structures on proton density–weighted images. However, pathologic areas of degeneration in motor neuron disease could demonstrate high signal intensity on proton density–weighted images (2).

The diagnosis of motor neuron disease is made on the basis of the pathologic, clinical, neurophysiologic, and muscle biopsy findings. I agree that the presence of high signal intensity in the pyramidal tract (bilateral precentral

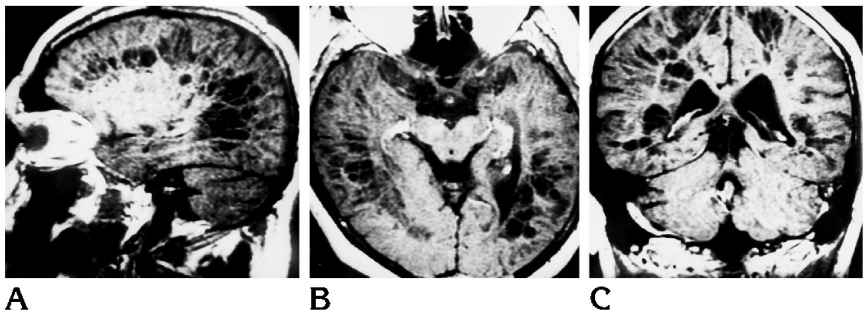
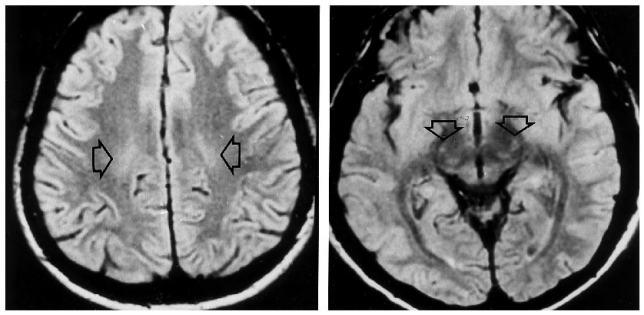
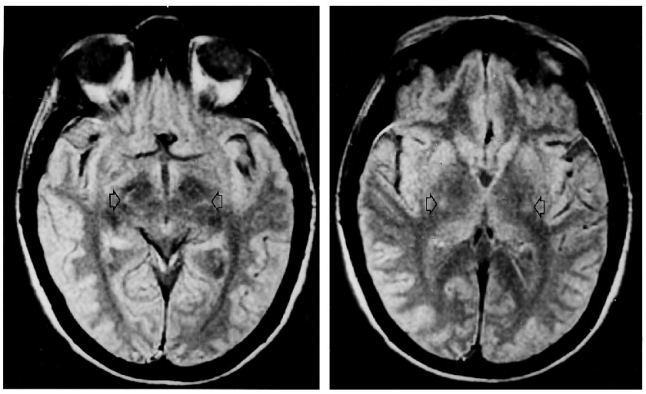


Fig 3. On sagittal (*A*) (400/15), axial (*B*) (400/16), and coronal (*C*) (400/16) T1-weighted spin-echo MR images, multiple lesions are seen in both cerebral hemispheres.



A B

Fig 4. Case 1 (primary lateral sclerosis). Axial proton density–weighted spin-echo MR images (1800/40/2) show symmetric hyperintensity (*arrows*) in the centrum semiovale (*A*) and in the cerebral peduncle (*B*).



A B

Fig 5. Case 2 (amyotrophic lateral sclerosis). Axial proton density–weighted spin-echo MR images (2240/40/2) show symmetric circumscribed hyperintensity (*arrows*) in the cerebral peduncle (*A*) and in the internal capsule (*B*).

cortices) on T2-weighted images is a useful MR finding supporting the diagnosis of motor neuron disease. However, this finding might be insufficient to assure the diagnosis of motor neuron disease. I believe that T2-weighted images showing areas of high signal intensity in the corticospinal tract must be analyzed with caution and compared with T1-weighted and, even more important, proton density–weighted images to differentiate normal areas from those of degeneration.

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1. Mascalchi M, Salvi F, Valzania F, et al. Corticospinal tract degeneration in motor neuron disease. *AJNR Am J Neuroradiol* 1995;16: 878–880
2. Mirowitz S, Sartor K, Gado M, Torack R. Focal signal-intensity variations in the posterior internal capsule: normal MR findings and distinction from pathologic findings. *Radiology* 1989;172:535–539

Reply

We appreciate the interest of Dr Guermazi in our report of symmetric signal abnormalities on MR images in the cerebral and cervical corticospinal tracts of two patients with motor neuron disease. Because we were aware of the important study of Mirowitz et al pointing out that symmetric hyperintensity in T2-weighted spin-echo images, possibly related to lightly myelinated fibers of the parietopontine tract, can be observed in the posterior internal capsule in about 50% of "normal" brain examinations, we did obtain proton-density spin-echo images corresponding to the T2-weighted images shown in the article. Sharing the view of Dr Guermazi that caution is needed in evaluating signal abnormalities of corticospinal tracts, we are pleased to show (Figs 4 and 5) that high signal intensity of the intracerebral corticospinal tracts in our two patients was present on proton density–weighted spin-echo images too. Unfortunately, axial T1-weighted images of the brain and axial T1-, proton density–, or T2-weighted spin-echo images of the cervical spinal cord were not obtained.

We would like, however, to note that corticospinal tract hyperintensity on proton density– and T2-weighted images of the brain of patients with motor neuron disease was previously emphasized in several articles which appeared in neurology journals were quoted in our report (1–3). In our opinion, the original contribution of our paper was the demonstration of the extension of the signal changes to the lateral columns of the cervical spinal cord. This feature, besides ruling out the possibility that the intracerebral signal changes in our patients correspond to the parietopontine tracts, might constitute an indirect clue to the diagnosis of motor neuron disease in patients referred for cervical spine MR imaging to exclude other causes of spastic tetraparesis with or without amyotrophy.

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1. Goodin DS, Rowley HA, Olney RK. Magnetic resonance imaging in amyotrophic lateral sclerosis. *Ann Neurol* 1988;23:418–420
2. Sales Luis ML, Hormigo A, Mauricio C, Alves MM, Serrao R. Magnetic resonance imaging in motor neuron disease. *J Neurol* 1990; 237:471–474
3. Marti-Fabregas J, Pujol J. Selective involvement of the pyramidal tract on magnetic resonance imaging in primary lateral sclerosis. *Neurology* 1990;40:1799–1800