Meningeal enhancement in multiple sclerosis truth or coincidence?

R I Grossman

AJNR Am J Neuroradiol 1992, 13 (1) 401-402
http://www.ajnr.org/content/13/1/401.citation

Commentary

Meningeal Enhancement in Multiple Sclerosis Truth or Coincidence?

Robert I. Grossman[1]

Barkhof et al (1) present an unusual report on a patient with definite multiple sclerosis including the recent onset of new symptoms developing over a 2-month interval. Long TR images demonstrate high intensity lesions in the periventricular region consistent with the clinical diagnosis of multiple sclerosis. Following intravenous administration of gadopentate dimeglumine, intraparenchymal enhancement is observed in the temporal lobe, as well as a most unusual magnetic resonance (MR) finding—diffuse enhancement of the leptomeninges. The radiologic observation is unambiguous. However, the etiology of this finding is shrouded. Many problems arise if one is to attribute the diffuse meningeal enhancement to multiple sclerosis with disseminated infiltration of the leptomeninges by inflammatory cells. These concerns are enumerated below:

1. The cerebrospinal fluid findings are not consistent with a pervasive leptomeningeal inflammatory process. Although possible, it would have been more suggestive if oligoconal bands and lymphocytes were present.

2. The patient, despite having widespread meningeal enhancement, was completely asymptomatic. As stated by the authors, patients with multiple sclerosis may have associated symptoms of leptomeningeal irritation, such as headache and photophobia. These symptoms have been attributed to lymphocytic infiltration of the leptomeninges (2). The pathologic explanation of the radiologic finding of enhancement, would be on firmer grounds if these symptoms had been present and the cerebrospinal fluid findings were positive.

3. With the liberal use of gadopentetate dimeglumine enhancement in routine MR, as well as many serial studies in multiple sclerosis, including patients with acute exacerbations, it is interesting that extensive leptomeningeal enhancement has not been reported (3–6). This is particularly troublesome if significant leptomeningeal involvement was demonstrated in 41% of autopsy-proven cases (7).

4. It is difficult to correlate what Guseo and Jellinger, Silberberg, and Adams have written with what the authors present (7, 8). In the cases described the meningeal infiltrations were usually found in close proximity to active demyelination, especially in the depths of sulci (7). Such areas should be focal and near plaques. The enhancement pattern of the leptomeninges described by Barkhof et al (1) is not focal but is diffuse over the whole brain, involves the falx, and bears no relationship to the intraparenchymal lesions. The MR images do not seem consistent with the quoted pathologic descriptions.

5. No information is provided regarding the temporal relationship between the lumbar puncture and enhanced MR. Is it possible that the enhancement could have been related in some way to the lumbar puncture?

The authors have identified a number of different etiologies of leptomeningeal enhancement. I would be quite concerned that, given the above discrepancies, another, as yet unknown, source is responsible for the MR picture. While the presence of meningeal enhancement and the diagnosis of multiple sclerosis are present in the patient described, it would be wrong for this report to be taken out of context and for leptomeningeal enhancement to be an unequivocal MR sign of multiple sclerosis. For that matter, in patients displaying leptomeningeal enhancement and receiving steroids for acute multiple sclerosis, extreme caution is advised before attributing this MR finding to being consistent with multiple sclerosis (per this case report) rather than working the patient up for infection, etc. The postulate concerning the role of an altered blood-brain barrier in the pathogenesis of multiple sclerosis is intriguing and provocative, yet the present case description is unconvincing in support of this hypothesis.

[1] Department of Radiology, Hospital of the University of Pennsylvania, 3400 Spruce St., Philadelphia, PA 19104. Address reprint requests to R. I. Grossman.

Index terms: Sclerosis, multiple; Demyelinating disease; Contrast media, paramagnetic

AJNR 13:401–402, Jan/Feb 1992 0195-6108/92/1301-0401 © American Society of Neuroradiology

References

1. Barkhof F, Valk J, Hommes OR, Scheltens P. Meningeal Gd-DTPA enhancement in multiple sclerosis. *AJNR* 1992;13:397–400
2. Silberberg DH. Pathogenesis of demyelination. In: McDonald WI, Silberberg DH, eds. *Multiple sclerosis.* London: Butterworths, 1986:99–111
3. Grossman RI, Braffman BH, Brorson JR, Goldberg HI, Silberberg DH, Gonzalez-Scarano F. Multiple sclerosis: serial study of gadolinium-enhanced MR imaging. *Radiology* 1988;169:117–122
4. Miller DH, Rudge PI, Johnson G, et al. Serial gadolinium-enhanced magnetic resonance imaging in multiple sclerosis. *Brain* 1988;111:927–939
5. Kermode AG, Tofts PS, Thompson AJ, et al. Heterogeneity of blood-brain barrier changes in multiple sclerosis: an MRI study with gadolinium-DTPA enhancement. *Neurology* 1990;40:229–235
6. Harris JO, Patronas NJ, Frank JA, McFarlin DE, McFarland H. *The natural history of multiple sclerosis lesions in relapsing-remitting patients between clinical relapse by serial Gd-DTPA.* Presented at the 9th Meeting of The Society of Magnetic Resonance in Medicine, August 1990, New York
7. Guseo A, Jellinger K. The significance of perivascular infiltrations in multiple sclerosis. *J Neurol* 1975;211:51–60
8. Adams CW. The general pathology of multiple sclerosis: morphological and chemical aspects of the lesion. In: Hallpike JF, Adams CWM, Toutelotte WW, eds. *Multiple sclerosis: pathology, diagnosis and management.* London: Chapman and Hall, 1983:203–240