CORRESPONDÊNCIA / CORRESPONDENCE

Fulminant form of multiple sclerosis simulating brain tumor: a case with parkinsonian features and pathologic study. *Maranhão-Filho PA, Moraes L Filho, Camara LSA, Salema CC.* Arq Neuropsiquiatr 1995;53(3-A):503-508.

To the Editor:

I would like to comment on the article by Maranhao-Filho entitled "Fulminant form of multiple sclerosis simulating brain tumor". I must disagree with the diagnosis of multiple sclerosis (MS) in this case. The MRI images are very much against that diagnosis but on the contrary are pathognomonic for acute disseminated encephalomyelitis.A number of images of that type had been published. *See* Annals of Neurology 1994;36(S2):S231-S243 and Journal of Neurology Neurosurgery and Psychiatry 1995;59:467-470.

In that disease, as opposed to MS, gray matter, including the basal ganglia are not infrequently involved. I should add, however, that symptoms of basal ganglia involvement do occur in MS although they are quite unusual. The spinal fluid findings in both MS and acute disseminated encephalomyelitis can be identical, as in this case, although what may help differentiate between the two is the disappearance of such oligoclonal bands; something that never happens in MS where oligoclonal bands, when present, remain for the life of the individual. The biopsy findings are interesting especially because of the lack of inflammatory changes which again is not unusual after the acute phase of the disease has passed.

The apparent biphasic course of the illness may well have been the result of the prolonged effect of the corticosteroids which he received at the time of the initial episode although details of dosage, route of administration and duration of treatment are not given.

While acute disseminated encephalomyelitis is usually regarded as a single phase disease with the development of illness lasting a very short time, there are many cases in which the progression is slower extending over a matter of 4 to 6 weeks. Other types, including recurrent disseminated encephalomyelitis as well as multiphasic disseminated encephalomyelitis have been described in the two papers that I have referred you to.

The problem of differentiating this condition from a brain tumor on the basis of MRI should never occur: the excellent Fig.1B illustration leaves absolutely no doubt about the non-tumoral nature of the lesion since it would highly unlikely for a massive tumor to produce nothing more than "slight" right hemiparesis.

Boston, September 26, 1995

Charles M. Poser, M.D. Department of Neurology, Beth Israel Hospital, Harvard Medical School, 330 Brookline Avenue, Boston MA 02215, USA. FAX (1617)667-5216.

The Author's repply:

To the Editor:

Thank you for the letter containing Dr. Poser's comments on our manuscript entitled "Fulminant form of multiple sclerosis simulating brain tumor: a case with parkinsonian features and pathologic study" by PA Maranhão-Filho, L Moraes Filho, LSA Camara and CC Salema, which was published in Arq Neuropsiquiatr 1995;53:503-508. Let me now address the criticisms pointed in his letter:

1. Our patient did not have any viral disease or received any sort of vaccine prior to the disease onset, as it would be expected in acute disseminated encephalomyelitis (ADEM). The course was biphasic, again not typical of ADEM, a monophasic condition. The second episode was particularly severe. The patient is still under oral cortisone, following unsuccessful trials with methylprednisolone (1g intravenously daily for five days) and plasmapheresis. Even though it is impossible to differentiate multiphasic DEM (MDEM) from multiple sclerosis (MS) on a clinical basis[5]. This picture does not suggest typical MDEM, in which the prognosis is much better[3].

2. As far as the MR scans are concerned, we based our report on previous publications showing images similar to ours (*see* Br Med J 1992; 285:1616-1617, Arq Neuropsquiatr 1983; 41:171-181, Neuroradiology 1992; 34:150-154, J Neurol Neurosurg Psychiatry 1989;52:903-906, Radiology 1991;180:467-474, J Neurol Neurosurg Psychiatry 1982;45:802-808). We should now add Arq Neuropsiquiatr 1995;53(2):302-306, that was not cited in our article. In our case, it was remarkable the development of new images, which does not occur in recurrent DEM (RDEM)[3], and the presence of an ovoid white matter lesion paralleling the main cerebral axis, that was found in 86% of the MS cases[1]. Although basal ganglia involvement in MS is rare, it may occur most frequently as paroxysmal movement disorders such as paroxysmal dystonia[7] and less frequently as non paroxysmal movement disorders like chorea, ballism, choreoathetosis and parkinsonism[8]. Basal ganglia involvement in MR imaging has been found in 25% of MS patients[6].

3. The CSF examination is similar in DEM, MDEM, RDEM and MS. However, in DEM, in contrast to our case, oligoclonal bands are rare[3].

4. The biopsy does not help in the differential diagnosis, as in DEM, MDM, RDEM and MS there is demyelinization without inflammatory infiltrate, provided the lesion is chronic[2].

5. Concerning the differential diagnosis with brain tumor, it is noteworthy that the patient presented with headache, dysphasia, progressive right hemiparesis, together with CT (not shown) and MR scans showing a single left-sided temporo-parieto-occipital lesion enhanced by contrast with considerable perilesional oedema and mass effect. Such aspects are not found in DEM[3] (Fig 1a in our paper). This should promptly raise the possibility of a tumor. The lesion disappeared after corticoid administration, which eventually occur in primary central nervous system lymphomas. We believe therefore that the title of our article is perfectly adequate.

6. Finally, it should be emphasized that distinguishing ADEM from MS may be sometimes difficult and that both share similar treatments. ADEM may be a forme fruste of MS or part of a continuum of demyelinating diseases which includes MS[4].

Rio de Janeiro, October 20, 1995.

Péricles A. Maranhão Filho, Department of Neurosurgery, National Institute of Cancer (INCa), Praça Cruz Vermelha 23, 20230-130 Rio de Janeiro RJ, Brasil. FAX (5521) 439-5594.

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PS: We suppose that the first reference suggested by Dr. Poser in his comments should be Ann Neurol 1994;36(S2): S180-S193 and not S231-S243, as mentioned.