

Multiple sclerosis simulating brain tumor

Amer A. Shurbaji, MD, JBNS, Munir A. Dehayyat, MRCP, JBN, Imad A. Abu-Ruman, MRCP, Ammar F. Mubaidin, MRCP, JBN.

ABSTRACT

We report a case of a 29-year-old female patient with atypical magnetic resonance image appearance of multiple sclerosis simulating brain tumor on magnetic resonance images, that proved to be a demyelinating disease on brain biopsy. Steroid pulse therapy produced regression of the lesions on magnetic resonance images.

Keywords: Multiple sclerosis, brain tumor, magnetic resonance image, demyelination.

Neurosciences 2002; Vol. 7 (2): 134-137

Multiple sclerosis (MS) is the most common demyelinating disease of the central nervous system (CNS). The misdiagnosis rate of MS remains at 5 to 10%, and is attributed to over diagnosis based on brain magnetic resonance imaging (MRI) findings. Large intracerebral lesions that looked more like tumors or abscesses have been described in MS patients. The diagnosis of these lesions presents difficulties, and the differential diagnoses include MS and its variants, neoplastic, infectious and vascular processes. In this paper we report a case of a 29-year-old female patient with atypical MRI appearance of MS simulating brain tumor on MR images, that proved to be a demyelinating disease on brain biopsy.

Case Report. A 29-year-old female patient with no previous medical history of note was referred to the Neurosurgical Department at King Hussein Medical Center as a suspicious case of brain metastasis for further investigation and management. Her history dates back to 4 weeks prior to presentation, when she woke up one morning complaining of pain and blurry vision in the left eye. The pain she describes was dull, aching, increased by moving her eyes, but with no associated redness or discharge from her eyes. Vision in her left eye

deteriorated rapidly in the next few days to the extent that she was unable to see moving objects. There was no history of motor, sensory, or sphincteric disturbances. General physical examination revealed no abnormality. Neurological examination confirmed the presence of swollen left optic disc, along with a relative afferent pupillary defect in the left eye, with a visual acuity of 6/60 in the left eye. However, the rest of her neurological examination was unremarkable. A brain MRI was carried out and showed evidence of 2 rounded enhancing lesions in both frontal lobes (**Figure 1**), measuring approximately 3 x 3 cm each, surrounded by brain edema, with minimal mass effect (**Figure 2**), picture suggestive of multiple brain metastasis (**Figures 3 & 4**). She was fully investigated to detect any primary focus by chest, abdominal, and pelvic computerized tomography (CT) scan, mammogram, thyroid ultrasound; all were negative. Complete blood count, liver, and kidney function tests were also normal. A brain biopsy was carried out through small left lateral frontal craniotomy. No definite mass was detected even with the help of intra-operative ultrasound. The only macroscopic findings were abnormal white matter, which contained streaks of gray color tissue. Multiple biopsies were taken from different parts of the lesion, and the frozen section was not in favor of

From the Departments of Neurosurgery (Shurbaji) and Neurology (Dehayyat, Abu-Ruman, Mubaidin), King Hussein Medical Center, Amman, Jordan.

Received 3rd September 2001. Accepted for publication in final form 14th November 2001.

Address correspondence and reprint request to: Dr. Amer A. Shurbaji, Consultant Neurosurgeon, King Hussein Medical Center, PO Box 125, Amman 11118, Jordan. Tel. +962 (6) 5622267. Fax. +962 (6) 617433. E-mail: anoud@hotmail.com



Figure 1 - Brain magnetic resonance image showing 2 rounded lesions in both frontal lobes, measuring about 3 x 3 cm each.

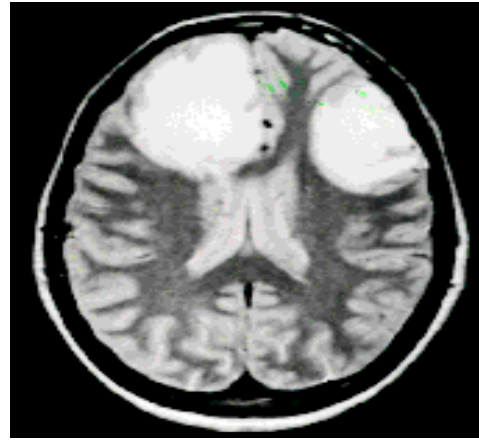


Figure 2 - Brain magnetic resonance image showing 2 rounded lesions in both frontal lobes, with surrounding edema and minimal mass effect.



Figure 3 - Coronal MRI, showing 2 rounded lesions in both frontal regions with surrounding edema picture suggestive of brain metastasis.

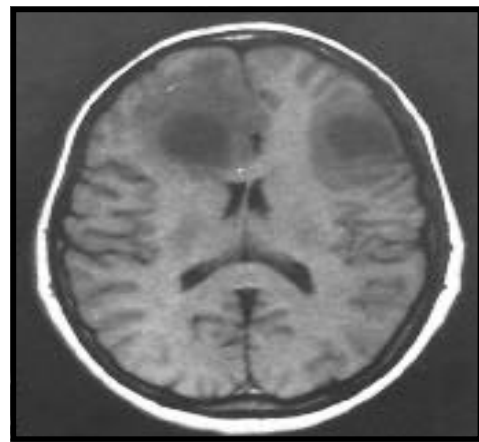


Figure 4 - Axial brain MRI, showing the 2 lesions of the same patient.

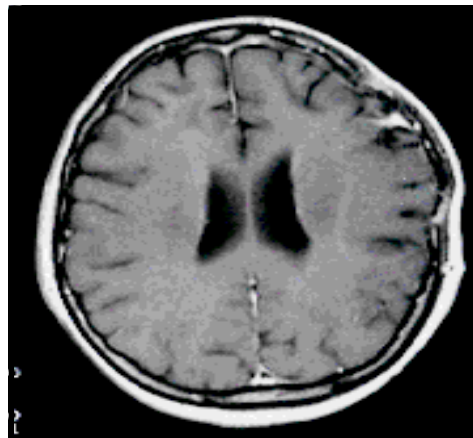


Figure 5 - Follow-up axial brain magnetic resonance image 6 months later showing complete resolution of the 2 lesions.

neoplastic process. The histopathological reports confirmed the diagnosis of demyelinating disease.

Cerebrospinal fluid analysis showed monocytosis of 12 cells, protein of 87 mg/dl, glucose of 65 mg/dl, and a positive oligoclonal band. Visual evoked potential was carried out, and showed evidence of delayed half and whole field from the left eye. Methylprednisolone 1gr/day for 5 days was initiated. A repeat brain MRI was carried out 6 months later which showed significant regression in the size of the previously mentioned lesions (**Figure 5**).

Discussion. Multiple sclerosis is the most common demyelinating disease of the CNS. The age of onset peaks between 20 and 30 years. Almost 70% of patients manifest symptoms between ages 21 and 40. Disease rarely occurs prior to 10 or after 60 years of age. However, patients as young as 3 and as old as 67 years of age have been described. Like other immune-mediated diseases, females are affected more frequently than males (1.4 to 3.1 times as many women than men affected). There appears to be a genetic element involved in the pathogenesis of MS. The data to date suggests that the inheritance of MS is multigenic. There is also a considerable body of evidence supporting a role for environmental agents in the pathogenesis of MS. Infectious diseases have been the most popular, of the proposed environmental factors.

Multiple sclerosis causes inflammation within the brain and spinal cord, primarily in white matter. The inflammatory infiltrate consists of lymphocytes, plasma cells and macrophages. The mononuclear cell inflammation is most prominent in the acute phases of the illness. In more chronic lesions, there is astrocytosis and loss of oligodendrocytes, producing the "plaque" of MS. The cardinal feature of the pathology of MS is demyelination, namely, the loss of myelin with relative sparing of axons. The destruction of myelin in MS will ultimately produce either slowing of conduction or complete conduction block, due to altered conduction of nerve impulses through myelinated fibers of the CNS, which in turn results in the symptoms and signs of CNS dysfunction. It is important to note that patients with MS have subjective complaints and objective signs that frequently are not attributable to one specific lesion in the CNS. It is usually possible to distinguish at least 2 or more separate foci of involvement based on the clinical assessment of the patient.

Multiple sclerosis most often is characterized by episodes of neurological dysfunction followed by periods of stabilization or partial to complete remission of symptoms. These symptoms (relapses or exacerbation's) can appear over a few hours or days, can be gradually worsening over a period of a few weeks, or sometimes can present themselves acutely. Depending on a course and a subtype of the

disease, these symptoms will either persist or slowly resolve over weeks or months and may even culminate as a complete remission. A relapsing-remitting pattern is the most common and is characteristic for this disease, and seen in 55% to 85% of the cases. Older patients and men are more likely to have chronic progressive MS. Primarily progressive MS is seen in only 15% of patients.

In most cases of MS, the clinical course is variable with the vast majority of patients having mild to moderate disabilities that develop over the course of years.¹ At present there are no tests in which an abnormality is specific for MS. Nonetheless, clinical examinations and diagnostic testing including MRI, lumbar puncture and evoked potentials are very helpful.

With the advent of MRI in 1981, imaging of MS entered a new era. Unfortunately, the misdiagnosis rate of MS remains at 5 to 10%. Misdiagnosis in the current era of medical sophistication is attributable to overdiagnosis based on brain MRI findings. Nevertheless, unusual conditions can mimic MS. Certain features are typical of MS on MRI. Bright lesions on T2-weighted images, especially in a periventricular location (often radiating out from the ventricle, "Dawson's fingers"); a lesion >5 mm and lesions below the tentorium, especially in the cerebellar peduncle, help confirm the diagnosis.² Some acute MS lesions enhance after gadolinium infusion but chronic plaques often do not, pointing to lesions separated in time. Up to now, there is no pathognomonic test to ascertain the diagnosis, and in 10 to 15% of MS cases there are problems in differential diagnosis.

Large intracerebral lesions that looked more like tumors or abscesses have been described in MS patients. The lesions were sharply demarcated cystic formations with margins that enhanced with contrast. The diagnosis of these lesions presents difficulties, and the differential diagnoses include MS and its variants, neoplastic, infectious and vascular processes.³⁻¹⁰ These lesions may be characterized on T2-weighted images by a well-defined rim of increased signal intensity and a concentric region of higher signal intensity. On T1-weighted images these lesions were evident as regions of low signal intensity, often with a rim of contrast enhancement or increased signal intensity. These appearances tended to be shown by new, evolving lesions.¹¹

This case report, in conjunction with those in the literature, indicates that conservative treatment with steroid therapy and serial MRI imaging should establish the diagnosis of MS without the need for surgical intervention.⁸ If a clear recovery was not achieved in a relatively short term, or when the clinical presentation begins with signs suggestive of a neoplasm, a stereotactic brain biopsy is recommended, so that neoplastic and other non-

tumorous lesions can be excluded.^{12,13}

References

1. Jaspersen J, Jones AG. A case of rapid deterioration: acute multiple sclerosis of the Marburg type. *J Neurosci Nurs* 1998; 30: 350-355.
2. Offenbacher H, Fazekas F, Schmidt R, Freidl W, Flooh E, Payer F et al. Assessment of MRI criteria for a diagnosis of MS. *Neurology* 1993; 43: 905-909.
3. Einig M, Higer HP, Mauz M, Ernst JP. Intracranial tumor-like lesions in children and young adults with multiple sclerosis. *Rofo Fortschr Geb Rontgenstr Neuen Bildgeb Verfahr* 1992; 157: 384-389
4. Giang DW, Poduri KR, Eskin TA, Ketonen LM, Friedman PA, Wang DD et al. Multiple sclerosis masquerading as a mass lesion. *Neuroradiology* 1992; 34: 150-154.
5. Ohkawa S, Mori E, Ohsumi Y, Tabuchi M, Yamadori A. A case of acute multiple sclerosis mimicking tumour on the neuro-imaging studies. *Rinsho Shinkeigaku* 1992; 32: 1277-1280.
6. Kurihara N, Takahashi S, Furuta A, Higano S, Matsumoto K, Tobita M, et al. MR imaging of multiple sclerosis simulating brain Tumour. *Clin Imaging* 1996; 20: 171-177.
7. Domzal T, Orłowska E. Multiple sclerosis with the symptoms of brain tumour. *Neurol Neurochir Pol* 1990; 24: 98-103.
8. Paley RJ, Persing JA, Doctor A, Westwater JJ, Roberson JP, Edlich RF. Multiple sclerosis and brain tumor: a diagnostic challenge. *J Emerg Med* 1989; 7: 241-244.
9. Dierckx IM, Appel B, Mortelmans LL. Multifocal intracranial tumoral lesions: neuroimaging and differential diagnosis with non-oncologic pathology. *J Belge Radiol* 1990; 73: 7-14.
10. Maranhao-Filho PA, Moraes Filho L, Camara LS, Salema CC. Fulminant form of multiple sclerosis simulating brain tumor: a case with parkinsonian features and pathologic study. *Arq Neuropsiquiatr* 1995; 53: 503-508.
11. Yetkin Z, Haughton VM. Atypical demyelinating lesions in patients with Multiple sclerosis. *Neuroradiology* 1995; 37: 284-286.
12. Braus DF, Schwechheimer K, Volk B, Munding F. The significance of stereotaxic brain biopsy in atypical multiple sclerosis. *Nervenarzt* 1989; 60: 700-705.
13. Amer Ferrer G, Isla A, Diez Tejedor E, Roda JM, Hernandez Perez MA, Barreiro Tella P. Multiple sclerosis lesions