

# Simultaneous Radiologic Isolated Syndrome and Glioblastoma Multiforme in an Adult Patient

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## ABSTRACT

A young known case of multiple sclerosis was evaluated with chief complaint of gradually progressed left lower limb weakness over 3 weeks. Pathologic examination of a mass in brain magnetic resonance imaging confirmed the diagnosis of glioblastoma multiforme (GBM). Given the importance of the definite diagnosis of malignant glioma and its effect on patient's management, GBM should be considered as a differential diagnosis in cases with coexisting mass like lesion and demyelinating plaques.

**Keywords:** Radiologic Isolated Syndrome; Glioblastoma Multiforme; Tumefactive Multiple Sclerosis

ICNSJ 2015; 2 (4) :149-151

www.journals.sbmu.ac.ir/neuroscience

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**Received:** March, 2016

**Accepted:** March, 2016

## INTRODUCTION

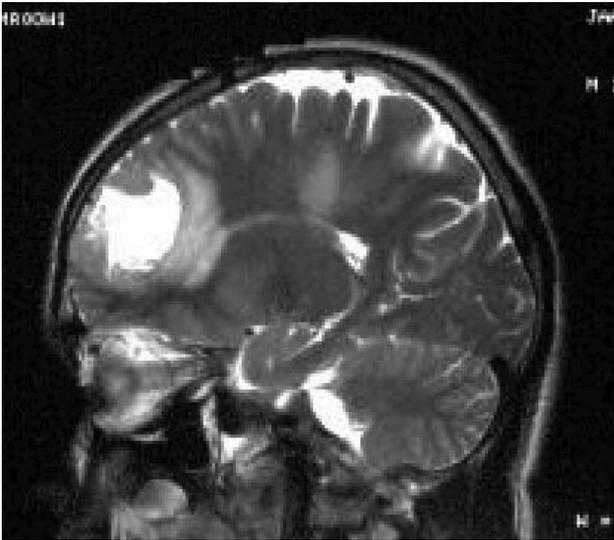
Multiple sclerosis (MS) is an inflammatory disease of central nervous system, which is characterized by myelin damage in the brain and spinal cord. Although it is uncommon, the concurrence of space occupying lesions along with multiple sclerosis plaques has been reported<sup>1</sup>. It is important to differentiate tumefactive MS from other space occupying lesions, such as neoplasms because management and prognosis are different. In comparison with high grade malignancies, there are more reports about the coexistence of MS and low grade tumors<sup>2</sup>. We reviewed a case with a rare coexistence of MS and glioblastoma multiforme (GBM).

## CASE PRESENTATION

A young patient, admitted in our department with chief complaint of left lower limb weakness which gradually progressed over 3 weeks. The patient had no prior history of neurological problems. Systemic physical examinations were completely normal. Abnormal findings in neurological examination were decreased muscle power of the left lower limb, left Babinski sign and impaired Luria test (a test for evaluating frontal lobe).

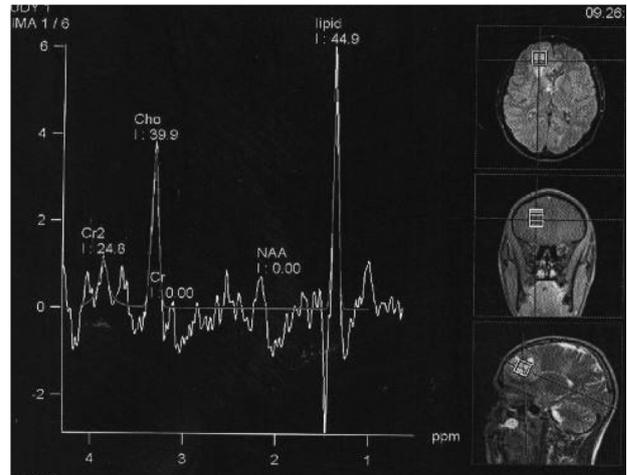
Laboratory test including vasculitis marker, Venereal Disease Research Laboratory, serum angiotensin-converting enzyme and human immunodeficiency virus antibody were negative. Axial brain CT scan revealed a hypodense mass lesion in the right frontal lobe with surrounding edema. In brain magnetic resonance imaging (MRI) the lesion was hyper signal in T2 (Figure 1) and FLAIR sequence, hypo signal in T1 and showed incomplete ring enhancement in post contrast images. It was iso to hypointense in diffusion weighted (DWI) imaging (Figure 2) and showed no restricted diffusion in apparent diffusion coefficient (ADC) map. Also multiple oval shape plaques were found in periventricular and pericallosal regions, one of them had Dawson's finger characteristic. These lesions were hyper signal in T2 and FLAIR, isointense in T1 and were hypersignal in both DWI and ADC map. Cervical MRI and visual evoked potential requested and the results were normal.

MRS (magnetic resonance spectroscopy) revealed reduction in N-acetylaspartate (NAA) peak with increase in choline, lipid and choline/Cr ratio (Supplemental Figure 3). Considering the findings of MRI and MRS of right frontal lesion, two most possible differential



**Figure 1.** Sagittal T2 MRI image showing a cortical hyperintense lesion in frontal lobe with surrounding edema along with a hyperintense lesion emanating radially from the body of the corpus callosum (Dawson's finger).

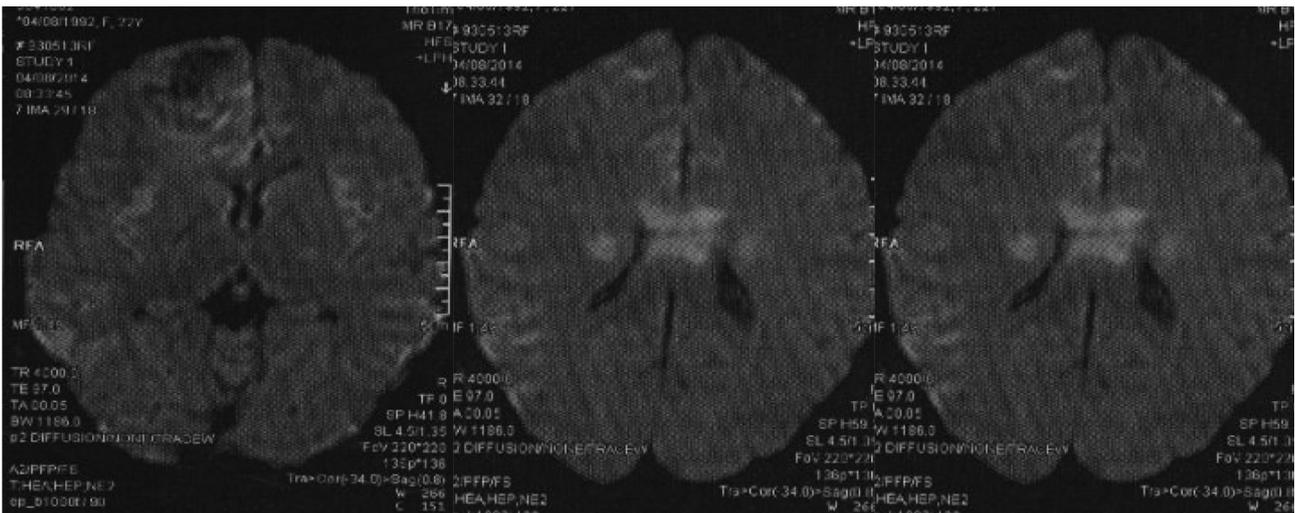
diagnosis were tumefactive MS and low grade glioma. Because of this important diagnostic dilemma the patient referred for histological assessment. Finally pathology of the lesion revealed GBM (Figure 4). Paraffin sections of the neoplasm showed Foci of necrosis surrounded by glial cells with palisading pattern were evident. Hypercellularity and increased mitotic activity were other notable findings. The final histopathologic diagnosis was Glioblastoma Multiforme). Periventricular lesions had typical characteristic of demyelination disease. Indeed, our patient had radiologically isolated syndrome with simultaneous GBM<sup>3</sup>.



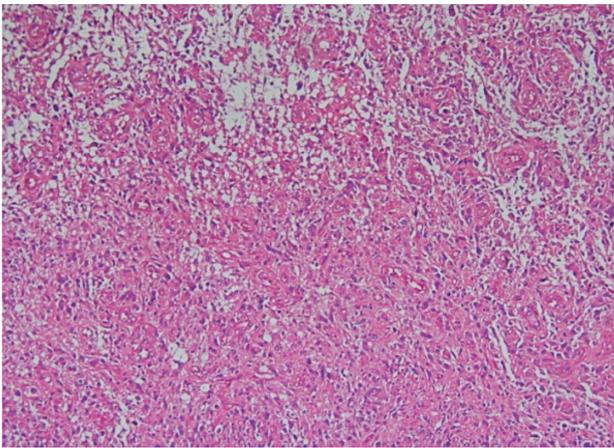
**Figure 3.** MRS of lesion revealed reduction in NAA peak with increase in choline, lipid and cholin/Cr ratio.

### DISCUSSION

MS is an important prevalent neurological disease<sup>4</sup>. Characteristic imaging findings of MS include multiple supratentorial and infratentorial white matter lesions, usually in the periventricular white matter with a slightly surrounding edema and mass effect proportional to the lesion size<sup>5</sup>. Demyelination plaques are hypersignal in T2 and FLAIR. In acute early phase they show reduced diffusion on ADC map which converts to increase ADC map in later stage due to peripheral vasogenic edema<sup>6</sup>. Tumefactive MS is classically present with mass like lesion, with contrast enhancement<sup>7</sup> and may mimic brain tumor or abscess<sup>8</sup>. Some MRI characteristics may help in distinguishing tumefactive MS from brain tumors or abscess. These findings are open ring enhancement, peripheral restriction on DWI and venular enhancement<sup>9</sup>.



**Figure 2.** Axial diffusion-weighted MRI showing no restricted diffusion in ADC map.



**Figure 4.** Paraffin sections of the neoplasm showed proliferation of atypical glial cells and Foci of necrosis with palisading pattern along with Hypercellularity and increased mitotic activity. The final histopathologic diagnosis was Glioblastoma Multiforme.

Significant differences in the *N*-AA/ Creatine ratio in central regions, Is a finding that may help to differentiate Tumefactive demyelinating lesions and high-grade gliomas on MRS<sup>10</sup>. Elevation of the glutamate/glutamine peaks is another MRS useful findings which can help to differentiate these two entity<sup>11</sup>.

Finally in equivocal cases histologic examination will solve the diagnostic dilemma<sup>12</sup>. There are case reports of concurrent MS and brain tumors. Brain tumors are diagnosed more frequently in patients with MS than general population. This increase may be explained by frequent neuroimaging of the central nervous system in patients with MS that reveals symptomatic or asymptomatic brain tumors<sup>13</sup>. An earlier study in Swedish patients with MS reported an increase in the diagnosis of low-grade brain tumors rather than high-grade malignancy<sup>14</sup>.

## CONCLUSION

Although it is not a common presentation, in any case of demyelinating plaque that mimics a brain tumor, even with documented diagnosis of previous MS, the possibility of a concurrence of MS and brain tumor should be strongly considered<sup>15</sup>.

As far as we know there are limited reports of coexisting clinically silent MS and GBM in literature. Given the importance of the definite diagnosis of malignant glioma and its effect on patient's management, GBM should be considered as a differential diagnosis in cases with coexisting mass like lesion and demyelinating plaques.

## ACKNOWLEDGEMENT

This study is supported by clinical research development center of Loghman Hakim hospital, Shahid

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## REFERENCE

1. Nahser HC, Vieregge P, Nau HE, Reinhardt V. Coincidence of multiple sclerosis and glioma. Clinical and radiological remarks on two cases. *Surg Neurol.* 1986 Jul;26(1):45-51.
2. Hofer S, Linnebank M, Weller M, Bahmanyar S, Montgomery SM, Hillert J, et al. Cancer risk among patients with multiple sclerosis and their parents. *Neurology.* 2010 Feb 16;74(7):614-5;
3. Okuda DT, Mowry EM, Beheshtian A, Waubant E, Baranzini SE, Goodin DS, et al. Incidental MRI anomalies suggestive of multiple sclerosis: the radiologically isolated syndrome. *Neurology.* 2009 Mar 3;72(9):800-5.
4. Kurihara N, Takahashi S, Furuta A, Higano S, Matsumoto K, Tobita M, et al. MR imaging of multiple sclerosis simulating brain tumor. *Clin Imaging.* 1996 Jul-Sep;20(3):171-7.
5. Lolekha P, Kulkantrakorn K. Tumor-like manifestation, uncommon form of multiple sclerosis: report of a patient. *J Med Assoc Thai.* 2006 May;89(5):721-6.
6. Balashov KE, Aung LL, Dhib-Jalbut S, Keller IA. Acute multiple sclerosis lesion: conversion of restricted diffusion due to vasogenic edema. *J Neuroimaging.* 2011 Apr;21(2):202-4.
7. Lucchinetti CF, Gavrilova RH, Metz I, Parisi JE, Scheithauer BW, Weigand S, et al. Clinical and radiographic spectrum of pathologically confirmed tumefactive multiple sclerosis. *Brain.* 2008 Jul;131(Pt 7):1759-75.
8. Jain D, Rajesh LS, Vasishtha RK, Radotra BD, Banerjee AK. Demyelinating disease simulating brain tumours: a histopathologic assessment of seven cases. *Indian J Med Sci.* 2006 Feb;60(2):47-52.
9. Malhotra H, Jain K, Agarwal A, Singh M, Gupta R. Characterization of tumefactive demyelinating lesions using MR imaging and in vivo proton MR spectroscopy. *Mult Scler.* 2009; 15:193-203.
10. Saindane AM, Cha S, Law M, Xue X, Knopp EA, Zagzag D. Proton MR spectroscopy of tumefactive demyelinating lesions. *AJNA Am J Neuroradiol.* 2002; 23: 1378-86.
11. Cianfoni A, Niku S, Imbesi SG. Metabolite findings in tumefactive demyelinating lesions utilizing short echo time proton magnetic resonance spectroscopy. *AJNR Am J Neuroradiol.* 2007 Feb;28(2):272-7.
12. Xia L, Lin S, Wang ZC, Li SW, Gao CC. Tumefactive demyelinating lesions: nine cases and a review of the literature. *Neurosurg Rev.* 2009; 2:171-179.
13. Bahmanyar S, Montgomery SM, Hillert J, Ekblom A, Olsson T. Cancer risk among patients with multiple sclerosis and their parents. *Neurology.* 2009 Mar 31;72(13):1170-7.
14. Montgomery S, Hassan A, Bahmanyar S, Brus O, Hussein O, Hiyoshi A, et al. Mortality following a brain tumour diagnosis in patients with multiple sclerosis. *BMJ Open.* 2013 Nov 11;3(11):e003622.
15. Maranhão-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 Sep;53(3-A):503-8.