

Brain radiation and possible presentation of multiple sclerosis

Vahid Shaygannejad^{1,2}, Mohammad Zare^{1,2}, Helia Maghzi², Parisa Emami²

¹Department of Neurology, Isfahan Neurosciences Research Center, ²Department of Neurology, Medical School, Isfahan University of Medical Sciences, Isfahan, Iran

Radiation therapies are commonly used for malignant or metastatic brain tumors for curative and palliative use. The radiation-induced neurotoxicity includes both parenchymal and vascular damage. Here we report the case of a 43-year-old woman who developed Multiple sclerosis (MS) 9 months after she underwent the last session of radiotherapy for the diagnosed meningioma. Conventional doses of radiation might trigger MS.

Key words: Meningioma, multiple sclerosis, radiation therapy

INTRODUCTION

Multiple sclerosis (MS) is thought to be an autoimmune, neurodegenerative and demyelinating disease of central nervous system, which predominantly affects females between ages of 20 and 40 years and is amongst neurological disorders with high-rate of morbidity.

Some evidences have demonstrated the concurrence of MS with central nervous system tumors such as gliomas and also higher prevalence of malignant brain neoplasms in MS patients compared to the general population.^[1-3]

Meningiomas are intracranial tumors which originate from arachnoid cap cells, and are mostly seen in females and older ages.^[4] Some studies reported meningiomas in MS patients in which in a few cases meningioma growth were related to interferon-beta therapy.^[2,5-9]

Unlike other studies here we report a patient with meningioma who developed MS 2 years after the diagnosis of meningioma; and discuss the potential relationship between MS and meningioma with a review of current literature.

CASE REPORT

A 43-year-old woman was admitted to hospital 2 years ago complaining of progressive generalized headache and bilateral blurred vision, with no history of hemiparesis or seizure. Her past medical and familial history was unremarkable for any neurological disorders or other disease. General clinical assessment was within normal limits. Laboratory exams were also

found to be normal. Neurological exam, including visual field and ophthalmoscopy was within normal limits; on her magnetic resonance imaging (MRI) of brain a tumor in optic chiasm was observed.

The mass was restricted and surgically removed. Histological examination revealed meningothelial meningioma, and a diagnosis of meningioma was confirmed. Patient underwent 28 session of radiotherapy and 15 months after the last session of radiotherapy there was no sign of meningioma.

Nine months after the last session of radiotherapy she was referred to the clinic complaining of blurring vision in the right eye. General clinical assessment was normal. Neurological examination revealed visual acuity of 20/200 in right eye, increased deep tendon reflexes (3+) and also bilateral extensor plantar reflexes. The remaining neurological examination and fundoscopy was normal. On her MRI, radiological finding showed multiple lesions in periventricular, centrum semiovale and corpus callosum [Figure 1] in which it showed compatibility with a diagnosis of MS according to 2005 MC Donald's criteria.

Drug therapy for MS with beta-interferon 1a (Avonex) was begun.

DISCUSSION

Amongst the primary non-gliar intracranial tumors, meningiomas are the most common with 20% of all intracranial tumors in men and 38% in women. Smoking, allergy and some genes are thought to play a role in susceptibility to meningioma.^[10,11]

Address for correspondence: Dr. Helia Maghzi, Department of Neurology, Medical School, Isfahan University of Medical Sciences, Isfahan, Iran.
E-mail: helia.maghzi@yahoo.com

Received: 08-01-2013; **Revised:** 11-02-2013; **Accepted:** 16-02-2013

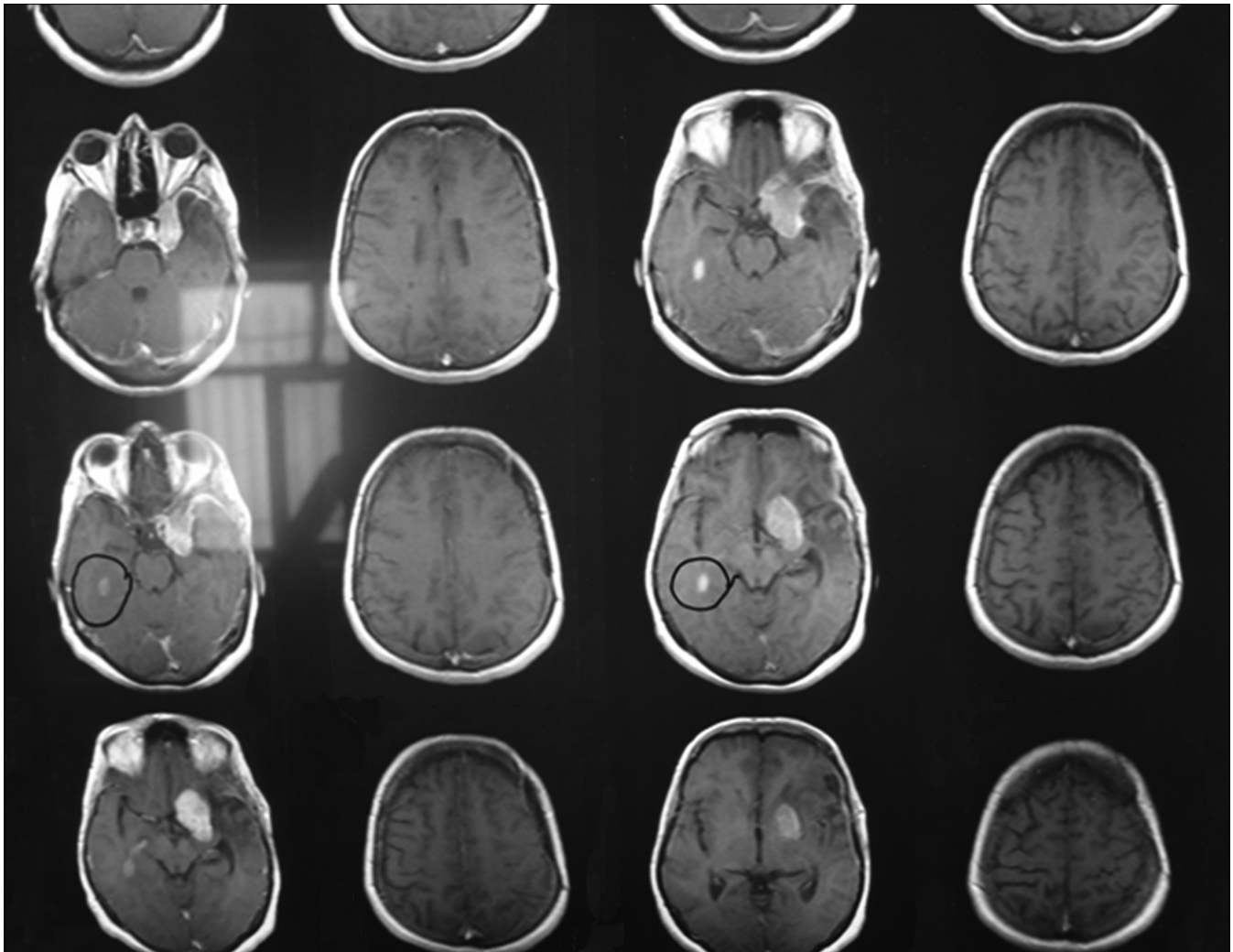


Figure 1: Multiple lesions in periventricular, centrum semiovale and corpus callosum in magnetic resonance imaging of the patient

Radiation therapies are commonly used for malignant or metastatic brain tumors for curative and palliative use. Early delayed radiation induced neurotoxicity which occurs 6-12 week after radiation leads to reversible damages however, the late delayed radiation-induced neurotoxicity is the more severe and irreversible consequence in which it includes both parenchymal and vascular damage.^[12,13]

Nine months after the last session of radiotherapy for meningioma our patient was diagnosed with MS. These results may be due to the role of radiotherapy in damaging blood brain barrier and causing the interaction between immune and central nervous system antigen with white matter, which lead to production of demyelination plaque.

In line with our findings is the case of a patient with clinically quiescent MS who underwent radiotherapy for parotid carcinoma in which it caused exacerbation of symptoms and the newly hyper intensive regions on MRI correspond to regions that are defined by the 50% isodose radiation field.^[14] Also, another case of exacerbation of quiescent

MS was observed after radiotherapy for glomusjugulare tumor with plaque confined to radiation field.^[15] On animals models the radiation-induced demyelination has been reported. A study conducted on radiated rats demonstrated neurological signs and perivascular cellular infiltration of experimental allergic encephalomyelitis.^[16] These results can be due to the reported development of disseminated plaques of demyelination which is seen after radiotherapy.^[15] However, similar lesions can be seen in MS patients because of underlying pre-disposition to demyelination.

CONCLUSION

Here we have reported the case of MS development after patient underwent radiotherapy for the diagnosed meningioma. These results might be due to the effect of radiation on blood brain barrier and the interaction between immune system antigens and whit matter and the production of demyelination plaques. Our findings contribute to the fact that conventional doses of radiation might trigger central nervous system autoimmunity.

REFERENCES

1. Shuangshoti S, Hjarndermaal GM, Ahmad Y, Arden JL, Herman MM. Concurrence of multiple sclerosis and intracranial glioma. Report of a case and review of the literature. *Clin Neuropathol* 2003;22:304-8.
2. Spaar FW, Wikström J. Multiple sclerosis and malignant neoplasms in the central nervous system: A clinical anatomical report of three cases. *J Neurol* 1978;218:23-33.
3. Green AJ, Bollen AW, Berger MS, Oksenberg JR, Hauser SL. Multiple sclerosis and oligodendroglioma. *Mult Scler* 2001;7:269-73.
4. Marosi C, Hassler M, Roessler K, Reni M, Sant M, Mazza E, *et al.* Meningioma. *Crit Rev Oncol Hematol* 2008;67:153-71.
5. Salvi F, Mascalchi M, Plasmati R, Michelucci R, Calbucci F, Dal Pozzo G, *et al.* Multiple lesions in cerebral white matter in two young adults with thoracic extramedullary tumours. *J Neurol Neurosurg Psychiatry* 1992;55:216-8.
6. Batay F, Al-Mefty O. Growth dynamics of meningiomas in patients with multiple sclerosis treated with interferon: Report of two cases. *Acta Neurochir (Wien)* 2002;144:365-8.
7. Costa MF, Novis SA, Niemeyer Filho P, Pimentel ML, Novis RF, Duarte F. Multiple sclerosis, spinal cord ependymoma and intracranial meningioma: Case report. *Arq Neuropsiquiatr* 2000;58:1133-7.
8. Drevelegas A, Xinou E, Karacostas D, Parissis D, Karkavelas G, Milonas I. Meningioma growth and interferon beta-1b treated multiple sclerosis: Coincidence or relationship? *Neuroradiology* 2005;47:516-9.
9. Gama HP, Rocha AJ, Silva CJ, Mendes MF, Veiga JC, Lancellotti CL, *et al.* Meningioma growth during interferon beta-1A treatment for multiple sclerosis. *Arq Neuropsiquiatr* 2008;66:402-4.
10. Vranic A, Peyre M, Kalamarides M. New insights into meningioma: From genetics to trials. *Curr Opin Oncol* 2012;24:660-5.
11. Bondy M, Ligon BL. Epidemiology and etiology of intracranial meningiomas: A review. *J Neurooncol* 1996;29:197-205.
12. Kim JH, Brown SL, Jenrow KA, Ryu S. Mechanisms of radiation-induced brain toxicity and implications for future clinical trials. *J Neurooncol* 2008;87:279-86.
13. LAMPERT PW, DAVIS RL. Delayed effects of radiation on the human central nervous SYSTEM; "Early" and "Late" delayed reactions. *Neurology* 1964;14:912-7.
14. Murphy CB, Hashimoto SA, Graeb D, Thiessen BA. Clinical exacerbation of multiple sclerosis following radiotherapy. *Arch Neurol* 2003;60:273-5.
15. McMeekin RR, Hardman JM, Kempe LG. Multiple sclerosis after x-radiation. Activation by treatment of metastatic glomus tumor. *Arch Otolaryngol* 1969;90:617-21.
16. Paterson PY, Richarson WP, Drobish DG. Cellular transfer of experimental allergic encephalomyelitis: Altered disease pattern in irradiated recipient lewis rats. *Cell Immunol* 1975;16:48-59.

How to cite this article: Shaygannejad V, Zare M, Maghzi H, Emami P. Brain radiation and possible presentation of multiple sclerosis. *J Res Med Sci* 2013;18:S93-S5.

Source of Support: Nil, **Conflict of Interest:** None declared.