

Coexistence of Multiple Sclerosis and Brain Tumor: An Uncommon Diagnostic Challenge

Abstract

Nonneoplastic demyelinating processes of the brain with mass effect on magnetic resonance imaging can cause diagnostic difficulties. It requires differential diagnosis between the tumefactive demyelinating lesion and the coexistence of neoplasm. We document the case of 41-year-old woman with clinical and radiological findings suggestive of multiple sclerosis. Additional investigations confirmed the coexistence of astrocytoma. This report emphasizes the importance of considering brain tumors in the differential diagnosis of primary demyelinating disease presenting with a cerebral mass lesion.

Keywords: *Astrocytoma, brain tumor, multiple sclerosis*

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Introduction

Multiple sclerosis (MS) is a chronic inflammatory demyelinating disease characterized by heterogeneity in clinical symptoms and for that reason may simulate or masquerade other central nervous system (CNS) diseases.^[1,2] Primary demyelinating disease of CNS generally does not produce a focal diffuse mass lesion, a feature that has been used to distinguish demyelinating lesions from the tumor.^[3] MS plaques on magnetic resonance imaging (MRI) generally appear as multiple, well-demarcated, homogenous, small ovoid lesions, lacking mass effect, and often oriented perpendicular to the long axis of the lateral ventricles.^[4] However, patients with MS may develop space-occupying lesions that may be mistaken as neoplasia, and this form of MS is eventually called tumefactive MS.^[5] The occurrence of tumor-like demyelination is reported rare and more commonly occurs in women and young adults, which causes a diagnostic enigma for both clinicians and radiologists.^[5-7] On the other hand, neoplasia may simulate MS at its initial presentation.^[8] Moreover, though uncommon, there might be a coincidence of MS and a CNS neoplasm in the same patient.^[9-12]

We report a case of a patient with a coexistence of MS and astrocytoma.

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Case Report

A 41-year-old right-handed woman who had complained of a 3-year history of progressive ataxia and dizziness was visited by a neurologist. Throughout these years, she was visited several times by general physicians. She had a history of falling along with ataxia, and no blurring of vision was noted. Neurological examination revealed no cranial nerve deficit. MRI demonstrated high-signal lesions in the left frontal lobe involving corpus callosum on fluid-attenuated inversion recovery imaging [Figure 1].

After that, with a diagnosis of MS, according to McDonald criteria (2005) the patient was treated by β -interferon 1a for about 1 year. She had been complained of nausea, vomiting, fatigue, and drowsiness as side effects of β -interferon 1a during therapy. The ataxia, as chief complaint was also continued. After 1 year and with the stress of her husband death, she presented with slowing and clumsiness in a way that she could not walk without help more than 50 m. Neurological examination revealed impaired tandem gait, positive Romberg sign, and positive bilateral Babinsky. New MRI showed that the lesion extended to left frontoparietal lobe [Figure 2].

Although brain tumor was suspected, the patient treated with β -interferon 1a for more

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six months until a magnetic resonance spectroscopy (MRS) was done and a commission of neurologists and neurosurgeons suggested astrocytoma and suspected MS for the patient.

Finally, a neurosurgeon did stereotaxis, and pathology revealed Grade II astrocytoma. With this diagnosis the tumor excised by craniotomy and followed by 1 month of radiotherapy. After that, ataxia and clumsiness reduced but she was not visited by neurologist and received no therapy for MS for 6 months. After that following a severe cold, she presented with difficulty in walking and slowing in a way that could not walk without help more than 10 m. Hence with a diagnosis of relapse of MS she received pulse therapy of corticosteroid for 5 days, twice in 2 weeks and followed by Extavia (β -interferon) for 2 months. With therapy walking ability improved and she could walk about 100 m without help.

During follow-up, neurological examination revealed impaired tandem gate, positive Romberg sign, and positive bilateral Babinsky. Cranial nerves were intact, but quadriplegia (more severe in right side), and clumsiness was present. Last MRI showed multiple high-signal plaques around the corpus callosum and high-signal lesion in left parasagittal area due to excision of the tumor [Figure 3].

Discussion

MS is usually diagnosed by demonstrating clinical and/or radiographic evidence of dissemination of disease in time and space.^[13] Although the usual appearance of MS is that of multiple, small, demyelinating plaques, in some cases, it can simulate a mass lesion, which it would be hard to distinguish from a brain tumor.^[14] One series of MS patients who underwent autopsy showed that 6% of cases diagnosed as MS were, in fact, cases with other diagnosis, including a small number of CNS primary neoplasms (0.57%).^[11] The most common neoplasm mistaken for MS is CNS lymphoma. MRI findings of glioma may also look similar to those of MS with T2-hyperintense lesions in the cerebral white matter extending into corpus callosum and variable gadolinium enhancement.^[3] However, as in our case, low-grade astrocytomas may be sometimes a difficult diagnosis based only on neuroimaging techniques and may be only established by biopsy or radical removal.^[11,15] On the other hand, MS may present as a mass lesion and brain biopsy is generally necessary to confirm the diagnosis.^[5] MRS has shown to be a helpful tool in differentiating astrocytic tumors from demyelinating lesions.^[12] Reduction of N-acetylaspartate and increase in choline may resemble those of brain tumors and acute MS plaques.^[12,14,16] The chronic demyelinating plaque, however, shows a completely different pattern.^[15] A stereotactic biopsy and histologic examination of the lesion is the final diagnostic approach in equivocal cases.^[14] It is safe and reliable, especially if specimens from multiple sites within the lesion are targeted. It has a diagnostic accuracy of

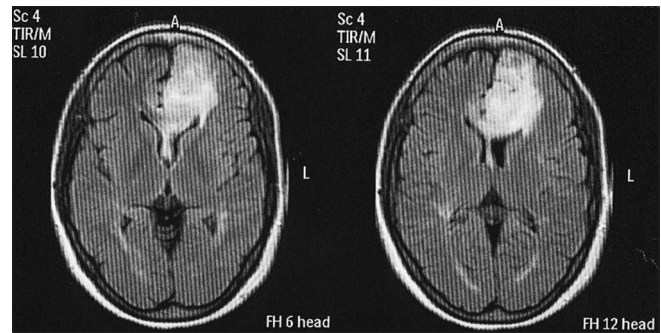


Figure 1: High-signal lesions in the left frontal lobe involving corpus callosum on fluid-attenuated inversion recovery imaging

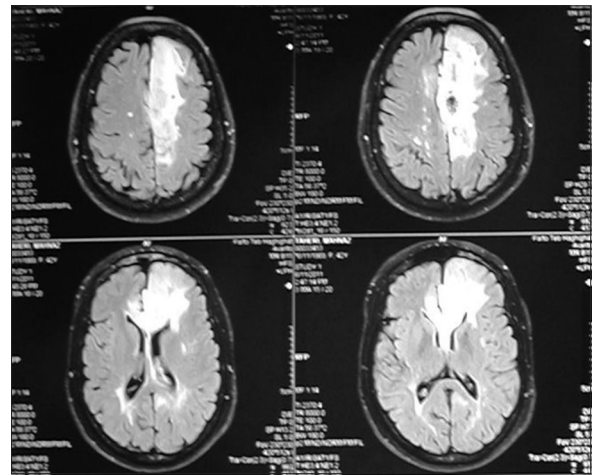


Figure 2: High-signal lesions in the left frontoparietal lobe involving corpus callosum on fluid-attenuated inversion recovery imaging

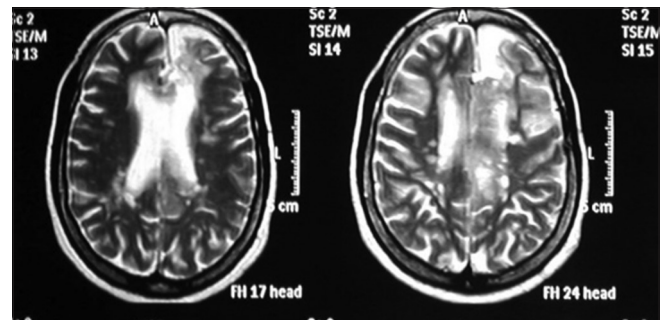


Figure 3: High-signal plaques around the corpus callosum and high-signal lesion in left parasagittal area in T2-weighted imaging

82–99%.^[17] In our case, MRS suggested brain tumor and stereotactic biopsy endorsed the diagnosis. Butteriss *et al.* also reported a case of oligodendroglioma in MS that was diagnosed by surgical removal of the lesion, but that had been considered to be tumefactive MS on preoperative MRI.^[12]

The occasional development of gliomas in cases of MS is now an established fact, but the number of reported cases is still small. In our case as previous cases, the sequence of tumor occurring in the context of longstanding MS went unrecognized.^[12] These reports emphasize the importance

of considering brain tumors in the differential diagnosis of primary demyelinating disease presenting with a cerebral mass lesion.

Conclusion

This scenario will suggest that when we are dealing with a patient with atypical clinical and MRI presentation suggesting MS or brain tumor, and there is an obstacle for brain biopsy: A short course of steroid therapy and follow-up, including imaging, may play a role in clarifying the diagnosis and leading to correct root of therapy. When there is no improvement with steroid therapy or extensions of the single lesion in MRI, other diagnosis such as brain tumor should be ruled out as soon as possible. In the other hand, when brain tumor coexists with MS, after managing the tumor, continuing of correct therapy for MS and following up for possible relapses is very important.

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Conflicts of interest

There are no conflicts of interest.

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