

NMO presenting as MS worsening on natalizumab

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INTRODUCTION

Neuromyelitis optica (NMO) is a disabling CNS demyelinating disease. Treatment is usually general immunosuppression or B-cell targeted therapy. There are conflicting reports about the benefit of natalizumab in patients with NMO.¹⁻³

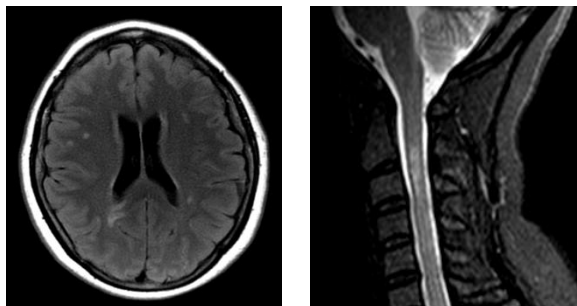
METHODS

Case report of a patient presenting as MS with negative serum NMO Ab who significantly worsened on natalizumab.

RESULTS

In 2008, a 51 year-old Hispanic woman developed bilateral, sequential optic neuritis with incomplete recovery, followed quickly by partial myelitis. MRIs showed multiple enhancing brain lesions and 5-6 small, non-enhancing spinal lesions. Serum NMO-IgG was negative at that time. She was initiated on Copaxone and did well for 2 years.

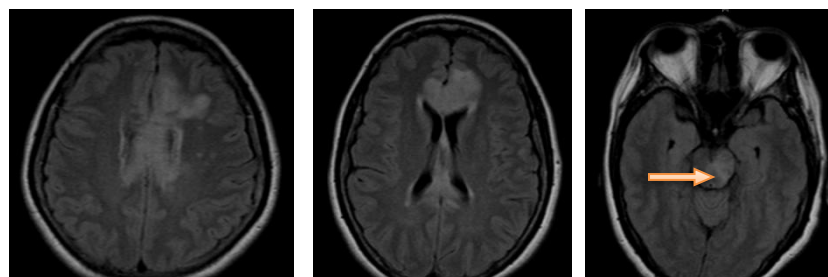
She then had a relapse (fall of 2010) with new MRI lesions. She was switched to Betaseron, but within 4 months had a new relapse and increased LFTs. Due to limited resources, she was switched back to Copaxone. Approximately 6 months later (July 2011) she had another relapse. By this time, she was insured and was started on natalizumab with a negative serum JCV Ab. She received six monthly infusions.



MRI prior to natalizumab

RESULTS CONTINUED

She did well for six months, then developed confusion (asking repetitive questions), aphasia, right sided weakness, numbness and apraxia. Her MRI revealed large, bilateral, T2 hyperintense, subcortical white matter lesions with patchy enhancement. Due to concerns about PML, she was started on PLEX. She had a lumbar puncture and JCV PCR was sent, which came back negative, twice. Initial CSF - WBC 19 with 44N, 28L, glucose 33, protein 153. Repeat CSF - WBC 27, glucose 56, protein 53. Viral studies, gram and stain and culture all negative.



MRI after 6 doses of natalizumab

She continued to decline with worsening right sided weakness. MRI of her spine revealed subtle enhancement at T3-5. With this new finding, her current symptoms were felt to be due to a MS relapse. Therefore she was started on IV methylprednisolone. She had minimal improvement and was transferred to rehabilitation.

By follow-up on 5/22/12 in our clinic, she had significant improvement. After discussion with the patient and her family, it was decided to start her on Cytoxin. She received 2 doses of Cytoxin. Shortly thereafter, she developed left hemiparesis. Repeat MRI C- and T-spine revealed a longitudinally extensive (C1-C7) lesion. Due to this she was re-tested for NMO-Ig G, which came back positive at >160.



MRI June 2012

RESULTS CONTINUED

She was admitted to the hospital on 7/19/12 with confusion and dizziness. She was found to have a multi-drug resistant UTI and was treated with IV antibiotics. During this admission, she developed acute left optic neuritis and was treated with IVMP x 5 days followed by PLEX x 6. After PLEX, she received her first dose of Rituximab.

CONCLUSION

While natalizumab has been shown to inhibit migration of T and B cells across the blood-brain barrier,⁴ it may be that the increased peripheral B-cells, particularly the CD19+ cells, may increase disease activity in patients with NMO. In addition, this case highlights how closely NMO can mimic MS and the importance of re-testing patients for NMO Ab, if clinical suspicion is high.

REFERENCES

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