hospitals in the Mountain West. The treatment regimens and outcomes in this population may inform approaches to current management and future clinical trials.

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A Case of Recurrent Cerebral Cortical Encephalitis in MOG Antibody-Associated Disease

Laura Cacciaguerra, John J. Chen, Eoin P. Flanagan

Objective

NA.

Background

Cerebral cortical encephalitis is a recently recognized syndrome of myelin-oligodendrocyte-glycoprotein-antibody-associated disease (MOGAD), yet most descriptions report single episodes without recurrence.

Design/Methods

Case report of recurrent cerebral cortical encephalitis in MOGAD.

Results

This is a case report of a 32-year-old patient who initially developed meningoencephalitis when he was three. He was stable until age 31, when he developed headaches followed by status epilepticus secondary to cerebral cortical encephalitis with accompanying unilateral right temporal cerebral cortical T2-hyperintensity with leptomeningeal enhancement. Cerebrospinal fluid analysis revealed 505 white cells/μl (normal, 0-5) but oligoclonal bands were negative. Over the subsequent two weeks he developed bilateral severe vision loss (20/400 bilaterally) accompanied by bilateral optic disc edema, and MRI orbits showed bilateral anterior segment optic nerve enhancement extending >50% of the length of each nerve, confirming bilateral optic neuritis. MOG-IgG was tested in serum and was positive at a titer of 1:100. He was treated with intravenous methylprednisolone 1 gram daily for 5 days and a subsequent slow oral prednisone taper along with anti-epileptic treatment with levetiracetam, later transitioned to oxcarbazepine, although he later self-discontinued all anti-epileptics. He received maintenance intravenous immunoglobulins (IVIg) 0.4 g/Kg weekly, that was later weaned to 1 g/Kg every 4 weeks. He then developed recurrent cerebral cortical encephalitis 18 months after his prior episode manifesting with seizures and left occipito-temporal T2-hyperintensity and swelling with leptomeningeal enhancement. He was treated with high dose IV steroids, an oral prednisone taper and his IVIg dose was changed back to 0.4 g/Kg once weekly and anti-seizure medications reinitiated.

Conclusions

Cerebral cortical encephalitis in MOGAD can be recurrent. Close scrutiny of the MRI in patients with MOGAD who develop seizures after prior cerebral cortical encephalitis is important as it can discriminate a recurrent cerebral cortical encephalitis attack from a breakthrough seizure related to prior damage.

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Development of New or Enlarging MRI Lesions Outside of Clinical Attacks in MOG-Antibody-Associated Disease

Stephanie Syc-Mazurek, John Chen, Padraig Morris, Elia Sechi, Jayawant Mandrekar, Jan-Mendelt Tillema, Alfonso Lopez, Claudia Lucchinetti, Nicholas Zalewski, Laura Cacciaguerra, Marina Buciuc, Karl Krecke, Steven Messina, M. Tariq Bhatti, Sean Pittock, Eoin Flanagan

Objective

To determine the frequency of new/enlarging T2 or enhancing asymptomatic lesions in myelin-oligodendrocyte-glycoprotein-antibody-associated-disease (MOGAD) and compare to multiple sclerosis (MS) and aquaporin-4 antibody-positive-neuromyelitis-optica-spectrum-disorder (AQP4+NMOSD).

Background

Data on new asymptomatic lesions in MOGAD is limited.

Design/Methods

We retrospectively identified Mayo Clinic MOGAD patients with inclusion criteria of: 1)MOG-IgG positivity by live-cell-based-assay; 2) Fulfilling current MOGAD diagnostic criteria; 3) Baseline and follow-up paired MRIs without interval attacks. Paired MRIs (baseline and follow-up) were categorized as either attack-to-remission or remission-to-remission scans. A neurologist and neuroradiologist reviewed MRIs (T2-FLAIR brain, T2 spine, and T1-post-gadolinium brain and spine) to identify new/enlarging lesions. A subset of MOGAD patients matched for follow-up interval were compared to MS and AQP4+-NMOSD patients.

Results

We included 105 MOGAD patients (median age, 31 years[range, 3-80]; 60% female) with 373 paired MRIs (brain, 213, spine 160). In total, 13/373 (3%) scans (10/105 patients) had one or more new/enlarging T2-lesions (brain, 12/213[5.6%]; spine, 1/160[0.6%]) and 8/367 (2%) had enhancing lesions. New spinal lesions were rare across all groups (0-4%). T2 lesions occurred more commonly in attack-remission scans (8/171[4.7%]) then remission-remission scans (5/202[2.4%]). Clinical characteristics did not differ between patients who developed new/enlarging lesions and those who did not. Maintenance immunosuppressants were used in 44/105 (42%) patients. New/enlarging lesions did not predict future clinical relapse. New brain lesions were less in MOGAD (1/25[4%]) than MS (14/26[54%], $p < 0.0001$) but did not differ from AQP4+NMOSD (1/13[8%], $p = 1.0$) in subgroup analysis.

Conclusions

New brain MRI lesions rarely develop outside of attacks in MOGAD which differs from MS. Surveillance MRI in MOGAD may have limited utility as a surrogate biomarker of disease activity in clinical practice and for clinical trials.

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