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Recurrent Brainstem Lesions as a Manifestation of MOGAD

Rumyar Ardakani, Kyle Blackburn

Objective

NA.

Background

MOG antibody-associated disease (MOGAD) is an inflammatory and demyelinating disease of the central nervous system. Brainstem and cerebellar involvement in MOGAD is relatively common and typically occurs in conjunction with lesions in other parts of the central nervous system such as the optic nerve, supratentorial white matter, or spine. Isolated attacks of the brainstem and cerebellum are rare—especially in a relapsing form.

Design/Methods

A case report.

Results

We present the case of a 60 year-old female who developed new onset weakness and incoordination of her left arm and leg. Her brain MRI

revealed restricted diffusion and edema involving the left cerebellar peduncle. She was diagnosed with a stroke and discharged home on antiplatelet therapy. One month later, she developed a left facial droop and was diagnosed with a Bell's palsy. She received oral steroids and had improvement of her facial droop, though it returned a few weeks after completion of her oral steroids. A repeat brain MRI at that time revealed enlargement of the left cerebellar lesion with extension into the left pons. She was again treated with oral steroids and her symptoms improved. She presented again 3 months later with dysarthria and pseudobulbar affect with her brain MRI revealing diffuse T2/FLAIR hyperintensities and edema throughout the bilateral pons and cerebellar peduncles. She received 3 days of high-dose IV methylprednisolone with marked improvement. She ultimately had serum MOG antibody testing performed which returned positive (titer of 1:1000). She was initiated on rituximab, and was clinically stable for over 24 months.

Conclusions

Although brainstem involvement in MOGAD usually occurs in conjunction with other lesions, our case demonstrates that relapsing attacks isolated to the brainstem and cerebellum are within the clinical spectrum of MOGAD.

Disclosure: Dr. Ardakani has nothing to disclose. Dr. Blackburn has received personal compensation in the range of \$500-\$4,999 for serving on a Scientific Advisory or Data Safety Monitoring board for Genentech.

Spinal Central Canal Dilation in MOG Antibody-Associated Disease Versus Other CNS Demyelinating Disorders

Lauren Webb, Laura Cacciaguerra, John Chen, Elia Sechi, Vyanka Redenbaugh, Divyanshu Dubey, Sean Pittock, Eoin Flanagan

Objective

To assess the frequency of spinal cord central canal dilation on magnetic resonance imaging (MRI) in patients with myelin-oligodendrocyte glycoprotein antibody-associated disease (MOGAD) myelitis compared to myelitis patients with aquaporin-4-positive-neuromyelitis optic spectrum disorder (AQP4+NMOSD) and multiple sclerosis (MS).

Background

In MOGAD myelitis, a sagittal T2-hyperintense line accompanied by axial T2-signal restricted to the spinal cord gray matter forming an H-sign have been recognized and occur more frequently than in AQP4+NMOSD and MS myelitis. Pseudo-dilation of the ependymal canal has also been highlighted in cases of MOGAD myelitis, but detailed studies of this are lacking.

Design/Methods

The spinal cord MRIs of myelitis patients with MOGAD (n = 63), AQP4+NMOSD (n = 37), and MS (n = 26) were evaluated for central canal dilation, defined as T2-hyperintensity with similar consistency to cerebrospinal fluid within a myelitis T2-lesion. Clinical data were collected from the medical record for MOGAD patients. The expanded disability status scale (EDSS) was used to quantify disability at nadir.

Results

The median EDSS score at nadir for the MOGAD myelitis patients was 6 (range: 0-8). MOGAD patients experienced weakness (55/63[87%]), urinary retention/incontinence (50/63[79%]), numbness/paresthesias (48/63[76%]), and stool incontinence/constipation (36/63[57%]). Central canal dilation was more frequent in patients with MOGAD (23/63[37%]) than MS (0/26[0%]); p < 0.001) but did not differ from AQP4+NMOSD (14/37[38%]; p = 0.89). Spinal canal dilation resolved on follow-up axial MRI for most MOGAD (29/34[85%]) and AQP4+NMOSD (13/14[93%]) patients.

Conclusions

Central canal dilation is a common radiologic accompaniment of acute MOGAD and AQP4+NMOSD myelitis, but not MS myelitis. The resolution of central canal dilation on follow-up MRI in most patients suggests it is transient and related to the acute inflammatory edema. Central canal dilation may differentiate MOGAD and AQP4+NMOSD from MS. Its recognition could facilitate earlier testing for MOG-IgG and AQP4-IgG, accurate diagnosis, and treatment.

Disclosure: Dr. Webb has nothing to disclose. Ms. Cacciaguerra has received personal compensation in the range of \$500-\$4,999 for serving on a Scientific Advisory or Data Safety Monitoring board for Roche. Ms. Cacciaguerra has received personal compensation in the range of \$500-\$4,999 for serving on a Speakers Bureau for BMS Celgene. Ms. Cacciaguerra has received personal compensation in the range of \$500-\$4,999 for serving on a Speakers Bureau for Sanofi. John Chen has nothing to disclose. Dr. Sechi has nothing to disclose. Dr. Redenbaugh has nothing to disclose. The institution of Dr. Dubey has received personal compensation in the range of \$500-\$4,999 for serving as a Consultant for UCB. The institution of Dr. Dubey has received personal compensation in the range of \$5,000-\$9,999 for serving as a Consultant for Astellas. Dr. Dubey has received personal compensation in the range of \$0-\$499 for serving on a Speakers Bureau for AGRIMS. 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Exposure to TNF Inhibitors is Rare at MOGAD Diagnosis

Vyanka Redenbaugh, Eoin Flanagan, Valentina Floris, MD, Pietro Zara, M. Tariq Bhatti, Francisco Sanchez Moreno, Matthew Koster, Sara Mariotto, Sean Pittock, John Chen, Alberto Cauli, Paolo Solla, Elia Sechi

Objective

To assess the potential association between TNF-inhibitors and MOGAD

Background

The association of tumor necrosis factor- α (TNF)-inhibitors with MS has previously been suggested, whereas little is known about MOG-IgG-associated disease (MOGAD) in the context of these drugs. We recently encountered two patients who developed MOGAD while receiving TNF-inhibitors, prompting a search for similar cases in the literature and clinical practice.

Design/Methods

The two cases were seen at Mayo Clinic, Rochester (bilateral optic neuritis) and the University-Hospital of Sassari (brainstem syndrome). Three additional cases of MOGAD presenting during treatment with TNF-inhibitors were identified through Pubmed. We searched the medical records of 336 MOGAD patients seen at the Mayo Clinic, to assess if they had been treated with TNF-inhibitors.

Results

A total of 5 patients were identified. The median age at MOGAD presentation was 40 years (range, 36-49); 4/5 were male (80%). The median time from TNF-inhibitor initiation to MOGAD presentation was 6.5 years (range, 2-18). Of 4 patients who discontinued the TNF-inhibitor due to MOGAD onset, two subsequently had a MOGAD relapse. While in another patient, neurological symptoms subsided with corticosteroids despite TNF-inhibitor being maintained. The frequency of MOGAD presenting during TNF-inhibitors treatment at Mayo Clinic was 0.3% (1/336 cases).

Conclusions

We found that MOGAD is unlikely to present during treatment with TNF-inhibitors. The outcomes in these patients seemed not to be influenced by TNF-inhibitor treatment duration or discontinuation. These findings suggest the benefit of TNF-inhibitor withdrawal is not obvious, and the choice of discontinuing vs maintaining treatment with TNF-inhibitors should be weighted based on symptoms severity and activity status of the underlying systemic disorder. When withdrawal is considered, immunosuppression with agents potentially effective for both MOGAD and the immune-mediated disease originally managed by the TNF-inhibitor, could serve as dual purpose treatment.

Disclosure: Dr. Redenbaugh has nothing to disclose. The institution of Dr. Flanagan has received personal compensation in the range of \$500-\$4,999 for serving on a Scientific Advisory or Data Safety Monitoring board for Alexion. Dr. Flanagan has received personal compensation in the range of \$500-\$4,999 for serving on a Scientific Advisory or Data Safety Monitoring board for Genentech. Dr. Flanagan has received personal compensation in the range of \$500-\$4,999 for serving on a Scientific Advisory or Data Safety Monitoring board for Horizon Therapeutics. Dr. Flanagan has received personal compensation in the range of \$500-\$4,999 for serving on a Speakers Bureau for Pharmacy times. The institution of Dr. Flanagan has received research support from Viela Bio. Dr. Flanagan has a non-compensated relationship as a Member of medical Advisory Board with The MOG Project that is relevant to AAN interests or activities. Dr. Flanagan has a non-compensated relationship as a Editorial board member with Journal of The Neurologic Sciences that is relevant to AAN interests or activities. Dr. Flanagan has a non-compensated relationship as a Editorial board member with Neuroimmunology Reports that is relevant to AAN interests or activities. Dr.

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