Conclusions

Co-existent CSF anti-NMDAR and anti-GAD65 AE is not well-described in the literature. While this patient's serum GAD65 titer was in a range that correlates with neurologic autoimmunity, it was not as high as is typically reported in AE, and could simply be a marker of her insulin-dependent diabetes. However, it was also definitively positive from the CSF, which usually correlates well with GAD65-associated AE. Clinically, the patient had AE features that can be seen associated with either NMDAR and/or GAD65 antibodies. It is interesting to consider whether the GAD65 positivity is most likely to correlate with the patient's adult-onset diabetes, her AE, or both.

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Evidence for and Against Subclinical Disease Activity and Progressive Disease in MOG Antibody Disease and Neuromyelitis Optica Spectrum Disorder

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Objective

To investigate the evidence for and against relapse-independent clinical progression and/or subclinical disease activity in patients with Myelin Oligodendrocyte Glycoprotein Antibody Disease (MOGAD) and Aquaporin-4 IgG Seropositive Neuromyelitis Optica Spectrum Disorder (AQP4-IgG+ NMOSD).

Background

MOGAD and AQP4-IgG+ NMOSD are generally relapsing disorders, without clinical progression or subclinical disease activity outside of relapses. With advances in the diagnosis and treatment of these conditions, prolonged periods of remission without relapses can be achieved, and the question of whether progressive disease courses can occur has re-emerged.

Design/Methods

We conducted a narrative literature review in Ovid MEDLINE and Embase, exploring the evidence for and against relapse-independent progression in MOGAD and AQP4-IgG+ NMOSD. We classified the results in four categories : 1)Clinical observations, 2)MRI findings, 3) Retinal imaging, and 4)Fluid-based biomarkers.

Results

As the optic nerve is the major site of involvement in MOGAD and AQP4-IgG+ NMOSD, much of the data comes from the visual pathway studies. Possible pathophysiologic mechanisms of the OCT abnormalities in the absence of symptomatic optic neuritis are: 1) primary neurodegenerative process in retina, 2) subclinical inflammation of the optic nerve, 3) chiasmal involvement leading to abnormal findings in the unaffected eye, and 4)trans-synaptic retrograde degeneration originating from inflammatory lesions in the posterior visual pathway. Except for chiasmal involvement, these mechanisms are not specific to the visual pathway, and are considered as potential explanations for subclinical disease activity outside of the retina. Outside of the visual pathway, there is a lack of sufficient evidence to support the existence of subclinical disease activity and/or relapse-independent clinical progression in MOGAD and AQP4-IgG+ NMOSD, although recent fluid-based biomarker data (serum NfL and GFAP) support that neuro-axonal and/or astrocytic damage may be ongoing between attacks in these diseases.

Conclusions

This review highlights the many unknowns that remain in our understanding of the pathophysiology and clinical course of MOGAD and AQP4-IgG+ NMOSD.

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Coexistance of Probable Neurosarcoidosis and Generalized Myasthenia Gravis: Case Report

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Objective

NA.

Background

Sarcoidosis is an immune mediated disorder characterized by the formation of non-necrotizing granulomas in multiple organs. The broad range of manifestations of sarcoidosis in the nervous system includes cranial neuropathy, meningitis or myelopathy. Generalized Myasthenia Gravis is a neuromuscular disorder characterized by the formation of antibodies against the acetylcholine receptor, and typically presents with muscle weakness and fatigue. Association of Myasthenia Gravis and Neurosarcoidosis is infrequent.

Design/Methods

NA.

Results

Case report: A thirty-five year old woman, natural from Senegal was admitted to the hospital with a two month history of left peripheral facial nerve palsy, progressive dysphonia and dysphagia, associated with fever, headache and asthenia. Examination revealed, left facial numbness and weakness (V and VII nerve) and right vocal cord paresis (X nerve). Chest ray showed bilateral hilar lymphadenopathy and cranial MRI revealed enhancement of both Meckel cavum, bilateral trigeminal nerves and canalicular segment of the left facial nerve. CSF examination detected elevated protein and lymphocytic pleocytosis. For further study a PET/TC was done which revealed supradiaphragmatic and

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