Myelin oligodendrocyte glycoprotein antibody-associated disease

- Understanding Mechanisms of Whole Brain and Regional Grey Matter Atrophy in Children With MOGAD
- Comorbidities Are Associated With Unfavorable Outcome in Aquaporin-4 Antibody Positive Neuromyelitis Optica Spectrum Disorders and Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease: Exploratory Study From the CROCTINO Cohort
- Latin American RAND/UCLA modified Delphi consensus recommendations for management and treatment of adult MOGAD patients in clinical practice
- Autoimmune neuro-ophthalmic disorders: pathophysiologic mechanisms and targeted biologic therapies
- Pediatric cerebrospinal fluid immune profiling distinguishes pediatric-onset multiple sclerosis from other pediatric-onset acute neurological disorders
- Ofatumumab successfully treats myelin oligodendrocyte glycoprotein antibody-associated disease accompanied by Epstein-Barr viral infection: a case series
- 18 F-FAZA, 18 F-FDG, and 123 I-IMP Imaging Reveal Hypoxia and Metabolism in Acute MOGAD
- CNS B cell infiltration in tumefactive anti-myelin oligodendrocyte glycoprotein antibody-associated disease

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Myelin oligodendrocyte glycoprotein antibody-associated disease, often referred to as MOG-AD, is a rare autoimmune disorder that affects the central nervous system (CNS). It is characterized by the presence of antibodies against the myelin oligodendrocyte glycoprotein (MOG), which is a protein found on the surface of myelin-producing cells called oligodendrocytes. Myelin is the protective covering that surrounds nerve fibers and facilitates the proper transmission of nerve signals.

MOG-AD shares some similarities with multiple sclerosis (MS) and neuromyelitis optica spectrum disorder (NMOSD), but it is considered a distinct clinical entity. MOG-AD primarily affects the optic nerves and spinal cord, leading to various neurological symptoms. These symptoms can include optic neuritis (inflammation of the optic nerve) resulting in vision problems, transverse myelitis (inflammation of the spinal cord) causing weakness and sensory disturbances, and other neurological deficits.

Diagnosing MOG-AD involves clinical evaluation, neuroimaging (such as MRI scans), cerebrospinal fluid analysis, and testing for the presence of MOG antibodies in the blood. It's important to note that the presence of MOG antibodies doesn't necessarily indicate the presence of the disease, as these antibodies can also be found in other conditions. Therefore, a comprehensive assessment is required to establish a diagnosis.

Treatment for MOG-AD typically involves managing acute attacks with immunosuppressive medications, such as corticosteroids and other immune-modulating drugs. Long-term management may involve medications to prevent relapses and maintain disease stability. Given the rarity of the condition and the evolving nature of research, treatment approaches can vary, and patients are often closely monitored by a team of neurologists.

It's essential for individuals suspected of having MOG-AD to work closely with healthcare professionals

experienced in treating autoimmune neurological disorders to receive accurate diagnosis and appropriate management. If you suspect you or someone you know has MOG-AD, it's advisable to seek medical attention promptly for proper evaluation and guidance.

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOG-AD) is an immune-mediated neuroinflammatory disorder leading to demyelination of the CNS. Interleukin (IL)-6 receptor blockade is under study in relapsing MOGAD as a preventative strategy, but little is known about the role of such treatment for acute MOGAD attacks.

McLendon et al. discuss the cases of a 7-year-old boy and a 15-year-old adolescent boy with severe acute CNS demyelination and malignant cerebral edema with early brain herniation associated with clearly positive serum titers of MOG-IgG, whose symptoms were incompletely responsive to standard acute therapies (high-dose steroids, IV immunoglobulins (IVIGs), and therapeutic plasma exchange).

Both boys improved quickly with IL-6 receptor inhibition, administered as tocilizumab. Both patients have experienced remarkable neurologic recovery.

They propose that IL-6 receptor therapies might also be considered in acute severe life-threatening presentations of MOGAD [1)]

1)
McLendon LA, Gambrah-Lyles C, Viaene A, Fainberg NA, Landzberg EI, Tucker AM, Madsen PJ, Huh J, Silver MR, Arena JD, Kienzle MF, Banwell B. Dramatic Response to Anti-IL-6 Receptor Therapy in Children With Life-Threatening Myelin Oligodendrocyte Glycoprotein-Associated Disease. Neurol Neuroimmunol Neuroinflamm. 2023 Aug 15;10(6):e200150. doi: 10.1212/NXI.0000000000200150. PMID: 37582615.