Title: Neuro-inflammatory involvement of the spinal cord: what's new in Neuromyelitis Optica and MOG-Antibody-Associated Disease, their differential diagnosis and treatment.

Abstract:

Neuromyelitis Optica (NMO) and MOG-Antibody-Associated Disease (MOGAD) are autoimmune demyelinating disorders that predominantly affect the central nervous system, with a distinct predilection for the spinal cord and optic nerves. While NMO is characterized by the presence of aquaporin-4 antibodies (AQP4-IgG), MOGAD is associated with myelin oligodendrocyte glycoprotein antibodies (MOG-IgG). Both conditions can present themselves as recurrent attacks of transverse myelitis, leading to significant morbidity and disability. However, the clinical spectrum of both diseases can be larger.

The pathogenesis of spinal cord involvement in NMO involves an autoimmune response against AQP4, a water channel predominantly expressed on the astrocytic end-feet. This results in complement-mediated astrocyte damage, inflammation, and demyelination. In MOGAD, antibodies against MOG lead to an inflammatory cascade leading to subsequent myelin destruction.

Distinguishing between NMO and MOGAD is essential due to differences in prognosis and treatment strategies. The clinical presentation, imaging findings, and serological markers play a pivotal role in establishing an accurate diagnosis. Additionally, distinguishing these entities from other demyelinating diseases, such as multiple sclerosis, is imperative for tailored patient management.

Current treatment strategies in NMO and MOGAD aim to suppress inflammation, prevent relapses, and mitigate disability. High-dose corticosteroids remain the first-line therapy during acute attacks. Long-term immunosuppression, including rituximab, azathioprine, and mycophenolate mofetil, has shown efficacy in reducing relapse rates. Emerging therapies, such as anti-IL-6 receptor, anti-CD19, anti-C5, anti-FcRn monoclonal antibodies offer promising alternatives. Individualized treatment plans should consider the specific clinical and serological features of each patient.

In conclusion, spinal cord involvement in NMO and MOGAD represents a significant clinical challenge, necessitating a multidisciplinary approach for accurate diagnosis and optimal management. Advances in our understanding of the pathogenesis and ongoing research into targeted therapies provide hope for improved outcomes in patients affected by these debilitating conditions.