MOGAD Masterclass:

Extending Approaches to Diagnosis and Management

December 13th, 2024 Lyon, France



About myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD)

MOGAD is a rare autoimmune demyelinating disease of the central nervous system that is distinct from multiple sclerosis (MS) and neuromyelitis optica spectrum disorder (NMOSD).1



1-Day event

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14 Global **MOGAD** experts

Exchanging insights with about 60 specialists coming from 15 countries



High-profile content

The masterclass provides the very latest cutting-edge knowledge and advancements in MOGAD

Learning objectives

- Evaluate diverse myelin oligodendrocyte glycoprotein (MOG) antibody assays, discerning their unique attributes and optimal clinical utility to refine diagnosis
- Summarize emerging blood and cerebrospinal fluid (CSF) biomarkers for MOGAD
- Review pathogenic pathways in MOGAD
- Summarize new magnetic resonance imaging (MRI) approaches and emerging algorithms for diagnosing MOGAD, emphasizing advancements to improve accuracy and treatment decisions
- Describe assessments employed for characterizing retinal and optic nerve involvement in MOGAD
- Review the treatment landscape for MOGAD, including both established and emerging therapies
- Summarize best practices for the management of pediatric patients with MOGAD

Session 1: Clinical and Biological Considerations

Key take home

MOGAD's typical clinical features include optic neuritis, transverse myelitis, and acute disseminated encephalomyelitis but the spectrum of phenotypes is expanding.¹ The international diagnostic criteria, published in 2023,¹ require one core clinical event and positive MOG-IgG, with cell-based assays recommended for testing. Serum is the preferred specimen type for MOG-IgG testing, but cerebrospinal fluid (CSF) testing is also useful in some cases. Novel biomarkers like Neurofilament Light Chain (NfL) and glial fibrillary acidic protein (GFAP) levels are being studied for monitoring MOGAD. Pathogenesis involves antibody-mediated cell cytotoxicity and potentially complement activation, with animal models aiding research. MOGAD is highly heterogeneous (clinical presentation and neuropathology) and may require an individualized treatment-based approach.

Presentations within the session:

- Clinical features from a neurologist perspective
- **Refining assays for MOG antibodies**
- **CSF MOG antibodies and emerging** biomarkers in blood and CSF
- **Dissecting pathogenic pathways** in models of MOGAD
- Pathologic pathways in MOGAD



Session 2: What's New in Imaging

Key take home

Imaging is essential in diagnosing MOGAD, especially when antibody status is unclear. Distinguishing MRI features include poorly defined lesions and optic nerve head swelling. Regular MRI follow-up is generally not recommended unless clinically indicated. Advanced MRI techniques and machine learning help differentiate MOGAD from MS and NMOSD, which is crucial for accurate early disease discrimination and appropriate management. Optic neuritis is the most common adult MOGAD phenotype, characterized by severe vision loss, bilateral lesions, and high steroid responsiveness, with about 5% of patients experiencing severe disability despite treatment.

Presentations within the session:

- **Conventional and nonconventional** approaches to assess MOGAD
- Algorithms and combinations to differentiate MOGAD from other demyelinating diseases
- **MOGAD** ophthalmological involvement: key clinical features, optical coherence tomography (OCT), and ocular MRI differentiating MOGAD from non-MOGAD optic neuritis



Session 3: Current and Future Management

Key take home

MOGAD has a significant disease and treatment burden. High relapse rates in MOGAD patients highlight the need for approved therapies and standardized treatment guidelines. Several new treatments targeting pathways like cluster of differentiation 19 (CD19), interleukin-6 (IL-6) receptors, and neonatal Fc receptor (FcRn) are under investigation in clinical trials. Clinical presentation of MOGAD varies with age and more studies are needed to understand the best management in children. Predicting relapse risk is complex and influenced by factors like age, phenotype, and treatment options, requiring tailored treatment approaches.



These events were initiated, organised and funded by UCB.

Visit the Rare Disease Connect in Neurology (RDCN) portal for valuable resources on MOGAD. www.ucbrarediseaseconnect.com

Reference: 1. Banwell B, et al. Lancet Neurol. 2023;22(3):268-282.

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Intended for a Healthcare Professional audience.



Third Masterclass

GREAT EXPECTATION