



Title	Treatment of central nervous system inflammatory demyelinating disorders: bedside to the bench
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Multiple sclerosis (MS), the most important inflammatory demyelinating central nervous system (CNS) disease complex, is characterised by heterogenous immunopathogenetic pathways, various clinical entities and disease courses, and finally, inhomogeneous and unpredictable treatment effects. Therefore, identification of MS-specific biological markers has continuously gained importance over the last decade.

There is accumulating evidence from immunological, pathological, and therapeutic studies that B cells (and antibodies) are critically involved in the pathophysiology of MS. B cells (and antibodies) seem to play various roles in the initiation and propagation of inflammatory demyelinating processes at different disease stages of MS and its variants. Recent therapeutic trials indicated that monoclonal antibodies that specifically target B cells are effective in MS and neuromyelitis optica (NMO).

This lecture will review the current status and (potential) applicability of antibodies in cerebrospinal fluid (CSF) and/or serum as biological markers for:

- (1) diagnosis: value of CSF oligoclonal IgG bands for MS (differential) diagnosis, value of anti-MOG and –AQP4 antibodies to distinguish different CNS demyelinating disorders (paediatric and adult ADEM, CIS and NMO)
- (2) disease progression: value of antibodies to myelin (eg MOG, MBP) and non-myelin antigens (eg neurofascin)
- (3) repair and regeneration: IgM antibodies promoting remyelination, antibodies blocking inhibitory molecules, eg NoGo
- (4) monitoring disease-modifying therapies: neutralising antibodies (against interferon beta and natalizumab), risk assessment and treatment stratification (anti-JC virus antibody assays)

Treatment of Central Nervous System Inflammatory Demyelinating Disorders: Bedside to the Bench

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Central nervous system inflammatory demyelinating disorders (CNS IDD) are important diseases affecting patients of a wide age range, and are potentially treatable. CNS IDD can result in major neurological disabilities and even mortality. Both classical multiple sclerosis (CMS) and neuromyelitis optica are predominantly characterised by relapsing attacks of CNS inflammatory demyelination. The diagnosis of which may be difficult especially in the early phase. Excitement has arisen from increasing numbers of disease-modifying drugs (DMDs) available for CMS in recent years. Beta-interferon and glatiramer acetate are certainly first-line DMDs. Natalizumab, fingolimod and mitoxantrone are approved second-line DMDs for relapsing multiple sclerosis patients. Other DMDs that may be used in refractory patients include alemtuzumab, teriflunomide and rituximab. However, availability, costs and rare but serious side-effects are practical issues that may limit the choice of treatment for these patients. How about novel therapies?