

Modern concepts of myelitis etiopathogenesis and targeted therapeutic approaches

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Background: Myelitis is an acute or subacute inflammatory-demyelinating disorder of the spinal cord that frequently leads to long-term disability and severe neurological deficits. Recent advances have demonstrated that many cases previously classified as idiopathic are actually driven by specific autoimmune biomarkers (such as anti-AQP4 and anti-MOG antibodies) and post-infectious neuroinflammatory cascades. Unraveling the early pathogenetic mechanisms and implementing targeted immunobiological therapies are crucial for optimizing clinical outcomes.

Objective: To evaluate the modern pathogenetic and clinical-immunological features across distinct etiologic forms of myelitis and to assess the efficacy of targeted therapeutic modalities, including monoclonal antibody therapy.

Materials and Methods: A combined retrospective and prospective analysis was conducted on clinical, laboratory, and neuroimaging data from 52 patients diagnosed with transverse and disseminated myelitis. Diagnostic confirmation was achieved using high-field MRI (1.5–3.0 Tesla), detection of cerebrospinal fluid (CSF) oligoclonal IgG bands, and cell-based assays (CBA) alongside ELISA to measure serum anti-aquaporin-4 (anti-AQP4) and anti-myelin oligodendrocyte glycoprotein (anti-MOG) autoantibodies.

Results:

- **Etiologic Heterogeneity & Biomarkers:** Neuromyelitis optica spectrum disorder (NMOSD, AQP4-seropositive) was identified in 42.3% of patients, MOG antibody-associated disease (MOGAD) in 21.1%, and post-infectious autoinflammatory mechanisms (secondary to SARS-CoV-2, EBV, or VZV) in 19.2% of cases.
- **Acute Management:** High-dose intravenous methylprednisolone pulse therapy (1000 mg/day) combined with early therapeutic plasma exchange (PLEX) or intravenous immunoglobulin (IVIG) during the acute phase yielded a 35% improvement in the rate of motor recovery.
- **Targeted Biotherapy:** For maintenance of remission and relapse prevention, B-cell-depleting monoclonal antibodies (Rituximab, Inebilizumab) and IL-6 receptor antagonists (Satralizumab) reduced relapse risk by 78% compared to

conventional immunosuppressants, alongside marked improvements on the Expanded Disability Status Scale (EDSS).

Conclusion: Identifying distinct autoimmune immunophenotypes (anti-AQP4 and anti-MOG) is central to the contemporary etiopathogenetic framework of myelitis. Prompt, aggressive immunomodulatory intervention (corticosteroid pulse + PLEX) during the acute phase, followed by maintenance with targeted biological agents, significantly curtails relapse rates and improves long-term quality of life.