



Articles appearing in the March 2020 issue

Frequency of myelin oligodendrocyte glycoprotein antibody in multiple sclerosis: A multicenter cross-sectional study

Objective To address the frequency of myelin oligodendrocyte glycoprotein (MOG) antibody (Ab) in an unselected large cohort of adults with MS.

Methods This is a cross-sectional study in 2 MS expert centers (Lyon and Strasbourg University Hospitals, France) between December 1, 2017, and June 31, 2018. Patients aged ≥18 years with a definite diagnosis of MS according to 2010 McDonald criteria were tested for MOG-Ab by using a cell-based assay (CBA) in Lyon and subsequently included. Positive samples were tested by investigators blinded to the first result with a second assay in a different laboratory (Barcelona, Spain) by using the same plasmid and secondary Ab.

Results Serum samples from 685 consecutive patients with MS were analyzed for MOG-Ab. Median disease duration at sampling was 11.5 (interquartile range, 5.8–17.7) years, and 72% were women. Two (0.3%) patients resulted to be MOG-Ab-positive. The 2 patients were women aged 42 and 38 at disease onset and were diagnosed with secondary and primary progressive forms of MS, respectively. This positive result was confirmed by the CBA in Barcelona.

Conclusions Our findings indicate that MOG-Ab are exceptional in MS phenotype, suggesting that the MOG-Ab testing should not be performed in typical MS presentation.

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Anti-IGLON5 disease: A new case without neuropathologic evidence of brainstem tauopathy

 $\begin{tabular}{ll} \textbf{Objective} To describe the neuropathologic features and the molecular data of phosphorylated tau (pTau) in a new case of anti-IgLON5 disease. \end{tabular}$

Methods Review of clinical data, postmortem neuropathologic examination. Biochemical analyses of pTau were performed in brain samples from the present case and from a previously described patient with anti-IgLON5 with the characteristic brainstem tauopathy.

Results The patient was a 71-year-old man with a clinical syndrome consisting of sleep disturbance and bulbar symptoms. IgLON5 antibodies of predominant IgG4 subtype were detected in serum and CSF. He carried the HLA DRB1*10:01-DQB1*05:01 haplotype. Despite treatment with IV immunoglobulins, he unexpectedly died during sleep 2 years after disease onset. Histology showed neurofibrillary pathology and β -amyloid deposits consistent with Alzheimer disease (AD) of intermediate severity. pTau deposits were absent in the brainstem. There were few perivascular CD8⁺ T-cell infiltrates in the posterior hypothalamus, amygdala, and brainstem with microglial activation. The pTau immunoblot showed a pattern of bands consistent with AD, which was different from that observed in the patient with anti-IgLON5 with brainstem tauopathy who presented a differential band around 56 KDa.

Conclusions The absence of pTau deposits in the brainstem of the present patient suggests that the tauopathy of patients with anti-IgLON5 disease may be a late, secondary event. The antiIgLON5 brainstem tauopathy has a specific molecular signature different from primary tauopathies. pTau deposits restricted to the hippocampus/limbic regions of patients with antiIgLON5 may represent an age-related comorbidity.

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