Abstracts

Articles appearing in the January 2018 issue

Neurofilament light chain predicts disease activity in relapsing-remitting MS

Objective To investigate if serum neurofilament light chain (NfL) and chitinase 3-like 1 (CHI3L1) predict disease activity in relapsing-remitting multiple sclerosis (RRMS).

Methods A cohort of 85 patients with RRMS was followed for 2 years (6 months without disease-modifying treatment and 18 months with interferon- β -1a). Expanded Disability Status Scale was scored at baseline and every 6 months thereafter. MRI was performed at baseline and monthly for 9 months and then at months 12 and 24. Serum samples were collected at baseline and months 3, 6, 12, and 24. We analyzed serum levels of NfL using a single-molecule array assay and CHI3L1 by ELISA, and estimated the association with clinical and MRI disease activity using mixed-effects models.

Results NfL levels were significantly higher in patients with new T1 gadolinium-enhancing lesions (37.4 pg/mL, interquartile range [IQR] 25.9–52.4) and new T2 lesions (37.3, IQR 25.1–48.5) compared to those without (28.0 pg/mL, IQR 21.9–36.4, $\beta = 1.258$, $p < 0.001$; and 27.7 pg/mL, IQR 21.8–35.1, $\beta = 1.251$, $p < 0.001$, respectively). NfL levels were associated with the presence of T1 gadolinium-enhanced lesions up to 2 months before ($p < 0.001$) and 1 month after ($p = 0.009$) the time of biomarker measurement. NfL levels fell after initiation of interferon- β -1a treatment ($p < 0.001$). Changes in CHI3L1 were not associated with clinical or MRI disease activity or interferon- β -1a treatment.

Conclusion Serum NfL could be a promising biomarker for subclinical MRI activity and treatment response in RRMS. In clinically stable patients, serum NfL may offer an alternative to MRI monitoring for subclinical disease activity.

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A multiplex family with GAD65-Abs neurologic syndromes

Objective Neurologic autoimmune syndromes associated with anti-glutamate acid decarboxylase 65 antibodies (GAD65-Abs) are rare and mostly sporadic.

Method We describe a niece and her aunt with GAD65-Abs neurologic syndromes. High-resolution human leukocyte antigen (HLA) typing of class I and class II alleles was performed using next-generation sequencing.

Results The proband had cerebellar ataxia and probable limbic encephalitis features while her niece had stiff-person syndrome. Both had high titer of GAD65-Abs in the serum and CSF and showed signs of inflammation in the CSF. Both affected members carried the same rare recombinant DRB1*15:01:01~DQA1*01:02:01~DQB1*05:02:01 haplotype, which may or may not be involved in disease susceptibility. Interestingly, other unaffected members of the family had either the same HLA haplotype but normal serum GAD65-Abs or had different HLA types but high titer of serum GAD65-Abs without neurologic symptom, suggesting cumulative effects.

Conclusion This unique association strengthens the concept that hereditary factors, possibly including specific HLA haplotypes, play a role in neurologic syndromes associated with GAD65-Abs.

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