In Focus

Spotlight on the February 11 Issue

Robert A. Gross, MD, PhD, FAAN
Editor-in-Chief, *Neurology*®

Potassium channel KIR4.1-specific antibodies in children with acquired demyelinating CNS disease

The authors measured KIR4.1-IgG in children with acquired demyelinating disease, other neurologic diseases, autoimmune diseases, and controls. Serum antibodies to KIR4.1 were found in the majority of children with acquired demyelinating disease but not in children with other diseases or in controls. These findings suggest that KIR4.1 is an important target of autoantibodies in childhood acquired demyelinating disease.

See p. 470

Distinction between MOG antibody-positive and AQP4 antibody-positive NMO spectrum disorders

This study investigated 215 patients with neuromyelitis optica spectrum disorders for clinical findings that distinguish those with myelin oligodendrocyte glycoprotein (MOG) antibodies, those with aquaporin-4 antibodies, and those seronegative for both antibodies. The

16 patients with MOG antibodies had more restricted phenotypes and fewer attacks with better recovery. The features of MOG antibody-positive cases suggest a different underlying pathophysiology.

See p. 474

From editorialists Weinshenker & Wingerchuk: "Whether the recent observations with anti-MOG will prove as important for clinical diagnosis and pathogenic understanding of NMO as anti-AQP4 will require further careful prospective longitudinal studies with adequate sample size, relevant controls, and consistent assay methodology."

See p. 466

Genotype-phenotype correlations in alternating hemiplegia of childhood

The authors observed that alternating hemiplegia of childhood (AHC) patients with the E815K mutation in *ATP1A3* had more severe symptoms than other mutation groups, especially those with D801N mutation. Although AHC is not generally seen as a progressive disorder, it should be considered a disorder that deteriorates abruptly.

See p. 482; Editorial, p. 468

Guillain-Barré syndrome associated with preceding hepatitis E virus infection

Neuralgic amyotrophy and hepatitis E virus infection

Is it possible that hepatitis E virus has been misnamed? Hepatitis E virus infection may trigger both Guillain-Barré syndrome and brachial neuritis with substantial neurologic illness, without much evidence of hepatitis.

See p. 491 and p. 498

High plasma estradiol interacts with diabetes on risk of dementia in older postmenopausal women

The authors investigated the association of endogenous total- and bioavailable-estradiol (E2) and total-testosterone in a cohort of 5,644 postmenopausal women, in part consisting of a random subcohort of 562 women not using hormone therapy and 132 incident dementia cases. High E2 level is an independent predictor of incident dementia, particularly in postmenopausal women with diabetes.

See p. 504

Satiety-related hormonal dysregulation in behavioral variant frontotemporal dementia

The authors explored postmeal hormone levels in 19 patients with behavioral variant frontotemporal dementia (bvFTD), 17 with Alzheimer dementia, and 18 controls. Aberrant hormone levels may represent a compensatory response to the behavioral (tendency to overeat) or neuroanatomical abnormalities of bvFTD.

See p. 512

Myoinositol and glutamate complex neurometabolite abnormality after mild traumatic brain injury

This study measured in vivo myoinositol and glutamate plus glutamine in 26 patients with mild traumatic brain injury (mTBI) and 13 controls. The concentrations of neurometabolites relative to creatine, as well as absolute myoinositol concentration, were higher in patients with mTBI compared with controls, suggesting a complex glial and excitatory response to injury without concomitant neuronal loss.

See p. 521

NB: "Pseudo-Popeye syndrome: Extramedullary plasmacytoma manifesting in skeletal muscle," see p. 544. To check out other NeuroImages, point your browser to Neurology.org.

Podcasts can be accessed at www.neurology.org

Neurology®

Spotlight on the February 11 Issue

Robert A. Gross

Neurology 2014;82;465

DOI 10.1212/WNL.0000000000000140

This information is current as of February 10, 2014

Updated Information & Services

including high resolution figures, can be found at:
<http://n.neurology.org/content/82/6/465.full>

Permissions & Licensing

Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at:
http://www.neurology.org/about/about_the_journal#permissions

Reprints

Information about ordering reprints can be found online:
<http://n.neurology.org/subscribers/advertise>

Neurology® is the official journal of the American Academy of Neurology. Published continuously since 1951, it is now a weekly with 48 issues per year. Copyright © 2014 American Academy of Neurology. All rights reserved. Print ISSN: 0028-3878. Online ISSN: 1526-632X.