Editors' Note: In response to an inquiry by Drs. Lerner and Miodownik, American Academy of Neurology (AAN) guideline authors Bhidayasiri et al. discuss how relevant studies are analyzed and classified for potential inclusion in AAN practice guidelines. The painstaking process used in the development of AAN practice guidelines is described in detail in the Clinical Practice Guideline Process Manual (<http://tools.aan.com/globals/axon/assets/9023.pdf>) available to AAN members and the public on the AAN Web site.

—Megan Alcauskas, MD, and Robert C. Griggs, MD

DISTINCTION OF SEROPOSITIVE NMO SPECTRUM DISORDER AND MS BRAIN LESION DISTRIBUTION

Ilya Kister, Yulin Ge, Joseph Herbert, New York; Tim Sinnecker, Jens Wuerfel, Friedemann Paul, Berlin: Matthews et al.¹ attempted to differentiate seropositive neuromyelitis optica spectrum disorders (NMOSD) from multiple sclerosis (MS) based on brain MRI records. They suggested that none of the patients with NMOSD exhibited “Dawson fingers” on brain MRI.

James Dawson described these characteristic lesions in MS pathologically as “wedge-shaped areas with broad base to the ventricle, and extensions into adjoining tissue in the form of finger-like processes or ampullae, in each of which a central vessel could usually be found.”² Ultra-high-field MRI allows for in vivo visualization of small central veins within Dawson fingers.

Our 2 groups used ultra-high-field MRI to image brains in NMOSD and MS and independently reported that periventricular lesions are rare in NMOSD and lack central venule.^{3,4} This supports the authors’ finding that the presence of Dawson fingers constitutes strong evidence against the diagnosis of NMOSD. However, for this criterion to be useful in clinical practice, an unambiguous definition of what constitutes Dawson finger on conventional brain MRI must be adopted.

It would be helpful if the authors could supply a definition based on their experience with NMOSD and MS that would more formally specify lesion morphology. This should include details on borders, dimensions, and orientation on axial and sagittal T2-weighted sequences. In addition, a defined

distance from lateral ventricles and other periventricular lesions would be helpful.

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VARICELLA-ZOSTER VIRUS ACUTE MYELITIS IN A PATIENT WITH MS TREATED WITH NATALIZUMAB

Bertrand Bourre, Romain Lefaucœur, Patrick Ahtoy, Floriane Travers, Damien Fetter, Rouen, France: Yeung et al.¹ studied a case of varicella-zoster virus (VZV) myelitis in a patient with MS treated with natalizumab (NTZ). We wanted to stress another manifestation of VZV infection in NTZ-treated patients. A patient with MS recently came to our institution for thunderclap headaches. She had received NTZ for 14 months. She was afebrile and complained of photophobia but had no meningeal signs. Neurologic examination was normal and she had no skin lesions. Brain imaging revealed neither bleeding nor vascular malformation and we performed a lumbar puncture. We noted elevated protein (0.68 g/L), normal glucose (3.5 g/L), and 33 leukocytes with a majority of lymphocytes and no organisms. CSF PCR analyses were negative for herpes simplex virus, Epstein-Barr virus, cytomegalovirus, adenovirus, and enterovirus, but positive for VZV. She was treated with IV acyclovir for 14 days with disappearance of headaches. Repeat CSF examination after 10 days was normal.

Yeung et al. had difficulty differentiating VZV myelitis from a relapse and concluded that neurologists should not underestimate VZV infection in NTZ-treated patients. We also wanted to emphasize the multifaceted presentation of VZV infection and

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Ilya Kister, Yulin Ge, Joseph Herbert, et al.

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