

load of varicella-zoster virus (VZV) is higher in patients with MS at relapse phase than those in remission. The status of the disease course in all patients with MS should be determined before sampling.

Four out of the 15 patients with MS with relapse were on interferon (IFN)- β treatment. It has been shown that the administration of IFN- β can lead to the reduction of JCV genome³ and may result in a false-negative due to decreasing the JCV titer and T-cell response. Thus, patients taking IFN- β should be excluded from this study.

In addition, we would not have included clinically isolated syndrome (CIS) in this study cohort. Approximately 80% of patients with CIS develop MS, while the rest do not.⁴ Patients with CIS should not be considered for evaluation of JCV-specific T-cell response under corticosteroid therapy.

Author Response: Renaud A. Du Pasquier, Mathieu Canales, Myriam Schlupe, Lausanne, Switzerland:

We thank Mr. Zahednasab for his interest in our article. As we explained in the Methods, we enrolled only patients with MS who had a relapse severe enough to warrant 3 days of IV corticosteroids followed by tapering oral prednisone. The mean delay between the onset of symptoms and steroid treatment was 11.6 days (range 0–54 days). Concerning VZV and MS, the paper of Sotelo et al. has been challenged.⁵ In addition, VZV is not JCV so it is difficult to draw any conclusions from this comparison.

Regarding IFN- β , as we mentioned: “If a patient exhibited no T-cell response against a given virus before and after CS, then this patient was not taken into account in our analyses for the given virus and the given assay.” This was the case in 2 of 4 patients on IFN- β , who are not part of the JCV-specific cellular immune response part of our article and thus not included into the analysis of corticosteroids effects. Finally, in the patients with CIS, diseases other than MS were carefully ruled out. Currently, 4 of 8 of these patients with CIS have converted to definite MS, confirming that their inclusion was appropriate.

© 2013 American Academy of Neurology

1. Antoniol C, Jilek S, Schlupe M, et al. Impairment of JCV-specific T-cell response by corticotherapy: effect on PML-IRIS management? *Neurology* 2012;79:2258–2264.
2. Sotelo J, Martinez-Palomo A, Ordonez G, et al. Varicella zoster virus in cerebrospinal fluid at relapses of multiple sclerosis. *Ann Neurol* 2008;63:303–311.
3. Delbue S, Guerini FR, Mancuso R, et al. JC virus viremia in interferon-beta-treated and untreated Italian multiple sclerosis patients and healthy controls. *J Neurovirol* 2007; 13:73–77.
4. Fisniku LK, Brex PA, Altmann DR, et al. Disability and T2 MRI lesions: a 20-year follow-up of patients with relapse onset of multiple sclerosis. *Brain* 2008;131:808–817.
5. Burgoon MP, Cohrs RJ, Bennett JL, et al. Varicella zoster virus is not a disease-relevant antigen in multiple sclerosis. *Ann Neurol* 2009;65:474–479.

CORRECTIONS

Randomized controlled trial of trigeminal nerve stimulation for drug-resistant epilepsy

In the article “Randomized controlled trial of trigeminal nerve stimulation for drug-resistant epilepsy” (*Neurology*[®] 2013;80:786–791) by DeGiorgio et al., two corrections are needed. The first is in the abstract, where the confidence interval should read “Subjects in the treatment group were more likely to respond than patients randomized to control (odds ratio 1.73, confidence interval 0.59–5.1).” The second correction is in the level of evidence statement. Although there was improvement within the active treatment group alone, there was no significant difference in effect between the treatment and control groups. The study was insufficiently powered to exclude an important difference. Therefore, the level of evidence statement should read “Because of a lack of statistical precision, this Class II study provides insufficient evidence to determine the efficacy of trigeminal nerve stimulation in patients with DRE.” The editors regret the error and the misstatement.

WriteClick: Do acute phase markers explain body temperature and brain temperature after ischemic stroke?

In the WriteClick Author Response “Do acute phase markers explain body temperature and brain temperature after ischemic stroke?” by J.M. Wardlaw et al. (*Neurology*[®] 2013;80:778), there is an error in one of the author affiliations. It should read Bartosz Karaszewski, Gdansk. The authors regret the error.

Author disclosures are available upon request (journal@neurology.org).

Neurology®

WriteClick: Do acute phase markers explain body temperature and brain temperature after ischemic stroke?

Neurology 2013;81;98

DOI 10.1212/WNL.0b013e31829d3ed6

This information is current as of July 1, 2013

Updated Information & Services

including high resolution figures, can be found at:
<http://n.neurology.org/content/81/1/98.2.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 © 2013 American Academy of Neurology. All rights reserved. Print ISSN: 0028-3878. Online ISSN: 1526-632X.

