

4. Southam CM, Moore AE. Induced virus infections in man by the Egypt isolates of West Nile virus. *Am J Trop Med Hyg* 1954;3:19.
5. Sampson BA, Armbrustmacher V. West Nile encephalitis: the neuropathology of four fatalities. *Ann N Y Acad Sci* 2001;951:172-8.

Address for correspondence: Vijay Kumar Krishnamoorthy, Department of Internal Medicine, Advocate Illinois Masonic Medical Center, 836 West Wellington Street, Chicago, IL 60657, USA; fax: 773-296-5361; email: Vijay.Krishnamoorthy-MD@advocatehealth.com

“Acute” West Nile Virus Encephalitis (Response to Krishnamoorthy et al.)

To the Editor: In a letter to the editor, Krishnamoorthy et al. question the diagnosis of “acute West Nile encephalitis” in our case report. We did not use the word “acute” in the paper, but the patient did in fact have

an acute illness. We believe that this case report, in which West Nile virus (WNV) was isolated in cell culture, represents the best evidence for a WNV infection in a human in the United States. The diagnosis of West Nile encephalitis was based on clinical analysis (1); not everyone with the diagnosis undergoes an autopsy. In many instances, patients do recover. In our case, the patient had the clinical features of encephalitis consisting of unremitting fever associated with a rapid course of progressive confusion and lethargy followed by coma. In addition, increased depression of respiratory drive existed, pointing to brain stem involvement. We agree that the inflammatory changes in the brain were limited as compared to such changes in other reported cases of WNV; however, this limitation was attributable to the fact that the patient was both immunocompromised and neutropenic at the time of acute infection. Therefore, the usual inflammatory response cannot be expected. Even though the changes were limited, they were consistent with the histologic findings in previously published reports (1,2).

The second point by Krishnamoorthy et al. represents their hypothesis about a human chronic carrier state for WNV. Although a chronic carrier state is possible, the viremic period associated with arboviral infections is typically short (3). While one cannot rule out persistent infection with WNV, until our report attempts to recover the virus by isolation in North America in humans have been uniformly unsuccessful. Also, previous reports of successful WNV isolations by Israeli investigators in immunocompetent hosts (4) have been from blood specimens before seroconversion. These considerations indicate that the virus is not routinely found in the blood in substantial amounts by the time clinical symptoms consistent with WNV infection occur. We do not know, nor have we speculated, about the timing of the infection as the patient had no recollection of a mosquito bite. Tests for both immunoglobulin (Ig) G and IgM antibodies to WNV were negative in our patient. Because the patient was immunocompromised, a humoral response was not expected; therefore, this information cannot be used as

The image shows a screenshot of the CDC Emerging Infectious Diseases Journal homepage. The page features a search bar, a navigation menu, and several article highlights. A large, stylized 'SEARCH EID ONLINE' graphic is overlaid on the right side of the screenshot. Below the screenshot, the URL 'www.cdc.gov/eid' is displayed in a large, bold font.

evidence that the patient had an acute infection. However, observations that the patient had no manifestation of encephalitis during a previous episode of neutropenia and that she had an acute febrile illness associated with neurologic signs of encephalitis point to an acute infection. The figure, in which WNV copy numbers are correlated with leukocyte count, is not intended to pinpoint the time of infection. However, as stated in the paper, this figure did show that the virus was rapidly cleared after resolution of neutropenia.

A report by Camenga et al. (5) demonstrated that mice, infected with WNV develop only an inapparent infection. These mice will invariably die of fulminant encephalitis if only a single dose of cyclophosphamide is given. However, mice treated with one dose of cyclophosphamide demonstrate inflammatory changes in the brain. If a second dose of the drug is administered 5 days after infection, inflammation is completely suppressed in mice. Although mice are immunologically different from humans, this work, done almost 30 years ago, supports the argument for an acute infection in the current case report. If the patient in our study was a chronic carrier, she should have had manifestations of acute West Nile encephalitis immediately following the first course of combination chemotherapy, which was much more immunosuppressive than cyclophosphamide alone. This fact reemphasizes our major point in the article that patients who are immunocompromized and undergoing chemotherapy, which may cause neutropenia, should take extra precautions against being exposed to WNV.

Alexander Hindenburg*
and **Cinnia Huang†**

*Winthrop University Hospital, Mineola, New York, USA; †New York State Department of Health, Albany, New York, USA

References

1. Shieh WJ, Guarmer J, Layton M, Fine A, Miller J, Nash D, et al. The role of pathology in an investigation of an outbreak of West Nile encephalitis in New York, 1999. *Emerg Infect Dis* 2000;6:370–2.
2. Sampson BA, Armbrustmacher V. West Nile encephalitis. The neuropathology of four fatalities. *Ann NY Acad Sci* 2001;951:172–8.
3. Southam CM, Moore AE. Induced virus infections in man by the Egypt isolates of West Nile virus. *Am J Trop Med Hyg* 1954;3:19–50.
4. Hindiyeh M, Shulman LM, Mendelson E, Weiss L, Grossman Z, Bin H. Isolation and characterization of West Nile virus from the blood of viremic patients during the 2000 outbreak in Israel. *Emerg Infect Dis* 2001;7:748–50.
5. Camenga DL, Nathanson N, Cole GA. Cyclophosphamide-potentiated West Nile viral encephalitis: relative influence of cellular and humoral factors. *J Infect Dis* 1974;130:634–41.

Address for correspondence: Alexander Hindenburg, 200 Old Country Road, Mineola, NY 11501, USA; fax: 516-663-4613; email: ahindenb@winthrop.org

Instructions for Infectious Disease Authors

Letters

This section includes letters that present preliminary data or comment on published articles. Letters (500–1,000 words) should not be divided into sections, nor should they contain figures or tables. References (not more than 10) may be included.

Correction Vol. 9, No. 4

In the article, “Antimicrobial Drug Prescriptions in Ambulatory Care Settings, United States, 1992–2000” by Linda F. McCaig et al. errors occurred on pages 432, 434, and 446. On page 432, the correct affiliations are as follows: Linda F. McCaig, National Center for Health Statistics, Centers for Disease Control and Prevention (CDC), Hyattsville, Maryland, USA; Richard E. Besser and James M. Hughes, National Center for Infectious Diseases, CDC, Atlanta, Georgia, USA. In the abstract, the change in antimicrobial prescribing rate for amoxicillin/clavulanate is +69%. On page 434, second paragraph, Results section, the correct first sentence appears below:

During the study period, the antimicrobial prescribing rate at all ambulatory care visits declined for amoxicillin and ampicillin (–43%; $p < 0.001$), cephalosporins (–28%; $p < 0.001$), and erythromycin (–76%; $p < 0.001$) (Figure 5); the prescribing rate rose for azithromycin and clarithromycin (+388%; $p < 0.001$), quinolones among persons ≥ 15 years (+78%; $p < 0.001$), and amoxicillin/clavulanate (+69%; $p = 0.004$) (Figure 6).

On page 436, the correct caption to Figure 6 appears below:

Trends in increasing annual antimicrobial prescribing rates by drug class—United States, 1992–2000. Note: trend for amoxicillin/clavulanate $p < 0.001$; for quinolones among persons ≥ 15 years, $p < 0.001$; for azithromycin and clarithromycin among all ages, $p < 0.001$.

The corrected article appears online at <http://www.cdc.gov/ncidod/EID/vol9no4/02-0268.htm>.

We regret any confusion these errors may have caused.