

Author affiliation: University of the Punjab,
Lahore, Pakistan

DOI: 10.3201/eid1708.100950

References

1. Idrees M, Lal A, Naseem M, Khalid M. High prevalence of hepatitis C virus infection in the largest province of Pakistan. *J Dig Dis*. 2008;9:95–103. doi:10.1111/j.1751-2980.2008.00329.x
2. Martell M, Esteban JI, Quer J, Genesca J, Weiner A, Gomez J. Hepatitis C virus circulates as a population of different but closely related genomes: quasispecies nature of HCV genome distribution. *J Virol*. 1992;66:3225–9.
3. Jarvis LM, Ludlam CA, Simmonds P. Hepatitis C virus genotypes in multi-transfused individuals. *Haemophilia*. 1995;1(Suppl):3–7. doi:10.1111/j.1365-2516.1995.tb00123.x
4. Forns X, Maluenda MD, Lopez-Labrador FX, Ampurdanes S, Olmedo E, Costa J, et al. Comparative study of three methods for genotyping hepatitis C virus strains in samples from Spanish patients. *J Clin Microbiol*. 1996;34:2516–21.
5. Lau JY, Mizokami M, Kolberg JA, Davis GL, Prescott LE, Ohno T, et al. Application of six hepatitis C virus genotyping systems to sera from chronic hepatitis C patients in the United States. *J Infect Dis*. 1995;171:281–9. doi:10.1093/infdis/171.2.281
6. Idrees M. Development of an improved genotyping assay for the detection of hepatitis C virus genotypes and subtypes in Pakistan. *J Virol Methods*. 2008;150:50–6. doi:10.1016/j.jviromet.2008.03.001
7. Idrees M, Riazuddin S. Evaluation of three different HCV typing methods for the detection of mixed genotype infections. *J Dig Dis*. 2011; (in press). doi:10.1111/j.1751-2980.2011.00496.x
8. Zarkesh-Esfahani SH, Kardi MT, Edalati M. Hepatitis C virus genotype frequency in Isfahan province of Iran: a descriptive cross-sectional study. *Virol J*. 2010;7:69. doi:10.1186/1743-422X-7-69
9. Silva MB, Andrade TM, Silva LK, Rodart IF, Lopes GB, Carmo TM, et al. Prevalence and genotypes of hepatitis C virus among injecting drug users from Salvador-BA, Brazil. *Mem Inst Oswaldo Cruz*. 2010;105:299–303. doi:10.1590/S0074-02762010000300009
10. Viazov S, Widell A, Nordenfelt E. Mixed infection with two types of hepatitis C virus is probably a rare event. *Infection*. 2000;28:21–5. doi:10.1007/s150100050005

Address for correspondence: Muhammad Idrees, Division of Molecular Virology and Molecular Diagnostics, National Centre of Excellence in Molecular Biology, University of Punjab, 87 West Canal Bank Rd, Thokar Niaz Baig, Lahore 53700, Pakistan; email: idreeskhan@cemb.edu.pk

West Nile Virus Aseptic Meningitis and Stuttering in Woman

To the Editor: West Nile virus (WNV), a mosquito-borne flavivirus, is closely related to St. Louis encephalitis virus and Japanese encephalitis virus (JEV). Most cases of WNV have been mild, but neuroinvasive disease has been observed, especially among older persons and immunocompromised persons (1,2). The most common neurologic manifestations of WNV are aseptic meningitis, meningoencephalitis, and encephalitis with or without acute flaccid paralysis (3). Other less common neurologic manifestations include Guillain-Barré syndrome, chorioretinitis, stroke-like symptoms, and unilateral brachial plexopathy (4,5).

We report a case of WNV aseptic meningitis in a 39-year-old immunocompetent woman who had severe headache with new-onset stuttering. Her medical history included lumbar disc herniation and migraines, for which she was taking sumatriptan. Her symptoms started ≈2 weeks before hospitalization and included a severe generalized headache initially thought to be a migraine, but sumatriptan resulted in no improvement. A few days later, she had fever and was intermittently stuttering. She denied recent travel or animal exposure but admitted to

having received multiple mosquito bites during the preceding weeks.

At admission, she had a temperature of 101.3°F, pulse rate of 92 beats/min, blood pressure of 130/80 mm Hg, and respiratory rate of 16 breaths/min. She appeared mildly ill but was alert and oriented with no nuchal rigidity, photophobia, rash, or limb weakness. Results of a physical examination were unremarkable, and results of a neurologic examination were notable only for stuttering. Laboratory test results included a leukocyte count of 12,300 cells/mm³ (63% neutrophils, 29% lymphocytes, 7% monocytes, 1% basophils) and a platelet count of 204,000 cells/mm³. Other laboratory values were unremarkable, and levels of serum transaminases and creatinine phosphokinase were within reference ranges. Cerebrospinal fluid (CSF) was clear and contained 37 leukocytes/mm³ (2% neutrophils, 78% lymphocytes, 20% monocytes), 2 erythrocytes/mm³, a glucose level of 68 mg/dL, a protein level of 36 mg/dL, and a lactic acid level of 2.1 meq/L. No abnormalities were found on a cranial computed tomography scan.

The patient began treatment with acyclovir, 10 mg/kg intravenously, every 8 hours for 3 days. On hospital day 2, she underwent magnetic resonance imaging of the brain; results were within reference limits. On hospital day 3, her headache began to improve and she became afebrile, but she still stuttered occasionally. Results of CSF tests for enterovirus, herpes simplex viruses 1 and 2, and varicella zoster virus and PCR for human herpesvirus 6 were negative, and acyclovir was discontinued. On hospital day 5, she was discharged. Three days later, serum and CSF ELISA results for WNV were positive. A WNV ELISA was performed at ViroMed Laboratories (Minnetonka, MN, USA) by using a Focus Test Kit (Focus Diagnostics, Cypress, CA, USA), and the result was positive. The

patient subsequently reported that her stuttering had ceased.

A high degree of clinical suspicion for WNV infection should be considered in patients with a recent history of mosquito bites and an acute febrile illness associated with neurologic signs and symptoms (5). Typical CSF findings of infection with WNV include lymphocytic pleocytosis, elevated protein level, reference glucose and lactic acid levels, and no erythrocytes (6).

The clinical presentation of WNV infection varied widely from asymptomatic seroconversion to fatal encephalitis. It is possible, but unlikely, that the stuttering in the patient was an indication of a migraine aura. Initially, the patient reported that the headache might have been a migraine, but later reported that its associated symptoms, e.g., photophobia, were not as severe and did not last as long as her usual migraines. Further argument against migraine aura is the lack of response to her migraine medication and the fact that the stuttering continued after the headache resolved.

Because WNV resembles JEV, it is interesting to note that a case of stuttering in a young adult infected with JEV has been reported (7). However, the mechanism of stuttering associated with WNV is unknown. One possible explanation is myoclonic contractions of the tongue, i.e., vocal myoclonus.

**Nardeen Mickail,
Natalie C. Klein,
and Burke A. Cunha**

Author affiliations: Winthrop-University Hospital, Mineola, New York, USA; and State University of New York School of Medicine, Stony Brook, New York, USA

DOI: 10.3201/eid1708.101691

References

1. Kramer LD, Li J, Shi PY. West Nile virus. *Lancet Neurol.* 2007;6:171–81. doi:10.1016/S1474-4422(07)70030-3

2. Klein NC, Johnson DH, Cunha BA, Minniganti V, Hansen E. West Nile encephalitis: the Long Island experience. *Infectious Diseases in Clinical Practice.* 2000;9:303–8. doi:10.1097/00019048-200009070-00008
3. Sejvar JJ, Marfin AA. Manifestations of West Nile neuroinvasive disease. *Rev Med Virol.* 2006;16:209–24. doi:10.1002/rmv.501
4. Weiss D, Carr D, Kellachan J, Tan C, Phillips M, Bresnitz E, et al. Clinical findings of West Nile virus infection in hospitalized patients, New York and New Jersey, 2000. *Emerg Infect Dis.* 2001;7:654–8. doi:10.3201/eid0704.010409
5. Cunha BA. Differential diagnosis of West Nile encephalitis. *Curr Opin Infect Dis.* 2004;17:413–20. doi:10.1097/00001432-200410000-00005
6. Tyler KL, Pape J, Goody RJ, Corkill M, Kleinschmidt-DeMasters BK. CSF findings in 250 patients with serologically confirmed West Nile virus meningitis and encephalitis. *Neurology.* 2006;14:361–5. doi:10.1212/01.wnl.0000195890.70898.1f
7. Chen WH, Peng MC. Acquired stuttering in a patient with encephalitis [in Chinese]. *Gaoxiong Yi Xue Ke Xue Za Zhi.* 1993;9:183–5.

Address for correspondence: Burke A. Cunha, Infectious Disease Division, Winthrop-University Hospital, Suite 432, 222 Station Plaza North, Mineola, NY 11501, USA; email: llusardi@winthrop.org

No Evidence of Dengue Virus Circulation in Rural Gabon

To the Editor: Dengue virus (DENV) is a mosquito-borne RNA virus belonging to the family *Flaviviridae*. It is composed of 4 closely related serotypes designated DENV-1–4. There are 2 transmission cycles for this virus. The endemic/epidemic cycle involves humans and the mosquito species *Aedes aegypti* and *Ae. albopictus*. The zoonotic or sylvatic cycle involves monkeys and

sylvatic *Aedes* spp. mosquitoes (1).

Despite occasionally severe clinical forms, human dengue usually consists of a self-limited febrile disease often associated with asthenia, headache, rash, arthralgia, and myalgia. DENV is widely distributed throughout Asia, the Pacific, Central and South America, the Middle East, and Africa (2,3). In Africa, most DENV outbreaks have been reported in the eastern regions, and episodic cases have occurred in western regions. However, few data are available for central regions.

In Gabon, concurrent of transmission of DENV and chikungunya virus was documented in 2007 during a large outbreak of dengue (4). This outbreak affected Libreville and major cities in northwestern Gabon and was caused by DENV-2. DENV isolates were closely related to strains from Asia, suggesting that the outbreak resulted from recent introduction of the virus. Epidemic DENV strains are constantly moving from one region to another, and local DENV transmission from sylvatic to urban areas has been documented in some countries in Africa (5,6).

To examine possible circulation of DENV in Gabon, we tested the following for antibodies against dengue: villagers living in rural areas, pet monkeys in the same areas, and wild monkeys killed in forests for bushmeat. A total of 4,341 persons and 186 pet monkeys were sampled during July 2005–May 2008 in 220 randomly selected villages, which represented 10.3% of all villages in Gabon. Fifty wild monkeys were also sampled during October 2009–August 2010 in different regions of Gabon (Table).

DENV-specific immunoglobulin (Ig) G and IgM were detected by using capture ELISA kits (Panbio; Brisbane, Queensland, Australia) (7) according to the manufacturer's instructions. All samples were tested with an IgG assay, which was designed to detect high antibody titers usually associated