Powassan Virus Encephalitis, Minnesota, USA

To the Editor: Birge and Sonnesyn report the first death of a Minnesota resident caused by Powassan virus (POWV) (*1*). However, they provide an inaccurate description of several critical diagnostic and surveillance issues concerning POWV.

The 17 POWV infections detected in Minnesota residents from 2008 through 2011 (6 cases were identified through 2010, not 8 as reported by Birge and Sonnesyn) (Minnesota Department of Health [MDH], unpub. data) were found through enhanced surveillance. Health alerts to Minnesota medical providers described POWV as a possible etiologic agent for viral meningitis and encephalitis. Providers consulted with MDH on suspected cases and submitted serum and cerebrospinal fluid specimens to MDH. MDH conducted serologic testing for endemic arboviruses (including POWV) and reverse transcription PCR (RT-PCR) for flaviviruses and POWV. MDH would not have detected any POWV infections without enhanced surveillance. Limited field studies also identified POWV-infected ticks in 4 Minnesota counties (not 2 as reported [*1*]) (MDH, unpub. data).

Commercial laboratories do not provide testing for POWV, and only a few state health department laboratories and the Centers for Disease Control and Prevention offer testing. Serologic testing (enzyme immunoassay with plaque-reduction neutralization testing confirmation) is preferred (*2*) because POWV RT-PCRs are not validated, and the short viremic periods of flaviviruses limit their usefulness (*3*).

Few POWV infections are identified by lineage (prototype vs. deer tick virus); Minnesota's first case in 2008 was identified as a deer tick virus infection, but the lineage was unknown for the other 16 cases. However, many case-patients had likely exposure to *Ixodes scapularis* ticks (blacklegged ticks), the tick species associated with deer tick virus transmission, and viruses from all POWV-positive tick pools were confirmed as deer tick virus by sequencing. The distribution of the 2 lineages in North America is poorly understood, and most cases likely go undetected without specific POWV surveillance efforts.

David F. Neitzel, Ruth Lynfield, and Kirk Smith

Author affiliation: Minnesota Department of Health, St. Paul, Minnesota, USA

DOI: http://dx.doi.org/10.3201/eid1904.121651

References

1. Birge J, Sonnesyn S. Powassan virus encephalitis, Minnesota, USA. Emerg Infect Dis. 2012;18:1669–71. http://dx.doi.org/10.3201/eid1810.120621
2. Hinten SR, Beckett GA, Gensheimer KF, Pritchard E, Courtney TM, Sears SD, et al. Increased recognition of Powassan encephalitis in the United States, 1999–2005. Vector Borne Zoonotic Dis. 2008;8:733–40.
3. Lanciotti RS. Molecular amplification assays for the detection of flaviviruses. Adv Virus Res. 2003;61:67–99. http://dx.doi.org/10.1016/S0065-3527(03)61002-X

Address for correspondence: David F. Neitzel, Minnesota Department of Health, Infectious Disease Epidemiology, 625 Robert St N, PO Box 64975, St. Paul, MN 55164, USA; email: david.neitzel@state.mn.us

Hepatitis E Virus and Porcine-derived Heparin

To the Editor: Cases of sporadic, locally acquired hepatitis E have been increasingly identified in industrialized countries over the last few years (*1*). In this setting, hepatitis E is thought to be a zoonotic infection, with pigs as the primary host. Consumption of uncooked or lightly cooked pork meat products is thought to be a key route of infection, but other routes of transmission have been documented (*2*). For example, there have been several iatrogenic cases after transfusion of hepatitis E virus (HEV)–contaminated blood products (*3*) and transplantation of an HEV-infected donor liver (*4*). However, in most cases the source and route of infection are uncertain.

In May 2011, a 42-year-old woman sought care at the Royal Cornwall Hospital in Truro, United Kingdom, for a 1-week history of malaise, diarrhea, nausea, and vomiting. Physical examination results were normal. Her liver function test results, however, indicated hepatitis: alanine aminotransferase 2,785 IU/L (reference range 10–36 IU/L), alkaline phosphatase 319 IU/L (reference range 30–130 IU/L), and bilirubin 30 μmol/L (reference range <21 μmol/L). HEV IgM and IgG serologic test results for the patient were positive, and HEV genotype 3 was identified in her blood by reverse transcription PCR and sequencing. Other causes of viral hepatitis and hepatocellular jaundice, including hepatitis viruses A, B, and C; Epstein-Barr virus; and autoimmune hepatitis, were excluded by testing. As with most immunocompetent persons with HEV, the patient made an uneventful clinical recovery after 12 weeks, and her liver function tests returned to normal after 8 weeks.