CARTAS AL EDITOR

Factores que intervienen en la génesis de una epidemia por dengue

Señor editor: la fiebre por dengue es la enfermedad viral transmitida por vector más importante a escala mundial,[1] y cada vez es más urgente obtener información que nos permita establecer cuáles son las condiciones que preceden a la aparición de una epidemia, en especial aquellas en las que se presentan casos con manifestaciones severas de la enfermedad: fiebre hemorrágica por dengue y síndrome de choque por dengue (FHD y SCD, respectivamente); por tal razón, es importante la aportación de trabajos como el de Espinoza-Gómez F y colaboradores,[2] en el cual describen la transmisión de la enfermedad en ausencia aparente de un brote epidémico, basados en la presencia de IgM en muestras de suero de personas asintomáticas. Sin embargo, habría que llevar a cabo trabajos que incluyan la mayor cantidad posible de información, ya que las epidemias son el producto de un conjunto de variables (mosquito vector, clima, estado nutricio, la capacidad virulenta de la cepa viral, movimientos poblacionales, etcétera) que coinciden en tiempo y espacio; de esta forma, el análisis en conjunto de todas estas variables nos permitiría identificar aquellos factores presentes en periodos interepidémicos que están involucrados en la subsiguiente aparición de epidemias. Por ejemplo, en el caso de factores climatológicos se han reportado brotes de dengue asociados con fenómenos como "El Niño";[3] asimismo, se ha observado que el incremento en el número de casos de dengue se asocia con el desplazamiento de la cepa viral predominante por una nueva en una región endémica específica.[4,5] Estas cepas nuevas pueden tener características de virulencia distintas, adquiridas mediante diversos mecanismos evolutivos. Estos eventos, así como otros (movimientos poblacionales, por ejemplo) pueden estar sucediendo en periodos interepidémicos. Tomando en cuenta lo anterior, sería interesante poder determinar en el trabajo de Espinoza-Gómez F y colaboradores, qué virus (serotipo/genotipo) estuvo asociado con esta transmisión interepidémica y si es el mismo virus asociado con epidemias anteriores o posteriores; por otra parte, estos datos podrían ser comparados con lo que ocurre en otras regiones endémicas en nuestro país.

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Detection of antibodies to West Nile and Saint Louis encephalitis viruses in horses

To the editor: West Nile virus (WNV) and Saint Louis encephalitis virus (SLEV) belong to the Japanese encephalitis antigenic complex (family *Flaviviridae*, genus *Flavivirus*).[1-3] As with other members of this complex, WNV and SLEV are usually maintained in cycles between birds and *Culex* species mosquitoes. Humans, horses and other mammals are typically dead-end hosts. WNV and SLEV infections in humans are usually asymptomatic or characterized by a mild febrile illness, although fatal meningoencephalitis or encephalitis may occur. WNV infection may also lead to fatal disease in horses, whereas clinical manifestations have not been reported in horses infected with SLEV.

The geographic ranges of WNV and SLEV include the Americas.[3,4] WNV activity has been reported in almost all of the continental United States,[5] southern Canada[6] and Mexico.[7-12] SLEV is endemic throughout the United States, particularly in central and eastern states and in the southwest, Mexico and Central America.[3,13] Studies were conducted to determine the prevalence of WNV and SLEV infections in horses in Nuevo León State, México.

Veterinary practitioners obtained blood samples from 88 horses between March 17 and April 4, 2003. The horses were from 29 randomly selected study sites in the municipalities of Pesquerías, Monterrey, Dr. Arroyo, Zuazua, Guadalupe, Santiago and Escobedo. All study sites were on privately owned properties. None of the horses had ever been outside of Nuevo Leon State. All horses were healthy at the time of serum collection and none had a history of WNV-like illness. None of the horses had been vaccinated against WNV.

All sera were tested for flavivirus-specific antibodies by epitope-blocking enzyme-linked immunosorbent assay (ELISA) as previously described.[14] Blocking ELISAs were performed using the flavivirus group-reactive monoclonal antibody (MAb) 6B6C-1 or the WNV-specific MAb 3.1112G. Sera positive for flavivirus antibodies by ELISA were tested by plaque reduction neutralization assay (PRNT) to identify the infecting virus. PRNTs were done using WNV (strain NY99-35261-11) and SLEV (strain TBH-28). Virus stocks were obtained from the World Health Organization Center for Arbovirus Reference and Research maintained at the Centers for Disease Control and Prevention, Division of Vector-Borne Infectious Diseases, Fort Collins, CO. PRNTs were performed using Vero cells. Sera were tested using a starting dilution of 1:20. Titers were expressed as the reciprocal of serum dilutions yielding ≥90% reduction in the number of plaques ($PRNT_{90}$).

Twenty-six (29.5%) of 88 horses had evidence of flavivirus-specific antibodies by both blocking ELISA and PRNT. Twenty (22.7%) horses were confirmed to have had a WNV infection, 1 (1.1%) was confirmed to have had a SLEV infection and 5 (5.7%) had antibodies to a flavivirus of undetermined etiology. The WNV-infected horses were from 3 municipalities: Pesquerías (n= 13), Zuazua (n= 6) and Guadalupe (n= 1). The WNV seropositive horses from Pesquerías were sampled at 3 study sites: Ejido Francisco Villa (n= 6), Sabinal (n= 5) and Los Dorados (n= 2). The WNV seropositive horses from Zuazua and Guadalupe were from the sites in Granja La Palma and Montada Guadalupe, respectively. One horse was confirmed to have had a SLEV infection: a 10 year-old stallion from the municipality of Pesquerías.

The mean age of flavivirus-infected horses (7±2.0 years) did not differ significantly from those horses that had not been infected (6±2.5 years). Of the horses with antibodies to flaviviruses, 77% were male and 23% were female (n= 20 and n = 6, respectively). Similarly, of the flavivirus-negative horses, 72% were male and 27% were female (n= 44 and n= 17, respectively).

Previously, we detected WNV RNA in brain tissue from a horse that died in June 2003 in the municipality of Juárez in Nuevo León State.[15] Nucleotide sequencing and phylogenetic studies demonstrated that this WNV was most similar to isolates collected in Texas in 2002. WNV infections have also been reported in horses or birds from the northern Mexican states of Coahuila, Tamaulipas and Chihuahua.[7-10] Taken together, our findings and data from previous surveillance efforts, suggest that WNV activity is now widespread in northern México.

Our studies complement recent serological surveys that have provided evidence of SLEV transmission in México. Ulloa and colleagues detected antibodies to SLEV in 20 of 202 (9.9%) domestic animals sampled in Chiapas State in 2001.[12] In addition, one of 102 (1.0%) resident birds captured in Yucatán State in 2002 had antibodies to SLEV.[16]

In summary, we report a high prevalence of antibodies to WNV in horses in Nuevo León State. Presumably, the geographic distribution of WNV in the Americas will continue to expand; thus, enhanced WNV surveillance in México, and south of there, should remain a priority.

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