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Short communication

Characteristics of an outbreak of West Nile virus encephalomyelitis in a previously uninfected population of horses

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Abstract

Equine West Nile virus (WNV) encephalomyelitis cases – based on clinical signs and ELISA serology test results – reported to Texas disease control authorities during 2002 were analyzed to provide insights into the epidemiology of the disease within a previously disease-free population. The epidemic occurred between June 27 and December 17 (peaking in early October) and 1698 cases were reported. Three distinct epidemic phases were identified, occurring mostly in southeast, northwest and then central Texas. Significant ($P < 0.05$) disease clusters were identified in northwest and northern Texas. Most (91.1%) cases had no recent travel history, and most (68.9%) cases had not been vaccinated within the previous 12 months. One-third of cases did not survive, 71.2% of which were euthanized. The most commonly reported presenting signs included ataxia (69%), abnormal gait (52%), muscle fasciculations (49%), depression (32%) and recumbency (28%). Vaccination status, ataxia, falling down, recumbency and lip droop best explained the risk of not surviving WNV disease. Results suggest that the peak risk period for encephalomyelitis caused by WNV may vary substantially among regions within Texas. Recumbent horses have a poor prognosis for survival. Vaccines, even if not administered sufficiently in advance of WNV infection within a district, may reduce the risk of death by at least 44%.

Keywords: Equine; West Nile virus; Epidemiology; Outbreak; Risk factors; Geostatistical analysis

1. Introduction

Horses are particularly susceptible to West Nile virus (WNV) infection and may present with acute clinical signs of encephalomyelitis, such as ataxia,

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rearlimb paresis, muscle tremors and fasciculations, and recumbency. Although 80% of affected horses recover in 3–4 weeks, a small proportion may have persistent neurological deficients. Treatment is supportive, including the use of anti-inflammatories, vitamins, fluids and antibiotics (Cantile et al., 2000; Murgue et al., 2001; Ostlund et al., 2001; Porter et al., 2001; Trock et al., 2001; Salazar et al., 2004; Ward et al., 2004). The presence of migratory birds, a susceptible horse population, and climatic and environmental conditions suitable for proliferation of infected mosquito vectors are factors that may result in an outbreak of equine WNV encephalomyelitis (Cantile et al., 2000). In temperate zones, disease risk appears to be greatest during a 3-month period between August and October, whereas in subtropical regions (such as Florida) the period of risk is longer and the outbreak pattern may not be as predictable (Murgue et al., 2001; Porter et al., 2001; Trock et al., 2001; Ward et al., 2004). Temperature and rainfall have been associated with some WNV epidemics in equine (Cantile et al., 2000; Ward et al., 2004) and human populations (Hayes, 2001). WNV was first detected in the state of Texas in 2002. During 2001, no human or equine cases were reported, WNV infection was not detected in sentinel flocks maintained in 28 Texas counties, and WNV was not identified in mosquitoes collected in 20 Texas counties (USGS, http://cindi.usgs.gov/hazard/event/west_nile/west_nile_2001.html. Accessed May 23, 2005). However, WNV was isolated from one human and five equine cases in neighboring Louisiana, and from six birds in Louisiana and Oklahoma. The objective of this study was to describe the first outbreak of WNV encephalomyelitis in the Texas equine population during 2002, and to identify variables associated with the risk of death of affected cases.

2. Materials and methods

Clinical cases of WNV encephalomyelitis occurring in the Texas equine population during 2002 were reported by owners and veterinarians to the Texas Department of State Health Services' Zoonoses Control Branch. Clinical signs were judged consistent with WNV disease by investigating veterinarians or TDSHS personnel, and cases were confirmed by IgM enzyme-linked immunosorbent assay (MAC-ELISA $\geq$ 1:400). Clinical signs and a positive MAC-ELISA are sufficient criteria for a probable case (Ostlund et al., 2001), the MAC-ELISA being a highly sensitive assay. WNV IgM antibodies are detectable 6–10 days post-infection, and persist for $<$2 to 3 months (Castillo-Olivares and Wood, 2004; Ostlund et al., 2001). Vaccination is unlikely to produce false positive IgM ELISA results (Hathaway et al., 2004).

Data available for cases included date of onset and county. In addition, most cases were investigated by Zoonoses Control Branch staff and a standard equine neurological disease report was completed. Ancillary data available from these reports included age (years), gender (male, male castrated, female), breed (specific breed types), presenting signs (excitable, fever, ataxia, falls down, recumbency, abnormal gait, muscle fasciculations, lip droop, difficulty eating or drinking, depressed, head pressing), WNV vaccination status and date (dates of administration up to 1 year prior to disease onset), history of travel outside the county of residence during 60 days preceding disease onset, case outcome (recovered, euthanized, died) and date, and clinical sequelae at the time of reporting. From a free-text field describing other presenting signs, the following variables were created based on the most frequently reported signs: lethargy, sweating, seizures, reluctance to walk, blindness, hypersensitivity and teeth-grinding. Information on the number of equine on case premises and number of other equine ill with signs suggestive of WNV disease was also recorded. In this report, only results of analyses of data describing confirmed (MAC-ELISA positive) cases in Texas are presented.

Disease attack rates (cases/population at-risk) were calculated for each Texas county (n = 254) in which a case of WNV disease was reported during 2002, using county equine census data for 2002 as the denominator (NASS, http://www.nass.usda.gov/census/census02/profiles/tx/index.htm. Accessed May 23, 2005). A frequency distribution of case age, gender, breed, vaccination status, travel history and reported clinical signs was formed. The odds of a case not surviving (versus surviving) were estimated for each of these variables by calculating unadjusted exposure odds ratios (ORs) and 95% confidence intervals (CIs) by use of a logarithmic approximation (SPSS version 12. SPSS Inc., Chicago, IL). Reference categories were