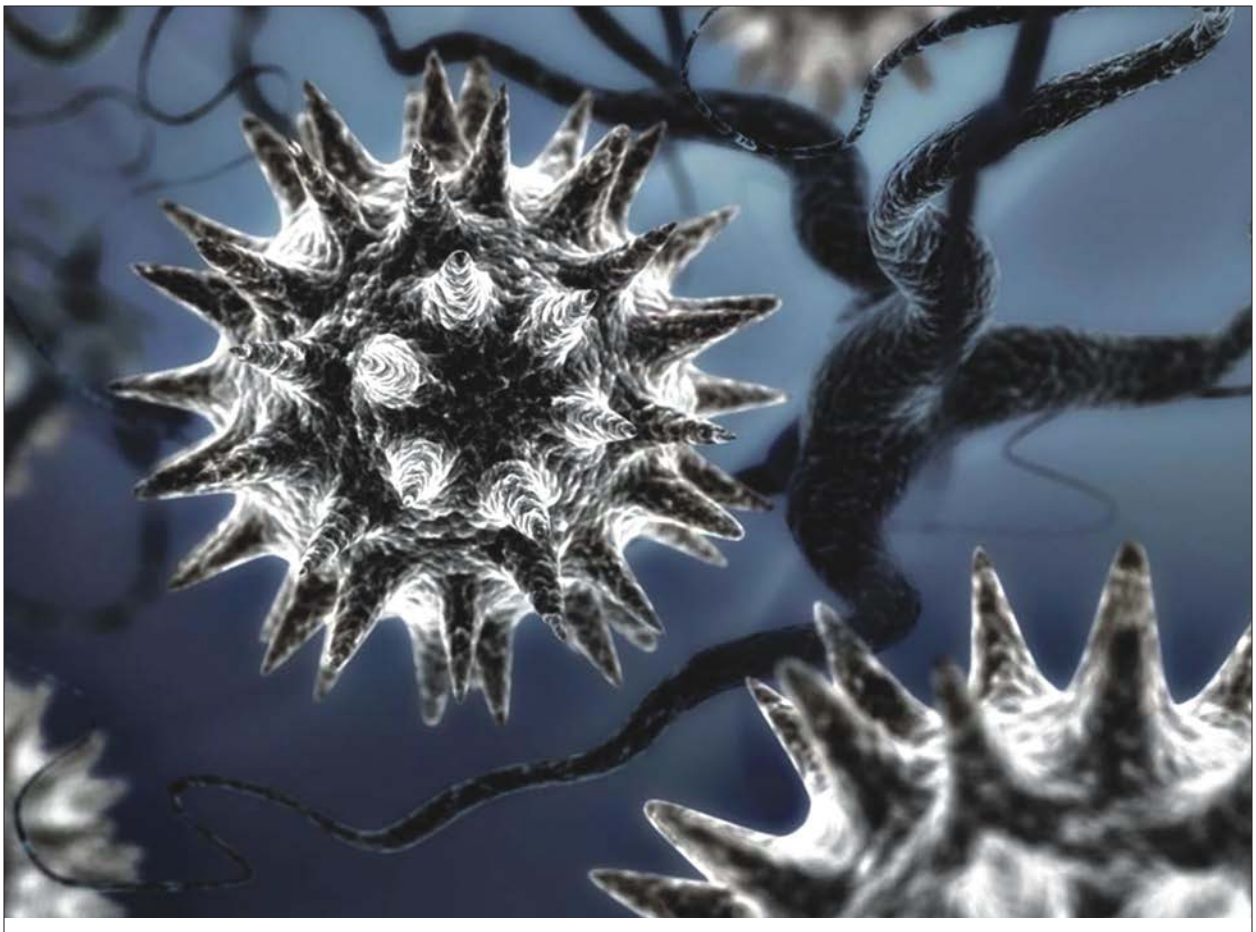

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Incidence of *West nile virus* in Al-Ahsa, Saudi Arabia

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ABSTRACT

West nile virus (WNV) causes neurological disease in both humans and horses that can be devastating in some cases. In the horse clinical manifestation of the disease may include ataxia, paresis or paralysis of the limbs, recumbency, hyperexcitability and hyperaesthesia. The goal of this study is to examine the incidence of WNV in horses in Al-Ahsa, Saudi Arabia. Clinical examination and serologic testing based on ELISA were performed on 63 horses. Clinical examination for neurologic signs detected no significant findings while serology positively identified WNV antibodies among 33.3% of tested population. Despite the fact no clinically ill horses were detected, the presence of seropositive animals and the wide range of hosts of the virus require further observation of the epidemiology of WNV in the future.

Key words: *West nile virus*, horses, clinical and serologic tests, antibodies

INTRODUCTION

West nile virus (WNV), an enveloped ssRNA virus member of the genus *Flavi virus*, family Flaviviridae (Monath and Heinz, 1996) causes neurological disease in both humans and horses (Petersen and Roehrig, 2001). During the past two decades, WNV has become a re-emerging essential pathogen, where outbreaks have occurred more frequently with higher proportion of neurological disease cases have been reported (Cantile *et al*., 2000; Chowers *et al*., 2001; Hubalek and Halouzka, 1999; Murgue *et al*., 2001; Weiss *et al*., 2001). During these outbreaks a remarkable increase in the number of severe human cases, significant increase in the severity of neurological disease in horses or high bird mortality were clearly noticed.

In Egypt, epidemiological studies detected viral activity in birds with seropositivity rate of 54% in horses and only one fatal case was reported (Schmidt and Elmansoury, 1963). Human cases with deaths were reported from Algeria in 1994 (Le Guenno *et al*., 1996), Romania in 1996 (Tsai *et al*., 1998), Morocco in 1996 (Tber-Aldelhaq, 1996), Tunisia in 1997 (Triki *et al*., 2001), Russia in 1999 (Platonov *et al*., 2001) and in Israel from 1998-2000 (Malkinson *et al*., 2002). In Israel, more than 400 people were infected, 325 of whom were hospitalized and 33 died. At least 75 horses were affected with encephalitis and 15 died, but, very unusually, there was also extensive mortality in birds, particularly geese (Banet-Noach *et al*., 2003; Steinman *et al*., 2002).

In the summer of 1999, WNV was reported for the first time in the western hemisphere. Fifty nine people had to be hospitalized as a result of severe neurological illness, including seven deaths, in the New York City metropolitan area (Anonymous, 2003; Asnis *et al*., 2000). At the same time, approximately 20 horses from Long Island were confirmed as WNV encephalitis cases (Trock *et al*., 2001). Since, then over 25,000 horses were reported with WNV (http://www.aaep.org/wnv.htm). Later investigations indicated that in contact animals were also seropositive to WNV (approximately 20%). There was significant avian mortality, in particular in crows, which in this case preceded the human and horse disease reports. Despite the cold New York

winter, the infection did not disappear and virus activity was first detected in 2000 when WNV was isolated from a Red-tailed hawk in New York in February 2000, as well as from adult *Culex* mosquitoes overwintering in protected areas in January and February 2000 (Anderson *et al*., 1999). By the end of the year, the Centers for Disease Control and Prevention had received reports of 21 human cases, 63 equine cases, 4304 infected dead birds and 6 infected other mammals (Petersen and Roehrig, 2001) in a total of 7 other northeastern states. WNV spread further to all the USA, Mexico, Canada and the Caribbean during the next three years.

The natural cycle of WNV involves birds as the main amplifying host and several species of mosquitoes as vectors typically but not exclusively *Culex* species (Komar, 2000).

WNV infection in horses is usually mild disease not associated with clinical illness. However, in recent years WNV saw an increased proportion of neurological disease in both humans and horses (Petersen and Roehrig, 2001). Approximately 10% of horses and around 1% of humans infected with WNV showed neurological disorders. Clinical signs of WNV in horses are mostly of neurological nature that reflect the pathology in the Central Nervous System (CNS). These occur predominantly in the spinal cord, rhombencephalon and mesencephalon being the cerebral cortex less often affected (Cantile *et al*., 2001). Such pathology may cause ataxia, paresis or paralysis of the limbs, which can affect one, two (usually the hind limbs) or all four limbs, the latter usually progress to recumbency. Hyperexcitability or even aggression, and hyperaesthesia may develop. A transitory febrile period might occur after infection although this is not always observed in some epidemics, e.g., outbreak in Italy 1998 (Cantile *et al*., 2001). A proportion of horses suffering from WNV infection do not recover and die spontaneously or, more often, are euthanased on humane grounds. Mortality rates among clinically affected horses have been estimated around 38, 57.1 and 42% during outbreaks in the USA in 2000, France in 2000 and Italy in 1998, respectively (Murgue *et al*., 2001; Ostlund *et al*., 2001). In contrast to the human disease, severe neurological disease in horses does not appear to occur preferentially in old individuals. Treatment is aimed at reducing CNS inflammation, preventing self-inflicted injuries and providing fluid and nutritional care.

The aim of this study was to determine if horses in Al-Ahsa were exposed or infected with WNV. Such study will provide valuable information on epidemiology and zoonotic potential of the disease.

MATERIALS AND METHODS

During the spring and summer of 2007, horse farms in Al-Ahsa, Saudi Arabia were visited in which 63 animals were examined physically. Horse age ranged between 3 and 12 years. Clinical changes including systemic signs associated neurolgic manifestation were observed. Signs of fever, off-feed, decrease performance as well as neurologic disorders such as ataxia, dysmetria, hyperexcitability, paresis or paralysis of the limbs, recumbency and muscle tremors were observed. In the meantime, 63 serum samples were collected from these horses residing horse. Samples were examined for WNV antibodies using ELISA as previously described (Wernery and Kennie, 2007).

RESULTS

Physical examination for systemic signs including fever, off-feed, decreased performance associated with neurological disease characterized by ataxia, dysmetria, hyperexcitability, paresis or paralysis of the limbs, recumbency and muscle tremors, failed to detect any significant findings. Signs of fever and decreased performance were detected in about 25% of the horses, however these changes were attributed to respiratory disease. On the other hand, serologic testing for

WNV antibodies detected antibodies titer in 33.3% of the examined animals. The horses that showed WNV titer had no evidence of neurological disease. Follow up calls with horse owners for one year revealed no case fatalities among WNV positively identified horses.

DISCUSSION

Data on WNV in horses in Saudi Arabia is rarely available. The incidence of seroconversion against WNV reported in this study is similar to reports in other countries (Schmidt and Elmansoury, 1963; Trock *et al*., 2001). Although, none of these seropositive horses showed any obvious clinical signs suggestive of WNV infection, the data reveal an alarming signs to veterinarian, health officials and horse owners. The presence of antibody titer against WNV indicates exposure to the disease however, it is very likely that these animals developed natural resistance overtimte. The horse is known to be a dead host of WNV since the amount of circulating virus in the blood is not adequate to allow the virus to be transmitted by the mosquitoes (http://www.aaep.org/wnv.htm). Nonetheless, additional work has to be done to determine the incidence of the virus in birds since they are the amplifying vector.

Antibody titer against WNV was also detected in a population of camels residing the same region of Saudi Arabia (Wernery *et al*., 2001 personal communication). In addition, studies showed the presence of arthropde vectors of WNV in Saudi Arabia (Al-Ali *et al*., 2008). The existence of the vector of the disease as well as the seroconversion in multiple animal species should be considered carefully in monitoring programs of WNV in the country. Severe forms of the disease in neighboring countries in multiple species could result from strain variation or differences in the host immunity experience (Malkinson *et al*., 2002; Banet-Noach *et al*., 2003; Steinman *et al*., 2002). This implicate that chances for emergence of more virulent form of the disease in both human and horse is not unlikely to occur.

Despite the fact that no clinical manifestation suggestive of WNV was found in our study since these systemic signs (fever and reduced performance) that were observed in some animals were attributed to a respiratory disease. Such finding was confirmed using serology and animals tested positive for EHV (unpublished data). The follow up work of these affected animals reported no case fatalities during this episod. *West nile virus* (WNV) causes devastating disease in the horse that requires aggressive veterinary care due to the neurologic manifestation, recumbency and muscle damage (Smith, 2002). Such care may not be available to horses in Saudi Arabia if were to be applied and so death of these clinically ill animals is inevitable. Therefore, early detection and prevention of disease spread are extremely crucial to save horses. In human a more severe form of the disease was observed in the United States.

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