

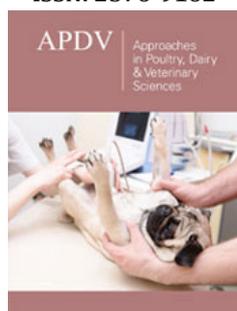
West Nile Fever in Brazil

Contarini de Oliveira SF¹, Perin Klippel DL¹, Lemos VZ¹, Coelho EC¹, Rondon DA¹, Moscon LA¹, Teixeira MC² and Pereira MC^{1*}

¹Centro Universitário do Espírito Santo (UNESC), Brazil

²Centro Universitário Ritter dos Reis (UniRitter), Brazil

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***Corresponding author:** Clairton Marcolongo Pereira, Centro Universitário do Espírito Santo (UNESC), Colatina, ES, Brazil

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Abstract

West Nile Fever is a zoonotic disease caused by the West Nile virus, which belongs to the family *Flaviviridae*. The transmission happens due to the bite of mosquitoes *Culex spp.* and it was first recorded in West Africa, but time by time the virus spread to others continents. Equines have quite a predisposition for this pathology and birds are the main reservoir. Once within the organism, the pathogen starts to replicate until it gets to the central nervous system, which is when it triggers encephalitis, and the neurological symptoms begin. Histologically, it can present inflammation of the neuronal tissue, variable degrees of necrosis and gliosis, and in the necropsy no lesions could be found. The diagnose is made with the physical examination, serological analysis and PCR test. The treatment is supportive for infected patients.

Keywords: West Nile virus; Zoonosis; Vector; Fever

Mini Review

West Nile Fever is caused by a flavivirus called West Nile Virus (WNV), which belongs to the family *Flaviviridae*. This pathogen is transmitted by mosquitoes and has a zoonotic character [1]. The first record of this virus parasitizing a horse was dated in 1956 in Egypt, and since the 90's decade, the number of this disease's reports increased significantly [1]. Equines are the most susceptible to the infection by the virus and lesions caused are restricted to the central nervous system [2]. Horses are infected by the bite of a mosquito that had previously fed on an infected animal such as birds, hamsters and eastern chipmunks [3]. Birds are the main reservoir of this virus. It is suggested that among the genus of mosquitoes that can play the role of vector, *Culex* deserves emphasis. That being said, *Culex pipiens*, *Culex quinquefasciatus*, *Culex nigripalpus*, and *Culex tarsilis* are some of the main species involved [4]. Even though it is not so common, it is possible to transmit the virus without the action of the arthropods. It might happen orally, in blood transfusion or in organ transplants [5]. It is still uncertain the exact mechanism and places where the virus will replicate once inside the body. However, it is believed that it might happen first on skin and lymph nodes and then it might reach the central nervous system [5]. When the horse is infected, the clinical signs are muscle tremors, ataxy, drowsiness, apathy, facial paralysis, difficulty getting up, fever and blindness [6]. Usually in the necropsy, no lesions can be found, but microscopically the lesions are located mainly in the brainstem and spinal cord [2]. Still histologically, the neuronal tissue might present inflammatory infiltration of the parenchyma, neuronal necrosis microglial nodules and gliosis [7]. It might appear, as well, some degrees of lymphocytic polioencephalomyelitis in the brainstem [8].

In 1999, WNV was reported in the United States of America for the first time, infecting humans and horses. Moreover, it did not take long to spread towards Latin America. There were records in 2005 in Colombia, 2006 in Argentina, for example. In Brazil, there has been reporting of serological evidences since 2008 [4]. The diagnose is based on clinical signs, serological analysis, and PCR tests [5]. When closing a diagnose of this pathology, it is fundamental to report this case, since this disease is one of the notifiable arboviral encephalitis [9]. As the WNV, Japanese encephalitis virus (JEV) and Saint Louis encephalitis virus (SLEV) have the same antigen complex, it might occur cross serological reaction, which hinders the

differentiation among them [1]. The main cause of encephalitis in horses in Brazil is rabies, but WNV cases are increasing significantly since 2018 [4] and should be included in the differential diagnosis of horses with neurologic signs. One way of avoiding this rise is reducing the number of vectors by eliminating mosquitoes breeding sites, avoiding stagnant water and small pools of water [9]. Once diagnosed, there is no specific treatment for this disease [3].

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