Focus Article: Equine Rabies

Dr Daniel L. Horton MA VetMB MSc PhD MRCVS
Wildlife Zoonoses and Vector Borne Diseases, Virology Department, Veterinary Laboratories Agency Weybridge, UK

Rabies is a fatal zoonotic disease, with a virtually global distribution and wide host range. It is a disease of great significance since the incubation period can vary from several days to many years. It is a disease that should not be overlooked in imported animals, particularly in those showing neurological signs.

Rabies virus is maintained in endemic cycles in a variety of mammals worldwide (including dogs, racoons, foxes, skunks) and in both terrestrial animals and bats in the Americas. Several countries such as the UK, Australia and much of Europe are free from disease. Cases in horses are rare, usually sporadic consequences of ‘spill-over’ transmission from a reservoir species. The risk therefore depends upon the occurrence of infected reservoir species. Of the 6,155 rabies cases reported in United States in 2010, only 37 (0.6%) were in horses or donkeys, which is comparable to the average of 38 cases per year over the preceding three years. The elimination of canine rabies, and rise in wildlife rabies in North America suggest that rabid wildlife pose the largest threat to horses in North America. In South America vampire bats play a significant role, infecting horses when taking a blood meal. In many other parts of the world including much of Africa and Asia, dog rabies remains endemic and therefore rabid dogs represent a source of infection to horses and other equines. In addition to rabies virus, there are ten other virus species in the genus lyssavirus. All are capable of causing rabies, and most have been isolated from bats. Only rabies virus has been associated with deaths in horses, but the repeated discovery of other lyssavirus species reinforces the need for characterisation of virus strains.

Virus Transmission
Rabies virus is most commonly transmitted in saliva via bites or scratches, but these events are often not witnessed in horses, and the wounds heal by the time clinical signs develop. In over 20 confirmed naturally occurring equine rabies cases in North America, none had visible bite wounds. Virus gains entry to the neurons and moves up the peripheral nerves into the central nervous system (CNS), is then distributed

Fig.1: Electron micrograph depicting the rabies virus
widely within the CNS and subsequently disseminates to multiple organs. Histopathological changes within the CNS include varying degrees of inflammation and classic cytoplasmic inclusion bodies known as Negri bodies in neurons. The degree of inflammation varies depending on the viral isolate, host and individual.

**Incubation Period**

The incubation period is commonly one to two months but can vary from one week to several years. A specific incubation period for equine rabies not been well documented, as exposure to the virus is rarely witnessed. One experimental study in horses showed a mean incubation period of 12.3 days but the dose and route of inoculation, site and severity of the wound, and pathogenicity of the viral strain will all have an effect on the incubation period. Clinical signs in horses do not appear to adhere strictly to the classic ‘furious’ or ‘dumb’ manifestations of disease. Instead there is a wide spectrum of signs, which overlap and it is not infrequent for horses to present with choke. The most common presenting signs in a case series of naturally infected North American horses were ataxia & paresis (43%), lameness (29%), recumbency (14%), pharyngeal paralysis (10%) and colic (10%). In 21 experimentally infected horses the most common signs were muzzle tremors (81%), lethargy (71%), ataxia (71%) and pharyngeal paralysis (71%). Death typically occurs within 7 days from the onset of clinical signs.

The clinical signs of rabies in the horse include: pyrexia, muzzle tremors, lethargy, ataxia and paresis, hyperaesthesia, pharyngeal paresis/paralysis, lameness, recumbency, tail, perineal and anal sphincter hypotonia, aggression, tenesmus, biting, convulsions, colic, head tilt, circling, hypersalivation and abnormal vocalisation.

**Diagnosis**

The wide range of clinical signs and frequent absence of relevant history make clinical diagnosis difficult. Laboratory confirmation ante mortem is also challenging as CSF may be normal. Most cases in horses are only confirmed post mortem using CNS samples, primarily brainstem, cerebellum, hippocampus and medulla. Direct fluorescent antibody testing (FAT) of fixed brain smears will detect viral antigen, and confirmation of diagnosis can be made by inoculation of tissue culture, inoculation of mice or molecular tests. The most recent developments of real-time quantitative polymerase chain reaction (real-time PCR) not only allow rapid diagnosis but also differentiation between lyssavirus species.

**Prevention**

Despite current interest in experimental therapy for humans, there are still no effective treatments and therefore control depends crucially on prevention of the disease developing. Immediate thorough wound cleaning is the most simple and effective preventive measure after potential exposure but that is rarely applicable for horses if a bite is not witnessed. Vaccination with modern tissue culture based vaccines is safe and effective for preventing disease. Although rabies in horses is rare, vaccination is generally recommended where possible in endemic countries. The difficulties in clinical diagnosis, combined with close contact between people and horses make vaccination also justified to reduce the risk to humans. This is particularly important for animals with exposure to large numbers of people, such as those in petting zoos or riding stables.
Although vaccinating horses will prevent disease, it has been shown repeatedly that the best way to reduce spill-over infections in humans and domestic animals is to control or eliminate disease in the reservoir species. Rabies is a notifiable disease in the UK and any clinical suspicion should be reported to the local Animal Health Office.