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A Review on Equine Rabies

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Abstract

Equines are very susceptible to the disease, and horses that come into contact with rabid animals are particularly at risk. The virus in the horse's body continues from days to months before signs develop, and once signs appear, it is fatal. Rabies is a global zoonotic disease, however due to successful prevention standards few countries are declared to be rabies free, like Islands that have a strict quarantine program. Clinical signs of rabies in the early stages are confused with other diseases like colic and normal aggressive tendencies. Diagnosis of rabies must be verified with confirmative laboratory tests. As a result of the concentration of the rabies virus in the secreted saliva from the infected horse it is mainly transmitted through its bite or if the infected saliva come in contact with the eyes, nose, or mouth, or broken skin of other animal or human. There is no treatment for it till now, so it must be prevented through vaccination, and infected animal should be euthanized, in addition to effective quarantine program.

Keywords: Prevention • Etiology • Rhabdovirus • Pathogenesis • Cardiorespiratory

Introduction

Rabies occurs worldwide in mammals, bats, and wild carnivorous which are the principle reservoirs. It remains a feared zoonotic disease throughout much of the world and kills more than 55,000 people and millions of animals worldwide [1]. Horses are extremely susceptible to infection and require lower dose of virus compared to other domestic species. Rabies is an acute, progressive encephalomyelitis caused by lyssaviruses, which are usually confined to one major reservoir species in a given geographic area, although spillover to other species is common [2]. Rabies in horses usually manifest as difficulty swallowing, ataxia, and progressive paralysis [3]. It is a notifiable disease in most countries, including UK, USA, Canada and Australia. It persists in wildlife populations throughout the United States, Canada, Mexico, and other parts of the world. It remains a threat to all domestic species, including the horse. Equine rabies has been reported in most countries. It remains a significant threat to horses in South and Central America. On the other hand, there are some countries that are free from the disease like England, New Zealand, Hawaii, Australia, and some Caribbean and Pacific Islands are free from the disease [4]. The veterinarians' ability to diagnose the disease has been improved greatly due to the advances in the technology used to detect the infection. Signs generally divided to three forms which are neurologic signs with 'paralytic', 'dumb' and 'furious' forms. Its diagnosis includes history, signs, post mortem diagnosis; immunohistochemistry of the hippocampus, cerebellum and brainstem. Because of the increasing urbanization of areas in which the disease is endemic in wildlife populations, the risk of exposure continues to be a concern for veterinarians and horse owners too. There is no specific age for infection as all ages affected. Till now there is no treatment for rabies infection and its prognosis is invariably fatal, but inactivated vaccine available as preventive method for the disease [5].

Literature Review

Etiology

Rabies is caused by lyssaviruses, *Lyssavirus* genus in the Rhabdovirus family. There are more than 16 different Lyssaviruses have been described. Globally, rabies virus is the most important member of the genus. The viruses contain a single linear molecule of minus sense ssRNS (11 to 12 kb), and consists of a lipid envelope containing glycoprotein "spikes" surrounding a helically wound nucleocapsid and forms a 170 × 70 nm bullet-shaped virion typical of all Rhabdoviridae [6]. The morphology of the active form is cylindrical shape with one rounded and one planer end.

There are knob shaped projections are regularly arranged over the surface of the virion (Figure 1).

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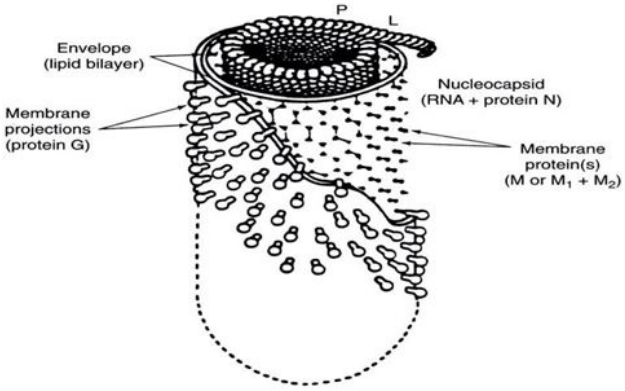

Figure 1. Knob shaped projections are regularly arranged over the surface of the virion.

Rabies virus could be inactivated by heating at 56°C for 30 min, ultraviolet light, by 1% formalin, 3% cresol, 0.1% beta propiolactone, and organic solvents, detergents. It could persist in infected brain tissue for 7-10 days at room temperature and for several weeks at 4°C. Rabies virus grows in cell culture at 37°C in an atmosphere of 0.5% carbon dioxide and 90% humidity. It replicates in cytoplasm of host cell, forming typical "Negri bodies", containing viral rib nucleoprotein.

Transmission and pathogenesis of rabies

Transmission of rabies infection usually occurs by the bite of a rabid animal and results in the inoculation of rabies-infected saliva in to sub-epithelium and striated muscle [7]. Firstly, the virus replicates at the site of the bite wound in the muscle cells and sub epithelial cells. Infection of motor and sensory nerves once sufficient concentration of virus is reached centripetal spread from site of inoculation to spinal cord and CNS *via* neuronal axons.

Sometimes long incubation periods noted with this disease may be related to viral replication in the muscle cells before viral entry into the nervous system [8]. It may transmit by the infected saliva contact with broken skin or intact mucous membrane from a rabid animal. Saliva is infectious before or when, the time clinical signs start. Lyssaviruses are highly neurotropic. Rabies virus travels *via* the peripheral nerves to the spinal cord and ascends to the brain, or it travels *via* peripheral nerves to the salivary glands and other organs after reaching the brain. When the infected animal is capable of transmitting rabies *via* its saliva, virus will be detectable in the brain. From CNS virions move centrifugally in peripheral nerves to all highly innervated organs, including salivary glands then it is bud from plasma membranes of mucous cells and is released in high concentrations in saliva [9].

Virus is shed intermittently in the saliva. Aerosol infection may occur *via* direct attachment of the virus to olfactory nerve endings [10]. After replication in the CNS, clinical signs developed, and at the end of this phase rabies virus may be found in nearly every innervated organ. Incubation period is very variable and depends on the local replication of virus in muscle at wound site before migrating to nervous tissues, site of bite wound and its closer to the CNS, dose of inoculated virus, and pathogenicity of strain of inoculated virus.

Generally, Incubation period from 2-9 weeks, but can be several months, and death usually occurs within 2-10 days after onset of clinical signs [11].

Clinical signs

Clinical signs of rabies are variable and confusing as early signs, such as lameness, choke or colic are general signs for many diseases. Rabies Signs ranged from obvious signs of neurologic disease and behavioral changes to lameness and colic [12]. Clinical signs dependent on sites of viral replication within CNS accordingly, there are 3 forms described in the horse, but many of the clinical signs overlap between forms. Most horses exhibit some combination of ataxia, fever, hyperesthesia, followed by ascending paralysis, recumbence and loss of tail and anal sphincter tone (Figure 2).

Figure 2. Clinical signs of rabies are variable and confusing as early signs.

Dumb or brainstem form

In this form the viral replication occur in brainstem. This form mainly characterized by difficulties in swallowing, progressive Pharyngeal paralysis, drooling, depression and ataxia may be observed. Horses with this form may present for choke.

Paralytic or spinal cord form

Viral replication occurs in spinal cord. The horse here suffers from ascending paralysis. As disease progresses get recumbency, loss of sensory perception of hind limbs and loss of tail and anal sphincter tone [16].

Furious or cerebral form

In this form the virus replication occur in cerebral cortex. The main signs vary between aggressiveness, convulsions, photophobia, hydrophobia, and hyperesthesia may be observed. In horses, the furious form of rabies has been noted in 43% of experimentally infected cases.

There are other signs observed about rapid horses but less common like sweating, muscle tremors, circling, head tilt, teeth grinding,

blindness, abnormal vocalization and roaring, they also may experience seizures. There are no reports of horses recovering from rabies infections. Signs like, fever, abdominal pain, behavioral changes, and cardiorespiratory dysfunction may be the result of anoxic/ischemic injury, particularly in the cerebral cortex and hypothalamic control centers of the brain which may lead to death. The disease is invariably fatal in horses, but survival time after the onset of signs may range from 2 to 5 days and can be as long as 2 weeks [17].

The length of the incubation period, strength of the clinical signs, and the survival time are related to the dose and pathogenicity of the viral strain, the host's immune status, and the proximity of the bite wound site to the CNS.

Discussion

Samples

Brain tissue, and Studies have shown that submission of spinal cord in livestock, including horses, leads to enhanced (3.5x) detection of lesions compared to submission of brain alone. The unfixed tissue refrigerated or fixed in 10% buffered formalin to be stored. All laboratory samples from rabies suspects should be handled carefully and labeled appropriately. The brain should not be frozen or placed in fixative because these procedures can interfere with fluorescent antibody test.

Identification of Negri bodies in brain tissue; pathognomonic. histopathology requires sample to be submitted in 10% buffered formalin [18].

FA technique for viral antigen in the brain requires refrigerated unfixed specimens. The rabies virus-specific fluorescent antibody test is the most widely used method for confirming infections and can identify 98% of infected brain tissues correctly. The procedure has been well standardized for brain specimens, and commercial conjugates are readily available. This test for the most part has displaced the need to confirm the diagnosis by histologic detection of Negri bodies.

PCR detection of the virus can be done on fixed or unfixed tissue; latter preferred. Polymerase chain reaction assays also can be performed using primers that amplify genomic RNA and viral mRNA sequences from ante mortem or postmortem specimens.

Serum and CSF neutralization tests and fluorescent antibody tests on CSF, skin, tactile hair follicles, cornea, saliva, or salivary gland have been used as antemortem immunodiagnostic tests, but difficulties in their interpretation because of false-negatives and false-positives has limited their usefulness. Electron microscopic examination and fluorescent antibody studies from humans and animals that died from the disease revealed that almost all neurons in the CNS were infected with the virus Negri bodies, which are intracytoplasmic inclusions of viral ribo nucleoprotein matrices found most commonly in the hippocampus and Purkinje's cells in the cerebellum, are pathognomonic for the disease. These inclusions have been identified as areas of active viral replication by the identification of rabies viral antigen.

They generally are easier to find if the incubation period has been long and the clinical course protracted. In addition to the brain, spinal cord and peripheral nerve should be examined.

Zoonotic importance of rabies virus

Rabies is a major zoonotic hazard. It is a member of the Rhabdovirus family, the genera of which includes two zoonotic pathogens, both transmissible to human beings from horses: Lyssavirus (rabies virus) and Vesiculovirus (vesicular stomatitis virus).

Rabies has the highest case fatality of any infectious disease. When a person is exposed to an animal suspected of having rabies, the risk of rabies virus transmission should be evaluated carefully. The virus has been transmitted by transplantation of tissues and organs from infected people. CSF may harbor low levels of the virus, and the zoonotic risk, combined with the often impracticality of CSF collection in the field, should be considered.

Treatment

As rabies is a highly contagious viral infection with no effective treatment up to now, all treatments which done is symptomatic, but there is no treatment for the infection. Bite wounds should be thoroughly washed with soap and water. The supportive treatment of the symptomatic patient is only indicated in humans, and euthanasia is recommended once clinical signs have developed.

Prevention and control

Quarantine of animals when imported into rabies-free countries from countries where rabies is endemic strictly for 6 months period.

Disease prognosis is fatal so, vaccination programs of wildlife reservoirs and stray dogs are required to control and eradicate the disease (Figure 3).

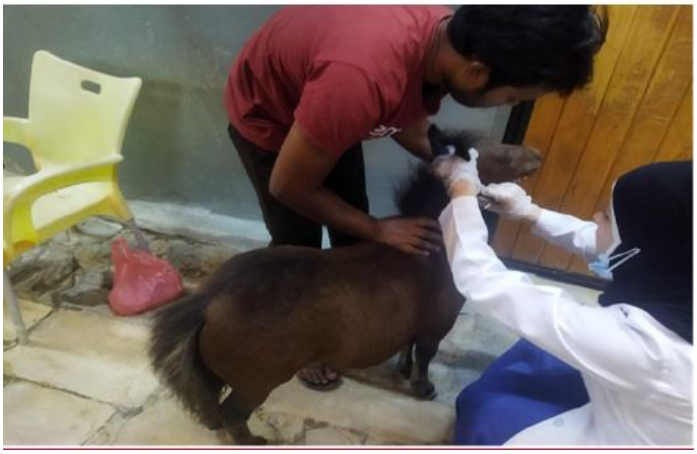

Figure 3. Vaccination program.

All staff that exposed to confirmed or suspected case of equine rabies like veterinarians, horse owners, nurses or other personnel should be reported to appropriate authorities to determine need for rabies treatment or prophylaxis.

All current licensed equine rabies vaccines are inactivated (killed), and it is recommended for intramuscular injection, and appears safe and effective. Primary vaccination of horses 3 months or older, second dose at 1 year of age, followed by 6 monthly or annual boosters. Studies have shown that after the initial vaccine series, neutralizing antibodies may persist for at least three years.

It is not recommended to give pregnant Mares rabies vaccine, to provide safety during gestation period because of lack of safety studies. It should therefore receive boosters before breeding. Modified live rabies vaccines should not be used in horses.

The rabies vaccines are considered highly protective, yet failure or breaks in vaccine protection has been described in young horses reportedly vaccinated for rabies, suggesting that a two-dose primary vaccination series for foals should be evaluated. Success of vaccination must be checked using a blood test; animals must show a rabies antibody titer ≥ 0.5 IU/ml. Vaccination results in circulating neutralizing antibodies within 3 weeks. T and B lymphocyte responses necessary for clearance. Immune response from vaccination can prevent rabies, but response to natural infection does not aid in recovery. Following exposure, intervention by neutralizing antibody (produced by previously vaccinated host animal or passively administered to immunologically naïve animals must occur prior to nerve entry.

Conclusion

Recombinant of thymidine kinase deficient vaccinia or canary pox virus with rabies glycoprotein gene. Used for oral vaccination of wildlife, and in some European countries it has been successful.

The vaccinated animal is considered immunized if it was vaccinated at least 30 days previously, while the exposed but vaccinated horse should be revaccinated immediately and observed for 45 days. The unvaccinated exposed horse should be euthanized immediately, or, if its owner is unwilling, the horse should be kept quarantined under observation for 6 months, and if it developed neurologic signs during quarantine, it should be euthanized. As rabies is a reportable disease, the management of a rabies suspect should be undertaken in consultation with state or federal public health authorities. For histopathological examination and fluorescent antibody test the head of the rapid horse should be removed and shipped in a well-sealed and labeled container under refrigeration (or cold-packed), and it must sent to a qualified laboratory.

The risk of transmission of rabies from an infected horse to a human being should be considered carefully, for instance the exposure of horse owners, animal health technicians, veterinarians, or other personnel to a confirmed equine rabies case or a rabies suspect should be reported immediately to a physician who can determine the need for rabies treatment and prophylaxis. Veterinarians, animal health technicians, and animal caretakers should be vaccinated prophylactically against rabies.

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