

African Horse Sickness

Perdesiekte, Pestis Equorum, La Peste Equina, Peste Equina Africana

Importance

African horse sickness is a serious viral infection of horses spread by midges. Mortality can be as high as 95% in some forms of this disease. Asymptomatic or mild infections may occur in zebras, African donkeys, and horses previously infected by another serotype of the virus. Potential arthropod vectors may exist in the United States.

Etiology

African horse sickness is caused by the African horse sickness virus, an arthropod-borne orbivirus in the family Reoviridae. There are nine serotypes of the virus.

Species affected

African horse sickness can affect horses, donkeys, mules, zebra, and camels. Zebras may be asymptomatic carriers in Africa, and horses and mules may be accidental hosts. Dogs are susceptible to experimental infections. Serum samples from African elephants are positive in complement fixation tests, but there is no evidence of virus replication in this species.

Geographic distribution

African horse sickness is endemic in sub-Saharan central and east Africa. This disease often spreads to southern Africa and occasionally to northern Africa. Outbreaks have been seen in Egypt and other parts of the Middle East, as well as in Spain.

Transmission

African horse sickness is not contagious. The causative virus is transmitted by midges in the genus *Culicoides*. Field vectors include *Culicoides imicola* and *C. bolitinos*; *Culicoides imicola* appears to be the most important vector. Potential *Culicoides* vectors may exist in the United States.

Transmission by insects other than midges is thought to be a minor source of infection. Mosquitoes have been implicated as biological vectors, and biting flies in the genera *Stomoxys* and *Tabanus* may be able to transmit the virus mechanically.

Incubation period

In experimental infections, the incubation period can range from 2 to 14 days; most often, clinical signs appear 5 to 7 days after infection. In natural infections, the incubation period appears to be approximately 7 to 14 days.

Clinical signs

Four different forms of African horse sickness may be seen: the peracute or pulmonary form, the subacute edematous or cardiac form, the acute or mixed form, and horsesickness fever.

The Peracute or Pulmonary Form

This form of African horse sickness usually begins with an acute fever, followed by the sudden onset of severe respiratory distress. Infected animals often stand with forelegs spread, head extended, and nostrils fully dilated. Other clinical signs may include tachypnea, forced expiration, profuse sweating, spasmodic coughing, and a frothy serofibrinous nasal exudate. Dyspnea usually progresses rapidly, and the animal often dies within a few hours after the respiratory signs appear.

The Subacute Edematous or Cardiac Form

The cardiac form of African horse sickness usually begins with a fever that lasts for 3 to 6 days. Shortly before the fever starts to subside, edematous swellings appear on the supraorbital fossae and eyelids. These swellings later spread to involve the cheeks, lips, tongue, intermandibular space, laryngeal region, and sometimes the neck, shoulders, and chest. However, it is important to note that no edema of the lower legs is observed. Other clinical signs, usually seen in the terminal stages of the disease, can include severe depression, colic, and petechiae under the ventral surface of the tongue and in the conjunctivae. Death often occurs from cardiac failure. If the animal recovers, the swellings gradually subside over the next 3 to 8 days.

The Acute or Mixed Form

In this form of African horse sickness, symptoms of both the pulmonary and cardiac forms are seen. In most cases, the cardiac form is subclinical and is followed by severe respiratory distress. Occasionally, mild respiratory signs may be followed by edema and death from cardiac failure. The mixed form of African horse sickness is rarely diagnosed clinically, but is often seen at necropsy in horses and mules.

Horsesickness Fever

In horsesickness fever, the clinical signs are mild. The characteristic fever usually lasts for 3 to 8 days; morning remissions and afternoon exacerbations are often seen. Other symptoms are generally mild and may include mild anorexia or depression, congested mucous membranes, and an increased heart rate. This form of the disease is rarely fatal.

Post mortem lesions

In the pulmonary form of African horse sickness, the typical lesion is edema of the lungs or hydrothorax. In the most acute cases, extensive alveolar edema and mottled hyperemia of the lungs may be seen. In more prolonged cases, there may be extensive interstitial and subpleural edema, and hyperemia may be less apparent. Occasionally, extensive fluid accumulation may be noted in the thoracic cavity, with near normal appearance of the lungs. The lymph nodes, particularly the nodes in the thoracic and abdominal cavities, are usually enlarged and edematous. Less often, there may be subcapsular hemorrhages in the spleen, congestion in the renal cortex or gastric fundus, and edematous infiltration around the aorta and trachea. Hyperemia and petechial hemorrhages may be apparent in the small and large intestines, and the pericardium may contain petechiae.

In the cardiac form, a yellow gelatinous infiltrate can be seen in the subcutaneous and intermuscular fascia of the head, neck, and shoulders, and occasionally the brisket,

ventral abdomen and rump. Hydropericardium is common. The epicardium and endocardium often contain petechial and ecchymotic hemorrhages. Lesions may also be found in the gastrointestinal tract, resembling the pulmonary form. In addition, prominent submucosal edema may be noted in the cecum, large colon, and rectum. In the cardiac form, the lungs are usually normal or slightly engorged, and the thoracic cavity rarely contains excess fluid.

In the mixed form, the post-mortem lesions are a mixture of typical findings from both the cardiac and pulmonary forms.

Morbidity and Mortality

Morbidity and mortality vary with the species of animal and previous immunity. Horses are particularly susceptible to this disease. In this species, the mortality rate varies from 50% to 95%, depending on the form of the disease. In the cardiac form, mortality is usually 50-70% and, in the mixed form, greater than 80%. The pulmonary form is almost always fatal, but horsesickness fever rarely results in death. In other species of Equidae, African horse sickness is generally less severe. In mules, the mortality rate is approximately 50%, and in European and Asian donkeys, 5-10%. Death is rare in African donkeys and zebra.

Animals that recover from African horse sickness develop good immunity to the infecting serotype, and there is partial immunity to other serotypes. A vaccine is available in endemic countries.

Diagnosis

Clinical

African horse sickness should be suspected in animals with typical symptoms of the cardiac, pulmonary, or mixed forms of the disease. The supraorbital swellings are particularly characteristic of this disease. The horse sickness form can be difficult to diagnose.

Differential diagnosis

The differential diagnosis includes encephalosis, equine viral arteritis, equine infectious anemia, equine morbillivirus pneumonia, purpura hemorrhagica, and equine piroplasmiasis.

Laboratory tests

African horse sickness can be diagnosed by isolating the virus or detecting its nucleic acids or antigens. More than one test should be used to diagnose an outbreak. Virus isolation is particularly important when outbreaks are seen outside endemic areas. Suitable cultures for inoculation include baby hamster kidney (BHK-21), monkey stable (MS) or African green monkey kidney (Vero) cells. Isolation is also possible in embryonic eggs or newborn mice. The virus isolate should be serotyped by virus neutralization or other methods.

Viral antigens can be detected by enzyme-linked immunosorbent assays (ELISAs). A reverse-transcription polymerase chain reaction (RT-PCR) technique is used to detect viral RNA.

African horse sickness may also be diagnosed by serology. Antibodies can be detected within 8 to 12 days after infection and may persist for one to four years. The African horse sickness virus does not cross-react with other known orbiviruses. Available serologic tests include complement fixation, ELISAs, immunoblotting, and virus neutralization. The indirect ELISA and complement fixation tests are the prescribed tests for international trade. The virus neutralization test is used for serotyping. Immunodiffusion and hemagglutination inhibition tests have also been described.

Samples to collect

Before collecting or sending any samples from animals with a suspected foreign animal disease, contact the AVIC. These samples should only be sent under secure conditions, by authorized personnel, and to authorized laboratories to prevent the spread of disease.

In live animals, uncoagulated blood samples should be taken for virus isolation. Success is most likely if these samples are collected early during the febrile stage. Necropsy samples for virus isolation should include small (2-4 g) samples of the spleen, lung, and lymph nodes. The samples for virus isolation should be stored and transported at 4°C. Serum should also be taken for serology.

Recommended actions if African horse sickness is suspected

Notification of authorities

African horse sickness should be reported immediately to state or federal authorities upon diagnosis or suspicion of the disease. Federal: Area Veterinarians in Charge (AVICS)

http://www.aphis.usda.gov/vs/area_offices.htm

State vets: <http://www.aphis.usda.gov/vs/sregs/official.html>

Quarantine and Disinfection

Susceptible animals from Asia, Africa, and the Mediterranean are quarantined in insect-proof facilities at the point of entry for a minimum of 60 days.

If African horse sickness is detected in a country where it is not endemic, a strict quarantine zone should be established. All Equidae should be sprayed with insect repellants and, at a minimum, stabled from dusk to dawn. If possible, animals should be stabled in insect-proof housing. Each susceptible animal should have its temperature taken regularly (optimally, twice daily). Those animals that develop a fever should be kept in insect-free stables until the cause of the fever has been established, or killed. These control measures should be implemented upon suspicion of the disease. Vaccination should be considered once the diagnosis has been confirmed.

The African horse sickness virus can be inactivated by formalin, β -propiolactone, acetyleneimine derivatives, or radiation. It is also destroyed at a pH less than 6, or pH 12 or greater.

Public health

Humans are not natural hosts for the African horse sickness virus, and no cases have been seen after contact with field strains. However, a neurotropic vaccine strain, adapted to mice, can cause encephalitis and retinitis in humans.

For More Information

World Organization for Animal Health (OIE)
<http://www.oie.int>

OIE Manual of Standards
http://www.oie.int/eng/normes/mmanual/a_summry.htm

OIE International Animal Health Code
http://www.oie.int/eng/normes/mcode/A_summry.htm

USAHA Foreign Animal Diseases book
http://www.vet.uga.edu/vpp/gray_book/FAD/

References

“African Horse Sickness.” In *Manual of Standards for Diagnostic Tests and Vaccines*. Paris: World Organization for Animal Health, 2000, pp. 178-188.

“African Horse Sickness.” In *The Merck Veterinary Manual*, 8th ed. Edited by S.E. Aiello and A. Mays. Whitehouse Station, NJ: Merck and Co., 1998, pp. 496-7.

Erasmus, B.J. “African Horse Sickness.” In *Foreign Animal Diseases*. Richmond, VA: United States Animal Health Association, 1998, pp. 41-51.