African Horse Sickness

Perdesiekte,
Pestis Equorum,
Peste Equina,
Peste Equina Africana

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Importance

African horse sickness (AHS) is a serious arthropod-borne viral disease of equids, with a mortality rate that can reach 95% in some species, such as horses. At present, AHS virus (AHSV) is only endemic in Africa; however, suitable vectors exist outside this area, and infected animals or vectors may carry the virus into AHS-free regions. The potential for dissemination is especially high in animals that tend to develop mild or subclinical infections, such as zebras (*Equus burchelli*) and donkeys, or horses with partial immunity. One extensive epidemic in 1959–1961 affected the Middle East and parts of Asia, as well as Africa, and is thought to have been responsible for the deaths of 300,000 equids. An outbreak in Spain lasted from 1987, when the virus was introduced in imported zebras, to 1990, and spread to Portugal and Morocco. Within Africa, additional AHSV serotypes have recently spread to some areas where only one serotype was previously found. Although vaccines are available, cross-protection between serotypes is limited, and the introduction of a new serotype into an area may result in outbreaks.

Etiology

African horse sickness results from infection with the African horse sickness virus (AHSV), a member of the genus *Orbivirus* in the family Reoviridae. There are nine serotypes of this virus, and while some serotypes are cross-protective (e.g., serotypes 6 and 9), others are not.

Species Affected

Equids including horses, donkeys, mules and zebras are the primary hosts for AHSV; however, this virus is also known to affect dogs. Among equids, the most serious infections occur in horses and mules, which are thought to be accidental hosts. Zebras, which are often asymptomatic, are thought to be the natural reservoir hosts in most regions of Africa.

Antibodies to AHSV have been reported in a number of other species, although there are sometimes inconsistencies between studies. Seropositive animals have included various wild carnivores, such as hyenas (*Crocuta crocuta*), jackals (various *Canis* spp.), African wild dogs (*Lycaon pictus*), cheetahs (*Acinonyx jubutus*), lions (*Panthera leo*) and large-spotted genets (*Genetta maculata*), which might be exposed by feeding on infected zebras. Some authors have reported that carnivores may have antibodies to AHSV serotypes (e.g., serotype 4) that are not necessarily common among equids in the area. There are also reports of seropositive herbivores including dromedary camels (*Camelus dromedarius*), sheep, goats, African elephants (*Loxodonta africana*), black rhinocerus (*Diceros bicornis*) and white rhinocerus (*Ceratotherium simum*). Attempts to establish experimental infections resulted in seroconversion with no evidence of virus replication in African elephants, and seroconversion in hyenas, while mink (*Mustela vison*) did not seroconvert or replicate virus. The significance of seropositive animals is still unclear, and no animals other than equids are thought to be important in maintaining or amplifying AHSV.

Zoonotic potential

African horse sickness is not zoonotic.

Geographic Distribution

African horse sickness is endemic in sub-Saharan Africa. Serotype 9 is widespread in the endemic region, while serotypes 1 to 8 occur in limited areas. The greatest virus diversity has been reported in southern and eastern Africa. Some serotypes have recently caused outbreaks in countries where they were not previously found. In particular, serotypes such as 2, 4, 6, 7 and 8 have been detected in regions where only serotype 9 was once common.

African horse sickness outbreaks have occurred outside Africa in the Middle East, the Mediterranean region of Europe and parts of Asia (e.g., the Indian subcontinent). Although all outbreaks, to date, were eventually eradicated, AHSV was able to persist for years in some areas.

Transmission

AHSV is transmitted by the midges *Culicoides imicola* and *C. bolitinos* in Africa. Additional species of *Culicoides* might also be able to act as vectors. They include species found outside the endemic region, such as *C. variipennis,* which occurs in North America and is an efficient AHSV vector in the laboratory, and *C. brevitarsis*, which is common in Australia. Wind has been implicated in the dispersal of infected vectors during some epidemics. AHSV is capable of overwintering, at least in milder climates where *Culicoides* adults can survive. Other arthropods might also be capable of transmitting AHSV, but are thought to be, at most, only minor sources of infection. Mosquitoes have been implicated as possible biological vectors, and biting flies in the genera *Stomoxys* and *Tabanus* may be able to transmit the virus mechanically. AHS virus was also isolated from the camel tick *Hyalomma dromedarii* in Egypt, and the dog tick, *Rhipicephalus sanguineus*, could transmit AHSV from dogs in the laboratory.

Zebras are thought to be the natural reservoir hosts for AHSV, but horses, mules and donkeys can also develop viremia sufficient to infect *Culicoides*. How long equids remain infectious for the vector is not certain; however, AHSV could be isolated from the blood of some experimentally infected zebras for up to 40 days, and from the spleen at 48 days. In experimentally infected horses, this virus has been recovered from blood for as long as 21 days, although viremia typically lasts only 4-8 days. Donkeys are reported to remain viremic for up to 28 days, and viral RNA has been detected longer in some studies (e.g., 47 days or more in some experimentally infected animals inoculated with serotype 4). African horse sickness is not transmitted between equids by casual contact.

Dogs can be infected if they eat contaminated horsemeat, and experimental infections have been established by the oral route as well as by subcutaneous or intravenous inoculation. AHSV is reported to persist for some time in frozen meat, and it is reported to survive for up to 6 months at 4ºC in medium containing serum, and for more than 2 years in putrid blood. Although arthropods including ticks can transmit AHSV from dogs to other dogs or horses in the laboratory, viremia is usually low and transient, and dogs are not thought to be significant in the epidemiology of African horse sickness. In addition, dogs are not usually hosts for *Culicoides* in the endemic region.

Disinfection

AHSV can be inactivated in the laboratory with formalin, β-propriolactone, acetylethyleneimine derivatives or radiation. It is resistant to liquid solvents such as ether. It is also destroyed at pH < 6 or pH ≥ 12. Acidic disinfectants such as 2% acetic or citric acid have been recommended for decontamination when it is warranted. AHSV is relatively heat stable, and it was not inactivated by heating in citrated plasma at 55-75°C for 10 minutes.

Incubation Period

The incubation period for African horse sickness in equids is approximately 3 days to 2 weeks (usually < 9 days), with the cardiac form typically developing later than the pulmonary form. Experimental infections suggest that the incubation period is potentially be as long as 21 days.

Clinical Signs

Four different forms of African horse sickness exist: the peracute (pulmonary) form, the subacute edematous (cardiac) form, the acute (mixed) form, and horsesickness fever. Sudden death can also occur without preceding signs. Symptomatic infections are seen most often in horses and mules, with the pulmonary and mixed forms usually predominating in susceptible populations of horses. Zebras and donkeys rarely develop serious clinical signs. The mildest form, horsesickness fever, tends to develop in resistant species such as donkeys, or in horses with partial immunity. This form can also occur in zebras, although most infections in this species are asymptomatic.

The peracute or pulmonary form

The pulmonary form of African horse sickness usually begins with an acute fever, followed within a day or two by the sudden onset of severe respiratory distress. Animals with this form 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 less than a week. Shortly before the fever starts to subside, edematous swellings appear in the supraorbital fossae and eyelids. These swellings later spread to involve the face, tongue, intermandibular space, laryngeal region, and sometimes the neck, shoulders and chest. Edema of the lower legs is absent. Other clinical signs, usually seen in the terminal stages of the disease, can include severe depression, colic, petechiae or ecchymoses on the ventral surface of the tongue, and petechiae in the conjunctivae. Death often occurs from cardiac failure. If the animal recovers, the swellings gradually subside over a few days to a week.

The acute or mixed form

Clinical signs of both the pulmonary and cardiac forms are seen in the mixed form. 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. Although the mixed form is common, it may not be recognized except at necropsy.

Horsesickness fever

Horsesickness fever is characterized mainly by fever, often with morning remissions and afternoon exacerbations. Other signs are generally mild and may include mild anorexia or depression, edema of the supraorbital fossae, congested mucous membranes and an increased heart rate. Death would be unusual.

Infections in dogs

The pulmonary form is reported to be the most common form in dogs. Fatal cases have been described in dogs that ate infected meat during epidemics. In one outbreak, 13 of 17 dogs died with unspecified clinical signs, while two animals that survived longer had fever, depression, dyspnea, moist rales and white foam around the nostrils, and died within 24 hours despite intensive care. Some dogs living in endemic regions are seropositive, suggesting that milder cases or subclinical infections also occur, and AHSV has been isolated from feral dogs not reported to be ill. In early experiments, which used crude preparations of virus (e.g., filtered blood from infected horses), some dogs had no apparent signs of illness, while others developed febrile reactions and survived, or died with evidence of severe pulmonary disease.

Post Mortem Lesions   📷 **Click to view images**

Equids

In the pulmonary form of African horse sickness, the characteristic lesions are interlobular edema of the lungs and hydrothorax. In the most acute cases, frothy fluid is present in the trachea and pulmonary airways, and may flow from the nostrils and the cut surface of the lungs. The lungs are typically mottled red (with distended interlobular septae) noncollapsed and heavy. In more prolonged cases, there may be extensive interstitial and subpleural edema, and hyperemia may be less apparent. Fluid may be found in the thoracic cavity (hydrothorax) and abdominal cavity. Occasionally, there can be extensive fluid accumulation 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. In some cases, there may also be subcapsular hemorrhages in the spleen, congestion in the renal cortex, edematous infiltration around the aorta and trachea, and petechial hemorrhages on various serosal and pleural surfaces. Gastrointestinal lesions can include hyperemia and petechiae in the small and large intestines, and hyperemia of the gastric fundus. Cardiac lesions are not prominent, although petechiae may be found on the pericardium, and there may be increased pericardial fluid.

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. Ascites can also be seen. The lungs are usually normal or slightly edematous/engorged in this form of AHS, 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.

Dogs

The gross lesions reported in dogs have been consistent with pulmonary disease; the main lesions were hydrothorax and pulmonary congestion and edema. Red-tinged foam was noted in the airways of some animals. In some cases, the fluid in the lungs (clear and straw-colored) gelled on exposure to the air. Areas of emphysema and/or areas of hepatization were also reported in some lungs. Other lesions, such as hyperemia of the intestinal mucosa, petechiae and ecchymoses on the endocardium, and congestion of the liver and other internal organs have been noted in experimentally infected or naturally infected dogs.

Diagnostic Tests

African horse sickness is often diagnosed by virological methods. More than one test should be used to diagnose an outbreak (particularly the index case) whenever possible. AHSV can be isolated from the blood of live animals, or from tissue samples, especially spleen, lung and lymph nodes, collected at necropsy. Successful isolation from the blood is most likely if these samples are collected early during the febrile stage. AHSV can be recovered in various cell lines including baby hamster kidney (BHK-21), monkey stable (MS) or African green monkey kidney (Vero) cells, as well as *Culicoides* (e.g., KC cells) and mosquito insect cell lines, and in embryonated eggs, Intracerebral inoculation of newborn mice can also be performed. The isolate should be serotyped using virus neutralization or other methods, to allow the selection of an appropriate vaccine strain.

AHSV antigens can be detected in the blood and tissues (e.g., spleen) with enzyme-linked immunosorbent assays (ELISAs). Various reverse-transcription polymerase chain reaction (RT-PCR) assays are used to detect viral RNA. Some RT-PCR assays can also be used for rapid serotyping of field isolates.

Serology can also be used to diagnose African horse sickness, with antibodies usually detected within 8 to 14 days after infection. Paired serum samples are recommended, and are particularly important in areas where the disease is endemic. Available serologic tests include ELISAs, complement fixation, immunoblotting and virus neutralization. Complement fixation is now used infrequently in many areas, although is still employed in some endemic regions. The virus neutralization test is used for serotyping. Immunodiffusion and hemagglutination inhibition tests have also been described. Cross-reactivity

between AHSV serotypes is variable, and AHSV does not cross-react with other known orbiviruses.

Treatment

There is no specific treatment for African horse sickness, other than supportive care. Treatment may also be needed for secondary infections.

Control

Disease reporting

A quick response is vital for containing outbreaks in AHS-free regions. Veterinarians who encounter or suspect this disease should follow their national and/or local guidelines for disease reporting. In the U.S., state or federal veterinary authorities should be informed immediately.

Prevention

Live attenuated vaccines are routinely used to control African horse sickness in endemic regions. Monovalent or polyvalent vaccines may be employed, depending on the viruses circulating in the area. Reactivity to some vaccine strains is reported to be better than to others, and protection may be incomplete in some cases: clinical cases and mild or subclinical infections have been reported in some horses that had received as many as 5 vaccine doses in Africa. The currently available vaccines are teratogenic in pregnant mares, and vaccine strains may be transmitted by *Culicoides* vectors. No killed or subunit vaccines are currently manufactured commercially.

Stabling equids in insect-proof housing, especially from dusk to dawn (when *Culicoides* are most active), can also reduce the risk of infection. One study suggested that open stables might provide some degree of protection from *C. imicola,* but did not protect horses from *C. bolitinos*. Instead, concentrations of the latter vector seemed to increase inside open stables that contained horses. Vector control measures such as insect repellents and targeted applications of insecticides or larvicides might also be helpful.

When outbreaks occur in endemic areas, they have generally been controlled by vaccination and movement restrictions on equids. Some authors have recommended that surveillance systems be established to provide early warnings of outbreaks and detect the introduction of new serotypes in an area. Donkeys, which are not usually vaccinated for AHS, might be monitored by serology.

Most non-endemic countries test and quarantine imported equids to prevent them from introducing AHSV. The quarantine period may be extensive (e.g., 60 days in the U.S., if the horse is from an endemic country). If a virus is introduced into a non-endemic region, control measures may include the establishment of quarantine zones and movement controls, vaccination campaigns, and possibly the euthanasia of infected animals, depending on the situation. Stabling equids in insect-proof housing, at least overnight, can provide some protection to uninfected animals, as well as

reduce the risk that infected animals will transmit the virus to vectors. Vector control measures may reduce the number of *Culicoides*, although they are unlikely to be completely eliminated. Monitoring for fever can be helpful in detecting cases early. Each susceptible animal should have its temperature taken regularly (optimally, twice daily). The onset of cold weather can end epidemics, but the virus has sometimes re-emerged in the spring, at least in climates with mild winters (e.g., in Spain).

Morbidity and Mortality

Significant numbers of equids in Africa (e.g., 34% of equids and up to 50% of donkeys in Ethiopia) have been exposed to AHSV. Antibodies have also been documented in other animals, including 4-8% of dogs in some areas, although the significance of this finding is still unclear. Outbreaks of African horse sickness tend to occur in late summer and autumn, with cycles occurring at irregular intervals in some regions. Climatic conditions reported to favor epidemics are heavy rain alternating with hot, dry periods. Some countries have reported that there are fewer outbreaks since the number of free-ranging wild zebras decreased. In other regions, African horse sickness outbreaks seem to have increased recently, and have sometimes been caused by serotypes not usually found in that area.

Morbidity and mortality differ, depending on the viral strain, species of animal, previous immunity and form of the disease. In horses that develop clinical signs, the pulmonary form of African horse sickness is nearly always fatal, and the mortality rate in the cardiac form is usually 50% or higher. In the mixed form, mortality rate estimates vary from approximately 70% to greater than 80%, while horsesickness fever is not fatal. The mixed and pulmonary forms tend to predominate in naive horse populations, and the mortality rate is usually 50-95%. African horse sickness is generally less severe in other equids. The mortality rate is approximately 50% in mules during epidemics, and 5-10% in European and Asian donkeys. Deaths are rare among donkeys and zebra in endemic areas of Africa. Little is known about morbidity and mortality rates in dogs, but both subclinical and fatal infections have been reported. Once pulmonary signs appear, the prognosis in this species also appears to be poor. In one outbreak, 15 of 17 dogs died after being fed horsemeat from a sick horse. Intensive treatment was attempted in 2 of these animals, but it was unsuccessful.

Equids that recover from African horse sickness develop good immunity to the infecting serotype and partial immunity to other serotypes.

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.

Internet Resources

[The Merck Veterinary Manual](#)

[United States Animal Health Association. Foreign Animal Diseases](#)

[World Organization for Animal Health (WOAH)](#)

[WOAH Manual of Diagnostic Tests and Vaccines for Terrestrial Animals](#)

[WOAH Terrestrial Animal Health Code](#)

Acknowledgements

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