

AFRICAN HORSE SICKNESS

(*Perdesiekte, Pestis Equorum, La Peste Equina*)

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Definition [top](#)

African horsesickness (AHS) is a highly fatal, viscerotropic, insect-borne viral disease of horses and mules and generally a subclinical disease in other Equidae. The clinical signs and lesions result from selective increased vascular permeability and are characterized by an impairment of the respiratory and circulatory systems.

Etiology [top](#)

The etiological agent of AHS is a typical orbivirus measuring 68-70 nm in diameter, and the virion is composed of a double-layered protein shell.

The virus is present in the blood and certain organs such as spleen, lung, and lymph nodes in reasonably high concentration, whereas only traces are found in serum, tissue fluids, excretions, and secretions (10). Viremia generally lasts for about 4-8 days and roughly parallels the febrile reaction. In exceptional cases, viremia may last as long as 17 days in the horse and 28 days in zebra and donkeys.

The AHS virus is relatively heat stable, particularly in the presence of protein. It can be stored for at least 6 months at 4° C in saline containing 10 percent serum. Blood in OCG preservative (500 ml glycerin, 500 ml distilled water, 5 g sodium oxalate, and 5 g carbolic acid) can remain infective for more than 20 years; lyophilization may preserve infectivity for as long as 40 years. The virus is readily inactivated at pH values lower than 6.3, but it is relatively stable between values ranging from 6.5 to 8.5.

Nine distinct serotypes of AHS virus are known, the last of which was isolated in 1960. This suggests that, despite its segmented genome, the virus can be regarded as genetically stable and that new serotypes do not readily develop. The present nine serotypes probably evolved over many centuries.

Host Range [top](#)

Horses, mules, and donkeys have historically been known as hosts for AHS virus, as reflected in the name of the disease. In view of the high mortality rate suffered by horses and mules, these species should be regarded as accidental or indicator hosts. That AHS failed to establish itself outside the tropical regions of Africa tends to indicate that neither horses nor mules or donkeys remain long-term carriers of AHS virus and are therefore not essential for the permanent persistence of the infection in a particular region. Zebra may fulfill this role, but no irrevocable proof has been found to substantiate this view.

The dog has long been known to be susceptible to experimental infection (23). Infection of dogs also readily occurs following ingestion of infected horse meat (3). However, it is extremely unlikely that this species becomes infected by insect bites, and it is generally accepted that dogs play no role in the spread or maintenance of AHS (16).

Camels can ostensibly become inapparently infected with AHS virus, but few details are available as to the level and duration of viremia in this species and its

role, if any, in the epizootiology of the disease. A high percentage of African elephant serum samples reacted positively against AHS virus in complement fixation tests (7), but no neutralizing antibodies could be demonstrated in such samples. No evidence of virus replication could be found in elephants artificially infected with AHS virus (12). It can therefore be concluded that the African elephant is not susceptible to infection and that the putative serological evidence resulted from abnormal reactions of elephant sera in a complement fixation test.

Geographic Distribution [top](#)

African horse sickness appears to be endemic in tropical regions of central Africa from where it regularly spreads southwards to southern Africa. The Sahara Desert forms a formidable barrier against northward spread. Occasionally the infection does reach northern African countries, either by spread along the Nile valley or along the West Coast of Africa. The disease has also occurred outside Africa on a few occasions, the most notable of which was the major outbreak in the Near and Middle East from 1959 through 63 and in Spain (1966 and 1987-1990) (15).

In temperate regions such as South Africa, AHS has a definite seasonal occurrence. The first cases are usually noticed towards midsummer, and the disease disappears abruptly after the onset of cold weather in autumn. The disease is most prevalent in warm, low-lying moist areas such as valleys and marshes.

Transmission [top](#)

African horse sickness is a noncontagious disease, and the virus was the first shown to be transmitted by midges (*Culicoides* spp.) (9). The most significant vector seems to be *Culicoides imicola*, but other species, such as *C. variipennis*, which is common in many parts of the United States, should also be considered as potential vectors (4).

The virus is transmitted biologically by midges, and these insects are most active just after sunset and at about sunrise.

Although other insects such as mosquitoes have been implicated as biological vectors, and large biting flies (e.g., *Stomoxys*, *Tabanus*) may transmit AHS virus mechanically, the role of these insects in the epizootiology of the disease is regarded as absolutely minimal compared with that played by the *Culicoides* species.

Generally, midges disperse only a few kilometers from their breeding sites, but it

has been postulated that they can be borne for longer distances on air currents (21). Analysis of field observations on the progression of outbreaks indicates that wind-borne spread of midges may assist the short-distance spread of the disease but that long-distance jumps of the infection are invariably the result of movement of infected Equidae.

Incubation Period [top](#)

In experimentally induced cases the incubation period usually varies between 5 and 7 days, but it may be as short as 2 days and rarely as long as 14 days. Circumstantial evidence indicates that, following natural infection, the incubation period varies from 7 to 14 days.

Clinical Signs [top](#)

Four clinical forms of AHS can be distinguished (10).

The Peracute or Pulmonary Form [top](#)

This form is characterized by very marked and rapidly progressive respiratory involvement. An acute febrile reaction may be the only clinical sign for a day or two, reaching a maximum of about 104-106° F (40-41° C). This is followed by various degrees of respiratory distress. The breathing may increase to 60 or even 75 respirations per minute, and the animal tends to stand with its forelegs spread apart, its head extended, and the nostrils fully dilated. Expiration is frequently forced with the abdomen showing heave lines. Profuse sweating is common, and spasmodic coughing may be observed terminally with frothy, serofibrinous fluid exuding from the nostrils (Fig. 3). The onset of dyspnea is usually very sudden, and death often occurs within 30 minutes to a few hours after its appearance.

The Subacute Edematous or Cardiac Form [top](#)

The incubation period of this form varies between 7 and 14 days, and the onset of the clinical disease is marked by a febrile reaction of 102-106° F (39-41° C) that lasts for 3-6 days. Shortly before the decline of the fever, characteristic edematous swellings appear. These initially involve the supraorbital fossae and the eyelids (Figs. 4, 5, and 6) and later extend to the lips, cheeks, tongue, intermandibular space, and laryngeal region. Subcutaneous edema sometimes extends a variable distance down the neck towards the chest, often obliterating the jugular groove. Interestingly, no edema of the lower limbs is observed. Terminally, petechial hemorrhages develop in the conjunctivae and under the ventral surface of the tongue. The animal becomes very depressed and may lie

down frequently but for very short periods only. Occasionally, signs of colic may develop. Finally, the animal remains prostrate and dies from cardiac failure about 4-8 days after the onset of the febrile reaction. In cases that recover, swellings gradually subside within a period of 3-8 days.

The Acute or Mixed Form [top](#)

This form represents a mixture of the pulmonary and cardiac forms. Although seldom diagnosed clinically, it is seen at necropsy in the majority of fatal cases of AHS in horses and mules. The disease manifests itself in various ways. Initial pulmonary signs of a mild nature that do not progress are followed by edematous swellings and effusions, and death results from cardiac failure. In the majority of cases, however, the subclinical cardiac form is suddenly followed by marked dyspnea and other signs typical of the pulmonary form.

Horsesickness Fever [top](#)

This is the mildest form and is frequently overlooked in natural outbreaks. The febrile reaction is usually of the remittent type, with morning remissions and afternoon exacerbation, and lasts for 3-8 days but rarely exceeds 104° F (40° C). Apart from the febrile reaction, other clinical signs are rare and inconspicuous. The conjunctivae may be slightly congested, the pulse rate may be increased, and a certain degree of anorexia and depression may be present. This form of the disease is usually observed in donkeys and zebra or in immune horses infected with a heterologous serotype of AHS virus.

Gross Lesions [top](#)

The lesions observed at necropsy examination depend largely on the clinical form of disease manifested by the animal before death (10). In the peracute form the most characteristic changes are edema of the lungs or hydrothorax (Figs. 7 and 8). In very peracute cases, extensive alveolar edema and mottled hyperemia of the lungs are seen, whereas in cases with a somewhat more protracted course extensive interstitial and subpleural edema is also present, but hyperemia is less evident. Occasionally the lungs may appear reasonably normal, but the thoracic cavity may contain as much as 8 L of fluid. Other less commonly observed lesions are periaortic and peritracheal edematous infiltration, diffuse or patchy hyperemia of the glandular fundus of the stomach, hyperemia and petechial hemorrhages in the mucosa and serosa of the small and large intestines (Fig. 9 and 10), subcapsular hemorrhages in the spleen, and congestion of the renal cortex. Most of the lymph nodes are enlarged and edematous, especially those in the thoracic and abdominal cavities. Cardiac lesions are usually not conspicuous, but epicardial

and endocardial petechial hemorrhages are sometimes evident.

In the cardiac form the prominent lesion is a yellow gelatinous infiltration in the subcutaneous and intermuscular fascia primarily of the head, neck, and shoulders (Fig. 11). Occasionally the lesion may also involve the brisket, ventral abdomen and rump. Hydropericardium (Fig. 12) is a common feature, and there are extensive petechial and ecchymotic hemorrhages on the epicardium and endocardium, particularly of the left ventricle. The lungs are usually normal or only slightly engorged, and the thoracic cavity rarely contains excess fluid. The lesions in the gastrointestinal tract are generally similar to those found in the pulmonary form, except that submucosal edema of the cecum, large colon, and rectum tends to be far more pronounced.

In the mixed form the lesions seem to represent a combination of those found in the pulmonary and cardiac forms.

Morbidity and Mortality [top](#)

In susceptible horse populations, the fatalities range between 70 and 95 percent, and the prognosis is extremely poor. In mules, the mortality rate is about 50 percent and in the European and Asian donkey about 5-10 percent. No mortality is observed among African donkeys and zebra.

In enzootic regions, the mortality rate is modified in proportion to the immunity acquired by the equine population as a result of previous vaccination or exposure to natural infection.

Diagnosis [top](#)

Field Diagnosis

During the early febrile phase of AHS, a field diagnosis may be virtually impossible. However, a presumptive diagnosis should be possible once the characteristic clinical signs have developed and, more particularly, at necropsy.

Specimens for the Laboratory [top](#)

Confirmation of a presumptive diagnosis is based on virus isolation and identification. This is of particular importance whenever outbreaks occur outside the enzootic regions. The AHS virus can be isolated quite readily from blood collected during the early febrile phase (preferably in heparin or else in other

anticoagulants) as well as from spleen, lung, and lymph nodes collected at necropsy (10).

Specimens for virus isolation should be shipped to the laboratory refrigerated, NOT FROZEN.

Horses that survive infection develop specific antibodies within 10-14 days after infection that reach a peak about 10 days later. It is always advisable to use paired (acute and convalescent phase) serum samples. Serological tests can demonstrate AHS antibodies for 1 to 4 years after infection.

Differential Diagnosis [top](#)

The clinical signs of AHS, particularly when not fully developed, may be confused with other infections, notably equine encephalosis and equine viral arteritis (EVA). The former disease occurs under the same epizootiological conditions as AHS, and in South Africa the two diseases frequently occur simultaneously. Horses suffering from equine encephalosis usually do not have characteristic lung edema or subcutaneous edema, and the mortality rate is considerably lower than in AHS.

Severe cases of EVA may readily be confused with AHS. The presence of ventral edema (in EVA), particularly of the lower limbs, and the much lower mortality rate should allow differentiation. In countries where piroplasmiasis occurs, the early stage of this disease, before blood parasites can be demonstrated and anemia develops, may be confused with AHS.

The necropsy lesions of AHS can be confused with those found in cases of purpura hemorrhagica. In the latter condition, the hemorrhages and edema seem to be more severe and widely distributed than in AHS and usually involve the limbs and lower abdomen. The highly sporadic occurrence of purpura also aids in differentiation.

Vaccination [top](#)

The work of Alexander and de Toit (1,2) has resulted in the development of a live attenuated vaccine that has been used successfully for several decades. However, the adaptation of the virus to the brains of adult mice resulted in a neurotropic vaccine that occasionally caused encephalitis in horses mules and particularly in donkeys (20). This necessitated an alternate and safer method of attenuation achieved by plaque selection in Vero cell cultures (11). The vaccine currently used in South Africa consists of two quadrivalent vaccines that are administered 3 weeks apart. Strategic reserves of monovalent vaccines are also maintained.

Extensive work is presently under way to develop potent inactivated and recombinant vaccines that should widen the choice in the near future.

Control and Eradication [top](#)

Preventive Measures [top](#)

The most important means of introducing AHS into a hitherto disease free country is by the introduction of equid animals incubating the disease. Zebra and African donkeys that do not develop any clinical sign of disease are particularly dangerous. Equid animals imported from infected countries should be quarantined in insect-proof facilities at the point of entry. At present, there is a minimum 60-day quarantine period for horses brought into the United States from Asia, Africa, and the Mediterranean countries.

Once the disease is introduced into a country, several preventive measures should be taken to prevent further spread and eventually to eradicate the scourge in the shortest possible time. It is essential to isolate and identify the causal virus, but it is imperative that control measures be implemented even before the final diagnosis has been made.

Officials should delineate area of control, taking into consideration geographical borders such as mountains and rivers. The movement of all equid animal within, into, and out of the control zone should be stopped and this restriction rigidly enforced. Furthermore, all equid animals should be stabled, at least from dusk to dawn, and sprayed with insect repellents to reduce the risk of insects feeding on the animals. If sufficient stabling facilities are not available, barns could be used. Even if not insect-proof, such housing will reduce the risk of infection. Additionally, the rectal temperatures of all equid animals in the zone should be taken regularly (preferably twice daily) to detect infected animals as early as possible because overt disease is generally preceded by viremia for about 3 days. Animals with fever should be killed or housed in insect-free stables until the cause of the fever has been established.

Once the diagnosis has been finalized, vaccination of all susceptible animals with the relevant monovalent AHS vaccine should be considered. This decision will be guided largely by the success of measures already taken.

Natural Immunity [top](#)

Animals that recover from the disease develop a solid life-long immunity against

the infecting virus and a partial immunity against heterologous serotypes. Foals from immune dams have a passive immunity that may protect them for up to 6 months.

Public Health [top](#)

There is no evidence that man can become infected with field strains of AHS virus, either through contact with infected animals or from working in laboratories. However, it has been shown that certain neurotropic vaccine strains may cause encephalitis and retinitis in humans following transnasal infection (22).

GUIDE TO THE LITERATURE [top](#)

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