AFRICAN HORSE SICKNESS

AETIOLOGY

Classification of the causative agent

African horse sickness (AHS) is caused by a virus of the family *Reoviridae* of the genus *Orbivirus.* There are 9 antigenically distinct serotypes of AHS virus (AHSV) identified by virus neutralization, but some cross-reaction has been observed between 1 and 2, 3 and 7, 5 and 8, and 6 and 9. No cross-reactions with other known orbiviruses have been observed.

Resistance to physical and chemical action

Temperature: Relatively heat stable, especially in presence of protein. AHSV in citrated plasma still infective after heating at 55–75°C/131–167°F for 10 minutes. Minimal loss of titre when lyophilized or frozen at –70°C/–94°F with Parker Davis Medium. Infectivity is remarkably stable at 4°C/39°F, particularly in the presence of stabilizers such as serum and sodium oxalate, carbolic acid and glycerine: blood in OCG can remain infective >20 years. Can be stored >6 months at 4°C/39°F in saline with 10% serum. Fairly labile between –20°C /–4°F and –30°C/–22°F.

pH: Survives pH 6.0–12.0. Readily inactivated below pH 6.0. Optimal pH is 7.0 to 8.5.

Chemicals/Disinfectants: Inactivated by formalin (0.1%) for 48 hours, β□-propiolactone (0.4%), and binary ethyleneimine. Resistant to lipid solvents. Inactivated by acetic acid (2%), potassium peroxymonosulfate/sodium chloride – Virkon® S (1%), and sodium hypochlorite (3%).

Survival: Putrefaction does not destroy the virus: putrid blood may remain infective for >2 years, but virus is rapidly destroyed in meat by rigor mortis (lowering pH). Vaccine strains survive well in lyophilised state at 4°C/39°F.

EPIDEMIOLOGY

- Infectious disease is transmitted by *Culicoides spp*. that occurs regularly in most countries of sub-Saharan Africa
- At least two field vectors are involved: *Culicoides imicola* and *C. bolitinos*
- The disease has both a seasonal (late summer/autumn) and an epizootic cyclical incidence, with disease associated with drought followed by heavy rain
- Major epizootics in southern Africa are strongly linked with warm (El Niño) phase of the El Niño/Southern Oscillation (ENSO)
- Mortality rate in horses is 70-95%, mules around 50%, and donkeys around 10%.
  - other than mild fever, infection in zebra and African donkeys is subclinical
  - viraemia may be extended in zebra (up to 40 days)

Hosts

- Usual hosts are equids: horses, mules, donkeys and zebra
- Reservoir host are believed to be zebras
- Antibody is found in camels, African elephants, and black and white rhinoceroses, but their role in epidemiology is unlikely to be significant
- Dogs have peracute fatal infection after eating infected horsemeat, but are not a preferred host by *Culicoides* spp. and unlikely to play a role in transmission

Transmission

- Not contagious by contact
- Usual mode of transmission is the biological vector *Culicoides* spp. *C. imicola* and *C. bolitinos* are known to transmit AHSV in the field; *C. imicola* appears to be the principal vector

- The North American species *C. variipennis* is an efficient vector in the laboratory
- Occasional mode of transmission: mosquitoes – *Culex, Anopheles* and *Aedes* spp.; ticks – *Hyalomma, Rhipicephalus*; and possibly biting flies – *Stomoxys* and *Tabanus*
- Moist mild conditions and warm temperatures favour the presence of insect vectors
- Wind has been implicated in dispersal of infected *Culicoides* in some epidemics
- Movement of *Culicoides* spp. over long distances (700 km over water, 150 km over land) via wind has been postulated

Sources of virus

- Viscera and blood of infected horses
- Semen, urine and nearly all secretions during viraemia, but no studies have documented transmission
- Viraemia usually lasts 4–8 days in horses but may extend up to 21 days; in zebras viraemia may last up to 40 days
- Recovered animals do not remain carriers of the virus

Occurrence

AHS is endemic in the central tropical regions of Africa, from where it spreads regularly to Southern Africa and occasionally to Northern Africa. All serotypes of AHS occur in eastern and southern Africa. Only AHS serotype 9, 4 and 2 have been found in North and West Africa from where they occasionally spread into countries surrounding the Mediterranean.

A few outbreaks have occurred outside Africa in the Near and Middle East (1959–63), Spain (1966, 1987–90), Portugal (1989), Yemen (1997) and the Cape Verde Islands (1999). But recent northward expansion of the main African vector (Afro-Asiatic species *C. imicola*) and bluetongue virus into the Mediterranean Basin of Europe now threatens that region and beyond to AHS.

DIAGNOSIS

Incubation period is usually 7–14 days, but may be as short as 2 days. For the purposes of the OIE *Terrestrial Code*, the infective period for AHSV shall be 40 days for domestic horses.

Clinical diagnosis

- There are four principal manifestations of disease
- In the majority of cases, the subclinical cardiac form is suddenly followed by marked dyspnoea and other signs typical of the pulmonary form
- A nervous form may occur, though it is rare
- Morbidity and mortality vary with the species of animal, previous immunity and the form of the disease
    - Horses are particularly susceptible where mixed and pulmonary forms tend to predominate; mortality rate is usually 50% to 95%
    - Mules: mortality is about 50%; European and Asian donkeys: mortality is 5–10%; African donkeys and zebra: mortality is rare
- Animals that recover from AHS develop good immunity to the infecting serotype and partial immunity to other serotypes

Subclinical form (Horse sickness fever)

- Fever (40–40.5°C/104°F–105°F)
- Mild form; general malaise for 1–2 days
- Very rarely results in death

Subacute or cardiac form

- Fever (39–41°C/102–106°F)
- Swelling of the supraorbital fossa, eyelids, facial tissues, neck, thorax, brisket and shoulders
- Mortality usually 50% or higher; death usually within 1 week

Acute respiratory or pulmonary form

- Fever (40–41°C/104–106°F)
- Dyspnoea, spasmodic coughing, dilated nostrils with frothy fluid oozing out
- Redness of conjunctivae
- Nearly always fatal; death from anoxia within 1 week

Mixed form (cardiac and pulmonary)

- Occurs frequently
- Pulmonary signs of a mild nature that do not progress, oedematous swellings and effusions
- Mortality: about 70–80% or greater

Lesions

- Respiratory form:
  - interlobular oedema of the lungs
  - hydropericardium, pleural effusion
  - oedema of thoracic lymph nodes
  - petechial haemorrhages in pericardium
  - mucosa and serosa of small and large intestines may exhibit hyperaemia and petechial haemorrhages

- Cardiac form:
  - subcutaneous and intramuscular gelatinous oedema
  - epicardial and endocardial ecchymoses; myocarditis
  - hemorrhagic gastritis