CHAPTER 3.1

African horse sickness

Fast facts

African horse sickness (AHS) is an exotic arthropod-borne viral disease of *Equidae* transmitted by certain species of *Culicoides*.

Organism

AHS virus (AHSV):

- has nine different serotypes
- multiplies in regional lymph nodes and is followed by primary viraemia, with subsequent dissemination to target organs.

Susceptible species

AHS affects:

- all species of *Equidae* (although not all show clinical signs)
- dogs and camels (dead-end hosts).

Economic impact

There is potential for significant economic loss associated with high mortality in horses, as well as restrictions on animal movements and international trade.

Epidemiology

AHS:

- distribution is determined by the presence of competent vectors
- morbidity depends on species, previous immunity and the form of disease
- can cause mortalities, with horses particularly susceptible (up to 95 per cent).

Transmission

Transmission occurs primarily through *Culicoides* vectors.

Clinical signs

The disease may present as a peracute (pulmonary) form, subacute oedematous (cardiac) form, and acute mixed form or as horse sickness fever. Clinical signs may include:

- pyrexia
- laboured breathing and coughing
- pulmonary oedema
- oedema of the head, conjunctiva, neck, brisket, ventral thorax and abdomen
- death (which is common and can occur suddenly).

Post-mortem

Gross lesions vary depending on the form of the disease, and lesions are not pathognomonic. These may be found:

- subpleural and interlobular oedema
- subcutaneous and intermuscular connective tissue oedema.

Samples

Collect:

- blood samples (EDTA and plain tubes)
- lymph nodes (especially mesenteric and bronchial), lungs, spleen from dead horses.

Actions to take

If you suspect a case of AHS:

- call the Emergency Animal Disease Watch Hotline (1800 675 888) immediately or contact a government veterinarian in your state or territory
- isolate suspected cases and implement biocontainment protocols (including movement controls and protection of animals from vectors) until advised by government veterinary authorities.

Introduction

African horse sickness (AHS) is an arthropod-borne infectious (but non-contagious) viral disease affecting all species of *Equidae*.

Disease agent and susceptible species

African horse sickness virus (AHSV):

- is a double-stranded RNA virus belonging to the genus *Orbivirus* and family *Reoviridae*
- has nine different serotypes and some serotypes are cross protective
- mainly affects equids, with horses, mules, donkeys and zebras the primary hosts
- causes the most serious infections in horses (mortality up to 95 per cent)
- is also known to affect dogs, usually (but not exclusively) following ingestion of virus-infected meat. However, dogs are considered dead-end hosts
- is thought to be maintained in the environment in most regions of Africa by zebras which are often subclinically infected natural reservoir hosts
- antibodies have been reported in camels and African elephants but these species are not considered significant in the epidemiology.

Distribution

AHS:

- geographical distribution and seasonal occurrence are dependent on competent vectors, and studying the dynamics and behaviour of *Culicoides* spp. is therefore essential to understanding the disease
- appears to be seasonal in endemic areas, where it is preceded by seasons of heavy rain alternating with hot and dry climatic conditions
- occurs in eastern and southern Africa (all serotypes of AHSV)
- virus serotypes 2, 4 and 9 have been found in North and West Africa, from where they occasionally spread into countries surrounding the Mediterranean.

For the latest information on the distribution of AHS, refer to the WAHIS information database website of the World Organisation for Animal Health (OIE) [http://www.oie.int].

Occurrences in Australia

None reported.

Epidemiology

Modes of transmission

AHS is not contagious and does not spread by direct contact between horses.

Transmission occurs via:

- **vectors,** during blood-feeding by *Culicoides* species—*Culicoides imicola* is the principal vector in Africa and is present in Australia but other Australian *Culicoides* species could be involved. The virus has also been isolated from *Rhipicephalus sanguineus* (a dog tick) and *Hyalomma dromedarii* (a camel tick) and has been experimentally transmitted by mosquitoes, including *Aedes aegypti* and *Culex pyriens*, both of which are present in Australia

- **wind,** which was implicated in the dispersal of infected *Culicoides* in some epidemics
- **mechanical transmission** by other biting flies, but these are unlikely to play a significant role
- **parenteral** injection of infectious blood.

Disease dynamics

Following ingestion by a competent female *Culicoides* vector, the virus replicates in the insect gut then translocates and replicates in the salivary glands before infection of the next mammalian host. Following infection of a mammalian host:

- multiplication of AHSV occurs in regional lymph node and is followed by primary viraemia, with subsequent dissemination to endothelial cells of target organs
- the incubation period lasts 2–10 days, depending on viral load, viral virulence and host factors
- the viraemic phase typically lasts only 2–8 days (for horses) but reservoir mammalian host species (such as zebra) have a longer infectious period
- lifelong immunity following recovery is rare as different serotypes are not necessarily cross-protective
- animals that survive infection do not become carriers of the virus
- morbidity and mortality can be as low as 30 per cent and 10 per cent respectively in endemic areas
- high morbidity and high case mortality (of up to 100 per cent) may occur in naïve horses.

Persistence of the agent

AHSV:

- is inactivated in the laboratory with formalin, Virkon® S or radiation
- is destroyed at a pH less than 6, or pH 12 or greater. Acidic disinfectants such as acetic or citric acid have been recommended for decontamination when warranted. Alkaline disinfectants such as sodium hypochlorite are also recommended
- can survive in frozen meat, but is inactivated at temperatures greater than 60°C
- is rapidly destroyed in carcasses that have undergone rigor mortis due to pH fluctuations.

Diagnosis and pathology

Clinical signs

The disease may present as a peracute (pulmonary) form, subacute oedematous (cardiac) form, an acute mixed form, or as horse sickness fever.

Signs of the **peracute pulmonary form** ('Dunkop') include:

- acute pyrexia, followed within a day or two by the sudden onset of severe respiratory distress
- short incubation period, usually 3–4 days
- tachypnoea, forced expiration, profuse sweating, spasmodic coughing and a frothy serofibrinous nasal exudate
- forelegs spread apart stance, extended head and dilated nostrils

- dyspnoea, which usually progresses rapidly and causes death within a few hours after respiratory signs appear
- pyrexia which subsides gradually while the breathing remains laboured for several days (in recovering animals).

Signs of the **subacute oedematous cardiac form** ('Dikkop') include:

- a more protracted and milder incubation than the pulmonary form
- pyrexia lasting less than a week
- oedematous swellings appear in the supraorbital fossae and eyelids, spreading to involve the face, tongue, intermandibular space, laryngeal region, and sometimes the neck, shoulders and chest
- absence of oedema of the ventral side and lower legs
- severe depression, colic, petechiae or ecchymoses on the ventral surface of the tongue, and petechiae in the conjunctivae (all are usually seen in the terminal stages of the disease)
- death from cardiac failure (which often occurs within 1 week of the onset of pyrexic reaction).

The **acute mixed form** is most commonly seen in outbreak cases, where the disease may causemortality of up to 80 per cent. Signs include:

- a mix of clinical signs from the pulmonary and cardiac forms of the disease
- pyrexia
- mild pulmonary or subclinical cardiac disease followed by oedema, cardiac failure or respiratory failure
- death, usually occuring 3–6 days after the onset of pyrexic reaction.

A combination of pathologies (often not clinically apparent) are usually found at post-mortem.

Horse sickness fever is:

- the mildest form of the disease
- frequently not clinically diagnosed as mild pyrexia in animals may be subclinical
- usually observed in donkeys, reservoir species (zebras) and partially immune horses infected with heterologous serotypes of the virus.

Pathology

Gross lesions are quite variable and depend on the form of the disease. In the respiratory forms of the disease, this may be found:

- interlobular oedema of the lungs
- hydropericardium, pleural effusion
- oedema of the thoracic lymph nodes
- petechial haemorrhages of the pericardium.

In the cardiac forms of the disease, this may be found:

- subcutaneous and intramuscular gelatinous oedema
- epicardial and endocardial ecchymosis, myocarditis
- haemorrhagic gastritis.

Differential diagnosis

Consider in the differential diagnosis:

- exotic diseases
  - dourine
  - equine encephalosis
  - equine piroplasmosis
  - equine viral arteritis (avirulent strains of the virus circulate in Australia)
  - Nipah virus infection
  - surra
- endemic diseases
  - anthrax
  - equine infectious anaemia
  - Hendra virus infection
  - purpura haemorrhagica (a complication of strangles)
- non-infectious causes
  - acute poisoning (plant or chemical)
  - heat stress.

Samples required

Sample collection

Collect these samples from clinically affected animals:

- **serum**, preferably paired sera
- **EDTA blood** from live animals (at least 10 if possible), preferably at early pyrexic stages (7–10 ml per animal)
- **fresh tissue** from lymph nodes (especially the mesenteric and bronchial nodes), lungs, spleen.

Transport of samples

For transport:

- chill blood samples and unpreserved tissue samples either at 4°C, or with frozen gel packs
- DO NOT FREEZE SAMPLES at –20°C; it reduces the sensitivity when used for virus isolation and molecular diagnostic tests
- send samples with dry ice if the journey is expected to take several days.

Sample submission

The relevant state or territory laboratory should coordinate sample packaging and consignment for delivery to CSIRO-AAHL.

Diagnostic tests

For AHS diagnosis:

- serological tests available include complement fixation test (CFT), competitive blocking ELISA (which is serogroup specific based on VP7) and viral neutralisation
- nucleic acid detection includes real-time reverse transcription polymerase chain reaction (real-time RT-PCR)
- viral isolates can be detected in blood during the early pyrexic stage.

Reporting requirements

AHS is an OIE-listed disease and Australia has an international obligation to report cases. If you suspect AHS, report it immediately by phoning the **Emergency Animal Disease Watch Hotline on 1800 675 888**, wherever you are in Australia. Alternatively, contact a government veterinarian in your state or territory.

Biocontainment and personal protective equipment

There are no public health implications for AHS, but you should implement biocontainment protocols until advised by government veterinary authorities. This includes isolating suspected cases (and protecting from vectors, if possible) and using and appropriately disposing of personal protective equipment such as gloves, coveralls and rubber boots (or disposable boots). You need to thoroughly disinfect and decontaminate clothing, vehicle and equipment before leaving the property.

Further information

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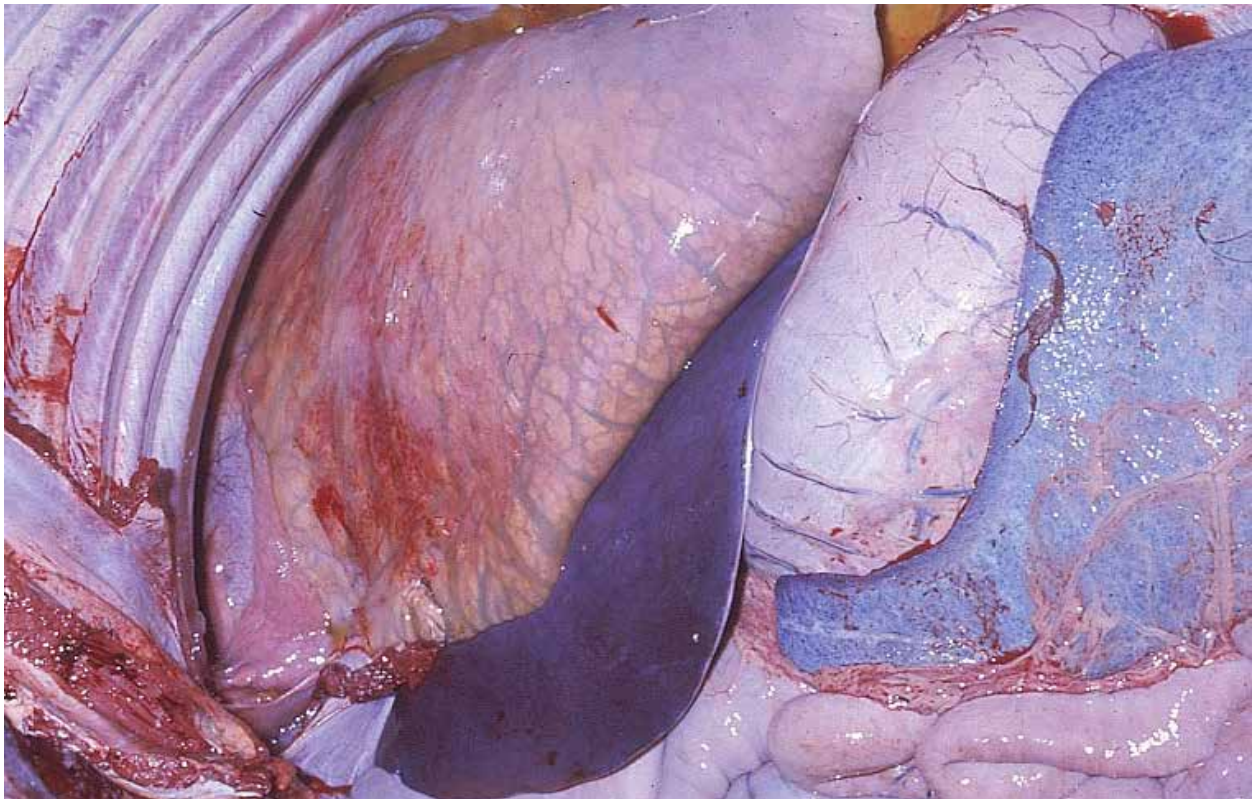
FIGURE 3.1.1 Abundant froth draining from the nostrils reflects severe pulmonary oedema

Image credit: PIADC and CFSPH

FIGURE 3.1.2 Severe interlobular oedema of the lungs with petechiae on the pulmonary pleura and the splenic capsule



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