African Horse Sickness

African horse sickness (AHS) is an insect-borne infectious disease of equids. The disease is caused by an orbivirus that is transmitted by *Culicoides* midges. At the current time AHS is not present in Europe and has not been reported in the UK. However, with climate changes, vector distribution alterations and the appearance of Blue Tongue virus in the UK last year there is a possibility that a case of AHS may occur in the UK. Blue Tongue is considered to be a reasonable indicator of the likelihood of the appearance of AHS in a country as it is a very closely related virus and is transmitted by similar *Culicoides* midges.

AHS is taken extremely importantly by veterinary surgeons and others associated with the horse industry as it an extremely aggressive disease in the horse with a mortality rate of up to 90%. Given this aggression and the possibility that AHS could become spread by midges within any area that it enters, swift diagnosis of early cases is vital in the control of the disease. In the field, the diagnosis of AHS relies initially on recognising the clinical signs.

The incubation period for AHS can range from 2–14 days. Clinical signs typically appear 5–7 days after infection. They are characterised by damage to the respiratory and circulatory system as a result of increased vascular permeability. The clinical signs of AHS range from the nonspecific to the classical forms of the disease. The clinical signs are centred on the respiratory and circulatory systems. Nonspecific clinical signs include pyrexia, inappetance, and, in some cases, sudden death. Classic clinical signs of AHS take a number of well recognised forms.

Clinical Forms of AHS that May Occur in the UK

Pulmonary Form ('Dunkop')

Clinical signs of the pulmonary form include pyrexia, severe dyspnoea/tachypnoea, 'air hunger' (extended head, open mouth, flared nostrils), paroxysmal coughing, sweating, recumbency and death. In some cases marked nasal exudate/froth is noted particularly at *post mortem*.

Figure 1: *Foam from nostrils classically seen in the pulmonary form of AHS.*

Cardiac Form ('Dikkop')

Clinical signs of the cardiac form include pyrexia, subcutaneous oedematous swelling of head, particularly the supraorbital fossae, lips, cheeks, tongue, intermandibular space and eyelids, dyspnoe, cyanosis and death. In some cases the eyelids are so swollen the horse cannot open its eyes and in severely affected cases there may be petichial or even frank haemorrhage from the conjunctiva and mouth. In some cases, colic and/or dysphagia may also occur.

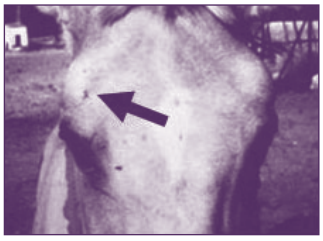

Figure 2: *Swelling of the supraorbital region classically seen in the cardiac form of AHS.*

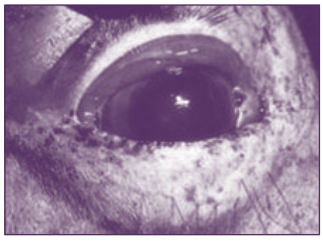

Figure 3: *Conjunctival haemorrhage classically seen in the cardiac form of AHS.*

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Mixed Form

Clinical signs of the mixed form usually include respiratory distress and subcutaneous oedema as described above.

Differential Diagnosis

In the UK the major differential diagnoses are anthrax (in sudden death cases), *purpura haemorrhagica*, EVA and EIA.

Diagnosis

The diagnosis of AHS is made on a combination of clinical signs, *post mortem* findings, virus isolation, serological identification. Blood samples from affected cases should be taken into heparin and stored at 4°C. AHS is a **notifiable disease** and should be notified to **Defra** immediately. It is not, however, a contagious disease (no horse to horse spread).

Control Measures

The main control measures for AHS in the world include arthropod vector control, vaccination of susceptible animals in countries where this is permitted, (preferably with the monovalent vaccine for the outbreak serotype) and euthanasia or isolation (in vector proof facilities) of equids showing clinical signs. In the UK, at the current time African Horse sickness is included in The Specified Diseases (Notification and Slaughter) Order 1992 in order to enable the implementation of the slaughter requirements of EU Council Directive 92/35/EEC, which lays down control rules and measures to combat African horse sickness.

Amended with thanks from data provided by Professor A. Guthrie.
Figures courtesy of Professor A. Guthrie.

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