African horse sickness

African horse sickness (AHS) is a highly fatal, insect-borne viral disease of equidae. A mortality rate of 90% can be expected when the disease occurs in an area for the first time.

Geographical distribution

During the 1980s, AHS was reported from most African countries south of the Sahara. In Europe the disease was introduced into Spain (1987) and extended to Portugal and Morocco in 1989.

Cause

Family Reoviridae; genus *Orbivirus*. There are nine different virus types. The AHS arbovirus is related to bluetongue subgroup of reoviruses.

The virus is relatively stable between pH 6 and 10.4, and is inactivated at temperatures above 60°C.

Host species

Under natural conditions only equines and dogs are affected by AHS. Horses are the most susceptible of the equidae.

Transmission

AHS is transmitted by insects - usually *Culicoides* species, as well as *Stomoxys calcitrans*, *Phlebotomus* and *Simulium*, and possibly other insect species. The insects can transmit the infection while sucking blood up to five weeks after the intake of virus. The virus appears able to persist in the absence of equine species. Zebras and elephants are believed to act as a reservoir for the virus. Dogs are rarely infected from eating infected horse meat.

Lifelong immunity follows recovery from AHS, and antibodies in colostrum protect foals for 3 - 5 months.

Clinical signs

Four different clinical forms of the disease are seen in susceptible equine populations.

Severe pulmonary disease is the most severe form, leading to death within 3 - 5 days. The incubation period is usually 3 - 5 days. A sudden fever is followed by severe dyspnoea due to massive pulmonary oedema. Frothy fluid is discharged from the nose. This is the form which normally affects dogs.

The subacute or cardiac form is caused by a less virulent virus strain. It has a longer incubation period and course. It characteristically causes oedematous swelling of the head, neck, eyelids, lips, cheeks, tongue, thorax and ventral abdomen. Bulging of the supraorbital fossa is a feature, and the tongue may prolapse. The temperature increases up to 41°C for 3-4 days and there is increasing dyspnoea. Circulatory failure usually leads to death after 10-12 days.

The abortive form is characterised by fever, reddened conjunctivae, accelerated pulse and dyspnoea. There may be a fever for 1-2 days, and a short period of general malaise. After a relatively short course, the animals recover spontaneously. This form is often observed in donkeys and in partially immune horses in endemic areas.

In the mixed form either pulmonary or cardiac symptoms can dominate.

Diagnosis

Seen at autopsy the pulmonary form is characterised by extensive oedema of the lungs. The pleural surfaces are unevenly raised owing to infiltration of clear yellow fluid in subpleural and interlobular tissues. The thoracic cavity contains a large volume of clear yellow fluid. The liver is frequently congested and kidneys are hyperaemic.

In the cardiac form the striking postmortem lesion is gelatinous infiltration of the subcutaneous and intermuscular tissue, commonly around the head, throat, abdomen and forelegs. Hydropericardium and extensive haemorrhages in the epi- and myocardium.

The diagnosis is confirmed by the isolation of virus or by serology. Heparinized blood samples are collected from febrile animals and serum samples from convalescent animals; spleen from dead animals.

Fig 1: Bulging of the supraorbital fossa is a feature of the cardiac form of African horse sickness.

Virus demonstration involves horse or mouse transmission tests and rapid demonstration of virus by immunodiffusion or immunofluorescence. Group specific antibodies are detected by complement fixation or gel diffusion tests and type specific antibodies by serum neutralization tests.

Risk of introduction

The risk of AHS being introduced to New Zealand is considered to be low.

To maintain itself AHS requires the presence of suitable insect vectors. The major vectors, *Culicoides* species, are not found in New Zealand. However, other biting insects may play a role as vectors and so should the virus be introduced to this country, an epizootic of AHS, cannot be ruled out. It is likely, however, that in most parts of New Zealand the population density of biting insects would be insufficient to sustain a major epizootic.

The disease is unlikely to establish in

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New Zealand, as the maintenance of AHS appears to depend on a population of suitable reservoir hosts (probably zebras and elephants). In the absence of such a reservoir population the disease is likely to die out, as it has done following extensive epizootics in North Africa, the Middle East and the Indian subcontinent.

Effects of introduction

In a fully susceptible horse population, the effects of AHS can be devastating. Mortality of up to 95% can be expected.

The cost to the New Zealand horse industries of an outbreak of AHS would be expected to exceed substantially the cost of an outbreak of equine influenza which has been estimated at nearly $175 million. This cost would be incurred by the cancellation of all horse gatherings for a period of time, and makes no allowance for mortality.

The presence of AHS in New Zealand would cause major disruption to the export of horses.

Meat inspection certificates for meat exports to a number of countries certify that New Zealand is free of AHS.

Prevention

No horses are imported from countries in which AHS occurs.

Control

Should the disease occur in New Zealand, the Chief Veterinary Officer would design ad hoc strategies based on the data gathered at the time of finding the index case(s), and the advice given by a team of experts.

Intensified surveillance of insects and the use of insecticides would be considered.

Polyvalent attenuated tissue culture vaccines are used successfully and without postvaccinal complications in enzootically infected areas. Immunity takes 5 months to develop fully after vaccination, but lasts for at least 6 years, possibly for life. Available vaccines do not protect fully against heterologous strains of AHS.

Culicoides are nocturnal sylvatic feeders and seldom enter buildings. Horses at risk in endemic areas should be stabled overnight.

Further reading – *refer page 27*

4, 5, 8, 10, 18, 19, 21

Further reading

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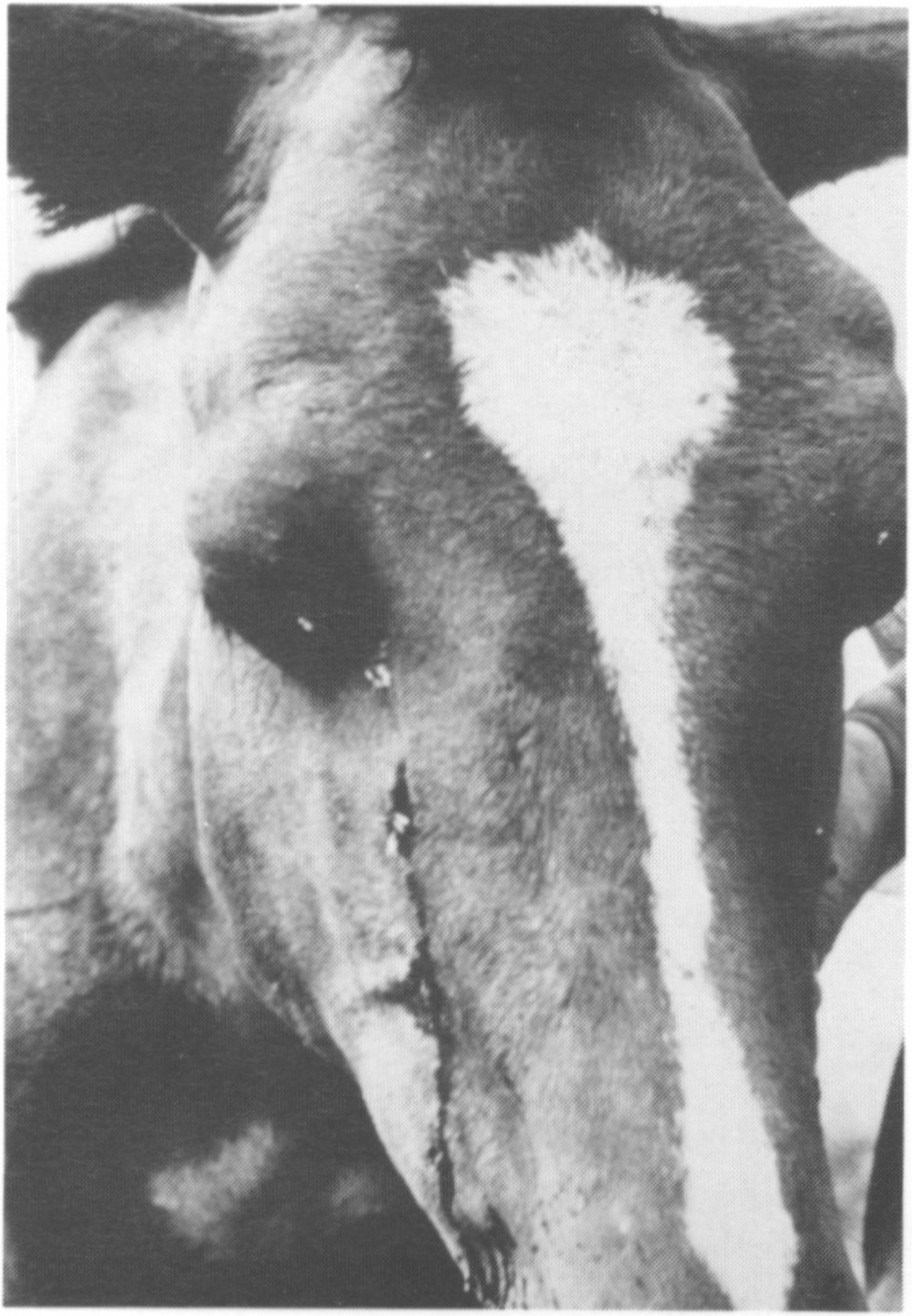

Fig 1: Bulging of the supraorbital fossa is a feature of the cardiac form of African horse sickness.