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## African Horse Sickness - ECO-VET

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### The Eco-Vet Way

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**Cause:** Infection by one of nine strains of virus from the genus Orbivirus.

**Cinical:** A number of factors may be found ranging from:

Severe depression

Fever

Oral haemorrhages

Rapid respiration

Nasal discharge of initially clear mucous

Marked swelling over the eyes

Slight impacted colic

**Diff Diagnosis:** There are a number of other viruses that may mimic AHS of which viral encephalosis is the most important.

**Diagnosis:** Is dependent on serum or tissues being tested in the laboratory.

**Idea:** Assume anything that looks like AHS is the disease until proved otherwise. I am not interested in hopefully saying it is just a virus. Assume the worst and nail it. Suspicion of AHS is a veterinary emergency, just as colic is.

**Prevention:** The OP vaccine has saved the lives of millions of horses. It can work very well but there are problems:

Young horses often require three courses of inoculation before being protected.

Well vaccinated horses over the age of thirteen, may be better off by vaccinating only every second year.

Vaccines must be maintained cold, check on them, have they been kept properly. Ask your supplier, ask your vet. Demand the best.

**Season:** Mainly from Dec to April, but react if disease is present in your area.

**Transmission:** The virus is injected directly into a horse when bitten by a fly of the Culicoides sp. thus transmitting the virus from one horse to another.

**Fly Control:** Is very difficult, but massive use of fly repellents must be carried out during the danger times

**Treatment:** It is a viral disease:

Antibiotics do not help and may be toxic

Anti-inflammatories reduce fever at a time when the fever is attempting to kill the virus

Vitamins, particularly VitC at six gms daily helps to boost immune system

Do not move them

TLC is enormously important, keep them warm

Work very quietly, they often sleep a lot

If sleeping a lot, keep turning every four hours quietly

Calcium intravenously may help

Do not euthanase as even the most desperate cases can get better.

**ECO-VET** My best success is with this regime that has saved at least **80%** of cases( serologically confirmed):

**ECO-HEAL** and **ECO-HEART** and **LUNGS-ECO** All of these at five mls given orally every hour for four doses, then at two hourly intervals for a further four doses, then four times daily.

**EPILEP-ECO** may be added where CNS involvement suspected.

**Future:** The development of new vaccine is very promising.

The development of a natural, non-toxic vaccine is also encouraging.

**Now for the longer version.**

## AFRICAN HORSE SICKNESS (Perdesiekte) IN DEPTH STUDY

### Introduction

African Horse Sickness (AHS) is an infectious disease of equines caused by one of the nine serotypes of **ORBIVIRUS**. Midges of the **CULICOIDES** species transmit the disease.

The disease occurs frequently in most countries of sub-Saharan Africa, and has been recorded since the

14th century. Records of the Dutch East India Company often refer to the disease as a significant problem, including a reference to an outbreak in **1719**, in Cape Town where some sixteen hundred horses were lost. Explorers such as Livingston also sustained severe losses. The **Voortrekkers** experienced many difficulties with **Perdesiekte**. A major reason for the placement of the Veterinary Institute at Onderstepoort was **Theiler's** interest in AHS; this being prevalent in the area. Major outbreaks historically seem to have occurred at intervals of 20-30 years.

The most severe was the **1855** outbreak in the **Cape** when some seventy thousand horses, making up nearly forty per cent of the total population, succumbed to infection. Remember that we are talking about massive losses occurring at a time when horses were of enormous importance as working and transport animals. It is not difficult to imagine the great disruption and distress.

It was as early as 1903 that evidence confirming the requirement for an insect vector was established, although it was as late as **1944** that **Du Toit** reported that the midge **Culicoides imicola** was implicated as the vector of AHS and a similar disease called Bluetongue in sheep.

### **Aetiology**

AHS viruses are classified in the genus Orbivirus and family Reoviruses. It has many relationships with other orbiviruses, including those that cause Bluetongue in sheep and the **equine encephalosis** viruses. There are nine serotypes of the virus numbered one to nine, It is important to note that there are certain affinities between some strains, such as 1&2, 3&7, 5&8 and 6&9. This does however indicate that for a vaccine to be effective all nine serotypes have to be included.

The virus is relatively heat stable and is capable of surviving in putrid blood for more than two years. Any infected substances, from mucous and snot to post mortem contaminants must be adequately cleaned and disinfected.

### **Epidemiology**

AHS is **endemic** in Central and Eastern Africa where it is available on an annual basis to spread mainly to Southern Africa. From time to time it may spread to North Africa, the Middle East and Spain.

**Serotype 9** caused havoc in Iran during 1959, from where it spread to the Persian Gulf as well as into India and Pakistan, Turkey and Afghanistan during **1960-61**. Estimates indicated that a total of some **300,000** horses were lost during this outbreak. Spanish outbreaks between 1987-90 were thought to have been carried by a group of **Burchells zebras** imported from Namibia.

Apart from the NE Transvaal the disease is not endemic in South Africa. The disease appears first there during Dec and Jan and gradually works its way Southwards, depending on climatic conditions being favourable for the breeding of the Culicoides midges. This is offered as the explanation for the regular non-appearance of the virus in many years in areas such as Gauteng, Free State and the Cape. Good early summer rains often indicate that virus will be a problem during that season.

In the summer rainfall areas of Southern Africa, AHS enjoys warm coastal regions and moist low-lying inland areas with valleys and marshes. In these areas the disease begins to flare up during February with most deaths during March and April. The disease normally disappears after the first frosts, although cases during May and June will still be found in the Lowveld.

With the absence of insect vectors during most normal winters the disease disappears completely until the following rainy season when the cycle once again is repeated from the North.

One serotype of the virus usually dominates during an outbreak. **Serotypes 1-8** may be associated with more than ninety per cent mortality, while serotype **9** is slightly less dangerous with mortality of about seventy five per cent.

Horses are the most susceptible to AHS while mules have a reduced mortality and donkeys and zebras are most resistant. It is suggested that certain breeds of horses in the North and West of Africa may have developed acquired natural resistance by virtue of the fact that they have been in Africa for more than two thousand years.

**Foals** born from immune mares gain antibody protection after they have drunk colostrum. This protects them until between the four and six month of age, after which they are fully susceptible. Foals born from susceptible mares have no protection.

Many people do not realise that **dogs** are highly susceptible to the disease both from natural infection caused by insect transmission but also by eating the meat of horses that died from the disease. Luckily Culicoides does not usually feed on dogs.

Antibodies to AHS have also been found in elephants but it is unlikely that these animals act as reservoir hosts.

AHS is infectious, but it is **not contagious**. The difference here is that there is a requirement for the insect to spread the virus by inoculating it directly into another horse, rather than by direct spread, as occurs in equine flu by directly coughing the virus into the air stream of another susceptible individual. Horses can of course spread the virus by travelling to other areas when they are incubating the disease. This was clearly demonstrated in the highly publicised outbreaks in the Cape during 2000.

#### **Culicoides species.**

There are now known to be many species of the fly that are vectors for AHS although many of them may be of local significance only. It is during the period between sunset and sunrise that the midges are most active. They will travel several km from their breeding sites, although factors such as winds may carry them to adjacent areas even over some considerable distances. In 1996 a single fly trap was recorded as catching in excess of **one million** midges in one night! We can thus begin to understand the risk factors that may prevail when climatic factors are favourable. The established ideas that stabled horses will not contact the disease as the midge will not go inside have been disproved. There are times when the environment is so full of hungry female flies needing a blood meal, that they will find a horse somewhere, anywhere! The risk years have been clearly associated with good early summer rains allowing excellent breeding conditions for the midges and a steady drift of infected flies from the North.

We are faced with the prospect that in exceptionally wet years the presence of overwhelming virus challenge may allow disease to develop in horses that have even normally considered levels of vaccination immunity.

When infection is expected to be high, foals may have to be vaccinated from three months of age.

The first four complete vaccination courses may have to be completed before two years of age.

Over vaccination may actually lead to lowered immunity.

Remember that transmission of the virus may be effected by injecting horses using the same syringe or needle.

Horses that recover from AHS do not become carriers of the virus.

#### **Pathogenesis**

There are a number of factors that decide the outcome in the horse that is bitten by a midge infected by AHS virus including the virulence of the individual virus serotype and the immune status of the horse. After the virus is inoculated into the body it is carried to the regional lymph nodes where it finds conditions favourable to its multiplication. Virus is released into the blood whereby it finds itself infecting the target organs, namely the lungs and the other lymphoid tissues of the body. This **viraemia** is associated with the red blood cells and lasts for about four to eight days. By the third day after inoculation the virus may be found in organs such as the spleen, lungs and pharynx as well as most lymph nodes. The **heart is not** a primary site for virus replication.

Theiler's original description of the four different types of disease are still valid:

1. The horse sickness **fever** form.
2. The peracute, pulmonary or **dunkop** form. The thin head!
3. The subacute oedematous, cardiac or **dikkop** form. The swollen head!
4. The **mixed** form.

#### **Biliary.**

Fully susceptible horses usually develop dunkop, while those with some immune awareness develop the dikkop form.

It is likely however that most cases are actually some variant of the mixed form. It is interesting that there is some evidence that horses who have **exercised hard** during the incubation of the disease will be more likely to develop dunkop.

Isolation of more than one serotype from an infected horse has never been recorded.

#### **CLINICAL**

The incubation period is from 2 – 10 days although more commonly between five and seven days.

**Dunkop.** This form is seen in fully susceptible horses, particularly foals. Also seen in dogs.

The body fever may be 41°C or higher for up to 36 even 48 hours,

The breathing is severely compromised, mouth may be open with the tongue extended, and the neck and head are stretched out.

Coughing may be paroxysmal,

Large quantities of frothy coloured fluid from nostrils, sometimes only after death.

May even be exercising when dramatically becomes ill with dyspnoea and death following quickly.

Appetite may remain reasonable although they may take feed into mouth and hold this without chewing it. The prognosis is that **less than 5%** will recover.

**Dikkop.** In this form the disease may develop more slowly:

Swelling of the supraorbital fossa may be the first sign as a large bulge above the eyes,

Swelling of head and or neck may develop,

Fever often is not the first sign that is seen,

Fever may last up to six days before reducing,

Oedema of the head and neck usually appears late as disease develops, but the earlier that it does develop worsens the prognosis,

Petechiation of the mucosa of the eyes and mouth are not good signs,

Colic may be present and affected animals may be restless and paw the ground,

Mortality rate is about fifty per cent with death occurring about four days after the start of the fever,

Swallowing may be difficult and water and food particles may drip out of the nose.

**Mixed form.** This is the most commonly diagnosed form at post mortem although we rarely diagnose this clinically. The temptation for the clinician is to try to be more specific.

All of the signs noted above are possible,

The Dikkop form may be evident then a rapid change to Dunkop takes place,

Mortality rate is about seventy per cent.

**Horsesickness fever.** This is an interesting case where horses that are immune to particular serotypes of the virus become affected by one of the other serotypes, against which they may have some cross protection immunity.

A mild form of disease,

Fever of 39-40°C lasting between one and five days,

Fever of unknown origin during an AHS outbreak should always be handled with suspicion,

Transient non-specific signs, such as loss of appetite, increased respiratory rate, increased heart rate and mucosal congestion may be seen.

**Biliary.** We must always be aware that many horses harbour dormant or sub clinical affection of biliary fever.

There is a well-recognised association between AHS and an acute flare up of biliary fever.

Particular care must be taken to establish, or rule out, the presence of biliary parasites during cases suspicious of AHS.

It is likely that many mild cases of AHS have eventually succumbed to an overwhelming and acute flare up of biliary as the immune system becomes suppressed by the virus.

All fever cases in horses must have biliary considered as a part of the disease process.

**Pathology.**

**Dunkop.**

Severe oedema of the lungs is considered to be the most significant finding,

Hydrothorax with several litres of slightly yellow, congealing fluid may be found in the thorax,

The lungs are very heavy, fluid oozes out of them when they are cut,

Looks like the horse has drowned,

Froth and fluid all through the trachea and bronchi,

May be haemorrhages in heart,

Lymph nodes show some enlargement,

Spleen is normal or slightly enlarged,

The glandular stomach is markedly congested,

**Dikkop.**

Oedema of the subcutaneous tissues of the head and neck, including between the muscles. This is yellow, thick and gelatinous,

In some cases will include tissues of the back, chest and shoulders,

The tongue is often hugely swollen, cyanotic and has petechial haemorrhages,

Lungs show only slight congestion or fluid,

Mucous membranes of caecum, colon and rectum may be marked.

**Diagnosis.** This is not always as simple as it seems, but:

1. The epidemiology is important. The right time of the year, the right area, other cases in the area,
2. Clinical signs such as swellings, fever and respiratory distress are suspicious,

3. Marked post mortem signs are indicative,
  4. Blood in heparin for virus examination during the fever stages is useful,
  5. Post mortem samples from spleen, lung or lymph nodes maintained at 4C are important,
  6. Virus may be identified by complement fixation, and other specialised immune typing techniques,
  7. Serotyping is carried out via virus neutralising testing,
  8. Recovered horses will have high complement fixation antibody titres,
  9. Many new and more rapid testing methods will be available very soon,
- Many owners and veterinarians become irritated by the time required to confirm a diagnosis of AHS, but it is time consuming and difficult to speed up.

**Confusing Diseases.** It is not possible to clinically differentiate the horsesickness fever form of the disease from any other disease that causes fevers in horses.

Equine encephalosis has many epidemiological factors in common with AHS: fever, oedema, supraorbital fossa swellings.

**Treatment.** There is no specific treatment but some guidelines:

TLC is very important. Horses respond well to lots of attention. No movement. Transport improves chances of death. Absolute minimum of stress, including injections. Recovered horses must not work for six weeks. Control biliary parasites although care must be observed with toxic drugs. Antibiotics are not indicated and may be toxic. There is growing evidence that homoeopathic medicines provide most success.

**Vaccination.**

Onderstepoort produces in excess of 150,000 doses of AHS vaccine per year.

The horse population of South Africa is probably in the region of 300,000 animals.

Since vaccination became widespread the numbers of clinical cases have dropped very significantly.

There are two different vaccines, one against serotypes 1,3,4 & 5; the second against serotypes 2,6,7 & 8.

These are normally given three weeks apart beginning from July, well before the expected AHS season.

Serotype 9 is not included as this is very rare in South Africa and does not get good cross protection from number six.

Vaccination reactions such as transient depression and fever are rare but do occur and on occasion, even blindness, encephalitis and other neurological disorders may be associated with fatal consequences. There have been four cases in laboratory workers handling the vaccine where loss of vision was a factor. It is important to follow the recommended vaccine schedule closely.

There has to be a balance between not working horses hard during the second week following vaccination and allowing them to go dilly in the paddock. The owner must exercise some common sense in this regard.

Many racehorse trainers do not vaccinate as they are convinced that side effects, relating to work intolerance are much higher than believed. There is no doubt in my own mind that exercising young horses during vaccination is dangerous.

It is likely that many horses require four different courses of vaccine before developing good immunity.

This does explain why many of the actual clinical cases seen involve 2 and 3 year olds.

**Foals.** In most cases colostrum provides a very effective means for immunising foals. The foal gains protection that is directly related to the degree of protection that the mare herself enjoys. In most cases this protection will last the foal only for 2-4 months, rarely six months. In view of the thoroughbred breeding season this means that most foals will require vaccination in Dec or Jan before the peak season for disease occurs.

Foals and yearlings should be vaccinated twice during their first and second years of life.

**Control.** It is a significant factor that in most normal seasons, stabling of horses from four pm until nine am seems to offer some reduction in Culicoides activity. Extensive applications of insecticides also have to be considered during seasonal fly activity.

**Pro-active.** Owners have the right to be a nuisance by advising, even insisting, that their neighbours also follow all control suggestions. There is nothing more irritating when following anti-fly control to the letter, only to observe that your neighbour with twenty horses does not even vaccinate.

**Future.**

Plans for a new, more efficient, dead vaccine with less toxicity are encouraging.

The use of DNA probes may simplify diagnosis in the near future.