

Keeping horses indoors during the tie when the midges are active is an effective additional prevention measure

African Horsesickness

Due to the unusually warm weather over large parts of the country this past winter, many horse owners fear that African Horse Sickness may be particularly severe this summer. With this in mind, Dr Rentia de Wet reviews this deadly disease and gives advice on how to best protect your horses against it.

African Horse Sickness (AHS) is a frequently fatal infectious disease of horses caused by an orbivirus of which there are nine serotypes. It is borne and transmitted by *Culicoides* midges and is associated with clinical signs and lesions compatible with impaired respiratory and circulatory functions. AHS is not contagious. The disease spreads either by movement of infected hosts (horses) or of the vector (midges).

As the disease is transmitted by *Culicoides* midges, it has a seasonal occurrence and its prevalence is therefore influenced by climatic and other conditions, which favour the breeding of midges. AHS is generally most prevalent in warm coastal regions or low-lying, moist inland areas during the second half of summer, some

time after the rainy season has commenced. In South Africa the first cases usually occur at the beginning of February, but the most serious outbreaks commonly occur in March and April. When the first frosts occur at the end of April or May the disease usually abruptly disappears.

AHS primarily affects equine animals. Horses are most susceptible (mortality rate 70-95%), while mules are less susceptible (mortality rate 50-70%). Donkeys and zebra are very resistant with most infections being subclinical. Foals born to immune dams are resistant as a result of passive immunity obtained through the colostrum. This immunity declines until it is completely lost after about five or six months. Foals born to fully susceptible mares are just as susceptible as unexposed adult horses.

Dogs are the only other species that may become ill and die following infection with AHS virus (after ingestion of infected horse-meat.)

The period between sunset and sunrise, when *Culicoides* midges are the most active, is when most animals acquire infection in open veld. Generally, these midges disperse only a few kilometres from their breeding sites.

Clinical signs

The incubation period usually varies between five and seven days, but it may be as short as 48 hours or as long as 21 days. Four forms of AHS are distinguished, namely:

Fever form

This form usually occurs in partially immune horses or in more resistant species such as donkeys and zebras. It is a very mild form of the disease and is frequently not clinically diagnosed. The most characteristic finding is a rising in body temperature lasting one to six days followed by a return of the temperature to normal one to two days later, followed by recovery. Some horses may show slight clinical signs of illness, such as partial loss of appetite, reddened eyelid membranes, slightly laboured breathing and increased heart rate.

Dunkop or lung form

Fully susceptible horses (foals that have lost their colostral immunity or horses that have never been exposed to the AHS virus) usually develop this form. It is also the usual form in dogs, the most characteristic sign being laboured breathing. A febrile reaction may be the only sign for a day or two, reaching a maximum of 41°C or higher in the course of two or three days, followed by death. The characteristic signs in this form of AHS are extremely difficult breathing, paroxysms of coughing and discharge of large amounts of frothy fluid from the nostrils. In some cases the discharge from the nose is seen only after death.

The onset of laboured breathing is generally very sudden, and death often occurs within a few hours of its appearance. Terminally the nostrils are dilated, the mouth is open, the tongue protrudes, the head and neck are extended and the animal sweats profusely. On post mortem inspection the impression is gained that the animal drowned as a result of the excessive amount of fluid in the lungs. Several litres of pale yellowish fluid are found in the chest cavity.

The prognosis of horses affected by the dunkop form is usually unfavourable; less than five percent of cases recover. In horses that recover, the temperature gradually drops but the breathing remains laboured for some time.

Dikkop or heart form

This form is characterised by swelling beneath the skin layers of the neck and head, particularly the hollow above the eyes. The febrile reaction may remain high for three to six days before declining; however, some animals hardly develop any temperature reaction. The swelling of the head and neck usually appears fairly late in the course of the disease but if it commences early, the course is always more rapid and the condition more serious, and death frequently follows.

As the swelling increases, laboured breathing and blue discolouration of the skin and mucous membranes may occur. In severe cases the eyelids, lips, cheeks, tongue, space under the lower jaw and sometimes also the neck, chest and shoulders, but not the lower parts of the legs, are involved. The swollen eyelids move with difficulty and are kept partly closed. Unfavourable signs during the terminal stages of the disease are bleeding in the mucosa (round purplish red spots) of the eyelid membranes, mouth and underside of the tongue.

Sometimes the animal repeatedly lies down or, when standing, often paws the ground with the front feet and stands restlessly as a result of severe abdominal pain and colic. Paralysis of the oesophagus (*slukpyp*) may be a complication. When the animal drinks, water containing food particles may be returned via the nostrils. In severely affected animals the oesophagus may be stuffed with food and acquire a cord-like appearance. In the dikkop form the course is always more protracted and milder than the dunkop form.

The mortality rate is about 50%, death usually occurring within four to eight days of the onset of the temperature reaction. Sometimes biliary fever may be a complication. This usually occurs in carrier animals whose resistance have been lowered by the AHS virus. In these cases yellow discolouration of the mucous membranes, paleness due to decreased red blood cell counts and constipation are evident.

On post mortem inspection clear and yellowish fluid (in some cases more than 2 litres) is found in the cavity around the heart. The lungs are usually normal and the chest cavity seldom contains an excess of fluid. Moderate to severe swelling of the mucous membranes of the gastrointestinal tract is present explaining the colic symptoms observed.

Mixed form

Although this is the most common form of AHS it is rarely diagnosed clinically; only during post mortem when lesions of both the dunkop and dikkop forms are seen. The incubation period varies from five to seven days. Horses affected by this form may either show signs of respiratory distress followed by swelling or affected horses may initially show signs of the dikkop form before they suddenly develop severe respiratory distress from which they may die. The mortality rate of horses affected by this form of the disease is about 70% and death usually follows three to six days after the onset of the febrile reaction.

Diagnosis

The area, clinical signs of AHS and lesions observed are often sufficiently specific to make a provisional diagnosis of the disease. To confirm the diagnosis, the virus should be isolated from blood collected during the febrile stage of the disease or from specimens of the lungs, spleen and lymph nodes collected at post mortem.

Control

Apart from supportive treatment there is no specific therapy for AHS. It is essential that affected animals should be carefully nursed, well fed and given rest as the slightest exertion may result in death. After recovery the horse should be rested for 4 to 6 weeks before it is returned to its normal work.

As equine babesiosis may be a complication of AHS, blood smears as well as the body temperature should be regularly taken and if smears are positive, animals should be appropriately treated.

Annual vaccination in September (some time before the peak AHS season to allow animals to respond adequately to the vaccine before they are challenged) with the live weakened polyvalent vaccine is the only practical means of control. The polyvalent vaccine contains eight serotypes of AHS virus – vaccine 1 contains serotypes 1,3,4 and 5 and vaccine 2 contains serotypes 2,6,7 and 8. These should be administered at least three weeks apart. Serotype 9 is not included in the vaccine because serotype 6 affords adequate cross-protection to serotype 9.

Generally vaccination has no or limited side effects. A slight temperature response might be observed between 5 to 13 days after inoculation.

Note that prophylactic vaccination against AHS, although efficient at preventing serious losses, cannot fully be relied upon to protect horses against infection or disease. The injection of several serotypes of weakened AHS virus in horses usually results in the production of antibodies against each serotype, although the degree of the response of individual horses may vary. Due to passive immunity acquired by foals born to immune mares (fully vaccinated), it is generally recommended that foals should not be inoculated before they are six months of age.

Insect control

Infection of susceptible horses can be prevented to a large degree by stabling them some hours before dusk and letting them out a few hours after dawn, as *Culicoides* midges are nocturnal and are not inclined to enter buildings. The application of insect repellents and the use of insecticides on the animals' coats are also recommended.

Note: *It is no longer necessary for a vet to administer horse sickness injections, unless your horse is going to the Cape or you are planning on competing in the Cape. Routine vaccination may now be done by a responsible person and noted in your horse's passport. SAH*



African Horsesickness is the biggest threat to horses in South Africa