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African horse sickness: a description of outbreaks in Namibia

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Summary

African horse sickness is a vector-transmitted viral disease that affects equidae. The authors report cases of disease that occurred in Namibia between 2006 and 2008. These were recorded by staff of the *Istituto Zooprofilattico Sperimentale dell'Abruzzo e del Molise 'G. Caporale'* and the Central Veterinary Laboratory in Windhoek. All cases described were confirmed by laboratory testing. This study received the support of Namibian state and private veterinarians and horse breeders.

Keywords

African horse sickness, *Culicoides*, Horse, Equid, Namibia, Virus.

Introduction

African horse sickness (AHS) is a viral disease of equidae that is endemic in sub-Saharan Africa. Cases of the disease have been reported in North Africa where seroconversion still occurs in Morocco, in the Iberian Peninsula and areas of middle-eastern Asia. As is the case for bluetongue, the aetiological agent is a RNA virus of the genus *Orbivirus*, family *Reoviridae*. It is transmitted by arthropod vectors that belong to various species of *Culicoides*. Nine serotypes have been identified to date (2).

The disease is often fatal in horses and mules, and is characterised by signs and pathological disorders caused by respiratory and cardiovascular impairment. The literature on infectious diseases describes four clinical forms, namely: pulmonary, cardiac, mixed and febrile. Clinical signs and lesions can be pathognomonic (2, 6, 9). However, laboratory confirmation of a suspected outbreak is required to identify the serotype responsible for the outbreak.

AHS must be differentiated from other diseases, such as purpura haemorrhagica, viral arteritis and babesiosis (2, 8). In Southern Africa, particular attention must be paid to equine encephalosis, caused by a virus from the same genus and family as the AHS virus. Encephalosis virus causes signs similar to a mild form of AHS (Fig. 1) but it also affects the central nervous system (3, 10). In contrast with AHS, mortality is low and does not exceed 5%. It is often confirmed by retrospective diagnostic serology.

In Namibia, AHS vaccination is performed using a live attenuated vaccine produced by Onderstepoort Biological Products in Onderstepoort, South Africa. The vaccine contains seven of the nine viral serotypes and is administered in two separate doses 21 days apart. The first dose consists of three serotypes and the second of four serotypes. Serotypes 5 and 9 are not included, the former because it was found to be insufficiently attenuated and the latter because it is considered that serotype 6 protects against serotype 9.

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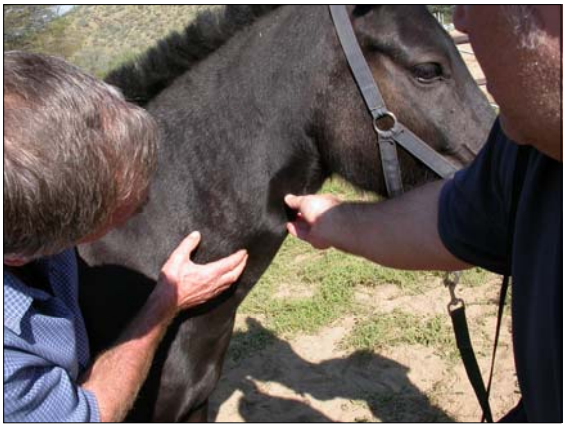

Figure 1
Equine encephalosis: oedema of the neck region

The aim of this paper is to report on cases of AHS diagnosed in Namibia between 2006 and 2008 by staff of the Istituto Zooprofilattico Sperimentale dell'Abruzzo e del Molise 'G. Caporale' (IZS A&M) and the Central Veterinary Laboratory (CVL) in Windhoek.

It is worth noting that this disease has a significant economic impact since Namibia is an exporter of pedigree horses.

The AHS outbreaks described in this study occurred in the area around the capital, Windhoek, and in the districts of Okahandja to the north, Gobabis and Omitara to the east and Mariental to the south (Fig. 2), the area has a radius of about 400 km around the capital.

The cases reported involved horses or foals from farms, riding schools and studs. They normally involved saddle horses that were generally well kept and monitored.

Materials and methods

Case data were collected using a form designed by the IZS A&M Veterinary Epidemiology, Planning and Information Centre. Necropsy was performed in accordance with the IZS A&M standard operating procedures.

Biological samples for diagnosis

Blood samples in ethylenediaminetetraacetic acid (EDTA) were taken from horses during the febrile phase, whereas organs including spleen, lungs, parotid gland and mandibular, mediastinal and mesenteric lymph nodes were taken from carcasses. Samples were refrigerated during transport and processed immediately on arrival at the laboratory. When tests could not be performed on the day after collection, samples were stored at –70°C.

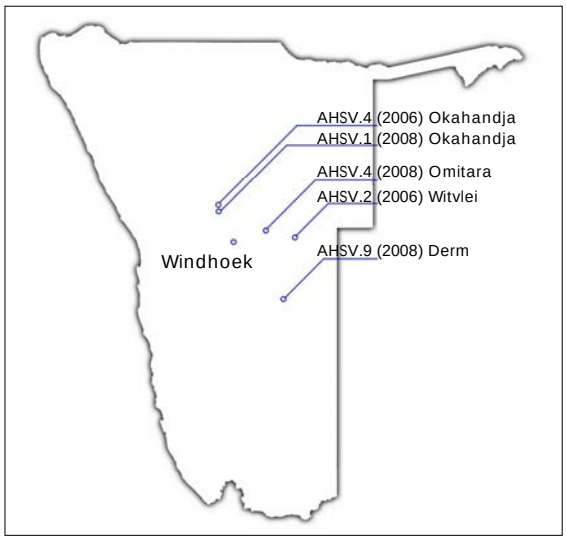

Figure 2
Area around Windhoek showing the location of cases and African horse sickness serotypes isolated
The area has a radius of about 400 km around the capital

Reverse transcriptase-polymerase chain reaction

The CVL used the reverse transcriptase-polymerase chain reaction (RT-PCR) to detect AHS virus in accordance with the method described by Stone-Marschat *et al.* (11).

Virus isolation

The virus was isolated according to the procedures described in the *Manual of diagnostic tests and vaccines for terrestrial animals* (1) on samples that gave positive results for the RT-PCR. The isolated viruses were then typed by serum neutralisation using serotype-specific hyperimmune sera provided by the Onderstepoort Veterinary Research Institute (1).

Description of outbreaks

Unvaccinated horses

Case 1: 2008

A cross-bred four-month-old foal died suddenly that had not presented obvious signs of illness. The foal belonged to a riding school in the Okahandja district, 120 km north of Windhoek. This school had 50 horses of all ages and about 400 zebras.

Results at PCR were positive for AHS virus but these were not confirmed by virus isolation.

Anatomopathological examination

External inspection revealed a slight oedematous swelling of the supraorbital fossa (Fig. 3). Visible mucosa was normal. There was some peritoneal, pleural and pericardial serous effusion, accompanied by mild lung congestion. Small blood haemorrhagic suffusions were observed on the epicardium and along the coronary arteries; white clots were detected within the thoracic cavity. Accentuated intestinal meterorism with numerous petechial haemorrhages on the serosa and mucosa of the small intestine and colon, with traumatic post-mortem rupture of the latter were also recorded.

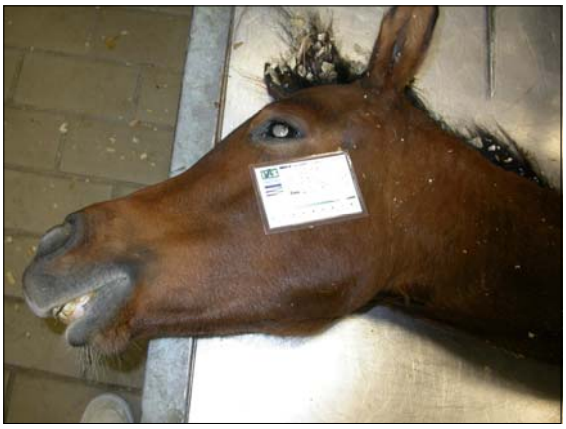

Figure 3
Mild oedema of the supraorbital fossa

Case 2: 2008

A cross-bred six-month-old foal, born to a mare that had been imported from northern Europe the previous year and that had not been vaccinated against AHS. The foal came from the same farm as case 1 in the Okahandja district. It was found dead in the morning without any appreciable premonitory clinical signs; the afternoon before death it was seen running in the paddock.

PCR results for AHS virus and virus isolation were positive and the strain isolated was identified as serotype 1.

Anatomopathological examination

Cutaneous and subcutaneous examination revealed slight swelling of the supraorbital fossa and eyelids. Visible mucosa was normal.

Subcutaneous haemorrhages were observed in the region of the head. There were no muscular lesions. A large volume of blood was present in the abdominal and thoracic cavities and in the pericardium. Haemorrhagic suffusions were seen on the epicardium and widespread haemorrhages on the endocardium and cardiac valves. Haemorrhages and congestion of the pulmonary parenchyma were also observed. Other findings included hepatic congestion, mild hyperaemia of the mucosa of the glandular fundus of the stomach and petechial haemorrhages on the intestinal serosa. The mandibular, retropharyngeal and mediastinal lymph nodes were enlarged, haemorrhagic and oedematous. The kidneys were congested and milky-coloured urine was observed in the pelvis and ureter.

Case 3: 2008

Case 3 was an eight-year-old saddle mare from a farm in the Mariental district, 250 km south of Windhoek. The animal was slaughtered because it was suspected of being affected by anthrax or rabies. Only the head was sent to the CVL. According to the owner, it had been transferred from the pasture to the stables due to emaciation and starvation. After eating normally for a week, it stopped feeding. It was then transferred to a pen with other horses, where it began eating again for a few days. One morning it was found on the ground with swollen eyes, a protruding tongue and blood loss from the anus. The animal had been sick for about two weeks.

The PCR results for AHS virus and viral isolation were positive and the strain isolated was identified as serotype 9.

Anatomopathological examination

Cutaneous and subcutaneous examination revealed swelling of the eyelids.

Case 4: 2006

A six-month-old foal which came from the same area as cases 1 and 2 (the Okahandja district). The day before death, the animal had fever, peaking at 40°C-41°C, it was lethargic, showing slight oedema of the supraorbital fossa and neck (Fig. 3) and haemorrhagic conjunctivitis (Fig. 4). According to the owner, death occurred in a few hours.

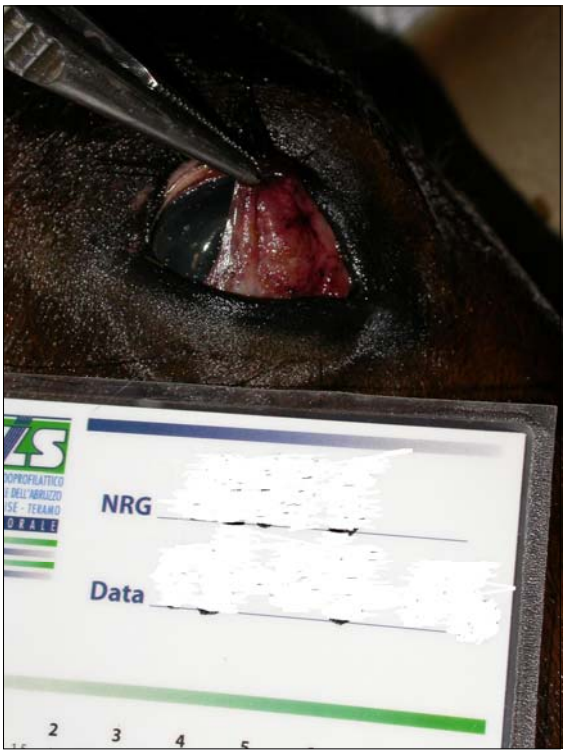

Figure 4
Haemorrhagic conjunctivitis

The PCR results for AHS virus and viral isolation were positive and the strain isolated belonged to serotype 4.

Anatomopathological examination

A slight gelatinous exudate was observed in the subcutaneous, subfascial and intramuscular tissues of the head, neck and dorsal thoracic area. Haemorrhages were noted in the neck muscles and salivary glands. Haemorrhagic suffusions were recorded throughout the parietal pleura, abdominal tunica muscolaris and at subpleural, pericardial, subpericardial, endocardial and subcapsular spleen levels.

Other findings included hydrothorax, mild ascites, ectasia of the blood vessels, pulmonary oedema, haemorrhages on the heart valves and tunica adventitia of blood vessels, mild liver congestion and hyperaemia of the glandular fundus of the stomach. Haemorrhagic suffusions were also observed on the mucosa of the oesophagus and trachea were foamy serous liquid was present. Lymph nodes in the area of the head were haemorrhagic, while those of the lungs were enlarged and haemorrhagic. The kidneys were congested, with haemorrhages on the bladder mucosa; the presence of haematuria was recorded.

Horses without a history of regular vaccination

Case 5: 2006

A male, seven-year-old horse from a farm in the Gobabis district, 230 km east of Windhoek. The animal had worked normally on the farm the day before the onset of clinical signs. Oedema of the supraorbital fossa, the head and neck, accompanied by haemorrhagic lacrimation were noted. Death occurred within a few hours. Six other animals had died previously with similar signs.

The PCR results for AHS virus and viral isolation were positive and the strain isolated was identified as serotype 2.

Anatomopathological examination

Oedema of the supraorbital fossa and haemorrhagic conjunctivitis were observed (Fig. 5). The head, neck and dorsal thoracic area showed marked oedema at subcutaneous, subfascial and intramuscular levels (Figs 6 and 7), in the thigh and subscapular muscles, oedema was only just visible. Haemorrhagic suffusions were recorded on the parietal pleura (Fig. 8), abdominal tunica muscularis and at the subpleural level; hydrothorax (Fig. 8) and ascites were also evident and were accompanied by pulmonary oedema (Figs 9 and 10), together with a considerable increase in lung weight and size. Hydropericardium and widespread pericardial, subepicardial, endocardial and valvular haemorrhagic suffusions were observed (Figs 11, 12 and 13).

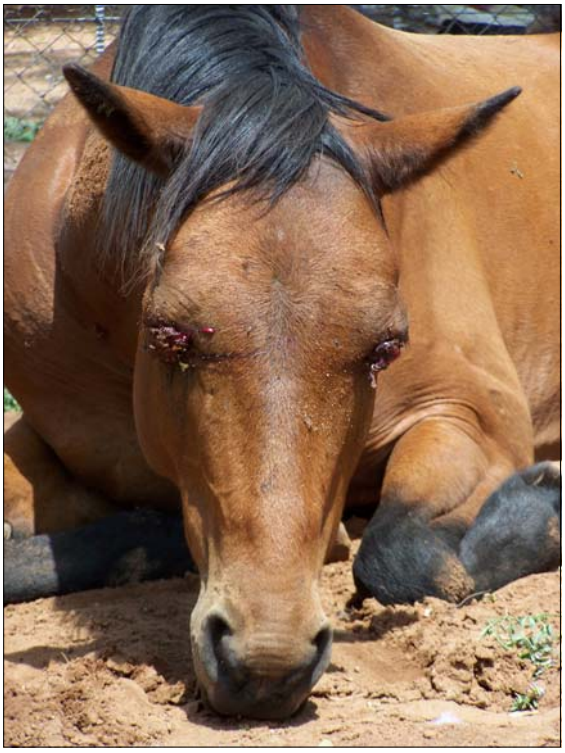

Figure 5
Oedema of the supraorbital fossa and haemorrhagic conjunctivitis

Figure 6
Subcutaneous haemorrhagic oedema

Widespread haemorrhagic suffusion was also seen on the omentum, serosa and mucosa of a large part of the enteric tract and the tunica adventitia of the blood vessels (Fig. 14). The liver was congested and glandular gastric mucosa hyperaemic. The spleen was slightly enlarged, with subcapsular blood suffusions. The cephalic and pulmonary lymph nodes were haemorrhagic, while the kidneys were congested with haemorrhages on the bladder mucosa. Haematuria was observed.

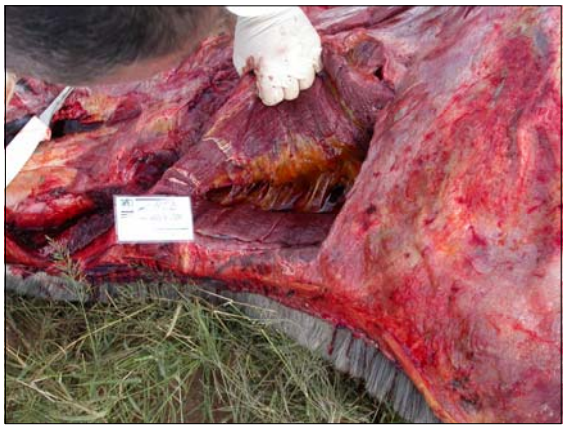

Figure 7
Subfascial oedema

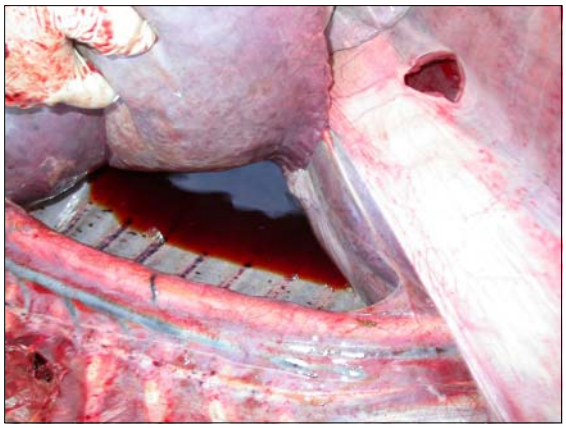

Figure 8
Hydrothorax, haemorrhagic suffusion on the pariental pleura

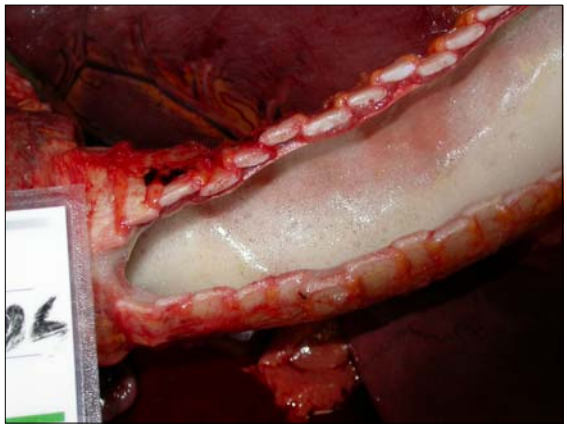

Figure 9
Pulmonary oedema, foamy serous liquid in trachea

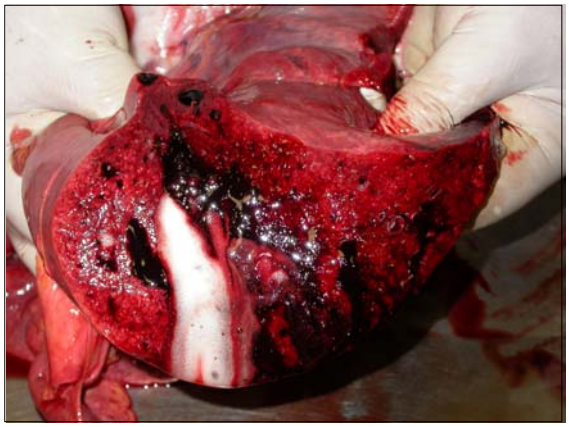

Figure 10
Pulmonary oedema

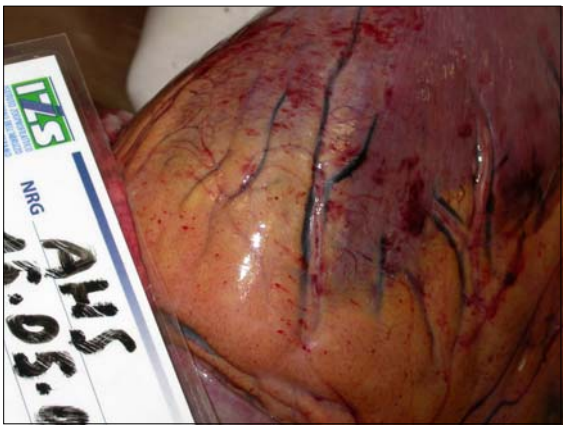

Figure 11
Epicardium, haemorrhagic suffusions

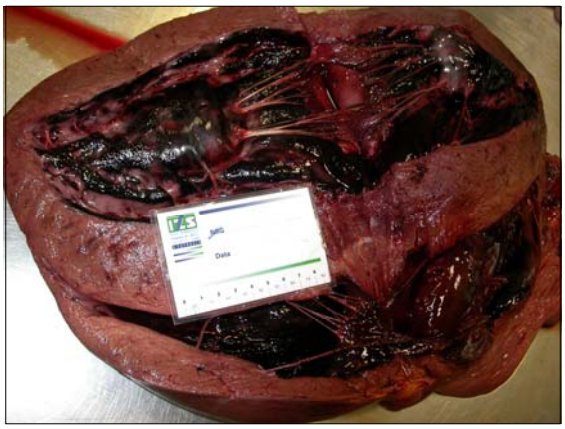

Figure 12
Endocardium, haemorrhagic suffusions

Vaccinated horses

Case 6: 2008

A five-year-old Arab stallion from a farm in Omitara, between the districts of Windhoek and Gobabis. The animal began showing signs of AHS in a stable in Swakopmund, on the Atlantic Coast, where it had been moved the previous month. It died about 24 h after the onset of clinical signs, namely: tachypnoea, oedema of the supraorbital fossa, of the eyelids and of the entire head and neck, in the absence of pyrexia. Phenylbutazone was administered intravenously as symptomatic treatment. The animal had been vaccinated about a month before the onset of signs, in view of its transfer to the coast.

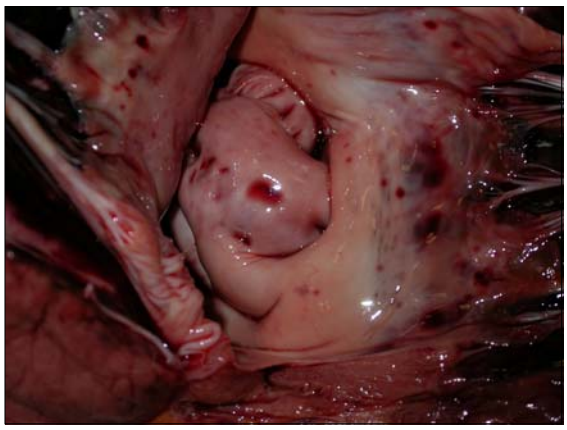

Figure 13
Cardiac valves, haemorrhagic suffusions

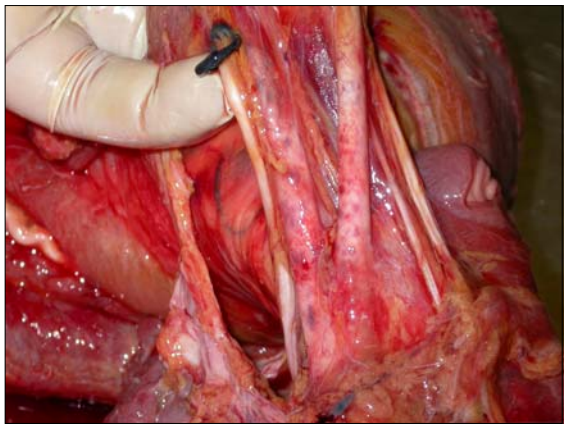

Figure 14
Blood vessels, haemorrhagic suffusion on the tunica adventitia

The PCR results for AHS virus and viral isolation were positive and the strain isolated belonged to serotype 2.

Anatomopathological examination

Necropsy revealed swelling of the supraorbital fossa and eyelids, with subcutaneous haemorrhagic suffusion that did not affect the

lower layers. The subcutaneous haemorrhages extended throughout the entire cephalic area. Oedema of the subcutaneous tissue and of the muscles of the neck, together with venous ectasia in the entire subcutaneous thoracic area, were observed. Hydrothorax and hydropericardium were seen. There were petechial haemorrhages on the pericardium, epicardium and endocardium and dilatation of the right heart. Food residues were found in the trachea, while the pulmonary parenchyma was congested, with haemorrhagic areas, and mild pulmonary oedema was present. Congestion and subcapsular haemorrhagic suffusions were observed in the liver (Fig. 15). Food residues were found in the oesophagus; the stomach was constipated, with the presence of fermented food and significant hyperaemia of the glandular mucosa, with numerous *Gasterophilus* spp. larvae present in the oesophageal part (Fig. 16). Petechial haemorrhages were present on the omentum and serosa of the small and large intestines (Figs 17 and 18). The spleen was slightly enlarged, with capsular petechial haemorrhages (Figs 19 and 20). The lymph nodes of the cervical area and mediastinum were haemorrhagic. The renal pelvis contained milky-coloured urine.

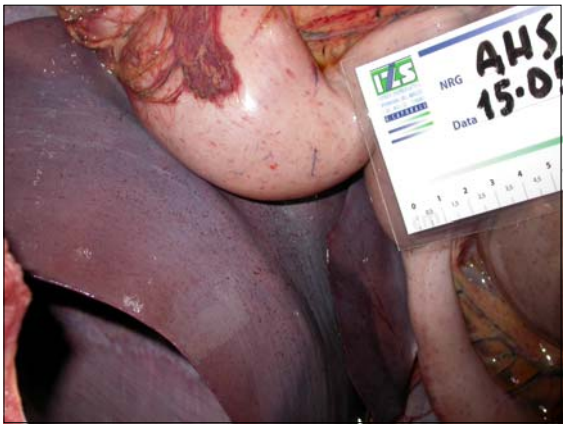

Figure 15
Liver, subcapsular haemorrhagic suffusions

Case 7: 2008

An eight-year-old mare from a farm on the northern outskirts of Windhoek that had been vaccinated seven months previously.

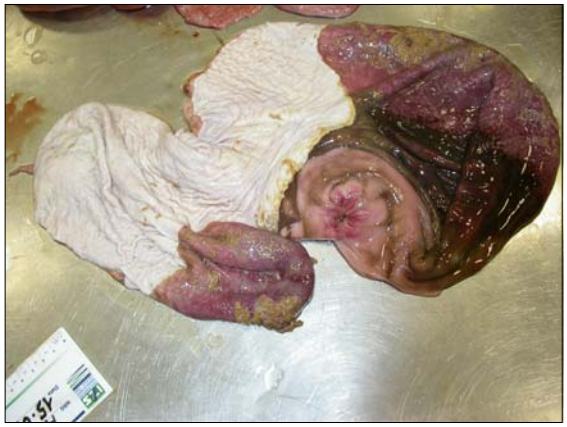

Figure 16
Stomach, hyperaemia of the glandular mucosa

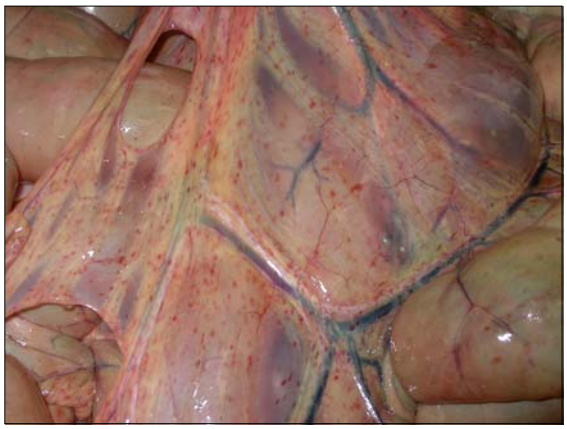

Figure 17
Omentum, petechial haemorrhages

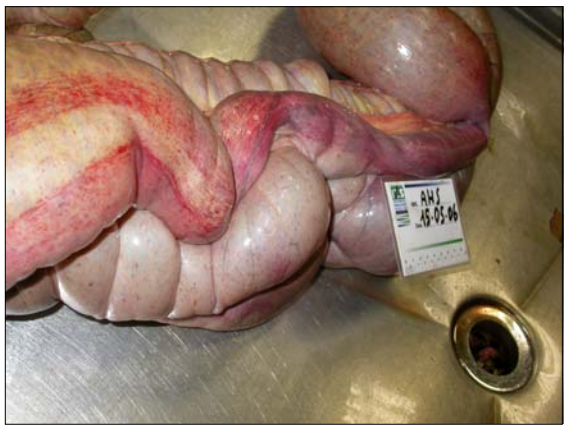

Figure 18
Large intestine,petechial haemorrhages on serosa

The PCR results were positive for AHS virus, but were not confirmed by virus isolation.

The animal was examined on a number of occasions. On the first veterinary examination,

it presented severe head and neck oedema, especially of the supraorbital fossa and eyelids, hyperthermia, tachypnoea, tachycardia, anorexia and lethargy. During the examination, a blood sample was taken and symptomatic treatment with phenylbutazone was administered.

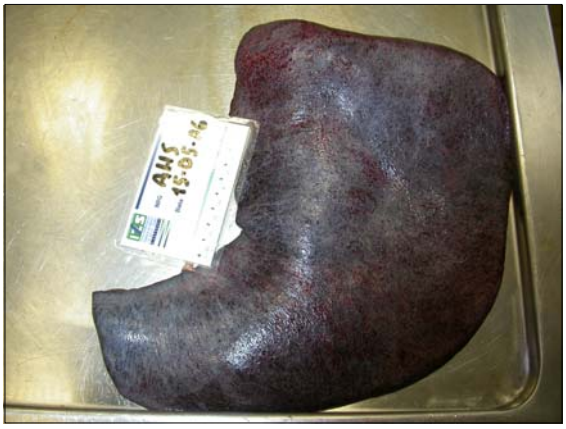

Figure 19
Spleen, subcapsular petechial haemorrhages

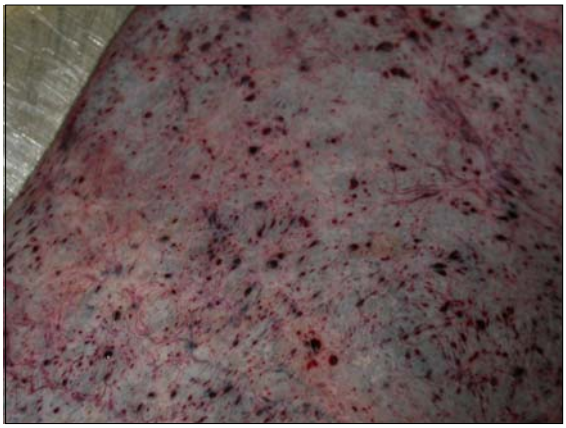

Figure 20
Spleen, subcapsular petechial haemorrhages, magnification

A subsequent examination seven days later revealed that the animal was well nourished with reasonable muscular tone but was slightly lethargic. Specific signs and behaviour were as follows: the head and neck were extended with the eyes half-closed. Very obvious oedema of the supraorbital fossa, eyelids, mandibular area and neck persisted. The skin was anelastic and there were signs of dehydration. Visible mucosa was slightly pale, with subclinical jaundice. Petechial haemorrhages were present on the ventral side of the tongue. The mandibular lymph nodes were enlarged. The rectal temperature was 35.5°C, the pulse weak and shallow at 24 beats/min, the respiratory rate 18 breaths/min, short and frequent. The owner reported complete anorexia, reduced defecation and urination.

The mare was re-examined 30 days later. Her nutritional status was good, muscle tone fair and sensory reactions slightly depressed. Her head and neck were still extended and eyes half-closed. The eyelid oedema, dehydration and lymph node enlargement were reduced. Visible mucosa was normal. The body temperature was 37.5°C, the pulse was recorded at 24 beats/min and the respiratory rate 10 breaths/min, both apparently normal. Major organ functions were reported to be normal. These findings led to the conclusion that the animal was recovering.

Case 8: 2008

A two-year-old South African Warmblood mare that was kept in a stable in the Okahandja district. The animal had undergone two vaccination cycles, the first at the age of five months and the second when a year old. At the age of about 20 months, it presented obvious oedema of the supraorbital fossa, lethargy and a rectal temperature of 38.9°C. The signs disappeared within a few days.

The PCR results were positive for AHS virus but these results were not confirmed by virus isolation.

Case 9: 2006

A male six-year-old horse that had received multiple vaccinations and was kept at the Okakambe Trails stables in the Swakopmund district, about 30 km north of the district capital. The animal presented a body temperature of 40°C-41°C, lethargy and neck and head oedema. The conjunctivae were congested. When contacted a month later, the breeder reported that the horse no longer presented any signs of disease. Tests were conducted on blood in EDTA.

The PCR results for AHS virus and viral isolation were positive and the strain isolated was identified as serotype 2.

Discussion

Of the four clinical forms – febrile, cardiac, mixed and pulmonary – described in the literature (4, 5), the first, characterised by a slightly or moderately raised temperature and oedema of the supraorbital fossa, was observed in only one vaccinated animal (case 8). This mare recovered in a few days. This form of the disease can pass unobserved by breeders and its frequency may thus be underestimated.

The cardiac, subacute, form is characterised by fever that lasts several weeks and subcutaneous oedema of the head, neck and dorsal thoracic area (2). Oedema of the supraorbital fossa and haemorrhagic conjunctivitis are common features. This form was observed in two horses (cases 7 and 9), which had both been vaccinated and which both recovered.

The mixed form, consisting of a combination of the cardiac and pulmonary forms, is normally the most common (2). This form was observed in a vaccinated male horse (case 6) that presented respiratory signs and subcutaneous oedema of the head and neck.

The anatomopathological lesions were typical of the cardiac form, namely:

- gelatinous exudate in the subcutaneous, subfascial and intramuscular tissues and lymph nodes
- hydropericardium and epicardial and endocardial haemorrhages
- petechial haemorrhages of the enteric tract serosa
- mild pulmonary oedema.

In addition, the typical lesions of the pulmonary form could be seen, as follows:

- hydrothorax
- ascites
- hyperaemia
- oedema of the glandular gastric mucosa.

It should be noted that neither the course of the disease, death within 24 h, nor the absence of pyrexia are mentioned in the literature (2).

The pulmonary form is the hyperacute form of AHS and can evolve so rapidly that no clinical signs are seen in the affected animal. Marked lethargy and hyperthermia (40°C-41°C) in the presence of respiratory signs often characterise this form. This was observed in cases 1, 2, 4 and 5.

These cases presented pulmonary lesions varying from mild congestion to oedema, with the presence of foamy serous liquid in the trachea in one case. Hydrothorax and ascites were always observed.

Moreover, and in contrast with reports in the literature, subcutaneous and subfascial oedema of the head and neck were seen in two cases and haemorrhagic suffusions throughout the enteric tract in all horses.

Case 3 cannot be attributed to any of the four forms as it was not possible to perform a necropsy on the animal as only the head was made available. The clinical history of this case suggests a subacute form of AHS.

Conclusions

In agreement with the literature consulted (2, 4, 7, 8), our observations revealed that:

- differentiation between the four forms of AHS described is not always possible or is unambiguous
- AHS serotypes 1, 2, 4 and 9 have been and/or are circulating in Namibia
- serotype 9, described as being of low pathogenicity, is capable of killing unvaccinated animals, following a subacute disease course.

With reference to immunisation, in three of the four unvaccinated animals, death was sudden and occurred in the absence of any significant signs, while in the fourth horse, death ensued after a lengthy course.

One of the four vaccinated animals showed a disease course similar to the unvaccinated horses, with death occurring within 24 h after appearance of clinical signs. In the remaining three cases, the animals recovered a month after the onset of signs.

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