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Review on African Horse Sickness

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Abstract: African horse sickness is an infectious but but non-contagious insect-borne viral disease affecting entire species of Equidae. It is caused by a virus of the genus Orbivirus that is spread by hematophagou midges of the genus Culicoides. Environmental factors influence the distribution of midges inside their overall range and hence the disease has a geographical distribution. Four clinical presentations (Pulmonary, cardiac, mixed and African Horse Sickness fever) of the African horse sickness have been described, each related to specific pathogenesis and mortality ranging between 95% (Pulmonary) to 0% (fever). Diagnosis is made on the basis of typical clinical signs and lesions in association with the seasonal incidence of competent vectors. There is no treatment for this disease. The disease has great economic impacts in countries where horses are necessary for transportation and as draft animals. The principles of control in endemic areas are vaccination and reduction of exposure to biting insects, whereas in non-endemic areas the objective is to prevent the introduction of the disease and eradication. Horse owners should be advised to stable their horses from early evening until morning while the Culicoides are most active and to spray horses' house with suitable insecticides. The objective of this paper is to review African Horse Sickness.

Key words: Environmental Factors · Equidae · Orbivirus

INTRODUCTION

African Horse Sickness (AHS) is a highly infectious noncontagious viral disease that affects all species of Equidae. The disease has no public health importance. The death rate varies depending on the form of the disease and the species but it can be as high as 95%. African Horse Sickness is widespread in sub-Saharan, central and east Africa. The disease sometimes spreads from central to southern or northern Africa. A number of outbreaks have been reported outside Africa in the 20th century including the Middle East, Spain, Portugal, Saudi Arabia, Yemen and the Cape Verde Islands [1].

African Horse Sickness is caused by the African Horse Sickness Virus (AHSV) which belongs to the genus Orbivirus. Orbiviruses are transmitted to animals mainly by arthropod vectors. The distribution of these viruses is very similar to the distribution of their particular vector and it is consequently influenced by climatic conditions. African Horse Sickness Virus is spread by midges of the genus Culicoides [2].

African Horse Sickness is prevalent to sub-Saharan Africa, including both the tropical and sub-tropical regions and happens from Senegal in the west to Sudan, Ethiopia and Somalia in the east and south to South Africa. The disease has been reported in North Africa, but it is mostly inhibited to the south of the Sahara due to the desert's large expanse that turns as a protective barrier. AHS has not been reported in Madagascar or several of the Indian Ocean islands [3].

The disease has both a periodic (late summer/autumn) and a recurrent incidence with main epizootics in Southern Africa during warm–phase occasions [4]. Death due to AHS is associated with the species of Equidae affected besides the strain or serotype of the virus. However, two field vectors are: Culicoidesimicola and *Culicoides bolitinos* [5].

African Horse Sickness is well adapted to Zebra, which is considered the natural host and reservoir of the virus in Africa. Other equine species and their crossbreeds are prone to AHSV infection and further known susceptible species are dogs [6].

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The most important vector of AHSV in the African continent is *C. imicola* and for several years this species was considered to be the only Culicoides involved in AHSV transmission in the field. Another vector was involved in the AHS spread in South Africa. In the year 2000, *C. bolitinos* was shown as a possible possible AHS vector in South Africa [7].

African Horse Sickness in horses regularly presents as any of the following four clinical forms: Pulmonary, cardiac, mixed and AHS fever [8].

There is no treatment for AHSV; the disease is managed by supportive treatment. Disease prevention is by vaccination with a polyvalent vaccine as all AHSV serotypes are present in South Africa and in most parts of sub-Saharan Africa. A number of methods are employed for the diagnosis of AHSV, including virus inoculation of cell cultures, mice inoculation, postmortem, serology and molecular assays [9].

The essential strict quarantine rules in place for the export of equines from South Africa have a paralyzing effect on the local equine industry [10].

Ethiopia is facing severe and repeated outbreaks of AHS in different regions. The virus might have been presented into Ethiopian equines by wind-borne infected midges (Culicoides) from prevalent regions of Africa [11].

Therefore, the objective of this seminar paper is:

- To review African Horse sickness

Definition of African Horse Sickness: African Horse Sickness is a highly fatal viscerotropic, insect-born viral infectious disease that distresses Equidae. The death rate fluctuates depending on the form of the disease and the species however it can be as high as 95% [1]. The clinical signs and lesions outcome from a selective increased in vascular permeability and are characterized by alterations in the respiratory and circulatory system [12].

Etiology: The African horse sickness virus is a member of the genus Orbivirus that belongs to the Reoviridae family, with several relationships in epidemiology to the prototype virus. Such as an orbivirus, AHSV has a double-stranded ribonucleic acid genome of ten segments [5].

The virion is an unenveloped particle of a size about 70 nm. The genome of AHS virus is composed of ten double-stranded ribonucleic acid (dsRNA) segments, encoding seven structural viral proteins (VP1-7), most of which have been entirely sequenced for AHSV serotypes 4, 6 and 9 and four nonstructural proteins (NS1, NS2, NS3, NS3A) [13].

Proteins VP2 and VP5 form the external capsid of the virion and proteins VP3 and VP7 are the main internal capsid proteins. Proteins VP1, VP4 and VP6 constitute minor inner capsid proteins. The NS3 proteins are the second most inconstant AHSV proteins [14], the most inconstant being the key outer capsid protein, VP2. This protein, VP2, is the factor of AHSV serotypes and together with VP5, the mark for virus neutralization activity and there are nine immunologically separate serotypes [15].

Epidemiology: African horse sickness is common to sub-Saharan Africa, including both the tropical and sub-tropical regions and happens from Senegal in the west to Sudan, Ethiopia and Somalia in the east and south to South Africa [16]. Because of the disease's correlation with vector populations; it has a seasonal and cyclical nature. The vectors for AHSV are the crepuscular biting midges of the Culicoidesgenus, which transmit the virus by biological means [17].

During an outbreak of AHS, one serotype will usually dominate, but it does not exclude other Serotypes [18]. Generally, serotypes 1-8 are mostly responsible for outbreaks in South Africa, while serotypes 4 and 9 have been recorded in outbreaks outside of Africa [19]; although this is becoming less clear [20]. Besides equids, dogs are the only other species that contract a highly fatal form of the disease after infection with AHSV [6].

Geographic Distribution and Occurrence: African Horse Sickness is restricted to the tropical and sub-tropical areas of sub-Saharan Africa. From 2008 to 2012, outbreaks of AHS have been reported in Angola, Botswana, Eritrea, Ethiopia, Gambia, Ghana, Lesotho, Namibia, Nigeria, Senegal, South Africa and Swaziland. The disease is suspected in several other African countries but has not been confirmed [21]. The disease has been reported in North Africa, but it is mostly constrained to the south of the Sahara due to the desert's large expanse that acts as a protective barrier. AHS has not been reported in Madagascar or any of the Indian Ocean islands [18].

Because of the disease's correlation with vector populations, it has a very seasonal nature. However, longer term studies have largely concluded its cyclical

nature as well. It is seasonal as it peaks during the late summer/autumn months and cyclical as it has been proposed that the main outbreaks have occurred following El Niño warm-phase events, which occur approximately every 20 years [17].

Generally, AHS first emerges during December/January with a peak in cases during March/April [3]. Interestingly, in East Africa, cases are seen from September to December with a peak in October and all nine serotypes have been detected in eastern and southern Africa, with serotype 9 being more dominant in the north-west and north-east regions of sub-Saharan Africa [22].

Host Range: The range of hosts for AHSV is confined principally to equine species. The most susceptible are horses up to 95% mortality followed by mules 70% mortality. African donkeys and zebras are the most resistant and apparently remain subclinical for AHS [23]. Additional host for AHS is the domestic dog. Generally, the dog only becomes viraemic after the ingestion of infected meat or experimental infection, however, they are not considered to play any role in the transmission of AHS [24].

Pigs, cats and monkeys are resistant to infection. The OIE reports that there is no evidence that humans could become infected with AHSV. However, it has been described previously that certain neurotropic vaccine strains may cause encephalitis and retinitis of the eyes in humans following aerosol infection in vaccine production [25].

Transmission and Source of Infection: African horse sickness virus is incidentally transmitted to equids by haematophagous arthropods. Direct transmission has only been seen in dogs after oral contamination with infected meat. According to the OIE, certain mosquito and tick genera may establish an occasional mode of transmission [5]. A lot of vectors have been found infected with AHSV, in specific, mosquitoes of the Aedes, Culexand Anophelesgenera, or ticks of the Hyalomma or Rhipicephalus genera [26]. In sub-Saharan Africa, *Culicoides imicola* has been accepted as the major vector of AHSV: *C. imicola*is found throughout sub-Saharan Africa, the Mediterranean Basin and South East Asia [27].

The distribution of *C. imicola*, geographically and seasonally, is dependent on a range of environmental factors. Temperature is probably the most significant extrinsic factor affecting the transmission, infectivity and virogenesis of AHSV in Culicoidesvectors and the survival of the midges themselves. As temperatures raise the infection rates of Culicoidesmidges increase and transmission can occur faster than in cooler temperatures. Interestingly, midge survival rates decrease as temperature increases. Consequently, the rate of transmission results from the interface of these two variables [28]. The spread of the virus to areas where it does not usually exist occurs both by the movement of infected animals such as zebras and horses and by the transportation of midges by wind and the virus is present in all body fluids and tissues of affected animals from the onset of fever until recovery and clinically affected Equidae are the chief source of the virus during an outbreak [12].

Risk Factors: The virus could have been sustained during the cold season through low-level transmission between reduced insect populations and infected donkeys or mules. In these animals, the viraemia has been shown to last longer than in horses [29].

The spatial evolution of AHS outbreaks depends on the vector biotope. The biotopes with conditions favorable for larval development and adult survival are wet and areas near waterholes where the shade maintains humidity. AHS outbreaks are directly associated to periods of vector activity, which is greatest in hot and humid climates, i.e. Subtropical areas, after the rainy season and the rates of AHSV infection of Culicoides vectors and of virogenesis is temperature dependent. As temperature increases, infection rates tend to increase, virogenesis is more rapid and transmission can arise earlier. But, midge survival rates decrease and vice versa [30].

Environmental Risk Factors: African Horse Sickness predominates in the warm coastal areas or low-lying, moist, inland areas and occurs predominantly in and around lakes, pans and rivers. Heavy, early summer rainfall, followed by a drier period appears to favor the development of epidemics and the occurrence of the disease is often seasonal because of the seasonal variations in the number of Culicoides species present and possibly other weather related factors including topography, influence the distribution of midges within their overall range and the disease has a geographical distribution [31].

Generally, the disease first emerges during December/January with a peak in cases during March/April and interestingly, in East Africa, the majority of cases are seen from September to December with a peak in October [22].

Host Risk Factors: Natural infection occurs in equidae and, the most severely affected animals are horses, mules, donkeys and zebras in order of lesser degrees of susceptibility. Following the natural infection or vaccination- immunity specific to that specific strain will be developed, but not to heterologous strains [12].

Zebras are believed to be the reservoirs for AHSV since they naturally experience a subclinical course of infection. The fact that the virus has not become established outside of sub-Saharan Africa provides further evidence. Also, the extremely high rates of mortality exhibited by the virus infected horses and mules are consistent with these animals being accidental hosts. Even though Culicoidesare active year-round through much of the geographical disease range, the virus can nevertheless be maintained in biting midges during their low activity periods and/or when temperatures are too low to allow for viral replication in the vector; thus the disease can be maintained in midges for up to 11.5 months [4].

Mortality and Morbidity: The socio-economic influence of AHSV drops principally on two separate equine populations within Africa. Morbidity and mortality from the disease within working equids may limit the draft power of these animals provided in low-income countries, thus affecting food security, poverty alleviation and gender equality [32].

African Horse Sickness Virus types 1-8 are considered to be highly pathogenic for horses and infection results in high mortality of up to 95%. The type 9 AHS virus seems to be less pathogenic and infection may result in lower mortality i.e.70%. Mortality in mules may reach 50% while in donkeys it is around 10% and generally morbidity and mortality rates are dependent on the species of animal, previous immunity acquired (mortality and morbidity decrease) and the form of the disease [14].

The Status of the Disease in Ethiopia: Ethiopia is facing severe and frequent out breaks of AHS in different regions. The virus could have been introduced in to Ethiopian equines by wind- borne infected midges (Culicoides) from common regions of Africa. The vectors were recognized to be wind- driven and migrate, carrying the virus over 700Km [11].

The virus neutralization test specified that two serotypes of AHS were involved in the outbreak that occurred in 2002-2003 in southern Ethiopia (Awassa, Hossana, Wondogenet and Hagerselam), western Ethiopia (Jimma, Bedelle, Nekemte, Horroguduru and Chaliya) and central Ethiopia (Debrezeit, Meki, Zeway, Filtimo and Bekejo), Serotypes 9 and 6 were isolated from blood, spleen and lymph nodes collected from sick and died animals. It is well documented that despite its widespread distribution, serotype 9 of AHSV has a lower virulence than other serotypes; killing few horses in endemic areas [33].

Multiple types of AHSV were known circulating in Ethiopia currently, including AHSV-2, 4, 6, 8 and 9. This signifies the first identification of AHSV-4, AHSV-6 and AHSV-8 in Ethiopia. AHSV-9 was the dominant serotype recognized followed by serotype 4. The detection of formerly exotic serotypes (AHSV-4, -6 and -8) in the Oromia region of Ethiopia suggests an important shift in the global epidemiology of AHSV and may indicate a significantly increased risk to equids in the region. Ethiopia also represents a potential source of infection, for further emergence of the disease into adjacent regions and/or other geographical areas [34].

Pathogenesis: Zebras are the natural host and reservoir; transmission is via biting arthropods and midges (Culicoides species) are the most significant vector. Initial viral multiplication takes place in the regional lymph nodes resulting in a primary viraemia that disseminates the virus throughout the body via the blood and leads to the infection of target organs such as the heart, lungs, spleen and lymphoid tissues. The primary organs affected by all serotypes are heart and lung tissue followed by the spleen [35].

Infection results in damages to the circulatory and respiratory systems resulting in severe effusion and hemorrhage in different organs and tissues. Horses is bitten by an infected midge →initial viral replication in regional lymph nodes →primary viremia →infection of target organs (endothelial cells and mononuclear cells of the lung, spleen and lymphoid tissue) secondary viremia →virally induced →endothelial cell damage and activation of infected macrophages with subsequent cytokine production →increased vascular permeability

→edema. Distinct serotypes demonstrate individual tropisms for pulmonary and cardiac endothelial cells and account for the four frequently over lapping clinical forms of AHS: pulmonary form, cardiac form, mixed form and horse sickness fever form [36].

Clinical Signs: The incubation period ranges from 2 to 21 days but is usually on Average 7–14 days long. When animals are a source of infection for Culicoidesmidges' incubation period may reaches 40 days for domestic horses [21].

In experimental infections in horses, the incubation period lasts 5-7 days with a minimum of 2 days and a maximum of 10 days. This has been found to be dependent on the dose and virulence of the virus. Clinical signs develop as a result of the damage to the endothelial cells in blood vessels and reduced function of the circulatory and respiratory systems [18]

The disease in horses usually presents as one of the following four clinical forms: Pulmonary, cardiac, mixed and AHS fever, but the occurrence of one or another form of the disease is determined by innate resistance, acquired heterologous resistant and virulence of the infecting strain of the virus [6].

Pulmonary or per Acute Form: The pulmonary form, known as dunkop('thin head' in Afrikaans) is per acute and may develop so rapidly that an animal can die without prior signs of illness. Frequently, there will be marked depression and fever (39–41°C), followed by the onset of respiratory distress and severe dyspnoea. Coughing spasms may occur, the head and neck are extended and severe sweating develops. In the terminal stages large quantities of frothy fluid may be discharged from the nostrils and death rates from this form commonly exceed 95% [37].

Cardiac or Sub-acute Form: The cardiac form of the disease is characterized by fever (39–40°C) lasting a number of weeks. The main clinical finding is subcutaneous oedema, mostly of the head including the supraorbital fossae, neck and chest. Equideas suffering from this form of the disease are said to have the *dikkop* form of the disease ('thick head'), referring to the pronounced supraorbital swelling which is usually observed and mortality rates may exceed 50% [37].

Mixed Form: The mixed form is the most common form of AHS and the combination of clinical signs that can be seen in both cardiac and pulmonary forms of the disease are observed. The mortality rate is approximately 70% and death usually occurs within three to six days of fever onset [37].

Horse Sickness Fever: Horse sickness fever is always mild and not lethal, usually involving only mild to moderate fever and oedema of the supraorbital fossae. It is more commonly encountered in infected African donkeys and zebras [37]. This form is the merely form of the disease that occurs in zebras [12].

Diagnosis: There are various methods used for the diagnosis of AHSV, including virus inoculation of cell cultures, mice inoculation, postmortem, serology and molecular assays [38].

The traditional methods of virus identification are serological techniques that are used to isolate virus as antigen, some of the most commonly used serological techniques are:

- Serum neutralization test: Neutralizing antibodies can be detected as early as three weeks post infection by the serum neutralization test and can continue for many years. Their identification allows for the typing of circulating viruses. Some cross-neutralization between the virus serotypes has been observed, in particular between serotypes 9 and 6 and 5 and 8 [39].
- Enzyme-Linked Immunosorbent Assay (ELISA): Several indirect or competitive ELISAs have been developed for the detection of the virus group antigens for the last 40 years. They appear to have satisfying sensitivity and now broadly implemented in diagnostic laboratories [40].
- Laviadaand his Colleague have developed a DIVA assay (i.e. one that Differentiates Infected from Vaccinated Animals), based on recombinant NS3 from serotype 4 virus, for the detection of anti-NS3 antibodies; such antibodies are usually only induced after infection with AHSV or vaccination with a live vaccine [41].

Confirmatory diagnosis of AHS is better achieved through virus detection techniques than through serology, because many horses will die before the induction of the virus antibodies, antibodies being detectable 10 to 14 days' post infection [39].

Recently, but, the use of Real-Time Polymerase Chain Reaction (RT-PCR) assays, which allow rapid virus identification and typing, has replaced serological techniques in most laboratories. RT-PCR assays amplify

conserved genomic sequences (1, 3, 5, 7 or 8 dsRNA segments) for group diagnosis, or highly variable segments (segment 2 encoding VP2 in particular) for serotyping [42].

Post Mortem Finding: African Horse Sickness affects the vascular endothelium of many organs and causes a selective increased permeability of blood vessels in specific organs, producing oedema of the subcutaneous tissues and lungs, pronounced submucosal congestion of the fundus of the stomach, petechiae and hemorrhages in internal organs, ascites, hydrothorax and hydro pericardium[43].

Gross lesions at necropsy depend on the clinical form of the disease: In the pulmonary form, alveolar and interstitial oedema of the lungs and hydrothorax are the key findings. In the cardiac form, oedematous infiltration of the head and neck musculature is the most noticeable lesions, together with subcutaneous oedema of the head. In the mixed form, a combination of these lesions occurs and it is the most fatal form of AHS, with lesions of the cardiac or the pulmonary form predominating [6].

Differential Diagnosis: The disease shares a number of clinical signs and symptoms that are seen following infections by the closely associated Orbivirus, Equine Encephalosis Virus. Other differential diagnoses for AHS include Babesiosis, Purpura Haemorrhagica, Equine Viral Arteritis, Equine Infectious Anaemia and Equine Morbillivirus Pneumonia [43].

Treatment, Prevention and Control

Treatment: There is no treatment for AHSV; the disease is managed by supportive treatment. Affected animals should be well nursed, fed and rested for at least four weeks before being returned to light work [9].

Prevention: The most practical approach and primary means to the prevention of viral diseases is vaccination. The all nine AHSV serotypes are distributed throughout South Africa and most part of Sub-Saharan Africa, although they may differ temporally. For this reason, a polyvalent, attenuated vaccine was developed by Onderstepoort Biological Products. There have been a number of different vaccines developed for AHS over the last century. Before 1930, the approach was to confer active immunity by inoculating horses with virulent AHSV and passive immunity by administering anti-AHSV sera. Despite the good protection that these vaccines provided, they occasionally resulted in serious side-effects with some horses dying of encephalitis [3].

Onderstepoort Biological Products manufactures two quadrivalent vaccines containing live, attenuated strains. The seed virus is selected from genetically stable macroplaques from Vero cells. The first vaccine contains serotypes 1, 3 and 4 (AHS1), while the second contains serotypes 2, 6, 7 and 8 (AHS2). The vaccines must be administered at least three weeks apart. Serotypes 5 and 9 are not included in the vaccine as they are cross protected by serotypes 6 and 8, respectively and hence in *vivo* cross protection was so confirmed between serotypes 5 and 8 and serotypes 6 and 9. Repeated vaccinations over time are believed to assist the animal in gaining greater immunity to the serotypes contained in the vaccines [44].

Inactivated vaccines are an alternative to live; attenuated vaccines and advantageous in that they do not contain a potentially dangerous live agent. However, they are expensive to produce and require multiple inoculations. Complete vaccine inactivation may also be difficult [45].

African Horse Sickness Virus (AHSV) VP2, VP7 and NS3 have also been used as vaccine candidates by using a recombinant modified Ankara vaccine (MVA) [46]. In 2011, the VP2-recombinant MVA vaccine (of serotype 4), was also successfully tested, although only in a mouse model [47]. Vaccination of horses with this new vaccine prevented the horses from becoming viraemic after inoculation with live AHS virus and resulted in appropriate levels of circulating antibodies. This vaccine represents a significant advance in the successful prevention of AHS, but it is monovalent and the infecting serotype will have to be determined prior to immunization with the vaccine [48].

In endemic areas where AHS occurs almost every year, that is most parts of Africa south of the Sahara; annual vaccination of horses is a very useful means of control. Although prophylactic immunization against AHS is an efficient method of preventing serious losses, it cannot be relied upon fully to protect horses against infection or disease. Onderstepoort Biological Products (OBP) currently produces a polyvalent vaccine containing attenuated strains prepared in two components one of which is trivalent (serotypes 1, 3 and 4) and the other of which is quadrivalent (serotypes 2, 6, 7 and 8). The administration of these component vaccines three

to four weeks apart will protect horses against the disease. Serotypes 5 and 9 are not included in the vaccines as they are cross protected by serotypes 8 and 6, respectively. Here vector controls are paramount by the use of repellents and insecticides, the elimination of insect breeding areas and the housing of animals in insect-proven buildings at dawn and at dusk when insect activity is maximum [11].

In non-endemic areas, if an outbreak of AHS occurs, much more stringent control measures must be taken which involve quarantine, slaughtering of viraemic animals, vaccination, stabling and controlling Culicoides [49].

Control: As Culicoides species midges are active at dusk and dawn, husbandry measures include housing animals from before dusk to after dawn and preventing access of midges to stables and the application of insecticides to the animals' coats may prevent the midge from biting. These measures aim to limit the amount of time the animals can be exposed to the vector. Even before the vector species was identified, these measures were found to be highly effective at preventing infections as well as are still effective today [50].

Controlling vector population's targets to reduce the number of potential bites that susceptible animals receive. Eradicating the midge population entirely is not possible, nor is it wise, from an ecological perspective point of views. Monitoring the vector population includes altering their habitat, adultciding, larvaciding and the use of repellents. If AHSV should emerge outside AHS endemic areas, the control programme implemented should contain three components: quarantine, vector control and vaccination [51].

Economical Importance of the Disease: Economically it is currently predicted that a widespread outbreak of this disease would have a devastating effect on the horse industry of any country affected. The disease was tremendous economic concern in southern Africa when horses were important for transportation and as draft animals. It is currently an economic concern because of the costs associated with preventive measures in endemic areas, monitoring of the introduction of disease in neighboring unaffected areas and restrictions on the importation of horses from countries in which the disease is endemic. The high case fatality rate and morbidity of the disease in outbreaks are other sources of loss [12].

The essential strict quarantine rules in place for the export of equines from South Africa have a paralyzing effect on the local equine industry and international competitiveness of South African race and sport horses is seriously hindered, with costly and lengthy quarantine periods now in place to export a horse from South Africa to the European Union [10].

The economic importance of AHS in South Africa is tremendous and According to Grewar *et al.* [37] the horseracing industry has made a substantial contribution to the national economy of South Africa and the contribution to the gross domestic product in 2009 was R2.7 billion. The 2011 AHS outbreak in Western Cape, South Africa affected approximately 70 animals and the total laboratory costs amounted to approximately R850 000. South Africa exports around 200 horses per year and the revenue loss due to this outbreak was projected at R20 million per year, with foreign investment losses projected at R200 million per year [37].

African Horse Sickness is a major animal health concern in Namibia and the economic effect of AHS in Namibia affects mainly the pedigree horse industry and exportation. There are about 61 902 horses in Namibia specifically bred for races, sport and the thoroughbred industry. Horses are exported to the Arabian Peninsula, Europe and South Africa and the expected income from exports is estimated at approximately N$ 60000 per horse. In 2011, approximately 1000 horses were lost to AHS, exports and equestrian events being halted by such mortalities [52].

CONCLUSION AND RECOMMENDATIONS

African horse sickness is a serious, often fatal arthropod-borne viral disease of equidae, prevalent to Africa. It is transmitted by arthropod vectors principally Culicoides species-biting midges, with death in horses as high as 95%. This disease has greater risk to regions near to Sub-Saharan Africa is by accidental introduction of wind –born infected midges and such regions must continually be on the alert of the disease. Horses are the most susceptible species of animals while donkeys and mules are considerably less susceptible and generally develop milder disease. The principle of control in endemic areas are vaccination and reduction of exposure of horses to biting insects, where as in non-endemic areas the goal is to prevent introduction of the disease and eradication if it is introduced and the establishment of a protection zone of at least 100km radius around infected premises.

Based on the above conclusion the following recommendations are forwarded:

- When the disease is established the government should implement policy for total prohibition on the movement of equidae.
- Horse owners should be advised to stable their horses from early evening until morning while the Culicoides are most active and to spray horses' house with the suitable insecticides.
- Annual vaccination should be applied for donkeys, mules and horses
- The epidemiological status of the diseases should be studied in domestics as well as wild animals.
- Suspect cases should always be immediately reported to veterinary authorities so that appropriate control measures can be implemented.

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