Review on Nairobi Sheep Disease

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Abstract: Nairobi sheep disease is a noncontiguous, tick-borne, viral infection of sheep and goats characterized by hemorrhagic gastroenteritis and high mortality. It is associated with the Nairobi sheep disease virus; transmitted by an Ixodid ticks, both transtadially and transovarially. The virus causes acute gastroenteritis with mortality rate of 90% in susceptible population. It is originally thought to be endemic only in East Africa, particularly endemic in Kenya; but now a day it is reported from maney places in India and Srilanka. Sera positive for Nairobi sheep disease virus antibody were confirmed in an outbreak in haired sheep in Harar Province of Ethiopia. Nairobi sheep disease can causes a similar clinical picture with other important diseases which are endemic in Ethiopia such as, heart water, Rift Valley fever, anthrax, peste des petits ruminants and coccidiosis. The endemicity of the disease in the neighboring country, Kenya and the similarity of the disease with other highly prevalent diseases in Ethiopia through many conditions such as, clinical sign, mortality and morbidity lead Nairobi sheep disease in to consideration. Because if its once occur it may deteriorate the economy of the country, Ethiopia. Therefore, this paper reviews to familiarize Nairobi sheep disease and organize recent information regarding to epidemiology and management of Nairobi sheep disease. The disease can be diagnosed by clinical and laboratory methods. No effective treatment is available for Nairobi sheep disease. It can be controlled by dipping or spraying of the animal by acaricides.

Key words: Nairobi sheep disease • Sheep

INTRODUCTION

Nairobi sheep disease is a noncontiguous tick transmitted disease of small ruminants, particularly sheep associated with the Nairobi sheep disease virus (NSDV) and characterized by fever, hemorrhagic gastroenteritis, abortion and high mortality [1]. Partly due to climatic change and partly due to change of human habitat occupation, the impact of tick borne virus is increasing. Nairobi sheep disease (NSD) first identified at the beginning of 20th century by Montgomery as disease affecting sheep and goats in part of Kenya [2] and was originally thought to be endemic only East Africa. It is reported in Kenya, Uganda, Tanzania, Somalia, Ethiopia, Botswana, Mozambique and Republic of Congo [3]. However, recent sequence data showed the same virus can also be found in money places in India and Srilanka where it is called Ganjam virus [4]; it is reported in the USA [3].

Montgomery showed that NSD is tick borne gastro-enteritis of sheep and goat in East Africa; was caused by a filterable virus and transmitted most commonly by the brown ear tick. The transmission of the disease by this tick confirmed by Daubney and Hudson who demonstrated also that tick infected in any instars usually transmitted the disease [5].

The virus spread by hard tick (Ixodide ticks) and appears to be dependent on the tick vectors for dissemination with no direct transmission between animals [2]. Animals in endemic areas are usually immune and the virus can persists in the ticks for longer period, more than 2 years in unfed adult [1]. Clinical disease occurs when susceptible animals are moved into enzootic area or when there have been breakdown in tick control measures [6].

As there is no any effective treatment available for NSD, the disease causes severe illness, prognosis in susceptible sheep and goats is poor. Nairobi sheep
Nairobi Sheep Disease

Characteristics of Nairobi Sheep Disease Virus: Nairobi sheep disease virus is the prototype of the tick-borne NSD serogroup, genus Nairovirus; family Bunyaviridae. It is highly pathogenic for sheep and goat [8]. Nairoviruses are small, enveloped RNA viruses in which the genome consists of three segments of single stranded, negative sense RNA, designated Large (L), Medium (M) and Small (S). The S, M and L segments encode, respectively, the nucleocapsid protein (N), at least two envelope glycoprotein (Gn and Gc) and the viral RNA-dependent RNA-polymerase. The carboxyterminal half of this protein contains most of the polymerase motifs, while the amino-terminal part is largely of unknown function [4]. Circular nucleocapsids are formed by base paired terminal nucleotides, not by covalent bond. The envelope glycoprotein is responsible for neutralization and haemagglutination [9].

Nairobi sheep disease virus has been shown to be identical with or very closely related to Ganjam virus, which has been isolated from ticks and mosquitoes in India. The virus of NSD also shows some serological relationships on complement fixation with Dugbe virus of cattle in West Africa and certain strains of Crimean Congo hemorrhagic fever virus in human [6]. The virus replicates in the cytoplasm and bud from Golgi membrane. Because of their segmented genome, closely related the virus can undergo genetic reassortment [10]. Their S RNA genome segments and encoded nucleocapsid proteins were found to be 1590 nucleotides and 482 amino acids in length and differed by only 10 and 3% at nucleotide and amino acid levels, respectively. Genetic and serologic data demonstrate that Ganjam virus is an Asian variant of NSD virus. These viruses were phylogenetically more closely related to Hazara virus than Dugbey virus [8].

Arbovirus is a term used to groups of virus that are transmitted by arthropod vector. Nairovirus are spherical or pleomorphic in shape, 80-120 nm in diameter [10].

Epidemiology

Geographical Distribution: Nairobi sheep disease is found in East and Central Africa. Serological evidence suggests that this disease may also be present in Botswana and Mozambique [11]. The disease has been reported most frequently in Kenya in Kikuyu country between Nairobi and Mount Kenya as well as in Uganda, Tanzania and Somalia [7]. The distribution of NSD in East Africa is closely linked with the distribution of Rhipicephalus appendiculatus and incidence of the disease increases with increased ticks’ activity. Animals in endemic areas tend to be immune but native animal introduced in such area suffer a sever disease. Outbreak may also occur when there is a breakdown of tick control measures [12].

Morbidity and Mortality: Sheep and goats in enzootic regions are often immune to NSD and maternal antibodies protect their off springs from the diseases. Outbreaks are usually seen when animals without immunity are exposed to the virus. Outbreaks can also be seen when ticks population temporarily expand their range during a period of high rainfall or other ecological change. In animals showing clinical sign, the prognosis is generally poor [11]. Mortality rate in the field is as high as 70-90% for indigenous breeds of sheep and 30% for exotic and cross breed. The mortality rates in goats are 80% has been reported [3].

Species Affected: Laboratory and domesticated animals other than sheep and goats are resistant to infection with NSDV([7]. All breeds of sheep and goats are affected with NSD. Goats are less susceptible than sheep and adult sheep and goats affect a more sever diseases than lambs and kids. There are differences in susceptible among different breeds of sheep and goats and unlike other most diseases, the indigenous breeds are susceptible than exotic breeds [1].

Transmission: Nairobi sheep disease is not contagious and is only transmitted by ticks. Transmission by contact does not occur [12]. Experimentally, NSD can be transmitted by the inoculation of infectious blood, serum,
or organ suspensions into susceptible animals [7]. Nairobi sheep disease is transmitted either transovarially or transtadially by the Rhipicephalus appendiculatus (brown ear tick) in which it can survive up to 800 days. Other Rhipicephalus species and Amblyomma vargienatum tick also may transmit the disease. The virus is shed in urine and face, but the disease is not spread by contact [3]. The virus transmitted by any stage of the tick of the brown ear ticks (Figure 1) [10].

Pathogenesis: Following inoculation by an infected ticks the virus replicate in the endothelium, in liver, spleen, lung and other organs of endothelial cell [14]. The NSDV has particular predilection in the vascular endothelial cell in which the cytopathic effect includes endothelial swelling, edema and necrosis. The necrosis of abomasums, small intestine, gall bladder and female genital tract results in congestion, hemorrhagic and catarrhal inflammation and finally desquamation of the necrotic epithelium [12].

Clinical Findings: Clinical sign begin with a steep rise in body temperature (41-42°C) that persists for 1-3 days follows an incubation period of 4-5 days. Sometime, the fever is biphasic. Leucopenia and viraemia usually coincide with the febrile phase of the diseases. Illness is manifest by depression, anorexia, dyspnea, fetid dysentery that cause painful straining and mucopurulent, blood stained nasal discharge. Death may occur in the early febrile viremic phase or follow 2 days after remission of the fever [3].

Superficial prescapular and precrucal lymph node are often palpable. Conjunctivitis may also be seen. Death is also seen later, from hemorrhagic diarrhea and dehydration ([11]. Pregnant animal most frequently aborted. Case mortality rate is 30-90%, but is lower in goat [1]. Ocular discharge, groaning and dullness may be seen [12] the presence of colostral immunity not only protects lambs and kids from early exposure to infection but also allows development of active immunity, enabling survival in tick infested area [3].

Necropsy Findings
Gross Lesions: Hemorrhagic diathesis and consists of hemorrhages on serous surface of the visceral organs and mucosal surface, particularly the abomasums, colon and female genital tract [1]. Petechial and ecchymotic hemorrhage in the mucosa of the cecum and colon frequently appear as longitudinal striation or zebra striping and are sometime the only lesion evident. Subserosal hemorrhage may be seen in the cecum, gallbladder and kidney. Conjunctivitis with dried crust around the nostril is often noted [3]. Extensive ulceration may be present in the cecum abomasums colon and distal ileum and around the iliocecal valve (CFSPH, 2009). Lymph nodes are enlarged and edematous. The uterus and fetal skin are hemorrhagic. Ticks are likely to be found in the body especially in the ear and head ([1]. The spleen may be several times its normal size and engorged with blood [7]. Pulmonary congestion and alveolar edema may be evident. The heart become pale, flaccid and contains unclothd blood. The bone marrow of long bones becomes gelatinous and bright red [12].

Microscopic Lesions: The histopathological lesions outside the gastrointestinal tract include myocardial degeneration, nephritis and necrosis of the gallbladder ([1]; Hyperplasia of lymphoid tissue [3] and degeneration of tubular epithelium, accumulation of hyaline materials and cellular cast in the tubule [12].

Diagnosis
Clinical Diagnosis: Nairobi sheep diseases should be suspected in the sheep or goats with severe gastroenteritis and nasal discharge in or near an enzootic area [11]. This is particularly true if the incidence of illness in sheep high, is low in goat and is absent in cattle and other animals [7]. The presence of attached ticks is supportive [11]. Recent introduction of animals in an endemic area is an important epidemiological factor to be considering [12].

Laboratory Diagnosis: Specimen for laboratory diagnosis includes uncoagulated blood or plasma, mesenteric lymph node, spleen and serum safely to avoid aerosol infection [1]. Little or no virus can be found in the blood after the body temperature falls. Paired acute and convalescent
serum should be submitted for serology. The sample for virus isolation should be kept cool, but freezing may decrease the virus recovery [11].

Laboratory confirmation is necessary for definitive diagnosis [7]. The virus first isolated in tissue culture or in infant mice and the disease can be reproduced in susceptible sheep [1]. Nairobi sheep disease virus can be diagnosis by virus isolation. Nairobi sheep disease virus can be identified directly by in clinical sample with AGID [7]. This test can be carried out in laboratory without tissue culture facilities at field investigation. The test animals of choice are suckling mice inoculated intracerebrally or laboratory raised sheep [11]. Inoculation of cell culture with suspension of infected organs or plasma and subsequent staining of cell culture by the direct fluorescent antibody test or indirect fluorescein conjugated antibody test provide the most reliable means of identifying NSDV. The use of fluorescein conjugated antibody test allows detection of the virus within 24-48 hours [7].

Intracerebral inoculating of suckling mice is an excellent method of isolation NSDV. The brain materials from infected mice can be determined by FAT or CFT [7]. Antibody in infected or recovered animal can be detected by immune diffusion, IFAT, haemagglutination and ELISA [3].

**Differential Diagnosis:** Other diseases that resemble to NSD include heartwater, Rift Valley fever, peste des petits ruminants, salmonellosis, coccidiosis and some toxicity like arsenic poisoning [15]. Differentiation from other viral or rectttesial disease is based on geographical location of the outbreak, species of animal affected, cross-immunity studies, serologic investigation and viral isolation [7]. Hepatic necrosis and death occur in all age; particularly in lambs Rift Valley fever is diagnostic. Exposure to ticks may also results in heart water at the same time with Nairobi sheep disease affecting some animal, if Rhipicephalus and Amblyomma ticks are present in the same area as frequently the case in part of East Africa, but hearwater is usually characterized by nervous manifestation despite the possible occurrence of diarrhea and tenesmus [16]. Demonstration of Cowdria ruminantium in the cytoplasm of endothelial cells of blood vessel or Giems stained lymph node or brain biopsy smear will confirm heartwater. Salmonellosis can be confirmed by isolation and characterization of salmonella species from the intestinal lesions or intestinal content [12].

**Managements of Nairobi Sheep Disease:** No effective treatment is available for Nairobi sheep disease. Fluid therapy is recommended in diarrheic or dysenteric case. Sick animal should be isolated and well nursed [12]. In areas where NSD does not occur, the disease might be eradicated by movement control, quarantine and euthanasia of infected animals together with tick control measure. Sheep and goat can be protected from tick vector by dipping or spraying with acaricides. Dead animal should be buried or incinerated [15]. Vaccination of susceptible risk group such as native animal before being introduced in to endemic area is recommended. Modified live virus or inactivated virus vaccines are available. However, vaccination is very not commonly practiced in endemic area because animal tend to be immune and losses sporadic [12].

**Nairobi Sheep Disease in Ethiopia:** Sera positive for NSDV antibody were confirmed in an outbreak in haired sheep in Harar province of Ethiopia [7]. Nairobi sheep disease may exist in south east of Ethiopia [5].

**Public Health Significance:** Nairobi sheep disease can cause a mild influenenza like disease in human. The clinical sign include fever, headache, back pain abdominal pain, joint pain, nausea and vomiting. The infection may acquired by ticks bite, needle stick injuries or other means. Antibody to NSDV has also found among the general population, laboratory worker and agricultural worker in Uganda, India and SriLanka. Investigators should take precaution to prevent infection [16].

**Economic Significance:** Sheep are an important part of global agricultural economy. Small ruminants are reared mainly for four functions, namely; meat, milk, skin and wool according to order of importance [17]. Nairobi sheep disease can cause death of the animal, increase cost of preventive programme, threat of spread to new geographical area and protein deficiency [18]. The presence of disease can limit trade and export, import of new breed, loss of animal protein for human consumption and affect people livelihood [19].

**CONCULUSION**

Nairobi sheep disease is noncontiguous disease of sheep and goats caused by Nairobi sheep disease virus. The disease transmitted by Rhipicephalus appendiculatus and may be by other species of Rhipicephalus
and Amblyomma varginatum. It is mostly severe in sheep than goats, but does not affect cattle and other animals other than sheep and goat. The disease is severe in sheep and goats and has no treatment; prognosis in susceptible sheep and goats is poor. It develops similar clinical picture with important disease endemic in Ethiopia.

Based on the above conclusion the following recommendations are forward:

- As Nairobi sheep disease is very important sever disease in sheep and goats and is prevalent in neighboring countries, Ethiopia should give emphasis to avoid introduction of this disease in to the country.
- To control Nairobi sheep disease countries where the disease is prevalent should protect their susceptible sheep and goats’ population from the vector by acaricides dipping and spraying, controlling sheep and goats’ movement into endemic areas and burial or incineration of dead sheep.
- As Nairobi sheep disease is prevalent in Kenya and their livestock frequently get access into Ethiopia, the status of the disease should be evaluated by epidemiological study.

REFERENCES