African Trypanosomiasis

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African trypanosomiasis, also known as "Nagana", is an acute or chronic infectious disease of domestic animals characterized by intermittent fever, anemia, edema, and rapid loss of condition, often terminating in death. It is a collective term comprising infection with *Trypanosoma congolense, T. vivax.* The trypanosomes can complete their developmental cycle only in flies of the genus *Glossina,* the tsetse flies, but may be mechanically transmitted by other hemophagous arthropods.

History and Distribution

The Boer settlers in South Africa left accounts of the ravages of "Tsetse Disease" in 1836, and 1857, David Livingstone described the disease and incriminated the tsetse fly. Sir David Bruce (1894) showed that trypanosomes were the causative agents of the disease in ZuZuland. It was he who first publicized the name "Nagana", a Zulu term meaning "to be in low or depressed spirits".

Nearly two-thirds of tropical and subtropical Africa (4.5 million square miles) is infested with the tsetse fly. This area extends from the Sudan in the north to Rhodesia in the south. *Trypanosoma vivax* has been reported from Central and South America and the West Indies. In parts of Africa, Nagana appears to be spreading. It was eradicated from South Africa in 1962.

Etiology

Nagana is caused by single or mixed infection with the following trypanosomes:

1. *Trypanosoma congolense,* the smallest pathogenic trypanosome is monomorphic, has no free flagellum, and is 8-21 um long and 1-27 um broad. Cattle and dogs are highly susceptible, but it can cause disease in all species of domestic animals. Dogs often undergo chronic infection. *Trypanosoma congolense* is the main cause of Nagana in East Africa, but does occur in other regions of Africa.
2. *Trypanosoma brucei* (in the subgenus Trypanozoon) is polymorphic with 3 forms appearing in blood smears: short forms without flagella, long forms with falgella, and intermediate forms. Horses, dogs, pigs, and camels are highly susceptible. It is related to *T. rhodesiense* and *T. gambiense,* the causes of "sleeping sickness" in man. These species both infect domestic animals. *Trypanosoma rhodesiense* has a host range similar to *T. brucei; T. gambiense* infects man and pigs.
3. *Trypanosoma vivax* is monomorphic and large, measuring 18-26 um by 3 um with a distinct flagellum. It is very active in fresh blood smears. Cattle, sheep, and goats are primarily affected; and it is less virulent for cattle than *T. congolense.* Dogs may undergo chronic infections. *Trypanosoma uniforme* is similar to *T. vivax* but smaller and causes a similar disease. *Trypanosoma vivax* readily persists in the absence of tsetse flies, for example in South America. It is the main cause of Nagana in West Africa.

Transmission

Probably all flies of the genus *Glossina* (tsetse flies) are capable of biologic transmission of the disease. These are large flies characterized by a "cleaver-shaped" pattern on their wings. Females extrude 1 live larva every 10-15 days. Tsetse flies are found only in Africa and the southern Arabian peninsula. Mechanical transmission by tsetse and other flies does occur, and there is evidence that Nagana is spreading into areas free of tsetse flies.

Host Range

Cattle, sheep, goats, pigs, horses, camels, and monkeys are susceptible to Nagana. The small West African shorthorned cattle are relatively resistant to trypanosomes. Rats, mice, guinea pigs, and rabbits are useful laboratory species. The large game animals are resistant but may circulate the parasites and serve as reservoirs. Thirty species of wild animals have been found to harbor pathogenic trypanosomes of man and animals.

Clinical Signs

A. *Trypanosoma congolense* is the cause of peracute, acute, or chronic forms of Hagana in cattle and dogs especially. A febrile response occurs 4-10 days after infection and is followed by intermittent febrile episodes. Listlessness, depression, lassitude, and lowering of the head are common signs as are salivation, lacrimation, and nasal discharge. As the disease progresses, one sees loss of condition and hair

color changes from black to metallic brown. The back is often arched and the abdomen tucked up. Accelerated pulse and jugular pulsation occurs and breathing is difficult. Anemia is a prominent sign. The organisms are readily demonstrable in blood smears, especially early in the infection. *Trypanosoma congolense* is a hematic trypanosome. In the more chronic forms of the disease, enlarged lymph nodes are seen and organisms are most readily demonstrated in lymph node smears.

B. *Trypanosoma brucei* caused acute disease in horses and mild or sub-clinical disease in cattle. A febrile response occurs 4-14 days after infection. This is followed by recurrent febrile reactions. The heartbeat and respiration may be accelerated, and loss of condition and weakness is seen while the appetite remains good. Progressive anemia and icterus and edema of the ventral regions, especially the male genitalia, are characteristic. The organisms are not always easily demonstrated in blood smears, and since this is a humoral trypanosome, it is best demonstrated in tissue smears or sections, (e.g. lymph nodes). Infected animals die in a few weeks or several months depending on the virulence of the *T. brucei* strain.

C. *Trypanosoma vivax* causes disease in cattle, sheep, and goats, especially. It is less virulent than *T. congolense* and may cause sub-clinical infections. Horses are also affected, but the disease induced by *T. vivax* is much milder than *T. brucei* infection. The organisms are often observed only in lymph node smears.

Pathological Changes

No pathognomonic changes are seen in Nagana. Anemia, edema, and serous atrophy of fat are commonly seen. Subcutaneous edema is particularly prominent and is usually accompanied by ascites, hydropericardium, and hydrothorax. The liver may be enlarged or atrophic, and edema of lymph nodes if often seen. The spleen may be swollen, normal or atrophic. Necrosis of the kidneys are heart muscle and subserous petechical hemorrhages commonly occur. Gastroenteritis is common, and focal polioencephalomalacia may be seen. The anemic blood changes are anisocytosis, poikilocytosis, polychromasia, and punctate basophilia. Organisms are quite easily demonstrated in blood smears in the early stages of *T. congolense* infection. With *T. congolense* and *T. vivax,* the hematic trypanosomes, anemia is the main lesion; but with *T. brucei,* a humoral parasite, extensive inflammatory, degenerative, and necrotic changes in parenchymatous organs are the major lesions.

Diagnosis

The history, symptoms, and lesions, together with the presence of vectors, allow tentative diagnosis, but confirmation is essential by demonstration of trypanosomes in thick blood or lymph node smears. This is best achieved during the early febrile reaction. A microhematocrit centrifugation procedure is widely used for concentration of tryponosomes prior to making diagnostic smears. In chronic cases, lymph node smears are the most reliable source of trypanosomes. Hematological examination is important. Leucocytosis alternates with leucopenia, and mitotic figures are found in leucocytes. Marked reduction of erythrocytes is seen. Animal inoculation is an important diagnostic tool, with the guinea pig, mouse, rat, rabbit, dog, and cat serving as laboratory animals. Rodent inoculation is not reliable for the diagnosis of *T. vivax* infection. Complement-fixation, agglutination, and agar-gel-diffusion tests are used in diagnosis, and an excellent indirect fluorescent antibody test is available using dried blood samples.

Treatment

Hold animals in the shade and treat them with the appropriate trypanosomal drugs. Some of the older drugs are still quite good, e.g., potassium antimony tartrate, dimidium bromide, and "Antimosan" are good for *T. vivax* and *T. congolense* but not good for *T. brucei.* "Antrycide Prosalt" (quinapyramine) is effective against all 3 and is still widely used. The most widely used drugs today are "Berenil" (diminazine aceturate), "Ethidium" and "Novidium" (homidium salts), and "Samorin" and "Trypamidium" (isometamidium Cl). Drug resistance to all these and other therapeutic compounds has been reported. This may be overcome by using 2 drugs simultaneously.

Prophylaxis and Control

The disease may be controlled by tsetse fly eradication with DDT, BHC, Dieldrin, or other suitable insectivides. Experiments on fertility reduction (sterile male technique) are underway as are studies on tsetse parasites. Bush clearing and game control are effective but ecologically unacceptable control methods. Infection of animals and treatment under controlled conditions are practised but are of questionable value. Experimental vaccines, both live and inactivated, are being tested, but there is no immediate prospect of their widespread use since too many antigenic variants of the trypanosomes exist. The development of trypanotolerant cattle strains is being attempted, but thus far cross-breeding experiments have not produced productive cattle strains with adequate resistance.

Conclusion

Nagana renders vast areas of Africa unsuitable for domestic animals and thus for man. This disease results in loss of the 3 big "M's" sorely needed in Africa, namely meat, milk, and manure. dogs are often chronically infected and could serve as an easy method of importation of trypanosomes into clean countries. *Trypanosoma vivax* does not need tsetse flies to persist and can become

established where tabanids, *Stomoxys, Simulium, Chrysops,* and other biting flies are prevalent. It could be introduced into the United States of America and become established, creating a problem of enormous economic significance.

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Bluetongue and Related Diseases

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Identification

A. *Definition.* Infectious, non-contagious viral diseases of ruminants transmitted by insects and characterized by inflammation and congestion of the mucous membranes, leading to cyanosis, edema, hemorrages and ulceration.

B. *Etiology.* Bluetongue (BT) virus is the type species of the genus *Orbivirus* in the family Reoviridae, which are icosahedral, double-stranded RNA viruses. Orbiviruses are arthropod-borne viruses approximately 50 to 70 nanometers in diameter characterized by extreme lability at low pH and resistance to inactivation by lipid solvents such as ether, chloroform, and deoxycholate.[9,20,22,45,88,975,116,117,118] Temperature sensitivity of BT virus is peculiar as the virus has been found to survive in a preserved defibrinated blood sample for 25 years at room temperature[91] and is very stable at refrigerator temperatures but freezing has a deleterious effect on the virus.

Twenty serotypes of BT virus have been found in the world.[21,44,46,91,104] Serotypes 10, 11, and 13 exist in the U.S. and other parts of the world including Africa,[44] but serotype 17 has only been found in the U.S.[5] Epizootic Hemorrhagic Disease (EHD) virus, an orbivirus related to BT virus, have been found in North America.[5] Two serotypes of EHD or EHD-like virus have also been isolated from gnats in Nigeria, but it is not known if these represent new serotypes.[86] Ibaraki virus was isolated from cattle in Japan in the early 1960's [94] and has been found to be very closely related if not identical to EHDV.[16]

C. *History.* In the late 1800's BT was described as a disease of imported European breeds of sheep in South Africa although it had apparently been recognized since the first importations of sheep in the 1700's.[20,43,47,48] In 1934, BT virus was isolated and identified from cattle in South Africa with a disease described as "Pseudo-Foot-and-Mouth."[8] During the 1940's, BT appeared throughout medeastern Asia in Palestine, [33,60] Syria, and Turkey,[35,120] and in 1943, the most severe epizootic of BT known occurred on the island of Cyprus.[30,109] The disease appeared in Portugal and Spain in the mid 1950's in both sheep and cattle.[63,101] In the United States, a clinical entity called "mycotic stomatitis" in cattle was described between 1889 and 1904 which was identical to the description of BT in cattle in South Africa and elsewhere, and which is now recognized as being caused by BT or EHD virus.[85] BT was described in Texas sheep in 1948 and reported as "Soremuzzle" but it wasn't until 1952 that the virus was isolated and identified in California sheep.[79,80] BT virus was first recovered from U.S. cattle in 1959 from cases clinically diagnosed as "mycotic stomatitis".[14]

Epizootic Hemorrhagic Disease (EHD) virus was first recovered from a white-tailed deer in a major dieoff in New Jersey in 1955.[110] The virus has since been recovered from cattle with clinical disease identical to BT in the U.S.[84] and has been isolated from gnats in Nigeria.[62,86]

Ibaraki virus, an orbivirus closely related to EHD virus, was isolated from a major epizootic in cattle in Japan in 1959.[93,94,95]

Signs

A. *Clinical features.* Bluetongue in U.S. sheep is much less severe than the disease seen in Africa or Mideastern Asia.[20] In the U.S., bluetongue epizootics usually occur in the late summer months and early fall until the first frost in the more temperate areas. In the southern states the disease