# RHODESIENSE SLEEPING SICKNESS: RE-EMERGING AS A PUBLIC HEALTH PROBLEM

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## General Overview

Early last century, Sleeping sickness was perceived by the colonial power by far the most important public health problem in Tanzania and continental wide. Towards the end of the 1950s prevalence of sleeping sickness was believed to be successfully reduced in all endemic foci in Tanzania to less than 0.1%. Towards the end of 1960s government was either lacking in resources or has diverted resources to other pressing health problems. Breakdown of sleeping sickness specialized mobile teams, deterioration of control activities, severe disruption of health facilities, population movement into high-risk areas and change of health policy resulted in sleeping sickness return with a vengeance. Sleeping sickness due to *Trypanosoma brucei rhodesiense* seems to be quiescent and less widespread in Africa but it is very active in Tanzania and Uganda. Very few cases are reported from Zambia and Malawi. Rhodesiense sleeping sickness is returning with a vengeance in Tanzania but does not receive due attention, probably because its impact is so patchy and few cases are reported from few endemic districts.

Sleeping sickness was first recorded in 1922 in Maswa district, south of Lake Victoria (1). It then spread throughout mainland Tanzania, and is then presently endemic in 8 regions namely Arusha, Lindi, Ruvuma, Kagera, Kigoma, Tabora, Mbeya and Rukwa. The annual average for the last decade was 264 cases, but this is highly under estimated, due to difficulty to diagnose and

remoteness of affected area. Although during the past ten years 8 regions were accounted for presence of sleeping sickness, the disease tend to be more concentrated in certain districts e.g. Kasulu and Kibondo in Kigoma Region) whereas some have hardly reported any cases in this decade. It is noted that during the past ten years about 90% of the sleeping sickness cases were recorded from Kasulu and Kibondo districts in Kigoma region. It is true that these figures are relatively small compared to other tropical diseases affecting Tanzanians. African trypanosomiasis, without intervention, has the potential to develop into epidemics and this characteristic makes it a major public health problem.

African trypanosomiasis, in addition to infecting man also infects domestic animals. It is one of the primary factors in limiting the production of domestic farm animals in Tanzania and Africa at large. Therefore in addition to causing severe human morbidity and mortality, it has limited agricultural development. Infection of domestic animals has had human public health consequences as it has critically reduced the amount of protein in the African's diet (2).

**The Disease**

Sleeping Sickness is caused by protozoan haemoflagellates of the genus *Trypanosoma* and is transmitted by tsetse flies (*Glossina*). The disease occurs in two forms, the one referred to as West African trypanosomiasis is caused by *Trypanosoma.b. gambiense* and is more chronic in nature; it can last up to 6 years. It is predominantly man to man infection, however some evidence of animal reservoir has been found. Another form referred to as East African form caused by *T.b. rhodesiense,* it is acute and lasts only for weeks and rarely can last more than 9 months. It has variety of vertebrate reservoir hosts including man's domestic livestock, moreover, the existence of reservoir influences the epidemiology of the infection. Tanzania is affected by only acute form of the disease caused by *T.b. rhodesiense.*

The epidemiology of sleeping sickness is complex and transmission cycles are subject to interactions between humans, tsetse flies and trypanosomes and significantly in T.b rhodesiense sleeping sickness, domestic and wild animals. Game-fly-human cycle is typical in rhodesiense sleeping sickness, during epidemic *T.b.rhodesiense*, however, man-fly-man or animal-fly human cycles predominate.

**Diagnosis**

Except in advanced stages, the clinical features of African human trypanosomiasis are not diagnostic of the disease. Diagnosis depends upon demonstration of parasites in blood, lymph nodes aspirates; followed by lumbar puncture to determine whether there is involvement of central nervous system (CNS). Sometimes because of the periodicity of the parasitaemia, the number of parasites in blood my be very low at times, multiple sampling and concentration techniques are often necessary to be able to detect the parasites. *Trypanosoma b. gambiense* infection can be particularly difficult to diagnose due to scarcity and infrequent appearance of parasites in the blood.

A number of tools exist for the diagnosis of sleeping sickness patients. Thick or thin blood film is still good method for detecting parasites. The use of concentration techniques including Haematocrit Centrifugation Technique (HCT) or Miniature Anion Centrifugation Technique (MAECT) or Quantitative Buffy Coat (QBC) can increase the sensitivity of trypanosome detection by several orders of magnitude (3). All these concentration techniques have been adapted for use in field surveys. Immunodiagnostic methods are also in use for detection of latent infections and for mass surveys or other epidemiological investigations. Card Indirect Agglutination Test for Trypanosomiasis (CIATT) is one of the serological tests that detects antigens and therefore, current infection. It is applicable in both types of sleeping sickness. Card Agglutination Test for Trypanosomiasis (CATT) and antibody detection test is specific for gambiense sleeping sickness.

The criteria used to determine a CNS involvement as follows: white blood cell counts above 5 cells/$mm^3$ or the demonstration of trypanosomes or protein levels above normal (>37mg/100ml) of cerebrospinal fluid.

**Treatment**

The combination of active case detection and successful treatment is the cornerstone of prevention and control sleeping sickness. But implementation of this strategy is difficulty; very few donors are funding sleeping sickness control programmes and drugs are quite toxic, irregularly produced and too expensive. Moreover, drug resistance has recently being reported from several endemic countries (4).

With one exception, no new drug has become available for treatment of either rhodesiense or gambiense sleeping sickness in over 40 years. Suramin developed in 1922 is used to treat early stage of the *T.b. rhodesiense* infection that has not involved the central nervous system. Melarsoprol (MelB) developed in 1948, is the drug of choice for the late stage treatment of both gambiense and rhodesiense sleeping sickness in which cerebral manifestations occur. Pentamidine, developed in 1937 is used for the early stage of gambiense sleeping sickness. Only recently a new drug become available;

it is Eflornithine developed in 1990, which is unfortunately efficacious in treatment of only gambiense sleeping sickness (5,6). All these drugs have adverse side effects, melarsoprol may cause reactive encephalopathy in 5-10% of patients with a fatal outcome in 1-5% (7).

It is mandatory to systematically follow-up all treated patients to ascertain that they have cured. It is recommended that patients are seen every 6 months over a period of two years and all procedures of parasitological examination and lumbar puncture should be carried. However, due to painful lumbar puncture, most patients resent coming back for follow-up once they feel better

**Surveillance and Control**

Sleeping sickness in Tanzania can be controlled, but control requires continuous surveillance of the population at risk for infection and treatment of all infected humans found. Regular medical surveillance, which involves case detection and early treatment, and vector control, is the backbone of the strategy for the effective control of sleeping sickness. With available tools, control should be a continuous process as it has already been shown that where control efforts are interrupted, soon or later there will be resurgence of the disease. One example of this phenomenon is re-emerging of sleeping sickness in Serengeti National Park, which was silent focus for more than a decade. The report of tourists being infected by *T.b. rhodesiense* sleeping sickness in National Park it does mean that may be the disease is rising to an epidemic level among the local population.

**Social and Economic Impact of Sleeping Sickness**

Social and economic impact of sleeping sickness is often underestimated. Agriculture -based economy and workers in rural areas are at risk contracting the disease and consequently labour force is reduced. In social life from family levels, mental confusion, personality and behaviour changes which are characteristics of late stage of the disease in which central nervous system is involved, may lead to divorce and break up in homes. Some times these people become mentally disturbed, suicidal and violent to the community. It has been reported that past history of sleeping sickness in children had influence on physical growth and attainment of sexual maturity (8). There are other impacts such as population movements, which may render problem in case detection, case reporting and control of the disease. Studies in other endemic countries like Uganda have demonstrated that sleeping sickness had an adverse impact on the functioning of households which include increased poverty, lack of basic food security, children's education and problems to women who may be stigmatised and rejected by their spouses.

**Conclusion**

The demonstration of the parasite is mandatory before treatment of sleeping sickness patient; this is due to toxicity of the drugs. Due to the costs of staffing and equipping sleeping sickness treatment facilities there are few health centres and dispensaries equipped for treating the disease. All endemic districts in Tanzania have poor coverage of sleeping sickness fixed post health units for diagnosis and treatment. Therefore most patients travel long distances to receive treatment. The problem of poor accessibility to diagnosis and subsequently treatment may be alleviated by the use of mobile teams

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