AFRICAN TRYPANOSOMIASIS (SLEEPING SICKNESS)

Dr. Muhammad Zakria

Abstract: It is caused by trypanosomes conveyed to human by bites of infected tsetse flies and unique to sub-saharan African. It is caused by protozoa of the species trypanosomes brucei. Clinical features occur in two stages including fever, headache, arthralgias, itching, lymphadenopathy, day time sleep episodes and night time period of wakefulness, confusion, hemipariesis. Trypanosomiasis should be considered in any febrile patient from endemic area. It is diagnosed by thick and thin blood fill. Treatment varies according to stage of disease including I/V or I/M pentamidine or I/V suramin. For second stage a regimen involving combination of nifurtimox and eflornithine.

Key words: Trypanosomiasis, Encephalopathy, Parasitemia, thick and thin blood film, Lymphadenopathy.

African trypanosomiasis, is caused by trypanosomes conveyed to humans by the bites of infected tsetse flies, and is unique to sub-Sahara Africa.[1] It is caused by protozoa of the species Trypanosoma brucei. There are two types that infect humans, Trypanosoma brucei gambiense (TbG) and Trypanosoma brucei rhodesiense (TbR). TbG causes over 98% of reported cases.[2]

The disease has been recorded as occurring in 37 countries, all in sub-Saharan Africa. It occurs regularly in southeast Uganda and western Kenya, and killed more than 48,000 Africans in 2008.[3] In 2015 it caused around 3,500 deaths, down from 34,000 in 1990.[4,5] More than 80% of these cases are in the Democratic Republic of the Congo. Three major outbreaks have occurred in recent history: one from 1896 to 1906 primarily in Uganda and the Congo Basin and two in 1920 and 1970 in several African countries.[2] The number of people being affected by the disease has declined. At this rate, sleeping sickness elimination is a possibility. The World Health Organization plans to eradicate sleeping sickness by the year 2020.[6]

Signs and symptoms

African trypanosomiasis symptoms occur in two stages.

The first stage (hematolymphatic phase) 1, is characterized by fever, headaches, joint pains, and itching. Fever is intermittent, with attacks lasting from a day to a week, separated by intervals of a few days to a month or longer. Invasion of the circulatory and lymphatic systems by the parasites is associated with severe swelling of regional lymph nodes, often to tremendous sizes

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(Winterbottom's sign), the tell-tale swollen lymph nodes along the back of the neck, may appear.[7] Occasionally, a chancre (red sore) will develop at the location of the tsetse fly bite. If left untreated, the disease overcomes the host's defenses and can cause more extensive damage, broadening symptoms to include anemia, endocrine, cardiac, and kidney dysfunctions.

The second phase (encephalitic phase) 1, begins when the parasite invades the central nervous system by passing through the blood–brain barrier. Disruption of the sleep cycle is a leading symptom of this stage and is the one that gave the disease the name 'sleeping sickness.' Infected individuals experience a disorganized and fragmented 24-hour rhythm of the sleep-wake cycle, resulting in daytime sleep episodes and nighttime periods of wakefulness.[7]

Other neurological symptoms include confusion, tremor, general muscle weakness, hemiparesis, and paralysis of a limb. Parkinson-like movements might arise due to non-specific movement disorders and speech disorders. Individuals may also exhibit psychiatric symptoms such as irritability, psychotic reactions, aggressive behaviour, or apathy which can sometimes dominate the clinical diagnosis.[8] Without treatment, the disease is invariably fatal, with progressive mental deterioration leading to coma, systemic organ failure, and death. An untreated infection with T. b. rhodesiense will cause death within months[9] whereas an untreated infection with T. b. gambiense will cause death after several years.[10] Damage caused in the neurological phase is irreversible.[3]

Investigations

Trypanosomiasis should be considered in any febrile patient from an endemic area. In rhodesiense infections, thick and thin blood films, stained as for the detection of malaria, will reveal trypanosomes. The trypanosomes may be seen in the blood or from puncture of the primary lesion in the earliest stages of gambiense infections, but it is usually easier to demonstrate them by aspiration of a lymph node. Concentration methods include buffy coat microscopy and miniature anion exchange chromatography.

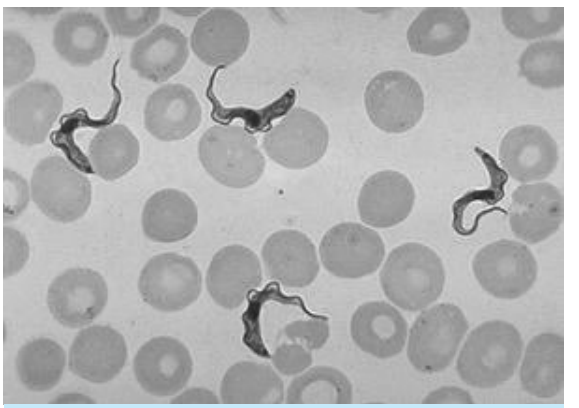

Fig 1: Trypanosoma forms in a blood smear

Due to the cyclical nature of parasitaemia, the diagnosis is often made by demonstration of antibodies using a simple, rapid screening card agglutination trypanosomiasis test (CATT), followed by parasitological confirmation. If the CNS is affected, the cell count (> 20 × 109 leucocytes per litre) and protein content of the CSF are increased and the glucose is diminished. A very high level of serum IgM or the presence of IgM in the CSF is suggestive of trypanosomiasis. Recognition of CNS involvement is critical, as failure to treat it might be fatal.[1]

Treatment

First stage

The current treatment for first-stage disease is intravenous or intramuscular pentamidine for T. b. gambiense or intravenous suramin for T. b. rhodesiense.[1]

Second stage

For T. b. gambiense a regimen involving the combination of nifurtimox and eflornithine, nifurtimox-eflornithine combination treatment (NECT), or eflornithine alone appear to be more effective and result in fewer side effecfs. These treatments may replace melarsoprol when available[11] with the combination being first line.[12] NECT has the benefit of requiring less injections of eflornithine.[11]

Intravenous melarsoprol was previously the standard treatment for second-stage (neurological phase) disease and is effective for both types. Melarsoprol is the only treatment for second stage T. b. rhodesiense; however, it causes death in 5% of people who take it. Resistance to melarsoprol can occur.[12]

Prognosis

If untreated, T. b. gambiense almost always results in death, with only a few individuals shown in a long-term 15 year follow-up to have survived after refusing treatment. T. b. rhodesiense, being a more acute and severe form of the disease, is consistently fatal if not treated.[12] Disease progression greatly varies depending on disease form. For individuals which are infected by T. b. gambiense, which accounts for 98% of all of the reported cases, a person can be infected for months or even years without signs or symptoms until the advanced disease stage, where it is too late to be treated successfully.

For individuals affected by T. b. rhodesiense, which accounts for 2% of all reported cases, symptoms appear within weeks or months of the infection. Disease progression is rapid and invades the central nervous system, causing death within a short amount of time.[13]

Prevention

Currently there are few medically related prevention options for African Trypanosomiasis (i.e. no vaccine exists for immunity). Although the risk of infection from a tsetse fly bite is minor (estimated at less than 0.1%), the use of insect repellants, wearing long-sleeved clothing, avoiding tsetse-dense areas, implementing bush clearance methods and wild game culling are the best options to avoid infection available for local residents of affected areas.[14]

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