Trypanosomiasis

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Key Points
➢ Trypanosomiasis is infectious disease. ➢ Causative agent is protozoan parasite. ➢ Trypanosomiasis can cause behavioral disturbances

African trypanosomiasis is an infectious disease of humans and animals of similar aetiology and epidemiology. The causative agents of the disease are protozoan parasites of the genus *Trypanosoma* that live and multiply extracellularly in blood and tissue fluids of their mammalian hosts and are transmitted by the bite of infected tsetse flies (*Glossina* sp.). African animal trypanosomiasis or nagana disease is caused by *T. congolense*, *T. vivax* and *T. brucei* spp. In wild animals, these parasites cause relatively mild infections while in domestic animals they cause a severe, often fatal disease. All domestic animals can be affected by nagana and the symptoms are fever, listlessness, emaciation, hair loss, discharge from the eyes, oedema, anaemia, and paralysis.[1]

Figure:1 *Tsetse fly (glossina specie)*[1]

Effects

There are various effects of trypanosomiasis:

Severe headaches. Irritability. Extreme fatigue. Fever. Swollen lymph nodes. The trypanosome can cross the placenta and infect the fetus.

Stages of infection:

In the first stage, the trypanosomes multiply in subcutaneous tissues, blood and lymph. This is also called haemo-lymphatic stage, which entails bouts of fever, headaches, enlarged lymph nodes, joint pains and itching. In the second stage the parasites cross the blood-brain barrier to infect the central nervous system. This is known as the neurological or meningo-encephalic stage. Changes of behaviour, confusion, sensory disturbances and poor coordination. Disturbance of the sleep cycle, which gives the disease its name, is an important feature.[2]

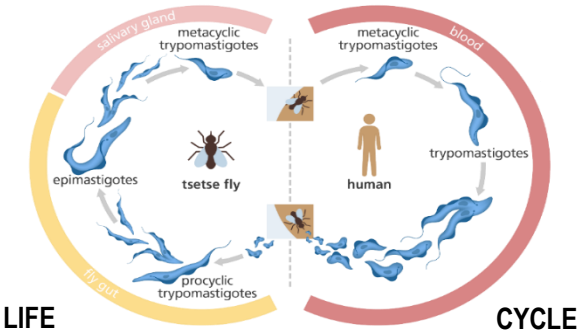

Figure:2 *Life cycle of tsetse fly*[2]

The life cycle of *Trypanosoma cruzi* involves two intermediate hosts the invertebrate vector (triatomine insects) and the vertebrate host (humans) and has

three developmental stages namely, trypomastigotes, amastigotes and epimastigotes.

The cycle started with the insect sucking of bloodstream trypomastigotes of the infected vertebrates. Most of the ingested trypomastigotes are broken down in the stomach of the insect while the surviving trypomastigotes transform into either spheromastigotes (spherical stage) or into epimastigote stage few days later

In the mammalian bloodstream, the trypomastigotes have different forms:

- Long slender form
- Short, stumpy form
- Intermediate form between the two.

The short stumpy form is pre-adapted for survival in the tsetse fly so is the form that differentiates into the next stage of the life cycle when the fly takes a blood meal from an infected mammalian host.

Once inside the midgut of the tsetse fly, the trypomastigotes transform into procyclic trypomastigotes, which multiply in the gut.

Finally, the epimastigotes transform into short, infective metacyclic trypomastigotes and detach from the wall of the salivary gland, ready to be injected into a new host when the fly takes another blood meal.[3]

Treatment

Anyone diagnosed with African Trypanosomiasis should be treated, with specific drug and treatment course depending on type of infection (*T. b. gambiense* or *T. b. rhodesiense*) and disease stage (i.e., presence or absence of central nervous system involvement). Pentamidine, the recommended drug for first stage *T. b. gambiense* infection, is available in the United States. The other drugs (suramin, melarsoprol, eflornithine, and nifurtimox when used in combination with eflornithine) used to treat African trypanosomiasis are not commercially available in the United States but can be obtained from CDC.

There is no test of cure for African trypanosomiasis. After treatment, patients should be closely followed for 24 months and monitored for relapse. Recurrence of symptoms will require examination of body fluids, including CSF, to detect the presence of trypanosomes.

Only four drugs are available for the chemotherapy of human African trypanosomiasis or sleeping sickness; **Suramin, pentamidine, melarsoprol and eflornithine**.

- **Pentamidine** – used to treat first stage *Trypanosoma brucei gambiense*, it generally has no adverse side effects.
- **Suramin** – used to treat first stage *Trypanosoma brucei rhodesiense*, it causes some side effects such as urinary tract infections and allergic reactions.
- **Melarsoprol** – used to treat second stage *Trypanosoma brucei rhodesiense*. It is derived from arsenic and has many unwanted side effects. In extreme cases, it has been seen to cause reactive encephalopathy (disorder of the brain) which can be fatal. There has also been a spread of resistance to melarsoprol in trypanosomes found in central Africa.
- **Eflornithine** – originally developed as an anti-cancer drug, it is used to treat second stage *Trypanosoma brucei gambiense*. It is less toxic than melarsoprol but can cause vomiting, diarrhoea and anaemia.[4]

Prevention

Preventive measures include;

- Wearing long-sleeved shirts and long trousers to limit the amount of exposed skin
- Wearing clothes in neutral colours as tsetse flies are attracted to bright colours

- Avoiding bushes where the tsetse fly rest during the hottest part of the day as they will bite if disturbed
- Using insect repellent.

Control of African trypanosomiasis currently centres around two key strategies:

Population screening to ensure early treatment of infected people and help reduce the number of people carrying trypanosomiasis. Fewer people carrying the parasite means the tsetse fly is less likely to become infected when they take a blood meal and less likely to pass the parasite on to another individual at their next blood meal.

Reducing transmission via the tsetse fly by using insecticides to kill the insect and deploying fly traps and/or screens in homes to reduce the number of flies entering.

Figure:3 *Preventive measures of African sleeping sickness*[4]

References:

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