Sleeping sickness

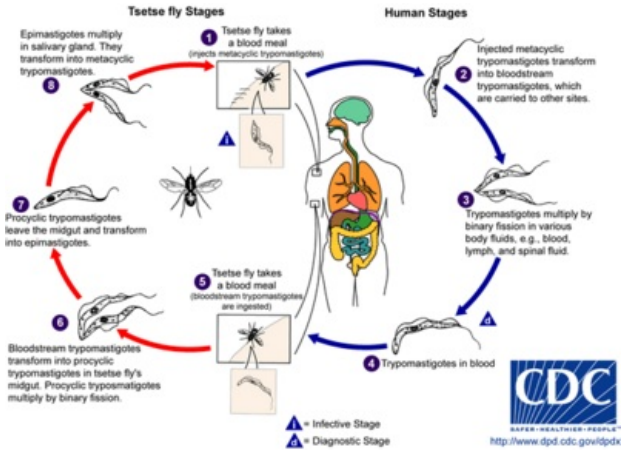
Template:Infobox - onemocnění **Sleeping sickness** or **african trypanosomiasis** is a disease caused by flagella of the genus *Trypanosoma*. It can be caused by both *T. gambiense (brucei)* and *T. rhodesiense*, but the course of the disease then varies considerably. Template:Deatils

T. gambiense vs. T. rhodesiense

	T. gambiense	**T. rhodesiense**
Course:	chronic (with CNS involvement)	acute
Onset:	slow	quick
Death:	> 4 years	< 9 months
V:	months or years	days

Patogenesis

1. The tsetse fly **stings** → trypanosomes enter **the lymph**;
2. They are carried by lymph into the nearest **lymph node**, which drains the relevant part of the body;;
3. from there they enter the thoracic duct and **into the bloodstream**;
4. they can cross the blood-brain barrier (HE) into the **cerebrospinal fluid**.



Clinical piscture

Two phases: **1. phase**:

- **skin lesions (skin scab)**: local **skin inflammation** occurs at the site of the sting → skin **edema** develops → **ulcerates** the site over time → heals with a **scar** = disappears spontaneously.
- **Winterbottom symptome**:
  - it is a swelling of the lymph nodes in the neck area;
  - it develops at an early stage when trypamosomes have not yet crossed the blood-brain barrier (so they are only in the blood and lymph);
  - typical **fevers** (in waves), **headaches and joint pain**, **malaise** , **anemia**, etc.

2. phase:

- **edema** of a number of organs associated with **CNS ingection**;
- at this stage, the trypanosomes have already crossed the **HE barrier**.

Periodic fevers

- Trypanosomes have a mantle on the surface composed of many copies of a single glycoprotein (GP) = almost the entire trypanosome population in the body is therefore **homogeneous**;
- furthermore, trypanosomes have at least **100 genes**, in their genetic makeup that encode different glycoproteins = in certain circumstances, they may therefore **"exchange"** their glycoprotein mantle for another;
- at the moment when the trypanosome in the organism multiplies sufficiently (5-7 days), the human organism starts to respond with **IgM** → production → trypanosomes are subsequently **lysed** by complement;
- however, out of the whole lysed population having a mantle from one specific GP (eg GP A), there is about **1 %** rypanosome with a different GP mantle (eg GP B) → the population with GP B starts **to multiply again** → the organism responds to them again by producing antibodies against **GP B** → however, there is still a certain percentage of trypanosomes that have GP C on their surface, and so it goes on and on;
- whenever the majority of the parasite,population is lysed ***a fever*** occurs.

Diagnostics

- **Microscopy = direct proof:**
  - we take material from ulcers, blood, cerebrospinal fluid;
  - **must not be taken at a time of fever** (trypanosomes are lysed).

Therapy

Untreated ends in death *1. phase:* **suramin**; *2. phase:* **melarsoprol** – an arsenic compound passing through the blood-brain barrier. It is a toxic but effective antiparasitic. It is currently hardly used (in 5–20% of fatal encephalopathies). It was colloquially referred to by doctors as "arsenic in antifreeze" (dissolved in propylene glycol) and among patients as "fire in the veins" (painful application).).[1][2]

Links

Related articles

- Chagas disease

References

1. https://www.lekari-bez-hranic.cz/spava-nemoc,
2. https://www.newscientist.com/article/mg18524821.800-curing-diseases-modern-medicine-has-left-behind/

Used literature

- BEDNÁŘ, M, et al. *Lékařská mikrobiologie.* 1. edition. Marvil, s. r. o., 1996. ISBN 80-238-0297-6.
- RNDr. Eva Nohýnková, Ph.D. [přenáška z parazitologie]