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Microbiology

Doctor 2017 | Medicine | JU

Sheet

Slides

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Protozoal Infections

Recap:

Parasitic kingdom includes three phyla: 1- *Protozoa* 2- *Helminths* 3- *Arthropods*

Protozoa can be classified according to the: 1- Organ of locomotion. 2- Habitat.

A. According to the organ of locomotion they are classified into 4 classes:

1. Class *sarcodina* or Rhizopoda (*Amoebae*).
2. Class *Ciliata* (*Ciliates*).
3. Class *Zoomastigophora* (*Flagellates*)
4. Class *Sporozoa* (*Plasmodia & Coccidia*)

B. According to the habitat they are classified into:

- 1- *Intestinal protozoa*, which are further divided into
 - a) *Amoeba* (*Entamoeba Histolytica*)
 - b) *Ciliates* (*Balantidium Coli*)
 - c) *Flagellates* (*Giardia lamblia*)
 - d) *Coccidia* (*Cryptosporidium*)
- 2- *Blood protozoa*
- 3- *Tissue protozoa*
- 4- *Urogenital protozoa*

**Last lecture we already talked about Amoeba and its characteristics.*

**Today we will complete our discussion about other types of protozoa that are involved in intestinal infections.*

GIARDIA LAMBLIA (INTESTINAL FLAGELLATE)

also referred to as *Giardia duodenalis* or *Giardia intestinalis*

- 1) It is the causative agent of ***giardiasis***, popularly known as **beaver fever**.
- 2) The only common pathogenic protozoan found in the proximal bowel of humans.
(duodenum+ jejunum = Proximal Bowel)
- 3) Distributed worldwide.

4) Risk Factors associated with **Giardia** infections (Factors that Contribute to the development of giardia) include:

- a) poor sanitary conditions.
- b) consumption of contaminated water.

Morphological forms of Giardia:

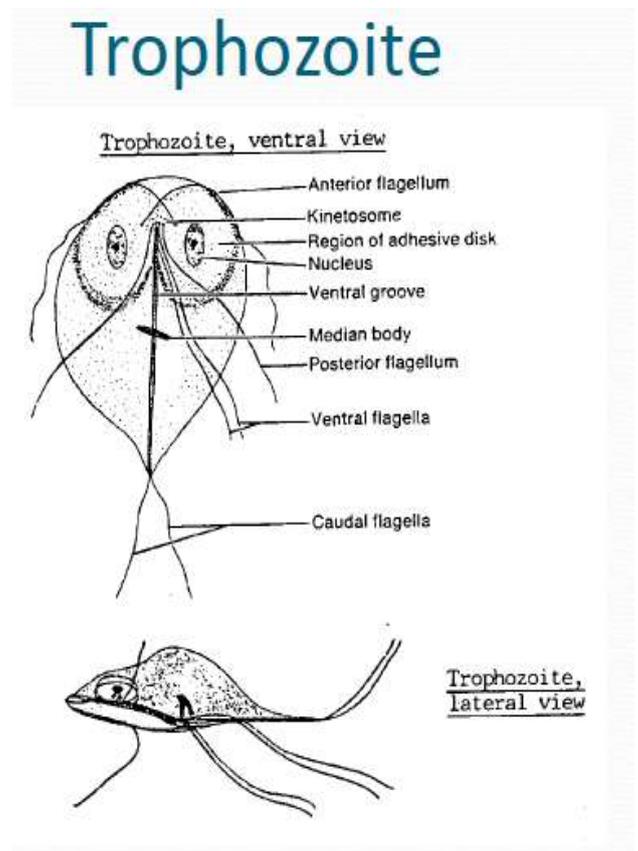
Giardia lamblia exists in two forms:

1) An active form, called a trophozoite:

This form attaches to the lining of the small intestine with a “Sucker”, it is also responsible for the signs and symptoms of giardiasis.

Its Features:

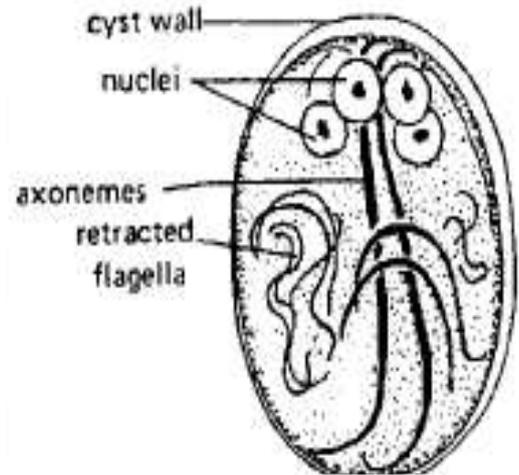
1. heart-shaped organism.
2. Has 4 pairs of flagella. (2 anterior ,2 Posterior, 2 ventral and 2 caudal).
3. Has 2 nuclei with prominent central karyosome*, giving it the appearance of a face.
4. Composed of 2 axostyles* (*Sheets of Microtubules, involved in motility and provides support for the cell*).
5. Approximately 15 µm in length.
6. The swaying or dancing motion of the trophozoites in fresh preparations is unmistakable (Can be seen clearly)



2) An inactive form called a cyst:

Its Features:

1. It is ellipsoid, thick-walled, highly resistant (it's resistant to chlorinated water of swimming pools, meaning that it can even contaminate swimming pool water).
2. 8–14 μm in length.
3. If the Cyst is immature, it contains two nuclei. On the other hand, if it is mature, it contains four nuclei.
4. Found in the stool, often in enormous numbers.
5. Cysts can survive in water for up to 3 months.



***Karyosome**: Concentrated mass of chromatin found in the cell's nucleus.

***Axostyle**: an axial rod composed of microtubule sheets that arises from many parasitic flagellates and participates in Motile and Supportive Functions.

Note: Trophozoites Contain A large concave sucking disk on the ventral surface which helps the organism adhere to intestinal villi. As the parasites pass into the colon, they typically encyst (Become enclosed in a cyst), and the cysts are passed in the stool.

CYST	TROPHOZOITE
Transmission state of protozoans	Disease-causing state of protozoans
Non-motile	motile
Infective	Non-infective
Non-reproductive structures	Reproduce through binary fission
Resistance to water and dessication	Not Resistance structures
Can survive outside the host	Cannot survive outside the host

Visit www.pediaa.com

*This table contains a summary of the differences between the trophozoite and the cyst, make sure to memorize the table since it contains additional differences.

Pathology and Pathogenesis:

- Giardia lamblia is usually a weak pathogenic organism when it comes to humans.
- Cysts may be found in large numbers in the stools of entirely asymptomatic persons (where the patient is a carrier for the disease but experiences no symptoms).
- In some people, however, **large numbers** of parasites attached to the bowel wall may cause irritation and low-grade inflammation of the duodenal or jejunal mucosa, with consequent acute or chronic diarrhea, associated with epithelial cell damage (crypt hypertrophy, villous atrophy or flattening).

Note: Giardia trophozoites only **attach and colonize** on the intestinal mucosa, without **invading** the epithelium, Also, the small intestinal morphology may appear normal using a light microscope, despite being infected.

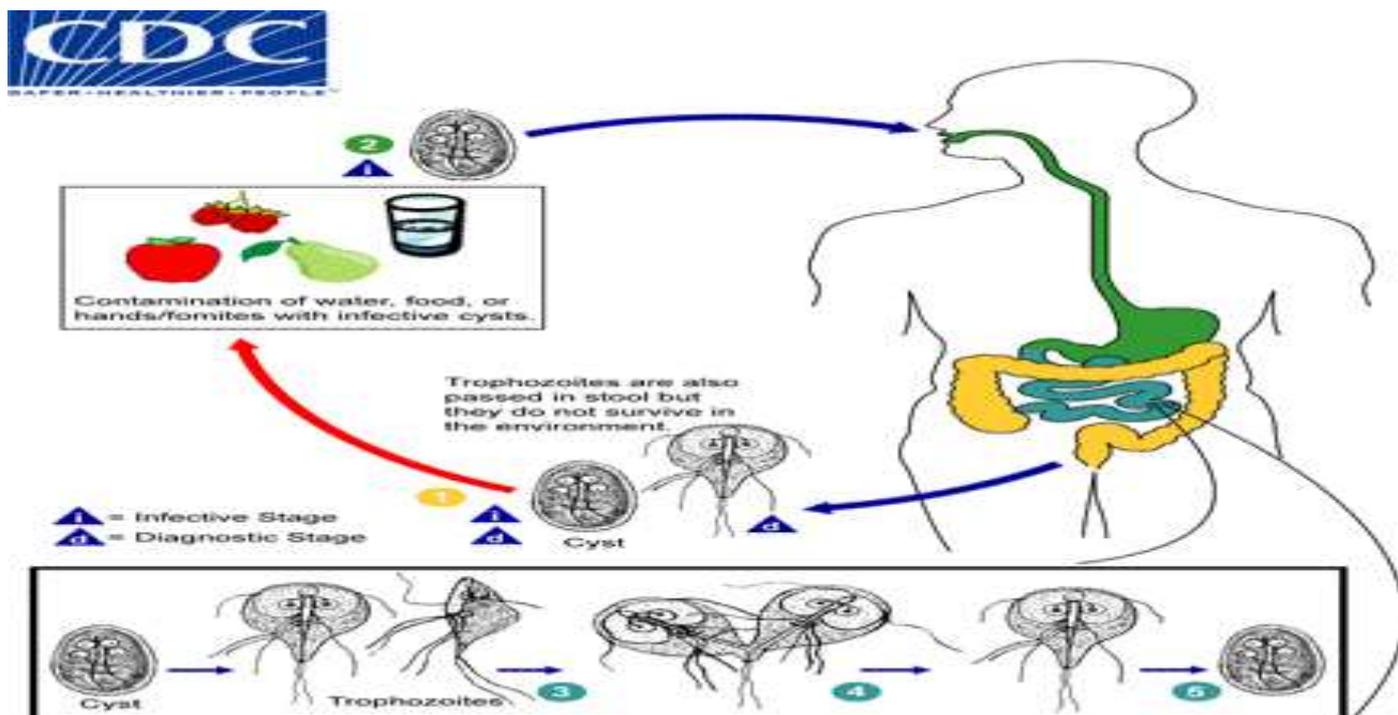
Transmission:

- Giardiasis is transmitted via the fecal-oral route with the ingestion of cysts.
- Primary routes are **personal contact** and **contaminated water and food**, which might occur in day care centers, refugee camps, and institutions, or during **oral-anal sex**, people who **have contact with animals who carry the disease**, like beavers (hence its nickname), and **men who have sex with men**.
- **Epidemic outbreaks** (sudden outbreaks), have been reported at resorts, due to overloading the sewage facilities or contamination of the water supply.
- Symptoms usually begin 1 to 3 weeks after exposure.

The Life Cycle of Giardia lamblia:

- 1- Ingestion of the **cyst** (the infective form or stage).
- 2- A process known as **excystation** takes place to produce trophozoites. (each cyst produces two trophozoites)
- 3- Trophozoites multiply by **binary fission** in the colon, and can be **free** or **attached to the mucosa** by a ventral sucking disk.
- 4- **Encystation** then occurs as the parasites transit toward the colon. The cyst is the stage found most **commonly in nondiarrheal feces**.

- 5- Because the **cysts** are infectious when passed in the stool or shortly afterward, person-to-person transmission is possible (*for example, when someone comes into contact with fecal matter (poop) from an infected person (especially a child in diapers).*)



Further explanation: The Giardia *enters the body as a cyst*, when it reaches the *small intestines*, *excystation takes place* and the Giardia can reproduce, which causes its symptoms, once it gets to the *colon*, *the environment becomes inappropriate to reproduce*, so the Giardia becomes *encysted again*, symptoms like diarrhea disappear, but the **cysts** are still found in samples of the non-diarrheal feces.

Clinical Aspects:

- The disease varies from *asymptomatic carriage* to *severe diarrhea and malabsorption*.
- Subclinical infections (*asymptomatic infections*) is common in *endemic areas* (where the infection occurs constantly at a certain rate in a specific geographic area)
- In acute outbreaks, at various times during the course of the infection, Stools may be *watery, semisolid, greasy* (difficult to flush), *bulky*, and *foul smelling* (having an extremely unpleasant smell) and does not contain mucous or blood.

Methods for diagnosis of Giardia:

- 1- Immunologic enzyme-linked *immunoassays(EIAs)* and *immunofluorescence microscopy* that detect Giardia antigen in the stool are now commercially available. (easy and quick to use)
- 2- A *trichrome stain* of preserved stool is another method used to detect giardia.

- The diagnosis of giardiasis is made by finding:
 - a) **The cyst** in formed stool (non-diarrheal stool).
 - b) **The trophozoite** in diarrheal stools, duodenal secretions, or jejunal biopsy specimens.

Note: Cysts usually need *saline wet mount preparation*, While Trophozoites need a *permanent slide preparation*.

Treatment:

- 1- **Quinacrine** and **Metronidazole** are given for 5 to 7 days, they have 70%-95% efficacy in treatment (very effective) and are preferred for *patients capable of ingesting tablets*.
- 2- **Tinidazole**, Given as a single-dose therapy.
- 3- **Furazolidone** found usually as a suspension and is used by pediatricians.
- 4- During pregnancy, **paromomycin** is the preferred treatment drug.

CRYPTOSPORIDIUM (INTESTINAL SPOROZOA)

General characteristics:

- They appear as *small spherical structures* arranged along the line of epithelial cells.
- Their cell walls are unique as they exhibit **acid-fast staining** (they react positively to acid-fast staining).
- *Cryptosporidium complete its life cycle within a host, including its asexual and sexual reproductive cycles.*
- Cryptosporidium inhabits the *brush border of mucosal epithelial cells of the gastrointestinal tract*, especially the surface of villi of the lower small bowel.
- The Infective stage of Cryptosporidium is called **Sporulated oocyst**.

Pathology and Pathogenesis:

- Usually asymptomatic and transient.
- Cryptosporidium species, typically **C hominis**, **C parvum** can infect the intestine in immunocompromised persons (e.g., those with AIDS) and cause severe, intractable diarrhea (*hard to control or deal with*).

- They have long been known as parasites of *rodents*, *fowl* (eggs/flesh of a bird), *rhesus monkeys*, *cattle*, and *other herbivores* (animals that feed on plants).
- They used to be an *unrecognized cause of self-limited, mild gastroenteritis* and *diarrhea* in humans.

Transmission:

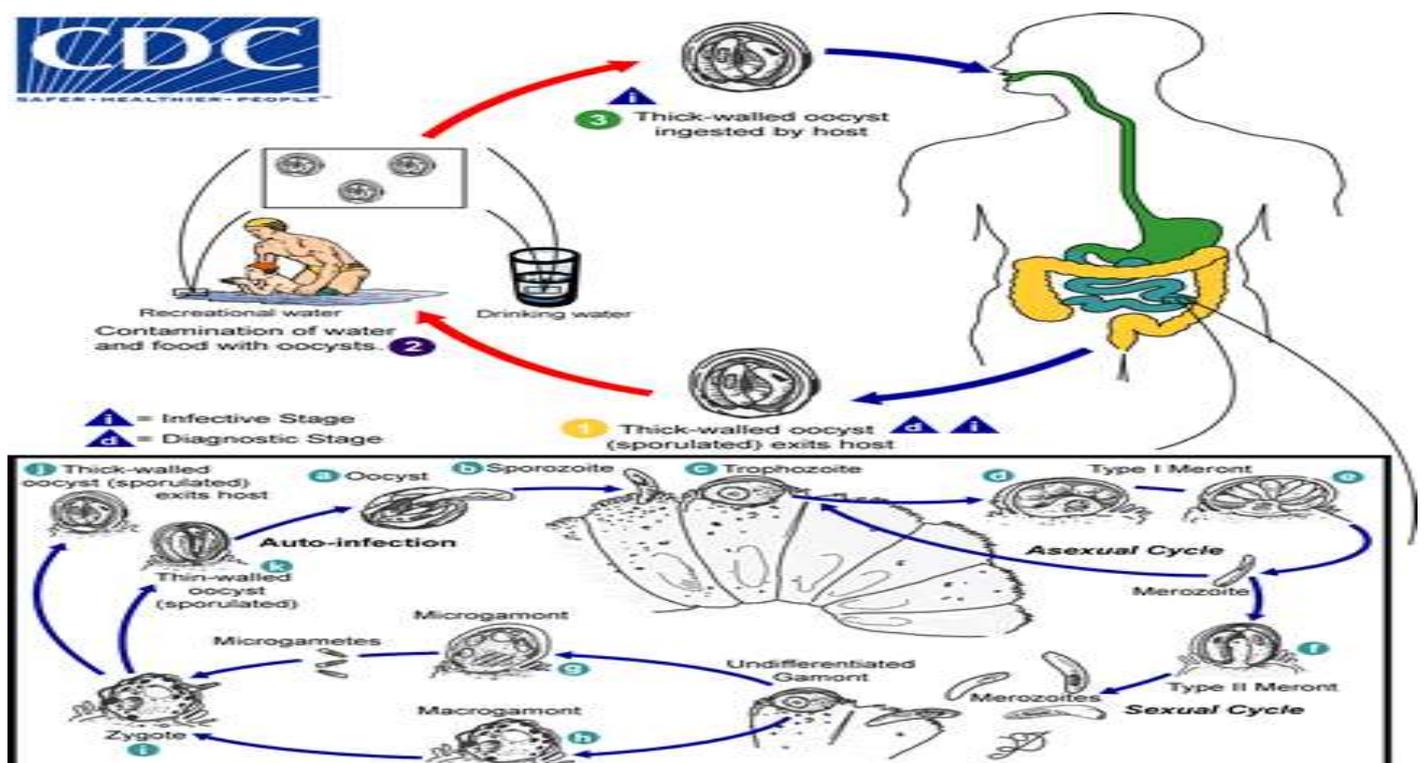
- Transmission of *Cryptosporidium parvum* and *C. hominis* occurs mainly through contact with *contaminated water and food*.
- Transmission can occur also through *exposure to infected animals or exposure to water contaminated by feces of infected animals*.

The Life Cycle of Cryptosporidium:

1- Ingestion or inhalation of *Sporulated oocysts* (the infective stage).

2- A process known as *excystation* takes place, the sporozoites are released and parasitize epithelial cells of the *gastrointestinal tract* or other tissues such as the *respiratory tract*.

Note: Oocysts are infective upon excretion immediately, thus permitting direct and immediate fecal-oral and person to person transmission.



Further explanation: Similar to Giardia, cryptosporidium has an inactive infectious form which is transmitted usually through contaminated food, once it gets to a favorable environment (GI or Respiratory tracts), it undergoes excystation, releasing its sporozoites, which reproduce sexually or asexually, causing its symptoms.

Clinical aspects:

- Clinically, symptoms differ according to the immune system of patients:
 - a) In Immunocompetent patients (Humans that have a normal immune response):
asymptomatic infection or *self-limited watery diarrhea*.
 - b) In immunocompromised patients: *chronic, severe, non-bloody diarrhea* with *nausea, vomiting, abdominal pain*, and *anorexia* resulting in weight loss and *death*
- Symptoms in immunocompetent patients are usually short lived (1 to 2 weeks).

Methods for diagnosis of Cryptosporidium:

- Diagnosis depends on *detection of oocysts* in fresh stool samples.
- Stool concentration techniques using *a modified acid fast stain* are usually necessary.
- Stool antigen detection by *direct fluorescent antibody* or *EIA* tests are now available.

Remember: A *direct fluorescent antibody* (DFA), also known as "direct immunofluorescence microscopy", is used also to detect Giardia.

Treatment:

Nitoxoxanide, is a synthetic drug and has been approved for use in all patients over 1 year of age in the U.S and is reported to have a cure rate of 72% to 88% by the CDC.

CYCLOSPORA (INTESTINAL SPOROZOA)

The Life Cycle of Cyclospora:

The life cycle of *Cyclospora* is similar to the *Cryptosporidium* and appears to involve only a single host. (capable of completing its lifecycle within a single host)

The main difference is that **Cyclospora oocysts** are not immediately infectious when freshly passed in stools (since, sporulation occurs after days or weeks), so person to person transmission is uncommon.

Further explanation note: Since sporulation occurs after many days, the oocysts of cyclospora die from the unfavorable environment, before being able to sporulate and survive these harsh environments.

Clinical aspects:

It causes **altered mucosal architecture** with **shortening of intestinal villi** due to diffuse edema and infiltration of inflammatory cells which leads to diarrhea, anorexia, fatigue, and weight loss.

Diagnosis:

By examining stools for oocysts which are (8–10 μm) in diameter and are **acid-fast positive** (reddish color).

Treatment:

Cyclospora infections are treatable with **trimethoprim-sulfamethoxazole** (TMP-SMZ).

Balantidium coli (Intestinal Ciliated protozoa)

General characteristics:

- It causes **balantidiasis** or **balantidial dysentery**.
- The **largest** intestinal protozoa of humans.
 - The **trophozoite** stage is a ciliated oval organism (60 X 45 μm or larger). It has a steady progression and rotation around the long axis motion (Rotary motility).

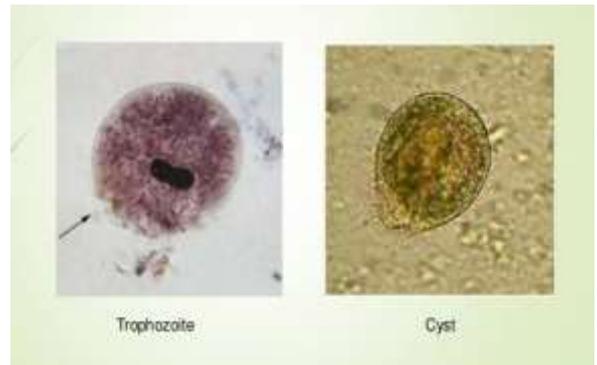
I found this video usefull for visualizing the motion of balantidium coli

<http://www.youtube.com/watch?v=QivUzq8GS3s>

Remember: Cysts are the parasite stage responsible for **transmission** while trophozoites are **disease-causing and not infective**.

Clinical aspects:

- Most infections are apparently *harmless* in endemic areas. However, rarely, in acute outbreaks the trophozoites *invade the large bowel and terminal ileum causing erosions and ulceration*.
- Some trophozoites invade the wall of the colon and multiply and leading to the presence of *mucous* and *blood* in the stool.



Treatment:

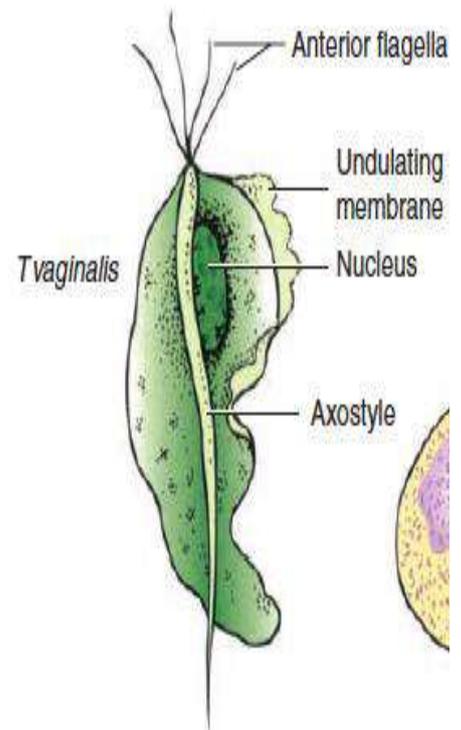
- **Oxytetracycline**, which may be followed by **Iodoquinol** or **Metronidazole**.

Sexually transmitted protozoan infection

Trichomonas (Urogenital flagellated protozoa)

General characteristics:

- Trichomonas are flagellated protozoa with 3-5 anterior flagella.
- There are three members of the genus Trichomonas parasitize humans:
 - 1- **Trichomonas hominis** → parasitize the **intestine**.
 - 2- **Trichomonas tenax** → parasitize the **mouth**.
 - 3- **Trichomonas vaginalis**:
 - a) parasitize the **Vagina** in females.
 - b) parasitizes in **Prostate, seminal vesicle** and **Urethra** in males.
- **Trichomonas vaginalis** cause **trichomoniasis** in humans.
- It is **pear-shaped** with **undulating membrane** (which enhances motility of the parasite in a viscous fluid, such as blood) lined with a **flagellum** and **4 anterior flagella**.
- It is about 5-30 X 2-14 µm. it moves with **wobbling or rotating motion**.



Note: only **T-vaginalis** is an established pathogen. And it attaches to the host cell by its axostyle.

Pathology and Pathogenesis:

- Direct contact of **T-vaginalis** with the squamous epithelium of the **genitourinary tract** results in:
 - a) Destruction of the involved epithelial cells.
 - b) Development of a neutrophilic inflammatory reaction.
 - c) Petechial hemorrhages (tiny pinpoint red mark).
 - d) Pain during urination (dysuria) or sexual intercourse.
- In females, it causes low-grad inflammation limited to **vulva vagina** and **cervix**, **causing vaginitis** with frothy yellow or creamy discharge.
- In Males , it may infect the **Prostate**, **Seminal Vesicles** or the **urethra**
- It is a sexual transmitted disease (STD) but sometimes, (nonvenereal transmission) can occur (non-sexual transmission) for example babies can *get infected as they move through the birth*.

Diagnosis:

In most symptomatic cases, **wet mount examination** for motile trophozoites is sufficient/enough.

Note: There is no **Cyst** stage within **Trichomonas**, so we depend on **Trophozoite** morphology for diagnosis.

Treatment:

- Topical and systemic → **Metronidazole**. (*From the slides*)
- **Tinidazole** and **Ornidazole** are equally effective with fewer side effects.
These drugs belong to (Nitroimidazoles) family.

Note: Alcohol consumption should be avoided during treatment with nitroimidazoles, to reduce the possibility of a disulfiram-like reaction.

They are not highly recommended during Pregnancy since they have teratogenic effects (an agent that can disturb the development of the embryo or fetus).

Blood and tissue protozoan infections

Hemoflagellate: contains only two genera that parasitize humans

1- Trypanosoma

2- Leishmania

- They are invasive either in tissues or in the blood.
- They produce high morbid, frequent and *lethal diseases*.
- They usually need intermediate hosts (vectors, which help transmit an infection from one host to another).
- Hemoflagellates have several morphologic forms:

a) **Amastigote** (rounded form): Found in intracellular Environments (Tissues)

b) **Epimastigote** and **Promastigote**.: Found in insect hosts

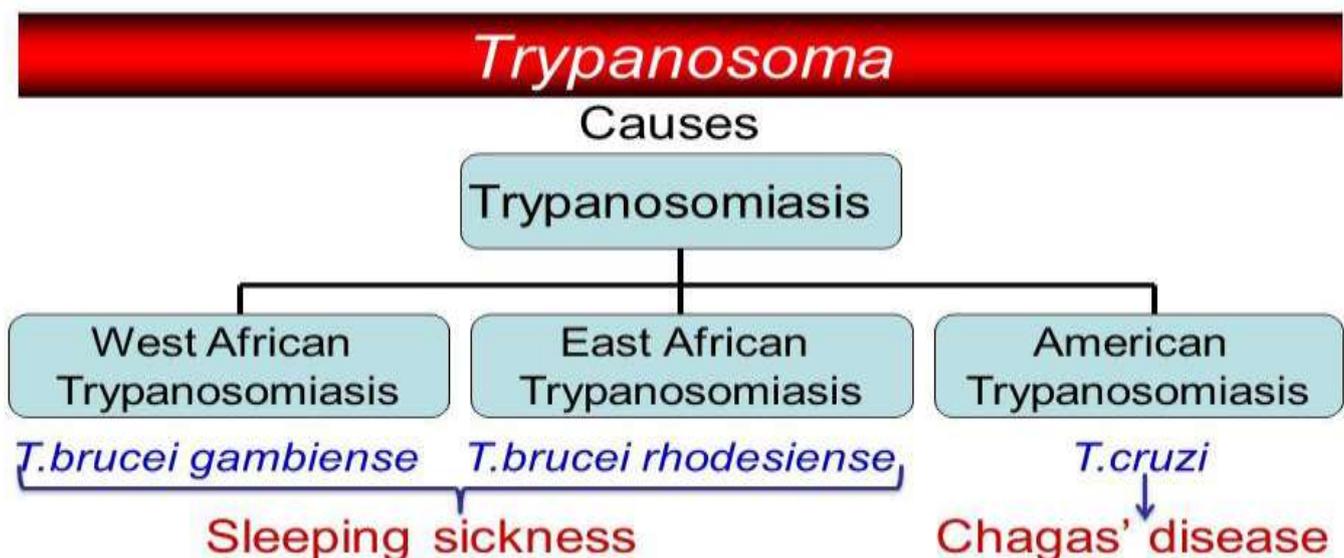
c) **Trypomastigote** : Found in mammalian Bloodstream

For additional information on this topic: http://dna.kdna.ucla.edu/parasite_course-old/cruzi_files/subchapters/morphology%20and%20life%20cycle.htm .

<http://mibr.asm.org/content/mibr/66/1/122/F1.large.jpg>

Trypanosoma:

- There are two types of trypanosomiasis that affect humans, they are divided according to etiological agents as well as geographical location.



- **West African Trypanosomiasis** causes slowly developing chronic disease, while **East African Trypanosomiasis** causes acute disease.

African Trypanosomiasis

- The Vector: **tsetse fly** (*Glossina* species), Which is found only in rural Africa.

- a) *Glossina palpalis* transmits → Trypanosomiasis *brucei* (T. b.) *gambiense* .
- b) *Glossina morsitans* transmits → T. b. *rhodesiense*.

- Tsetse flies of both sexes transmit pathogenic African trypanosomes (if we compare it to malaria, only female anopheles mosquito can transmit the disease).
- A bite by the tsetse fly is often **painful** and can develop into a red sore, also called a **Painful chancre** (*Chanchroid*) then **parasitemia** occurs and the infection invades the **central nervous system**.
- The infection leads to Insomnia at night, and Sleepiness during the day (may be uncontrollable).
- The African trypanosomes shows two morphologic forms: ***Trypomastigote*** and ***Epimastigote***.
- The African trypanosomes only attach to RBCs, without invading them, unlike malaria, which invades RBCs.

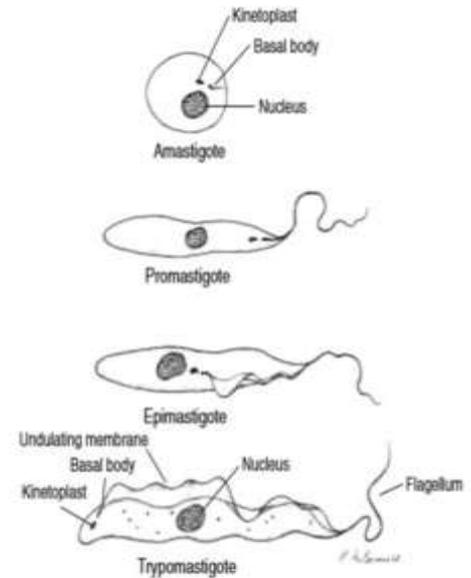


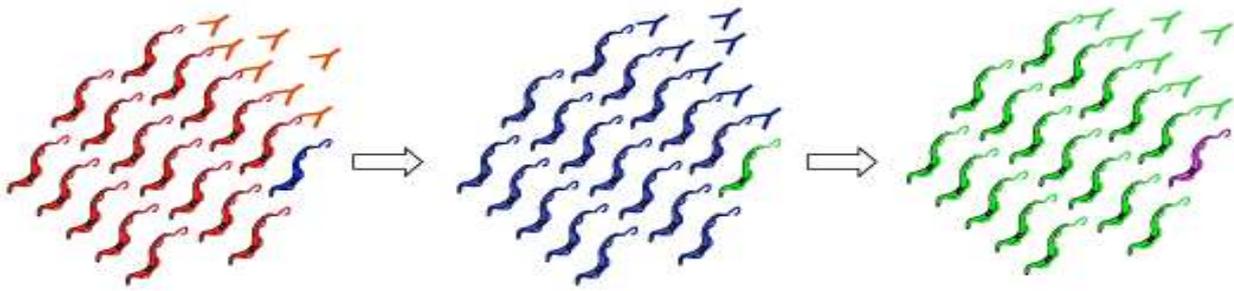
Figure 49-8 Characteristic stages of species of *Leishmania* and *Trypanosoma* in human and insect hosts. (Illustration by Nobuko Kitamura.)

IMPORTANT Note: the doctor said that it also shows *Promastigote* and *Amastigotes* forms!!! but all other sources say 2 forms. I'll try to contact with Dr.Nader, if he insists that it shows 4 forms I will edit it immediately. 😊

Note: A unique feature of African trypanosomes is **their ability to change the antigenic surface coat** of the outer membrane of the *trypomastigote*, helping to evade the host immune response, since *trypomastigote* surface is covered with a dense coat of **variant surface glycoprotein (VSG)**.

- So each time the antigenic coat changes, the host does not recognize the organism and must **undergo a new immunologic response**.

The picture below shows that some **trypomastigotes** escape and change their antigens before recognizing them by immune's antibodies.



American Trypanosomiasis

- The Vector: reduviid bugs (also called Triatomine bugs, kissing bugs).
- Causes zoonotic disease (disease which can be transmitted to humans from animals)
- It has 3 developmental stages:
 - a) **Epimastigote**, Inhabits insects, not mammals.
 - b) **Trypomastigote**, Inhabits the bloodstream.
 - c) **Amastigotes**, inhabits Tissues.
- Definitive hosts include: Humans, dogs, cats, rats...etc.

Amastigote: is usually found in heart, liver and brain, so it's a serious infection.

- Reduviid bug (defecates) near wounds while taking a **blood meal**, Then a chagoma forms (an inflammatory nodule at the bite site of the reduviid bug)
- On the other hand, in African trypanosomiasis, tsetse fly bites humans and inoculates parasites that are found in its **saliva**.

Leishmaniasis

Leishmaniasis is divided into clinical syndromes according **to what part of the body is affected most**:

1- **Cutaneous Leishmaniasis** it is also called (Baghdad boil, oriental sore), caused by (*L.tropica*) and (*Leishmania major*), *Leishmania major* is the most common type in Jordan especially in areas near to Al Aqaba city.



2- *Mucocutaneous leishmaniasis* is caused by (L. braziliensis).



3- *Visceral Leishmaniasis* (black fever, kala-azar), is caused by (L.donovani).



Back to leishmaniasis generally,

- It is a flagellated protozoan
- Life cycle requires two hosts:
 - a) **Vertebrate**; mammalian host
 - b) **Invertebrate vector**; female sand fly
- Obligate intracellular organism. (which means that it cannot reproduce outside its host cell, meaning that the parasite's reproduction is entirely reliant on intracellular resources.)
- Infects primarily **phagocytic cells and macrophages**.
- The incubation period ranges from 10 days to 2 years.

Transmission:

1. Bite of sand fly.
2. Transfusion blood and transplantation.
3. Mother to baby.
4. Direct contact; from man to man through nasal secretion.

Plasmodium (Blood sporozoa)

- Plasmodium is a genus of parasitic alveolates, many of which cause malaria in their hosts.
- The parasite always has two hosts in its life cycle: **Dipteran insect host** (type of insects) and a **vertebrate host**.
- The life cycle of Plasmodium can be divided into two distinct phases:
 - 1- **The asexual cycle**, called schizogony, occurs in *vertebrate hosts like humans*.
 - 2- **The sexual cycle**, occurs in *insect host like mosquitoes*.
- The vector for malaria is the female anopheline mosquito.

Species of malaria parasites that commonly infect humans:

1. Plasmodium malariae:
 - causes **quartan fever**, a malarial fever that typically recur (occur again) every 72 hours or every fourth day (at three-day intervals)
 - Also causes classical malaria in humans
2. P. vivax:
 - the most frequent and widely distributed cause of malaria.
 - cause **tertian fever**, a malarial fever that typically recur every 48 hours or every third day (two-day intervals).
3. P. ovale:
 - is relatively uncommon.
 - Also causes **tertian fever**.
4. P. falciparum:
 - it is the major species associated with deadly infections throughout the world, (*The most dangerous type*). It causes **malignant fever** which recurs every 48 hours but with more severe symptoms, the temperature can reach more than 42 degrees affecting the brain and cerebral cortex.
5. P. knowlesi:
 - It causes malaria in monkeys and can infect human as well.

The name of the fever describes how long it takes for the fever to recur (quartan = four days, tertian= three days)

Recur: occur again periodically or repeatedly.

Mechanism of Infection:

1. The **vector mosquito** takes a blood meal.
2. Sporozoites contained in *the salivary glands of the mosquito* are discharged into the *punctured wound* (deep wound).
3. Within an hour, these infective sporozoites are carried via the *blood to the liver*.
4. They penetrate *hepatocytes and begin to grow*, initiating the *pre-erythrocytic or primary exoerythrocytic cycle*.
 - **exoerythrocytic cycle**: is the developmental stage of the malaria parasite in liver parenchyma cells of the vertebrate host before the red blood cells become infected.
5. The sporozoites become *round or oval and begin dividing repeatedly*.
 - **Schizogony** results in large numbers of exoerythrocytic merozoites (a small ameboid sporozoan trophozoite)
6. Once these merozoites leave the liver, they invade the red blood cells (RBCs), this marks the end of the *exo-erythrocytic cycle* thus initiating the *erythrocytic cycle* (Infection of RBCs).
7. Once the RBCs and reticulocytes (immature red blood cell) have been invaded, the parasites *grow and feed on hemoglobin*.
8. Within the RBC, the merozoite (or young trophozoite) becomes vacuolated, ring shaped, more or less ameboid, and uninucleate.
9. Merozoites progress into mature **trophozoites**, Once the nucleus begins to divide, the **trophozoite** is called a **developing schizont** (contains many *merozoites*, which rupture releasing *merozoites* into the bloodstream).
10. The excess **protein and hematin** present from the *metabolism of hemoglobin* (hemolytic anemia) combine to form malarial pigment.

*(The number of **merozoites** in the single **schizont** depends on the species.)*

Note: A dormant schizogony may occur in **P. vivax** and **P. ovale** organisms, which remain quiescent (inactive) in the liver. These resting stages have been termed hypnozoites and lead to a true relapse, often within 1 year or up to more than 5 years later.

True relapse: recurrence of symptoms of a disease after a period of improvement, similar to an incubation period.

Laboratory diagnosis (Includes all species of malaria parasites):

1- Routine Methods:

Thick and thin blood films, At least 200 to 300 oil immersion fields should be examined on both films before a negative report is issued.

- Blood is collected using **EDTA anticoagulant**.
 - **Stains we use in blood films:**
Giemsa stain, Wright's stain and **Fluorescent nucleic acid stains**, such as **acridine orange**.
- Through the routine methods, we can determine the presence and which type of plasmodium is causing the disease.

2- Antigen antibody detection

Therapy:

1. **QUINOLINES**, are Antimalarial drugs such as **Chloroquine** that is used for all types except for *P. falciparum* infections due to acquired resistance.
2. *P. falciparum* infections are treated by **combination therapies**.
3. **Primaquine** Treats Hypnozoites
4. **ARTEMISININS** can also be used as antimalarial drugs.
5. **Tetracycline, doxycycline, and clindamycin** are used increasingly in combination with other antimalarial drugs to improve their efficacy.

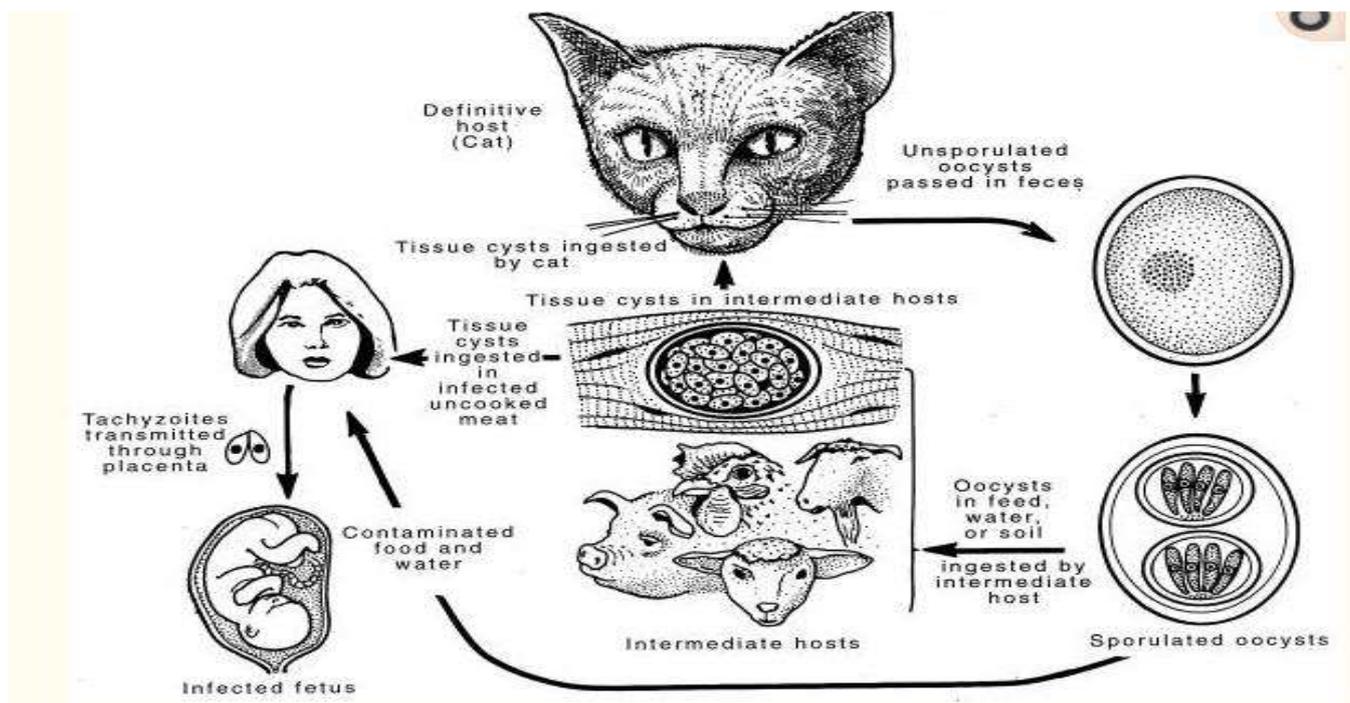
These drugs are classified into the following, according to the stage of malarial growth that they target:

1. **Tissue schizonticides** (which kill tissue schizonts).
2. **Blood schizonticides** (which kill blood schizonts).
3. **Gametocytocides** (which kill gametocytes).
4. **Sporonticides** (which prevent formation of sporozoites within the mosquito, inhibiting the life cycle of malarial parasite).

Tissue protozoa

Toxoplasma gondii (Tissue sporozoa)

- It is a **coccidian protozoa** with worldwide distribution that infects wide range of animals and birds but does not appear to cause disease in them.
- The normal final hosts are strictly the **cats and its relatives**, the only host of which the oocyst-producing sexual stage of toxoplasma can develop.
- When oocysts are ingested, they can either repeat its sexual life cycle in a cat, or if ingested by a **human they can establish an infection in which it can reproduce asexually**, where it opens up and releases sporozoites to duodenum then invade various cells especially macrophages where they form **tachyzoites** which spread infection to lymph nodes and other organs.
- Latent infections occur with Toxoplasma (parasites in tissue **cysts** are called **bradyzoites**).
- it produces either congenital or postnatal toxoplasmosis.
- Congenital infections occur in non-immune mothers during pregnancy.



THE END 😊