Intestinal flagellates

- Giardia intestinalis
- Trichomonas spp
- Dientamoeba fragilis
- Trichomonas vaginalis

Hemoflagellates

- Leishmania donovani
- Trypanosoma cruzi

Leishmania donovani

- a vector-borne disease
- transmitted by sandflies
- caused by obligate intracellular protozoa of the genus *Leishmania*

- Promastigotes
- amastigotes

- in the sandfly's midgut, the parasites differentiate into promastigotes

Mexico
Central America,
South America
southern Europe
Asia (not Southeast Asia),
the Middle East,
and Africa

**Symptoms**

**Cutaneous leishmaniasis (L.tropica)**

- one or more sores on their skin.
- the sores can change in size and appearance over time.
- they often end up looking somewhat like a volcano, with a raised edge and central crater.
- a scab covers some sores.
- the sores can be painless or painful.
- some people have swollen glands near the sores (for example, in the armpit if the sores are on the arm or hand).

**Visceral leishmaniasis (kala-azar)(L.donovani)**

- fever,
- weight loss,
- enlarged spleen and liver (usually the spleen is bigger than the liver).
- Some patients have swollen glands.
- Certain blood tests are abnormal. low blood counts, including a low red blood cell count (anemia), low white blood cell count, and low platelet count.
- Some patients develop post kala-azar dermal leishmaniasis.
- Visceral leishmaniasis is becoming an important opportunistic infection in areas where it coexists with HIV.

**Diagnosis**

- Giemsa-stained slides
- culture (using for example the diphasic NNN medium)
Antibody detection

Trypanosoma cruzi

Chagas disease,

a zoonotic disease

transmitted to humans

by blood-sucking triatomine bugs (kissing bug)

Chronic Chagas disease is a major health problem in many Latin American countries

trypomastigotes

amastigotes

trypomastigotes

amastigotes

Trypanosoma cruzi can also be transmitted through blood transfusions, organ transplantation, transplacentally, in laboratory accidents.

Symptoms

fever,

anorexia,

lymphadenopathy,

mild hepatosplenomegaly,

myocarditis.

Chronic stage may not occur for years or even decades after initial infection.

cardiomyopathy (the most serious manifestation);
• megaesophagus
• megacolon;
• weight loss.
Chronic Chagas disease and its complications can be fatal.

Diagnosis

Microscopic examination:
• of fresh anticoagulated blood, or its buffy coat, for motile parasites;
• of thin and thick blood smears stained with Giemsa, for visualization of parasites.

Isolation of the agent by:
• inoculation into mice;
• culture in specialized media (e.g. NNN, LIT);
• xenodiagnosis, where uninfected reduviid bugs are fed on the patient's blood, and their gut contents examined for parasites 4 weeks later.

Treatment

• benznidazole or nifurtimox
• In the chronic stage, e.g., pacemaker for heart block

Trypanosoma brucei

• T. b. gambiense - West African sleeping sickness
• T. b. rhodesiense - East African sleeping sickness.
• T. b. brucei - under normal conditions does not infect humans

Symptoms

• a trypanosomal chancre can develop on the site of inoculation.
• a hemolymphatic stage (fever, lymphadenopathy, and pruritus.)

• the meningoencephalitic stage (headaches, somnolence, abnormal behavior, loss of consciousness and coma)

• the course of infection is much more acute with *T. b. rhodesiense* than *T. b. gambiense*.

**Diagnosis**

• microscopic examination of chancre fluid, lymph node aspirates, blood, bone marrow, or, in the late stages of infection, cerebrospinal fluid.

• a wet preparation should be examined for the motile trypanosomes, and in addition a smear should be fixed, stained with Giemsa (or Field), and examined.

• the Card Agglutination Trypanosomiasis Test (CATT) test is of value for epidemiologic surveys or screening of *T. b. gambiense*

**Amebas**

**Intestinal amebas**

• *Entamoeba histolytica*

• *Entamoeba coli*

• *Balantidium coli*

**Free-living amebas**

• *Naegleria fowleri*

• *Acanthamoeba castellani*

*Naegleria fowleri* - *Acanthamoeba* spp

• commonly found in lakes, swimming pools, tap water, and heating and air conditioning units.
• only one species of *Naegleria* is known to infect humans, several species of *Acanthamoeba* are implicated

**Symptoms**

• Acute primary amebic meningoencephalitis (PAM) - *Naegleria fowleri*.
• severe headache and other meningeal signs, fever, vomiting, and focal neurologic deficits, and progresses rapidly (<10 days) and frequently to coma and death.
• *Acanthamoeba* spp. - subacute or chronic granulomatous amebic encephalitis (GAE), with a clinical picture of headaches, altered mental status, and focal neurologic deficit, which progresses over several weeks to death.
• granulomatous skin lesions
• more seriously, keratitis and corneal ulcers following corneal trauma or in association with contact lenses.

**Diagnosis**

*Naegearia* infections

• CSF
• a wet mount may detect motile trophozoites,
• Giemsa-stained smear trophozoites
*Acanthamoeba* infections,
• biopsy specimens (brain tissue, skin, cornea) or of corneal scrapings, which may detect trophozoites and cysts.

• cultivation
• direct immunofluorescent antibody

*N.fowleri*                           *Acanthamoeba* spp.

**Treatment**

• Eye and skin infections caused by *Acanthamoeba* spp. are generally treatable. Topical use of 0.1% propamidine isethionate (Brolene) plus neomycin-polymyxin B-gramicidin
ophthalmic solution

- keratoplasty is often necessary in severe infections
- Amphotericin B has been successfully used to treat PAM caused by *Naegleria fowleri* Sporozoans

- **Blood sporozoans**
  - *Plasmodium vivax*
  - *Plasmodium malariae*
  - *Plasmodium ovale*
  - *Plasmodium falciparum*
  - *Babesia microti*

- **Other**
  - *Isospora belli*
  - *Sarcocystis bovihumanis*
  - *Cryptosporidium parvum*
  - *Toxoplasma gondii*

  *Plasmodium spp.*

- *Plasmodium vivax*
- *Plasmodium malariae*
- *Plasmodium ovale*
- *Plasmodium falciparum*
• A single species, *P. reichenowi*, which infects chimpanzees, is known to be a close sister lineage of *P. falciparum*.

• This new species has been isolated in two chimpanzees (*Pan troglodytes*) kept as pets by villagers in Gabon (Africa).

• *The risk of transfer and emergence of this new species in humans must be now seriously considered given that it was found in two chimpanzees living in contact with humans and its close relatedness to the most virulent agent of malaria.*

• The malaria parasite life cycle involves two hosts:
  • a malaria-infected **female Anopheles mosquito** - sporozoites - **the human host**
  • sporozoites infect liver cells - into schizonts, which rupture and release merozoites.
  • *P. vivax* and *P. ovale* a dormant stage [hypnozoites] can persist in the liver and cause relapses by invading the bloodstream weeks, or even years later.
  • initial replication in the liver (exo-erythrocytic schizogony) - asexual multiplication in the erythrocytes (erythrocytic schizogony).
  • merozoites infect red blood cells.
  • the ring stage trophozoites mature into schizonts, which rupture releasing merozoites.
  • some parasites - into sexual erythrocytic stages (gametocytes).
  • male (microgametocytes) and female (macrogametocytes), are ingested by an *Anopheles* mosquito during a blood meal.
  • parasites’ multiplication in the mosquito - the **sporogonic cycle**.
  • in the mosquito's stomach, the microgametes penetrate the macrogametes generating zygotes.
• the zygotes in turn become motile and elongated (ookinetes) which invade the midgut wall of the mosquito where they develop into oocysts.

• the oocysts grow, rupture, and release sporozoites, which make their way to the mosquito's salivary glands.

• Inoculation of the sporozoites into a new human host perpetuates the malaria life cycle.

Symptoms

The most frequent symptoms

• fever
• chills,
• headache,
• myalgias,
• arthralgias,
• weakness,
• vomiting,
• diarrhea.

• Other clinical features
  — splenomegaly,
  — anemia,
  — thrombocytopenia,
  — hypoglycemia,
  — pulmonary or renal dysfunction,
  — neurologic changes.
• **P. falciparum** (cerebral malaria),
  - acute renal failure,
  - severe anemia,
  - adult respiratory distress syndrome.
• **P. vivax** - splenomegaly (with, rarely, splenic rupture),
• **P. malariae** - nephrotic syndrome.

• **Ring**: early developmental stage of the asexual erythrocytic parasite;
• **Trophozoite**: next developmental stage of the asexual erythrocytic parasite; it has lost its "ring" appearance, and has begun to accumulate pigment
• **Schizont**: late developmental stage of the asexual erythrocytic parasite; it has begun its division into merozoites, and thus is characterized by the presence of multiple contiguous chromatin dots
• **Gametocyte**: sexual erythrocytic stage.

**Treatment**

**Quinine** (in chloroquine resistant *P.falciparum*)

**Quinidine** (IV form of quinine)

**Chloroquine** (acute attack) (prophylaxis)

**Mefloquine** (prophylaxis in chloroquine resistant cases)

**Prymethamine-sulfadoxine** (in chloroquine resistant *P.falciparum*)

**Artemisinine** (**Qinghaosu**) (in chloroquine resistant *P.falciparum*)

**Tetracycline and doxycycline**

**Halofantrine**

**Chloroguanide**
**Primaquine** (eliminates exo erythrocytic forms in the liver)

**Prophylaxis**

**Chloroquine** 500 mgr per week starting 1-2 wks before travel, during travel and 4 wks post travel.

**Chloroquine resistant P.falciparum mefloquine** 250 mgr.per week, 1 week before, during travel and 4 wks post travel. *Don’t use mefloquine during pregnancy.*

**Treatment**

**Chloroquine** 1gr po, 0.5 gr in 6 hrs, then 0.5gr.daily x 2d.

**Primaquine** 26.3 mgr.po daily x14d. *Check for severe G6PD deficiency.*

**Chloroquine resistant P.vivax** quinine sulphate 650 mgr+doxycycline 100 mgr.OR mefloquine 750 mgr.po, then 500 mgr. 12 hrs.later. for 7 days

**Chloroquine resistant P.falciparum** quinine sulphate 325 mgr.+doxycycline100mgr. for 7 days.

**Babesiosis**

hemoprotezoan parasites of the genus *Babesia*

**Babesia microti** and *Babesia divergens*

easily be misdiagnosed as *Plasmodium*

**Symptoms**

probably asymptomatic

fever

chills

sweating

myalgias

fatigue

hepatosplenomegaly

hemolytic anemia
Diagnosis

detection of parasites in patients' blood:
IFA
PCR

Toxoplasmosis

- eating undercooked meat of animals harboring tissue cysts.
- consuming food or water contaminated with cat feces or by contaminated environmental samples (such as fecal-contaminated soil or changing the litter box of a pet cat).
- blood transfusion or organ transplantation.
- transplacentally from mother to fetus.

Toxoplasma gondii

- asymptomatic infection.
- cervical lymphadenopathy
- a flu-like illness.
- In rare cases ocular infection with visual loss can occur.

Immunodeficient patients;
- central nervous system disease
- retinochoroiditis,
- pneumonitis,
- other systemic disease.

In patients with AIDS, toxoplastic encephalitis is the most common cause of intracerebral mass lesions.

- Congenital toxoplasmosis

- results from an acute primary infection acquired by the mother during pregnancy.
The incidence and severity of congenital toxoplasmosis vary with the trimester during which infection was acquired.

Ocular Toxoplasma infection, can be the result of congenital infection,

Observation of parasites in patient specimens, such as bronchoalveolar lavage material from immunocompromised patients, or lymph node biopsy.

Isolation of parasites from blood or other body fluids, by intraperitoneal inoculation into mice or tissue culture.

Detection of parasite genetic material by PCR, especially in detecting congenital infections in utero.

*Serologic testing is the routine method of diagnosis.* (T.gondii IgM and IgG)

Reference

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