Toxoplasma gondii

Parasitology (MA301)

Medical Analysis Department

Summer School

Lecture 4

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Toxoplasma gondii

Case Study

A 22-year-old woman presented with mild fever, fatigue, and enlarged cervical lymph nodes. She reported frequent contact with cat litter and consumption of undercooked meat. Serological testing demonstrated antibodies consistent with recent **Toxoplasma gondii** infection. She was monitored and received appropriate treatment based on her clinical status, while being advised to avoid undercooked meat and use gloves and proper hygiene when handling cat litter, leading to clinical improvement

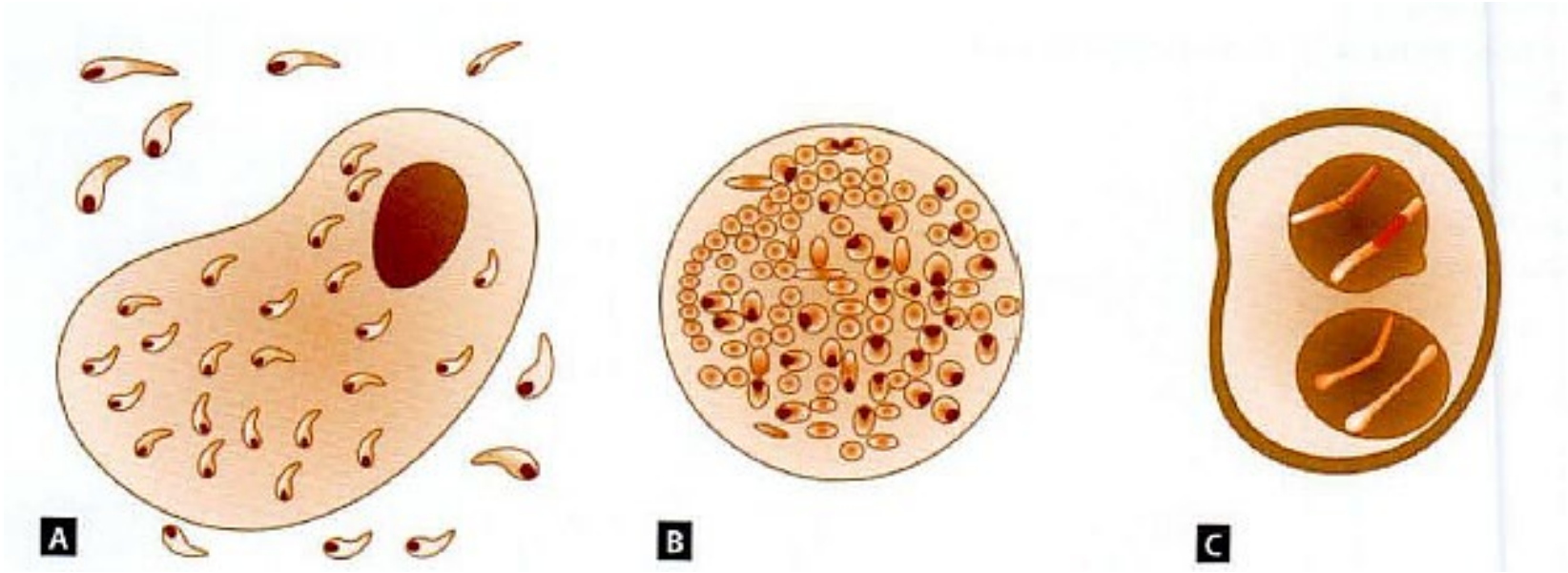
Introduction

- Toxoplasma gondii is an obligate intracellular coccidian parasite
- The name Toxoplasma is derived from the Greek word Toxon, meaning arc or bow, referring to the curved shape of the trophozoite.
- Toxoplasma is now recognized as the most common protozoan parasite globally, with the widest range of hosts spread over 200 species of birds, reptiles, and mammals, including humans.

Morphology

T. gondii occurs in three forms:

1. Trophozoite (A)
2. Tissue cyst (B)
3. Oocyst (C)



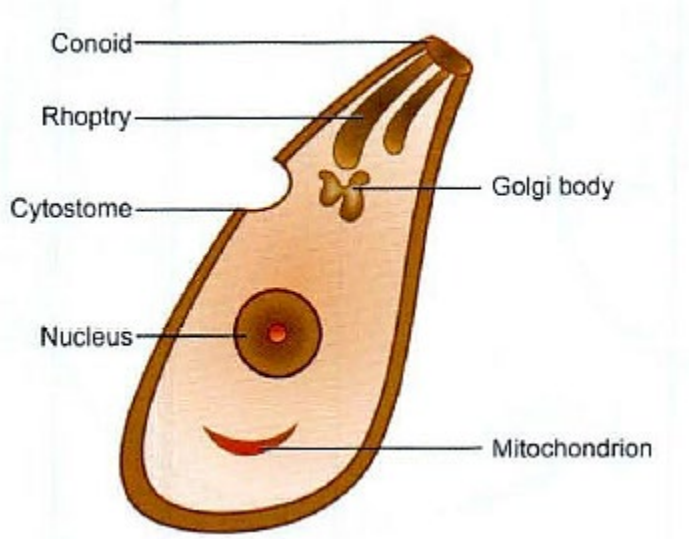
Morphology

- The trophozoite and tissue cyst represent stages in asexual multiplication (schizogony), while the oocyst is formed by sexual reproduction (gametogony or sporogony).
- All three forms occur in domestic cats and other felines, which are the definitive hosts and support both schizogony and gametogony.
- Only the asexual forms, trophozoites and tissue cysts are present in other animals, including humans and birds, which are the intermediate hosts.
- All three forms are infectious to man.

Morphology

Trophozoites (Tachyzoites)

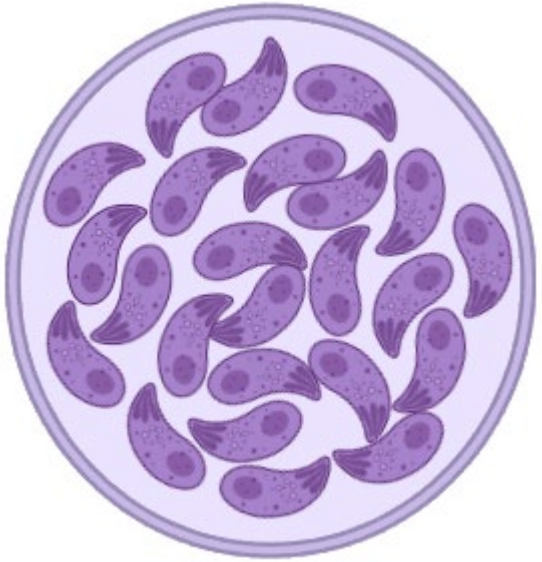
- The trophozoite is crescent-shaped, with one end pointed and the other end rounded.
- It measures 3-7 µm in length. The nucleus is ovoid and is situated at the blunt end of the parasite.
- The rapidly proliferating trophozoites in acute infection are called tachyzoites.

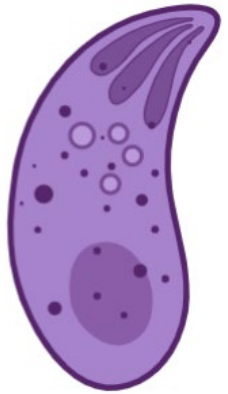


Morphology

Tissue cyst (Bradyzoite)

- Tissue cysts are the resting form of the parasite.
- They are found during chronic stage of the infection and can be found in the brain (most common site), skeletal muscles and various other organs.
- The cyst is round or oval, 10-20 µm in size and contains numerous bradyzoites. Cysts remain viable in tissue for several years.
- In immunologically normal hosts, the cysts remain silent, but in the immunodeficient subjects, they may get reactivated, leading to clinical disease.
- lhey reach various tissues and organs through blood and
- lymphatic dissemination.

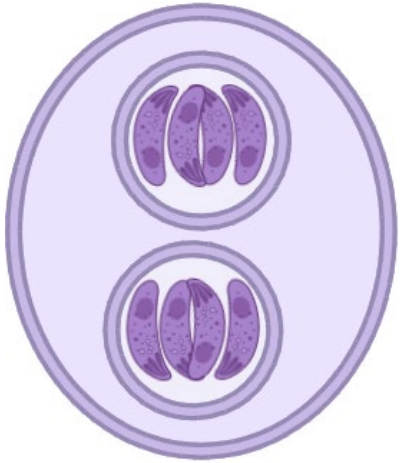




Morphology

Oocyst

- Oocysts develop only in definitive hosts in the intestine of cats and other felines, but not in humans.
- It is oval in shape and measures 10-12 µm in diameter. Each cyst is surrounded by a thick, resistant wall.
- The oocysts are formed by sexual reproduction (gametogony).
- Cats shed millions of oocysts which undergo sporulation in the soil with the formation of two sporocysts, each containing four sporozoites. The sporulated oocyst is infective.
- Oocyst is very resistant to environmental conditions and can remain infective in soil for about a year.
- When the infective oocyst is ingested, it releases sporozoites in the intestine, which initiate infection.

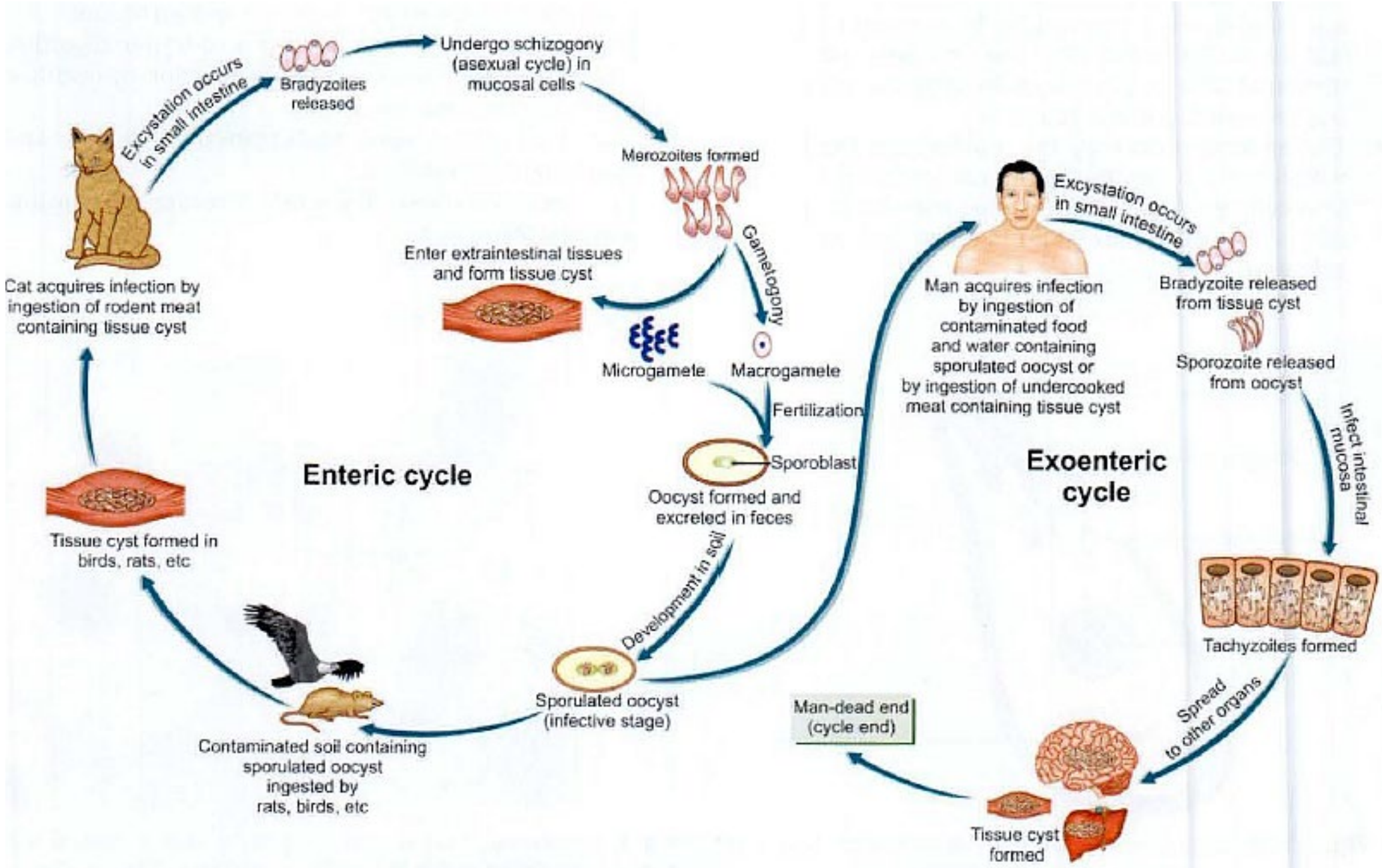


Life cycle

Host: T. gondii completes its life cycle in two hosts.

- Definitive hosts: Cats and other felines, in which both sexual and asexual cycles take place.
- Intermediate hosts: Man and other mammals, in which only the asexual cycle takes place.
- T. gondii has two types of life cycles:

1. Enteric cycle
2. Exoenteric cycle.


Enteric cycle
Cat acquires infection by ingestion of rodent meat containing tissue cyst
Excystation occurs in small intestine
Bradyzoites released
Undergo schizogony (asexual cycle) in mucosal cells
Merozoites formed
Enter extraintestinal tissues and form tissue cyst
Gametogony
Microgamete
Macrogamete
Fertilization
Sporoblast
Oocyst formed and excreted in feces
Development in soil
Sporulated oocyst (infective stage)
Contaminated soil containing sporulated oocyst ingested by rats, birds, etc
Tissue cyst formed in birds, rats, etc
Exoenteric cycle
Man acquires infection by ingestion of contaminated food and water containing sporulated oocyst or by ingestion of undercooked meat containing tissue cyst
Excystation occurs in small intestine
Bradyzoite released from tissue cyst
Sporozoite released from oocyst
Infect intestinal mucosa
Tachyzoites formed
Spread to other organs
Man-dead end (cycle end)
Tissue cyst formed

Enteric Life cycle

- In cats, both asexual (schizogony) and sexual (gametogony) reproduction occur in the small intestine.
- Infection is acquired by eating tissue cysts in prey or oocysts from feces.
- Bradyzoites released in the intestine multiply into merozoites; some form tissue cysts, while others develop into gametocytes.
- Fertilization produces oocysts that are excreted in feces, where they sporulate in soil.
- Mature oocysts with sporozoites are infective to other animals, continuing the cycle.

Exoenteric life cycle

- In humans and other intermediate hosts, infection occurs by eating undercooked meat (especially pork, lamb) with tissue cysts, ingesting oocysts from cat feces, congenital transmission, or rarely by transfusion or transplant.
- Sporozoites or bradyzoites invade intestinal cells, multiply into tachyzoites, and spread via blood and lymph to organs such as brain, eye, liver, and muscles, where they form tissue cysts with bradyzoites.
- These may persist for years and reactivate during immunosuppression.
- Humans are dead-end hosts; the full cycle is maintained mainly between cats and mice.

Pathogenicity and Clinical Features

- The outcome of Toxoplasma infection depends on the immune status of the infected person.
- Active progression of infection is more likely in immunocompromised individuals. Toxoplasmosis has acquired great importance as one of the major fatal coccidian complications in acquired immunodeficiency syndrome (AIDS).
- Most human infections are asymptomatic.
- Clinical toxoplasmosis may be congenital or acquired.

Clinical manifestation

Congenital toxoplasmosis

results when T gondii is transmitted transplacentally from mother to fetus.

This occurs when the mother gets primary toxoplasma infection, whether clinical or asymptomatic, during the pregnancy.

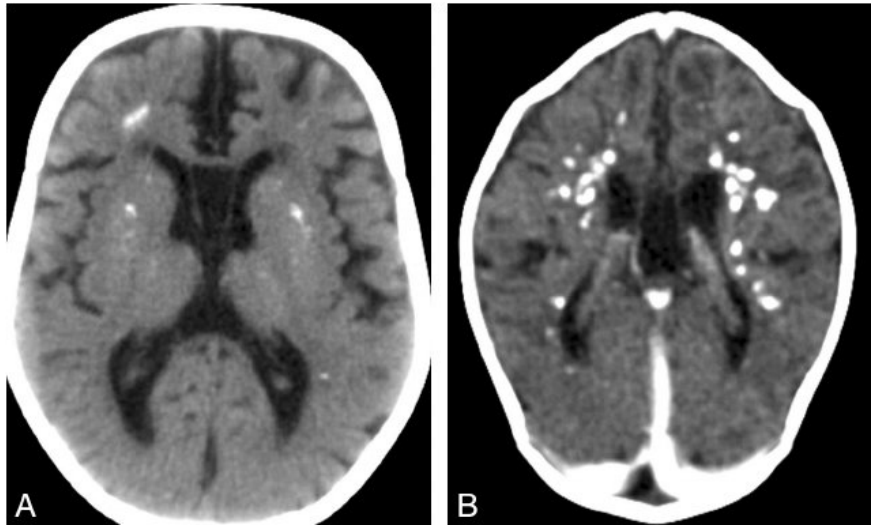
The risk of fetal infection increases with advancing pregnancy (about 25% in 1st trimester, up to 65% in 3rd), but damage is most severe when infection occurs early

Mothers with chronic infection usually do not transmit it, though reactivation during pregnancy can infect the fetus.

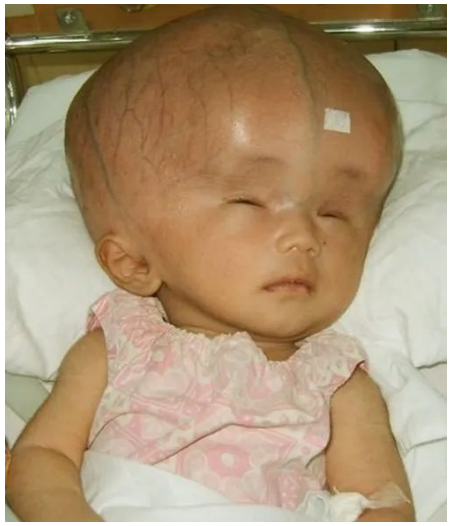
Clinical manifestation

Congenital toxoplasmosis

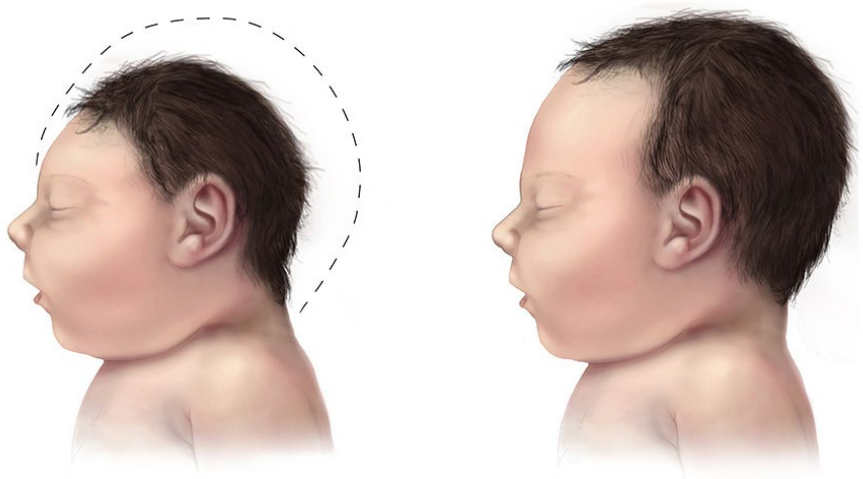
Most newborns are asymptomatic, but some develop disease weeks to years later. Manifestations include chorioretinitis, cerebral calcifications, hydrocephalus, microcephaly, seizures, mental retardation, deafness, blindness, and strabismus. A few present at birth with acute disease such as fever, jaundice, rash, lymphadenopathy, hepatosplenomegaly, myocarditis, eye defects, and CNS involvement.



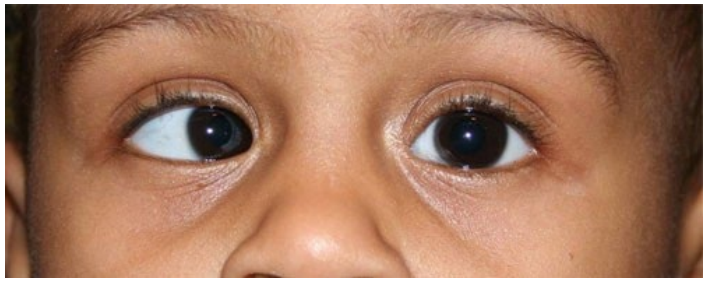
Cerebral calcification



Hydrocephalus



Microcephalus



Strabismus

Clinical manifestation

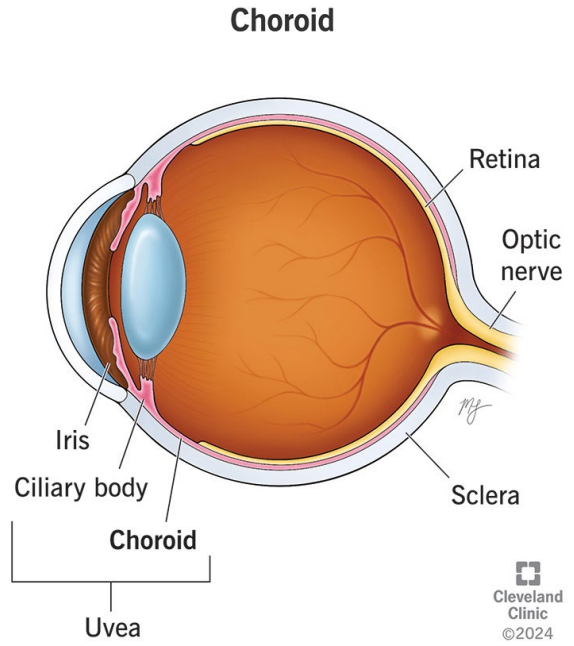
Acquired Toxoplasmosis

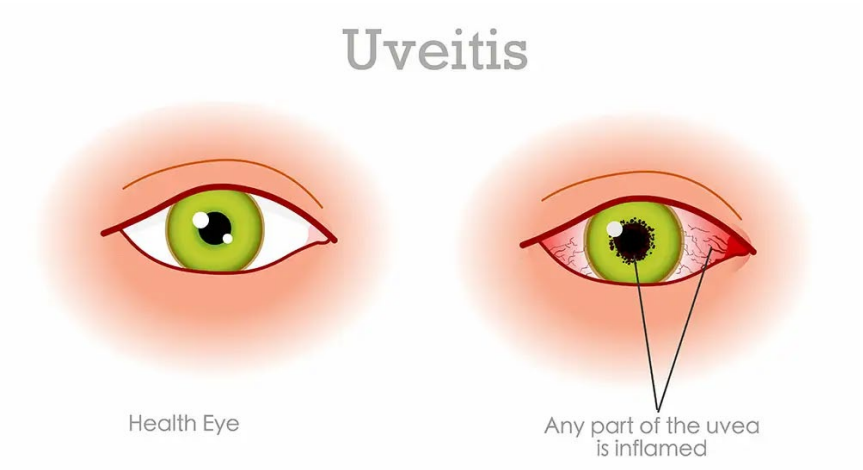
- Infection acquired postnatally is mostly asymptomatic.
- The most common manifestation of acute acquired toxoplasmosis is lymphadenopathy; the cervical lymph nodes being most frequently affected.
- Fever, headache, myalgia, and splenomegaly are often present. the illness may resemble mild flu and is self-limited, although the lymphadenopathy may persist.
- In some cases, there may be a typhus-like exanthema with pneumonitis, myocarditis and meningoencephalitis, which may be fatal.

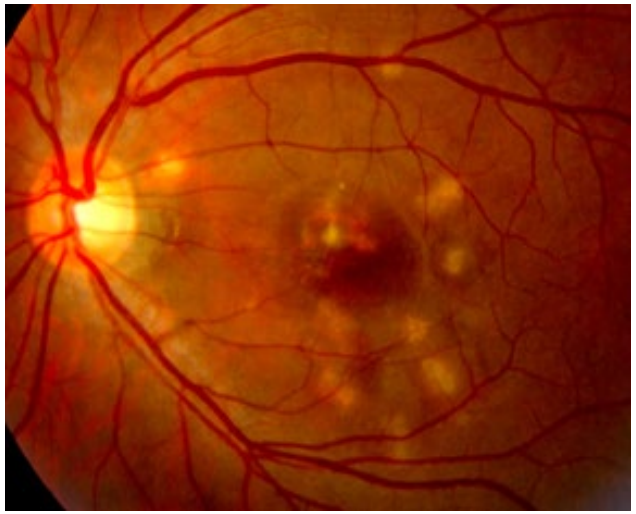
Clinical manifestation

Ocular toxoplasmosis

Is an eye infection caused by Toxoplasma.It can appear as inflammation of the eye such as uveitis, choroiditis, or chorioretinitis.







Laboratory Diagnosis

The diagnosis of acute toxoplasmosis is made mainly by demonstration of trophozoites and cysts in tissue and body fluids and by serology

1. Microscopy

- Tachyzoites and tissue cysts can be detected in various specimens like blood, sputum, bone marrow aspirate, cerebrospinal fluid (CSF), amniotic fluid, and biopsy material from lymph node, spleen and brain.
- Smear made from earlier specimens is stained by Giemsa, PAS, or Gomori methenamine silver (GMS) stain.

Laboratory Diagnosis

2. Serology

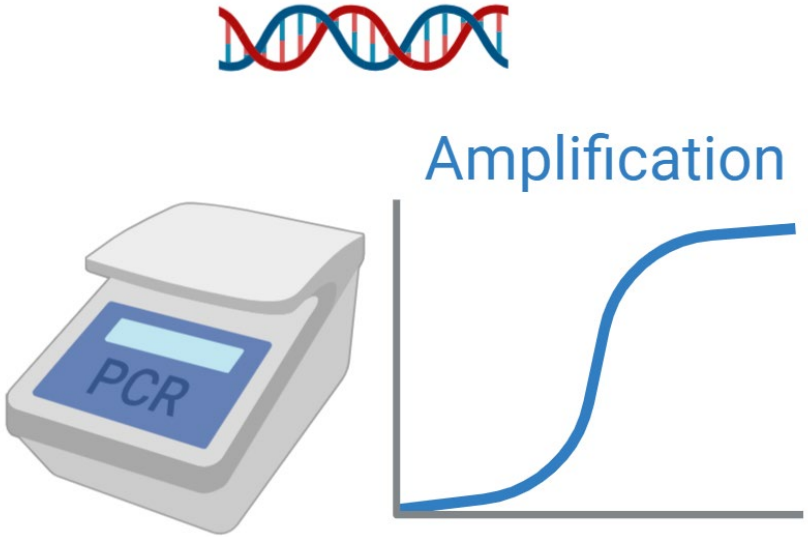
- **Antibody detection:** Diagnosis of acute infection with T. gondiican be made by detection of thesimultaneous presence of lgM and lgG antibodies.

- **Antigen detection:** Detection of antigen by ELISA indicates recent Toxoplasma infection.

Laboratory Diagnosis

3. Molecular Method

- Deoxyribonucleic acid (DNA) hybridization techniques and polymerase chain reaction (PCR) are increasingly used to detect Toxoplasma from different tissues and body fluids.
- B, gene of T. gondii can be detected by PCR of the amniotic fluid in case of congenital toxoplasmosis.



Laboratory Diagnosis

Imaging

Magnetic resonance imaging (MRI) and computed tomography (CT) scan are used to diagnose toxoplasmosis with central nervous system involvement.

Ultrasonography (USG) of the fetus in utero at 20-24 weeks of pregnancy is useful for diagnosis of congenital toxoplasmosis.

Treatment

- **Congenital toxoplasmosis:** Pyrimethamine (1 mg/kg daily) + sulfadiazine (100 mg/kg) with folinic acid for 1 year; corticosteroids if chorioretinitis is present.
- **Immunocompetent patients:** Usually no treatment unless severe; ocular disease → pyrimethamine + sulfadiazine or clindamycin for 1 month, with folinic acid to prevent marrow toxicity.
- **Immunocompromised patients (e.g., AIDS):** With CD4 <100/µL, give prophylaxis against toxoplasma encephalitis. Trimethoprim–Sulfamethoxazole is first choice