Toxoplasma gondii

Overview and Incidence

The incidence of toxoplasmosis varies greatly by country and by age, but may affect up to one-third of the global human population.

The majority of immunocompetent adults,

pregnant women and children infected with *Toxoplasma gondii* experience no or mild symptoms during acute infection.

Infants of women :This incidence ranges from who sero convert during pregnancy may develop

congenital toxoplasmosis. one to ten per 10,000 live births. Immuno compromised

individuals are at risk for reactivation of latent infection, including potentially fatal encephalitis.

Causes and Risk Factors

The house cat and other members of the family Fieldale serve as definitive hosts in which the sexual stages of the parasite develop.

The life cycle of *Toxoplasma gondii* begins when a cat ingests toxoplasma-infected tissue from an intermediate host,usually a rodent.
Tissue cysts within the muscle fibers or brain are digested in the cat's digestive tract.
The parasite then undergoes sexual development, multiplies in the intestine of the cat and is eventually shed in cat feces, mainly into litter boxes and garden soil. produce a resistant spore (oocyst) which is able to survive between hosts.- have very complex life cycles involving asexual and sexual phases

Human infection may be acquired in several ways:-

A) Ingestion of undercooked infected meat

B) Ingestion of the oocyst from fecally. contaminated hands or food

C) Organ transplantation or blood transfusionand Transplacental transmission

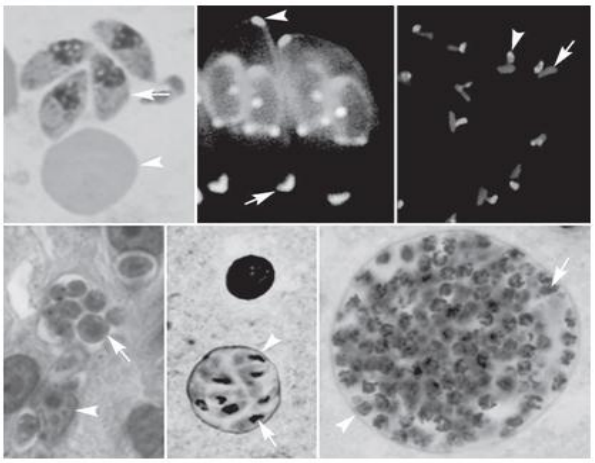
Toxoplasma gondii stages

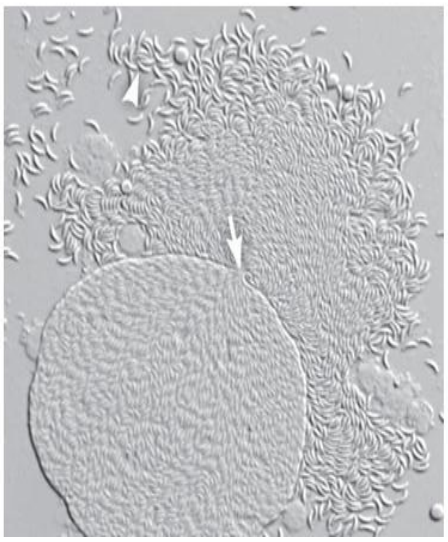
1-Tachyzoite: The quickly multiplying forms are responsible for initial spread of infection, single(free or intracellular)or in masses (pseudocyst) If pregnant women becomes infected tachyzoites can infect the fetus via the blood stream.

2-_ Bradyzoite: Similar to tachyzoites but less active (metaboilically)in tissue cyst.

3-Both oocyst and tissue cyst transform to tachyzoites shortly after ingestion. Tachzoites localize in neural and muscle tissues and developed into tissue cyst bradyzoites.

4-Immature oocyst. Sporulated oocyst.



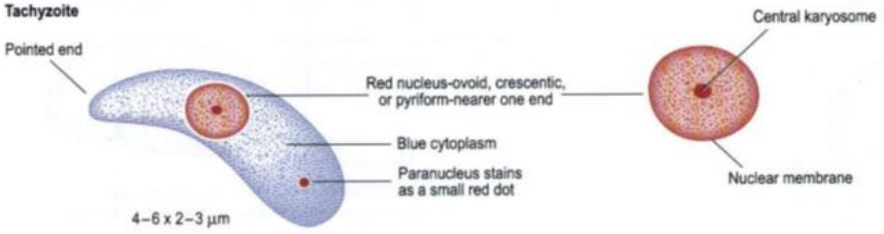


Tissue protozoa

Toxoplasma gondii

Toxoplasma has a very wide mammalian host range.

Morphology



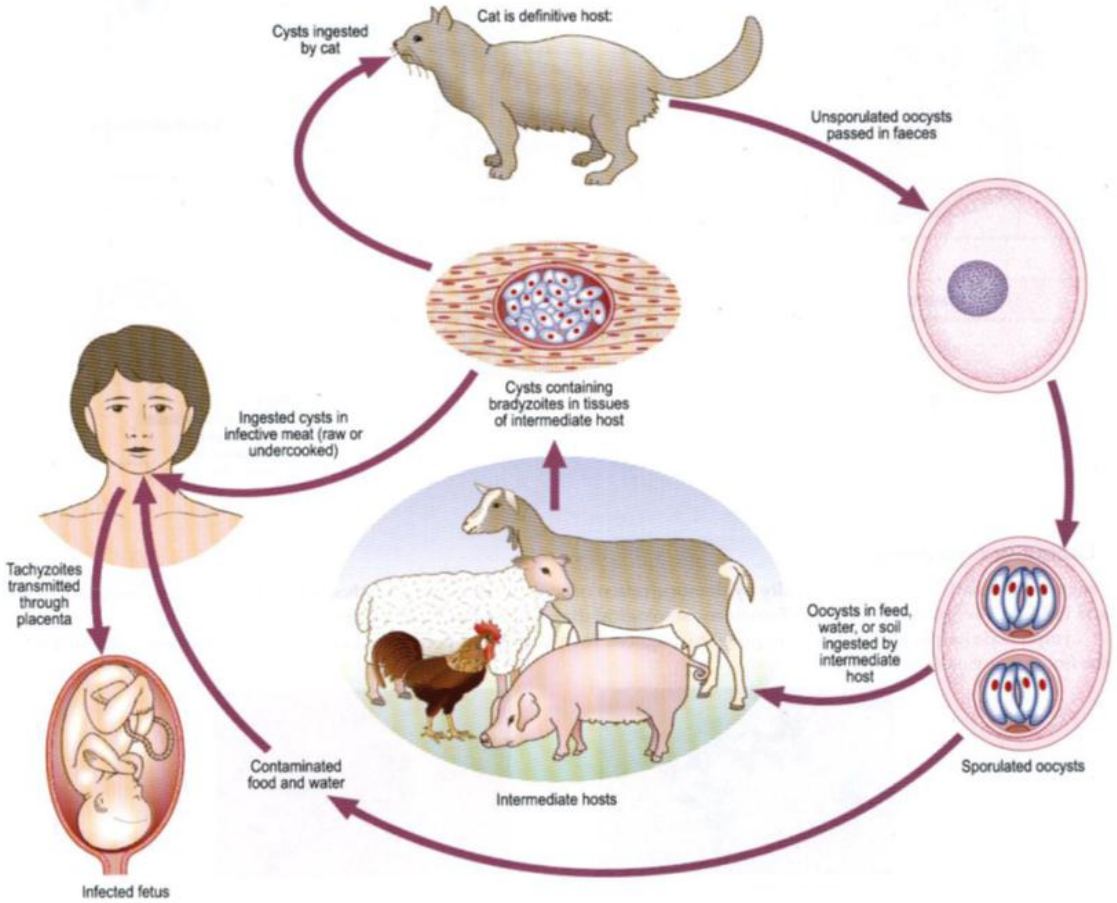
Habitat

Tachyzoites: single (free or intracellular) or in masses (pseudocysts)

In nucleated cells, especially macrophages

Bradyzoites (similar to tachyzoites but less active metabolically) in tissue cysts

Life cycle



Pathology

T. gondii invades numerous organs, infecting a broad spectrum of cell types.Tachyzoites infect macrophages and are disseminated through the blood to many organs, where they invade, asexually multiply and cause cellular disruption, leading to cell death. The resulting necrosis attracts inflammatory host cells, such as lymphocytes and monocytes. This inflammatory response that causes the major pathology in infected individuals.

As host resistance develops, usually around 3 weeks post infection, tissue cysts may form in many organs, primarily in brain and muscle. These quiescent cysts enable *Toxoplasma gondii* to evade the adaptive host immune. As tissue cysts periodically rupture, the released bradyzoites are killed by the host immune system.

If immune surveillance becomes compromised, such as due to chemotherapy or AIDS, these bradyzoites develop into tachyzoites, causing active toxoplasmosis.

Clinical Manifestations

Over 80-90% of primary infections produce no symptoms. The incubation period for symptoms is 1 to 2 weeks.
Mild symptoms of primary infection include localized, painless cervical or occipital lymphadenopathy, usually persisting 4-6 weeks, or nonspecific symptoms including myalgia, headache, rash or sore throat that persist for one month or longer.Recently, newer more virulent strains causing severe symptoms in immunocompetent individuals have been reported.

Congenital toxoplasmosis:

is caused by infection with *Toxoplasma gondii* in a pregnant woman. Infants born to women who were infected before conception do not develop disease due to protection by maternal antibodies. In contrast, new infections with detectable maternal parasitemia are associated with up to a 50% transmission rate to the fetus.

The likelihood of transmission and severity of disease in the fetus are inversely proportional. Mothers who develop acute toxoplasmosis in the first trimester have a much lower fetal transmission rate than in the third trimester, but fetuses exposed early are at much higher risk for severe symptoms or death and spontaneous abortion.

Diagnosis :-

It is relies on either indirect serological tests or direct detection of the organism. Serologic tests, indicating recent or past infection, are most effective in immunocompetent adults who are able to mount a humoral response to the parasite.

These include ELISA, IFA, complement fixation and in the past, the Sabin-Feldman dye test to detect IgG antibodies develop 1-2 weeks post infection and then persist and therefore will not distinguish between recent and past infection.

Rising titers on serial examination may be indicative of active infection. The presence of a high IgM titer in the absence of a significant IgG titers indicates early stages of primary infection.

A negative IgM titer is helpful for ruling out recent infection. However, due to considerable variability in tests and a high false positive rate, a positive IgM test should be verified in a reference laboratory.

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In infants, it is important to distinguish between maternal antibodies found in a non-infected infant versus a high titer of antibodies being produced by an infected infant. In this regard, the simultaneous measurement of maternal and infant IgG antibodies specific for the parasites is critical An infant: maternal IgG ratio of four or higher is indicative of new infection. This test should be repeated in the infant at 4 months of age.

In addition, the presence of high titers of specific IgM antibodies in the infant's serum is diagnostic. Current procedures allow diagnosis of an active infection in the fetus in utero by means of PCR of amniotic fluid obtained by amniocentesis.

The majority of infants will appear normal on prenatal ultrasound although findings may include intracranial calcifications, ventricular dilatation,

hepatic enlargement, increased placental thickness and ascites.

Treatment of new infections in pregnant women is controversial because of the toxicity of the medications, but treatment is still advocated.

Congenitally-infected New borns are treated aggressively Medications to treat the infection include: pyrimethamine (25-100 mg/d × 3-4wks) plus either trisulfa pyrimidines or sulfadiazine (1-1.5 gm qid 3-4 wks).