Lec.5 **Protozoa** **3rd class**

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Toxoplasma gondii

Toxoplasmosis is a disease of cats as well as other mammals and birds Caused by a parasite called Toxoplasma gondii (*T. gondii*) . Toxoplasma infection is common , but full-blown disease is rare. T.gondii is important because virtually all warm-blooded animals, including man, can become infected with it. Domestic, wild, & feral cats can transmit toxoplasma infection to humans. This parasite infect large number of the vertebrates host including man , mammals, birds and reptile.

- **Habitual: epithelial cells of small intestine or other tissue of the host.**

***Toxoplasma* stages**

The Infective stage: Sporozoite comes from three forms

a) Sporozoites within mature oocyst: this is found in feces of cat and other felidue family ((including cats, tigers, pumas Jacques ...ext) this found in fresh passed stool sample of cat with double wall of sub-spherical sporoblast contain a nucleus and in soil outside the body, the immature oocysts will develop sporoblast and from mature oocysts with 2 sporocysts each with 4 crescentric sporozoites.

b) Tachyzoites with pseudocyst: it is found in the acute stage of the parasite in any reticuloendothelial system or parenchymal tissue of man or other mammals, their number is usually 6-16 in one cell.

c) Bradyzoites with true cyst contain large number of 50 or more, it is present in chronicstage of the parasite.

In case of mature oocyst, it found only in cat family, but other are found in othermammals including cats also.

Life cycle of *Toxoplasma gondii*

Divided into:

a) Intestinal or enteroepithelial or isosporian life cycle, this found completed in cat and other felidne family (tiger, lion ...ect.)

Intestinal life cycle: in cat and other felidue family only: they get infection by ingestion of mature oocysts from infected cat; in the small intestine of the cat the sporozoites are released. Some of these sporozoites will initiate the isosporian phase (asexual &sexual multiplication) .More or less resemblance to that of *Isospora belli*. The merozoites will continue one or more Schizogony cycle and these continue to form both male and female gametocytes, so there will be male gametocyte that will divided a lot of (large number of male gametocytes) while the female gametocyte will form one ovum only. Male gametocyte will fertilize the ovum and form the zygote which secrete and surround them selves by a wall to form the immature oocysts and then shed out epithelium lining of small intestine and go out with feces to outside where maturation take place (sexual and asexual cycle need21-24 days). & after maturation the oocysts is ready to infect other cats.

b) Extra-intestinal or toxoplasma phase.

while the other sporozoites take their way through intestinal wall & go by blood stream, At acute stage ,they go to paranchymal cell &RES (Mcrophage, Neutrophil & Monocytes), in these cells , they divide to form Tachyzoites (pseudocyst) contain multiplied asexual (contain 6-16 Tachyzoites &again invade other paranchymal cells or RES with development of immunity, the multiplication of Tachyzoites will ceased down & form bradyzoites surrounded by a cystic wall & contain 50 or more bradyzoites) in chronic stage form in Brain ,Eye, muscle Lung of

infected cat, & viable for about one year ago to small intestine and release their sporozoites and cause intestinal and extra-intestinal phase maturation **the oocysts is ready to infect other cats.**

Cats -> final host

Mature oocyst -> infective stage

■1. I.M.H rapture of oocyst.

2. Liberation of sporozoites.

3. Penetration of sporozoites to epithelial cells and traveled various organs of The body such as brain, heart, liver, muscle.

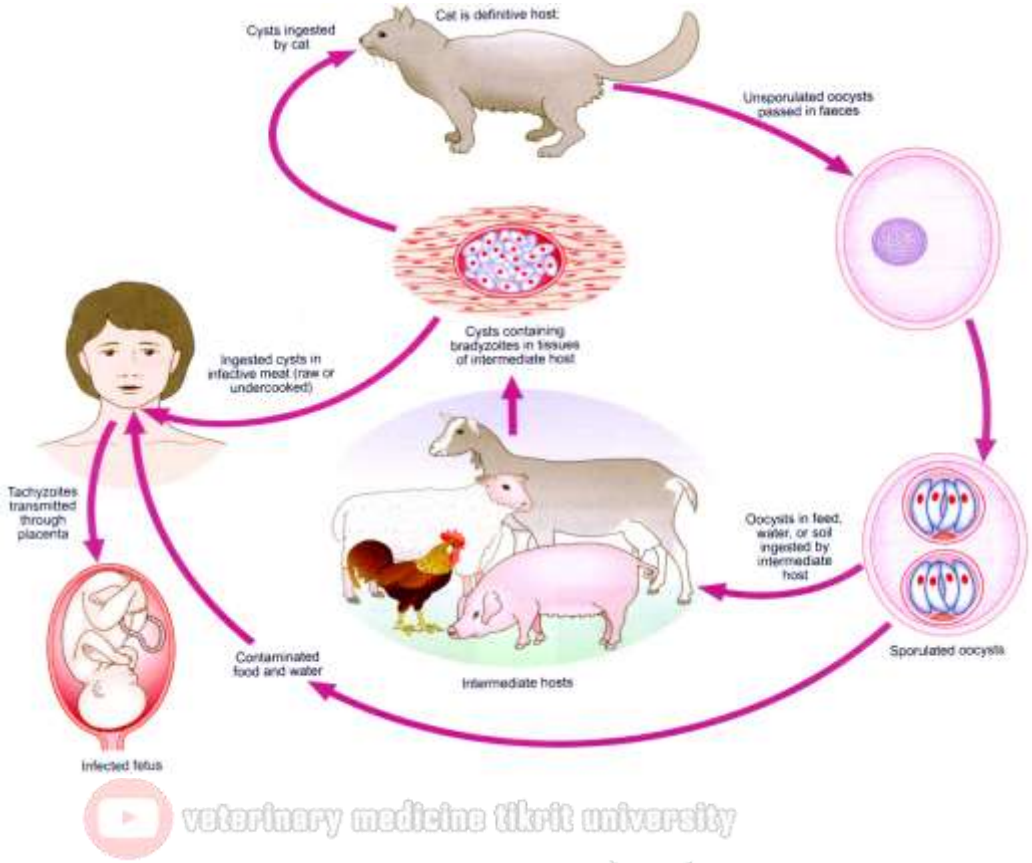
In chronic s stage Bradyzoites is found in brain, eyes, muscle and lung of infected cats and remain viable for 1 year. So the cat and its family considered as complete host because both (intestinal and extra-intestinal take place in it). If the mature oocyst from the cat is ingested by another host, man, other mammals, birds and reptile they become infected with the parasite of mature oocyst, in the small intestine is ruptured and sporozoites are released.

All of the sporozoites will take their way through intestinal wall to any parenchymal and other RES, in the acute stage they will form tachyzoites and in the chronic stage they will form brady zoites.

Cat could be infected by eating raw meat or undercooked meat or meat of other *mammals containing pseudocyst or true cyst, in the small intestine either* Tachyzoites or bradyzoites are released and some of them will initiate intestinal or i.sosporian phase and other will initiate extra-intestinal phase. •

In man and other carnivores family, if they ingest undercooked meat cow and sheep containing true and pseudocyst, in small intestine, all are released and all of them will pass through the intestinal wall and form tachyzoites in acute stages and bradyzoites in chronic stages.

In case of man and other mammals are called incomplete host because only asexual life cycle occur. In case of man the infection is in blind end so man is not regarded as a source of infection but only if he is eaten by a carnivore in forest.



Transmission of *T.gondii*

Major Routes of Transmission:

1-Ingestion of under cocked meat contaminated with *T.gondii.*

2-Ingestion of contaminated H2O.

3-Because the parasite can cross Transplacentally ,so it can infect the fetus from infected

mother.

Minor Routes of Transmission.

1-Blood transfusion from donors to recipient

2-Organ Transplantation.

3-Drink not pasteurizing milk from infected cow

Pathogenesis of toxoplasmosis

Most cases of toxoplasmosis in human are probably acquired by the ingestion of either tissue cysts with infected meat or oocysts in food contaminated with cat feces. Bradyzoites from the tissue cyst & Sporozoite released from oocysts penetrate the intestinal epithelial cells &multiply in the intestine, *T. gondii* may spread both locally to mesenteric lymph nodes &to distant organs by invading lymphatic's &blood.

The clinical picture is determined by the extent of injury to these organs, especially to vital & vulnerable organs, such as eye, heart &adrenals. *T. gondii* does not produce toxins, necrosis is caused by intracellular multiplication of Tachyzoites. Opportunistic toxoplasmosis in AIDS patients usually represents reactivation of chronic infection. The predominant lesion of toxoplasmosis ,encephalitis in these patients is necrosis which results in multiple abscesses, some as large as a tennis ball.

Symptoms

The symptoms are divided into two main groups:

a-Neonatal (Congenital) Toxoplasmosis

b-Acquired (Postnatal) Toxoplasmosis

a-Neonatal Toxoplasmosis: If the fetus get infected transplacentally from asymptomatic mother during 3rd trimester of pregnancy. At acute stage; it may lead sporadic abortion (onlyone) or still death, at birth or shortly after that (1-2) weeks,

the infant shows signs &symptoms of Sabine's Tetrad & these are:

1-Intracerebral calcification

2-Retino-choroditis (birth)

3-Hydrocephalous

4-Microcephalous

5-Psychomotor disturbance

6-Generalized convulsion

b-Acquired (post-Natal) Toxoplasmosis

Acquired (post-Natal) Toxoplasmosis: 90% of man &animals, show no symptoms or signs and the other 10% have the most common forms(4 signs)

a-Lymphadenitis with fever, headache &malacia, the lymph nodes is either superficial or deep &mostly the L.N of the neck region, also(1-2 weeks) ,Splenomegaly, Erythematous rash.

b- Typhus like Xanthomeatus, disease produce Myocarditis ,Meningeocephalitis, Atypical pneumonia……..death occur.

c-In rare cases , primary involve CNS &death occur.

d-Retino-choraditis of non-congenital infection in which the ocular lesion begins in Retina &spread to the choroid and in sever rare cases it causes enucleation of the eye (one eye) **while in congenital it involves both eyes.**

Diagnosis

1) The demonstration of the Toxoplasma gondii organism in blood, body fluids, or tissue.

2) **Detection** of **Toxoplasma gondii antigen** in blood or body fluids by enzyme-linked immunosorbent assay (**ELISA**) technique.

3) **The Sabin-Feldman dye test:** is a sensitive and specific neutralization test. It measures IgG antibody and is the standard reference test for toxoplasmosis. High titers suggest acute disease.

4) **Serologically**: IgM fluorescent antibody test detects IgM antibodies within the first week of infection, but titers fall within a few months.

5) **P**olymerase **C**hain **R**eaction on body fluids, including CSF, amniotic fluid, and blood.

6) **Skin test** results showing delayed skin hypersensitivity to Toxoplasma gondii antigens.

7) **Antibody levels** in aqueous humor or CSF may reflect local antibody production and infection.

8) **Animal inoculation**: inoculation of suspected infected tissues into experimental animals.

9) **Culture**: inoculation of suspected infected tissues into tissue culture**.**

Treatment

Most people will be treated with a combination of medicines called pyrimethamine, sulfadiazine and folinic acid. These medications usually need to be taken for at least four to six weeks. Pregnant women may be treated with spiramycin .

SARCOCYSTIS sp.

•Important stage of genus found in the intermediate host

•Both schizontsin the endothelium of the blood vessels and the bradyzoitcysts in the skeletal and cardiac muscles .

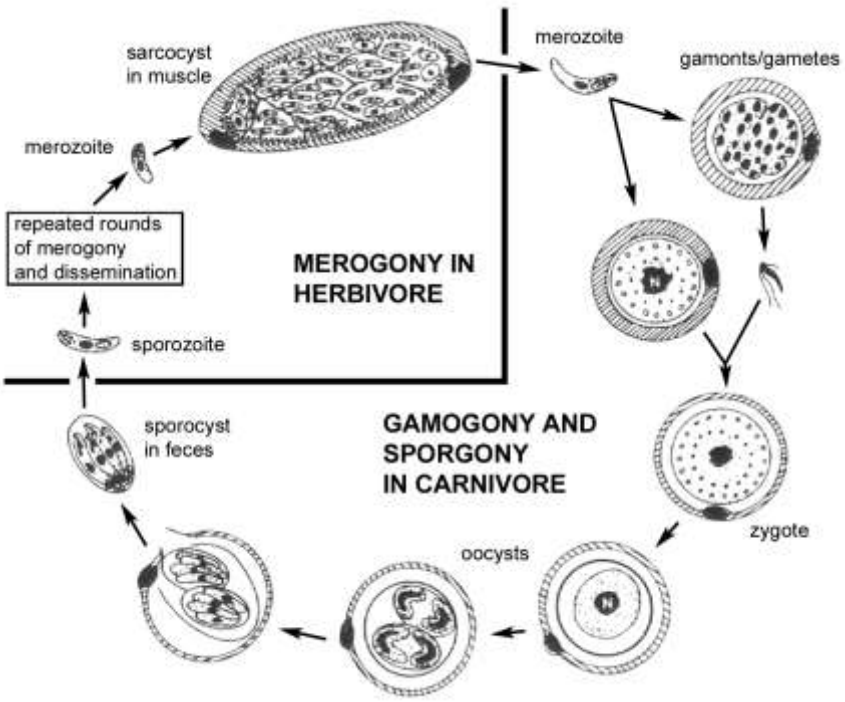
•Final hosts :dogs ,cat, and man

•Intermdiate host : ruminants ,pigs, and horses

•Site in final host : small intestine

*****Site in intermediate host :schizontsin endothelial cells of the blood vessels ,large cysts contain bradyzoitin muscle

Life cycle:



Clinical sign

Infection in the final host is normally non-pathogenic

•In the heavy infection of the intermediathost:

•Anorexia

•fever,

•anemia,

•loss of weight,

•recumbancy ,

•in lambs dog sitting

In the catle:

• Sarcocystis spp infections are quite prevalent in farm animals; In cattle severely affected by *S. cruzi*, the signs include fever, anorexia, cachexia, decreased milk yield, diarrhea, muscle spasms, anemia, hyperexcitability, weakness, prostration, and death. Cows infected in the last trimester of pregnancy may abort.

Pathology

•Some species of Sarcocystis are pathogenic to the herbivore intermediate host.

***** Acute lesions characterized by hemorrhage, edema, and necrosis are associated with the maturation of second generation meronts. Macroscopic lesions observed postmortem may include generalized serous atrophy of fat, excessive yellowish fluid in all body cavities, watery blood, petechicalhemorrhagein the heart and pericardium, serosa of the gastrointestinal tract and urinary bladder, edema and hemorrhage of lymph nodes, and alternate pale and dark striping or mottling of skeletal muscles. Microscopically, hemorrhage may be seen in all organs, and mononuclear cell infiltration into the perivascular tissues of the hear, skeletal muscles, lung, liver, and kidney may be mild to severe. Regenerative changes are most often associated with the myocardium.

*Chronic lesions charactgerized by muscle atrophy and myositis are associated with mature sarcocysts. Specific macroscopic lesions may not always be seen postmortem. Microscopic lesions may include myositi psand myocarditis. Most definitive hosts are clinically unaffected by Sarcocystis infection