

Toxoplasma

		Parasite			Phylum	Subphylum	Superclass	Class	Subclass	Order	Suborder	Genus
					Sarcomasti- gophora	Mastigophora (having one or more flagella)		Zoomastigopho- rea		Kinetoplastida	Trypanosomatina	Trypanosoma Leishmania
Protozoa (unicellular)				Helminths (multicellular)						Retortamonadida		Retortamonas Chilomastix
Kingdom-Protista				Kingdom-Animalia						Diplomonadida	Enteromonadina Diplomonadina	Enteromonas Glardia
-	-		•							Trichomonadida		Trichomonas Dientamoeba
Amoebae	Flagellates	Sporozoa	Ciliates			Sarcodina (pseudopodia present)	Rhizopoda	Lobosea	Gymnamoe- bla	Amoebida	Tubulina	Entamoeba Endolimax Iodamoeba
Entamoeba Naegleria	Giardia Trichomonas	Plasmodium Babesia	Balantidium	201						Schizopyrenida	Acanthopodina	Acanthamoeba Naegleria
	Nematodes	Toxoplasma		Trematories	Apicomplexa			Sporozoea	Coccidia	Eucoccidia	Emeriina	Crypto- sporidium hospora Sarcocystri Toxoplasma
	Trematores			Temptoodo							Haemosporina	Plasmodium
									Piroplasmia	Piroplasmida		Babesia
	Ascaris Ancylostoma	Ascaris Taenia Ancylostoma Echinoco		Fasciola Schistosoma	CiBophora			Kinetofragmino- phorea	Vestibuliferia	Trichostomastida	Trichostomatina	Balantidium
					Microspora			Microsporea		Microsporida	Apansporoblas- tina	Enterocytozoon Encephalito- zoon Microsporum

The coccidia are unicellular protozoa and belong to the

Phylum Apicomplexa Class sporazoea

subclass cocccidia

They live intracellularly, at least during a part of their life cycle and at some stage in their life cycle, they possess a structure called the apical complex.

All coccidian have a sexual sporogonic phase and an asexual schizogonic phase.

Many of them also show an alteration of hosts; a definitive host and an intermediate host

Toxoplasma Gondii

Toxoplasma gondii is an obligate intracellular coccidian parasite.

Toxoplasmosis is a zoonotic infection of humans and animals, caused by the opportunistic protozoan (Toxoplasma gondii), a parasite belonging

phylum Apicomplexa

Class sporazoea

subclass cocccidia .

<u>History</u>

The first described in 1908 by Nicolle and Manceaux in a small North American rodent called gondii.

Its importance as a human pathogen was recognized much later, when Janku in 1923 observed the cyst in the retina of a child with hydrocephalus

The discovery of the life cycle of the parasite has been completed only in 1970 when domestic cats were recognized as the definitive hosts.

In 1980 T.gondii was regarded as important opportunistic infection in immune compromised subjects



1



The name Toxoplasma is derived from the Greek word 'Toxon' meaning arc or brow referring to the curved shape of the trophozoite

Geographic Distribution:

toxoplasmosis is one of the most common human infections throughout the world.

It has been estimated that 30% of the world population has been infected .

prevalence depends on the locale and the age of the population.

Generally, hot arid climatic conditions are associated with a low prevalence of infection.

In the United States and most European countries, the seroprevalence increases with age

in the United States, 5-30% of individuals 10-20 years old

and 10-65% of those >50 years old .

A high prevalence of infection in China 15%, 20% in UK

in France(50-75%) has been related to a preference for eating raw or undercooked meat.

while a high prevalence in Central America has been related to the frequency of stray cats in a climate favoring survival of oocysts and soil exposure

prevalence of T. gondii total Abs among young women attending the public health laboratory in Mosul was around 50% with only 10% positive IgM anti-Toxoplasma Abs.

48% of Patients with Leukemia in Mosul showed presence of anti-T.gondii Abs in their sera and 28% of them were positive for IgM Abs.

seroprevalence in Ramadi show (wives 30.7%), while 13.1% in husbands only.

in Duhok showed 59% of pregnant ladies with bad obstetric history (abortion, or dead fetus)

anti- Toxoplasma Abs by latex agglutination test and IgM Abs were found in 1% of them

<u>Morphology</u>

T. gondii occurs in 3 forms :

€ Trophozoite

€ Tissue cyst

€ Oocyst.

The trophozoite and tissue cyst represent stages in asexual

multiplication (schizogony),

while the the oocyst is formed by sexual reproduction (gmaetogony or sporogony).



All 3 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 the 3 forms are infectious to man.

Trophozoites (Tachyzoites)

The trophozoite is crescent shaped, $3-7 \mu m$ in length with one end pointed and the other end rounded

The nucleus is ovoid and is situated at the blunt end of the parasite

Electron microscopy reveals an apical complex at the pointed end The trophzoite stains well with Giemsa stain, the cytoplasm

appearing blue and the nucleus red .



3

The actively multiplying trophozoite is seen intracellularly in various tissues during early acute infection

Extracellular trophozoites can also be seen in impression smears

It can invade any nucleated cell and replicate within cytoplasmic vacuoles by a process called endogony (internal budding), where in 2 daughter trophozites are formed, each surrounded by a membrane, while still within the parent cell.

When the host cell becomes distended with the parasite, it, releasing disintegrates, releasing the trophozoites that infect other cells

During acute infection, the proliferating trophozoites within host cell may appear rounded and enclosed by the host cell membrane. This is, called pseudocyst or colony and can be differentiated from tissue cysts by staining reactions.

The rapidly proliferating trophozoites in acute infection are called tachyzoites

The trophozoites are susceptible to drying, freezethawing





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 tissue cyst is round or oval, 10–20 µm in size and contains numerous bradyzoites.

The cyst wall is eosionophilic stains weakly and the parasites inside are stained deeply with silver,

In contrast to the pseudocyst with periodic acid Schiff (PAS) stain,

The slowly multiplying parasites within the cyst are called bradyzoite

Bradyzoites: Similar to tachyzoites in morphology

Cysts remain viable in tissue for several years.

In immunocompetent hosts, the cysts remain silent, but in the immunodeficient subjects, they may get reactivated, leading to clinical disease.

when the raw or undercooked meat containing the cysts is eaten, infection occurs.

The cyst wall is disrupted by peptic or tryptic digestion and the released parasites initiate infection by invading intestinal epithelial cells.

They reach various tissues and organs through blood and lymphatic dissemination.

Cysts are susceptible to freezing, and thawing, and heat above 60°C.

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 $10-12 \ \mu m$ in diameter.

Each cyst is surrounded by a thick resistant wall.

The oocysts is formed by sexual reproduction (gametogony).

Cats shed millions of oocysts per day in feces for about 2 weeks during the primary infection.

The freshly passed oocyst is not infectious.

They undergo sporulation in the soil with formation of 2 sporocysts, each containing 4 sporozoites with in several days to several weeks the sporulated oocyst is become infective.



Sporulated oocyst



Oocyst is very resistant to environmental conditons and can remain infective in soil for about 3 years

When the infective oocyst is ingested, it releases sporozoites in the intestine, which initiates infection. The incubation period ranges from 5- 24 days. If the cat ingest infective oocyst and initiate intestinal infection, the animal will pass oocyst within 21-24 days

If the cat feed in its tissue cysts, the oocyst will appear in the cat's feces within 5-10 days During acute infections, cats excrete unsporulated oocysts (noninfectious) in their feces ,after several days to several weeks, depending on environmental conditions.

the oocysts sporulate and become infectious. Under favorable conditions (i.e., in warm, moist soil)



A- Unsporulated oocyst B- sporulated oocyct with 2 sporocysts ,4 sporozoites are clear in one sporocyst



I: Toxoplasma gondii. A. Smear from peritoneal fluid of infected mouse, showing crescentic tachyzoites—extracellular trophozoites and intracellular within macrophage; B. Thick-walled tissue cyst containing rounded forms- bradyzoites; C. Oocyst containing 2 sporocysts with sporozoites inside

Risk factors for T. gondii infection in human being:

- 1-having poor hand hygiene.
- 2- seropositive cats in farming areas
- 3-cleaning the cat litter box .
- 4- eating raw or undercooked pork, beef.
- 5- gardening .
- 6- eating raw or unwashed vegetables or fruits.
- 7- contact with soil .
- 8-infrequent washing of kitchen knifes .
- 9-butchers do not always wear gloves during work when handling raw meat



Domestic cats : reservoir source of infection.

Cat feces is the chief source of infection.

All the 3 forms are infectious to man (Tachyzoite, mature oocyst and tissue cyst are the infective stage).

Transmission:

T. gondii infection usually is transmitted from infected cat and domestic animals to man (zooontic infection).

There are several transmission ways

1-Oral transmission: T. gondii infection is acquired by eating raw or undercooked meat (chicken, pork and goat meat) containing the tissue cyst or ingesting food ,water vegetables and fruits contaminated with mature oocysts form in cat feces

2-Congenital transmission: the infection is transmitted from the infected pregnant mother to the fetus, by the tachyzoits passing through the placenta.

The prevalence of T. gondii infection in pregnant women 25% in China, Austra (35%), France (43%) and Brazil (53%)

3-Other modes of transmission: laboratory infection is caused by accidental self-inoculation of tachyzoites, It is less common.

The infection may be transmitted by blood transfusion, unpasteurised milk, and organ transplantation





T. gondii completes its life cycle in 2 hosts .

The widest range of hosts spread over 200 species of birds, reptiles, and mammals, including humans

<u>Definitive host:</u> Cats and other felines, in which both sexual and asexual cycle takes place.

Intermediate hosts: Man and other mammals, in which only the asexual cycle takes place.

T. gondii has 2 types of life cycles:

€ Enteric cycle

€ Exoentric cycle.

Enteric cycle

Enteric cycle occurs in cat and other definitive hosts .

Both sexual reproduction (gametogony) and asexual reproduction (schizogony) occur within the mucoscal epithelial cells of the small intestine of the cat.

Cat acquires infection by ingestion of tissue cysts in the meat of rats and other animals or by ingestion of oocysts passed in its feces.

The bradyzoites are released in the small intestine and they undergo asexual multiplication (schizogony) leading to formation of **merozoites**

Some merozoites enter extraintestinal tissues resulting in the formation of tissue cysts in other organs of the body.

Other merozoites transform into male and female gametocytes and sexual cycle (gametogony) begins, with the formation of microgamete and macrogamete.

A macrogamete is fertilized by motile microgamete resulting in the formation of an oocyst, which passes through maturation stages (sporulation) in the soil after being excreted from host through feces with in few days or weeks.

A mature oocyst containing 8 sporozoites is the infective form which may be ingested by cats or other mammals to repeat the cycle







Exoenteric cycle

Exoenteric cycle asexual cycle occurs in humans, mice, rats, sheep, cattle, pigs and birds , which are the intermediate hosts.

Humans acquire infection after:

1-Eating uncooked or undercooked infected meat, particularly lamb and pork containing tissue cysts

2-Ingestion of mature oocysts through food, water, or fingers flies) (contaminated with cat feces directly or indirectly

3-Intrauterine infection from mother to fetus (congenital toxoplasmosis)€€

4-Blood transfusion or transplantation from infected donors.

5- laboratory infected after contact with contaminated needles or glassware or with infected tissue.

Sporozoites from the oocysts and bradyzoites from the tissue cysts enter into the intestinal mucosa and multiply asexually and tachyzoites are formed (endodyogeny).

Tachyzoites continue to multiply and spread by lymphatic system and blood.

Some tachyzoites also spread to distant extraintestinal organs like brain, eye, liver, spleen, lung, and skeletal muscles and form tissue cysts.

The slowly multiplying forms inside the tissue cysts are known as bradyzoites, which remain viable for years.

The dormant bradyzoites inside the cyst may be reactived in immune suppression causing renewed infection in the host. Human infection is a dead end for the parasite





Pathogenicity and Clinical Features

The outcome of Toxoplasma infection depends on the immune status of the infected person and the strain of the parasite.

T. gondii is found inside the reticuloendothelial cells and many other nucleated cells of the host.

The disease (toxoplasmosis) occur in the immunocompetent hosts or in the immuno- compromised hosts.

The rate of the organ or tissue to get parasite infection depends on

1- Vascular supply

2-Immunity (local and general)

3- The regenerative ability of the host cells.

Toxoplasmosis has acquired great importance as one of the major fatal complications in acquired immunodeficiency syndrome (AIDS).

-Most human infections are asymptomatic (90%).

-Clinical toxoplamosis may be congenital or acquired (10%)

In the acute infection, the proliferation of tachyzoites in the gastro-intestinal tract as well as in the extra-intestinal sites, cause disruption and death of cells, resulting in the foci of necrosis, surrounded by an intense mononuclear cell reaction.

Parasitology

10



The development of both the humoral and cell mediated immunities in the immunocompetent hosts, resolve the acute infection and associated with the disappearance of tachyzoites from various tissues, especially from the extra-neural tissues and the formation of tissue cysts.

The tachyzoites may persist in the (CNS) and even in the eye due to the absence of circulating antibodies in the tissue

In the immunodeficient hosts and even some apparently normal hosts, the acute infection does not resolve but progress to cause severe necrotising lesions such as acute necrotising encephalitis, pneumonitis and myocarditis, even death.

The presence of cysts in many organs through out the life of the host is probably lead to clinicle feature of the infection.

In chronic infection, these cysts remain in a viable latent form and retain their potential for reactivation. Reactivation of chronic infection possibly results from the rupture of cysts.

this causes recurrent parasitaemia frequently seen in some asymptomatic patients with chronic infection

Rupture of cyst also liberates many tachyzoites, which cause toxoplasmosis in the immunodeficient hosts or adults suffering from congenital toxoplasmosis.

The heart, liver, kidney and various other organs in the immunocompetent hosts or immunodeficient hosts are involved in disseminated toxoplasmosis.

These organs show areas of necrosis with or without inflammatory cells and the presence of tachyzoites and cysts.

Toxoplasmosis in man occurs as congenital, acquired, ocular infections in the immunocompetent hosts or an infection in the immunocompromised host

The most common manifestation

of acute toxoplasmosis is cervical lymphadenopathy.

The nodes may be single or multiple, are usually not tender lymphadenopathy occurs in 20–30% of symptomatic patients. Between 20-40% of patients with lymphadenopathy also have headache, malaise, fatigue, and fever.

A smaller proportion of symptomatic individuals have myalgia, sore throat, abdominal pain, maculopapular rash, meningoencephalitis, and confusion.

Rare complications associated with pneumonia, myocarditis, encephalopathy, pericarditis, and polymyositis.

Signs and symptoms associated with acute infection usually resolve within several weeks.



Congenital toxoplasmosis

Congenital toxoplasmosis results from an acute primary infection acquired by the mother during pregnancy (20%) of women infected with T. gondii develop clinical signs of infection

Congenital toxoplasmosis occurs approximately (20%) of infants born to pregnant women, who acquire the infection during first trimester of pregnancy.

Most infected newborns are asymptomatic at birth, Some develop clinical manifestations of toxoplasmosis weeks, months, and even years after birth

Congenital infection lead to a wide variety of manifestations in the fetus and infant including spontaneous abortion, still-birth, a newborn with classic signs of congenial toxoplasmosis such as hydrocephalus or microcephalus, cerebral calcifications and retinochoroiditis

Women with documented acute toxoplasmosis should be counseled to use appropriate measures to prevent pregnancy for 6 months after infection.

T. gondii infection in pregnant women may cause poor obstetric outcomes such as spontaneous abortion, hydatidiform mole, and sterility

In pregnancy, if the mother becomes infected during the first trimester, the incidence of transplacental infection is lowest (20%), but the disease in the neonate is most severe.

If maternal infection occurs during the third trimester, the incidence of transplacental infection is greatest (65%), but the infant is usually asymptomatic at birth.

Infection of the fetus during last trimester of pregnancy, is more likely to be mild or asymptomatic at birth.

the severity of fetal damage is highest, when infection is transmitted in early pregnancy chronic infection, the tissue cyst may be reactivated during pregnancy and liberate trophozoites, which may infect the fetus

The risk of fetal infection rises with progress of gestation; from 20% in first trimester to 65% in the third trimester.

Acquired toxoplasmosis

Acquired infection with Toxoplasma in immunocompetent persons is generally an asymptomatic infection (90%).

However, 10-20% of patients with acute infection may develop cervical lymphadenopathy and/or a flu-like illness. Fever, headache, myalgia, and splenomegaly are often present.



Rare cases of human gastrointestinal tract infection with T. gondii have presented as ulcerations in the mucosa

The clinical course is benign and self-limited; symptoms usually resolve within a few months to a year

Immunodeficient patients often have central nervous system (CNS) disease but may have chorioretinitis , or pneumonitis .

In patients with AIDS, toxoplasmic encephalitis is the most common cause of intracerebral mass lesions duto reactivation of chronic infection.

Toxoplasmosis in patients being treated with immunosuppressive drugs may be due to either newly acquired or reactivated latent infection

Occular Toxoplasmosis

Acute infection of eye begins as single or multiple foci of necrosis of retina .

Infection with T. gondii is estimated to cause 35% of all cases of chorioretinitis in the United States and Europe.

Both the tachyzoites and tissue cysts are found in the retinal lesions.

Ocular manifestations of congenital toxplasmosis in the new born infants often asymptomatic until the second or third decade of life.

when lesions develop in the eye. Unilateral chorioretinitis with photophobia blurred vision and pain in the eye are the frequent clinical manifestation

Ocular toxoplasmosis

- Diagnosis is usually clinical.
- Usually in congenital or immunocompromised cohorts. However can occur occasionally in immunocompetent adults.
- Supportive evidence of +ve toxoplasma IgG abs
- PCR on vitreous fluid for difficult cases.
- Exclude syphilis infection.





Infection In the Immunocompromised host

All types of T. gondii infections that occur in the immunocompetent hosts, are also seen in the immunocompromised hosts.

The infection is more serious in immunosuppressed patients receiving immunosuppressive therapy for malignancy or persons receiving organ transplantations drugs and AIDS.

toxoplasmic encephalitis is one of the most commonly recognized manifestation of the infection in patients with AIDS.

the disease progresses rapidly and death is the frequent out come of the condition.

The signs and symptoms of acute toxoplasmosis in immunocompromised patients principally involve the CNS .

More than 50% of patients with clinical manifestations have intracerebral involvement.

Clinical findings at presentation range from nonfocal to focal dysfunction.

CNS findings include encephalopathy, meningoencephalitis, and mass lesions.

Patients may present with altered mental status (75%), fever (70%), seizures (33%), headaches (56%), and focal neurologic findings (60%).

Laboratory Diagnosis

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

<u>Microscopy</u>

Tachyzoites and tissue cysts can be detected in various specimens like blood, sputum, bone marrow aspirate, cerebrospinal fluid (CSF), aminiotic fluid, and biopsy material from lymphnode, spleen, and brain.

Smear made from above specimens is stained by Giemsa, PAS, or Gomori methanamine silver (GMS) stain

Tachyzoites appear as crescent-shaped structures with blue cytoplasm and dark nucleus Tachyzoites or cyst can also be demonstrated effectively by fluroscent conjugated antibody technique in tissue biopsy or impression smear.

Presence of only tissue cysts does not differentiate between active and chronic infection. The presence of cysts in placenta or tissues of newborn establishes congenital toxoplasma infection



Toxoplasma can be isolated by inoculating body fluids,

blood, or tissue specimes by intraperitoneal inoculation in mice or in tissue culture.

Mice should be examined for Toxoplasma in their peritoneal exudate after 7–10 days of inoculation.

<u>Serodiagnosis</u> is the main stay for diagnosis of toxoplasmosis.

<u>Antibody detection</u> Diagnosis of acute infection with T. gondii can be made by detection of IgM and IgG antibodies.

Tests include:

€€ Enzyme-linked immunosorbent assay (ELISA)

€€ Indirect fluroscent antibody test (IFAT)

€€ Latex agglutination test

Sabin-Feldman dye test

Positive IgG can be detected as early as 2–3 weeks after infection. Peak level of antibody is observed in blood 4–8 weeks after infection

A positive IgM antibody titer indicates an early primary infection.

The serum IgM titer can be measured by double sandwich IgM ELISA , more specific and sensitive.

Negative IgM titer and postive IgG titer indicate distant infection.

Double sandwich IgA-ELISA test is used for detecting congenital infection in newborns

IgG antibodies :

The IgG for usually appear within 2 weeks of the infection and peak within 1-2 months. However, they decline over months to years .

IgG antibodies avidity testing can be usefull in difficult cases during pregnancy for establishing when infection may have occurred

IgM antibodies can be detected as early as one week after the primary infection, peak within 2-4 weeks, then drop to below the detection limit within a few weeks

The detection of both T. gondii-specific IgM and IgA antibodies after birth confirms the diagnosis of congenital Toxoplasmosis ,because both of them can not cross the placenta Serologic diagnosis in congenital toxoplasmosis based on the persistence of IgG antibody or a positive IgM titer after the first week of life (a time frame that excludes placental leak).



The IgG determination should be repeated every 2 months.

An increase in IgM beyond the first week of life is indicative of acute infection.

Up to 25% of infected newborns may be seronegative and have normal routine physical examinations

Guide to general interpretation of IgG IgM	Toxoplasma gondii serology results					
Negative Negative	No serological evidence of infection with Toxoplasma					
Negative Equivocal	Possible early acute infection or false positive IgM reaction.					
Negative Positive	Possible acute infection or false-positive IgM .					
Equivocal Negative	Indeterminate: obtain a new specimen for testing or retest this specimen for IgG in a different assay.					
Equivocal Equivocal	Indeterminate: obtain a new specimen for both IgG and IgM testing.					
Equivocal Positive	Possible acute infection with Toxoplasma.					
Positive Negative	Infected with Toxoplasma for .1 year.					
Positive Positive	Possible recent infection within the last 12 months, or false-positive IgM reaction.					

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

In AIDS and other immonocompromised patients, antigen detection is very useful.

Detection of antigen in amniotic fluid is helpful to diagnose congenital toxoplasmosis

<u>Skin test of Frenkel</u> Diluted toxoplasmin is injected intradermally and delayed positive reaction appears after 48 hours. This test is not very reliable for diagnosis of toxoplasma

<u>Molecular Methods</u> DNA hybridization techniques and polymerase chain reaction (PCR) are increasingly used to detect Toxoplasma from different tissues and body fluids.

T. gondii can be detected by PCR of the aminiotic fluid in case of congenital toxoplasmosis.

<u>Imaging</u> Magnetic resonance imaging (MRI) and computed tomography (CT) scan are used to diagnose toxoplasmosis with (CNS) involvement.

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

<u>Sabin-Feldman dye test.</u> the first serological method to be described by Sabin and Feldman (1948) to detect circulating antibodies in toxoplasmosis

Dye test is a highly sensitive and specific test with no false positives reported.

it is now used only in a reference laboratory as a reference serological test.



16



Microscopy	Serodiagnosis	Molecular	Imaging	Others	1
achyzoites and ssue cysts detected blood, sputum and one marrow aspirates tains used: Giemsa PAS GMS	Antibody detection: Test for detecting IgG antibody: ELISA IFAT Latex agglutination test Sabin-Feldman dye test Test for detecting IgM antibody: Double sandwich IgM ELISA IgM-ISAGA Test for detecting IgA antibody: Double sandwich IgA ELISA Antigen detection: busic for detection: for detection: busic for detection: busic for detection: for d	diagnosis • PCR	MRI and CT scan for central nervous system involvement USG for congenital toxoplasmosis	Animal inoculation Skin test of Frenkel	

Treatment

<u>Congenital Toxoplasmosis</u> Neonates with congenital infection are treated with oral pyrimethamine (1 mg/kg) daily and sulfadiazine (100 mg/ kg) with folinic acid for 1 year.

Systemic corticostoriod may be added to reduce chorioretinitis

Immunocompetent Patients and older children, who have only lymphadenopathy, do not require specific therapy unless they have persistent, severe symptoms

Patients with ocular toxoplasmosis are treated for 1 month with pyrimethamine plus either sulfadiazine or clindamycin

Prophylaxis and control

Tatis in bost - -

1-Individuals at risk, particularly pregnant women, children, and immunocompromised persons should avoid contact with cat and its feces.

2- Proper cooking of meat (Meat cooked to 50-60 C° for 4 – 6 minutes or freezing at – 20 C° for 48 hours)

3- Proper washing of hands and washing of vegetables and fruits before eating.

4- Blood or blood products from seropositive persons should not be given and screening for T. gondii antibody should be done in all blood banks. The seroprevalence of T. gondii in blood donors in China (20%) in Egypt (59%) and Malaysia (28%)

5-It is difficult to control toxoplasmosis because of wide range of animal reservoirs.

6-Some occupations required people to have contact with animals and meats and these frequently possess higher risk of infection with the parasite need education ,such as dairy & slaughterhouse & meat-processing workers, veterinarians, meat sellers and cooks.

7-Detection of antibodies is very important for pregnant women and women of childbearing age.

This is an effective way to find the infection, and then to provide treatment. It is also an efficient way to stop congenital toxoplasmosis in newborns

Currently, there is **no effective vaccnine available for humans.** a genetically engineered vaccine is under development for use in cats