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Review article

Toxoplasmosis: Prevalence, Aetiology, Symptoms, Diagnosis, and Treatment

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ABSTRACT

Toxoplasmosis is a worldwide contagious disease of humans and other warm-blooded animals, including birds. *Toxoplasma gondii (T. gondii)* is an obligate intracellular protozoan parasite responsible for Toxoplasmosis disease, which is clinically manifested chiefly in pregnant women and people with weakened immune systems. Clinical complications include abortion and stillbirths, encephalitis, pneumonia, brain and eye damage, and neonatal mortality. The genus *Toxoplasma* was first proposed in 1908 by Nicolle and Manceaux following the identification of asexual stages of similar parasites in the tissues of birds and mammals, and merozoites in the blood of North African rodents, *Ctenodactylus gundi.* At about the same time, Splendore independently described *Toxoplasma* in laboratory rabbits in Sao Paulo, Brazil, and Darling probably found it in man in Panama in the same year. Although several species were named, during the 1930s, it was shown that these were identical to the type species *T. gondii.* During the 1960, scientists provided evidence for the coccidian nature of the parasite. Then, between 1960 and 1970, the heterogeneous life cycle was elucidated by the discovery of sexual stages in the small intestine of cats, which followed the induction of infection in intermediate hosts by inoculation with cat feces. This review seeks to study the parasite and its responsibility to cause disease, condition prevalence, clinical manifestations accompanied by infection, and treatment protocols.

Introduction

Toxoplasmosis was first discovered in 1908 by Splendor in Brazil and Nicolle and Manceaux in Tunisia, who found the parasite in the blood, spleen, and liver of the North African rodent [1]. The first case of congenital toxoplasmosis, including ocular disease, was probably observed in 1923 [2]. The clinical importance of *Toxoplasma gondii* (*T. gondii*) infection was established during the 1930s with the recognition of *T. gondii* as the etiological agent of encephalomyelitis in neonates, and the description of the classic triad of human congenital toxoplasmosis: retinochoroiditis, hydrocephalus, and encephalitis with cerebral calcification [3, 4].

In 1939, the protozoan parasite was identified in tissues of a congenitally infected infant [5]. Sabin reported the first case of encephalitis due to *T. gondii* [6]. Acutely acquired human disease and vertical transmission in humans were both recognized in the 1940s when the classic tetrad of symptoms of congenital toxoplasmosis was described: retinochoroiditis, hydrocephalus or microcephalus, cerebral calcification, and psychomotor disturbances [7]. *T. gondii* was recognized as a causative agent of lymphadenopathy in the early 1950 [8]. Feldman in 1953 had diagnosed 103 children, of which 99% had eye lesions, 63% had intracranial lesions, and 56% had psychomotor retardation [9]. The major sequelae of congenital toxoplasmosis and recognition of risks to patients with malignancies were described in the 1960s, with recognition of *T. gondii* as an opportunistic pathogen in AIDS patients in the 1980s [10, 11]. Its complete life cycle was described in 1970 [5].

The name Toxoplasma (toxon =arc, plasma = form, Greek) is derived from its crescent shape. The parasite belongs to the Sarcocystidae family, as listed in (Table 1), which revealed the taxonomy of *T. gondii* [12].

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Table 1. Taxonomy of T. gondii [13]

Phylum	Apicomplexa
Class	Sporozoa
Sub class	Coccidia
Order	Eucoccidia
Suborder	Eimeria
Family	Sarcocystidae
Genus	*Toxoplasma*
Species	*Toxoplasma gondii*

The morphology of *T. gondii* varies during its developmental cycle. Banana- or crescent-shaped forms of the parasite (Figure 1-A) are ingested with food and enter the body via the pharyngeal and intestinal mucosa [14]. In intermediate hosts and humans, these multiply during the acute phase of infection by cell division within a fine network of cells. At this stage of their development, the protozoa are known as tachyzoites (also: trophozoites, endozoites), which are illustrated in (Figure 1-A) [15]. These 4-7 micron long, 2-4 micron wide forms of the pathogen with a nucleus in the lower half of the cell (Figure 1-B) are distributed via the blood and lymph to the entire body, where they cause lysis of host cells and go on to infect adjacent cells [16]. This process gradually gives rise to tissue damage in the form of necrotic foci surrounded by inflammation.

Damage occurs especially in the brain, heart muscle, skeletal muscle, liver, and eyes, though any organ in the body can be affected. Later, intracellular cysts are formed (Figure 1-C), mostly in skeletal muscle, including the diaphragm, and in the heart muscle and the brain. If the infected organ forms antibodies to the pathogen, the free and intracellular forms of the parasite disappear and are replaced by tissue cysts with a diameter of 100-300 microns [17]. These cysts, which are surrounded by a wall, can contain several thousand bradyzoites (also: cystozoites) (Figure 1-D). These cause clinically inconspicuous infection and can persist in the host for years, as the cyst protects them from attack by the immune system. Probably, infection is always followed by lifelong persistence of cysts and by seropositivity.

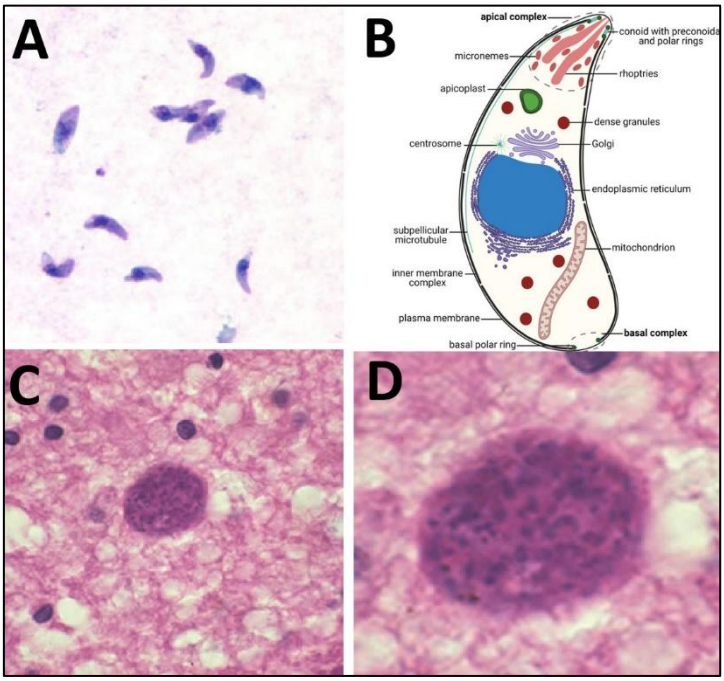

Figure 1. Toxoplasma gondii. A: Tachyzoites and crescent shape of pathogen [18]. B: Microscopic structure [19]. C: Cyst in infected brain cells [18]. D: Cyst magnification [18]

A third form of the organism is that of oocysts, which to date have been found only in members of the cat family. This round to oval form measuring 9x14 microns is protected by a resistant cyst wall and can survive in the environment for up to five years [20]. Oocysts arise as the final product of a sexual cycle that takes place in the intestinal epithelium

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of the host animal. Shedding of oocysts in the animal's feces is followed within three to four days by sporulation. Only then do the oocysts become infectious. They can survive for up to 18 months in a humid environment, but are destroyed by dry heat or boiling water. The parasite life cycle is illustrated in (Figure 2).

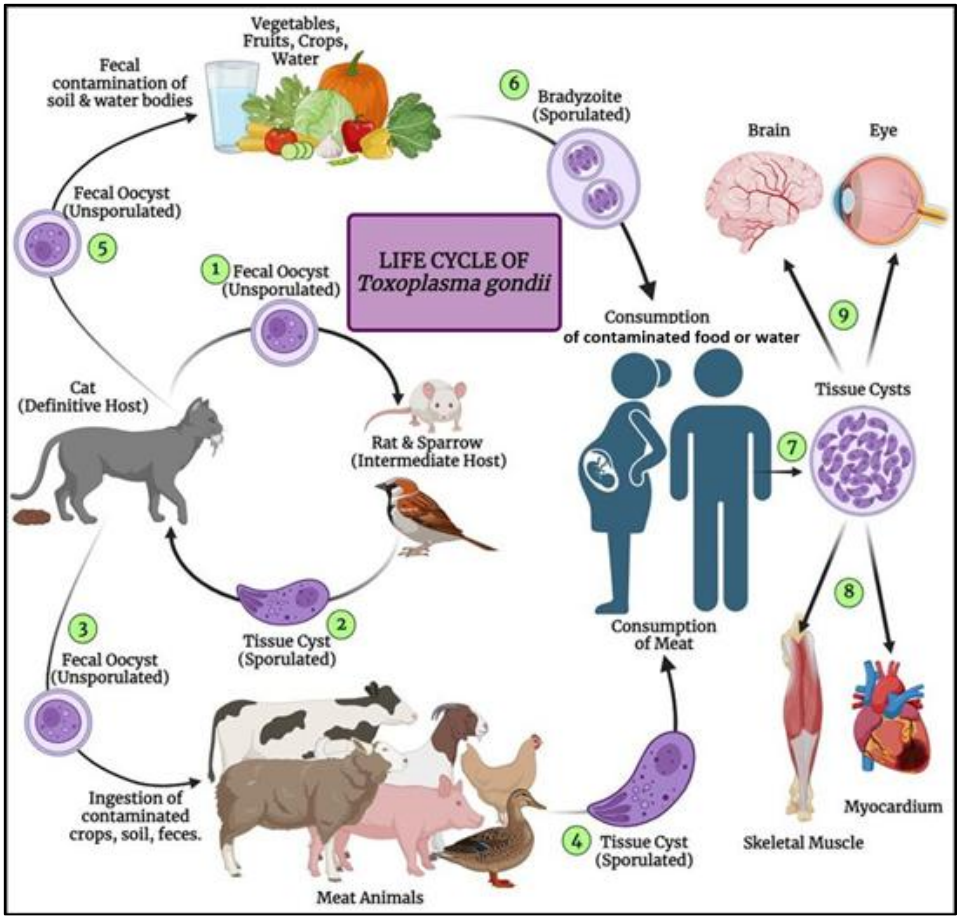

Figure 2. Lifecycle of T. gondii [21]

The intracellular parasites (tachyzoites) are approximately 2 by 6 μm, crescent-shaped organisms that are enclosed in a parasite membrane to form a cyst measuring 10-12 μm in size. Cysts in cat feces (oocysts) are 10-13 μm in diameter [22]. The oocyst contains two sporocysts, each of which contains four sporozoites. Thus, resemble the oocysts of isospora species. Only cats will produce and pass *Toxoplasma*. As seen in (Figure 3), the life cycle of *T. gondii* has two phases. The sexual part of the life cycle (coccidian-like) takes place only in members of the Felidae family (domestic and wild cats), which makes these animals the parasite's primary host. The asexual part of the life cycle can take place in any warm-blooded animal, like other mammals (including felines) and birds [23].

In the intermediate hosts (including felines), the parasite invades cells, forming intracellular so-called parasitophorous vacuoles containing bradyzoites, the slowly replicating form of the parasite. Vacuoles form tissue cysts mainly within the muscles and brain. Since they are within cells, the host's immune system does not detect these cysts [24].

Resistance to antibiotics varies, but the cysts are very difficult to eradicate. Within these vacuoles, *T. gondii* propagates by a series of binary fissions until the infected cell eventually bursts and tachyzoites are released [25]. Tachyzoites are the motile, asexually reproducing form of the parasite. Unlike the bradyzoites, the free tachyzoites are usually efficiently cleared by the host's immune response, although some manage to infect cells and form bradyzoites, thus maintaining the infection. Tissue cysts are ingested by a cat (e.g, by feeding on an infected mouse), the cysts survive passage through the stomach of the cat, and the parasites infect epithelial cells of the small intestine, where they undergo sexual reproduction and oocyst formation. Oocysts are shed with the feces. Animals and humans that ingest oocysts (e.g, by eating unwashed vegetables, etc.) or tissue cysts in improperly cooked meat become infected. The parasite enters macrophages in the intestinal lining and is distributed via the bloodstream throughout the body [26, 27].

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Prevalence

The prevalence of human infection by *Toxoplasma* varies greatly between countries. Factors that influence infection rates include diet (prevalence is possibly higher where there is a preference for less-cooked meat) and proximity to cats. Many studies had reported cases of toxoplasmosis such as a study from Austria reported frequent symptoms in children with congenital toxoplasmosis in 1957, a study from France showed that the seroprevalence (evidence of past infection) in pregnant women in Paris was 85% with a high risk of *T. gondii* infection in sero negative in 1965, and a large study from France had reported of 374 infected pregnancies in 1974 [28-30].

Austria implemented a toxoplasmosis screening program in 1975. Nearly all women who were pregnant were serologically screened early in pregnancy, and if found to be negative initially, were tested again during the second and third trimesters. Women with *Toxoplasma* infections were treated as soon as the infection was detected. Although sero-positivity rates among pregnant Austrian women have declined from approximately 50.0% during the late 1970s to 36.7% during the early 1990s, the incidence of congenital *T. gondii* infection has declined even more, from 50-70 cases per 10,000 births before the program to 1 per 10.000 births during the early 1990s [31, 32]. An epidemiological study conducted in Norway in 1978 showed a significantly higher prevalence of *T. gondii* antibodies among blind and partially sighted children, mentally retarded children, and children with speech or behavior disorders than in healthy controls [33].

Moreover, the European Research Network on Congenital Toxoplasmosis was established in 1993 and has sponsored several studies regarding public health interventions for congenital toxoplasmosis. Most recently, a multi-center study was conducted to evaluate the effectiveness of toxoplasmosis treatment administered during pregnancy in preventing transmission of maternal infection to the fetus. Pregnant women who visited one of five European university medical centers for prenatal care were screened for *T. gondii* antibodies at their first prenatal visit. Seronegative women were retested at least once every trimester in two centers and monthly in the other centers, until the birth of the infant. For women who seroconverted during pregnancy, prenatal treatment was started, and their infants were followed up for one year after birth. Treatment regimens consisted of spiramycin or a combination of pyrimethamine and sulfadiazine. Furthermore, the earlier antibiotics were administered after infection, the less likely sequelae were detected in the infant [34].

A long-term study in Denmark conducted a newborn screening for toxoplasmosis to determine the feasibility of screening newborn infants for congenital toxoplasmosis in an area with low prevalence. The seroprevalence of antibodies to *T. gondii* among women during this study was 28%. Approximately 90.000 infants were screened for *T.* gondii-specific IgG antibodies 5-10 days after birth. Infants born for one year to mothers who serologically confirmed congenital infections [35]. A prospective study to determine the seroprevalence of maternal and neonatal toxoplasmosis, cytomegalovirus antibodies, and hepatitis -B antigens in a rural Egyptian area. The study was done on a randomly selected sample of pregnant woman and their newborn infants (n=150). Sera were collected from the mothers during the first antenatal visit, and at the time of delivery, and cord blood specimens (paired samples taken from their infants to be tested for *T. gondii* IgG and IgM antibodies). Out of the 150 pregnant women, 64 (43%) were *T. gondii* immune at the first antenatal visit, and their newborns were *T. gondii* IgG positive. *T. gondii*-specific IgM antibody was detected in only three mothers at the time of delivery. The rate of maternal infection susceptible pregnancies was 4%, and the maternal fetal transmission rate was estimated to be 33%, as only one newborn infant had *T. gondii* IgM antibody at birth. This denoted a prevalence of congenital *T. gondii* infection < 1,0 % to non – immune mothers. There were no clinical features of congenital infection in an infant with *T. gondii* IgM antibody. The mothers infected during pregnancy had known risk factors for *T. gondii* infection, and the transplacental transmission rate in a rural Egyptian area was high compared to other countries [36].

Another study to evaluate the prevalence rate of, and the environmental and personal factors that may contribute to infection, processed serum samples of 197 pregnant women aged 17 to 45 years attending the maternity hospital in Makkah were tested for anti-T. *gondii* IgG and IgM antibodies using enzyme-linked immunosorbent assay (ELISA). The sero-prevalence of anti-T. *gondii* IgG was 29.4%, whereas IgM sero-positivity was 5.6 %. The highest IgG and IgM seroprevalence were among participants aged 35 to 43 years (48.8% and 12%, respectively). No statistically significant relation was observed between *T. gondii* seroprevalence and the other variable factors studied, indicating that there is a considerable rate of *T. gondii* infection among pregnant women in Makkah and supporting the concern that Saudi women may be vulnerable to that infection [37].

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In Jordan, out of the 280 pregnant women, sera were tested during the period between January 2000 and May 2001. Blood samples were taken after the first antenatal visit. Serum was separated and tested for *T. gondii* IgG antibodies using an indirect fluorescent antibody. Sero-prevalence gradually increased with age, from 31.7% at 15-24 years to 90.0% at 35-45 years. Regression analysis showed that sero-prevalence of toxoplasmosis is positively correlated with age and residence. Consumption of undercooked meat and contact with soil were significant risk factors [38].
In Duhok, northern Iraq, a study revealed that 77.4% of cases had single or multiple fetal loss as evidence of infection. All the women were examined for the presence of *T. gondii*-specific IgM antibodies by ELISA; only three cases tested positive. Also, researchers tested 187 of the women by the latex agglutination test; 55 tested positive [39].
In general, toxoplasmosis infects approximately one-third of the world's population [40]. Seroprevalence ranges widely, from 10% to over 90% in different regions, with higher rates observed in certain areas like Africa and South America [41]. (Table 2) lists the percentage of regional variations in global prevalence.

Table 2. Regional variations in the global pooled prevalence of Toxoplasmosis

Region	Percentage	Reference
Africa	51%	[42]
South America	49%.	
Europe	47%.	
Australia	43%	
Asia	36%.	
North America	23%	

Infection rates globally reach above 80% in some parts of the world. Studies indicate an average global seroprevalence rate of around 25.7%, according to a systematic review published in PubMed, with a range from 0.5% to 87.7%. The following (Figure 3) showed adjusted global prevalence at the age of 22 years [43].

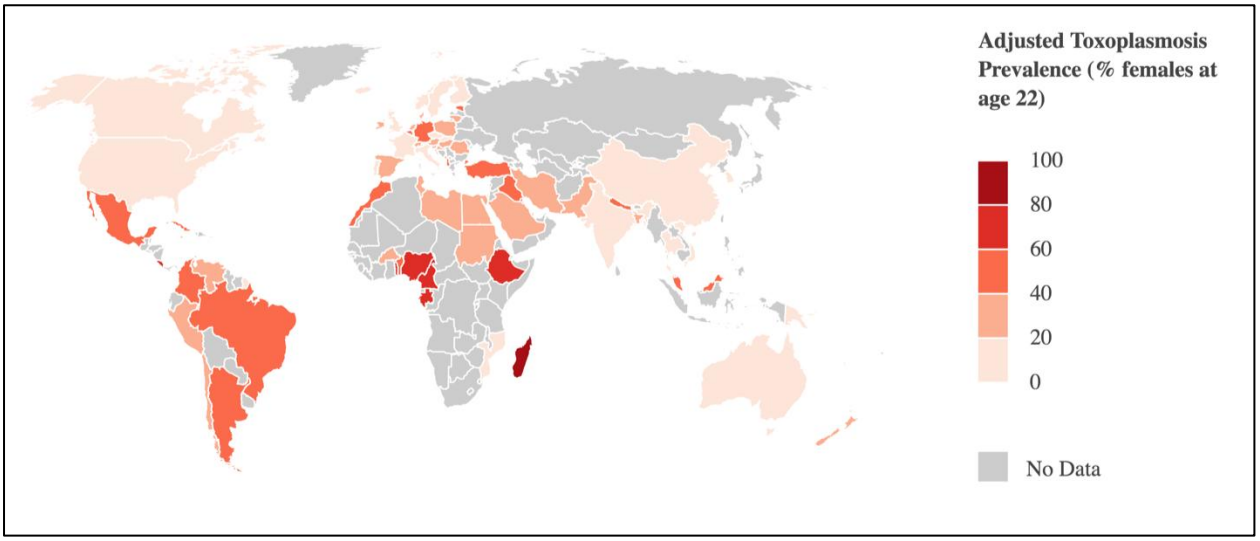

Figure 3. Adjusted toxoplasmosis prevalence corresponding to age of 22 years [44]

Transmission

Toxoplasmosis is not passed from person-to-person, except in instances of mother-to-child (congenital) transmission and blood transfusion or organ transplantation [45]. People typically become infected by three principal routes of transmission, namely: foodborne, animal-to-human (zoonotic), and mother-to-child (congenital) transmission.
Foodborne transmission: The tissue form of the parasite (a microscopic cyst consisting of bradyzoites) can be transmitted to humans by food. People become infected by eating undercooked, contaminated meat (especially pork, lamb, and venison), accidental ingestion of undercooked contaminated meat after handling it and not washing hands thoroughly (*Toxoplasma* cannot be absorbed through intact skin), or eating food that was contaminated by knives, utensils, cutting boards, or other foods that had contact with raw, contaminated meat [46].

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Animal-to-human (zoonotic) transmission: Cats play an important role in the spread of toxoplasmosis. They become infected by eating infected rodents, birds, or other small animals. The parasite is then passed in the cat's feces in an oocyst form, which is microscopic. Kittens and cats can shed millions of oocysts in their feces for as long as three weeks after infection. Mature cats are less likely to shed *Toxoplasma* if they have been previously infected. A *Toxoplasma*-infected cat that is shedding the parasite in its feces contaminates the litter box. If the cat is allowed outside, it can contaminate the soil or water in the environment as well. People can accidentally swallow the oocyst form of the parasite. People can be infected by accidental ingestion of oocysts after cleaning a cat's litter box when the cat has shed *Toxoplasma* in its feces. Accidental ingestion of oocysts after touching or ingesting anything that has come into contact with a cat's feces that contains *Toxoplasma*. Accidental ingestion of oocysts in contaminated soil (e.g, not washing hands after gardening or eating unwashed fruits or vegetables from a garden). Drinking water contaminated with the *Toxoplasma* parasite is another way of such transmission [47-50].
Mother-to-child (congenital) transmission: A woman who is newly infected with *Toxoplasma* during pregnancy can pass the infection to her unborn child (congenital infection). The woman may not have symptoms, but there can be severe consequences for the unborn child, such as diseases of the nervous system and eyes [51]. Rare instances of transmission: Organ transplant recipients can become infected by receiving an organ from a *Toxoplasma*-positive donor [52]. Rarely, people can also become infected by receiving infected blood via transfusion [53]. Laboratory workers who handle infected blood can also acquire infection through accidental inoculation [54].

Symptoms

The acute stage of toxoplasma infections can be asymptomatic, but often gives flu-like symptoms in the early acute stages, and like flu can become, in very rare cases, fatal [55]. The acute stage fades in a few days to months, leading to the latent stage. Latent infection is normally asymptomatic; however, in the case of immunocompromised patients (such as those infected with HIV or transplant recipients on immunosuppressive therapy), toxoplasmosis can develop [56]. The most notable manifestation of toxoplasmosis in immunocompromised patients is toxoplasmic encephalitis, which can be deadly. If infection with *T. gondii* occurs for the first time during pregnancy, the parasite can cross the placenta, possibly leading to hydrocephalus, intracranial calcification, and chorioretinitis, with the possibility of spontaneous abortion or intrauterine death [57].
T. gondii infections can change the behavior of rats and mice, making them drawn to rather than fearful of the scent of cats. This effect is advantageous to the parasite, which will be able to sexually reproduce if its host is eaten by a cat. The infection is almost surgical in its precision, as it does not impact a rat's other fears, such as the fear of open spaces or of unfamiliar-smelling food [58]. There has been speculation that human behavior may also be affected in some ways, and correlations have been found between latent *Toxoplasma* infections and various characteristics such as decreased novelty-seeking behavior, slower reactions, feelings of insecurity, and neuroticism [59]. Several independent pieces of evidence point towards a possible role of *Toxoplasma* infection in some cases of schizophrenia and paranoia, but this theory does not seem to account for many cases.
A recent study has indicated that toxoplasmosis is also correlated strongly with an increase in boy births in humans, leading to an alteration of the human sex ratio. According to the researchers, "depending on the antibody concentration, the probability of the birth of a boy can increase up to a value of 0.72, which means that for every 260 boys born, 100 girls are born" [60, 61]. Symptoms in persons with otherwise healthy immune systems can include: enlarged lymph nodes in the head and neck, headache, fever, mild illness similar to mononucleosis, muscle pain, and sore throat. Toxoplasmosis is the second leading cause of death among foodborne illnesses in the US [62].

Diagnosis

The tachyzoites or bradyzoites can be seen under a microscope in smears from infected tissues after staining (acute infection). Smears of lymph gland biopsy, bone marrow aspiration, cerebrospinal fluid, and peritoneal or pleural fluids, or even sputum, all of which can harbor parasites. Other tests that can be used to investigate the presence of organisms, such as serological tests, ELISA (IgG, IgM), and PCR (Polymerase chain reaction) [63-66].

Treatment

Most healthy people don't require toxoplasmosis treatment. But if you're otherwise healthy and have signs and symptoms of acute toxoplasmosis, your doctor may prescribe the following drugs:

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Pyrimethamine (Daraprim). This medication, typically used for malaria, is a folic acid antagonist. It may prevent your body from absorbing the B vitamin folate (folic acid, vitamin B-9), especially when you take high doses over a long period. For that reason, your doctor may recommend taking additional folic acid. Other potential side effects of pyrimethamine include bone marrow suppression and liver toxicity [67, 68].
Sulfadiazine. This antibiotic is used with pyrimethamine to treat toxoplasmosis. The drug used against *T gondii* acts on the tachyzoites rather than on the bradyzoites in tissue cysts. Hence, they may control active infection, but they cannot eliminate chronic infection [69].
Other treatments may include prenatal spiramycin, which is usually given to reduce the risks of mother-to-child transmission. Infants with congenital toxoplasmosis are treated with clindamycin, while acute adult cases, such as immunosuppressed patients and ocular toxoplasmosis, are generally treated by pyrimethamine and sulfonamides (sulphadiazine or sulphadoxine) plus folinic acid to protect bone marrow from the suppressive effects, and corticosteroids are used to reduce inflammation [70]. There are patient groups in which toxoplasmosis may cause severe disease. The outcome of the disease is, however, constantly improving through the development and refinement of the diagnostic screening procedures and the treatment protocols.

Conclusion

The current review indicates that there is a considerable rate of *Toxoplasma* infection among aborted women worldwide, especially in Africa and South America. Moreover, it shows the need to provide health education to pregnant women in order to reduce or to prevent primary infection during early months of their pregnancy. Seroprevalence of toxoplasmosis is positively correlated with age, residence, education, and knowledge of transmission of the disease. Contact with cats and consumption of undercooked meat were found to be significant risk factors, which should be avoided as much as possible in all age periods and during early pregnant women stages in every place. According to study of role of distribution of carrier animals in specific areas in comparison to role of eating habits focusing on consuming of undercooked meat, results showed that infections recorded was higher in areas dealing with carrier animals when compared with same way of consuming habits in both areas.

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Disclosure of conflict of interest
The authors have no disclosures to declare.

Compliance with ethical standards
The work is compliant with ethical standards.

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