The Prevalence of Toxoplasmosis among Iraqi Population

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Abstract

Toxoplasma gondii is an obligatory intracellular zoonotic protozoon, with a worldwide distribution infecting humans and almost all warmblooded animals. This review highlights the seroprevalence of toxoplasmosis in Iraq, and focus on the effects of variable risk factors, clinical features, and preventive strategies. Toxoplasmosis is recognized as a considerable public health issue in Iraq, numerous studies reported variable seroprevalences among populations. The variability in seropositivity is influenced by geographical location, age, hygienic and socioeconomic conditions. Higher seroprevalence is particularly reported among rural communities, pregnant women, and individuals with frequent exposure to animals or contaminated soil. The main routes for acquiring toxoplasmosis are eating or drinking contaminated food or water, being in close contact with contaminated soil, eating undercooked infected meat, and congenitally. Clinically, toxoplasmosis can be either asymptomatic, or exhibits mild flu-like symptoms to severe complications or even death in immunocompromised people and congenital infections in infants. Screening programs for pregnant women and serological testing for immunocompromised individuals can aid in early diagnosis and timely intervention. In conclusion toxoplasmosis seroprevalence is influenced by variable socio-demographic and environmental factors that affect its distribution and transmission. Further research is required to determine the actual seroprevalence and the associated factors in order to improve the control measures, and prevent the occurrence of new cases. Therefore, the introduction of health education programs and sanitary measure among the community are necessary to minimize the burden of toxoplasmosis and its related health consequences.

Keywords: Toxoplasmosis, Seroprevalence, Risk factors, Stages, Life cycle, Pathogenesis, Epidemiology

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Introduction

Toxoplasma gondii is an obligatory intracellular zoonotic apicomplexan. It is widely distributed across the human population and is regarded as the primary contributor to global morbidity (Murad and Eassa 2023). The incidence of *T. gondii* among the population is greatly variable because it is influenced by numerous environmental and socioeconomic situations, in addition to cultural customs (Mareze et al., 2019). It is estimated that approximately 30% of people worldwide are afflicted with this parasite (Dubey, 2021). Infection in healthy adults may be experienced in the form of flu-like symptoms, while immunocompromised individuals face severe Toxoplasmic encephalitis, whereas infections during pregnancy can cause neurological or visual harm to the fetus or even result in miscarriage (Attias et al., 2020).

Two hosts are used in the life cycle of *Toxoplasma gondii*; cats and other members of the Felidae family are the definitive hosts, in which the parasite undergoes sexual reproduction in their intestinal lining cells, while humans and all warm-blooded animals serve as intermediate hosts which harbor the asexual stages (Mosawi et al., 2019; Dubey, 2021).

The parasite possesses three body forms including the oocyst, bradyzoite, and tachyzoite (Waldman et al., 2020). The tachyzoite is characterized by its ability to rapidly divide resulting in the cell's death in the early phases of the illness. The bradyzoite; is found in tissue cysts and is relatively slower in growth and the sporozoite is specific to the sexual cycle, present in the sporulated oocyst (Mosawi et al., 2019). When the oocyst sporulates in the environment, it becomes infectious and is accidentally consumed by other warm-blooded animals that act as the parasite's intermediate hosts (Guiton et al., 2017; Mosawi et al., 2019).

Genetically, *T. gondii* can be classified into three distinct strains or genotypes: Type I, Type II, and Type III (Dubremetz and Lebrun, 2012). These types differ in their virulence, and they can alter the mechanism of infection and the intensity of symptoms. Although types II and III are significantly less virulent than type I, type I is very pathogenic to outbred mice. Therefore, finding the genetic type can aid in comprehending the illness and potentially determining the best course of therapy (Fuentes et al., 2001; Alghamdi et al., 2016). This chapter aims to provide insight into the current status of *Toxoplasma gondii* infections in Iraq and to highlight strategies for effective prevention, diagnosis, and management. Through the determination of its seroprevalence among different categories of the Iraqi population using immunological tests, exploring its epidemiological patterns, associated risk factors, and potential public health implications.

1. Historical Aspects of Toxoplasma gondii and its Classification

Over a century ago, *Toxoplasma gondii* was initially identified in Tunis at the Pasteur Institute (Splendore, 1908; Nicolle and Manceaux, 1909). Nicolle and Manceaux first named it *Leishmania gondii* because they believed they had found a new species of *Leishmania* (Nicolle, 1908). They rapidly realized the error, and, in 1909, Nicolle and Manceaux (1909) created a new protozoan genus named *Toxoplasma*, this genus contained only one species the *T. gondii* based on the structure of the parasite (toxo which in Greek means arc or bow and plasma meaning shape). The first time *T. gondii* was described as an obligatory intracellular parasite was in 1937, when two virologists studying guinea pig brains, Sabin and Olitsky, observed that the parasite could only multiply within a live cell. Furthermore, the cats' small intestine included the sexual phase of *T. gondii*, which completed the parasite's life cycle (Sabin and Olisky, 1937). *Toxoplasma gondii* is classified according to Robert and Janovy (2005), as shown in (Figure 1).

Kingdom	Protista
Sub Kingdom	Protozoa
Phylum	Apicomplexa
Class	<u>Conoidasida</u>
Order	Eucoccidiorida
Sub Order	<u>Eimeriorina</u>
Family	Sarcocystidae
Subfamily	<u>Toxoplas matinae</u>
Genus	Toxoplasma
Species	<i>T. gondii</i> (Nicolle and <u>Manceaus</u> , 1909)

Fig. 1: Taxonomic classification of *Toxoplasma gondii*

2. Morphology and Stages of Toxoplasma gondii

Toxoplasma gondii, possess three stages:

1. The oocyst, resistant to environmental conditions, excreted in cat feces and

is responsible for disease transmission.

2. The bradyzoite:exist in tissue cyst, divid slowly, responsible for disease transmission

when undercooked meat is eaten.Occur in all worm-blooded animals.

3. The tachyzoite, rapidly dividing stage, determine the acute stage of the disease, found

in all worm-blooded animals.All these stages are connected to the life cycle of the parasite.



Fig. 2: A longitudinal scanning and transmission electron microscopy section of *T. gondii* tachyzoite showing its primary structures and organelles.

4.1 Tachyzoites

This word, which was coined by Frenkel, describes the rate at which this stage multiplies throughout the infection's acute phase. Tachyzoite morphologically resembles a crescent (Figure 2) (Jones and Dubey, 2010).

One of the main features of the tachyzoite is its strong polar structure, which is arranged around a clear apical pole and contains two sets of secretory organelles known as micronemes and rhoptries, in addition to a special microtubule-made appendage called a conoid. A variety of these products have already been identified, and a repertoire has been discovered to encourage cell movement and invasion of the host cells (Frénal et al., 2017).

4.2 Bradyzoite and Tissue Cysts

A "bradyzoite" is an organism that multiplies slowly by recurrent endodyogeny inside a tissue cyst (Brady means slow in Greek), it is also known as a cystozoites and can be seen in the chronic stage of the illness (Tobin et al., 2010).

In comparison to tachyzoites, bradyzoites are thinner and less susceptible to destruction by proteolytic enzymes (Robert-Gangneux and Dardé, 2012). Mature bradyzoites may contain hundreds of zoites (Figure 3). Additionally, the bradyzoites release the contents of their organelles into the parasitophorous vacuole (PV) matrix, which progressively causes the formation of an intra-cystic network and a cyst wall in association with the PV membrane (Attias et al., 2020). Although they can form tissue cysts in the kidneys, liver, or lungs, most frequently are seen in the cardiac and skeletal muscles, as well as in the brain and eyes. Whole tissue cysts may survive for a very long time inside their host and are safe without triggering any inflammatory reaction (Dubey, 2016).



Fig. 3: Scanning and transmission electron microscopy scheme of *Toxoplasma gondii* tissue cyst demonstrating: numerous bradyzoites within the cyst. b. A closer inspection, of the tissue cyst surrounded by a membrane, with a substantial cyst wall deposited beneath it. Modified from Attias *et al.* (2020).

4.3 Oocysts

The oocyst of *T. gondii* is formed in cats or other felids as a result of the sexual reproduction of the parasite in these hosts. Both domestic and wild cats emit *T. gondii* oocysts in their feces. Oocysts excreted in cat feces are non-sporulated and non-infectious (Figure 4A). Sporulation occurs under favorable environmental conditions, yielding two sporocysts (Figure 4B), each of which has four sporozoites (Ferguson and Dubremetz, 2014). The sporulated oocysts can survive under severe weather conditions, contaminating the ecosystem (Jones and Dubey, 2010).



Fig. 4: Oocyte. A. Unstained unsporulated oocyst, B. Unstained sporulated oocyst.

3. The Toxoplasma gondii Life Cycle

The life cycle of *T. gondii* (Figure 5) is "indirect" since the parasite reproduces both sexually and asexually, endures in the environment, and under the effect of their immune systems, lays dormant in hosts (Jokelainen, 2013), and includes:

5.1 Asexual Life Cycle (Intermediate Hosts)

The ingested sporulated oocyst ruptures releasing its sporozoites into the gut lumen, and then they enter the cells in the lamina propria after passing through the gut epithelium in the intermediate host. The second way of infection is by ingesting tissue cysts in undercooked meat, the released bradyzoites will behave in a similar way as the sporozoites. Both of them develop into the quickly proliferating tachyzoite in the intestinal epithelium from there they spread throughout the body, and then enter host cells, where they rapidly divide till the cell ruptures (Weiss and Kim, 2000).

5.2 Sexual Life Cycle (Definitive Hosts)

The epidemiology of toxoplasmosis depends on cats and wild felids since they are the only definitive hosts that can spread the oocysts through their feces (Bayarri et al., 2012). A cat can acquire infection through two main methods: either by ingesting sporulated oocysts from the contaminated environment or consuming the tissue cysts present in the organs and tissues of an intermediate host prey that is chronically infected (like mice) (Dubey and Lappin, 2006). Following tissue cyst ingestion, the bradyzoites are released due to the rupture of the cyst wall by the action of the digestive enzymes. The released bradyzoites invade the intestinal epithelium, then they undergo five distinct developmental stages in addition to systemic dispersion following change to the invasive stage of tachyzoites, which, within two days of infection, produces both micro and macro gametocytes (Dubey et al., 1998). Followed by the fusion of these gametes forming the zygote, which develops into oocysts and secretes a cyst wall. For several days or weeks, oocysts are excreted in cat feces after rupturing the intestinal epithelial cells. The released oocysts sporulate under environmental conditions to infect new hosts (Dubey et al., 1998; Dabritz and Conrad, 2010).



Fig. 5: Life cycle of *Toxoplasma qondii*

4. Pathogenesis

Toxoplasma gondii is regarded as one of the most aggressive parasites, capable of entering and multiplying in all nucleated cells of warmblooded organisms, including humans (Zhu et al., 2019). The parasite can live in a parasitophorous vacuole (PV) inside the cell (Sonaimuthu et al., 2016). According to Saeij and Frickel (2017), the PV membrane (PVM) protects the parasite from intracellular cytoplasmic defense systems that have advanced to identify cytoplasmic pathogens. Most primary infections do not cause any symptoms because the parasite stays in the tissues, such as the liver, eyes, or lungs. The person is normal and there is no inflammation until an immunodeficiency arises, which activates the parasite (Hassan et al., 2019).

5. Clinical Characteristics of Toxoplasmosis in Human

Human clinical manifestations of toxoplasmosis vary depending on host parameters including immunological status and genetic background, as well as parasite characteristics like strain virulence and inoculum size (Weiss and Dubey, 2009). These clinical features can be divided into:

5.1 Acquired Toxoplasmosis in Immunocompetent Postnatal Individuals

Toxoplasma gondii infections are asymptomatic in more than 80% of cases and result in a benign sickness in 20% of cases (Robert-Gangneux and Dardé, 2012). Symptoms include fever, lymphadenopathy, and asthenia. For a few days or weeks, the patient may have a mild fever, but it will go away completely on its own. Usually, the disease's progression is benign and self-limiting (Garweg, 2016). A significant opportunistic illness among individuals with acquired immunodeficiency syndrome (AIDS) is an indication of toxoplasmosis. In HIV patients, immunological suppression, can reactivate latent infections and cause potentially deadly encephalitis (Laboudi et al., 2020).

5.2 Congenital Toxoplasmosis

It results from infection of the fetus during pregnancy. Pregnant women who experience a primary infection in the early stages of their pregnancy are most commonly affected, but immunocompromised pregnant women might be at risk of transmission due to tissue cyst reactivation during a chronic illness (Goldstein et al., 2008). Based on the pregnancy stage at which infection occurs, congenital toxoplasmosis can cause an abortion or severe symptoms (Chaichan, 2017). Furthermore, either from birth or as they develop during childhood, congenital toxoplasmosis can cause significant visual impairment, irreversible neurological abnormalities, and macrocephaly (Bahia-Oliveira et al., 2017).

6. Ocular Toxoplasmosis

Although it was previously believed that congenital infections were the cause of the majority of ocular toxoplasmosis cases, the majority of the cases are now understood to be caused by a postnatal infection. In immunocompromised patients, it is also seen as a localized form of reactivation Blurred or impaired vision is the result of ocular involvement in the retina (Kalogeropoulos et al., 2022). Vision-threatening consequences such as retinal detachment, choroidal neovascularization, and glaucoma may develop at any point during the clinical course with ocular toxoplasmosis, which is a progressive and chronic necrotizing retinitis (Park and Nam, 2013).

7. Immunocompromised Patients Toxoplasmosis

In immunocompetent people, most instances of toxoplasmosis have been reported to be asymptomatic; nevertheless, several risk factors can change the infection's course and cause symptoms, including severe ones. Immunocompromised individuals (such as those with cancer or acquired immunodeficiency syndrome (AIDS), immunosuppressed organ transplant recipients, and fetuses in cases of congenital infection are high-risk populations for the disease's severe progression (Fang et al., 2021). In these patients, toxoplasmosis usually results from the reactivation of latent infections that were acquired before immune suppression, or it can develop from a recent acute infection with the parasite (Kodym et al., 2015). Immunocompromised individuals may experience more severe symptoms and consequences such as pneumonitis, encephalitis, splenomegaly, retinochoroiditis, and fulminant myocarditis with heart failure (Mahmoudi and Rahmati 2020).

8. Toxoplasma gondii Transmission Routes

Humans may acquire toxoplasmosis in several ways (Figure 6), including drinking water or eating vegetables contaminated with *T. gondii* sporulated oocysts, which are extremely resistant to the standard chlorination disinfection procedures employed in the water supply sector (Laboudi et al., 2020). The eating of infected undercooked or raw meat that contains tissue cysts (Muhie and Keskes, 2014). Once a woman acquires an infection during pregnancy, it can spread via the placenta and cause a congenital disease in the fetus (Singh, 2016). The acquisition of the disease from organ transplants or during blood transfusions although these methods are rare, can occur (Robert-Gangneux and Dardé, 2012).

9. Epidemiology

Toxoplasmosis is a serious global public health issue, with approximately 2 billion people infected (Rahmanian et al., 2020). After listeriosis and salmonellosis, *T. gondii* is thought to be the third most frequent cause of fatal foodborne illness (Murat et al., 2013). The prevalence of *T. gondii* is constantly changing and varies widely between nations as well as between regions within a single nation. It is impacted by a variety of complicated risk factors, including social, environmental, and health-related behaviors (Pappas et al., 2009).

In Iraq, the prevalence of toxoplasmosis varies from region to region depending on climatic conditions, personal hygienic measures, and community sanitation. Because there is so much research on this topic, only those published after 2012 were reviewed in Table 1. These epidemiological studies showed considerable variations in Toxoplasmosis in different Cities and Governorates of Iraq.



10. Diagnostic Approaches for Toxoplasma gondii

The diagnosis of T. gondii in humans and animals is crucial for clinical therapy, epidemiological research, prevention, and control (Mohamed, 2020). *T. gondii* can be found in biological and environmental materials using different techniques, like microscopy, serological, and molecular methods (Su et al., 2010).

12.1. Microscopy

Under the microscope, *T. gondii* has traditionally been detected in fecal, tissue, water, and environmental materials (Liu et al., 2015). Tachyzoites and bradyzoites are typically identified under the microscope using Hematoxylin and Eosin (Hand E) and Giemsa stains (da Silva et al., 2010).

Cities/Governora Host		Techniques	Prevalence (%) of		Peferences
			InC	IgM	
Dubok	Fomala	ELICA	igg	11.1	Damadhan & Canloga 2010
DUIIOK	Program twom on	ELISA	44.4	11.1	Ahmad at al. 2000
		ELISA	57.1	2.59	Allilled et al., 2020
	women	ELISA	11.5	0.63	Mizuri & Mero, 2020
	Female	ELISA	36.3	0.76	Salih et al., 2020
	Males	777.704	20.75	4.72	
	Females	ELISA	12.8	4.8	Abdulla et al., 2022
Amede	Diabetic Women	ELISA	21	1.5	Golek, 2023
	Healthy Women		12	1	
Akre	Women	ELISA	54.46	4.44	Saadi & Ahmed, 2020
Erbil	Pregnant Women	ELISA	34.8	12.93	Abdullah & Mahmood, 2017
	Healthy Populations	LAT	8.4	15.6	Al-Daoody et al., 2019
	Leukemia	ELISA	44.44	12.5	Mawlood, 2021
	Patients				
	Aborted Women	ELISA	27.03	0.74	Mohammed & Hamad, 2023
Sulaimani	Cancer Patient	ELISA	39.8		Mohammed et al., 2023
	Healthy		24.4		
Kirkuk	Pregnant and	ELISA	20.3	1.5	Aljumaili et al., 2014
	Non-Pregnant		37.2	0.4	
	Diabetic Male	ELISA	9.09	2.27	Nihad & Hamad, 2017
	Diabetic Female		15	9	
	Female	ELISA	9.0	3.33	Barzinij, 2021
Nineveh	Aborted Woman	LAT	62		Al-Omer & Al-Marsoomy, 2021
		ELISA	73.33	9.09	
Tikrit	Pregnant Women	ELISA	30	20.68	Mohammed, 2018
Samara	Aborted Women	VIDAS	31.2		Anwar & Al-Bayati, 2018
Salahdin	Human	ELISA	26	9	Al-doury et al., 2018
Diyala	Women	ELISA	38	4	Shaker et al., 2018
	Hemodialysis Patients	ELISA	54.1		Hussein & Molan, 2019.
	Healthy Control		38.2		, 2
Baquba	Human	LAT	41		AL-Khafaji et al., 2020
		ELISA	22		
Baghdad	Males	ELISA	30	2.5	Mahmood et al., 2013
		LAT	34	5	
	Females	ELISA	20	1.5	AL-Mossawei et al., 2016
	Pregnant Women	ELISA	28.8	12.4	Mohammed et al., 2022
	Aborted Woman	LAT	44.8	77	Fadhil et al. 2022
	Aborted Wollian	ELISA	24.8	6.8	1 uumi et ui., 2025
Karbala	Human	FLISA	18	40.8	Merzah 2016
	Aborted Women	FLISA	57.14	40.0	Farhan 2020
Babylon	Aborted Women	FLISA	5/.14	42.05	Hadi et al. 2016
Babyion	Healthy Women	ELISA	44	4	11aul et al., 2010
	Prograph Women	FLICA	35.4	3.2	AL Issuei at al 2010
Al Najof	Human	ELISA	20.5	_	Neori 2021
Al-Najai	Huillall	LLISA	33.7	5	N0011, 2021
	Malaa		42.1	0	
	Males	ELISA	54.83	51.61	Mandi et al., 2023
T 4.7 • .	Females	777.704	39.78	10.73	
vvasit	women	ELISA	17	0.8	Al-Sray et al., 2019
	Pregnant Women	ELISA	25.81	8.19	Al-Gharibawi & Alwaaly,2021
Thi-Qar	Human	ELISA	22	2	AL-Aboody & AL-Rekaby, 2017
AI-Muthanna	Males	ELISA	13.04	2.2	AlSaadawi & Alkhaled, 2015
	Females		13.9	0	
Basra	Cancer Patients	ELISA	82.1	23.2	Al-Tameemi et al., 2018
	Women	ELISA	41.4	0	Al-Mothafer & Awad, 2022

Table 1: The seroprevalence of *T. gondii* among humans in different Cities and Governorates of Iraq.

12.2. Techniques for Detecting *Toxoplasma gondii* using Molecular and Serological Methods

Toxoplasmosis can be diagnosed serologically by utilizing the enduring presence of certain antibodies in serum after parasite infection (Liyanage et al., 2021). IgM and IgG antibodies, as well as occasionally IgA directed against parasite protein antigens, are the main markers

used in the serological diagnosis of toxoplasmosis (Garg et al., 2019; Liyanage et al., 2021). The first generation of M-class antibodies is triggered by the presence of the parasite during the first contact with it. Accordingly, the presence of IgM antibodies may suggest an acute infection that was acquired recently (Garg et al., 2019). IgG immunoglobulins are then produced and remain in the body for years (Liyanage et al., 2021). The most widely utilized technique in clinical laboratories is enzyme-linked immunosorbent assay (ELISA) (Liyanage et al., 2021).

ELISA is widely used to identify IgA, IgG, and IgM anti-*Toxoplasma* antibodies (Liu et al., 2015). Numerous samples can be examined concurrently using ELISA due to its automated capabilities (Hamid, 2017). Additionally, the immunosorbent agglutination assay (ISAGA), modified agglutination test (MAT), indirect fluorescent antibody test (IFAT), and indirect hemagglutination assay (IHA) have also been employed (Garg et al., 2019).

Biological and environmental materials containing *T. gondii*-specific DNA or RNA can be amplified using molecular techniques such as polymerase chain reaction (PCR) (Su et al., 2010). Numerous PCR methods exist, such as nested, real-time, and traditional PCRs (Fallahi et al., 2014).

Nested PCR yields higher sensitivity and specificity for the B1 and REP 529 genes of *T. gondii* than conventional PCR (Fallahi et al., 2014). Very few numbers of target genes can be identified using real-time PCR, in contrast to nested and conventional PCRs (Liu et al., 2015).

11. Prevention and Control

To reduce or eliminate the danger of infection, several strategies can be used. Among these maintaining personal cleanliness and avoiding eating raw or undercooked meat, especially by pregnant women, should be careful because of the possibility of transplacental transmission (Van Knapen and Overgaauw, 2008). Wearing gloves when handling or gardening is recommended since the sporulated oocyst from cat stool can spread and persist in the environment for several months (Darbitz and Conrad, 2010; Liu et al., 2015). In addition, it is recommended to prevent contact with sporulated oocysts by frequently washing the cat litter box, using gloves, refraining from eating or smoking while gardening, as well as avoiding unwashed, raw vegetables (Van Knapen and Overgaauw, 2008).

Conclusion

The prevalence of *Toxoplasma gondii* in Iraq is high, with significant variation across different regions and populations, including both genders. Major risk factors identified include consumption of undercooked meat, age, occupation, residency, and contact with cats. These findings underscore the urgent need for improved diagnostic tools, public health education, and targeted interventions to minimize the prevalence of toxoplasmosis. Effective control measures, including routine screening and preventive strategies in high-risk groups, are essential to mitigate the disease burden in Iraq. Hand washing correctly after handling meat, contact with cats, or outdoor activity with soap and water before performing other tasks will prevent the transmission of *T. gondii* to humans. Further research is needed to provide a more precise understanding of the situation of this disease.

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