Toxoplasmosis among rich Sudanese females

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This book discusses about toxoplasma gondii infection and its epidemiology, transmission, life cycle, pathology and pathogenesis, symptoms, complications, diagnosis, treatment, prevention and control of that infection. Also it studied prevalence of toxoplasmosis among selected group of rich females. In this book you will find more information about toxoplasmosis as an opportunistic infection and as a zoonotic disease which affect huge number of people around the world, especially females whom trasmit infection to their children and in certain cases they loss them due to abortion or they born them with severe complications such as mental retardation so which take care from our wifes and our entire family and we should protect ourselves and our pets mainly cats from infection with toxoplasmosis.

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Toxoplasmosis among Rich Females

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Toxoplasmosis among rich females

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Contents

Dedication

To my Mother

And

To my father

&

To

All persons who help me at different stages of my life

Acknowledgements

By the grace of Almighty Allah and his help I completed this study, all praise to him and special thanks to volunteers who we so cooperative and hospitable.

Abstract

Background: toxoplasmosis is a disease that results from infection with the Toxoplasma gondii parasite, one of the world's most common parasites1. T. gondii has an environmental stage oocysts are shed in cat feces, sporulate, and disperse in the environment, where intermediate hosts get infected. Oocysts are an important source of infection for both animals and human.

Objectives:

The purpose of this study was to determine the prevalence of toxoplasmosis among selected group of rich Sudanese females.

Materials and methods:

A total of 45 rich volunteers females diagnosed serologically by latex agglutination method at parasitology laboratory, Faculty of Medical laboratory, Elrazi University, Sudan.

Result:

From a total of 45 rich volunteers' females diagnosed serologically by latex agglutination test, 33.3 % were seropositive and 67.7 were seronegative.

Discussion:

Toxoplasmosis is one of the most important diseases, which is more commonly diagnosed serologically. When we compare our study result with the results of previous studies we observed the lowest percentage of toxoplasmosis among rich Sudanese girls so toxoplasmosis is suspected to be reduced by improving the lifestyle of people.

Conclusion:

In summary we conclude that rich volunteer's girls is associated with low prevalence of toxoplasmosis, another studies is recommended.

Introduction:

Toxoplasmosis is a disease that results from infection with the Toxoplasma gondii parasite, one of the world's most common parasites [1] It's an important cause of reproductive failure in man and farm animals resulting in significant socio-economic losses worldwide[2]. Toxoplasmosis as other parasitic infections are dynamic in their distribution –some are endemic while many ubiquitous. The environment plays a key role in their survival and transmission often time[3].A toxoplasma infection occur 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) ,eating food that was contaminated knives , utensils ,cutting boards and other food that have had contact with raw, contaminated meat, drinking water contaminated with toxoplasma gondii, accidentally swallowing the parasite through contact with cat feces that contain toxoplasma gondii, mother – to-child (congenital) transmission, receiving an infected organ transplant or infected blood via transfusion [4], it can be also sexually transmitted infection with serious clinical consequence [5].

In most cases toxoplasmosis does not cause any symptoms and the person is not aware they are infected but in 10-20% of people infected with toxoplasmosis will develop symptoms similar to flu or glandular fever such as, high temperature (fever) of 38C OR overaching muscle, tiredness feeling sick, sore throat, swollen glands, these symptoms are usually mild and will normally pass within a few weeks. Toxoplasmosis can be serious if a women becomes infected while she is pregnant or few weeks before conceiving. This is because there is a chance the infection could be passed to her baby and if the infection spreads to her baby , it can cause ,miscarriage , stillbirth and congenital toxoplasmosis ,that cause serious problems that either noticeable from birth or develop several months or years later, such as brain damage, hearing loss and vision problems[6].

Toxoplasmosis is present in every country and seropositivity rates range from less 10% to 90%. The causative agent, Toxoplasma gondii, has a complex life cycle and is an important food borne pathogen. Human infection can result from the ingestion or handling of undercooked or raw meat containing tissue cyst (bradyzoite). Alternatively, it can result from direct contact with cats or from the consumption of water or food contaminated by oocysts excreted in the faeces of infected cats [7].

Background

Toxoplasmosis is caused by infection with the protozoan *Toxoplasma gondii,* an obligate intracellular parasite. The infection produces a wide range of clinical syndromes in humans, land and sea mammals, and various bird species. *T. gondii* has been recovered from locations throughout the world, except Antarctica.

Nicolle and Manceaux first described the organism in 1908, after they observed the parasites in the blood, spleen, and liver of a North African rodent, *Ctenodactylus gondii*. The parasite was named *Toxoplasma* (arc like form) *gondii* (after the rodent) in 1909. In 1923, Janku reported parasitic cysts in the retina of an infant who had hydrocephalus, seizures, and unilateral microphthalmia. Wolf, Cowan, and Paige (1937-1939) determined that these findings represented the syndrome of severe congenital *T gondii* infection.

There are 3 major genotypes (type I, type II, and type III) of *T gondii*. These genotypes differ in their pathogenicity and prevalence in people. In Europe and the United States, type II genotype is responsible for most cases of congenital toxoplasmosis.

T gondii infects a large proportion of the world's population (perhaps one third) but uncommonly causes clinically significant disease. However, certain individuals are at high risk for severe or life-threatening toxoplasmosis. Individuals at risk for toxoplasmosis include fetuses, newborns, and immunologically impaired patients.

Congenital toxoplasmosis is usually a subclinical infection. Among immunodeficient individuals, toxoplasmosis most often occurs in those with defects of T-cell–mediated immunity, such as those with hematologic malignancies, bone marrow and solid organ transplants, or acquired immunodeficiency syndrome (AIDS).In most immunocompetent individuals, primary or chronic (latent) *T gondii* infection is asymptomatic. A small percentage of these patients eventually develop retinochoroiditis, lymphadenitis, or, rarely, myocarditis and polymyositis. [8]

Epidemiology & Risk Factors

Toxoplasmosis is not passed from person-to-person, except in instances of mother-to-child (congenital) transmission and blood transfusion or organ transplantation. People typically become infected by three principal routes of 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)
- Eating food that was contaminated by knives, utensils, cutting boards, or other foods that had contact with raw, contaminated meat.

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 3 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 contain *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.

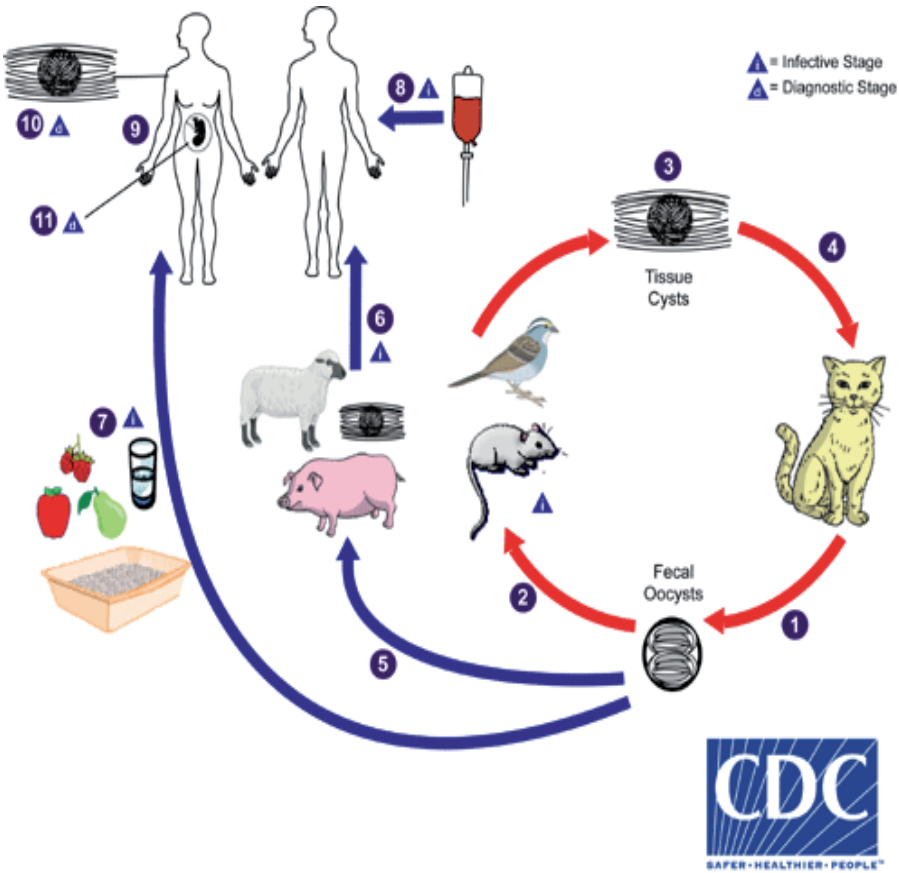
Mother-to-child (congenital) transmission

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.

Rare instances of transmission

Organ transplant recipients can become infected by receiving an organ from a *Toxoplasma*-positive donor. Rarely, people can also become infected by receiving infected blood via transfusion. Laboratory workers who handle infected blood can also acquire infection through accidental inoculation. [9]

Life Cycle:


= Infective Stage
= Diagnostic Stage
Tissue Cysts
Fecal Oocysts
CDC
SAFER·HEALTHIER·PEOPLE™

The only known definitive hosts for *Toxoplasma gondii* are members of family Felidae (domestic cats and their relatives). Unsporulated oocysts are shed in the cat's feces [1]. Although oocysts are usually only shed for 1-2 weeks, large numbers may be shed. Oocysts take 1-5 days to sporulate in the environment and become infective. Intermediate hosts in nature (including birds and rodents) become infected after ingesting soil, water or plant material contaminated with oocysts[2]. Oocysts transform into tachyzoites shortly after ingestion. These tachyzoites localize in neural and muscle tissue and develop into tissue cyst bradyzoites[3]. Cats become infected after consuming intermediate hosts harboring tissue cysts[4]. Cats may also become infected directly by ingestion of speculated oocysts. Animals bred for human consumption and wild game may also become infected with tissue cysts after ingestion of speculated oocysts in the environment[5]. Humans can become infected by any of several routes:

- Eating undercooked meat of animals harboring tissue cysts[6].
- Consuming food or water contaminated with cat feces or by contaminated environmental samples (such as fecal-contaminated soil or changing the litter box of a pet cat)[7].
- Blood transfusion or organ transplantation[8].
- Transplacentally from mother to fetus[9].

In the human host, the parasites form tissue cysts, most commonly in skeletal muscle, myocardium, brain, and eyes; these cysts may remain throughout the life of the host. Diagnosis is usually achieved by serology, although tissue cysts may be observed in stained biopsy specimens [10]. Diagnosis of congenital infections can be achieved by detecting *T. gondii* DNA in amniotic fluid using molecular methods such as PCR [11].[(10)]

Pathogenesis:

Most cases of toxoplasmosis in humans are acquired by ingestion of infected meat containing tissue cysts with bradyzoites or food contaminated with cat feces containing oocysts. Bradyzoites or sporozoites penetrate intestinal cells and then spread locally to the mesenteric lymph nodes and then to distant organs via the lymphatics and blood. Focal areas of necrosis may develop in a variety of organs and the clinical manifestations reflect injury to specific tissues. Tissue death is not the result of a Toxoplasma toxin, but is a consequence of the egress of the tachyzoites which destroys the host cell. Only 10-30% of toxoplasma infections are symptomatic and the most common clinical manifestation in immunocompetent adults is lymphadenitis and lymphadenopathy.

The most common symptom is swollen lymph nodes which may be associated with fever, headache, muscle pain, anemia and sometimes lung complications. Any lymph node can be infected but commonly the deep cervical nodes of the neck are involved. There is malaise, fever, and lymphocytosis which mimics infectious mononucleosis. The infection usually resolves on its own in weeks or months.

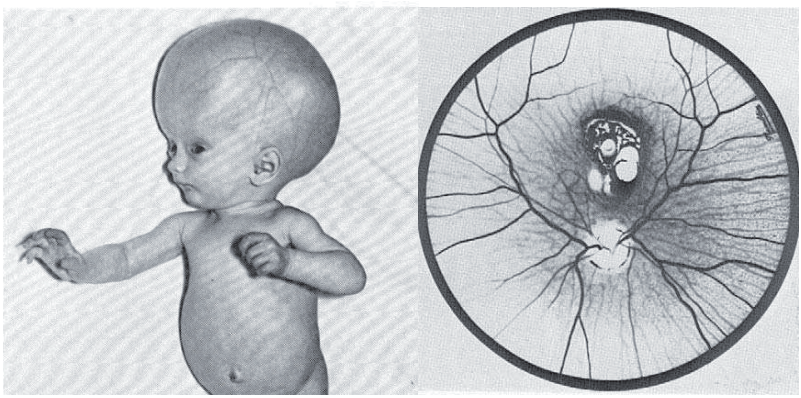
Although toxoplasmosis in humans is usually of little consequence there are two conditions where it can be devastating and lethal:

Toxoplasmosis during Pregnancy

In the child of a woman who acquires the infection for the first time during pregnancy. About 0.8% of pregnant females are expected to sero-convert. If *T. gondii* is acquired during the first pregnancy congenital toxoplasmosis will occur 45% of the time. Most severe effects are acquired during the first trimester, but if a chronically infected female becomes pregnant then the child will not suffer from toxoplasmosis.

Transplacentally infections (by tachyzoites) results in:

Subclinical	60%
Encephalitis Hydrocephalus Microcephalus Retinochoroiditis	30%
Abortion	10%



Hydrocephaly in a baby (left) and retinochoroiditis (right)

Newborns with toxoplasmosis may have fever, convulsions, mental retardation, blindness, pneumonitis, and hepatosplenomegaly. Recovery from congenital toxoplasmosis is rare and treatment is usually useless.

An Opportunistic Disease:

In the 1980's toxoplasmosis became one of the opportunistic diseases associated with immunocompromised patients, such as AIDS patients, transplantation patients and cancer patients treated with immunosuppressive drugs. In AIDS patients 25-50% develop encephalitis due to reactivation of chronic or latent infections, and if left untreated 90% will die from toxoplasma induced encephalitis due to necrosis and multiple abscesses. Symptoms include headache, fever, lethargy, altered mental status and can progress to focal neurological defects and convulsions. Lesions can be seen in CT scans and MRI imaging. Ocular involvement can also occur. Reactivation probably results from rupture of the cyst and renewed multiplication of bradyzoites into tachyzoites. The cause of cyst rupture is unknown. Myocarditis and pneumonitis may also occur. [11]

Complications of toxoplasmosis

For most people, toxoplasmosis causes no or few symptoms and passes without any further Problems. However, some people can develop serious complications.

Serious problems are more likely to develop if you become infected while you're pregnant or if you have a weak immune system.

Some of the main complications associated with toxoplasmosis are outlined below.

Ocular toxoplasmosis

The parasite that causes toxoplasmosis can lie dormant (inactive) in the back of the eye (retina) for many years.

If it becomes active again – for example, if you receive treatment that weakens your immune system – it can cause inflammation and scarring in the eye. This is known as ocular toxoplasmosis.

Ocular toxoplasmosis can affect one or both eyes and can cause:

- blurred vision
- floaters (small shapes floating in your field of vision)
- reduced vision or loss of vision

Medications are usually given to treat the infection, and steroids are often used to reduce any swelling in your eye. The scarring caused by toxoplasmosis will not clear up, but treatment may prevent it getting worse.

Congenital toxoplasmosis

If a woman becomes infected with toxoplasmosis for the first time during pregnancy or a few weeks before conceiving, there's a risk the infection could spread to her unborn baby. This is known as congenital toxoplasmosis.

Congenital toxoplasmosis can cause a range of problems that are either noticeable from birth or develop months or years later. The severity of the condition varies depending on when the mother became infected.

The baby's symptoms will usually be more severe if the mother is infected early on in the pregnancy, and less severe if they're infected later on.

Problems caused by congenital toxoplasmosis can include:

- hydrocephalus (fluid on the baby's brain)
- brain damage
- epilepsy – a condition that causes repeated seizures (fits)
- hearing loss
- ocular toxoplasmosis
- learning disabilities
- jaundice – yellowing of the skin and the whites of the eyes
- an enlarged liver or spleen
- cerebral palsy – a condition that affects a child's movement and co-ordination

Early treatment of congenital toxoplasmosis may help reduce the risk of serious or long-term problems, although it cannot reverse damage that has already occurred.

Cerebral toxoplasmosis

If you have a weakened immune system and you become infected with toxoplasmosis, the infection can spread to organs such as your eyes and brain because your immune system may not be able to fight off the infection.

If toxoplasmosis begins to affect the brain, it can cause a serious and life-threatening infection called cerebral toxoplasmosis.

Signs and symptoms of toxoplasmosis encephalitis and toxoplasmosis infections in people with immune deficiency can include:

- headaches
- confusion
- poor co-ordination

- seizures (fits)
- a high temperature (fever) of 38C (100.4F) or over
- slurred speech
- ocular toxoplasmosis

Medication can be used to treat the infection and reduce swelling in the brain, although it may not be able to cure the condition completely. [12]

Diagnosis

Diagnosis of toxoplasmosis is typically made by serologic testing. A test that measures immunoglobulin G (IgG) is used to determine if a person has been infected. If it is necessary to try to estimate the time of infection, which is of particular importance for pregnant women, a test which measures immunoglobulin M (IgM) is also used along with other tests such as an avidity test.

Diagnosis can be made by direct observation of the parasite in stained tissue sections, cerebrospinal fluid (CSF), or other biopsy material. These techniques are used less frequently because of the difficulty of obtaining these specimens.

Parasites can also be isolated from blood or other body fluids (for example, CSF) but this process can be difficult and requires considerable time.

Molecular techniques that can detect the parasite's DNA in the amniotic fluid can be useful in cases of possible mother-to-child (congenital) transmission. Ocular disease is diagnosed based on the appearance of the lesions in the eye, symptoms, course of disease, and often serologic testing. (13)

Morphology of Toxoplasma gondii

There are only two morphologic forms of trophozoites seen in humans, tachyzoites and bradyzoites. The infective form for humans is the oocyst. This form mas be encountered on occasion, especially where veterinary parasitologic techniques are performed. Thus, all three of these morphologic forms are discussed in this section. Oocyst. The typical infective form of Toxoplasma gondii, the oocyst, is similar in appearance to that of Isospora belli. The most notable difference between the two organisms is that T. gondii is smaller. The round to slightly oval form measures 10 to 15 µm long by 8 to 12 µm wide. The transparent oocyst contains two sporocysts, each with four sporozoites. The organism is bordered by a clear, colorless, two-layered cell wall. Tachyzoites. The actively multiplying, crescent-shaped tachyzoites range in size from 3 to 7 µm by 2 to 4 µm (Fig. 7-13; Table 7-9). One end of the organism often appears more rounded than the other end. Each tachyzoite is equipped with a single centrally located nucleus, surrounded by a cell membrane. A variety of other organelles are present, including a mitochondrion and Golgi apparatus; however, these structures are not readily visible.

Bradyzoites. Although there is evidence to support an antigenic difference, the typical bradyzoite basically has the same physical appearance as the tachyzoite, only smaller. These slow-growing viable forms gather in clusters inside a host cell, develop a surrounding membrane, and form a cyst in a variety of host tissues and muscles outside the intestinal tract. Such cysts may contain as few as 50 and up to as many as several thousand bradyzoites. A typical cyst measures from 12 to 100 µm in diameter.

Epidemiology

T. gondii has been found worldwide in many species, including carnivorous and herbivorous mammals and birds. It has also been found in every population group of humans investigated. However, the definitive host was shown to be cats, which have been associated with transmission of the parasite in every population investigated. In the United States, most infection occurs through ingestion of contaminated meat, especially pork and lamb. One survey of pigs in the US found a seroprevalence of 42% in breeder pigs and 23% in market pigs. Another study in the 1960s found *T. gondii* in 32% of pork chops and 4% of lamb chops in grocery stores. In humans the seroprevalence is 22.5% at the national level, according a study of 17,658 people from 1988 to 1994. Because there is the extra concern of congenital infection in mothers, additional Attention given to the prevalence in women of child-bearing age. The seroprevalence in the group of 15 to 44 year old women was reported to be 15%. [14]

TOXOPLASMOSIS

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:

- **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.

- **Sulfadiazine.** This antibiotic is used with Pyrimethamine to treat toxoplasmosis.

Treating people with HIV/AIDS

If you have HIV/AIDS, the treatment of choice for toxoplasmosis is also Pyrimethamine and sulfadiazine, with folinic acid (leucovorin). An alternative is Pyrimethamine taken with clindamycin (Cleocin).

Treating pregnant women and babies

If you're pregnant and infected with toxoplasmosis, treatment may vary depending on where you receive medical care.

If infection occurred before the 16th week of pregnancy, you may receive the antibiotic spiramycin. Use of this drug may reduce your baby's risk of neurological problems from congenital toxoplasmosis. Spiramycin is routinely used to treat toxoplasmosis in Europe, but is still considered experimental in the United States.

If infection occurred after the 16th week of pregnancy, or if tests show that your unborn child has toxoplasmosis, you may be given pyrimethamine and sulfadiazine and folinic acid (leucovorin). Your doctor will help you determine the optimal treatment.

If your infant has toxoplasmosis or is likely to have it, treatment with pyrimethamine and sulfadiazine and folinic acid (leucovorin) is recommended. Your baby's doctor will need to monitor your baby while he or she is taking these medications. [15]

Preventing Toxoplasmosis

You can prevent toxoplasmosis by:

- washing hands carefully
- keeping your food preparation areas clean
- cooking and storing your food at the appropriate temperatures
- being careful when dealing with animals
- using caution when gardening

Additional important information if you are pregnant or have a weakened immune system

Minimizing Your Risk

Wash your hands

- Wash hands after using the bathroom and changing diapers, and before handling or eating any food.
- Always wash hands after contact with farm animals, pets, animal feces, and animal environments.
- Hand Hygiene

Keep your food preparation areas clean

- Keep raw meat separate from produce and other foods when shopping for and storing groceries.
- Wash hands, cutting boards, countertops, cutlery, and utensils after handling uncooked meat.
- Wash raw fruits and vegetables before eating.
- Cross-Contamination
  Food and kitchen tools and surfaces may become contaminated from raw food products.

Cook and store your food at the appropriate temperatures

- Freeze meat for several days before cooking to inactivate the parasite and reduce the likelihood of infection.
- Thoroughly cook raw meat and poultry. Cook all meat to an internal temperature of 160° F and until it is no longer pink in the center or until the juices become clear.
  - Do not taste meat before it is fully cooked.

- Storage and Cooking Temperatures
  Learn more about storage and cooking temperatures

- Defrost food in the refrigerator, in cold water, or in the microwave. Food should be stored in a refrigerator that is 40°F or cooler or a freezer that is 0°F or cooler.

Be careful when dealing with animals

- Always wash hands after contact with farm animals, pets, animal feces, and animal environments.

Use caution when gardening

- Wear gloves when you garden or do anything outdoors that involves handling soil.

- Wash your hands well with soap and water after outdoor activities, especially before you eat or prepare any food.

Additional Information If You are Pregnant or Have a Weakened Immune System

- Avoid changing the cat litter box yourself; let someone else do it.

- If you have to change it, wear disposable gloves and wash your hands with soap and water afterwards.

- Change the litter box daily because the parasite is not infectious until 1-5 days after it is shed in the feces.

- Help prevent your cat from becoming infected by keeping it indoors and feeding it only canned or dry cat food.

- Do not feed your cat raw meat.
  Avoid stray cats and kittens and cover your outdoor sandboxes.

- Do not get a new cat while you are pregnant.[16]

Toxoplasmosis and HIV coinfection:

Toxoplasmosis associated with HIV infection is typically caused by reactivation of a chronic infection and manifests primarily as toxoplasmic encephalitis. This disease is an important cause of focal brain lesions in HIV-infected patients. Characteristically, toxoplasmic encephalitis has a subacute onset with focal neurologic abnormalities frequently accompanied by headache, altered mental status, and fever. The most common focal neurologic signs are motor weakness and speech disturbances. Patients can also present with seizures, cranial nerve abnormalities, visual field defects, sensory disturbances, cerebellar dysfunction, meningismus, movement disorders, and neuropsychiatric manifestations. Toxoplasmosis rarely presents as a rapidly fatal form of diffuse encephalitis.

Diffuse toxoplasmic encephalitis should be considered in patients with anti-*T gondii* immunoglobulin G (IgG) antibodies and CD4 T-cell counts of <100/µL who present with unexplained neurologic disease.

HIV-infected patients may develop extracerebral toxoplasmosis with or without concomitant encephalitis. Ocular and pulmonary disease are the most common presentations in patients with extracerebral toxoplasmosis. Patients with chorioretinitis present with blurred vision, scotoma, pain, or photophobia. Ophthalmologic examination reveals multifocal, bilateral lesions that typically are more confluent, thick, and opaque than those caused by cytomegalovirus (CMV). Vitritis may be accompanied by anterior uveitis. *T gondii* is a much less common cause of chorioretinitis in HIV-infected patients than CMV.

Patients with pulmonary toxoplasmosis have a clinical presentation that may be difficult to distinguish from *Pneumocystis jiroveci* pneumonia. A highly lethal syndrome of disseminated toxoplasmosis that consists of fever and sepsis like syndrome with hypotension, disseminated intravascular coagulation, elevated lactic dehydrogenase, and

pulmonary infiltrates has been described in HIV-infected patients. [17]

toxoplasmosis and behaviors of patient:

Toxoplasma gondii is a single-celled brain parasite spread by cats. Our feline companions are its preferred home and only in their bodies can it mature and reproduce. So like most parasites, *T.gondii* has a complex life cycle designed to get it into its final host. If it finds itself in another animal, it travels to the brain and changes the host's behavior to maximize its chances of ending up in a cat. For rodents, this means being eaten and infected individuals are less fearful of cats and more active, making them easier prey.

Humans can also contract the parasite, through contact with soil contaminated by the faeces of carriers or through eating infected meat. But since cats are very unlikely to eat humans, *T.gondii* reaches a cul-de-sac in our bodies. Still, there is nothing to stop the parasite, evolutionarily speaking, from trying out the strategies that work so well in other hosts. In rare cases, *T.gondii* infection causes a disease called toxoplasmosis that produces mild flu-like symptoms and only really threatens fetuses and those with weak immune systems. But in most instances, the parasite acts more subtly.

Carriers tend to show long-term personality changes that are small but statistically significant. Women tend to be more intelligent, affectionate, social and more likely to stick to rules. Men on the other hand tend to be less intelligent, but are more loyal, frugal and mild-tempered. The one trait that carriers of both genders share is a higher level of neuroticism – they are more prone to guilt, self-doubt and insecurity.

In individuals cases, these effects may seem quirky or even charming but across populations, they can have a global power. *T.gondii* infection is extremely common and rates vary greatly country to country. While only 7% of Brits carry the parasite, a much larger 67% of Brazilians are infected. Given that the parasite alters behavior, infection on this scale could lead to sizeable differences in the general personalities of people of different nationalities. This is exactly what Lafferty found.

Neuroticism is one of the most widely-studied of all psychological traits and Lafferty found that levels in different countries correlated well with the levels of *T.gondii* infection. The parasites' presence was also related to aspects of culture associated with neuroticism. Countries where infection was common were more likely to have 'masculine sex roles', characterized by greater differences between the sexes and their part in society and a stronger focus on work, ambition and money rather than people and relationships. Strongly infected societies were also more likely to avoid risk and embrace strict rules and regulations.

Obviously, different countries are also not just uniform populations, and increasing rates of migration mean that many countries are very ethnically and culturally mixed. However, This works in favor of Lafferty's theory as any mixing would serve to mask the link between infection and culture. If anything, the link is stronger than seen in this study.

These results are obviously very controversial and it would be imprudent to suggest that *T.gondii* is the major driver of human culture. It is just one of a number of influences that include genes, our physical environment and our histories. And Lafferty himself is quick

to point out caveats to his own results.

For a start, they do not imply that the parasite is causing these personality types; it could be that people with these traits are more likely to become infected. To establish the true direction of causality, Lafferty will need to find out how the parasite manipulates the mind. The general idea is that infection alters levels of the immune system's communication chemicals – the cytokines – which in turn alter levels of neurotransmitters like dopamine. But the details remain a mystery.

Nonetheless, the results are striking and one implication is that climate could have a larger effect on culture than previously thought. *Toxoplasma gondii*'s oocyst live longer in humid, low regions so variations in climate could influence the global distribution of cultural traits. Perhaps, this could explain why men and women perform more distinct roles in society in countries in warmer climates. Other factors can also affect the risk of infection, including cat ownership and national cuisines that include undercooked meat.

We like to think of culture as something governed by the collective actions of free-thinking and free-acting humans. But Lafferty's analysis shows us that if environmental factors like parasites can affect our thoughts and actions, no matter how subtly, they can have a strong effect on national cultures. In many cases, these effects could be much stronger than the agents that we normally believe to drive cultural trends. After all, more people around the world are infected with *Toxoplasma* than are connected to the internet.[18]

Symptoms of toxoplasmosis

In most cases, toxoplasmosis doesn't cause any symptoms and the person isn't aware they're infected.

This is because the immune system is normally strong enough to fight the infection and stop it causing serious illness.

However, some people will develop flu-like symptoms. There's also a risk of more serious problems if a woman becomes infected while she's pregnant, or if someone with a weak immune system becomes infected.

Flu-like symptoms

About 10-20% of people infected with toxoplasmosis will develop symptoms similar to flu or glandular fever, such as:

- a high temperature (fever) of 38C (100.4F) or over
- aching muscles
- tiredness
- feeling sick
- sore throat
- swollen glands

These symptoms are usually mild and will normally pass within a few weeks. [19]

toxoplasmosis and organs donation:

Toxoplasmosis in organ transplant patients can be a result of donor-transmitted infection, or reactivation of latent infection, or *de novo* infection. Solid organ transplants including heart, liver, kidney, pancreas and small bowel, and hematogenous stem cell transplants have been implicated in the risk of acquiring infection. In contrast to a benign course in immunocompetent individuals, the spectrum of illness is severe in transplant recipients. Clinical manifestations usually occur within the first 3 months of transplant and may present as encephalitis, pneumonitis, chorioretinitis, meningitis, and disseminated toxoplasmosis with multi-organ involvement. The diagnosis of toxoplasmosis in organ transplant patients is often difficult and is an integration of clinical, radiological, and microbiological workup. Preventive measures include pretransplant evaluation and chemoprophylaxis in view of rapidly progressing and fatal outcome of toxoplasmosis in immunocompromised individuals. [20]

Toxoplasmosis vaccine:

Toxoplasmosis, caused by an intracellular protozoan parasite, *Toxoplasma gondii*, is widespread throughout the world. The disease is of major medical and veterinary importance, being a cause of congenital disease and abortion in humans and domestic animals. In addition, recently it has gained importance owing to toxoplasma encephalitis in AIDS patients. In the last few years, there has been considerable progress towards the development of a vaccine for toxoplasmosis, and a vaccine based on the live-attenuated S48 strain was developed for veterinary uses. However, this vaccine is expensive, causes side effects and has a short shelf life. Furthermore, this vaccine may revert to a pathogenic strain and, therefore, is not suitable for human use. Various experimental studies have shown that it may be possible to develop a vaccine against human toxoplasmosis. Recent progress in knowledge of the protective immune response generated by *T. gondii* and the current status of development of a vaccine for toxoplasmosis are highlighted. [21]

pediatric toxoplasmosis:

History

Congenital toxoplasmosis is the consequence of transplacental hematogenous fetal infection by *T gondii* during primary infection in pregnant women. Primary infection in an otherwise healthy pregnant woman is asymptomatic in 60% of cases. Symptoms during pregnancy are frequently mild. The most common manifestations are fatigue, malaise, a low-grade fever, lymphadenopathy, and myalgias. Latent *Toxoplasma* infection with reactivation during pregnancy may lead to congenital infection only in immunocompromised women (most commonly, those with AIDS).

The classic triad of chorioretinitis, hydrocephalus, and intracranial calcifications cannot be used as a strict diagnostic criterion for congenital toxoplasmosis because a large number of cases would be missed. Congenital toxoplasmosis may occur in the following forms:

- Neonatal disease
- Disease occurring in the first months of life
- Sequelae or relapse of previously undiagnosed infection
- Subclinical infection

When clinically recognized in the neonate, congenital toxoplasmosis is very severe. Spontaneous abortions, prematurity, or still birth may result. Signs of generalized infection, such as the following, are usually present:

- Intrauterine growth restriction
- Fever
- Chorioamnionitis (usually bilateral)
- Cerebral calcification
- Abnormal cerebrospinal fluid (xanthochromia and pleocytosis)
- Vomiting
- Eosinophilia
- Abnormal bleeding
- Jaundice
- Hepatomegaly
- Splenomegaly
- Lymphadenopathy
- Rash

Neurologic signs are severe and always present. They include the following:

- Microcephaly or macrocephaly
- Bulging fontanelle
- Abnormal muscle tone
- Seizures

- lay of developmental milestone acquisition

De Most cases of chorioretinitis result from congenital infection, although patients are often asymptomatic until later in life. Symptoms include blurred vision, scotoma, pain, photophobia, and epiphora. Impairment of central vision occurs when the macula is involved, but vision may improve as inflammation resolves. Relapses of chorioretinitis are frequent but are rarely accompanied by systemic signs or symptoms.

Latent toxoplasmosis may reactivate in women with human immunodeficiency virus (HIV) and result in congenital transmission. Congenital toxoplasmosis in the infant with HIV appears to run a more rapid course than in infants without HIV. [22]

Evolution of Toxoplasma

T. gondii is unique among the apicomplexa parasites in that it has an extremely wide range of intermediate hosts - almost all warm-blooded vertebrates - although sexual reproduction is limited to felines. T. gondii also is unusual in that there are only three widespread genotypes. These are termed type I, II and III. Constructed a phylogenetic tree of the Apicomplexa using the rRNA and showed that these three clonal lines of T. gondii formed a single branch with two other apicomplexa which had a common ancestor around 12 M years ago. When they used the ITS1 region, which evolves more rapidly, they got a separation of H. hammondi and Toxoplasma from two other species.

Divergence of the Three Clonal Strains of T. gondii:

To determine the relative divergence between the three clonal strains of T. gondii, Su et al. looked at the frequency of single nucleotide changes (SNPs) in noncoding regions. Only two nonancestral changes were observed within the 40,670 bp analyzed.

However, when they looked at a set of three "exotic" strains of T. gondii, they observed 42 nonancestral mutations in 12,201 bp analyzed. They then estimated the rate of neutral mutations and found that a star like phylogeny best described the data. This indicates a genetic bottleneck followed by radiation. An age of 10^4 years was obtained for the origin of these three strains, whereas the origin of the exotic strains was more than 10^6 years.

The Acquisition of Oral Infectivity may have been the Selective Pressure to allow this Process to Occur

All three clonal lines of *T. gondii* can be transmitted orally via tissue cysts and can circumvent sexual reproduction in the cat. The exotics were less infective orally. This evidence suggests that there was a single meiotic event around 10,000 years ago which gave rise to the current population pattern of *T. gondii*. This is approximately equivalent to the time of human agriculture expansion and adaption of the cat as a domestic animal.[(23)]

Toxoplasmosis as zoonotic disease :

Toxoplasmosis is caused by the parasitic protozoan *Toxoplasma gondii*. People with weakened immune systems and infants whose mothers are infected during pregnancy can develop severe illness from this parasite. Most people infected with *Toxoplasma*, however, show no overt signs of disease.

Cats can acquire *Toxoplasma* by eating infected rodents, birds, or anything contaminated with feces from another infected animal. An infected cat can shed the parasite in its feces for up to two weeks. Once shed in the feces, the parasite must mature for one to five days before it becomes capable of causing infection. However, it can persist in the environment for many months and continue to contaminate soil, water, gardens, sandboxes, or any place where an infected cat has defecated. Although pregnant women or immunosuppressed individuals are often advised to remove cats from the household to reduce the risk of toxoplasmosis, direct contact with cats is very unlikely to spread infection with this organism.

Cats can transmit *Toxoplasma* to people through their feces, but humans most commonly become infected by eating undercooked or raw meat, or by inadvertently consuming contaminated soil on unwashed or undercooked vegetables. The symptoms of toxoplasmosis include flu-like muscle aches and fever, and headache. In rare cases, more advanced symptoms such as confusion, seizures, vomiting, or diarrhea may be observed.

Basic hygiene can prevent the spread of *Toxoplasma* from cats to humans. Wear gloves when handling potentially contaminated material (for example, when gardening or scooping the litter box, and be sure to wash your hands afterwards. Cover children's sandboxes when not in use to prevent wandering cats from defecating in them.

Pregnant women or immunosuppressed individuals are safest when other household members clean the litter box. [24]

neurotoxoplasmosis :

epidemiology:

Toxoplasma gondii is found ubiquitously and antibodies to the organism can be identified in 30% of all humans. The rate varies greatly from population to population and has a wide reported prevalence: from 6-90%. In most cases, the infection is asymptomatic. However, in immunocompromised patients (especially those with HIV/AIDS), infection can become established. Cerebral toxoplasmosis is found in 10-34% of autopsies on patients with HIV/AIDS.

The infection most likely occurs once the CD4+ count has dropped below 200 cells/mm

Clinical presentation :

In immunocompetent patients, acute encephalitis is extremely rare. Even in the immunocompromised symptoms are typically vague and indolent. Development of new neurological symptoms in these patients should raise high suspicion of cerebral toxoplasmosis.

Pathology:

Toxoplasma gondii is an intracellular parasite that infects birds and mammals. Its definitive host is the cat and other *Felidae* species. Excretion of oocytes in its faecal content followed by human contaminated uncooked consumption can lead to human infection. In immunocompetent individuals, it primarily causes a subclinical or asymptomatic infection. In immunocompromised individuals (e.g. AIDS patients), toxoplasmosis is the most common cause of a brain abscess.

Pathologically, parenchymal toxoplasma lesions have three distinct zones:

- a central avascular zone of coagulative necrosis
- an intermediate vascular zone containing numerous organisms
- An outermost zone of encysted organisms: Toxoplasma lesions do not have capsule. [25]

Congenital toxoplasmosis :

Overview :

- There are approximately 400 to 4,000 cases of congenital toxoplasmosis each year in the United States.
- Most infected infants appear healthy at birth.
- It can also cause serious and progressive visual, hearing, motor, cognitive, and other problems in a child.

Congenital toxoplasmosis is a disease that occurs in fetuses infected with *Toxoplasma gondii*, a protozoan parasite, which is transmitted from mother to fetus. It can cause miscarriage or stillbirth. It can also cause serious and progressive visual, hearing, motor, cognitive, and other problems in a child.

Symptoms of congenital toxoplasmosis:

- premature birth — as many as half of infants with congenital toxoplasmosis are born prematurely
- abnormally low birth weight
- eye damage
- jaundice, yellowing of the skin and whites of the eyes
- diarrhea
- vomiting
- anemia
- difficulty feeding
- swollen lymph nodes
- enlarged liver and spleen
- macrocephaly, an abnormally large head
- microcephaly, an abnormally small head
- skin rash
- vision problems
- hearing loss
- motor and developmental delays
- hydrocephalus, a buildup of fluid in the skull
- intracranial calcifications, evidence of areas of damage to the brain caused by the parasites
- seizures
- mild to severe mental retardation

causes of congenital toxoplasmosis :

You can get the *T. gondii* parasites in several ways:

- by eating uncooked or undercooked meat
- from unwashed produce
- by drinking water that is contaminated with the parasites or their eggs, though it is rare to get the parasites from water in the United States
- by touching contaminated soil or cat feces and then touching your mouth

If you become infected with the parasites during your pregnancy, you can pass them to your unborn child during pregnancy or delivery.

Diagnosis of congenital toxoplasmosis :

Your doctor may perform a blood test to detect the parasites. If you test positive for the parasites, they may perform the additional tests during your pregnancy to determine if your unborn baby is also infected. These tests include:

- ultrasound to check for fetal abnormalities, such as hydrocephalus
- polymerase chain reaction, or PCR, amniotic fluid testing, although this test may produce false negative or false positive results
- fetal blood testing

If your baby shows symptoms of congenital toxoplasmosis after birth, your doctor may perform one or more of the following tests:

- antibody test on the umbilical cord blood
- antibody test on your baby's cerebrospinal fluid
- blood test
- eye exam
- neurological exam
- CT or MRI scan of your baby's brain.

Treatment of congenital toxoplasmosis :

Some form of medication is typically used to treat congenital toxoplasmosis:

Drugs given during pregnancy :

- spiramycin, or Rovamycine, to help prevent the transmission of parasites from you to your fetus
- pyrimethamine, or Daraprim, and sulfadiazine may be given to you after the first trimester if it has been confirmed that your fetus is infected with the parasites

- folic acid to protect from bone marrow loss in you and your fetus, caused by pyrimethamine and sulfadiazine
- pyrimethamine, sulfadiazine, and folic acid, usually taken for one year

- steroids if your baby's vision is threatened or if your baby has high protein levels in their spinal fluid

Medication given after birth :

In addition to medication, your doctor may prescribe other treatments, depending on your baby's symptoms.

Prevention:

Congenital toxoplasmosis in the United States can be prevented if you, as an expecting mother:

- cook food thoroughly
- wash and peel all fruits and vegetables
- wash your hands frequently and any cutting boards used to prepare meat, fruits or vegetables
- wear gloves when gardening or avoid gardening altogether to avoid contact with soil that may contain cat waste
- Avoid changing the litter box. [26]

history of toxoplasmosis

In 1908, while working at the Pasteur Institute in Tunis, Charles Nicolle and Louis Manceaux discovered a protozoan organism in the tissues of a hamster-like rodent known as the gundi, *Ctenodactylus gundi*. Although Nicolle and Manceaux initially believed the organism to be a member of the genus *Leishmania* that they described as *"Leishmania gondii"*, they soon realized they had discovered a new organism entirely. They named it *Toxoplasma gondii*, a reference to its morphology (*Toxo*, from Greek (toxon); arc, bow, and (plasma); i.e., anything shaped or molded) and the host in which it was discovered, the gundi (gondii). The same year Nicolle and Mancaeux discovered *T. gondii*, Alfonso Splendore identified the same organism in a rabbit in Brazil. However, he did not give it a name.

The first conclusive identification of *T. gondii* in humans was in an infant girl delivered full term by Caesarean section on May 23, 1938, at Babies' Hospital in New York City. The girl began having seizures at three days of age, and doctors identified lesions in the maculae of both of her eyes. When she died at one month of age, an autopsy was performed. Lesions discovered in her brain and eye tissue were found to have both free and intracellular *T. gondii'*. Infected tissue from the girl was homogenized and inoculated intracerebrally into rabbits and mice; the animals subsequently developed encephalitis. Later, congenital transmission was found to occur in numerous other species, particularly in sheep and rodents.

The possibility of *T. gondii* transmission via consumption of undercooked meat was first proposed by D. Weinman and A.H Chandler in 1954 In 1960, the cyst wall of tissue cysts was shown to dissolve in the proteolysis enzymes found in the stomach, releasing infectious bradyzoites into the stomach (and subsequently into the intestine). The hypothesis of transmission via consumption of undercooked meat was tested in an orphanage in Paris in 1965; yearly acquisition rates of *T. gondii* rose from 10% to 50% after adding two portions of barely cooked beef or horse meat to the orphans' daily diets, and to 100% after adding barely cooked lamb chops. Such an experiment would nowadays be considered unethical.

In 1959, a study in Bombay found the prevalence of *T. gondii* in strict vegetarians to be similar to that found in nonvegetarians. This raised the possibility of a third major route of infection, beyond congenital and carnivorous transmission in 1970, the existence of oocysts was discovered in cat feces, and the fecal-oral route of infection via oocysts was demonstrated. [27]

Literature review:

A study done by Daryani A, et al, showed that the overall seroprevalence rate of toxoplasmosis is among general population in Iran was 39.3% [28]. A study done by Nebiye, et al showed that of 684 women, the prevalence of toxoplasmosis was determined to be 58.3%, .employment as seasonal farm worker, increasing age and having had three or more pregnancies were found to be the crucial associated risk factors that affect the prevalence of T.gondii infection [29]. A study done by K.Mohamed et al, showed that the seroprevalence rate of toxoplasmosis among 1146 serum samples was 43.6% [30].

Although most immunocompetent individuals infected with toxoplasmosis remain asymptomatic throughout life, worldwide this parasite cause a large amount of visual loss and morbidity, in addition to fatal infections in immunocompromised patients. Hygienic measures are cost-effective and can reduce the chance of transmission.

Justification:

Toxoplasmosis is a serious infectious disease that affect all age group and all people whether they are poor or rich and due to few data about prevalence of toxoplasmosis among rich people this study was conducted to fulfill the space with valuable data about the prevalence of toxoplasmosis among rich females.

Objectives:

The purpose of this study was to determine the prevalence of toxoplasmosis among selected group of rich volunteers Sudanese girls.

Materials and methods:

Study population:

A total of 45 rich Sudanese females from Khartoum state.

Data collection:

Data were collected from 45 rich volunteers Sudanese females from Khartoum state by parasitology staff at Elrazi University, Sudan.

Sample collection

5 ml of venous blood were collected from each rich volunteer Sudanese female in plain container and then serum was separated from each specimen.

Latex agglutination test:

Was used to screen the sera of each participant.

Data analysis:

Data of this study was analyzed by dividing the number of positive specimens to the whole specimens and then multiplies to 100 (percentage %)

(Number of positive specimens/all specimens) X100.

Ethical consideration:

This study was approved by the faculty of medical laboratory sciences, Elrazi University, and informed consent was obtained from each participant before sample collection.

Result:

Result	Number	Percentage %
Positive	15	33.3%
Negative	30	67.7%
Total	45	100%

Discussion:

Toxoplasmosis is one of the most important diseases, which is more commonly diagnosed serologically. When we compare our study result with the results of previous studies we observed the lowest percentage of toxoplasmosis among rich volunteers Sudanese girls so toxoplasmosis is suspected to be not affected by improving the lifestyle of people.

Conclusion:

In summary we conclude that high income is associated with normal prevalence of toxoplasmosis.

Recommendations:

Increasing the sample size and using another diagnostic methods are recommended to obtain more accurate and precise results.

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