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Public Health and Economic Significance of Toxoplasmosis

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Abstract: Toxoplasmosis is the most important worldwide zoonotic disease caused by the protozoan parasite known as *Toxoplasma gondii*. *Toxoplasma gondii* can be transmitted transplacentally from the mother to the fetus if the infection is contracted during pregnancy and causes abortion or congenital deformity. Postnatal infection is caused by ingestion of undercooked meat containing tissue cysts; ingestion of water, fruits, vegetables and shellfish contaminated with oocysts; or unintentional ingestion of cat feces or soil that contain oocysts. Toxoplasmosis leads to a myriad of diseases. In this manuscript the public health implication, economic consequence prevention and control strategies are reviewed. Timely treatment of man and animals with proper antibiotic, hygienic measures, proper disinfection and mass education are the measures to curtail the disease.

Key words: Economic Significance · Protozoa · Public Health · Toxoplasma

INTRODUCTION

Toxoplasma gondii is one of the well-studied parasites because of its medical and veterinary importance and its suitability as a model for cell biology and molecular studies with a unicellular organism. Toxoplasmosis is the most important worldwide zoonotic disease caused by the protozoan parasite known as *T. gondii* [1]. *T. gondii* infection represents the most prevalent parasitic zoonotic disease worldwide. This parasite is present on all continents and the rate of infection varies highly according to areas [2]. However, climate change has led to an increase of *T. gondii* infections in different regions of the world as a consequence of changing environmental conditions [3].

T. gondii has a complex life cycle with asexual reproduction taking place in diverse tissues of mammals and birds (secondary hosts) and sexual reproduction taking place in digestive epithelium of cats (primary host) [4]. Epidemiological distribution of toxoplasmosis is worldwide, but very high seroprevalence of *Toxoplasma gondii* is reported in South America and Africa including Ethiopia [1]. Toxoplasmosis infection occurs in every individual but sever in immunocompromised patients [5]. The public health relevance of toxoplasmosis relates to congenital and postnatal infection. The distribution of postnatal infection is highly variable worldwide, or even within a country, probably because of environmental, socioeconomic and cultural factors [6].

Felids, especially cats, are definitive hosts and represent the key element in the epidemiology of disease caused by this parasite. Almost all warm-blooded mammals, including livestock and human can serve as intermediate hosts [7]. *T. gondii* can infect all homeotherms and is responsible for many abortions and fetal malformations in human and animal. Toxoplasmosis is a major public health problem, with a high socioeconomic impact in terms of human suffering including the cost of caring sick, mentally retarded and blind children. The parasite is an extremely successful pathogen, responsive for significant morbidity and mortality, especially in congenitally infected and immuno-compromised individuals, although some subjects experience infection without overt disease or with mild symptoms [8]. In animals, the infection not only results in significant reproductive and economic losses,

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but also has implications for public health, since consumption of infected meat or milk can facilitate zoonotic transmission [9]. Therefore, the aim of this manuscript is to review the public health importance, economic significance, transmission way to human and animal and control of toxoplasmosis.

History of Toxoplasmosis: *Toxoplasma* was first isolated in 1908 in Tunis by Nicole and Manceaux from desert rodents which were maintained in the Pasteur institute. The rodents are called Gondi. In the same year it was also described in Brazil by Alfonso Splendore in rabbits. Then in 1909, the disease was differentiated from Leishmania and named as *Toxoplasma gondii*. Hence the name of organism is *Toxopalsma gondii* [10, 11]. The causative agent *Toxoplasma gondii* is a protozoan and a member of the sub order Eimeria. It is specific parasite of the definitive host members of the Felidae family, but has a wide range of intermediate hosts [12].

Epidemiology of Toxoplasmosis: *T. gondii* is widespread and capable of infecting many mammalian species. There is a high prevalence of toxoplasmosis throughout the world (20–90%), as well as a high resistance and persistence of the parasite in a broad spectrum of biological matrixes [13]. Epidemiological investigation in the US and elsewhere indicates that 60% cats are serologically positive to *Toxoplasma* antigen, the majority acquiring infection by predation. In Europe, parasitism rates in excess of 50% have been found in the meat of sheep and swine slaughtered in abattoirs. In Canada, the infection was found in 3.5 to 13.2% of pigs that underwent federal inspection, while in Japan, the rates are much lower. Cattle, on other hand, are more resistant to the infection; they have low, brief serological titers and parasites are isolated from them only rarely [14]. According to serological studies less than 1% in young adults in some areas, to 90% among older persons in other places. It is estimated that between 30 and 65% of all persons worldwide are infected with Toxoplasma [2].

Taxonomic Classification of Toxoplasmosis: Different species are assigned to Toxoplasma isolates based on the species of the host from which they were isolated. However, no biological and serological differences exist among the various isolates. Hence *Toxoplasma gondii* is the unique species of Toxoplasma organisms known to date [15]. The taxonomic classification of *T. gondii* is presented in Table 1.

Table 1: Taxonomic classification of *Toxplasma gondii*

Kingdom	Protista
Sub Kingdom	Protozoa
Phylum	Apicomplexa
Class	Conoidasida
Order	Eucoccidiorida
Sub order	Eimeriorina
Family	Sarcocystidae
Subfamily	Toxoplasmatinae
Genus	*Toxoplasma*
Species	*Toxoplasma gondii*

Sources: [2]

Growth and Survival Characteristics of Toxoplasma: Based on environmental circumstances, the time taken for oocysts to sporulate and become infectious ranges from one day to several weeks. Factors such as aeration and temperature affect the length of time required for sporulation to occur, with lower temperatures slowing the sporulation rate. *T. gondii* tissue cysts persist viable in infected meat stored at refrigeration temperatures of 4°C for up to 19 days. Cooking infected meat to internal temperatures of 67°C or higher inactivates the tissues cysts. Freezing meat at -10°C for 3 days or -20°C for 2 days or treatment with gamma irradiation at a dose of 75 krad is also sufficient to kill tissue cysts [16].

The duration of infectivity, however, decreases with increasing temperatures. Infectivity is maintained for at least 200 days in the temperature range of 10–25°C, for 1 month at 35°C, for 1 day at 45°C and sporulated oocysts become non-infective after 1 minute at 60°C. Unsporulated oocysts die within 24 hours when stored at 37°C, whereas sporulated oocysts can survive for over a month at 35°C and 9 days at 40°C. Constant freezing at -21°C kills unsporulated and sporulated oocysts within 1 and 28 days, respectively [17]. Tachyzoites that may be found in the milk of intermediate hosts are inactivated by pasteurization. Bradyzoites found in tissue cysts are resistant to gastric digestion, whereas tachyzoites are usually destroyed by the acid and proteolytic enzymes of the stomach. Experimental evidence indicates that tachyzoites may survive in acid-pepsin solution for up to 2 hours and the type of meal eaten may also increase the pH of the stomach and allow tachyzoites to traverse the stomach into the small intestine in an infective state [1].

Virulence and Infectivity: *T. gondii* virulence and infectivity are dependent on factors that control parasite-host cell interactions and/or moderate the host immune response [18]. The population structure of *T. gondii* is comprised of three highly abundant and overrepresented genetic lineages, commonly referred to as

genotypes I, II and III, amongst a diverse array of related genotypes. The three clonal lineages are very closely related but the small genetic differences result in distinct phenotypic differences in infectivity and virulence [19].

Most virulence studies have involved *genotypes I, II and III* and virulence has typically been assessed in a mouse pathogenicity model, with comparatively little known about human infection [18]. In the mouse model, highly virulent strains are typically *genotype I* whereas the vast majority of non-virulent strains are *genotype II* and *III*. In humans, the evidence for strain specific virulence is less well studied and relies predominantly on epidemiological evidence. The majority of human cases have been attributed to *genotype II* which is likely to be an artefact of an overrepresentation of this genotype in animals in Europe and the United States (US) where most human cases have been documented. The virulent nature of *genotype I* strains in mice may, however, extend to humans as severe ocular disease in otherwise immunocompetent adults have been attributed to *genotype I* strains [20]. Furthermore, non-*genotype II* strains have been associated with more severe disease at birth in congenitally infected newborns in the US. More recently, highly virulent atypical genotypes in French Guiana and Brazil have caused severe disease in immunocompromised individuals, foetuses and otherwise healthy individuals [1].

Mode of Transmission of Toxoplasmosis: The principal modes of *T. gondii* transmission are ingestion of fecal oocysts or tissue cysts and the transplacental transmission of tachyzoites from mother to unborn child. Infection with fecal oocysts may occur by accidentally ingesting contaminated soil (e.g. not washing hands after gardening or eating unwashed fresh produce), drinking untreated contaminated water, eating shellfish grown in contaminated water, or contact with cat feces (e.g. a cat litter box) [12]. Infection from tissue cysts may occur by consuming raw or undercooked meat, by accidentally consuming tissue cysts after handling raw meat and not washing hands thoroughly, or by cross-contamination of food prepared using unwashed utensils and chopping boards that have had contact with raw meat [21].

As tachyzoites are sensitive to environmental conditions they are usually killed rapidly outside the host and so are rarely involved in foodborne transmission of *T. gondii*. Organ transplant recipients can develop toxoplasmosis due to transmission of the parasite with the transplanted organ from a *Toxoplasma*-seropositive donor to a *Toxoplasma*-seronegative recipient. Heart transplantation is the most common type of organ transplantation procedure when this occurs, as cysts form in the cardiac muscles [22]. Infection of the feline definitive host occurs when a cat consumes an intermediate host (such as a mouse or bird) infected with tissue cysts. Upon ingestion of a tissue cyst by a susceptible cat, the walls of the cyst are digested by proteolytic enzymes and bradyzoites are released. The bradyzoites undergo asexual reproduction followed by sexual reproduction in intestinal epithelial cells to produce microgametocytes and macrogametocytes. The microgametocytes fertilize the macrogametocytes, leading to the production of zygotes. The zygotes differentiate into unsporulated oocysts and are shed in the feces of the definitive host [23].

Life Cycle of Toxoplasma: The life cycle of *T. gondii* includes both sexual and asexual multiplication. Sexual multiplication of *T. gondii* takes place in the gut of felines, making them the definitive hosts. Many feline species have been shown capable definitive hosts. If a cat ingests a *T. gondii* infected prey animal or meat, bradyzoites are released from the tissue cysts contained in their meal. In the previously uninfected cat, these bradyzoites invade epithelial cells of the cat's small intestine, where they start multiplying asexually [4, 8]. After five asexual stages of multiplication gametogony begins. Female macrogamonts and male microgamonts are formed and upon fertilization of the macrogamete by a microgamete, a zygote and an oocyst wall are formed. The nucleus divides twice and two sporoblasts (each with two nuclei) are formed. As the epithelial cells rupture, millions of oocysts containing sporoblasts are discharged into the intestinal lumen of the cat and eventually shed into the environment or cat litter box [15]. Depending on temperature and humidity these sporoblasts sporulate within 1 to 5 days to become infectious sporozoites with a haploid DNA content (4 sporozoites per sporoblast). Sporulated oocysts are infectious to cats (leading to another round of sexual multiplication), but even more so to an unequalled range of intermediate hosts: Probably all warm-blooded animals can be infected [6].

If an intermediate host ingests oocysts sporozoites will be released into the gut lumen and pass through the gut epithelium to enter cells in the lamina propria. In case an intermediate host ingests tissue cysts the released bradyzoites behave similarly to these sporozoites [3].

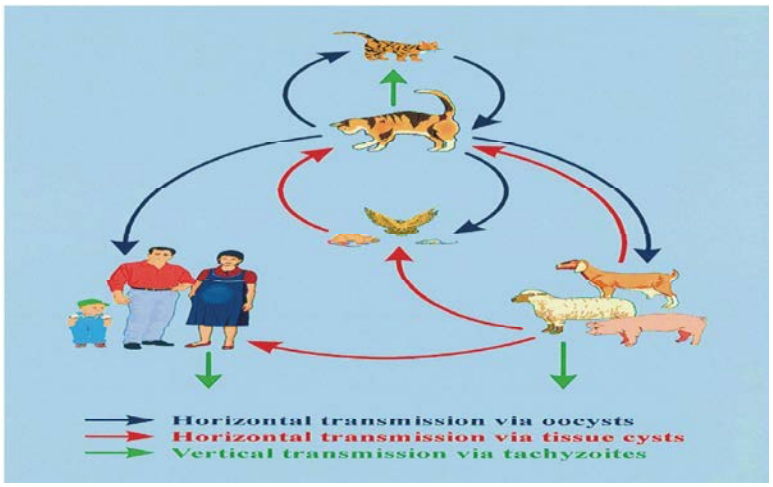

Fig. 1: Major routes of transmission of *T. gondii*. Source: [22]

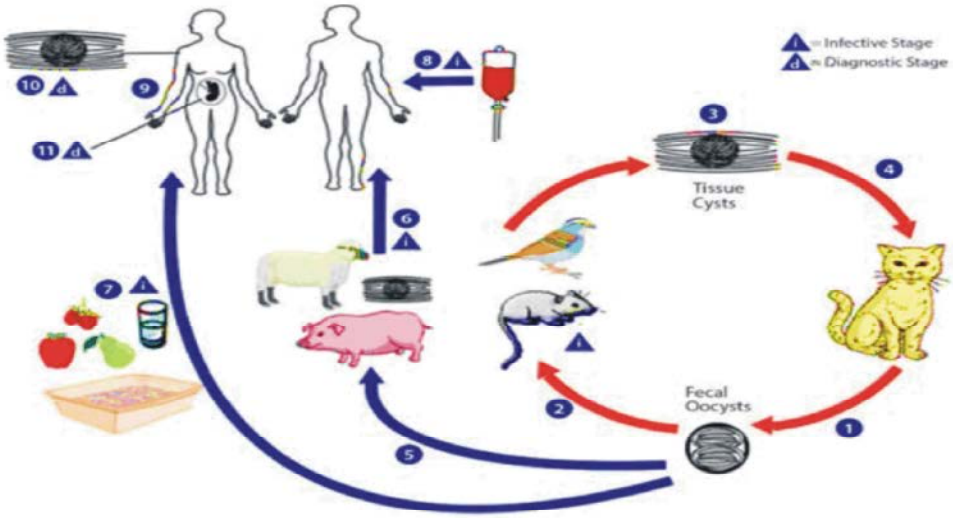

Fig. 2: Life cycle and mode of transmission of *Toxoplasma gondii* in different species of animals

Both sporozoites and bradyzoites transform into tachyzoites that enter a host cell where they divide rapidly until the cell bursts. Next, they continue to infect neighboring cells. Tachyzoites disseminate through the body by the circulation mostly intracellularly in leucocytes and finally enter various nucleated cells, but especially those in nervous and muscle tissue, where they transform into slowly dividing bradyzoites surrounded by a cyst wall [24].

The fate of these tissue cysts is not entirely clear. Tissue cysts seem to remain present lifelong in most hosts, although individual cysts are thought to rupture occasionally. This occasional cyst rupture is considered responsible for the persistence of antibodies in the host, because the released bradyzoites could stimulate the immune response in the immunocompetent host. Released bradyzoites transforming back into rapidly-dividing tachyzoites could explain the reactivation resulting in clinical symptoms or even fatal toxoplasmosis in immune-compromised individuals [17, 20]. Although intermediate hosts do not shed *T. gondii* they are infectious via carnivorism. Both felines and intermediate hosts are susceptible to infection via tissue cysts, which means that intermediate hosts are also infectious to each other. The ability to complete a cycle without the necessity to pass through the definitive host is quite unique in the world of parasites. Another interesting characteristic of *T. gondii* is the ability to change the behavior of rodents, causing them to specifically lose their aversion for cats. This trait provides an evolutionary advantage as it promotes *T. gondii* transmission [25].

- Unsporulated oocysts are shed in cat's feces.
- Intermediate hosts in nature (including birds and rodents) become infected after ingesting
- Sporulated oocysts in contaminated soil, water or plant material.
- Oocysts transform into tachyzoites shortly after ingestion. These tachyzoites localize in neural and muscle tissue and develop into tissue cyst bradyzoites.
- Cats become infected after consuming intermediate hosts harboring tissue cysts. Cats may also become infected directly by ingestion of sporulated oocysts.
- Food animals and wild game may also become infected with tissue cysts after ingestion of sporulated oocysts in the environment. Humans can become infected by multiple routes:
- Eating undercooked meat of animals harboring tissue cysts;
- Consuming oocysts in food or water contaminated with cat feces or by contaminated environmental samples (such as fecally contaminated soil or changing the cat litter box);
- Blood transfusion or organ transplantation; or
- Transplacental transmission of tachyzoites from mother to unborn child.
- Diagnosis is usually achieved by serology, although tissue cysts may be observed in stained biopsy specimens.

Source: [26]

Toxoplasmosis Manifestation in Livestock

Pig: Clinical toxoplasmosis in pigs is rare but there are cough, lack of coordination, tremors and diarrhea, with a 50% mortality rate, still-births, premature births and deaths soon after birth [1].

Cattle: Although cattle are considered a poor host for *T. gondii* can be successfully infected with *T. gondii* oocysts but due to innate resistance the parasite is removed or reduced to undetectable levels within a few weeks. There is no confirmed report of clinical toxoplasmosis in cattle but there is supposition as it causes abortion in cattle [27].

Camel: Acute toxoplasmosis is observed in camel with dysponea, many tachyzoites can be found in lungs and pleural exudates and *T. gondii* can be isolated from camel meat using cat biopsy [4].

Poultry: Toxoplasmic chickens show clinical signs like encephalitis, chorioretinitis, peripheral neuritis, torticollis, an inability to stand and lateral decumbency [7].

Sheep and Goat: Toxoplasmosis is common in sheep, goats, pigs and chickens as intermediate hosts; however, cattle and horses are notably resistant to the disease. In sheep, congenital infection is a leading cause of stillbirth and preterm lamb loss. Lambs that are born infected and survive usually exhibit normal growth, but they still represent a public health risk if their infected meat is consumed. Congenitally- infected lambs that survive the first week after birth usually grow asymptomatic and can be a source of infection for humans while adult goats can develop clinical toxoplasmosis involving liver, kidneys and brain [28, 29]. Toxoplasmosis can also occur in adult goats and the disease is more severe than in sheep. Congenital infection results in loss of kids before or after birth [14].

Pets: *Toxoplasma gondii* infection in cats is clinical significant, most severe in congenitally infected kittens and affected cats may appear depressed and anorexic and die suddenly with no obvious clinical signs [30]. Pneumonia is the most important clinical manifestation of feline toxoplasmosis and other common clinical manifestations are hepatitis, pancreatic necrosis, myositis, myocarditis, uveitis, dermatitis and encephalitis [31]. Primary toxoplasmosis in dogs is rare but common clinical signs of toxoplasmosis in dogs are pneumonia, hepatitis and encephalitis [32].

Public Health Importance and Economic Significance: Infection is asymptomatic in 80- 90% of non-pregnant, immunocompetent individuals and usually causes mild disease. Toxoplasmosis is a serious and often life-threatening disease in immunodeficient patients. Toxoplasmosis leads to a myriad of diseases. The risk-prone group of individuals including fetuses, new-born babies and immunological impaired patients develops chorioretinitis, lymphadenitis, or rarely, myocarditis and polymyositis [33]. It can cause more serious progression and complications such as abortion, when accompanied with some other infection such as human immunodeficiency virus (HIV) and catalyzes: birth defects, reproductive disorders and transmission of Hepatitis B virus (HBV). Children with acute congenital toxoplasmosis often die in the first month of life [34]. The economic

implication of *T. gondii* is mainly due to reproductive failure in animals including valuable livestock, disapproval of meat and wastage of milk, treatment cost in humans and vaccination cost in cats. Infection of dairy goats with *T. gondii* is widespread and constitutes a public health concern [35], resulting in significant reproductive losses [7].

Factors, such as management and hygienic standards in breeding, density of cats and ecological conditions are effective on the acquisition of *T. gondii* oocysts by animals. The most important risk factor for *T. gondii* infection was found to be under-cooked meat (lamb, beef and game). However, even true vegans can contract *T. gondii* infections, confirming the role of oocyst ingestion as a source of infection [36].

A multivariate analysis linked the risk of *T. gondii* infection with ingestion of raw ground beef and rare lamb, eating locally produced cured, dried, or smoked meat, working with meat, drinking unpasteurized goat's milk and having three or more kittens [37]. In one European multicenter study, between 30 and 63% of infections were recognized to consumption of undercooked or cured meat products and 6–17% to soil contact. Tachyzoites are killed by pasteurization and heating; therefore, it is advisable that milk, in specific goat's milk, should be pasteurized or boiled before human consumption. Any type of cooking would kill tachyzoites in eggs [2].

Clinical Symptoms of Toxoplasmosis: The infection presents with a wide range of clinical manifestations in man, land and sea mammals and various bird species [34]. Clinical manifestations of toxoplasmosis are caused by cell destruction due to multiplying tachyzoites, which most commonly affect the brain, liver, lungs, skeletal muscles and eyes. Oocyst-induced infection may be more severe than that induced by ingestion of tissue cysts. Signs may persist for one to twelve weeks but more severe disease is very rare in immunocompetent individuals [2].

Clinical Signs of Toxoplasmosis in Animals: The early symptoms include lethargy, persistent fever despite treatment with some antibiotics and anorexia. In some animals, toxoplasmosis may be characterized by hepatitis or pancreatitis. Central nervous system (CNS) signs, particularly common in older animals, vary with the site of the lesion and may include convulsions, restlessness, somnolence, head pressing, teeth grinding, behavior changes, hyperesthesia, atypical vocalizations, incoordination, trembling, opisthotonos or circling. Abortion, metritis and the birth of premature, can occur but seem to be rare. Ocular signs are common and may include generalized retinitis or irregular reddish, dark or pale retinal foci. Chronic low-grade infections may cause glaucoma, corneal opacity and panophthalmitis [38]

Clinical Symptoms of Toxoplasmosis in Human: Most persons infected by *T. gondii* after birth are asymptomatic unless immunosuppression occurs and the organism reactivates, however, some develop a mild disease or in rare cases, a more severe systemic illness [39]. Once infected, humans are believed to remain infected for life however; there is ongoing research on whether chronic *T. gondii* infection has an effect on reaction time [40].

A minority of healthy persons infected with *T. gondii* after birth develop mild symptoms such as fever, malaise and lymphadenopathy [39]. However, in rare cases, humans who were previously healthy have developed severe and even fatal disease, including pulmonary and multivisceral involvement, possibly from more virulent types of the organism [41]. Congenital toxoplasmosis generally occurs when a woman is newly infected with *T. gondii* during pregnancy and encephalitis is the most common clinical presentation of toxoplasmosis among persons with AIDS [42].

Among those chronically infected with AIDS, reactivated infection can result in encephalitis (inflammation of the brain), pneumonitis and neurologic diseases and can affect the heart, liver and inner ears, often with lethal outcome or less commonly, systemic toxoplasmosis. Rarely do infants who are born to mothers living with AIDS or mothers who are immunocompromised for other reasons, have chronic infection with *T. gondii* that was acquired congenitally in utero as a result of reactivated maternal parasitemia [43].

Diagnosis of Toxoplasmosis: The diagnosis is mainly carried out by microscopic examination, Culture or animal inoculation, Serological test and Polymerase Chain Reaction (PCR). *T. gondii* infection can be diagnosed indirectly with serological methods and directly by Polymerase Chain Reaction (PCR), hybridization, isolation and histology. Whereas indirect serological methods are widely used in immunocompetent patients, definitive diagnosis in immunocompromised people is mostly undertaken by direct detection of the parasite [44, 45].

Detection of *T. gondii* DNA in amniotic fluid by polymerase chain reaction assay has been shown to be a safe and accurate method of diagnosis. Serial fetal ultrasonographic examinations should be performed in cases of assumed congenital infection to detecting increase in size of the lateral ventricles of the central nervous system or other signs of fetal infection [32]. Measureable screening for IgG antibodies to *T. gondii* is used to determine the immune status of pregnant women and newborns. Anti-Toxo IgG antibodies may persist throughout life. Consequently, a steady anti-Toxo IgG titer shows earlier exposure, whereas a fourfold or greater rise shows active infection. Furthermore, among infants, serial determination of the anti-Toxo, IgG level will assist in determining between *T. gondii* infection that occurred congenitally (plateau level) or neonatally (increase in titer) [34].

Patients with HIV infection who are infected latently with *T. gondii* have variable titers of IgG antibody to *T. gondii* but rarely have IgM antibody. Although seroconversion and fourfold increase in IgG antibody titers may occur, the ability to diagnose active disease in patients with AIDS is impaired by immunosuppression. In HIV-infected patients who are seropositive for *T. gondii* IgG, *T. gondii* encephalitis is diagnosed presumptively on the basis of the presence of characteristic clinical and radiographic findings. If the infection does not respond to an empiric trial of anti- *T. gondii* therapy, demonstration of *T. gondii* organisms, antigen, or DNA in biopsied tissue, blood, or cerebrospinal fluid may be necessary to confirm the diagnosis [46].

Treatment of Toxoplasmosis

Treatment in Animals: There is no appropriate treatment for clinical toxoplasmosis in cats. Sulphonamides, trimethoprim, pyrimethamine and clindamycin, either alone or in combination, have been used to treat cats with clinical toxoplasmosis, with varying results. Ponazuril, an approved treatment for equine protozoal myelo-encephalitis caused by Sarcocystis neurona in horses, is excellent in treating acute toxoplasmosis in mice and in preventing recrudescent encephalitis in mice and should be evaluated in domestic cats [47].

Treatment in Human: Most individuals with healthy immune systems will not require treatment to *T. gondii* because the healthy immune can control the disease. The exception would be healthy mothers who acquire *T. gondii* for the first time after becoming pregnant as the fetus is in danger of acquiring the parasite [47]. The most effective treatment of toxoplasmosis is a combination of the oral antibiotic drugs pyrimethamine and sulfadiazine plus the B vitamin folinic acid [48]. Pyrimethamine is tolerated by most people, but it has some side effects like nausea, vomiting and diarrhea in the first few days of treatment while Sulfadiazine also causes skin rashes, itching and sensitivity to light, joint pain, fever and chills [49]. Sometimes, the combination of pyrimethamine + sulfadiazine may not be appropriate for everyone, therefore, another treatment option includes: pyrimethamine + clindamycin + folinic acid [50]. Spiramycin is one of the current drugs of choice for treatment of infected pregnant women. Treatment may decrease the severity of congenital toxoplasmosis or long term consequences, but possibly not the risk of transmission [2].

Control and Prevention: Feed/ration of the animals should not have access to cats which will help in avoiding feed contamination from cat feces. Preventive medication is to be given to young cats as they shed more oocytes in feces [51]. Educating people to prevent acquisition of new *Toxoplasma* infection and minimizing the risk of disease manifestations among HIV-*Toxoplasma* co-infected individuals is important. Two circumstances facilitate human postnatal *Toxoplasma* infection: the ingestion of bradyzoites in infected undercooked meat and the ingestion of oocysts via hands or food contaminated with the feces of infected cats. Hence, the control of human toxoplasmosis consists of avoiding these circumstances. Although the measures apply to everyone, pregnant women and immunodeficient individuals merit special attention, the former because of the possibility of congenital infection and the latter because of the risk of developing a severe case. Sanitary education should be directed particularly toward high-risk populations and it should focus on teaching people to avoid eating raw or undercooked meat and, in the case of food handlers, to prevent their hands from becoming contaminated [14].

There are general sanitation and food safety steps needed to be taken to prevent one from becoming infected with Toxoplasma. (i) Cats found to be shedding oocysts should be removed from the premises temporarily and treated to eliminate shedding. Since cats are usually meticulous groomers, it is unlikely that oocysts will be found on their fur. This means that regular handling will not be a significant risk. (ii) Microwave cooking, salting and smoking do not consistently kill all infective Toxoplasma stages. So meat should be frozen to –12°C for

at least 24 hours to kill Toxoplasma tissue cysts, but it must be noted that sporulated oocysts can survive at –20°C for up to 28 days [14]. (iii) Kitchen utensils and surfaces that have come in contact with raw meat should be washed with soap and scalding hot water to kill any bradyzoites or tachyzoites present. (iv) Individuals should always wash their hands thoroughly after contact with cat stool, litter or litter box. (v) Cat feces should be disposed of daily to reduce the risk of transmission. Feces and dirty litter can be disposed of in a septic system if the litter is biodegradable, sealed tightly in a plastic bag and placed in the garbage, or incinerated. Backyard compost units do not produce sufficient heat to destroy oocysts and other pathogens potentially present in fecal material. (vi) Keep cats out of sandboxes and other areas where children play to prevent the cats defecating there [2].

To prevent toxoplasmosis and other food-borne illnesses, food should be cooked to a safe temperature (71.1°C [160°F]). Fruits and vegetables should be peeled or thoroughly washed before they are eaten. Pregnant women should wear gloves when they are gardening or touching soil or sand, because of the possible presence of cat feces. Afterwards, they should wash their hands thoroughly, pregnant women should avoid nourishing the cats as well as avoid contact with cats and raw meat. Raw milk should not be drunk [51]. Avoiding the handling of stray cats, especially pregnant women and keeping cats indoors are good prevention measures [47].

T. gondii oocysts are resistant to most disinfectants but can be inactivated by iodine, formalin and ammonia. They are also destroyed with in10 minutes by temperatures greater than 66°C (150°F) and can be killed with boiling water. Tachyzoites and tissue cysts are susceptible to most disinfectants, including l% sodium hypochlorite and 70% ethanol. Tachyzoites are also inactivated at pH < 4.0. Freezing at –15°C for more than three days or –20°C for more than 2 days destroys a high percentage of the cysts [38].

CONCLUSIONS

Toxoplasmosis is triggered by the protozoan parasite, *Toxoplasma gondii* which commonly transmitted by ingestion of raw meat having tissue cysts; ingestion of water, fruits, vegetables and shellfish contaminated with oocysts; or accidental ingestion of cat feces or soil that contain oocysts. *Toxoplasma gondii* can also be conveyed transplacentally from the mother to the fetus and cause abortion or congenital deformity and severe in immunocompromised patients. Feed/ration of the animals should not have access to cats which will help in avoiding feed contamination from cat feces. Preventive medication is to be given to young cats as they shed more oocytes in feces. Educating the people to prevent acquirement of new *Toxoplasma* infection and diminishing the risk of disease manifestations among HIV-Toxoplasma co-infected individuals is important. Avoid the ingestion of bradyzoites in infected undercooked meat and the ingestion of oocysts via hands or food contaminated with the feces of infected cats is important.

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