

Review on Toxoplasmosis and its Current Status in Ethiopia

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Abstract: Toxoplasmosis is one of most important worldwide zoonotic disease caused by the obligate intracellular, protozoan parasite known as *Toxoplasma gondii*. Cats are definitive host for Toxoplasmosis but cattle, sheep and goat, poultry, pig and camel are reservoir host and facilitate the transmission of this disease to human. It is transmitted to humans by accidental ingestion of oocyst after cleaning an infected cat's litter box, accidental ingestion of oocyst with water and consumption of raw meat containing cyst causing fever, malaise, lymphadenopathy, pulmonary and multivisceral abnormality and encephalitis. The most commonly affected tissues are lungs, liver, brain, heart, placenta, eyes, spleen, lymph nodes and adrenal glands. Most forms of toxoplasmosis cases exposure result in subclinical infection with no clinical signs, but varies in different geographical areas of a country and it shows subclinical, sub-acute, acute and chronic. Diagnosis is by isolation of the parasite from patients and directly by polymerase chain reaction, hybridization, histology and more commonly by serological tests such as indirect fluorescent antibody and enzyme-linked Immunosorbent Assay. The aim of this review is to make a comprehensive document on current status of toxoplasmosis in Ethiopia. Toxoplasmosis common in Ethiopia and high prevalence is reported yet routine studies are needed to now accurate prevalence. Therefore, more studies in different geographical areas should be performed to design and implement appropriate intervention measures.

Key words: *Toxoplasma gondii* • Oocysts • Tissue Cysts

INTRODUCTION

Toxoplasmosis is one of the most prevalent parasitic infections of medical and veterinary importance due to its negative impacts on health and production. It is caused by an obligate intracellular parasite called *Toxoplasma gondii* [1]. *Toxoplasma gondii* is one of the most successful protozoan parasites due to its ability to manipulate the host immune system and establish a chronic infection [2].

Toxoplasmosis is found worldwide in nearly one-third of the human population [3]. Epidemiological distribution of toxoplasmosis is worldwide, but reported that very high seroprevalence of *T.gondii* in South America and Africa including Ethiopia [4]. Toxoplasmosis infection occurs in every individual but severe in immune compromised patients [5].

Toxoplasmosis transmission to the feline definitive host occurs when an immunologically naive cat ingests an infected intermediate host through predation. Animals and humans acquire infections mainly by ingesting food or water contaminated with sporulated oocysts and tissue cysts [6]. Major routes of transmission are different in human populations and depend on social culture, eating habits and/or environmental factors [7]. Although most infections are asymptomatic, reproductive losses in animals [8] and multiple disorders that include cognitive impairment and fatal encephalitis in humans could come about [2]. The congenital infection of fetuses results in hydrocephalus, intracranial calcification and retinochoroiditis [9]. Additionally, there are reports of associations between *T. gondii* infections with schizophrenia and bipolar disorder [10].

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Serological techniques such as the Sabin-Feldman dye test, Modified Agglutination Test (MAT), Enzyme-Linked Immunosorbent Assay (ELISA), indirect fluorescent antibody test (IFAT) and immune blotting are widely used to detect *T. gondii* specific antibodies [11].

Despite *T. gondii* being an important zoonotic pathogen and incidence of toxoplasmosis became increasing in Ethiopia, but there is only few documentation and understanding about the disease that addressed the multiple disorders it causes in humans, its impact in animal production, its temporal and spatial distribution and the risk factors associated with the occurrence of the disease in Ethiopia. Therefore, human awareness towards this disease can have a significant role in protecting public as well as pet. Taking this into account, the objectives of this paper is to review:

- ▶ Toxoplasmosis in animal and its public health importance
- ▶ Current status of Toxoplasmosis in Ethiopia

Toxoplasmosis

Etiology: *Toxoplasma gondii* is an intracellular protozoan organism with large number of intermediate hosts, including all warm-blooded animals and humans that belongs to the Kingdom Animalia, Phylum Apicomplexa, Class Protozoa, Subclass Coccidian, Order Eucoccidia, Family Sarcocystidae and Genus *Toxoplasma* [2]. The parasite *T. gondii* was discovered by scientists Charles Nicolle in North Africa and Alfonso Splendore in Brazil around 100 years ago. Felids, particularly the domestic cat, are its definitive hosts and the only animal species in which oocyst develops [6]. *Toxoplasma gondii* is regarded as one of the most successful parasites on the earth because of its broad host range, its high infection rates and its benign co-existence with the host [3].

Morphology: Tachyzoites are often crescent (banana) shaped and 2 μ m x 6 μ m in size. Tissue cysts vary in size from 5 μ m to 7 μ m and contain few to several hundred bradyzoites [11]. These bradyzoites differ only slightly from tachyzoites in having a nucleus situated towards the posterior end whereas the nucleus in tachyzoites is more central. Besides, bradyzoites are more slender than tachyzoites and less susceptible to destruction by proteolytic enzymes. Oocysts in freshly passed feces are unsporulated (non infective) sub-spherical to spherical in shape and 10 μ m to 12 μ m in diameter [3]. The sporulated oocyst contains four sporozoites and is 2 μ m x 6-8 μ m in size [12].

Life Cycle: The life cycle of *T. gondii* involves two phases. The sexual phase takes part in the definitive host and asexual phase in any warm-blooded animal including humans and in birds [2]. The sexual phase occurs in the cat's intestine after ingestion of any infective form. Members of the cat family can become infected by any of the three infective stages through ingesting of food or water contaminated with oocysts, by consuming infected tissues containing tachyzoites or bradyzoites, or trans placentally. Bradyzoites penetrate the epithelial cells of the small intestine and initiate a sequence of numerous generations where, after a variable number of schizogonies, microgamonts and macrogamonts are formed. The microgamonts fertilize the macrogamonts and oocysts are formed. The oocysts are excreted unsporulated and uninformative when passed in the feces [2]. Bradyzoite-induced cycle is known and the most efficient; nearly all cats fed tissue cysts can shed the oocyst, whereas less than 30% of cats fed tachyzoites or oocysts shed the oocysts [13].

Millions of oocysts are produced because of profuse multiplication of *T. gondii* in the feline intestine, usually without clinical signs. The asexual phase or extra-intestinal cycle occurs in any warm blooded animal after infection by any infectious stage after ingestion, sporozoites are released and invade the macrophages of the intestine. The sporozoites are differentiated into motile tachyzoites. Tachyzoites multiply rapidly by asexual process (endodyogeny) in a variety of cells and eventually encyst in several tissues, partly in the brain tissues. They are also responsible for congenital infections during pregnancy. When host immunity develops, the process slow down leading to chronic infection and the tachyzoites enter the bradyzoite (or cystozoites) stage, resulting in tissue cysts. Tissue cysts persist for a long time perhaps for the life of the host [14].

Epidemiology

Source of Infection: Three important source of toxoplasmosis are known, this include felines (cats), either (domestic and or wild), the environment (soil), intermediate host, the cat, may also be an intermediate hosts (any mammal, including man or birds). Note that the final host, the cat, may also be an intermediate host and harbor extra intestinal stages and infected cats shedding oocysts in their feces are the central source of infection, millions toxoplasma oocysts may be shed in a single stool of small animal (cats) and their

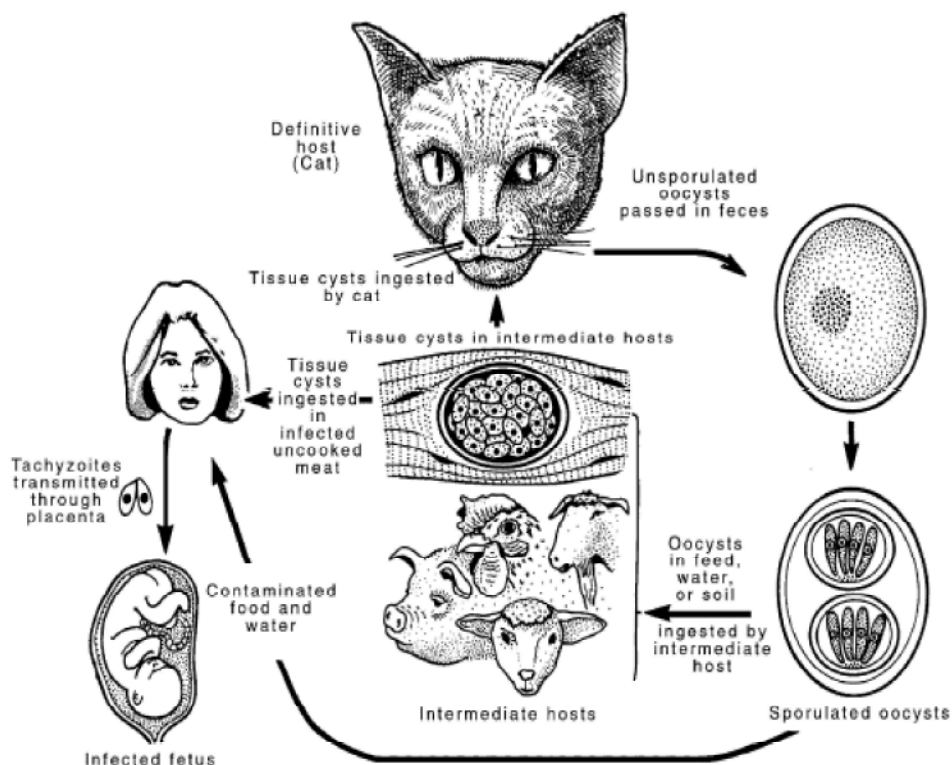


Fig. 1: Life cycle of *Toxoplasma gondii*[4].

survival depends on climatic and soil condition. For instance, oocysts could persist in moist soil for several months [15].

A wide range of intermediate hosts containing viable parasites in tissue cysts are a source of infection for small animals. Additionally, pasture or hay or stored feeds contaminated with feces are the main source of infection for herbivores. Among meat producing animals pigs, sheep and goats relatively often harbor *T. gondii* in edible tissue and therefore, raw or under cooked meat from these animals constitutes a major risk of infection for small animals including humans. On the other hand, as cattle appear to be naturally resistant of clearing the infection, beef is not generally regarded as an important source of small animal and human infection [9]. Moreover, in areas where goat's milk utilized, unpasteurized milk from acutely diseased goat is also an important source of infection, especially for cats. Although tachyzoites in the milk are likely to be destroyed by gastric juices. Penetration through sores in the oral mucosa might occur [16].

Geographical Distribution and its Occurrence:

Toxoplasmosis is worldwide in distribution and cats have a major influence in the epidemiology of the disease.

Islands with geographical isolation and absence of cats have been found to be free of toxoplasmosis. This fact is explained by the preying habits of this group and their diet that includes wild birds, rodents and *Toxoplasma* infected placentas and stillborn fetuses in some cases [17]. Historically, cats have been associated with domestic animals as an aid to rodent control [9].

Toxoplasma gondii infection in man and animals is widespread throughout the world, but varies in different geographical areas of a country. Causes for these variations are not yet known. Environmental conditions may determine the degree of natural spread of *T. gondii* infection. Infection is more prevalent in warm climates and in low-lying areas than in cold climates and mountain regions and in humid areas than in dry areas. This is probably related to conditions favoring sporulation and survival of oocysts in the environment [2].

Toxoplasmosis has been confirmed in 200 species of vertebrates, including primates, ruminants, swine, equine, carnivores, rodents, marsupial's, insectivores and numerous avian species. In general, among domestic animals' high reactor rates have been in cats, sheep, goats and swine, lower levels in horse and dogs and somewhat resistant in cattle, but in rare case it occurs with low level

[11]. From all, domestic cats have a key role in the epidemiology of *T. gondii* infection. Depending on the host species, the geographic area and the season of the year, up to 73% of small rodents and up to 71% of wild birds might be infected with *T. gondii*. In addition to this, the prevalence of *T. gondii* in feral cats is very high, when compared to owned cats, as they are more engaged at predated rodents and birds [18].

In Ethiopia, the prevalence of *T. gondii* is not well studied [19]. There are only few studies conducted in animals and the highest prevalence was reported in goats (82%) [20] and the lowest in cattle (6.6%) [21] and 96.77% of human infection in Addis Abeba abattoir worker [22].

Transmission routes: Widespread natural infection of the environment is possible since a cat may excrete millions of oocysts after ingesting as few as 1 bradyzoite or 1 tissue cyst and many tissue cysts may be present in one infected mouse [13]. Ingesting oocysts in water, soil or feed is probably the most common route for *T. gondii* infection in non-carnivorous mammals and birds [3]. The risk of *T. gondii* infection is higher in animals raised in extensive management than in intensive management. Sheep and goats on pasture show high sero-prevalence in many countries. Most *T. gondii* infections in pigs are due to oocysts via consumption of contaminated feed and soil [12].

Transmission to swine may also occur by the consumption of tissues of animals such as rodents and birds infected with *T. gondii* tissue cysts and by cannibalism. Studies have shown that *T. gondii* infection in pregnant sows can give rise to trans placental transmission, resulting in mummified fetuses, stillbirths and congenital infections, which have devastated the swine industry, causing significant economic losses [23].

Carnivorous animals get infected by feeding on chronically infected meat, prey and carrion [24]. *T. gondii* may survive in food animals for years in tissue cysts. Infection in felids and intermediate hosts, including humans, is acquired by ingesting uncooked or raw meat or carcasses contaminated with *T. gondii* cysts [25].

Risk Factor: A high rate of infection has been shown in sheep due to high rainfall, which allow a longer survival of oocysts on pasture. Sheep raised in high rainfall area without cat have almost no toxoplasmosis, whereas sheep raised in high rainfall with cats can have an infection rate as high as 32%. Direct sheep to sheep transmission might

occur by close contact with grossly infected placenta. Two risk factors for contracting toxoplasmosis in human being are; infants born to mothers who become infected with toxoplasma for the first time during or just before pregnancy and persons with severely weakened immune systems, such as those with HIV-AIDS. Illness may result from an acute toxoplasma infection or reactivation of an infection that occurred earlier in life [26].

Clinical Sign and Symptom: There are four forms of toxoplasmosis; subclinical, sub-acute, acute and chronic (latent). Most cases of exposure result in subclinical infection with no clinical signs. The sub-acute infections result in sudden death with few or no overt clinical signs. The acute form results from infection of tissues by tachyzoites and resultant tissue reactions. The organism affects various tissues and the clinical signs are preferable to the tissue involved. The most commonly affected tissues are lungs, liver, brain, heart, placenta, eyes, spleen, lymph nodes and adrenal glands. The latent form of toxoplasmosis refers to the quiescent tissue phase. Bradyzoites within cysts remain inactive and cause no tissue reaction. Recrudescence of these cysts to the acute stage may result from stress or other cause of immunosuppression [12].

Most infections are acquired via the gastrointestinal tract. In cats, the lesions that result from the intraepithelial cycle are usually not serious and do not produce clinical signs. In other animals tachyzoites causes damage to many organs. The clinical syndrome and the course of toxoplasmosis vary a great deal between species and age groups. The disease usually runs an acute course in cattle. Fever, dyspnea, nervous signs are seen earlier, followed by extreme lethargy and stillborn but not play significant role in causing bovine abortion. Pigs are highly susceptible and all ages can be affected. The principal manifestations in sheep are fetal resorption, abortion, mummified lambs and neonatal death [27].

The clinical disease in horse is rare. Natural outbreak in fowls has been reported and the parasite was transmitted to mice. In human beings, toxoplasma infections are associated with clinical signs such as mild fever and lymphadenopathy and may appear similar to mononucleosis or Hodgkin disease. Signs may persist for 1-12 weeks; more severe disease is very rare in immunocompetent individuals. Ocular toxoplasmosis (retinitis), encephalitis, hepatitis, myositis and pneumonia develop [28].

Worldwide toxoplasma encephalitis develops at some time in approximately 40% of individuals with AIDS. Approximately 10% of congenital toxoplasma infections result in abortion or neonatal death. Signs in this time include hydrocephalus, chorioretinitis, hepatosplenomegaly, microcephaly and small size fetus. Recently studies show that there is a cutaneous form of toxoplasmosis, where rare skin lesions may occur in the acquired form of the disease, including rosella and erythema multiform-like eruptions, prurigo-like nodules, urticaria and maculopapular lesions [29].

Ocular Toxoplasmosis: *Toxoplasma gondii* is the most common cause of retinochoroiditis in human worldwide, accounting for 28% to 55% of all cases of posterior uveitis [30]. Retinochoroiditis commonly occurs as a result of congenital infection but can also be due to an acquired or reactivated infection. It is usually a self-limiting disease with lesions healing within six weeks. Active lesions usually present with a white focus of crotizing retinochoroiditis close to old pigmented scars, these lesions are usually circular or oval in shape and they vary in size. In the congenital forms, the lesions are usually bilateral and central and in the acquired forms, the lesions are usually unilateral and solitary [31].

Parasites reach the eye as free tachyzoites or as cysts which rupture, releasing tachyzoites. Once infected cells lyse, tachyzoites invade the retina and multiply in the surrounding cells causing an inflammatory response [32]. Clinical manifestations of acute retinochoroiditis include tearing, pain, photophobia and progressive loss of vision over time, especially when there is macular or optic nerve involvement [4].

Congenital Toxoplasmosis: Toxoplasmosis is a significant cause of congenital disease. Congenital toxoplasmosis occurs in between 1 and 10 per 10, 000 live births in Europe. Transplacental transmission occurs when an immunocompetent woman acquires a primary infection during pregnancy, primary infections acquired four to six months before conception usually result in no transplacental transmission to the fetus [3].

Primary infection during pregnancy may result in severe damage or death of the fetus and long-term sequelae in the child, the risk of congenital infection increases from the first trimester (10-25%) to the third trimester (60-90%) with the development of a good blood flow [4]. The severity of the disease, however, is the

highest in the first trimester and lowest in the third trimester. Infection within the first two trimesters may result in death of the fetus in utero or spontaneous abortion. Infection in the last trimester usually results in newborns that are asymptomatic at birth, but may develop symptoms later in life [33].

Most children born with congenital toxoplasmosis are asymptomatic at birth; however, approximately 80% of them will develop neurological or ocular sequelae in life [28]. Approximately 10% of prenatal infections result in abortion or neonatal death and 10-23% of infected newborns show clinical signs of toxoplasmosis at birth. There may be mild disease such as reduced vision, or it may cause severe abnormalities such as blindness, mental retardation and epilepsy, the classic triad of signs is hydrocephalus, retinochoroiditis and intracranial calcifications and occurs in approximately 10% of all infected newborns [3].

Diagnosis: Diagnosis of toxoplasmosis is carried out by isolation of the parasite from patients and directly by polymerase chain reaction (PCR), hybridization, histology and more commonly by serological tests such as indirect fluorescent antibody (IFA) and enzyme-linked Immunosorbent Assay (ELISA) [34]. Adequately, serodiagnostic techniques have proved useful in diagnosing *Toxoplasma* infection in both humans and animals, for example modified agglutination test (MAT) ELISA. The MAT is the major recommended test for diagnosis of *T. gondii* in several animals and humans [2].

Treatment: The drugs which are used commonly are sulfadiazine (15-25 mg/kg) and pyrimethamine (0.44 mg/kg); they act synergistically. Though they cannot eradicate infection, the drugs are beneficial if given in the acute stage of the disease when there is active multiplication of the parasite. These drugs are believed to have little effect on the bradyzoite stage. Sulfonamides, trimethoprim, pyrimethamine and clindamycin have been used to treat cats with clinical [35].

Prevention and Control: Because *T. gondii* is transmitted by multiple modes and sources, it is difficult to establish the definite modes of transmission on an individual basis. But cats are keys to the transmission of *T. gondii* as they are the only definitive hosts that shed the oocysts. Therefore, prevention and control methods should target on Felidae [35]. Further options for the prevention and

control of toxoplasmosis in sheep and goats include; avoiding their exposure to the oocyst by keeping feeds in a closed container and by proper disposal of cat feces or chemoprophylaxis by adding the anticoccidial drug such as monensin to the feed is effective in reducing lamb losses [16].

Practicing good hygienic measures appears to be the best option to minimize transmission of *T. gondii* to humans. This can be done by increasing the public awareness through health education; especially for women of childbearing age, immune compromised groups and children. The lesson should include avoidance of consumption of raw or under cooked meat or unpasteurized goat's milk products and raw vegetables; wearing of gloves when cleaning cats litter and washing hands thoroughly with soap after handling suspected materials; washing kitchen materials with hot soapy water after contact with raw meat, poultry, or seafood, or with unwashed fruits or vegetables [14].

At present, there is no non-viable, effective vaccine to prevent *T. gondii* infection in animals and humans, with none on the horizon. The only available vaccine is a commercially produced live preparation for sheep. It consists of tissue culture grown 48 *T. gondii* tachyzoites attenuated by over 3000 passages in mice [36]. The vaccine stimulates effective protective immunity for at least 18 months following a single subcutaneous injection and sufficient reduction in reproduction wastage. The vaccine has a short shelf life and is a potential risk to humans [2].

Toxoplasmosis Disease Situation in Ethiopia:

Toxoplasma seroprevalence is variable, higher prevalence being observed in warm and moist areas than in cold or hot dry areas. Apart from this, variation may also be related to the age of the animals and husbandry practices. The overall prevalence recorded in sheep in Ethiopia and other African countries is 54.7% [37]. The overall seroprevalence of 26.7% recorded in goats from Ethiopia, the prevalence rates ranging from 11.5% to 39% have been recorded in various African countries including Ethiopia [38].

In Ethiopia, the prevalence of IgG antibodies to *T. gondii* has been determined by ELISA. One thousand and sixteen sera collected in different geographical regions were analyzed. Antibody titers >15 Iu/ml were detected in 74.4% of the specimens, out of which titers exceeding 200 Iu/ml in 1/3 were ELISA-positive sera. The highest antibody titers were found in children and 75% of young adults had sero-converted. As infection with the human immunodeficiency virus 1 (HIV-1) frequently leads to a resurgence of toxoplasmosis, the study conducted by and showed the occurrence of high anti toxoplasma antibodies in both HIV infected (74.2%) and non-HIV infected (83.3%) persons [39]. However, higher *T. gondii* antibody titers were recorded in persons infected with HIV when compared with HIV negative individuals in another study [40].

Table 1: Prevalence of animal toxoplasmosis in different part of Ethiopia

Author	Year of study	Location	Host	Method	Positive (%)
Zewdu <i>et al.</i> [41]	2010-2011	CE	Goat	ELISA	183 (19.74)
Gebremedhin <i>et al.</i> [42]	2010-2011	CE	Sheep	ELISA	357 (34.66)
Teshale <i>et al.</i> [20]	2005-2006	CSE	Sheep, goat	MDAT	480 (74.88)
Demissie and Tilahun [43]	2000-2001	CE	Sheep, goat	MDAT	159 (33.97)
Negashet <i>et al.</i> [44]	1999-2000	CE	Sheep, goat	MDAT/ELISA	75 (43.10)
Gebremedhin and Gizaw [45]	2013-2014	SE	Sheep, goat	ELISA	48 (26.09)
Gebremedhin <i>et al.</i> [45]	2011-2012	CE	Sheep, goat	DAT	111 (17.68)
Gebremedhin <i>et al.</i> [45]	2012-2013	CE	Camel	MAT	187 (48.57)
Tilahun <i>et al.</i> [46]	2012	CE	Chicken	MAT	48 (38.40)
Gebremedhin <i>et al.</i> [45]	2012-2013	CE	Chicken	MAT	183 (30.45)
Gebremedhin <i>et al.</i> [47]	2014	CE	Pig	DAT	129 (32.09)
Dubey [35]	2011	CE	Cat	MAT	33 (91.67)
Tiao [48]	2012	CE	Cat	MAT	41 (85.42)
Negash <i>et al.</i> [44]	1999-2000	CE	Cat	Coprology	5 (12.5)
Dubey [8]	2011	CE	Cat	Coprology	8 (22.22)

Note: CE, Central Ethiopia; CSE, Central and Southern Ethiopia; DAT, direct agglutination test; ELISA, enzyme linked immunosorbent assay; LA, latex agglutination test; MAT, modified agglutination test; MDAT, modified direct agglutination test; MDAT/ELISA, modified direct agglutination test and enzyme linked immunosorbent assay; n, sample size; NE, Northern Ethiopia; nr, not reported; SE, Southern Ethiopia; SFT, Sabin-Feldman dye test; WCSE, Western, Central and Southern Ethiopia.

Table 1: Prevalence of Human toxoplasmosis in different part of Ethiopia

Author	Year of study	Location	Host	Method	Positive (%)
Guebre Xabier <i>et al.</i> [49]	1990	CSE	human	ELISA	673 (73.23)
Woldemichael <i>et al.</i> [39]	1995-1996	CE	human	SFT/LAT	268 (78.82)
Negash <i>et al.</i> [40]	1999-2000	CE	human	MDAT	39 (60.00)
Shibre <i>et al.</i> [50]	2005	CE	human	ELISA	80 (88.89)
Shimelis <i>et al.</i> [51]	2007	CE	human	ELISA	297 (90.00)
Tedla <i>et al.</i> [52]	2009	CE	human	ELISA	434 (95.18)
Zemene <i>et al.</i> [53]	2011	SE	human	ELISA	163 (81.09)
Gebremedhin <i>et al.</i> [43]	2010-2011	CE	human	ELISA	346 (81.41)
Aleme <i>et al.</i> [54]	2011-2012	CE	human	ELISA	141 (94.00)
Muluye <i>et al.</i> [55]	2012-2013	NE	human	LAT	130 (76.47)
Tadesse <i>et al.</i> [56]	2012	NE	human	LAT	171 (40.52)
Endris <i>et al.</i> [57]	2010-2011	NE	human	LAT	341 (88.57)
Zewudu <i>et al.</i> [41]	2012-2013	NE	human	ELISA	51 (7.83)
Yohannes <i>et al.</i> [58]	2013	SE	human	ELISA	150 (88.24)

Note: CE, Central Ethiopia; CSE, Central and Southern Ethiopia; DAT, direct agglutination test; ELISA, enzyme linked immunosorbent assay; LA, latex agglutination test; MAT, modified agglutination test; MDAT, modified direct agglutination test; MDAT/ELISA, modified direct agglutination test and enzyme linked immunosorbent assay; n, sample size; NE, Northern Ethiopia; nr, not reported; SE, Southern Ethiopia; SFT, Sabin-Feldman dye test; WCSE, Western, Central and Southern Ethiopia

CONCLUSION AND RECOMMENDATIONS

Toxoplasmosis is one of the major diseases in pet animals and also cause risk for humans. The habits of consuming raw meat or under cooked mutton were significantly associated with toxoplasma infection in small animals. Since cats are the only definitive host, they cause great environmental contamination with oocysts. Likewise, the presence of cats in the house or in the nearby proximity correlated to *Toxoplasma gondii* infection for other animals. Thus, since small animals and humans can ingest raw meat or under cooked meat can pose to the infection. Older and younger (immuno suppressed) animals were comparatively affected including immuno deficiency (suppressed) humans. As a result of toxoplasmosis in human is zoonotic importance, this infection with the ever-increasing rate of the HIV/AIDS infection in Ethiopia, raw eating meat predisposing many HIV/AIDS infected person to serious complication from opportunistic pathogen. In line with the above observation the following recommendation forwarded:

- ▶ Practicing good hygienic measures appears to be the best option to minimize transmission of *T. gondii* to humans.
- ▶ Create public awareness on the infection in the rural and urban communities, veterinarian should provide advice on keeping health of small animals and reducing contamination of cats in pregnant women.
- ▶ Continuous community-based health education program should be set and all communication media utilized to create awareness about toxoplasmosis and associated risk factors and its transmission path way.

- ▶ Domestic and barn cats should be prevented from nesting and defecating in in hay, straw mows, grain stores or other loose piles of commodity of livestock feeds present in the farms.
- ▶ Cats should be provided with adequately cooked meat and should also be prevented from hunting birds and rodents.

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