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Review of Toxoplasmosis and its Zoonotic Importance

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Abstract: Toxoplasmosis is an important infection caused by single celled protozoan parasite, *Toxoplasma gondii* which is one of the world's most common parasites. Toxoplasmosis was first described in 1908 from a small rodent. Toxoplasmosis is considered to be one of the leading causes of death attributed to food-borne illness in the world. Most people affected never develop signs and symptoms. But for infants born to infected mothers and for people with compromised immune systems, toxoplasmosis can cause extremely serious complications. The parasite infects almost all worm blooded animals and serological evidence indicates that it is one of the most common of humans' infections throughout the world. *Toxoplasma gondii* is an obligate intracellular parasite that has a two-stage asexual cycle in warm-blooded animals and a sexual cycle in felidae. The disease is transmitted mainly by ingestion of infective stage of the parasite, organ transplant as well as blood transfusion in addition to the transplacental transmission which is very common. Toxoplasmosis can be presented in various forms of clinical manifestations depending on the immune status of the patient causing life threatening disease in AIDS patients. Pregnant women and sheep, cat owners, veterinarians, abattoir workers, children, cookers, butchers are considered as high risk groups. Timely treatment of man and animals with proper antibiotic, hygienic measures, proper disinfection, mass education and vaccination are the measures to prevent the disease. Toxoplasmosis is important parasitic zoonoses of wide range of animals including man and birds and a number of transmission ways in animals and from animal to humans, therefore this review is needed to make comprehensive document on current status the disease. In conclusion, the disease has high prevalence in farm animals and humans in different parts of the world. Therefore, more studies in different geographical areas should be performed to design and implement appropriate intervention measures.

Key words: Protozoa · *Toxoplasma gondii* · Toxoplasmosis · Pregnancy · Zoonosis

INTRODUCTION

Infection with the protozoan parasite *Toxoplasma gondii* has a worldwide distribution. This obligate intracellular parasite can infect humans as well as virtually all warm-blooded animals, including mammals and birds. While infection does not cause clinical illness in the majority of animal species, in some it causes acute life-threatening disease and in others, particularly sheep and goats, but also pigs, it manifests itself as a disease of pregnancy by multiplying in the placenta and fetus. Acute, potentially fatal, infections are recorded in New World monkeys [1], marsupials and certain other animals [2].

In sheep, goats and pigs a primary infection established during pregnancy may result in apparent infertility or in stillbirths and abortion, according to the stage of pregnancy at which infection was initiated. In a typical case of abortion, a ewe or doe infected in mid-gestation produces a stillborn lamb/kid a few days earlier than the predicted end of pregnancy. Theaborted fetus is often accompanied by either a weak sibling or a 'mummified' fetus [3]. For women, infection with *Toxoplasma* during or just before pregnancy can be particularly serious resulting in miscarriage, stillbirth or child disability .Infection in early pregnancy, when the fetus has only a rudimentary immune system, results in fetal death and resorption. Mothers that become infected

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in late pregnancy would be expected to produce infected but clinically normal offspring. Following an infection, either during or outside of pregnancy, the parasite would not be expected to cause abortion in any subsequent pregnancy. While recent research has questioned this conclusion [4, 5]; the majority of current thinking tends towards the view that recrudescence of a persistent infection during pregnancy leading to repeat abortions is not normally a significant occurrence [6, 7].

Toxoplasma gondii is an obligate intracellular parasite that has a two-stage asexual cycle inwarm-blooded animals and a sexual cycle in felidae. In the asexual cycle, the two developmental stages are the rapidly multiplying tachyzoite and the slowly multiplying bradyzoite. In acute infection, tachyzoites actively penetrate host cells where they multiply causing the cell to rupture and release organisms locally and into the bloodstream. As the host develops immunity, the parasite retains its overall size and shape but transforms into thebradyzoite stage and multiplies more slowly within tissue cysts to establish a persistent infection [8, 9].

Toxoplasma gondii readily infects human beings and while infection is relatively common (approximately 30% of the population depending on age and environment), clinical illness is relatively uncommon. Those particularly at risk of developing clinical illness include pregnant women, as the parasite can pose a serious threat to the unborn child if the mother becomes infected for the first time while pregnant and individuals who are immunosuppressed, such as tissue transplant patients, AIDS patients, patients with certain types of cancer and those undergoing certain forms of cancer therapy. These individuals are at risk of developing acute lethal infection if left untreated. The very young and very old may also be more susceptible [6, 10]. Toxoplasmosis is now also recognized to be a water-borne zoonosis. This method of transmission occurs where water treatment is ineffective or non-existent and there is a sizeable local felid population that contaminates surface water with oocysts [11, 12]. Linked to this there is now also an appreciation that marine mammals are becoming infected by waters from contaminated land and from untreatedurban sewage and recently, serious and life-threatening infections among immunocompetent people in French Guiana and Suriname and so, the objectives of this review are:

- To make comprehensive review on the status of the disease.
- To review the zoonotic significance of toxoplasmosis
- To review the disease situation in Ethiopia.

History of Toxoplasmosis: *T. gondii* was discovered by Charles Nicolle and Manceaux in 1908 in a North African rodent, Ctenodactylus gondii. The organism was named Toxoplasma because of its crescent shape (toxon, arc or bow as in bow and arrow, Greek and plasma, from, Greek) and gondii after its rodent host, the gondii. At about the same time *T. gondii* was described from a laboratory rabbit and then in a human in Panama. Interest in human infections began in 1923 when *T. gondii* was described in the retina of a hydrocephalic child and in congenital toxoplasmosis. Within 5 years Albert Sabin (of polio vaccine fame) had characterized the pathology of the congenital infection and in collaboration with Henry Feldman developed a specific serological "dye test" for toxoplasmosis. Using the dye test toxoplasmosis was determined to be a human infection worldwide in distribution [13].

Etiology: Toxoplasmosis is caused by *Toxoplasma gondii*, an obligate intracellular protozoan parasite in the order Coccidia and phylum Apicomplexa. It is a specific parasite of the definitive host felidae family, but has a wide range of intermediate hosts [14]. *T. gondii* has three infective stages: a) Tachyzoites -the rapidly multiplying form of the parasite present during the acute stage of infection in the intermediate hosts. b) Oocysts (containing sporozoites) -present only in the cat feces [15]. C) Tissue cysts: walled structures, often found in the muscles and central nervous system, containing dormant *Toxoplasma gondii* bradyzoites [6].

Host Range and Susceptibility: The cat plays a central role in the epidemiology of toxoplasmosis and the disease is virtually absent from areas where cats do not occur. Epidemiological investigations in the USA indicate that 60% of cats are serologically positive to toxoplasma antigen, the majority acquiring infection by predation. As might be expected infections are more prevalent in stray cats and the prevalence in farm animals and human being is shown below in Table 1 [16].

The true seroprevalence of toxoplasmosis in cattle is not known due to the inaccuracy of the standard serological tests for cattle, hence low in cattle, reflective of relative unimportance of toxoplasmosis in cattle [17]. In pigs an epizootic condition has been described in the USA, the symptoms, lesions and organisms were seen in

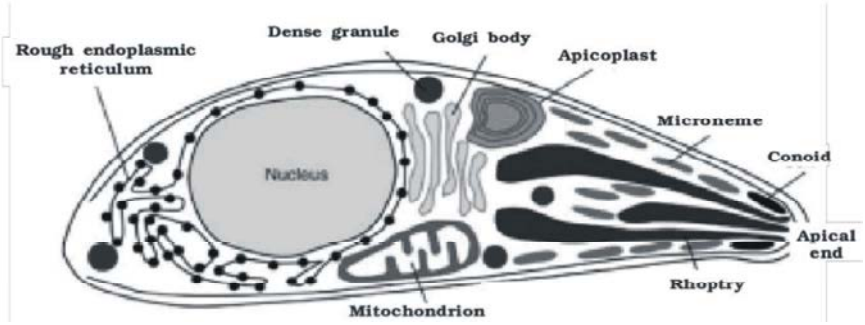

Fig. 1: Ultrastructure of *Toxoplasma gondii* [6]

Table 1: The seropositive prevalence of toxoplasmosis on worldwide basis on different animals and human being

Animals and human	Prevalence in %
Swine	22
Sheep	21
Goats	25
Human	>25

the lung, liver, kidneys and lymph nodes of the piglets and toxoplasma was recovered after mouse inoculation with material from the brain of the piglets' motherto explain. It the wide spread is diffic prevalence of toxoplasmosis in ruminants, particularly sheep, in view of the relatively low number of oocysts shed into the environment. Pregnant ewes are most commonly infected during periods of concentrate feeding prior to lambing, the stored food having been contaminated with cat feces in which millions of oocysts are present [16].

Tachyzoites have been demonstrated in urine, feces, milk, saliva and semen but are an unlikely source of infection. Tachyzoites cannot survive long in the environment and more susceptible to gastric digestion than bradyzoites. Oocysts may persist in the indoor and outdoor environment for more than a year [18].

Oocytes are extremely resistant to environmental influences and remain infective for a year in warm, moist climates and longer under cooler conditions. Recently flies, cockroaches, snails and earthworms have been implicated as transport hosts for toxoplasma oocytes. Uterine infection in woman may lead to the death of the child. Up to 1/3 of the world's population is estimated to carry a toxoplasma infection [17]. It isestimated that between 30-65% of world population is infected with toxoplasmosis [19].

Sources of Infection: Sources of *Toxoplasma gondii* infection is commonly acquire by ingesting food and water contaminated with the resistant stage of the parasite (oocystes) shed in the feces of infected cats or by ingesting the encysted stage of the parasite (tissue cysts) in infected meat. The common sources of *T. gondii* infection are: Cat feces -the sole source of infection for sheep, cattle and horses. Tissues of intermediate hosts -cats become infected as a result of ingesting tissues of intermediate hosts infected with the parasite, commonly these are rodents and small birds but all animals can be IH for *Toxoplasma gondii*. Rodents serve as reservoirs of infection for a long time. Feeds of animals contaminated with cat feces due to nests of cats. Actions of earthworms and other soil inhabitants - brings superficially buried feral cat feces to surfaces, which contaminates pastures. Ingestion of meat, dead rodents, cannibalized piglets and blood while tail or ear biting by pig [20].

Geographic Distribution: *T. gondii* occurs throughout the world. Seroprevalence rates are highly variable, but they are typically 10-30% in North America, northern Europe and Southeast Asia; 30-50% in Central and Southern Europe; and higher in Latin America and tropical regions of Africa. Small epidemics are seen occasionally, usually associated with contaminated food or water. Immunity is thought to be lifelong against most strains. This organism is especially prevalent in warm, humid climates, but significant numbers of animals and humans have been exposed even in very cold regions such as the Arctic [20].

Risk Factors: *Toxoplasma gondii* infection is associated with the following risk factors: Pathogen risk factors - oocysts are extremely resistant to external influences and can survive in the environment forat least one year. Oocysts are destroyed by exposure to temperatures between 90°C or 194°F for 30 s and 50°C for 2.5 min. Clinical disease occurrence is highly dependent on strains of *T. gondii* [11].

Environmental and management risk factors-the favorable environment for the survival of infective oocystes and feeding farm animals with cat feces contaminated pastures are associated with infection of *Toxoplasma gondii*. A high rate of infection has been shown in sheep due to high rainfall, which allow a longer survival of oocysts on pasture. Extensive animal farming is more risk full than intensive farming system. Host risk factors- 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 [11]. 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 and in infants (less than 5) and elders (greater than 65). Illness may result from an acute toxoplasma infection or reactivation of an infection that occurred earlier in life [21].

Transmission in Animals: *Toxoplasma gondii* is a tissue cyst forming protozoan microorganism that is vertically transmitted to fetus if a mother is infected during pregnancy. Premature birth, stillbirth and the birth of weak puppies are common in toxoplasmosis. Further spread of oocysts may occur via coprophagous insects which can contaminate vegetables, meat and animal fodder. Venereal transmission can occur in sheep. Other animals such as sheep, cattle and horses are infected due to the oocysts passed in the feces of the cat family [22].

The prevalence of infection is highest in young cats hunting for the first time. For the infection of cat the period of excretion of oocysts is short, approximately 2 weeks. Infection of farm animals occurs as the result of ingestion of feed or water contaminated with cat feces which contain infective oocysts and ingestion of contaminated stored feeds, contaminated pastures or contaminated water supplies [20].

Transmission to Human: The major modes of transmission of toxoplasmosis to human being include: Consumption of raw or undercooked meat containing toxoplasma cysts. Fecal - oral transfer of toxoplasma oocysts from cat feces directly or in contaminated food, water, soil, vegetable or fruit. Vertical transmission from mother to fetus, if primary infection occurs during pregnancy. Organ transplantation, Blood transfusion (iatrogenic), Transplacental transmission and accidental inoculation of tachyzoites in the skin are other modes of transmission [23].

Life Cycle of *Toxoplasma gondii*: Unsporulated oocysts of toxoplasma are passed in the feces of acutely infected cats. The oocysts usually sporulate in 1 to 5 days forming two sporocysts, each containing 4 infective sporozoites. When the oocysts are ingested by any warm-blooded host, the sporozoitesexcyst, invade intestinal cells and begin to divide asexually to produce tachyzoites. These then migrate throughout the body, invading tissue cells and multiplying until the cells rupture. Eventually, tachyzoitesexcyst, becoming bradyzoites within the cells of the central nervous system, muscles and sometimes other organs. If the host is eaten by other animals, the bradyzoitesexcyst in the intestine and the process is repeated, forming new tissue cysts. Only 20% of cats fed oocysts will develop a patent infection and the prepatent period may be 18 days or more [24].

Contrary to previous beliefs, studies have shown that oocysts can be shed in low numbers by previously infected cats that are challenged again with the parasite or that become immunosuppressed [26].

Pathogenesis: Infection from *Toxoplasma gondii* is acquired by carnivorism, ingestion of feces containing oocysts or congenitally [27]. In unexposed cat after ingestion of uncooked meat containing tissue cysts, *T. gondii* initiates entero epithelial replication. Bradyzoites are released from tissue cysts by digestion in the stomach and small intestine and invade intestinal epithelium by undergoing sexual replication, culminating in the release of oocysts in the feces. Oocysts first seen in the feces at three days after infection and released for up to 20 days after infection. After exposure to air for 24 hrs. Oocysts sporulate, become infective and may persist in the environment for up to one year. Cats generally develop immunity to *T. gondii* after the initial infection and therefore only shed oocysts once in their life time [21].

In all warm-blooded animals after ingestion of uncooked meat containing tissue cysts or feed contaminated with cat feces containing oocytes, *T. gondii* initiates extra intestinal replication. Bradyzoites and sporozoites, respectively are released and infect intestinal epithelium. After several rounds of epithelial replication,

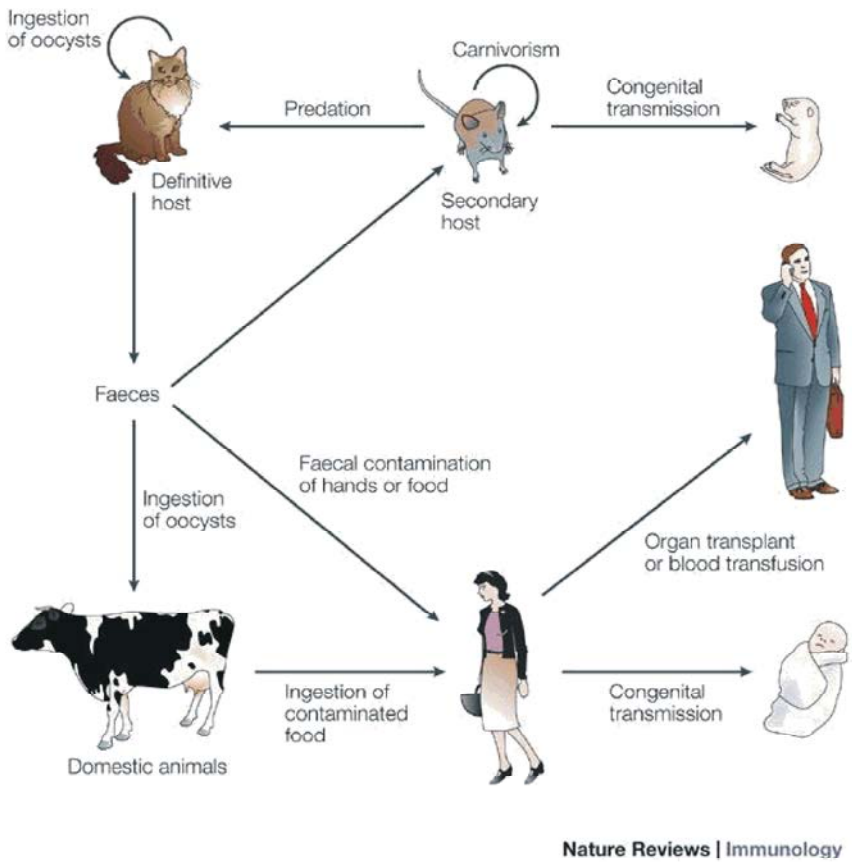

Fig. 2: Life cycle of toxoplasmosis [25]

Table 2: Major features of the life cycle of *Toxoplasma gondii* [24]

No	Features	Interpretation
1	Life cycle	Indirect or direct. Also between IH
2	Infective stage for FH	Bradyzoite cysts, tachyzoites and small oocysts
3	Infective stage for IH	Bradyzoites, tachyzoites and oocysts
4	Asexual phase	In many hosts
5	Sexual phase	In cats

tachyzoites emerge and disseminate via the blood stream and lymph. Tachyzoites infect tissues throughout the body and replicate intracellular until the cells burst, causing the tissue necrosis. Young and immunocompromised animals may succumb to generalized toxoplasmosis at this stage [21]. Older animals mount a powerful cell-mediated immune response to the tachyzoites. Tissue cysts are usually seen in the neurons and in cardiac and skeletal muscles. Tissue cysts remain viable in the host for many years [28].

Incubation Period: Incubation period varies according to infection dose. The incubation period in animals is probably similar to the 5-23 day incubation period in humans. In adults the incubation period for *Toxoplasma gondii* infection ranges from 10 to 23 days after the ingestion of undercooked meat and from 5 to 20 days after the ingestion of oocysts from cat feces [29]. Experimentally infected kittens developed diarrhea 5-6 days after inoculation. Reactivation can occur years after an animal was infected [20].

Clinical Signs and Symptoms: There are 4 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 referable

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 [24].

Clinical Symptoms Cats and Other Felids: The vast majority of infections in domesticated cats are asymptomatic. Most cases of toxoplasmosis seem to occur in young or immunocompromised cats, although older, apparently immunocompetent, animals have also been affected. Many cats develop respiratory signs, including dyspnea. Severe respiratory involvement is often fatal. Neurological signs may be prominent, especially in older cats. Kittens with encephalitis may sleep excessively or cry constantly [30].

Clinical Symptoms in Sheep Goats and Cattle: *T. gondii* usually infects adult sheep and goats without clinical signs; however, infections acquired during pregnancy can cause abortions, stillbirths and mummification or resorption of the fetus. The consequences are influenced by the timing of the infection. Toxoplasmosis seems to be very rare or absent in cattle, but fever, respiratory distress, nasal discharge and conjunctiva hyperemia were described in experimentally infected calves [30].

Clinical Signs in Man: Most healthy people who are infected with toxoplasmosis have no signs or symptoms and aren't aware that they're infected. Some people, however, develop signs and symptoms including: body aches, swollen lymph nodes, headache, fever and fatigue. Symptoms in immunocompromised patients are more severe and includes: headache, confusion, poor coordination and seizures. Blurred vision caused by severe inflammation of retina (ocular toxoplasmosis) [17].

Only a small number of babies who have toxoplasmosis show signs of the disease at birth. Often, infants who are infected don't develop signs which may include hearing loss, mental disability or serious eye infections until their teens or later [17]. 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 roseola and erythema multiform-like eruptions, prurigo-like nodules, urticaria and maculopapular lesions [15].

Complications: The toxoplasmosis infection can sometimes spread to the eyes and causes Ocular toxoplasmosis. This is called ocular toxoplasmosis and is possible even after the initial infection. The *T. gondii* parasite, which causes toxoplasmosis, can lie dormant (asleep) in the retina for many years. It can wake up at any time and start a new infection [11].

Ocular toxoplasmosis causes ocular lesions, Retinochoriditis, which are wounds in the eyes that are caused by inflammation and scarring. These can appear in: the retina (the nerve tissue that lines the back of the eye) and the choroid (the layer behind the retina that contains major blood vessels). The damage to the eyes is sometimes called retinochoroiditis and can cause eye problems including: loss of eyesight, a squint (when one eye looks in a slightly different direction to the other one), clouding of the eye lens (cataract), eye shrinking (microphthalmia) and loss of cells and tissue from the optic nerve, which connects the eye to the brain, resulting in poor vision (optic atrophy) [11].

Congenital Toxoplasmosis: In most cases, babies born with congenital toxoplasmosis develop normally after treatment with antibiotics. However, in up to four percent of cases, serious complications can developwithin the first years of life. These include: death, permanent brain damage and permanent visual impairment (partial or complete loss of sight) in both eyes [11].

Diagnosis of Toxoplasmosis

Diagnosis in Animals: Diagnosis of toxoplasmosis on clinical grounds alone is generally not possible because of the wide variety of clinical signs that can occur. Immunosuppression from drug administration or other means (e.g. canine distemper) may allow rapid multiplication and dissemination of toxoplasma organisms with production of clinical disease [31].

Fecal Examination: *T. gondii* is an intestinal coccidian of cats. Its oocysts are usually diagnosed using a standardfecal floatation. Oocysts are unsporulated in fresh feces and infective stages are crescent-shaped cells, approximately 5 micrometer long and 2 micrometer wide, with a pointed apical end and a rounded posterior end. They are enclosed by complex membrane called pellicle which helps to structural integrity and motility [32].

Mouse Bioassay: Diagnosis may also be confirmed by inoculation of suspicious material into mice and subsequent examination of exudates or tissues for

evidence of infection. Often a definitive diagnosis of toxoplasmosis is made only at autopsy. The possibility of immunosuppression resulting from the effects of canine distemper virus on thymus and other lymphatic tissues should be considered as a possible mechanism for activation of latent toxoplasma infection [16].

Diagnosis of Toxoplasmosis in Human Being

Physical Examination: The most common physical examination findings of toxoplasmosis include painless lymphadenopathy in immunocompetent individuals. Other findings include fever, malaise, myalgia and a maculopapular skin rash that spares the palms and the soles. In retinochoroiditis examination reveals multiple yellow-white cotton like patches with indistinct margins located in small clusters in the posterior pole. Vital signs are Fever and Tachypnea [16].

Serological Tests: Different serological tests often measure different antibodies that possess unique patterns of rise and fall with time after infection. A combination of serological tests is frequently required to establish whether an individual has been more likely infected in the distant past or has been recently infected. An IgM test is used to help determine whether a patient has been infected recently or in the distant past. Because of the significant potential of misinterpreting a positive IgM test result, confirmatory testing should be performed. Despite the wide distribution of commercial test kits to measure IgM antibodies, these kits often have low specificity and the reported results are frequently misinterpreted. In addition, IgM antibodies can persist for months to more than one year [20].

The Skin Test: The antigen is prepared by rapidly freezing and thawing Toxoplasma obtained from mouse inoculation. A dye test requires a minimum of 7 mm of either erythema or induration for positive reaction. It measures IgG and is the standard reference test for toxoplasmosis; however, it requires live *T. gondii* and thus it is not available in most laboratories [33].

Differential Diagnosis: Toxoplasmosis should be considered in differential diagnosis of multifocal CNS diseases, particularly in dogs that have been adequately vaccinated against distemper. Toxoplasmosis is rarely considered in a primary diagnostic list other than with problems of abortion and associated neonatal mortality. The differential diagnosis of abortion in cattle and sheep is related with brucellosis and in pig with leptospirosis. The cause of encephalitis in animals is related with viral infections (e.g. rabies), bacteria (*Listeria monocytogenes* and verminous encephalomyelitis related with migration of larva of parasitic species having somatic migration (Micronema *deletrix*, *Paraelaphostrongylustenuis*). Samples for confirmation of diagnosis are: a) Parasitology - fresh or chilled brain, lung, placenta, faeces b) Serology - fetal thoracic fluid c) Histology -placental cotyledons, lung, liver, brain, spinal cord, kidney and heart [22].

Necropsy and Laboratory Findings: In the early stages of infection in cats, oocysts can be demonstrated in feces by using conventional parasitological flotation techniques to concentrate and identify the organism. Examination of cerebrospinal fluid from animals with neurologic disease as a result of toxoplasma infection may reveal an increase in protein (greater than 35 mg/dl) and an increase in cell number, particularly neutrophils, reflecting the necrosis and hemorrhage present at sites throughout the nervous systems. The most characteristic gross lesion of toxoplasmosis is the presence of white chalky foci of necrosis and calcification up to 2 mm in diameter in cotyledons [34].

Morbidity and Mortality: The consequences of infection are most serious in pregnant women and immuno-compromised people. Estimates of the rate of congenital toxoplasmosis range from approximately 1person in 3000 births to 1 person in 10, 000 births. Most women pass *T. gondii* to the fetus only if they are first exposed during the pregnancy. However, a few immunosuppressed women have apparently transmitted reactivated organisms from previous infections [6].

In rare cases, a seropositive, immunocompetent woman has also produced a congenitally infected fetus, possibly either from a recrudescent infection or after she was infected by a different strain of the organism. The risk of transmission from an infected mother is estimated to be 25% during the first trimester, with the majority of these fetuses developing severe clinical signs. The fetal mortality rate at this time is high. Conversely, approximately 50-65% of infants are thought to become infected during the third trimester and 70-90% are asymptomatic at birth, although some will later develop clinical signs if they are not treated [6].

Treatment of Toxoplasmosis: Toxoplasmosis can be treated with a combination of drugs. If one is infected during pregnancy, the mother and the baby should be closely monitored during pregnancy and after the baby is

born. Persons with compromised immune systems, like AIDS patients, might need medication for the rest of their lives or for as long as they are immunosuppressed [22].

Treatment in Animals: Specific proven treatment for toxoplasmosis in exotic ruminants is lacking. Anti- inflammatory drugs may be beneficial in conjunction with antibiotic therapy for the treatment of series ocular inflammation. Treating with a combination of sulphamethazine and pyrimethamine has proved to be effective in mitigating the effects of experimentally induced toxoplasmosis in pregnant ewes. Treatment is administered over 3 days for 3 periods with an interval of 5 days. Chemotherapy with sulphadiazine (60 mg/kg/ day) for every 4-6 h and pyrimethamine (0.5-1mg/kg/day) as a single dose, limits the spread of infection until host immunity is acquired. These two drugs are synergistic and inhibitors of folate metabolism. Folinic acid (1 mg/kg/day) may be administered to prevent their toxic side effects [22].

Treatment in Human: In humans traditional therapy for clinical toxoplasmosis consists of a combination of pyrimethamine and sulphonamides but not recommended in pregnant women due to potential side effects on the fetus (due to inhibition of folate synthesis). Spiramycin, (a macrolide), is one of the current drugs of choice for pregnant women having toxoplasmosis. In order to prevent reactivation of disease in sero positive patients who develop AIDS, treatment with a trimethoprim - sulfamethoxazole, dapsone-pyrimetamine or fansider has been used. Antibiotics cannot destroy tissue cysts and may not be able to eradicate actively dividing parasites. If the presence of acute *T. gondii* infection in a pregnant woman is confirmed, treatment with spiramycin (Rovamycine) can be initiated in an effort to prevent transmission to the fetus [19].

Prevention and Control: There are two concerns in the control of toxoplasmosis in agricultural animals. The first one is to reduce the economic effects of infection in agricultural animals and the second one is to reduce the risk of human disease associated with consumption of infected meat especially meat of domestic animals [35]. On farms, control of toxoplasmosis is more difficult, but where possible animal feed should be covered to exclude access by cats and insects. Monensin and decoquinate have been administered to ewes in mid-pregnancy in attempts to control abortion due to toxoplasmosis.

Fortunately a live vaccine consisting of tachyzoites attenuated by a repeated passage in mice is now available for sheep. The vaccine consists of 10000-1000000 tachyzoites and it is given as a single dose IM at least 3 weeks prior to lambing [16]. Microwave cooking, salting and smoking do not consistently kill all infective toxoplasma organisms. Freezing meat to a -12°C for at least 24 h will kill most toxoplasma tissue cysts, but sporulated oocysts can survive at -20°C for up to 28 days. Washing kitchen, utensils and surfaces that have come in contact with raw meat with soap and scalding hot water will kill any bradyzoites or tachyzoites. Cats should be kept out of sandboxes and other areas where children play as cats may be inclined to defecate in them [19].

The following measures are important for the prevention of toxoplasmosis in general: Cooking all meat to a minimum temperature of 67°C or 153°F for 10 minutes; Peeling or thoroughly washing fruit and vegetables prior to consumption; Cleaning all surfaces and objects that come in contact with raw meat or unwashed fruit and vegetables; Avoiding contact with cat litter and garden soil, otherwise wearing gloves and washing hands thoroughly after gardening; Avoiding feeding raw meat to cats; Keeping cats indoors so they do not become infected by eating small prey by predation [27].

Zoonotic Importance: A major importance of toxoplasmosis in farm animals is its zoonotic potential while seroprevalence studies indicate relatively high rates of infection in farm animals, the infection is subclinical and *Toxoplasma gondii* has virtually no importance as a cause of clinical disease in farm animals with the exception of that associated with abortion and neonatal disease in sheep [36].

Humans are the intermediate hosts for *T. gondii* and approximately one half of the population of the United States is infected. Infection of man is acquired or congenital. Acquired infections occur in two ways: either from the ingestion of oocysts shed in the feces of cats by direct hand contamination or indirectly from ingestion of contaminated food and ingestion of undercooked meat containing toxoplasma cysts. Most infections probably arise from the ingestion of oocysts from cat feces that contaminate food or that are inadvertently ingested because of poor hygienic practices. However, human infection can also result from ingestion of bradyzoites and tachyzoites in meat or tissues that are eaten or handled. Beef is a minor source of infection, with pig, sheep and horse meat having greater risk [37]. Tachyzoites are secreted inthe milk of goats challenged with oocysts a toxoplasmosis, although the risk is minimal [38].

There is usually no clinical disease in humans infected with *T. gondii* or the disease is mild and self-limiting. Significant disease can occur in humans suffering from acquired immune deficiency syndrome (AIDS), malignancy, in those treated with cytotoxic or immunosuppressive drugs and in the very young and very old individuals. There is also a risk for abortion or congenital infection of the fetus such as hydrocephalus, intracranial calcification and retinochoroiditis. Toxoplasmosis poses an occupational risk for veterinarians, farmers and slaughterhouse workers who handle infected material. The risk is particularly high with contact with lambing ewes in infected flocks, veterinarians and farm workers, especially if pregnant, should take precautions to avoid infection when handling infected material [37].

The 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. Out of 1360 sera of domestic ruminants tested, in East Hararghe Zone, the presence of anti-*T. gondii* IgG antibodies was detected in 302 (22.2%). The highest seroprevalence of infection was observed in sheep (33.7%) and the lowest was in cattle (10.7%). in the study area [39].

In Ethiopia, the prevalence of IgG antibodies to *T. gondii* has been determined by ELISA in humans. 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, higher *T. gondii* antibody titers were recorded in persons infected with HIV when compared with HIV negative individuals [40].

CONCLUSION

Toxoplasmosis is important parasitic zoonoses of wide range of animals including man and birds, caused by protozoa known as *Toxoplasma gondi*i. Even though the disease has high rates of infection in farm animals; it has virtually no importance as a cause of clinical disease in farm animals except its association with abortion and neonatal disease in sheep. The major importance of toxoplasmosis is its zoonotic potential. Thus, the higher seroprevalence encountered in farm animal species used as a food source revealed the potential risk of *T. gondii* infection presented to people through consumption of their meat. Among the signs hydrocephalus, retinochoroiditis, convulsion and intracerebral calcifications in fetus and lymphadenitis and encephalitis in immunocompromised groups are the major findings of toxoplasmosis in human. Antibiotics and supportive therapy are used to treat clinical disease, but do not destroy the bradyzoites and do not eliminate infections.

In general, toxoplasmosis is highly important in its high-risk groups and in order to reduce its means of transmission, the following recommendations are forwarded;

- Adequate research should be carried out to estimate the public health importance of toxoplasmosis;
- Education of women of age 15-44 years, especially pregnant women, about the transmission, prevention and control of toxoplasmosis is required
- 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; Veterinarians, slaughter house and abattoir workers should take care when faced the sources of *T. gondii* in order to minimize its transmission;
- Individuals should always wash their hands thoroughly after contact with cat stool, litter or litter box;
- Thorough cooking of meat and washing of fruits and vegetables before eating.

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