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Toxoplasmosis: A global infection, so widespread, so neglected

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Abstract- Toxoplasmosis is an epidemiological paradox. It is one of the most prevalent and most widespread parasitic infections, yet one of the most ignored of all human infections. Between 30% and 65% of all persons worldwide are infected with Toxoplasma gondii, the causative organism. It is a coccidian parasite that infects mostly species of warm-blooded animals including man. It is asymptomatic among immunocompetent persons but presents a spectrum of clinical manifestations among the immunocompromised. Approximately 10% of congenital toxoplasmosis results in abortion or neonatal death. Infection may be associated with other diseases such as HIV/AIDS in humans or immunosuppressive therapy in any species. Toxoplasma encephalitis reportedly develops in approximately 40% of individuals with AIDS, and is fatal in 10-30% of these cases. This paper discussed updates and research trends on the biology, epidemiology, transmission, diagnosis, treatment of toxoplasmosis and made recommendations.

Index Terms- Toxoplasmosis, Cat faeces, *Toxoplasma gondii,* Neglected disease

I. INTRODUCTION

Toxoplasmosis is a parasitic infection with a worldwide distribution. The causative organism, *Toxoplasma gondii, is* a coccidian parasite that infects mostly species of warm-blooded animals including man. It was first described in 1908 when found in the blood, spleen, and liver of a North Africa rodent *Ctenodactylus gondii*. The parasite was named *Toxoplasma* (arc-like form) *gondii* (after the rodent). About a third of the world's human population is estimated to carry *Toxoplasma* parasite (Ryan and Ray, 2004). It is one of the most prevalent chronic infections that man has had to contend with (Jones *et al*., 2007). Wild cats play significant role in the spread of toxoplasmosis because they are the only animals that excrete resistant oocysts into the environment. All other animals, including man, serve as intermediate hosts in which the parasite may cause systemic infection, typically resulting in the formation of tissue cysts.

Cats generally acquirethe infection by feeding on infected animals, such as mice oruncooked household meat.During the first few weeks post-exposure, the infection typically causes a mild flu-like illness or no illness at all. Thereafter, the parasite rarely causes any symptoms in otherwise healthy adults. However, those with a weakened immune system, such as Acquired Immunodeficiency Syndrome (AIDS) patients or pregnant women, may become seriously ill, and occasionally may be fatal. The parasite can cause encephalitis (inflammation of the brain) and neurologic diseases, and can affect the heart, liver, inner ears, and eyes (chorioretinitis).

II. CLINICAL MANIFESTATIONS OF TOXOPLASMOSIS

The infection presents with a wide range of clinical manifestations in man, land and sea mammals, and various bird species (Akyar, 2011). When symptoms develop, they are nonspecific andinclude malaise, fever, sore throat, and myalgia. 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 (Tenter *et al*., 2000). Of clinical cases, quite few may develop ocular toxoplasmosis (retinitis), but this is more commonly associated with congenital infection (Perkins, 1990).

Approximately 10% of congenital toxoplasmosis results in abortion or neonatal death. Clinical signs of congenital Toxoplasmosis is not apparent at first in most cases butinfection acquired after birth isusually asymptomatic. Intrauterine meningoencephalitis could lead to the development of the following: cerebrospinal fluid (CSF)abnormalities, hydrocephalus, microcephaly, chorioretinitis,seizures, and deafness. Some of the severely affected infants die *in utero* or within a few days of birth. (Foulon *et al*, 1988). Other signs include maculopapular rash, generalized lymphadenopathy, hepatomegaly,splenomegaly, jaundice, and thrombocytopenia. The clinical course usuallyis benign and self-limited. Myocarditis, pericarditis, and pneumonitisare rare complications.Infants with congenital infectionare asymptomatic at birth in 70% to 90% of cases, although visualimpairment, learning disabilities, or mental retardation willbecome apparent in a large proportion of children several monthsto years later.

Infection may be associated with other diseases such as HIV/AIDS in humans or immunosuppressive therapy in any species (Akyar, 2011). *Toxoplasma* encephalitis reportedly develops in approximately 40% of individuals with AIDS, and is fatal in 10-30% of these cases (Patton, 1993). Among those chronically infected with AIDS, reactivatedinfection 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 (Remington *et al.,* 1995). 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.

III. THE BIOLOGY OF *T. GONDII*

T. gondii belongs to the Kingdom Protista, Subkingdom Protozoa, Phylum Apicomplexa, Class Sporozoasida, Order Euccidiorida, Family Sarcolystidae, Genus *Toxoplasma,* andSpecies *T. gondii.* The life cycle of *T. gondii* has two phases. The sexual phase of the life cycle takes place only in cats (family Felidae), which makes cats the primary host. The second phase, the asexual phase takes place in other warm-blooded animals, including cats, mice, man, and birds (Dubey *et al.,* 1998). In both the primary and secondary hosts, the *Toxoplasma* parasite invades cells and forms a vacuole. Inside this specialized vacuole, known as parasitophorous vacuole, the parasite forms bradyzoites, which are the slowly replicating versions of the parasite. The vacuoles containing the reproductive bradyzoites form cysts mainly in the tissues of the muscles and brain. Since the parasites are inside of cells, they are safe from the host's immune system, which does not respond to the cysts (Ira *et al;* 2009). Inside the vacuoles, *T. gondii* replicates itself (by endodyogeny) until the infected cell fills with parasites and bursts, releasing tachyzoites, the motile, asexual reproducing form of the parasite. Unlike the bradyzoites, the free tachyzoites are usually efficiently cleared by the host's immune system, although some of them manage to infect cells and form bradyzoites, thus maintaining the infection. (Wilson *etal*; 1980).

Tissue cysts are ingested by a cat during feeding, for example, on an infected mouse. The cysts survive passage through the stomach of the cat, and the parasites infect epithelial cells of the small intestine where they undergo sexual reproduction and oocyst formation. Oocysts are shed with the feces. Animals and man that ingest oocysts (for example while eating unwashed vegetables) or tissue cysts (while eating improperly cooked meat) become infected. The parasite enters macrophages in the intestinal lining and is distributed via the blood stream throughout the body (Dvorak, 2008). *Toxoplasma* is able to dysregulate host's cell cycle by holding cell division before mitosis. This dysregulation of the host's cell cycle is caused by a heat-sensitive secretion (with a molecular mass larger than 10 kDa). Infected cells secrete the factor which inhibits the cell cycle of neighboring cells. The reason for *Toxoplasma's* dysregulation is unknown, but studies have shown that infection is preferential to host cells in the S-phase and host cell structures with which *Toxoplasma* interacts may not be accessible.

IV. EPIDEMIOLOGY AND WORLDWIDE DISTRIBUTION 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 (Vaz *et al*., 2010). Serological studies have indicated incidence of *Toxoplasma* infections ranging from less than 1% in young adults in some areas, to 90% among older persons in other places (Montoya and Remington, 1995). It is estimated that between 30% and 65% of all persons worldwide are infected with *Toxoplasma* (Tenter *et al*., 2000). The interest in toxoplasmosis has been stimulated over the last few years by the finding that this infection is widespread biologically as well as geographically. It is widespread perhaps, because of its simple mode of contraction. Infection can occur simply by ingestion of oocysts following the handling of contaminated soil with cat litter or the consumption of contaminated water or food. However, no direct association has been found between cat ownership and the risk of toxoplasmosis in people (Walker *et al.,* 2008).

Tansmission of tachyzoites to the fetus can occur via the placenta following primary maternal infection. The incidence of prenatal *T. gondii* infections within the same or similar populations have been estimated to range from about 1 to 120 per 10,000 births (Patton, 1993). Rarely, does infection by tachyzoites occurs from ingestion of unpasteurized milk or by direct entry into the bloodstream through a blood transfusion or laboratory accident; but it does occur through transplantation of an organ that contain tissue cysts.

T qondii has been recovered from locations throughout the world, except Antarctica. Seroprevalence among adults could be as high as 90% in many countries (Akyar, 2011). Some studies have reported of incidence of primary maternal infection during pregnancy to range from about 1 to 310 per 10,000 pregnancies in different populations in Europe, Asia, Australia and the Americas (Opsteegh *et al*., 2011). In Brazil, a recent report has it that 53.03% of pregnant women were positive for IgG and 3.26% were positive for IgM (Vaz *et al.*, 2010). *T. gondii* seropositivity among pregnant women, their fetuses, neonates, and AIDS patients have been investigated in Qatar. Widespread occurrence is confirmed in East Mediteranean (Akyar, 2011). In many developing countries, the exact prevalence of toxoplasmosis is not well articulated unlike in the developed world (Lindstrom *et a*l., 2006), but there is large variation between countries. In France, for example, around 88% of the population are carriers, probably due to a high consumption of raw and lightly cooked meat (Ancha and Szyfres, 2003). In Germany, the Netherlands and Brazil there are high prevalence rates of 68%, 80% and 67% respectively (Henriquez *et al*, 2009). In Britain about 22% are carriers, while in South Korea the rate is 4.3% (Tenter *et al*., 2000). The *T. gondii* seroprevalence for the Dutch human population has decreased from 40.5% in 1995/1996 to 26.0% in 2006/2007 (Hofhuis *et al*., 2010). This is thought to be an effect of the decreased prevalence in consumption animals, especially in pigs, due to increased intensive indoor farming. A stable infection pressure from the environment is suggested by the unchanged seroprevalence in sheep when compared to studies in the eighties (Opsteegh *et al.,* 2011). However, differences may have been missed due to methodological differences between studies (Opsteegh *et al.,* 2011). In the United States, the Center for Disease Control and Prevention, reports that the overall seroprevalence as determined from specimens collected by the National Health and Nutritional Examination Survey (NHANES) between 1999 and 2004 was 10.8%, while seroprevalence among women of childbearing age

(15 to 44 years old) was 11% (Torda, 2001. In certain areas of western Europe and Africa, the sero-positivity rate is reported to be approximately 50 – 78% in individuals with AIDS (Montoya and Liesenfeld, 2004).

Toxoplasmosis has long been reported to be widespread in West Africa (UNAIDS, 2004). In sub-Saharan Africa, toxoplasmosis often remain undetected and untreated due to insufficient diagnostic procedures (Lindstrom *et al*, 2006). Several studies have shown a consistently high *T. gondii*-seroprevalence for this region, ranging from 35% to 84% in different African countries south of Sahara (Tenter et al., 2000). Considering that around 30–50% of those coinfected with HIV and *T. gondii* are expected to ultimately develop toxoplasmosis, the high seroprevalence combined with the HIV-pandemic indicate that 2.5–10 million people in this region may be at risk dying from toxoplasmosis (Lindstrom *et al*, 2006). Similarly, high incidences have been found in the Central Africa region (Dubey *et al*., 2005). There is paucity of published work on toxoplasmosis among countries in East Africa. However, a work carried out among three tribes: Baganda, Masai, and Bondei, using serological test showed widespread distribution of *T. gondii* (Tenter *et al*., 2000).

In Nigeria, toxoplasmosis has been reported both in man and some important animals. A work carried out among pregnant women attending Antenatal Clinics at University College Hospital, Ibadan, and St. Mary's Catholic Hospital, Ibadan, showed very high prevalence of *Toxoplasma* antibodies in the sera of both pregnant (75.4%) and postpartum (80.5%) women (Onadeko *et al*., 1996). However, it has been observed that the ELIZA method was not very sensitive in detecting *Toxoplasma* among Nigerian women (Onadeko *et al*., 1992). Reporting further on toxoplasmosis in Nigeria, Onadeka *et al*. (1996) observed that polydactylism, a common congenital abnormality, was traced to reinfection or recrudescence of toxoplasmosis which accounted for high antibody levels. He also observed an association between high prevalence of toxoplasmosis and overcrowding with poor environmental sanitation challenges, including considerable contamination with cat faeces. In the Middle belt region of the country, high prevalence of toxoplasmosis has been reported among pregnant women from Benue State. Among women of the 39-42 age bracket, 71.4% presented with serological evidence of toxoplasmosis (Olusi *et al*., 1996).

In Northern Nigeria, more work have been reported on veterinary toxoplasmosis. High seroprevalence of of toxoplasmosis have been reported among some animals of economic importance, such as sheep (Okoh *et al*., 1984), chicken in Zaria (Aganga, 1985), pet dogs in Zaria (Aganga and Ortese, 1984), and dogs in Maiduguri (Kamani *et al*., 2010). These studies reveal the high preponderance and spread of veterinary toxoplasmosis in Northern Nigeria. They also show that these animals reported as having high prevalence may represent possible animal source of infection to humans in the region (Clementino *et al*., 2010).

V. TRANSMISSION OF TOXOPLASMOSIS

Carnivorous animals are often infected with *Toxoplasma* through ingestion of bradyzoites from tissue cysts in infected prey, as are persons who eat undercooked meat, particularly that of pigs, sheep and goats. Infection can also be through the milk of sheep, goats and cattle, and sometimes through chicken eggs (Eyles, 2001). *Toxoplasma* cysts are less commonly found in poultry and rarely found in beef. Its prevalence in commercial farm animals has decreased significantly with the advent of intensive management practices (Clementino *et al*., 2009). Free range poultry, swine, small ruminants, marsupials and some wild game are more likely to harbour cysts. Tachyzoites are killed relatively easily by pasteurization, and uncommonly survive gastric digestion but any kind of cooking will definitely kill tachyzoites in an egg.

Oocysts are only shed by cats but unsporulated oocyst in fresh feces are not uninfective. Appropriate oxygen, humidity, and temperature are necessary for sporulation to occur. Sporulated oocysts are the most environmentally resistant life stage of the parasite (Halland, 2004). Ingestion of even as few as ten oocysts may infect an intermediate host, while ingestion of 100 or more oocysts can cause a patent infection in a cat, which may shed tons of hundreds of millions of oocysts (Patton, 1993).

In utero transmission of *Toxoplasma* occurs only if primary infection of the mother occurs during pregnancy. Parasitemia then results in placentitis and infection of the fetus. This is more likely to occur in man, sheep and goats, and sometimes in mice, cats and dogs. Under normal circumstances, a female that has been exposed to *Toxoplasma* 4-6 months prior to pregnancy will develop sufficient immunity to protect herself and the fetus for the rest of her life (Vaz *et al*., 2010). However, if the immune response is suppressed by drug therapy or disease such as AIDS in man, both the mother and the fetus may become susceptible to infection again (Tenter *et al*., 2000). The risk of vertical transmission to the fetus increases from the first trimester (10-24%) to the third trimester (60-90%), and the potential of congenital defect is more severe with earlier infections (Patton, 1993).

VI. ECONOMIC IMPORTANCE OF TOXOPLASMOSIS

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, mycocarditis and polymyositis (Jones *et al.*, 2003). 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 (Ouermi *et al*., 2009), reproductive disorders (Montoya and Liesenfeld, 2004), and transmission of Hepatitis B virus (HBV). Children with acute congenital toxoplasmosis often die in the first month of life (Akyar, 2011). It causes tremendous losses of animals too, including valuable livestock. Infection of dairy goats with *T. gondii* is widespread and constitutes a public health concern (Zhao et al., 2011), resulting in significant reproductive losses (Dubey, 2009; Walsh *et al*., 1999).

VII. BEHAVIORAL CHANGES ASSOCIATED WITH *TOXOPLASMA* INFECTION

One of the dramatic characteristics of *T. gondii* is its ability to change the behaviour of its host. It has been reported that infected rats and mice are less fearful of cats, and some of the infected rats seek out cat-urine-marked areas. This effect is advantageous to the parasite, as the setting catalyzes the proliferation of the parasites as the infected rat is eaten by the cat (Beyer *et al*; 1986). The mechanism for this change is not completely understood, but It could also be a result of subtle effects on the nervous system (Obendorf *et al.,* 1996; Berdoy *et al*., 2000). However, there is evidence that toxoplasmosis infection raises dopamine levels and concentrates in the amygdala in infected mice. (Henriquez *et al*., 2009). Perhaps, this could be as a result of low-grade encephalitis marked by presence of cysts in the brain, which induces the production of neurotransmitter (dopamine), which acts similarly to dopamine reuptake inhibitor type antidepressants and stimulants (Laing *et al*., 1996). This observation of behavioral change among rats and mice has led to speculation that *Toxoplasma* may have similar effects in man, even in the latent phase that had previously been considered asymptomatic (Tenter *et al*., 2000). Correlations have been found between latent *Toxoplasma* infections and various characteristics such as: decreased novelty seeking behavior, slower reactions, as well as lower rule-consciousness and greater jealousy (in men) Greater warmth, conscientiousness and moralistic behavior (in women).

VIII. DIAGNOSIS OF TOXOPLASMOSIS

Serologic tests are the primary means ofdiagnosis. Immunoglobulin (Ig) G-specific antibodies achieve a peak concentration during about one to two months after infection and remain positive indefinitely.For patients with seroconversion or a fourfold increase in IgGantibody titer, specific IgM antibody determinations shouldbe performed as the presence of *T gondii*-specific IgM antibodies may indicateacute or recent infection. Enzyme immunoassay tests are themore sensitive assays for IgM, detection, and this can be achieved 2 weeks after infection.Peak concentrations of IgM antibody ia achieved in one month, but decreases thereafter to an undetectable level within 6 to 9 months. This undetectable level can persist for as long as 2 years, and during this period, it confounds the differentiationof acute and remote infections. Tests to detect IgA and IgE antibodies,which decrease to undetectable concentrations sooner than IgMantibodies, are useful for the diagnosis of congenital infectionsand infections in other patients, such as pregnant women, forwhom more precise information about the duration of infectionis needed.

Detection of *T gondii* DNA inamniotic fluid by polymerase chain reaction assay has been shown to be a safe and accurate method ofdiagnosis. Serial fetal ultrasonographic examinations shouldbe performed in cases of suspected congenital infection to detectany increase in size of the lateral ventricles of the centralnervous system or other signs of fetal infection.Quantitative 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). (Akyar, 2011). If the diagnosis for an infant is unclear at the time of delivery,evaluation of the infant should include ophthalmologic, auditory,and neurologic examinations; lumbar puncture; and computed tomographyof the head. Attempt should be made to isolate *T gondii* fromthe placenta, umbilical cord, or blood specimen from the infantby mouse inoculation.

Patients with HIV infection who are infectedlatently with *T gondii* have variable titers of IgG antibodyto *T gondii* but rarely have IgM antibody. Although seroconversionand fourfold increases in IgG antibody titers may occur, theability 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 basisof the presence of characteristic clinical and radiographicfindings. If the infection does not respond to an empiric trialof anti-*T gondii* therapy, demonstration of *T gondii* organisms,antigen, or DNA in biopsied tissue, blood, or cerebrospinalfluid may be necessary to confirm the diagnosis.

IX. PREVENTION AND CONTROL OF TOXOPLASMOSIS

Improper handling of cat litter and not necessarily ownership of cat is accepted as a risk factor of toxoplasmosis (Walker *et al*., 2008). This definitely determines what measures would be effective in preventing or controlling the spread of toxoplasmosis. 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 –12ºC for at least 24 hours to kill *Toxoplasma* tissue cysts, but it must be noted that sporulated oocysts can survive at –20ºC for up to 28 days. (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 (Tenter *et al*., 2000). (vii) Unwashed fruits or vegetables as well as unpasteurised milk should not be eaten. (viii) One must ensure that all avenues that could bring one in contact with cat faeces either directly or indirectly are blocked, while the intermediate host population must be properly checked.

X. TREATMENT OF TOXOPLASMOSIS

Traditional drug therapy for clinical toxoplasmosis consists of a combination of pyrimethamine and sulfonamides. This combination can cause dose-related bone marrow suppression with resultant anemia, leucopenia, thrombocytopenia, and reversible acute renal failure. Leucovorin (folinic acid) could be added to the combination to prevent bone marrow suppression and to reduce the severity of congenital infection and increase the proportion of infants asymptomatic at birth (Daffos *et al*; 1988). 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 (Tenter *et al*., 2000).

XI. CONCLUSION

The preponderance of toxoplasmosis around the world is predicated on the anthropogenic activities favourable to the transmission of the parasite to man and animals. The paucity of research in developing countries is perhaps, caused by sophisticated and expensive methods for verification of infection, since the widespread occurrence of the infection in the area is not in doubt. Furthermore, health authorities must make a policy to monitor the clinical and laboratory parameters of pregnant women as regards their toxoplasmosis status. It is essential that both clinical and laboratory parameters be used in establishment of toxoplasmosis status to ensure better accuracy (Boyer *et al*. 2005; Mazzola *et al*. 2007; Mioranza *et al*. 2008). The war against toxoplasmosis is one we cannot afford to lose. Urgent steps are needed to launch enlightenment campaigns on the disease epidemiology to mitigate the tide of rapid spread of toxoplasmosis.

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