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REVIEW ARTICLE

Toxoplasmosis: A Food Borne Parasitic Zoonosis

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Abstract

Toxoplasmosis is one of the important zoonosis caused by *Toxoplasma gondii*, an obligate intracellular protozoan parasite with worldwide distribution. Felines are definitive host and only animal species to shed the infectious stage in their faeces and the intermediate hosts are vertebrates like amphibians, fish, reptiles and other warm blooded animals including man. Infection occurs by eating infected meat, particularly swine products, by ingesting water, soil, or food that has come into contact with infected animal's faecal matter, by transplacental route from infected mother to fetus during pregnancy, by organ transplantation and blood transfusion. Parasite can infect any cell of the body and hence clinical sign/symptoms are dependent on the organ affected. Main forms of disease in human are congenital and ocular. Persons at risk are pregnant women, cat owners, veterinarians, abattoir workers, butchers, children, cooks. Diagnosis is by microscopic examination, serological test, computed tomography and PCR based technique. Treatment with Pyrimethamine and Sulfadiazine is most effective. Control and prevention can be conducted by proper hygiene, proper cooking of food and mass education and awareness to persons at high risk.

Keywords: Transplacental, Zoonosis, Faeco-oral route, Computed tomography, PCR assay.

1. Introduction

Toxoplasmosis is a parasitic disease caused by the protozoan *Toxoplasma gondii* (Ryan and Ray, 2004). The word *Toxoplasma* is derived from toxan means Arch and plastos means shaped. They are crescent shaped obligate intracellular protozoan parasite enclosed in a parasitic membrane to form a cyst measuring 10-100 µ in size, with worldwide distribution under the genus *Toxoplasma* and the phylum Apicomplexa (Soulsby, 1982). The definitive host of *T. gondii* is the cat and other felids and the intermediate hosts are vertebrates like amphibians, fish, reptiles and other warm blooded animals including man (Hubalek, 2003). Infections are most common in cats, sheep, goats and swine (Chessa *et al.*, 2014). Lower infection rates are seen in dogs, horses and cattle seem to be resistant to infection. Cats are the only species to shed the infectious stage in their feces (Slavin *et al*., 1994). The parasite shows four infectious stages for both intermediate and definitive hosts i.e. oocyst, tachyzoites, bradyzoites and tissue cysts (Soulsby, 1982).

2. History

The organism was first described in Tunis in 1908 by Charles Nicolle and Louis Manceaux within the blood, spleen and liver of the Gundi (*Ctenodactylus gundi*, A North African rodent) (Weiss and Dubey, 2009), but was not identified as an agent of infectious disease until 1932. *T. gondii* was first identified as a human pathogen in 1939 (Ferguson, 2009). The parasite is known to cause congenital disease and abortion both in human and livestock. (Dubey and Beattie, 1988). One third of humanity has been exposed to this parasite. It became more widely recorded as a cause of morbidity in immunodeficient patients, including AIDS patients in 1983. *T. gondii* continues to be an important disease in the modern world, especially in pregnant women and immunocompromised patients. Although asymptomatic in immunocompetent adults, and can cause severe disease manifestation and even death in immunocompromised patients (Singh, 2003) .

3. Public Health Significance and Prevalence

Toxoplasmosis continues to be a significant public health problem. Recent publications have linked suicide and schizophrenia to *Toxoplasma* infection. *T gondii*–infected mothers had a relative risk of self-directed violence compared with noninfected mothers, and the risk seemed to increase with increasing IgG antibody level (Torrey *et al*., 2012).

Geographic area, age, and socioeconomic factors influence the prevalence of the disease. Regional prevalence varied with different climates, highest in tropical regions and lowest in cold regions of the world. Prevalence increases upto age of 20 years after which it remained steady. Among female patients, the prevalence of *Toxoplasma* antibodies was higher among female blood donors than among pregnant women (Luyasu, 1997). There is a progressive increase in the prevalence of positive antibodies to *T. gondii* with age (Choi *et al.*, 1997).

4. Transmission

In Animals, carnivores usually infected by the ingestion of bradyzoites encysted in another intermediate host. Herbivores get infected by ingesting sporulated oocysts shed by infected cats through fecal-oral route. Transmission can also occur through placenta as trachyzoites multiplication occurs within the placenta, through body excretion like milk and saliva and through sexual intercourse (Singh *et al.*, 1997).

In Human, various means of transmission include ingestion of uncooked infected meat containing cysts (Choi *et al.*, 1997), ingestion of sporulated oocysts from contaminated food and water with cat faeces or hands (Jones and Dubey, 2012), organ transplantation, blood transfusion, transplacental transmission (Jones *et al.*, 2001) and accidental inoculation of tachyzoites in the skin (Parija, 1996) through contaminated food, water, or dust or during cleaning of cat litter. The parasites are shed in the faeces of infected cats and become infectious after 1 to 5 days. Children can get toxoplasmosis by playing in sandboxes that contain cat faeces (Luyasu, 1997). Poor hygiene observed in India during handling of meat from slaughter house to kitchen can be a source of *T. gondii* infection.

5. Signs and Symptoms

In animals, early symptoms include lethargy, persistent fever, anorexia, hepatitis. Other CNS signs are convulsion, restlessness, head pressing, teeth grinding, personality changes, hyperesthesia, atypical vocalization, incoordination, trembling, opisthotonus, paralysis and depressed reflexes. Abortion, metritis and birth of premature can also occur (Dubey, 1988). Ocular signs are retinitis, irregular reddish, dark, pale retinal foci, congestion of vessels, hemorrhages and exudates cloud in vitreous humor. Chronic low grade infections may cause glaucoma, corneal opacity, and panopthalmitis (Dubey, 1988).

In Humans, **v**arious forms of toxoplasmosis is reported which includes:

Congenital toxoplasmosis in which transmission occur either by active primary infection during pregnancy or previously exposed mother before pregnancy with compromised immune. The risk of baby's infection depends partly upon the timing of the mother's infection which is highest in 3rd trimester**.** When the mother is infected between 10-24 weeks of gestation period the risk of severe problems in newborn is 5-6 % (Jeffrey, 2003). It is of two types.

Asymptomatic congenital toxoplasmosis in which up to 90% of the infected babies appear normal at birth but 80-90% will develop sight threatening eye infection months to years after birth and about 10% will develop hearing loss and/or learning disabilities.

Symptomatic congenital toxoplasmosis occurs in one out of 10 infected babies which has a severe *Toxoplasma* infection that is evident at birth. Some die within a few days of birth and those who survive sometimes suffer from mental retardation, severely impaired eyesight, retinochoroiditis, microcephalus/hydrocephalus, intra cerebral calcifications, convulsions and psychomotor retardation, abnormal CSF, jaundice and hepatosplenomegaly (Sever, 1988).

Ocular toxoplasmosis is often asymptomatic in immunocompetent individuals, but ocular lesions are seen in infected patients up to 20%. It is acquired as the late sequel of asymptomatic congenital Toxoplasmosis and reactivation of earlier infection. The signs are blurred vision, ocular pain, photophobia, necrotizing retinitis and uveitis retinochoroiditis. (Vallochi *et al.*, 2002).

Toxoplasmosis in AIDS patients can be due to recently acquired infection or more commonly due to reactivation of the latent infection. Approximately 3-10% of all AIDS patients die of Toxoplasmosis. Apart from major ocular signs, encephalitis, pneumonia, urinary tract infection, peritonitis, retinochoroiditis (Hunter and Remington, 1994).

Toxoplasmic encephalitis include signs like headache, confusion, ataxia, hemiparesis, retinochoroiditis, calcifying lesions, degeneration of ventricular walls, multicystic encephalopathy and perivascular edema (Alappat *et al.*, 2000).

Toxoplasma pneumonia recognized in the patients with AIDS who are not receiving appropriate anti-HIV drug or primary prophylaxis for toxoplasmosis. Signs are cough, rales, shortness of

breath, fever, diffuse infiltrate of both lungs, hepato-splenomegaly, lymphadenopathy (Dubey, 1988).

6. Diagnosis

It depends on history and clinical signs of patients. Diagnosis can be made by direct observation of parasite in the tissue or feces through microscope, by serological tests like antibody detection tests like Sabin–Feldman dye test, Indirect fluorescent antibody test, Indirect haemagglutination test, Latex agglutination test, Direct agglutination test and Enzyme linked immunosorbent assay (ELISA). Antibodies can also be detected by numerous serological tests using commercially available kits to detect *T. gondii* specific IgG, IgM, IgA or IgE antibodies (Singh, 2003; Hill and Dubey, 2002)**).** Isolation of parasite by intra-peritoneal inoculation into mice or tissue culture. PCR technique can be helpful for detecting congenital toxoplasmosis (sterkers *et al.*, 2011). It has allowed detection of *T. gondii* DNA in brain tissue, cerebrospinal fluid, vitreous and aqueous fluid, bronchoalveolar lavage fluid, urine, amniotic fluid and peripheral blood (Deboer *et al*., 1996).

7. Treatment

Oocyst shedding in infected cats can be reduced with combination of Pyrimethamine and Sulfadiazine (Frankel, 1973). Clindamycin has been shown to be effective treating ocular toxoplasmosis in humans and animals as a therapeutic alternative to Pyrimethamine (Dubey, 1988). Clindamycin has shown activity against acute and chronic toxoplasmosis (Dubey, 1988; Djurkovic *et al*., 2002). Congenital or occular toxoplasmosis can be treated with Pyrimethamine or Sulfa-diazine. Corticosteroids for vision-threatening lesions with tapering dosage are good. For women who acquire the infection during pregnancy, Spiramycin, a macrolide antibiotic decreases the incidence of congenital toxoplasmosis when administered early in the course of the disease (Boyer, 2005).

8. Prevention and Control

No vaccine is available for toxoplasmosis in humans. *T. gondii* infection is an important cause of abortion and mortality in sheep and goats throughout the world. A live vaccine using a nonpersistent strain of *T. gondii* is available in New Zealand, the United Kingdom, and Europe to prevent abortion caused by *T. gondii* infection in sheep. A live vaccine using a mutant strain of *T. gondii* (T-263) is being developed in the United States to reduce oocyst shedding by cats (Reddy, 2006). Killing of tachyzoites through inactivation at pH<4 or Freezing at -12°C, cooking to an internal temperature of 70°C, or using gamma radiation (5 kGy) kill tissue cysts in meat is another way. Several agents have been shown to be effective against the tachyzoite of *T. gondii*, but there is no chemotherapeutic agent that is effective against the encysted form (bradyzoite) of the parasite.

Health education and awareness to persons at high risk like pregnant women, cat owners, veterinarians, abattoir workers, children, cooks, butchers is important. Pregnant women should avoid nourishing the cats as well as avoid contact with cats, raw meat and raw milk or unpasteurized should not be consumed (Narladkar *et al.*, 2006).

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