SEROPREVALENCE OF *TOXOPLASMA* ANTIBODIES IN DOMESTIC ANIMALS- AN INDICATOR OF *TOXOPLASMA GONDII* IN THE ENVIRONMENT AND HUMAN

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**INTRODUCTION**

Toxoplasmosis, caused by *Toxoplasma gondii*, is an obligate intracellular parasite widespread in human beings and many other warm blooded animals. The disease, toxoplasmosis is generally asymptomatic except in immunocompromised adults and congenitally infected children.

**ETIOLOGY AND LIFE CYCLE OF TOXOPLASMOSIS**

Cats (both domestic and wild) are the only definitive hosts (Figure 1). Cats excrete *T. gondii* oocysts (~ 10 µm in diameter) in their faeces. Excreted oocysts are nonsporulated and, therefore, are non-infective. After defaecation, sporulation (development of infective sporozoites inside the oocysts) requires 1 to 5 days and can survive up to 18 months and is remarkably resistant to most disinfectants. Human beings and other warm-blooded animals become infected primarily by ingesting food or water contaminated with oocysts or by ingesting infective animal tissues (meat) that contain cysts of *T. gondii*.

![Figure 1. Life cycle of Toxoplasma gondii](Source: Dubey, J.P. (1994). JAVMA, 205: 1593-1598)

After ingestion of faecal-contaminated food/water, the oocysts ruptures in the intestine and releases 8 sporozoites. Sporozoites multiply in the intestinal cells and in associated lymph nodes, and tachyzoites (rapidly multiplying forms) are formed. Tachyzoites are dispersed to the rest of the body via blood and lymph, eventually encysting in the brain, skeletal and cardiac muscles and liver. Encysted *T. gondii* organisms are called bradyzoites or cystozoites (slowly multiplying forms). Cysts are microscopic and survive in tissues for the duration of the life of the hosts. Tachyzoites are found
during the acute phase, whereas encysted bradyzoites (tissue cysts) are often found predominantly in
the latent, chronic phase. It has been hypothesized that tissue cysts rupture occasionally and the
released bradyzoites are killed in immunocompetent hosts. However, in immunosuppressed hosts such
as AIDS patients, bradyzoites released from tissue cysts may multiply locally and spread to other
organs.

**Transmission of toxoplasmosis**

The three means by which it is mainly spread are ingestion of food or water contaminated
faeces with infective oocysts of cats and other wild Felids, ingestion of infective tissues cysts and
transplacental transmission by tachyzoites. Oocysts-induced are generally more severe than tissue
cyst-induced infections.

One usually catches the diseases by consuming food contaminated with litter/soils of
domestic cat accidentally by *Toxoplasma* oocysts. In areas like the Kumaon however (which is home
to the big cats rather than to the little domestic ones), the source of infection is probably quite
different. Studies suggest that the ingestion of poorly cooked mutton which harbour a developing
phase of *Toxoplasma* could be the mode of transmission. Toxoplasmosis has developed in human
beings that drink unprocessed goats milk. Therefore, goats milk should be boiled/pasteurised before
consumption by human beings particularly infants who are more susceptible to toxoplasmosis than
adults.

Nothing is known of the extent of *T. gondii* contamination of soils in India. Soil is a
continuous source of infection for animals and humans. From a public point of view, determination of
*T. gondii* infection in roaming chicken, and pigs (due to their feeding habits) would be useful in
understanding the epidemiology and should be a good indicator of the level of soil contamination with
oocysts shed by definitive hosts. Although, meat is generally cooked well before human consumption,
handling of infection meat and poor hygiene could be a source of infection. Ingestion of infected
tissue by cats can lead to oocysts shedding and spread of *T. gondii* in the environment.

**SEROPREVALENCE OF TOXOPLASMA GONDII IN HIMALAYAN REGION**

Only limited studies have been undertaken in Himalayan region. *Toxoplasma* antibodies were
detected in 23% of sheep (n=88) and 12.5% of goats (n=8) from Palampur (H.P.) region with titre
(reciprocal) ranging from 25 to 5000 by a Modified Agglutination Test (MAT). In Kumaon, 81.2% of
Pashmina goats (n=48) and 90% of the local goats (n=39) were positive (Figure 2). A nation wide
survey for *Toxoplasma* antibodies in human indicates an overall prevalence rate of ~20% among
Indians. But in kumaon, over 77% of the hill folk test positive for *Toxoplasma* antibodies. Singh and
Nautiyal (1991) reported a seroprevalence of 77% in female and 37% in males from Kumaon region. The high prevalence rate of T. gondii infection in hilly region of the Himalayan might play a role in the local epidemiology of toxoplasmosis in humans. Families often keep domestic cats as pets. In addition, feral cats (Felis catus) including leopard (F. leo) are common in the Himalayan belt. Ingestion of infected tissues by cats could lead to shedding of oocysts in the environment. Toxoplasma gondii oocysts are resistant to environmental influences and can survive freezing and drying. However, unlike oocysts, tissue cysts in meat are relatively susceptible to environmental influences. Tissue cysts are killed at 70°C for 10 min or 60°C for 30 min. Since meat is usually cooked well before consumption, transmission of T. gondii in humans in India via infected meat appears to be minimal. Therefore, transmission through the oocysts shed by cats appears to be the main source of T. gondii infection in man and animals.

CLINICAL SIGNS OF TOXOPLASMOSIS

*Toxoplasma gondii* parasitize most hosts without overt clinical disease. However, in human, it usually causes a febrile illness associated with some enlargement of lymph nodes, etc., while in pregnant women particularly; it poses a danger to the foetus by way of abortion, foetal death, foetal abnormalities, etc. Infection by either oocysts or tissue cysts during pregnancy can result in transplacental infection of the foetus with tachyzoites. Infants who survive and look apparently normal at birth can go on to develop blindness and mental retardation in later life.

Another area where it rears its ugly head is in the course of HIV infection. In AIDS patients, toxoplasmosis affects the nervous system. Encephalitis is reported to be the predominant clinical manifestations of toxoplasmosis in AIDS patients and is believed to be due to reactivation of latent infection. Diagnosis of toxoplasmosis in AIDS patient is difficult because their immunity is so depressed that they do not form antibodies to *Toxoplasma*. Only way to prevent and AIDS epidemic would be in the prompt identification of HIV positive cases and preventing its spread. Health education and prevention is the name of the game.

**Diagnosis and treatment of toxoplasmosis**

Diagnosis of *Toxoplasma* is also aided by serologic tests and by bioassay or xenodiagnosis in mice. In fact ELISA, the test for toxoplasmosis is popular and is usually diagnosed accidentally while handling infertility cases, as it is one of the causes of abortions. *Toxoplasma* screening should be routine procedure in all antenatal clinics since tests are not prohibitively expensive. Besides, health education should be a part for life saving for the unborn one.

**Control of toxoplasmosis**

Key factors in tackling toxoplasmosis lies in protecting pregnant women by preventing and promptly treating *Toxoplasma* infection. Human beings are immune to subsequent infection. No vaccine is available yet. Those whose tests reveal current infection, treatment are of paramount importance; treatment can prevent the birth of a deformed or retarded child.

Care should be taken to wash hands after gardening, handling meat, knifes, utensils that come in contact with uncooked meat. Meat should be well cooked. Pregnant women should avoid contact with cat faeces, soil and uncooked meat. Vegetables should be washed thoroughly before eating because of possible contamination with soils that contain cat faeces.

**REFERENCE**
