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Popular Article

TOXOPLASMOSIS: An Overview

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Abstract

This article presents a review of the zoonotic disease, Toxoplasmosis which is caused by a protozoan parasite, *Toxoplasma gondii*. The article addresses major components involved in the zoonotic spread of the disease from animals to humans, which include the aetiology of the parasite, its transmission, and the epidemiology involved along with a wider health perspective to elicit its significance on a global scale. Along with the aforementioned factors, the pathological, diagnostic, preventive and post-infection management of the disease is also included.

Keywords: *Toxoplasma gondi*, Clinical toxoplasmosis, Tachyzoite, Bradyzoite

Introduction

Toxoplasma is a zoonotic protozoan belonging to phylum apicomplexa affecting both cats and humans. The Toxoplasma genus has only one pathogenic species known as *Toxoplasma gondii*. It is a widespread disease that is most prevalent in areas with dense populations of stray cats. Around 30 million humans are thought to have a Toxoplasmosis-related infection, and 20%-60% of cats are diagnosed with Toxoplasmosis (Wingertzahn *et al*, 2022). *Toxoplasma gondii* has been thought of as relevant to public health primarily within the context of congenital toxoplasmosis or postnatally acquired disease in immune-compromised patients. Latent *T. gondii* infection has been increasingly associated with a wide variety of neuropsychiatric disorders and ocular disorders (Milne *et al*, 2020).

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Transmission

Toxoplasmosis is not passed from one human to another, except when it is passed trans-placentally or through blood transfusion. People typically acquire the infection in 3 ways, they are stated as follows:

- Foodborne
- Animal to human(zoonotic)
- Mother to child(congenital)

1. **Foodborne**

The tissue form of the parasite (a microscopic cyst consisting of bradyzoites) can be transmitted to humans by food. People become infected by:

- Eating undercooked, contaminated meat (especially pork, lamb, and venison) or shellfish (like oysters, clams, and mussels);
- Accidentally ingesting undercooked, contaminated meat or shellfish after handling it and not washing hands thoroughly (Toxoplasma cannot be absorbed through intact skin); and
- Eating food that was contaminated by knives, utensils, cutting boards or other foods that had contact with raw, contaminated meat or shellfish.
- Drinking unpasteurized goat's milk (tachyzoites).

2. **Zoonotic transmission**

Cats play a vital role in the spread of toxoplasmosis. They become infected by eating infected rodents, birds, or other small animals. The parasite is passed into the cat's faeces in an oocyst form, which is microscopic. Kittens and cats can shed millions of oocysts in their faeces for as long as three weeks after infection. Previously infected mature cats are less likely to shed Toxoplasma. Toxoplasma-infected cat that is shedding the parasite in its faeces contaminates the litter box. If the cat is allowed outside, it can contaminate the soil or water in the environment. People can be infected by:

- Accidental ingestion of oocysts after cleaning a cat's litter box when the cat has shed Toxoplasma in its faeces,
- Accidental ingestion of oocysts after touching or ingesting anything that has come into contact with a cat's faeces that contain Toxoplasma
- Accidental ingestion of oocysts in contaminated soil (e.g., not washing hands after gardening or eating unwashed fruits or vegetables from a garden)
- Drinking water contaminated with the Toxoplasma parasite.

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3. Congenital transmission

A newly infected woman with Toxoplasma during or just before pregnancy can pass the infection to her unborn child (congenital infection). The woman may not have symptoms, but there can be severe consequences for the unborn child, such as diseases of the nervous system and eyes (Centers for Disease Control and Prevention, 2023).

Epidemiology

In the United States, it is estimated that 11% of the population 6 years and older have been infected with Toxoplasma. In various places throughout the world, it has been shown that more than 60% of some populations have been infected with Toxoplasma. Infection is often highest in areas of the world that have hot, humid climates and lower altitudes because the oocysts survive better in these types of environments (Centers for Disease Control and Prevention, 2023).

According to the review published in Ecohealth (Aguirre *et al,* 2019), an estimated 8–22% of people in the USA are infected, and a similar prevalence exists in the UK (Dubey 2002; Dubey and Jones 2008; Jones *et al*. 2001, 2003, 2007). In Central America, South America, and continental Europe, estimates of infection range from 30 to 90% (Dubey and Jones 2008; Dubey 2010; Minbaeva *et al*. 2013; Wilking *et al*. 2016).

These infections have significant consequences affecting mortality and quality of life. In the USA, where over a million people are infected each year and approximately 2839 people develop symptomatic ocular disease annually, the cost of illness has been estimated to be nearly $3 billion and an 11,000 quality-adjusted life-year loss annually (Jones and Holland 2010; Batz *et al*. 2012; Hoffmann *et al*. 2012). Mead *et al*. (1999) suggested that T. gondii is one of three pathogens (along with Salmonella and Listeria) that account for > 75% of all deaths due to foodborne disease in the USA. Scallan *et al.* (2011) estimated that Toxoplasma caused 8% of hospitalizations and 24% of deaths in the USA resulting from foodborne illnesses (Aguirre *et al,* 2019).

In a study conducted by Singh *et al*, testing the seroprevalence and incidence of *Toxoplasma gondii* in Indian women of childbearing age has remained a contentious issue. Different laboratories have used different patient recruitment criteria, methods and variable results, making these data unreliable. There is no published pan-India seroprevalence study. Hence, a seroprevalence study was undertaken comprising 1464 women of reproductive age representing four distinct geographical regions of India. This resulted in an estimated prevalence of 22.4%; the highest prevalence being in South India (37.3%) followed by East India (21.2%) and North India (19.7%). West Indian women

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had the lowest seroprevalence (8.8%). This difference was highly significant. In this analysis, the possible risk factors of infection in these women were determined. These included lower socioeconomic status, residing in mud-plastered houses, consumption of raw salad, drinking untreated water, owning pets and advanced age. Overall, the incidence rate of toxoplasmosis was 1.43%. Extrapolating the data, it estimated that between 56,737 and 176,882 children a year may be born in India with a possible risk of congenital toxoplasmosis, which can manifest itself in utero or several years after birth (PubMed Central 2014).

Pathophysiology & life cycle

Infection of cats can occur in three ways, via the tachyzoite, the bradyzoite or the sporozoite. Upon ingestion of tachyzoites, they enter the host cells by active penetration of the cell membrane. After entering the host cell, the tachyzoite becomes ovoid and becomes surrounded by a parasitophorous vacuole, which is protected from host immune defences (Lindsay *et al*, 2004). Within the cell, tachyzoites multiply asexually by repeated endodygeny, a specialized form of reproduction in which two progeny form within the parent parasite. These tachyzoites proceed to infect other cells upon lysis of the host cell. Tachyzoites are capable of infecting numerous cell types inducing tissue necrosis, which is seen as acute infection within the host. After a few divisions, tachyzoites encyst in cells and form bradyzoites. This cycle known as the extraintestinal cycle, is the only known development of T. gondii that occurs in its non-feline hosts.

Bradyzoites are responsible for chronic infection of the host. Bradyzoites may be entered into the body via tissue cysts found in the meat of intermediate hosts. The cyst wall is degraded by the action of digestive enzymes releasing bradyzoites. Bradyzoites resist peptic and tryptic digestion (Soulsby, 2022). Bradyzoites enter the intestinal epithelial cells and initiate the formation of schizonts. Merozoites released from schizonts form male and female gametes, which fuse to form an oocyst. The coccidian phase of this entero-epithelial cycle is found only in the definitive feline host, and hence cats are known to produce T. gondii oocysts. These oocysts become infective in 1-5 days after excretion when sporulation occurs. Ingestion of such oocysts can also lead to infection.

As the entero-epithelial cycle progresses, bradyzoites or sporozoites acquired from tissue cysts and oocysts respectively, invade the intestinal tissues and transform into tachyzoites spreading in the body via blood or lymph. Tachyzoites transform into bradyzoites and form tissue cysts in the central nervous system (CNS), muscles, and visceral organs, and probably persist for the life of the host. Parasitemia during pregnancy can cause placentitis followed by the spread of tachyzoites to the foetus (Dubey *et al*, 2009).

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Clinical manifestations

Clinical toxoplasmosis is the most severe in trans placentally infected kittens. This can involve inflammation of the lungs, liver and CNS. This presents clinically as ascites, lethargy and dyspnoea. Encephalitic kittens may sleep most of the time or cry continuously. Most adult cats are asymptomatic and the disease precipitates in immune-compromised individuals infected with Feline Leukemia Virus or Feline Immunodeficiency Virus. Symptoms involve fever, lethargy and anorexia with pneumonia and jaundice. Ocular toxoplasmosis involves uveitis, retinitis, anterior or posterior iridocyclitis or chorioretinitis. Neurological disease was frequent in cats and involved infection of the brain, spinal cord and nerves causing manifesting as a lack of coordination, heightened sensitivity to touch, personality changes, circling, head pressing, ear twitching, difficulty chewing and swallowing food, seizures, and loss of control over urination and defecation (Calero-Berna *et al*, 2019; Cornell Feline Health Center 2018 and Dubey *et al*, 2009).

Diagnosis

Diagnosis is based on history and clinical signs and can be confirmed by various methods like serology, cytology, faecal examination, radiology or blood biochemistry. Traditionally detection of oocysts in faeces of suspected cats is done by direct wet mount method. Diagnosis based on faecal examination is not as accurate because cats excrete oocysts only for 1-2 weeks after first exposure and are not clinically ill during the process of shedding (Dubey *et al*, 2009). Elevated IgG antibody levels against T. gondii in a healthy cat indicate a prior infection, suggesting immunity and a low probability of oocyst shedding; whereas high IgM antibody levels imply an active infection (Cornell Feline Health Center 2018). Liver enzymes like Alanine Aminotransferase(ALT) and Aspartate Aminotransferase(AST) show increased activity attributed to hepatic infection in Toxoplasmosis (Lobetti *et al*, 2012).

Detection of tachyzoites in tissue samples via fine needle aspiration of lymph nodes, in the CSF and trans-tracheal or bronchoalveolar fluid provides definitive confirmation of the diagnosis of the disease (Hartmann *et al*, 2013). PCR of CSF, Aqueous humour or bronchoalveolar lavage can also be used for diagnosis. Radiographic findings show a diffuse interstitial to alveolar pattern with a mottled lobar distribution. Ocular Fundic Examination should be routine practice for febrile cats suspected with ocular toxoplasmosis (Calero-Berna *et al*, 2019).

Management

Clindamycin is the drug of choice, administered PO every 12 hours for 3–4 weeks at 10–12.5 mg/kg in dogs and 25–50 mg/kg in cats (MSD Vet Manual, 2021). Cats with systemic disease and

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uveitis should be treated with clindamycin in combination with topical, oral or parenteral glucocorticoids, to avoid secondary glaucoma and lens luxation [Hartmann *et al*, 2013]. Prednisolone acetate (1% solution) applied topically to the eye three to four times daily is generally sufficient.

Prevention

Cats should be fed commercially prepared dry or canned cat food to avoid the potential ingestion of tissue cysts and subsequent infection that may occur when feeding undercooked or raw meat. They should be prevented from hunting and eating potential intermediate hosts or mechanical vectors, such as cockroaches, earthworms, and rodents (Dubey *et al*, 2009). Cat litter boxes must be emptied daily as it takes 1-5 days for the oocysts to sporulate. Cleaning of litter boxes with hot water and soap is recommended. Common household products such as detergents, antimicrobial soaps, and bleach are not effective at killing oocysts, and their use for this purpose is not recommended (Wehbe *et al*, 2021). Gloves should be used during such activities and washing of hands with soap and water is an essential hygiene measure.

Pregnant women should avoid cleaning cat litter boxes. Vegetables obtained from gardens must be washed thoroughly as they may have been contaminated with cat faeces. Pregnant women should refrain from consuming raw or undercooked meat due to the potential presence of tissue cysts. These are killed by heating the meat throughout to 67°C (152.6°F) or by cooling to −13°C (8.6°F) (MSD Vet Manual, 2021). Humans and susceptible animal hosts can be exposed to *Toxoplasma gondii* oocysts in the environment through drinking water contaminated with felid faeces and thus filtered or bottled water should be consumed if living or travelling in an endemic region (Shapiro *et al*, 2019). Finally, direct contact with cats is unlikely to result in transmission of toxoplasmosis because most cats do not leave faeces on their fur for the 1 to 5 days required for oocyst sporulation (Kramer *et al*, 2009).

One health perspective on toxoplasmosis

A review published by Aguirre *et al*, via ecohealth suggests that, as a global strategy, One Health recognizes the interconnectedness of the health of people, animals, plants, and the environment from the local to the global levels and employs a holistic approach encouraging and expanding transdisciplinary collaborations, integrative research, capacity building, clinical practice, policy, and communication among many stakeholders. This approach can overcome bureaucratic boundaries and represents an opportunity for new partnerships focused on solutions for humans, animals, plants, and the environment (Zinsstag 2012; Rubin *et al*. 2014; Aguirre *et al*. 2016). Toxoplasmosis qualifies as a One Health disease because it significantly affects the health of human, domestic animals, wildlife,

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and ecosystems, and is perceived as a threat by those who rely on animal resources (Crozier and Schulte-Hostedde 2014; Jenkins *et al*. 2015).

The above-mentioned review article also states that toxoplasmosis is the second leading cause of death among foodborne illnesses in the USA (Scallan *et al*. 2011; Gao *et al*. 2016). In humans, symptoms, or lack thereof, at the time of infection do not predict disease manifestation later in life. The disease may be either acute or chronic and can cause active infection at any age (Boyer *et al*. 2011; Delair *et al.* 2011). Postnatal T. gondii infection may appear to be asymptomatic or cause fever and lymphadenopathy affect any organ, especially the eyes (Montoya and Remington 1995; Delair *et al.* 2011; Undseth *et al.* 2014), and cause seizures (McAuley *et al*. 1994). Virulence varies by strain and susceptibility based on an individual's genetic traits (Ngo *et al.* 2017). Genotypes in French Guiana, for example, cause significant damage and even death in adults who are not known to be immunocompromised (Carme *et al*. 2009). In the USA, an estimated 1.1 million people are infected with T. gondii each year, and approximately 10.4% of the population demonstrate seroprevalence linked to past exposure (Jones and Holland 2010; Jones *et al*. 2018).

An epidemiological survey conducted in India by Rashmi Thakur *et al*, it was found that in India, 56,737 and 176,882 children are born with a possible risk of congenital toxoplasmosis every year. A seroprevalence of 22% in pregnant women was reported. The first nationwide survey on T. gondii infection in humans in India conducted in 2005 indicated a seroprevalence of 24% (5611/ 23094). In North India, seroprevalence was highest in Chandigarh (20%) followed by Uttar Pradesh (19%); prevalence was 13% in people of Punjab (Thakur *et al*, 2019).

Conclusion

Toxoplasmosis is a substantial menace to the humankind. The clinical manifestations of this disease have major ramifications on human beings, some of which involve posterior retinitis, uveitis and lymphadenopathy in adult immune-compromised individuals. Trans-placentally transmitted infections most likely result in abortions and stillbirths, and cause severe birth defects in live-born infants, such as microencephaly, hydrocephalus, intracerebral calcifications and retinochoroiditis. Cats are the principal vectors of this life-threatening parasite; stray cats generally acquire the etiological agent via ingestion of infected rodents. Regular washing of hands and sanitizing as and when possible is pivotal in avoiding the infection and preventing its transmission. Foodborne spread of the disease can be controlled by implementing more stringent hygiene measures in meat factories as well as thoroughly cooking any kind of meat before consumption. Toxoplasmosis is an entirely preventable disease, spreading awareness to the most susceptible populations and following all norms

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of hygiene and cleanliness meticulously will most certainly result in minimal transmission and maximal prevention of this disease.

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