Toxoplasmosis
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Etiological agent

Toxoplasma gondii is the protozoan parasite that causes Toxoplasmosis.

Transmission

Infection by T. gondii usually occurs through one of the three following modes of transmission:

Foodborne - consumption of undercooked meat, or unwashed vegetables that have been contaminated by Toxoplasma gondii cysts. [1]

Congenital - In cases where a pregnant mother is infected by Toxoplasma gondii, the parasite can infect the fetus in utero. This is most likely to occur later in pregnancy.[1][4]

Zoonotic - This method of transmission occurs during direct infection from animal to human. [1]

In some rare instances, Toxoplasmosis has been known to have been transmitted through donated organs and blood. [1]

Reservoirs

Members of the family felidae (including domestic cats), humans, and other warm blooded animals are the primary reservoir of T. gondii. [2] Contaminated soil and water, which can contain oocysts, are another reservoir. [2]

General Characteristics

Toxoplasma gondii is an obligate, intracellular, protozoan belonging to the family Apicomplexa. [1][2] The life cycle of T. gondii alternates between a sexually reproducing cycle within members of the family Felidae (felids) and an asexually reproducing cycle that takes place in intermediate hosts.[2] Sexual reproduction only takes place within the epithelial cells of the intestinal walls of felids, which are considered the definitive hosts of T. gondii.[2] Depending on the stage of the parasite’s life cycle that is taking place, T. gondii will take one of four morphologically distinct cellular forms. When a cat ingests a tissue cyst from an infected rodent, for example, the cyst releases a form of the parasite called the bradyzoite, which is a dormant, slowly reproducing form that infects the small intestine of the cat host. [1] The bradyzoites then convert to a rapidly dividing form called merozoites which proliferate in advance of sexual reproduction. [1] In the final stage of the sexual cycle, the resulting zygotes form oocysts
containing the stage of the *T. gondii* organism called sporozoites, which are excreted in the feces of the host.

The mature oocyst, containing eight sporozoites, is protected by a resilient exterior, allowing it to survive months outside of a host. Initiation of the asexual cycle of *T. gondii* begins after ingestion of the oocyst by an intermediate host. [2] Once inside the stomach of the host, sporozoites are released which then infect the host’s intestinal epithelium before converting to the tachyzoite form. Tachyzoites then proliferate throughout the host and multiply asexually, eventually converting to bradyzoites which are incorporated into tissue cysts, primarily in muscle, liver and brain tissue. [1] The multiplication of the parasite within cells eventually leads to the rupture and death of host cells.

**Key tests for identification**

Toxoplasmosis is usually diagnosed through the use of serological tests that detect antibodies specific to *T. gondii*, specifically the Sabin-Feldman dye test which detects IgG antibodies.[4] The parasite can also be isolated and detected in affected tissues and body fluids by visual examination. An ophthalmic slit lamp can be used to examine the retina for signs of damage caused by *T. gondii*. [4] Detection of *T. gondii* DNA via PCR (polymerase chain reaction) within amniotic fluid is used in cases of suspected congenital Toxoplasmosis in which the mother may have transmitted the infection to her unborn fetus. [4]

**Signs and Symptoms**

One possible reason Toxoplasmosis is relatively prevalent may be due to the fact that individuals with healthy immune systems that are infected by *T. gondii* usually show no outward signs or symptoms. [2] Upon initial infection an individual may experience swollen lymph nodes, headache, muscle ache and fever. [1]

In individuals with compromised immune systems, the symptoms of Toxoplasmosis are usually much more severe and longer lasting. These symptoms include encephalitis, confusion, delayed reaction time, retinal damage, and in some cases behavioral and psychological changes. [6]

In cases of congenital Toxoplasmosis symptoms are not often seen until later. [3] Possible symptoms include chorioretinitis, hydrocephaly, cranial deformities, and mental retardation. [3][4]

**Virulence Mechanisms**

When *T. gondii* tissue cysts or oocysts are consumed by a host, the parasite is released within the intestinal lumen where it soon enters the epithelial cells of the host. [1] Most of the damage caused by the *T. gondii* parasite occurs as a result of multiplication within the cells of the host. *T. gondii* is able to evade the host’s immune system by isolating itself within vacuoles. This eventually leads to cell death and localized necrosis within the infected tissues. [2]
Historical information

*T. gondii* was discovered and first described by Nicolle and Manceaux in 1908. The first conclusively diagnosed and documented case of Toxoplasmosis was in 1938 of an infant in New York City, who acquired Toxoplasmosis congenitally. The life cycle of *T. gondii* as well as its transmission was not well understood until 1970.[6]

Control/Treatment

Treatment for Toxoplasmosis is usually only administered when a woman is pregnant to prevent congenital transmission, or in immunocompromised individuals in order to prevent more severe complications. Treatment consists of sulfonamides and pyrimethamine, which when combined are more effective than either alone. [2]

Prevention/Vaccine

Presently, no vaccine has been developed to prevent Toxoplasmosis. The primary preventive measures that can be taken are to ensure that meat is properly cooked and fruits and vegetables are washed completely.[3] Washing one’s hands before food preparation, and especially after coming into contact with a cat litter box, are other important precautions. [3][2] When a woman is pregnant, it is recommended that she avoid any contact with cat waste as a precautionary measure. [3]

Current Information:

Epidemiology and Prevalence

Toxoplasmosis is widespread both in the United States as well as globally. It is estimated that up 22.5 % of the U.S. population aged 12 and older has been infected. Globally, up to one third of the population of developed countries is infected. [8]

Local cases or outbreaks

No reliable data stating statistics for Toxoplasmosis in either Travis County or Texas was available and no documented outbreaks have been recorded. Within the U.S., The last reported outbreak occurred in Illinois in 1984 among members of a family farm, in which nine cases were documented. [7]

Global cases or outbreaks

Globally, Toxoplasmosis is quite widespread, with an estimated incidence of up to one third of the population in developed countries. In 1995, in Victoria, British Columbia, Canada, a contaminated municipal water supply lead to an outbreak of 2,985 to 7,118 cases of
Toxoplasmosis. [7] In 2010, an outbreak of 248 cases of Toxoplasmosis occurred in Tamil Nadu, India. This outbreak was also traced to contaminated drinking water. [7]

**Works Cited**


