Toxoplasmosis

By Chris Lucas-Melanson

Toxoplasmosis – Protozoan Disease

Etiological Agent - *Toxoplasma gondii* (1)

**Transmission:** Occurs by three ways

1. Congenitally from mother to unborn child
2. Through ingestion of uncooked meat containing oocysts of the disease
3. Through feline fecal matter contamination

**Reservoirs:** Cats are definitive hosts and many mammals and birds serve as intermediate hosts (1)

**General Characteristics:** *T. gondii* is an obligate intracellular protozoic parasite of the coccidia subclass that specifically goes through the sexual reproduction life cycle in the feline host. It has been known to infect various warm-blooded mammal as an intermediate host. *T. gondii* cysts prefer to reside in the brain, neural, and liver tissues. It commonly spreads through ingestion into the gastrointestinal tract from which it can infect macrophages and form a cyst which grows in the phagocyte and will burst releasing the cell into other tissues. *T. gondii* has a polymorphic life cycle with both motile and non-motile stages (1)

**Key Tests for Identification:** The Sabin-Feldman dye test is the most reliable test. Live virulent tachyzoites of *T. gondii* are used as antigen and are exposed to dilutions of the test serum and to a complement accessory factor resembling complement that is obtained from *Toxoplasma*-antibody free-human serum. This test is highly sensitive and specific for Toxoplasmosis, but dangerous due to the use of live antigen. Unfortunately it’s high cost means it is often unavailable in developing countries (1)

Many serological tests can be used to identify *T. gondii* in a host including the direct hemagglutination test, the latex agglutination test, and the enzyme-linked immunosorbent assay (ELISA). The ELISA test is specific and appears to be the standard test for *T. gondii* (1)

Serologic evidence for an acute acquired infection is obtained when antibody titers rise by a factor of 4 to 16 in serum taken 2 to 4 weeks after the initial serum collection, or when specific IgM antibody is detected(1)

This serological evidence only confirms that the patient may have been infected in the past. To confirm an acute acquired infection, antibody titers rising by a 4 to 16 rate taken 2 to 4 weeks after initial serum collection must be seen. (1)

For immunocompromised patients who may not develop antibodies, a smear identifying the organisms by their structure can be made from a biopsy of host tissue.(1)
**Signs/symptoms:** Lymphadenitis (inflammation of lymph nodes) accompanied by fever, malaise, fatigue, muscle pains, sore throat, and headache. Encephalitis is a severe symptom of the disease in immunocompromised patients and headache, disorientation, drowsiness, hemiparesis, reflex changes, and convulsions can occur (1)

Ocular *T gondii* can be the result of congenital infection. Congenitally infected patients are often asymptomatic until the second or third decade of life, when lesions can begin to develop in the eye. (2)

It’s most interesting symptom however, is its ability to alter the behavior of its host. In mice and rodents, *T. gondii* has been shown to increase levels of activity and decrease the fear of feline predators. This can cause mice and rodents to go as far as to seek out their feline predators. Otters with moderate to severe encephalitis have been shown to be 3.7 times more likely to be eaten by sharks. (5) Strong correlations have been found between rates of *T. gondii* infection and likelihood of committing suicide, especially in women (6)

In people of normal immunocompetence, little to no symptoms are likely to occur. This decreases however, with the quantity of *T. gondii* that is transmitted to the patient. (1)

**Historical information:** *T gondii* was first isolated from a North African gundi rodent by Nicolle and Manceaux in 1908. The name Toxoplasma gondii was based on the shape of the infectious stage, toxon means bow or arc in greek and plasma means life. Gondii may be a result of the misspelling of the gundi rodent where it was first found. (3)

**Virulence Mechanisms:** *T. gondii* has several virulence factors owing to its various life stages, making it a highly dynamic and successful parasite. One of these includes the ability to exit the phagosome before fusion of its vesicle with a lysozome containing vesicle. It also has the ability to enter a phagocyte, independent of a receptor mediated-endocytosis mechanism. Due to its polymorphic life cycle, it has the ability to be ingested in the gastro-intestinal tract both as a cyst in meat or as an oocyst in feces. It’s tachyzoite form has the ability to be motile, and can form new cysts to allow further intracellular reproduction. The bradyzoite forms are latent intracellular tissue cysts that are unresponsive to antibiotics. Once in the body tachyzoite *T. gondii* activates the inflammatory immune response, causing flu-like symptoms in acute cases. (4)

**Control/Treatment:** Though the majority of patients will not develop any symptoms during infection, there are drugs available if the infection becomes more severe. These include pyrimethamine sulfadiazine and leucovorin which are usually prescribed together. Other drugs are available depending on the patient specific reactions and allergies to the drugs. Spiramycin is commonly used to treat early maternal infections up to the early second trimester. After the second trimester then pyrimethamine, sulfadiazine, and leucovorin are commonly prescribed. AIDS and other immunocompromised patients require more specific treatment until a significant immunological improvement is shown. (2)

**Current Information and outbreaks/prevention:** Serological prevalence data indicate that toxoplasmosis is one of the most common human infections throughout the world, and affects approximately one third of the population. Variation from country to country is high. The particularly high prevalence in France has been linked to a culinary preference for raw and
undercooked meat, while the high prevalence in Central America has been related to the high frequency of stray cats in a climate favorable to the survival of the parasite. (2) In the U.S. the overall prevalence among people older than six is 12.4% and among women of childbirthing age is 9.1%. (2)

**Prevention:** There are no current vaccines available and taking preventative measures is often the best way to ensure you won’t become infected. Regularly cleaning the cat litter box, which stops oocytes from sporulating in one to five days is very important. Thorough hand washing and proper food preparation and cooking are also effective at limiting exposure.

Works Cited:


