Toxoplasmosis

Toxoplasmosis is a parasitic disease caused by the element *Toxoplasma gondii*. The disease has a wide range of clinical manifestations and occurs with varying prevalence around the world. It affects humans, mammals and birds.

Toxoplasmosis usually occurs subclinically, with only a small proportion affected by lymphadenitis, influenza symptoms, retinal and CNS involvement. Toxoplasmosis has serious consequences in fetuses, newborns and immunocompromised patients (especially in cases of T-cell immunity - hematological malignancies, bone marrow and organ transplantation, AIDS).[1]

Toxoplasma gondii

- element (Protozoa);
- obligately intracellular parasite;
- 3 main genotypes that differ in pathogenicity and prevalence in humans.[1]

Life cycle of *T. gondii*

Sex cycle

- takes place only in the definitive host, which is the cat.

Asexual cycle

- occurs in mammals, including humans, and some bird species.

2 forms:

- tachyzoite - a rapidly dividing form that occurs in the acute phase of infection;
- bradyzoites - a slow-growing firm that forms tissue cysts.

The cat becomes infected with T. gondii by ingesting contaminated raw meat from birds or mice. The sexual cycle begins in the cat's digestive tract. Macrogametocytes and microgametocytes are formed from digested bradyzoites, which merge to form zygotes. Zygotes encapsulate a solid wall (encapsulate) and become oocysts. Inside the oocyst, the zygote sporulates and divides to form sporozoites. An infected cat excretes oocysts in the faeces. Sporozoites become infectious after 24 hours or more. In a primary infection, a cat can excrete millions of oocysts daily for 1-3 weeks. Oocysts are very resistant and can remain infectious for more than a year in a warm humid environment.[1]

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Transmission

In humans, oocysts, tachyzoites and bradyzoites of T. gondii can cause infection. Humans are most often infected by ingesting oocysts from contaminated soil or cat excrement or by contaminated water or food (unwashed vegetables from the garden). However, oocysts are not infectious immediately after leaving the cat's digestive tract, but must undergo a developmental phase of sporulation in the external environment, after which they become infectious to all warm-blooded vertebrates. Therefore, sandpits where children play are risky, because in the case of contamination of the sandpit with cat feces, the development of spores may have ended. Rarely, transmission is possible with unpasteurized milk or blood transfusions. Transmission is also possible by ingesting tissue cysts in raw or undercooked meat (mainly pork in Europe and the USA). Rarely, infection with a tissue cyst organ transplant from an infected donor can also occur. Food can be contaminated secondarily, through secondary contamination of food by oocysts, which are transmitted by flies or cockroaches. The transmission of tachyzoites to the fetus takes place transplacentally during the primary infection of the pregnant woman.[1]

Pathogenesis

After ingestion of oocysts, bradyzoites or sporozoites begin to release and enter the host (nuclear) cells of the digestive tract. The tachyzoites divide within the host cell until the cell ruptures and the tachyzoites infect neighboring cells. They spread through the lymph and disseminate hematogenously through the tissues.

Tachyzoites proliferate, forming necrotic deposits surrounded by a cellular response. As the normal immune response develops, the tachyzoites disappear from the tissues. In immunocompromised patients, acute infections may progress further and result in pneumonia, myocarditis, and necrotizing encephalitis.

Tissue cysts form as early as 7 days after infection and remain throughout the host's life. It produces very little or no immune response.[1]

Clinical picture

Toxoplasmosis is accompanied by symptoms in only about 10-20% of cases. It can have a serious or life-threatening course in immunodeficient patients. Congenital toxoplasmosis has a wide range of symptoms that may appear in the perinatal period or later.[1]

Acute toxoplasmosis

Immunocompetent persons

In about 80-90% of cases, it is asymptomatic.

Clinical signs - resolve spontaneously:

- cervical lymphadenopathy with painful nodules up to 3 cm in diameter;
- fever, malaise, night sweats, myalgia, sore throat, abdominal pain due to retroperitoneal and mesenteric lymphadenopathy, maculopapular rash;
- retinochoroitis - "ocular form of toxoplasmosis" (more often unilateral; eye pain, visual disturbances; necrotic lesions on the back of the eye).[1]

Immunosuppressed individuals

It can be a primary infection or reactivation.

Clinical signs:

- CNS toxoplasmosis (in about 50% of immunosuppressed individuals; especially in AIDS patients) - convulsions, dysequilibrium, head nerve paresis, hemiparesis, mental disorders, headaches, visual disturbances, focal neurologic deficits;
  - encephalitis, meningoencephalitis, CNS lesions;
- flu-like symptoms and lymphadenopathy - as in immunocompetent individuals;
- myocarditis;
- pneumonia (prolonged febrile illness with cough and dyspnoea; evidence of T. gondii in bornchoalveolar lavage).[1]

Latent toxoplasmosis

- In many people, the disease "skips" the acute phase;
- a person is subjectively without problems, only antibodies are present in the blood;
- however, once **immunosuppression** occurs, the disease is **activated**.

After a short phase of acute toxoplasmosis, the infection enters a latent phase with the formation of tissue cysts, especially in the nerve and muscle tissue.

Toxoplasmosis causes typical behavioral changes in rodents (impairs motor and learning ability, reduces neophobia and fear, prolongs reaction time), which apparently facilitates transmission of the parasite to the final host, ie ingestion by a cat.[2]

According to some studies, latent toxoplasmosis in humans prolongs reaction time and changes personality.[3][4][5] Further research has found an increased seroprevalence of toxoplasmosis in road accident victims;[6][7] especially in Rhd) -negative individuals with high titers of anti-toxoplasmosis antibodies.[8]

Other studies suggest that chronic toxoplasmosis may be involved in the development of various mental disorders.[9] An increased prevalence of anti-toxoplasmosis antibodies has been demonstrated in schizophrenics and other severely mentally ill patients.[10] The pathogenesis is explained by the effect of T. gondii on neurotransmitters and its action in areas of the brain affected by schizophrenia.[11]

Further studies found a significantly increased incidence of anti-toxoplasmosis antibodies in patients with Parkinson's and Alzheimer's disease compared with controls.[12][13]

However, this hypothesis of the effect of toxoplasmosis on humans is based primarily on correlation studies. But the correlation says nothing about the causality of relationships[1] (https://cs.wikipedia.org/wiki/Korelace_neimplikuje_kauzalitu). In terms of causality [2] (https://cs.wikipedia.org/wiki/Kauzalita) it already has the value of longitudinal research[3] (https://cs.wikipedia.org/wiki/Longitudin%C3%A1ln%C3%AD_v%C3%BDzkum). It was performed on a random, representative sample and refutes this hypothesis[14].

Congenital toxoplasmosis

Toxoplasmosis is a fetal infection (TORCH). The infection has the most serious consequences at the beginning of pregnancy.

A significant proportion of children with congenital toxoplasmosis do not have detectable specific IgM antibodies after birth and in early childhood. About 67% of patients have no symptoms of infection.[1]

The clinical picture of congenital toxoplasmosis is dominated by CNS and retinal disorders:

- The so-called **Sabin's Triassic** or **Tetrada**:

1. chorioretinitis (approximately 15% of cases; more often bilateral)[1];
2. *cerebral calcifications* (approx. 10% of cases)[1];
3. *convulsions*;
4. *hydrocephalus*.

Newborns with congenital toxoplasmosis tend to have anemia, thrombocytopenia, and hyperbilirubinemia. Later, mental retardation, convulsions, visual disturbances, spasticity, hearing impairments (sensorineural hearing impairments) and other serious neurological manifestations manifest themselves.[1]

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Diagnostics

Direct detection of T. gondii in blood, body fluids (amniotic fluid, etc.) or tissues by culturing on cell cultures or by PCR.

Indirect detection of specific IgG and IgM antibodies.

Auxiliary imaging methods (CT/MRI of the brain, ultrasound of the brain in newborns,...).[1]

Therapy

Toxoplasmosis in immunocompetent pregnant women usually requires treatment.

Symptomatic treatment.

In indicated cases of eradication of tachyzoite using pyrimethamine (not effective against tissue cysts - bradyzoites), which is given in combination with folinic acid (folinic acid), which is the antidote to foliar cyst antagonists, which is pyrimethamine. Pyrimethamine is usually administered concomitantly sulfadiazine.[1]

Links

Related articles

- Fetal threatening infections• Congenital toxoplasmosis
- High-risk pregnancy and the newborn

Reference

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Used literature

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