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# Toxoplasma Gondii-Morphology, Prevention And Laboratory Diagnosis

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**Abstract**: This article deals with Toxoplasma gondii-morphology, prevention and laboratory diagnosis.

**Key words:** toxoplasma, HIV, encephalitis, cerebral toxoplasma, opportunistic infections.

Etiology and pathogenesis. The causative agent of the disease is Toxoplasma gondii. Its forms, i.e. oocysts, live in the small intestine of cats. Oocysts are excreted in the feces of these animals. Oocysts enter the human body mainly through the oral route. However, oocysts can enter the body through open skin wounds. Also, the infection is transmitted from the pregnant mother to the child through the placenta, i.e. transplacentally.

Parasites that enter the human body enter the epithelial cells of the intestines and begin to multiply there. Formed trophozoites spread to all organs by hematogenous and lymphogenous ways. Some of them die in large quantities, and toxic substances are formed from them. As a result of this, general allergy and hypersensitivity develop in the body. The organism begins to protect itself, that is, it produces antibodies against trophozoites. As the titer of antibodies increases in the body, trophozoites that have not had time to enter the cells and circulate in the blood begin to die. The trophozoites that have passed into the tissues form cysts. Cysts are often located in the brain, heart and skeletal muscles, uterine muscles and eyes. Cysts burst, parasites come out of them and spread to other organs by hematogenous way. At this time, the symptoms of the disease arise.

The pathogenesis of toxoplasmosis transmitted to the child through the transplacental route is somewhat different. An infection circulating in the blood of a pregnant woman is transmitted to the child through the placenta. During this period, the child's immune system is not yet formed. The risk of passing this infection to the child is high, especially in the I and II trimesters of pregnancy. Toxoplasmas have a teratogenic effect, they disrupt embryogenesis and often cause miscarriage. Even a healthy child can get toxoplasma infections. This is called congenital toxoplasmosis. Such children are born with various defects (hydrocephaly, microcephaly, anencephaly, body deformities).

Pathomorphology. Toxoplasma infections have the ability to pass through any barriers. They pass through GEB and damage brain ventricles, parenchyma, meninges and blood vessels. Choroidal

tangles and periventricular area are especially affected. Toxoplasmosis infection causes severe intoxication and allergy. As a result of the excitation of the choroidal tangles, the production of cerebrospinal fluid increases, IKG develops. Ventricles and subarachnoid spaces begin to expand. A reactive inflammatory process begins in choroidal tangles and meninges, that is, reactive chorioependymatitis and leptomeningitis develop. The places where toxoplasmas have fallen atrophy, and necrotic foci are formed in those places. Subarachnoid cisterns and brain ventricles expand due to atrophy of cortical neurons, soft tissue and periventricular area, and necrotic foci are formed in these areas. Serous-proliferative inflammatory reactions can be observed in these tissues. Calcium salts are collected in places where cysts break down and necrotic foci appear. Destructive changes also develop in areas covered with white matter of the brain, that is, myelin. A specific-reactive encephalitic process takes place in these places.

Since toxoplasmosis is an infectious-allergic, toxic-allergic process, lymph nodes enlarge, retinitis and choroiditis develop in the eyes. Cerebral toxoplasmosis is very rare. Damage to internal organs, especially the liver, spleen, lungs and heart, is almost always detected. Toxoplasma most often spreads from the liver and lungs. They live hidden in these tissues for many years.

Along with the brain, the spinal cord can also be damaged. In such cases, encephalomyelitis or toxic-allergic encephalomyelopathy develops. But myelitic symptoms are less pronounced than encephalitic symptoms.

Brain toxoplasmosis develops neurasthenia, encephalopathy, leptomeningitis and chorioependymatitis. So, in mild cases, only symptoms of neurasthenia are detected, and in severe cases, encephalopathy, leptomeningitis, and chorioependymatitis develop. Pseudotumorosis is typical for cysts with large foci.

The clinic of acute toxoplasmosis begins acutely. Body temperature rises to 39-40°C, headache, dizziness, nausea and general weakness appear. In most cases, acute toxoplasmosis begins with psychotic disorders, that is, delirious states, and in rare cases with epileptic attacks. Rigidity of the neck muscles, Kernig's symptom, pyramidal, extrapyramidal and vestibulo-coordinator disorders develop. Visual function is almost always reduced. Along with cerebral symptoms, there are symptoms of lymphadenopathy, cardiomyopathy and reactive hepatitis.

Latent toxoplasmosis is characterized by the complete absence of clinical symptoms. If the immune system is strong, toxoplasmosis is hidden. The presence of toxoplasmosis in a patient is accidentally detected during neuroimaging and laboratory tests. If foci of toxoplasmosis and calcifications are found in the brain on CT or MRI, eosinophilia is always detected in the blood. It is necessary to check the internal organs of such a patient (lungs, liver) and conduct specific laboratory tests. Latent toxoplasmosis is activated in extreme cases and clinical symptoms begin to appear.

Diagnosis. Early diagnosis is difficult. In most cases, the disease is detected during preventive examinations. However, if "unexplained" headache and subfebrile temperature, lymphadenopathy, general weakness, malaise, loss of appetite, nausea, abdominal pain, liver pain, eosinophilia are detected in the blood, the patient has parasitic diseases (toxoplasmosis, cysticercosis, echinococcosis). it can. Such a patient should undergo more serious medical examinations. Because the clinic of the disease may not appear for 15-20 years.

Comparative diagnosis. Comparative diagnosis is carried out with other etiological inflammatory diseases of the MNS (leptomeningitis, chorioependymatitis, encephalitis) and brain tumors.

Treatment. In acute toxoplasmosis, adults are prescribed 75 mg of pyrimethamine in 1-3 days,

25 mg in 4-7 days. Children are recommended to drink 2 mg/kg for the first 3 days, and 1 mg/kg for the next 4-7 days based on their body weight. In parallel, sulfadizin is taken 500 mg 4 times a day for 7 days. Also, spiramycin, roxithromycin, azithromycin may be recommended. The dose of drugs is determined depending on the severity and course of the disease. Antihistamines and general stimulants are also made. The effectiveness of drugs in chronic toxoplasmosis is very low. Because pharmacological agents and antibiotics have almost no effect on endozoites. In such cases, a surgical operation is used.

Prevention. Pregnant women undergo special examinations to prevent congenital toxoplasmosis. Detected infections are eliminated. Prevention of acquired toxoplasmosis consists in following general hygiene requirements.

Prognosis. Congenital toxoplasmosis has a poor prognosis. Most children born with this infection die within weeks. Those who survive will have severe brain injuries. They lag behind neurological and neuropsychological development. In chronic transient types of acquired toxoplasmosis, special treatment is not required. Patients with well-maintained immunity will recover. In acute viral infections (AIDS, herpes, etc.), chronic toxoplasmosis can become acute.

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