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Toxoplasmosis, a Disease as Old as it is New

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Abstract

Toxoplasmosis is an infectious disease caused by Toxoplasma Gondii, an obligatory intracellular parasite of all warm-blooded hosts worldwide, which is considered the most prevalent parasitic infection in humans and domestic animals in the world. Toxoplasmosis in an otherwise healthy person may have no symptoms or only a few swollen glands usually in the patient's neck, but in people with a weakened immune system may only have symptoms of swollen glands or an infection that attacks the brain and nervous system. Its diagnosis is based on the combination of the following examinations: a) serological examinations b) molecular c) histological d) imaging. The key to effective treatment is the combination of antiparasitic preparations, although tissue cysts are resistant to a large number of antimicrobial agents. Acquired parasitosis, even in the asymptomatic form, constitutes a serious public health and economic problem.

Keywords: Toxoplasmosis, Toxoplasma gondii, epidemiology, diagnosis, treatment.

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INTRODUCTION

General concepts

Toxoplasma gondii is an old parasite, but every day the medical world is more and more concerned about the many problems it causes. The disease related to this parasite has received different names over the years such as: "litter box disease"; "a mononucleosis-like illness"; "life-threatening CNS disease"; "flu-like" symptoms; "crazy cat-lady syndrome" Toxoplasmosis is a worldwide infectious disease from Alaska to Australia introduced in 1/3 of the human population (1). The causative agent is the obligate intracellular parasite*Toxoplasma gondii*, which appears in about 200 species of mammals and birds. *T. gondii* is one of the most successful protozoan parasite due to the large spectrum of affected hosts as it infects all warm-blooded animals, domestic, agricultural, birds including also humans who serve as intermediate hosts and complete the cycle life of the parasite (2).

Taxonomic characteristics

There is a scientific classification of it that summarizes: Domain Eukaryota; Kingdom Chromalveolata; Nentype: Alveolata; Type: Apicomplexa; Class: Conoidasida; Subclass: Coccidiasina; Order: Eucoccidiorida; Family: Sarcocystidae; Subfamily: Toxoplasmatinae; Genus: *Toxoplasma*; Type: *T. gondii*; Binomial name: *Toxoplasma gondii* (Nicolle &Manceaux 1908).

Historical data:

T. gondii was first described by the French scientists Nicolle and Manceaux in 1908 in the rodent Ctenodactylus gundi in North Africa. In 1909, they named toxoplasma for its half-moon arc shape (Greek: toxon- arc; plasma-form) and gondii (after the rodent). At the same time in 1908, Splendore described toxoplasma in a laboratory in Brazil (3). In 1909, the parasite was differentiated from *Leishmania* and was named *T. Gondii*. The discovery of the electron microscope in the 60s visualized the apical complex, making it possible to place *T. gondii* within the phylum Apicomplexa of the kingdom Protista (4).The complete life cycle of *T. gondii*was recognized in 1970 where the cat is defined as the definitive host for the pathogen and the oocysts are secreted in their faeces while humans, other mammals and birds are intermediate hosts. The first case was described in 1938 in a small girl in New York with convulsions three days after birth and death within a month. The autopsy showed the presence of the parasite in the brain, spinal cord, retina (5). The first case of congenital infection was seen in a 6-year-old boy in 1941 with headache and convulsions after being hit with a baseball bat. The doctors discovered the infection with *T. gondii* that developed acute inflammation of the brain, encephalitis that led to death (5). In 1958, Beverley & Beattie confirmed the link between lymphadenopathy and toxoplasmosis. In 1981-83, the first cases of cerebral toxoplasmosis in AIDS patients were recorded, and in 1984, *T. gondii* was recognized as an opportunistic pathogen in AIDS patients. In 1995, the largest recorded epidemic of acute infection in humans

(100 individuals aged 6–83 years) related to oocysts in municipal drinking water was seen.

Physiopathological forms

There are three physio-pathological forms of *T. gondii*: A) Tachyzoite (vegetative form): typical eukaryotic cells that replicate in the cells of the intermediate host and in the non-intestinal epithelial cells of the definitive host during the acute stage of infection. They invade the muscles of the heart, liver, spleen, lymphatic glands, and CNS and induce the humoral and cellular inflammatory response that eliminates most of the parasites but also leads to immune-related tissue destruction. B) Bradyzoite: Bradyzoite and cystozoite are the same thing. A tissue cyst is a collection of bradyzoites surrounded by a well-defined host cell membrane that is obtained from the gelling of an immature acorn and multiplies slowly. Cysts appear 7 days after infection and remain in the host for life. They do not produce or produce very little inflammatory response but cause severe disease in immunosuppressed persons. C) Sporozoites. This form of the parasite is found in oocysts that are shed by cats into the environment. Cats excrete millions of oocysts after swallowing a bradyzoite or an indoor cyst (6).

Biological cycle

The biological cycle contains two phases: A) Gametogonic/sexual phase, (enteroepithelial cycle): develops only in the intestinal epithelium of definitive hosts (domestic and wild cats). The bradyzoids that are released from the destruction of the cystic wall, in the enterocytes undergo a reproductive sexual process called "merogany" that ends with the formation of the zygote, the oocyst. This is eliminated with the cat's feces in the external environment as unsporulated oocysts, which are not infectious. The process of sporogony occurs in the outdoor environment after 48 hours. B) Schizogonic/asexual phase, (extraintestinal cycle): develops in intermediate hosts (birds, mammals, humans) after swallowing cats or consuming live meat with tissue cysts. During this phase, parasitaemia develops, the acute phase of the disease that leads to degenerative-necrotic lesions in the parasitized organs. There is a feature (internal inflammation) that leads to the formation of cysts that remain latent for a long time in muscles (skeletal, brain, eyes, liver, heart, lungs, uterus, pancreas, digestive tract) risking disease in immunocompetent persons (7).

Ecology

The parasite has no harmful effects on the environment and does not participate in any biochemical synthesis that damages or contributes positively to the biosphere. The main effect is at the individual level as an obligate intracellular parasite targeting warm-blooded vertebrates and their hosts. Ecology is related to factors such as: climatic factors, seasonal factors, geographical factors (8,9).

Ways of infection:

Humans can be infected by different routes: A) the oral route (accidental ingestion of parasitic oocysts through hands contaminated with feces or through food), B) the transcutaneous and mucosal

route, C) the respiratory route, D) the transplacental route, E) the nosocomial route (blood transfusion, organ transplant, laboratory accidents) (10,11).

Transmission mode

Several ways of transmission of the parasite are known: A) from cat to other animals, B) from other animals to cat, C) from animals to animals, D) from mother to offspring (12,13).

Epidemiological information

This versatile parasite infects more than a third of the human world (14). The prevalence of parasitosis in the world varies from 0 to 100% (15). Seroprevalence in Europe varies from 54% in southern European countries to 5-10% in northern Sweden and Norway. It is reported that 50% of the European population is infected in the 3rd decade of life, in France it was 80% (16).

Epidemiological factors

In addition to the climatic, seasonal and geographical factors mentioned in ecology, we describe also other factors such as: race, gender, age and age group, educational level (17-19).

Risky professions

There are several professions that are more vulnerable to this parasite, such as: veterinarians, animal keepers and breeders, slaughterhouse workers, cooks, archaeologists, farmers, gardeners, laboratory and health care workers (20-22).

The parasite and its food origin

This parasite is also closely related to food, since about 750 deaths are attributed to the parasite every year, where 375/50% are of food origin, making the parasite the third cause of food-related deaths in the USA (23).

Clinical data:

Exposure to the parasite does not necessarily lead to infection. The appearance of this infection will depend on the stage of the parasite, route of intake, virulence and parasite dose. Mode of infection: a) congenital b) acquired. Clinical stages include 3 stages: 1) primary septicemic stage, 2) secondary immune stage, 3) chronic tertiary stage. Based on these stages, we will have this clinical course: A) acute, B) subacute, C) chronic D) reactivated. The clinical categories include: a) acute toxoplasmosis acquired in immunocompetents (inapparent/asymptomatic and symptomatic forms), b) acute toxoplasmosis acquired or reactivated in non-HIV immunosuppressed persons, c) acute toxoplasmosis acquired or reactivated in persons with HIV, d) ocular toxoplasmosis, e) congenital infection. The literature talks about the strong connection of the chronic parasitic form with the neuropsychiatric changes that include schizophrenia; suicide; the development of psychosis, depression, anxiety. Studies in immunocompetent adults regarding the neuropsychiatric effects of *T. gondii* have been done (24). Jaroslav Flegr in 1992 at Charles University in Prague studied the effects of *T. gondii* on personality and human behavior, including female students, military recruits, and blood donors. Seopositive men were antisocial, dogmatic and suspicious. Seropositive women were superego (25). Some studies describe a

possible link between the parasite and Parkinson's disease, cryptogenic epilepsy or migraine (26, 27). The parasite increases the risk of suicide in infected people (28). We can also say that subjects with latent parasitosis have a significantly higher risk of road accidents compared to uninfected subjects. Studies on seropositivity in accident victims and the control group have been done (29). The negative effects of parasitosis on the psychomotor performance of men influence the rate of road accidents. The parasite has a negative effect on the psychomotor system of men, influencing the rate of road accidents (30). RhD phenotype plays an important role in the relationship between latent form and psychomotor performance, personality and intelligence. Jaroslav Flegr and colleagues suggest serology testing in Rh-negative pilots, air traffic controllers, and barge drivers (31).

People with high risk

There is a category of people more at risk for this parasite such as: people with HIV/AIDS, patients with malignant pathology, patients undergoing chemotherapy or under the use of steroid or immunosuppressive preparations, pregnant women, organ transplant recipients, people undergoing transfusion (32-34).

Diagnosis

The diagnosis of parasitosis is based on the combination of the following examinations: A) serological examinations B) molecular C) histological D) imaging (35-38).

Treatment

The key to effective treatment is the combination of antiparasitic preparations, although tissue cysts are resistant to a large number of antimicrobial agents (39,40).

Control and prevention

We must say that HIV positive people, those with damaged immunity or who take immunosuppressive medications, pregnant women or those who are planning a pregnancy, should be concerned about Toxoplasmosis. But should they give up their love for the cat? NO.By by knowing the routes of reception we can sufficiently minimize the risks of transmission. Programs for the education of pregnant women about the prevention of parasitosis have given positive results in changing risky behaviors, where a decrease in seroprevalence has been seen (41). From the first prenatal visit, health education/consultant workers should educate the pregnant woman about food hygiene and avoiding exposure to cat feces.

CONCLUSIONS

This material sheds light on a parasitic pathology that constitutes a serious public health and economic problem. This parasite appears in three important forms, such as lymphoglandular, ocular and cerebral. Global warming affects its spread. Toxoplasmosis is a disease present in all seasons of the year. The consumption of undercooked meat remains a major risk factor. It

is difficult to determine the evolutionary stage of parasitosis. Lymphadenopathy is the most frequent clinical manifestation of acquired infection in immunocompetents. The diagnosis of parasitosis is based on the general criteria of infectious diagnostics, which include epidemiological, clinical, immunological, imaging and therapeutic criteria. The treatment of parasitosis includes medications from different pharmacological groups that are often accompanied by not few and mild but not rare side effects and an insensitivity of the parasite to them.

RECOMMENDATIONS

a. Continuous screening of HIV patients is necessary to avoid severe neurological forms. Prophylaxis is life-saving.

b. Hemodialysis patients are a risk group, so serological examination before dialysis is recommended to avoid parasitic dissemination.

c. Pathological manifestations in transplants may eventually be related to parasitosis, so their screening is now necessary.

d. Toxoplasmosis testing of blood donors should be routine.

e. The presence of the infection doctor in many services should be increased, since the parasite itself shows many clinical manifestations.

f. Neuropsychic symptoms can be worn by our parasite, so we should be close to neuropsychiatric consultations.

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