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Review Paper

Toxoplasmosis: Clinical presentation, diagnosis and prevention

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Abstract

Introduction: Toxoplasmosis is a cosmopolitan parasitic disease caused by the protozoan *Toxoplasma gondii*. While it remains asymptomatic in most cases, a more severe course is seen especially in immunocompromised individuals as well as in neonates and infants with congenital toxoplasmosis.

Aim: The aim of this review article is to present the most important information on the course of the disease, its diagnostics and preventive measures targeted at reducing the risk of infection.

Material and methods: A literature search was conducted using electronic databases such as PubMed, Google Scholar and Willey Online Library. The analysis included the papers published between 1948 and 2024. Altogether, 38articles were subjected to analysis.

Results and discussion: The data obtained in the literature survey have been systematized and presented in 3 sections. A wide range of clinical presentations of toxoplasmosis dictates the need to include both clinical examinations and laboratory tests into the diagnostic process.
It is of key importance to diagnose the disease in its early stage and to institute the appropriate treatment.

Conclusions: The main risk factors for *T. gondii* infection are any practices which represent departures from sanitation and hygiene standards. Moreover, educational campaigns should be given a high priority, particularly those targeted at pregnant women. Screening tests aimed at an early detection of the infection should come into a wider use.

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1. INTRODUCTION

Toxoplasmosis is a cosmopolitan parasitic disease caused by the protozoan *Toxoplasma gondii*. This parasite commonly infects humans and most warm-blooded animals. The life cycle comprises both sexual and asexual reproduction stages. The former occurs in the definitive host: the domestic cat and other felids (lynx, puma, jaguar), while the asexual stage takes place in the intermediate host – humans – or in other mammals and birds.[1] In the majority of cases, the infection is either asymptomatic or presents with mild symptoms. A more severe course of the disease is seen particularly in immunocompromised individuals as well as in neonates and infants with congenital toxoplasmosis.[2] Epidemiological studies have shown that toxoplasmosis is a disease reported worldwide; it has been classified as a foodborne infection.[3]

The main routes of transmission include: consumption of raw or undercooked meat containing parasite cysts, drinking untreated water or ingestion of fruit or vegetables contaminated with oocysts from cat feces, and the vertical route.[4] Examples of iatrogenic transmission, which is relatively rare but very important from a medical point of view, are shown in Figure 1.

The analysis of polymorphic surface antigen glycoprotein SAG2 and dense granule antigen GRA4 identified three main strains with three different genotypes. While type I is a highly virulent strain (LD100 = 1), types II and III are considered to be of lower virulence (LD50 ≥ 1000).[5] In humans, type I is predominantly responsible for acquired infections with an acute clinical course, whereas type II is a common cause of congenital toxoplasmosis in Europe; it is also the most common strain to be isolated from animals. The type III strain of *T. gondii* was isolated mainly from the environment.[6,7] In Poland, the seroprevalence of *T. gondii* in humans varies between 40%–60%, depending on the study cohort.[8–11]

In recent years in Poland only congenital toxoplasmosis has been a reportable disease and, consequently, the prevalence of toxoplasmosis in humans may be underestimated. Clearly, there is a need to include information about other clinical presentations of the disease i.e. lymphadenopathy, ocular toxoplasmosis or neurotoxoplasmosis in the annual epidemiological reports.

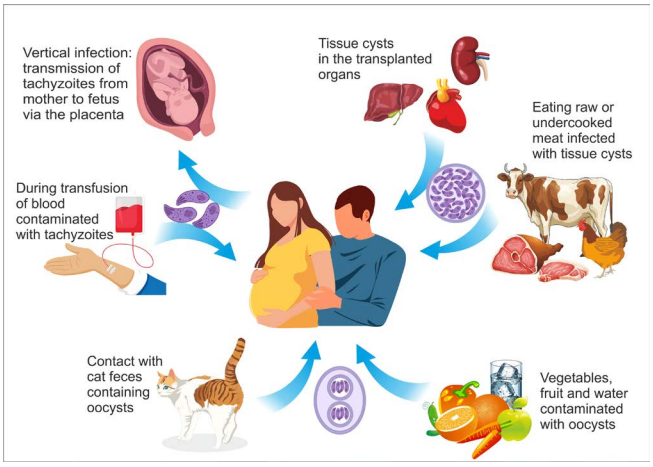

Figure 1. Major routes of transmission of *T. gondii*.

2. AIM

The aim of this review article is to present the most important information on the course of the disease, its diagnostics and preventive measures targeted at reducing the risk of infection.

3. MATERIAL AND METHODS

The literature search was conducted using electronic databases such as PubMed, Google Scholar and Willey Online Library. The analysis included the papers published between 1948 and 2024. Altogether, thirty eight articles were subjected to analysis. The keywords used in the article search were: 'Toxoplasmosis,' '*Toxoplasma gondii*,' 'Diagnosis,' 'Prevention,' 'Clinical manifestations.' The exclusion criteria were lack of Polish or English full-text version and publications on toxoplasmosis in animals.

4. RESULTS AND DISCUSSION

4.1. Clinical presentation

Toxoplasma gondii is responsible for causing toxoplasmosis in humans, either in its acquired or congenital form. The invasion is usually asymptomatic. In its acute form, acquired toxoplasmosis is characterized by fever and the symptoms on the part of the affected organs. The most commonly diagnosed forms are: lymphadenopathic, congenital and ocular toxoplasmosis. The clinical presentation and the severity of symptoms depend on a number of factors, including: the immune status of the patient, the route of transmission (acquired vs. congenital toxoplasmosis) and the virulence of the *T. gondii* strain.[12]

Lymphadenopathic toxoplasmosis

Lymphadenopathic toxoplasmosis is associated with the involvement of lymph nodes in the head and neck region. It may be accompanied by fever, malaise and muscle pain. Some authors describe this presentation of toxoplasmosis as a mononucleosis-like infection.[13]

The symptoms resolve over 1–2 months in 60% of immunocompetent patients if no antiparasitic treatment is used.[14]

Ocular toxoplasmosis

The clinical symptoms of ocular toxoplasmosis depend on the anatomical location of the lesion.[15] The disease typically begins with a focal retinitis, usually with secondary involvement of the choroid. The vitreous body shows signs of inflammation. The involvement of the macula and the optic disc, subretinal neovascularization and retinal detachment lead to visual impairment, resulting in complete vision loss.[13] Over the next 1–2 months the inflammation resolves spontaneously, leaving a characteristic finding in the form

of a scar, which is usually oval-shaped, often with black pigment granules visible on its peripheries. The recurrence of inflammation may occur following the reactivation of the existing *T. gondii* cysts. The secondary lesion tends to appear on the edges of the previous lesion, so, in consequence, the newly-formed inflammatory foci are adjacent to the existing scars, and are thus referred to as satellite foci. Over 50% of the patients can be expected to have recurrences of oculopathy despite antiparasitic treatment instituted during the periods of exacerbation.[16]

Congenital toxoplasmosis

Congenital toxoplasmosis occurs in children whose mothers acquired primary infection with the parasite *T. gondii* during pregnancy or shortly before gestation.

The risk of developing congenital toxoplasmosis and the severity of fetal damage depends on the time of the mother becoming infected. The risk of mother-to-child transmission is the lowest during the first, and the greatest during the third trimester of gestation.[17] Infecting the fetus in the first trimester may result in the loss of pregnancy, stillbirth, or delivering a child with major defects and abnormalities involving the brain and eyes (the Sabin–Pinkerton triad).[18] If the transmission occurs during the second or third trimester, the infection may be subclinical or asymptomatic at birth. Nevertheless, even 1/3 of the infected children will develop complications postnatally, of which choroiditis and retinitis are the most common.[19]

In Poland, according to the epidemiological data, the mean annual incidence figure for congenital toxoplasmosis over the years 2007–2021 was 2.6 per 10,000 live births.[20]

Toxoplasmosis in immunocompromised individuals

Immunocompromised individuals, including those with HIV-positive status and patients receiving immunosuppressive therapy, the course of the disease may be much more severe. The most common manifestation is the involvement of the central nervous system taking the form of toxoplasmic encephalitis, known to be rapidly progressive and potentially fatal.[21] *Toxoplasma gondii* has a pronounced tropism to a range of organs. Consequently, a disseminated toxoplasmosis may develop, characterized by cardiac, musculoskeletal, pulmonary, hepatic and renal involvement.[22]

4.2. Laboratory Diagnosis

Serologic testing

Serologic assays are used for the detection of specific antibodies, including Immunoglobulin M (IgM), G (IgG) and A (IgA) in the material obtained from the patient (blood serum, cerebrospinal fluid, amniotic fluid, and the material collected from the anterior chamber of the eye) Table 1.

Immunoglobulin M antibodies are the first to appear after about one week following the infection with *T. gondii*. Their level rises, reaching a peak value after 1–3 months. Subsequently, over the next 9 months, a gradual fall is observed. In some patients, IgM antibodies may be present for up to a few years. IgG antibodies, in turn, appear after approximately 2 weeks following the moment of infection, with the highest levels noted after 3 months. They may remain in the system, at low levels, even for a lifetime. As far as IgA antibodies are concerned, their peak values are observed later, as compared to IgM, and they are present for 3 to 4 months following the infection with the parasite.[23]

Table 1. Characteristics of tests used in serodiagnosis of toxoplasmosis.

Test	Patient Samples Antibody type test	Characteristic	Authors, reference number
Sabin-Feldman Dye test (SFDT)	IgG, IgM, IgA Serum	High sensitivity and specificity. Require a source of viable tachyzoites, and a high degree of technical expertise. Cannot accurately differentiate between acute or chronic infection. The test is not recommended in immunocompromised individuals as antibodies are produced in low levels and irregularly.	Sabin and Feldman.[33] Liu et al.[34]
Modified Agglutination Test (MAT)	IgG Serum	Relatively high specificity. Can detect IgG antibodies in acute infection, which is very useful in the diagnosis of toxoplasmosis in AIDS patients, especially for chronic and latent infections.	Liu et al.[34]
Enzyme-Linked Immunosorbent Assays (ELISA)	IgG, IgM, IgA Serum	Routine screening for infections because it is highly sensitive (allowing quantitative and semi-quantitative antibody measurements).	Li et al.[35]
Immunosorbent Agglutination Assay (ISAGA)	IgM Serum	Highly sensitive and specific. Suitable for the diagnosis of acute toxoplasmosis in immunocompetent patients and as a screening test for recent infection in pregnant women. Owing to its high sensitivity and the fact that IgM and IgA antibodies do not cross the placenta, the method is a valuable tool in assisting the diagnosis of congenital toxoplasmosis in infants and detection of its reactivation.	Desmonts et al.[36]
Indirect Fluorescent Antibody Test (IFAT)	IgG, IgM Serum	This widely used test does not use live tachyzoites. It is characterized by sensitivity of 80.4–100 % and specificity of 91.4–95.8 %. A fluorescent microscope is required for reading the results. cross-reactivity with rheumatoid factor and anti-nuclear antibodies may occur.	Arthur and Blewett.[37]
Indirect Haemagglutination Assays (IHA)	IgG Serum	With a sensitivity of 100 % and specificity of 98.5 %. It is recommended for screening tests. The detection of acute or congenital toxoplasmosis can pose a problem.	Eissa et al.[38]

IgG(–) and IgM(–). The absence of antibodies excludes the possibility of a recent infection. In such a case, all hygiene and sanitary recommendations should be observed. Pregnant women should undergo routine screening as well.[23,24]

IgG(+) and IgM(–). The presence of IgG antibodies provides evidence of infection. In pregnant women, the antibody avidity should be determined. Antibody avidity tests can differentiate between IgG antibodies of low avidity, which are produced in the initial period of the invasion, from those with a high avidity index, strongly binding to antigen and produced at the advanced, inactive stage of the invasion. The diagnosis of an early stage of *T. gondii* infection is based on demonstrating seroconversion or a significant rise in the specific IgG antibody concentration characterized by low avidity, with a simultaneous presence of positive values of IgA and IgM antibodies. According to the standard recommendation, another serologic test is performed 3 weeks later. In the case of high IgG avidity, either a new infection or reactivation is suspected. There is no need for a serologic test if such as situation occurs in an immunocompetent person. However, if IgG avidity is too low or inconclusive in a pregnant woman, the time of *T. gondii* acquisition cannot be determined and the appropriate treatment must be instituted.[23,25]

IgG(–) and IgM(+) Detection of IgM and absence of IgG are suggestive of a recent infection. Nevertheless, the presence of IgG antibodies are of key importance to confirm the infection and there is a need for a repeat test after 2 weeks.[23]

IgG(+) and IgM(+) There is a possibility of a recent infection within the last few months or of a false positive IgM reaction. If the patient is pregnant and is IgG/IgM positive, the IgG avidity test must be performed.[23]

Molecular detection

The gold standard to diagnose congenital toxoplasmosis is molecular detection, particularly when serologic tests fail to yield conclusive results. Moreover, this method is recommended to diagnose toxoplasmosis of the CNS in immunocompromised individuals. Molecular testing allows for the detection of the parasite not only in the serum, but also in the amniotic fluid, blood, cerebral and eye tissue or urine, which largely facilitates confirmation or exclusion of the infection in infants.[26,27]

It must be said that molecular tests used routinely are not standardized and their sensitivity ranges between 65% and 100%.[28–30]

4.3. Prevention steps against toxoplasmosis

Preventive measures against acquired toxoplasmosis include:

- Avoiding the consumption and tasting of raw, undercooked or cured meat (cysts in meat undergo inactivation at the temperature of 67°C (up to 3 minutes) or –12°C for 2 days).[31,32]
- Washing hands with soap and water thoroughly after handling raw meat. Likewise, all the utensils coming in contact with meat, including knives, chopping boards, working surfaces etc. should be thoroughly washed.
- Avoiding drinking of unboiled or untreated water. The protozoan is not killed with chlorine in the process of water treatment.
- Avoiding the consumption of raw or undercooked oysters and mussels.
- Washing fruit and vegetables thoroughly before eating.
- Using gloves while working in the field or in the garden.
- If cats are kept, the litter box should be cleaned regularly.
- Washing hands with soap and water after contact with cat feces.
- Never feeding cats with raw or undercooked meat.
- Testing antibody levels in pregnant women who were seronegative for toxoplasmosis
- Securing sandboxes against cats.
- Screening for toxoplasmosis while running educational campaigns.[2]

5. CONCLUSIONS

(1) Toxoplasmosis is a parasitic infection most commonly characterised by a mild clinical course, which may be more severe in immunocompromised individuals as well as in infants with the congenital form of the disease.
(2) Prior to planned pregnancy serologic tests should be performed in order to confirm/exclude the infection.
(3) With a view to preventing new cases of toxoplasmosis, health education measures should be taken and implemented across a range of age groups, with a special focus given to the main risk factors for the infection.

Conflict of interest

None declared.

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