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Toxoplasmosis, a zoonotic infection; A critical and updated analysis: A Review Article

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Abstract

Toxoplasmosis is a parasitic illness caused by *Toxoplasma gondii*, an obligate intracellular protozoan. It may infect humans and pets, as well as other warm-blooded mammals. Toxoplasmosis can be silent in the general public but it can be deadly in immunocompromised patients. This neurotropic protozoan has been linked to an increased risk of behavioral changes in infected animals and mental illnesses in human beings. The detection of a biological pathogen linked to schizophrenia is critical to comprehend the pathogen's biological influence on affected cases. Moreover, in schizophrenia cases, toxoplasmosis infection and lipid profile disturbance are frequent. The exact underlying pathology is yet unknown. Pregnant women are a particular risk group; they are liable to a higher risk of infection, leading to miscarriage, stillbirth, or permanent impairments in the unborn child. This review aimed to explore the up-to-date knowledge on this zoonotic parasite and critically analyze current information, inconsistencies, and implications on public health. We discuss the pathological pathways by which it produces its devastating effect on the central nervous system in both animals and human models, in addition to its enigmatic impact on lipid profile.

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Introduction

Toxoplasmosis, a disease caused by an intracellular parasite, *Tosoplasma gondii* is manipulating humans and animals with extensive distribution worldwide prevalent in a warm and moist environment (1,2). *Tosoplasma gondii* prevails in three forms: oocysts, tachyzoites, and bradyzoites. The life cycle is complex and includes two stages; the asexual cycle exists in any warm-blooded animal, like daogs, birds, pigs, in addition to humans (3). The sexual part started when the definitive host) final host; the cat) ingested the tissue cyst. The cyst stayed in the epithelial cells lining the small intestine (4). Then, the oocytes are excreted by the faeces of the cat and end the sexual part of the cycle. These oocytes can survive for more than one year (5). As for the prospective host, the human becomes infected following ingestion of undercooked meat contained the bradyzoites, which transforms into the sporozoites inside the body by the effect of CO_2, enzymes and bile salts to settle in macrophages of the small intestine. Later it will leave intestine toward the endothelial cells of blood vessels and multiplication asexually there to produce the first generation of schizonts and merozoites. The later invade the endothelial cells of capillaries to produce the second generation of schizonts and merozoites at this time due to body immunity increase the parasite encysted as a bradyzoites among the muscles' fibers. Finally, sporozoites transformed into motile tachyzoites spread in the different parts of the body. Tachyzoites have rapid multiplication leading to the spreading of infection alongside tissue destruction (6,7). *Tosoplasma gondii* life cycle is highlighted in Figure 1.

Moreover, it can cause fatal infection in pregnant women; it settles in the muscle tissue and the central nervous system and is transformed into bradyzoites or the tissue cyst (8). The diagnosis of toxoplasmosis can be direct by observing the *T. gondii* in the stained tissue, cerebrospinal

fluid, blood or tissue biopsy. Today owing to the difficulties of performing these specimens, they are seldom used (9). Serological testing is the typical and reliable method to diagnose toxoplasmosis since the most ordinary form of infection is latent (10). *Tosoplasma gondii* causes intense and persistent humoral immune reaction associated with measurable antibody titers, regardless of clinical features among infected persons (11). The first detectable antibody one week after illness is the IgM, the earliest and most sensitive diagnostic marker in acute infection; however, it can be present for months or even one year (12). On the other hand, IgG could be detected one to two weeks after infection and reach a peak during the first one to two months, then decline later; thus, it is the standard test for chronic infection and continues as a residual titer long life. Other modalities of antibodies detection include the standard Sabin-Feldman Dye Test, Agglutination, Immunochromatographic, and Western Blotting tests (13).

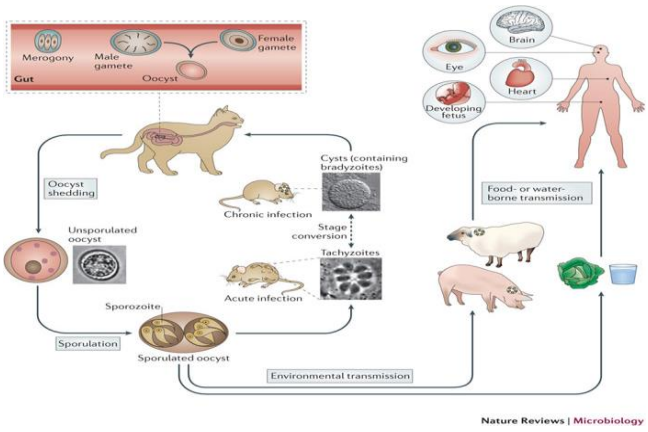

Figure1. *Tosoplasma gondii* life cycle (2).

Toxoplasmosis infection and mental illness

"Is insanity related to a microbial infection?" was a published editorial report in 1896 that started the debates on the infectious role in insanity pathogenesis (14). Emerging studies associated toxoplasmosis *Tosoplasma gondii* exposure with a wide range of mental illnesses, including autism, bipolar disorder, generalized anxiety disorder, pregnancy-related depression, and schizophrenia (15-17). Schizophrenia is a chronic disabling mental illness that affects how patients think, behave, display emotions, interpret reality, and interact with others (18).

Xu F *et al.* (19), conducted a retrospective cross-sectional study of confirmed schizophrenic cases and healthy controls. Their study highlighted that anti-toxoplasmosis serum IgG was significantly higher in cases than healthy controls (18% vs 7.4%, P-value 0.0001, OR 2.77). Earlier research confirmed the same results among patients presented with the first attack of schizophrenia (20); moreover, their findings showed that the intensity of txoplasmosis infection was linked to several clinical signs of schizophrenia, such as aggression, poor attention, and stereotyped behavior, the neurocognitive consequences behind *T. gondii* infection in the brain is a subject of several theoretical theories.

Toxoplasmosis impact on the onset of mental disease is accredited to prasite-induced biochemical mechanisms. Chronic *T. gondii* infection alters brain neurobiology, namely -aminobutyric acid synaptic and signal transduction. However, other researchers postulated the immunological role of *T. gondii* infection. Higher amounts of proinflammatory cytokines, particularly IL-6, was significantly elevated among schizophrenic cases, raised inflammatory cytokines are the biological hallmark of schizophrenia, support this hypothesis (21-23).

During pregnancy, *T. gondii* parasite targets the growing fetal brain and causes congenital disabilities. Brown *et al.* (24) conducted a retrospective study that utilized maternal IgG serum levels to see if toxoplasmosis exposure during pregnancy is linked to a higher incidence of schizophrenia and associated disorders in adult children. A positive correlation was confirmed for those patients with a high category of IgG antibody titer, as for cases with moderate or minimal increase of the IgG titer, no meaningful correlation was scored, thus, it may be conceivable that the findings were due to a change in the mother immunological state (24,25). Interestingly, Brown *et al.* (25) study included normal newborn babies upon delivery; no signs of congenital toxoplasmosis were demonstrated, which implies that toxoplasmosis infection produces comparable effects to those caused by rubella, influenza, and cytomegalovirus, and other infections linked to schizophrenia (26).

Toxoplasmosis infection and behavioral changes in animal

The effects of latent *T. gondii* infection on the central nervous system have been reviewed, a growing body of evidence supports neurologic and behavioral changes in animal models infected with latent toxoplasmosis, the infected rodents showed reduced learning, poor performance, and poor memory task achievements surprisingly, no cysts were identified in the hippocampi of afflicted animals, but they were located throughout the brain, suggesting that alterations in neurophysiology were to blame for the animal behavioral abnormalities (27).

Joanne *et al.* (28) have reported similar behavioral changes in mice and cats; the author interprets these changes as part of the invading neurotrophic parasite's manipulative function in completing its life cycle. Rodents' altered behavior makes them more vulnerable to predation by cats, the parasite's ultimate host. Later research has revealed that starting therapy can reverse *T. gondii* behavioral alterations. This discovery provides new avenues for the management of human schizophrenia.

Lipid profile

In confirmed cases, both txoplasmosis infection and a lipid profile disturbance are prevalent; however, the underlying pathophysiological processes are unidentified; therefore, exploring the impact of chronic infection is crucial to get insight into the development of target therapeutic medicines (29). In their study on schizophrenic patients, Xu *et al.* (19) discussed that high-density lipoprotein and serum triglycerides are significantly elevated in seropositive cases (P=0.000), cholesterol, on the other hand, indicated the opposite tendency, the strong connection between confirmed clinical cases emphasized these findings even more (OR=0.62, 95%CI=0.51-0.748, P=0.000).

Xu *et al.* (19) accredited these changes to the inability of *T. gondii* to produce cholesterol on its own; thus, it needs the host endocytosed low-density lipoprotein for cholesterol production; a key component of the parasitophorous vacuoles, because of increased absorption, *T. gondii* infection decreases blood cholesterol concentration in both infected humans and animals (19,30,31). Furthermore, gender differences in *T. gondii* infection were further highlighted in Sagud *et al.* (21) work that revealed higher blood concentrations of total cholesterol levels and low-density lipoprotein in *T. gondii* infected males. However, no meaningful changes were reported in schizophrenic female patients regarding lipid indicators.

Interestingly, Stürchler study investigated total cholesterol concentrations of serums, cord blood between seropositive and seronegative pregnant women; (280 vs. 279) respectively for cases and healthy controls. The difference was statistically insignificant (32). It is established that pregnancy is a state of immunological tolerance to the growing in utero fetus, which may modify the maternal response to infection; this may explain the lower total cholesterol in newborns (33). What backs up this explanation is the Marchioro results which showed that pregnant women with acute or chronic toxoplasmosis show less concentration of peripheral TNF-α. The latter is a proinflammatory cytokine, which is ideally increased in adult *T. gondii* infection (34).

Toxoplasmosis trend in pregnancy

The infection can have a devastating effect on the growing fetus, resulting in abortion, stillbirth, brain calcification, ventriculomegaly, premature labor, and death. The infection can be transmitted to the fetus by transplacental passage during a primary infection in the mother, or it can be existing before conception in rare cases (35-37).

The probability of transmission is 30%, and it is influenced by the gestational age and whether or not chemoprophylaxis is administered to pregnant women. The transmission rate is highest in the third trimester, with 70%, 46%, and 13% at 36, 26, and 13 weeks of pregnancy, respectively. The sequence of infection, on the other hand, is the least in the third trimester (38).

According to some studies, if therapy is begun as soon as possible, the chance of transmission is reduced by up to 75%. To capture this window of opportunity, comprehensive screening for maternal seroconversion is required. Confirming toxoplasmosis infection in the mother is a Delma. Although clinically speaking, the signs of illness are minor and mimic a flu-like sickness, 70% of people are clinically asymptomatic. As a result, establishing primary infection is dependent on maternal seroconversion from -ve to +ve IgG antibody, whereas IgM is a poor indicator of acute illness and can remain for years. The IgG avidity assay measures antibody binding strength *to T gondii*; low avidity indicates acute infection, whereas high avidity indicates chronic infection (39).

The avidity and stability of a high IgG titer over one-month rule out recent infection. Because of the catastrophic consequence of fetal toxoplasmosis, parents must be counselled about the significance of positive and negative testing since prevention is essential (40). It's vital to confirm the primary maternal infection to assess fetal transmission risk, give appropriate counselling, and begin antibiotics. Countries with high prevalence, like France, have implemented prenatal screening programs that have reduced the incidence by more than half (41). Olaru *et al.* (42). assessed the importance of starting therapy during pregnancy by comparing the newborn outcomes of infected women treated to those who were not. His findings revealed that treated mothers' babies had significantly lowered rates of ocular complications (38% vs 62%) and hydrocephalus (67% vs 92%). Thus, even in countries where routine screening and treatment are not consistently followed, the author recommends anti-parasitic therapy for verified cases. As the use of the medication throughout pregnancy resulted in improved clinical results. In summary, many zoonotic diseases exist (43-47); Toxoplasmosis still has many layers still to be unraveled.

Conclusion

In conclusion, this review confirms a link between persistent *T. gondii* infection and schizophrenia. As a result, additional pathogens' co-infection should be considered. We also speculated on the possibility of a strong link between *T. gondii* infection and serum lipid modification in schizophrenia patients. a critical problem to be addressed in the treatment intervention for schizophrenia patients' lipid levels. Aiming to reduce vertical transmission and the incidence of severe neonatal toxoplasmosis, we strongly recommend adopting *T. gondii* screening programs in expectant mothers and treatment for women who contract toxoplasmosis during pregnancy.

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Conflict of interest

The authors declare that have no conflict of interests regarding the publication of this manuscript.

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داء المقوسات، عدوى حيوانية المنشأ؛ تحليل نقدي محدث: دراسة مراجعة

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الخلاصة

داء المُقَوَّسَات هو مرض طفيلي تسببه المقوسة الكوندية، وهو طفيلي لا يستطيع العيش إلا داخل الخلايا. قد يصيب البشر والحيوانات الأليفة، وكذلك الثدييات ذوات الدم الحار. يمكن أن يكون داء المقوسات صامتا في عامة الناس، ولكنه قد يكون مميتا في المرضى الذين يعانون من نقص المناعة. تم ربط هذا البروتوزوان الموجه للأعصاب بزيادة مخاطر التغيرات السلوكية في الحيوانات المصابة والأمراض العقلية لدى البشر. يعد اكتشاف العامل البيولوجي المرتبط بالفصام أمرا بالغ الأهمية لفهم التأثير البيولوجي لمسببات المرض على الحالات المصابة. علاوة على ذلك، في حالات الفصام، تكون عدوى داء المقوسات واضطراب الدهون أمرا متكررا. إن سبب هذا الترابط والعلاقة بين الاثنين غامض وغير معروف حتى الآن. تعتبر النساء الحوامل فئة معرضة بشكل خاص؛ هم عرضة لخطر أكبر للإصابة بالعدوى، مما يؤدي إلى الإجهاض أو ولادة جنين ميت أو ضعف دائم في الجنين. يهدف هذا المقال إلى استكشاف المعرفة الحديثة حول هذا الطفيلي الحيواني المنشأ والتحليل النقدي للمعلومات الحالية والتناقضات والآثار المترتبة على الإصابة على الصحة العامة. نناقش المسارات المرضية التي ينتج من خلالها تأثيره المدمر على الجهاز العصبي المركزي في الحيوان والنموذج البشري، بالإضافة إلى تأثيره الغامض على ملف الدهون.