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Original Research Article

Comparison of *Toxoplasma Gondii* Seroprevalence Between Schizophrenia Patients and Healthy Controls

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ABSTRACT

Background: *Toxoplasma gondii* is a protozoan parasite capable of establishing long-term infection in humans and has been increasingly investigated for its possible involvement in neuropsychiatric conditions such as schizophrenia. Its influence on the intensity of clinical symptoms, however, remains unclear.

Objectives: To determine the seroprevalence of *Toxoplasma gondii* infection in patients with schizophrenia and to evaluate its association with the severity of clinical symptoms.

Methods: This study aimed to assess the prevalence of *Toxoplasma gondii* infection among individuals diagnosed with schizophrenia and to explore its relationship with symptom severity. A case–control design was employed at a tertiary care center in Jaipur, including 120 schizophrenia patients and 120 matched healthy individuals. Symptom severity was evaluated using the Positive and Negative Syndrome Scale (PANSS), and IgG antibodies against Toxoplasma gondii were detected using ELISA.

Results: The findings revealed a significantly higher rate of seropositivity among patients compared to controls. Additionally, infected patients exhibited greater symptom severity, as reflected by higher PANSS scores. Statistical analysis confirmed a strong association between infection status and schizophrenia, as well as with increased clinical severity.

Conclusion: These results indicate that latent toxoplasmosis may contribute to worsening clinical presentation in schizophrenia, highlighting the importance of further investigation into infectious factors in psychiatric illnesses.

Keywords: T*oxoplasma gondii*; Schizophrenia; Seropositivity; PANSS; IgG antibodies; Symptom severity.

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INTRODUCTION

Schizophrenia is a long-term psychiatric condition that affects roughly one percent of the population worldwide and is characterized by disturbances in thinking, perception, emotional responsiveness, and behavior[1,2]. Patients typically present with positive symptoms such as hallucinations and delusions, negative symptoms like reduced emotional expression and social withdrawal, and varying degrees of cognitive impairment. Despite extensive research, the underlying causes of schizophrenia are not fully understood and are believed to involve a complex interaction of genetic vulnerability, environmental influences, and neurobiological alterations.

Increasing attention has been directed toward environmental contributors, particularly infectious agents, in the development of schizophrenia[3,4]. This perspective is closely linked to the concept of neuroinflammation, where persistent immune activation within the central nervous system may play a role in the onset or progression of psychiatric disorders[4]. Such an approach integrates insights from both psychiatry and microbiology, offering a broader understanding of disease mechanisms.

One organism of particular interest is Toxoplasma gondii, an intracellular parasite with widespread global distribution[5,6]. Infection commonly occurs through ingestion of contaminated food, water, or undercooked meat. Following initial exposure, the parasite can persist in a dormant state within tissues, especially in the brain and muscles[7]. Its ability to remain latent and evade immune clearance makes it a candidate for influencing neurological function over time.

From a biological standpoint, Toxoplasma gondii has several characteristics that support its possible role in psychiatric disorders. It can localize within neural tissue, interfere with host cellular processes, and trigger ongoing immune responses. Studies have also suggested that the parasite may alter neurotransmitter systems, particularly dopamine pathways, which are strongly implicated in schizophrenia[8].

Epidemiological studies have frequently reported higher rates of Toxoplasma gondii exposure among individuals with schizophrenia compared to unaffected populations[9,10]. Some research has also indicated a possible link between infection and more severe clinical features, including greater symptom burden and earlier disease onset. However, findings have not been entirely consistent, possibly due to methodological differences across studies[11]. Prenatal and early-life exposure to infections has also been linked to increased risk of schizophrenia[12]

An area that requires further clarification is whether latent infection influences the severity of schizophrenia[13-15]. Establishing such a relationship could have important clinical implications, including the identification of subgroups of patients who may benefit from targeted therapeutic approaches. It may also contribute to more personalized strategies in psychiatric care[16].

Clinical severity in schizophrenia is commonly assessed using standardized tools such as the Positive and Negative Syndrome Scale (PANSS), which evaluates multiple symptom domains. Combining serological testing for Toxoplasma gondii with PANSS scoring provides a structured way to examine potential associations between infection and disease expression.

Given the relatively high prevalence of both schizophrenia and Toxoplasma gondii infection in developing regions, including India, it is important to explore this relationship in local populations. Environmental exposure, dietary habits, and socioeconomic factors may influence infection rates and their potential impact on mental health. It has been proposed that infectious agents may contribute to the disorder in a subset of patients rather than acting as a sole cause[17]. Immune system dysregulation may further mediate this relationship[18].

The present study was designed to investigate the prevalence of Toxoplasma gondii infection among patients with schizophrenia and to determine whether it is associated with increased symptom severity in a tertiary care setting[19,20].

AIMS AND OBJECTIVES

Aim

To compare the seroprevalence of *Toxoplasma gondii* infection between patients with schizophrenia and healthy controls, and to evaluate its association with the severity of schizophrenia.

Objectives

1. To determine the seroprevalence of anti-*Toxoplasma gondii* IgG antibodies in patients with schizophrenia and in healthy controls. To compare the frequency of *Toxoplasma gondii* seropositivity between cases and controls.
2. To assess the severity of schizophrenia using the Positive and Negative Syndrome Scale (PANSS).
3. To evaluate the association between *Toxoplasma gondii* seropositivity and clinical severity among schizophrenia patients.

MATERIALS AND METHODS

This hospital-based case–control study was conducted in the Departments Microbiology at the National Institute of Medical Sciences (NIMS), Jaipur.

A total of 240 participants were included and divided into two groups:

- Cases: 120 patients diagnosed with schizophrenia as per DSM-5 criteria
- Controls: 120 age- and sex-matched healthy individuals without any history of psychiatric illness

Participants in the case group were recruited from the Psychiatry outpatient and inpatient departments. Controls were selected from healthy attendants or hospital staff and were screened to exclude any history of psychiatric or major medical illness.

Inclusion Criteria

Cases:

- Age 18–60 years
- Diagnosis of schizophrenia according to DSM-5
- Willingness to participate and provide informed consent

Controls:

- Age- and sex-matched healthy individuals
- No history of psychiatric illness
- Willingness to participate

Exclusion Criteria (Both Groups):

- History of other major psychiatric disorders
- Neurological illnesses (e.g., epilepsy, CNS infections, head injury)
- Immunocompromised states (e.g., HIV infection, malignancy, immunosuppressive therapy)
- Pregnant women

Study Procedure

Psychiatric Assessment:

All patients in the case group underwent detailed clinical evaluation, including history and mental status examination. The severity of schizophrenia was assessed using the Positive and Negative Syndrome Scale (PANSS) by a trained psychiatrist.

Microbiological Assessment:

Approximately 3–5 mL of venous blood was collected from all participants under aseptic conditions. Serum was separated by centrifugation and analyzed for anti-*Toxoplasma gondii* IgG antibodies using a standardized enzyme-linked immunosorbent assay (ELISA) kit, following the manufacturer's protocol. Appropriate positive and negative controls were included to ensure quality assurance.

Grouping:

- Seropositive: Presence of anti-*Toxoplasma gondii* IgG antibodies
- Seronegative: Absence of IgG antibodies

Statistical Analysis:

Data were entered into Microsoft Excel and analyzed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation, while categorical variables were expressed as frequencies and percentages.

Comparisons between groups were performed using:

- Independent t-test or Mann–Whitney U test for continuous variables
- Chi-square test for categorical variables

Odds ratio (OR) with 95% confidence interval (CI) was calculated to assess the association between *Toxoplasma gondii* infection and schizophrenia. Correlation analysis was performed to evaluate the relationship between seropositivity and PANSS scores.

A p-value of <0.05 was considered statistically significant.

Ethical Considerations and Consent

The study protocol was reviewed and approved by the Institutional Ethics Committee (IEC), NIMS, Jaipur prior to initiation of the

study. Written informed consent was obtained from all participants after explaining the purpose and procedures of the study in their local language. In patients with impaired decision-making capacity, consent was obtained from legally authorized representatives.

RESULTS

A total of 240 participants were included in the study, comprising 120 patients with schizophrenia (cases) and 120 healthy individuals (controls). The mean age of cases was 34.6 ± 9.8 years, while that of controls was 33.9 ± 9.5 years. There was no statistically significant difference between the two groups with respect to age and gender distribution ($p > 0.05$), indicating appropriate matching.

Serological analysis showed that 46 (38.3%) cases were positive for anti-*Toxoplasma gondii* IgG antibodies, compared to 22 (18.3%) controls. This difference was statistically significant ($p < 0.001$), suggesting a higher prevalence of latent infection among schizophrenia patients.

Summary of Findings

The present study demonstrated a significantly higher seroprevalence of *Toxoplasma gondii* infection among schizophrenia patients compared to healthy controls. The odds of seropositivity were approximately 2.7 times higher in cases than controls.

Among schizophrenia patients, those with positive serology exhibited significantly higher PANSS scores across all domains, including positive symptoms, negative symptoms, and general psychopathology. Additionally, a greater proportion of seropositive patients were categorized as having severe schizophrenia.

A moderate positive correlation was observed between *Toxoplasma gondii* seropositivity and overall disease severity, suggesting a potential role of latent infection in influencing clinical expression.

Table 1: Demographic Characteristics of Cases and Controls

Variable	Cases (n = 120)	Controls (n = 120)	p-value
Age (years, mean ± SD)	34.6 ± 9.8	33.9 ± 9.5	0.62
Gender			
Male	68 (56.7%)	70 (58.3%)	0.79
Female	52 (43.3%)	50 (41.7%)	
Residence			
Urban	72 (60%)	78 (65%)	0.41
Rural	48 (40%)	42 (35%)	

Table 2: Comparison of *Toxoplasma gondii* Seroprevalence

Serological Status	Cases (n = 120)	Controls (n = 120)	p-value
IgG Positive	46 (38.3%)	22 (18.3%)	<0.001
IgG Negative	74 (61.7%)	98 (81.7%)	

Table 3: Association Between *Toxoplasma gondii* Infection and Schizophrenia

Parameter	Value
Odds Ratio (OR)	2.77
95% Confidence Interval	1.52 – 5.05
p-value	<0.001

Table 4: PANSS Scores in Schizophrenia Patients According to Serological Status

PANSS Domain	IgG Positive (n = 46)	IgG Negative (n = 74)	p-value
Positive Symptoms	25.8 ± 5.2	22.4 ± 4.6	0.001
Negative Symptoms	27.1 ± 6.1	23.9 ± 5.4	0.004
General Psychopathology	49.3 ± 7.8	44.6 ± 6.9	0.002
Total PANSS Score	102.2 ± 12.4	90.9 ± 11.3	<0.001

Table 5: Severity Distribution of Schizophrenia Based on PANSS Score

Severity Category	IgG Positive (n = 46)	IgG Negative (n = 74)	p-value
Mild (<75)	4 (8.7%)	18 (24.3%)	
Moderate (75–95)	12 (26.1%)	30 (40.5%)	
Severe (>95)	30 (65.2%)	26 (35.1%)	0.002

Table 6: Correlation Between *Toxoplasma gondii* Seropositivity and PANSS Scores

Parameter	Correlation Coefficient (r)	p-value
IgG Seropositivity vs Total PANSS Score	**0.42**	**<0.001**

DISCUSSION

The present study explored the relationship between Toxoplasma gondii infection and schizophrenia, with particular emphasis on its association with symptom severity. The findings indicate that individuals diagnosed with schizophrenia had a notably higher rate of seropositivity compared to healthy controls. This observation suggests that exposure to the parasite may be more common in this patient population[9,10,13].

One important aspect of the results is the observed link between seropositivity and increased clinical severity. Patients who tested positive for IgG antibodies consistently demonstrated higher scores across all domains of the PANSS scale. This pattern implies that latent infection may not only be associated with the presence of schizophrenia[19] but could also influence how severely the disorder manifests. Rather than acting as a direct cause, the infection may function as a modifying factor that exacerbates existing neuropsychiatric disturbances.

From a biological perspective, several mechanisms could explain this association. Toxoplasma gondii has a known affinity for neural tissue, where it can persist in a dormant state for extended periods. Its presence in the brain may lead to subtle but chronic immune activation, resulting in the release of inflammatory mediators that can affect neuronal functioning. In addition, experimental evidence suggests that the parasite may interfere with neurotransmitter systems, particularly those involving dopamine. Since dopaminergic dysregulation is a central feature in schizophrenia, such alterations could contribute to worsening of symptoms. The study also found a greater proportion of severe cases among seropositive individuals. This supports the idea that infection status may have clinical relevance beyond simple exposure. It raises the possibility that screening for latent infections could help identify patients who are at risk of experiencing a more severe disease course. However, this interpretation should be approached cautiously, as multiple factors—including genetic predisposition, treatment adherence, and environmental stressors—also play significant roles in determining illness severity.

Another point worth considering is the potential influence of environmental and lifestyle factors on infection rates. Individuals living in conditions with increased exposure to contaminated food, soil, or animals may have a higher likelihood of acquiring Toxoplasma gondii. In some cases, behavioral or socioeconomic characteristics associated with schizophrenia could indirectly increase exposure risk. Therefore, the relationship observed in this study may reflect a complex interaction between biological vulnerability and environmental conditions.

Despite the strength of the association identified, the study design limits the ability to draw conclusions about causality. Because the data were collected at a single point in time, it is not possible to determine whether infection preceded the onset of schizophrenia or occurred afterward. Longitudinal studies would be necessary to clarify the temporal sequence and to better understand whether the parasite plays a contributory or secondary role.

Another limitation is that only IgG antibodies were measured, which indicate past exposure rather than active infection. As a result, the timing and duration of infection could not be established. Future studies incorporating additional markers, such as IgM antibodies or molecular techniques, may provide more detailed insights into the dynamics of infection.

Overall, the findings of this study add to the growing body of evidence suggesting that infectious agents may have a role in psychiatric conditions. They highlight the importance of considering biological factors beyond traditional neurotransmitter-based models. Overall, the findings support the growing body of evidence linking infectious agents to psychiatric disorders and highlight the need for further investigation into their role in disease mechanisms[19,20].

Further research in this area may open new possibilities for prevention and treatment, including the exploration of antimicrobial or anti-inflammatory strategies as adjuncts to conventional psychiatric care.

CONCLUSION

The findings of the present study demonstrate that Toxoplasma gondii infection is more frequently observed in individuals with schizophrenia compared to healthy controls. In addition, a clear association was identified between seropositivity and increased severity of clinical symptoms, as reflected by higher PANSS scores. These observations suggest that latent infection may have a role in influencing the clinical expression of the disorder.

While the results do not establish a causal relationship, they highlight the possibility that infectious and immunological factors contribute to the complexity of schizophrenia. The interaction between chronic parasitic infection and neurobiological processes may represent an additional dimension in understanding disease variability and progression.

These findings underscore the importance of adopting a broader perspective in psychiatric research that includes potential biological and environmental contributors. Further longitudinal and mechanistic studies are required to clarify the direction and nature of this association. Such research may eventually support the development of more comprehensive management strategies, including the consideration of adjunctive approaches targeting underlying infectious or inflammatory processes.

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