(e) ISSN Online: 2321-9599

(p) ISSN Print: 2348-6805

ORIGINAL ARTICLE

Assessment of incidence of Toxoplasma gondii infection in schizophrenia patients

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ABSTRACT:
Background: Schizophrenia is a multifaceted and enduring mental health condition marked by a diverse range of symptoms, which include delusions, hallucinations, disorganized speech or behavior, and cognitive impairments. Toxoplasma gondii, an intracellular protozoan parasite, can infect humans through three primary routes: the ingestion of tissue cysts, the consumption of oocysts, or through congenital transmission involving tachyzoites. Hence; the present study was conducted for assessing incidence of Toxoplasma gondii infection in schizophrenia patients. **Materials & methods:** A total of 100 patients with confirmed diagnosis of schizophrenia were enrolled. Complete demographic details of all the patients were recorded. A diagnosis of schizophrenia was reached through an assessment of patient-specific signs and symptoms, as described in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5).Blood samples were obtained from all patients. Serum component was separated by centrifugation. Serum samples were analyzed by qualitative and quantitative methods for T. gondii IgG antibodies with the commercially available enzyme immunoassay kit "Toxoplasma IgG". All the results were recorded in Microsoft excel sheet and were subjected to statistical analysis using SPSS software. **Results:** A total of 100 patients were evaluated. Mean age of the patients was 49.2 years. There were 61 males and 39 females. Among these 100 subjects, Toxoplasma gondii infection was seen in 36 percent of the patients. Out of 61 males, Toxoplasma gondii infection was seen in 25 subjects while among 39 females, Toxoplasma gondii infection was seen in 11 subjects. majority proportion of subjects with Toxoplasma gondii infection were of rural residence, of lower socio-economic status and were labourers (p-value < 0.05). **Conclusion:** The correlation observed between T. gondii seropositivity and schizophrenia in this study might suggest a direct causal link between the infection and the onset of schizophrenia.
Key words: Toxoplasma gondii, Schizophrenia

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This article may be cited as: Saha G, Shah AA. Assessment of incidence of Toxoplasma gondii infection in schizophrenia patients. J Adv Med Dent Scie Res 2017;5(2):253-256.

INTRODUCTION

Schizophrenia is a multifaceted and enduring mental health condition marked by a diverse range of symptoms, which include delusions, hallucinations, disorganized speech or behavior, and cognitive impairments. The condition typically manifests early in life and follows a chronic trajectory, rendering it a debilitating disorder for numerous individuals and their families.[1] Disability in this context often arises from both negative symptoms, which are characterized by deficits or losses, and cognitive symptoms that affect attention, working memory, and executive functioning. Furthermore, the presence of positive symptoms, such as delusions, hallucinations, and suspiciousness, can lead to episodes of relapse. The significant variability observed in schizophrenia has contributed to ongoing debates regarding its diagnostic criteria, underlying causes, and pathophysiological mechanisms.[2, 3]

Toxoplasma gondii, an intracellular protozoan parasite, can infect humans through three primary routes: the ingestion of tissue cysts, the consumption of oocysts, or through congenital transmission involving tachyzoites. Following the acute phase, during which tachyzoites proliferate in various organs, the parasite preferentially forms cysts in the brain, leading to a chronic infection characterized by a dynamic equilibrium between the host's immune defenses and the parasite's strategies to evade these responses. Various types of brain cells, such as astrocytes and neurons, are susceptible to infection. In vitro investigations involving non-neuronal cells have revealed significant alterations in host cell gene expression due to the infection, affecting both immune response-promoting molecules and those related to signal transduction pathways, indicating that similar changes may occur in infected neural cells. Interferon-gamma plays a crucial role in mediating the immune response necessary to control T. gondii within the brain and to sustain the latency of chronic infection. Additionally, the infection triggers the release of numerous cytokines from microglia, astrocytes, and neurons, which can either enhance or inhibit inflammatory responses.[4- 6] Hence; the present study was conducted for assessing incidence of Toxoplasma gondii infection in schizophrenia patients.

MATERIALS & METHODS

The present study was conducted for assessing incidence of Toxoplasma gondii infection in schizophrenia patients. A total of 100 patients with confirmed diagnosis of schizophrenia were enrolled. Complete demographic details of all the patients were recorded. A diagnosis of schizophrenia was reached through an assessment of patient-specific signs and symptoms, as described in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5).[7] The DSM-5 states that "the diagnostic criteria [for schizophrenia] include the persistence of two or more of the following active-phase symptoms, each lasting for a significant portion of at least a one-month period: delusions, hallucinations, disorganized speech, grossly disorganized or catatonic behavior, and negative symptoms."At least one of the qualifying symptoms must be delusions, hallucinations, or disorganized speech.[7] Blood samples were obtained from all patients. Serum component was separated by centrifugation. Serum samples were analyzed by qualitative and quantitative methods for T. gondii IgG antibodies with the commercially available enzyme immunoassay kit "Toxoplasma IgG". All the results were recorded in Microsoft excel sheet and were subjected to statistical analysis using SPSS software. Chi-square test was used for evaluation of level of significance.

RESULTS

A total of 100 patients were evaluated. Mean age of the patients was 49.2 years. There were 61 males and 39 females. Among these 100 subjects, Toxoplasma gondii infection was seen in 36 percent of the patients. Out of 61 males, Toxoplasma gondii infection was seen in 25 subjects while among 39 females, Toxoplasma gondii infection was seen in 11 subjects. majority proportion of subjects with Toxoplasma gondii infection were of rural residence, of lower socio-economic status and were labourers (p-value < 0.05).

Table 1: Incidence of Toxoplasma gondii infection

Toxoplasma gondii infection	Number	Percentage
Present	36	36
Absent	64	64
Total	100	100

Table 2: Associate risk factors for Toxoplasma gondii infection

Variable	Toxoplasma gondii infection present	Toxoplasma gondii infection absent	p-value
Mean age	47.6	51.5	0.45
Males	25	36	0.25
Females	11	28	
Rural residence	25	31	0.001*
Urban residence	11	35	
Lower socio-economic status	28	30	0.021*
Labourers	23	12	

*: Significant

DISCUSSION

Schizophrenia is defined by a range of psychopathological features. The primary characteristics include positive symptoms, such as delusions and hallucinations—often referred to as psychotic symptoms that indicate a disconnection from reality—negative symptoms, which primarily manifest as diminished motivation, reduced spontaneous speech, and social withdrawal, and cognitive deficits. Collectively, individuals with schizophrenia exhibit poorer performance than control groups across various cognitive domains, although there is significant individual variability. Positive symptoms typically exhibit a pattern of relapse and remission, although some patients may endure persistent long-term psychotic symptoms. In contrast, negative and cognitive symptoms are generally chronic and have enduring impacts on social functioning. The initial episode of psychosis typically arises during late adolescence or early adulthood, often preceded by a prodromal phase or an "at risk mental state." In certain cases, premorbid cognitive and/or social impairments may be evident for many years prior to the onset of psychosis. Conversely, there are instances where the onset occurs abruptly in individuals who previously functioned well.[6-9]Toxoplasmosis, an infection resulting from the protozoan parasite Toxoplasma gondii, ranks among the most prevalent parasitic diseases affecting humans and various warm-blooded animals. This parasite has a global distribution, being identified in regions ranging from Alaska to Australia. Approximately one-third of the global population has encountered this organism. While the infection typically remains asymptomatic in healthy adults, it poses significant risks, including blindness and cognitive impairments in children infected in utero, as well as severe complications in individuals with weakened immune systems.[7-9]Hence; the present study was conducted

for assessing incidence of Toxoplasma gondii infection in schizophrenia patients.

A total of 100 patients were evaluated. Mean age of the patients was 49.2 years. There were 61 males and 39 females. Among these 100 subjects, Toxoplasma gondii infection was seen in 36 percent of the patients. Out of 61 males, Toxoplasma gondii infection was seen in 25 subjects while among 39 females, Toxoplasma gondii infection was seen in 11 subjects. majority proportion of subjects with Toxoplasma gondii infection were of rural residence, of lower socio-economic status and were labourers (p-value < 0.05). In the typical progression of toxoplasmosis, IgM antibodies specific to T. gondii are the earliest to appear in serum, typically within a few days post-infection. These antibodies generally become undetectable between weeks 4 and 12, although they may persist for several months or even years in a significant number of individuals. IgG antibodies emerge approximately two weeks after the detection of IgM, peaking between the second and third month, and they tend to remain at residual levels for the individual's lifetime. The presence of IgM antibodies, in the absence of IgG, suggests a recent infection, whereas the detection of IgG antibodies, particularly in the absence of IgM, indicates a chronic infection. Reactivation of a latent infection may lead to elevated levels of IgG and/or IgM; however, in immunocompromised patients, these antibodies may not be detectable.[11]The initial modern meta-analyses indicated that individuals who are Toxoplasma seropositive have an approximately 2.7-fold increased likelihood of developing schizophrenia. In contrast, more recent prevalence studies have frequently reported a reduction in the prevalence of toxoplasmosis among schizophrenia patients, rather than an increase. A recent meta-analytic investigation encompassing 43 case-control studies revealed an overall OR of 1.81, which further diminished to 1.43 after adequately addressing publication bias. It has been proposed that heightened awareness of patient rights may contribute to this observed decline. Consequently, only those patients who voluntarily provide informed consent—likely representing individuals with less severe manifestations of schizophrenia—are eligible for inclusion in contemporary research studies. Several investigations have indicated that patients with latent toxoplasmosis tend to exhibit more pronounced positive symptoms of schizophrenia.[12- 17]

CONCLUSION

The correlation observed between T. gondii seropositivity and schizophrenia in this study might suggest a direct causal link between the infection and the onset of schizophrenia. Hence; further studies are recommended for better exploration of results.

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