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ORIGINAL RESEARCH PAPER

Toxoplasma gondii infection of the central nervous system and suicide: A case-control study of decedents

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ABSTRACT

We sought to determine the association between *Toxoplasma gondii* (*T. gondii*) infection of the central nervous system and suicide in a sample of decedents in Mexico City. One hundred and forty-seven decedents (87 who committed suicide and 60 who did not commit suicide) were studied. Brain tissues (amygdala and prefrontal cortex) of decedents were examined for the detection of *T. gondii* using immunohistochemistry. Detection of *T. gondii* was positive in 7 (8.0%) of the 87 cases (6 found in prefrontal cortex and one in amygdala), and in one (1.7%) of the 60 controls (found in prefrontal cortex) (OR: 5.16; 95% CI: 0.61–43.10; $P = 0.14$). Results suggest that *T. gondii* infection in brain is not associated with suicide. Further studies to confirm this finding are needed.

KEYWORDS

Toxoplasma gondii, suicide, case-control study, immunohistochemistry, epidemiology

INTRODUCTION

Toxoplasma gondii (*T. gondii*) is an obligate intracellular protozoan [1]. This successful parasite cycles between definitive felid hosts and a broad range of intermediate hosts including humans [2]. Toxoplasmosis, the disease caused by *T. gondii*, is one of the most common parasitic infections of man and other warm-blooded animals [3]. It is estimated that 30% of the global human population is chronically infected by *T. gondii* [4]. In addition, *T. gondii* is a major problem for health economics in many countries [5]. Transmission of *T. gondii* usually occurs by ingestion of food or water that is contaminated with oocysts shed by cats or by eating undercooked or raw meat containing tissue cysts [6]. Primary

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infection with *T. gondii* during pregnancy may lead to congenital toxoplasmosis [7]. Transmission of *T. gondii* may also occurs by organ transplantation [8], or blood transfusion [9]. Toxoplasmosis has a wide spectrum of clinical outcomes [10]. Infection with *T. gondii* is generally asymptomatic but some patients experience lymphadenitis [11], ocular disease or, in immunocompromised patients, a life-threatening encephalitis [6]. *T. gondii* is able to persist in the central nervous system in a variety of hosts including humans [12]. After infection, *T. gondii* persists as intraneuronal cysts that are controlled, but not eliminated by the immune system [13]. This parasite can modify the structure and function of neurons leading to specific behavioral changes in the host [14]. Neuroinflammation associated with *T. gondii* infection may contribute to depression and suicidal behavior [15]. In murine models, researchers have shown that type 1 innate lymphoid cells regulate the onset of *T. gondii*-induced neuroinflammation [16], and the immunoproteasome subunits LMP2, LMP7 and MECL-1 are crucial along the induction of cerebral toxoplasmosis [17]. Infection with *T. gondii* has been associated with schizophrenia [18, 19], mixed anxiety and depressive disorder [20], generalized anxiety disorder, obsessive-compulsive disorder [21], and aggression and impulsivity [22]. Furthermore, seropositivity to *T. gondii* has been associated with suicidal behavior. Several studies have demonstrated a higher frequency of *T. gondii* exposure in suicide attempters than in controls [23–25]. However, other studies have reported no association between *T. gondii* exposure and suicide attempts [26–27]. These studies have searched for the link between *T. gondii* exposure and suicidal behavior in live persons, but the link between *T. gondii* infection and completed suicide has been scarcely studied. Therefore, in the present study we sought to determine the association between *T. gondii* infection of the central nervous system and suicide.

MATERIALS AND METHODS

Study design

An age- and gender-matched case-control study was performed.

Study population

One hundred and forty-seven decedents of whom 87 had committed suicide (cases) and 60 had not died by suicide (controls) were included in the study. Cases were enrolled at the "Instituto de Ciencias Forenses" in Mexico City, Mexico from November 2015 to December 2016, whereas controls were enrolled at the same forensic Institute from January to December 2016. Decedent cases were included in the study if they had a forensic diagnosis of completed suicide. Age and gender were not restrictive criteria for enrollment. Decedent controls were included in the study if they had a forensic diagnosis of death by causes other than suicide. Of the 87 decedent cases studied, 67 were male and 20 were females. They were 10–90 years old (mean age: 34.8 ± 17.4 years). Of the 60 decedent controls studied, 49 were males and 11 were females. They were 8–75 years old (mean age: 31.7 ± 14.8 years). No statistically significant difference in gender ($P = 0.49$) or age ($P = 0.25$) between cases and controls was found. Autopsies were performed >12 h after death.

Detection of *T. gondii* in brain by immunohistochemistry

Brain tissues (amygdala and prefrontal cortex) of decedents were examined for detection of *T. gondii* using immunohistochemistry. Brain tissues were formalin-fixed, and paraffin-embedded sections were examined using the Tinto Detector Immuno DNA System equipment (Bio SB, Santa Barbara, CA, USA) and Digital Pressure Cooker, Model PC-2000 (Bio SB). The Mouse/Rabbit Immunodetector HRP/DAB (Bio SB) was used for immunohistochemistry. Paraffin-embedded 2 µm tissue sections were used for immunostaining. We used the primary antibody "*T. gondii*, rabbit polyclonal" (Bio SB) and the positive control "*T. gondii* positive control slides" (Bio SB). All assays were performed according to the manufacturer's instructions. No information about the specificity of the polyclonal antibodies was included in the package insert. Slides were read by an anatomopathologist (author LFSA). A brown coloration of cysts or tachyzoites structural forms were considered positive results.

Statistical analysis

The software Epi Info version 7 and Microsoft Excel were used for the statistical analysis. Age of cases and controls were compared with the Student's *t*-test. The Fisher's exact test was used to compare the frequencies of *T. gondii* in brain tissues between the groups. Odds ratios (OR) and 95% confidence intervals (CI) were calculated. Statistical significance was set at a P value <0.05.

Ethical aspects

This project was approved by the Ethical Committee of the "Instituto de Ciencias Forenses" in Mexico City (Reference Number: CEI-014-2016).

RESULTS

Detection of *T. gondii* by immunohistochemistry was positive in 7 (8.0%) of the 87 decedent cases (6 found in prefrontal cortex and one in amygdala). Figure 1 shows *T. gondii* in brain (found in prefrontal cortex, case No. 25). Detection of *T. gondii* by immunohistochemistry was positive in 1 (1.7%) of the 60 decedent controls (found in prefrontal cortex, Fig. 2). No difference in the prevalence of *T. gondii* infection in brain by immunohistochemistry in cases and controls was found (OR: 5.16; 95% CI: 0.61–43.10; $P = 0.14$). Table 1 shows a stratification by age and gender and prevalence of *T. gondii* infection in brain by immunohistochemistry in cases and controls. No difference in

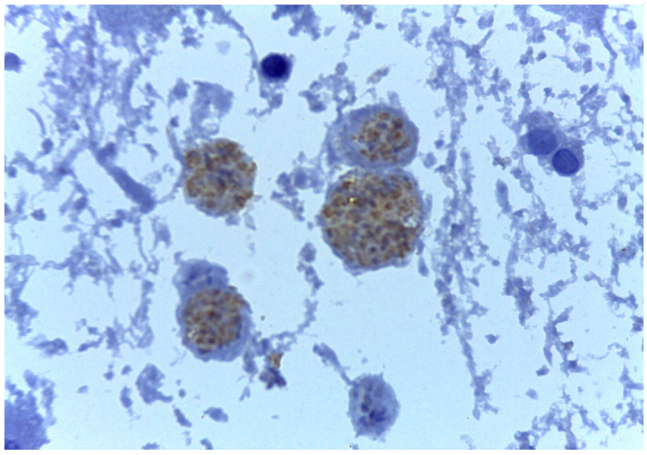

Fig. 1. T. gondii cysts in brain tissue (prefrontal cortex, case No. 25)

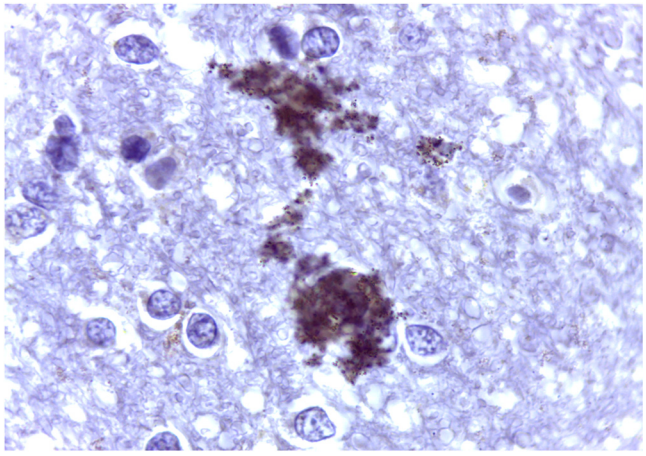

Fig. 2. Free *T. gondii* tachyzoites in brain tissue (prefrontal cortex, control No. 8)

prevalence rates of *T. gondii* infection in brain with respect to gender and age between cases and controls was found.

DISCUSSION

The link between exposure to *T. gondii* and suicidal behavior has been examined in a number of epidemiological studies [23–28]. Those studies have been based on serological analysis of anti-*T. gondii* antibodies in live persons. However, to the best of our knowledge, studies of the link between *T. gondii* infection of the brain and suicide using an age- and gender-matched case-control study design of decedents have not been reported. Therefore, in the present study, we assessed the association between *T. gondii* infection of the brain and completed suicide in decedents received for postmortem examinations in a forensic institute in Mexico City. Controls were similar to cases with respect to age, gender, and forensic center. Our approach was examining the infection with *T. gondii* in two sites (prefrontal cortex and amygdala) of the brain of decedents using immunohistochemistry. We found that persons who died by suicide had a somewhat higher, but not statistically significant, prevalence of *T. gondii* infection of the brain detected by immunohistochemistry than persons who died by causes other than suicide. Our findings thus suggest that infection with *T. gondii* in the central nervous system is not associated with completed suicide. We are not aware of another study about the association of suicide and *T. gondii* infection in brain detected by immunohistochemistry and therefore, we cannot compare our results with others of similar studies. There are few studies that have linked completed suicide to *T. gondii* seroprevalence rates. In a study about the seroprevalence of *T. gondii* infection in people who died due to sudden dead in Poland, a significantly higher *T. gondii* seroprevalence in suicide cases aged 38–58 years than in controls was found [29]. Suicide rates were positively associated with *T. gondii* seroprevalence rates in postmenopausal women of 20 European countries [30]. Comparison of results of such studies with the ones found in our study should be interpreted with care since different biological samples among the studies were analyzed. We examined prefrontal cortex and amygdala because these brain sites have been found consistently infected with *T. gondii* in experimentally

Table 1. Association between prevalence of *T. gondii* infection in the brain by immunohistochemistry and suicide: a stratification by gender and age groups

Characteristic	Cases			Controls			OR	95% Confidence interval	*P* value
	No. tested	Positivity to *T. gondii*		No. tested	Positivity to *T. gondii*				
		No.	%		No.	%			
Gender									
Male	67	5	7.5	49	1	2.0	3.87	0.43–34.24	0.39
Female	20	2	10.0	11	0	0.0	–	–	0.52
Age (years)									
≤30	42	1	2.4	36	1	2.8	0.85	0.05–14.15	1.00
31–50	30	5	16.7	16	0	0.0	–	–	0.14
>50	15	1	6.7	8	0	0.0	–	–	1.00
All	87	7	8.0	60	1	1.7	5.16	0.61–43.10	0.14

infected mice [31, 32]. In addition, these brain sites are involved in clinical outcomes of *T. gondii* infection. For instance, infection of cortex and amygdala resulted in memory impairment in mice [33], and infection in amygdala was linked to reduced predator aversion in rats [34]. Furthermore, the amygdala and prefrontal cortex form a circuit implicated in emotion regulation, the pathogenesis of major depressive disorder, and might be related to the pathogenesis of suicidal behavior since structural and functional abnormalities in these brain sites were demonstrated in patients with major depressive disorder and suicide attempts [35]. Little is known about the distribution of *T. gondii* in brain in humans using immunohistochemistry. We selected a sample of prefrontal cortex because the frontal lobe of brain has been successfully used to detect *T. gondii* in humans by immunohistochemistry [36]. *T. gondii* can also be found in choroid plexus by immunohistochemistry [36]. In a study of postmortem examinations of 17 AIDS patients with cerebral toxoplasmosis, choroid plexus infection was found in 53% of all cases by immunohistochemistry [37]. The precise location of *T. gondii* in brain in humans is limited, and a study of 102 autopsy cases on the presence of parasite DNA in four regions of the brain showed no specific distribution [38]. The choroid plexus might be an important location for detection of *T. gondii*. Experiments in mice have shown a close interaction between *T. gondii* infection at the choroid plexus and the impairment of the blood-cerebrospinal fluid barrier function indicating that infection-related neuroinflammation is initiated in the choroid plexus [39]. In a recent study of decedents, we found no association between completed suicide and the presence of anti-*T. gondii* antibodies in serum [40]. In the present study, we assessed this association beyond seropositivity by analyzing the presence of *T. gondii* directly in the central nervous system using immunohistochemistry in brain samples. To the best of our knowledge, this strategy has not been used in case-control studies of suicide decedents. In a cross-sectional study of decedents who committed suicide, we found an association between a history of depression and *T. gondii* infection in brain using immunohistochemistry [41]. Therefore, it is possible that the association between *T. gondii* infection in brain and completed suicide, albeit no currently observed in general in the present study, might be found in subsets of decedents with a history of clinical conditions including depression. The frequency of *T. gondii* infection in brain of decedents by immunohistochemistry found in the present study is lower than the 31% *T. gondii* seroprevalence reported in the population in Mexico City by indirect immunofluorescence [42].

We cannot rule out a link between suicide and *T. gondii* infection because our study have some limitations: 1) the sample size was small; 2) we examined only two brain sites by immunohistochemistry; and 3) we did not use other laboratory tests for increasing the sensitivity of detection of *T. gondii* infection in brain including molecular assays. Therefore, the lack of association between completed suicide and infection with *T. gondii* in brain found in our study should be confirmed in other studies with large sample sizes, examination of several brain sites, and using a diversity of tests for detection of *T. gondii* in brain. In addition, studies using not only qualitative tests but also quantitative tests for detection of *T. gondii* in brain to determine the association between suicide and *T. gondii* infection should be conducted.

CONCLUSIONS

Results suggest that *T. gondii* infection in brain is not associated with suicide. Further studies to confirm this finding are needed.

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Authors contributions: LAML and FGD obtained samples and data of the study population. LFSA read the immunohistochemistry slices. EIAS performed the immunohistochemistry tests. JHT and LFSA obtained funding. JHT, ARS, MAS, ASA and CAE performed the data analysis. CAE performed study concept and design, laboratory tests, analysis and interpretation of data, statistical analysis, and wrote the manuscript.

Conflicts of interest: The authors declare that no conflict of interest exists.

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