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Epilepsy and seropositivity rates of *Toxocara canis* and *Toxoplasma gondii*

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Summary

Purpose: Increased seropositivity for *Toxoplasma gondii* and *Toxocara canis* have been observed in epileptic patients. Our aim is to determine whether there is any relationship between these agents and epilepsy in our cryptogenic epilepsy group.
Material and methods: We studied specific IgG antibodies against *T. gondii* and *T. canis* in 100 cryptogenic epileptic patients and 50 healthy volunteers that had no history of epilepsy in their first degree relatives. We studied *T. gondii* and *T. canis*-specific IgG antibody serum levels and compared the values of these two groups.
Result: We found similar *T. gondii* and *T. canis* serum IgG antibodies in patients with cryptogenic epilepsy and in the control group, even though the control group included more animal owners.
Conclusion: We did not show any relationships between epilepsy and positive *T. gondii* and *T. canis* serology in our epileptic patients.

Introduction

Epilepsy is defined as recurrent seizures that are not the immediate result of an acute cerebral insult. When the cause is not found it is classified as either idiopathic or cryptogenic epilepsy. If there is a cause such as congenital abnormalities, trauma, hypoxia, tumor, cerebral infarction, degenerative diseases or infections, it is known as secondary epilepsy. In this group, infectious agents play a significant role especially in developing countries, and knowledge of these agents and the prevention of secondary outcomes of these agents such as epilepsy is important. Approximately 30% of epilepsy is associated with neurocysticercosis in developing countries. Poor sanitation and the consumption of contaminated foods, including vegetables and undercook meat,

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are causes of infection.[1] Other parasites, malariasis, toxoplasmosis and toxocariasis can cause not only acute symptomatic seizure but also remote symptomatic epilepsy.[2]

Toxoplasmosis, which is caused by *Toxoplasma gondii*, is widespread in the tissues of animals and birds, and infection occurs through eating undercooked meat. At the same time, cats, which are the definitive hosts, pass the oocysts in their faeces and contaminate the soil, so that humans may be infected.[3] Toxocariasis is known to be caused by *Toxocara canis*, a nematode of the dog, or *Toxocara cati*, which infects felines. Infection is by ingestion of eggs with active embryos.[4] *T. canis* larvae can be found in the human brain,[5] and *Toxoplasma cysts* remain inactive in the host's body. Following an infection with Toxocara eggs, a large number of larvae are able to remain in the tissues for many years. It is thought that the immune reaction which is triggered may lead to generalized seizures, while granulomas may lead to focal seizures.[3] While some studies have found a link between seroprevalency for these parasites and epileptic seizures,[6,7] others have found that the parasites are not responsible for epileptic pathogenesis but only that seroprevalency was high in epilepsy patients.[3,8] Consumption of food related to pork is very restricted in Turkey, so neurocysticercosis cases are anecdotal. Dogs and cats roam free without owners or routine veterinary care especially in rural areas. Therefore, we planned to screen for *T. canis* and *T. gondii* seroprevalency in normal controls and patients with cryptogenic epilepsy and to find whether there was a causal relationship between epilepsy and these parasites in our region.

Material and method

This study was conducted on 100 consecutive patients (46 women and 54 men) with idiopathic epilepsy aged between 13 and 64 (mean 28.88 ± 3.2 years) who were evaluated and followed up with clinical history, cranial imaging and other analyses in the Neurology Department of the Medical School of Adnan Menderes University in 2003. The types of seizure were defined according to the ILAE 1981 classification. The control group consisted of 29 male and 21 female volunteers aged between 11 and 60 (27.56 ± 1.71 years) with comparable epidemiological characteristics and without any complaints or history of previous seizures in either themselves or their families. All patients' demographic and lifestyle characteristics were derived by a standard questionnaire. Five milliliters of venous blood was taken from each patient and the sera of these blood samples were separated by centrifugation at 3000 rpm and then stored at $-20\,^{\circ}C$ until the analyses were carried out. Antibodies to Toxocara were measured with in house ELISA as described by Savigny et al. using excretory/secretory (E/S) antigens.[9] Positive and negative control sera and Toxocara E/S antigens were kindly provided by Dr. H. Auer (Institute of Hygiene, University of Vienna). Antibodies to Toxoplasma were measured with in house ELISA. Antigen preparations were made from tachyzoites of the TRH strain of *T. gondii*. Tachyzoites were obtained from the peritoneal exudates of mice infected 2 days earlier. These antigens were routinely used for the serological Toxoplasma tests in our laboratory.[10] The consistency between in house ELISA and commercial ELISA IgG kit had been found to be high in an earlier study performed in the parasitology laboratory (kappa = 0.960, $p = 0000$).[11] In another study, a significant correlation among IFAT, in house ELISA and IHA techniques was also observed.[12] For these two tests previously worked-out chess-board titrations with different antigen and conjugate concentrations were performed to establish optimal test conditions. Tests were performed in duplicate. Briefly, the wells in micro titer plates were sensitized with the antigen overnight at $4\,^{\circ}C$. The plates were then given three washes of 3 min each in phosphate buffer saline (PBS pH 7.4) containing 0.05% Tween 20 (PBS Tween). All sera were tested in 1:100 dilutions. Sera to be assayed were diluted in PBS Tween, and 100 μl volumes were added to each well. After 1 h of incubation at $37\,^{\circ}C$ the plates were washed five times with PBS Tween. The wells were then filled with 100 μl of anti-human IgG labeled with alkaline phosphatase conjugate (Sigma, A-3187) diluted 1:10,000 in PBS Tween, and incubated for 1 h at $37\,^{\circ}C$. After five washings, 100 μl substrate (Sigma N-2765) was added to each well. The enzymic hydrolysis of the substrate was stopped after 30 min by the addition of 100 μl NaOH. After 30 min, the optical density was measured with a spectrophotometer at 405 nm by Micro plate Reader. Results were considered positive when the extinction value 6—8 negative control sera rose to three times the standard deviation.

Statistical evaluations were performed with the 10.0 version of SPSS software. Chi-square test and Fisher's exact test were used in comparing the results.

Results

Sociodemographic characteristics of the patient and control groups (age, sex, occupation, place