Jordan Medical Journal

ORIGINAL ARTICLE

Investigating the possible association between *Toxoplasma gondii* infection and Multiple Sclerosis using a combination of serological and MRI indices

Nawal Hijjawi [1*], Ali Al-Radaideh[2, 3], Nawal El-Haj[1]

[1] Department of Medical Laboratory Sciences, Faculty of Applied Medical Sciences, The Hashemite University, Zarqa, Jordan.

[2] Department of Medical Imaging, Faculty of Applied Medical Sciences, The Hashemite University, Zarqa, Jordan.

[3] Department of Medical Radiography, College of Health Sciences, Doha University for Science and Technology, Doha, Qatar.

*Corresponding author:

nhijjawi@hu.edu.jo

Received: October 20, 2024

Accepted: May 1, 2024

DOI:
https://doi.org/10.35516/jmj.v59i3.1888

Abstract

Background: The present study was established to investigate if there is any association between multiple sclerosis (MS) and toxoplasmosis using a combination of serological and MRI indices.

Methods: 100 relapsing-remitting MS (RRMS) patients were recruited in the present study from the neurology clinic at Al-Bashir Hospital along with 34 age-matched healthy controls (HCs). MS patients as well as controls were tested for the presence of *Toxoplasma* IgG and IgM antibodies using ELISA technique. Magnetic resonance imaging (MRI) was performed on 1.5 T Siemens MR system. 3D T1 and T2 weighted images were acquired and processed to extract the white matter T2-lesion load.

Results: 86 RRMS patients (86%) and 12 HCs (35.3%) were tested positive for the *T. gondii* IgG antibodies. This difference in IgG seropositivity between MS patients and HCs was statistically significant ($p<0.05$). On the other hand, 19 RRMS patients (19%) and 4 HCs (11.7%) were tested positive for the *Toxoplasma gondii* IgM antibodies, and this difference was statistically insignificant ($p>0.05$). Neither IgG nor IgM concentration levels showed any significant association ($p>0.05$) with MS disease's indices.

Conclusions: A strong significant association between the presence of anti-toxoplasma IgG antibodies and MS disease was found. Serum antibodies for *T. gondii* should be tested regularly among MS patients and anti-parasitic therapy should be provided, when necessary to prevent any reactivation and dissemination of the disease among these immunocompromised patients.

Keywords: *Toxoplasma gondii*, Multiple Sclerosis (MS), *Toxoplasma* IgG and IgM antibodies, ELISA, Magnetic Resonance Imaging.

INTRODUCTION:

Toxoplasma gondii (*T. gondii*) is an obligate intracellular protozoan parasite, which replicates in the mammalian cells with a complex life cycle consisting of sexual cycle in its feline definitive host (cat) and asexual cycle in its intermediate host (livestock, birds, humans) [1]. Globally it is

estimated that *T. gondii* approximately infect one-third of the world's population [2,3]. The risk of getting infected with *T. gondii* is mainly associated with a low socio-economic status, where the infection is acquired by several routes either upon the ingestion of sporulated oocysts released with cat faeces contaminating food and water or upon the consumption of raw/ undercooked meat containing tissue cysts, or when the parasite is congenitally transmitted from mother to fetus and during blood transfusion and organ transplantation [4,5]. The clinical presentation of *T. gondii* infection is generally asymptomatic in immunocompetent humans [6], however, it is severe and life-threatening in congenital infections and among the immunocompromised patients, mainly those with acquired immune deficiency syndrome (AIDS) giving rise to more severe clinical manifestations, such as encephalitis [7–9].

Toxoplasmosis can be diagnosed by different biological, histological, serological and molecular methods [11,12,13]. Currently, serology is regarded as the gold standard method for toxoplasmosis diagnosis using enzyme linked immunosorbent assay (ELISA) test, which can detect specific antibodies Immunoglobulin G (IgG) and M (IgM) in patient's serum [10,11].

Multiple Sclerosis (MS) is regarded nowadays as one of the most frequent immune-mediated, inflammatory demyelinating autoimmune diseases, which leads to the destruction of nerves in the central nervous system (CNS) affecting many individuals worldwide mostly young adults (20 to 50 years old) [12–15]. The symptoms, severity, and course of MS disease vary widely depending on the sites of the plaques as well as the extent of the nerve demyelination [14]. Most MS patients (80%) initially experience relapsing-remitting MS (RRMS), featuring axonal demyelination and incomplete remyelination. Progression leads to Secondary Progressive MS (SPMS), marked by extensive degeneration, axonal loss, and atrophy with minimal or no remyelination [16].

In several previous studies, toxoplasmosis was investigated for its possible association with the pathogenesis of many autoimmune diseases. These autoimmune diseases include MS, rheumatoid arthritis, inflammatory bowel disease, lupus erythematosus and psychiatric/ neurological disorders where higher levels of anti-toxoplasma (IgG) were detected in the serum of patients suffering from the above-mentioned diseases compared to controls [17–21].

More specifically, the role of *T. gondii* infection in MS disease has been investigated in many studies and was found to be controversial [22–27]. While some studies indicated the presence of a strong and significant association between toxoplasmosis and MS, others found no significant association between the two diseases [28,29]. Furthermore, some studies showed that the seropositivity of toxoplasmosis tends to play a protective role against the development and progression of MS by excreting an immunomodulatory effect [25,30,31]. Therefore, the main aim of the present study was to observe if there is any significant association between toxoplasmosis and MS disease indices by investigating the seroprevalence of *T. gondii* IgG and IgM among MS patients and comparing them with age and sex-matched HCs. Furthermore, any possible association between the seroprevalence of *T. gondii* IgG and IgM and white matter T2 lesion load among MS patients was also investigated.

MATERIALS AND METHODS

Study participants

One hundred relapsing-remitting MS (RRMS) patients (67 females, 33 males; mean age 37.7 years; age range 18-60 years), were recruited in the present study from the neurology clinic at Al-Bashir Hospital from July to September 2017 after receiving ethical approval to conduct the study from the Hashemite University Institutional Review Board (HU-IRB) NO: 1610795/222/A. Thirty-four age-matched HCs (18 females, 16 males, mean age 34 years, age range 20–60 years) agreed to participate in this case-control study. The inclusion criteria for this study were as follows: age between 18 and 60 years, had a clinically definite MS (RRMS) based on revised McDonald's criteria 2010, had not experienced a relapse or received corticosteroids treatment in the last four weeks preceding the study, no previous history of other central nervous system diseases such as demyelinating and neurodegenerative diseases, brain tumor or surgery, head injury, cerebrovascular disease. Only 65 RRMS patients (41 females, 24 males, mean age 32 years; age range 18–58 years) had magnetic resonance imaging (MRI) scanning. The present study was carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki). All participants gave informed consent according to local ethics approval. However, the 65 RRMS patients who had an MRI scan were asked to fill out the MRI safety questionnaire before participating in the study. Neurological examinations were carried out by a consultant neurologist who was qualified to perform the EDSS assessments according to the standardized scoring system in Neurostatus (http://www.neurostatus.net/scoring/index.php). Table 1 summarizes the characteristics of all RRMS patients and HCs.

Table 1: Descriptive characteristics of the study participants.

Subjects	Controls	MS Patients
N	34	100
Sex (F/M)	(18/16)	(67/33)
Age range, years	20-60	18 – 60
Mean age ± SD, years	34 ± 7.1	37.7 ± 9.87
Disease Duration range, years	-	0.1 – 33
Mean Disease Duration ± SD, years	-	6.71 ± 6.73

Collection of blood samples and serum preparation

Five millilitres blood samples were collected from each RRMS patient and HC in vacutainer plain tubes with gel to separate the serum. The blood samples were allowed to clot at room temperature (22–25 °C) before being centrifuged at 4000 rpm for 5 min using Roto Fix 32A Centrifuge (Germany) to separate serum from other blood components. Thereafter, the serum was decanted in 2 ml Eppendorf tubes and frozen at −70 °C until subsequent analysis.

Toxoplasma gondii IgG and IgM detection using ELISA assay

All the collected serum samples from the recruited MS patients (n=100) and controls (n=34) were tested for the presence of T. gondii IgG and IgM using a commercially available enzyme linked immunosorbent assay (ELISA) kits (RD-Ratio diagnostic, Germany) according to the manufacturer's instructions. The validity of the ELISA kit was checked and validated. Briefly, serum

samples from MS patients and controls were individually mixed with diluent to reach the dilution of 1: 100 and dispensed into wells in the strip. Positive and negative control were included in each test. After incubation, all samples were washed with washing solution and *T. gondii* IgG, anti-IgG conjugate was dispensed into each well, while *T. gondii* IgM, Toxo-HRP- conjugate was added and incubated and washed repeatedly before adding the substrate solution. Finally, stopping solution was dispensed to all samples and the absorbance was measured within 15 minutes at a wavelength of 450 nm (*T. gondii* IgG and IgM kit). The concentration of *Toxoplasma* -specific IgG and IgM antibodies were measured in IU/ml.

Magnetic resonance imaging (MRI):

Magnetic resonance imaging was performed on a 1.5 T Siemens MR system (Siemens Medical Systems, Erlangen, Germany), equipped with 8-channel phase-array head coil. The imaging protocol included 3D T1- weighted Magnetization Prepared – Rapid Acquisition Gradient Echo (MPRAGE) (TR = 900 ms; TE = 4.43 ms; TI = 604 ms; 1.2 mm isotropic spatial resolution), and 3D T2-weighted Fluid-Attenuated Inversion Recovery (FLAIR) (TR = 8210 ms; TE = 100 ms; TI = 2500 ms; 1.5 × 1.5 × 4 mm spatial resolution).

Calculation of white matter's T2 lesion load:

White matter T2 lesions segmentation was performed in Jim software (Jim version 7; Xinapse Systems, Northants, England). The T2-weighted FLAIR images were spatially co-registered to the high resolution, T1-weighted MPRAGE images using rigid-body registration with six degrees of freedom to make the two volumes dimensionally similar. The co-registered T2-FLAIR images were used to define "seed" points in the center of each MS lesion, which were used in combination with the T1 and T2 images to find the MS lesions based on the Fuzzy Connectedness algorithm [32]. Figure 1 shows 3D rendering of segmented MS lesions in one patient.

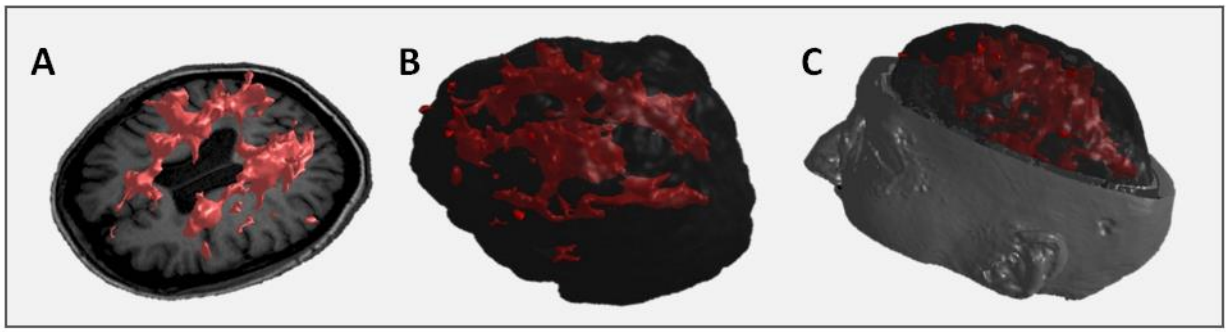

Figure 1: Three-dimensional (3D) rendering of segmented MS lesions derived from high-resolution MRI scans and displayed on A) axial image, B) skull stripped brain-rendered image, and C) whole head-rendered image.

Statistical Analysis

All statistical analyses were performed in SPSS 23 (SPSS, Chicago, IL, USA). Statistical differences between RRMS patients and controls for the IgG and IgM seropositivity and the differences between RRMS patients with IgG seropositive and those with seronegative for any of the MS disease indices were assessed using independent samples t-test. *T. gondii* IgG and

IgM concentration levels were tested for any possible association with the calculated MS disease indices using Pearson correlation. The significant level was tested at p-value<0.05.

RESULTS

The frequencies of anti-*T. gondii* IgG and IgM in MS patients and HCs are shown in Table 2. Eighty-six RRMS patients (86%) and 12 HCs (35.3%) were tested positive for the *T. gondii* IgG antibodies. This difference in IgG seropositivity between MS patients and HCs was statistically significant (p<0.05). IgG seropositivity difference was reflected in the concentration of *T. gondii* IgG, which was significantly (p <0.05) higher in the serum of RRMS (44.70 IU/ml) than that in the serum of HCs (23.47 IU/ml). Unlike the IgG results, 19 RRMS patients (19%) and 4 HCs (11.7%) were tested positive for the *Toxoplasma gondii* IgM antibodies, and this difference was statistically insignificant (p >0.05).

Table 2: Frequency of anti-*T. gondii* IgG and IgM seropositivity in MS patients and controls.

Subjects/Number	Frequency of anti-T. gondii IgG seropositivity (%)	*P*-value	Frequency of anti-T. gondii IgM seropositivity (%)	*P*-value
MS patients / 100	86 (86%)	0.000	19 (19%)	0.118
Controls / 34	12 (35.3%)		4 (11.7%)	
Abbreviations: MS-Multiple sclerosis; IgG- immunoglobulin G; IgM- immunoglobulin M				

No significant difference (p >0.05) was detected between RRMS patients with IgG seropositive and those with seronegative for any of the MS disease indices (EDSS, Disease duration, Number of relapses, and white matter T2 lesion load). Similar findings were detected for the *Toxoplasma gondii* IgM positives. Both *T. gondii* IgG and IgM concentration levels were tested for any possible association with the above MS disease indices. Neither IgG nor IgM concentration levels showed any significant association (p >0.05) with these indices.

DISCUSSION:

An emerging field of research has started to examine the association between infectious pathogens and autoimmune and neurological diseases [33]. *Toxoplasma gondii* is among the investigated infectious agents which attracted scientists to investigate possible association with the pathogenesis of many autoimmune diseases among them is MS where higher levels of anti-toxoplasma IgG were detected in the serum and compared to age and sex matched controls [17–21].

Toxoplasma gondii has a clear predilection for the CNS where tissue cysts are usually formed in neurons of the brain and sometimes in the retina as well as in the muscle cells [34]. The status of infection by *T. gondii,* whether acute or chronic, can be best determined by the detection of IgM or IgG antibodies, respectively [35]. According to previous studies, the presence of anti-toxoplasma IgG antibodies in MS patients ranges between 32.4% -39.1% depending on the sample size and the population involved in the research [36]. In the present study, higher percentage of MS patients have anti-toxoplasma IgG (86%) compared to HCs group (35.3%) and the *T. gondii* IgG absorbance and concentrations were significantly higher in MS patients compared to HCs (p < 0.05). This relatively high anti-toxoplasma IgG percentage detected in this study could be attributed to the high probability of exposure to the *T. gondii* parasite

by MS patients in Jordan, and the nature of the immunosuppressive medications. However, it might be hard to decide whether the MS patients acquired toxoplasmosis before they are diagnosed with MS or not, but the presence of higher levels of IgG compared to HCs indicates chronic nature of toxoplasmosis. Moreover, since some of them have IgM antibodies, which indicate recent infections (acute toxoplasmosis), the fact that these MS acquired during the MS course of infection is very likely and can be partially attributed to the nature of the immunosuppressive medication.

Several previous studies have suggested an association between chronic *T. gondii* infection and MS, but conflicting results have been reported, indicating that *T. gondii* can exert a positive or a negative role on different autoimmune condition [22–27]. A recently published metanalysis of different studies assessing the association between MS and *T. gondii* has demonstrated a statistically lower, but still not significant, protective effect of the parasite on the risk of being diagnosed with MS [25]. Upon inspecting the included studies in the above meta-analysis study, one can observe that the sample size is relatively small. Therefore, to overcome this limitation, a new large population-based study was conducted to inspect the relationship between *T. gondii* and MS [27]. The results of the case-control study revealed a negative association between *T. gondii* and MS. This implies a potential protective role of the parasite, aligning with the hygiene hypothesis, which states that limited early-life exposure to pathogens may elevate the risk of autoimmune diseases such as MS and allergies due to immunological imbalance [27].

In the current study, *T. gondii* IgM antibodies in the serum of MS patients were not significantly ($p<0.05$) different from those detected in the serum of HCs. This could be attributed to the relatively few inflammatory changes during the RRMS stage. Furthermore, 19 IgM seropositive patients had multiple attacks and flairs within short period of time compared to IgM seronegative patients, and were severely affected by the symptoms of MS. Therefore, the presence IgM should not be overlooked among MS patients, and it should be treated immediately to prevent any chance of dissemination of the parasite among these immunocompromised patients, who have active toxoplasmosis.

At this stage, elucidating the importance or significance of the correlation between toxoplasmosis and MS proves challenging. Further research and investigations, encompassing larger sample sizes and diverse MS phenotypes, are urgently needed. The present study recommended that the serum antibodies for *T. gondii* should be tested regularly among MS patients and anti-parasitic therapy should be provided, especially for the IgM positive patients, to prevent any reactivation and dissemination of the disease among these immunocompromised patients.

CONCLUSION:

The findings of the present study revealed relatively high anti-toxoplasma IgG percentage among the studied MS patients, which could be attributed to the high probability of exposure to the *T. gondii* parasite by MS patients in Jordan and the nature of the immunosuppressive medications. Therefore, serum antibodies for *T. gondii* should be tested regularly among MS patients and anti-parasitic therapy should be provided when necessary to prevent dissemination and complications from this parasitic disease.

Declarations:

- Competing interests

The authors declare that they have no conflict of interest.

- Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. N.A.E received a small master studentship from the Hashemite University.

- Authors' contributions

The authors, all contributed to different aspects of this multidisciplinary piece of work and all of them are aware of and approve the manuscript as submitted to this journal:

Nawal Hijjawi, PhD: Proposed the concept and design the study, supervised Toxoplasma gondii IgG and IgM detection and contributed to the writing of the manuscript.

Ali Al-Radaideh, PhD: Proposed the concept and design of the study, supervised the MRI physics, analyzed the MRI data and wrote the first draft of the manuscript.

Nawal Adel El-Haj, M.Sc: Collected blood samples, performed Toxoplasma gondii IgG and IgM detection and contributed to the writing of the manuscript.

- Acknowledgements

We would like to thank the Ministry of Health and Al-Bashir hospital for their great help and support and for allowing us to use their 1.5 TMRI in this study.

REFERENCES

1. Tenter AM, Heckeroth AR, Weiss LM. Tenter AM, Heckeroth AR, Weiss LM. Toxoplasma gondii: from animals to humans. Int J Parasitol 2000; 30:1217. https://doi.org/10.1016/S0020-7519(00)00124-7.
2. Innes. A brief history and overview of Toxoplasma gondii. Zoonoses Public Health 2010; 57:1–7. https://doi.org/10.1111/j.1863-2378.2009.01276.x.
3. Montoya J, Liesenfeld O. Toxoplasmosis. Lancet 2004; 363:1965–76. https://doi.org/10.1016/S0140-6736(04)16412-X.
4. Barsoum RS. Parasitic infections in organ transplantation. Experimental and Clinical Transplantation 2004; 2:258–67.
5. Foroutan-Rad M, Majidiani H, Dalvand S, Daryani A, Kooti W, Saki J, et al. Toxoplasmosis in Blood Donors: A Systematic Review and Meta-Analysis. Transfus Med Rev 2016; 30:116–22. https://doi.org/10.1016/J.TMRV.2016.03.002.
6. Wormser GP, Tanowitz HB, Weiss LM. Toxoplasmosis: A Comprehensive Clinical Guide. vol. 34. Oxford Academic; 2002. https://doi.org/10.1086/340624.
7. Robert-Gangneux F, Dardé M. Epidemiology of and diagnostic strategies for toxoplasmosis. Clin Microbiol Rev 2012; 25:264–96. https://doi.org/10.1128/CMR.05013-11.
8. Mahadevan A, Arvinda Hanumantapura R, Satishchandra P, Avindra N, Udaykumar R, Shankar Susarla K. Neuropathological correlate of the “concentric target sign” in MRI of HIV associated cerebral toxoplasmosis. J Magn Reson Imaging 2013; 38:488. https://doi.org/10.1002/JMRI.24036.
9. Roche AD, Rowley D, Brett FM, Looby S. Concentric and Eccentric Target MRI Signs in a Case of HIV-Associated Cerebral Toxoplasmosis. Case Rep Neurol Med 2018; 2018:1–3. https://doi.org/10.1155/2018/9876514.
10. Jamshaid Iqbal, Nabila Khalid. Detection of acute Toxoplasma gondii infection in early pregnancy by IgG avidity and PCR analysis. J Med Microbiol 2007; 56:1495–9. https://doi.org/10.1099/JMM.0.47260-0.
11. Sakikawa M, Noda S, Hanaoka M, Nakayama H, Hojo S, Kakinoki S, et al. Anti-Toxoplasma

Antibody Prevalence, Primary Infection Rate, and Risk Factors in a Study of Toxoplasmosis in 4,466 Pregnant Women in Japan. Clin Vaccine Immunol 2012; 19:365. https://doi.org/10.1128/CVI.05486-11.

12. Reiber H, Teut M, Pohl D, Rostasy K, Hanefeld F. Paediatric and adult multiple sclerosis: age-related differences and time course of the neuroimmunological response in cerebrospinal fluid. Mult Scler 2009; 15:1466–80. https://doi.org/10.1177/1352458509348418.
13. Ramagopalan S V., Sadovnick AD. Epidemiology of Multiple Sclerosis. Neurol Clin 2011; 29:207–17. https://doi.org/10.1016/J.NCL.2010.12.010.
14. Sinnecker T, Mittelstaedt P, Dörr J, Pfueller CF, Harms L, Niendorf T, et al. Multiple sclerosis lesions and irreversible brain tissue damage: a comparative ultrahigh-field strength magnetic resonance imaging study. Arch Neurol 2012; 69:739–45. https://doi.org/10.1001/ARCHNEUROL.2011.2450.
15. Schoonheim MM, Vigeveno RM, Lopes FCR, Pouwels PJW, Polman CH, Barkhof F, et al. Sex-specific extent and severity of white matter damage in multiple sclerosis: implications for cognitive decline. Hum Brain Mapp 2014; 35:2348–58. https://doi.org/10.1002/HBM.22332.
16. Confavreux C, Vukusic S. Natural history of multiple sclerosis: a unifying concept. Brain 2006; 129:606–16. https://doi.org/10.1093/BRAIN/AWL007.
17. Brown AS, Schaefer CA, Quesenberry CP, Liu L, Babulas VP, Susser ES. Maternal exposure to toxoplasmosis and risk of schizophrenia in adult offspring. Am J Psychiatry 2005; 162:767–73. https://doi.org/10.1176/APPI.AJP.162.4.767.
18. Zinger H, Sherer Y, Goddard G, Berkun Y, Barzilai O, Agmon-Levin N, et al. Common infectious agents prevalence in antiphospholipid syndrome. Lupus 2009; 18:1149–53. https://doi.org/10.1177/0961203309345738.
19. Petríková J, Agmon-Levin N, Shapira Y, Barzilai O, Ram M, Gilburd B, et al. Prevalence of Toxoplasma antibodies among patients with various autoimmune diseases. Ann Rheum Dis 2010;69: A8–A8. https://doi.org/10.1136/ARD.2010.129577T.
20. Arango M-T, Shoenfeld Y, Cervera R, Anaya J-M. Infection and autoimmune diseases, El Rosario University Press; 2013.
21. Carter CJ. Toxoplasmosis and Polygenic Disease Susceptibility Genes: Extensive Toxoplasma gondii Host/Pathogen Interactome Enrichment in Nine Psychiatric or Neurological Disorders. J Pathog 2013; 2013:1–29. https://doi.org/10.1155/2013/965046.
22. Serdar O, Fatima K, Hayri Demirbas, İnci Ç, Özge Yılmaz K, Özlem M, et al. Relationship of Toxoplasma Gondii Exposure with Multiple Sclerosis. Electronic Journal of General Medicine 2016; 13:58–63. https://doi.org/10.15197/EJGM.01429.
23. Marjan S, Mehdi T. Seroepidemiological Study of Toxoplasma gondii in Patients with Multiple Sclerosis in Ahvaz, Southeastern Iran. Medical Laboratory Journal 2017; 11:6–9. https://doi.org/10.18869/acadpub.mlj.11.3.6.
24. Stascheit F, Paul F, Grittner U, Harms L, Rosche B. Toxoplasma gondii Seropositivity Is Negatively Associated With Multiple Sclerosis (P1.121). Neurology 2015;84.
25. Reza Saberi, Mehdi Sharif, Shahabeddin Sarvi, Sargis A Aghayan, Seyed Abdollah Hosseini, Davood Anvari, et al. Is Toxoplasma gondii playing a positive role in multiple sclerosis risk? A systematic review and meta-analysis. J Neuroimmunol 2018; 322:57–62. https://doi.org/10.1016/J.JNEUROIM.2018.06.011.
26. The role of Toxoplasma gondii in multiple sclerosis: A matched case-control study. | Neurology Asia | EBSCOhost n.d. https://openurl.ebsco.com/EPDB%3Agcd%3A11%3A18537051/detailv2?sid=ebsco%3Aplink%3Ascholar&id=ebsco%3Agcd%3A150990999&crl=c (accessed February 3, 2024).

27. Nicoletti A, Cicero CE, Giuliano L, Todaro V, Lo Fermo S, Chisari C, et al. Toxoplasma gondii and multiple sclerosis: a population-based case–control study. Scientific Reports 2020 10:1 2020; 10:1–7. https://doi.org/10.1038/s41598-020-75830-y.
28. Serdar O, Fatima K, Hayri Demirbas, İnci Ç, Özge Yılmaz K, Özlem M, et al. Relationship of Toxoplasma Gondii Exposure with Multiple Sclerosis. Electronic Journal of General Medicine 2016; 13:58–63. https://doi.org/10.15197/EJGM.01429.
29. Marjan S, Mehdi T. Seroepidemiological Study of Toxoplasma gondii in Patients with Multiple Sclerosis in Ahvaz, Southeastern Iran. Medical Laboratory Journal 2017; 11:6–9. https://doi.org/10.18869/acadpub.mlj.11.3.6.
30. Stascheit F, Paul F, Grittner U, Harms L, Rosche B. Toxoplasma gondii Seropositivity Is Negatively Associated With Multiple Sclerosis (P1.121). Neurology 2015;84.
31. Koskderelioglu A, Afsar I, Pektas B, Gedizlioglu M. Is Toxoplasma gondii infection protective against multiple sclerosis risk? Mult Scler Relat Disord 2017; 15:7–10. https://doi.org/10.1016/J.MSARD.2017.04.004.
32. Al-Radaideh A, Athamneh I, Alabadi H, Hbahbih M. Cortical and Subcortical Morphometric and Iron Changes in Relapsing-Remitting Multiple Sclerosis and Their Association with White Matter T2 Lesion Load: A 3-Tesla Magnetic Resonance Imaging Study. Clin Neuroradiol 2019; 29:51–64. https://doi.org/10.1007/S00062-017-0654-0.
33. Delogu LG, Deidda S, Delitala G, Manetti R. Infectious diseases and autoimmunity. J Infect Dev Ctries 2011; 5:679–87. https://doi.org/10.3855/JIDC.2061.
34. Schlüter D, Barragan A. Advances and Challenges in Understanding Cerebral Toxoplasmosis. Front Immunol 2019;10. https://doi.org/10.3389/FIMMU.2019.00242.
35. Hill D, Dubey JP. Toxoplasma gondii: transmission, diagnosis and prevention. Clin Microbiol Infect 2002; 8:634–40. https://doi.org/10.1046/J.1469-0691.2002.00485.X.
36. Reza Saberi, Mehdi Sharif, Shahabeddin Sarvi, Sargis A Aghayan, Seyed Abdollah Hosseini, Davood Anvari, et al. Is Toxoplasma gondii playing a positive role in multiple sclerosis risk? A systematic review and meta-analysis. J Neuroimmunol 2018; 322:57–62. https://doi.org/10.1016/J.JNEUROIM.2018.06.011

دراسة العلاقة المحتملة بين الإصابة بالتوكسوبلازما غوندي والتصلب اللويحي المتعدد باستخدام مزيج من المؤشرات المصلية والتصوير بالرنين المغناطيسي

نوال حجاوي[1]، علي الردايدة [2,3]، نوال الحاج[1]

[1] قسم العلوم الطبية المخبرية، كلية العلوم الطبية التطبيقية، الجامعة الهاشمية، الاردن.

[2] قسم التصوير الطبي، كلية العلوم الطبية التطبيقية، الجامعة الهاشمية، الاردن، الاردن.

[3] قسم التصوير الاشعاعي الطبي، كلية العلوم الصحية، جامعة الدوحة للعلوم والتكنولوجيا، الدوحة، قطر

Received: October 20, 2024

Accepted: May 1, 2024

DOI:

https://doi.org/10.35516/jmj.v59i3.1888

الملخص

الخلفية والاهداف : تم عمل الدراسة الحالية لدراسة احتمالية وجود علاقة بين مرض التصلب اللويحي المتعدد (MS) وطفيل التوكسوبلازما جوندي باستخدام مزيج من المؤشرات المصلية والتصوير بالرنين المغناطيسي.

منهجية الدراسة : تم عمل الدراسة على 100 مريض يعانون من التصلب اللويحي المتعدد من عيادة الأعصاب في مستشفى البشير بالإضافة إلى 34 شخصاً سليماً تطابقاً في العمر. تم اختبار المرضى الذين يعانون من التصلب المتعدد والاشخاص الاصحاء لوجود نوعين من الاجسام المضادة لطفيل التوكسوبلازما (IgG و IgM) باستخدام تقنية الELISA .

تم إجراء التصوير بالرنين المغناطيسي باستخدام جهاز الرنين المغناطيسي من شركة سيمنز وبقوة 1.5 تسلا، حيث تم تصوير المرضى عدة انواع من صور الرنين المغناطيسي وهي (T1 and T2 FLAIR) ليتم بعدها معالجة وتحليل الصور لاستخراج الآفات في المادة البيضاء.

النتائج: تبين وجود اجسام مضادة للتوكسوبلازما IgG عند86 (86%) مريض تصلب لويحي و12 (35.3%) شخص سليم ($p<0.05$). من ناحية اخرى، تبين وجود الاجسام المضادة للتوكسوبلازما IgM عند19 (19%) مريض تصلب لويحي و4 (11.7%) شخص سليم ($p>0.05$). لم تظهر مستويات تركيز IgG أو IgM أي علاقة احصائية قوية ($p> 0.05$) مع مؤشرات مرض التصلب اللويحي.

الاستنتاجات: هناك ارتباط احصائي قوي بين وجود أجسام IgGالمضادة للتوكسوبلازما ومرض التصلب اللويحي المتعدد. ينبغي اختبار أجسام IgG المضادة للتوكسوبلازما بانتظام بين مرضى التصلب اللويحي المتعدد وينبغي توفير العلاج المضاد للطفيل عند الضرورة لمنع أي إعادة تنشيط وانتشار لهذا المرض بين هؤلاء المرضى الذين يعانون من مناعة ضعيفة.

الكلمات الدالة: توكسوبلازما جوندي، التصلب اللويحي المتعدد (MS)، الاجسام المضادة للتوكسوبلازما (IgG)، الاجسام المضادة للتوكسوبلازما (IgM)، التصوير بالرنين المغناطيسي (MRI) ، ELISA.