ASSOCIATION BETWEEN *TOXOPLASMA GONDII* INFECTION IN WOMEN AND THE PRESENCE OF CARDIOLIPIN AND PHOSPHOLIPID ANTIBODIES

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Abstract

IgG and IgM cardiolipin and phospholipid antibodies were measured, by an ELISA technique, in the sera of patients with toxoplasmosis. Immunological methods for autoantibodies had been applied by ELISA. Regarding Anti-cardiolipin it was revealed that 24.7% of toxoplasmosis cases versus 0% of control group showed significant positive result (P=0.0412) . Whereas, 12.5% of toxoplasmosis patients and 12% of healthy controls had anti-cardiolipin antibodies, with no significant differences (P=0.554) between these two groups.

Keywords: Toxoplasma gondii , cardiolipin, phospholipid antibodies

Introduction

There are several causes for triggering of autoimmune diseases and the infectious agents are one of these important causes (1). The relationship between infections and autoimmune disease has been studied extensively over many years and described the association between microbiological infection (bacterial, viral and parasite) and development of autoimmunity (2, 16). The identification of microbial peptides that similar to self- tissue by molecular mimicry is a true factor that inducing or promoting autoimmune diseases triggering by certain infections (2).According to these association between the infectious agents and autoimmune triggering ,this study suggests to identify if there is a correlation between *T. gondii* infection and autoimmune response throughout investigate the presence of anti-cardiolipin (aCL) and anti-phospholipid (aPL)) antibodies, in the sera of toxoplasmosis patients.

Materials and methods

Fifty four and thirty two sera of women were diagnosed with toxoplasmosis were investigated for IgM and IgG anti-cardiolipin and anti-phospholipid antibodies tests respectively, in addition to 14 and 10 sera from healthy women as control groups randomly selected to compare the presence of these antibodies respectively . All these sera were collected from women who attended to the hospitals in AL-Najaf and AL-Qadissyha provinces in Iraq, from September 2008 to February 2009.

Results

1-Anti- cardiolipin Antibody (aCL)

By ELISA, 13 (24.7%) of 54 toxoplasmosis cases showed positive anti-cardiolipin (concentration ≥ 7 u/ml), while no one (0%) of 14 healthy women showed positive result(P = 0.0412) (Table 1).

Table (1): Anti-cardiolipin Seropositivity in Toxoplasmosis Cases and Controls.

		Anti-cardiolipin			
Groups	No.	Positive (≥ 7 u/ml)	%	Negative < 7u/ml	%
Toxoplasmosis cases	54	13	24.7	41	75.3
controls	14	0	0	14	100

$X^2 = 4.17$ p= 0.0412

2- Anti- Phospholipid Antibody (aPL)

By ELISA, 4 (12.5%) of 32 toxoplasmosis cases showed positive aPL (concentration ≥ 10 u/ml), versus 2 (20%) of 10 healthy women (Table 2).

Table (2): Anti- Phospholipid Seropositivity in Toxoplasmosis Cases and Controls

		Anti- Phospholipid			
Groups	No.	Positive (≥ 10 u/ml)	%	Negative (< 10u/ml)	%
Toxoplasmosis cases	32	4	12.5	28	87.5
controls	10	2	20	8	80

$X^2 = 0.35$ P= 0.554

Discussion

Infection agent continues to be among the leading cause of morbidity and mortality worldwide. In addition, they are also implicated in the pathogenesis of indirect consequences such as induction of the autoimmune diseases (7, 11, 12).

In the present study, two markers were examined to determine the autoimmune response in toxoplasmosis patients.

1- Anti- Cardiolipin Antibody (aCL)

By ELISA, the study revealed that 13(24.7%) of 54 sera of toxoplasmosis patients showed positive aCL, while no one (0%) of 14 sera from healthy women showed positive aCL. There is significant difference ($P<0.05$) in the frequency of seropositive aCL between patients group and healthy control group. This result revealed a positive correlation between the disease and aCL. This result corresponding to the result of (10) in France and (1) in Italy, they recorded that, the levels of anticardiolipin (IgG & IgM) antibodies among those with toxoplasmosis were 29.3% and 27.4% respectively.

The explanation of this correlation is attributed firstly, to the mechanism of molecular mimicry, i.e. immunological cross-reactivity between the parasite and components of host tissues (8, 13), and secondly , Because aCL are strongly associated with recurrent pregnancy loss (3, 4); therefore, common abortions in patients with toxoplasmosis may be partially attributed to the presence of aCL.

In the present study there is no one (0%) of healthy control group had positive aCL and this result is disagreement with the finding of (9) who recorded that 10% of 100 healthy adults had positive aCL. This low aCL ratio in the current study may be due to the decrease number of tested healthy women.

2- Anti- Phospholipid (aPL)

The seropositivity of aPL antibodies were evaluated for 32 toxoplasmosis cases and 10 healthy women by ELISA technique. Four (12.5%) toxoplasmosis cases and two (20%) healthy woman showed positive aPL, but with no significant difference (P>0.05) between them.

This result is compatible with the result of (15); who showed that, the aPL antibodies in Leishmaniasis was revealed significant difference when compared with toxoplasmosis and malaria patients. While the current study is incompatible with (17); who estimated the prevalence of IgG and IgM antibodies to *T. gondii* in patients with antiphospholipid syndrome and found a significantly high prevalence of IgM antibodies only. But (5) confirmed that, the rising of IgM antibodies in antiphospholipid syndrome patient was false positive.

The difference in results of aPLseropositivity in toxoplasmosis patients may be attributed to genetic factors (6), environmental factors or presence of other co-infectious agents (14) and performance of ELISA kit which used in each study .

References:

Barrile, A.; Quattrocchi, P.; Bonanno, D.; Crisafe, A.; *et al.*, (1992). Presence and significance of anticardiolipin antibodies in infectious diseases. Recenti. Prog. Med. 83(6):350-353.

Berlin ,T.,Zandman-Goddard,G., Blank,M., Shoenfeld,Y. (2007). Autoantibodies in non autoimmune individuals during infections. Ann N. Y. Acad. Sci., 1108:584-593.

Buchanan, R.R.C., Wardlow,J.R., Riglar,A.G., Little John,G.O., and ,M.H.(1989). Anti-phospholipid antibodies in connective tissue diseases : The relation to anti –phosphlipid syndrome and forme frusta disease .J. of Rheumatol. 16:757-761.

Bustos, D.; Moret, A.; Tambutti, M.; Gogorza, S.; Testa, R.; Ascione, A. and Prigoshin, N. (2006). Autoantibodies in Aigintine women with recurrent pregnancy loss. Americ. J. Repord. Immunol., 55:201-207.

De-Carolis, S.; Botta, A.; Santocci, S.; Garofalo, S.; Martino, C.; Perrelli, A.; Salvi, S,; Ferrazzani, S.; Caforio, L. and Scambia, G. (2009). Clin. Rev. allergy Immunol [Epub ahead of print].

Dighiero, G., and Rose, N.R.(1999). Critical self- epitopes are key to the understanding of self- tolerance and autoimmunity. Immunol. Today; 20:423-428.

Gill S.R., Pop M., Deboy R.T.(2006).Metagenomic analysis of human distalgut microbiome.Scince.,312 :1355-1359.

Kalra, S.; Tuli, A.; Goyal, U.; Choudhary, R. and Raheja, S. (2002). Correlation of anticardiolipin antibody IgM with first trimester recurrent abortions. J. Anat. Soc. India., 51(10): 10-13.

Klok, A. M.; Geertzen, R.; Rothova, A.; Baarsma, G. S. and Kijlstra, A. (1992). Anticardiolip in antibodies in uvieitis. Curr. Eye. Res.; 11(1):209-13.

Lafeuillade, A.; Delbeke, E.; Chaffanjon, P.; Aubert, L.; Dhiver, C.; Gastaut, J. A. and Quilichini, R. (1990). Value of anticardiolipin antibody assay in human immunodeficiency virus infection. Presse med. 19(26):1225-1227.

Lederberg ,J.(2000).Infectious history .Science;288:287-293.

Ley,R.E., Peterson,D.A., and Gordon,J.I..(2006).Ecological and evolutionary forces shaping microbial diversity in the human intestine.Cell.,124 :837-

Norberg,R., Nived,O., and Sturfelt,G.(1987).Anticardiolipin and complement activation :relation to clinical symptums. J.Rheumatol.,14:149.

Sherer, Y., Blank, M. and Shoenfeld, Y.(2007).Antiphospholipid syndrome (APS): where does it come from ? Best Practice and Research Clinical Rheumotology, 21(6):1071-1078.

Skouri, H., Gandouz, R., Kraiem, I., Harrabi, L. and Bensaid, M.(2008). Antibodies to anionic phospholipid and co- factor in Kala-azar .Coparative study with malaria,toxoplasmosis and autoimmune diseases. Clinic.Exp. Rheumatol., 26(5):894-902.
Zandman, G., Berkun, Y., Barzilai, O., Boaz, M., Ram, M., Anaya, J.M., and Shoenfeld, Y. (2008). Neuropsychiatric lupus and infectious triggers . Lupus , 17:380-384 .
Zinger, H.; Sherer, Y.; Goddard, G.; Berkun, Y.; Brazilai, O.; Agmon- Levin, N.; Ram, M.; Blank, A.; Tincani, B.; Rozman, B.; Cervera, R. and Shoenfeld, Y. (2009). Common infections against prevalence in antiphosphplipid syndrome, Lupus, 18: 1149-1153.