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RESEARCH ARTICLE

Impact of Anti-*Toxoplasma gondii* and adipose hormones with Insulin Resistant on obese aborted women

Abeer J. Hassan[1], Nazar Sh. Mohammed[2], Muhannad Shweash[3], Hatem M. Hadeed[3]

[1]*Department of Medical Technology, Medical-Technical College, Baghdad, Middle Technical University (MTU), Iraq*
[2]*Department of Community, Medical-Technical Institute, Baghdad, Middle Technical University (MTU), Iraq*
[3]*Department of Clinical Laboratories Sciences, College of Pharmacy, University of Anbar Ramadi, Iraq*

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ABSTRACT

This study was carried out at Baghdad hospital in, for the period from November 2018 to July 2019. The study included (151) aborted obese women whose ages ranged between (18–41) years with positive *Toxoplasma gondii* infection. They were divided into two groups according to the body mass index (BMI) value: Group 1: consisted of 61 women with BMI <30, Group 2: consisted of (90) women with BMI >30. The control group included (52) healthy volunteer women aged 19–41 years with negative *Toxoplasma* for comparison of the results. The case and controls were matched for age and gender. Serum samples were tested for fasting blood sugar, insulin, IgG, and IgM of *Toxoplasma*, Leptin, and Adiponectin as well as insulin resistance index. The results showed that the age factor was not significant between group 1 and group 2 when compared with the healthy group, and there was no significant change between group 2 comparing to group1. In this study, the result of BMI showed substantial increase in group 1, while highly marked increase in group 2 when both groups were compared with the control group. Finally, the levels of *Toxoplasma* IgG and IgM antibodies showed a highly significant increase in the two patient groups in comparison with the control group. An increase in mean value of leptin concentration was noticeable in group 1 and group 2 with a highly significant difference when compared with the control group. No significant difference was found in the levels of fasting blood glucose in Group 1 and Group 2 compared to the control group. Also, a significant difference in HOMA-IR and QUICK- IR was observed in the patient groups once associated control group. Data revealed a considerable difference with the glucose/insulin ratio in group 1, but a highly significant was noticed in group 2 when compared with the control group. HOMA-AD results showed a significant difference in Group 1 and a highly significant decrease in Group 2.

Keywords: Abortion, Insulin resistance, Obese, *Toxoplasma gondii*.

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INTRODUCTION

T. gondii is intracellular pathogenic parasites that have the ability to infect mammalian nucleated cells through invading host macrophages as carrier vehicles.[1] Numerous signals secreted by adipose tissue due to its function as an endocrine organ, these including cytokines, growth factor and protein hormones such as leptin, adiponectin and resisting which have been stimulated both glucose and lipid metabolism[2] through many mechanisms the adipose tissue impair glucose metabolism and muscle adenosine triphosphate (ATP)synthesis via releasing adipokines, pro-inflammatory factors and free fatty acids (FFAs).[3] In addition, change insulin signaling,[4] through promote the synthesis of toxic lipid metabolites via stimulation of glucose uptake, suppression of triglyceride hydrolysis, and glycerol release with free fatty acids released into the bloodstream.[5] Adipose tissues are derived from pre-adipocytes and they are metabolically dynamic organs and are the primary sites for excessive energy storage. However, they act as endocrine organs able to synthesize many biologically active compounds that regulate the metabolic homeostasis.[6] Several types of research have focused on the two prototypic adipokines (leptin and adiponectin), which show beneficial effects on insulin action and lipid metabolism.[7] Insulin resistance (IR) is a situation that the target cells are unable to reply to normal circulating insulin levels. IR can also develop into another acquired condition and genetic like obesity, non-insulin-dependent diabetes, polycystic ovary syndrome, and metabolic syndrome.[8] In fatty tissues, insulin resistance is considered as a disorder of early and irreversible state that can explain to its association with adipocytes dysfunction

**Author for Correspondence: nazarnazar909@yahoo.com*

and disorder of peripheral tissue.[8] Many adipokines play key roles in IR development.[9] Changing insulin sensitivity in the target organs such as liver and muscles happened by the systemic or local function and their effects become obvious during immune mechanism and neuroendocrine autonomous. Moreover, adipose tissue stromal cell is included within the metabolism of sex hormones and glucocorticoids, influencing adipogenesis, metabolism of lipids and carbohydrates as well as cardiovascular functions.[8] Group of obese type 2 diabetic patients, adipose tissues are important sources for inflammatory factors due to its ability to secrete different cytokines by the adipocyte cells and its role in the allowed pro-inflammatory macrophages to infiltrate in the site of injury.[10] *Moreover, T. gondii* can disturb the metabolism of lipids through decreasing muscle lipase lipoprotein secretion and modifying the action of tissue lipoprotein lipase through chronic toxoplasmosis to improve the delivery of triglycerides into adipose tissues.[11,12] Individuals suffering from *T. gondii* infection have more risk factor in gaining obesity, then to the individuals are seronegative *T. gondii*.[13]

MATERIAL AND METHODS

Blood Sample Collection

Venous blood (5ml) samples were collected from each patient and healthy control using sterile disposable syringes. The blood samples were left for clotting at room temperature, and after separated by centrifugation for 10 minutes at (3000) rpm for serum collection. Serum samples were separated into 2 Eppendorf tube and kept at deep freeze (-20) until used. Serum samples were processed for the determination of fasting blood sugar, fasting insulin concentration, leptin, adiponectin, and anti toxoplasma antibody IgG and IgM levels.

Estimation of Toxoplasma IgG and Direct agglutination test (DAT)

To detect the suspected *Toxoplasma* infections in the samples collected from aborted women. The test is sensitive and easy, simple and rapid to perform. It measures both IgG and IgM Abs.

Estimation of Glucose by the Enzymatic Colorimetric Method

Basically, glucose oxidase used for the oxidized Glucose into D-gluconate with the formation of hydrogen peroxide. The production of this structure enhanced the oxidized of phenol and 4-amino antipyrine (4-AA) into red quinone imine dye related to estimating glucose level in all samples.

Estimation of Leptin Hormone

Serum leptin was estimated by the human leptin ELISA kit. The Demeditec ELISA for Leptin DEE007 is known with Sandwich-Assay designed to use 2 specific antibodies in more affinity. The first antibody is coated on a microtiter well plate to bind in leptin in the added samples and then immobilized leptin binds with the second specific anti-leptin antibody. The Biotinylated second antibody is applied in a mixture buffer with a Streptavidin-Peroxidase-Enzyme Conjugate. In the final substrate reaction time, stoop solution add, and the change of the color is analyzed quantitatively depending upon the leptin-amount in samples.

Estimation of Adeponectin Hormones

The Demeditec ELISA for Adiponectin DEE009 is branded as Sandwich-Assay. The same principles and methods were used for the estimation of adiponectin hormones as described above, with Demeditec ELISA for Leptin DEE007.

Homeostasis model Assessment–Adiponectin (HOMA-AD)

Homeostasis model Assessment–Adiponectin (HOMA-AD) index, an adapted form of the model, which includes the total serum adiponectin level in the denominator of the index, after that it adds an indirect adiposopathy measurement, and a final adjustment to the individual adiposity degree. The HOMA-AD index is established to be a crucial alternative indicator to detect IR among adult women and present similar demonstrations compared to the HOMA1-IR index.

Statistical Analysis

Data analysis made by SPSS Vr.24 programme, t–test and Monte Carlo tests (MCP) at 5% & 1% were applied for the determination of levels of significance.

RESULTS

The clinical characteristics of the patients and healthy control are presented in Table 1. The age factor showed a non-significant relationship between group 1 (28.43 ± 5.68) and group 2 (28.87 ± 5.97) when compared with the healthy control group (27.77 ± 6.01). The result of BMI in this table also displayed a substantial increase ($p < 0.05$) for group 1 (24.57 ± 3.61), while a highly significant increase ($p < 0.01$) was found in group 2 (32.38 ± 1.55) when compared with the control group (22.31 ± 1.30).

Table 1: Basic clinical data of the studied groups

	(Control) n = 52		*G1(BMI< 30) n = 61*		*G2(BMI>30) n = 90*	
Parameter	*Mean ± Std.*		*Mean ± Std.*	*p-value*	*Mean ±Std.*	*p-value*
Age	27.77	6.01	28.43 ± 5.68	0.89 NS	28.87 ± 5.97	0.001 NS
BMI	22.31	1.30	24.57 ± 3.61	0.032 S	32.3 ± 1.55	0.000 S
IgG	0.38	0.20	2.44 ± 1.59	0.000 HS	2.69 ± 2.46	0.000 HS
IgM	0.48	0.23	2.12 ± 1.13	0.000 HS	1.93 ± 1.30	0.000 HS

p-value < 0.01 is High significant, P value > 0.05 significant, p value < 0.05 is non significant.

Data in Table 2 revealed a marked increase ($p < 0.05$) in insulin level in group 1 (9.77 ± 6.94) comparing with a group of control (6.19 ± 2.78). In contrast, a highly significant difference ($p < 0.01$) was found between group 2 (18.4 ± 0.92) and the control group. There was an increase in mean leptin concentration values in group 1(1(29.32 ± 17.03) and group 2 (53.85 ± 7.01) with a marked significant difference ($p < 0.01$) when compared with the control group (7.31 ± 1.67).

Table 3 demonstrated no marked differences ($p > 0.05$) in the levels of fasting blood glucose in 1 (91.12 ± 10.74) and group 2 (92.52 ± 8.86) in comparison to control group (87.81 ± 10.16). Also, a significant difference ($p < 0.05$) was shown in HOMA-IR and QUICK- IR in Group 1 (2.22 ± 1.66) and (3.34 ± 0.64) respectively in comparison to control group (1.34 ± 0.63) and (3.32 ± 0.37) respectively. While a very significant difference ($p < 0.01$) was detected in group 2 (4.2 ± 0.47) and (2.73 ± 0.12), respectively when compared with the healthy group. Data in this table revealed a significant difference ($p < 0.05$) in glucose/insulin ratio in group 1 (16.18 ± 11.68), but a highly significant was noticed in group 2 (5.02 ± 0.52) when compared with the control group (16.85 ± 6.66)**,** while HOMA-AD results presented a significant difference ($p < 0.05$) in Group 1 (7.02 ± 4.39) and a highly significant decrease in group 2 (9.74 ± 3.91).

Correlation between hormonal levels and *Toxoplasma gondii* antibody in patient groups

Data in Table 4 demonstrated a significant negative correlation between Anti-*Toxoplasma* antibody and insulin levels in group 1 (r = –0.283,–0,317) ($p < 0.05$) and in group 2 with a non-significant negative correlation (r = – 0.001, –0.075) ($p > 0.05$). Also, a significant negative correlation was found between the toxoplasma antibody and leptin concentration in Group 1 (r = –0.314,-0.341) 9P <0.05), while in Group 2,

Table 2: Hormonal results (Leptin, Adeponectin, Adiponectin/Leptin, Insulin) between obese and overweight group compared to the control group.

	(Control) n = 52	*G1(BMI < 30) n = 61*		*G2 (BMI > 30) n = 90*	
Parameter	*Mean ± Std.*	*Mean ± Std*	*P-value*	*Mean±Std.*	*P value*
Insulin (mIU/L)	6.19 ± 2.78	9.77 ± 6.94	0.037 S	18.45 ± 0.92	0.000 HS
Leptin (ng/ml)	7.31 ± 1.67	29.32 ±17.03	0.000 HS	53.85 ± 7.01	0.000 HS
Adeponectin (mg/ml)	11.08 ± 2.99	4.37±2.78	0.000 HS	2.32 ± 0.91	0.000 HS
Adeponectin/leptin	1.62 ± 0.64	0.24 ± 0.22	0.000 HS	0.04 ± 0.02	0.01

p-value < 0.01 is High significant, p-value > 0.05 significant, p-value <0.05 is non significant

Table 3: Glycemic indices among studied groups

		G1(BMI < 30) n = 61		*G2(BMI > 30) n = 90*	
	(Control) n = 52	*Mean ± Std*	*Pvalue*	*Mean ± Std*	*Pvalue*
F.B.S (mg/dL)	87.81 ± 10.61	91.12 ± 10.74	0.218 NS	92.52 ± 8.86	0.008 HS
IR-HOMA	1.34 ± 0.63	2.22 ± 1.66	0.025 S	4.20 ± 0.47	0.000 HS
IR-QUICK	3.32 ± 0.37	3.34 ± 0.64	0.895 NS	2.73 ± 0.12	0.000 HS
Glucose/Insulin	16.85 ± 6.66	16.18 ± 11.68	0.606 NS	5.02 ± 0.52	0.000 HS
HOMA-AD	14.89 ± 8.27	7.02 ± 4.39	0.000 HS	9.74 ± 3.91	0.040 S

p value < 0.01 is High significant, P value >0.05 significant, P value <0.05 is non significant

Table 4: Correlation between hormonal levels and *Toxoplasma gondii* antibody in patient groups

		G1(BMI<30) n = 61		*G2(BMI>30) n = 90*	
IgG		*IgM*	*IgG*	*IgM*	
Insuline (mIU/L)	R	-.283*	-.317*	-.001	-.075
	Sig.	.027	.013	.996	.482
Leptin(ng/mL)	R	-.314*	-.341**	.219*	.385**
	Sig.	.014	.007	.038	.000
Adeponectin(mg/ml)	R	.208	.049	.011	.022
	Sig.	.107	.708	.920	.836
Adeponectin/leptin	R	.309*	.183	.026	.051
	Sig.	.015	.159	.805	.630

** Correlation is highly significant at the 0.01 level, *. Correlation is significant at the 0.05 level.

Table 5: Correlation between insulin resistances IR-HOMA, IR-QUICK, glucose/Insulin, HOMA- adeponectin with anti-*toxoplasma* antibodies

		G1(BMI<30) n = 61		*G2(BMI>30) n = 90*	
IgG		*IgM*	*IgG*	*IgM*	
IR-HOMA	r	-.274*	-.305*	.006	.037
	Sig.	.032	.017	.956	.731
IR-QUICK	r	.239	.322*	.009	.073
	Sig.	.063	.012	.931	.497
Glucose/Insulin	r	.239	.327*	.027	.115
	Sig.	.063	.010	.802	.279
HOMA - AD	r	-.193	-.408**	.016	.029
	Sig.	.137	.001	.883	.786

**. Correlation is significant at the 0.01 level, *. Correlation is significant at the 0.05 level

there was a positive correlation. Furthermore, adiponectin concentration.

Data in Table 5 revealed a significant negative relation among *T. gondii* antibodies and HOMA-IR in Group 1 (r = –0.274, – 0,305) ($p < 0.05$), while a significant positive relation was detected among Group 2 (r = – 0.006, –0.037) ($p > 0.05$) and the control Group. Also, a significant negative correlation was seen between Anti- *Toxoplasma* antibodies and HOMA-AD concentrations in group 1 (r = –0.193,-0.408) ($p < 0.05$), while in Group 2 there was a positive correlation with the control group. Furthermore, IR-QUICK showed a significant positive correlation in both groups 1 (r = 0.239, 0,322) ($p > 0.05$) and Group 2 (r = 0.009, 0.073) ($p > 0.05$) with Anti-*Toxoplasma* antibody levels.

DISCUSSION

Toxoplasma gondii is understudied pathogen in great interest of obesity researches. Experimentally, rats infected for 30 days inoculation with *T. gondii* exhibited a marked increase in weight and followed by weight loss within the next 60 days. Researchers suggested that increased weight occur because of some direct essential properties such as behavioral changes, like high food intake associated with *T. gondii* cysts infection in brain or directed dominant effects such as changes in hypothalamic functions including appetite regulation due to peripheral tissue inflammations. Weight associated effects of toxoplasmosis might be affected by *Toxoplasma gondii* strains. In additional *in vivo* animal study, there are two diverse *Toxoplasma* strain showed opposite impacts on the body weight (Kannan, G.,*et al*., 2010).[14] Reeves *et al*., demonstrated that people with positive results for *T. gondii* IgG showed nearly twice the chances of being overweight when compared with negative persons, while they could not decide if there is a causative association among obesity and *positive of T. gondii*.[11] The mean leptin value was noticeable in Group 1 (1(29.32 ± 17.03) and group 2 (53.85 ± 7.01) through an extremely significance ($p < 0.01$) differences after comparing with the group of control (7.31 ± 1.67). Leptin amount were high and relate to both Body Mass Index (BMI) and the percentage of body fat (Shah, N.R. and Braverman, E.R., 2012). In a study of (Nosaka, K., Hunter, M. and Wang, W., 2016)[15] they found that leptin hormone increases weight gain in obese individuals, but for non-obese people infected with toxoplasmosis also leptin hormone was increased in this group compared to the control, which is explained by the fact that leptin may be classified as a cytokine because of the similarity between leptin's structure and its receptors with cytokines. Leptin structurally resembles IL-2 in particular and is a crucial T-cell growth factor.[16] Circulating leptin concentrations is proportional to the size of fat mass (Kearns., *et al.,* 2006). Serum leptin increases predisposition to infections by decreasing the T-helper (Th) cells with direct effect on thymic functions.[17] Leptin has stimulating effects on Th1 cells and inhibiting effects on Th2 cells. In the context of the cellular immune response to infections, leptin plays a key role in the activation of Th1 cell and the elevation of IL-2, IFN-y and TNF-α level, which are produced by the Th1 cells. The secretion of both hormones is changed and characterized by a reduced adiponectin production, an increased leptin production as well as its association with the insulin resistance.[1] Furthermore, several recent studies showed that adiponectin/leptin and/or leptin/adiponectin ratios are highly correlated with surrogate measures of insulin resistance in comparison with those of adeponectin or leptin alone. Under an obesity-related condition, leptin level is greater and adeponectin level is lower, therefore, the Adeponectin/Leptin may be relatively low. Adeponectin plays a role in elevating the insulin receptor sensitivity as an anti-atherogenic and anti-inflammatory. The results of this study were in agreement with a previous study which indicated that the plasma concentration of adeponectin is correlated highly with sensitivity of insulin which recommends that a low level of adeponectin is associated with insulin resistance.[18] The HOMA-AD guide is shown to be a beneficial alternative indicator for IR detection between adult females and exhibited a comparable action when compared to the HOMA–IR guide.[19] Until now, there is no studies in Iraq examined the relation between HOMA–AD index regarding to IR assessment. The result for HOM–AD is considered a useful marker which may assist physicians and researchers in determining IR. The homeostasis model assessment for IR (HOMA-IR) index was commonly trailed as a measure tool of IR in clinical practice studies and many studies of epidemiology.[20] It was positive correlations with HOMA-IR, QUICK_IR, Glucose/Insulin concentrated were found in group 1, while in Group 2, there was a negative correlation. Furthermore, Adeponectin/Leptin showed a significant negative correlation with insulin in Group 1 and a positive correlation with HOMA-IR, QUICK-IR, and Glucose/Insulin in Group 1. These findings agreed with

Shin MJ, Park E, Lee JH, and Chung N. 2006, who found that many studies reported that lipid peroxidation was increased by insulin resistance and compensatory hyperinsulinemia, indicating the linkage to each other. We found an association between the plasma antioxidant condition, lipid peroxidation and the insulin resistance index HOMA-IR in non-diabetic patients with hypercholesterolemia.[21,22]

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