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# Lipid Profile Parameters and Adipokines among Adolescents Infected with Toxoplasmosis

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**Abstract**

The lipid profile and adipokines of an adolescent may be affected by some parasite infections. Recently, it has been discovered that these parasites are connected to body mass index (BMI), lipids and adipokines. The current study, therefore, aimed to specify how *Toxoplasma gondii* (*T. gondii*) affect BMI, lipid profile and adipokines. This study was conducted in Al Madain hospital, Baghdad from October to December 2022. An ELISA test was performed to examine the anti-*T. gondii* IgG and IgM for a group of adolescents attending the hospital. Based on this examination ninety adolescents were chosen to be involved in the study. They were separated in to two groups: individuals who tested positive for the parasite (n=45) and those who tested negative for the parasite, control group (n=45). All participating adolescents were subjected to some measurements which included body mass index (BMI), lipid profile parameters (cholesterol, triglycerides, HDL, LDL, and VLD) and adipokines (adiponectin and chemerin). Significant high BMI was noticed in *T. gondii* positive group (29.65±0.6 Kg/m$^2$) compared to control. The results also indicated that there was a significant increase in all tests of the lipid profile among *T. gondii* positive group compared to the control. However, the matter reversed in HDL examination as its level increased significantly in the control compared to the *T. gondii* positive group. Finally, both adiponectin (78.6±0.037 ng/ml) and chemerin (3.43±0.1 ng/ml) raised up significantly in *T. gondii* positive group versus control group.

**Keywords:** Adiponectin, Adolescents, Chemerin, Toxoplasmosis

# مؤشرات ملف الدهون والحركيات الدهنية لدى المراهقين المصابين بداء المقوسات الكوندية

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**الخلاصة**

ان ملف الدهون و الحركيات الدهنية يمكن ان يتأثر ببعض الطفيليات. مؤخراً، تم اكتشاف علاقة بين الاصابة بهذه الطفيليات وكل من مؤشر الكتلة الحيوية ، مؤشرات ملف الدهون و الحركيات الدهنية. وعلى هذا الاساس هدفت الدراسة الحالية الى تحديد تأثير الاصابة بداء المقوسات الكوندية على هذه المؤشرات. اجريت الدراسة في مستشفى المدائن–بغداد من تشرين الاول و لغاية كانون الاول من العام 2022.تم اجراء فحص الامتزاز المناعي المرتبط بالانزيم للكشف عن الاضداد المضادة لطفيلي المقوسات الكوندية لمجموعة من

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المراهقين الذين راجعوا المستشفى اعلاه. اعتمادا على هذا الفحص تم اختيار 90 منهم ليتم شمولهم بالدراسة. تم تقسيمهم الى مجموعتين :مجموعة اظهروا فحصا موجبا لطفيلي المقوسات (العدد: 45) ومجموعة اظهروا فحصا سالبا للطفيلي (العدد:45) و الذين تم عدهم مجموعة سيطرة. خضع كل المشاركين في الدراسة لبعض الفحوصات و التي تضمت قياس مؤشر الكتلة الحيوية، مؤشرات ملف الدهون (الكوليسترول، الدهون الثلاثية، البروتين الشحمي عالي الكثافة، البروتين الشحمي قليل الكثافة، البروتين الشحمي قليل الكثافة للغاية) و الحركيات الدهنية (الاديبونكتين و الكيميرين). اظهر مؤشر الكتلة الحيوية ارتفاعا معنوياً لدى الموجبين لفحص طفيلي المقوسات (29.65±0.6 كغم/م$^2$) مقارنة بمجموعة السيطرة. كما ظهر ارتفاع معنوي في كل مؤشرات ملف الدهون لدى الموجبين لطفيلي المقوسات مقارنة بالسيطرة خلافا لمستوى البروتين الشحمي عالي الكثافة الذي كان مرتفعا و بشكل معنوي لدى الموجبين لطفيلي المقوسات. من جهة اخرى اظهر كل من الاديبونيكتين (78.6±0.037 نانوغرام/مل) و الكيميرين (3.43±0.1 نانوغرام /مل) مستويات مرتفعة و بشكل معنوي لدى الموجبين لطفيلي المقوسات مقارنة بالسيطرة .

## 1. Introduction

*Toxoplasma gondii* (*T. gondii*) is an agent that causes the zoonotic disease toxoplasmosis. The protozoan parasite *T. gondii* is an obligate intracellular member of the phylum Apicomplexa and the order Eucoccidioridia [1]. It is estimated that this parasite infects up to one-third of all people on earth [2]. *T. gondii* has both sexual and asexual phases in its life cycle. The "definitive" hosts are cats as they are the only animals who undergo the sexual life cycle. In the cat gut epithelium, *T. gondii* develops into male and female gametocytes, facilitating sexual reproduction. Cats are capable of releasing millions of sporulated oocyst, each of which contains one pair of sporocysts, and inside each sac there are four sporozoites [3]. Many methods of transmission, including ingestion of raw meat, vertical transmission from an infected mother, exposure to oocytes in cat faeces, blood transfusions, and organ transplants, can cause human infection [3]. Toxoplasmosis is asymptomatic and is often associated with short self‑limiting illnesses in immunocompetent individuals [4]. There have been some studies on the adolescent's age group. In one of these studies, which was carried out in Italy between 1987 and 1991, 48.5% of participants had IgG antibodies against *T. gondii* [6]. Another study in young Iranians found that 56.3% of the subjects had toxoplasmosis [5]. In a different study from Pakistan, lgG *T. gondii* antibodies were looked for in schoolchildren living in the suburbs of Islamabad and 17.4% of particpants were found to be affected overall [6]. With numerous research, toxoplasmosis has recently received a lot more attention in Iraq. Many researchers have been interested in the role of serum biochemical parameters in humans infected with some parasites, including the levels of cholesterol, triglycerides, high density lipoprotein (HDL), very low-density lipoprotein (VLDL), low density lipoprotein (LDL), total protein, total globulin and total albumin [7]. Some parasites can develop on lipid-rich medium without serum, according to *in vitro* research [8]. Adipokines, often referred to as adipocytokines, are cytokines made by adipose tissue that play a part in the body's energy and metabolism, inflammation, obesity and other processes. Leptin, adiponectin, interleukin-6 and other substances are examples of adipokines [9]. Adipokines, including adiponectin and chemerin, are produced and secreted by adipocyte tissues [10]. The feature that sets adiponectin apart from the majority of adipokines is that it has a negative relationship with obesity. It is primarily produced in white adipose tissue [11]. Due to the production of numerous cytokines, adipose tissue is now recognized as an endocrine organ in addition to being a storage organ. Adipokines are associated with a variety of physiological and pathological processes in the human body. In addition to adipose tissue, chemerin is expressed in the liver, adrenal glands, lungs, fibroblasts, chondrocytes, pancreas, kidney, epithelial cells and platelets [14]. Numerous earlier research in Iraq have noted the seroprevalence of *T. gondii* in a number of certain populations such as women who are

pregnant, haemodialysis patients, immunocompromised individuals, blood donors, etc [12, 13, 14, 15]. Hence, the prevalence of *T. gondii* in Iraq is higher than it is in the neighbouring nations [16],[17]. This study set out to assess the adipokines and lipid profile characteristics in teenagers who had been exposed to *T. gondii*.

## 2. Materials and Methods:

### 2.1 Individuals and Study Design

This study was a case control study achieved between October to December 2022, at Al-Madain Hospital, Baghdad-Iraq. ELISA test was performed to detect anti-*T. gondii* antibodies on a group of adolescents who attended this hospital. They provided serum samples which were then tested using the " IgG and IgM" enzyme immunoassay kit for anti-*T. gondii* IgG and IgM antibodies (Bioassay Technology Laboratory, China). The procedure was completed following the manufacturer's instructions. Based on this examination, ninety individuals were selected to be involved in the study. They were divided in to two groups: individuals who tested positive for the parasite (n=45) and control group, individuals who tested negative for the parasite (n=45). There were 45 (50%) males and 45 (50%) females with ages ranging between 14 to 19 years. The local ethics committee of the College of Science, University of Baghdad, authorized the study protocol (Ref. : CSEC/1022/0124). Also, each participant consented to taking part in the study, going through all examinations and providing the necessary information.

### 2.2 Body Mass Index Calculation

Both weight and height were measured using an electronic weighing scale and a free-standing portable stadiometer. Each participant was requested to remain motionless on the scale while their weight was recorded. When the participants were weighed, they had no shoes on. BMI was calculated by dividing the weight (kg) by the square of the height (m) [18].

### 2.3 Lipid Profile

All individuals' total cholesterol, triglycerides, high density lipoprotein (HDL), low density lipoprotein (LDL) and very low-density lipoprotein (VLDL) levels were assessed using a lipid profile kit (Linear Chemicals in Barcelona, Spain). All evaluations were conducted in accordance with the manufacturer's instructions.

### 2.4 Adiponectin and Chemerin Assessment

According to the instructions provided by the manufacturer, sandwich enzyme-linked immunosorbent assay (ELISA) kits (Bioassay Technology Laboratory, China) were used to measure adiponectin and chemerin levels. A 96-well plate was used to analyse all samples. The plate was then examined using an ELISA reader at 450 nm wavelength.

### 2.5 Statistical Analysis

The data was statistically analysed using SAS 9.1 (Statistical Analysis System).
Chi square ($X^2$) was used to analyse the distribution of IgG and IgM among *T. gondii* positive adolescents. As well as the mean BMI, cholesterol, triglycerides, HDL, LDL, VLDL, adiponectin and chemerin levels were compared between the *T. gondii* positive and the control groups, using the student T-test. Statistical significance was defined as a *P-value* of less than 0.05. The mean and standard deviation (mean ±SD) was used to express the data.

## 3. Results

The results of anti-*Toxoplasma* IgG and IgM of the infected adolescents with *T. gondii* (n=45) is presented in Table 1. Significant difference (*P<0.05*) was reported in the distribution of anti-*Toxoplasma* IgG and IgM. The majority (95.55%) of them were IgG+ and IgM+ cases. Two of them (4.45%) were IgG- IgM+ cases. While no cases of IgG+ IgM- were noticed. On other hand, results showed that the BMI differed between *T. gondii* positive and the control groups. BMI was significantly (*P<0.05*) higher (29.65±0.6) in those adolescents who had positive antibodies against *T. gondii* versus low BMI in the control group (22.29±0.5) (Table 2).

Findings revealed significant differences (*P<0.05*) in cholesterol levels between the *T. gondii* positive and the control groups (Table 3). Adolescents who tested positive for anti-*T. gondii* antibodies had the highest mean cholesterol levels (205.84±0.7 mg/dL) versus (195.2±1.1 mg/dL) the control group. The results of triglyceride also varied significantly (*P<0.05*) between *T. gondii* positive group and control. *T. gondii* positive group showed the highest mean of triglycerides (207.38±0.5mg/dl) versus the control group (191.18±1.1 mg/dL) (Table 4).

**Table 1:** The distribution of anti-*Toxoplasma* IgG and IgM among infected group (n=45)

| ***Groups*** | ***N (%)*** | ***$X^2$ (p-value)*** |
|---|---|---|
| All | 45 | - |
| Anti *Toxoplasma* IgG+ IgM+ | 43 (95.55%) | 78.53(0.00) |
| Anti-*Toxoplasma* IgG- IgM+ | 2 (4.45%) | |
| Anti-*Toxoplasma* IgG+ IgM- | 0 (0.00%) | |

**Table 2:** BMI of *Toxoplasma gondii* positive group and the control group

| ***Groups*** | ***BMI (Mean ± SD) Kg/$m^2$*** | ***T-test*** | ***P-value*** |
|---|---|---|---|
| *Toxoplasma gondii* positive | 29.65±0.6 | 7.8 | <0.05 |
| *Toxoplasma gondii* negative (Control) | 22.29±0.5 | | |

**Table 3:** Cholesterol level of *Toxoplasma gondii* positive group and the control group

| ***Groups*** | ***Cholesterol (Mean ± SD) mg/dL*** | ***T-test*** | ***P-value*** |
|---|---|---|---|
| *Toxoplasma gondii* positive | 205.84±0.7 | 7.79 | <0.05 |
| *Toxoplasma gondii* negative (Control) | 195.2±1.1 | | |

**Table 4:** Triglycerides level of *Toxoplasma gondii* positive group and the control group

| ***Groups*** | ***Triglycerides (Mean ± SD) mg/dL*** | ***T-test*** | ***P-value*** |
|---|---|---|---|
| *Toxoplasma gondii* positive | 207.38±0.5 | 13.6 | <0.05 |
| *Toxoplasma gondii* negative (Control) | 191.18±1.1 | | |

Findings showed that the control group's HDL level significantly increased (P<0.05). They had an HDL level of 48.94 0.7 mg/dL. HDL levels were determined to be lower in the *T. gondii* positive group than in the control group (Table 5). The results of LDL varied

significantly (P<0.05) between the two groups. *T. gondii* positive group revealed the highest mean of LDL (129.70±1.8mg/dl) compared control group (108.15±1.4mg/dl) (Table 6).

**Table 5:** HDL level of *Toxoplasma gondii* positive group and the control group

| *Groups* | *HDL (Mean ± SD) mg/dL* | *T-test* | *P-value* |
|---|---|---|---|
| *Toxoplasma gondii* positive | 34.66±1.7 | 7.9 | <0.05 |
| *Toxoplasma gondii* negative (Control) | 48.94±0.7 | | |

**Table 6:** LDL level of *Toxoplasma gondii* positive group and the control group

| *Groups* | *LDL (Mean ± SD) mg/dL* | *T-test* | *P-value* |
|---|---|---|---|
| *Toxoplasma gondii* positive | 129.70±1.8 | 9.3 | <0.05 |
| *Toxoplasma gondii* negative (Control) | 108.15±1.4 | | |

Results also revealed that the VLDL level increased significantly (P<0.05) in *T. gondii* positive adolescents while it was lower in control group. Their VLDL means were 41.47±0.1mg/dL and 38.23±0.2mg/dL respectively (Table 7).

**Table 7:** VLDL level of *Toxoplasma gondii* positive group and the control group

| *Groups* | *VLDL (Mean ± SD) mg/dL* | *T-test* | *P-value* |
|---|---|---|---|
| *Toxoplasma gondii* positive | 41.47±0.1 | 13.06 | <0.05 |
| *Toxoplasma gondii* negative (Control) | 38.23±0.2 | | |

Results presented significant increase in serum adiponectin concentration among *T. gondii* positive group compared to control group. Their adiponectin concentration was 78.6±0.037 ng/ml and 31.94±0.032 ng/ml respectively (Table 8). The results of chemerin did not differ from the results of adiponectin as *T. gondii* positive group had the highest mean of chemerin (3.43±0.1 ng/ml) versus low level in control group (1.75±0.06 ng/ml) (Table 9).

**Table 8:** Adiponectin level of *Toxoplasma gondii* positive group and the control group

| *Groups* | *Adiponectin (Mean ± SD) ng/ml* | *T-test* | *P-value* |
|---|---|---|---|
| *Toxoplasma gondii* positive | 78.6±0.037 | 20.86 | <0.05 |
| *Toxoplasma gondii* negative (Control) | 31.94±0.032 | | |

**Table 9:** Chemerin level of *Toxoplasma gondii* positive group and the control group

| *Groups* | *Chemerin (Mean ± SD) ng/ml* | *T-test* | *P-value* |
|---|---|---|---|
| *Toxoplasma gondii* positive | 3.43±0.1 | 12.12 | <0.05 |
| *Toxoplasma gondii* negative (Control) | 1.75±0.06 | | |

## 4. Discussion

This study demonstrated that majority cases of Toxoplasmosis were IgG+ IgM+, while two cases only of IgG- IgM+ were found among *Toxoplasma* positive group. The presence of IgM but not IgG indicated that these cases were acute. Subacute toxoplasmosis occurred if both antibodies were detected, however chronic infection requires the absence of IgM [19]. IgM

can persist for several months to years after an acute infection, thus making the distinction between an acute and a chronic infection challenging [20].

The distribution of anti-*Toxoplasma* antibodies depends on different factors such as sample size, social and cultural habits, geographic factors, climate, transmission route and age differences [19]. This study produced intriguing findings that supported *T. gondii*'s alteration of some metabolic markers. As well as it indicated that body mass index could correlate with *T. gondii* positive status. High BMI and anti-*Toxoplasma* seropositive status were found to be related. This finding is consistent with that of [21] who discovered a correlation between obesity and a high concentration of anti-*T. gondii* antibodies, while other investigations [22, 23, 24] found no evidence of a connection between toxoplasmosis and obesity. The findings of the current study contradict those studies. On other hand, the lipid profile results showed an increase in cholesterol and triglyceride levels as well as a degree of lipid peroxidation marked by a significant increase in HDL. Numerous researchers have been interested in finding the correlation between cholesterol levels in persons who have certain parasite infections. As *T. gondii* cannot produce cholesterol on its own, it must obtain LDL-derived cholesterol from the host cells via LDL-mediated endocytosis or protein associated with the LDL receptors [25]. *T. gondii* entrance into cells could be regulated by a mechanism that involves host cholesterol rather than parasite cholesterol [26]. According to these findings, cholesterol plays a crucial role in the pathophysiology of *T. gondii* infection. However, information on the lipid basis of *T. gondii* is extremely few and the molecular mechanisms by which Toxoplasma recovers cellular lipids are largely unknown [27]. Those with protozoan infections had elevated lipoprotein concentrations, according to earlier research [28]. A few additional illnesses, such as HIV infection and antiviral medication, have been linked to changes in these lipoproteins [29]. Some researchers found contradictory results which may be related to the chronic character of the condition and that was largely the case in our investigation. Triglyceride levels increased slightly while HDL and LDL levels fell in case of acute infection [30]. Another study found that acute phase HDL was altered, with decreased HDL and antioxidant activity as well as other structural and compositional changes and interactions with inflammatory proteins [31]. According to a different study, the rapid acute phase response associated with hypertriglyceridemia may be to responsible for the HDL reduction [32]. The features of the HDL in the chronic and acute stages of toxoplasmosis require more study. Adiponectin level was significantly higher in *T. gondii* positive group compared to the control group. As showed previously, the mean of BMI in the positive group was significantly higher as well. Adolescents who have high BMI may develop metabolic syndrome as a result of *T. gondii* infection which may be related to the pro-inflammatory cytokines-adipocytokine-metabolic connection. This connection was demonstrated by higher levels of the proinflammatory adipokines and a dominating Th1-cytokine profile [10]. Increasing adiponectin levels in some individuals are linked to greater weight gain. Adipocytes' increased production of adiponectin may indicate "healthy" adipose tissue with increased potential to retain fat [33]. More over results of the current study revealed that chemerin increased significantly in those who had positive anti-*T. gondii* antibodies. This result confirms the previous result concerning adiponectin. As an adipokine, chemerin is a chemoattractant protein released by adipocytes and is linked to several chronic infections and inflammations [34]. *T. gondii* infection may have an impact on the onset of metabolic syndrome in adolescents with high BMI which may be related to the pro-inflammatory cytokines-adipocytokine-metabolic link. The dominant Th1-cytokine profile, defined by IFN- production as a marker of low-grade chronic inflammation, elevated proinflammatory adipokine levels which are mirrored by elevated serum chemerin levels, and the generation of insulin were used to demonstrate this link [12]. This result agrees with the result of [35] who

demonstrated that the chemerin levels of the *T. gondii* IgG seropositive group were higher than those of the *T. gondii* IgG seronegative group. Based on the aforementioned finding, we can conclude that the positive status of the *T. gondii* parasite can be associated with an increase in biomass as it can change lipid levels and increase the level of adiponectin and chemerin.

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