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Biochemical and Hormonal Alterations in Toxoplasma Gondii-Infected Women: Evidence from Kirkuk City, Iraq

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Abstract: Toxoplasmosis is a zoonotic disease caused by an obligate intracellular parasite, Toxoplasma gondii, that infects all warm-blooded animals except insects and the definitive host is members of the family Felidae. The present study was designed to assess the effect of T. gondii infection on some biological and biochemical markers (dopamine, oxytocin, adiponectin, and chemerin) and also lipidogram parameters including lipid profile, Ca and Cu. During the period between 1st of June to 1th of July 2024, 50 of infected women and 30 of healthy controls were enrolled in Al-Jumhuri General Hospital, Kirkuk city. Results: The level of oxytocin, adiponectin, chemerin, total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), and very low density lipoprotein (VLDL) and dopamine were significantly high in the patients infected while triglycerides (TG), low density lipoprotein cholesterol (LDL-C), calcium, and copper were significantly low in infected patients ($P \leq 0.05$).These results show that T. gondii infection induces major changes in neuroendocrine control, regulation of adipokines, and lipid–mineral metabolism during toxoplasmosis, emphasizing the relevance of these biomarkers on diagnosis and comprehension about T. gondii infection.

Keywords: Toxoplasma gondii, dopamine, oxytocin, adiponectin, chemerin, lipid profile, calcium, copper

Introduction

Toxoplasmosis is the most common zoonosis increasingly transmitted by *Toxoplasma gondii*, an obligate intracellular protozoan parasite. Warm-blooded creatures like humans perform the role of intermediate hosts, and members of the cat family are definitive hosts, which enable sexual reproduction of the parasite (Mahmood et al., 2022). In *T. gondii*, migration through numerous organs occurs, including muscle and the gastrointestinal epithelium. Infection is acquired or congenital, with the latter being severe, especially when maternal infection occurs in early pregnancy (Mohammed, 2011). It is known that the transmission to humans generally happens via consumption of food or water contaminated with oocysts released in cat feces, but also by ingestion of raw or undercooked meat harboring tissue cysts. Vertical transmission from infected mothers to their fetuses is also a major route of infection (Addo et al., 2023). An estimated one-third of the world's human population is infected with *T. gondii*, and clinically the disease varies from asymptomatic in immunocompetent hosts to severe and fatal in immunosuppressed patients (Ali et al., 2022; Hiro, 2016).

Oxytocin is one of the neurohormones which was studied in relation to toxoplasmosis. It is produced in the hypothalamus and released by the posterior pituitary, controlling reproductive events, parturition, and postpartum function (Audunsdottir & Quintana, 2022). It has been demonstrated in the past that *T. gondii* infection could alter oxytocin signaling at the brain level, resulting in increased levels of circulating (Samira & Ajai, 2021). Another important neurotransmitter is dopamine, which makes up roughly 80% of the brain's catecholamine levels and serves as a powerful signaling compound between neurons in the brain and plays a crucial role in mood, attention, and voluntary movement (Tannin et al.).Other than neurohormones, adipokines, including chemerin and adiponectin, have also been identified to play roles in connecting infection with metabolism and the immune response. Chemerin is an immune cell attractant and an adipose tissue protein with a role in metabolism. Then again, Chemerin have been related to obesity and chronic inflammatory state (Shi et al., 2016; Iskandar et al., 2017; Haberl et al., 2018; Ba et al., 2019; Buechler et al., 2019). The most abundant adipokine in human circulation, adiponectin, a 224-amino-acid protein secreted by white adipose tissue (WAT), also has protective roles in metabolic and inflammatory processes [6]. While expressed to a lower level than in adipose tissue, it is also found in various tissues including bone, liver, skeletal muscle and placenta (Modagan et al., 2018; Tanabe et al., 2020; Khoramipour et al., 2021). In addition, mounting evidence suggest that trace minerals including selenium and zinc and acutely toxic elements from heavy metals regulate immunity and host susceptibility to parasitic infection [3–6]. Moreover, the interactions of selenium with other trace and toxic elements in sheep supplementary Se have been proven a vital role of selenium in not only the animal health but also for the environmental protection (Palani et al., 2019). It highlights the broader importance of mineral and metabolic pathways in host–parasite synchronisation. Therefore, the present work was designed to study the effect of Toxoplasma gondii infection on some biochemical and physiological markers like dopamine, oxytocin, adiponectin, and chemerin, in addition to lipid profiles as cholesterol and triglycerides, calcium, and copper to clarify the possible role of these parameters in diagnosis or in understanding of toxoplasmosis.

Materials and Methods

Study design and participants

A cross-sectional study at Al-Jumhuri General Hospital, Kirkuk, Iraq from 1st of June 2024 to 1st of July 2024. Eighty women of reproductive age (20-40 years) were divided into 50 with

diagnosed T. gondii and 30 healthy women (control group). Clinical features and laboratory data were used to diagnose the case. Each participant provided written informed consent before enrolment.

Sample collection and preparation

From all the participants, blood samples were collected in sterile conditions and specifically venous blood samples. Blood was allowed to clot and the supernatant was harvested following centrifugation to obtain serum. The serum was aliquoted and stored following standard operating procedures until analysis.

Biochemical and hormonal assays

We were directed to assess the influence of T. gondii infection on the level of selected physiological markers such as dopamine, oxytocin, adiponectin, chemerin, calcium, and copper. Levels for these parameters were examined using standard commercially available ELISA kits (Cloud-Clone Corp., USA) per manufacturer instructions.

Lipid profiles were determined as follows:

- **Total cholesterol (TC):** was determined by the method of Allain et al. (1974).
- **Triglycerides (TG):** were measured using a method by Fossati and Prencipe (1982).
- **HDL-C:** estimated using the procedure of Gotto (1988).
- **LDL-C and VLDL:** estimated by using Friedewald formula (Friedewald et al., 1972).

Statistical analysis

All statistical analysis was done using Statistical Package for Social Sciences (SPSS), version 27. Values were presented as mean ± SD. The Independent-Samples t-test was performed to compare between infected patients and healthy cases. A P-value < 0.05 was considered as statistically significant.

Results

The objective of the present study was to determine serum neurohormones, adipokines, lipid profile parameters, and some minerals in women with T. gondii infection as compared to the healthy control group. The descriptive statistics (mean ± SD) are presented in Table 1, and group comparisons are shown in Figures 1–11.

The serum oxytocin, adiponectin, and chemerin were significantly ($P \leq 0.05$) increased, while total cholesterol (T.C), high-density lipoprotein cholesterol (HDL-C), and very low-density lipoprotein cholesterol (VLDL) of infected groups were significantly higher than the control group. Nevertheless, patients also demonstrated a significant decrease ($P \leq 0.05$) in dopamine, T.G, LDL-C, Ca, and Cu compared to the control.

The mean ± SD of various parameters is also indicated in Table 1, from which the level of dopamine declined significantly from 632 ± 14.87 pg/mm to a significantly lower value of 265.4 ± 11.42 pg/mm. In contrast, the level of oxytocin increased nearly threefold from 110 ± 25.34 pg/ml in healthy individuals to an average of 423 ± 20.78 pg/ml in infected ones. Adiponectin and chemerin were 1.7 times higher in the infected women compared to the controls.

Lipid Profile:

- T.C (mg/dl): 145.76 ± 10.32 vs. 177.87 ± 7.87
- HDL-C (mg/dl): 25.78 ± 3.54 vs. 35.89 ± 4.87
- VLDL (mEq/L): 46.87 ± 6.54 vs. 69.67 ± 14.7

However, in this group, there was a decreased concentration of T.G levels (156.89 ± 3.76 vs. 149.76 ± 6.65 mg/dl) and a lower LDL-C for diabetic patients (85.81 ± 14.15 mg/dl).

Parameter	Control (n=30) Mean ± SD	Patients (n=50) Mean ± SD
Dopamine (pg/ml)	632 ± 14.87	265.4 ± 11.42
Oxytocin (pg/ml)	110 ± 25.34	423 ± 20.78
Adiponectin (ng/ml)	30.67 ± 5.43	65.8 ± 6.87
Chemerin (ng/ml)	2.87 ± 0.98	4.56 ± 1.20
T.C (mg/dl)	145.76 ± 10.32	177.87 ± 7.87
T.G (mg/dl)	156.89 ± 3.76	149.76 ± 6.65
HDL-C (mg/dl)	25.78 ± 3.54	35.89 ± 4.87
LDL-C (mg/dl)	85.87 ± 5.31	78.98 ± 5.66
VLDL (mg/dl)	46.87 ± 6.54	50.67 ± 7.41
Ca (ng/ml)	9.45 ± 1.35	6 ± 1.76
Cu (ng/ml)	29.65 ± 3.54	34.87 ± 3.44
Note: Significant differences were considered at $P \leq 0.05$.		

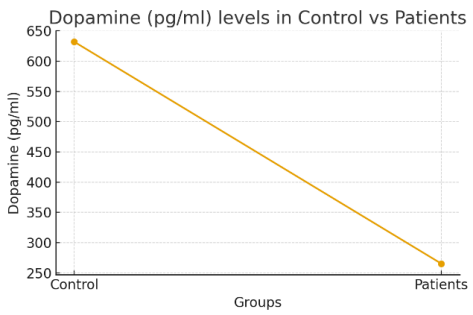
Table 1. Mean ± SD of biochemical and hormonal parameters in control and Toxoplasma gondii-infected patients

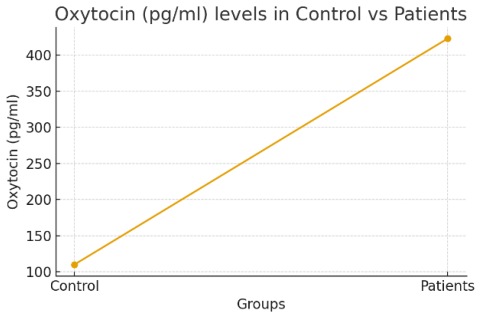
Minerals:

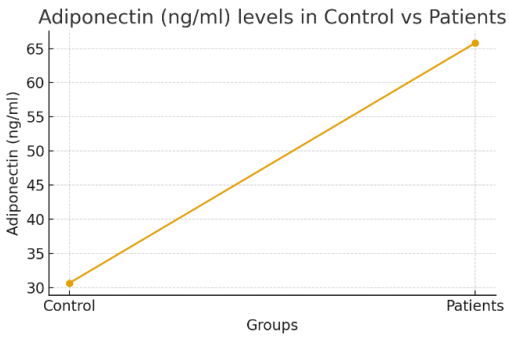
- Calcium (ng/ml): 9.45 ± 1.35 vs. 6 ± 1.76
- Copper (ng/ml): 29.65 ± 3.54 vs. 34.87 ± 3.44

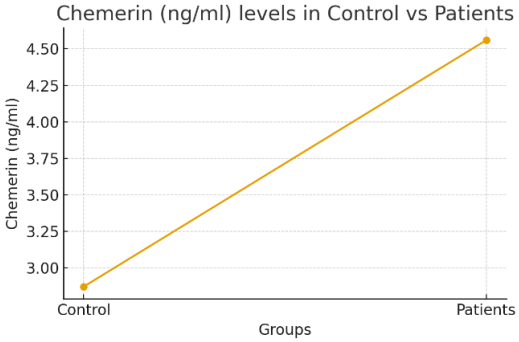
All these differences are visible in Figs. 1 (Dopamine), 2 (Oxytocin), 3 (Adiponectin), 4 (Chemerin), 5 (TC) Total cholesterol, 6 (TG) Triglycerides, data refer to Figure 7, 8 (VLDL), 9 (Calcium), and 10 (Copper).

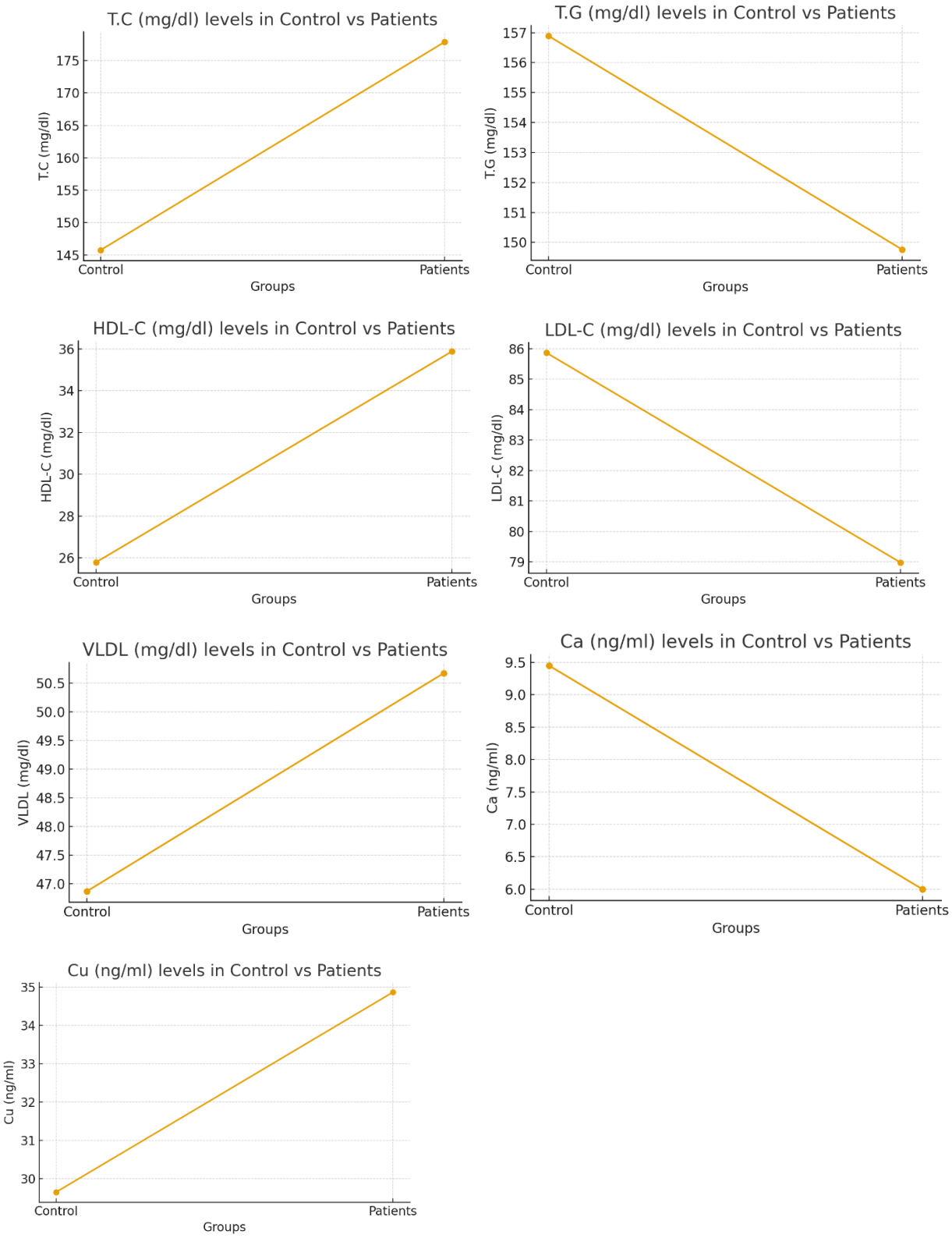
Collectively, these results indicate that T. gondii infection markedly alters the profiles of hormones, metabolites, and minerals in infected women.











Figures 1–11. Serum biochemical and hormonal profiles in *Toxoplasma gondii*–infected patients compared with healthy controls.

Discussion

The results of the present study confirmed increased levels of oxytocin in infected women vs. negative controls. This finding is consistent with those of Abdulai and Vyas (2021) and Erzaiq (2023), who found higher levels of oxytocin in toxoplasmosis patients. Such elevation could be related to hypothalamic–amygdala signaling circuits, for example, from the paraventricular nucleus (PVN) to the medial dorsal amygdala

(Ferguson et al., 2000), or it may reflect parasite-induced modulation of oxytocin release during early labor (Nakajima et al., 2014).

However, as opposed to Mirzaeipour et al. (2021) and Rahdar et al. (2023), the level of this neurotransmitter was significantly decreased in the infected females. They described an elevation in dopamine release during chronic, but not acute, toxoplasmosis. These differences could be due to strain variation, stage of disease, or the effect of drug treatment (Prandovszky et al., 2011; Blader et al., 2001; Skallova et al., 2006). Changes in dopamine pathways might underlie some of the behavioral and psychiatric symptoms observed in infected individuals.

For adipokines, the current findings showed higher levels of adiponectin and chemerin in infected women, results that also concur with those obtained by Yousif (2024). Elevated adiponectin may be linked to metabolic adaptation and fat accumulation (Hivert et al., 2013), whereas chemerin is reportedly associated with inflammation and chronic infections (Mancuso, 2016). These results introduce T. gondii infection into the adipocyte-related signaling pathways (Abdul-Aziz & Zghair, 2014).

Lipid profiles showed increased levels of total cholesterol, HDL-C, and VLDL, but decreased triglycerides and also LDL-C. These findings are consistent with those of Al-Kuraishi (2013) and Al-Dori (2022), but conflict with the report of Ali (2021), which showed lowering effects on cholesterol and HDL-C. It has been demonstrated that lipid metabolism is essential in host–parasite interactions because T. gondii relies on host lipids for replication and survival (Crook, 2012; Feingold & Grunfeld, 2012). Cholesterol, in particular, is important for parasite entry into host cells (Coppens et al., 2000; Coppens & Joiner, 2003).

Regarding minerals, serum calcium showed a significant decrease in the infected group as compared with control patients, which was consistent with Al-Zuhairy (2008). This decrease in calcium could be due to parasite use of host calcium as some parasites multiply (Bouchot et al., 1999; Pingret, 1996). On the other hand, copper levels were higher, similar to Seyrek (2004). Elevations could potentially be due to increased ceruloplasmin production during inflammation (McLaren-Howard & Grant, 1998). Notably, the expanded function of heavy metals in host health and disease is emerging from recent research. For instance, the cooperative and antagonistic response of selenium and zinc has shown to affect animal health (Palani et al., 2022a), whereas changes in the content of heavy metals in animal manure may also affect human health along with the environment (Palani, et al 2022b).

In addition, parasitic infections may not only affect biochemical parameters within an endemic region due to toxoplasmosis. Studies, such as Qadir et al. As demonstrated in the results of a study conducted in 2022 (7), checking the existence of Giardia lamblia in Sulaimani children,there is a need to monitor parasitic infections because of their metabolic observation. Furthermore, potential drugs are now being studied on the basis of natural bioactive compounds. The antioxidant and healing power of the Crocus sativus L (Mhamad et al (2025) may at would have a prophylactic benefit in oxidative stress due to parasitic infection. Taken together, these results underscore the near-universal rulethat T. gondii infection requires a cascade of reprogramming eventsbiochemical adjustments from different signalling pathways such ashormonal regulation, adipokine secretion, fat metabolismand mineral homeostasis. Moreover, these changes show the ability of the parasite to manipulate host biology and highlight the necessity of including heavy metals, co-infections, and natural therapeutic compounds in the search for the broader spectrum of health effects in the context of toxoplasmosis.

Conclusion

This current study confirmed that any significant metabolic abnormality associated with Toxoplasmosis infection is manifested by elevated concentrations of these markers: oxytocin, adiponectin, chemerin, copper, TC, HDL-C, and VLDL, and decreased dopamine, triglyceride, and LDL-C levels. From this, we conclude that these changes result from the parasite's interference with neurohormonal, adipokine, and mineralocorticoid balance, emphasizing the importance of monitoring these parameters to better understand, diagnose, and manage toxoplasmosis.

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