

Circulating inflammatory mediators CCL2 and CXCL16 as biomarkers in Iraqi women infected with toxoplasmosis

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Abstract

The protozoan parasite *Toxoplasma gondii* is the cause of the widespread but sometimes disregarded parasitic illness toxoplasmosis. Cytokines play an indispensable role in fostering an environment that is more conducive to pregnancy. Their intricate network of interactions allows for the observation of numerous modulatory consequences, such as cell migration, invasion, and gene expression. Among these, CCL2 (MCP-1) and CXCL16 have emerged as promising immunological biomarkers reflecting disease activity, inflammation, and tissue involvement. Monocyte chemoattractant protein 1 (MCP-1), another name for C-C motif ligand 2 (CCL2), is a crucial chemotactic factor that attracts macrophages for the immunological response. CXCL16 is a unique chemokine that can be found in both transmembrane and soluble forms. Numerous inflammatory conditions in humans, such as interstitial lung disease, rheumatoid arthritis, vascular disease, and hepatic injury, have been linked to CXCL16 expression. This study aimed to evaluate the correlation between *T. gondii* seropositivity and concentrations of plasma markers (CCL2 and CXCL16) in Iraqi pregnant women. This study comprised 68 serum samples of Iraqi women, attended Al-Yarmouk Teaching Hospital, Baghdad. The levels of CCL2 and CXCL16 were measured using sandwich ELISA. Results of circulatory marker levels revealed a significant variation across all groups. Also, results revealed that the chemokines CCL2 and CXCL16 showed distinct timing in their concentrations throughout pregnancy. Their higher levels in late pregnancy among healthy pregnancies suggested a normal immune adaptation, while their elevated early levels in aborted cases may signal inflammatory processes linked to pregnancy loss. In conclusion, these results indicated that CCL2 and CXCL16 could serve as immunological biomarkers for monitoring pregnancy progression and identifying risk of pregnancy loss.

Keywords: *Toxoplasma gondii*, Toxoplasmosis, Biomarkers, chemokine (C-C motif) ligand 2 (CCL2), chemokine (C-X-C motif) ligand 16 (CXCL16), ELISA.

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Introduction

The coccidian parasite *Toxoplasma gondii* has an exceptionally broad intermediate host range that includes a range of warm-blooded animals and uses felids as definitive hosts. Globally, one-third of persons have a persistent toxoplasmosis infection.¹ Worldwide, infections with *T. gondii* are associated with substantial morbidity and mortality, and the infection has wide clinical expressions in both immunocompetent and immunocompromised individuals. Although pregnant women and immunocompromised patients may occasionally experience consequences that impair their brain, eyes, or fetuses, the illness is often asymptomatic in humans.² But it can also result in several potentially fatal clinical problems.³ Also, in congenitally infected infants, as well as immunocompromised humans with profound defects in cellular immunity (due to acquired immunodeficiency syndrome [AIDS] or transplants), sporadic instances of severe, fulminant, life-threatening toxoplasmosis were described.⁴

Although most women are often asymptomatic, toxoplasmosis infection during pregnancy can result in disease transmission through the placenta, which can cause serious problems like miscarriage, abortion, various degrees of mental or physical impairments, blindness, and hydrocephalus.^{5,6} The third trimester of pregnancy has the highest incidence of congenital toxoplasmosis, whereas the first and second trimesters have the most severe infection, which can cause abortion and stillbirth.^{7,8} After passing through, *T. gondii* can multiply in the lamina propria of the intestinal cells after crossing the epithelial barrier. *T. gondii* infection responds to complex immunity, in which distinct leucocytes participate in diverse ways, which is the response to the parasite infection.⁹

One essential function of the immune system is cell migration. A carefully thought-out and regulated migratory process is required due to a variety of morphogenetic events, angiogenesis, the formation of intercellular connections, and intricate interactions between

the immune systems and cells of the mother and the fetus.¹⁰

Chemokines are a family of small, secreted proteins involved in the mediation of directional immune cell migration, i.e., chemotaxis. Chemokines are divided into four subfamilies, CXC, CC, CX3C, and C, according to the number and disulfide bonding arrangement pattern of conserved cysteine residues located near the N-terminus.^{8,11} Chemokines are crucial for the selective recruitment of neutrophils, monocytes, and lymphocytes and for initiating chemotaxis via the activation of G-protein-coupled receptors.¹² The chemokine family is crucial in enabling an efficient innate immune response to *T. gondii* infection, allowing it to enable an effective innate immune response. There is significant sequestration of the wide variety of chemokines during the pathophysiology of toxoplasmosis.¹³ One of the primary chemokines that controls monocyte and macrophage migration, and invasion is monocyte chemoattractant protein-1 (MCP-1) or chemokine (C-C motif) ligand 2 (CCL2). It was shown that CCL2 and its receptor CCR2 are both induced and involved in a number of illnesses.¹⁴ Although the pretreatment of human monocyte-derived macrophages with MCP-1/CCL2 was shown to cause *T. gondii* suppression, the impact of chemokines on MCP-1/CCL2 secretion has been questioned thus far. However, recent research on human subjects, indicated that human fibroblasts exposed to live tachyzoites (strain RH) significantly increase MCP-1/CCL2 production and secretion. During infection, these chemokines may draw Th1 cells in addition to "monocytes," natural killer "NK" cells, and dendritic cell "DCs".^{13,15}

CXC-chemokine ligand 16 (CXCL16) is a multifunctional cytokine, highly expressed in diverse immune cells, including macrophages, dendritic cells, and B and T lymphocytes. It is also expressed in other cell types like fibroblasts and activated endothelial cells, as well as trophoblasts.¹⁶ The PI-3K/PDK1/AKT/cyclin D1 pathway is activated by CXCL16 and CXCR6, which are expressed as a single pair of chemokines and chemokine receptors at the maternal-fetal interface. As a result, endometrial decidualization is improved.¹⁷

Although CXCL16 is implicated in normal physiology and cellular functions it has also been shown to contribute to diverse pathologies.¹⁸ Not much is known, however, about the role of CXCL16 during parasitic infections. Previous research has shown that *Leishmania donovani*, another protozoan parasite, induces CXCL16 expression in murine macrophages and increases CXCL16-mediated chemo-taxis of CXCR6-expressing cells.¹⁹ Also, a recent report described the presence of CXCL16-secreting liver macrophages following *Trypanosoma brucei* infection in mice, potentially contributing to the disease pathology through the recruitment of CXCR6+CD4+ T cells.²⁰

Consequently, this study aimed to evaluate the correlation between *T. gondii* seropositivity and concentrations of plasma markers (CCL2 and CXCL16) in Iraqi pregnant women.

Materials and Methods

Immunoassay of CCL2 and CXCL16

The study included 68 Serum levels of human MCP-1/CCL2 and human CXCL16 were determined for each of the groups under study. Sandwich Enzyme-Linked Immunosorbent Assay (ELISA) was performed using commercial kits (Bioassay Technology Laboratory, China), in accordance with the manufacturer's instructions. ELISA plates (96-well) were used, and ELISA reader (Promega Instrument) was used to analyze the results. The absorbance was measured at a wavelength of 450 nm. CCL2's sensitivity was 6.4 pg/ml, while CXCL16's was 0.262 pg/ml.

Statistical Analysis

The Statistical Package of Social Science (SPSS) software (2019) was used to analyze the study parameters of various groups. The t-test and Least Significant Difference (LSD) were used to compare the significance between means. The chi-square test was used to compare the significant percentages (at $p < 0.05$ and $p < 0.01$ probability). The correlation coefficient between variables in this study was estimated ²¹

Results

Samples

Women (n=68) who were attending the clinics of Al-Yarmouk Teaching Hospital in Baghdad-Iraq, were enrolled in this study during the period from March 2025 and May 2025. Blood samples were collected, sera separated and then stored at -10°C until time of testing.

Laboratory investigations were performed for the following groups:

-Pregnant women (n=30) testing positive (+ve) for toxoplasmosis: These included only one healthy pregnant woman in her first trimester, two healthy pregnant women in their second trimester, and 27 healthy pregnant women in their third trimester.

-Apparently healthy pregnant women (n=15) in their third trimester testing negative (-ve) for toxoplasmosis.

Abortion group: this included:

- Toxoplasmosis subgroup (n=17): Ten aborted women in their first trimester, six in their second trimester, and only one in the third trimester, while the non-toxoplasmosis-infected aborted pregnant women (n=6) included five patients from 1-3 months and only one from 4-6 months.
- Non-toxoplasmosis subgroup (n=6): included 5 patients in their third trimester and only one in its second trimester.

Age of study subjects

Table 1 showed a significant difference between positive and negative pregnant women, where the 21–25 and 26–30 age groups had the highest frequencies. A statistically significant association was also noticed between CCL2 and CXCL16 biomarkers with age of both pregnant women positive ($p = 0.0451$) and pregnant women negative ($p = 0.0498$). Statistics of the abortion group indicated that most of the positive cases were concentrated within the 21–25 age group, but the overall association with age was not significant ($p = 0.2367$), while the negative group attributed a borderline significant association ($p = 0.050$), with higher proportions seen in the 21–25 and 31–35 age groups (Table 1).

Table 1. Distribution of chemokine (C-C motif) ligand 2 (CCL2) and CXC-chemokine ligand 16 (CXCL16) in study sample according to age groups.

Age	Pregnant +ve	Pregnant -ve	Abortion +ve	Abortion -ve
15-20	4 (13.3%)	3 (20%)	4 (23.5%)	1 (16.6%)
21-25	11 (36.6%)	6 (40%)	7 (41.1%)	3 (50%)
26-30	10 (33.3%)	5 (33.3%)	3 (17.6%)	0 (0%)
31-35	5 (16.6%)	1 (6.6%)	3 (17.6%)	2 (33.3%)
Total	30	15	17	6
<i>p</i> -value	0.0451	0.0498	NS	0.050

p > 0.05 is not significant (NS).

Gestational Periods

Pregnant and abortion groups showed significant variability of the CCL2 and CXCL16 positive distribution across the gestational periods. In pregnant women, positivity markedly increased with advancing age of pregnancy, with the majority of positive cases (90–100%) observed at the third trimester, unlike the first and second trimester which showed very few positives. On the contrary,

highest positivity is noticed in the abortion group during early pregnancy (first trimester: 58.8%), which progressively declined and became minimal by the third trimester (5.8% or 0%). Hereby, statistically significant differences are observed for all parameters ($p < 0.05$ – $p < 0.01$), confirming a strong association between periods of pregnancy and the distribution of CCL2 and CXCL16 expression (Table 2).

Table 2. Distribution of chemokine (C-C motif) ligand 2 (CCL2) and CXC-chemokine ligand 16 (CXCL16) in the study sample according to the gestational periods.

Month	Pregnant +ve	Pregnant -ve	Abortion +ve	Abortion -ve
First trimester	1 (3.3%)	0 (0%)	10 (58.8%)	5 (83.3%)
Second trimester	2 (6.6%)	0 (0%)	6 (35.2%)	1 (11.1%)
Third trimester	27 (90%)	15 (100%)	1 (5.8%)	0 (0%)
Total	30	15	17	6
* <i>p</i> -value	<0.0001	<0.0001	<0.0076	<0.03166

* $p \leq 0.05$ is significant.

Serum level of CCL2 and CXCL16 serum

This study assessed the serum levels of chemokines CCL2 and CXCL16 in relation to toxoplasmosis infection status among both pregnant women and abortion cases. Both CCL2 and CXCL16 were significantly increased in women tested positive for toxoplasma infection, with stronger differences observed in

the abortion cases. With regard to CCL2 serum levels in pregnant women, the results are as follow: toxoplasma-positive cases showed significantly higher CCL2 levels than toxoplasma-negative ones ($p = 0.0387$). While in abortion cases, a significant increase in CCL2 was observed in toxoplasma-positive women compared with negatives ($p = 0.0001$). Regarding CXCL16 serum concentrations in pregnant

women, the levels were significantly higher in toxoplasma-positive cases than in negative cases ($p=0.0052$) and CXCL16 levels in abortion

cases were also significantly elevated in toxoplasma-positive women compared with negative cases ($p=0.0063$) as shown in Table 3.

Table 3. Relation of chemokine (C-C motif) ligand 2 (CCL2) and CXC-chemokine ligand 16 (CXCL16) concentrations (pg/ml) with Toxoplasmosis/Pregnancy and Abortion.

Variables	Pregnant		<i>p</i> -value	Abortion		<i>p</i> -value
	+Ve	-Ve		+Ve	-Ve	
CCL2	378.00 ±16.82	322.13 ±13.95	0.0387	619.29 ±18.52	419.47 ±12.93	0.000)
CXXL16	639.47 ±27.08	516.80 ±17.66	0.0052	619.29 ±15.38	488.00 ±22.46	0.0063

*:T-test. * $p \leq 0.05$ is significant.

Discussion

Women (n=68) with toxoplasmosis were enrolled in the investigations of this study to detect the potential of CCL2 and CXCL16 serum marker levels in both pregnant and aborted Iraqi women. Approximately, one-third of people worldwide are exposed to infection with the *T. gondii* protozoan parasite. Notably, innate immune detection mechanisms for *T. gondii* in humans (an obligatory intracellular parasite), are mostly unknown.²²

To establish an infection and ongoing parasite persistence, it is essential to highlight the complicated relationship between toxoplasmosis and the immune response, which allow the determination of disease immunological and pathological pathways along therapy.²³ This study evaluated the distribution of CCL2 and CXCL16 positivity among pregnant and aborted women according to age groups. It showed a statistically significant association between age and chemokine levels in most study groups. Of note, we found that serum levels of CCL2 and CXCL16 was most commonly elevated among pregnant women of 21–25 years age group, followed by those of 26–30 years, with a statistically significant association between age and chemokine expression. This may be explained by younger reproductive-age women having enhanced immune modulation that supports pregnancy. Pregnant negative cases showed a similar age pattern with a significant association, reinforcing age as an important factor in chemokine status during pregnancy.^{24,25} In contrast, non-significant association with age was noticed, despite that

abortion positive cases were most frequent in the 21–25-year-old group, possibly due to smaller sample size or multiple contributing factors to abortion.^{17,26} Additionally, abortion negative cases showed a borderline significant association, with higher proportions in women of 31–35 years age group, emphasizing that as the maternal age advances there may be associated alterations in immune or inflammatory profiles.^{16,27}

In this study, when the relation between the gestational periods and the CCL2 and CXCL16 chemokine levels was explored, results demonstrated a significant association between gestational periods and the distribution of CCL2 and CXCL16 levels among both pregnant and aborted women. Besides, these results showed that chemokine levels differ significantly across pregnancy stages. Most CCL2/CXCL16-positive cases in pregnant women are seen in the third trimester, constituting 90% of cases ($p=0.0001$). Negative cases in pregnant women also reflected this trend, with 100% recorded in the same period, again demonstrating high statistical significance ($p=0.0001$). These findings suggested that chemokine expression is strongly affected by gestational progression and may increase as pregnancy advances, reflecting enhanced immune and inflammatory regulation required for fetal growth and preparation for parturition.^{28,29} Similarly, aborted negative cases were predominantly observed during the first trimester (83.3%), with a statistically significant association ($p=0.0316$). This pattern reflected the known clinical observation that the majority of spontaneous abortions occur during early

pregnancy and may be related to early placental development and immune tolerance mechanisms. Maternal-placental-perinatal unfavorable pregnancy outcomes are frequently more common in women with a history of FRSA.^{25,30}

Recent studies recorded that there is enhanced production of plasma inflammatory mediators; CCL2, CXCL16, and interleukin (IL)-33 in *T. gondii*-seropositive pregnant women. Additionally, multiparous women have a higher risk of contracting *T. gondii* as compared to primiparas women.^{1,8} These analyses demonstrated significant alterations in serum levels of CCL2 and CXCL16 in women with toxoplasmosis, both during pregnancy and in cases of abortion, indicating an active immunological response associated with *T. gondii* infection.

CCL2 concentrations were significantly elevated in Toxoplasma-seropositive pregnant women compared to sero-negative controls (378.00 ± 16.82 vs. 322.13 ± 13.95 pg/ml; $p = 0.0387$). This increase suggested enhanced recruitment of monocytes/macrophages during infection, reflecting activation of innate immunity aimed at controlling the parasite during pregnancy.^{31,32} Notably, a more pronounced elevation in CCL2 levels was observed in aborted women with toxoplasmosis (619.29 ± 18.52 pg/ml) compared to sero-negative aborted controls (419.47 ± 12.93 pg/ml), ($p = 0.0001$). This marked elevation indicated profound inflammatory signaling, a possible factor which may contribute to placental inflammation, tissue damage, and pregnancy loss. Hence, this can reinforce the hypothesis that dysregulated chemokine production plays a role in the pathogenesis of toxoplasmosis-associated abortion.^{29,33}

At the maternal-fetal interface, CCL2 is expressed by trophoblasts, decidual tissue, the myometrium, and other tissues. Meanwhile, a number of physiological regulators of CCL2 that are involved in tissue remodeling, angiogenesis, and immunocyte recruitment were identified by a previous research.²⁹ The most common cause of autogenic pregnancy death before the 24th week of pregnancy is spontaneous abortion, often known as miscarriage.³⁴ Spontaneous

abortions, or miscarriage, is the most frequent cause of autogenic pregnancy demise before the 24th gestational week.³⁵ The main threat is chromosomal abnormalities, and abnormal chemokine levels are considered an extra risk.³⁶ A previous study revealed that CCL2 mRNA was more prevalent in the decidual and chorionic tissues of a spontaneous abortion cohort than in the healthy group.³⁷

Similarly, CXCL16 levels were significantly higher in Toxoplasma-seropositive pregnant women compared to seronegative pregnant controls (639.47 ± 27.08 vs. 516.80 ± 17.66 pg/ml; $p = 0.0052$). CXCL16 effectively functions in T-cell recruitment and Th1 immune responses, a fundamental element for controlling intracellular pathogens such as *T. gondii*. Nevertheless, excessive Th1-type responses during pregnancy may disrupt maternal-fetal immune tolerance.^{18,38} In aborted women, significant elevation in CXCL16 concentrations was also recorded in seropositive cases compared to seronegative ones (619.29 ± 15.38 vs. 488.00 ± 22.46 pg/ml; $p = 0.0063$). This increment suggested a strengthened inflammatory milieu, a potential factor which exacerbates placental dysfunction and contributing to fetal loss.^{10,39}

In conclusion, this study concluded that age significantly influences CCL2 and CXCL16 distribution, especially during pregnancy, being the younger age groups associated with higher expression and older age potentially linked to adverse outcomes. Overall, the results refer to significant upregulation of CCL2 and CXCL16 in both pregnancy and abortion associated with toxoplasmosis. Although moderate elevation in chemokines during pregnancy may reflect protective immune activation, the markedly higher levels observed in aborted women suggested an exaggerated inflammatory response that may compromise pregnancy maintenance. These findings highlighted the dual role of chemokines in host defense and immunopathology and support their potential use as biomarkers for immune imbalance and adverse pregnancy outcomes in toxoplasmosis.

Author Contributions

All authors designed the concepts. GMH, MAN, and MMA: performed the investigations. SHF and AFA: analyzed the data. GMH and AFA: wrote the manuscript. All authors read and approved the manuscript.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Ethical approval

The protocol of the study was reviewed and approved by the Ethical Committee of the College of Science, University of Baghdad (NO: CSEC/0126/0013).

Informed consent

Each volunteer or the patient's legal companion (parent or elder sibling) gave a vocal affirmation of their objective consent to sample collection under medical supervision.

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