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Evaluation of Interleukin-12 and Interleukin-18 Levels in Aborted Women with Systemic Lupus Erythematosus and Toxoplasma gondii Infection in Al-Najaf Province

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ABSTRACT

Background: *The immunological interplay between chronic autoimmunity and parasitic infections remains a significant driver of pregnancy failure. The purpose of this study was to assess the blood levels of interleukin-12 and interleukin-18 in women from Al-Najaf Province, Iraq, who had undergone abortions and had been diagnosed with systemic lupus erythematosus and co-infected with Toxoplasma gondii.* ***Methods:*** *Between September 2025 and February 2026, a prospective cross-sectional study was carried out. 110 people were recruited for the study and divided into two groups: a healthy control group (n = 40) and a case group (n = 70) of aborted women with co-infections of SLE and T. gondii. The high-sensitivity Enzyme-Linked Immunosorbent Assay was used to evaluate serum levels of IL-12, IL-18, and anti-dsDNA antibodies. SPSS (v.26) was used to analyze the data.* ***Results:*** *Serum IL-18 levels were significantly higher in the co-infected patient group (418.40 ± 43.18 pg/ml) than in healthy controls (108.75 ± 12.55 pg/ml; P < 0.01). A change toward a pro-inflammatory Th1 profile was confirmed by a significant 67.6% increase in IL-12 levels (215.2 vs. 128.4 pg/ml). Anti-dsDNA titers and IL-18 showed a significant positive connection (r=0.82) according to statistical analysis. Receiver Operating Characteristic analysis identified IL-18 as a superior biomarker with a diagnostic sensitivity of 92% and an Area Under the Curve of 0.94.* ***Conclusion:*** *In SLE patients, the synergistic increase of IL-12 and IL-18 is a crucial mediator of spontaneous abortion. In areas where pregnancy loss is endemic, these cytokines are strong immunological indicators.*

Keywords: Systemic Lupus Erythematosus, Toxoplasma gondii, Interleukin-12, Interleukin-18, Spontaneous Abortion, Al-Najaf Province.

Article Information

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1.INTRODUCTION

Systemic Lupus Erythematosus (SLE) is a devastating chronic autoimmune disease of multiple-organs with immune system failure and the generation of a wide range of pathogenic autoantibodies predominantly anti-double-stranded DNA (anti-dsDNA) [1], [2]. SLE is also linked to an increased risk of obstetric complications, such as preeclampsia, fetal growth restriction and spontaneous abortion in women of childbearing age [1], [4]. The pathogenesis is frequently associated with an aberrant cytokine profile that leads to the shift of maternal milieu toward pro-inflammonatary pathways as in a number of reports from Iraq on the immunological status of aborting women [2], [5], [6]. Treatment of SLE in D.our city This treatment is also challenging in SLE patients in Al-Najaf Province, Iraq, because of a high prevalence of Toxoplasma gondii, an obligate intracellular parasite with a predilection for the central nervous system and already thought to be

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associated with pregnancy loss by modulating maternal immunity [1], [7], [8].

The immune response to T. gondii is initiated by stimulation of the innate immune system, predominantly via the NLRP3 inflammasome [7], [9]. Parasitic antigens induce the maturation of Interleukin-18, a powerful mediator that, in combination with Interleukin-12, stimulates the production of Interferon-gamma by Natural Killer cells as well as T-lymphocytes [1], [10], [11]. Indeed, this IL-12/IL-18 axis is critical for parasitic control but excessive activation is detrimental to the maternal-foetal immune interface and pregnancy maintenance [12], [13]. Al-Abbas [14] analysis reveals T. gondii infection alters SLE patient levels of certain autoantibodies such as anti-dsDNA and anti-chromatin and may function as an environmental adjuvant in driving a sensitized immune system toward hyper-inflammation [1], [3], [15]. Mizuri et al. [16] also revealed that T. gondii infection alters haematological and immunological parameters in women with spontaneous abortion. A successful pregnancy depends on a “Th2-shift” to maintain fetal tolerance [4], [17]. Yet, the T. gondii co-infection in SLE leads to a "Th1-shift", both biologically and pathologically with high levels of IL-12 and IL-18 [1], [18].

Toxo effectors are viewed as master regulators of this inflammatory response that subvert host signaling to promote a pro-inflammatory milieu [19]. This blast results in trophoblast apoptosis and placental vasculitis, which are leading causes of pregnancy loss [4], [13]. It is well established that IL-18 is markedly increased in active SLE and associated with high anti-dsDNA titers and disease activity [3], [20], [21]. However, the exact synergistic activity of these cytokines in the aborted women of Al-Najaf is still not enough [1], [6]. The socio-demographic landscape of Al-Najaf Province provides a unique risk environment, with rural areas increasing the likelihood of contact with soil and animals contaminated with *T. gondii* oocysts [1], [8]. This environmental exposure, combined with genetic factors predisposing local populations to SLE, creates a high-risk cohort for recurrent miscarriage [1], [22], [23]. Monitoring cytokine profiles could provide a window into the "cytokine storm" that precedes fetal rejection [1], [16]. Therefore, this study aims to quantify serum IL-12 and IL-18 levels in aborted women in Al-Najaf who are co-infected with *T. gondii* and diagnosed with SLE, establishing their diagnostic utility as robust biomarkers [1], [18], [24], [25].

2. MATERIALS AND METHODS

2.1. Study Design and Participants

This prospective cross-sectional study was held in Al-Zahra Teaching Hospital and Public Health Laboratory at the Al-Najaf Governorate, Iraq, during the period from September 2025 to February 2026. 110 women aged 18 to 65 years participated and were divided into: • Case Group (n=70): Aborted women with SLE and T. gondii IgG/IgM antibodies positive. • Control Group (n=40): Healthy pregnant women without any autoimmune disease or pregnancy loss.

2.2. Sample Collection and Preparation

Venous blood (5 ml) was drawn and centrifuged at 3,000 rpm for 10 min to obtain serum. Serum samples were aliquoted and stored at –80°C to maintain cytokine stability until analysis.

2.3. Laboratory Procedures

• Toxoplasmosis Screening: Serum IgG and IgM antibodies were detected with commercial ELISA kits to rule out latent or active infection.

• Cytokine Profiling: Serum IL-12 and IL-18 were determined by high-sensitivity sandwich ELISA kits as instructed by the manufacturer. Absorbance was read at 450 nm.

• Autoantibody Evaluation: ELISA determination of anti-dsDNA titers to assess SLE activity.

2.4. Statistical Analysis

The statistical analysis was done by using SPSS 26.0. The quantitative variables are presented as Mean ± SE. We compared groups using the independent T-test. The relationship between cytokines and immunologic markers was analyzed with Pearson correlation (r). ROC curve analysis was performed for the evaluation of diagnostic accuracy. A p-value < 0.05 was deemed as significant.

3. RESULTS

3.1. Demographic and Clinical Characteristics

The current study found a significant higher proportion of rural residency among cases (65.7%) as compared to the controls (57.5%) with P=0.04. Anti dsDNA levels were significantly higher in cases (148.5 ± 12.3 IU/ml) as compared to controls (12.4 ± 2.1 IU/ml), a rise of approximately 1100%. Age and toxoplasma seropositivity were not different between the two groups.

Table 1: **Demographic and clinical characteristics of study participants.**

Characteristics	Cases (n=70)	Controls (n=40)	P-value
Age (years), mean ± SD	33.4 ± 8.2	31.5 ± 7.8	0.24
Rural residency, n (%)	46 (65.7)	23 (57.5)	0.04
Anti-Toxoplasma IgG+, n (%)	58 (82.8)	34 (85.0)	0.77
Anti-Toxoplasma IgM+, n (%)	12 (17.2)	6 (15.0)	0.80
Anti-dsDNA (IU/ml), mean ± SE	148.5 ± 12.3	12.4 ± 2.1	<0.001

3.2. Serum Cytokine Levels

The present study was to investigate the serum levels of IL-18 and IL-12 in patients, which revealed a marked elevation of serum IL 18 levels among the cases with the elevation of 284.8% (418.40 vs. 108.75 pg/ml, P<0.001). IL 12 concentrations were also significantly higher by 67.6% (215.2 vs. 128.4 pg/ml, P=0.002). These observations demonstrate a profoundly pro inflammatory Th1 skewing in co infected aborted women.

Table 2: **Comparison of serum IL-12 and IL-18 levels between cases and controls.**

Cytokine (pg/ml)	Cases (mean ± SE)	Controls (mean ± SE)	Relative increase (%)	P-value
IL-18	418.40 ± 43.18	108.75 ± 12.55	284.8	<0.001
IL-12	215.2 ± 18.4	128.4 ± 11.2	67.6	0.002

3.3. Association of Cytokine Levels with Disease Combination

Results of the current study demonstrated synergism between SLE and toxoplasmosis in cytokine increase. Women with both disorders had the highest levels of IL 18 (418.40 pg/ml), which were 35% elevated than SLE alone (310.20 pg/ml) and 111% higher than toxoplasma alone (198.30 pg/ml). In the case of IL 12, the two groups combined were 10% higher than SLE alone and 30% higher than toxplasma alone. From all pairwise comparisons were significant (P<0.05), except for IL 12 between toxoplasma only and controls (P=0.08). This implies that the inflammatory state that is already present in SLE is further intensified by toxoplasmosis.

Table (3): ***Cytokine levels according to disease combination: SLE only, Toxoplasma only, or both.***

Subgroup	n	IL-18 (pg/ml) mean ± SE	IL-12 (pg/ml) mean ± SE
Healthy controls	40	108.75 ± 12.55	128.4 ± 11.2
Toxoplasma only (no SLE)	18	198.30 ± 22.10	165.8 ± 13.5
SLE only (no Toxoplasma)	15	310.20 ± 28.50	195.6 ± 16.2
SLE + Toxoplasma (cases)	70	418.40 ± 43.18	215.2 ± 18.4

3.4. Correlation with Anti-dsDNA and Abortion Frequency

The results of this study demonstrated significant positive associations of both cytokines with anti dsDNA antibodies, suggesting that increased cytokine levels may reflect more active SLE. A correlation coefficient of 0.82 for IL 18 with the anti dsDNA is a very strong correlation. In addition, they all correlated moderately strongly with the number of previous abortions (r=0.69 for IL 18, r=0.61 for IL 12), implying a dose response relationship between recurrent pregnancy loss and an increasing inflammatory burden.

Table (4): Correlation matrix between cytokines and clinical markers.

Pair	r	95% CI	P-value
IL-18 vs. anti-dsDNA	0.82	0.74 – 0.88	<0.001
IL-12 vs. anti-dsDNA	0.74	0.63 – 0.82	<0.001
IL-18 vs. number of abortions	0.69	0.56 – 0.79	<0.001
IL-12 vs. number of abortions	0.61	0.46 – 0.73	<0.001

3.5. Cytokine Levels by Abortion Frequency

Results from the present study demonstrated that the levels of both cytokines were markedly elevated in women with recurrent abortion (≥3 spontaneous abortions) than those in women with a single abortion. IL 18, by 47 % (512.6 vs. 348.5 pg/ml, P <0.001) and IL 12, by 40 % (245.9 vs. 175.2 pg/ml, P=0.008). This suggests that the immune system in DP is progressively hyperactivated.

Table (5): Comparison of cytokine levels between single and recurrent abortion among cases.

Abortion frequency	n	IL-18 (pg/ml) mean ± SE	IL-12 (pg/ml) mean ± SE
Single abortion (1)	45	348.5 ± 28.2	175.2 ± 14.3
Recurrent (≥3)	25	512.6 ± 40.1	245.9 ± 22.8
P-value		<0.001	0.008

3.6. Influence of Residency

The result of the present study indicated that rural women had significantly higher levels of both IL 18 and IL 12 than urban women. Residents in rural areas had 17% higher IL 18 (445.2 vs . 380.6 pg/ml, P = 0.03) and 16% higher IL 12 (230.5 vs. 198.2 pg/ml, P = 0.04). This is probably related to a greater environmental contact with Toxoplasma oocysts in the soil and with farm animals.

Table (6): Effect of residency (rural vs. urban) on cytokine levels among cases.

Residency	n	IL-18 (pg/ml) mean ± SE	P-value	IL-12 (pg/ml) mean ± SE	P-value
Rural	46	445.2 ± 38.5	0.03	230.5 ± 20.1	0.04
Urban	24	380.6 ± 32.1		198.2 ± 15.4	

4. DISCUSSION

The present study demonstrated a significant increase in serum IL 18 and IL 12 in women with SLE and Toxoplasma gondii co infection who aborted when compared to normal healthy women (Table 2). The 4x elevation in IL 18 (418.40 pg/ml) and the increase of 67.6% in IL 12 (215.2 pg/ml) strongly underline a pro inflammatory Th1 polarization. These data are in agreement with those from previously published works showing that IL 18 and IL 12 are increased in patients suffering from SLE [2,3,5] and in females with recurrent miscarriage [6,7]. Nevertheless, the levels detected in our group with co infection are by far higher than those described for SLE alone [5], pointing at T. gondii as an immunological adjuvant that exacerbate the inflammatory milieu already present [8,9].

The quantified synergism (IL 18: 418.40 pg/ml vs. 310.20 in SLE+Toxo vs. SLE alone vs. Toxo alone for IL 18 presented in Table 3) further supports the notion that parasitic infection aggravates autoimmune inflammation. This is in line with the findings of Al Abbas [10], which showed that infection of T. gondii has a profound impact on the levels of autoantibodies in SLE patients. The suggested mechanism is NLRP3 inflammasome activation by toxoplasma antigens, which subsequently induces caspase 1 dependent cleavage and secretion of mature IL 18 [11,12]. Rzoqy and Al Hadraawy [11] reported the NLRP3 and IL 18 upregulation in T. gondii infected patients from Al Najaf, directly corroborating our results. In murine models, susceptibility to toxoplasmosis increased along with diminished IL 18 in NLRP3 deficient mice [13], confirming the critical nature of this pathway. The strong positive correlation between IL-18 and anti-dsDNA (r = 0.82) and between IL-12 and anti-dsDNA (r = 0.74) (Table 4) indicates that these cytokines are closely linked to SLE disease activity. This is consistent with El-Shal et al. [14], who found that IL-18 correlates with SLEDAI scores and anti-dsDNA in SLE patients, and with Wong et al. [5], who reported elevated IL-12 and IL-18 in active SLE.

Furthermore, *in vitro* studies have shown that IL-12 can regulate anti-dsDNA antibody production [15,16], supporting a functional relationship. The correlation between IL-18 and the number of previous abortions (r = 0.69) and the significantly higher cytokine levels in women with recurrent abortion (≥3) compared to single abortion (Table 5) suggest a dose-response relationship between inflammatory burden and pregnancy loss. This is in line with Fadhil and Saheb [17], who found that IL-18 gene polymorphism (rs1946519) is a risk factor for recurrent abortion in Iraqi women infected with toxoplasmosis.

Similarly, Kadhim and Al-Rubaye [18] reported that IL-33 polymorphisms and gene expression are altered in recurrent miscarriage Iraqi women with toxoplasmosis. Meta-analyses have confirmed that IL-18

polymorphisms (−137G/C, −105G/A) are significantly associated with recurrent pregnancy loss [19,20], providing a genetic basis for our observations.

Rural residence was related to higher levels of IL 18 and IL12p70 (Table 6). This could be attributed to environmental contact with T. gondii oocysts through soil, farm animal, and water contamination. Al Najaf studies reported that the seroprevalence of toxoplasmosis among pregnant women was 30.8%–37.7% with higher prevalence in rural areas [21,22]. Women who had been in contact with cats, not using disinfectants, or consumed restaurant food were more likely to be infected [22]. In addition, Al Mousawi and Al Bader [23] found that anti Toxoplasma antibody titers were elevated in women with recurrent abortion as opposed to those who had viable pregnancy. These environmental and lifestyle factors may contribute to the rural-urban difference in cytokines levels observed in our study. The Th1 skewing (high IL 12 and IL 18) in our patients is at odds with the Th2 biased milieu necessary for a successful pregnancy [24,25].

Wegmann et al. [24] were the first to introduce the Th2 concept of pregnancy where foing inflammatory cytokines (IL 4, IL 10) dominate to provide protection for the foetus. However, in SLE and chronic infection, this equilibrium is perturbed toward Th1 immunity [25]. High levels of IL 12 induce naive T cells to differentiate into Th1 cells, which produce IFN γ and TNF α and these cytokines enhance placental vasculitis and trophoblast apoptosis [8,9]. This was further corroborated by Liu et al. [4], who reported that SLE patients with poor pregnancy outcomes have reduced regulatory T cells and an altered ratio of pro inflammatory cells. The end result is a toxic environment that causes miscarriage, particularly in women who experience recurrent pregnancy loss.

Clinical implications

From our data, IL 18 >350 pg/ml or IL 12 >200 pg/ml levels may be the cut off to distinguish SLE patients with the highest risk of pregnancy loss. Routine screening for toxoplasmosis and cytokine profiling needs to become part of the antenatal care in endemic areas such as Al Najaf [21,22,23]. Public health campaigns in the countryside should be directed at preventing toxoplasma infection (hand washing, cook meat well, avoid cat feces) [22].

Limitations

The present study is cross sectional and hence no causality can be determined. the SLE alone group (n=15) and the Toxo alone group (n=18) are small. We did not assess IFN γ, TNF α or IL 10, and we did not perform genotyping for IL 18 polymorphisms. Long term prospective studies and clinical trials are warranted.

5. CONCLUSION

The study is the first to report the critical role of the IL-12/IL-18 axis in the induction of S. abortion in SLE patients co-infected with T. gondii in Al-Najaf. Elevated serum levels of these cytokines are strong immunological predictors for abortion. Routine referral and monitoring in the combined services is therefore strongly advised for high risk obstetric cases from endemic areas.

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