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Study the relationship between TGF-β gene expression and TH17 in influencing recurrent miscarriages in women with autoimmune thyroid disease

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ABSTRACT

Background: TGF-β is the main regulator of immune responses and plays an important role in the development of autoimmune diseases in humans, and is strongly implicated in autoimmune thyroid diseases. **Objective:** This study aimed to evaluate the immunological and gene expression of TGF-β and their relationship to recurrent miscarriages. **Methodology**: The study was conducted in Iraq in Salahuddin province, during which 90 samples were collected from women, 70 of whom were infected with AITD with positive TPOAb. They were compared with 20 healthy women. **Results**: During this study, highly significant differences in TGF-β gene expression were observed at $p \leq 0.001$ between patients with AITD and healthy individuals. The means were (1.739±0.214) and (1.208±0.347), with highly significant differences in the immunological TGF-β levels at $p \leq 0.001$ between pregnant patients and patients with RM, and the means were (1.7511±0.4955) and (1.0145±0.1960). It was also found that significant differences in the level of TH17 at $p \leq 0.001$ existed between pregnant patients and those with RM; the mean was (1.0111±0.2284) and (1.5298±0.2922), respectively. **Conclusion**: Through this study, it was found that the presence of the TGF-β gene and its high levels maintain pregnancy and work to suppress the cytokines secreted by Th17, which stimulate miscarriage. Additionally, the high level of the gene enhances its immune response, while its low level activates Th17 cells and increases the secretion of inflammatory cytokines.

Key words: TGF-β, Th17, Autoimmune thyroid disease (AITD).
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INTRODUCTION

Autoimmune thyroid disease (AITD) is one of the most common thyroid diseases and is of two types: hypothyroidism and hyperthyroidism. Autoimmune thyroid diseases occur either due to the effect of lymphocytes on the thyroid gland or due to the production of autoantibodies to the gland's proteins, such as thyroid peroxidase antibody (TPOAb), thyroglobulin antibody (TgAb) and thyroid-stimulating hormone receptor antibody (TRAb) (1,2) Recurrent miscarriages(RM): are defined as losing the fetus twice or more before reaching the 20th week. There are several causes, including genetic, immunological, anatomical, and physiological factors. They constitute about 15% of miscarriages with unknown causes (3). TGF-β, a master regulator of immune response, is deeply implicated in the complex pathophysiology and development of autoimmune thyroid diseases. Based on the close interplay between thyroid autoimmunity and TGF-β, scientific interest has shifted to understanding the possible role of this molecule in the diagnosis and development of thyroid diseases. TGF-β is a member of a transforming growth factor superfamily, which includes TGF-β, bone morphogenetic proteins, and growth re-differentiation factors, among other molecules (4). TGF-β exists in three isoforms: TGF-β1 (the most abundant), TGF-β2, and TGF-β3. TGF-β is synthesized in the rough endoplasmic reticulum and secreted by almost all human cells, including all white blood cell lineages. It plays an instrumental regulatory role in the generation, maturation, proliferation, and apoptosis of

various cell types, as well as in maintaining vascular, skeletal, and connective tissue homeostasis, regulating physiological immune responses and tolerance, and limiting the development of inflammation, fibrosis, tumor surveillance, and autoimmunity (5). The role of the signaling pathways associated with TGF-β activation and function is fundamental regarding various human cell, tissue, and system properties and homeostasis. Transforming growth factor beta (TGF-β) plays a pivotal role in the proper function of the human immune system and is implicated in the pathophysiological spectrum of thyroid autoimmunity (6). Relationship between AITD and TGF-B in recurrent miscarriage: The pathogenesis of recurrent miscarriage is related to immune disorders, which can lead to decreased maternal and fetal tolerance (7, 8). To the mother, the fetus is a semi-allograft with maternal and paternal genes. Under typical physiological conditions, the maternal immune system is expected to mount an immune response (9). However, the mother can achieve a successful pregnancy due to the establishment of a unique immune tolerance environment. TGF-β is a cytokine that can control harmful immune responses to the embryo. Among them, TGF-β has immune suppressive effects (10). This is equivalent to creating an environment of immune tolerance. Several studies showed that TGF-β levels were lower in patients with recurrent miscarriage than in people with normal pregnancies. It is consistent with reduced maternal-fetal tolerance in patients with recurrent miscarriage. Several statements regarding the mechanism of reduced maternal-fetal tolerance (9). Transforming growth factor-beta (TGF-B) Genes: TGF-b, an evolutionarily conserved secreted protein consisting of three isoforms, TGF-b1 (the most common), TGF-b2, and TGF-b3, which map to regions of human chromosomes TGF-B1 19q13.2, TGF-B2 1q41, and TGF-B3 14q23-24 (11). Th17 cells Like Th1 and Th2 lymphocytes, Th17 cells develop from naive CD4 cells (1 out of 4 cell groups: Th1, Th2, Th17, and Treg), differentiate from naive T cell precursors by the polarizing cytokines transforming growth factor-β (TGF-β) and IL-6, under the control of the transcription factor retinoic-acid-receptor-related orphan nuclear receptor alpha and gamma (RORα/γ)(12) (13). Th17 cells were described in 2003 by Aggarwal (14, 15). The discovery of this lymphocyte subtype was initiated and named after the identification of IL-17A in 1993 (14). They have been described as producing IL-17A, IL-17F (27), and IL-22, distinguishing them from Th1 and Th2 cells, which do not produce these cytokines (16). Th17 also produces IL-21; however, it might be secreted by other Th cells (16). The non-pathogenic Th17 cells naturally occur in the gut, and they are responsible for maintaining the homeostatic microbiota and fighting against pathogenic microbes. They have no autoreactive features. Unlike non-pathogenic ones, the pathogenic Th17 cells arise in the presence of IL-23. They are mainly responsible for developing autoimmune reactions to self-antigens and in organ-specific autoimmunity, transplantation, and acute transplant rejection (17, 18, 19)**.** The aim of this study was to investigate the relationship between TGF-β and TH17 levels in women who suffer from recurrent miscarriages and have immune gland diseases, as well as to compare the gene expression levels between patients and the control group.

METHODOLOGY

2.1 Subjects: This study was conducted in Saladin Governorate from February 20, 2024, until June 20, 2024, and gene expression tests were conducted in the College of Science-University of Tikrit laboratories.

2.2 Sample collection: A total of 90 samples were collected for the study, all of which were from women. The study was divided into two groups: 70 patients with AITD and 20 healthy controls. Then, 70 patients were divided into 35 pregnant patients and 35 patients with recurrent miscarriages (all 70 patients suffered from AITD). The following tests were performed for all study samples for diagnosis: triiodothyronine (T3), thyroxine (T4), TSH, and anti-TPOA. The specialist physician diagnosed 70 patients with hypothyroidism, where the average TSH for pregnant patients was 9.67 MIU/L. The average for patients with recurrent miscarriage was 43.85 MIU/L with a positive result for anti-TPOAb for all patients (pregnant and recurrent miscarriage). In contrast, the results of the control samples were normal TSH 2.13 MIU/L with a negative result for anti-TPOA. Blood samples were taken from pregnant women with AITD at different periods of pregnancy, and none of them had ever had a previous miscarriage. As for women who also suffered from recurrent miscarriages, blood samples were taken at different periods after the miscarriage, and none of them had become pregnant during the period of sample collection. The control group comprised married women who had not become pregnant during the sample collection period.

2.3 Blood Sampling: 10 ml of venous blood was drawn for all samples. The blood samples were divided into three parts: 5 ml was collected in gel tubes for inducting immune tests (TGF-B, Th17 cell, anti-TpoAb), and 4 ml for performing physiological tests (TSH, T3, T4). The blood serum was separated by centrifugation at 3500 rpm for 15 minutes, 250 µl of blood were added to a tube containing 750 ml of Trizo for conducting for gene expression all samples were stored at -20 °C.

2.4 Primers: The following primers were used in gene expression:

These primers were provided from Scientific Research Co. Ltd Iraq, following Table (1)

Primers	Sequence 5'-3'		Product size
TGF-β	F	GGGACTATCCACCTGCAAGA	165bp
	R	CTGTTGTACAGGGCGAGCAC	
GAPDH gene	F	TGCCACCCAGAAGACTGTGG	129bp
	R	TTCAGCTCAGGGATGACCTT	

2.5 Quantitative Real-Time PCR (qPCR): Real-time quantitative PCR was used to identify the TGF-β gene normalized by the housekeeping gene (GAPDH) in patients (pregnant women, recurrent miscarriages) and healthy controls according to the protocol followed by the manufacturer Bioneer/ Korea

2.6 RNA extraction, cDNA synthesis, and RT-qPCR:

1- RNA was extracted by following the manufacturer's instructions and steps Bioneer/ Korea.

2- The extracted RNA was converted to cDNA according to the manufacturer's instructions in Promega/USA using a Thermocycler.

Table (2): Thermocycler conditions for cDNA synthesis

Step	Temperature	Time
cDNA synthesis (RT step)	42 °C	1 hour
Heat inactivation	95 °C	5 minutes

3- RT-qPCR: After obtaining cDNA, it is used in an RT-PCR device according to the instructions of Bioneer/Korea and according to the steps shown in the Table (3).

qPCR steps	Temperature	time	Repeat cycle
Initial denaturation	95 °C	5 min	1
Denaturation	95 °C	20 sec	45
Annealing/extension	60 °C	30 sec	
Melting curve	65-95 °C		

4- Data Analysis of qRT-PCR: The data results of q RT-PCR for the target and housekeeping gene were analyzed by the relative quantification of gene expression levels (fold change) (The ΔCT Method Using a reference gene) as the following equation.

ΔCTP = CT target - CT GAPDH

ΔCTC = CT control - CT mean GAPDH

ΔΔCT = ΔCTP - ΔCTC

Folding = $2^{-\Delta\Delta CT}$

2.7 Detection of Human Immune Assays TH17, TGF-β, in ELISA kit:
Standard solutions were prepared per the manufacturer's Shanghai YL Biont/China protocol. 50 µl each of standard solutions and streptavidin-HRP were added to the wells of the standard solutions. 40 µl of samples and 10 µl of conjugated antibodies were added to the sample wells, and then 50 µl of streptavidin-HRP was added to all sample wells. Then we covered the plate with a special cover, shook it a little for mixing, and placed it in the incubator at 37 ˚C for 60 min. the washing solution was prepared by adding 20 ml of concentrated washing solution to 980 ml of distilled water. Then the plate cover was removed and washed, and the process was repeated five times. Then we dry the plate on the filter paper for one minute. Then, 50 µl of each chromogen solution A and B were added to each well and mixed well, then incubated for 10 min at 37 ˚C away from light. We then noticed the appearance of the blue color at different concentrations, indicating the antigen reaction and its presence. Then 50 µl of the stop solution was added to each hole to stop the reaction and we noticed that the blue color turned yellow immediately. The absorbance was measured at a wavelength of 450 nm within 10min.
2.8 Statistical Analysis: Statistical analysis was performed for all study samples and for all immunological, physiological, and genetic tests using the statistical analysis program Statistical Package for Social Sciences version 27.0(SPSS)- One-Way ANOVA. To obtain the mean, standard deviation, and correlation coefficient.

RESULTS

3.1 Serum level of T17: The results in Table (4) showed no significant differences when comparing the group of AITD patients (pregnant + recurrent miscarriages) with the control group. However, the results in Table (5) showed significant differences between patients with AITD.

Table (4): Comparison between patients (pregnant + recurrent miscarriage) and controls in TH17 levels

TH17	Mean ±SD		P value
	Patients	Controls	
	1.27045 ± 0.26995	1.2725 ± 0.2959	0.245 NS

Table (5): Comparison between pregnancy and recurrent miscarriage in TH17 levels

TH17	Mean ±SD		P value
	pregnancy	miscarriage	
	1.0111 ± 0.2284	1.5298 ± 0.2922	≤0.001 **

3.2 Serum level of Transforming Growth Factor- Beta (TGF-β): TGF-B is a multifunctional cytokine important for immune cells, helping them perform many functions [26]. Therefore, TGF-β was studied in women with AITD and Anti TPOAb-positive and compared with healthy. Table (6) shows the results among patients with thyroid disease compared with controls.

Table (6): Comparison between patients (pregnant + RM) and controls in TGF-β levels

TGF-β	Mean ± SD		P value
	Patients	Controls	
	1.3827 ± 0.370858	1.7510 ± 0.3488	≤0.005 *

Table (7) indicates the comparison between pregnant women and those with recurrent miscarriages, all of whom have AITD. The results showed significant differences at p≤0.001 for women suffering from recurrent miscarriages compared to pregnant women. The mean and SD for the two groups are shown in Table (7).

Table (7): Comparison between pregnancy and miscarriage in TGF-B levels

TGF-β	Mean ± SD		P value
	pregnancy	miscarriage	
	1.7511 ± 0.4955	1.0145 ± 0.1960	≤0.001 **

3.3 Gene Expression: We note from Table (8) and through the comparison of the results of gene expression between patients and controls and GAPDH that there is a genetic difference between patients and controls.

Table (8): Comparison between patients and controls in TGF-B gene expression

Group	Mean of CT	Mean ofΔCT	Mean of fold	P value
patients	26.248	5.502	1.739	
controls	21.338	4.946	1.208	
GAPDH			1.00	≤0.001 **

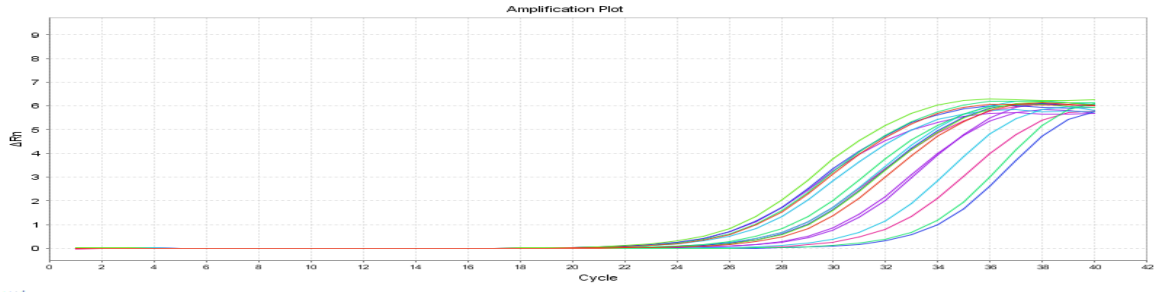

Figure (1): TGF-β amplification plots samples from all study groups by qPCR. The photograph was taken directly from the Bio Systems7500 qPCR machine. The colors in the diagram above shows the number of cycles required for DNA replication, when replication begins and when it ends

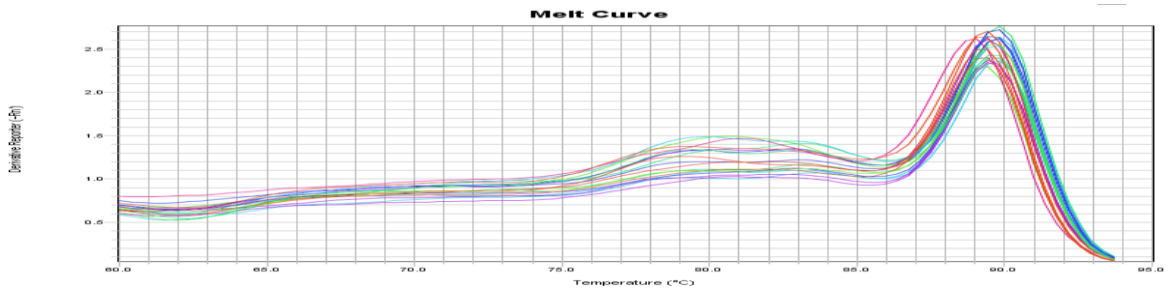

Figure (2): TGF-β melt curve by qPCR, which includes all study groups. The photograph was taken directly from the Bio Systems7500 qPCR machine. (It is a melting curve and shows the temperature at which the double strand of DNA begins to disintegrate into a single strand.

Table (9): shows the comparison between the study groups

Fold of TGF-β Gene	Mean ± SD				P value
	patients	controls	pregnancy	miscarriage	≤0.001 * *
	1.739 ± 0.214	1.35 ± 0.347	2.13 ± 0.119	1.21 ± 0.201	

Table (9) shows the comparison between the study groups. Table (9) shows significant differences at $p < 0.001$ and superiority of pregnant women in gene expression.

DISSCUSION

The results of this study indicate an increase in the levels of Th17 cells in women with recurrent miscarriages and AITD. This is because increased concentrations of anti-TPO Ab lead to increased concentrations of Th17 and decreased Treg cells lead to the creation of an inflammatory environment inside the uterus that causes miscarriage and is consistent with (20, 21). Also, the increase in Th17 concentrations leads to an increase in the inflammatory cytokines it secretes (IL-17, TNF-α) and a decrease in Treg cell cytokines (IL-10), which is consistent with (22, 23). The effect of the STAT3 factor, an essential regulatory factor that regulates the differentiation of human Th17 cells in addition to IL-6 and TGF-B, and any defect in its signals leads to autoimmune diseases (24, 25). This factor also can increase Th17 cells and thus increase the secretion of the inflammatory cytokine IL-17, which leads to the occurrence of abortion (26). The results also showed an increase in TGF-β concentrations in the control group. This is because TGF-β is a protein in the cytoplasm of almost all cells in the body and performs basic physiological functions under normal conditions, such as cell proliferation, differentiation, apoptosis, and maintaining immune homeostasis. Therefore, the increases in the control group are consistent with (27, 28). It is also showed an increased TGF-β concentration in pregnant women and decreased concentrations in women with recurrent miscarriages. One reason for this decrease of TGF-β is normally found in the cell in a dormant state, so to function, it must be activated by one of the activating factors, which are either acids, bases, reactive oxygen species (ROS), thrombospondin-1 (TSP-1), and proteases. It then becomes active, and without this activation, it cannot bind to the receptor and trigger the signals responsible for all its mechanisms of action (27) .The effect of TGF on the natural killer cells(NKc) present in the placenta, which are the primary immune cells that maintain immune tolerance between the mother and the fetus, as TGF-β works to inhibit the toxic activity of these cells by inhibiting the expression of CD16 present on the surfaces of their cells and the expression of DC39 specific to T cells, as well as increasing the expression of NKc expressing CD56, which can provide immune tolerance to the fetus. Therefore, when its concentrations decrease, it leads to adverse results. These results were consistent with (29). TGF gene expression increased in the patient group compared to the control group. This is because the thyroid follicular cells proliferate under the influence of two crucial factors, TSH and TGF-β. TGF-β works through a dual inhibitory or enhancing effect on the growth and proliferation of follicular cells, as well as preventing the increased infiltration of white blood cells into the gland cells and their destruction. Therefore, we find it related to autoimmune diseases of the thyroid gland and a significant modification in the immune cells Th1 and Th2. These results are consistent with (30). As for the increase in gene expression in pregnant women, this was due to the effect of the estrogen hormone, which works to modify the production of this factor from the thyroid gland follicular cells by activating the signals of the transcription factors SMADs (31).

CONCLUSION

Our study showed a relationship between the TGF-B gene and AITD, and that high levels of the gene in pregnant patients suppress the inflammatory effect of Th17 cells, suppress the secretion of their cytokines, and maintain pregnancy, unlike patients who have a decrease in the TGF-B gene, which negatively affects the fetus, leading to its loss.

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دراسة العلاقة بين التعبير الجيني ل TGF-β وTh17 في التأثير على حالات الإجهاض المتكررة لدى النساء المصابات بأمراض الغدة الدرقية المناعية الذاتية

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الخلاصة

خلفية البحث: TGF-β هو المنظم الرئيسي للاستجابات المناعية يلعب دورًا مهمًا في تطور الأمراض المناعية الذاتية لدى البشر وله تورط قوي في أمراض الغدة الدرقية المناعية الذاتية. الإجهاض المتكرر هو فقدان أكثر من حملين متتاليين قبل أن يصل الجنين إلى الأسبوع العشرين من الحمل. **الهدف من البحث:** هدفت هذه الدراسة إلى تقييم التعبير المناعي والجيني لـ TGF-β وعلاقتهما بالإجهاض المتكرر. **المواد والطرق:** أجريت هذه الدراسة في العراق في محافظة صلاح الدين وتم خلالها جمع 90 عينة من النساء، 70 منهن مصابات بـ AITD مع TPOAb إيجابية. تم تقسيمهن إلى 35 امرأة حامل مصابة بـ AITD و35 مصابة بالإجهاض المتكرر مع .AITD. تمت مقارنتهن بـ 20 امرأة سليمة. **النتائج:** وجدنا اختلافات كبيرة للغاية في التعبير الجيني لـ TGF-β عند $p \leq 0.001$ بين المرضى المصابين بـ AITD والأصحاء. وكان المتوسط (1.739±0.214) و (1.208±0.347) فروق ذات دلالة إحصائية عالية في مستويات TGF-β المناعية عند $p \leq 0.001$ بين المريضات الحوامل والمصابات بـRM ، وكان المتوسط (1.7511±0.4955)(1.0145±0.1960).(كما وجدنا فروق ذات دلالة إحصائية في مستوى TH17 عند $p \leq 0.001$ بين المريضات الحوامل والمصابات بـ RM وكان المتوسط (1.0111±0.2284).

الكلمات المفتاحية: عامل النسخ (TGF-B)،الخلايا المساعدة (Th17)، امراض الغدة الدرقية المناعية الذاتية.