Med. J. Cairo Univ., Vol. 93, No. 3, Accepted 3/6/2025
DOI: 10.22608/MJCU. 1155-1163, September 2025
www.medicaljournalofcairouniversity.net

Thyroid Dysfunction in Pregnancy as a Risk Factor for Gestational Diabetes Mellitus

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Abstract

Background: Gestational diabetes mellitus (GDM) and thyroid dysfunction are two significant endocrine disorders in pregnancy, with impact on maternal and fetal health. Growing evidence suggests a potential association between thyroid autoimmunity and the development of GDM.

Aim of Study: To study the association of thyroid autoimmunity and/or dysfunction with occurrence of GDM.

Subjects and Methods: A cross-sectional study comprised 50 pregnant females with GDM and 50 without GDM, all were recruited after week 24 of gestation and without known thyroid illness. Thyroid function tests (TSH, FT4) and TPOAb, along with GDM diagnostic testing (OGTT) were done to all patients.

Results: A significant association was found between GDM on one side and TPOAb positivity and TSH on the other.

Conclusions: Thyroid dysfunction may be a risk factor for developing GDM. Screening for thyroid dysfunction and thyroid autoantibodies in early pregnancy may help identify women at risk for GDM. Also, GDM patients should be screened for thyroid dysfunction.

Key Words: *Thyroid Dysfunction – Pregnancy – GDM.*

Introduction

PREGNANCY is a unique physiological state that brings about substantial hormonal, metabolic, and immunological changes to support fetal development and maternal adaptation. Among the key endocrine systems affected is the thyroid axis, which plays a central role in regulating metabolism, fetal neurodevelopment, and maternal health. Dysregulation of thyroid function or the presence of thyroid autoimmunity during pregnancy has been increasingly recognized as a contributor to adverse maternal and perinatal outcomes **[1]**.

GDM is one of the most prevalent metabolic complications of pregnancy, defined by impaired glucose tolerance with onset or first recognition during gestation. It usually manifests between 24–28 weeks of pregnancy, a period characterized by heightened insulin resistance due to placental hormones such as human placental lactogen and progesterone **[2]**. GDM not only increases the risk of obstetric complications, such as macrosomia and preeclampsia, but also poses a long-term risk for the development of type 2 diabetes mellitus (T2DM) in the mother and metabolic disorders in the offspring **[3]**.

In parallel, autoimmune thyroid diseases, including Hashimoto's thyroiditis and Graves' disease, are among the most common autoimmune conditions affecting women of reproductive age. These disorders are marked by the presence of thyroid-specific autoantibodies, notably TPOAb and thyroglobulin antibodies (TgAb), which can impair thyroid hormone production **[4,5]**. Even in the absence of overt hypothyroidism or hyperthyroidism, thyroid autoimmunity is associated with pregnancy complications such as miscarriage, preterm delivery, and impaired glucose tolerance **[6]**.

Recent research has explored the potential interplay between thyroid autoimmunity and GDM. Shared pathophysiological mechanisms have been proposed, including chronic low-grade inflammation, altered immune responses, and hormonal imbalances affecting both thyroid and glucose metab-

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olism [7]. Thyroid hormones are known to influence hepatic glucose production, peripheral insulin sensitivity, and pancreatic β-cell function. Subclinical hypothyroidism may exacerbate insulin resistance, while thyrotoxicosis can increase hepatic glucose output and contribute to β-cell stress [8].

Several epidemiological studies and meta-analyses have indicated that the presence of thyroid autoantibodies, particularly TPOAb, may be an independent risk factor for GDMeven in euthyroid women [9]. Subclinical hypothyroidism has also been associated with a higher incidence of GDM, especially in populations with sufficient iodine intake [10,11]. However, findings across studies remain heterogeneous, and the causal relationship remains to be definitively established.

Understanding the potential link between thyroid autoimmunity and GDM is of critical importance. Both conditions independently increase the risk of adverse pregnancy outcomes and may exert synergistic effects when coexistent. Moreover, early identification of thyroid dysfunction or autoimmunity could serve as a predictive marker for GDM risk, allowing for earlier interventions and improved maternal-fetal care [6,12].

This study aims to explore the association between thyroid autoimmunity and/or dysfunction and the development of GDM in a cohort of pregnant women. Through a controlled cross-sectional cohort, it investigates the prevalence of thyroid autoantibodies and abnormal thyroid function tests in pregnant women with and without GDM.

Patients and Methods

Patients were recruited from Kasr Al-Ainy Obstetrics and Gynecology Department and Endocrinology Outpatient Clinic, Cairo University Hospital, over a period from January 2024 to January 2025. The study was approved by the scientific committee in March 2023. An informed written consent was obtained from all participants.

The study comprised a total of 100 pregnant women, after 24 weeks gestation, divided into two groups: Group A: 50 pregnant women diagnosed with GDM, Group B: 50 pregnant women without GDM. Patients with pre-existing type 1 or type 2 diabetes, or prediabetes, those with chronic systemic diseases (e.g., autoimmune disorders, cardiovascular diseases), pregnancies resulting from assisted reproduction methods and patients with known thyroid disorders that were being treated with levothyroxine (LT4) or antithyroid drugs were all excluded from the study cohort.

All subjects underwent thorough history taking and clinical examination. Serum TSH, and FT4 as well as TPOAb were measured besides routinely done investigations.

Diagnosis of GDM:

Glucose Tolerance Testing:

GDM was diagnosed using the one-step 75gram oral glucose tolerance test (OGTT), as per the guidelines established by the American Diabetes Association (ADA) 2025. This test was performed at 24-28 weeks of gestation following an overnight fast of 8 hours, during which only water was permitted. All participants were instructed to refrain from consuming food or beverages other than water during the fasting period.

1- Fasting Blood Glucose Measurement: A fasting blood sample was taken to determine the baseline glucose level.
2- Glucose Consumption: A glucose solution containing 75g of glucose was ingested by the patient.
3- Post-Glucose Blood Samples: Blood samples were collected at 1 hour and 2 hours following the ingestion of the glucose solution to evaluate glucose metabolism.

The diagnosis of GDM was confirmed if any one of the following thresholds were exceeded during the 75gram OGTT:

- Fasting Plasma Glucose: ›_92mg/dL (5.1mmol/L)
- 1-hour Plasma Glucose: ›_180mg/dL (10.0mmol/L)
- 2-hour Plasma Glucose: ›_153mg/dL (8.5mmol/L)

Any participant with one or more values exceeding the specified thresholds was diagnosed with GDM.

Data collection and statistical analysis:

The data collected from clinical evaluations and laboratory investigations were documented in a pre-designed format.

Statistical methods:

Data management and analysis were performed using Statistical Package for Social Sciences (SPSS) vs. 27. Numerical data were summarized using means and standard deviations or medians and/or ranges, as appropriate. Categorical data were summarized as numbers and percentages. Estimates of the frequency were done using the numbers and percentages. Numerical data were explored for normality using Kolmogrov-Smirnov test and Shapiro-Wilk test. Chi square or Fisher's tests were used to compare between the independ-

ent groups with respect to categorical data, as appropriate.

Comparisons between two groups for normally distributed numeric variables were done using the Student's *t*-test while for non-normally distributed numeric variables, comparisons were done by Mann-Whitney test.

To measure the strength of association between the normally distributed measurements, Pearson's correlation coefficients was computed (*r* is the correlation coefficient & it ranges from -1 to +1),

- +1 indicates positive correlation.
- –1 indicates negative correlation.
- 0 indicates no correlation.

Spearman's correlation coefficients were calculated for non-normally distributed variables, r -values:

- From 0 to 0.25 (–0.25) = Little or no correlation;
- From 0.25 to 0.50 (–0.25 to 0.50) = Fair degree of correlation;
- From 0.50 to 0.75 (–0.50 to -0.75) = Moderate to good correlation;
- Greater than 0.75 (or –0.75) = Very good to excellent correlation.

Pearson's and Spearman's correlation tests were used for linear correlation between variables. The (+) sign was considered as indication for direct correlation i.e. increase frequency of independent lead to increase frequency of dependent & (–) sign as indication for inverse correlation i.e. increase frequency of independent lead to decrease frequency of dependent, also we consider values near to 1 as strong correlation & values near 0 as weak correlation.

To measure the independent effect of different factors on occurrence of GDM, factors which had significance level less than 0.10 were selected to enter into stepwise logistic regression analysis. Logistic regression was done to give adjusted odds ratio and magnitude of the effect of different risk factors in relation to gestational diabetes. Odds Ratio (OR) and 95% Confidence Interval (95% CI) were done also (95% CI that doesn't contain 1.0 is considered significant).

Receiver operating characteristic curve (ROC curve) was done to determine the best cutoff point, sensitivity, specificity and area under the curve for TSH and thyroid peroxidase antibodies. The accuracy of the test depends on how well the test separates the group being tested into those with and without gestational diabetes. Accuracy is measured by the area under the ROC curve. A larger area under a ROC curve (AUC) indicates superior test performance, with 1 representing 100% sensitivity and specificity and 0.5 representing no discriminatory utility. The cutoff limit for an abnormal test result that produces the point nearest the upper left corner on the ROC graph is optimal if false-positive and false negative results are equally undesirable. Criteria to qualify for AUC were as follows: 0.90 – 1 = Excellent, 0.80-0.90 = good, 0.70-0.80 = Fair; 0.60-0.70 = Poor; and 0.50-0.6 = Fail. The optimal cutoff point was established at point of maximum accuracy. All tests were two tailed & Probability (*p*-value) ≤ 0.05 is considered significant.

Results

Demographic data analysis showed no statistically significant differences between both groups. The mean age in group A and B was 26±2 and 25±4 respectively. The blood pressure, complete blood count, liver function tests and kidney function tests were normal in both groups with no statistically significant differences.

TSH and thyroid peroxidase antibodies were significantly higher among pregnant females with GDM (group A) compared to pregnant females without GDM (group B), *p*-value <0.001. There was no statistical difference between the two groups as regarding FT4. Table (1) shows mean values of thyroid function tests, TPOAb as well as classification of patients according to thyroid status. Comparison between the 2 groups regarding TSH and TPOAb is illustrated in Fig. (1) and Fig. (2) respectively.

Table (1): Thyroid profile of study groups.

	Group A n=50 (%)	Group B n=50 (%)	*p*-value
Thyroid profile:			
Euthyroid	47 (94)	50 (100)	0.242
Subclinical hypothyroid	3 (6)	0 (0)	
	Median (IQR1)	Median (IQR1)	
TSH (mU/L)	4 (3.2-7.3)	2.1 (1.5-4.8)	<0.001
FT4 (pmol/L)	11.8 (10.4-12.8)	11.9 (11-13)	0.262
TPOAb (IU/ml)	47 (2.4-404)	20.6 (4.4-188)	<0.001

[1]IQR interquartile range (25th-75th)

p-value <0.05 is considered significant.

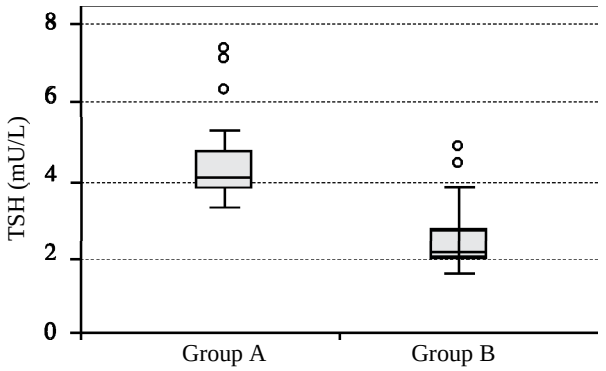

Fig. (1): Boxplot representing TSH level among study groups, median value(IQR) 4 mU/L (3.2-7.3) in Group A, 2.1 mU/L (1.5-4.8) in Group B. *p*-value <0.001.

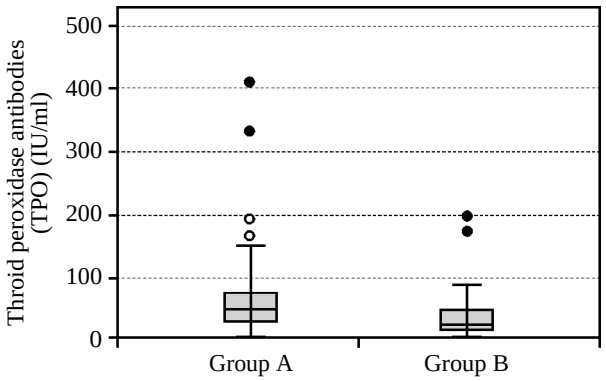

Fig. (2): Boxplot representing thyroid peroxidase antibodies level among study groups, median value (IQR) 47 IU/ml (2.4-404) in Group A, 20.6 IU/ml (4.4-188) in Group B. *p*-value <0.001.

Linear correlation was done to test for TSH and TPOAb against other variables, with little significant impact. A positive correlation was found between TSH and TPOAb. Correlation coefficients are shown in Table (2).

Multivariate analysis was done to test for independent risk factors for GDM. Potential factors with significant correlation with GDM occurrence were entered into the model. TSH was found to be independently correlated with GDM development. Odds ratio and coefficient are shown in Table (3).

The regression coefficient shows the effect of each variable after controlling the effect of other variables in the model. The model shows that TSH level was the most important predictor of gestational diabetes. For every unit increase in TSH level, the risk of GDM increases by 30 times.

Receiver operating characteristic curve (ROC) was done to test for the specificity and sensitivity of TSH and TPOAb as potential risk factors for GDM development. TSH showed a higher performance in predicting GDM with 100% sensitivity and 92% specificity. Values are shown in Table (4). ROC curves for TSH and anti-TPO are illustrated in Fig. (3) and Fig. (4) respectively.

Table (2): Correlation between TSH and TPOAb with different factors.

	TSH		TPOAb	
	r	*p*-value	*r*	*p*-value
Age	0.19	0.054	0.12	0.24
Number of gravidities	0.38	0.001	0.27	0.007
Number of parities	0.31	0.002	0.18	0.082
Number of abortions	0.28	0.004	0.33	<0.001
Systolic blood pressure	0.06	0.589	-0.14	0.16
Diastolic blood pressure	0.12	0.25	-0.05	0.639
TPOAb	0.44	<0.001	0.44	<0.001
Free T4	–0.21	0.035	-0.16	0.107
Hemoglobin	0.14	0.167	0.09	0.395
White blood cells	0.08	0.434	-0.02	0.873
Platelet	0.03	0.797	0.08	0.411
Albumin	0.07	0.483	0.18	0.079
Total bilirubin	–0.03	0.776	-0.02	0.811
AST	–0.05	0.623	-0.08	0.41
ALT	0.05	0.631	0.14	0.161
Serum creatinine	–0.03	0.735	-0.08	0.427
Urea	0.01	0.945	0.05	0.638

r is the correlation coefficient & it ranges from -1 to +1.
p-value <0.05 is considered significant.

Table (3): Shows the variable which was significant in the stepwise logistic regression.

Variables	B	SE	OR	95.0% CI for OR	*p*-value
TSH	3.4	0.7	30.4	8-115.3	<0.001

B : Regression coefficient. OR: Odds ratio.
SE: Standard error. CI : Confidence interval.

Table (4): Receiver operating characteristic curve for (TSH and TPOAb) for prediction of GDM.

Variables	Cutoff point	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	AUC	95% CI for AUC	*p*-value
TSH	>2.9	100	92	93	100	0.96	0.90-0.99	<0.001
TPOAb	>24.1	80	56	65	74	0.72	0.62-0.81	<0.001

PPV: Positive predictive value. AUC: Area under ROC curve.
NPV: Negative predictive value. *p*-value <0.05 is considered significant.

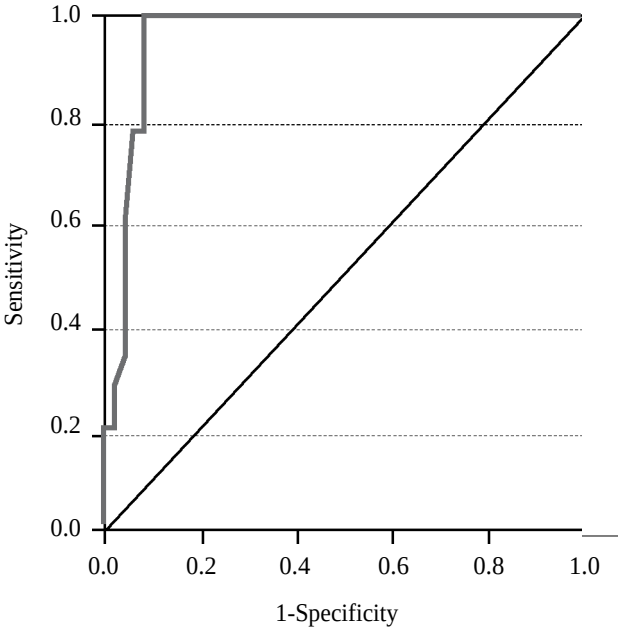

Fig. (3): ROC curve for TSH for diagnosis of gestational diabetes.

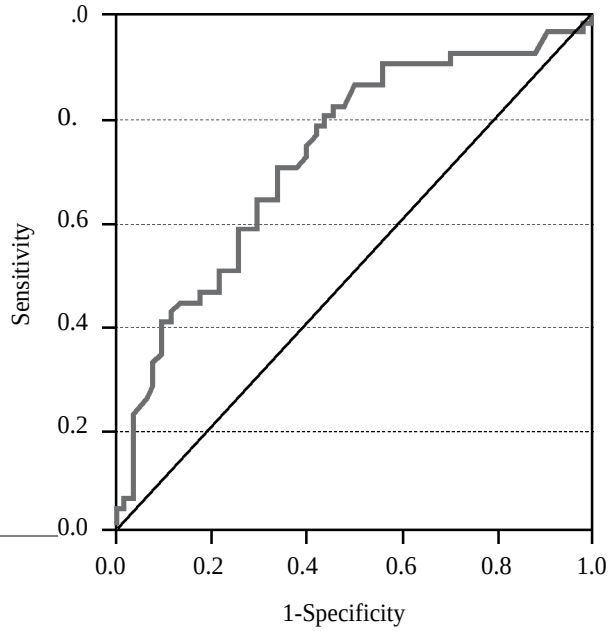

Fig. (4): ROC curve for Thyroid peroxidase antibodies for diagnosis of gestational diabetes.

Discussion

In this study, findings demonstrated a significant association between thyroid function parameters and thyroid autoimmunity markers and the presence of GDM. Pregnant women with GDM exhibited significantly higher levels of TSH compared to their non-diabetic counterparts, with median TSH values of 4 mU/L versus 2.1mU/L respectively (p<0.001). Furthermore, TPOAb titers were considerably elevated among pregnant women diagnosed with GDM, reflecting a robust association between thyroid autoimmunity and GDM. In contrast, FT4 levels showed no significant difference between both groups (p=0.262). Additionally, subclinical hypothyroidism was present exclusively within the GDM group (6%), whereas euthyroid status was maintained by all women without GDM.

The significant elevation in TSH and TPOAb among GDM patients observed in our study aligns with several published findings. Recent literature supports the concept that elevated TSH levels and thyroid autoimmunity markers are independently associated with higher risks of GDM. For instance, a comprehensive meta-analysis involving 44 studies concluded that subclinical hypothyroidism significantly increases the risk of developing GDM (OR 1.54; 95% CI: 1.03–2.30) and noted a strong association between elevated TPOAb levels and GDM occurrence (OR 1.49; 95% CI: 1.07–2.07) ***[13]***.

Our results are further corroborated by Huang et al. ***[14]***, who found that isolated positive TPOAb-significantly increase the risk of GDM independent of maternal FT4 levels. Similar conclusions were reached by Sitoris et al. ***[7]***, who reported a significant association between elevated TPOAband GDM, particularly in older pregnant women. The potential mechanistic link between TPOAband impaired glucose tolerance during pregnancy involves chronic inflammatory responses mediated by pro-inflammatory cytokines, such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), as previously described ***[17,18]***. This inflammatory state can impair insulin receptor sensitivity, promote pancreatic beta-cell dysfunction, and consequently result in insulin resistance and glucose intolerance.

Interestingly, our findings showed no significant differences in FT4 levels between groups, indicating that overt hypothyroxinemia was not a prominent feature in this sample population. This aligns with Yang et al. ***[15]*** who similarly reported no strong association between early trimester euthyroid states with thyroid antibodies and subsequent development of GDM, reinforcing the complexity of the relationship and highlighting that TPOAb, rather than FT4 levels, might be a more sensitive marker for predicting GDM ***[15]***.

The identification of subclinical hypothyroidism exclusively in the GDM group in our study

further supports the clinical relevance of elevated TSH levels as a potential marker for glucose intolerance during pregnancy. Osinga et al. [16] recently emphasized this clinical importance, suggesting routine screening of thyroid function, especially in women at higher risk of GDM or with elevated TPOAb, to mitigate potential metabolic and obstetric complications [16].

The mechanisms underlying the association between thyroid dysfunction and GDM likely involve disrupted glucose homeostasis through impaired insulin sensitivity and secretion, possibly mediated by chronic inflammation and autoimmune processes inherent to thyroid autoimmunity [17,18]. Additionally, thyroid hormones exert essential regulatory effects on pancreatic beta-cell function and insulin sensitivity, thus further supporting the biological plausibility of these clinical findings [18].

The current analysis also demonstrated significantly higher TSH levels among women diagnosed with GDM. Furthermore, these elevated TSH levels were associated with an increased prevalence of thyroid peroxidase antibody (TPOAb) positivity, indicating a potential underlying autoimmune thyroid process. The observed positive correlation between elevated TSH levels and TPOAb titers aligns with findings from other studies. Huang et al. [14] demonstrated a similar significant association between TSH and TPOAb, emphasizing thyroid autoimmunity's role as an independent predictor of GDM risk. They proposed that positive TPOAbcould represent early markers of autoimmune-mediated thyroid dysfunction, thereby increasing maternal insulin resistance and subsequently influencing glucose metabolism adversely .Osinga et al. [16], also demonstrated a positive relationship between elevated TPOAb and elevated TSH, suggesting a likely autoimmune etiology contributing to subclinical hypothyroidism and insulin resistance in pregnancy. This relationship indicates that autoimmune thyroiditis, marked by elevated TPOAb, is closely linked to subclinical thyroid dysfunction and may exacerbate maternal metabolic disturbances leading to GDM.

The observed correlation between increased TSH and higher gravidity and parity in women with GDM is consistent with previous reports highlighting a relationship between multiparity and thyroid dysfunction. A recent individual participant data meta-analysis identified multiparity as a risk factor for developing elevated TSH levels and thyroid autoimmunity during pregnancy [16].This association suggests that repeated pregnancies could enhance susceptibility to autoimmune thyroid dysfunction, possibly due to immune modulation or cumulative stress on thyroid function across pregnancies.

Additionally, the absence of significant correlations between TSH and routine biochemical parameters (such as liver and kidney function tests, blood pressure, and complete blood counts) in our study suggests that elevated TSH may exert its influence on pregnancy primarily through metabolic and autoimmune pathways rather than through overt systemic dysfunction. This finding aligns with previous research showing limited interactions between mild thyroid dysfunction (subclinical hypothyroidism) and standard biochemical or hematological parameters in pregnancy [13,15]. Alongside, the current results did not show significant correlations between TPOAb and routine biochemical parameters such as liver function tests, kidney function tests, blood counts, or blood pressure measurements. This finding aligns with observations from Yang et al. [15] who reported no significant biochemical disturbances associated with thyroid autoantibodies in early pregnancy among euthyroid women. This suggests that TPOAb predominantly affect pregnancy outcomes through autoimmune and inflammatory pathways rather than direct systemic biochemical alterations.

Another critical aspect revealed by this study is the indirect impact of gravidity and parity on TPOAb positivity. Women in our GDM group, who also showed higher gravidity and parity, had significantly elevated TPOAb titers. The association between increased parity and thyroid autoimmunity was previously noted by Sitoris et al. [7].

Indicating that cumulative pregnancies may heighten maternal susceptibility to autoimmune diseases, potentially through repeated immune system activation and hormonal fluctuations during successive pregnancies. Thus, multiparity appears to potentiate thyroid autoimmunity risk, indirectly contributing to increased GDM susceptibilityFrom a clinical perspective, the authors strongly suggest that screening for thyroid autoimmunity, particularly TPOAb, should be considered routinely during early pregnancy, especially in women with higher gravidity and parity. Early detection of elevated TPOAb could enable prompt interventions, such as closer metabolic monitoring and lifestyle modifications, potentially reducing GDM incidence and related adverse pregnancy outcomes.

In conclusion, the observed correlation between TPOAb and TSH levels in women with GDM reinforces the hypothesis that thyroid autoimmunity significantly contributes to GDM risk. It highlights

the need for targeted clinical strategies aimed at early identification and management of thyroid autoimmunity in pregnancy.

Future research should focus on larger, multicentre prospective studies to validate findings, evaluating the cost-effectiveness of universal vs. targeted screening.

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الارتباط بين الاضطراب المناعى أو الوظيفي للغدة الدرقية فى السيدات الحوامل المصابين بسكرى الحمل

يعد سكرى الحمل (GDM) وأمراض المناعة الذاتية للغدة الدرقية (AITDs) من أكثر الاضطرابات الغدد الصماء شيوعًا خلال فترة الحمل، ولهما تأثيرات بالغة على صحة الأم والجنين. يحدث سكرى الحمل نتيجة اضطراب فى تحمل الجلوكوز يُكتشف لأول مرة أثناء الحمل، بينما تشمل أمراض الغدة الدرقية المناعية الذاتية مثل التهاب هاشيموتو ومرض جريفز، وتنتج عن مهاجمة جهاز المناعة لأنسجة الغدة الدرقية.

تهدف هذه الرسالة إلى دراسة العلاقة بين وجود اضطرابات في وظيفة الغدة الدرقية أو وجود أجسام مضادة مناعية ضدها وبين حدوث سكرى الحمل، من خلال مراجعة علمية ودراسة تحليلية لحالات واقعية.

سكرى الحمل: سكرى الحمل هو اضطراب مؤقت فى التمثيل الغذائى للكربوهيدرات يظهر خلال الثلث الثانى أو الثالث من الحمل. تتسبب التغيرات الهرمونية فى زيادة مقاومة الجسم للأنسولين، مما يؤدى إلى ارتفاع مستويات السكر فى الدم لدى النساء ذوات القابلية الوراثية أو وجود عوامل خطر مثل السمنة أو تاريخ عائلي للسكرى.

التشخيص والعلاج: يعتمد التشخيص على اختبار تحمل الجلوكوز، ويوصى بالفحص بين الأسبوع ٢٤ و٢٨ من الحمل. يشمل العلاج تعديل النظام الغذائى والنشاط البدنى، وفي بعض الحالات استخدام الإنسولين أو أدوية خافضة للسكر. بعد الولادة، يختفى المرض غالبًا لكن تظل المرأة معرضة لسكرى النوع الثانى مستقبلاً.

أمراض الغدة الدرقية المناعية الذاتية أثناء الحمل : تشمل هذه الأمراض التهاب الغدة الدرقية المزمن (هاشيموتو) وفرط نشاطها (جريفز)، وتتميز بوجود أجسام مضادة مثل anti–TPO وanti–Tg وTRAb. تؤثر الاضطرابات فى مستويات هرمونات الغدة (TSH، T3، T4) على التمثيل الغذائى والوظائف الحيوية، وقد تؤدى إلى مضاعفات فى الحمل.

التشخيص والعلاج: يتم الاعتماد على قياس TSH وFT4 واختبارات الأجسام المضادة، بالإضافة إلى التصوير بالموجات فوق الصوتية. يشمل العلاج استخدام ليفوثيروكسين فى حالات القصور، وأدوية مضادة للدرقية فى حالات فرط النشاط.

العلاقة بين أمراض الغدة الدرقية وسكرى الحمل:

تشير الأدلة الحديثة إلى وجود علاقة بيولوجية ومناعية بين أمراض الغدة الدرقية المناعية وسكري الحمل. تشمل الآليات المحتملة:

- الالتهاب والمناعة الذاتية: حيث تؤدى الالتهابات المزمنة ووجود أجسام مضادة إلى زيادة مقاومة الأنسولين.
- تأثيرات الهرمونات: تؤثر هرمونات الغدة الدرقية بشكل مباشر على تنظيم سكر الدم ووظائف خلايا بيتا فى البنكرياس.
- زيادة TSH أو وجود anti–TPO: أظهرت دراسات عديدة أن ارتفاع مستويات TSH ووجود anti–TPO يرتبطان بزيادة خطر الإصابة بسكرى الحمل حتى فى النساء ذوات الوظائف الدرقية الطبيعية (يُطلق عليهن «يوثايرود»).

الدراسة: أجريت دراسة حالة وشاهد فى مستشفى القصر العينى على ١٠٠ سيدة حامل (٥٠ مصابة بسكرى الحمل و٥٠ سليمة). تم تقييم مستويات TSH، FT4، anti–TPO، وanti–Tg. وأظهرت النتائج:

- ارتفاع معدلات الأجسام المضادة (خصوصًا anti–TPO) بين المصابات بسكرى الحمل.
- ارتباط واضح بين القصور الدرقى دون أعراض (Subclinical hypothyroidism) وحدوث سكرى الحمل.

الاستنتاجات والتوصيات:

- وجود خلل فى وظائف الغدة الدرقية أو الأجسام المضادة المناعية يمثل عامل خطر مستقل لحدوث سكرى الحمل.
- يُنصح بإدراج اختبارات الغدة الدرقية والأجسام المضادة ضمن فحوصات الحمل، خاصة للنساء المعرضات للخطر.
- الإدارة المتكاملة بين أطباء الباطنة والتوليد والتغذية مهمة لتقليل المضاعفات وتحسين النتائج.
- هناك حاجة لمزيد من الدراسات لتحديد جدوى الفحص الشامل لجميع الحوامل ومدى تأثير العلاج المبكر على تقليل المضاعفات.

التوصيات:

١. توسيع نطاق الفحص المبكر لوظائف الغدة الدرقية (TSH وFT4) والأجسام المضادة (anti–TPO وanti–Tg) لجميع الحوامل، خاصة في المجموعات عالية الخطورة.

٢. اعتبار الأجسام المضادة للغدة الدرقية (anti–TPO) مؤشرًا هامًا للتنبؤ بإمكانية الإصابة بسكرى الحمل حتى فى غياب أعراض أو قصور واضح فى الغدة.

٣. دمج رعاية الغدة الدرقية والسكرى ضمن متابعة الحمل، من خلال تعاون بين تخصصات الباطنة والغدد الصماء وأمراض النساء.

٤. إجراء دراسات مستقبلية واسعة النطاق (متعددة المراكز) لتقييم الفائدة الاقتصادية والسريرية من تطبيق الفحص الشامل وتحديد تأثير العلاج المبكر على نتائج الحمل.

٥. تثقيف الكوادر الطبية والنساء الحوامل حول أهمية المتابعة الدورية لوظائف الغدة الدرقية أثناء الحمل لتقليل احتمالات المضاعفات.