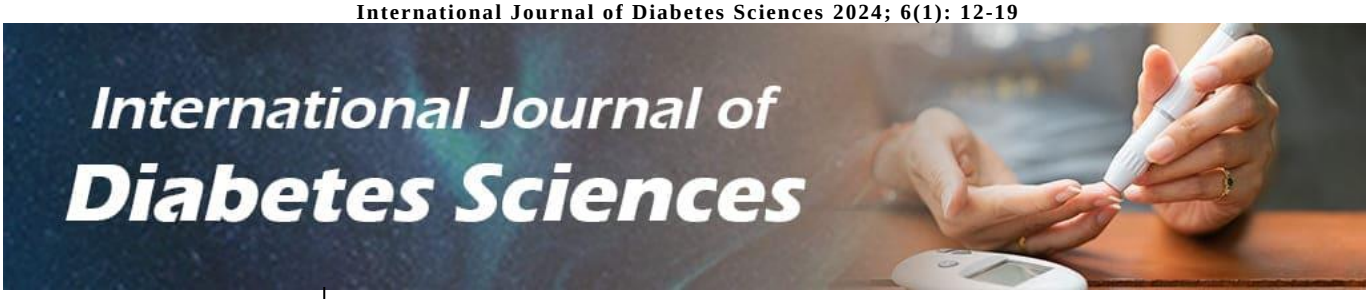

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Evaluating the relationship between thyroid function and insulin resistance in gestational diabetes

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Abstract
Background: Gestational diabetes mellitus (GDM) and thyroid dysfunction during gestation (GTD) are the two most prevalent endocrinopathies during pregnancy.
Aims of the study: Provide an overview of the peculiar aspects of GDM and thyroid disorder and highlight the potential interactions and clinical consequences of these two frequent clinical conditions.
Methodology: This case-control study included 50 pregnant women suffering from gestational diabetes who were diagnosed by doctors at Maysan Children's and Maternity Hospital for the period from 5/11/2023 to 5/20/2024, along with 50 pregnant women who did not have gestational diabetes and 50 non-pregnant women who did not have gestational diabetes as a healthy control group. Ethical approval was obtained from all research participants; 5 ml was withdrawn from all participants, placed in a gel tube, and left at room temperature for 15 minutes until coagulation, after which it was separated using a centrifuge for 15 minutes, and the serum was stored at a temperature of -20 Celsius until use. Fasting glucose levels were measured using a spectrophotometer, and HBA1c and insulin levels were measured using Cobas e411. As for thyroid hormones, their levels were measured using Avias-6 device.
Result: The results showed higher levels of fasting and cumulative blood sugar and insulin levels in pregnant women compared to the control group in the first and second age groups. The results also showed that there was no statistical significance in T_3 levels, while thyroxine showed a decrease in the gestational diabetes group, and thyroid-stimulating hormone showed a significant increase in the same group in the first and second age groups.
Conclusions: Low levels of thyroxine and high levels of thyroid-stimulating hormone indicate an imbalance in thyroid secretions. High levels of sugar and insulin may also be related to thyroid disorders, as thyroid hormones can affect the metabolism of sugar and its levels in the blood.

Keywords: Gestational diabetes mellitus, hypothyroidism, insulin resistance, hemoglobin A1C, and thyroid function test

Introduction
Gestational diabetes mellitus (GDM) is characterized by any level of impaired glucose tolerance that occurs for the first time or is first identified during pregnancy. Gestational diabetes mellitus GDM can be categorized into two types, A1GDM and A2GDM. Diet-controlled gestational diabetes (GDM), also known as A1GDM, refers to the management of gestational diabetes without the use of medication, relying solely on nutritional therapy [1]. These hormones include growth hormone, prolactin, corticotropin-releasing hormone, and progesterone play a role in causing insulin resistance and elevated blood sugar levels in pregnancy [2, 3]. Gestational diabetes impacts (2 - 10%) of pregnancies in the United States. Women who develop gestational diabetes (GDM) are at a heightened risk of developing diabetes mellitus within 10 to 20 years post-pregnancy, with a likelihood increase ranging from 35% to 60% [4, 5]. During pregnancy, a specific percentage of women experience thyroid disorders. In a recent meta-analysis study, the prevalence rates of TPO Ab positivity and SCH were reported as 7.5% and 3.1%, respectively. Thyroid disorders have a strong association with diabetes. The occurrence of thyroid disorders is higher in women with either type 1 or type 2 diabetes mellitus, and thyroid disorders also elevate the risk of developing type 2 diabetes mellitus. Despite numerous studies, there is no conclusive evidence to demonstrate the impact of thyroid disorders on the occurrence of gestational diabetes mellitus (GDM) [6, 7]. A comprehensive retrospective study of 27,513 Chinese women revealed that elevated FT_4 levels have a protective effect against gestational diabetes mellitus (GDM) [8].

Nevertheless, two studies conducted in the United States failed to establish any noteworthy correlations [9]. Similarly, other studies have reported positive [10], negative [8], or no associations [11]. Both hypothyroidism/subclinical hypothyroidism (SCH) and thyroid peroxidase antibody (TPO Ab) positivity have been indicated to have a heightened correlation with an increased risk of gestational diabetes mellitus (GDM). This risk is further amplified when these conditions coexist. Nevertheless, this pattern failed to find support in various other studies, as no statistically significant connections between these factors were found. These studies only examined one or two thyroid disorder statuses, primarily subclinical hypothyroidism, while disregarding other thyroid disorders. Further research utilizing comprehensive thyroid markers and disorders is necessary to enhance the existing literature and gain deeper insights into this area [12, 13, 14]. Insulin resistance is characterized by a diminished physiological reaction to the stimulation of insulin in specific tissues, such as the liver, muscle, and adipose tissue. Hyperinsulinemia results from the beta cells' compensatory increase in insulin synthesis and the body's inability to digest glucose effectively being hampered by insulin resistance (IR) [15]. High blood pressure (Hypertension), abnormal lipid levels (Dyslipidemia), visceral adiposity the buildup of fat around internal organs hyperuricemia higher blood uric acid levels increased inflammation markers endothelial dysfunction and a prothrombic state are just a few of the metabolic consequences of insulin resistance (IR). Type 2 diabetes mellitus (T2DM), non-alcoholic fatty liver disease (NAFLD), and metabolic syndrome (MetS) may all arise from the emergence of insulin resistance (IR) [16].

Methodology

This case-control study included 50 pregnant women suffering from gestational diabetes who were diagnosed by doctors at Maysan Children's and Maternity Hospital, along with 50 pregnant women who did not have gestational diabetes and 50 non-pregnant women who did not have gestational diabetes as a healthy control group. Ethical approval was obtained from all research participants. The study was conducted for the period between 5/11/2023 and 5/20/2024, and all patient data, such as age and nutritional nature, were recorded. 5 ml of blood was drawn from each participant, placed in a gel tube, and left for 15 minutes at room temperature until coagulation, after which it was separated using a centrifuge for 15 minutes at a speed of 3000 rpm. The blood serum is kept at a temperature of -20 Celsius. Fasting glucose levels were measured using a spectrophotometer, and HBA1c and insulin levels were measured using cobas e411. As for thyroid hormones, their levels were measured using Avias-6 device.

Statistical analysis

Statistical analysis is often used to analyze quantitative data and provides methods for data description and simple inference for continuous and categorical data. The procedure involves the collection of data, leading to a test of the relationship between two statistical data sets. In this study, all data are presented as mean ± standard deviation. The statistical analyses were performed using SPSS (version 26) and using dependent t-tests (two-tailed) and independent t-tests (two-tailed) for normally distributed variables, whereas the Mann-Whitney and Wilcoxon tests weret used for those variables that were not normally distribute. $p<0.05$ was considered statistically significant.

Ethical approval

Before the samples were taken, all of the patients who were going to be part of this study were properly informed and gave their verbal permission. The Committee on Publication Ethics at the Maysan Maternity Hospital, gave its approval to the study.

Results

FBS, HbA1C and insulin levels of (25-30) years in Gestational diabetes mellitus pregnant (GDM), normal pregnant and control groups

The results of the study show significant statistical differences between the three study groups: pregnant women with gestational diabetes (GDM), normal pregnant women, and the control group, in the means and standard deviations of morning glucose levels (FBS), glycated hemoglobin (HbA1C), and insulin levels. For pregnant women with gestational diabetes, the values were 173.58±33.48 for FBS, 8.22±0.82 for HbA1C, and 13.46±4.05 for insulin. For normal pregnant women, values were recorded as 113.03±6.78, 5.65±1.31, and 9.62±1.81, respectively. While for the control group, the values were 94.58±10.12 for FBS, 4.89±0.86 for HbA1C, and 6.24±1.87 for insulin. The P value for all measures was 0.000, indicating significant statistical differences between the three groups.

The levels of T_3, T_4 and TSH hormone of (25-30) years in Gestational diabetes mellitus pregnant (GDM), normal pregnant and control groups.

The study shows the differences in the means and standard deviations of the levels of T_3, T_4, and TSH hormones for the following study groups: pregnant women with gestational diabetes (GDM), normal pregnant women, and the control group for the age group (25–30) years. For the group with gestational diabetes, values were recorded as follows: for T_3, it was 2.50±0.51 nmol/L, for T_4, it was 115.00±35.48 nmol/L, and for TSH, it was 4.05±1.45 µIU/mL. For normal pregnant women, the values were 2.33±0.66, 152.03±20.51, and 2.36±0.70, respectively. For the control group, the values were 2.45±0.53, 134.94±19.88, and 2.47±0.65, respectively. The P value indicates that there were no statistically significant differences in T_3 levels between groups (P = 0.646), while differences in T_4 and TSH levels were statistically significant (P = 0.000), indicating a significant effect of gestational diabetes on these levels in the study.

Table 1: Mean ±SD of FBS, HbA1C and insulin levels of (25-30) years of Gestational diabetes mellitus pregnant (GDM), normal pregnant and control groups

Parameters	FBS	HbA1C	insulin
GDM pregnant	173.58±33.48	8.22±0.82	13.46±4.05
Normal pregnant	113.03±6.78	5.65±1.31	9.62±1.81
Control group	94.58±10.12	4.89±0.86	6.24±1.87
P. value	0.000	0.000	0.000

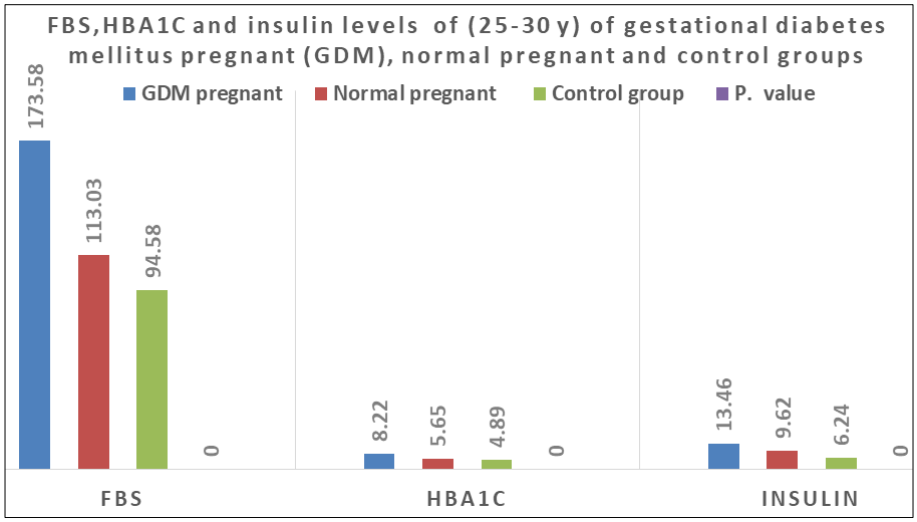

Fig 1: The difference in the FBS, HbA1c and insulin levels of (25-30) years in Gestational diabetes mellitus pregnant (GDM), normal pregnant and control groups

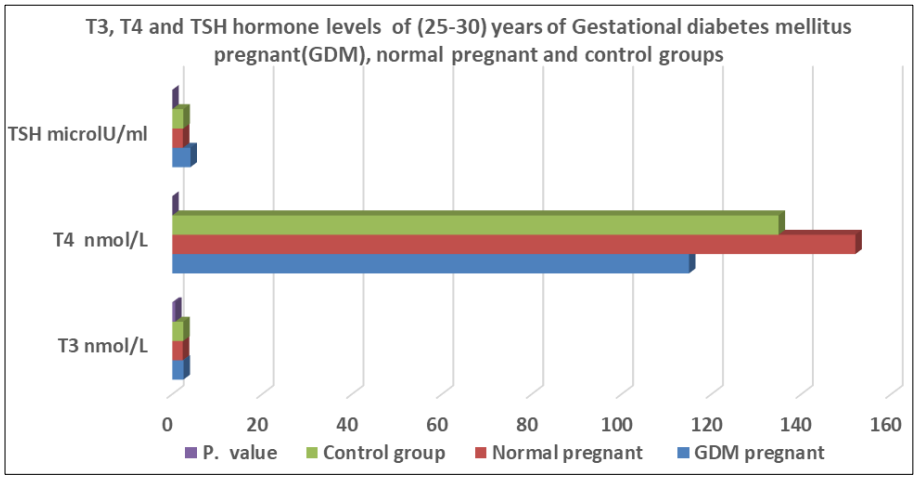

Fig 2: The difference in the T_3, T_4 and TSH hormone levels of (25-30) years in Gestational diabetes mellitus pregnant (GDM), normal pregnant and control groups

Table 2: Mean ±SD of T_3, T_4 and TSH hormone levels of (25-30) years in Gestational diabetes mellitus pregnant (GDM), normal pregnant and control groups

Parameters	T_3 nmol/L	T_4 nmol/L	TSH microl U/ml
GDM pregnant	2.50±0.51	115.00±35.48	4.05±1.45
Normal pregnant	2.33±0.66	152.03±20.51	2.36±0.70
Control group	2.45±0.53	134.94±19.88	2.47±0.65
P. value	0.646	0.000	0.000

The levels of FBS, HbA1C and insulin of (31-36) years in Gestational diabetes mellitus pregnant (GDM), normal pregnant and control groups: The results highlight the significant differences between the study groups for the age group (31-36) years, which include pregnant women with gestational diabetes mellitus (GDM), normal pregnant women, and the control group, in terms of means and standard deviations of fasting glucose levels (FBS), glycated hemoglobin percentage (HbA1C), and insulin levels. For the group with gestational diabetes, FBS levels were 192.18±40.65, HbA1C 8.45±0.96, and insulin 13.54±4.75. For normal pregnant women, the values were 111.17±7.36, 5.59±1.17, and 9.02±1.60, while for the control group, they were 92.39±11.52, 5.69±1.36, and 6.16±1.72, respectively. The P value for all measures was 0.000, indicating the presence of statistically significant differences between the three groups, which confirms the importance of distinguishing in health follow-up between these groups.

Table 3: Mean ±SD of FBS, HbA1C and insulin levels of (31-36) years in Gestational diabetes mellitus pregnant (GDM), normal pregnant and control groups.

Parameters	FBS	HbA1C	insulin
GDM pregnant	192.18±40.65	8.45±0.96	13.54±4.75
Normal pregnant	111.17±7.36	5.59±1.17	9.02±1.60
Control group	92.39±11.52	5.69±1.36	6.16±1.72
P. value	0.000	0.000	0.000

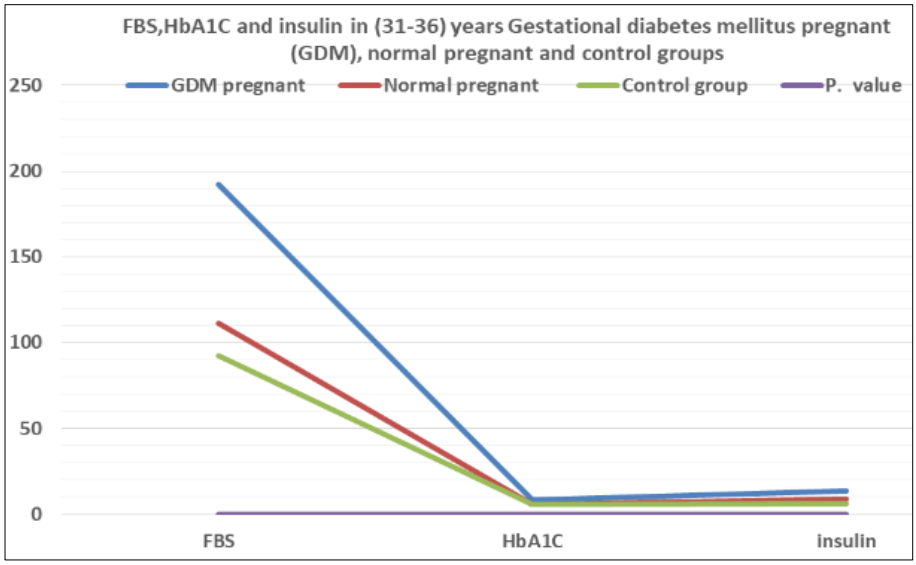

Fig 3: The difference in the FBS, HbA1C and insulin levels of (31-36) years in Gestational diabetes mellitus pregnant (GDM), normal pregnant and control groups.

The levels of T_3, T_4 and TSH hormone of (31-36) years in Gestational diabetes mellitus pregnant (GDM), normal pregnant and control groups.

The study provides in-depth data on the means and standard deviations of the levels of T_3, T_4, and TSH hormones among three groups: pregnant women with gestational diabetes (GDM), normal pregnant women, and a control group, for the age group (31-36) years. For pregnant women with gestational diabetes, the values were found to be as follows: T_3 2.41±0.80 nmol/L, T_4 125.28±38.94 nmol/L, and TSH 3.81±1.46 µIU/mL. For normal pregnant women, the values recorded were T_3 2.39±0.71, T_4 154.47±17.45, and TSH 2.67±0.69. While the control group values were T_3 2.40±0.72, T_4 146.43±22.73, and TSH 2.37±0.73, The P value for T_3 levels was 0.851, indicating that there were no significant statistical differences between the three groups in the level of this hormone, while the differences in T_4 and TSH levels were statistically significant (P = 0.001 and P = 0.000, respectively), indicating a significant effect of diabetes status. Pregnancy affects the levels of these hormones.

Table 4: Mean ±SD of T_3, T_4 and TSH hormone levels of (31-36) years in Gestational diabetes mellitus pregnant (GDM), normal pregnant and control groups

Parameters	T_3 nmol/L	T_4 nmol/L	TSH microlU/ml
GDM pregnant	2.41±0.80	125.28±38.94	3.81±1.46
Normal pregnant	2.39±0.71	154.47±17.45	2.67±0.69
Control group	2.40±0.72	146.43±22.73	2.37±0.73
P. value	0.851	0.001	0.000

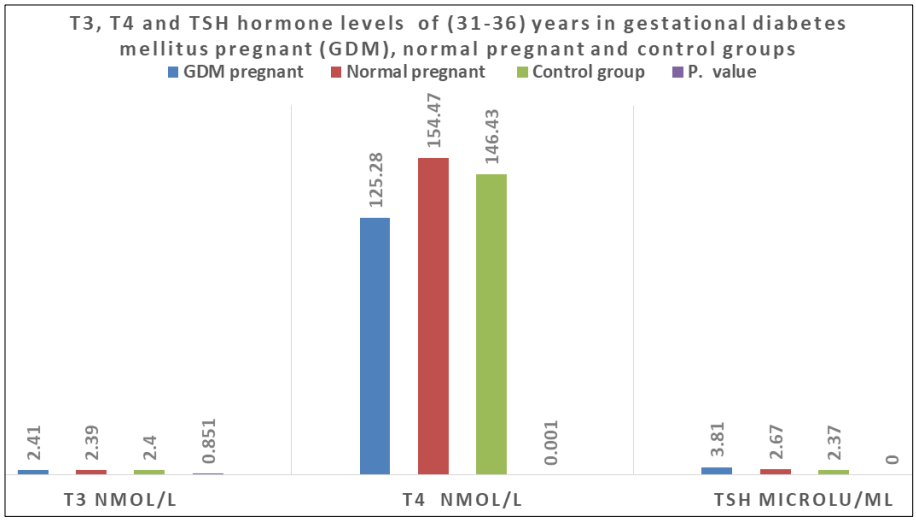

Fig 4: The difference in the T_3, T_4 and TSH hormone levels of (31-36) years among the study group

Discussion

Pregnancy is associated with hyperinsulinemia and insulin resistance (IR), which can potentially elevate the likelihood of diabetes in specific women. Gestational diabetes is a condition characterized by the presence of glucose intolerance that is either observed or identified for the first time during pregnancy. The current definition does not exclude the possibility that there was undetected glucose intolerance before the pregnancy. As a result, the Endocrine Society has suggested using the phrase "hyperglycemia in pregnancy" as a more appropriate alternative. When high blood sugar is first found during pregnancy, the International Association of Diabetes and Pregnancy Study Groups (IADPSG) describes it as either gestational diabetes

mellitus (GDM) or "overt diabetes." In 2013, the World Health Organization (WHO) proposed a way to name high blood sugar levels first noticed during pregnancy. They offered two terms: "diabetes mellitus (DM) in pregnancy" or "gestational diabetes mellitus (GDM) [17]. The study groups, consisting of women with gestational diabetes who were classified as normal weight, overweight, obese, or having high blood pressure, showed a substantial increase in fasting sugar, cumulative sugar, and insulin levels compared to the control group. The findings are consistent with [18, 19]. A progressive intrauterine growth restriction (IR) occurs during a regular pregnancy, starting around the middle of the pregnancy and progressing throughout the third trimester. Potential factors contributing to insulin resistance (IR) during pregnancy encompass hormones and adipokines released by the placenta, such as human placental lactogen, human placental growth hormone, and tumor necrosis factor (TNF)-α. Elevated levels of progesterone, estrogen, and cortisol additionally contribute to a disruption in the equilibrium between glucose and insulin during pregnancy. During pregnancy, the pancreas increases its secretion of insulin in response to peripheral insulin resistance. Gestational diabetes mellitus (GDM) occurs when a woman's pancreas is unable to produce enough insulin to meet the metabolic needs caused by internal resistance. Moreover, this condition of relatively reduced ability to process glucose is worsened by higher caloric consumption, reduced physical activity, and greater accumulation of fat in the mother's body [20, 21]. During the initial phases of pregnancy, there is an increase in the secretion of insulin, while the sensitivity to insulin either remains constant, declines, or possibly increases. Insulin sensitivity steadily decreases during the middle of pregnancy and continues to diminish until the late third trimester, when it reaches its maximum level of decline. The placenta rebounds upon delivery. As a result, gestational diabetes mellitus (GDM) usually appears during the later part of the second trimester of pregnancy and disappears right after childbirth [20]. There are multiple risk factors that are associated with the development of GDM. The most common risk factors for type 2 diabetes mellitus include obesity, advanced maternal age, a strong family history of diabetes mellitus, chronic glucosuria, belonging to an ethnic group with a high incidence of the disease, and older maternal age. Other risk factors for gestational diabetes mellitus (GDM) include a medical history of giving birth to kids with a birth weight of 4000 g or more, multiple miscarriages, unexplained fetal deaths, and essential hypertension or pregnancy-induced hypertension [22]. Rajput evaluated the diagnostic accuracy of HbA1C levels taken during the 24th and 28th weeks of pregnancy in 607 women to detect gestational diabetes mellitus. The diagnosis of GDM was determined based on the discovery that HbA1C, with a threshold value of 5.95%, had an accuracy of 80.5%, a sensitivity of 28.6%, and a specificity of 97.2% [23]. Furthermore, when the cutoff point for HbA1C was established at 5.45% or above, the sensitivity for detecting GDM was 85.7% and the specificity was 61.1%. A threshold of 5.35% for HbA1C levels in the first trimester and 5.75% between 20 and 24 weeks of pregnancy has been identified as most effective for detecting gestational diabetes mellitus at 24-28 weeks. When comparing two studies, it seems that the HbA1C threshold levels might increase as the pregnancy progresses [24]. However, this syndrome is often caused by prolonged insulin resistance during pregnancy, resulting in impaired function of pancreatic β-cells. β-cells perform the primary task of storing and releasing insulin in response to the buildup of glucose in the bloodstream [25, 26]. During pregnancy, insulin sensitivity changes as the growing baby needs energy to meet its metabolic needs. In the early stages of pregnancy, there is a heightened responsiveness to insulin, allowing for better absorption of glucose into fat cells. In later stages of pregnancy, the body needs more energy, so this gets it ready for that [27]. It is thought that sustained fuel excess is the main cause of β-cell malfunction, which is typified by uncontrolled insulin production. Insulin resistance is facilitated by the elevation of certain hormones, including oestrogen, progesterone, leptin, cortisol, placental lactogen, and placental growth hormone, as pregnancy advances [28]. β-cell changes can happen at any point in the insulin signaling chain, including changes that happen after the protein is translated, the production of β-cells, and gene changes that are linked to insulin signaling [26]. $GLUT_4$, which moves glucose molecules across the cell's plasma membrane, can't do its job as well when insulin signals are changed. The rate at which insulin speeds up the absorption of glucose into cells is 54% slower in pregnant women with gestational diabetes mellitus (GDM) than in pregnant women who are healthy [27]. Progesterone levels increase gradually throughout pregnancy, from the first to the third trimester. These levels hinder the phosphoinositol-3kinase (PI3K) pathway by decreasing the production of IRS-1 and obstructing the movement and absorption of glucose facilitated by $GLUT_4$ [29]. Pregnancy is characterized by a significant elevation in estrogen levels, which is associated with a reduction in insulin function. Human placental lactogen (hPL) has dual functionality, acting as both an insulin mimic and an anti-insulin agent. Raised amounts of the molecule are linked to pregnancy, which increases the mother's ability to absorb glucose and synthesize glycogen [30]. Hypoglycemia is caused by pituitary and human placental growth hormones, which also impair hepatic gluconeogenesis and lower glucose uptake and glycogen production. These hormonal consequences help diabetes develop [31]. An excessive amount of pro-inflammatory cytokines and adipokines builds up in fatty tissues during pregnancy. This makes the body inflamed, which makes leptin come out and insulin resistance happen. Too much production of leptin sets off the hypothalamus, which makes people eat too little and causes high blood sugar during pregnancy [32]. Adiponectin makes it easier to stop gluconeogenesis, insulin signaling, and the burning of fats. AMP-activated protein kinase (AMPK) and the transcription factor PPARα (Peroxisome proliferator-activated receptor alpha) are turned on in liver cells to make this happen. In turn, this makes IRS-1 work harder in cells that are sensitive to insulin and [33]. Adipose tissue macrophages that have multiplied in liver cells release pro-inflammatory cytokines, including interleukin 6 (IL-6), interleukin-1β (IL-1β), and tumor necrosis factor-α (TNF-α). These cytokines are associated with chronic inflammation in patients with gestational diabetes mellitus (GDM). Insulin resistance is brought on by either degrading IRS-1 through the $STAT_3$-SOCS3 pathway or by increasing IRS-1 serine phosphorylation to lower the activity of insulin receptor (IR) tyrosine kinase [34]. Our research indicates that the occurrence of GDM has shown a steady rise in correlation with higher BMI levels before pregnancy. The prevalence of

GDM was found to be approximately 13.44%, a significantly higher rate compared to women of European and American ancestry. According to research, Asians are more likely to have diabetes than people of other races, even though their body mass index (BMI) stays the same. Possible explanations for this racial disparity include the following points: Firstly, it is crucial to acknowledge that centripetal obesity mellitus acts as a separate indicator, and Asians amass visceral and abdominal fat to a higher degree than their European and American counterparts with the same waist size. Furthermore, Asian societies have a higher frequency of insulin resistance, even though they weigh less than those of Europe and America [36]. Thirdly, mitochondrial dysfunction influences insulin resistance and central obesity. Studies have shown that in East Asian populations, having more of certain variations in mitochondrial DNA 16–189 is linked to getting type 2 diabetes, but not in European people. Diabetes is a problem all over the world, but it needs more attention in Asia than anywhere else [37]. The results demonstrated that there was no statistically significant disparity in T_3 levels between the control group and any group of pregnant women with gestational diabetes. In contrast, the group of women diagnosed with gestational diabetes experienced a reduction in T_4 levels when compared to the control group. In contrast, there was a significant increase in TSH levels observed in all groups of pregnant individuals diagnosed with gestational diabetes. The study groups consisted of women who had gestational diabetes and women who did not, in contrast to the control group. The findings provided here support this observation [38]. The low FT_4 level functions as an independent risk factor for GDM was shown by the steady decline in the association between FT_4 levels and the prevalence of GDM. The metabolism of glucose depends critically on thyroid hormones. Glucose metabolism is mostly dependent on the hormone T_3. Approximately 80% of T_3 in the bloodstream is transformed outside of the thyroid gland through the process of mono-deiodination of T_4 and the activity of deiodinase [39, 40]. More precisely, a reduction in FT_4 level was linked to an elevation in FT_3 and BMI. Also, being overweight was linked to a higher risk of getting GDM. There was also a study that found the relationship between FT_3/FT_4 levels and BMI went up in pregnant women who were euthyroid and had low FT_4 levels. This points to a rise in the activity of peripheral deiodinase [41]. Studies have also shown that too much energy use makes the change from peripheral T_4 to T_3 go up. This finding suggests that the amount of energy used might affect how well peripheral deiodinase works. The studies reported earlier suggest a correlation between diabetes and a low FT_4 level [40, 41]. Studies conducted on the general population have discovered that the control of glucose metabolism by thyroid hormone is influenced by many processes. Hormones in the thyroid can shorten the time it takes for insulin to break down, speed up the process, and make more inactive types of insulin available. They also raise the level of glucose transporter 2 on the outer layers of liver cells, which increases the release of glucose in the liver [42]. The cAMP makes β-adrenergic receptors more sensitive, which in turn makes catecholamines more sensitive. Catecholamines are strong chemicals that speed up the breakdown of glycogen. Researchers have found a number of issues with the links between the hypothalamic-pituitary axis and the T_3 receptor on thyroid cells in people who have diabetes. Consequently, multiple separate connections between glucose metabolism and thyroid function have been discovered. These connections serve as shared routes through which thyroid failure and diabetes might arise. Currently, nonetheless, the literature that elucidates the role of maternal thyroid hormones in the regulation of glucose metabolism during pregnancy is sparse. Our research results support earlier studies that have indicated insulin resistance in those with subclinical or hypothyroidism diagnoses [43]. Research done *in vitro* and *in vivo* in living creatures has indicated that this resistance may be brought on by a decrease in peripheral tissue glucose utilization or a decrease in insulin need [42, 43]. It was found in the Fremantle Diabetes Study that there is a strong negative link between blood TSH levels and insulin sensitivity in people with type 2 diabetes. Kapadia *et al*. discovered that people with mild hypothyroidism had a lot more signs of insulin resistance than a control group [44]. The scope of our analysis spans a wide range of advantages. Patients that were excluded from the study had different types of thyroiditis and immune system illnesses, including diabetes caused by immunological disorders. This is so because type 1 diabetes and autoimmune thyroiditis have been strongly linked in prior studies. Patients on thyroid replacement therapy and those using hormone drugs that may impair thyroid function either before or during pregnancy were not included in our study. This made it possible for us to investigate in real-life possible correlations between early pregnancy thyroid hormone levels and gestational diabetes mellitus (GDM). The trial did not look at FT_3 levels in later stages of pregnancy. The prevalence of thyroid dysfunction in pregnant women with type 1 diabetes was reported to be 40.9% 8 and 40.7% 7 in the studies conducted by Gllas PR and Lois Jovanic P, respectively, which is higher than our own findings. The reason for this difference can be related to the fact that 80% of our patients had type 1 diabetes, whereas only 20% had type 2 diabetes. Conversely, every participant in the research conducted by Gllas PR and Lois Jovanic P had type 1 diabetes, a condition that is linked to a greater occurrence of thyroid dysfunction.The numbers are 7 and 8. In a separate study involving Iranian women with diabetes who were not pregnant, it was discovered that thyroid dysfunction was present in 40% of those with type 1 diabetes mellitus and in 20% of those with type 2 diabetes mellitus [45, 46].

Conclusion

An imbalance in thyroid secretions is indicated by low levels of thyroxine and excessive amounts of thyroid-stimulating hormone. Elevated concentrations of sugar and insulin have been found to have a potential correlation with thyroid problems, since thyroid hormones can influence the sugar metabolism and its concentrations in the bloodstream.

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