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Iron status and its clinical implications in women with diabetes and thyroid disorder: a cross-sectional study

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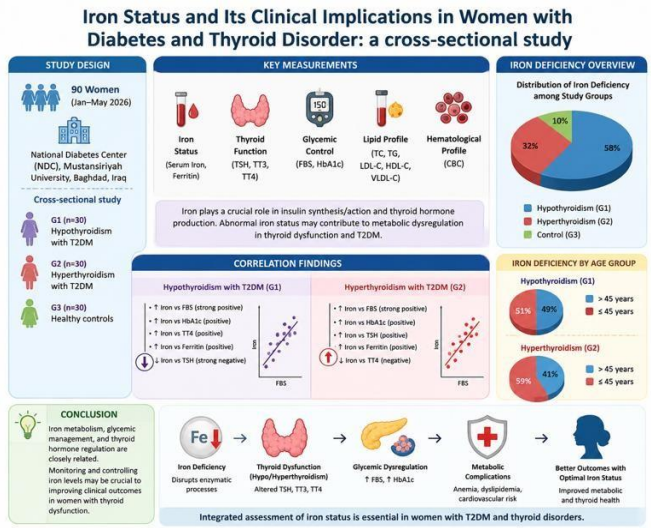
Abstract

Introduction: Thyroid dysfunction and Type 2 Diabetes Mellitus are intimately linked to iron status. Because iron plays a crucial role in the synthesis and action of insulin as well as in the generation of thyroid hormone through its involvement in important enzymatic activities, maintaining appropriate iron levels is vital for correct metabolic function. **Objective:** This study aimed to assess iron status in women with Type 2 Diabetes Mellitus and thyroid dysfunction. **Methods:** Ninety participants were collected from the National Diabetes Center (NDC) at Mustansiriyah University. **Results:** They were divided into three groups: G1: (30) hypothyroidism patients with T2DM, G2: (30) hyperthyroidism patients with T2DM, and G3: (30) healthy controls there was a strong positive correlation between iron vs FBS and positive correlation with iron and HbA1c, TT4, and serum ferritin levels in hypothyroidism patients, while A strong negative Correlation between iron level and TSH in hypothyroidism patients. **Conclusion:** A strong positive correlation between iron vs FBS and positive correlation with iron vs HbA1c, TSH and serum ferritin levels in hyperthyroidism patients, while A negative Correlation between iron level and TT4 in hyperthyroidism patients. Conclusion, iron metabolism, glycemic management, and thyroid hormone regulation are closely related. Monitoring and controlling iron levels may be crucial to improving clinical outcomes for individuals with thyroid dysfunction since changes in iron status may contribute to metabolic dysregulation in both hypothyroid and hyperthyroid states.

Keywords: Hematological profile. Hypothyroidism. Hyperthyroidism. Iron level. T2DM.

Graphical Abstract



Source: Own authorship.

Introduction

The two most prevalent endocrine illnesses seen in clinical practice are thyroid conditions and diabetes mellitus. Thyroid issues and diabetes have long been linked, and it has been demonstrated that the two conditions have an impact on one another **[1,2]**. Thyroid hormones play a role in controlling pancreatic function and the metabolism of carbohydrates, while diabetes has varying effects on thyroid function tests **[3,4]**.

Women with diabetes are much more likely to get thyroid problems. The most prevalent condition is

hypothyroidism, which can result in severe hypoglycemia by slowing metabolism and increasing insulin action. For diabetic women, annual thyroid screening is necessary because both hypothyroidism and hyperthyroidism affect glycemic control [5]. Since iron is necessary for the thyroid peroxidase enzyme, which is responsible for the synthesis of thyroid hormones, iron deficiency (ID) in a diabetic woman is strongly associated with an increased risk of thyroid dysfunction, especially hypothyroidism [6]. Compared to men, diabetic women, particularly those with Type 2 Diabetes Mellitus (T2DM), are more susceptible to this interaction, which frequently leads to worse thyroid-related outcomes [7].

Thyroid dysfunction and Type 2 Diabetes Mellitus are intimately linked to iron status. Because iron plays a crucial role in the synthesis and action of insulin as well as in the generation of thyroid hormone through its involvement in important enzymatic activities, maintaining appropriate iron levels is vital for correct metabolic function [8]. Effective insulin action, normal thyroid hormone (T3 and T4) synthesis, and general metabolic balance are all supported by adequate iron status [9]. On the other hand, abnormalities in iron levels, whether excessive or insufficient, might negatively impact thyroid function and glycemic regulation, resulting in additional metabolic issues [10].

This study aimed to assess iron status in women with Type 2 Diabetes Mellitus and thyroid dysfunction, and to investigate its clinical implications by examining its relationship with thyroid hormone levels, glycemic control, and associated metabolic abnormalities.

Material and Methods

Study Design

This study followed a cross-sectional design in accordance with the STROBE guidelines (link: https://www.strobe-statement.org/). Accessed on January 12, 2026.

Participants and Variables

Ninety participants were collected from the National Diabetes Center (NDC) at Mustansiriyah University between January 2026 and May 2026. They were divided into three groups: G1: (30) hypothyroidism patients with T2DM, G2: (30) hyperthyroidism patients with T2DM, and G3: (30) healthy controls. Every participant had to undergo a clinical examination to rule out any other illnesses. Each participant's demographic information, including sex, age, height, weight, and BMI, was recorded. Blood samples were taken for laboratory investigation which included: FBS, HbA1c, TC,TG, LDL-C,HDL-C , VLDL-C were measured by using automated analyzer (cobas c111) Germany. Serum Ferritin, TT3, TT4 and TSH were measured by Minividas with Biomeruix Kits. Complete Blood count was measured by automated system SYSMIX. Mean ± SD were expressed for all data.

Statistical Analysis

Statistical analysis was performed using LSD, considering $p<0.05$ as the lowest limit of significance. Analysis of variance (ANOVA) for equality of means (testing of coincidence). It's used SPSS programs, version 22 (SPSS, Chicago, IL, USA).

Ethical Approval

The study was approved by the institutional ethics committee in Mustansiriyah University, Baghdad, Iraq, and adheres to the ethical principles outlined in the declaration of Helsinki, 2025. Informed consent was obtained from all participants involved in the study, with all procedures explained in detail before participation.

Results

Table 1. Clinical parameters among groups of study (Hypothyroidism with T2DM, Hyperthyroidism with T2DM and Control).

Parameters	Hypothyroidism with T2DM (G1)	Hyperthyroidism with T2DM (G2)	Control (G3)	ANOVA (F value) (Post Hoc Significance)
	Means ±SD	Means ±SD	Means ±SD	
	n=30	n=30	n=30	
Age (years)	39.33±13.68	40.46±12.23	41.86±10.19	F = 0.020
BMI (kg/m^2)	29.66±3.58	25.85±2.53	25.27±3.93	F = 4.138(a,b)
FBS (mg/dL)	203.26±58.56	193.6±39.36	99.8±5.76	F = 7.764(a,b,c)
HbA1c (%)	8.59±1.46	8.43±1.36	4.83±0.31	F = 0.361(b,c)
TC (mg/dL)	232.1±53.44	214.96±44.43	158.96±28.87	F = 2.359(b,c)
TG (mg/dL)	269.5±58.96	244.63±336.60	1.56±4.2812	F = 3.668 (b,c)
HDL (mg/dL)	34.7±1.36	43.73±2.95	53.06±5.34	F= 0.441(b,c)
LDL (mg/dL)	108.12± 8.23	105.31±6.84	82.65±9.54	F = 6.416(b,c)
VLDL (mg/dL)	18.25.36±1.23	20.51±1.26	23.25±1.86	F = 1.223(b,c)
TT_3 (nMol/L)	0.83±0.25	4.29±1.08	1.98±0.46	F = 1.185(a,b,c)
TT_4 (ng\dL)	47.18±10.60	127.87±13.93	97±14.67	F = 108.5016 (a,b,c)
TSH (IU/mL)	27.44±15.26	0.05±2.12	2.44±1.51	F = 47.229 (a,b,c)

*= The p-value is < 0.0001. The result is significant at p < 0.05. **= The p-value is< 0.05. The result is insignificant at p < 0.05; a= Difference between hypothyroidism Vs. hyperthyroidism groups. The result is significant at p < 0.05; b= Difference between hypothyroidism Vs. control groups. The result is significant at p < 0.05; c= Difference between hyperthyroidism Vs. control groups. The result is significant at p < 0.05. Source: Own authorship.

Table 2. Hematological profile levels among groups of study (Hypothyroidism, Hyperthyroidism and Control).

Parameters	Hypothyroidism with T2DM (G1)	Hyperthyroidism with T2DM (G2)	Control (G3)	p-value
	Means ±SD	Means ±SD	Means ±SD	
	n=30	n=30	n=30	
Ferritin (ng/mL)	12.69±4.43	17.53±6.24	36.98±8.60	F = 3.659 (a,b,c)
Iron (ng/mL)	55.43±11.04	68.26±8.91	89.06±9.16	F = 4.716(a,b,c)
Hb%	8.8±0.48	8.83±0.87	13±0.67.13	F = 3.263(b,c)
WBC(10^3µL)	1.33±5.22	2.35±6.2.12	1.03±9.22	F = 3.081(a,b,c)
Platelet	16.7±202.18	17.13±216.12	14.3±255.12	F = 3.505 (a,b,c)

*= The p-value is < 0.0001. The result is significant at p < 0.05; **= The p-value is < 0.05. The result is insignificant at p < 0.05; a= Difference between hypothyroidism Vs. Hyperthyroidism groups. The result is significant at p < 0.05; b= Difference between hypothyroidism Vs. Control groups. The result is significant at p < 0.05; c= Difference between hyperthyroidism Vs. Control groups. The result is significant at p < 0.05. Source: Own authorship.

In Table 3 there was a strong positive correlation between iron vs FBS and positive correlation with iron and HbA1c, TT4, and serum ferritin levels in hypothyroidism patients, while A strong negative Correlation between iron level and TSH in hypothyroidism patients. A strong positive correlation between iron vs FBS and positive correlation with iron vs HbA1c,TSH and serum ferritin levels in hyperthyroidism patients, while A negative Correlation between iron level and TT4 in hyperthyroidism patients.

Table 3. Correlation Coefficient of iron level with (Hypothyroidism with T2DM, Hyperthyroidism with T2DM).

	Iron levels (ng/mL)	
	Hypothyroidism with T2DM	Hyperthyroidism with T2DM
	Correlation Coefficient(r)	Correlation Coefficient(r)
Iron vs FBS	0.702**	0.536**
Iron vs HbA1c	0.321*	0.269*
Iron vs TT4	0.334*	0.343*-
Iron vs TSH	0.678**-	0.280*
Iron vs ferritin	0.282*	0.232*

**Correlation is significant at p< 0.01; *Correlation is significant at p< 0.05. Source: Own authorship.

As shown in Figure 1, distribution percentage of iron level deficiency among study groups (Hypothyroidism, Hyperthyroidism and Control). In Figure 2, Distribution percentage of iron level deficiency in Hypothyroidism patients according age groups(more than 45 years and less than 45 years). In figure 3, Distribution percentage of iron level deficiency in Hyperthyroidism patients according age groups (more than 45 years and less than 45 years).

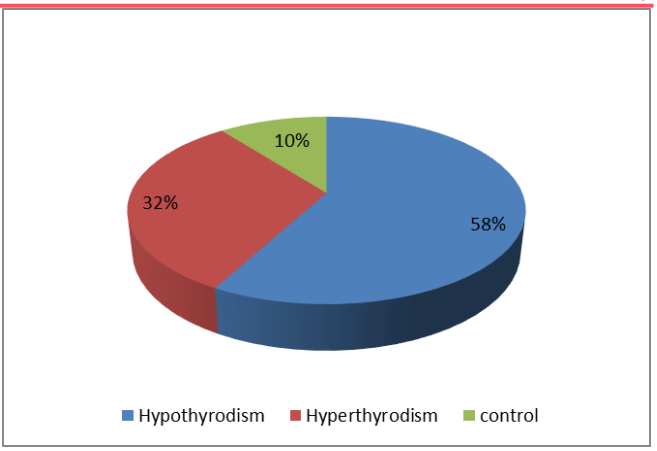


Figure 1. Distribution of iron deficiency among study groups (Hypothyroidism, Hyperthyroidism and Control). Source: Own authorship.

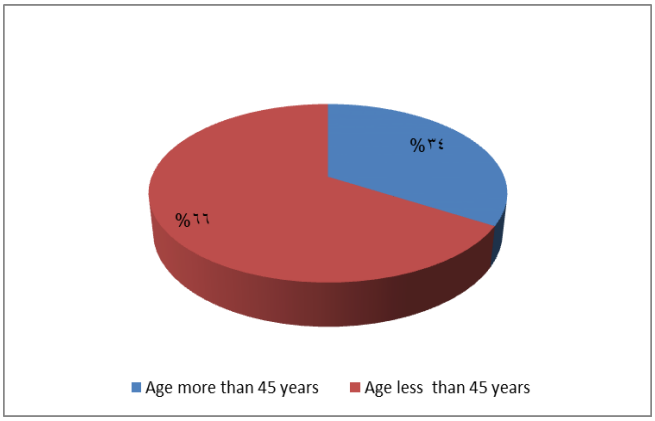


Figure 2. Distribution of iron deficiency in Hypothyroidism patients according age groups. Source: Own authorship.

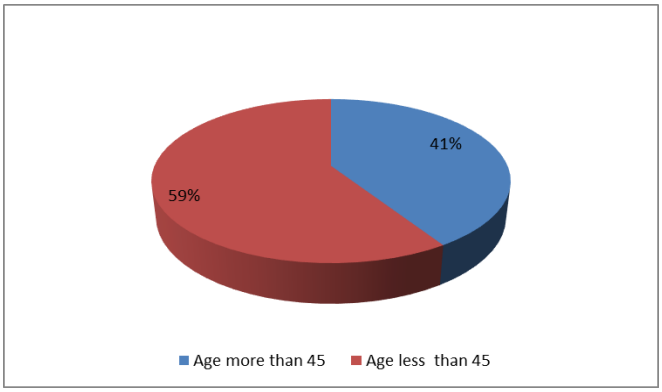


Figure 3. Distribution of iron deficiency in Hyperthyroidism patients according age groups. Source: Own authorship.

Discussion

In comparison to healthy controls, the current study demonstrated that women with thyroid dysfunction had distinct hematological and metabolic changes, with the hypothyroid group exhibiting the most severe abnormalities. This result lends credence to the idea that thyroid dysfunction, especially in women with concomitant endocrine and metabolic disorders, is strongly connected with poor glucose homeostasis, lipid abnormalities, and iron imbalance **[11,12]**.

Age did not significantly differ across the groups under investigation in the current study, suggesting that the observed biochemical changes were less likely to be age-driven. BMI, on the other hand, differed significantly, with the hypothyroid group having greater levels. This could be explained by hypothyroidism's decreased basal metabolic rate, fluid retention, and propensity for weight gain. Additionally, poor glycemic status, cardiometabolic burden, and insulin resistance may all be exacerbated by elevated BMI **[13]**.

When compared to controls, the patient groups' fasting blood sugar levels were noticeably higher, and this increase was particularly pronounced in hypothyroid women. The overall trend still points to poor glycemic control in thyroid dysfunction, even though HbA1c showed less noticeable variance. Hepatic glucose generation, insulin sensitivity, pancreatic beta-cell activity, and carbohydrate metabolism are all known to be impacted by thyroid hormones. Thus, through distinct mechanisms, both hypothyroidism and hyperthyroidism may impair glucose control **[14,15]**.

The lipid profile also showed significant disturbances, particularly increased total cholesterol, triglycerides, and LDL levels in the thyroid-disorder groups. These findings are in agreement with previous studies demonstrating that hypothyroidism is strongly associated with dyslipidemia due to reduced LDL receptor activity, impaired lipid clearance, and altered hepatic lipid metabolism **[16-18]**.

In terms of thyroid hormones, the current findings demonstrated the anticipated hormonal pattern, with hypothyroid patients having lower TT4 and higher TSH **[10–13]**. This supports the biological validity of the groups under study and validates decreased thyroid gland activity. Since iron is required for thyroid peroxidase activity, which is required for thyroid hormone synthesis, iron status may have a direct bearing on this finding. Therefore, thyroid dysfunction itself may hinder iron uptake and use, and iron shortage may exacerbate thyroid dysfunction **[19,20]**.

The iron profile revealed significantly lower ferritin and serum iron levels in the patient groups, especially in hypothyroid women. Hemoglobin was also lower in those groups, suggesting an increased tendency toward iron deficiency and anemia. This agrees with recent studies reporting that women with hypothyroidism are more likely to have depleted iron stores and lower ferritin concentrations **[21,22]**. One possible explanation is that hypothyroidism reduces gastrointestinal motility and gastric acid secretion, both of which may impair iron absorption. In addition, menstrual abnormalities and chronic inflammation may further aggravate iron deficiency in women with thyroid disease **[21,23]**.

The fact that each parameter represents a distinct biological process explains why some biomarkers rise while others fall. For instance, because thyroid dysfunction impairs insulin sensitivity and lipid metabolism, fasting blood sugar, total cholesterol, triglycerides, and LDL may rise. Conversely, decreased iron reserves, poor absorption, altered erythropoiesis, or elevated metabolic demand may result in a decrease in ferritin, serum iron, and hemoglobin **[20,23]**. Consequently, it is not paradoxical for elevated metabolic markers and lowered hematological markers to coexist; rather, this indicates the multisystem impact of thyroid dysfunction **[21,22]**.

Iron and fasting blood sugar (FBS) were shown to be strongly positively correlated in both groups, with the link being stronger in hypothyroidism than in hyperthyroidism. This implies that increased oxidative stress and decreased insulin sensitivity may be two ways that excessive iron levels may impede glucose metabolism. The stronger connection in hypothyroid patients may be explained by the fact that excess iron is known to enhance insulin resistance and pancreatic β-cell dysfunction **[24]**.

In both the hypothyroid and hyperthyroid groups, iron significantly positively correlated with HbA1c. This suggests that poor long-term glycemic management may be associated with greater iron status. These results are in line with other research indicating that iron overload may exacerbate long-term hyperglycemia **[25-27]**. In terms of thyroid hormones, both hypothyroidism and hyperthyroidism showed a strong positive association between iron and total thyroxin (TT4). This could be related to iron's function as a cofactor in the production and metabolism of thyroid hormones. Thyroid peroxidase activity, which is critical for hormone synthesis, depends on adequate iron levels **[28]**.

On the other hand, the two groups had different relationships between iron and thyroid-stimulating hormone (TSH). There was a strong negative association in hypothyroidism and a positive correlation in hyperthyroidism. Increased iron levels may be linked to better thyroid function, which lowers TSH levels, according to the inverse relationship in hypothyroid patients. This lends credence to the idea that hypothyroidism may be made worse by iron shortage **[29,30]**. Furthermore, there was a significant correlation between iron levels and ferritin in both the hyperthyroidism and hypothyroidism groups, which is to be expected given that ferritin represents the body's iron reserves. The very weak relationships, however, imply that under thyroid dysfunction, circulation and

stored iron may be regulated differently [31].

According to Figure 1, decreased metabolic activity and decreased gastrointestinal absorption of iron may account for the greater incidence of iron insufficiency in hypothyroid patients. Reduced thyroid hormone levels may result in inefficient iron intake and utilization since thyroid hormones are essential for controlling erythropoiesis and iron usage [32]. Furthermore, menorrhagia in females is frequently linked to hypothyroidism, which can exacerbate iron loss. On the other hand, iron shortage was still seen in certain patients despite the fact that hyperthyroidism is usually linked to an elevated metabolic rate. Increased iron requirement, faster turnover, and occasionally malabsorption could be the cause of this. If consumption falls short of the elevated physiological requirements, the hypermetabolic condition may result in the depletion of iron reserves [33].

Iron deficiency was present in both age categories of hypothyroid patients, as seen in Figure 2, but it was more common in those under 45 than in those over 45. This could be explained by younger people's higher metabolic demands and increased iron loss, especially in females. On the other hand, older individuals had a relatively reduced prevalence, which may have been caused by many underlying causes such chronic illness and impaired iron metabolism [34].

Figure 3 shows that iron deficiency was present in both age groups of hyperthyroidism patients, but it was more common in those under 45 than in those over 45. Elevated metabolic needs and iron turnover linked to hyperthyroid conditions, together with other variables such female menstruation loss, may be responsible for the higher frequency in younger individuals. On the other hand, elderly individuals showed a comparatively reduced prevalence, perhaps as a result of underlying comorbid illnesses and variations in iron metabolism [35].

Conclusion

It was concluded that iron metabolism, glycemic management, and thyroid hormone regulation are closely related. Monitoring and controlling iron levels may be crucial to improving clinical outcomes for individuals with thyroid dysfunction since changes in iron status may contribute to metabolic dysregulation in both hypothyroid and hyperthyroid states.

CRediT

Author contributions: Conceptualization; Data Curation; Formal Analysis: Naji, and Al-Noori; Investigation: Methodology: Project Administration: Supervision: Naji, and Al-Noori; Al-Ani and Tahir; Writing -Original Draft, Writing -Review & Editing: Al-Ani and Tahir.

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Ethical Approval

The study was approved by the institutional ethics committee in Mustansiriyah University, Baghdad, Iraq, and adheres to the ethical principles outlined in the declaration of Helsinki, 2025.

Informed Consent

Informed consent was obtained from all participants involved in the study, with all procedures explained in detail before participation.

Funding

Not applicable.

Data Sharing Statement

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request, and all data is stored following privacy and ethical guidelines.

Conflict of Interest

The authors declare no competing interests.

Similarity Check

It was applied by Ithenticate[@].

Application of Artificial Intelligence (AI)

Not applicable.

Peer Review Process

It was performed.

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