Evaluation of Hepcidin , Hephaestin and Iron Homeostasis in Newly diagnosed Subclinical Hypothyroid patients

Dlveen M Ali Aziz[1], Sherwan F. Salih[2]*

[1] Department of Medical Laboratory, College of Health Sciences, University of Duhok, Duhok, Iraq
[2] Department of Medical Chemistry, College of Medicine, University of Duhok, Duhok, Iraq

*Corresponding author email:dlveenduhoki11@gmail.com (Tel.: +9647508435885)

ABSTRACT

Subclinical hypothyroidism (SCH) is one of the most frequent endocrine diseases, which is defined as high levels of serum thyroid-stimulating hormone (TSH) with normal serum free thyroxine (FT4) levels, Thyroid hormones are important for the proper regulation of iron metabolism and a dysfunctional thyroid gland could disrupt iron balance by influencing the regulation of iron-regulating proteins such as hepcidin and hephaestin. Nevertheless, the connection of these biomarkers with newly diagnosed subclinical hypothyroidism is still unknown. The objective of this study was to evaluate serum hepcidin, hephaestin and iron homeostasis parameter in newly diagnosed subclinical hypothyroid patients and compare them with apparently healthy control.This cross sectional study was done at Duhok City – Kurdistan Region – Iraq from October 2025 to January 2026. A total of (160) samples were collected, (80) were newly diagnosed subclinical hypothyroidism and (80) were apparently healthy for the control group. Serum thyroid stimulating hormone(TSH), free thyroxin (FT4), free triiodothyronin (FT3) and anti thyroperoxidase antibody(Anti-TPO), iron , unbound iron binding capacity (UIBC), hepcidin, hephaestin, were all evaluated. Transferrin saturation (TS%) and total iron binding capacity (TIBC) were calculated, TIBC, serum UIBC and hepcidin concentrations were significantly higher in subclinical hypothyroid patients than in controls (356.46±4.03),(286.97±5.11),(639.43±54.46) respectively, ($p<0.05$), whereas, Serum iron was significantly lower in subclinical hypothyroid patients than in controls ($p<0.05$). SCH patients had higher prevalence of iron deficiency than controls (32.5% vs. 8.7%; $p<0.001$). There were no significant associations of iron parameters, hepcidin, and hephaestin subclinical hypothyroid autoimmunity. However, Serum hephaestin was significantly associated with subclinical ypothyroid severity ($p=0.042$).In conclusion, reduced serum iron and transferrin saturation(TS%), together with elevated total iron-binding capacity, unsaturated iron-binding capacity, and hepcidin concentrations and a greater incidence of iron deficit, were indicative of poor iron homeostasis in newly diagnosed SCH.

KEYWORDS: subclinical hypothyroidism, iron Homeostasis, hepcidin, hephaestin, thyroid Hormons.
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1. Introduction

Subclinical hypothyroidism (SCH) is one of the most common endocrine disorders worldwide, characterized by elevated serum TSH levels with normal levels of serum FT4 (Gosi *.et al,* 2024).Despite being often without clinical manifestations, recent researches show the association between SCH and several pathological conditions, including metabolic syndrome, cardiovascular disease, oxidative stress, and dysregulation of iron metabolism (Jasim, S.*et al,*2023)(Obeagu, E.I.*et al*, 2025). It is estimated that SCH found in about 3% to 10% of the entire adult population, while the prevalence increases to 10% to 20% amongst women and the elderly (Hamid, I. *et al*, 2024). The thyroid hormones have an important function in the regulation of erythropoiesis, iron metabolism, and cell metabolism activities (Köhrle, J.*et al*, 2023.). The dysfunctioning of the thyroid gland can lead to the disturbance in iron metabolism through the changes in iron absorption in the intestines, synthesis of erythropoietin, and the functioning of iron regulatory proteins (Hussein *et al*., 2025).

Iron metabolism is strictly regulated through the use of several proteins, where hepcidin and hephaestin have gained scientific importance in recent times (Manolova, V. and Bachmann, M.F.*et al*, 2021). Hepcidin is a hormone that acts as a key regulator of iron metabolism within the body and is mainly produced by the liver. This hormone binds itself with ferroportin to regulate the iron level within the body(Nemeth, E.*et al*, 2023.). Hephaestin is an iron-oxidizing ferroxidase enzyme, mainly found in enterocytes, necessary for the conversion of ferrous iron (Fe2+) to ferric iron (Fe3+) Lead to inefficient export of iron from the intestine which can lead to iron deficiency and imbalances in iron homeostasis (Hussein *et al.,* 2025).

Although the important involvement of thyroid hormones in iron metabolism has been well established. However, hepcdin-hephaestin levels

together with other iron biomarkers in recently diagnosed cases of SCH are inadequately studied. Some recent studies have found that those with thyroid gland disorders can display abnormal levels of serum ferritin, iron, transferrin saturation(TS%), and hepcidin (Güngör, N.D. and Uzunlulu, M.*et al* , 2022.) (Agha, J.T. and Hayder, Z.S.*et al*, 2025.). This implies a connection between thyroid gland health and iron regulation processes (Ganz *et al* ., 2022). Nonetheless, a proper understanding of the link between SCH and iron balance still remains ambiguous, especially among new SCH without any form of treatment. Therefore the objective of this study was to evaluate serum hepcidin, hephaestin and iron homeostasis biomarkers in newly diagnosed subclinical hypothyroid patients and compare them with apparently healthy control. As well as, to evaluate the association between these biomarkers with autoimmunity and severity of SCH.

2. Materials and Methods

This case-control study was conducted at Duhok University, College of Health Science, Medical Laboratory Department. The study duration was covered from October 2025 to January 2026. A total of (160) participants from Duhok province were included in this study, in which (80) patients with newly diagnosed subclinical hypothyroidism (aged equal or more than 20 years) collected in Azadi Teaching Hospital, and (80) individuals were healthy volunteers involved as controls , selected from medical staff, university employees and public (age and gender matched with the subclinical hypothyroid group). Biochemical parameters measurement was performed at Vin Private Medical Laboratory. The diagnosis of subclinical hypothyroidism is based on the criteria when serumTSH is elevated and serum free T4, freeT3 are normal (Ambasta, A.T.*et al,* 2025.).

Study participants were chosen by convenience, non-random sampling method. All participants completed a questionnaire form which included personal information and a special code for each participant to ensure their privacy. Personal information (Name of the participants and gender, ages, height, weight, and family history of thyroid disorders) and special code for each participants. The BMI was calculated by dividing the weight (kg) by square of height (m) As the following equation: BMI = weight (kg) / (height (m))² (WHO, 2024).

After an overnight fast, sample collection included 8mililiters of the blood was collected from the participants, then transferred to serum separation tubes , labeled by their unique ID code. Serum separation tubes were applied to centrifuge to separate serum from the red blood cell. Serum TSH, FT4, FT3, anti -TPO, iron, UIBC and ferritin measurements were performed by Roche/Hitachi Cobas 6000. Transferrin saturation was calculated by dividing serum iron by TIBC multiplying by 100% as the equation: TS% = serum iron/TIBC×100, and total iron binding capacity was calculated by serum iron plus UIBC as the equation: TIBC = Serum Iron + UIBC (Dortez, S.*et al,* 2023.). Anti -TPO, TSH, FT4, FT3 and ferritin levels were measured by electrochemiluminescence immunoassay method, whereas serum iron and UIBC levels were measured by using a colorimetric method. Hepcidin and hephaestin were measured by using Human hepcidin and hephaestin ELISA kit - BT LAB depending on the sandwich principle that utilizes two antibodies (Mishra & Chopra.*et al,* 2022). Individuals showing the following conditions were excluded from the study like known of thyroid diseases; pregnancy, any systemic diseases, or liver diseases or anemia that could alter study biochemical parameters.

Ethical approval:

The ethical approval was gained from the scientific committee of College of Health Science – University of Duhok and the Directorate of Health in Duhok governorate with the reference number (15102025-8-11) and the date (25 Oct. 2025). Each participant has voluntarily signed a written consent before participating. The study followed Helsinki and CIOMS guidelines to ensure confidentiality and voluntary participation.

Statistical analysis:

Recording and data analysis was done by using Statistical Package for Social Sciences (SPSS) version (26) and normality was assessed using Shapiro-Wilk test. General data and lab results were recorded as number, percentage, means ± standard error (SE) and p-values. One way ANOVA one-way and independent t-test were used for the comparison between the groups, and the Pearson chi-square test to determine significant association between categorical variables. Pearson Correlation was used to determine the relationship between serum hepcidin, hephaestin levels and study biochemical parameters in subclinical hypothyroids . Statistical significance was defined as a p-value (< 0.05).

3. RESULTS

Table 1. The Demographic and anthropometric characters between studied groups were shown in table 1.

characteristics	SCH N=80 Mean ±SE, N(%)	Controls N=80 Mean ±SE, N(%)	*p* value
Gender Male Female	 20(25%) 60(75%)	 20(25%) 60(75%)	 1.000*
Age(years) 20-29	36.63±1.01 22(27.5%)	36.54±1.01	0.951**

30-39 40-49 ≥50	26(32.5%) 24(30%) 8(10%)	23(28.7%) 25(31.3%) 24(20%) 8(10%)	0.998**
WC (cm)	95.24±1.51	90.81±1.57	**0.043***
BMI(kg / m²)	29.83±0.70	27.44±0.57	**0.009***
FH of thyroid disorders Positive Negative	 33(41.3%) 47(58.8%)	 17(21.3%) 63(78.8%)	**0.010***

The results in table 1 show that the females covered more prevalence of the SCH than males (75%; 25%) respectively. Patient with SCH had significantly higher WC and BMI than control group (95.24±1.51), (29.83±0.70) respectively. Furthermore patients with SCH had more prevalence of family history of thyroid disorders than control group (41%; 21.3%), (p = 0.01) respectively.

Table 2. Descriptive Statistics of Biochemical parameters between studied group.

Parameters	**SCH N=80**	**Controls N=80**	***p* value**
	Mean ±SE N (%)	**Mean ±SE N (%)**	
TSH (mIU/L)	6.46±0.21	2.03±0.95	**<0.001**
FT3 (pmol/L)	4.63±0.09	4.87±0.13	0.130
FT4 (pmol/L)	14.74±0.22	14.95±0.29	0.560
Anti-TPO (IU/mL)	107.11±12.02	22.31±5.76	**<0.001**
Ferritin (ng/mL)	88.98±9.42	90.46±10.30	0.916
Iron (µg/dL)	66.57±2.54	79.13±3.67	**0.006**
TIBC (µg/dL)	356.46±4.03	331.19±4.36	**<0.001**
TF%	22.44±0.75	25.56±1.12	**0.021**
UIBC (µg/dL)	286.97±5.11	253.90±5.67	**<0.001**
Iron deficiency	26 (32.5%)	7 (8.7%)	<0.001
Hepcidin (ng/mL)	639.43±54.46	473.17±39.57	**0.015**
Hephaestin(ng/mL)	7.65±0.49	8.93±1.05	0.272

The biochemical parameters of the two groups are presented in Table 2. Serum iron levels and transferrin saturation (TS%) were significantly lower in subclinical hypothyroid patients (66.57±2.54), (22.44±0.75) respectively. Whereas TIBC, UIBC and hepcidin levels were significantly higher in patients with SCH compared to subjects of control group (356.46±4.03), (286.97±5.11), (639.43±54.46) respectively, (all $p < 0.05$), Furthermore hephaestin levels were insignificantly lower in subclinical hypothyroid patients than control group (7.65±0.49), ($p < 0.272$). The prevalence of iron deficiency significantly higher in SCH group (32.5%) than in control group (8.7%) ($p < 0.001$).

Table 3. Biochemical parameters in subclinical hypothyroid patients with Anti-TPO levels.

Serum Anti-TPO in SCH Patients			
Parameters	**<34 N=36**	**≥34 N=44**	**p value**
	Mean ±SE	**Mean ±SE**	
Ferritin (ng/mL)	91.08±17.13	87.26±10.06	0.814
Iron (µg/dL)	68.72±3.39	64.53±3.85	0.466
TIBC (µg/dL)	355.73±5.59	357.36±5.86	0.842
TF%	22.91±1.05	21.85±22.92	0.487
UIBC (µg/dL)	284.68±7.39	289.78±6.97	0.623
Hepcidin (ng/mL)	635.71±96.46	672.4±78.72	0.766
Hephaestin (ng/mL)	8.09±0.74	7.11±0.60	0.318

Table compares the biochemical parameters between SCH patients with negative anti-TPO (<34 IU/mL, n = 36) and positive anti-TPO (≥34 IU/mL, n = 44). Patients with positive anti-TPO had lower mean serum ferritin (87.26 ± 10.06 vs. 91.08 ± 17.13 ng/mL; p = 0.814), iron (64.53 ± 3.85 vs. 68.72 ± 3.39 µg/dL; p = 0.466), transferrin saturation (TS%) (21.85 ± 22.92 vs. 22.91 ± 1.05%); p = 0.623), and hephaestin (7.11 ± 0.60 vs. 8.09 ± 0.74 ng/mL; p = 0.318) compared with patients with negative anti-TPO. However, the positive anti-TPO group had significantly higher mean serum TIBC (357.36 ± 5.86 vs. 355.73 ± 5.59 µg/dL; p = 0.842), UIBC (289.78 ± 6.97 vs. 284.68 ± 7.39 µg/dL; p = 0.766) and hepcidin (672.4 ± 78.72 vs. 635.71 ± 96.46 ng/mL; p = 0.766). However, these differences were not statistically significant (all p > 0.05).

Table 4. Biochemical parameters in subclinical hypothyroid patients with TSH levels.

Serum TSH in SCH patients			
parameters	**4.2 – 9.9 N=57**	**≥10.0 N=23**	**p value**
	Mean ±SE	**Mean ±SE**	
Ferritin (ng/mL)	100.75±20.64	84.23±10.34	0.431
Iron (µg/dL)	68.57±4.96	65.79±2.96	0.623
TIBC (µg/dL)	352.23±4.12	366.96±9.42	0.098
TF%	24.01±1.66	21.80±0.81	0.186
UIBC (µg/dL)	283.95±5.04	294.48±12.75	0.355
Hepcidin (ng/mL)	577.36±78.54	687.59±79.46	0.416
Hephaestin (ng/mL)	9.21±0.77	7.02±0.59	**0.042**

The results of table 4 demonstrate that the difference in the biochemical markers between different grade of SCH (mild and sever). No statistically significant differences were found in ferritin, iron, TIBC, transferrin saturation, UIBC, and hepcidin levels between the two groups of TSH (all $p > 0.05$). However, serum hephaestin level was significantly lower in the sever SCH compared to the mild SCH (7.02 ± 0.59 vs. 9.21 ± 0.77, $p = 0.042$).

4. DISCUSSION

In the current study, females were the majority of patients with newly diagnosed SCH, indicating the increased sensitivity of them to thyroid gland dysfunction, principally due to the larger frequency of autoimmune thyroid disorders among female (Ferrara, M.*et al,* 2024.) . Patients with SCH were substantially higher in BMI, WC, positive family history of thyroid gland disorders, and anti-thyroid peroxidase antibody (anti-TPO) level than healthy controls. These results are in agreement with earlier data demonstrating that obesity, genetic susceptibility, and thyroid autoimmunity are key factors to SCH(Sulaiman, E.A.*et al*, 2025). Decreased thyroid gland activity may contribute to weight gain through decreased basal metabolic rate, thermogenesis, and lipid metabolism, whereas increased anti-TPO levels suggest autoimmune thyroiditis as the main underlying etiology of SCH and indicate a higher risk of progression to overt hypothyroidism(Sulaiman, E.A.*et al*, 2025).

The present study found substantial abnormalities in iron homeostasis in patients with SCH. Compared with healthy controls, the subclinical hypothyroid patients showed a substantial reduction in serum iron and TS% and a significant increase in TIBC and UIBC with unaffected serum ferritin concentrations. These data suggest that changes in iron metabolism emerge early in thyroid gland dysfunction, and they indicate that early changes in thyroid dysfunction lead to decreased circulatory iron availability, even with normal iron stores. Subsequent research has reported that functional iron insufficiency occurs before the depletion of stored iron (Iolascon, A.*et al,* 2024.). Thyroid gland has a crucial role in several physiological functions related to iron metabolism, such as stomach acid production, intestinal iron absorption, enterocyte maturation, erythropoiesis, and cellular metabolism(Pasricha, S.R.*et al,* 2021.). Thus, even modest thyroid gland dysfunction can impair iron intake and utilization and lead to low circulating iron concentrations with normal ferritin levels. The increased TIBC and UIBC in the present study further suggest decreased iron availability, as hepatic transferrin production rises as serum iron decreases (Yang, J.*et al,* 2023.). In agreement with these biochemical findings, iron shortage was far more prevalent in patients with SCH than in healthy controls, highlighting the intimate physiological connection between thyroid gland function and iron metabolism (Auerbach, M.*et al*, 2025.).

One of the main findings of this study was a significant rise in serum hepcidin and a significant drop in serum hephaestin levels in patients with SCH. Increased hepcidin and decreased hephaestin levels may lead to poorer intestinal iron transport and lower circulating iron despite intact iron reserves. This can be explained by the effect of TSH and thyroid hormone deficiency on the pathways regulating iron metabolism. The increase in hepcidin action will result in an increase in ferroportin degradation and iron release from enterocytes and macrophages into the bloodstream. Hephaestin will have reduced action, which will inhibit the oxidation of Fe^{2+} to Fe^{3+}, thus inhibiting iron release via the ferroportin pathway. The present findings suggest that impaired intestinal iron transport is a significant mechanism contributing to early alterations in iron homeostasis in patients with SCH and that combining the assessment of hepcidin, hephaestin, and conventional iron parameters may enhance the evaluation of iron metabolism in these individuals.

The effect of thyroid autoimmunity on iron homeostasis was evaluated by comparing anti-TPO-positive and anti-TPO-negative subclinical hypothyroid patients. Anti-TPO-positive patients had insignificantly lower serum iron, ferritin levels, TS% and insignificantly higher TIBC, UIBC levels. These findings suggest that the changes in the iron metabolism are related to the thyroid dysfunction as well as the thyroid autoantibody positivity. Anti-TPO antibodies are accurate markers of autoimmune thyroid disease and predictors of progression to overt hypothyroidism, but they are not independent markers of impaired iron homeostasis; similar results have been published in subsequent research (Garofalo, V.*et al,* 2023). Furthermore, there were no significant differences in hepcidin and hephaestin levels between anti-TPO-positive and anti-TPO-negative patients , suggesting that intestinal iron transport is similarly affected in the early stage of

subclinical hypothyroidism, irrespective of autoimmune status. The present study investigated the effect of raising serum TSH levels (severity of SCH) on iron metabolism in SCH. No significant difference was seen in ferritin, serum iron, TS%, TIBC, UIBC, or hepcidin concentrations between mild SCH patients and sever SCH. However, The present study observed significantly lower serum hephaestin concentrations in patients with sever SCH, indicating that molecular alterations in intestinal iron transport might precede changes in conventional biochemical markers. These data are in agreement with previous evidence indicating that metabolic abnormalities in SCH are not only dependent on the TSH concentration but also on the duration of disease, nutritional status, tissue sensitivity to thyroid hormones, and individual physiological variability (Garofalo, V.*et al,* 2023). The lack of changes in hepcidin further suggests that minor elevations in TSH alone may not have a major effect on systemic iron control.

Conclusion: In this study, newly diagnosed subclinical hypothyroid patients have shown significant disturbance in iron homeostasis in terms of low serum iron and transferrin saturation (TS%) along with high TIBC and UIBC while maintaining their ferritin concentration levels, indicating the existence of functional iron deficiency in early stages without any depletion of iron storage. Higher serum hepcidin and lower hephaestin levels signify the disturbance in the process of absorption of iron in the intestine as a possible cause of such disturbances in iron homeostasis. Furthermore, significant reduction in hephaestin levels in the case of severe subclinical hypothyroidism can be considered as an early biomarker of changes in the metabolism of iron in the intestine. Since there were no significant changes in the parameters of iron metabolism, hepcidin, and hephaestin on anti-TPO , it can be that it is dysfunction, but not the autoimmunity of the thyroid gland, that is responsible for the impairment of iron metabolism in early subclinical hypothyroid patients.

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AUTHOR CONTRIBUTIONS

The authors collectively participated in the study design and data interpretation. All authors have examined and approved the final manuscript. [Sherwan F. Salih] conceptualized the study and designed the methodology. [Dlveen M Ali] collected and analyzed the data.

DECLARATION

The authors declare no conflicts of interest related to this study.

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