Rzeszów University Press 2025
ISSN 2544-1361 (online)
doi: 10.15584/ejcem.2025.3.12

*http://www.ejcem.ur.edu.pl*
*European Journal of Clinical and Experimental Medicine*
*Eur J Clin Exp Med 2025; 23 (3): 596–604*

**ORIGINAL PAPER**

# Hormonal profiles and metabolic changes in women diagnosed with concomitant Hashimoto's thyroiditis and polycystic ovary syndrome via sonography

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**ABSTRACT**

**Introduction and aim.** Women with both Hashimoto's thyroiditis (HT) and polycystic ovary syndrome (PCOS) often experience hormonal imbalances and metabolic changes. We investigated the correlation between sonographic changes and hormonal abnormalities in women with Hashimoto's thyroiditis and PCOS.

**Material and methods.** A case-control study including 150 women with PCOS and Hashimoto's thyroiditis, and 50 healthy women as a control group, was conducted at Al-Habobbi Teaching Hospital from 7/1/2023 to 7/10/2024. Lipid, blood sugar, luteinizing hormone (LH), follicle-stimulating hormone (FSH), prolactin, testosterone, and thyroid hormones were assessed, the groups had similar mean ages and smoking rates.

**Results.** When the case group was compared with the control group, significant hormonal and metabolic differences were observed. Specifically, levels of LH were significantly higher in the case group (14.68±1.21 vs. 3.31±1.03 mIU/mL, p=0.001), as were levels of FSH (14.85±1.07 vs. 5.26±0.51 mIU/mL, p<0.001), prolactin (28.90±1.34 vs. 7.02±1.16 ng/dL, p<0.001), and testosterone (57.71±2.61 vs. 12.41±2.27 ng/dL, p<0.001). In terms of lipid profile, the case group showed elevated total cholesterol (229.93±14.61 vs. 134.51±9.38 mg/dL, p<0.001), triglycerides (287.78±41.43 vs. 128.04±10.20 mg/dL, p<0.001), low-density lipoprotein (LDL) (136.98±20.02 vs. 58.67±11.45 mg/dL, p<0.001), and very low-density lipoprotein (VLDL) (57.55±8.28 vs. 25.60±2.04 mg/dL, p<0.001), while levels of high-density lipoprotein (HDL) were significantly lower (35.39±3.54 vs. 50.23±4.55 mg/dL, p<0.001). Regarding thyroid function, thyroxine (T4) levels were significantly reduced in the case group (9.80±0.77 vs. 15.02±1.25, p<0.001), while thyroid-stimulating hormone levels were elevated (6.25±1.10 vs. 2.17±0.74 µIU/mL, p<0.001).

**Conclusion.** These findings suggest a potential complex interaction between the thyroid and reproductive glands, which may influence the pathogenesis and metabolic effects of these endocrine disorders. However, the individual and combined effects require further detailed investigation.

**Keyword.** Hashimoto's thyroiditis, polycystic ovarian syndrome, sonographic changes

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Received: 25.11.2024 / Revised: 24.03.2025 / Accepted: 6.04.2025 / Published: 30.09.2025

Ahmed AW, Abdul-QaderKhuder H, Jasim SAH., Mohsein OA. Hormonal profiles and metabolic changes in women diagnosed with concomitant Hashimoto's thyroiditis and polycystic ovary syndrome via sonography. *Eur J Clin Exp Med.* 2025;23(3):596–604. doi: 10.15584/ejcem.2025.3.12.

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## Introduction

Polycystic ovary syndrome (PCOS) is one of the most prevalent endocrine and metabolic disorders among women of reproductive age, with a global prevalence ranging between 3% and 15%. This complex disorder affects not only reproductive function but is also strongly associated with metabolic and mental health issues, which may collectively compromise overall health and well-being.[1,2] PCOS is a heterogeneous disorder that varies based on factors such as age, genetics, race, and environmental influences. Despite this variability, PCOS is classified into four phenotypes, which are distinguished by three key factors such as polycystic ovary morphology, ovulatory dysfunction, and elevated androgen levels.[3] Thyroid disorders are also common and often overlapped with PCOS, aside from reproductive dysfunction. Women with PCOS are at an increased risk of thyroid disease such as sub-clinical hypothyroidism and autoimmune thyroiditis (AIT). Researchers have found that women with PCOS are significantly more likely to have hypothyroidism, with a prevalence of 11% to 14%, compared to only 1% to 2% among women without the disorder. Both conditions commonly share metabolic abnormalities such as insulin resistance, dyslipidemia, and obesity.[4,5] The most common autoimmune thyroid disorder is HT, first reported by Hakaru Hashimoto in 1912. However its ability to cause inflammation was not fully realized until the 1950s. Epidemiologic data suggests that the most common cause of hypothyroidism is HT.[6] The disease is characterized by lymphocytic infiltration of the thyroid gland and may present in various forms, including painless (silent) thyroiditis and subclinical or overt hypothyroidism. HT could be secondary to immune-modulating medical therapies like interferon-alpha or monoclonal antibodies against Cytotoxic T-lymphocyte-associated protein 4 (CTLA-4).[7,8] Abnormal thyroid function is a common hormonal imbalance in both PCOS and HT. Hypothyroidism is often caused by an autoimmune response in which the immune system produces antibodies against thyroid proteins such as thyroid peroxidase (TPO) and thyroglobulin (TG). This autoimmune activity may interfere with estrogen metabolism. Elevated estrogen levels are associated with enhanced humoral immunity, while androgen and progesterone levels are generally reduced in women with PCOS.[9,10] The metabolic disturbances observed in both conditions such as insulin resistance, dyslipidemia, and weight gain are significantly more pronounced in women with concurrent PCOS and hypothyroidism.[11] Dysfunction of thyroid has been linked with a more severe metabolic profile in the population of PCOS. Patients with either condition have higher triglycerides or fasting insulin or homeostasis model assessment of insulin resistance (HOMA-IR) than subjects with PCOS alone. Obese women with HT and longstanding PCOS show significantly greater body mass index (BMI), fasting blood glucose and cholesterol levels than controls or those with HT alone.[12,13] Moreover, the coexistence of PCOS and hypothyroidism has been associated with more severe metabolic and hormonal disturbances. Studies have shown that correcting thyroid hormone levels in hypothyroid patients leads to significant improvements in metabolic health. These findings support the growing body of evidence linking thyroid dysfunction with the metabolic abnormalities commonly observed in patients with PCOS.[14,15]

## Aim

The aim of this study is to investigate the correlation between sonographic changes and hormonal abnormalities in women with both HT and PCOS. The study will analyze sonographic changes in the ovaries and thyroid, correlating these with hormone levels such as thyroid hormones and androgens.

## Material and methods

This case-control study included 150 women diagnosed with both HT and PCOS. Diagnosis of PCOS was confirmed by physicians using the Rotterdam criteria, which require at least two of the following: irregular ovulation, clinical or biochemical signs of hyperandrogenism, and polycystic ovarian morphology on ultrasound. Participants in the case group were consecutively recruited as they presented and met the inclusion criteria. The control group consisted of 50 age-matched healthy women who were randomly selected from individuals attending routine check-ups. The health status of the control participants was verified through clinical evaluation, detailed medical history, and laboratory screening to exclude thyroid or reproductive disorders. These sample sizes of cases and controls were selected to achieve adequate statistical power and robust comparisons, given the estimated prevalence of PCOS and HT. This 3:1 ratio optimized data collection from cases while preserving sufficient controls. The age range (18–45 years) was chosen to encompass the reproductive years, guiding enrollment based on study endpoints and supported the feasibility and objectives of the study. All information was recorded by participants and transcribed. The study was conducted at Al-Haboubi Teaching Hospital for the period between 1/7/2023 to 10/7/2024. Women with hyperthyroidism, those on treatment, and patients who had had their thyroid removed were excluded. 5 mL was collected from each participant and placed in a gel tube and left for 15 minutes at room temperature until clotting. Serum was separated using a centrifuge at 3500 rpm for 15 minutes. The blood serum was isolated and kept at a temperature of minus 20°C until use. Lipid profile levels (total cholesterol, low-density lipoprotein (LDL), very low-density lipoprotein (VLDL) and

high-density lipoprotein (HDL)) and fasting blood sugar (FBS) were estimated using colorimetric methods using a spectrophotometer (Biolabo, Firance). The levels of triiodothyronine (T3), thyroxine (T4), thyroid-stimulating hormone (TSH), thyroid peroxidase antibodies (TPOAb), luteinizing hormone (LH), follicle-stimulating hormone (FSH), prolactin, and testosterone were estimated using the Cobas E411 device (Roche, German). Thyroid hormone levels were measured in enzyme-linked immunosorbent assay (Biotechne, USA). Additionally, all assays were validated in terms of their sensitivity (limit of detection), specificity, and variability (coefficient of variation, CV%), which were closely monitored to ensure the reliability of the results. These measures guarantee the robustness of the findings and enhance the overall credibility of the study.

### *Statistical analysis*

Statistical analysis was conducted using SPSS version 26 (IBM, Armonk, NY, USA). Data were expressed as frequencies, percentages, and means±SD. Independent and dependent t-tests were applied for normally distributed variables, while Mann-Whitney U, Wilcoxon, and Chi-square tests were used for non-normal data. A p-value <0.05 was considered statistically significant.

### *Ethical approval*

Prior to sample collection, all participants were thoroughly informed about the study's objectives and procedures. Written informed consent was obtained from each participant, confirming their voluntary participation. The study was approved by the Committee on Publication Ethics at the Thi-Qar Health Directorate (Al Habbobi Teaching Hospital) and Ninevah Health Directorate (Al Batool Teaching Hospital) under approval form No. 3997, dated January 7, 2023.

## Results

### *Socio-demographic characteristics and clinical profiles of patients with concomitant HT and PCOS*

The study revealed significant demographic and clinical differences between the control and case groups. While there was no significant difference in mean age (years) (p=0.92) or smoking status (p=0.77), BMI was notably higher in the case group (32.72±2.95 vs. 23.63±1.80 kg/m$^2$, p<0.001). Physical activity was lower in the case group (2.1±1.1 vs. 3.5±1.2 hours/week, p=0.02), and a higher proportion of the case group had a family disease history (40% vs. 10%, p<0.001). Differences in education (secondary education: 30% vs. 50%, p=0.05), marital status (unmarried: 60% vs. 40%, p=0.03), and socioeconomic status (low class: 50% vs. 20%, p<0.001) were also significant, as detailed in Table 1.

**Table 1.** A comparative analysis of demographic, lifestyle, and clinical parameters

| Parameters | Control group (n=50) Mean±SD | Case group (n=150) Mean±SD | p |
|---|---|---|---|
| Age (years) | 28.98±7.10 | 29.09±7.35 | 0.92 |
| BMI (kg/m$^2$) | 23.63±1.80 | 32.72±2.95 | <0.001 |
| Smoking status (%) | 15% (7) | 18% (27) | 0.77 |
| Physical activity (hours/week) | 3.5±1.2 | 2.1±1.1 | 0.02 |
| Family history (%) | 10% (5) | 40% (60) | <0.001 |
| Education level | High school: 50% (25) | High school: 30% (45) | 0.05 |
| | College: 30% (15) | College: 40% (60) | |
| | University: 20% (10) | University: 30% (45) | |
| Marital status (%) | Single: 40% (20) | Single: 60% (90) | 0.03 |
| | Married: 60% (30) | Married: 40% (60) | |
| Socioeconomic status | Low: 50% (25) | Low: 20% (30) | <0.001 |
| | Medium: 40% (20) | Medium: 50% (75) | |
| | High: 10% (5) | High: 30% (45) | |

### *Ultrasound findings*

Transvaginal ultrasound imaging revealed characteristic features in patients presenting with ovarian and thyroid abnormalities. As shown in Figure 1, both ovaries appeared bilaterally enlarged with borderline dimensions. Multiple small follicles, each numbering more than ten, were observed to be arranged peripherally a typical finding consistent with polycystic ovary morphology. In Figure 2, a significantly enlarged right ovary was noted, exhibiting a peripheral distribution of follicles and a centrally located echogenic stroma, further supporting the diagnosis of ovarian dysfunction. Regarding thyroid abnormalities, Figure 3 demonstrates a longitudinal scan of a female patient diagnosed with nodular Hashimoto's thyroiditis. The image reveals a solid, hypoechoic, homogeneous nodule with poorly defined margins, while the background thyroid parenchyma appears normal. In contrast, Figure 4 displays a transverse scan from another patient with the same diagnosis, showing a solid, hyperechoic, homogeneous nodule with sharply defined margins and a thin hypoechoic halo. The background parenchyma in this case exhibits a micronodular pattern, which is commonly associated with chronic autoimmune thyroiditis.

### *Lipid profile alterations in patients with concomitant HT and PCOS*

The study demonstrated significant differences in lipid profiles between the case and control groups. The case group had higher mean total cholesterol (229.93±14.61 vs. 134.51±9.38 mg/dL, p<0.001), triglycerides

(287.78±41.43 vs. 128.04±10.20 mg/dL, p<0.001), LDL (136.98±20.02 vs. 58.67±11.45 mg/dL, p<0.001), and VLDL (57.55±8.28 vs. 25.60±2.04 mg/dL, p<0.001). In contrast, HDL levels were lower in the case group (35.39±3.54 vs. 50.23±4.55 mg/dL, p<0.001). These findings highlight a significant impact of disease state on lipid profiles, as shown in Table 2.

**Table 2.** A comparative study of serum lipid levels between control and case groups

| Parameters | Control group (n=50) Mean±SD | Case group (n=150) Mean±SD | p |
|---|---|---|---|
| TC (mg/dL) | 134.51±9.38 | 229.93±14.61 | <0.001 |
| TG (mg/dL) | 128.04±10.20 | 287.78±41.43 | <0.001 |
| HDL (mg/dL) | 50.23±4.55 | 35.39±3.54 | <0.001 |
| LDL (mg/dL) | 58.67±11.45 | 136.98±20.02 | <0.001 |
| VLDL (mg/dL) | 25.60±2.04 | 57.55±8.28 | <0.001 |

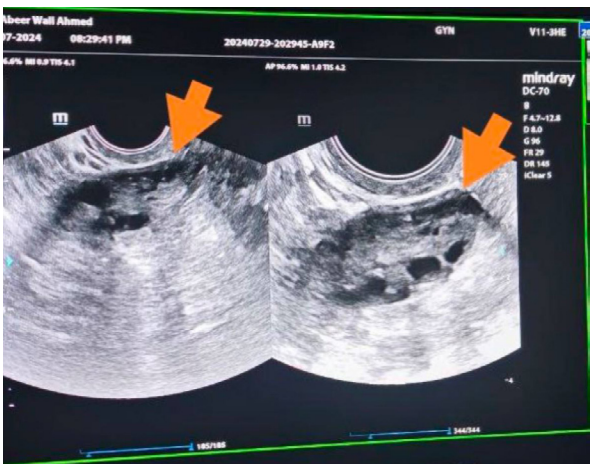


**Fig. 1.** Transvaginal ultrasound image shows bilaterally enlarged ovaries with a borderline size, multiple small follicles, more than 10 in number, are located peripherally

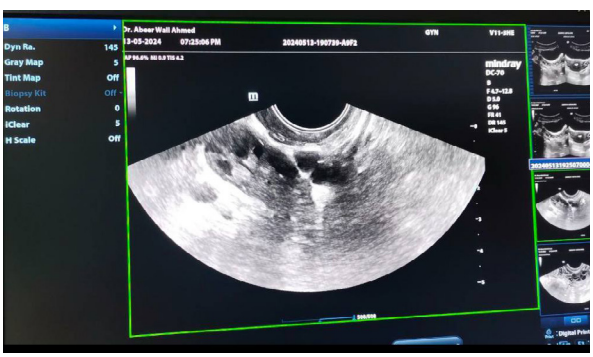


**Fig. 2.** Transvaginal ultrasound reveals a significantly enlarged right ovary, characterized by peripherally distributed follicles and a centrally located echogenic stroma

### *Hormonal profile differences in patients with HT and PCOS*

The study revealed significant hormonal differences between the case and control groups. The case group had a significantly higher mean level of LH (14.68±1.21 vs. 3.31±1.03 mIU/mL, p=0.001), FSH (14.85±1.07 vs. 5.26±0.51 mIU/mL), prolactin (28.90±1.34 vs. 7.02±1.16 ng/dL, p<0.001), and testosterone (57.71±2.61 vs. 12.41±2.27 ng/dL, p<0.001). These findings indicate notable changes in circulating hormones, demonstrating the differential effects of disease on hormone levels, as shown in Table 3.

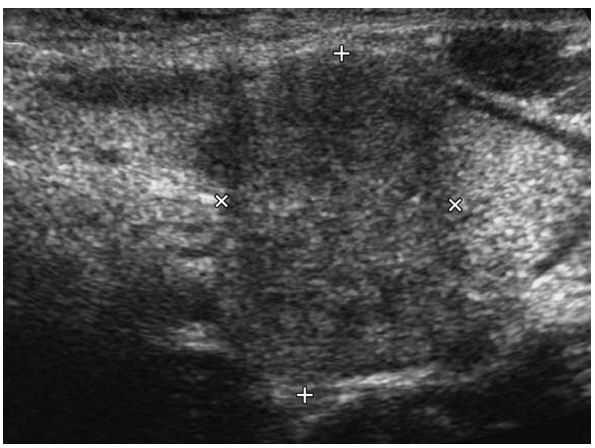

**Fig. 3.** Woman with nodular HT, longitudinal scan shows solid hypoechoic homogeneous poorly marginated nodule (cursors), background parenchyma is normal

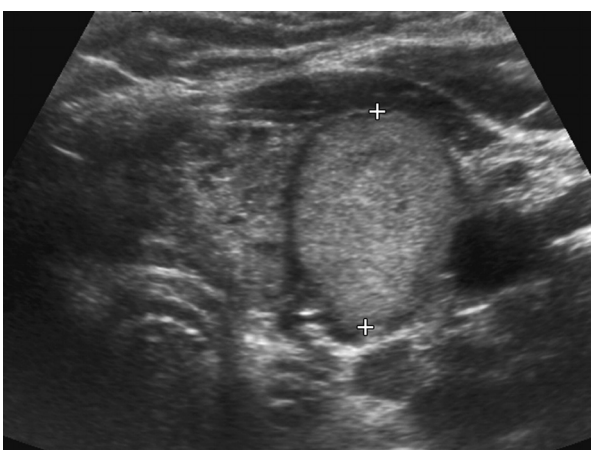

**Fig. 4.** Woman with nodular HT, transverse scan shows solid hyperechoic homogeneous sharply marginated nodule (cursors) with thin hypoechoic halo, background parenchyma is micronodular

**Table 3.** A comparative analysis of LH, FSH, prolactin, and testosterone levels

| Parameters | Control group (n=50) Mean±SD | Case group (n=150) Mean±SD | p |
|---|---|---|---|
| LH (mIU/mL) | 3.31±1.03 | 14.68±1.21 | <0.001 |
| FSH (mIU/mL) | 5.26±0.51 | 14.85±1.07 | <0.001 |
| Prolactin (ng/mL) | 7.02±1.16 | 28.90±1.34 | <0.001 |
| Testosterone (ng/dL) | 12.41±2.27 | 57.71±2.61 | <0.001 |

***Comparative study of glycemic and thyroid function parameters in patients with HT and PCOS***

The study showed significant differences in thyroid function between the case and control groups. While FBS and T3 levels were similar between the groups (p=0.53 and p=0.52, respectively), T4 levels were significantly lower in the case group (9.80±0.77 vs. 15.02±1.25 μg/dL, p<0.001) and TSH levels were significantly higher (6.25±1.10 vs. 2.17±0.74 μIU/mL, p<0.001). These findings highlight the significant impact of disease status on thyroid function, as shown in Table 4.

**Table 4.** Analysis of fasting blood sugar and thyroid hormone levels between control and case groups

| Parameters | Control group (n=50) Mean±SD | Case group (n=150) Mean±SD | p |
|---|---|---|---|
| FBS (mg/dL) | 95.54±8.81 | 96.46±9.05 | 0.53 |
| T3 (ng/dL) | 4.46±0.54 | 4.52±0.55 | 0.52 |
| T4 (μg/dL) | 15.02±1.25 | 9.80±0.77 | <0.001 |
| TSH (μIU/mL) | 2.17±0.74 | 6.25±1.10 | <0.001 |

***Correlation analysis of age, BMI, lipid profile, hormonal levels, and glycemic parameters in patients with HT and PCOS***

The Table 5 shows correlations between key clinical parameters using Spearman's test. Significant relationships were found between some key variables. For example, LDL, VLDL, and TG levels showed strong negative correlations with each other (p<0.01), indicating an interdependence between these lipids. A significant positive correlation was also observed between TSH, T4, and T3 (p<0.01), indicating a hormonal interaction between these substances that play a role in thyroid regulation. On the other hand, testosterone showed a moderate correlation with BMI (p<0.05), reflecting its potential effect on lipids or body weight. FBS levels also showed relatively weak correlations with most other parameters. An asterisk (*) indicates statistical significance, with one asterisk meaning p<0.05 and two asterisks meaning p<0.01, reflecting different levels of statistical significance of these relationships.

## Discussion

Thyroiditis is a diverse disorder that impacts the thyroid gland and has multiple underlying causes. HT, or chronic lymphocytic thyroiditis, is an AIT that frequently occurs in young women and is a prevalent form of thyroid inflammation. HT can occur simultaneously with clinical hypothyroidism, normal thyroid function, or hyperthyroidism.[16] Significant differences between the control and case groups have been observed at the level of various patient demographic, lifestyle, and clinical parameters, as described below. Age (p=0.92) is matched between groups, removing a confounder of age found in studies including, which used age-matched cohorts to minimize bias.[17] The much higher BMI in the case group (32.72±2.95 vs. 23.63±1.80 kg/m$^2$, p<0.001) emphasizes the contribution of obesity in making people more susceptible to the disease, like, which elevated BMI as a predictor of metabolic disorders; however, Serin et al. pointed out that only calculating fat mass or BMI is not enough and should be placed in the context of body composition and factors reflecting metabolic activity with the call for a better metabolic fingerprint.[18,19] Given that there was no substantial difference in smoking status (p=0.77), we agree with Arduc et al., who described the variability in smoking exposure impact across cultural contexts in contrast to the findings of Ho et al., who highlighted smoking as a major risk factor.[20,21] The reduction in physical activity in the case group (2.1±1.1 hours/week vs. 3.5±1.2 hours/week, p=0.02) is concordant with, making associations between sedentary behavior and chronic disease risk; however, argued that differences in dietary intake may play a more significant role.[22] The crystal-clear family history (40% vs. 10%, p < 0.001) risk positioned the case group up significantly which underpins genetic susceptibility as supported by,

**Table 5.** Evaluating interrelationships among key clinical variables[a]

| Parameters | BMI | TC | TG | HDL | LDL | VLDL | LH | FSH | Pro. | Test. | FBS | T3 | T4 | TSH |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| BMI | 1.000 | -0.070 | 0.018 | 0.065 | -0.070 | 0.018 | -0.147 | 0.015 | 0.002 | 0.190* | 0.000 | 0.005 | -0.013 | 0.053 |
| TC | -0.070 | 1.000 | -0.287** | -0.281** | 0.898** | -0.287** | 0.028 | -0.033 | 0.106 | 0.009 | 0.035 | -0.070 | 0.152 | -0.194* |
| TG | 0.018 | -0.287** | 1.000 | 0.128 | -0.646** | 0.287** | 0.073 | 0.094 | -0.140 | 0.089 | 0.000 | 0.046 | -0.320** | 0.270** |
| HDL | 0.065 | -0.281** | 0.128 | 1.000 | -0.435** | 0.128 | -0.040 | -0.083 | -0.115 | -0.040 | 0.128 | -0.057 | -0.040 | 0.248** |
| LDL | -0.070 | 0.898** | -0.646** | -0.435** | 1.000 | -0.646** | -0.003 | -0.048 | 0.156 | -0.023 | 0.036 | -0.060 | 0.251** | -0.297** |
| VLDL | 0.018 | -0.287** | 0.287** | 0.128 | -0.646** | 1.000 | 0.073 | 0.094 | -0.140 | 0.089 | 0.000 | 0.046 | -0.320** | 0.270** |
| LH | -0.147 | 0.028 | 0.073 | -0.040 | -0.003 | 0.073 | 1.000 | 0.094 | 0.070 | 0.089 | -0.062 | -0.070 | 0.046 | 0.270** |
| FSH | 0.015 | -0.033 | 0.094 | -0.083 | -0.048 | 0.094 | 0.094 | 1.000 | 0.070 | 0.094 | -0.014 | -0.044 | -0.017 | 0.047 |
| Pro. | 0.002 | 0.106 | -0.140 | -0.115 | 0.156 | -0.140 | 0.070 | 0.070 | 1.000 | -0.041 | -0.093 | 0.069 | -0.035 | -0.096 |
| Test. | 0.190* | 0.009 | 0.089 | -0.040 | -0.023 | 0.089 | 0.089 | 0.094 | -0.041 | 1.000 | 0.278 | 0.119 | -0.138 | 0.038 |
| FBS | 0.000 | 0.035 | 0.000 | 0.128 | 0.036 | 0.000 | -0.062 | -0.014 | -0.093 | 0.278 | 1.000 | 0.026 | 0.063 | -0.088 |
| T3 | 0.005 | -0.070 | 0.046 | -0.057 | -0.060 | 0.046 | -0.070 | -0.044 | 0.069 | 0.119 | 0.026 | 1.000 | -0.091 | -0.006 |
| T4 | -0.013 | 0.152 | -0.320** | -0.040 | 0.251** | -0.320** | 0.046 | -0.017 | -0.035 | -0.138 | 0.063 | -0.091 | 1.000 | -0.156 |
| TSH | 0.053 | -0.194* | 0.270** | 0.248** | -0.297** | 0.270** | 0.270** | 0.047 | -0.096 | 0.038 | -0.088 | -0.006 | -0.156 | 1.000 |

[a] * – p<0.05, ** – p<0.01

who highlighted heredity as risk factor, while Mukherjee et al. that environmental changes can reduce genetic susceptibility.[23,24] Differences in education level with a moderate link (p=0.05) suggest that higher education facilitates better health management. The greater proportion of singles within the case group (60% vs. 40%, p=0.03) highlights a possible association with stress and limited social support. Additionally, the elevated socioeconomic status in the case group (p<0.001) challenges traditional assumptions and indicates region-specific factors or sample-related biases. These findings suggest multifactorial relationships genetic, lifestyle, and socioeconomic that require further investigation in regional and cultural contexts, as these parameters collectively influence human health and protection against various pathologies.[25,26] The values in table 2 demonstrate a statistically significant difference in serum lipids observed in the case group versus controls, with total cholesterol, triglycerides, LDL, and VLDL being higher and HDL being lower in the case group, all with p<0.001. These findings strongly point towards dyslipidemia in the case group, an established determinant of cardiovascular and metabolic disorders. In the case group, the total cholesterol and LDL were higher (229.93±14.61 and 136.98±20.02 mg/dL, respectively), a finding in agreement with Cai et al., emphasized the importance of hypercholesterolemia for atherosclerosis risk.[27] Contrarily, Zhao et al., These findings suggest that the majority of lipid dysregulations are a function of abnormal diets, and not due to genetic or pathophysiological predispositions.[28] The high levels of triglycerides (287.78±41.43 mg/dL) and VLDL (57.55±8.28 mg/dL) are similar with.[29] Establishing connections with each of these parameters with insulin resistance and metabolic syndrome. However, Arduc et al. indicated that such elevations could be related to genetic factors, especially in populations with a history of dyslipidemia.[30] Reduced HDL levels (35.39±3.54 in the case group vs. 50.23±4.55 mg/dL in controls) were in accordance with Fan et al. and noted that HDL provided a protective effect and the absence of it also increased cardiovascular risk. Such dietary interventions have been shown to increase HDL levels, and so even though HDL levels are affected by risk factors at their origins as mentioned, this leaves open the possibility for corrective action to be taken to address the imbalance.[31] As proposed by Palomba et al. (2023), the dyslipidemic profile in the case group can be attributed on a scientific basis to chronic inflammation, oxidative stress and changes in lipid metabolism pathway.[32] Studie like Batóg et al., also support this interpretation, which stated that systemic inflammation directly disrupts lipid metabolism by reducing the production of HDL and increasing the production of LDL and VLDL. This highlights the need for timely interventions such as lifestyle changes, medications to manage lipids to reduce the risk of chronic diseases resulting from poor lipid profiles. Future work should investigate the genetic and molecular pathways associated with myogenic lipids in finer detail to better align therapeutic targeting approaches.[33] The case group has higher total cholesterol, triglycerides, LDL, and VLDL levels, and lower HDL levels, which means more significant atherosclerosis risk and cardiovascular diseases are mediated. Management of dyslipidemia involves lifestyle modifications (diet, exercise) and medicinal therapy (statins or fibrates) to correct lipid abnormality to normal. "Indeed, the low HDL levels most specifically demand treatment programs designed to raise HDL, such as weight loss and aerobic exercise. Dysfunction of thyroid hormones aggravates aberrations of lipid metabolism mediated by the way of decreased hepatic lipase activity and disrupted cholesterol transport.[31,32] Levothyroxine therapy for correcting hypothyroidism may also improve the lipid profile. Elevated androgen levels contribute to increased insulin resistance, which exacerbates dyslipidemia. Both androgen excess and lipid disturbances can be managed with insulin-sensitizing agents, such as metformin. Addressing the complex interaction between hormonal and lipid abnormalities requires a multidisciplinary approach involving endocrinologists, gynecologists, and dietitians. Early diagnosis and targeted treatment are essential to prevent long-term complications, such as type 2 diabetes, cardiovascular diseases, and infertility.[32,33] The key is to monitor thyroid function and lipid profiles regularly, which allows for adjustment of therapy to promote optimal patient outcomes. Tailored therapy, according to the severity of hormonal and metabolic derangements, may result in increased effectiveness and less adverse effects. Chronic low-grade inflammation, pathological in PCOS and thyroid disorders for example, may also play a role in modulating metabolic profiles. Anti-inflammatory agents (e.g., omega-3 fatty acids or antioxidants) may supplement standard therapies.[33] Table 3 shows the differences in hormonal levels between control and case groups, noting that levels of LH, FSH, prolactin and testosterone were higher in the case group with p<0.001. Strong evidence suggest disruption of hypothalamic-pituitary-gonadal axis which may be associated with bas pathology or some physiological conditions. The significantly high LH and FSH levels of case group are similar to Lee et al., who noted similar hormonal spikes in diseases such as PCOS or gonadal dysfunction. Heightened gonadotropins might represent compensatory mechanisms to correct disrupted gonadal feedback or increased androgen synthesis.[34] However, Batóg et al., further proposed that such elevations could be attributed to hypothalamic hyperactivity or pituitary adenomas, warranting additional clinical evaluation.[35] Prolactin were significantly higher in the case group in agreement with Davoudi et al., associated hyperprolactinemia with

stress or the pituitary disorders. Elevated prolactin can inhibit gonadotropin secretion leading to a feedback dysregulation worsening hormone dysregulation.[36] Contrastingly, Elnour et al., postulated that transient elevation in prolactin levels could also stem from medication or physiological stress, necessitating distinction from pathological causes.[37] The excessively high testosterone levels are indicative of hyperandrogenism, the classic sign of a disease such PCOS or adrenal hyperplasia. This observation is in agreement with what was observed by Ma et al., who identified androgen excess as a key driver of metabolic and reproductive abnormalities.[38] However, Kim et al. indicated that elevations in testosterone could be derived from exogenous supplementation or tumors, thus requiring more extensive diagnostic workup.[39] These hormonal derangements might be indicative of systemic inflammation, oxidative stress, or genetic mutations that influence endocrine pathways, according to Abdul-Ameer. Finally, lifestyle factors with obesity and insulin resistance, which commonly come along with dysregulated testosterone and prolactin levels.[40] Data in Table 4 demonstrated significant variance in T4 levels of the control and case group however, FBS and T3 levels showed non-significant variance. The similar results were observed for FBS and T3, indicating that these parameters are not significantly affected by the condition of interest, consistent with Elslimani et al., who stated that increased T3 in the presence of normal FBS could reflect early thyroid dysfunction or non-metabolic conditions.[41] However, Novais et al. suggested that glucose metabolism changes may happen in later stages, and longitudinal studies are needed to confirm.[42] On the contrary, T4 levels in the case group were markedly lower ($p<0.001$), while TSH levels were higher ($p<0.001$). This pattern is consistent with primary hypothyroidism, as outlined by Gaberšček et al. further recognized lower T4 and higher TSH as key evidence of dysfunction of thyroid gland.[43] Du et al. also note that elevated TSH reflects compensatory activity in the pituitary gland in response to insufficient thyroid hormone production. Elevated TSH and reduced T4 levels indicate subclinical or overt hypothyroidism, likely linked to Hashimoto's thyroiditis. Thyroid hormone replacement therapy with levothyroxine is essential to restore euthyroid status, improve metabolic functions, and mitigate long-term cardiovascular risks.[44] Extreme dysfunction of the thyroid gland may be due to autoimmune processes, chronic inflammation or iodine deficiency.[45] also found that hypethyroidization of the hypothalamic-pituitary-thyroid axis due to systemic stress might also be involved. In contrast to Shanmugham et al., observing small reductions in T4 without elevated TSH, highlight the heterogeneous nature of thyroid dysfunction, potentially influenced by genetic and environmental factors.[46] The elevation of both LH and testosterone in the case group indicates hyperandrogenism, a hallmark of PCOS, strongly linked with menstrual irregularities, infertility and hirsutism. The treatment performed is going to lower amount of androgen in the body and this consist of combined oral contraceptive or anti-androgens like spironolactone. Elevated FSH levels and alterations in the LH:FSH ratios might decrease ovarian folliculogenesis. Ovulation induction agents such as clomiphene citrate or letrozole may be used to restore ovulatory cycles. Hyperprolactinemia was also noticed in the case group which can be a sign of pituitary invasion or an alteration of the endocrine system was related to stress. In order to normalize the serum prolactin levels and concomitant symptoms such as galactorrhea and infertility, pituitary function tests and dopaminergic medications such as cabergoline should be discussed.[47]

### *Study limitations*

Although a single-center design is superior for research focused on clinical needs, the paradigm could be stretched to multicenter operations to advance generalizability of findings. A lack of demographic characteristics could impact results, and controlling for them would make stronger conclusions, as seen from differences between the case and control groups. Likewise, greater diversity would enhance the applicability of the study to different ethnic backgrounds, allowing for more global conclusions. Implementing a longitudinal design that considers key lifestyle aspects will advance the awareness of the interrelationship of these conditions and the potential implications they will have throughout lives, laying foundations for further deep and actionable research.

## Conclusion

In summary, the results show significant differences regarding the hormonal and lipid profiles between HT and PCOS women and healthy control group. The evidence indicates a potential bidirectional relationship between the thyroid and reproductive gland that may contribute toward the pathogenesis and metabolic consequences of these endocrine disorders. Nonetheless, more extensive exploration of their isolated and collective roles is warranted. The absence of separate subgroup analysis in the present study hampers the possibility of exploring the effects of PCOS or HT in isolation or in combination. Future studies will need to revisit the design to allow subgroup comparisons and potentially to elucidate the relationship between these two endocrine disorders.

## Declarations

### *Funding*

This study received no funding.

*Author contributions*

Conceptualization, O.A. and A.W.; Methodology, O.A.; Software, O.A.; Validation, H.A., S.A. and Z.Z.; Formal Analysis, A.W.; Investigation, O.A.; Resources, H.A.; Data Curation, S.A.; Writing – Original Draft Preparation, O.A.; Writing – Review & Editing, A.W.; Visualization, S.A.; Supervision, H.A.; Project Administration, O.A.; Funding Acquisition, A.W.

*Conflicts of interest*

The authors declare that they have no competing interests.

*Data availability*

The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

*Ethics approval*

Ethical approval was obtained from the Ethics Committee at the Thi-Qar Health Directorate, Al Habbobi Teaching Hospital, and Ninevah Health Directorate, Al Batool Teaching Hospital (Approval No. 3997, dated January 7, 2023).

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