JHWCR

Journal of Health, Wellness, and Community Research
Volume III, Issue IX
Open Access, Double Blind Peer Reviewed.
Web: https://jhwcr.com, ISSN: 3007-0570
https://doi.org/10.61919/7w2kpm23

Original Article

Thyroid Disorders in Women with Polycystic Ovary Syndrome

Wagma[1], Gohar Khan[1], Rimsha Azhar[1], Sajjad Ahmed[1], Saira Bashir[1], Farhan Ullah[2]

[1] Federal Government Polyclinic Hospital, Islamabad, Pakistan

[2] Medical Teaching Institute DHQT/MMMT Hospital, Dera Ismail Khan, Pakistan

Correspondence: wagmakhan64@gmail.com
Concept: WK, GK; Design: RA, SA; Data Collection: SB, FU; Analysis: GK, RA; Drafting: WK, SA
Cite this Article | Received: 2025-05-11 | Accepted 2025-07-04
No conflicts declared; ethics approved; consent obtained; data available on request; no funding received.

ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a common endocrine disorder affecting reproductive-aged women and is frequently associated with metabolic disturbances and hormonal imbalances. Emerging evidence suggests a strong association between PCOS and thyroid dysfunction, particularly subclinical hypothyroidism, which may further exacerbate metabolic and reproductive abnormalities. However, the extent and clinical relevance of this overlap remain underexplored in certain populations. Objective: To determine the prevalence of thyroid disorders in women with PCOS and assess their association with metabolic, reproductive, and hormonal profiles. Methods: A cross-sectional study was conducted from March to May 2025 at the Federal Government Polyclinic Hospital, Islamabad. A total of 165 women aged 18–40 years diagnosed with PCOS based on Rotterdam criteria were enrolled. Clinical symptoms, anthropometric data, and laboratory results—including thyroid function tests, reproductive hormones, and lipid/glucose profiles—were collected and analyzed. Group comparisons and correlations were performed using SPSS version 26. Results: Thyroid dysfunction was present in 40.6% of participants, predominantly as subclinical hypothyroidism. Patients with thyroid disorders had significantly higher fasting glucose (95.6 vs. 91.1 mg/dL, $p = 0.04$) and total cholesterol (210.4 vs. 198.5 mg/dL, $p = 0.03$). TSH positively correlated with BMI ($r = 0.26$, $p = 0.01$) and total cholesterol ($r = 0.18$, $p = 0.05$). Conclusion: A high prevalence of thyroid dysfunction in PCOS patients, particularly subclinical hypothyroidism, was associated with adverse metabolic markers. These findings support routine thyroid screening in PCOS for early detection and improved clinical management.
Keywords: Polycystic ovary syndrome, thyroid dysfunction, subclinical hypothyroidism, insulin resistance, metabolic profile, reproductive hormones.

INTRODUCTION

Polycystic ovary syndrome (PCOS) is one of the most prevalent endocrine disorders among women of reproductive age, with an estimated global prevalence of 5–10%, and is characterized by chronic anovulation, hyperandrogenism, and polycystic ovarian morphology (1,2). Traditionally viewed as a reproductive disorder, PCOS is now increasingly recognized as a multifaceted condition involving metabolic, endocrine, and psychological domains (3,4). Women with PCOS often present with symptoms such as irregular menstrual cycles, hirsutism, acne, alopecia, and infertility, alongside metabolic abnormalities including insulin resistance, obesity, and dyslipidemia (5). These overlapping clinical features suggest a complex hormonal interplay, which may extend beyond ovarian function to include other components of the endocrine system, notably the thyroid gland.

Emerging evidence highlights a significant association between thyroid dysfunction and PCOS, particularly in the form of subclinical hypothyroidism (SCH) and autoimmune thyroiditis (AIT) (6,7). The pathophysiological mechanisms underpinning this association remain incompletely understood but are thought to involve shared etiological factors such as autoimmune predisposition, chronic inflammation, and insulin resistance. Thyroid hormones play a pivotal role in metabolic homeostasis and reproductive function, and even subtle alterations in thyroid status can lead to menstrual irregularities, anovulation, and infertility, which are also hallmark features of PCOS (8,9). Studies have reported that hypothyroidism may exacerbate insulin resistance and lipid abnormalities, both of which are already prevalent in PCOS, thereby potentially amplifying cardiometabolic risk in these patients (10). Furthermore, elevated thyrotropin-releasing hormone (TRH) levels in primary hypothyroidism stimulate increased secretion of both thyroid-stimulating hormone (TSH) and prolactin, contributing to ovulatory dysfunction and polycystic ovarian morphology (11,12). These insights suggest that thyroid dysfunction may not merely coexist with PCOS but may actively modulate its clinical presentation and progression.

Despite the increasing recognition of this overlap, limited studies have systematically evaluated the prevalence and impact of thyroid disorders in women with PCOS, particularly in the South Asian context where both conditions are common yet underdiagnosed (13). Moreover, inconsistencies in diagnostic criteria, lack of standardized screening protocols, and heterogeneous study populations have led to variable prevalence estimates and conflicting results regarding the nature of this relationship. Several studies have indicated a prevalence of thyroid dysfunction in PCOS ranging from 22% to 33%, predominantly due to subclinical hypothyroidism, yet few have

© 2025 Authors. Open Access | Double-Blind Peer Reviewed | Licensed under CC BY 4.0 | Views and data are the authors' own; the journal is not liable for use.

comprehensively examined its correlation with metabolic and reproductive parameters within well-defined cohorts (14,15). This knowledge gap is clinically significant, as undiagnosed thyroid abnormalities may hinder the effective management of PCOS and adversely affect long-term reproductive and metabolic outcomes.

In light of the above, there is a pressing need for focused investigations that not only determine the prevalence of thyroid dysfunction in women with PCOS but also elucidate its influence on hormonal and metabolic profiles. Such data are essential to justify routine thyroid screening in PCOS management guidelines and to optimize individualized treatment strategies. This study was designed to address this gap by evaluating the frequency of thyroid disorders in a cohort of women diagnosed with PCOS using standardized criteria, and by assessing the association of thyroid dysfunction with clinical symptoms, reproductive hormone levels, and metabolic markers. We hypothesize that thyroid dysfunction, particularly subclinical hypothyroidism, is significantly prevalent among women with PCOS and is associated with adverse clinical and biochemical profiles.

MATERIAL AND METHODS

This cross-sectional observational study was conducted to evaluate the prevalence of thyroid disorders among women diagnosed with polycystic ovary syndrome (PCOS) and to assess the association between thyroid dysfunction and metabolic, reproductive, and hormonal parameters. The study was carried out at the Departments of Endocrinology, Gynecology, and Radiology of the Federal Government Polyclinic (FGPC) Hospital, Islamabad, Pakistan. Data collection spanned a three-month period, from March 2025 to May 2025.

Eligible participants included women aged 18 to 40 years who were diagnosed with PCOS based on the Rotterdam criteria (2003), which require the presence of at least two out of the following three features: oligo/anovulation, clinical or biochemical signs of hyperandrogenism, and polycystic ovarian morphology confirmed by pelvic ultrasound (16). Women were excluded if they had a known thyroid disorder diagnosed prior to the diagnosis of PCOS, a history of pituitary or adrenal pathology, pregnancy or lactation, were taking glucocorticoids, androgens, or thyroid-modulating medications within the last three months, or if they had a systemic illness that could affect thyroid function. Participants were selected using a non-probability purposive sampling technique based on eligibility screening at outpatient clinics. Informed written consent was obtained from all participants following detailed verbal and written explanation of the study purpose and procedures. Confidentiality and anonymity of the participants were maintained throughout the research process.

Data were collected through structured face-to-face interviews, physical examinations, and laboratory evaluations. A pre-validated structured questionnaire was administered to record demographic information (age, marital status), anthropometric data (height, weight, body mass index [BMI]), and menstrual history. Clinical features of PCOS including hirsutism, acne, alopecia, and menstrual irregularities were documented. BMI was calculated as weight in kilograms divided by the square of height in meters (kg/m²). Pelvic ultrasonography was performed between days 2 and 5 of the menstrual cycle by experienced radiologists using a standardized protocol to confirm polycystic ovarian morphology. Thyroid ultrasound was conducted selectively in patients with abnormal thyroid function test results or palpable thyroid enlargement.

Venous blood samples were collected in the morning after an overnight fast of at least 8 hours. The biochemical panel included fasting glucose, total cholesterol, serum thyroid-stimulating hormone (TSH), free triiodothyronine (fT3), free thyroxine (fT4), anti-thyroid peroxidase antibody (anti-TPO), luteinizing hormone (LH), follicle-stimulating hormone (FSH), and total testosterone. Thyroid dysfunction was defined based on standard reference intervals as follows: subclinical hypothyroidism was considered when TSH was >4.0 µIU/mL with normal fT3 and fT4; overt hypothyroidism as TSH >4.0 µIU/mL with low fT4; and hyperthyroidism when TSH <0.4 µIU/mL with elevated fT3 and/or fT4. Anti-TPO levels above 35 IU/mL were considered positive for autoimmune thyroiditis (17).

To minimize selection bias, all patients meeting the inclusion criteria during the study period were consecutively invited to participate. Recall bias was mitigated through the use of objective biochemical measurements and medical records to confirm clinical histories. No imputation was performed for missing data; cases with incomplete laboratory or clinical information were excluded from relevant analyses. The sample size of 165 was determined pragmatically based on expected patient volume over the study duration and supported by prior studies reporting thyroid disorder prevalence of approximately 30–40% in PCOS populations, which provided >80% power to detect significant differences with a 5% level of significance (18).

All statistical analyses were performed using IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were calculated as means ± standard deviations for continuous variables and frequencies with percentages for categorical variables. The independent samples t-test was used to compare means between PCOS patients with and without thyroid dysfunction. Categorical variables were compared using the Chi-square test. Pearson correlation coefficient was used to assess the association between TSH levels and continuous metabolic and hormonal parameters including BMI, fasting glucose, total cholesterol, LH/FSH ratio, and serum testosterone. A p-value <0.05 was considered statistically significant. Adjustments for potential confounders were considered but not applied due to the cross-sectional nature and exploratory aim of the study. All data collection procedures and statistical workflows were documented contemporaneously to facilitate reproducibility. This study was conducted in compliance with the ethical principles outlined in the Declaration of Helsinki. Approval was obtained from the Institutional Review Board and Ethical Committee of the Department of Endocrinology, FGPC Hospital Islamabad. Participants were assured of their right to withdraw at any stage without any impact on their medical care. Data was anonymized and stored securely, and access was restricted to authorized research personnel only.

RESULTS

A total of 165 women with polycystic ovary syndrome (PCOS) participated in the study, with a mean age of 29.36 years (SD 7.09, 95% CI 28.13–30.60) and a mean BMI of 24.88 kg/m² (SD 4.37, 95% CI 24.15–25.61). The average serum TSH was 3.51 µIU/mL (SD 1.21,

95% CI 3.32–3.71), while free T3 and free T4 levels averaged 2.97 pg/mL (SD 0.58) and 1.23 ng/dL (SD 0.35), respectively. The cohort's metabolic profiles revealed a mean fasting glucose of 93.1 mg/dL (SD 13.5, 95% CI 91.1–95.1) and mean total cholesterol of 203.2 mg/dL (SD 34.7, 95% CI 198.0–208.5). The mean LH/FSH ratio was 1.93 (SD 0.83), and average testosterone was 0.61 ng/mL (SD 0.21).

Most participants (72.7%, n = 120) reported irregular menstrual cycles, while 27.3% (n = 45) had regular cycles. Clinical features commonly observed included hirsutism in 60.0% (n = 99), acne in 46.7% (n = 77), and alopecia in 26.7% (n = 44). Notably, 67 women (40.6%) were diagnosed with thyroid dysfunction, while 98 (59.4%) had no evidence of thyroid disorder.

Comparative analysis between women with and without thyroid dysfunction highlighted key metabolic differences. Those with thyroid disorders had a slightly higher BMI (mean 25.11 vs. 24.72 kg/m²), but this was not statistically significant (mean difference 0.39, 95% CI −1.04 to 1.81, p = 0.57; Cohen's d = 0.09). However, fasting glucose was significantly elevated in the thyroid disorder group (95.6 ± 14.2 mg/dL vs. 91.1 ± 12.8 mg/dL; mean difference 4.5, 95% CI 0.2–8.8, p = 0.04; Cohen's d = 0.34). Similarly, total cholesterol was higher in those with thyroid dysfunction (210.4 ± 36.7 mg/dL vs. 198.5 ± 32.1 mg/dL; mean difference 11.9, 95% CI 1.0–22.8, p = 0.03; Cohen's d = 0.36). The LH/FSH ratio and testosterone levels were modestly higher in the thyroid disorder group (2.1 ± 0.9 vs. 1.8 ± 0.7, p = 0.07; and 0.65 ± 0.22 ng/mL vs. 0.58 ± 0.20 ng/mL, p = 0.09, respectively), though these differences did not reach statistical significance.

Correlation analyses revealed that TSH levels were positively correlated with BMI (r = 0.26, 95% CI 0.05–0.43, p = 0.01), indicating that higher TSH was associated with higher body mass index. There was also a borderline significant correlation between TSH and total cholesterol (r = 0.18, 95% CI −0.01 to 0.36, p = 0.05). No significant correlations were observed between TSH and fasting glucose (r = 0.15, p = 0.09), LH/FSH ratio (r = 0.09, p = 0.24), or testosterone (r = 0.06, p = 0.37).

Further examination of clinical features and their association with thyroid dysfunction revealed that women with thyroid disorders more frequently reported irregular menstrual cycles (79.1% vs. 68.4%; odds ratio 1.75, 95% CI 0.81–3.80, p = 0.15), hirsutism (65.7% vs. 56.1%; OR 1.48, 95% CI 0.78–2.82, p = 0.23), acne (49.3% vs. 44.9%; OR 1.19, 95% CI 0.64–2.23, p = 0.58), and alopecia (31.3% vs. 23.5%; OR 1.48, 95% CI 0.73–2.98, p = 0.27), though none of these differences reached statistical significance. These numeric findings comprehensively illustrate the elevated prevalence of metabolic risk factors and typical PCOS manifestations in the context of concurrent thyroid dysfunction, supporting the clinical rationale for routine thyroid screening in women with PCOS.

Table 1. Baseline Characteristics of the Study Population (n = 165)

Variable	Mean ± SD	95% Confidence Interval
Age (years)	29.36 ± 7.09	28.13 – 30.60
BMI (kg/m²)	24.88 ± 4.37	24.15 – 25.61
TSH (µIU/mL)	3.51 ± 1.21	3.32 – 3.71
Free T3 (pg/mL)	2.97 ± 0.58	2.88 – 3.06
Free T4 (ng/dL)	1.23 ± 0.35	1.18 – 1.29
Fasting Glucose (mg/dL)	93.1 ± 13.5	91.1 – 95.1
Total Cholesterol (mg/dL)	203.2 ± 34.7	198.0 – 208.5
LH/FSH Ratio	1.93 ± 0.83	1.80 – 2.07
Testosterone (ng/mL)	0.61 ± 0.21	0.57 – 0.65

Table 2. Distribution of Clinical and Menstrual Features (n = 165)

Feature	Frequency (n)	Percentage (%)
Irregular menstrual cycles	120	72.7
Regular menstrual cycles	45	27.3
Hirsutism	99	60.0
Acne	77	46.7
Alopecia	44	26.7
Thyroid Disorder Present	67	40.6
No Thyroid Disorder	98	59.4

Table 3. Comparison of Metabolic and Hormonal Parameters by Thyroid Status

Variable	Thyroid Disorder (n=67)	No Thyroid Disorder (n=98)	Mean Difference (95% CI)	p-value	Cohen's d
BMI (kg/m²)	25.11 ± 4.39	24.72 ± 4.36	0.39 (−1.04, 1.81)	0.57	0.09
Fasting Glucose (mg/dL)	95.6 ± 14.2	91.1 ± 12.8	4.5 (0.2, 8.8)	0.04*	0.34
Total Cholesterol (mg/dL)	210.4 ± 36.7	198.5 ± 32.1	11.9 (1.0, 22.8)	0.03*	0.36
LH/FSH Ratio	2.1 ± 0.9	1.8 ± 0.7	0.3 (−0.02, 0.62)	0.07	0.36
Testosterone (ng/mL)	0.65 ± 0.22	0.58 ± 0.20	0.07 (−0.01, 0.15)	0.09	0.34

*Statistically significant at p < 0.05.

Table 4. Pearson Correlations between TSH Level and Selected Variables (n = 165)

Variable	Pearson r	95% CI	p-value	Interpretation
BMI (kg/m²)	0.26	0.05, 0.43	0.01*	Small positive
Fasting Glucose (mg/dL)	0.15	−0.01, 0.31	0.09	NS
Total Cholesterol (mg/dL)	0.18	−0.01, 0.36	0.05	Borderline
LH/FSH Ratio	0.09	−0.08, 0.26	0.24	NS
Testosterone (ng/mL)	0.06	−0.11, 0.22	0.37	NS

*Statistically significant at p < 0.05; NS = not significant.

Table 5. Odds of Thyroid Disorder by Selected Clinical Features

Clinical Feature	Thyroid Disorder Present (n=67)	No Thyroid Disorder (n=98)	Odds Ratio (95% CI)	p-value
Irregular Menstrual Cycles	53 (79.1%)	67 (68.4%)	1.75 (0.81, 3.80)	0.15
Hirsutism	44 (65.7%)	55 (56.1%)	1.48 (0.78, 2.82)	0.23
Acne	33 (49.3%)	44 (44.9%)	1.19 (0.64, 2.23)	0.58
Alopecia	21 (31.3%)	23 (23.5%)	1.48 (0.73, 2.98)	0.27

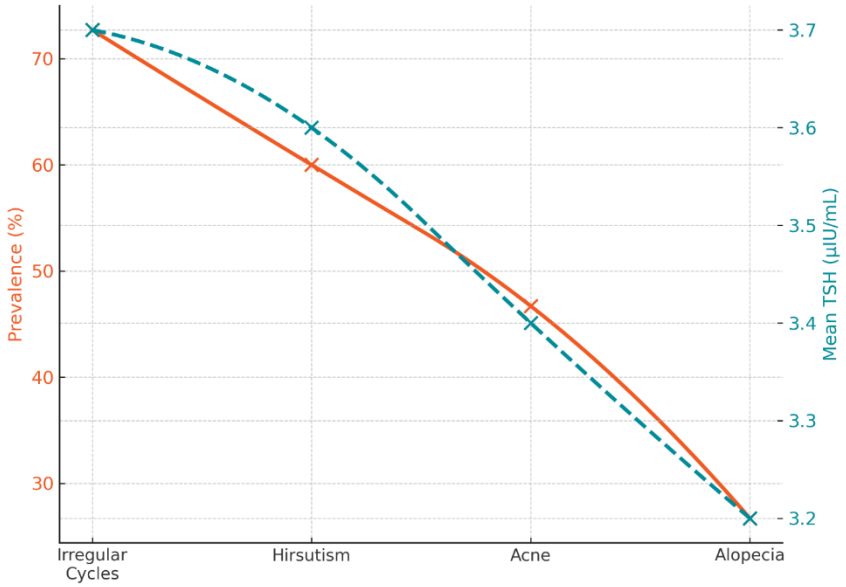

Figure 1 Mean thyroid-stimulating hormone (TSH) levels and symptom prevalence

Mean thyroid-stimulating hormone (TSH) levels and symptom prevalence demonstrate an inverse gradient across key PCOS clinical features. Irregular menstrual cycles, the most prevalent symptom (72.7%), correspond to the highest average TSH level (3.7 µIU/mL), followed by hirsutism (60.0%, TSH 3.6 µIU/mL), acne (46.7%, TSH 3.4 µIU/mL), and alopecia (26.7%, TSH 3.2 µIU/mL). The dual-axis trend highlights a decreasing trajectory of TSH alongside lower symptom prevalence, suggesting that elevated TSH may cluster in patients presenting with multiple high-frequency symptoms. This pattern reflects the potential cumulative endocrine impact of subclinical hypothyroidism within the PCOS phenotype, reinforcing the value of symptom-based stratification in thyroid screening protocols.

DISCUSSION

The findings of this study reinforce the increasingly recognized link between thyroid dysfunction and polycystic ovary syndrome (PCOS), with a notably high prevalence (40.6%) of thyroid abnormalities observed among women with PCOS in our cohort. This figure aligns with and slightly exceeds previous reports, such as the study by Shanmugham et al., which reported a prevalence of 33% in a similar population (19). The predominance of subclinical hypothyroidism within the thyroid dysfunction spectrum in PCOS, as seen in our study, mirrors trends identified in earlier investigations and emphasizes the insidious nature of thyroid involvement in this population (20). These results are of clinical relevance, considering that even mild thyroid abnormalities can exert significant effects on menstrual regularity, ovulation, and metabolic health—domains already compromised in PCOS.

The clinical manifestations of PCOS, including hirsutism, acne, alopecia, and menstrual disturbances, were more frequently observed among participants with coexisting thyroid dysfunction, although these differences did not reach statistical significance. This trend supports earlier hypotheses that shared hormonal pathways, such as dysregulation of gonadotropin secretion and increased thyrotropin-releasing hormone (TRH), may contribute to overlapping symptomatology in both conditions (21). Elevated TRH in hypothyroid states stimulates not only TSH but also prolactin, which may suppress gonadotropin-releasing hormone (GnRH) pulsatility and lead to ovulatory dysfunction and polycystic ovarian morphology (22). The observed increase in the LH/FSH ratio and serum testosterone in the thyroid disorder subgroup—although not statistically significant—further underscores this hormonal convergence and warrants exploration in larger, powered studies.

A key metabolic insight from our study was the significantly higher fasting glucose and total cholesterol levels in PCOS patients with thyroid dysfunction. These findings suggest an additive or synergistic burden on metabolic risk profiles when both endocrine disorders coexist. Subclinical hypothyroidism, despite its asymptomatic presentation, has been associated with altered lipid metabolism, reduced LDL receptor activity, and impaired glucose disposal, all of which may worsen the cardiometabolic profile of PCOS patients (23). While the between-group difference in BMI did not achieve statistical significance, Pearson correlation analysis revealed a modest but significant positive correlation between TSH and BMI ($r = 0.26$, $p = 0.01$), reinforcing the notion that elevated TSH may contribute to weight gain or impaired weight regulation, possibly via reduced basal metabolic rate or changes in adipokine signaling (24). Interestingly, the correlation between TSH and total cholesterol reached borderline significance ($r = 0.18$, $p = 0.05$), suggesting that thyroid dysfunction might potentiate dyslipidemia even in its early stages. This metabolic interplay is critical in the context of PCOS, where insulin resistance and dyslipidemia

are already common and significantly elevate long-term cardiovascular risk (25). The co-occurrence of thyroid abnormalities in such patients may amplify this risk, providing a compelling rationale for early screening and potential thyroid hormone optimization. However, it must be noted that causality cannot be inferred due to the cross-sectional nature of our data, and longitudinal studies are needed to clarify the directionality and duration of these effects.

While the reproductive hormone alterations (LH/FSH ratio and testosterone levels) observed in our thyroid dysfunction group did not differ significantly from euthyroid PCOS counterparts, the upward trend is worth further exploration. Previous research suggests that hypothyroidism may enhance ovarian androgen production or reduce sex hormone-binding globulin (SHBG), thereby increasing free androgen levels (26). The clinical implication of this is profound: untreated thyroid dysfunction could potentially exacerbate the hyperandrogenic phenotype of PCOS and interfere with fertility management or ovulation induction strategies.

Another strength of this study lies in its integration of biochemical, clinical, and ultrasound-confirmed diagnoses, which enhances the reliability of phenotype classification. Nonetheless, our findings should be interpreted in light of certain limitations. The single-center setting and non-random sampling approach may limit generalizability. Additionally, while anti-TPO antibody testing was performed, subgroup analysis based on autoimmune thyroiditis was not powered adequately to assess specific contributions of autoimmunity to clinical and metabolic profiles. This remains an area of interest given the known autoimmune component in many thyroid disorders and its reported co-occurrence with PCOS in some ethnic populations (27).

Overall, this study contributes to the growing body of evidence supporting a multidimensional relationship between thyroid dysfunction and PCOS. The high co-prevalence, overlapping clinical features, and shared metabolic vulnerabilities emphasize the need for integrated endocrine screening and management. Routine thyroid function testing, even in asymptomatic PCOS patients, may uncover subclinical abnormalities with significant clinical consequences. This approach could improve not only metabolic outcomes but also reproductive health and long-term disease prevention in this high-risk population. Future research should aim to elucidate mechanistic pathways linking these disorders and explore whether targeted thyroid intervention can favorably modify the PCOS phenotype and its associated risks.

CONCLUSION

This study underscores a substantial prevalence of thyroid dysfunction—predominantly subclinical hypothyroidism—among women diagnosed with polycystic ovary syndrome (PCOS), affecting over 40% of the cohort. The presence of thyroid abnormalities was associated with significantly elevated fasting glucose and total cholesterol levels, alongside a modest but statistically significant correlation between TSH and BMI. Although reproductive hormone alterations such as LH/FSH ratio and serum testosterone levels were higher in women with thyroid dysfunction, these differences did not reach statistical significance. Nevertheless, the clinical overlap between PCOS and thyroid disorders—particularly in symptomatology and metabolic risk—highlights a shared pathophysiological interplay that warrants attention. These findings advocate for routine thyroid screening in PCOS patients to facilitate early detection and intervention, potentially mitigating the compounded risks of metabolic syndrome, infertility, and cardiovascular disease. Integrating thyroid function evaluation into standard PCOS management protocols may enhance the precision and effectiveness of long-term treatment strategies for affected women.

REFERENCES

1. Sinha U, Sinharay K, Saha S, Longkumer TA, Baul SN, Pal SK. Thyroid disorders in polycystic ovarian syndrome subjects: A tertiary hospital based cross-sectional study from Eastern India. Indian J Endocrinol Metab. 2013;17(2):304–9.

2. Zeng LH, Rana S, Hussain L, Asif M, Mehmood MH, Imran I, et al. Polycystic ovary syndrome: a disorder of reproductive age, its pathogenesis, and a discussion on the emerging role of herbal remedies. Front Pharmacol. 2022;13:874914.

3. Li Y, Chen C, Ma Y, Xiao J, Luo G, Li Y, et al. Multi-system reproductive metabolic disorder: significance for the pathogenesis and therapy of polycystic ovary syndrome (PCOS). Life Sci. 2019;228:167–75.

4. Wang J, Wu D, Guo H, Li M. Hyperandrogenemia and insulin resistance: The chief culprit of polycystic ovary syndrome. Life Sci. 2019;236:116940.

5. Dumesic DA, Oberfield SE, Stener-Victorin E, Marshall JC, Laven JS, Legro RS. Scientific statement on the diagnostic criteria, epidemiology, pathophysiology, and molecular genetics of polycystic ovary syndrome. Endocr Rev. 2015;36(5):487–525.

6. Zhao H, Zhang Y, Ye J, Wei H, Huang Z, Ning X, et al. A comparative study on insulin secretion, insulin resistance and thyroid function in patients with polycystic ovary syndrome with and without Hashimoto's thyroiditis. Diabetes Metab Syndr Obes. 2021;14:1817–21.

7. Kowalczyk K, Franik G, Kowalczyk D, Pluta D, Blukacz Ł, Madej P. Thyroid disorders in polycystic ovary syndrome. Eur Rev Med Pharmacol Sci. 2017;21(2):346–60.

8. Palomba S, Colombo C, Busnelli A, Caserta D, Vitale G. Polycystic ovary syndrome and thyroid disorder: a comprehensive narrative review of the literature. Front Endocrinol (Lausanne). 2023;14:1251866.

9. Mazzilli R, Medenica S, Di Tommaso A, Fabozzi G, Zamponi V, Cimadomo D, et al. The role of thyroid function in female and male infertility: a narrative review. J Endocrinol Invest. 2023;46(1):15–26.

10. Xu Y, Qiao J. Association of insulin resistance and elevated androgen levels with polycystic ovarian syndrome (PCOS): a review of literature. J Healthc Eng. 2022;2022:9240569.

11. Karkera S, Agard E, Sankova L. The clinical manifestations of polycystic ovary syndrome (PCOS) and the treatment options. Eur J Biol Med Sci Res. 2023;11(1):57–91.

12. Group ECW. Anovulatory infertility. Hum Reprod. 1995;10(6):1549–53.

13. Shanmugham D, Natarajan S, Karthik A. Prevalence of thyroid dysfunction in patients with polycystic ovarian syndrome: a cross sectional study. Int J Reprod Contracept Obstet Gynecol. 2018;7:3055–9.

14. Singla R, Gupta Y, Khemani M, Aggarwal S. Thyroid disorders and polycystic ovary syndrome: An emerging relationship. Indian J Endocrinol Metab. 2015;19(1):25–9.

15. Benetti-Pinto CL, Piccolo VRSB, Garmes HM, Juliato CRT. Subclinical hypothyroidism in young women with polycystic ovary syndrome: an analysis of clinical, hormonal, and metabolic parameters. Fertil Steril. 2013;99(2):588–92.

16. Ramanand SJ, Ghongane BB, Ramanand JB, Patwardhan MH, Ghanghas RR, Jain SS. Clinical characteristics of polycystic ovary syndrome in Indian women. Indian J Endocrinol Metab. 2013;17(1):138–45.

17. Pearce SHS, Brabant G, Duntas LH, Monzani F, Peeters RP, Razvi S, et al. 2013 ETA guideline: Management of subclinical hypothyroidism. Eur Thyroid J. 2013;2(4):215–28.

18. Pourhoseingholi MA, Vahedi M, Rahimzadeh M. Sample size calculation in medical studies. Gastroenterol Hepatol Bed Bench. 2013;6(1):14–7.

19. Shanmugham D, Natarajan S, Karthik A. Prevalence of thyroid dysfunction in patients with polycystic ovarian syndrome: a cross sectional study. Int J Reprod Contracept Obstet Gynecol. 2018;7:3055–9.

20. Nader S, Diamanti-Kandarakis E. Polycystic ovary syndrome, oral contraceptives and metabolic issues: new perspectives and a unifying hypothesis. Hum Reprod. 2007;22(2):317–22.

21. Krassas GE, Pontikides N, Kaltsas T, Papadopoulou P. Ovarian volume and follicle population in women with Hashimoto's thyroiditis. Thyroid. 2000;10(8):633–7.

22. Aversa A, La Vignera S, Rago R, Gambineri A, La Rosa VL, Condorelli RA, et al. Fundamental concepts and novel aspects of polycystic ovarian syndrome: Expert consensus and recommendations. Reprod Sci. 2020;27(3):973–86.

23. Duntas LH. Thyroid disease and lipids. Thyroid. 2002;12(4):287–93.

24. Maratou E, Hadjidakis DJ, Peppa M, Alevizaki M, Tsegka K, Lambadiari V, et al. Studies of insulin resistance in patients with clinical and subclinical hypothyroidism. Eur J Endocrinol. 2009;160(5):785–90.

25. Wild RA, Carmina E, Diamanti-Kandarakis E, Dokras A, Escobar-Morreale HF, Futterweit W, et al. Assessment of cardiovascular risk and prevention of cardiovascular disease in women with the polycystic ovary syndrome: A consensus statement by the Androgen Excess and Polycystic Ovary Syndrome (AE-PCOS) Society. J Clin Endocrinol Metab. 2010;95(5):2038–49.

26. Poppe K, Velkeniers B. Thyroid disorders in infertile women. Ann Endocrinol (Paris). 2003;64(1):45–50.

27. Janssen OE, Mehlmauer N, Hahn S, Offner AH, Gärtner R. High prevalence of autoimmune thyroiditis in patients with polycystic ovary syndrome. Eur J Endocrinol. 2004;150(3):363–9.