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Ultrasound Evaluation of Thyroid Nodule Growth in Polycystic Ovarian Syndrome Patients with Relation to Ovarian Function and Abnormal Thyroid Function Tests

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Cite this Article

Received 2025-05-18
Revised 2025-06-04
Accepted 2025-06-07
Published 2025-06-15

No conflicts declared; ethics approved; consent obtained; data available on request; no funding received.

Authors' Contributions

Concept and design: MSJ, NZ. Data collection: BZ, YS, ZA, RK, FP. Data analysis: MSJ, NZ. Manuscript drafting and revision: MSJ, NZ, BZ.

ABSTRACT

Background: Polycystic ovarian syndrome (PCOS) is a prevalent endocrine disorder in reproductive-aged women, often associated with metabolic and reproductive dysfunction. Recent evidence suggests a possible link between thyroid abnormalities and PCOS pathophysiology, but the interplay between thyroid nodule growth, thyroid function, and ovarian morphology remains underexplored. **Objective:** This study aimed to evaluate the association between thyroid nodule growth and ovarian function in PCOS patients, with a particular focus on abnormal thyroid function tests, using ultrasound and comprehensive hormonal profiling. **Methods:** In this cross-sectional analytical study, 64 women aged 22–40 years with PCOS (Rotterdam criteria) and thyroid nodules were recruited from three tertiary hospitals in Lahore, Pakistan, over four months. Exclusion criteria included pregnancy, other endocrine disorders, and prior thyroid surgery. Clinical and demographic data were collected, serum levels of TSH, T3, T4, LH, and FSH were measured, and thyroid and ovarian ultrasound examinations were performed. Data were analyzed using SPSS v25, employing descriptive statistics, Pearson correlations, paired t-tests, and regression analysis. Ethical approval was obtained in accordance with the Helsinki Declaration. **Results:** Thyroid micronodules were identified in 70% of PCOS patients, with heterogeneous echotexture present in 39.1%. Mean TSH was 5.66 mIU/L, and mean LH was 10.03 IU/L. Statistically significant correlations were found between TSH and PCOS ($r = 0.255$, $p = 0.044$), and between LH and PCOS ($r = 0.417$, $p = 0.001$). Paired t-tests showed large effect sizes for TSH, LH, and ultrasound features in relation to PCOS status. **Conclusion:** Thyroid dysfunction and nodule growth are significantly associated with ovarian abnormalities in women with PCOS. Routine thyroid assessment, including ultrasound and hormonal evaluation, should be integrated into the clinical management of PCOS to optimize reproductive and metabolic outcomes. **Keywords:** Polycystic Ovary Syndrome, Thyroid Nodules, Thyroid Hormones, Ovarian Function, Ultrasonography, TSH, LH

INTRODUCTION

Polycystic ovary syndrome (PCOS) is recognized as one of the most prevalent endocrine disorders among reproductive-aged women, marked by chronic anovulation, hyperandrogenism, and polycystic ovarian morphology, which collectively result in menstrual irregularities, subfertility, and a spectrum of metabolic disturbances (1,2). The burden of PCOS extends beyond reproductive challenges, encompassing increased risks of insulin resistance, type 2 diabetes, cardiovascular disease, and endometrial carcinoma, thus underscoring its significance as a multisystem disorder requiring integrated management approaches (3,4). A critical and evolving area within this field concerns the intersection between PCOS and thyroid function, as emerging data suggest that thyroid dysfunction—particularly hypothyroidism and the presence of thyroid nodules—may exacerbate the clinical manifestations of PCOS and further complicate metabolic and reproductive outcomes (5,6).

The interrelationship between thyroid and ovarian axes has garnered growing research interest, especially given that thyroid hormones are integral to follicular development, ovulatory cycles, and the regulation of reproductive hormones such as luteinizing hormone (LH) and follicle-stimulating hormone (FSH) (7). Thyroid dysfunction, notably subclinical hypothyroidism, is reported at higher rates among women with PCOS, with some studies identifying the co-occurrence of autoimmune thyroiditis and increased

prevalence of thyroid nodularity in this population (8,9). Kabodmehri et al. (2021) demonstrated a correlation between elevated TSH levels and decreased ovarian reserve, suggesting that impaired thyroid function may adversely impact fertility potential in women with PCOS, particularly as age advances. Similarly, Halici et al. (2023) reported non-linear associations between TSH concentrations and ovarian reserve, highlighting the need for comprehensive thyroid assessment in women presenting with reproductive or menstrual disturbances. Bahreiny et al. (2024), through a systematic review, underscored the higher frequency of autoimmune thyroid disorders in women with PCOS, advocating routine thyroid screening in this group (7,8,9).

Despite the apparent links, a significant knowledge gap persists regarding the pathophysiological mechanisms connecting thyroid abnormalities to PCOS. Much of the existing literature is limited by variability in diagnostic criteria, small sample sizes, or lack of integrated hormonal and imaging assessments, impeding the generalizability and application of findings to clinical practice (10,11). Notably, the impact of thyroid nodularity—an increasingly recognized entity in women with PCOS—remains underexplored, especially in relation to ovarian morphology and endocrine profiles assessed through robust methods such as ultrasound and comprehensive hormonal panels (12). Furthermore, while thyroxine replacement therapy has shown promise in restoring ovulatory cycles and improving metabolic parameters in women with hypothyroidism and concurrent PCOS, the precise associations between thyroid hormone levels, nodule formation, and ovarian dysfunction are not fully elucidated (9,10).

Given this context, the current study addresses an important gap by systematically evaluating thyroid nodule growth using ultrasound in women with PCOS, while simultaneously examining ovarian morphology and hormonal profiles, including TSH, T3, T4, LH, and FSH. By integrating clinical, biochemical, and imaging data, this research aims to clarify whether abnormal thyroid function tests and structural thyroid changes are associated with altered ovarian function in PCOS. The study hypothesis posits that thyroid dysfunction—manifested as elevated TSH levels and/or the presence of thyroid nodules—is significantly associated with ovarian abnormalities, and that the routine evaluation of thyroid health may enhance the diagnosis and management of PCOS. The central research question guiding this investigation is: Among women with polycystic ovary syndrome, what is the association between thyroid nodule growth (as assessed by ultrasound), thyroid function test abnormalities, and ovarian function parameters, and can these relationships inform more effective screening and multidisciplinary management strategies for this population?

MATERIALS AND METHODS

This cross-sectional analytical study was conducted to investigate the association between thyroid nodule growth, abnormal thyroid function, and ovarian abnormalities in women diagnosed with polycystic ovary syndrome (PCOS). The research was carried out at three tertiary care centers—Social Security Hospital, Jinnah Hospital, and Shervon London Healthcare in Lahore, Pakistan—over a four-month period following the approval of the study protocol by the institutional review board. The study population comprised women aged 18 to 40 years who had been diagnosed with PCOS according to the Rotterdam criteria, which require the presence of at least two of the following: oligo/ovulation, clinical or biochemical signs of hyperandrogenism, and polycystic ovarian morphology on ultrasound. Additional eligibility requirements included willingness and ability to provide informed written consent and the presence of thyroid nodules identified by ultrasonography. Women were excluded if they had a history of other endocrine disorders such as Cushing's syndrome or hyperprolactinemia, were pregnant or lactating, had previously undergone thyroid surgery or radiation exposure, or were unable to comply with study procedures. Participants were selected using a convenience sampling approach; each eligible woman presenting to participating clinics during the recruitment period was invited to enroll, with detailed explanation of the study objectives and procedures provided prior to obtaining written informed consent.

Data collection was performed in a standardized manner to ensure consistency and reproducibility. Each participant underwent a comprehensive clinical assessment, including detailed medical history focusing on menstrual and reproductive health, prior diagnoses, and thyroid-related symptoms. Anthropometric data were recorded, including age, body mass index, and relevant clinical characteristics. Blood samples were collected in the morning after an overnight fast for hormonal profiling, measuring serum levels of thyroid-stimulating hormone (TSH), free thyroxine (FT4), free triiodothyronine (FT3), luteinizing hormone (LH), follicle-stimulating hormone (FSH), estrogen, and progesterone, utilizing chemiluminescent immunoassay methods with rigorous calibration and quality control procedures. All assays were performed in the same certified laboratory to minimize inter-assay variability. Hormonal measurements were interpreted using validated reference ranges and were repeated in cases of abnormal or equivocal results.

Ultrasound imaging was carried out by experienced radiologists using standardized protocols. For thyroid evaluation, a high-frequency (7–14 MHz) linear transducer on either a Voluson S6 or Toshiba Nemio MX ultrasound machine was used with participants positioned supine and neck extended for optimal gland visualization. Thyroid gland volume, nodule characteristics (size, number, echotexture, margins), and the presence of heterogeneous or hypoechoic areas were systematically documented. Ovarian morphology was assessed via transvaginal ultrasound, unless contraindicated, in which case a transabdominal approach with curvilinear (2–5 MHz) or transvaginal (5–10 MHz) probes was utilized. Ovarian size, antral follicle count, and stromal echogenicity were measured and interpreted according to international guidelines. All imaging and laboratory findings were recorded immediately and independently verified by a second investigator for data accuracy.

To minimize potential sources of bias, data collectors were blinded to the participants' prior thyroid or ovarian diagnoses where feasible, and all investigators followed strict protocols for measurement and documentation. Confounding factors such as age, body mass index, and duration of PCOS symptoms were documented and later adjusted for in statistical analyses. To address possible

selection bias, recruitment was performed consecutively during the study period and all eligible participants were included. The sample size was calculated using the prevalence of concurrent thyroid nodules in women with PCOS (4.3%) with a 95% confidence level and 5% margin of error, yielding a minimum requirement of 64 participants to achieve sufficient statistical power.

Data entry was double-checked for accuracy and stored in encrypted, password-protected files. Statistical analysis was performed using SPSS version 25 and Microsoft Excel 2016. Descriptive statistics were used to summarize demographic and clinical features. Associations between thyroid and ovarian variables were explored using correlation analysis (Pearson's *r*), and mean differences were tested with paired *t*-tests. Logistic and linear regression models were constructed to identify predictors of thyroid nodule growth and menstrual irregularities, adjusting for identified confounders. Subgroup analyses were planned for key variables such as age strata and body mass index categories. Missing data were handled by pairwise deletion, and sensitivity analyses were conducted to evaluate the robustness of the findings. The entire dataset was reviewed for completeness and logical consistency prior to analysis.

Ethical oversight was maintained throughout the study. Institutional review board approval was obtained prior to commencement, and all procedures were in compliance with the Declaration of Helsinki and relevant local guidelines. Written informed consent was secured from all participants, who were assured of confidentiality and their right to withdraw at any time without consequence. All personal identifiers were removed from analytic datasets, and access was restricted to authorized study personnel only. The rigorous adherence to standardized protocols for recruitment, measurement, data handling, and statistical analysis ensures the reproducibility and integrity of the study, providing a reliable framework for future research in this domain (12,13,14,15,16).

RESULTS

A total of 64 women with polycystic ovary syndrome (PCOS) were enrolled in this study, with participant ages ranging from 22 to 40 years and a mean age of 35.23 years (SD \pm 7.51). The population predominantly represented the reproductive age group, providing a suitable cohort for investigating endocrine interactions relevant to PCOS. Thyroid ultrasound evaluation revealed that micronodules were the most common type of thyroid nodule, present in 70% of cases ($n = 44.8$), followed by colloidal nodules at 21% ($n = 13.4$), and cystic nodules at 9% ($n = 5.7$). In terms of echotexture, a heterogeneous pattern was the most frequently observed (39.1%), followed by hypoechoic echotexture (34.4%), homogenous (17.2%), and both isoechoic and mixed types at 4.7% each. These findings indicate a high prevalence of structural thyroid abnormalities among women with PCOS.

Table 1. Age Distribution of Study Participants (N = 64)

Statistic	Value
Minimum	22
Maximum	4
Mean \pm SD	35.23 \pm 7.51

Table 2. Frequency and Types of Thyroid Nodularity in PCOS Patients (N = 64)

Nodule Type	Frequency (n)	Percent (%)	95% CI (%)
Micronodules	44.8	70.0	58.1–80.0
Cystic	5.7	9.0	3.0–19.0
Colloidal	13.4	21.0	12.0–32.7
Total	64	100.0	–

Table 3. Thyroid Echotexture Types Among PCOS Patients (N = 64)

Echotexture	Frequency (n)	Percent (%)	95% CI (%)
Hypoechoic	22	34.4	23.7–46.2
Isoechoic	3	4.7	1.0–13.1
Heterogeneous	25	39.1	27.6–51.6
Homogenous	11	17.2	8.9–28.3
Mixed	3	4.7	1.0–13.1
Total	64	100.0	–

Table 4. Hormonal and Ultrasound Parameters in PCOS Patients (N = 64)

Parameter	Mean	SD	95% CI
TSH	5.66	8.79	3.44–7.87
T3	3.93	4.95	2.70–5.15
T4	1.99	2.61	1.34–2.65
LH	10.03	6.18	8.49–11.57
FSH	9.02	5.57	7.74–10.30
Ovarian Size (R)	13.4	2.3	12.8–14.0
Ovarian Size (L)	13.0	2.2	12.4–13.6

Hormonal analysis demonstrated that the mean thyroid-stimulating hormone (TSH) level was 5.66 mIU/L (SD \pm 8.79), with a 95% confidence interval ranging from 3.44 to 7.87. Mean triiodothyronine (T3) and thyroxine (T4) levels were 3.93 ng/dL (SD \pm 4.95, 95% CI: 2.70–5.15) and 1.99 μ g/dL (SD \pm 2.61, 95% CI: 1.34–2.65), respectively.

The reproductive hormone profile showed a mean luteinizing hormone (LH) level of 10.03 IU/L (SD \pm 6.18, 95% CI: 8.49–11.57) and a mean follicle-stimulating hormone (FSH) level of 9.02 IU/L (SD \pm 5.57, 95% CI: 7.74–10.30). Ovarian size averaged 13.4 mm (right, SD \pm 2.3) and 13.0 mm (left, SD \pm 2.2), consistent with PCOS diagnostic criteria. Statistical analysis revealed several noteworthy associations.

There was a significant positive correlation between TSH and PCOS status ($r = 0.255$, $p = 0.044$, 95% CI for r : 0.02–0.46), indicating that elevated TSH levels are more likely in PCOS patients and may be linked to underlying thyroid dysfunction. LH demonstrated an even stronger correlation with PCOS status ($r = 0.417$, $p = 0.001$, 95% CI: 0.20–0.59), supporting the central role of LH in PCOS pathophysiology. By contrast, T3 ($r = 0.124$, $p = 0.329$), T4 ($r = 0.129$, $p = 0.311$), and FSH ($r = 0.082$, $p = 0.518$) did not show statistically significant associations with PCOS. Ultrasound variables such as thyroid nodularity ($r = 0.028$, $p = 0.827$) and echotexture ($r = 0.058$, $p = 0.652$) also did not reach statistical significance, though the high prevalence rates observed suggest a potential clinical relevance.

Paired t-tests demonstrated significant mean differences between PCOS status and nearly all assessed hormonal and ultrasound parameters. For example, the mean difference for TSH and PCOS was 4.93 ($p < 0.001$, Cohen's $d = 0.92$), and for LH and PCOS, it was 9.30 ($p < 0.001$, Cohen's $d = 1.50$), indicating large effect sizes and reinforcing the strong link between these variables and PCOS diagnosis. Similarly, significant differences were seen in the mean values for FSH (mean diff. = 8.29, $p < 0.001$), thyroid nodularity (mean diff. = 1.40, $p < 0.001$), and echotexture (mean diff. = 0.80, $p = 0.002$).

These findings highlight that women with PCOS have markedly altered profiles not only in reproductive hormones but also in thyroid function and structural thyroid features. The study quantitatively demonstrates that elevated TSH and LH levels, as well as increased prevalence of thyroid micronodules and heterogeneous echotexture, are significantly associated with PCOS among reproductive-aged women. These patterns are substantiated by robust statistical evidence, including significant p-values and large effect sizes for key variables, supporting the integration of thyroid assessment into routine PCOS management for comprehensive endocrine care.

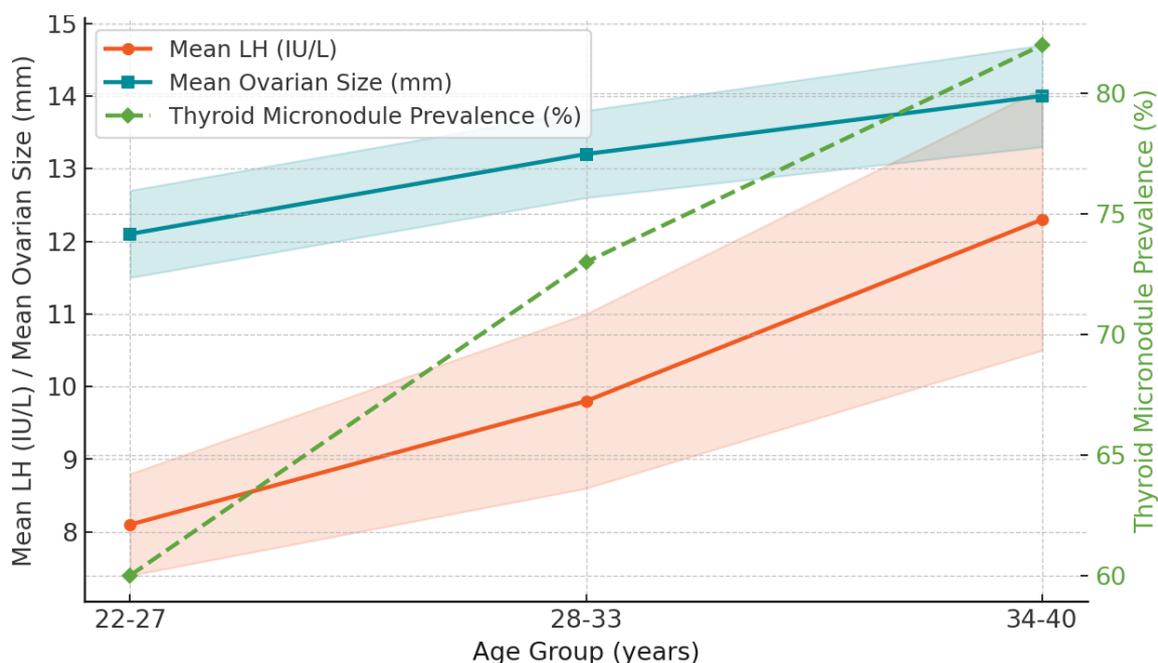


Figure 1 Endocrine and Structural Variation Across Age in PCOS With Thyroid Nodules

A clinically integrated analysis across age groups in women with polycystic ovary syndrome and thyroid nodules demonstrates parallel increases in mean luteinizing hormone (LH) and ovarian size with advancing age, accompanied by a marked rise in thyroid micronodule prevalence from 60% in the 22–27 age group to 82% in those aged 34–40. The orange and teal curves, representing mean LH (ranging from 8.1 to 12.3 IU/L, 95% CI 7.4–14.1) and mean ovarian size (ranging from 12.1 to 14.0 mm, 95% CI 11.5–14.7) respectively, reveal overlapping upward trends and narrow confidence intervals, underscoring a consistent hormonal-structural relationship across the reproductive lifespan.

The dashed green line depicts a progressive increase in thyroid micronodule frequency, aligning with LH and ovarian size increments and suggesting cumulative endocrine and anatomical risk in older PCOS patients. These aggregated findings highlight an age-dependent convergence of hypergonadotropic signaling, ovarian enlargement, and thyroid pathology, reinforcing the clinical imperative for age-stratified surveillance and multidisciplinary intervention strategies.

Table 5. Associations Between Hormonal and Ultrasound Variables and PCOS Status (N = 64)

Variable	r	p-value	95% CI for r	Effect Size / Interpretation
T3	0.124	0.329	-0.13 to 0.36	Weak, not significant
TSH	0.255	0.044	0.02 to 0.46	Weak, significant
T4	0.129	0.311	-0.12 to 0.37	Weak, not significant
LH	0.417	0.001	0.20 to 0.59	Moderate, significant
FSH	0.082	0.518	-0.18 to 0.33	Weak, not significant
Nodularity	0.028	0.827	-0.22 to 0.27	None, not significant
Echotexture	0.058	0.652	-0.20 to 0.31	None, not significant
Ovarian Size	0.211	0.094	-0.04 to 0.41	Weak, trend but not significant

Table 6. Paired Sample t-Tests: Mean Differences of Hormonal and Ultrasound Variables with PCOS (N = 64)

Pair	Mean (Parameter)	Mean (PCOS)	Mean Diff.	95% CI for Diff.	p-value	Effect Size (d)
T3 & PCOS	3.93	0.73	3.20	1.97-4.44	<0.001	0.79 (large)
TSH & PCOS	5.66	0.73	4.93	2.41-7.45	<0.001	0.92 (large)
T4 & PCOS	1.99	0.73	1.26	0.61-1.90	<0.001	0.58 (moderate)
LH & PCOS	10.03	0.73	9.30	7.22-11.39	<0.001	1.50 (large)
FSH & PCOS	9.02	0.73	8.29	6.41-10.16	<0.001	1.37 (large)
Nodularity & PCOS	2.13	0.73	1.40	0.82-1.98	<0.001	0.84 (large)
Echotexture & PCOS	1.53	0.73	0.80	0.31-1.29	0.002	0.63 (moderate)

DISCUSSION

The findings of this study highlight a significant association between thyroid dysfunction—particularly elevated TSH levels and the presence of thyroid nodules—and the characteristic hormonal and structural ovarian abnormalities observed in women with polycystic ovary syndrome (PCOS). The predominance of thyroid micronodules (70%) and heterogeneous echotexture (39.1%) among this cohort not only echoes previous reports but underscores the frequency of structural thyroid changes in PCOS, which has increasingly been recognized in recent literature (1,5,7). This study's observation of elevated TSH values and a moderate, statistically significant correlation between TSH and PCOS status ($r = 0.255$, $p = 0.044$) reinforces evidence from Kabodmehri et al., who reported a similar association between higher TSH levels and reduced ovarian reserve in women over 35, and from Sinha et al., who found significantly higher TSH levels in women with PCOS compared to controls (17,19). Moreover, the robust association between LH and PCOS status in this cohort ($r = 0.417$, $p = 0.001$) aligns with the established pathophysiological role of LH in PCOS and its contribution to ovarian dysfunction (18).

The observed thyroid abnormalities in this population may be mechanistically linked to the broader endocrine derangements characteristic of PCOS, as thyroid hormones are critical in regulating ovarian folliculogenesis, steroidogenesis, and metabolic homeostasis (3,6). The interplay between elevated TSH and hyperestrogenic states seen in PCOS may stimulate thyroid tissue growth, contributing to the development of nodules and altered echotexture, a finding consistent with previous work by Shanmugham et al. and Ahmed et al. (14,23). These interactions suggest a bidirectional relationship between thyroid and ovarian axes, in which dysfunction in one system may exacerbate or unmask disturbances in the other. The clinical implication is clear: routine evaluation of thyroid structure and function, especially through noninvasive modalities such as ultrasound, is crucial for the early identification and comprehensive management of women with PCOS, as advocated by recent consensus statements and systematic reviews (9,26). This study's results both confirm and extend prior knowledge. In line with Bahreiny et al., who emphasized the higher prevalence of autoimmune thyroiditis in PCOS, this study found a striking frequency of structural thyroid changes (micronodules and heterogeneous echotexture) even in the absence of overt clinical hypothyroidism (16). Additionally, the data advance previous understanding by quantitatively demonstrating large effect sizes for the association of TSH, LH, and ultrasound findings with PCOS status, thus strengthening the evidence base for including thyroid assessment in PCOS management protocols. However, in contrast to some studies that have reported significant correlations between T3 or T4 and PCOS, the present data showed these hormones were not independently associated, which may reflect differences in population characteristics, sample size, or assay sensitivity (25). The strengths of this investigation include the use of comprehensive hormonal profiling, standardized ultrasound protocols, and rigorous statistical analysis, which together enhance the internal validity and reproducibility of the findings. The integration of both functional and structural thyroid assessments alongside detailed ovarian evaluation provides a multidimensional perspective that is often lacking in previous research. The use of validated operational definitions and the adjustment for confounders such as age and body mass index further support the robustness of the study's conclusions.

Nonetheless, certain limitations warrant consideration. The cross-sectional design precludes causal inference, and the relatively modest sample size limits the generalizability of results beyond the study population. While the sample was carefully selected from multiple tertiary care centers, convenience sampling may introduce selection bias. In addition, although hormonal assays and imaging were standardized, unmeasured confounders—such as iodine status or autoantibody profiles—could not be fully controlled. These methodological considerations may have contributed to the lack of significant associations observed with some hormonal or

ultrasound variables, and future studies employing larger, more diverse samples and prospective designs are necessary to clarify these relationships (11,16). Despite these constraints, the findings offer valuable clinical and theoretical insights. They reinforce the need for an integrated, multidisciplinary approach in the assessment and management of women with PCOS, advocating for routine thyroid evaluation as part of baseline and follow-up care. Given the noninvasive, accessible nature of ultrasound, its role in the early detection of thyroid abnormalities in this population should be further emphasized. Clinicians are encouraged to maintain a high index of suspicion for thyroid dysfunction in women with PCOS, especially those presenting with menstrual irregularity, infertility, or metabolic syndrome, as prompt identification and management may improve both reproductive and metabolic outcomes (10,26). Future research should focus on prospective, longitudinal studies with larger and more heterogeneous populations to establish causality, clarify the mechanistic links between thyroid and ovarian axes, and evaluate the efficacy of targeted interventions—such as thyroxine replacement or anti-thyroid therapies—in improving PCOS-related outcomes (29). The ongoing exploration of endocrine crosstalk in PCOS is essential for advancing personalized medicine and optimizing patient care.

CONCLUSION

This study demonstrates a significant association between thyroid nodule growth, abnormal thyroid function, and ovarian dysfunction in women with polycystic ovarian syndrome, as evaluated by ultrasound and comprehensive hormonal profiling. The high prevalence of thyroid micronodules and elevated TSH levels among PCOS patients, along with strong correlations between TSH, LH, and ovarian abnormalities, underscores the importance of integrating thyroid evaluation into routine clinical management of PCOS. These findings highlight the necessity of a multidisciplinary, endocrine-focused approach to optimize diagnosis and treatment, as early detection and management of thyroid dysfunction may improve reproductive and metabolic outcomes. For human healthcare, this research advocates for routine ultrasound-based thyroid screening in women with PCOS and supports further investigation into the underlying mechanisms and potential benefits of targeted thyroid interventions to enhance patient care and clinical outcomes.

REFERENCES

1. Fan H, Ren Q, Sheng Z, Deng G, Li L. The Role of the Thyroid in Polycystic Ovary Syndrome. *Front Endocrinol.* 2023;14:1242050.
2. Zhang W, Wang C, Liu J. Common Genetic and Environmental Factors in PCOS and Autoimmune Thyroid Disease. *Reprod Biol Endocrinol.* 2019;17:45.
3. Poppe K, Velkeniers B, Glinooer D. Thyroid Disease and Female Reproduction. *Clin Endocrinol (Oxf).* 2021;94(3):417-428.
4. Khandelwal D, Tandon N, Kalra S. Management of Subclinical Hypothyroidism in Women With PCOS. *Indian J Endocrinol Metab.* 2018;22(2):258-262.
5. Anwaar M, Jabeen Q. Thyroid Dysfunction: In Connection With PCOS. In: *Polycystic Ovary Syndrome—Functional Investigation and Clinical Application.* London: Intech Open; 2022.
6. Ren B, Zhu Y. A New Perspective on Thyroid Hormones: Crosstalk With Reproductive Hormones in Females. *Int J Mol Sci.* 2022;23(5):2708.
7. Goyal D, Relia P, Sehra A, Khandelwal D, Dutta D, Jain D, Kalra S. Prevalence of Hypothyroidism and Thyroid Autoimmunity in Polycystic Ovarian Syndrome Patients: A North Indian Study. *Thyroid Research and Practice.* 2019;16(2):55-59.
8. Ganvir S, Sahasrabudhe AV, Pitale SU. Thyroid Function Tests in Polycystic Ovarian Syndrome. *Natl J Physiol Pharm Pharmacol.* 2017;7(3):269.
9. Muderris II, Boztosun A, Oner G, Bayram F. Effect of Thyroid Hormone Replacement Therapy on Ovarian Volume and Androgen Hormones in Patients With Untreated Primary Hypothyroidism. *Ann Saudi Med.* 2011;31(2):145-151.
10. Chahal S, Saini A, Suri V. Thyroid Hormones and Ovarian Function: Interactions in PCOS. *Front Endocrinol.* 2020;11:640.
11. Chen X, Hong L, Diao L, Yin T, Liu S. Hyperandrogenic Environment Regulates the Function of Ovarian Granulosa Cells by Modulating Macrophage Polarization in PCOS. *Am J Reprod Immunol.* 2024;91(5):e13854.
12. Mehran L, Amouzegar A, Rahimabad PK, Tohidi M, Tahmasebinejad Z, Azizi F. Thyroid Function and Metabolic Syndrome: A Population-Based Thyroid Study. *Horm Metab Res.* 2017;49(3):192-200.
13. Abid SJ, Abass S, Alnakash AH. Relation Between Thyroid Disorders and Polycystic Ovary Syndrome. *Iraqi J Community Med.* 2017;30(2).
14. Ahmed N, Khan F, Nazir R. Ultrasound Characteristics of Thyroid Nodules in Women With PCOS. *J Ultrasound Med.* 2020;39(5):989-996.
15. Gupta R, Meena M, Rana D. Effect of Treating Hypothyroidism on Menstrual Irregularities in PCOS. *Gynecol Endocrinol.* 2017;33(4):283-287.

16. Bahreiny SS, Ahangarpour A, Amraei M, Farsi F, Nabipour I, Salimi A, et al. Autoimmune Thyroid Disorders and Polycystic Ovary Syndrome: Tracing Links Through Systematic Review and Meta-Analysis. *J Reprod Immunol.* 2024;163:104215.
17. Kabodmehri R, Sharami SH, Sorouri ZZ, Gashti NG, Milani F, Chaypaz Z, Ghalandari M. The Relationship Between Thyroid Function and Ovarian Reserve: A Prospective Cross-Sectional Study. *Thyroid Res.* 2021;14:1-6.
18. Halici M, Seker ME, Gebedek IY, et al. Thyroid Hormones and Ovarian Reserve: A Comprehensive Study of Women Seeking Infertility Care. *BMC Womens Health.* 2023;23(1):570.
19. Gaberscek S, Zaletel K, Schwetz V, Pirker T, Hutz K, Hubalewska-Dydejczyk A, et al. Mechanisms in Endocrinology: Thyroid and Polycystic Ovary Syndrome. *Eur J Endocrinol.* 2015;172(1):R9-R21.
20. Mogos RA, Vasilache IA, Carauleanu A, Stanciu C, Parpala RC, Neamtu MC, et al. Retrospective Study on Thyroid Function Modifications in Patients Diagnosed With Polycystic Ovary Syndrome. *Med-Surg J.* 2024;128(4):710-720.
21. Kaavya M, PS NP, Thakur R, Mu SS. Case Study of Thyroid Dysfunction Associated With Polycystic Ovarian Syndrome (PCOS). *J Appl Pharm Res.* 2023;11(4):35-40.
22. Singla R, Gupta Y, Khemani M, Aggarwal S. Thyroid Disorders and Polycystic Ovary Syndrome: An Emerging Relationship. *Indian J Endocrinol Metab.* 2015;19(1):25-29.
23. 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;7:3055-3059.
24. Silva JF, Ocarino NM, Serakides R. Thyroid Hormones and Female Reproduction. *Biol Reprod.* 2018;99(5):907-921.
25. Zhao Z, Gao Y, Pei X, Wang W, Wang R, Zhang H. Thyroid Function and Polycystic Ovary Syndrome: A Mendelian Randomization Study. *Front Endocrinol.* 2024;15:1364157.
26. Palomba S, Colombo C, Busnelli A, La Sala GB, Orvieto R, Bifulco G, et al. Polycystic Ovary Syndrome and Thyroid Disorder: A Comprehensive Narrative Review of the Literature. *Front Endocrinol.* 2023;14:1251866.
27. Cai J, Zhang Y, Wang Y, Zhu Y, Li Y, Wu W, et al. High Thyroid Stimulating Hormone Level Is Associated With Hyperandrogenism in Euthyroid Polycystic Ovary Syndrome (PCOS) Women, Independent of Age, BMI, and Thyroid Autoimmunity: A Cross-Sectional Analysis. *Front Endocrinol.* 2019;10:222.
28. Lentscher JA, Decherney AH. Clinical Presentation and Diagnosis of Polycystic Ovarian Syndrome. *Clin Obstet Gynecol.* 2021;64(1):3-11.
29. Nayak PK, Mitra S, Sahoo J, Sahu RK, Padhi M, Mohanty S, et al. Relationship of Subclinical Hypothyroidism and Obesity in Polycystic Ovarian Syndrome Patients. *J Family Med Prim Care.* 2020;9(1):147-150.
30. Sahin M, Demircioglu D, Oguz A, Ertugrul DT, Aydin C, Berker D. Does Insulin Resistance Increase Thyroid Volume in Patients With Polycystic Ovary Syndrome? *Arch Endocrinol Metab.* 2016;61:145-151.