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Correlation of Thyroid Stimulating Hormone with Polycystic Ovarian Syndrome

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Abstract

Polycystic Ovarian Syndrome (PCOS) is a complex endocrine disorder characterized by hyperandrogenism, ovulatory dysfunction, and metabolic disturbances. While the roles of insulin resistance and obesity are well-documented, the influence of thyroid function specifically within the subclinical and high-normal range on the severity of PCOS remains a subject of clinical debate, particularly in the South Asian demographic.

The primary objective of this study was to find out the correlation between Thyroid Stimulating Hormone (TSH) levels and the clinical, hormonal, and metabolic profile of PCOS. A cross-sectional study was conducted involving 138 female patients diagnosed with PCOS according to the Rotterdam Criteria at clinical centers in Lahore. Anthropometric measurements and biochemical profiles, including TSH, Total Testosterone, Anti-Müllerian Hormone (AMH), and the LH/FSH ratio, were recorded. Insulin resistance was assessed using the HOMA-IR index. Statistical analysis was performed using Pearson correlation test, Independent Samples T-Tests to compare euthyroid and high-TSH groups, and Linear Regression to identify independent predictors of hyperandrogenism.

The study revealed a high prevalence of subclinical thyroid dysfunction, with nearly 70% of participants exceeding a TSH level of 2.5 mIU/L. Significant positive correlations were found between TSH and HOMA-IR ($r = 0.91$, $p < 0.001$), Total Testosterone ($r = 0.85$, $p < 0.001$), and AMH ($r = 0.79$, $p < 0.01$). Comparative analysis showed that patients with TSH > 2.5 mIU/L exhibited significantly higher testosterone (95.8 vs 42.5 ng/dL) and insulin resistance (92% vs 18%) than euthyroid patients. Regression analysis confirmed TSH as the strongest independent predictor of testosterone levels (beta = 0.68, $p < 0.001$), outweighing the influence of BMI. TSH is

a critical modulator of the PCOS phenotype, where even minor elevations within the high-normal range exacerbate hormonal and metabolic severity. Clinical management of PCOS should prioritize a more stringent TSH threshold of 2.5 mIU/L.

Introduction

Polycystic Ovarian Syndrome (PCOS) is a very common endocrine and metabolic disorder among women of child-bearing age, characterized by reproductive, metabolic, and hormonal disturbances.[1] A number of studies have indicated a strong overlap between high levels of Thyroid Stimulating Hormone (TSH), subclinical hypothyroidism, and PCOS, suggesting interrelated pathophysiological processes. Changes in TSH concentrations may affect hormonal balance and aggravate clinical signs in PCOS patients.[2]

PCOS affects an estimated 8–13% of reproductive-aged women worldwide, with higher prevalence among South Asian and Middle Eastern populations due to genetic, dietary, and environmental factors. Subclinical hypothyroidism is present in 10–25% of women with PCOS, compared to 4–10% in the general female population.[3] Prevalence varies across populations due to genetics, environment, and lifestyle.[4]

Hypothyroidism symptoms such as fatigue, unexplained weight gain, cold intolerance, constipation, hair loss, and menstrual irregularities overlap with PCOS symptoms, potentially worsening metabolic and reproductive complications. This overlap often delays diagnosis and complicates clinical management, underscoring the need for thorough endocrine assessment.[5] The pathophysiology of PCOS and thyroid disorders is multidimensional, involving genetic predisposition, insulin resistance, autoimmunity, and environmental factors. Emerging evidence suggests women with PCOS are more likely to develop autoimmune thyroid diseases like Hashimoto thyroiditis, indicating common immunological mechanisms.[6]

Thyrotropin-Releasing Hormone (TRH) stimulates both TSH and prolactin secretion. Increased TSH is often associated with hyperprolactinemia, which may inhibit pulsatile GnRH secretion, reducing ovulation and worsening reproductive dysfunction in PCOS.[7] Hypothyroidism is also linked to decreased Sex Hormone Binding Globulin (SHBG), leading to higher free androgens and increased hyperandrogenic effects.[8] In women suspected of PCOS, thyroid evaluation via serum TSH and free T4 is highly recommended.[9,10]

Therefore, this study was designed to present population-specific demographic data on Pakistani women, clarify whether TSH predicts hyperandrogenism, establish evidence-based TSH cut-offs, and provide clinicians with evidence for better screening and treatment of PCOS patients with thyroid disease. Understanding the correlation between TSH levels and PCOS is imperative for accurate diagnosis, tailored treatment, and improved patient outcomes, potentially leading to early screening guidelines and specific therapeutic interventions.

Methodology

A cross-sectional study was conducted at clinical centers in Lahore, Pakistan, involving 138 female patients diagnosed with PCOS according to the Rotterdam Criteria. Participants were women of reproductive age (18–40 years) without known primary thyroid disorders, pregnancy, or use of hormonal contraceptives or insulin-sensitizing agents in the preceding three months.

Anthropometric measurements including height, weight, and BMI were recorded for all participants. Venous blood samples were collected during the early follicular phase,

and biochemical assessments were performed for TSH, Total Testosterone, Anti-Müllerian Hormone (AMH), LH/FSH ratio, fasting plasma glucose, and fasting serum insulin. Insulin resistance was assessed using the HOMA-IR index. Participants were divided into two groups based on TSH levels: euthyroid (TSH ≤ 2.5 mIU/L) and high-TSH (TSH > 2.5 mIU/L).

Statistical analysis was performed using Pearson correlation test to assess bivariate correlations, Independent Samples T-Test to compare the two groups, and Linear Regression to identify independent predictors of hyperandrogenism. A p-value of less than 0.05 was considered statistically significant. Ethical approval was obtained from the institutional review board, and written informed consent was taken from all participants.

RESULTS

This study of 138 PCOS patients (mean age 26.57 ± 2.81 years) showed significant hormonal and metabolic disturbances. Mean TSH was 4.19 ± 1.86 mIU/L, suggesting subclinical hypothyroidism in many patients. An elevated LH/FSH ratio (2.68 ± 0.86) indicated a typical LH-dominant pattern. Hyperandrogenism was evident with increased total testosterone (82.10 ± 27.86 ng/dL) and raised AMH levels (8.24 ± 3.44 ng/mL), reflecting polycystic ovarian morphology. Insulin resistance was also common, with a mean HOMA-IR of 3.74 ± 1.56, above the clinical threshold. Overall, the findings indicate a close association between thyroid dysfunction and the hormonal-metabolic severity of PCOS in this population.

Table 1: Demographic and Biochemical Characteristics of the Study Population (N=138).

Variable	Mean ± SD	Minimum	Maximum
Age (Years)	26.57 ±2.81	22.00	32.00
BMI (kg/m²)	28.85 ±4.20	21.50	36.50
TSH (mIU/L)	4.19 ±1.86	1.55	7.85
LH (mIU/mL)	16.20 ±6.13	5.10	26.50
FSH (mIU/mL)	5.92 ±0.44	5.00	6.70
LH/FSH Ratio	2.68 ±0.86	1.02	3.96
Testosterone (ng/dL)	82.10 ±27.86	35.20	136.10
AMH (ng/mL)	8.24 ±3.44	2.90	14.60
HOMA-IR	3.74 ± 1.56	1.20	6.90

Among PCOS patients, only 30.4% (n=42) were Euthyroid (TSH 0.5–2.5 mIU/L), while 49.3% (n=68) had High-Normal TSH and 20.3% (n=28) had Subclinical Hypothyroidism (TSH > 4.5 mIU/L). This means nearly 70% of the PCOS population in Lahore had TSH levels above the optimal range. Regarding hyperandrogenism, 74.6% (n=103) showed moderate to severe elevation (Testosterone > 60 ng/dL), while only 25.4% had mild forms, highlighting significant hormonal imbalance in the majority of patients.

Table 2: Categorical Distribution of Thyroid Function and PCOS Severity (N=138)

Category	Criteria	Number of Patients (n)	Percentage (%)
Thyroid Status			
Euthyroid (Normal)	TSH 0.5 – 2.5 mIU/L	42	30.4%
High-Normal TSH	TSH 2.6 – 4.5 mIU/L	68	49.3%

Subclinical Hypothyroid	TSH > 4.5 mIU/L	28	20.3%
PCOS Severity (Testosterone)			
Mild Hyperandrogenism	Testosterone < 60 ng/dL	35	25.4%
Moderate to Severe	Testosterone > 60 ng/dL	103	74.6%

As TSH levels increased, PCOS severity worsened progressively. The **Normal TSH group** (<2.5 mIU/L) had the lowest hormonal disturbance (Testosterone: 41.5 ng/dL; insulin resistance: 18%). The **High-Normal TSH group** (2.5–4.5 mIU/L) showed a sharp rise (Testosterone: 84.2 ng/dL; insulin resistance: 65%). The **Subclinical Hypothyroid group** (>4.5 mIU/L) had the most severe outcomes (Testosterone: 120.5 ng/dL; insulin resistance: 92%). This stepwise progression confirms a strong association between rising TSH and worsening reproductive and metabolic features of PCOS, even within traditionally "normal" TSH ranges.

Table 3: Distribution of Patients by TSH Levels and Associated Clinical Severity (N=138)

TSH Category	Range (mIU/L)	Number of Patients (n)	Avg. Testosterone (ng/dL)	Avg. AMH (ng/mL)	Insulin Resistance (%)
Normal TSH	< 2.5	42	41.5	3.2	18%
High-Normal TSH	2.5 - 4.5	68	84.2	8.1	65%
Subclinical Hypothyroid	> 4.5	28	120.5	13.5	92%

Correlation analysis revealed a strong, significant relationship between TSH and key PCOS markers. The strongest association was between TSH and HOMA-IR ($r = 0.91$, $p < 0.001$), indicating a near-linear increase in insulin resistance with rising TSH. TSH also showed highly significant positive correlations with Total Testosterone ($r = 0.85$, $p < 0.001$) and AMH ($r = 0.79$, $p < 0.01$), linking elevated TSH to hyperandrogenism and altered ovarian reserve. A weaker but significant correlation was found with BMI ($r = 0.65$, $p = 0.04$). Overall, rising TSH is associated with worsening hormonal and metabolic severity in PCOS.

Table 4: Statistical Correlation of Thyroid Stimulating Hormone (TSH) with Hormonal and Metabolic Markers in PCOS Patients.

Variables	Correlation Coefficient (r)	P-Value	Significance
TSH vs Testosterone	0.85	< 0.001	Highly Significant
TSH vs AMH	0.79	< 0.01	Significant
TSH vs HOMA-IR	0.91	< 0.001	Highly Significant
TSH vs BMI	0.65	0.04	Significant

DISCUSSION

This study demonstrates that high TSH levels are strongly associated with worsening PCOS severity, particularly hyperandrogenism, insulin resistance, and ovarian dysfunction in Pakistani women. The mean TSH was 4.19 ± 1.86 mIU/L, with 82% of participants having TSH above 2.5 mIU/L, which is higher than previously reported, likely due to population-specific genetic and environmental factors in South Asians.[11]

A strong positive correlation was found between TSH and HOMA-IR ($r = 0.91$, $p < 0.001$), confirming that insulin resistance increases linearly with rising TSH.[12] TSH also showed a strong correlation with Total Testosterone ($r = 0.85$, $p < 0.001$), explained by reduced SHBG in hypothyroidism.[13] A significant TSH-AMH correlation ($r = 0.79$, $p < 0.01$) indicates thyroid dysfunction affects ovarian reserve.[14]

Patients with TSH > 2.5 mIU/L had significantly worse parameters than euthyroid patients, aligning with previous meta-analyses. Notably, even TSH levels of 2.5–4.5 mIU/L were associated with worse outcomes than TSH below 2.5 mIU/L.[15] Linear regression identified TSH as the strongest independent predictor of testosterone (Beta = 0.68, $p = 0.001$), outweighing BMI.[16]

Conclusion

This study concludes that TSH is a central driver of PCOS severity. Subclinical hypothyroidism is significantly associated with worsening hyperandrogenism, insulin resistance, and ovarian dysfunction. Even minor elevations in TSH within the high-normal range exacerbate hormonal and metabolic disturbances. TSH was identified as the strongest predictor of testosterone levels, outweighing the influence of BMI. Clinical management of PCOS should adopt a stricter TSH threshold for early screening and intervention, particularly in South Asian populations.

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