Journal of Chemical Health Risks

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JCHR (2025) 15(2), 1685-1690 | ISSN:2251-6727

Thyroid Stimulating Hormone Level in Polycystic Ovarian Syndrome and Its Correlation with Insulin Resistance

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(Received: 16 February 2025 Revised: 20 March 2025 Accepted: 11 April 2025)

KEYWORDS

Thyroid Stimulating Hormone, Insulin Resistance, Polycystic Ovarian Syndrome.

ABSTRACT:

Background: Polycystic Ovarian Syndrome (PCOS) is a major endocrine ailment in reproductive-age women. It relates to female infertility, with insulin resistance (IR) considered the main factor in pathogenesis. The nature and degree of IR vary among ethnicities. Thyroid dysfunction, mainly subclinical hypothyroidism, is common in PCOS subjects and has been postulated as a determinant of IR. This study aims to estimate thyroid-stimulating hormone levels in polycystic ovarian syndrome and their association with insulin resistance.

Methods: This cross-sectional study was conducted in the Reproductive Endocrinology and Infertility OPD under the Department of Obstetrics and Gynaecology, Dhaka Medical College and Hospital, from June 2022 to May 2023. 75 diagnosed PCOS patients of reproductive age (15-40 years) were included. A 2 ml venous blood sample was taken to measure TSH and HOMA-IR. TSH was measured by Micro particle Enzyme Immunoassay (MEIA). TSH above 2.5 mIU/L was considered high. HOMA-IR measured insulin resistance. SPSS v.23 was used for analysis.

Results: Out of 75 PCOS patients, 39(52.0%) belonged to the age group 18-24 years, with a mean age of 24.7±5.2 years. Low serum TSH (≤2.5 mIU/L) was found in 37(49.3%) and high serum TSH (>2.5 mIU/L) in 38(50.7%). Mean serum TSH was 2.49±1.20 mIU/L. Serum TSH was higher in an insulin-resistant group than the non-resistant group (68.9% vs 23.3%) with an odds ratio of 7.276, 95% CI (2.532-20.906). Significant correlation (r=0.542; p=0.001) was found between serum TSH and HOMA-IR.

Conclusion: Serum TSH level is significantly elevated in PCOS patients with insulin resistance compared to those without insulin resistance.

Introduction

Polycystic ovarian syndrome, also known as PCOS, is the most common endocrine disorder in women [1]. It is a hyperandrogenic and metabolic condition that affects between 5 to 10 percent of women of reproductive age [2, 3]. The presence of at least two out of three of the following symptoms, oligo or anovulation, hyperandrogenism, and enlarged polycystic ovaries on ultrasound, is required for a diagnosis of PCOS according to the Rotterdam updated diagnostic criteria [4, 5].

Women with PCOS present manifestations of menstrual and hormonal irregularities culminating in anovulation, infertility, hyperandrogenism, hirsutism, and insulin resistance [6]. The regression of these symptoms is potentially attainable by the reduction of hyperinsulinemia [7]. Several studies have mentioned that women with PCOS are more insulin resistant than would be expected on the basis of their age and body mass index [8]. The mechanism underlying this phenomenon is not fully understood.

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JCHR (2025) 15(2), 1685-1690 | ISSN:2251-6727

Although hyperandrogenemia may play a role, other factors such as thyroid function may also be involved [9, 10].

Women with PCOS have a high prevalence of increased thyroid-stimulating hormone (TSH) levels, which may additionally contribute to their phenotype [11]. PCOS cases with thyroid-stimulating hormone levels above 2.5 mIU/l exhibited a significantly higher body mass index, higher fasting insulin concentrations, and changed insulin resistance indices [12]. According to Mueller et al., PCOS women with TSH levels of 2 mIU/l or above were younger, had a higher BMI, and were more insulin-resistant [6]. Though the genetic background for IR is well established, however multifactorial pathogenesis has been proposed. The pathophysiologic connection between SCH and PCOS is complex. Yet, Singla et al. reported that adiposity, IR, high leptin, and deranged autoimmunity contribute individually and interconnectedly to both disorders mentioned above [13].

Some studies also show controversial findings. Enzevaei et al. and Ravi and Gokaldas et al. could not reveal any significant correlation between serum TSH, serum insulin, and BMI [14, 15]. Rojhani et al. showed there was no statistically significant association between PCOS status and subclinical hypothyroidism [16].

Women with PCOS have a high prevalence of increased TSH levels, with frequent prevalence of concomitant insulin resistance (IR) and metabolic syndrome (MS) [17]. Despite extensive data supporting the role of IR in PCOS, it remains unclear whether the association is causal, propagative, or existential. Based on the relationship between hypothyroidism, insulin resistance, and reproductive disorders, it is conceivable that a possible imbalance in thyroid function may initiate, maintain, or worsen the PCOS features [18].

The present study was undertaken to estimate the frequency distribution of IR and TSH levels among PCOS subjects and to explore the association of TSH and IR in this disorder in pursue of providing more individualized, efficient management plans.

Objective

The objective of this study was to estimate the thyroid-stimulating hormone level in polycystic ovarian syndrome and its association with insulin resistance.

Methodology & Materials

This was a cross-sectional observational study conducted in the Department of Obstetrics and Gynaecology, Dhaka Medical College Hospital (DMCH), Dhaka, Bangladesh. The study was carried out over 12 months, from June 2022 to May 2023. A total of 75 patients were included, all diagnosed with polycystic ovarian syndrome (PCOS) and aged between 18 and 40 years. These participants were selected from those attending the Reproductive Endocrinology and Infertility Outpatient Department (OPD) of DMCH during the study period.

Sample Selection: Participants were selected using a purposive sampling technique based on specific eligibility criteria.

Inclusion Criteria:

1. Women aged 15 to 40 years.
2. Diagnosed with PCOS based on the Rotterdam Criteria (2004).
3. Attending the Reproductive Endocrinology and Infertility OPD at DMCH.

Exclusion Criteria:

1. Women with PCOS already diagnosed with diabetes mellitus.
2. Presence of hyperprolactinemia or any known ovarian/adrenal neoplasm.
3. History of clinical hypothyroidism or previous thyroid disorder treatment.
4. Use of steroids or insulin sensitizers in the six months preceding the study.

Data Collection Procedure: Eligible patients were enrolled after obtaining informed written consent. A structured questionnaire was used to collect socio-demographic details, clinical features, obstetric and menstrual history, and anthropometric measurements. On the third day of a spontaneous or progesterone-induced menstrual cycle, 3 mL of fasting venous blood was collected after a 12-hour overnight fast. Laboratory tests were conducted for fasting blood glucose, fasting insulin, and serum thyroid-stimulating hormone (TSH). Additionally, a 2-hour post-glucose (75g) blood glucose test was performed. All biochemical analyses were conducted at accredited laboratories using standardized methods and equipment to ensure accuracy and reproducibility.

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JCHR (2025) 15(2), 1685-1690 | ISSN:2251-6727

Study Procedure: Each participant's clinical data was recorded following the inclusion/exclusion criteria. Fasting insulin was measured using ARCHITECT insulin assay kits, employing Chemiluminescent Microparticle Immunoassay (CMIA), while fasting plasma glucose was measured via the Glucose Oxidase method (GOD-PAP). Insulin resistance was calculated using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), using the formula:

HOMA-IR = (Fasting glucose \[mmol/L] × Fasting insulin \[μIU/mL]) / 22.5

Participants with HOMA-IR >1.9 were classified as insulin resistant. Serum TSH was measured using Microparticle Enzyme Immunoassay (MEIA), and values >2.5 mIU/L were considered elevated, in line with the American Thyroid Association (ATA) 2017 guidelines. Participants were categorized into insulin-resistant and non-resistant groups based on HOMA-IR, and comparisons were made between these groups regarding TSH levels and other clinical/biochemical variables.

Ethical Considerations: The study protocol was reviewed and approved by the Ethical Review Committee of Dhaka Medical College. Informed written consent was obtained from all participants after explaining the purpose, procedures, potential risks, and benefits of the study. Participation was voluntary, and confidentiality was strictly maintained. Subjects were informed of their right to withdraw at any point without consequence. Laboratory tests were performed only after obtaining necessary departmental permissions.

Statistical Analysis: Data were analyzed using the Statistical Package for Social Sciences (SPSS) version 23.0 (SPSS Inc., Chicago, IL, USA). Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Student's t-test was used for comparing continuous variables, and the Chi-square test was applied for categorical variables. Pearson's correlation coefficient was used to assess the relationship between serum TSH levels and insulin resistance (HOMA-IR). A p-value <0.05 was considered statistically significant.

Results

Table 1: Sociodemographic characteristics of the respondents (n=75)

Socio-demographic characteristics		Group I (N=45)		Group II (N=30)		P value
		N	%	N	%	
Age (years)	18-24	24	53.3	15	50	
	25-34	18	40	14	46.7	
	34-40	3	6.7	1	3.3	
Mean±SD		25.3±5.7		24.6±5.2		0.591
Educational status	Primary	14	31.1	12	40	0.405
	SSC	25	55.6	11	36.7	
	HSC	4	8.9	4	13.3	
	Graduation & above	2	4.4	3	10	
Occupational status	House maker	39	86.7	27	90	0.479
	Others	6	13.3	3	10	
Monthly family income (Taka)	10000-25000	25	55.6	18	60	0.703
	>25000	20	44.4	12	40	

Table 1 shows that age, educational status, occupational status, and monthly family income were statistically not significant between the two groups (p>0.05).

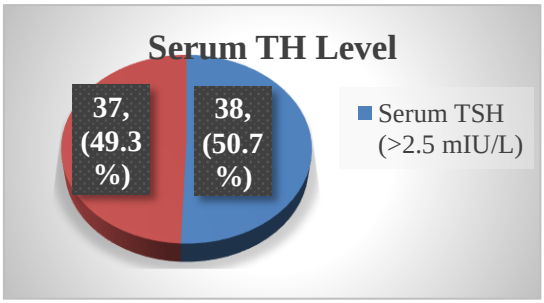

Figure 1: Distribution of the study patients according to serum TSH (n=75).

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JCHR (2025) 15(2), 1685-1690 | ISSN:2251-6727

Figure 1 reveals that serum TSH ≤2.5 mIU/L was found in 37(49.3%) patients and serum TSH >2.5 mIU/L was seen in 38(50.7%) women. The mean serum TSH value was 2.49±1.20 mIU/L.

Table 2: Comparison of mean serum TSH level with insulin-resistant and nonresistant groups (n=75)

Mean Serum TSH	Group I (N=45)	Group II (N=30)	P value
Serum TSH (mIU/L)	2.97±1.28	1.77±0.54	0.001
Range (min-max)	1.11-8.6	0.69-2.65	

Table 2 presents that the mean serum TSH level was significantly higher in insulin-resistant group than non-resistant group (2.97±1.28 vs 1.77±0.54 mIU/L).

Table 3: Association between cut off value of serum TSH level with insulin resistant (n=75)

Parameter	Group I (N=45)		Group II (N=30)		OR (95% CI)	P value
	n	%	n	%		
Serum TSH (>2.5 mIU/L)	31	68.9	7	23.3	7.276	0.001
Serum TSH (≤2.5 mIU/L)	14	31.1	23	76.7	(2.532-20.906)	

Table 3 exhibits that serum TSH was significantly higher in patients with insulin-resistant group (68.9% vs 23.3an %) with an odds ratio of 7.276, 95% CI (2.532-20.906). This table suggested that the odds of serum TSH above 2.5 mIU/L were 7.267 times higher in insulin-resistant PCOS women than in nonresistant cases.

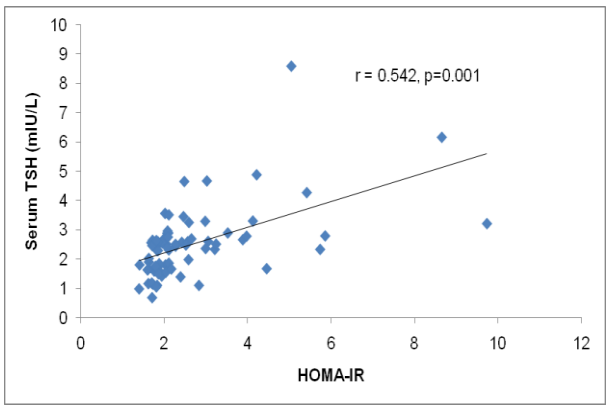

Figure 2: Scatter diagram showing Pearson's correlation between serum TSH and HOMA-IR.

Figure 2 displays that a significant positive correlation (r=0.542; p=0.001) was found between serum TSH and HOMA-IR in PCOS patients. Here, the value of the correlation coefficient (r=0.542) indicates a moderate positive association between the above parameters. From our study result, r2 = 0.293. It means 29.3% of the variability of insulin resistance in PCOS cases can be explained due to high serum TSH value above 2.5mIU/L and vice versa.

Discussion

PCOS is the most common heterogenous polygenic endocrine disorder, usually manifested in the reproductive age group, where hyperandrogenism is considered a key feature. Though the exact pathophysiology of PCOS is not clearly understood, IR is thought to be an important contributory factor. Over the decades, many studies have investigated the correlation between insulin resistance and high TSH levels [14]. Like many other disorders of metabolic syndrome, insulin resistance is thought to be a central phenomenon where many of the other abnormalities converge [19]. Considering contradictory results in this field, this study aimed to estimate the thyroid-stimulating hormone level in polycystic ovarian syndrome and its association with insulin resistance.

In this study we observed that more than half 24(53.3%) patients belonged to age group 18-24 years in group I (Insulin resistant) and 15(50.0%) in group II (Insulin nonresistant) 32(42.7%) belonged to age group 25-34 years and 4(5.3%) belonged to age group 35-40 years. The mean age was 25.3±5.7 years in group I and 24.6±5.2 years in group II. The difference was not statistically significant between the two groups (p>0.05). Similarly, Saha et al. reported that the majority of patients belonged to the age group 21-30 years in both the insulin resistant and insulin sensitive groups; the mean age was 26.03±4.12 years in the insulin resistant group and 26.5±4.07 years in the insulin sensitive group [19].

Regarding TSH level in this study demonstrated that serum TSH (≤2.5 mIU/L) was found in 37(49.3%) and high serum TSH (>2.5 mIU/L) was found in 38(50.7%) patients. The mean serum TSH was 2.49±1.20 mIU/L. Benetti-Pinto et al. revealed that the mean serum TSH level was 2.71±1.57 mIU/L [20], and Lee et al. documented that the mean TSH value was 2.39±1.34 mIU/mL [21]. The findings mentioned above are almost identical to this current study. Yu and

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Wang demonstrated that the mean TSH level was 5.11±22.7 mIU/mL which is different from our mean value [22].

This study revealed that the mean serum TSH level was significantly higher in insulin insulin-resistant group than non-resistant group (2.97±1.28 vs 1.77±0.54 mIU/L). Saha et al. reported that the mean serum TSH level was higher in the insulin resistance group (3.87±3.44 mIU/L) than in the insulin sensitive group (2.29±1.66 mIU/L) [19]. Moreover, Azargoon et al. observed a significant difference in mean serum TSH value between insulin-resistant and nonresistant groups (3.63±2.89 vs 2.93±1.91 mIU/L) [23]. The above-mentioned study findings are almost similar to this study. An identical result was also found in the study of Liu et al., where serum TSH levels in insulin. The resistance group was higher than in the non-insulin resistance group (3.1±1.1 mU/L vs. 2.3±0.8 mU/L) [24].

In this study, the distribution of PCOS cases who had TSH values above 2.5 mIU/L in insulin-resistant and nonresistant groups was 68.9% and 23.3%, respectively, with an odds ratio of 7.276. Saha et al. (2021) found that almost two-thirds (63.9%) of patients had serum TSH level >2.5 mIU/L in insulin insulin-resistant group and 31.3% in insulin insulin-sensitive group, with an odds ratio of 4.19 [19]. The above-mentioned study findings were almost similar to this study. According to Azargoon et al., 24.8% of cases of the insulin-resistant group and 13.6% of the nonresistant group had serum TSH value >2.5 mIU/L, which differs from our percentage [23].

In the present study, we observed that significant positive Pearson's correlation (r=0.542; p=0.001) between serum TSH and HOMA-IR in PCOS women. Saha et al. demonstrated a significant positive correlation between insulin resistance (HOMA-IR) and serum TSH above the threshold of 2.5mIU/L (r= 0.254; p= 0.034) [19]. Similarly, Fatima et al. and Velija-Asimi et al. showed a positive correlation of TSH and fasting insulin and HOMA IR in PCOS subjects [25, 26]. Dittrich et al. revealed that there was a significant positive correlation (r=0.387, p<0.05) between TSH with HOMA-IR in women with TSH ≥2.5 mIU/l [12]. The above-mentioned research findings were almost analogous to this study. On the other hand, Celik et al. observed no significant difference in insulin resistance parameters and fasting insulin levels between the two groups of SCHES and euthyroidism [27]. Ganie et al. showed no association of insulin sensitivity as measured by HOMA-IR with subclinical hypothyroidism [28]. However, they used the threshold HOMA IR value 2.29 for detection of insulin resistance, which is much higher than our cutoff value of 1.9. Naher et al. suggested no significant correlation between insulin sensitivity and TSH among the study subjects (r=0.081, P=0.323) [29]. Above mentioned results differ from our study. The variations could be due to disparity of sample size, variation in subject selection, differences in study designs, confounding variables, and threshold values.

As serum TSH and insulin resistance have a positive association, adequate screening and patient guidance considering insulin resistance, thyroid status allows clinicians to render PCOS patients with more individualized, logical, and effective management schemes.

Limitations of the study

This study has several limitations. First, the study population from a single tertiary care center may limit generalizability to broader populations. Second, the short duration and modest sample size may reduce statistical power and result in robustness. Lastly, financial constraints prevented assessment of important biochemical markers of hyperandrogenism, such as total testosterone, sex hormone-binding globulin (SHBG), and dehydroepiandrosterone (DHEA), which may have impacted the comprehensive evaluation of hyperandrogenism among participants.

Recommendations

Early detection and management of thyroid dysfunction in patients with polycystic ovarian syndrome (PCOS) may improve insulin sensitivity. To validate these findings, future research should involve multicenter trials with a larger sample size and extended duration, including participants with and without PCOS. Additionally, incorporating alternative measures of insulin resistance, such as the Quantitative Insulin Sensitivity Check Index (QUICKI) and the Fasting Glucose to Insulin Ratio (FGIR), could enhance insulin resistance assessment accuracy.

Conclusion

The study clearly demonstrated that serum TSH level is significantly elevated in PCOS patients with insulin resistance compared to PCOS patients without insulin resistance. Based on the study results, a significant positive correlation was found between high TSH (>2.5 mIU/L) value with HOMA-IR in women with PCOS. So, women

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JCHR (2025) 15(2), 1685-1690 | ISSN:2251-6727

with PCOS should be candidates for screening to identify any disturbances in thyroid function and any changes in metabolic parameters, as well as IR indices.

Financial support and sponsorship

No funding sources.

Conflicts of interest

There are no conflicts of interest.

Ethical approval

The study was approved by the Institutional Ethics Committee.

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