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Original Article

Study on Correlation between Endocrine and Metabolic Parameters in Polycystic Ovary syndrome and Thyroid-Stimulating Hormone: Should Normal Thyroid-Stimulating Hormone Level be Redefined?

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Abstract

Introduction: Polycystic ovary syndrome (PCOS) is a fairly common disorder of women in the reproductive age. Thyroid-stimulating hormone (TSH) affects endocrine and metabolic parameters in PCOS are claimed by some researchers**.**

Purposes: This study was carried out to clarify if there are correlations with endocrine and metabolic parameters in PCOS with TSH.

Methods: A total of 100 PCOS patients of age group between 20 and 35 years and body mass index (BMI) 18.5-30kg/m2 were recruited. Forty (*n*=40) healthy female subjects matched for age and BMI were also enrolled as controls. Cases were divided into two groups: Group I (TSH<2.1) and Group II (TSH≥2.1). All variables were again compared between two groups of PCOS cases.

Results: The mean serum BMI, TSH, fasting glucose, fasting insulin, homeostatic model assessment-insulin resistance, total cholesterol, low-density lipoprotein, triglyceride, and very low-density lipoprotein levels were significantly higher in Group II PCOS than Group I PCOS. Moreover, the luteinizing hormone (LH) and LH/follicle-stimulating hormone (FSH) levels were significantly lower in the patients with Group II PCOS than Group I PCOS (*P*< 0.05). Serum FSH, total testosterone, and high-density lipoprotein were unaltered in both groups.

Conclusion: TSH altered endocrine and metabolic parameters in PCOS.

Key words: Polycystic ovary syndrome, Thyroid stimulating hormone, Insulin resistance

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a fairly common disorder of women in the reproductive age. It is characterized by hyperandrogenism and chronic anovulation.[1,2] It affects 5%–6% of women during the reproductive age group.[3]

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Obesity, insulin resistance (IR), and dyslipidemia, which may predispose patients to metabolic syndrome, are common in PCOS.[4] Hyperandrogenism usually suggested by the presence of hirsutism (occurs in approximately 80% of PCOS women) and can be documented by measuring androgen levels in the blood. Free testosterone is the most frequently elevated steroid in the blood in PCOS. Circulating levels of total testosterone, androstenedione, and dehydroepiandrosterone are also elevated.[5] Chronic anovulation may present as irregular menstrual periods or amenorrhea. It is not essential to document anovulation by ultrasonography or progesterone measurements in the presence of a clear clinical history. In fact, PCOS occurs in 85%–90% of women with oligomenorrhea and in

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30%–40% of women with amenorrhea.[6] Many women with PCOS (between 38% and 88%) have been found to be overweight and obese, and the studies reported that obese PCOS women have more severe hyperandrogenism and related clinical features (such as hirsutism, menstrual abnormalities, and anovulation) than normal weight PCOS women.[7] It is associated with IR. IR, defined as a metabolic state characterized by a decrease in cellular ability to respond to insulin signaling, appears to be an essential pathophysiologic mechanism in the development of all metabolic complications of PCOS.[8] It has been suggested by some investigators that IR is present in all PCOS patients.[9] However, others have reported that IR is not a universal finding, but rather is present in no more than 40%–70% of PCOS patients.[10]

Hypothyroidism also causes similar problems that are occurred in PCOS like cystic ovaries, ovulatory dysfunction, and anovulation.[11] Hypothyroidism has been shown to cause many metabolic derangements, such as decrease in glucose disposal or its uptake by muscles or adipose tissues in response to insulin, increase in the level of sex hormone-binding globulin, weight gain, and hyperlipidemia, all of which can lead to IR.[12,13] In the presence of hypothyroidism, ovarian morphology becomes polycystic. Hence, thyroid disorders are one of the exclusion criteria before making a diagnosis of PCOS in any women. Cystic changes with raised ovarian mass have also been reported in hypothyroidism. Two facts make the picture more interesting, first that both have different etiopathology and second that reportedly thyroid disorders are more common in PCOS subjects.[12,14]

Recently, in a study by Ganie *et al.* confirmed that the chronic lymphocytic thyroiditis (CLT) girls had higher body mass index (BMI), waist circumference, and systolic blood pressure ($P< 0.001$) than non-CLT age-matched control.[15] An another study conducted in young women with PCOS concluded that there was no difference in two groups (with or without subclinical hypothyroidism) with respect to BMI, waist circumference or Ferriman–Gallwey score.[16] Hence, the results were conflicting, but the question is whether normal level of thyroid hormone, particularly thyroid-stimulating hormone (TSH) has any relation with endocrine and metabolic parameters in PCOS.

Therefore, this study was carried out to clarify if there are correlations with endocrine and metabolic parameters in PCOS with TSH.

MATERIALS AND METHODS

Study Design

A community-based case control study was carried out from 2007 to 2011 among women aged 20–35 years who were permanent residents of West Bengal. PCOS patients with BMI (18.5–30kg/m^2) were recruited from the OPD clinics of the Department of Gynecology in Institute of Postgraduate Medical Education and Research (IPGME&R), Kolkata. This clinical study was approved by the Institutional Ethics Committee (IPGME&R) Kolkata. All of the participants signed informed consent to be included in the study.

Sample Size

Totally 100 PCOS patients between age group between 20 and 35 years were recruited. Healthy age-matched controls (n=40) women without PCOS and matched for age BMI were recruited.

Selection of Cases and Control f rom Sources

Operational definition

Amenorrhea

Amenorrhea was defined as the absence of periods for at least 3 of the previous cycle in patient who had been menstruating previously.

Oligomenorrhea

Oligomenorrhea was considered when length of menstrual cycle was greater than 35 days. It is indirect marker for anovulation in absence of any hormonal evidence.

Normal TSH level – 0.4–4.0 μ/L

Homeostatic model assessment-insulin resistance (HOMA-IR)

Insulin resistance was measured with Homeostatic Model Assessment index (HOMA) = [fasting serum insulin level (μU/mL) x Fasting plasma glucose (mg/dL)]/405. A value more than two is insulin resistant.

Clinical hyperandrogenism

Hyperandrogenism was defined as presence of hirsutism (modified Ferriman-Gallwey score >5) and or severe acne.

Polycystic ovaries

Polycystic ovaries was defined having ovarian follicles 2–9 mm in diameter and ≥12 in number or ovarian volume ≥10 cm3 in one or both ovaries on trans-abdominal pelvic ultrasonography (USG). There should be no dominant follicle with size greater than 10 mm in diameter.

Participants selected were undergone three stages of operation.

Stage I: Questionnaire:
Administration of the questionnaire "probable cases" and "probable controls" was identified during the cross-sectional survey.

A "probable case:" A "probable case" was defined as a woman with symptoms suggestive of PCOS (i.e., oligo/amenorrhea/or clinical features of hyperandrogenism) as defined above.

A "probable control": A "probable control" was defined as a woman with regular menses and no clinical features of PCOS. Probable control group matched for age and BMI of probable PCOS cases were selected. A detailed history of taking drugs was elucidated and all participants who were on oral contraceptive pills, oral hypoglycemic agents and anti-thyroid drugs were excluded from study. They were then selected for Stage 2 examination.

Stage 2: Clinical examination and biochemical investigations

Selected women were examined for the presence of hirsutism, acne, or alopecia. Hirsutism was routinely graded by two physicians independently using the common modified Ferriman–Gallwey (FG) score. If the FG score differed by more than two, reevaluation by a third physician was done and median values were used. Nine areas were examined - upper lip, chin, chest, upper abdomen, lower abdomen, upper back, lower back, thighs, and upper arms. Each area is scored 0–4, resulting maximum score 36. Hirsutism was diagnosed when a score above five was evaluated.

Biochemical Investigations

Venous blood (5 ml) was drawn from both probable cases and probable controls. Blood samples were taken during the 3rd day of the menstrual cycle. Hemolyzed sera were discarded. Serum total testosterone was measured to diagnose biochemical evidence of androgen excess or hyperandrogenemia. Hyperandrogenemia was diagnosed when serum total testosterone level was greater than 55ng/dl Upper normal level of serum total testosterone level was 55ng/dl in our laboratory. Kit was supplied by Radio-pharmaceutical and isotope technology, BARC, Mumbai.

Stage 3: Ultrasound scanning

Pelvic ultrasound scanning on women identified as probable cases and probable controls. Pelvic ultrasound scanning was done on subjects to note the presence of polycystic ovaries.

Inclusion criteria of cases

The diagnostic criteria for PCOS were based on the unified standards formulated by the Rotterdam International Conference in 2003.[17] Patients with any two of the following three conditions were diagnosed with PCOS: (1) infrequent ovulation or anovulation; (2) hyperandrogenism or clinical manifestations of high blood androgen; and (3) polycystic ovaries on USG–multiple small follicles (>10–12) and (2–9 mm in diameter) tightly spaced along the periphery of the ovary.

Exclusion criteria of cases such as thyroid dysfunction, hyperprolactinemia, congenital adrenal hyperplasia, androgen secreting tumors, Cushing syndrome, and other diseases were included in the study. Patients were on organizational culture profile (OCP), hypolipidemic, and hypoglycemic drugs and were excluded from the study.

Inclusion criteria of controls

Patients in the control groups exhibited normal menstruation, no clinical or biochemical signs of hyperandrogenism, normal ovaries as defined by ultrasonic examination, and no family history of PCOS and they should be age and BMI matched with cases.

Participants on OCP or conceived were excluded from the study. Chronic kidney disease liver disease cancer patients were also excluded from the study.

A total of 100 PCOS patients between age group between 20 and 35 years were recruited. Healthy age-matched controls (n=40) women without PCOS and matched age, BMI recruited.

Other Biochemical Parameters

Blood samples were taken during the 3rd day of the menstrual cycle. Hemolyzed sera were discarded. All assays were completed within 3 days. Serum glucose estimation done by glucose oxidase-peroxidase with kit manufactured by Monozyme India Limited, Secunderabad, insulin by radioimmunoassay with module supplied by Radiopharmaceutical and Isotope Technology, Mumbai. Total serum follicle-stimulating hormone (FSH) and luteinizing hormone (LH) were also measured. Serum TSH, total cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL), very low-density lipoprotein (VLDL), and triglyceride (TG) were also measured.

Statistical Analysis

Statistical analysis was done by descriptive statistics in statistical analysis were carried out using XLSTAT 2018. Descriptive statistics are presented as mean ± standard deviation for normally-distributed variables. Non-normally distributed variables were compared by the Kolmogorov Smirnov test. Student's t-test was used to compare variables with normal distribution and $P < 0.05$ was considered significant. The degree of association between continuous variables was calculated by the Pearson correlation coefficient.

Tables 1: Basic parameters

Basic parameters	Cases=100		Controls *n*=40	
	Range	Mean	Range	Mean
Age years	20–35	26.42	20–35 years	26.89
BMI kg/m^2	18.5–30	23.49	18.5–30	22.46

BMI: Body mass index

RESULTS

Hormonal and metabolic parameters were screened in the patients with PCOS and in the healthy control subjects.

Basic Parameters [Table 1]

We studied 100 patients with PCOS (mean age 26.42 ± 4.58 years, range 20–35 years; BMI, 23.39± 3.22 kg/m^2) and 40 years age and BMI-matched healthy controls (mean age 26.89±3.91 years, range 20–35 years; BMI, 22.46± 2.6 kg/m^2).

Difference between mean age and BMI of 75 obese PCOS cases and mean age obese controls is not statistically significant.

Comparing Parameters between in Women with PCOS and Controls

The mean serum TSH, LH, LH/FSH, fasting insulin, HOMA-IR, total testosterone, TG, and VLDL levels were significantly higher, and the HDL levels were significantly lower in the patients with PCOS than controls (P< 0.05). Serum FSH and glucose were total cholesterol unaltered in both groups [Table 2].

Determination of Cutoff Value of TSH

TSH is positively correlated with HOMA-IR (r=0.525, P<0.0001). The correlation between TSH and IR was shown in Figure 1.

The association between TSH and IR, evaluated using the ROC curve, showed a cutoff value for TSH of 2.1mIU/L. At this value, sensitivity of 80.9% and specificity of 61.8% for diagnosis of IR using HOMA-IR (value above 2 is insulin resistant) [Figure 2].

Based on cutoff value, PCOS cases were divided into two groups: Group I (TSH< 2.1) and Group II (TSH≥ 2.1). All variable were again compared between two groups of PCOS case as shown in Table 3.

All Variable between Two Groups of PCOS Cases

The mean serum BMI, TSH, fasting glucose, fasting insulin, HOMA-IR, total cholesterol, LDL, TG, and VLDL levels were significantly higher in Group II PCOS than Group I PCOS. Moreover, the LH and LH/FSH levels were significantly lower in the patients with Group II PCOS than Group I PCOS (P< 0.05). Serum FSH, total testosterone, and HDL were unaltered in both groups.

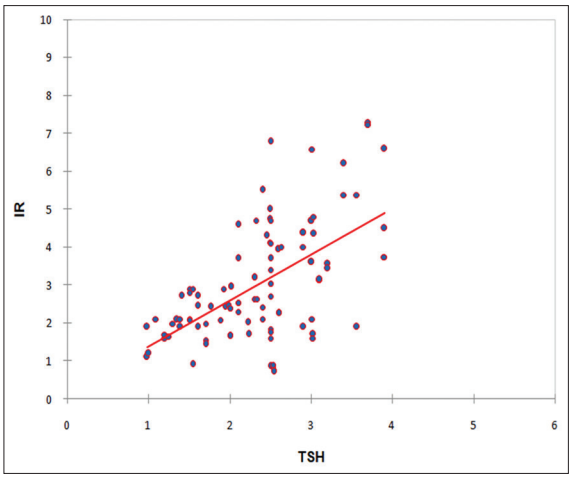

Figure 1: The correlation between thyroid-stimulating hormone and insulin resistance (IR) (homeostatic model assessment IR)

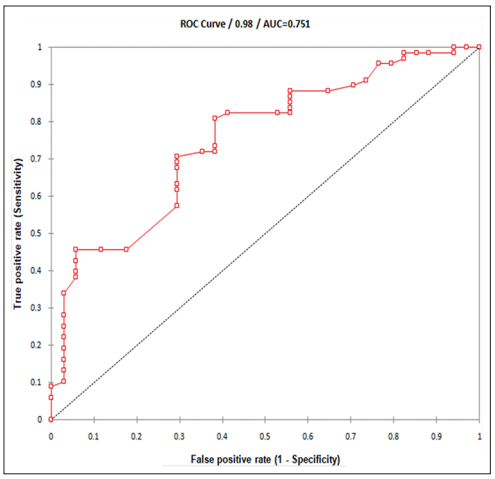

Figure 2: Receiver operating characteristic curve to evaluate the association between thyroid-stimulating hormone and IR

The Correlations between Biochemical and Hormonal Parameters with TSH

These were shown in Table 4.

A significant positive correlation was found between the TSH with glucose (r=0.235, P=0.017), insulin (r=0.492, P=0.001), HOMA-IR (r=0.525,<0.001), testosterone (r=0.298, P=0.02), cholesterol (r=0.232, P=0.019), LDL (r=0.199, P=0.045), TG (r=0.310, P=0.002), and VLDL (r=0.305, P=0.02). Negative correlation was found between TSH with LH/FSH (r= −0.249, P=0.012) and LH(r= −0.363, P=0.01). However, no correlation were found between TSH with FSH (r=0.28, P=0.00) and HDL (r=0.106, P=0.289).

DISCUSSION

This study showed that TSH levels >2.1 mIU/L being associated with IR and other hormonal and metabolic variables evaluated were also altered at that

Table 2: Comparing parameters between women with PCOS cases and controls

Parameters	PCOS cases *n*=100	PCOS controls *n*=40	*P* value	Significant or not
Age	26.43±4.58	26.89±3.92	0.491	Not significant
BMI kg/m^2	23.49±3.228	22.46±2.6	0.078	Not significant
TSH	2.37±0.708	1.6±0.65	0.001	Significant
LH, μ/ml	11.73± ±5.99	8.61±3.86	0.002	Significant
FSH,μ/ml	4.42±1.622	4.56±1.99	0.621	Not significant
LH/FSH	2.2±0.76	0.92±0.287	<0.001	Significant
Glucose mg/dl	87.24±9.51	88.98±11.04	0.353	Not significant
Insulinμ/ml	15.02±7.28	8.97±5.3	0.001	Significant
HOMA-IR	3.7±1.49	1.98±1.3	<0.001	Significant
Total testosterone ng/dl	85.99±41.31	8.91±5.41	0.0001	significant
Cholesterol mg/dl	175.79±17.77	170.64±21.89	0.972	Not significant
LDL mg/dl	105.23±16.26	104.71±21.38	0.88	Not significant
HDL mg/dl	42.4±4.65	47.43±4.5	0.0001	Significant
TG mg/dl	134.66±43.15	117.36±28.32	0.022	Significant
VLDL mg/dl	27.31±8.9	23.48±5.17	0.01	Significant

n: Number. BMI: Body mass index, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, VLDL: Very low-density lipoprotein, TG: Triglyceride, HOMA-IR: Homeostatic model assessment-insulin resistance, LH: Luteinizing hormone, FSH: Follicle-stimulating hormone, TSH: Thyroid-stimulating hormone, PCOS: Polycystic ovary syndrome

Table 3. Variable compared between two groups of PCOS cases

Parameters	PCOS Group I *n*=32	PCOS Group II *n*=68	*P* value	Significant or not
Age	25±5.47	26±3.98	0.063	Not significant
BMI kg/m^2	21.99±2.44	28.68±4.69	<0.001	Significant
TSH	1.65±0.34	2.82±0.466	0.001	Significant
LH, μ/ml	7.22±3.76	12.53±4.87	0.002	Significant
FSH, μ/ml	4.6±1.778	4.28±1.52	0.33	Not significant
LH/FSH	1.99±0.	542.59±0.9	<0.001	Significant
Glucose mg/dl	83.92±7.1	89.17±10.26	0.007	Significant
Insulin μ/ml	12.86±4.42	16.6±8.25	0.012	Significant
HOMA-IR	2.64±1.01	3.58±1.65	0.02	Significant
Testosterone ng/dl	77.56±31.99	81.8±45.36	0.228	Not significant
Cholesterol mg/dl	164.66±22.97	183.29±19.67	<0.001	Significant
LDL mg/dl	97.68±14.5	110.404±15.4	<0.001	Significant
HDL mg/dl	43.29±5.26	41.9±4.24	0.153	Not significant
TG mg/dl	116.58±40.74	146.94±40.9	0.001	Significant
VLDL mg/dl	23.31±8.15	29.57±8.6	0.001	Significant

BMI: Body mass index, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, VLDL: Very low-density lipoprotein, TG: Triglyceride, HOMA-IR: Homeostatic model assessment-insulin resistance, LH: Luteinizing hormone, FSH: Follicle-stimulating hormone, TSH: Thyroid-stimulating hormone, PCOS: Polycystic ovary syndrome

Table 4: Correlations between biochemical and hormonal parameters with TSH

Parameters	PCOS cases *n*=100 *r* value	*P* value
BMI	0.419	<0.0001
LH/FSH	−0.249	0.012
LH	−0.363	0.01
FSH	0.28	0.00
Glucose	0.235	0.017
Insulin	0.492	0.001
HOMA-IR	0.525	<0.001
Testosterone	0.298	0.02
Cholesterol	0.232	0.019
LDL	0.199	0.045
HDL	0.106	0.289
TG	0.310	0.002
VLDL	0.305	0.02

BMI: Body mass index, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, VLDL: Very low-density lipoprotein, TG: Triglyceride, HOMA-IR: Homeostatic model assessment-insulin resistance, LH: Luteinizing hormone, FSH: Follicle-stimulating hormone, TSH: Thyroid-stimulating hormone, PCOS: Polycystic ovary syndrome

defined cutoff value. In our study, we found following abnormalities.

Abnormalities Related to Glucose Concentration and Insulin Resistant

We found a linear correlation between TSH and fasting glucose concentrations and there was difference between two groups of PCOS but no differences between PCOS cases and controls. A study in India did not find any relation with serum TSH and glycemia.[18] Mueller *et al.* found that in women with PCOS, TSH ≥2.0 μIU/mL is associated with IR independently of BMI and age, and hypothyroid disturbances and elevated TSH levels are common findings among women with PCOS.[19] PCOS women show a high prevalence of metabolic disturbances including IR, dyslipidemia, and chronic low-grade inflammation.[20]

These findings bring to mind the question that does the hypothyroidism intensity have additional impact over IR? And is this IR in fact due to changes in thyroid hormones.

Abnormalities Related to Lipid Profile

In our study, we concluded gross alteration in all lipid parameters except HDL between two groups of PCOS cases. No alteration in HDL between two groups of PCOS and we could not explained it. Conflicting results were there on relationship among lipid parameters and SCH. Tuzca *et al.* concluded higher LDL in the SCH with PCOS and no changes in TG and HDL when compared with controls.[2,3] Ganie *et al.* concluded high triglyceride in the SCH group with PCOS with the control group[18,21,22] comparing to the control group.[23,24] Some studies claimed that no alteration in lipid levels in patients with PCOS and SCH compared to euthyroid case. Larger multi-centric studies are needed.

Gonadotropin Abnormalities

In our study, LH is lower in Group II PCOS (TSH ≥2.1) than Group I PCOS. No data were available on this finding. It might me due to effect of BMI.

A possibility arises that some obesity-associated factor may suppress GnRH pulse amplitude or pituitary LH responsiveness. Leptin acting at the level of the pituitary or hypothalamus may dampen LH secretion in obese PCOS individuals. Some authors have proposed this hypothesis, like La Zovic *et al.* noticed that there is significant positive correlation between LH and serum leptin level in non-obese PCOS negative correlation between these serum LH and leptin in obese PCOS cases.[25] We can assume this study that leptin modulates hypothalamic-pituitary-gonadal axis, and attenuation of serum LH obese PCOS is due to leptin resistance state. A second possibility is decreased LH secretion in obese PCOS is due to beta-endorphin.[26]

CONCLUSION

- TSH altered endocrine and metabolic parameters in PCOS
- This study showed that TSH levels >2.1 mIU/L being associated with IR and other hormonal and metabolic variables evaluated were also altered at that defined cutoff value.
- Our study also offers the possibility that the potential use of TSH as a predictor that can be involved in PCOS-related metabolic disturbances.

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