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Original Research Article

Thyroid dysfunction and autoimmune thyroid disease in women with polycystic ovarian syndrome: a case-control study

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ABSTRACT

Background: Polycystic ovarian syndrome (PCOS) is one of the most prevalent endocrine disorders affecting women of reproductive age. It is a multifactorial condition characterized by chronic anovulation, hyperandrogenism, and polycystic ovarian morphology. Emerging evidence suggests a link between PCOS and thyroid dysfunction, indicating shared pathophysiological and possibly autoimmune mechanisms. The main objective was to determine the prevalence of thyroid dysfunction and thyroid autoimmunity in women with PCOS and evaluate their association with clinical, metabolic, and endocrine parameters.
Methods: This hospital-based case–control study consisted of 126 women, comprising 63 cases diagnosed with PCOS and 63 age- and parity-matched healthy controls. Clinical, biochemical, and hormonal parameters were evaluated, including thyroid function tests (TSH and FT4), anti-thyroid peroxidase antibodies (anti-TPO), lipid profile, glucose metabolism, and gonadotropin levels. Data were analysed using SPSS version 20, with $p<0.05$ considered statistically significant.
Results: Thyroid dysfunction was significantly higher among PCOS women (26.98%) than controls (7.9%) ($p=0.015$). Subclinical hypothyroidism was the most frequent thyroid disorder (23.8% vs 7.9%). Anti-TPO antibody positivity was observed in 15.9% of PCOS women compared to 1.6% of controls ($p=0.005$). PCOS participants had higher BMI, triglycerides, total cholesterol, fasting glucose, HbA1c, LH and LH/FSH ratio, and free testosterone levels than controls.
Conclusions: Thyroid dysfunction and autoimmunity are significantly more prevalent among women with PCOS and appear to contribute to the metabolic and hormonal derangements characteristic of the syndrome. Routine screening for thyroid function and autoimmunity should be integral to the clinical evaluation and management of PCOS.

Keywords: Polycystic ovary syndrome, Thyroid dysfunction, Autoimmunity, Subclinical hypothyroidism, Anti-TPO antibodies

INTRODUCTION

Polycystic ovarian syndrome (PCOS) is a multifactorial endocrine condition affecting approximately 15-20% of women in their reproductive years.[1] It is a heterogenous disorder characterized by ovulatory dysfunction, clinical or biochemical features of hyperandrogenism, and polycystic ovarian morphology, as defined by the Rotterdam diagnostic criteria.[2] In addition to reproductive manifestations, PCOS is strongly linked with several metabolic disturbances, including insulin resistance, obesity, dyslipidemia, and abnormalities in glucose metabolism.

Thyroid disorders, particularly hypothyroidism, often present with symptoms that overlap with those of PCOS, such as menstrual irregularities, infertility, and increased body weight.[3] Autoimmune thyroiditis represents the most frequent cause of hypothyroidism in young women and has been reported to occur more commonly in individuals with PCOS. Increasing evidence suggests that both conditions may share common pathogenic mechanisms involving

genetic susceptibility, hormonal imbalance, insulin resistance, and immune dysregulation. Factors such as estrogen excess, thyroid-stimulating hormone receptor antibodies, and metabolic dysfunction may play an important role in linking PCOS with thyroid abnormalities.[4]

In view of these shared clinical and biological characteristics, evaluation of thyroid function and thyroid autoimmunity in women with PCOS is clinically important. Timely identification and management of thyroid dysfunction may contribute to improved reproductive health as well as better metabolic and cardiovascular outcomes. Hence, the present study was designed to assess the prevalence of thyroid dysfunction and autoimmune thyroid disease in women with PCOS and to analyze their association with clinical, metabolic, and hormonal parameters.

METHODS

This was a hospital-based case-control study conducted at the Department of Obstetrics and Gynaecology, Kasturba Hospital, University of Delhi, between August 2023 and October 2024. It included a total of 126 women, consisting of 63 cases diagnosed with PCOS and 63 age- and parity-matched healthy controls. The diagnosis of PCOS was confirmed according to the Rotterdam criteria (2003), which require the presence of at least two of the following: Oligo-ovulation or anovulation, evidence of hyperandrogenism by clinical and/or biochemical features and polycystic ovarian morphology on ultrasonography.

The control group comprised women attending the hospital for routine health evaluation, with regular menstrual cycles and no history of infertility or endocrine disorders.

Women with previously diagnosed thyroid disease, diabetes mellitus, Cushing's syndrome, hyperprolactinemia, congenital adrenal hyperplasia, or those receiving medications known to influence glucose or lipid metabolism were excluded from the study.

All participants underwent detailed clinical evaluation, including assessment of menstrual history, body mass index (BMI), waist-hip ratio (WHR), presence of acne, hirsutism, and acanthosis nigricans. Laboratory investigations included fasting plasma glucose, HbA1c, lipid profile, thyroid function tests (TSH and free T4), anti-thyroid peroxidase (anti-TPO) antibodies, luteinizing hormone (LH), follicle-stimulating hormone (FSH), prolactin, and free testosterone levels. Subclinical hypothyroidism was defined as a serum TSH level greater than 4.2 mIU/L in the presence of normal free T4 levels.

Statistical analysis was performed using SPSS software version 20.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean±standard deviation and compared using the independent t test. Categorical variables were analyzed using the Chi-square test or Fisher's exact test where applicable. A p value of less than 0.05 was considered statistically significant.

RESULTS

Baseline characteristics

The mean age of PCOS women and controls was comparable (25.82±4.85 vs 25.33±4.36 years; p>0.05), with most participants falling in 21-30 age group. Women with PCOS had a significantly higher mean BMI (26.42±4.20 kg/m²) compared to controls (23.68±1.33 kg/m²; p<0.001). Similarly, the mean WHR was greater among PCOS women (0.86±0.06 vs. 0.80±0.04; p<0.001). Menstrual irregularity were reported by 74.6% of women with PCOS in contrast to 12.7% of controls (p<0.001) (Table 1).

Thyroid function and autoimmunity

Thyroid dysfunction was observed significantly more often in women with PCOS (26.98%) compared to the control group (7.9%) (p=0.015). Subclinical hypothyroidism was the most common abnormality, affecting 23.8% of PCOS patients versus 7.9% of controls. Overt hypothyroidism was identified in 3.2% of women with PCOS and was absent in the control group (Table 2).

Anti-TPO antibody positivity was detected in 15.9% of women with PCOS, whereas only 1.6% of controls tested positive, indicating a significantly higher prevalence of autoimmune thyroiditis in the PCOS group (p=0.005) (Table 2 and Figure 1).

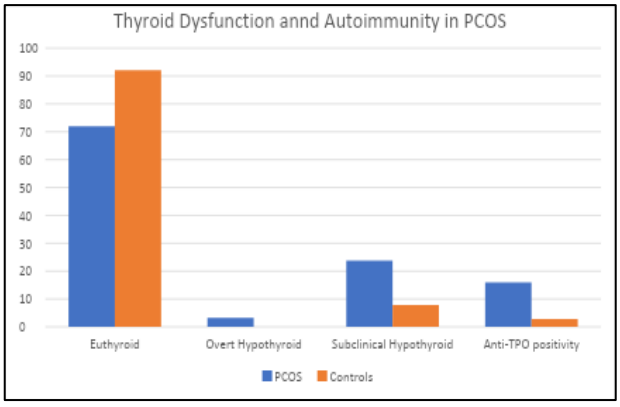

Figure 1: Thyroid function and autoimmunity in PCOS and controls.

Clinical, metabolic, and hormonal correlates

Comparison between euthyroid and hypothyroid women within the PCOS group revealed that infertility, menstrual disturbances, weight gain, hirsutism, and polycystic ovarian morphology were more frequent among those with thyroid dysfunction; however, these differences did not reach statistical significance (p>0.05) (Table 3).

Metabolic abnormalities, including higher BMI, increased WHR, and dyslipidemia, were more commonly observed in PCOS women with thyroid dysfunction, although these differences were also not statistically significant (Table 4).

Thyroid status did not significantly influence hormonal parameters/glycemic control, as LH, FSH, free testosterone, and HbA1c levels were comparable between euthyroid and hypothyroid PCOS patients.

Table 1: Demographic clinical, and hormonal characteristics.

Parameters	PCOS, (n=63)	Controls, (n=63)	P value
Mean age (in years)	25.82±4.85	25.33±4.36	0.30
BMI (kg/m²)	26.42±4.20	23.68±1.33	<0.001
WHR	0.86±0.06	0.80±0.04	<0.001
Menstrual irregularity (%)	74.6	12.7	<0.001
Fasting glucose (mg/dl)	93.83±14.20	89.06±4.43	0.012
HbA1c (Mean)	5.26±0.66	4.94±0.19	0.001
Triglycerides (mg/dl)	128.09±45.50	111.35±20.92	0.009
Total cholesterol (mg/dl)	169.96±31.26	159.65±17.76	0.025
LH (mIU/ml)	8.86±5.45	2.70±0.52	<0.001
FSH (mIU/ml)	5.60±1.60	3.21±0.75	<0.001
FSH: LH ratio	0.85±0.52	2.71±0.54	<0.001
Free testosterone (ng/dl)	3.76±8.04	0.30±0.11	<0.001
Prolactin (ng/ml)	15.38±5.2	11.87±2.78	0.001

Table 2: Thyroid function and autoimmunity.

Parameters	PCOS (%)	Controls (%)	P value
Euthyroid	72.02	92.1	
Overt hypothyroid	3.2	0	0.015
Subclinical hypothyroidism	23.8	7.9	
Mean TSH	3.31±2.15	2.34±0.94	0.001
Anti-TPO positivity	15.9	1.6	0.005

Table 3: Comparison of clinical parameters in different thyroid status within the cases.

Parameters	Euthyroid		Hypothyroid (SCH+Overt)		P value
	n=46	%	n=17	%	
HMB	5	10.9	2	11.8	0.920
Infrequent cycles	42	91.3	17	100	0.209
Weight gain	29	63	9	52.9	0.467
Acne	9	19.6	2	11.8	0.469
Infertility	29	63	14	82.4	0.144
Hirsutism	15	32.6	6	35.3	0.841
PCOM	43	93.5	16	94.1	0.926

Table 4: Comparison of metabolic and hormonal parameters in different thyroid status within the cases.

Parameters	Euthyroid	Hypothyroid	P value
Mean BMI	26.80±4.28	25.38±3.92	0.235
Mean HDL (mg/dl)	44.42±7.37	49.11±14.67	0.098
Mean triglycerides (mg/dl)	123.89±36.53	139.47±63.88	0.231
Mean cholesterol (mg/dl)	167.75±27.08	175.94±40.90	0.360
Mean HbA1c (%)	5.30±0.70	5.14±0.52	0.387
Mean FSH (mIU/ml)	5.63±1.50	5.55±1.90	0.863
Mean LH (mIU/ml)	9.10±5.83	8.20±4.35	0.564
Mean FSH: LH	0.84±0.50	0.86±0.58	0.883
Mean prolactin (ng/ml)	14.81±5.40	16.92±4.43	0.156
Mean testosterone (ng/dl)	3.40±7.37	4.75±9.80	0.558

DISCUSSION

The present study demonstrated a significant association between PCOS and thyroid dysfunction with thyroid abnormalities being considerably more common among women with PCOS than controls (26.98% vs 7.9%; p=0.015). Subclinical hypothyroidism (SCH) emerged as the most frequent thyroid abnormality (23.8% vs 7.9%). Similar findings have been reported by Sinha et al who observed SCH in 22.5% of women with PCOS compared to 8.75% of controls.[5] Likewise, Raj et al reported significantly higher mean TSH levels in women with PCOS (5.01±1.02 mIU/l) than controls (3.42±0.76 mIU/l; p<0.00001), with SCH being significantly more prevalent in the PCOS group (43.5% vs 20.5%; p<0.00001).[6] These findings strengthen the evidence supporting a close relationship between PCOS and thyroid dysfunction.

The significantly higher prevalence of anti-TPO antibody positivity among women with PCOS (15.9% vs 1.6%) further supports the hypothesis of an autoimmune link between these two disorders. Garelli et al reported anti-TPO positivity in 23% of women with PCOS compared to 6% of controls.[7] Similarly, Janssen et al observed elevated thyro-peroxidase (TPO) or thyroglobulin (TG) antibodies in 26.9% of women with PCOS compared to 8.3% of controls (p<0.001).[8]

Several common pathophysiological mechanisms may explain this association. Insulin resistance, obesity, and chronic low-grade inflammation are common features of both disorders. Increased adiposity leads to elevated leptin levels, which may stimulate hypothalamic thyrotropin-releasing hormone secretion, thereby increasing TSH levels and contributing to thyroid hyperplasia. In addition, the oestrogen-dominant hormonal milieu in PCOS may enhance immune activation through B-cell stimulation and cytokine-mediated inflammatory pathways, predisposing these women to autoimmune thyroid disease.[9]

Women with PCOS and thyroid dysfunction in the present study exhibited higher BMI, fasting glucose, triglycerides, and total cholesterol, indicating an increased metabolic risk profile. Although the differences were not statistically significant, the mean BMI was highest in women with overt hypothyroidism (28.88±5.21 kg/m²), followed by euthyroid PCOS women (26.80±4.28 kg/m²) and women with SCH (24.91±3.69 kg/m²); (p=0.235, p=0.193).

Yu et al similarly reported significantly higher BMI among SCH-PCOS women compared to euthyroid PCOS women (32.70±4.9 kg/m² vs 30.9±15.1 kg/m²). Both hypothyroidism and PCOS independently contribute to weight gain; hypothyroidism through reduced basal metabolic rate and fluid retention, and PCOS through insulin resistance and abnormal fat distribution.

Thyroid hormone deficiency also adversely affects lipid metabolism by reducing triglyceride clearance and increasing cholesterol synthesis. Concurrently, insulin resistance in PCOS enhances very-low-density lipoprotein production, thereby aggravating hypertriglyceridemia. Although higher lipid levels were observed among hypothyroid women with PCOS in the present study, the differences did not reach statistical significance. Elevated triglyceride levels were noted in 41.2% of hypothyroid women compared with 19.6% of euthyroid women (p=0.08). Among thyroid dysfunction subtypes, raised triglycerides were observed in 60% of women with SCH and 50% of those with overt hypothyroidism. Similar findings were reported by Celik et al who demonstrated significant dyslipidaemia in women with PCOS and SCH, particularly involving triglycerides.[11] The mean triglyceride level in SCH-PCOS women was significantly higher than in euthyroid PCOS women (143.26±99.86 mg/dl vs 88.56±37.56 mg/dl). Comparable observations were also made by Pergialiotis et al who noted greater insulin resistance and dyslipidaemia in hypothyroid women with PCOS.[12]

The FSH:LH ratio, which is typically reduced in women with PCOS, was similarly distributed across the euthyroid and hypothyroid groups, with no significant difference in either the distribution (p=0.937) or the mean ratio (p=0.883). Elevated serum free testosterone levels were observed in 50% of women with overt hypothyroidism, 33.3% of women with SCH, and 30.4% of euthyroid women. Yu et al observed that mean LH, FSH and FSH:LH were similar in both groups suggesting no statistically significant difference.[10] However, the mean free testosterone in SCH PCOS was 23.09±6.4 pg/ml compared to 12.2±5.7 pg/ml in euthyroid PCOS (p>0.05). These findings highlight the complex interaction between thyroid and ovarian function. Hypothyroidism may alter gonadotropin secretion, impair ovulatory mechanisms, and exacerbate hyperandrogenism through reduced sex hormone-binding globulin levels, thereby contributing to menstrual irregularities, infertility, and metabolic dysfunction.

Serum prolactin levels were largely within the normal range in both euthyroid and hypothyroid women (97.8% vs 94.1%), with no statistically significant difference between groups (p=0.156). This suggests that prolactin levels may remain relatively unaffected by thyroid status in women with PCOS. Similar findings were reported by Van der Ham et al who observed comparable rates of elevated prolactin levels in both groups (1.3% vs. 3%), indicating no significant association between thyroid status and hyperprolactinemia in women with PCOS.[13]

In the present study, thyroid autoimmunity was associated with a higher prevalence of infrequent menstrual cycles, heavy menstrual bleeding, and infertility. Infertility was observed in 80% of anti-TPO-positive women compared with 66% of antibody-negative women. Serin et al similarly reported significantly higher infertility rates among women with PCOS and Hashimoto's thyroiditis compared to women with PCOS alone (82.6% vs 56.5%; p=0.007), although menstrual irregularity rates were

comparable between groups (80.4% in PCOS vs 71.7% in PCOS with Hashimoto's Thyroiditis, p=0.26).[14] No significant differences were observed in acne or hirsutism. Likewise, Arduc et al found no significant difference in BMI (25.7±2.0 kg/m² vs 25.1±4.0 kg/m²) and lipid profile of both anti-TPO positive and negative patients.[15] The mean HDL in anti-TPO positive and negative groups were similar 49.8±12.9 mg/dl and 49.7±14.6 mg/dl respectively (p=0.996).

The high prevalence of SCH (23.8%) and autoimmune thyroiditis (15.9%) observed in the present study underscores the importance of comprehensive endocrine evaluation in women with PCOS. Routine screening for thyroid function abnormalities and thyroid autoantibodies may facilitate early diagnosis and timely intervention, potentially improving reproductive as well as metabolic outcomes.

Overall, the present findings support the growing body of evidence suggesting a strong epidemiological and pathophysiological association between PCOS and thyroid disorders.[16,17] Further large-scale longitudinal studies are required to better elucidate the causal relationship between these conditions and to determine whether correction of thyroid abnormalities can positively influence the long-term clinical and metabolic outcomes of women with PCOS.

Strength and limitations

The study evaluated a set of clinical, hormonal, and metabolic parameters using a case control study. However, other contributing factors such as genetic susceptibility, environmental influences, and additional biomarkers of thyroid autoimmunity were not assessed. Furthermore, as the sample was derived from a hospital-based gynecology outpatient population within a specific geographic region, the findings may not be generalizable to the broader population.

CONCLUSION

The increased prevalence of thyroid dysfunction, particularly subclinical hypothyroidism, among women with PCOS highlights the importance of early identification and management of thyroid abnormalities. Thyroid dysfunction contributes to the metabolic and hormonal disturbances seen in PCOS, reinforcing the need for routine thyroid screening as part of comprehensive care. Early diagnosis and appropriate treatment of even subtle thyroid abnormalities may lead to meaningful improvements in clinical symptoms, endocrine balance, and metabolic health in women with PCOS.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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