Journal of Population Therapeutics & Clinical Pharmacology

RESEARCH ARTICLE
DOI: 10.53555/y43bf735

PREVALENCE OF THYROID DYSFUNCTION AND AUTOIMMUNITY IN WOMEN WITH POLYCYSTIC OVARY SYNDROME

Humaira Alam[1*], Sayeba Akhter[2], Kazi Farhana Begum[3]

[1*]Assistant Professor, Department of Obstetrics and Gynaecology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh
[2]Former Professor, Department of Obstetrics and Gynaecology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh.
[3]Medical Officer, Department of Obstetrics and Gynaecology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh

***Corresponding Author:** Humaira Alam
*Assistant Professor, Department of Obstetrics and Gynaecology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh

Received: 14-09-2017 **Revised:** 19-10-2017 **Accepted:** 08-12-2017 **Published:** 23-12-2017

Abstract

Background: Polycystic ovary syndrome (PCOS) is a common endocrine disorder characterized by ovulatory dysfunction, hyperandrogenism, and polycystic ovarian morphology. Recent studies suggest an increased prevalence of thyroid dysfunction and autoimmunity among women with PCOS, potentially compounding metabolic and reproductive complications. This study aimed to evaluate the prevalence of thyroid dysfunction and autoimmune thyroiditis in women with polycystic ovary syndrome.

Methods: A cross-sectional observational study was conducted at the Department of Obstetrics and Gynaecology (Infertility), Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh, from July 2009 to June 2010. A total of 550 women diagnosed with PCOS based on the Rotterdam criteria were enrolled. Clinical evaluation, laboratory investigations including thyroid function tests (TSH, FT4), anti-thyroid antibodies (anti-TPO, anti-TG), and metabolic assessments were performed. Data were analyzed using SPSS v25.0, with statistical significance set at $p < 0.05$.

Results: Thyroid dysfunction was present in 17.27% of participants, with subclinical hypothyroidism accounting for 15.64%. Autoimmune thyroiditis, defined by at least one positive antibody marker, was detected in 26.36%. Women with subclinical hypothyroidism had significantly higher HOMA-IR, fasting insulin, LDL cholesterol, and triglyceride levels, and lower HDL cholesterol compared to euthyroid counterparts ($p < 0.01$ for all). Fasting glucose levels were not significantly different.

Conclusion: Thyroid dysfunction and autoimmunity are prevalent in PCOS and are associated with adverse metabolic profiles. Routine thyroid evaluation in PCOS patients may aid in early identification and management of comorbidities.

Keywords: PCOS, thyroid dysfunction, autoimmune thyroiditis, subclinical hypothyroidism.

INTRODUCTION

Polycystic ovary syndrome (PCOS) is the most frequent endocrine disorder in women of reproductive age, which is characterized by ovulatory dysfunction, hyperandrogenism, and polycystic ovarian morphology [1]. The prevalence is between 6 and 20 percent, depending on the diagnostic criterion employed in different parts of the world [2]. The Rotterdam consensus (2003) defines PCOS as a condition based on the presence of two of the following three characteristics: (a) oligo/anovulation, (b) clinical or biochemical hyperandrogenism, and (c) polycystic ovaries on ultrasound [1]. Besides reproductive consequences, PCOS is connected with insulin resistance, metabolic syndrome, and cardiovascular risk [3,4].

There is an emerging body of evidence that indicates that PCOS significantly overlaps with thyroid pathology, mainly autoimmune thyroiditis and subclinical hypothyroidism (SCH) [5,6]. Thyroid hormones are very much important in the regulation of metabolism and reproductive physiology, and a derangement in the thyroid status may worsen the menstrual irregularities, ovulatory disturbances and metabolic abnormalities that are frequently observed in PCOS [7]. The prevalence of subclinical hypothyroidism in PCOS is reported to be 10 to 25 percent, with various studies reporting an elevated level of thyroid-stimulating hormone (TSH) and a normal amount of free thyroxine (FT4) [8,9].

Autoimmune thyroiditis, or Hashimoto thyroiditis in particular features the manifestation of thyroid autoantibodies, including anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin (anti-TG) antibodies. Overstimulation of the estrogen receptors, a condition enhanced by estrogen dominance that is widely common in anovulatory PCOS patients, is believed to increase immune activity, creating vulnerability to other autoimmune diseases like thyroiditis [10].

Thyroid dysfunction has also proven to be associated with insulin resistance, which is characteristic of PCOS. The possibility is raised that hyperinsulinemia can affect the growth and functioning of the thyroid tissue in its mitogenic role [11]. It was noted by Ganie et al. that women with PCOS and SCH were more insulin-resistant and had impaired lipid profiles when compared to euthyroid women [8]. This metabolic load can be involved in poor cardiovascular outcomes, and it is why it is clinically relevant to diagnose and treat thyroid dysfunction in PCOS.

Although there is mounting evidence of comorbid thyroid issues with PCOS, there is still a question whether it should become a routine screen, especially in asymptomatic patients. The majority of the generated literature is in Western or South Asian populations, and little information on Bangladesh. In addition, autoimmune thyroiditis is a common disease with little research on its correlation with metabolic measures among PCOS women in this region.

This study aims to assess the prevalence of thyroid dysfunction and autoimmune thyroiditis among women with PCOS at a tertiary care center in Bangladesh. It also looks into the performance of subclinical hypothyroidism on metabolic items in this population. The study is relevant in filling a gap in the literature as it supplies regional information and establishes a case in support of the view that a thorough endocrine assessment in the treatment of PCOS is necessary.

METHODOLOGY & MATERIALS

This cross-sectional observational study was conducted at the Department of Obstetrics and Gynaecology (Infertility), Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh, between July 2009 and June 2010. A total of 550 women diagnosed with polycystic ovary syndrome (PCOS) according to the Rotterdam criteria were enrolled.

Inclusion Criteria:

I. Women aged 18 to 40 years diagnosed with PCOS based on Rotterdam criteria (presence of at least two of the following: oligo/anovulation, clinical or biochemical hyperandrogenism, polycystic ovaries on ultrasound).
II. Willingness to participate and provide informed consent.

Exclusion Criteria:

I. Pregnant or lactating women.
II. Women with previously diagnosed thyroid disease or on thyroid medication.
III. Those with chronic systemic illnesses such as diabetes mellitus, cardiovascular disease, or autoimmune disorders other than thyroiditis.
IV. Women on medications known to affect thyroid function or insulin sensitivity.

Data were collected through a combination of clinical evaluation, structured interviews, and laboratory investigations. Anthropometric measurements and clinical signs of hyperandrogenism were recorded. Blood samples were drawn for hormonal assays, including thyroid function tests (TSH, FT4), anti-thyroid antibodies (anti-TPO, anti-TG), fasting glucose, insulin, and lipid profile. Pelvic ultrasound was performed to assess ovarian morphology. Data collection followed standardized protocols to ensure accuracy and reproducibility. Informed consent was obtained from all participants, and confidentiality of personal information was maintained throughout the study. Data were analyzed using SPSS version 25.0. Descriptive statistics, including mean ± standard deviation for continuous variables and frequencies with percentages for categorical variables, were calculated. Inferential analyses involved independent samples t-tests for comparing means between groups and chi-square tests for categorical data. A p-value < 0.05 was considered statistically significant.

RESULTS

Table 1: Baseline characteristics of the study population (n=550)

Characteristics		Frequency (n)	Percentage (%)
Age group (years)	18–25	162	29.45
	26–30	208	37.82
	31–35	130	23.64
	36–40	50	9.09
BMI category (kg/m²)	< 25 (Normal weight)	180	32.73
	25–29.9 (Overweight)	240	43.64
	≥ 30 (Obese)	130	23.64
Menstrual irregularities	Oligomenorrhea	432	78.55
	Absent	118	21.45
Hirsutism (mFG score ≥ 8)	Present	215	39.09
	Absent	335	60.91
Acne	Present	162	29.45
	Absent	388	70.55
Family history of thyroid disease	Positive	134	24.36
	Negative	416	75.64

Table 1 presents the baseline characteristics of the 550 women with PCOS included in this study. The majority of participants were between the ages of 26 and 30 years (37.82%), followed by those aged 18–25 years (29.45%). In terms of body mass index (BMI), 43.64% were overweight (BMI 25–29.9), 32.73% had normal BMI (<25), and 23.64% were obese (BMI ≥30). A high proportion (78.55%) reported menstrual irregularities in the form of oligomenorrhea. Clinical signs of hyperandrogenism

were prevalent, with 39.09% exhibiting hirsutism and 29.45% reporting acne. Additionally, a positive family history of thyroid disease was noted in 24.36% of participants.

Table 2: Thyroid dysfunction in women with polycystic ovary syndrome (n=550)

Thyroid Status	Frequency (n)	Percentage (%)
Subclinical hypothyroidism	86	15.64
Overt hypothyroidism	9	1.64
Hyperthyroidism (subclinical/overt)	5	0.91
Total with thyroid dysfunction	95	17.27
Euthyroid	455	82.73

Table 2 shows the distribution of thyroid dysfunction among the PCOS cohort. Thyroid abnormalities were present in 17.27% of participants. Specifically, 15.64% had subclinical hypothyroidism (SCH), 1.64% had overt hypothyroidism, and 0.91% had hyperthyroidism (subclinical or overt). The remaining 82.73% were classified as euthyroid, indicating normal thyroid function.

Table 3: Autoimmune thyroiditis (n=550)

Autoimmune Marker	Frequency (n)	Percentage (%)
Anti-TPO positive	115	20.91
Anti-TG positive	89	16.18
Hashimoto's thyroiditis (clinical + ab)	112	20.36
Autoimmune thyroiditis (≥1 marker)	145	26.36
No autoimmune markers	405	73.64

Table 3 describes the prevalence of autoimmune thyroiditis based on the presence of thyroid autoantibodies. Anti-thyroid peroxidase (anti-TPO) antibodies were found in 20.91% of the women, while anti-thyroglobulin (anti-TG) antibodies were positive in 16.18%. A total of 20.36% met clinical and serological criteria for Hashimoto's thyroiditis. Overall, autoimmune thyroiditis, defined as the presence of at least one positive marker, was detected in 26.36% of the participants. The remaining 73.64% showed no autoimmune markers.

Table 4: Metabolic profile in SCH vs Euthyroid PCOS women (n=541)

Parameter	SCH (n = 86)	Euthyroid (n = 455)	p-value
HOMA-IR	3.5 ± 1.3	2.7 ± 1.0	<0.001
Fasting insulin (μIU/mL)	18.2 ± 5.1	15.3 ± 4.3	0.001
Fasting glucose (mg/dL)	94.1 ± 11.2	91.9 ± 10.4	0.076
LDL cholesterol (mg/dL)	132 ± 24	119 ± 21	<0.01
Triglycerides (mg/dL)	155 ± 45	139 ± 38	0.001
HDL cholesterol (mg/dL)	45 ± 9	50 ± 11	0.001

Table 4 compares the metabolic profiles of women with subclinical hypothyroidism (SCH) versus those who were euthyroid. The SCH group had significantly higher insulin resistance as indicated by HOMA-IR (3.5 ± 1.3 vs. 2.7 ± 1.0; $p<0.001$) and fasting insulin levels (18.2 ± 5.1 μIU/mL vs. 15.3 ± 4.3 μIU/mL; $p=0.001$). LDL cholesterol (132 ± 24 mg/dL vs. 119 ± 21 mg/dL; $p<0.01$) and triglycerides (155 ± 45 mg/dL vs. 139 ± 38 mg/dL; $p=0.001$) were also significantly elevated in the SCH group, while HDL cholesterol was lower (45 ± 9 mg/dL vs. 50 ± 11 mg/dL; $p=0.001$). Fasting glucose showed no significant difference between the two groups ($p=0.076$).

DISCUSSION

This study examined the prevalence of thyroid dysfunction and autoimmune thyroiditis among women with polycystic ovary syndrome (PCOS) and explored the metabolic implications of subclinical hypothyroidism (SCH) within this population. Our results show that there is a notable burden of pathologic thyroid parameters and autoimmune indicators among PCOS women, which complements and builds upon the already existing body of knowledge.

Subclinical hypothyroidism was the most widespread (15.64%), followed by the overall prevalence of thyroid dysfunction in the study groups at 17.27%. It can be compared with earlier results of Benetti-Pinto et al, who have reported SCH in 18.2 percent of PCOS women [12], and Ozdemir et al., who did not notice any difference in a smaller range of thyroid abnormalities [13]. The prevalence is observed to be more than in the general population, whose SCH normally strikes between 4 and 10 percent of women at the reproductive age [14]. These observations indicate a possible pathophysiological intersection between PCOS and thyroid malfunction that may result via standard hormonal and immunological mechanisms.

The prevalence in the PCOS cohort was 26.36 percent who had autoimmune thyroiditis in the presence of anti-thyroid antibodies. The most common parameters were anti-TPO antibodies (20.91%), followed by anti-TG antibodies (16.18%). These estimates are consistent with those of the previous research results, which showed a 28 percent prevalence of thyroid autoantibodies among PCOS patients [15] and another study indicating that almost a quarter of patients with PCOS had autoimmune thyroiditis [16]. These results also confirm the hypothesis of immune dysregulation in PCOS, which may be due to the presence of unopposed levels of estrogens that increase the production of autoantibodies [10].

Interestingly, the positive family history of thyroid disease was reported in 24.36% of participants and added more support to the genetic predisposition and family clustering of autoimmune thyroid disorders as highlighted by Ong et al. [17].

The comparison of the metabolism of women with SCH and women who were euthyroid provided statistically significant differences. SCH was related to increased insulin resistance (HOMA-IR), fasting insulin, LDL cholesterol and triglycerides levels, and decreased HDL cholesterol levels. The current results support those of Enzveai et al. (2013), who reported that insulin resistance is higher in SCH-PCOS patients than in euthyroid controls [18], as well as those of Trummer et al. (2015), according to which high concentrations of TSH have a negative effect on lipid metabolism in PCOS [19]. It is uncertain how this happens, but thyroid hormones have been shown to regulate glucose and lipid levels, and even mild hypothyroidism has the ability to disrupt metabolism [7].

Comparison of fasting glucose revealed no significant results as compared to the SCH and euthyroid groups in our data, unlike the findings of Sravan Kumar et al., who discovered elevated fasting glucose in SCH-PCOS patients [20]. This highly differs, and this could be as a result of population differences, sample sizes or differences in dietary and lifestyle influences.

The frequently overlapping issues of PCOS and thyroid autoimmunity stir up the notion of a potential shared etiology. PCOS-induced estrogen dominance could increase immune activation, leading to stronger autoantibodies against thyroid antigens [21]. Moreover, a potential pathogenic pathway might exist in the form of a chronic low-grade inflammation of both diseases. Oxidative stress caused by inflammation has been shown to cause PCOS as well as autoimmune thyroid disease, so perhaps immune-metabolic interplay could be the root cause of such results.

The presence of SCH and thyroid autoantibodies in PCOS women affects reproductive and metabolic health. Thyroid dysfunction may exacerbate anovulation, worsen lipid profiles, and reduce insulin

sensitivity, compounding metabolic risks in PCOS. Early identification of thyroid dysfunction could improve reproductive outcomes and reduce cardiovascular risk. Michalakis et al. demonstrated that normalization of TSH levels in women undergoing assisted reproductive technology led to improved outcomes, reinforcing the need for thyroid assessment in subfertile women [22].

While routine thyroid screening in PCOS patients remains debated, our findings support targeted evaluation, particularly in those with thyroid symptoms, family history, or infertility. The detection of anti-thyroid antibodies—even in euthyroid individuals—may warrant monitoring due to risk of progression to overt hypothyroidism.

Study limitations include its cross-sectional design, which precludes causal inferences, and the exclusion of non-PCOS controls, limiting broader generalization. Only baseline thyroid and metabolic parameters were assessed; longitudinal data would offer insights into the progression of these conditions. This study adds valuable regional data on the interplay between PCOS and thyroid abnormalities, supporting findings from Western and South Asian cohorts. The prevalence of thyroid dysfunction and autoimmune thyroiditis underscores the need for awareness among clinicians managing PCOS, advocating for integrated endocrine assessment.

CONCLUSION

This study reveals a substantial prevalence of thyroid dysfunction and autoimmune thyroiditis among women with polycystic ovary syndrome (PCOS), with subclinical hypothyroidism being the most common abnormality. Thyroid autoantibodies were present in over one-fourth of the participants, suggesting a strong association between PCOS and autoimmune thyroid involvement. Moreover, women with subclinical hypothyroidism exhibited significantly adverse metabolic profiles compared to their euthyroid counterparts. These findings underscore the need for routine thyroid screening in PCOS patients to identify and manage comorbid endocrine and metabolic disorders effectively.

REFERENCES

1. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). Human reproduction. 2004 Jan 1;19(1):41-7.
2. Azziz R, Woods KS, Reyna R, Key TJ, Knochenhauer ES, Yildiz BO. The prevalence and features of the polycystic ovary syndrome in an unselected population. The Journal of Clinical Endocrinology & Metabolism. 2004 Jun 1;89(6):2745-9.
3. Dunaif A. Insulin resistance and the polycystic ovary syndrome: mechanism and implications for pathogenesis. Endocrine reviews. 1997 Dec 1;18(6):774-800.
4. Legro RS, Arslanian SA, Ehrmann DA, Hoeger KM, Murad MH, Pasquali R, Welt CK. Diagnosis and treatment of polycystic ovary syndrome: an Endocrine Society clinical practice guideline. The Journal of Clinical Endocrinology & Metabolism. 2013 Dec 1;98(12):4565-92.
5. Kachuei M, Jafari F, Kachuei A, Keshteli AH. Prevalence of autoimmune thyroiditis in patients with polycystic ovary syndrome. Archives of gynecology and obstetrics. 2012 Mar; 285:853-6.
6. Sinha U, Sinharay K, Saha S, Longkumer TA, Baul SN, Pal SK. Thyroid disorders in polycystic ovarian syndrome subjects: A tertiary hospital based cross-sectional study from Eastern India. Indian journal of endocrinology and metabolism. 2013 Mar 1;17(2):304-9.
7. Pearce EN, Farwell AP, Braverman LE. Thyroiditis. New England Journal of Medicine. 2003 Jun 26;348(26):2646-55.
8. Ganie MA, Marwaha RK, Aggarwal R, Singh S. High prevalence of polycystic ovary syndrome characteristics in girls with euthyroid chronic lymphocytic thyroiditis: a case–control study. European journal of endocrinology. 2010 Jun;162(6):1117-22.
9. Shirsath A, Aundhakar N, Kamble P. Do the thyroid hormonal levels alter in polycystic ovarian disease? A comparative cross-sectional study. Indian J Basic Appl Med Res. 2015; 4:265-71.

10. Cutolo M, Sulli A, Straub RH. Estrogen's effects in chronic autoimmune/inflammatory diseases and progression to cancer. Expert review of clinical immunology. 2014 Jan 1;10(1):31-9.
11. Mueller A, Schöfl C, Dittrich R, Cupisti S, Oppelt PG, Schild RL, Beckmann MW, Häberle L. Thyroid-stimulating hormone is associated with insulin resistance independently of body mass index and age in women with polycystic ovary syndrome. Human reproduction. 2009 Nov 1;24(11):2924-30.
12. Benetti-Pinto CL, Piccolo VR, Garmes HM, Juliato CR. Subclinical hypothyroidism in young women with polycystic ovary syndrome: an analysis of clinical, hormonal, and metabolic parameters. Fertility and sterility. 2013 Feb 1;99(2):588-92.
13. Ozdemir D, Cuhaci N, Balkan F, Usluogullari A, Ersoy R, Cakir B. Prevalence of thyroid pathologies in patients with polycystic ovary syndrome. InEndocrine Abstracts 2011 Apr 1 (Vol. 26). Bioscientifica.
14. Hollowell JG, Staehling NW, Flanders WD, Hannon WH, Gunter EW, Spencer CA, Braverman LE. Serum TSH, T4, and thyroid antibodies in the United States population (1988 to 1994): National Health and Nutrition Examination Survey (NHANES III). The Journal of Clinical Endocrinology & Metabolism. 2002 Feb 1;87(2):489-99.
15. Hefler-Frischmuth K, Walch K, Huebl W, Baumuehlner K, Tempfer C, Hefler L. Serologic markers of autoimmunity in women with polycystic ovary syndrome. Fertility and sterility. 2010 May 1;93(7):2291-4.
16. Novais JD, Benetti-Pinto CL, Garmes HM, Menezes Jales R, Juliato CR. Polycystic ovary syndrome and chronic autoimmune thyroiditis. Gynecological Endocrinology. 2015 Jan 2;31(1):48-51.
17. Ong KK, Kuh D, Pierce M, Franklyn JA, Medical Research Council National Survey of Health and Development Scientific and Data Collection Teams. Childhood weight gain and thyroid autoimmunity at age 60–64 years: the 1946 British birth cohort study. The Journal of Clinical Endocrinology & Metabolism. 2013 Apr 1;98(4):1435-42.
18. Enzevaei A, Salehpour S, Tohidi M, Saharkhiz N. Subclinical hypothyroidism and insulin resistance in polycystic ovary syndrome: is there a relationship? Iranian journal of reproductive medicine. 2014 Jul;12(7):481.
19. Trummer C, Schwetz V, Giuliani A, Obermayer-Pietsch B, Lerchbaum E. Impact of elevated thyroid-stimulating hormone levels in polycystic ovary syndrome. Gynecological Endocrinology. 2015 Oct 3;31(10):819-23.
20. Sravan Kumar P, Ananthanarayanan PH, Rajendiran S. Cardiovascular risk markers and thyroid status in young I ndian women with polycystic ovarian syndrome: A case–control study. Journal of Obstetrics and Gynaecology Research. 2014 May;40(5):1361-7.
21. Fénichel P, Gobert B, Carré Y, Barbarino-Monnier P, Hiéronimus S. Polycystic ovary syndrome in autoimmune disease. Lancet (London, England). 1999 Jun 26;353(9171):2210-.
22. Michalakis KG, Mesen TB, Brayboy LM, Yu B, Richter KS, Levy M, Widra E, Segars JH. Subclinical elevations of thyroid-stimulating hormone and assisted reproductive technology outcomes. Fertility and sterility. 2011 Jun 30;95(8):2634-7.