

Type of the Paper (Research article)

## Association between Ovarian Reserve Parameters and Thyroid Disorders in Polycystic Ovary Syndrome

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Received: 12 December, 2023

Accepted: 29 January, 2024

Reviewed: 18 January, 2024

Published online: 16 July, 2024

### Abstract:

**Introduction:** The most prevalent endocrine condition affecting women in their reproductive years, polycystic ovarian syndrome (PCOS) affects 4–12% of the general population. Amenorrhea, infertility, hyperandrogenism, hyperinsulinemia, and metabolic syndrome are all linked to it. Patients with this illness should be properly monitored and given the proper diagnosis as soon as possible, since the underlying pathology may put them at risk for further comorbidities.

**Aim of the study:** to assess the relationship between ovarian reserve parameters and thyroid issues in PCOS.

**Subjects and Methods:** The purpose of this cross-sectional study was to assess the relationship between thyroid diseases and ovarian reserve tests. Two hundred participants with PCOS were enrolled in this study based on the Rotterdam Criteria 2004.

**Results:** Serum TSH and FSH levels showed a statistically significant positive linear minor association. In contrast, a linear association between LH and TSH was not statistically significant. There is a statistically significant linear negative link between TSH serum level and AFC, while AMH and TSH serum level similarly exhibit a statistically significant negative linear correlation.

**Conclusion:** Thyroid dysfunction may be present to some extent in the majority of PCOS patients. Thyroid autoimmunity should be examined along with the evaluation of ovarian reserve.

**Keywords:** Ovarian Reserve Parameters; Thyroid Disorders; Polycystic Ovary Syndrome

## 1. Introduction

The quantity and calibre of oocytes within the ovary are called "ovarian reserve". Except for antral follicle counts, most

established baseline tests for ovarian reserve offer an approximate indication of the antral follicle cohort size. AMH, which plays a role in

folliculogenesis, has a strong relationship with antral follicle numbers, age, and the success of *in vitro* fertilization [1].

Thyroid gland evaluation is frequently recommended for women with undiagnosed PCOS since thyroid disease has been linked to infertility issues. Disorders leading to overt hyperthyroidism or hypothyroidism can also have an impact on fertility and cause menstrual irregularities. Long-term steroid hormone treatment has been proven to affect all receptors in the signaling pathway of thyroid hormone activity, including thyrotropin-releasing hormone (TRH), thyroid-stimulating hormone

(TSH), and thyroid hormones. These receptors are present in the uterus of monkeys [2].

Pervin et al. (2020) assessed and identified thyroid impairment in PCOS individuals. They showed that 30% of PCOS patients have thyroid abnormalities. Compared to hyperthyroidism, hypothyroidism is about three times as common [3]. Infertile women's ovarian reserve and thyroid function-related indicators did not significantly correlate, according to Wu et al. (2021) evaluation of the association between ovarian reserve, thyroid function, and AMH levels in infertile patients [4].

## 2. Subjects and Methods

### 2.1. Subjects

Two hundred patients with PCOS diagnoses are included in this observational cross-sectional study. The following conditions apply to the study's eligible patients:

#### *Inclusion criteria*

The age range of 18–39, more than a year of infertility, PCOS cases diagnosed based on the updated European Society of Human Reproduction and Embryology (ESHRE) and American Society of Reproductive Medicine (ASRM) criteria, if two of the three criteria are met, normal hysterosalpingogram, and normal seminal profile is required.

#### *Exclusion criteria*

Individuals who met any of the following criteria were not allowed to participate in the study: age < 18 or > 39; patient refusal; previous thyroidectomy; prior radioactive iodine treatment; prior cervical surgery/radiotherapy; prior oophorectomy; malignant/autoimmune pathology; chronic kidney and liver disease; and any other medical conditions.

### 2.2. Methods

#### *Rotterdam Diagnostic Criteria of Polycystic Ovarian Syndrome*

To diagnose PCOS, at least two of the following three characteristics must be present [5].

### ***Oligo/anovulation***

Women who have menstrual periods longer than 35 days apart or, on the other hand, shorter than 21 days tend to have oligo/anovulation. It's crucial to keep in mind that anovulatory women might have regular periods as well. The luteal progesterone level, measured on day 21 of a 28-day cycle, will indicate the ovulatory status of these women.

### ***Hyperandrogenism***

Clinical (male pattern baldness, hirsutism), biochemical (increased free androgen index, FAI), or free testosterone.

It is challenging to evaluate hirsutism because most women conceal it from scrutiny. The most accurate way to measure hyperandrogenemia is with free testosterone, which can be obtained from bioavailable, calculated, or FAI sources. To rule out late-onset congenital adrenal hyperplasia and virilizing tumors, more research is necessary if there is a marked elevation in free testosterone or signs of rapid virilization.

### ***Polycystic ovaries on ultrasound***

When at least 12 follicles are visible in each ovary, the follicular diameter ranges from 2 to 9 mm, and the increased ovarian volume exceeds 10 mm<sup>3</sup>, polycystic ovaries are diagnosed using ultrasound technology. It's necessary to rule out additional causes such as congenital adrenal hyperplasia, tumors that

secrete testosterone, Cushing syndrome, thyroid malfunction, and hyperprolactinemia.

### ***Tools***

Patients who consented to take part in the trial gave their written and verbal consent. The following were applied to each patient.

### ***Careful history taking***

Taking a history includes the following: a personal history (including a full menstrual history, hirsutism, acne, oligomenorrhea or amenorrhea, infertility, and galactorrhea): prior experiences with thyroid problems, diabetes mellitus, hypertension, autoimmune diseases, and PCOS in the family. Abdominal examination included the following; Inspection of hair distribution, and Palpation for pelvi-abdominal masses. The pelvic examination included P/V for the exclusion of adnexal masses

### ***Transvaginal ultrasound***

Using a 5–9 MHz transvaginal probe, all patients had transvaginal ultrasonography in lithotomy position to diagnose PCO and detect any abnormalities or uterine or ovarian mass, such as ovarian cysts, uterine fibroid, and pelvic endometriosis [6].

The medical professional can inspect and search for anomalies inside the vagina by inserting an ultrasonography probe. It is possible to measure the endometrium's thickness. "PCO is now defined by the revised Rotterdam criteria

as having 12 or more tiny (2 to 9 mm) follicles in each ovary." After locating the ovary, the probe was rotated to measure its longest diameter and was then saved as a single frame on a split screen. To obtain the ovary's true transverse axis, the probe was then rotated 90 degrees. The ovary's biggest longitudinal, transverse, and anteroposterior (AP) diameters were measured in centimeters, and the volume of the ovary was computed.

The longest portion of the ovary was used to count the number and location of antral follicles; for better results, the entire ovary was tallied by doing a 2D sweep across the entire ovary. When the number of follicles is significantly higher, as in PCO, this procedure is very practical and dependable.

**Type of machine:** Mindray DP-15.

### ***Laboratory investigations***

1. Determination of AMH: The automated chemiluminescence assay Beckman Access-2 was used to determine AMH. Using a serum separator tube, we centrifuged the samples for 20 minutes at a weight of roughly 1000 xg after letting them clot for two hours at room temperature or overnight at 4°C. Newly-made serum was either used right away for analysis or stored in an aliquot at -20°C for subsequent use. Freeze/thaw cycles were not repeated. Samples with hemolysis were not included. Hemolysis was avoided because it could affect the outcome of the sample. This assay's

analytical sensitivity was 0.014 ng/mL. The coefficients of variance within and between assays were less than 12.3% and 14.2%, respectively.

2. Measurement of serum TSH, serum FT<sub>3</sub> and FT<sub>4</sub>: Each research participant had 6 ml of venous blood drawn from their median cubital vein using a disposable syringe by protocol. To prevent hemolysis, the nozzle was removed from the needle, and the blood from the syringe was gently pushed into a clean, dry test container. Every biochemical test was conducted as soon as feasible. In the event of a postponement, the specimen was preserved at -20°C to prevent degradation and impurity.

### ***Interpretation of thyroid function test results***

The findings of the thyroid function test can be categorized as follows based on the serum free T3 (FT3), free T4 (FT4), and TSH level; TSH, FT3, and FT4 are within normal limits in euthyroid patients. Primary hypothyroidism: TSH levels beyond the normal upper limit (>5.0 pmol/L) and FT4 and FT3 values below normal. Primary hyperthyroidism is defined as FT3, FT4, and TSH levels that are either undetectable or below the lower limit of normal (<0.3 pmol/L). Subclinical hypothyroidism: TSH that is just slightly high and normal FT3, FT4, and TSH levels. Subclinical hyperthyroidism: FT3, FT4, and TSH levels are within normal range and TSH levels are negligible or absent [7].

### 2.3. Statistical Analysis

After being gathered, edited, coded, and entered, the data were added to IBM SPSS, a statistical package for social science, version 20. Quantitative data were presented as means, standard deviations, and ranges when their distribution was parametric, whereas qualitative data were expressed as numbers and percentages. The chi-square test and/or Fisher exact test were employed in place of the chi-square test when comparing two groups using

qualitative data where the expected count in any cell was less than 5. The independent sample t-test was used to compare quantitative data having a parametric distribution between two independent groups. The allowable margin of error was set at 5%, while the confidence interval was set at 95%. Therefore, the following criteria were used to determine if the p-value was significant:  $P > 0.05$  = non-significant (NS);  $P < 0.05$  = significant (S); and  $P < 0.001$  = highly significant (HS). Certain information was illustrated with simple graphs.

### 3. Results

200 women with PCOS diagnoses based on the Rotterdam Consensus participated in the cross-sectional study that was carried out in this instance. The baseline data of the women under study are presented in **Table 1**. The women's ages varied from 18 to 38 years old, with an average age of  $(25.69 \pm 5.54)$ . Out of the 200

women in the study, 116 (58%) had primary infertility and 84 (42%) had secondary infertility. All of them had been diagnosed with infertility. Merely 8 (4%) instances exhibit comorbidities (DM and HTN) about linked comorbidities.

**Table 1:** Baseline data of the studied women (N= 120).

Variables	Descriptive statistics	
Age (years)	Range (Min-Max)	18.0 - 38.0
	Mean $\pm$ SD	25.69 $\pm$ 5.54
Infertility Type; N (%)	Primary	58 (48.3%)
	Secondary	62 (51.7%)
Duration of infertility (Years)	Range (Min-Max)	1.0 - 12.0
	Mean $\pm$ SD	3.78 $\pm$ 2.32
Co-morbidities	Yes	8 (4%)
	No	192 (96%)

Table (2) demonstrates the hormonal parameters and ovarian reserve assessment among the studied women. TSH ranged from (0.08) to (8.97) with an average of (2.45±1.35), LH ranged from (2.60) to (36.0) with an average of (2.45 ±1.35), FSH ranged from (1.80) to

(14.70) with an average of (5.71 ±2.47), AMH was ranged from (1.20) to (18) with an average of (6.04 ±3.01) ng/ml, and AFC was ranged from (10) to (50) with an average count of (23.89 ±3.01).

**Table 2:** Hormonal parameters and ovarian reserve assessment among the studied PCO women (N= 200).

Variables	Min- Max	Mean ±SD
TSH (mIU/ml)	0.08-8.97	2.52±2.15
LH (IU/L)	2.60-36.00	2.45±1.35
FSH (IU/L)	1.80-14.70	5.71±2.47
AMH (ng/ml)	1.20-18.00	6.04±3.01
AFC (count)	10-50	23.89±10.29

Table 3 demonstrated the association between TSH and type of infertility, there was a non-statistically significant association between

type of infertility and TSH serum level ( $p >0.05$ ).

**Table 3:** Association between TSH and type of infertility among studied women (N= 200).

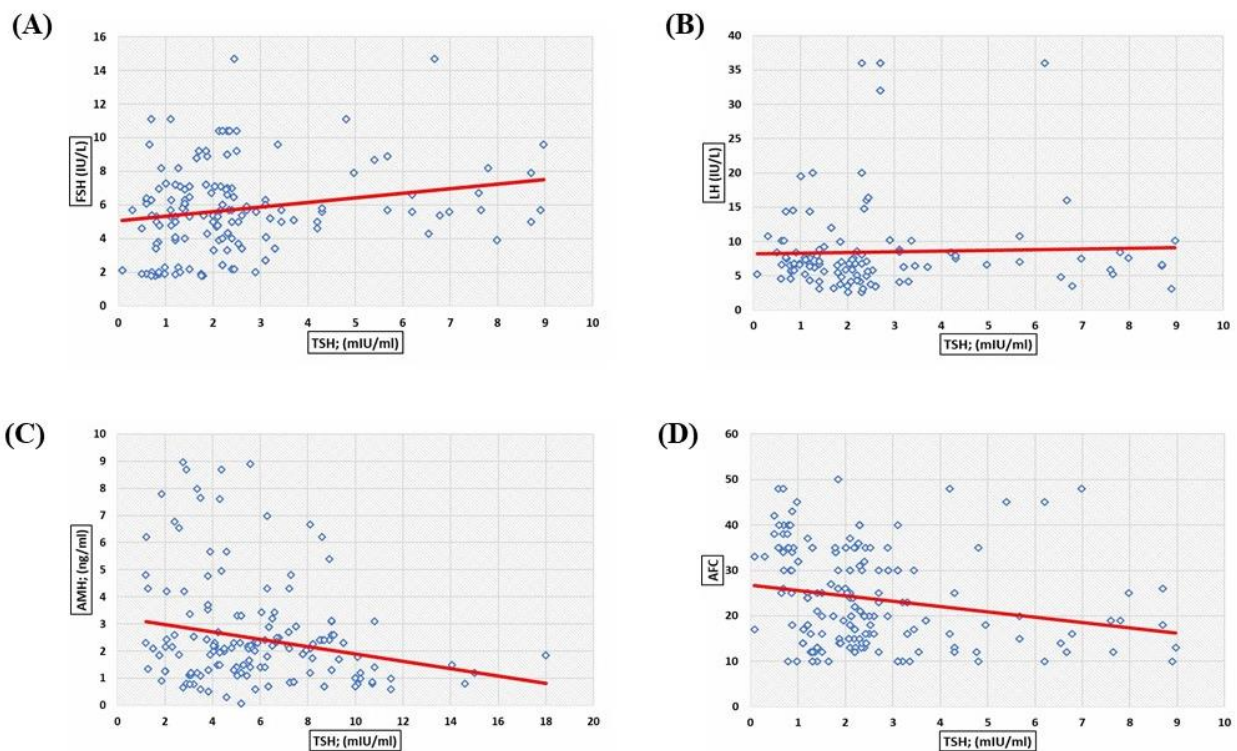
Variables	Min- Max	Mean ±SD	P- value
1ry infertility	0.3-8.97	2.53±1.96	0.312
2ry infertility	0.081-8.9	2.27±1.44	

Table 4 shows the relationship between the study women's ovarian reserve assessment and TSH with hormonal indicators. There was a non-statistically significant linear correlation between LH and TSH ( $p >0.05$ ); however, there was a statistically significant positive linear minor association between serum TSH and FSH

levels ( $r = 0.202$ ,  $p =0.006$ ). AMH and TSH serum levels displayed a statistically significant negative linear correlation ( $r =-0.230$ ,  $p =0.001$ ) (**Figure 1**). AFC and TSH serum levels similarly exhibited a statistically significant linear negative correlation ( $r =-0.201$ ,  $p =0.004$ ) (**Figure 1**).

**Table 4:** Correlation between TSH with hormonal parameters and ovarian reserve assessment among the studied women (N= 200).

Variables	r	P- value
LH	0.029	0.704
FSH	0.202	0.006*
AMH	-0.230	0.001*
AFC	-0.201	0.004*



**Figure 1:** Correlation between TSH with (A) FSH, (B) LH, (C) AMH, and (D) AFC.

## 4. Discussion

The most common cause of anovulatory infertility is polycystic ovarian syndrome (PCOS), which affects 5-10% of women [8]. Ovarian follicle abnormalities in PCOS patients lead to twofold abnormalities in ovarian follicles: first, excessive early follicular growth leads to an excess of tiny

antral follicles in women with PCOS, which are two to three times more numerous than in women with normal ovaries. Second, there is no selection of a single follicle from the larger pool of selectable follicles, and that single follicle does not mature into a dominant follicle. Follicle arrest (FA) is the term

for this second anomaly in folliculogenesis that accounts for PCOS ovulatory dysfunction [9].

According to the Rotterdam Criteria, four different clinical forms of Polycystic Ovary Syndrome (PCOS) can be distinguished. Phenotype A is the most common, exhibiting polycystic ovarian morphology (PCOM) in USG, menstrual disruption (oligo/amenorrhea), and clinical and/or biochemical hyperandrogenism (HA). The presence of HA+OA indicates phenotypic B, the presence of HA+PCOM indicates phenotype C and the presence of OA+PCOM defines phenotype D, the fourth phenotype [10]. A growing amount of data points to a connection between PCOS and autoimmune. Anti-granulosa cell antibodies have previously been proposed as a potential cause of PCOS development [11], but this idea hasn't been validated by Petrikova et al. (2015) [12]. Nevertheless, Benetti-Pinto et al. (2012) highlighted the connection between autoimmune illnesses, particularly Hashimoto's thyroiditis (HT), and PCOS [13]. Research indicates that the prevalence of autoimmune thyroid disease varies between 18% and 40% in women with PCOS, contingent upon the PCOS diagnostic criteria utilized and the ethnic background of the patients.

The intricate etiology of HT stems from aberrant interactions among T cells, antigen-presenting cells, and thyrocytes. Thyrocyte lysis is a result of TH1-type induced autoimmunity, which is a characteristic of HT. In genetically susceptible individuals, nongenetic factors such as environmental and hormonal factors may act as triggers for an aberrant autoimmune response [14].

Ovarian reserve function is a function that steadily declines with age and can be interpreted as a reflection of a woman's endocrine system and fertility. A decreased capacity for reproduction combined with a low response to ovarian stimulation characterizes diminished ovarian reserve or DOR. Unknown is the reason and mechanism of DOR, which still affects certain young women [15].

The granulosa cells of pre-antral and antral follicles generate anti-Mullerian hormone (AMH), a glycoprotein that belongs to the transforming growth factor- $\beta$  (TGF- $\beta$ ) family. AMH plays a crucial function in the development and maturation of follicles [16]. Gonadotropin-releasing hormone (GnRH) agonists and oral contraceptives have no effect on AMH levels, which are not influenced by the menstrual cycle. Therefore, serum AMH measurement is often performed during the first work-up for infertility and has been used widely in clinical practice for assessment of ovarian reserve [17].

The number of tiny follicles seen in PCOS is increased two to three times, which is consistent with serum AMH levels in PCOS patients being two to three times greater than in ovulatory women with normal ovaries. The elevated AMH may prevent follicle selection, leading to follicular halt at the small antral phase and failure of dominance, by decreasing follicle sensitivity to FSH and estrogen production [18]. AMH levels should range from 2 to 4 ng/mL; however, more than 3 ng/mL is indicative of PCOS. It has been claimed that the most sensitive and specific diagnostic sign for PCOS is a blood



level of AMH  $\geq 5$  ng/mL. 5 to 7 ng/mL is considered high normal [19].

Although the exact reason of PCOS's extremely high AMH levels is unknown, there has been evidence of a positive association between AMH and serum androgen levels, suggesting that PCOS's overproduction of androgens is caused by an intrinsic abnormality in thecal cells. Granulosa cells may exhibit inherent dysregulation, such as an overexpression of the AMH receptor type 2 (AMHR2) [20].

AMH causes ovarian aromatase expression and FSH receptor expression to drop dramatically, resulting in "follicular arrest." The "follicular arrest" may potentially be caused by the absence of luteinizing hormone (LH)-induced downregulation of AMHR2 expression [21]. Inducing ovulation is the treatment for oligo- or anovulatory infertility. First-line treatment for patients involves inducing ovulation with clomiphene citrate (CC). After 6–9 cycles of medication, 75–80% of females had an ovulation rate, and 70–75% had a cumulative pregnancy rate. CC resistance is defined as the inability to ovulate following a minimum of 150 mg per day beginning on the third day of the menstrual cycle and continuing for five days or six months [22].

The National Institute for Health and Care Excellence Guideline on Fertility states that in an in vitro fertilization (IVF) cycle, a follicle-stimulating hormone (FSH) level of less than 4 IU/l, an anti-Müllerian hormone (AMH) level greater than 25 pmol/l, or an antral follicle count (AFC) greater than

16 will likely result in an excessive ovarian response [23].

Vembu and Reddy demonstrated that the antral follicle count (AFC) is not a reliable indicator of the ovarian response, but rather the anti-Müllerian hormone (AMH) level is [24]. Transvaginal ultrasound scanning is used to visualize antral follicle count (AFC), which has garnered significant interest as an ovarian reserve diagnostic [25].

The thyroid gland is a butterfly-shaped gland near the base of the neck that weighs only 20 grams, but the hormones it secretes are vital to metabolism and growth. The gland is a key player in controlling every bodily function. Thyroid problems can take many various forms; hypothyroidism, hyperthyroidism, or euthyroidism, which occurs when thyroid hormone levels are within normal limits, are the conditions that might cause them. Thyroid problems affect 0.8% to 5% of adults, with fertile women having a 4–7 times higher chance of developing thyroid abnormalities. Thyroid hormone dysfunction and anatomic anomalies rank among the most prevalent endocrine gland diseases. Since thyroid abnormalities frequently manifest subtly, it has long been known that thyroid insufficiency has a significant impact on the reproductive system of women. The autoimmune nature of thyroid problems may be the cause of the high prevalence of thyroid illnesses in women, despite the fact the exact cause is unknown. Many metabolic processes are linked to anomalies in the thyroid hormone supply to the peripheral tissues [26].

The most prevalent pathological hormone shortage is primary hypothyroidism, with a prevalence of 4.3% for subclinical disease and 0.3% for overt disease. Abnormal thyroid hormone levels affect various organs, including the female reproductive system. Prolonged hypothyroidism raises serum prolactin levels, which can disrupt gonadotropin secretion. Anovulation and/or luteal phase malfunction result in clinical signs such as irregular menstruation and decreased fertility [27].

The most accurate and precise marker for the identification and treatment of thyroid dysfunction is the pituitary hormone thyrotropin (TSH) test [28]. Michalakis et al. (2011) have demonstrated that infertile women's TSH levels were greater than those of typically fertile women. Furthermore, infertile patients' DOR was linked to higher blood TSH levels [29].

It has been reported that PCOS and thyroid disorders have profound effects on fertility and reproductive biology. More interestingly hypothyroidism can initiate, maintain or worsen the syndrome [30]. Regarding thyroid disorders in PCOS, Janssen et al. (2004) have tried to explore the PCOS-thyroid interface. They showed a higher incidence of elevated TSH levels and a four times higher prevalence of autoimmune thyroiditis in PCOS subjects [31]. The age range in the current study was 18 to 38 years old, with an average age of  $25.69 \pm 5.54$  years. This outcome is similar to what has been reported by Roy et al. (2009) according to them, the average age was 28.2 years [32]. In addition, Sunj et al. (2013) included 96 infertile

PCOS women with a mean age of  $29.3 \pm 3.31$  years [33]. Zwain and Aziz (2026) showed that nearly half of the study patients were in the age group 26 - 35 years (48%) with PCOS followed by less than 25 years old patients [34]. Pervin et al. (2020) evaluated and detected thyroid dysfunction in patients with PCOS. A total of 150 women of reproductive age (15 - 45 years) were diagnosed with PCOS (2 of the 3 Rotterdam criteria). The mean age of the study subjects was  $24.57 \pm 4.27$  years [3]. Wu et al. (2021) evaluated the relationship between the ovarian reserve, thyroid function, and AMH levels in infertile patients. The average age of the patients was  $30.31 \pm 4.50$  years old (21-44 years old) and Izhar et al. (2021) determined the cut-off for the antral follicle count and the anti-Müllerian hormone level predictive of ovarian hyperstimulation syndrome with a mean age of the study participants was  $31.32 \pm 3.82$  years [35].

In our study, TSH ranged from 0.08 to 8.97 ( $2.45 \pm 1.35$ ). Pervin et al. (2020) found that 105 (70%) had normal TSH levels (TSH 0.3 - 5.0 pmol/L), 12 (8%) had decreased (TSH  $<0.3$  pmol/L) levels of TSH and 33 (22.0%) had elevated TSH level (TSH  $>5.0$  pmol/L) [3]. Elevated FSH levels cannot be used as an early predictor of reduced fertility [36]. Sequential basal FSH measurements may be useful as a short-term predictor [37]. In our study, FSH ranged from 1.8 to 14.70 ( $5.71 \pm 2.47$ ). An elevated FSH, if greater than 10 IU/L on more than one occasion, the ovaries are unlikely to be ovulating regularly and will also be resistant to exogenous stimulation. When serum concentration of FSH is above 15 IU/L, the chance of ovarian

activity is low and levels greater than 25 IU/L suggest menopause or POF. If a woman has amenorrhea and an elevated serum FSH concentration ( $> 20$  IU/L) on more than two occasions, she likely has POF. The longer the period of amenorrhea and the higher the FSH level, the greater the likelihood that the ovarian failure is permanent [38].

In our study, AMH ranged from 1.2 to 18 with an average of  $6.04 \pm 3.01$  ng/ml. Li et al. (2010) reported a mean AMH level of 9.85 ng/mL [39], while Catteau-Jonard et al. (2007) reported 6.59 ng/mL in patients with PCOS [40]. Piltonen et al. (2005) concluded that the AMH level in patients with PCOS was always two or three times higher than in the normal population (16–44 years) [41]. Hestiantoro et al. (2016) found that the mean value of the AMH level was around 7.51 ng/mL [42]. Izhar et al. (2021) found that the mean AMH was  $6.36 \pm 1.10$  ng/ml among the cases. Wu et al. (2021)<sup>4</sup> found that the average AMH was  $5.13 \pm 4.30$  ng/ml (0.08-18 ng/ml) [35].

In our study, AFC ranged from 10 to 50 with an average count of  $23.89 \pm 3.01$ . Izhar et al. (2021) found that the mean antral follicle count among the cases was  $14.32 \pm 3.69$  [35]. Our patients were diagnosed with infertility, out of the 200 women, 116 (58%) had primary infertility and 84 (42%) had secondary infertility. Our study demonstrated the association between TSH and type of infertility, as there was a non-statistically significant association between type of infertility and TSH serum level.

Zwain and Aziz (2026) showed that the overall prevalence of infertility was 46% in which 32% of them had primary infertility and 14% of cases had secondary infertility [34]. Pervin et al. (2020) observed that among a total of 120 married study subjects; primary infertility was detected in 58 PCOS women as the highest percentage (48.33%) followed by the normal fertility status (42.5%) and (9.17%) subjects with secondary infertility [3].

Our study demonstrated the correlation between TSH with hormonal parameters and ovarian reserve assessment among the studied women. There was a statistically significant positive linear slight correlation between serum TSH and FSH levels. On the other hand, there was no statistically significant linear correlation between LH and TSH. AMH showed a statistically significant negative linear correlation with TSH serum level, and there is also a statistically significant linear negative correlation between AFC and TSH serum level. Wu et al. (2021) showed that there was no significant correlation between thyroid hormones and ovarian reserve in women with infertility [4].

Zwain and Aziz (2026) stated that thyroid disorders were commonly detected among PCOS patients and the most common thyroid disorder in PCOS was hypothyroidism [34]. Sinha et al. (2013) reported that subclinical hypothyroidism (SCH) was present in 22.5% and clinical hypothyroidism was present in 2.5% of cases [43]. Among these hypothyroid patients, autoimmune hypothyroidism was present in 22.5% of patients. Janssen et al. (2004) observed a prevalence of autoimmune

thyroiditis (biochemically) in 26.9% of their 175 PCOS patients [31].

Other studies reported a higher prevalence of autoimmune thyroiditis in PCOS subjects. Thyroid pathologies were observed in half of the patients among 107 women with PCOS [11]. Kachuei et al. (2012) showed a significantly higher prevalence of autoimmune thyroiditis and goiter in PCOS patients than in control subjects [44]. In another study, 20 (40%) of 50 patients with PCOS showed subclinical hyperthyroidism [45]. In a study, Michalakis et al. (2011) reported prevalence of SCH was 23% among patients seeking infertility treatment [29].

Adamska et al. (2020) investigated the relationship between serum concentrations of TPOAbs and ovarian reserve in different PCOS phenotypes [46]. They noted no significant differences between the PCOS phenotypes and the control group of thyroid function tests (TSH, fT3, and fT4) and thyroid volume. Pervin et al. (2020) demonstrated that thyroid disorders are prevalent in 30% of PCOS patients. Hypothyroidism is almost three-fold more prevalent than hyperthyroidism [3].

Therefore, thyroid disorders can interact with the ovaries through both a direct effect on ovarian function and autoimmunity pathways. Assessment of serum TSH is mandatory in the workup of all PCOS women especially those presenting with menstrual irregularities. Therefore, TSH screening of all PCOS females of early reproductive age groups should be done to detect

subclinical or overt thyroid problems and to prevent the burden of infertility.

## Conclusion

We concluded that the majority of PCOS patients may have some degree of thyroid malfunction based on the results that were collected. Thyroid autoimmunity should be examined along with the evaluation of ovarian reserve. It is suggested that large sample sizes and multicenter cohort studies be used to ascertain the optimal AMH cutoff level for the ovarian response of sensitivity and specificity. Furthermore, before the results of the current work are made available globally, we advise validating it on a sizable patient population in subsequent research. To determine the accurate prevalence of thyroid dysfunction in patients with polycystic ovarian syndrome, a prospective multicenter investigation should be conducted. Additional investigation is necessary to determine the association between the length of impaired thyroid function and ovarian reserve. Further research to examine the molecular mechanisms that underlie the reduced ovarian reserve observed in women diagnosed with thyroid disorders. There should be a suitable procedure in place for ladies who are receiving stimulation. To prevent women from suffering negative consequences as a result of a lack of cooperation between reproductive treatment centers and other emergency departments where they might present in the event of hyperstimulation, all clinicians participating in patient care should be aware of this condition. To guarantee that care is not compromised and that no cases are overlooked, all

sinologists should be able to recognize the symptoms on ultrasound and work in tandem with

the gynaecologist and the patient.

**Funding:** This study is not funded.

**Conflicts of Interest:** All authors declare they have no conflicts of interest.

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