Study thyroid gland hormones in female with polycystic ovary syndrome: A Review

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Abstract

One metabolic syndrome is polycystic ovarian syndrome (PCOS) marked by polycystic ovary the hyperandrogenism, and anovulation. PCOS affects 6–10% of women in their reproductive years. It is a common condition. When no other cause can be identified, polycystic ovaries, hyperandrogenism, and/or irregular ovulation are the hallmarks of polycystic ovarian syndrome (PCOS). Since frank (overt) hypothyroidism is linked to insulin resistance, dyslipidemia, weight gain, anovulatory cycles, decreased levels of SHBG, and infertility, it shares several characteristics with the PCOS phenotype. Thyroid disorder screening is part of the recommended baseline screening for women who may be suspected of having PCOS, irregular menses, or infertility. Insulin resistance plays a role in PCOS pathogenesis.

Keywords: polycystic ovary syndrome; thyroid disease.

I. INTRODUCTION

Polycystic Ovarian Syndrome (PCOS) is a diverse illness with an unclear cause. Ovulatory dysfunction, "polycystic ovaries" on ultrasonographic or histopathological examination, hirsutism, hyperandrogenaemia, abnormal gonadotrophin concentrations, and, most recently, insulin resistance and hyperinsulinemia are just a few of the features that have been linked to the disorder[1].

One of the most prevalent endocrine conditions affecting women of reproductive age is the polycystic ovary (PCO) syndrome [2], which is caused by intricate and poorly understood interactions between genetic and environmental factors [3].

Currently, the condition is described as primarily hyperandrogenic, with patients exhibiting a range of symptoms and indicators that must include polycystic ovarian morphology, ineffective ovulation, and/or biochemical and/or clinical hyperandrogenism [4]. PCO syndrome has many side effects, including cutaneous manifestations, irregular menstruation and ovulation, and metabolic syndrome traits. Gaining more insight into this intricate condition and identifying possible disease biomarkers could be a big step toward preventing PCO syndrome, diagnosing it early, and treating it successfully[5].

Many patients with polycystic ovary syndrome (PCOS) are obese or overweight [6, 7], and the visceral fat in the abdomen is more common in these women [8]. Furthermore, PCOS affects 50–70% of women, and compensatory hyperinsulinism and insulin resistance are common[6], and insulin resistance is common in PCOS patients even when they are not obese [9].

The vicious cycle that these women may have started early in life or even during pregnancy explains the connection between excess androgen, abdominal fat, insulin resistance, and metabolic
abnormalities in women with PCOS. Specifically, in PCOS women, androgen excess that favors the accumulation of fat in the abdomen further encourages androgen secretion by the ovaries and adrenal glands. [10].

One of the main problems in the The precise measurement of androgens, especially testosterone, is necessary for the diagnosis of hyperandrogenic states like PCOS[11]. Hyperandrogenism, ovarian dysfunction, and metabolic abnormalities are the key aberrations in The balance of gonadotropins (GnRH), follicle stimulating hormone (FSH), and Luteinizing hormone (LH) is disrupted in PCOS due to a disturbance in the secretion pattern of GnRH . There is increased pulsatile release of GnRH [12]. The LH cells are more sensitive to faster GnRH pulse, and this creates a relatively high LH level compared to FSH.

In people without PCOS, the LH/FSH ratio is between 1 to 2 and in people with PCOS, the LH/FSH hormone of androgen production in adult Leydig cells[14]. raised to 2 or 3 folds [13]. LH is the main so the high LH level drives excess androgen production. FSH, on the other hand, stimulates the development of follicles in the ovary and in PCOS where its level is decreased relative to LH, many follicles do not mature and remain as cysts in the ovaries which is one of the common features seen in PCOS[15].This imbalance blocks release of eggs(ovulation) and without ovulation, progesterone hormone would not be produced. Normally, progesterone acts to slow down the LH pulses and this is missing in PCOS. Several defects in hormone regulation contribute to the overproduction of androgens by the ovaries. The theca cells of the ovaries produce excess androgen, and this is worsened by the increased insulin level and the increased luteinizing hormone (LH) level relative to FSH level which is seen in PCOS [16]. Patients with hypothyroidism often exhibit similar symptoms, including insulin resistance, weight gain, dyslipidemia, infertility, menstrual irregularities, and signs of hyperandrogenism [17, 18]. Researchers have concluded that PCOS is linked to a higher incidence of subclinical hypothyroidism (SCH) in PCOS patients’ thyroid function when compared to the normal population [17]. These characteristics are also associated with SCH, which is defined as an increased level of thyroid-stimulating hormone (TSH) combined with normal thyroid hormone levels and no evidence of hypothyroidism[17,18].

Polycystic ovarian morphology is observed in hypothyroidism. Therefore, one of the criteria used to rule out PCOS in any woman is thyroid disorders( In Figure 1) the underlying pathophysiology has been described. Increased thyroid stimulating hormone (TSH) and prolactin are caused by a rise in thyrotropin-releasing hormone (TRH) in primary hypothyroidism. By suppressing ovulation as a result of altered follicle stimulating hormone (FSH) and luteinizing hormone ratios as well as increased adrenal gland dehydroepiandrosterone, prolactin plays a role in polycystic ovarian morphology. Since elevated TSH affects FSH receptors indirectly, it also plays a role. It has also been proposed that hypothyroidism causes increased collagen deposition in the ovaries[19].
II. MATERIALS AND METHODS

Two endocrine disorders that are prevalent in the general population are thyroid conditions and PCOS, or polycystic ovarian syndrome. Despite having entirely distinct etiopathogenesis, hypothyroidism and PCOS share a number of characteristics. Primary hypothyroidism has been linked to changes in the ovary's cystic composition and an increase in ovarian volume. Conversely, it is becoming more widely acknowledged that thyroid issues are more prevalent in PCOS-affected women than in the general population [20]. It has not yet been determined whether this is because of a pathophysiological link between the two disorders or because there are common factors that predispose a person to both disorders.

In conclusion, polycystic ovarian morphology can result from hypothyroidism. Although the morphology may change depending on the degree and length of hypothyroidism, there is no proof that primary hypothyroidism causes PCOS.

3. PCOS

The Pathophysiology and Transmission of PCOS Across Generations

Daughters of PCOS mothers are five times more likely to inherit the syndrome [21]. Daughters of PCOS mothers are more metabolically and androgenically at risk, and their infant girls have a longer anogenital distance (AGD) [22,4]. In women with PCOS, baby AGD was predicted by mother testosterone levels [23]. It is unclear exactly how the daughters are exposed to HA, even though serum anti-Müllerian hormone (AMH) might be involved [24]. It was previously believed that the mechanism worked by causing the placenta's aromatase activity to increase through the action of AMH. It has been demonstrated that high AMH levels occur in women with PCOS during their second and third trimesters [25].
In humans, only one follicle is usually selected for sequential terminal maturation and ovulation due to the coordination of factors that influence follicular growth. There are roughly 6-7 million ovarian follicles at the peak of the gestational cycle, which decreases to 2-3 million primordial follicles at birth. The production of Theca cells produce androgens is stimulated by luteinizing hormone (LH), but insufficient follicle-stimulating hormone (FSH) levels and androgen conversion to estradiol prevent the choice of an influential follicle and lead to chronic anovulation [26]. Granulosa cells (GCs) secrete AMH, which is crucial for maintaining this equilibrium because it prevents the division of primordial into primary follicles [27, 4].

Numerous enzymes that produce steroids are expressed by the adrenal cortex, theca cell, and the zona reticularis. Hormones including androstenedione, DHEA (dehydroepiandrosterone), and DHEA sulfate are secreted by the zona reticularis. It is increasingly evident that 11-hydroxyandrostenedione is one of the steroidogenic repertoires of the adrenal and possibly theca cell, which is ultimately transformed into the powerful androgen 11-ketotestosterone [28]. When compared to control women, the serum levels of 11-oxygenated androgens were higher in PCOS-affected women, including 11-hydroxyandrostenedione, 11-ketoandrostenedione, 11-hydroxytestosterone, and 11-ketotestosterone [29].

4. PCOS And Thyroid Disorders Hormone

Do Women who suffer from PCOS are more likely to autoimmune thyroid disease (AITD) than those without PCOS? In the first comprehensive prospective study addressing this subject, 168 healthy controls, who were 28 years old on average [30] and 175 PCOS patients were included. Eight percent of controls and 26.9% of PCOS patients had increased TPO or Tg antibody levels, which are specific for HT. TPO and Tg antibody levels were also noticeably greater in young PCOS-affected women than in control groups, as was TSH.

Just 6.5% of controls and 42.3% of PCOS patients, respectively, exhibited the hypoechoic thyroid ultrasonography pattern associated with HT [30]. In this study, 36 PCOS patients and 11 controls had elevated antibodies to the thyroid and hypoechoic thyroid ultrasonography patterns (20.6 and 6.5%, respectively). When compared to controls, HT was more common in PCOS patients than shown to be three times higher when considered collectively [30].

Between 113 Italian patients (mean age = 24) and 100 controls (mean age = 27 vs. 8%), it was recently showed that PCOS-affected women had a higher prevalence of HT than did controls [31]. With a mean age of 24 years, the 168 young Brazilian women with PCOS exhibited a greater frequency 149 (88.7%) of them had normal thyroid function, while TSH levels in 19 (11.3%) cases were in the range of 4.5 to 10 mIU/l, suggesting subclinical hypothyroidism. This is a higher percentage compared to the general population in PCOS cases of subclinical hypothyroidism [32]. According to the Colorado Thyroid Disease Prevalence Study, 4-5% [30] of women in their 24s should have elevated TSH values.

A cross-sectional study conducted in Asia on 80 PCOS patients from Eastern India, ages 13 to 45, found that the prevalence of TPO-positive autoimmune thyroiditis was higher in these patients than in the 80 controls (22.5 vs 1.25%). Compared to controls, PCOS patients had greater mean TSH levels, a higher incidence of goiter (27.5 vs. 7.5%), and a higher frequency of a hypoechoic thyroid ultrasonography pattern (12.5 vs. 2.5%) [33]. A recent case-control study conducted in Iran has shown that the prevalence of clinically confirmed goiter and average quantity of TPO antibodies were higher in PCOS patients than in controls without PCOS [34].

TSH and Tg antibody concentrations, however, did not vary between PCOS and control subjects [34]. While thyroid disorders were more common in patients, only one Turkish study was unable to demonstrate that 84 PCOS-affected women had greater levels of TPO and Tg antibodies than 81
control participants [35]. Based on the bulk of research, we can say that PCOS and HT often co-occur.

III. Conclusion

Thyroiditis is thought to exacerbate PCO symptoms and be a risk factor for those with polycystic syndrome. Individuals who have autoantibodies against thyroid peroxidase and thyroglobulin are at risk of developing thyroid dysfunction in later life.

IV. REFERENCES


