

An Interrogative Study on Possible Clinical Interrelationship and Dietary Cautions among Polycystic Ovarian Syndrome and Thyroid Diseases

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Abstract

Polycystic ovarian syndrome (PCOS) is a widespread endocrine disorder with hyperandrogenic anovulation and polycystic ovaries, which is linked with diverse metabolic abnormalities and endocrine-related health issues, including thyroid disorders (TDs). The most recent studies on PCOS and TDs have been explored to find out the underlying association. Evidence revealed that TSH levels are correlated with BMI in females with PCOS. BMI impacts glucose and insulin levels, leading to increased levels of free testosterone, prolactin, luteinizing hormone to follicle-stimulating hormone ratio, triiodothyronine, and thyroxine. It also causes menstrual irregularities, oligomenorrhea, secondary amenorrhea, and polycystic ovaries. Moreover, three genetic polymorphisms have been identified as having a role in PCOS and TDs. Subclinical hypothyroidism and autoimmune thyroid diseases were also found to be more common in PCOS women, which may impair metabolic, hormonal, and reproductive functions. Changes in hormone levels brought on by TDS can increase ovarian volume, prevent ovulation, and lead to cysts. Furthermore, some hidden dietary cautions need to be addressed on having both clinical issues, like certain foods clinically proven beneficial for PCOS may cause trouble in the treatment of TDs and vice versa.

Keywords: polycystic ovarian syndrome, Thyroid disorders, Hypothyroidism, Hyperthyroidism, Autoimmune disease

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Introduction

1. Insight into Polycystic Ovarian Syndrome (PCOS)

Polycystic ovarian syndrome (PCOS) is a condition that affects the hormone levels of women during their years of childbearing (ages 15 to 44). PCOS affects between 2.2% to 26.7 % of women in this age range. PCOS is a “syndrome” that affects the ovaries and ovulation, and the term “polycystic” refers to “many cysts”. In PCOS, the ovaries develop a large number of tiny sacs known as follicles that are filled with fluid and hold an immature egg. The immature eggs are not fully developed hence the process of ovulation doesn't occur. In the absence of ovulation, the values of various hormones like luteinizing hormone (LH), progesterone, estrogen, and follicle-stimulating hormone (FSH) alter. In PCOS, the levels of androgen are enhanced while progesterone levels are lower as compared to normal conditions. Women with PCOS experience fewer periods than normal because excess male hormones interfere with the menstrual cycle (Watson & Kallen, 2021).

PCOS is considered a very common endocrine abnormality in women, with a global prevalence rate ranging from 5% to 15%. Generally, the condition seems to be a complicated genetic trait that dates back at least 50,000 years (Azziz, 2016). The worldwide annual incidence rates and age-standardized point prevalence for PCOS in 2019 were 1677.8 and 59.8 per 100,000, representing a 30.4% and 29.5% rise, respectively, since 1990. PCOS frequency and threshold rate increased in the ages 25-29 and 40-44, respectively (Safiri et al., 2022).

1.1. Symptoms and Diagnosis of PCOS

Common symptoms of PCOS as elaborated in Figure 1 (Chaudhuri, 2023) include no or irregular menstruation with ovarian eggs that are immature and fail to ovulate, scalp hair loss (alopecia), excess hair on face or body (hirsutism), face or body acne, dark skin patches (acanthosis nigricans), sleep apnea, anxiety, mood changes, low self-esteem, cardiovascular disease, poor body image and increased diabetes risk (Islander, 2018).

Women with PCOS create more masculine hormones than usual. This hormonal imbalance leads in individuals to miss menstrual periods and causes difficulty for them to conceive (Mukerjee, 2020). There is no single test for PCOS diagnosis; however, pelvic ultrasound exam using sonogram, ovarian exam for cysts & endometrial examination, and blood test for androgen hormone levels are suggested (Grigorescu et al., 2021).

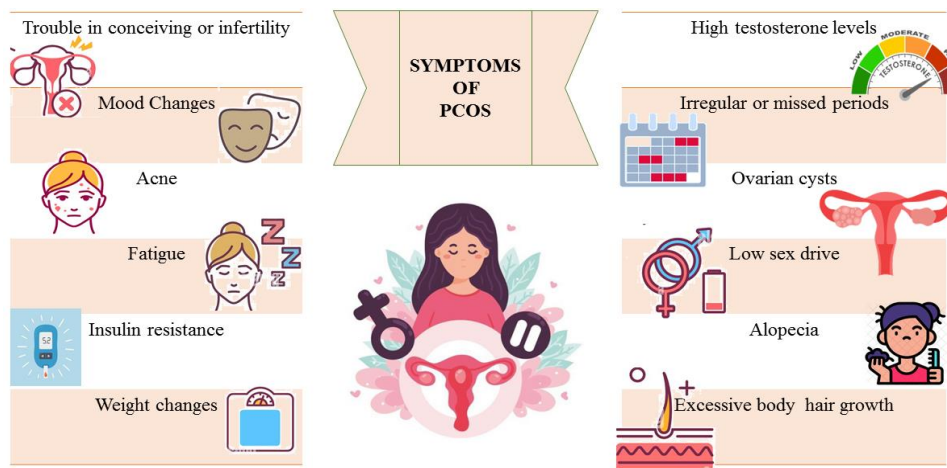


Fig. 1: Common symptoms of polycystic ovarian syndrome

1.2. Categories of PCOS

PCOS phenotypes can be classified into four categories as indicated in **Figure 2** (Sachdeva et al., 2019).

	Hyperandrogenism	Ovulatory Dysfunction	Polycystic Ovaries
Phenotype A <ul style="list-style-type: none"> Hyperandrogenism Ovulatory dysfunction Polycystic Ovaries 	Present	Present	Present
Phenotype B <ul style="list-style-type: none"> Hyperandrogenism Ovulatory dysfunction 	Present	Present	Absent
Phenotype C <ul style="list-style-type: none"> Hyperandrogenism Polycystic Ovaries 	Present	Absent	Present
Phenotype D <ul style="list-style-type: none"> Ovulatory dysfunction Polycystic Ovaries 	Absent	Present	Present

Fig. 2: Types of polycystic ovarian syndrome, along with their major presentations

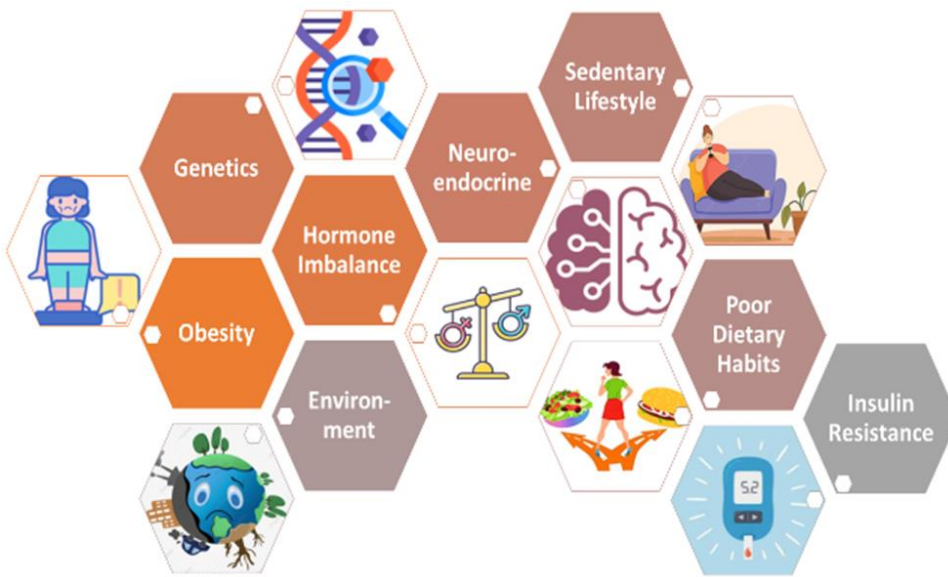
The "classic" version of the illness is represented by phenotypes A and B (hyperandrogenism + ovulatory abnormalities), with [A] and without [B] polycystic ovarian morphology [PCOM]. The so-called "ovulatory" PCOS (hyperandrogenism + PCOM alone) is phenotype C. Phenotype D is sometimes known as "non-hyperandrogenic" PCOS (ovulatory dysfunction plus PCOM alone). The extent to which the various phenotypes are associated with a high risk of metabolic dysfunctionalities and reproductive issues varies. A deeper knowledge of the adaptive causes of PCOS can provide additional insight into the variables underlying the etiology, occurrence, and duration of an illness that, at least on the surface, seems to be an adaptive contradiction (Azziz, 2016).

1.3. Causes and Comorbidities of PCOS

PCOS is one of the most common endocrine diseases in women of reproductive age, affecting at least 7% of infertile women and more than 10% of reproductive age women. PCOS can show up as many different phenotypes. Specific phenotypes, on the other hand, are linked to insulin resistance and an elevated possibility of acquiring metabolic syndrome over time (Mukerjee, 2020). Certain selective causative factors behind the PCOS pathogenesis have been depicted in Figure 3 (Witchel et al., 2019).

In a Mendelian Randomization study, testosterone level, obesity, fasting insulin, serum sex hormone-binding globulin concentrations, early menopause, anxiety, and baldness have all been linked to PCOS. Consequently, PCOS raises the chances of estrogen receptor-positive breast cancer while reducing the chances of endometrioid ovarian cancer and has no significant causative influence on coronary heart disease, type 2 diabetes, or stroke (Zhu & Goodarzi, 2022).

Fig. 3: Major causative factors behind PCOS pathogenesis



1.4. Treatment of PCOS and Its Associated Symptoms

In terms of medicinal approaches, combined contraceptives (COCs) are preferable with progestin for curing acne and hirsutism, as well as being beneficial in managing irregular periods. The World Health Organization advises that 35 g of estrogen combined with cyproterone acetate should be taken as a second option for hirsutism or severe acne (Sharpe et al., 2019). Letrozole was suggested as the primary treatment for women with PCOS and fertility problems because it promotes conception and fertility rates (Wang et al., 2019). Other than these, surgical procedures like bariatric surgery can also help to alleviate PCOS symptoms (Ortiz-Flores et al., 2018). Laparoscopic ovarian drilling is the least extensive surgical method that is used to rebalance and enhance ovarian function in PCOS women. Additionally, in a recent randomized controlled trial, obese women with PCOS received a three-component treatment consisting of exercise, diet, and cognitive behavioral therapy (CBT) (Hoeger et al., 2021).

1.5. Diet and PCOS Treatment

Dietary therapies, including low-calorie and low glycemic index (LGI) diets, are considered effective and safe for alleviating PCOS symptoms. Combining an LGI diet with exercise and omega-3 fatty acid supplementation has been shown to enhance HDL levels, increase sex hormone-binding globulin (SHBG), and reduce body fat. Flaxseed oil, a rich source of omega-3 alpha-linolenic acid, has demonstrated benefits in regulating sex hormones (Szczyk et al., 2021). Additionally, a meta-analysis indicates that long-term low-carbohydrate and ketogenic diets may help manage PCOS symptoms. The Mediterranean diet (MD), which is high in complex carbohydrates, fiber, and monounsaturated fats, is also suggested as an optimal dietary approach for PCOS (Chen & Pang, 2021).

Anti-inflammatory foods like extra virgin olive oil, berries, greens, and fatty salmon may help reduce inflammation-related symptoms. Additionally, the DASH diet is often recommended for PCOS patients to lower the risk of heart disease by restricting foods high in sugar and saturated fats (Dresden, 2021). Reducing dairy intake may aid some women with PCOS in weight loss and hormone balance, but those without sensitivity or intolerance can still consume dairy for essential nutrients like calcium and vitamin D (Santilli, 2019).

2. Introductory Insight into Thyroid Disorders (TDs)

Thyroid hormones, including thyroxine (T₄) and triiodothyronine (T₃), are secreted by the thyroid gland, which is made up of many grains and is situated in the lower neck region. The thyroid gland also has parafollicular cells, which are cells that make calcitonin, which, unlike T₃ and T₄, does not contain iodine (Mehdi et al., 2018). The small intestine absorbs iodine, a trace mineral that is necessary for the production of T₃ and T₄. Inadequate iodine can lead to iodine insufficiency and decreased thyroid hormone synthesis. Hypothyroidism, goiter, mental retardation, dementia, and myxedema can all be caused by a lack of iodine (Shahid & Sharma, 2022).

2.1. Hypo and Hyperthyroidism

An underactive thyroid gland is defined as hypothyroidism, which often causes cold sensitivity, bradycardia, exhaustion, and excess weight. In contrast, hyperthyroidism causes the thyroid gland to work more actively and appears as fine tremors, weight reduction, diarrhea, heat intolerance, and extreme muscle fatigue (Shahid & Sharma, 2022).

2.2. Symptoms and Diagnosis of Hypo- and Hyperthyroidism

The thyroid gland is necessary for our survival because hypothyroidism or its absence results in physical or mental damage and poor cold sensitivity. Its absence results in dwarfism and cognitive disability in children, while increased sweating, loss of weight, tachycardia, tremor, other mental issues, and anxiety are all symptoms of hyperthyroidism (Mehdi et al., 2018).

Thyroid-stimulating hormone (TSH) function is assessed through various diagnostic approaches. Blood tests measuring TSH, T₄, and T₃ levels are the first step in identifying thyroid disorders like hypothyroidism or hyperthyroidism. If thyroid dysfunction (TD) is detected, thyroid

antibody testing is conducted to diagnose autoimmune thyroid diseases (AITD) such as Graves' disease. Additionally, a thyroid scan helps evaluate the gland's shape, size, and location. Ultrasound is the most common method for examining thyroid nodules, and if a lump is found during imaging or a physical exam, a fine needle aspiration biopsy may be performed to check for cancer (Burch, 2017).

Radioiodine scanning, or radioiodine scintigraphy, is used to evaluate the thyroid gland's size and activity. The patient receives radioactive iodine, which helps assess thyroid function and structure before being excreted through urine. Another diagnostic method is a tissue biopsy, where a fine needle aspiration is used to collect a thyroid tissue sample for microscopic examination to detect malignant cells (Azziz, 2016).

2.3. Causes of TDs

Hypo and hyperthyroidism result from diseases that disrupt thyroid gland function. Approximately one in 4,000 babies is born with a malfunctioning thyroid, which, if untreated, may lead to physical and mental complications. Thyroid dysfunction can also stem from various conditions, including thyroiditis, an inflammation that affects hormone production; postpartum thyroiditis, a temporary condition affecting 5% to 9% of mothers after childbirth; and thyroid nodules, which are growths on or within the thyroid gland, with overactive nodules potentially causing hyperthyroidism (Warren, 2020).

Severe iodine deficiency can lead to goiter and hypothyroidism, as insufficient iodine levels hinder thyroid hormone production despite increased thyroid activity to absorb more iodine. Chronic iodine deficiency further stimulates thyroid function, increasing the risk of toxic nodular goiter and hyperthyroidism in affected populations (Zimmermann & Boelaert, 2015).

In iodine-deficient regions, common thyroid disorders include autoimmune thyroid disease (AITD), Hashimoto's thyroiditis, and Graves' disease. Hashimoto's thyroiditis, an autoimmune condition in which the immune system destroys thyroid cells, is one of the leading causes of hypothyroidism. The second most common cause is inadequate dietary iodine intake. Women are at least ten times more likely than men to develop Hashimoto's thyroiditis (Mincer & Jialal, 2022).

Graves' disease is an autoimmune disorder affecting the thyroid gland and is the primary cause of hyperthyroidism in 60% to 80% of cases. The estimated lifetime risk is 0.5% for men and 3% for women. The condition is caused by thyroid-stimulating immunoglobulin (TSI), also known as thyroid-stimulating antibody (TSAb), which binds to TSH receptors on thyroid cells. This stimulates TSH, leading to excessive thyroid hormone production and thyroid gland enlargement, resulting in hyperthyroidism (McCann & Weetman, 2021).

2.4. Treatments of TDs

Hyperthyroidism results from excessive thyroid hormone production and can be treated with antithyroid medications like methimazole and propylthiouracil, surgical thyroidectomy, or radioactive iodine ablation, the most common treatment in the United States (Kravets, 2016). For hypothyroidism, levothyroxine remains the gold standard treatment, while adding T₃ is not recommended, even for patients with persistent symptoms and normal TSH levels (Wilson et al., 2021).

To ensure complete absorption of medicines by the body, thyroid medication must be taken on an empty stomach. Consume it at least three to four hours after dinner or at least 30 to 60 minutes before breakfast. Taking this medication within 4 hours of eating foods containing calcium or iron is not advised (Kubala, 2019).

2.5. Diet and TD's Treatment

While hormonal issues cannot be reversed, their symptoms can be managed with appropriate dietary supplements. Thyroid hormone synthesis depends on essential nutrients such as iodine, iron, vitamin D, vitamin B12, and selenium (Dahiya et al., 2022). Healthcare professionals must consider interactions between dietary supplements and thyroid medications. For instance, calcium supplements can inhibit thyroid medication absorption and should be taken at least four hours apart. Caffeine and dietary fiber supplements should be consumed at least one hour before thyroid medication, while chromium picolinate, used for blood sugar control and weight management, should be taken three to four hours apart. Additionally, excessive intake of flavonoids found in fruits, vegetables, and tea may reduce thyroid activity (Babiker et al., 2020).

Cruciferous vegetables and soy products can interfere with thyroid hormone synthesis, but heating these goitrogenic foods may help reduce their impact by breaking down the goitrogenic compounds into less harmful metabolites (Babiker et al., 2020). Additionally, individuals with dairy and gluten allergies may benefit from avoiding these foods, as doing so can improve levothyroxine absorption (Larsen et al., 2022).

Maintaining serum selenium levels through diet can also help to avoid thyroid illness. Another nutrient, zinc, may have particular advantages for those with hypothyroidism. One small-scale investigation found that supplementing with zinc, either alone or in conjunction with selenium, enhanced thyroid function in hypothyroid females (Kubala, 2019).

3. A Collective Clinical Insight into PCOS and TDs

A worse metabolic profile was observed in PCOS and subclinical hypothyroid women. Thyroid autoantibody positivity rates were greater in PCOS women in comparison with the control group. Compared to controls, PCOS patients had a 3.6 times higher prevalence of overt TDs, and they received 3 times more medications for thyroid (Glintborg & Andersen, 2020). Two questions need to be addressed between thyroid conditions and PCOS: How are the ovaries affected by thyroid conditions? How does PCOS affect the thyroid?

A. How are ovaries affected by thyroid conditions?

In hypothyroidism, ovarian morphology can become polycystic, making thyroid issues an exclusion factor when diagnosing PCOS. Primary hypothyroidism leads to increased thyrotropin-releasing hormone (TRH), which raises prolactin and TSH levels. Prolactin suppresses ovulation by altering the FSH-LH ratio and increasing adrenal dehydroepiandrosterone, contributing to polycystic ovarian morphology. Additionally, elevated TSH affects FSH receptors, further influencing ovarian changes. While the severity of hypothyroidism can impact ovarian morphology, there is no evidence that hypothyroidism directly causes PCOS (Singla et al., 2015).

B. How does PCOS affect the thyroid?

The pathophysiological link between these two conditions has not yet been identified. The pathophysiological basis for the association between thyroid function and obesity is uncertain, still, there is sufficient data to conclude that individuals with high BMI have greater TSH levels. Obesity is associated with a changing environment, which includes elevated pro-inflammatory markers and insulin resistance. Through unidentified mechanisms, this lowers pituitary deiodinase-2 activity, leading to a relative T₃ deficiency and a rise in TSH levels (Singla et al., 2015).

3.1. Clinical Studies on PCOS with TDs

Thyroid issues and PCOS have been related for many reasons as depicted in **Figure 4** (Harada, 2022). A prospective cross-sectional study on reproductive ages (15 - 45 years) was carried out. In total, oligomenorrhea affected 54 of the participants. 45 people had menstrual irregularities. 120 (80 %) of the PCOS were married, 58 (48.33%) had primary infertility, and there were 11 (9.17 %) cases of secondary infertility. 105 (70%) of the total (150) study participants were euthyroid, 33 (22.0 %) were hypothyroid, and 12 (8 %) were hyperthyroid. The study concludes that TDs affect 30% of women with PCOS. Hypothyroidism affects approximately three times as many people as hyperthyroidism (Pervin et al., 2020).

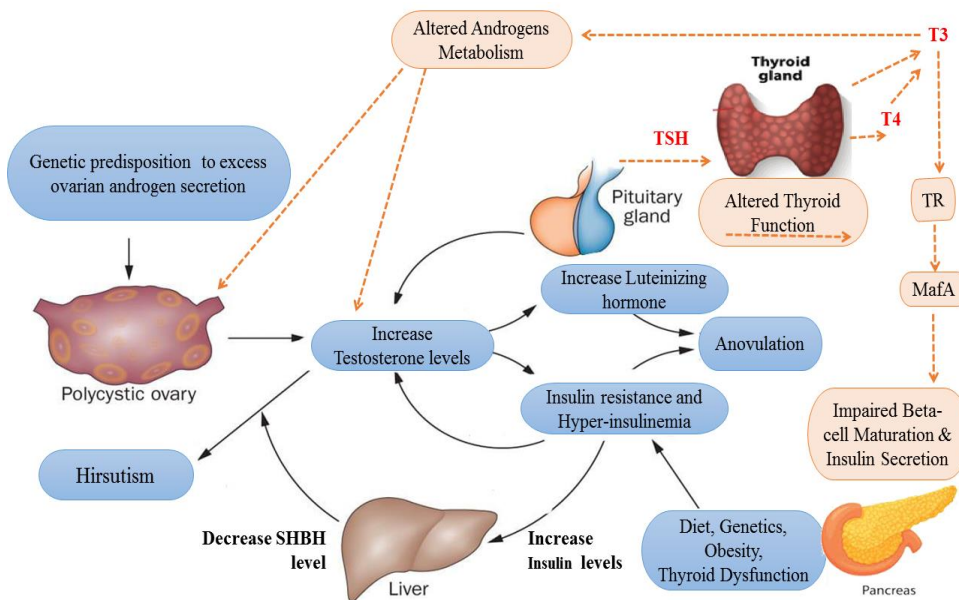


Fig. 4: Few possible Thyroid and PCOS interplay

Thyroid disorders (TDs) and PCOS are closely linked endocrine conditions, with PCOS affecting 6%-10% of women of reproductive age. The prevalence of Hashimoto's thyroiditis is three times higher in women with PCOS, and goiter is commonly associated with endocrine disorders (Anwaar & Jabeen, 2022). A study investigating serum TSH levels in women with PCOS found that 17% had subclinical hypothyroidism based on the Rotterdam 2003 criteria. Among obese women with PCOS, there was no statistical difference in LH:FSH ratio or insulin resistance between euthyroid and subclinical hypothyroid individuals. However, TSH levels significantly correlate with BMI in PCOS patients, contributing to various clinical symptoms (Patidar & Sapka, 2021).

It is crucial for detecting thyroid abnormalities in PCOS women. Serum-free testosterone, LH, and cholesterol levels are typically elevated in hypothyroidism and PCOS. As seen on ultrasonography, women with hypothyroidism who also have PCOS usually have larger ovaries as well as the existence of reciprocal polycystic ovaries. By stabilizing thyroid hormone levels, thyroid hormone replacement therapy reduces ovarian sores recurrence and ovarian volume (Vijey Aanandhi & John, 2018).

A cross-sectional investigation showed the incidence and causes of various TDs in PCOS women. The Rotterdam classification was used to diagnose PCOS between the ages of 18 and 35 years. Serum LH, FSH, TSH, fasting blood sugar, insulin levels, serum testosterone, serum prolactin levels, and dehydroepiandrosterone were all measured. It was found that the mean age of women with PCOS was 21.5 ± 4.7 years. All of the patients reported monthly irregularities, including oligomenorrhea, or secondary amenorrhea. Clinical hirsutism was observed in 72.5 % of the cases (Ferriman-Gallwey score >7). In 62.5 % of the cases, the LH/FSH ratio was more than 2. TSH levels in participants were 4.39 ± 1.91 (Kumar et al., 2020).

3.2. Clinical Studies on PCOS with SCH

While some studies suggest a link between subclinical hypothyroidism (SCH) and PCOS, this association is not widely recognized. A study involving 716 patients evaluated clinical, metabolic, and hormonal parameters using backward multiple regression. Both SCH and PCOS cases showed similar anthropometric and ovarian morphology, with PCOS primarily increasing C-peptide levels. There was no significant difference in insulin resistance or glucose intolerance between PCOS and PCO with SCH. TSH was correlated with insulin, prolactin, and total cholesterol, highlighting the need to standardize thyroid dysfunction exclusion criteria before diagnosing PCOS, as SCH should not automatically rule out PCOS (Freitas De-Medeiros et al., 2017). A meta-analysis further found that SCH patients with PCOS had higher HOMA-IR, LDL, FBG, FCP, TG,

TC, and PRL levels, while HDL and LH levels were lower (Xing et al., 2021).

A study was conducted in Pakistan to ascertain the occurrence of SCH in PCOS patients. Increased TDs, such as SCH, have been linked to PCOS. The endocrinology ward of a tertiary care hospital in Pakistan underwent case-control research from March 2020 to April 2021. 200 girls with PCOS who were aged between 18 to 30 years were involved in the study. The control group for the study consisted of 200 women without PCOS. After recording their demographics, blood was drawn from their cubital vein and submitted to the lab to be analyzed for TSH, T₄, and T₃. The report shows that participants were found to have a greater prevalence of SCH. People with PCOS were found to have SCH more frequently than participants without PCOS (43.5 % vs. 20.5 %) (Raj et al., 2021).

Hypothyroidism is more common in women with PCOS, making it essential to understand its clinical implications. A case-control study involving 190 women with PCOS phenotype A divided participants into three groups: PCOS with SCH (38 women), PCOS with normal thyroid function (76 women, control group), and PCOS with SCH on thyroid replacement therapy (76 women). Oral glucose tolerance, fasting insulin, fasting glucose, and serum lipid tests were conducted. There were no statistically significant differences in thyroid function measures among the groups. However, SCH in PCOS was associated with moderate changes in serum lipids, while BMI had a stronger influence on blood sugar and insulin levels (Li et al., 2022).

A study was conducted to investigate the connection between subclinical hypothyroidism with present and absent anti-thyroid antibodies, and PCOS symptoms and metabolic profile. The study's purpose is to see if SCH, with or without anti-thyroid antibodies (ATA), impacts the PCOS phenotypes and alters biochemical or clinical indicators. Retrospective cohort research took place in a tertiary referral center. The study included 367 women with PCOS, with 114 (31.1%) having SCH diagnoses and 16 (4.4%) having autoimmune thyroiditis diagnoses (AIT). In PCOS, SCH changes metabolic but not hormonal factors. The risk of PCOS is not disregarded by the SCH diagnosis. The possibility of a positive ATA had a rare effect (Gawron et al., 2022).

3.3. Clinical Studies on PCOS with AITD

PCOS has been associated with autoimmune disorders, particularly autoimmune thyroid disease (AITD). The prevalence of AITD in women with PCOS varies between 18% and 40%, depending on ethnicity and diagnostic criteria. A study by Romitti et al. (2018) compared PCOS patients to a reference group without PCOS, assessing methodological quality using the Ottawa-Newcastle Scale. AITD was found in 9.72% of control patients and 26.03% of PCOS patients, indicating a higher prevalence in those with PCOS. Given its potential impact on thyroid function, thyroid screening is recommended for women with PCOS.

A study to find TDs in PCOS patients showed Hashimoto's thyroiditis (HT), is far more prevalent in PCOS women than in the general population. Genetic and autoimmune histories have been identified as probable etiological variables. It has been determined that three genetic polymorphisms contribute to both PCOS and HT. Anovulatory cycles and high estrogen levels during pregnancy are the two main causes of high estrogen-to-progesterone ratios. The insufficient intake of vitamin D may contribute to the emergence of HT and PCOS. Further investigation into the stated common etiological variables linked to reproductive issues in HT and PCOS (Kowalczyk et al., 2022). The purpose of another study was to evaluate the hormonal, metabolic, and reproductive state of individuals with PCOS and associated thyroid pathology. The result shows that people suffering from diffuse endemic goiter (DEZ) and hypothyroidism as a result of chronic autoimmune thyroiditis (AIT) were included in the study. Furthermore, studies suggested that weight gain complaints were relatively common in the observation group (Ekaterina, 2019). Several studies found an effect of autoimmune thyroiditis on Metabolic, Immunological, Genetic, Hormonal, and Reproductive Factors that are linked to the prevalence of thyroid issues in women with PCOS are given below:

A. Metabolic Factors

Following Rotterdam's criteria for the signs and symptoms associated with the condition, it is observed that PCOS patients are obese and have hyperinsulinemia. Case-control research finds that obesity alters the ecology of the body, by increasing pro-inflammatory biomarkers and insulin sensitivity. Whereas, deiodinase-2, which is a major checkpoint in thyroid hormone signaling and controls cell-specific thyroid hormone activation, decreases its activity at the pituitary level and seems to cause a relative T₃ shortfall and an increase in TSH levels (Singh et al., 2020).

B. Immunologic Factors

Many of the investigations and procedures discovered through this search strategy also correlated with immune-related parameters including increased serum TSH and antibodies like anti-TPO and anti-TG. Numerous studies have shown that thyroid-related variables are more prevalent than non-specific autoimmune causes (Singh et al., 2020).

C. Genetic Factors

Polymorphisms in genes associated with PCOS, including fibrillin 3 (FBN3), gonadotropin-releasing hormone receptor (GnRHR), and CYP1B1 (which is involved in estradiol hydroxylation), are believed to play a role in the pathophysiology of both Hashimoto's thyroiditis (HT) and PCOS (Singh et al., 2020).

D. Hormonal Factors

One study shows that PCOS is also linked to hormonal problems caused by decreased progesterone levels, which the hypothalamus cannot regulate and which raise the pulse rates of gonadotropin-releasing hormone (GnRH) and LH (Singh et al., 2020). The hormonal issues that are common for the classic phenotype of PCOS, including a rise in total testosterone, a decrease in free androgen index, and a reduction in sex-steroid-binding globulin, were demonstrated to be made worse by euthyroid failure. The absence of a connection between tissue transglutaminase (TTG) and LH, LG, or FSH suggests that TD was caused by distinct mechanisms (Arkhyapkina et al., 2018).

E. Reproductive markers

Adolescent girls with PCOS and concurrent autoimmune thyroiditis are investigated for their metabolic and hormonal profiles. The analysis included 80 euthyroid PCOS patients. The study group comprised eighteen girls with AIT, while the test subjects included 62 girls without AIT. The amount of anti-thyroglobulin (anti-TG) and thyroid peroxidase (anti-TPO) antibodies were assessed. AIT was also identified using ultrasound imaging of the thyroid gland. The study's findings revealed no differences in metabolic profiles across the groups. Estradiol might contribute to the emergence of the autoimmune response, as shown by the elevated amounts of this hormone seen in girls with PCOS and AIT (Skrzyńska et al., 2021). In another study a total of 827 PCOS patients were seen for reproductive issues. Patients who presented predominantly with thyroid issues were eliminated. Laboratory testing and thyroid ultrasonography were used to screen all patients for the presence of AIT. AIT and PCOS patients had decreased rates of elevated testosterone, free androgen index, and hyperandrogenemia than those with only PCOS. In addition, testosterone levels were decreased in PCOS individuals who received AIT. Compared to controls, PCOS patients had a higher prevalence of AIT. AIT individuals with PCOS generally have lower hyperandrogenemia and hyperandrogenism (Ulrich et al., 2018).

4. Dietary Cautions or Collective Dietary Insight into PCOS + TDs

We all know that PCOS and TDs are inflammatory disorders. To minimize inflammation, it is critical to follow an anti-inflammatory diet. This includes spices like turmeric, colorful vegetables, fish, nuts, and gluten-free, dairy-free foods (Johnson, 2022; Luthra, 2020). High-fiber diets benefit PCOS by regulating blood sugar, while goitrogens found in certain foods can affect thyroid function (Whelan, 2021). Flaxseed is a miracle food for PCOS-afflicted ladies, but Flaxseed also contains goitrogens and cyanogen that may impact the development and functioning of the thyroid (Lay, 2017) Soy and processed foods should be avoided due to their hormonal and thyroid impacts (Antia, 2019).

5. Conclusion

Results revealed adequate evidence claiming that SCH or AITD are more prevalent in PCOS women. The majority of people with PCOS-SCH had thyroid dysfunction symptoms, which could lead to metabolic or endocrine disorders including infertility, dyslipidemia, insulin resistance, obesity, ovulatory dysfunction, abnormal menstrual cycles, and higher HOMA-IR values. Additionally, it appears that SCH and PCOS both are independent risk factors for metabolic syndrome and may lead to more worsened pathophysiological conditions resulting in increased years lived with disabilities and comorbidities. The clinical interrelationship of hyperthyroidism needs to be addressed in scientific investigations. Furthermore, some hidden dietary cautions need to be addressed, as certain foods that may have been proven clinically beneficial for PCOS may cause trouble in the treatment of TDs and vice versa. Inclusively, it is necessary to tackle either of the diseases very carefully giving special consideration to the prevention of the other one, particularly concerning fertility issues in young females, dietary recommendations, and nutritional remedial approaches prescribed.

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