

Coexistence of PCOS and Thyroid Disorders: An Endocrine Interplay

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Introduction

Polycystic Ovary Syndrome (PCOS) is the most prevalent endocrine disorder among women of reproductive age, characterized by oligo/anovulation, hyperandrogenism and polycystic ovarian morphology. Increasingly, clinical attention has turned toward the frequent coexistence of PCOS and thyroid dysfunction, particularly autoimmune thyroiditis and subclinical hypothyroidism. This association is not merely coincidental but appears to reflect a complex endocrine interplay with shared immunological, metabolic and hormonal pathways. Thyroid hormones play a crucial role in reproductive physiology and disturbances in thyroid function can exacerbate menstrual irregularities, infertility and metabolic dysfunction core features of PCOS. Conversely, the chronic inflammation, insulin resistance and hormonal dysregulation seen in PCOS may influence thyroid function or promote autoimmune activity. Studies have shown a higher prevalence of Hashimoto's thyroiditis and elevated thyroid antibodies in women with PCOS compared to the general population. Subclinical hypothyroidism in these patients may go unrecognized, yet it contributes to worsening insulin resistance, lipid abnormalities and anovulation. The co-occurrence of these two syndromes can create a vicious cycle, amplifying metabolic risk and reproductive impairment. This perspective explores the bidirectional relationship between PCOS and thyroid disorders, highlighting the need for comprehensive screening and integrated management strategies. Understanding this overlap offers opportunities to optimize hormonal balance, fertility outcomes and long-term health in affected women [1].

Description

The pathophysiological links between PCOS and thyroid disorders are multifaceted and begin with shared genetic and immunological susceptibilities. Both conditions exhibit strong familial aggregation and involve dysregulation of immune function. In PCOS, low-grade systemic inflammation and elevated levels of pro-inflammatory cytokines such as TNF- α , IL-6 and CRP are commonly reported. This inflammatory milieu may predispose to thyroid autoimmunity by facilitating the breakdown of self-tolerance. Indeed, several studies have reported significantly higher rates of Thyroid Peroxidase Antibody (TPOAb) positivity and Thyroid-Stimulating Hormone (TSH) elevation in women with PCOS, even when overt thyroid dysfunction is absent. Genetic studies suggest that polymorphisms in genes such as FOXP3, CTLA4 and PTPN22 may confer risk for both conditions by altering immune regulation. Additionally, estrogen excess in PCOS may influence immune reactivity, further contributing to thyroid autoimmunity. On the metabolic front, insulin resistance a hallmark of PCOS is known to affect thyroid hormone metabolism by altering deiodinase activity and increasing leptin levels, which in turn may influence TSH secretion. These intersecting

mechanisms demonstrate that the coexistence of PCOS and thyroid dysfunction is likely due to a common underlying endocrine-immune network rather than independent co-morbidity [2].

Thyroid dysfunction, especially hypothyroidism, can significantly influence the clinical manifestations of PCOS. Hypothyroidism may worsen menstrual irregularities and ovulatory dysfunction by disrupting gonadotropin secretion, lowering Sex Hormone-Binding Globulin (SHBG) and increasing peripheral estrogen levels through aromatization. The resultant estrogen dominance contributes to anovulation and polycystic ovarian morphology. Additionally, hypothyroidism-induced hyperprolactinemia can further suppress the hypothalamic-pituitary-ovarian axis. From a metabolic perspective, thyroid hormone deficiency impairs glucose utilization, worsens lipid profiles and promotes weight gain factors that already burden PCOS patients. Subclinical hypothyroidism may not produce overt symptoms but can subtly exacerbate insulin resistance and hyperandrogenism. The co-presence of thyroid dysfunction may also impact fertility treatments, as untreated hypothyroidism reduces conception rates and increases miscarriage risk. Therefore, thyroid evaluation should be considered a routine part of PCOS assessment, particularly in those with infertility, irregular menses, or poor response to ovulation induction. Addressing even mild thyroid dysfunction may improve reproductive outcomes and metabolic control in women with PCOS [3].

Conversely, PCOS may affect thyroid physiology and potentially contribute to the development or progression of thyroid disorders. Hyperinsulinemia, often present in PCOS, has been shown to stimulate thyroid cell proliferation and may play a role in goiter formation. Insulin and Insulin-Like Growth Factor-1 (IGF-1) receptors are expressed on thyroid tissue and their activation may influence thyroid growth and hormone output. Moreover androgen excess in PCOS could impact immune function and thyroidal responsiveness, although the exact mechanisms remain under investigation. Some studies have noted increased thyroid volume and nodularity in PCOS patients, even in the absence of biochemical thyroid dysfunction. The metabolic dysregulation characteristic of PCOS may also accelerate thyroid disease progression or worsen symptom expression. Additionally, the psychological stress and altered cortisol rhythms seen in PCOS may contribute to Hypothalamic-Pituitary-Thyroid (HPT) axis dysregulation. These observations suggest that PCOS is not just a passive associate but may actively influence thyroid pathophysiology. More research is needed to determine whether treatment of PCOS-specific features like insulin resistance or hyperandrogenism can positively affect thyroid function [4].

The clinical implications of coexisting PCOS and thyroid disorders are profound, underscoring the importance of interdisciplinary care. Women with both conditions are at higher risk of metabolic syndrome, cardiovascular disease, infertility and poor pregnancy outcomes. Managing one disorder in isolation may be insufficient, as untreated thyroid dysfunction may blunt the benefits of PCOS therapies and vice versa. Integrated treatment approaches should include levothyroxine replacement for hypothyroidism, insulin-sensitizing agents such as metformin and lifestyle interventions focusing on diet and physical activity. Ovulation induction agents may be more effective when thyroid function is optimized. Regular monitoring of TSH, TPOAb and metabolic parameters is essential for long-term management. Furthermore, patient

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Received: 02 June, 2025, Manuscript No. rtr-25-171746; Editor assigned: 04 June, 2025, PreQC No. P-171746; Reviewed: 16 June, 2025, QC No. Q-171746; Revised: 23 June, 2025, Manuscript No. R-171746; Published: 30 June, 2025, DOI: 10.37421/2684-4273.2025.9.117

education regarding the overlapping symptoms and risks can empower women to seek timely evaluation. Clinicians should maintain a high index of suspicion for thyroid abnormalities in all PCOS patients, especially those with treatment resistance, weight gain, or worsening lipid profiles. Early detection and a holistic therapeutic approach can significantly improve outcomes for women grappling with this dual endocrine burden [5].

Conclusion

In conclusion, the coexistence of PCOS and thyroid disorders reflects a complex interplay of endocrine, metabolic and immune pathways. This intersection contributes to worsened reproductive, metabolic and psychological outcomes and requires clinicians to adopt a comprehensive, multidisciplinary management strategy. Early identification of thyroid dysfunction in PCOS patients and vice versa is crucial for preventing complications and enhancing treatment efficacy. As research continues to unravel the shared mechanisms linking these conditions, individualized care based on hormonal profiles, antibody status and metabolic risk will become increasingly central. Recognizing and addressing this interplay is essential for improving the quality of life and long-term health of affected women.

Acknowledgement

None.

Conflict of Interest

None.

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How to cite this article: Miura, Rosario. "Coexistence of PCOS and Thyroid Disorders: An Endocrine Interplay." *Rep Thyroid Res* 09 (2025): 117.