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REPRODUCTIVE OUTCOMES IN WOMEN WITH AUTOIMMUNE THYROIDITIS

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Abstract

Autoimmune thyroiditis (AIT), commonly referred to as Hashimoto's thyroiditis, is a chronic immune-mediated disorder characterized by progressive destruction of thyroid tissue, often resulting in hypothyroidism. Women of reproductive age are predominantly affected, and AIT has been associated with reduced fertility and adverse pregnancy outcomes. This review examines the mechanisms by which autoimmune thyroiditis influences reproductive health, including hormonal dysregulation, menstrual disturbances, ovulatory dysfunction, diminished oocyte quality, and increased risks of miscarriage and preterm delivery. Diagnostic strategies, including evaluation of thyroid hormone levels and thyroid autoantibodies, are discussed, alongside therapeutic approaches with emphasis on levothyroxine replacement therapy. The role of assisted reproductive technologies (ART), particularly in vitro fertilization (IVF), is analyzed, highlighting the importance of achieving euthyroidism and monitoring thyroid autoantibody titers. Evidence indicates that timely diagnosis and proper management significantly improve reproductive outcomes in women with autoimmune thyroiditis.

Keywords: Hashimoto's thyroiditis, hypothyroidism, autoimmune thyroiditis, fertility, reproductive health, menstrual irregularities, ovulatory dysfunction, thyroid autoantibodies, in vitro fertilization,

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Introduction

Autoimmune thyroiditis is the leading cause of hypothyroidism in women of reproductive age. It is characterized by lymphocytic infiltration of the thyroid gland and the production of thyroid-specific autoantibodies, primarily anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin (anti-TG) antibodies. These autoimmune processes can progressively impair thyroid function, leading to subclinical or overt hypothyroidism. Thyroid hormones are essential regulators of metabolic processes and reproductive function, influencing the hypothalamic–pituitary–ovarian axis, follicular maturation, ovulation, endometrial receptivity, and early embryogenesis. Dysregulation of thyroid homeostasis, particularly in the context of autoimmunity, is associated with subfertility, recurrent pregnancy loss, and obstetric complications. Consequently, autoimmune thyroiditis should be considered a significant endocrine factor during infertility evaluation.

This article aims to review current evidence on the impact of autoimmune thyroiditis on female fertility, discuss diagnostic and therapeutic approaches, and examine the role of assisted reproductive technologies in affected women.

Pathophysiology of Autoimmune Thyroiditis

Autoimmune thyroiditis arises from aberrant immune activity in which thyroid-specific autoantibodies mediate cytotoxic damage to thyroid follicular cells. Over time, this leads to impaired synthesis of thyroxine (T4) and triiodothyronine (T3), resulting in hypothyroidism. Thyroid hormones exert multiple effects on reproductive physiology:

- Regulation of gonadotropin-releasing hormone (GnRH) and luteinizing hormone (LH)/follicle-stimulating hormone (FSH) secretion.
- Support of folliculogenesis and oocyte maturation.
- Promotion of endometrial receptivity.

Hypothyroidism disrupts these processes, contributing to ovulatory dysfunction, altered menstrual cycles, and compromised implantation. Furthermore, thyroid

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autoantibodies may directly interfere with ovarian function or endometrial immune tolerance, exacerbating reproductive challenges.

Diagnosis of Autoimmune Thyroiditis

Evaluation involves both biochemical and immunological assessment:

- TSH: Elevated levels indicate hypothyroidism.
- Free T4 and Free T3: Determine the degree of thyroid hormone deficiency.
- Anti-TPO and Anti-TG antibodies: Detect autoimmune activity against the thyroid.

Early detection is essential, particularly for women planning conception or undergoing fertility treatment.

Management

Thyroid Hormone Replacement Therapy

Levothyroxine is the primary treatment for hypothyroid women with autoimmune thyroiditis. The therapeutic goal is to achieve euthyroidism, with TSH levels ideally maintained below 2.5 mIU/L in women attempting conception or during pregnancy. Adequate therapy restores ovulatory function, regularizes menstrual cycles, and reduces the risk of miscarriage and obstetric complications.

Immunomodulatory Considerations

In selected patients with high antibody titers or recurrent pregnancy loss, immunomodulatory therapy (e.g., corticosteroids) may be considered. Such interventions require careful individualization due to potential risks and limited supporting evidence. For women who fail to conceive naturally, ART, particularly IVF, offers an effective alternative. Success depends on preconception endocrine optimization:

1. Achieving Euthyroidism: Proper thyroid hormone levels improve oocyte quality, endometrial receptivity, and implantation potential.
2. Monitoring Autoantibody Levels: Elevated thyroid autoantibodies may reduce IVF success and increase miscarriage risk.

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3. Close Hormonal Surveillance: Ovarian stimulation can affect thyroid function; dose adjustments of levothyroxine may be necessary.

4. Immunomodulation: In selected patients, adjunctive treatments (low-dose aspirin, heparin, or corticosteroids) may enhance implantation success.

When managed appropriately, IVF success rates in women with autoimmune thyroiditis are comparable to euthyroid women without thyroid pathology.

Prognosis

With timely diagnosis and proper endocrine management, the majority of women with autoimmune thyroiditis can achieve successful conception and carry pregnancies to term. Poorly controlled hypothyroidism and persistently elevated autoantibody levels remain major risk factors for infertility and adverse obstetric outcomes.

Conclusion

Autoimmune thyroiditis poses significant challenges to female reproductive health, affecting hormonal balance, ovulatory function, and pregnancy maintenance. However, early detection, effective thyroid hormone replacement, and individualized fertility management, including ART when indicated, significantly improve reproductive outcomes. Close collaboration between endocrinologists and reproductive specialists is essential to optimize fertility and ensure healthy pregnancies in affected women.

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