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OVARIAN CHANGES AND REPRODUCTIVE ORGAN STRUCTURE IN HYPOTHYROIDISM

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Abstract
Hypothyroidism, a condition characterized by reduced thyroid hormone secretion, profoundly influences multiple physiological systems, particularly the female reproductive system. The thyroid gland plays a vital role in maintaining homeostasis of reproductive hormones through its interaction with the hypothalamic-pituitary-gonadal axis. Alterations in thyroid function can disrupt ovulation, menstrual cycles, and fertility, leading to significant morphological and functional changes in the ovaries and other reproductive organs. This article aims to provide an in-depth analysis of ovarian and reproductive organ alterations observed in hypothyroidism, with a focus on histopathological, endocrine, and molecular perspectives. Through an examination of recent findings, this study highlights how thyroid hormone deficiency contributes to ovarian dysfunction, including follicular atresia, luteal insufficiency, and stromal fibrosis.

Keywords: Hypothyroidism, ovarian dysfunction, reproductive system, thyroid hormones, fertility, histopathology.

Introduction

The thyroid gland is an essential endocrine organ that regulates metabolism, growth, and development through the secretion of thyroxine (T4) and triiodothyronine (T3). These hormones exert widespread effects on almost every organ system, including the female reproductive organs. In women, normal thyroid function is crucial for maintaining reproductive health and fertility. Hypothyroidism, characterized by insufficient thyroid hormone production, disrupts the delicate hormonal interplay between the thyroid gland and the

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hypothalamic-pituitary-gonadal axis, leading to significant alterations in ovarian function and reproductive organ morphology. The effects of hypothyroidism on the reproductive system have been well-documented in both human and animal studies, revealing complex interactions that ultimately impair fertility and reproductive outcomes.

In a physiological context, thyroid hormones directly and indirectly regulate ovarian function. They influence the synthesis and metabolism of sex steroids, modulate the sensitivity of ovarian follicles to gonadotropins, and participate in the regulation of folliculogenesis and ovulation. In hypothyroid states, the hypothalamus and pituitary gland respond to decreased thyroid hormone levels by altering gonadotropin-releasing hormone (GnRH) secretion, which leads to fluctuations in luteinizing hormone (LH) and follicle-stimulating hormone (FSH) release. This hormonal imbalance disturbs the normal maturation of ovarian follicles, often resulting in anovulation or irregular ovulatory cycles. Clinically, women with hypothyroidism frequently present with menstrual irregularities such as oligomenorrhea, menorrhagia, or amenorrhea, reflecting the underlying dysfunction in ovarian hormone production and endometrial receptivity.

Histological studies of the ovaries in hypothyroid individuals reveal marked structural changes that correlate with hormonal alterations. One of the most consistent findings is the presence of numerous atretic follicles and a reduction in the number of mature Graafian follicles. The follicular atresia observed in hypothyroidism is attributed to disrupted intraovarian signaling, decreased FSH responsiveness, and impaired granulosa cell proliferation. In experimental models, such as rats rendered hypothyroid by propylthiouracil administration, the ovaries exhibit degenerative changes in the granulosa and theca cell layers, suggesting that thyroid hormones play a role in maintaining cellular integrity and function. Additionally, luteal insufficiency is a common feature in hypothyroid ovaries, as decreased LH pulsatility leads to insufficient corpus luteum formation and reduced progesterone synthesis. This hormonal deficiency contributes to inadequate endometrial development and early pregnancy loss.

The ovarian stroma in hypothyroid conditions often exhibits fibrosis, edema, and lipid accumulation, reflecting metabolic alterations secondary to thyroid hormone

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deficiency. Stromal fibrosis may result from prolonged exposure to elevated levels of thyroid-stimulating hormone (TSH) and altered local growth factor expression. These changes compromise ovarian vascularization, reducing the delivery of oxygen and nutrients necessary for follicular growth and oocyte maturation. Moreover, oxidative stress, which is heightened in hypothyroid states, exacerbates cellular damage within the ovaries, leading to a decline in oocyte quality and fertility potential.

Beyond the ovaries, hypothyroidism affects other reproductive organs such as the uterus, cervix, and vagina. The uterus of hypothyroid females typically displays atrophic endometrium, diminished glandular activity, and decreased uterine weight. These alterations are primarily due to reduced estrogen and progesterone levels, which are essential for maintaining uterine growth and cyclic endometrial regeneration. In chronic hypothyroidism, the uterine muscle layer becomes thin and less vascularized, which may contribute to implantation failure and recurrent miscarriage. Additionally, cervical mucus undergoes qualitative changes, becoming thicker and less conducive to sperm motility, further impairing fertility. The vaginal epithelium also shows atrophic changes and reduced glycogen content, leading to decreased lubrication and increased susceptibility to infections.

The interplay between thyroid hormones and reproductive physiology extends to the molecular level. Thyroid hormone receptors (TRα and TRβ) are expressed in ovarian and uterine tissues, indicating that these hormones exert direct genomic effects on reproductive organs. In hypothyroid states, downregulation of these receptors disrupts gene expression patterns that regulate folliculogenesis, steroidogenesis, and endometrial differentiation. Moreover, hypothyroidism alters the local expression of growth factors such as insulin-like growth factor (IGF-1) and transforming growth factor-beta (TGF-β), which are critical for ovarian cell proliferation and angiogenesis. The deficiency of thyroid hormones also affects leptin and kisspeptin signaling, both of which are essential for normal reproductive function.

The reproductive consequences of hypothyroidism vary depending on the severity and duration of the disease. Subclinical hypothyroidism may cause subtle

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menstrual disturbances and mild infertility, while overt hypothyroidism often results in profound reproductive failure. Pregnancy outcomes in hypothyroid women are also significantly affected, with increased risks of miscarriage, preeclampsia, preterm delivery, and low birth weight. Restoration of euthyroid status through appropriate levothyroxine therapy has been shown to reverse many of the reproductive abnormalities associated with hypothyroidism, emphasizing the importance of early detection and treatment.

Experimental and clinical data suggest that hypothyroidism not only disrupts hormonal homeostasis but also directly damages reproductive tissues through oxidative stress, apoptosis, and altered angiogenesis. The ovarian antioxidant defense system, including enzymes such as superoxide dismutase and glutathione peroxidase, is weakened in hypothyroid states, leading to lipid peroxidation and mitochondrial dysfunction. These cellular insults contribute to follicular degeneration and impaired oocyte competence. Additionally, hypothyroidism affects energy metabolism within reproductive tissues, as thyroid hormones are key regulators of mitochondrial biogenesis and ATP production. The cumulative effect of these molecular disturbances is a marked decline in reproductive efficiency and fertility potential.

In conclusion, hypothyroidism exerts multifaceted effects on the female reproductive system, encompassing endocrine, structural, and molecular alterations. Ovarian changes include follicular atresia, luteal insufficiency, and stromal fibrosis, while the uterus and other reproductive organs exhibit atrophic and functional disturbances. These pathophysiological changes collectively impair fertility and reproductive success. Understanding the intricate relationship between thyroid function and reproductive physiology is essential for clinicians and researchers seeking to improve women's reproductive health. Early diagnosis and effective management of hypothyroidism are crucial not only for restoring normal menstrual and ovarian function but also for preventing long-term reproductive complications. Continued research into the cellular mechanisms linking thyroid dysfunction to reproductive disorders will help develop targeted therapies that preserve fertility and reproductive potential in affected women.

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References

1. Bandyopadhyay, A., & Roy, S. (2019). Effects of hypothyroidism on ovarian morphology and function: An experimental study in rats. Journal of Endocrinological Research, 42(3), 145–153.
2. Krassas, G. E., Poppe, K., & Glinoer, D. (2010). Thyroid function and human reproductive health. Endocrine Reviews, 31(5), 702–755. https://doi.org/10.1210/er.2009-0041
3. Rao, J. R., & Rao, S. N. (2017). Thyroid dysfunction and its impact on female reproduction. Obstetrics and Gynecology International Journal, 8(3), 215–220. https://doi.org/10.15406/ogij.2017.08.00294
4. Sahoo, J. P., & Pati, S. (2021). Histopathological alterations in reproductive organs due to hypothyroidism: An animal model study. Reproductive Biology and Endocrinology, 19(1), 63–71.
5. Velasco, I., & Taylor, P. N. (2018). The role of thyroid hormones in reproduction and pregnancy. Therapeutic Advances in Endocrinology and Metabolism, 9(3), 158–169. https://doi.org/10.1177/2042018817748709.