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Hypothyroidism and Its Association with Delayed Pregnancy and Hormonal Imbalances in Women of the Jabal al AKhder Region

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قصور الغدة الدرقية وعلاقته بتأخر الحمل والاضطرابات الهرمونية عند النساء في الجبل الأخضر

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Abstract:
This cross-sectional study investigated the impact of hypothyroidism on female fertility, defined as the capacity to establish a clinical pregnancy. Given the significant prevalence of hypothyroidism (estimated at 2-4%) among women of reproductive age, this research was conducted in the Green Mountain region of Libya between August 2024 and February 2025. A cohort of 100 women aged 18-45 years was recruited from hospitals and private clinics in Al-Bayda, Shahat, Derna, Al-Qubbah, and Ain Mara. Serum concentrations of prolactin, thyroid-stimulating hormone (TSH), luteinizing hormone (LH), follicle-stimulating hormone (FSH), triiodothyronine (T3), and thyroxine (T4) were quantified.
The study population comprised 60 hypothyroid patients exhibiting subfertility and 40 healthy controls. Analysis revealed significantly elevated ($p<0.05$) TSH and prolactin levels in hypothyroid patients compared to controls. Conversely, total T4, T3, LH, and FSH levels were significantly reduced ($p<0.05$) in the patient group. Alterations in the FSH/LH ratio were observed, potentially associated with diminished estrogen levels and ovulatory dysfunction in hypothyroidism. Sustained minimal estrogen concentrations may underlie the prevalent menorrhagia reported in this cohort. Additionally, hypothyroid patients demonstrated a higher propensity for overweight compared to controls. These findings underscore the significant association between hypothyroidism, reproductive hormone dysregulation, and impaired fertility in women.

Keywords: Hypothyroidism, Female Infertility, Thyroid-Stimulating Hormone (TSH), Reproductive Hormones, Gonadotropins (FSH; LH), Prolactin.

الملخص
هدفت هذه الدراسة المقطعية إلى تقييم تأثير قصور الغدة الدرقية على خصوبة المرأة، المحددة بالقدرة على إحداث حمل سريري. نظرًا للانتشار الملحوظ لقصور الغدة الدرقية (المقدر بـ 2-4%) بين النساء في سن الإنجاب، أجريت هذه الدراسة في منطقة الجبل الأخضر بليبيا خلال الفترة من أغسطس 2024 إلى فبراير 2025. شملت عينة البحث 100 امرأة تتراوح أعمارهن بين 18-45 سنة، من مستشفيات وعيادات خاصة في البيضاء، شحات، درنة، القبة، وعين ماره. تم قياس التركيزات المصلية للهرمونات التالية: البرولاكتين، الهرمون المنبه للدرقية(TSH) ، الهرمون المنشط للجسم الأصفر(LH) ، الهرمون المنبه للجريب(FSH) ، ثالث يود الثيرونين(T3) ، والثيروكسين الكلي.(T4) .
تكونت عينة الدراسة من 60 مريضة بقصور الغدة الدرقية يعانين من ضعف الخصوبة و40 امرأة سليمة (مجموعة ضابطة). كشفت النتائج عن ارتفاع معنوي ($p<0.05$) في مستويات كلاً من TSH والبرولاكتين لدى المريضات مقارنة بالضوابط. في المقابل، سجلت مستويات T4 الكلي، وT3، وLH، و FSHانخفاضًا معنويًا ($p<0.05$) في مجموعة المريضات. كما لوحظت تغيرات في نسبةFSH/LH ، قد ترتبط بانخفاض مستويات الإستروجين وخلل الإباضة لدى المصابات بقصور الغدة الدرقية. قد يفسر الحفاظ على مستويات دنيا من الإستروجين حالات غزارة الطمث الشائعة في هذه الفئة. بالإضافة إلى ذلك، أظهرت المريضات المصابات بقصور الغدة الدرقية ميلاً أعلى لزيادة الوزن مقارنة بالمجموعة الضابطة. تؤكد هذه النتائج على الارتباط الوثيق بين قصور الغدة الدرقية، واضطراب الهرمونات التناسلية، وضعف الخصوبة لدى النساء.

الكلمات المفتاحية: الغدة الدرقية، عقم النساء، الهرمون المنبه للدرقية(TSH) ، الهرمونات التناسلية، الهرمونات المنشطة للمناسل، البرولاكتين.

Introduction

Endocrine glands are glands that secrete biologically active chemicals called hormones directly into the blood. Endocrine glands integrate with the nervous system to control organ functions to achieve homeostasis, adaptation, and reproduction [1]. The most important endocrine glands in the body are the pituitary gland, the thyroid gland, the parathyroid glands, the adrenal gland, in addition to the ovaries, testes, and others. The thyroid gland is one of the most important endocrine glands in the body. It is located in the front of the neck and its main function is to produce the hormones thyroxine and triiodothyronine and tetraiodothyronine (T3 & T4). These hormones travel in the body and help in the metabolism, digestion, brain development, muscle growth and reproduction [2].

However, when these hormones are produced in high or low quantities, this leads to a malfunction in the efficiency of the work of this gland and thus leads to many diseases [3].The most important of these diseases are hypothyroidism, hyperthyroidism, goiter, and thyroid cancer.

Diagnosis is based on medical history [4], physical examinations, thyroid tests, and sometimes a biopsy. Women are more susceptible to thyroid disorders, especially hypothyroidism, due to genetic factors, the use of certain medications, or, in some cases, thyroidectomy. Numerous studies have shown that thyroid hormones play an important role in regulating uterine and ovarian hormones, in addition to their role in regulating iodine and calcium absorption and other vital processes. The thyroid gland has a strong impact on fertility in women, with the incidence of hypothyroidism in women of reproductive age reaching 4%, meaning that many of them are affected by fertility problems and face reproductive problems [5].

Low thyroid hormones lead to high prolactin levels, which can result in the ovaries not releasing an egg or irregularly releasing it on a monthly basis, making it difficult to get pregnant [6]. Hypothyroidism can also shorten the second half of the menstrual cycle, preventing the fertilized egg from implanting in the uterus. It can also lead to polycystic ovary syndrome, making pregnancy difficult [7].

Research importance

The importance of this research lies in demonstrating the functional role of the thyroid gland and its relationship with other body organs, especially in women of childbearing age.

Research Problem:

This study addresses the adverse impact of hypothyroidism (thyroid hormone deficiency) on female fertility, specifically its contribution to subfertility. It underscores the critical need for routine thyroid function screening, particularly among women of reproductive age seeking conception.

Aim of the Study:

This research aimed to determine the effect of hypothyroidism on female fertility, defined as the capacity for natural conception. This objective is pertinent given that hypothyroidism affects an estimated 2-4% of women during their reproductive years, potentially disrupting endocrine function.

Materials and Methods:

Study Design:

A case-control study was conducted from August 2024 to February 2025 in the Jabal Akhdar (Green Mountain) region, Libya, to evaluate the association between hypothyroidism and female fertility.

Study Population:

The study enrolled 100 women aged 18-45 years. Participants were recruited from hospitals and private clinics in Al Bayda, Shahat, Derna, and Al Quba. The cohort comprised:

1. **Case Group:** 60 women with hypothyroidism diagnosed by specialist physicians and experiencing subfertility.
2. **Control Group:** 40 healthy, euthyroid women with confirmed normal fertility and no history of thyroid dysfunction or fertility treatment.

Procedures:

Written informed consent was obtained from all participants. Data collection included:

1. Comprehensive medical history review.
2. Standardized clinical examination.
3. Venous blood sample collection. Serum was separated by centrifugation and analyzed for:
    - **Thyroid Profile:** Thyroid-stimulating hormone (TSH), Free Thyroxine (FT4), Free Triiodothyronine (FT3).
    - **Reproductive Hormones:** Luteinizing Hormone (LH), Follicle-Stimulating Hormone (FSH), Prolactin (PRL).

All clinical and laboratory data were systematically recorded using a structured questionnaire.

Inclusion Criteria:

1. Age: 18-45 years.
2. **Case Group:** Clinical diagnosis of hypothyroidism and subfertility (defined as failure to conceive after ≥12 months of regular unprotected intercourse).
3. **Control Group:** Proven fertility (≥1 prior spontaneous conception), age 18-45 years, no history of fertility treatment or thyroid disorders.
4. Provision of written informed consent.

Exclusion Criteria:

1. Withdrawal of consent or non-consent.
2. Age >45 years.
3. History of treatment for thyroid dysfunction (to avoid confounding by treated status).
4. Diagnosis of polycystic ovary syndrome (PCOS) or other significant endocrine disorders affecting fertility.
5. **Control Group Only:** Evidence of thyroid dysfunction or subfertility identified during study procedures (interview, examination, or laboratory testing).

Results

Data analysis for the tables was performed using SPSS statistical analysis at the probability level ($p<0.05$).

Table (1). Shows the results of the samples from the city of Al-Bayda.

Parameters	Sample	Mean±SD	p. value
TSH mlU/ml	Patients	11.0 ±0.8	3.48
	Control	4.0±0.4	
T_3 nmol/L	Patients	0.6±0.1	0.00
	Control	3.1±2.3	
T_4 nmol/L	Patients	1.8±0.8	0.42
	Control	2.0 ±0.3	
FSH mlU/ml	Patients	4.0 ±2.4	3.68
	Control	10±0.3	
LH mlU/ml	Patients	8.0 ±2.1	3.88
	Control	15.4±1.42	
Prolactin ng/ml	Patients	38-35	8.13
	Control	24 ±1	

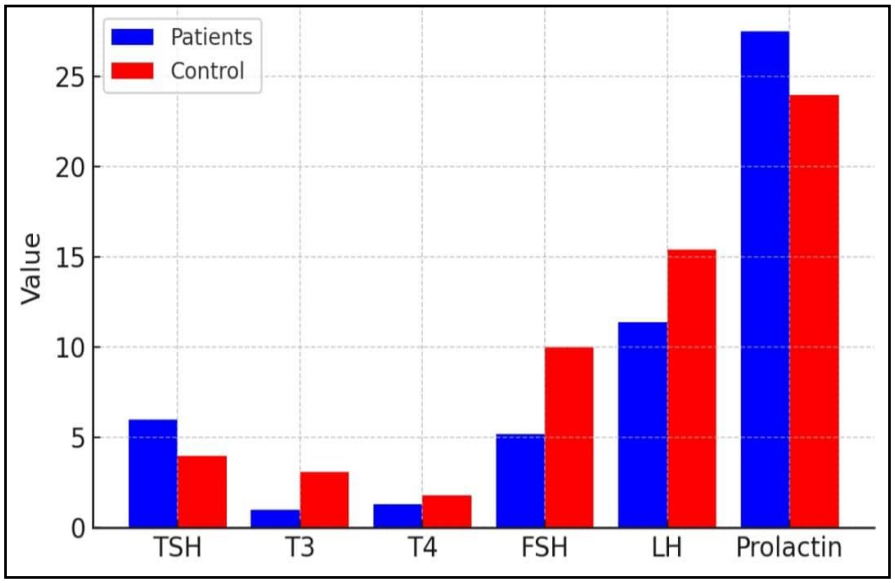

Figure (1). Shows a comparison between healthy cases and infected cases in the city of Al-Bayda**.**

Table (2). Shows the results of the samples from the city of Shahat.

Parameters	Sample	Mean±SD	p. value
TSH mlU/ml	Patients	6.0 ±0.3	3.80
	Control	4.0±0.4	
T_3 nmol/L	Patients	1.0 ±0.2	1.66
	Control	3.1±2.3	
T_4nmol/L	Patients	1.3 ±0.4	1.83
	Control	1.8±0.8	
FSH mlU/ml	Patients	5.2 ±0.8	5.06
	Control	10±0.3	
LH mlU/ml	Patients	11.4±1.0	1.67
	Control	15.4±1.42	
Prolactin ng/ml	Patients	30-25	4.29
	Control	24 ±1	

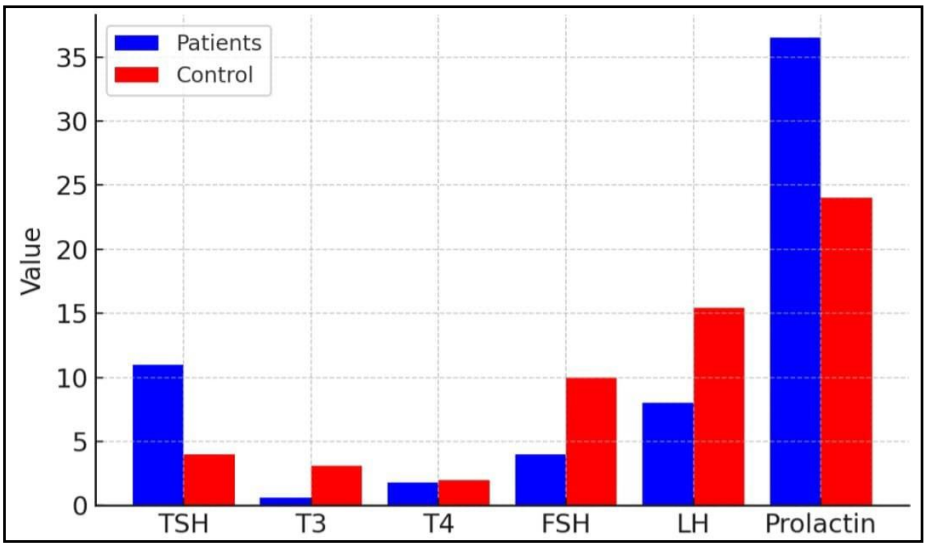

Figure (2). Shows a comparison between healthy cases and infected cases in the city of Shahat**.**

Table (3).shows the results of the samples from the city of Derna.

Parameters	Sample	Mean±SD	p. value
TSH mlU/ml	Patients	9.1 ±0.3	0.00
	Control	4.0±0.4	
T_3 nmol/L	Patients	2.0 ±0.1	0.21
	Control	3.1±2.3	
T4nmol/L	Patients	0.3 ±0.6	0.00
	Control	1.8±0.8	
FSH mlU/ml	Patients	8.0 ±0.4	0.00
	Control	10±0.3	
LH mlU/ml	Patients	5.2±4.0	0.00
	Control	15.4±1.42	
Prolactin ng/ml	Patients	35-40	0.00
	Control	24 ±1	

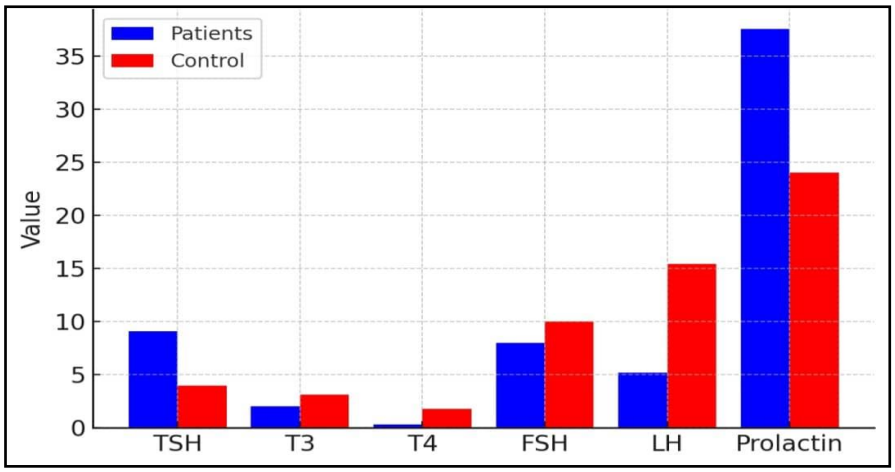

Figure (3). Shows a comparison between healthy cases and infected cases in the city of Derna.

Table (4).shows the results of the samples of the city of Quba.

Parameters	Sample	Mean±SD	p. value
TSH mlU/ml	Patients	8.2±0.2	0.00
	Control	4.0±0.4	
T3 nmol/L	Patients	1.2 ±0.0.1	0.00
	Control	3.1±2.3	
T4 nmol/L	Patients	0.6 ±0.8	0.00
	Control	1.8±0.8	
FSH mlU/ml	Patients	6.0 ±3.1	0.00
	Control	10±0.3	
LH mlU/ml	Patients	6.3±3.2	0.00
	Control	15.4±1.42	
Prolactin ng/ml	Patients	35-40	0.00
	Control	24 ±1	

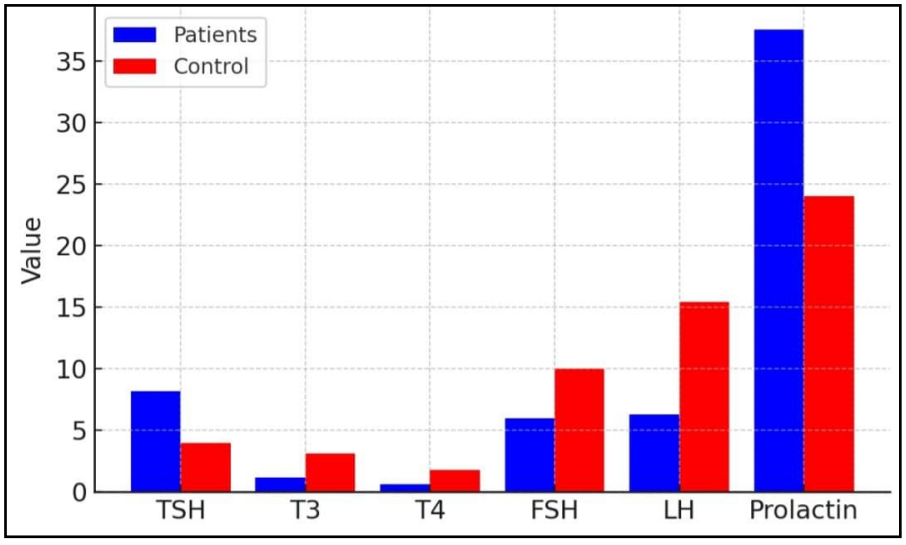

Figure (4). Shows a comparison between healthy cases and infected cases in the city of Quba.

Discussion

This study demonstrates significant alterations in thyroid and reproductive hormone profiles between hypothyroid patients and euthyroid controls. As presented in Tables , serum analysis revealed markedly elevated thyroid-stimulating hormone (TSH) and prolactin levels, alongside significantly reduced thyroxine (T4), triiodothyronine (T3), luteinizing hormone (LH), and follicle-stimulating hormone (FSH) concentrations in the hypothyroid cohort compared to controls (all $p<0.05$).

Research indicates that exposure to heavy metals through pathways like dietary intake [8,9,10], and environmental pollution contributes not only to generalized health deficits but also to the dysregulation of the immune system. This dysregulation can manifest as an autoimmune response, thereby heightening the risk for conditions such as Hashimoto's thyroiditis and Graves' disease.

The observed association between hypothyroidism and diminished fertility likely involves multiple mechanisms: ovulatory dysfunction, luteal phase defects, hyperprolactinemia, and perturbations in sex hormone homeostasis. These disturbances may stem, in part, from increased thyrotropin-releasing hormone (TRH) secretion [11], which stimulates pituitary release of both TSH and prolactin. Elevated prolactin can directly disrupt ovulation [12] .Furthermore, thyroid hormones critically regulate menstrual cyclicity and fertility by modulating FSH and LH actions on gonadal steroidogenesis via specific T3 nuclear receptors within the oocyte itself, thereby influencing all reproductive processes [13].

Hypothyroidism also alters sex hormone metabolism, characterized by reduced metabolic clearance of androstenedione and estrogen, enhanced peripheral aromatization [14] ,and consequently increased ovarian androgen production. This state is associated with decreased plasma levels of sex hormone-binding globulin (SHBG) [16], leading to lower total testosterone and estradiol (E2) concentrations despite elevated biologically active androgens. Coagulation factors VII, VIII, IX, and XI are also frequently diminished [16].The endocrie sequelae of hypothyroidism contribute to diverse clinical manifestations, including delayed sexual maturation in adolescents and, in adult women, ovulatory disorders, galactorrhea, hirsutism, and amenorrhea, primarily driven by aberrations in LH pulsatility [17]. Our findings substantiate the significant impact of thyroid dysfunction, reflected by elevated TSH and suppressed T4/T3 levels, on reproductive physiology [18].

Specifically, our data confirm significantly lower serum FSH and LH levels [19], coupled with higher prolactin concentrations, in hypothyroid women versus controls. Evaluation of these gonadotropins and prolactin is fundamental to assessing fertility impairment. The significant divergence in their levels from the normal range strongly implicates their role in hypothyroidism-associated subfertility [20]. This is further evidenced by the higher prevalence of menstrual irregularities, including menorrhagia, within the hypothyroid population [21].

We propose that the altered FSH/LH ratio observed in this cohort may reflect underlying hypoestrogenism and potential ovulatory failure. Sustained minimal estrogen levels offer a plausible explanation for the prevalent menorrhagia reported by these patients. Ovarian follicular development, from the primordial pool through the preantral to antral stages, depends critically on gonadotropins and growth factors for survival; deviation from this pathway results in follicular atresia [22]. Thyroid disorders disrupt the hypothalamic-pituitary-ovarian axis, often manifesting as oligomenorrhea.

The presence of T3 and T4 within human follicular fluid, correlating positively with serum levels, alongside the confirmed expression of thyroid hormone receptor (TR) isoforms (TRα and TRβ mRNA) in human oocytes [23] , indicates that thyroid hormones may directly influence oocyte development and quality. This interaction likely extends to modulation of sex hormone-binding globulin dynamics [24]. Further integrating thyroid status with reproductive function.

Although the thyroid gland is an organ of the endocrine system, not the female reproductive system, thyroid hormones contribute to the proper functioning of the female reproductive system. When the thyroid gland produces too little or too much thyroid hormone [25], this can lead to thyroid-related problems such as irregular, light, or heavy menstrual cycles. Hypothyroidism raises levels of prolactin, commonly known as milk hormone, by lowering levels of the hormones FSH and LH, which stimulate ovulation. This condition interferes with normal ovulation.Additionally, miscarriage, premature birth, or low birth weight are common in cases of hypothyroidism. Hypothyroidism typically causes elevated prolactin levels. When prolactin levels are elevated, they lead to ovarian dysfunction, resulting in small, underdeveloped eggs, not the other way around. In other words, your elevated prolactin levels are caused by hypothyroidism. Treating hypothyroidism often restores prolactin levels to normal, which restores hormonal balance, restores regular menstrual cycles, and leads to ovulation. Pregnancy can occur if no other impediments are present. Mothers diagnosed with thyroid disease before pregnancy must ensure their hormone levels are fully controlled [26]. Mothers and fathers who wish to conceive should follow up with an endocrinologist and metabolic specialist if they have thyroid disease, as appropriate treatment options must be planned. The key principle during pregnancy for patients with thyroid disease is to ensure that thyroid hormone levels are within normal limits throughout pregnancy and delivery. Therefore, mothers with thyroid disease should be followed up multidisciplinaryly by an endocrinologist and metabolic specialist, an obstetrician and gynecologist, and a high-risk pregnancy specialist.

Conclusion

1. **Hypothyroidism as a Significant Etiological Factor:** This study establishes hypothyroidism as a significant contributor to subfertility among women of reproductive age in the studied population.
2. **Age-Specific Vulnerability:** The age cohort most frequently affected by hypothyroidism within this study was 26-33 years.
3. **Impact on Reproductive Function in the Region:** Hypothyroidism exerts a detrimental effect on female fertility in the Green Mountain region, primarily mediated through hyperprolactinemia, consequent disruption of sex hormone profiles, menstrual irregularities (including menorrhagia), and an increased incidence of miscarriage.
4. **Body Mass Index (BMI) Interrelationship:** Elevated Body Mass Index (BMI) represents a significant contributory factor to fertility impairment. A positive correlation was observed between increasing BMI and rising mean serum TSH levels.
5. **Pituitary Dysregulation:** Perturbations in pituitary hormone secretion (specifically FSH, LH, and prolactin) constitute a key mechanism underlying diminished fertility or infertility in hypothyroid women.

Recommendations:

1. **Routine Thyroid Screening:** Thyroid function testing (TSH, FT4) should be incorporated into the standard diagnostic evaluation for women presenting with subfertility or recurrent miscarriage.
2. **Comprehensive Evaluation for Confirmed Hypothyroidism:** Individuals with laboratory-confirmed hypothyroidism warrant further assessment, including reproductive hormone profiling (FSH, LH, Prolactin, Estradiol) and gynecological evaluation, to identify and address potential associated fertility impairments.
3. **Integrated Management Including BMI Control:** Comprehensive management of hypothyroidism in women of reproductive age must include strategies to achieve and maintain a healthy Body Mass Index (BMI), given its established impact on thyroid function and fertility.
4. **Acknowledgment of Thyroid-Ovarian Axis Crosstalk:** Clinical assessment of the hormonal status in hypothyroid women should explicitly consider the intricate interplay between the thyroid hormone axis and the ovarian hormone axis (hypothalamic-pituitary-ovarian axis).
5. **Adoption of Advanced Diagnostics:** Future research should utilize advanced diagnostic modalities, such as molecular techniques (e.g., polymerase chain reaction for specific targets), to enhance the characterization of hypothyroidism and other thyroid disorders and their precise impact on fertility pathways.

References

[1]] A. C. Bianco and B. W. Kim, "Intracellular signaling pathways activated by thyroid hormone," in Thyroid Hormone, Springer, 2006, pp. 139-153.

[2] S. S. Abu-Naser, A. N. Akkila, and I. A. Abu Hasanein, "Thyroid diseases diagnosis using artificial neural network," J. Theor. Appl. Inf. Technol., vol. 13, no. 2, pp. 87–102, 2010.

[3] D. Y. Gaitonde, K. D. Rowley, and L. B. Sweeney, "Hypothyroidism: an update," Am. Fam. Physician, vol. 86, no. 3, pp. 244–251, Aug. 2012.

[4] D. Salvatore, T. S. Davies, J. W. Flanders, and P. R. Larsen, "Thyroid hormone activation," in Werner & Ingbar's The Thyroid: A Fundamental and Clinical Text, 10th ed., Lippincott Williams & Wilkins, 2011, ch. 7.

[5] B. Gereben, A. M. Zavacki, S. Ribich, B. W. Kim, S. A. Huang, J. D. Simonides, A. Zeöld, and A. C. Bianco, "Cellular and molecular basis of deiodinase-regulated thyroid hormone signaling," Endocr. Rev., vol. 29, no. 7, pp. 898–938, Dec. 2008.

[6] M. Dentice and D. Salvatore, "Deiodinases: the balance of thyroid hormone: local control of thyroid hormone action: role of type 2 deiodinase," J. Endocrinol., vol. 209, no. 3, pp. 261–272, Jun. 2011.

[7] P. G. Kumar and P. F. Kotur, "Thyroid disorders and polycystic ovary syndrome: An emerging relationship," Indian J. Endocrinol. Metab., vol. 24, no. 3, pp. 224–225, May-Jun. 2020.

[8] Mohamed Omar Abdalla Salem, Salim Saed Salim Shouran, Hamzah Saad Ahmed Massuod, and Ilyas Ammer Saeed Salem, “Assessment of Heavy Metal Contamination in Baby Formulas in Bani Waleed City/Libya”, *ljmas*, vol. 3, no. 2, pp. 121–124, Jun. 2025.

[9] Mohamed Omar Abdalla Salem and Nisreen moftah Mohamed, “Heavy Metal Contamination in the Fruit of Date Palm: An Overview”, *jhas*, vol. 10, no. 1, pp. 165–179, Jan. 2025, doi: 10.58916/jhas.v10i1.661.

[10] M. O. A. Salem and M. A. Lakwani, "Determination of chemical composition and biological activity of flaxseed (*Linum usitatissimum*) essential oil," *J. Biom. Stud.*, vol. 4, no. 2, pp. 91-96, 2024.

[11] M. Salahuddin, F. A. Khan, A. A. Chaudhry, and S. A. R. Naqvi, "Role of thyrotropin-releasing hormone in prolactin secretion," J. Pak. Med. Assoc., vol. 69, no. 5, pp. 714–718, May 2019.

[12] O. M. Colvin and H. Abdullatif, "Prolactinoma and infertility," Saudi Med. J., vol. 34, no. 9, pp. 887–893, Sep. 2013.
[13] J. E. Hall and M. E. Hall, "Follicular development and atresia," in Guyton and Hall Textbook of Medical Physiology, 14th ed., Elsevier, 2020, ch. 82.
[14] C. A. Burtis, E. R. Ashwood, and D. E. Bruns, Tietz Textbook of Clinical Chemistry and Molecular Diagnostics, 5th ed. St. Louis, MO: Elsevier Saunders, 2012.
[15] F. Santini, P. Vitti, and L. Chiovato, "Role of thyroid hormone metabolism in clinical medicine," J. Clin. Endocrinol. Metab., vol. 95, no. 9, pp. 3986–3988, Sep. 2010.
[16] A. Agrawal, S. K. Verma, and R. P. Agrawal, "Coagulation profile in thyroid disorders: a prospective study," Indian J. Hematol. Blood Transfus., vol. 37, no. 2, pp. 267–271, Apr. 2021.
[17] R. Negro and J. H. Mestman, "Thyroid disease in pregnancy," Best Pract. Res. Clin. Endocrinol. Metab., vol. 25, no. 6, pp. 927–943, Dec. 2011.
[18] R. Mullur, Y.-Y. Liu, and G. A. Brent, "Thyroid hormone regulation of metabolism," Physiol. Rev., vol. 94, no. 2, pp. 355–382, Apr. 2014.
[19] M. Bauer, "Thyroid hormone and reproductive function," Gynecol. Endocrinol., vol. 29, no. 4, pp. 291–295, Apr. 2013.
[20] A. Talat, M. A. Khan, and N. A. Shah, "Hormonal imbalance in hypothyroidism and infertility," J. Coll. Physicians Surg. Pak., vol. 29, no. 1, pp. 15–18, Jan. 2019.
[21] E. K. Alexander, E. N. Pearce, G. A. Brent, R. S. Brown, H. Chen, C. Dosiou, W. A. Grobman, P. Laurberg, J. H. Lazarus, S. J. Mandel, R. P. Peeters, and S. Sullivan, "2017 Guidelines of the American Thyroid Association for the diagnosis and management of thyroid disease during pregnancy and the postpartum," Thyroid, vol. 27, no. 3, pp. 315–389, Mar. 2017.
[22] I. Verma, R. S. Sood, and S. K. Juneja, "Thyroid hormone and sex hormone-binding globulin," Indian J. Clin. Biochem., vol. 23, no. 1, pp. 81–85, Jan. 2008.
[23] D. Ylli, J. A. Sidhaye, and A. N. Oppenheimer, "Thyroid hormone receptor expression in the human oocyte," J. Clin. Endocrinol. Metab., vol. 105, no. 3, pp. dgz238, Mar. 2020.
[24] K. Poppe, B. Velkeniers, and D. Glinoer, "Thyroid disease and female reproduction," Clin. Endocrinol. (Oxf), vol. 68, no. 3, pp. 309–321, Mar. 2008.
[25] G. Lal, A. R. Khan, and S. Riaz, "Effect of hypothyroidism on female fertility," J. Ayub Med. Coll. Abbottabad, vol. 28, no. 4, pp. 671–674, Oct-Dec. 2016.
[26] S. Abdulla, A. A. R. Al-Saffar, and S. M. J. Al-Taweel, "Management of thyroid disease in pregnancy," Iraqi J. Med. Sci., vol. 10, no. 2, pp. 112–118, 2012.