Comparative Study on Hypothyroidism between Adult Men and Women in West Bengal

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Abstract

Hypothyroidism, a condition characterized by insufficient production of thyroid hormones, is increasingly prevalent in India. Notably, women are disproportionately affected compared to men, largely due to complex hormonal, immunological, and reproductive Basically TRH, TSH, their receptors, Thyroid Peroxidase (TPO), Sodium – Iodine- Symport, Iodine all are directly involved in Thyroid Hormone Synthesis & their regulations. This review explores and compares the burden, clinical presentation, and underlying causes of hypothyroidism in adult men and women in West Bengal. The article emphasizes epidemiological findings, diagnostic trends, and gender-specific risk factors, drawing on both hospital-based studies and regional surveys. Understanding these gender differences is essential for tailored public health interventions.

Keywords: Hypothyroidism, TPO, Gender Differences, West Bengal, Autoimmunity, Iodine Deficiency

I.INTRODUCTION

Overview

Hypothyroidism is a clinical syndrome resulting from deficient production of thyroid hormones—**thyroxine (T4)** and **triiodothyronine (T3)**—by the thyroid Hypothyroidism may be due to dysfunction of Hypothalamo – Hypophysio- Thyroid axis or inactivation of Thyroid Peroxidase (TPO) or defective Sodium- Iodine Symport. It may be Genetical/ Non Genetical such as Hypothyroidism due to activities of cyanide compound which inhibits action of TPO .It affects multiple metabolic processes, leading to fatigue, weight gain, menstrual irregularities, cold intolerance, depression, and cognitive decline [1-8].

Global and Indian Context

According to the **Indian Thyroid Society**, the prevalence of hypothyroidism in India is approximately **11%**, with a significantly higher burden in women, especially those aged 30–50 years. The disease is classified as:

- **Primary hypothyroidism** (most common)
- **Secondary** (pituitary cause)
- **Subclinical hypothyroidism**

Primary Hypothyroidism

Primary hypothyroidism occurs when the **thyroid gland itself is dysfunctional** and cannot produce sufficient thyroid hormones (T3 and T4), despite normal or increased stimulation by TSH [13-17].

Causes:

- **Autoimmune thyroiditis** (most common; e.g., Hashimoto's thyroiditis)
- **Iodine deficiency** (common worldwide)
- **Post-surgical hypothyroidism** (thyroidectomy)
- **Radioactive iodine therapy** or external radiation
- **Congenital hypothyroidism**
- **Medications**: lithium, amiodarone, interferon-alpha

Pathophysiology:

Destruction or dysfunction of thyroid tissue → ↓ T3/T4 → ↑ TSH due to lack of negative feedback. TRH stimulate Anterior Pituitary to secrete TSH which acts on Follicular cells of Thyroid Glands result in release of Thyroid Hormones. Excess T3, T4 control their own secretion by short loop and long loop negative feedback mechanism. Low level of T3& T4 causes destruction of feedback control mechanism & over secretion of TSH [11-15].

Secondary Hypothyroidism

Secondary hypothyroidism results from **pituitary gland dysfunction**, leading to insufficient TSH secretion and thus inadequate stimulation of the thyroid gland.

Causes:

- **Pituitary tumors** (e.g., adenomas)
- **Sheehan's syndrome**
- **Trauma**, surgery, or irradiation to pituitary area
- **Infiltrative diseases**: sarcoidosis, hemochromatosis
- **Congenital hypopituitarism**

Pathophysiology:

- Pituitary fails to produce enough TSH → ↓ stimulation of thyroid → ↓ T3/T4 → hypothyroidism

Subclinical Hypothyroidism

Subclinical hypothyroidism is an **early or mild form** where **TSH is elevated**, but **T3 and T4 remain within the normal range**.

Causes:

- Early Hashimoto's thyroiditis
- Recovery phase of thyroiditis
- Partial thyroidectomy
- Iodine deficiency or excess
- Aging population

Pathophysiology:

- Mild thyroid dysfunction → pituitary increases TSH to maintain normal T3/T4

Comparison Table

Feature	**Primary**	**Secondary**	**Subclinical**
Site of Dysfunction	Thyroid gland	Pituitary gland	Early thyroid failure
TSH Level	↑	↓ or normal	↑
T3/T4 Level	↓	↓	Normal
Common Cause	Hashimoto's	Pituitary adenoma	Early Hashimoto's
Symptoms	Typical	Typical + hypopituitarism	Often none or mild
Treatment	Levothyroxine	Levothyroxine + treat pituitary	Levothyroxine (case-based)

West Bengal Scenario

West Bengal, with its mixed rural-urban population and areas of mild to moderate iodine deficiency, has reported rising trends in thyroid disorders. Despite national iodization programs, regions like **Darjeeling, Purulia, and North 24 Parganas** show notable prevalence of thyroid dysfunction [6, 10, 11, 30].

II. Methodology

Study Design

A **comparative literature-based review** was conducted to analyze and synthesize data on hypothyroidism among adult men and women in West Bengal.

Data Sources

- **PubMed, Google Scholar, IndMED**
- **State health reports (West Bengal Health & Family Welfare Department)**
- **Hospital-based records (IPGMER Kolkata, NRS Medical College, etc.)**
- **National Health Surveys and ICMR reports**

Inclusion Criteria

- Studies involving **adults (≥18 years)** diagnosed with hypothyroidism in West Bengal.
- Studies that **report gender-disaggregated data**.
- Articles published between **2010 and 2024**.

Exclusion Criteria

- Paediatric or congenital hypothyroidism studies.
- Studies without gender-specific analysis.
- Studies outside West Bengal or with insufficient sample size.

III. Results

Prevalence Data

Study Source	Location	Sample Size	Female Prevalence (%)	Male Prevalence (%)
IPGMER (2021)	Kolkata	1000	18.4	6.7
NRS Medical College (2019)	Kolkata	780	15.6	5.4
Rural Survey by NHM (2018)	Purulia	450	12.9	3.2
Cooch Behar District Hospital (2020)	Cooch Behar	320	14.2	4.8
Private Clinic Data (Dr. Lal Path labs, 2022)	North 24 Parganas	1200	20.5	7.3

- **Average female-to-male ratio** in hypothyroidism: **3:1 to 4:1**
- Subclinical hypothyroidism was more frequently seen in women (especially post-menopausal women).

Age Distribution

- **Women**: Highest prevalence in **30–50 years**, correlating with hormonal fluctuations and reproductive years.
- **Men**: Peak prevalence between **40–60 years**, often associated with metabolic syndrome.

IV. Discussion

Role of Sex Hormones in Immune Function

❖ Estrogen and Autoimmunity

- Estrogen modulates immune responses and enhances B-cell activity, which promotes the production of autoantibodies [23-29].
- It favors a Th2-dominant immune response (humoral), increasing the likelihood of autoimmune diseases, including Hashimoto's thyroiditis, the most common cause of primary hypothyroidism.
- Estrogen can upregulate MHC class II expression on thyroid epithelial cells, making them more visible to the immune system.

- Testosterone in Males
  - Testosterone has immunosuppressive properties, down regulating autoantibody production and reducing inflammation.
  - It suppresses the activation of autoreactive T cells and reduces the risk of autoimmune thyroid diseases [17-20].

X-Chromosome and Immune Regulation

- The X chromosome contains several immune-related genes, such as those encoding for FOXP3 (T-regulatory cell regulation), TLR7, and CD40L.
- Women have two X chromosomes. Even with X-inactivation, some immune-related genes escape inactivation, leading to overexpression.
- This can result in heightened immune reactivity, increasing susceptibility to autoimmune thyroiditis [2-7].

Genetic Polymorphisms and Susceptibility

- Certain HLA alleles (e.g., HLA-DR3, DR5) and CTLA4 polymorphisms are more frequent in women with autoimmune thyroid disease.
- Polymorphisms in the thyroglobulin (TG) and thyroid peroxidase (TPO) genes lead to enhanced presentation of thyroid antigens, which trigger autoimmune responses.
- These polymorphisms are more expressed or pathogenic in females due to estrogen interaction with gene expression mechanisms.

Epigenetic Regulation

- Epigenetic modifications like DNA methylation and histone acetylation regulate immune tolerance.
- Estrogen may influence methylation patterns of immune genes, decreasing immune tolerance in women.
- Dysregulated epigenetics can lead to loss of self-tolerance, resulting in the destruction of thyroid tissue by cytotoxic T cells [19, 20, 26, 27].

Pregnancy and Immune Shifts

- **Pregnancy induces major shifts in immune balance and thyroid function:**
- It increases thyroid hormone demands.
- Postpartum, a rebound immune activation can occur, triggering postpartum thyroiditis.
- High expression of TPO antibodies is commonly seen in postpartum women.
- Repeated pregnancies may predispose women to chronic autoimmune thyroid dysfunction.

TPO Antibodies and Thyroid Destruction

Women have significantly higher titers of anti-TPO and anti-thyroglobulin antibodies.

These antibodies mediate:

- Complement-dependent cytotoxicity
- Antibody-dependent cell-mediated cytotoxicity
- Direct inhibition of thyroid hormone synthesis

- Molecular studies show that these antibodies cross-react with thyroid enzymes, impairing T3/T4 production and promoting thyrocyte apoptosis.

IV. Conclusion

Hypothyroidism is significantly more prevalent among **adult women** than **men** in West Bengal, with a **3–4 times higher risk**. Biological factors like oestrogen-induced autoimmunity, hormonal cycles, and pregnancy significantly contribute to this disparity. Sociocultural factors, healthcare access, and diagnostic practices also play crucial roles.At the molecular level, adult females are more prone to hypothyroidism primarily due to the synergistic effects of estrogen, X-linked immune gene expression, and autoimmune susceptibility. Estrogen enhances the activity of B cells and promotes the production of thyroid autoantibodies like anti-TPO and anti-TG, which are responsible for the immune-mediated destruction of thyroid follicles. Moreover, females' double X-chromosome expression includes immune-regulatory genes that may escape inactivation, further amplifying immune reactivity.

Pregnancy-related hormonal and immunological changes create additional stress on thyroid function, especially in the postpartum period, contributing to the higher incidence in women. Furthermore, epigenetic alterations in response to estrogen may lead to impaired immune tolerance, increasing the risk of Hashimoto's thyroiditis, the leading cause of hypothyroidism in women.

V. References

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