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HORMONAL PROFILING OF SERUM TSH, T3, AND T4 LEVELS IN HYPOTHYROID AND HYPERTHYROID PRE-MENOPAUSAL AND POST-MENOPAUSAL FEMALES: A CROSS-SECTIONAL STUDY

Original Research

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ABSTRACT

Background: Thyroid hormones play a crucial role in regulating metabolism, growth, and reproductive function. Imbalances in these hormones, particularly in women, can lead to menstrual disturbances and endocrine dysfunction. Both hypothyroidism and hyperthyroidism are prevalent among females, especially during the premenopausal and postmenopausal phases, contributing to alterations in hormonal stability and reproductive health.

Objective: To evaluate serum thyroid hormone levels (TSH, T3, and T4) in premenopausal and postmenopausal females with thyroid dysfunction and to assess the association between thyroid abnormalities and menstrual irregularities.

Methods: A cross-sectional study was conducted at a tertiary care hospital in Lahore from March to June 2025. A total of 250 female participants were divided into five groups (n = 50 each): premenopausal hypothyroid (Group A), premenopausal hyperthyroid (Group B), postmenopausal hypothyroid (Group C), postmenopausal hyperthyroid (Group D), and euthyroid controls (Group E). Venous blood samples (3–5 mL) were collected, and serum levels of TSH, T3, and T4 were analyzed using the ELISA technique. Data were statistically analyzed using ANOVA with a significance level of $p < 0.05$.

Results:TSH, T3, and T4 levels showed statistically significant differences between groups ($p = 0.032$, $p < 0.05$). Mean TSH levels were 17.9 ± 13.7 µIU/mL and 11.0 ± 3.0 µIU/mL in pre- and postmenopausal hypothyroid females, respectively, compared to 0.20 ± 0.94 µIU/mL and 0.30 ± 0.96 µIU/mL in hyperthyroid females and 3.28 ± 0.39 µIU/mL in controls. Hypothyroid females exhibited low T4 (3.2 ± 0.8 µg/dL) while hyperthyroid females showed elevated T4 (12.4 ± 3.0 µg/dL). Menstrual irregularities were present in 72% of premenopausal hypothyroid and 62% of hyperthyroid females, with no menstrual disturbances reported in postmenopausal groups.

Conclusion: Thyroid dysfunction significantly affects serum TSH, T3, and T4 levels and is strongly associated with menstrual abnormalities in premenopausal women. Routine thyroid screening in women with menstrual irregularities or unexplained endocrine symptoms is recommended for early diagnosis and timely management.

Keywords: Endocrine disorders, Hyperthyroidism, Hypothyroidism, Menopause, Menstrual irregularities, Thyroid hormones, Thyrotropin.

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INTRODUCTION

Hormonal balance plays a pivotal role in maintaining human physiological stability, with estrogen, progesterone, and testosterone being particularly essential to female health and endurance throughout different life stages (1). Across her lifespan, a woman experiences several hormonally driven transitions—puberty, pregnancy, and menopause—each influencing the endocrine and reproductive systems in distinct ways (2). Among the many hormonal regulators, thyroid hormones have a profound impact on metabolic, reproductive, and neurological functions, highlighting their essential contribution to overall wellbeing (3). Thyroid dysfunction, encompassing both hypothyroidism and hyperthyroidism, is recognized as a global endocrine disorder, disproportionately affecting females, particularly in advanced age (4). Hypothyroidism, characterized by insufficient thyroid hormone secretion, often manifests insidiously with vague symptoms that delay diagnosis for months or even years (4,5). Conversely, hyperthyroidism involves excessive thyroid hormone production, commonly presenting with palpitations, heat intolerance, weight loss, and menstrual irregularities, yet frequently overlooked due to its subtle symptomatology (5,6). Despite the global burden of thyroid dysfunction, regional data—especially among older women in Thailand—remain limited, underscoring the need for localized epidemiological assessment.

Reproductive health is particularly sensitive to thyroid hormone fluctuations. The hypothalamic–pituitary–ovarian (HPO) axis governs the menstrual cycle, and any disruption in this intricate hormonal network can result in menstrual irregularities (7). Thyrotropin-releasing hormone (TRH) serves as a critical mediator between thyroid activity and reproductive function, influencing the release of gonadotropin-releasing hormone (GnRH) and prolactin, which in turn regulate ovarian activity (8). In hypothyroidism, elevated TRH can increase prolactin levels, leading to amenorrhea or irregular menstruation (9). Additionally, thyroid autoimmunity has been identified as a primary cause of thyroid dysfunction in reproductive-age women (10). Hormonal interactions extend further: hypothyroidism is associated with reduced sex hormone-binding protein (SHBP) levels, elevated free testosterone, and disrupted estrogen metabolism (11). Conversely, hyperthyroidism enhances SHBP synthesis, altering estrogen metabolism and androgen–estrogen conversion (12). These hormonal perturbations directly affect ovarian function and menstrual regularity. Globally, thyroid diseases rank immediately after diabetes in prevalence among endocrine disorders (13,14). Given these interrelations, the present study aims to examine thyroxine secretion patterns and thyroid hormone profiles in premenopausal and postmenopausal females with thyroid dysfunction. Using enzyme-linked immunosorbent assay (ELISA) to quantify TSH, T3, and T4 levels, the study seeks to elucidate the association between thyroid profiles in hypothyroid and hyperthyroid women and compare the strength of these relationships across reproductive phases. The objective is to better understand how thyroid hormone variations contribute to reproductive hormonal imbalances and menstrual irregularities in women.

METHODS

A cross-sectional study was conducted over a duration of four months, from March 2025 to June 2025, at a tertiary care hospital in Lahore. The study aimed to investigate the relationship between thyroid hormone levels and menopausal status in females diagnosed with thyroid dysfunction. Ethical approval for the study was obtained from the Institutional Review Board (IRB) of the respective tertiary care hospital and written informed consent was obtained from all participants prior to data and sample collection, ensuring adherence to ethical standards of the Declaration of Helsinki. A total of 250 female participants were enrolled using a purposive sampling technique. Participants were divided into five groups, each comprising 50 individuals. Group A included premenopausal females with hypothyroidism, Group B comprised premenopausal females with hyperthyroidism, Group C consisted of postmenopausal females with hypothyroidism, Group D represented postmenopausal females with hyperthyroidism, and Group E served as the control group, consisting of euthyroid females with normal thyroid hormone activity. Inclusion criteria included females clinically diagnosed with thyroid dysfunction by an endocrinologist, either hypothyroid or hyperthyroid, with no prior history of other significant endocrine or systemic disorders. Exclusion criteria included women who were pregnant, lactating, or using hormonal replacement therapy or oral contraceptives, as well as those with hemolyzed, lipemic, or contaminated blood samples. Venous blood samples (3–5 mL) were drawn from each participant after an overnight fast using aseptic precautions. The samples were allowed to clot at room temperature and subsequently centrifuged at 3000 rpm for 10 minutes to separate the serum. The serum aliquots were stored at –20 °C until biochemical

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analysis. All laboratory procedures were performed in the biochemistry section of the same tertiary care hospital using standardized protocols to ensure consistency and accuracy of results.

The thyroid hormone profile was determined using an Enzyme-Linked Immunosorbent Assay (ELISA). Thyroid-stimulating hormone (TSH), triiodothyronine (T3), and thyroxine (T4) concentrations were measured using a two-step sandwich immunoassay based on chemiluminescence principles. For TSH determination, biotinylated and ruthenium-labeled monoclonal TSH antibodies were used, with streptavidin-coated microparticles facilitating solid-phase binding. Magnetic capture followed by chemiluminescent signal measurement was performed, and quantification was based on a two-point calibration curve. The reference range of 0.4–4.0 µIU/mL was used to interpret results, with optical density (OD) readings below 0.4 considered normal and values exceeding 4.0 considered positive for hypothyroidism. The T3 assay was also performed using the sandwich immunoassay principle, with biotinylated monoclonal T3 antibodies and ruthenium-labeled antibodies forming immune complexes that bound to streptavidin-coated microparticles. After washing, chemiluminescence was quantified using a master calibration curve, and OD thresholds were used to determine positive or negative readings. Similarly, T4 concentration was measured using a comparable two-incubation sandwich immunoassay method with a biotinylated monoclonal T4 antibody conjugated to a ruthenium complex. After magnetic capture, chemiluminescent signals were recorded by a photomultiplier at 450 nm, and results were calculated using a two-point calibration curve. Substances showing an OD ≥4.5 were categorized as positive, while values below this were classified as negative. Data obtained from the assays were entered into and analyzed using the Statistical Package for Social Sciences (SPSS) software version 22. Descriptive statistics, including mean and standard deviation, were used to summarize quantitative data, while frequencies and percentages were used for categorical variables. Inferential analysis was performed using the independent sample t-test and one-way ANOVA where applicable. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 250 female participants were included in the study and categorized into five groups, each comprising 50 subjects: premenopausal hypothyroid, premenopausal hyperthyroid, postmenopausal hypothyroid, postmenopausal hyperthyroid, and euthyroid controls. The serum thyroid hormone profile showed significant differences between the groups ($p = 0.032$, $p < 0.05$), confirming a statistically significant alteration in thyroid function across premenopausal and postmenopausal women. Among hyperthyroid females, mean TSH levels were markedly suppressed, with values of 0.20 ± 0.94 µIU/mL in premenopausal and 0.30 ± 0.96 µIU/mL in postmenopausal subjects, compared with 3.28 ± 0.39 µIU/mL in controls. In contrast, T3 and T4 levels were significantly elevated. The mean T3 level in premenopausal hyperthyroid women was 2.80 ± 0.60 ng/mL and in postmenopausal 2.70 ± 0.70 ng/mL, whereas the control group exhibited 1.20 ± 0.20 ng/mL. Similarly, mean T4 levels were 12.4 ± 3.0 µg/dL in premenopausal hyperthyroid, 13.0 ± 2.5 µg/dL in postmenopausal hyperthyroid, and 7.8 ± 1.0 µg/dL in control females. In hypothyroid participants, a reverse hormonal trend was observed. The mean TSH level was markedly elevated at 17.9 ± 13.7 µIU/mL in premenopausal and 11.0 ± 3.0 µIU/mL in postmenopausal hypothyroid females, compared to 3.0 ± 0.5 µIU/mL in the control group. Mean T3 levels were lower in both premenopausal and postmenopausal hypothyroid women (1.1 ± 0.3 ng/mL and 1.0 ± 0.4 ng/mL, respectively) compared to controls (1.5 ± 0.3 ng/mL). T4 levels followed a similar pattern, recorded at 3.2 ± 0.8 µg/dL and 2.8 ± 0.7 µg/dL for premenopausal and postmenopausal hypothyroid females, respectively, versus 8.0 ± 1.2 µg/dL among controls.

Clinical assessment revealed a strong relationship between thyroid dysfunction and reproductive abnormalities. Menstrual irregularities were reported in 72% of premenopausal hypothyroid females and 62% of premenopausal hyperthyroid females, while no significant disturbances were reported among euthyroid controls. Although postmenopausal women naturally lacked menstrual activity, they demonstrated more severe hormonal dysregulation, with higher serum TSH and reduced T3/T4 in hypothyroid women and suppressed TSH with persistently elevated T3/T4 in hyperthyroid subjects, indicating intensified endocrine imbalance post-menopause. Overall, the study established a consistent pattern of high TSH with low T3 and T4 in hypothyroidism and the opposite trend in hyperthyroidism. These differences were statistically significant across both premenopausal and postmenopausal cohorts, underscoring the profound impact of thyroid dysfunction on hormonal equilibrium and menstrual physiology. To further explore the quantitative associations between thyroid hormones, correlation and mean difference analyses were performed. Pearson's correlation revealed a strong inverse relationship between TSH and both T3 ($r = –0.72$) and T4 ($r = –0.89$), indicating that as TSH increased, T3 and T4 levels significantly decreased across the study groups. Conversely, T3 and T4 demonstrated a strong positive correlation ($r = 0.93$), suggesting a parallel rise in both hormones in response to thyroid hyperactivity. Post hoc mean difference analysis showed a negligible difference in TSH between premenopausal and postmenopausal hyperthyroid females ($\Delta = 0.1$ µIU/mL), while a more pronounced difference was observed

between premenopausal and postmenopausal hypothyroid groups (Δ = 6.9 µIU/mL), reflecting a decline in TSH levels after menopause in hypothyroid women. The mean difference between hyperthyroid and hypothyroid females for T3 was 1.6 ng/mL and for T4 was 8.9 µg/dL, reinforcing the substantial hormonal divergence between the two thyroid dysfunction types. These results underscore significant interhormonal relationships and strengthen the evidence of a distinct endocrine pattern across menopausal groups.

Table 1: Thyroid hormonal parameters of pre-menopausal hyperthyroid females and postmenopausal hyperthyroid females with control group

Hormonal Parameter (units)	Pre-menopausal Hyperthyroid (n = 50)	Post-menopausal Hyperthyroid (n = 50)	Control (n = 50)
TSH (µIU/mL)	0.20 ± 0.94	0.30 ± 0.96	3.28 ± 0.39
T3 (ng/mL)	2.80 ± 0.60	2.70 ± 0.70	1.20 ± 0.20
T4 (µg/dL)	12.4 ± 3.0	13.0 ± 2.5	7.8 ± 1.0

Table 2: Thyroid hormonal parameters of pre-menopausal hypothyroid females and postmenopausal hypothyroid females with control group

Hormonal Parameter (units)	Pre-menopausal Hypothyroid (n = 50)	Post-menopausal Hypothyroid (n = 50)	Control (n = 50)
TSH (µIU/mL)	17.9 ± 13.7	11.0 ± 3.0	3.0 ± 0.5
T3 (ng/mL)	1.1 ± 0.3	1.0 ± 0.4	1.5 ± 0.3
T4 (µg/dL)	3.2 ± 0.8	2.8 ± 0.7	8.0 ± 1.2

Table 3: Correlation and Mean Difference Analysis of Thyroid Hormones

Parameter Comparison	Correlation Coefficient (r) / Mean Difference
TSH vs T3	r = –0.72
TSH vs T4	r = –0.89
T3 vs T4	r = +0.93
Pre- vs Post-menopausal Hyperthyroid (TSH)	Δ = 0.1 µIU/mL
Pre- vs Post-menopausal Hypothyroid (TSH)	Δ = 6.9 µIU/mL
Hyperthyroid vs Hypothyroid (T3)	Δ = 1.6 ng/mL
Hyperthyroid vs Hypothyroid (T4)	Δ = 8.9 µg/dL

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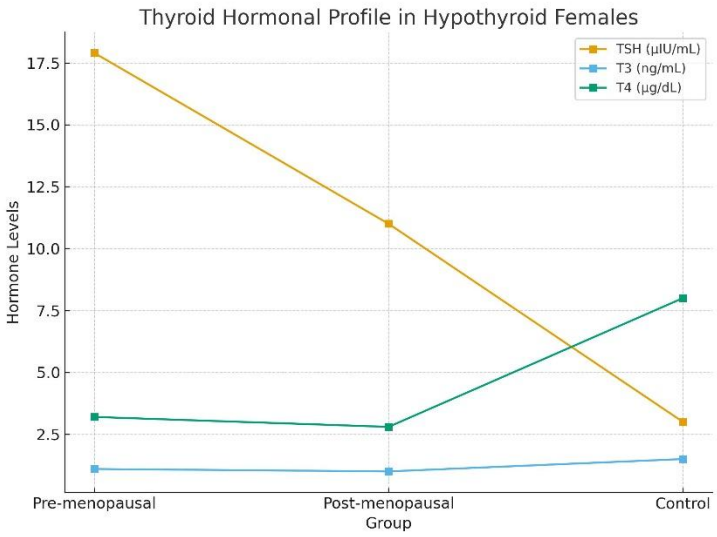

Figure 2 Thyroid Hormonal Profile in Hypothyroid Females

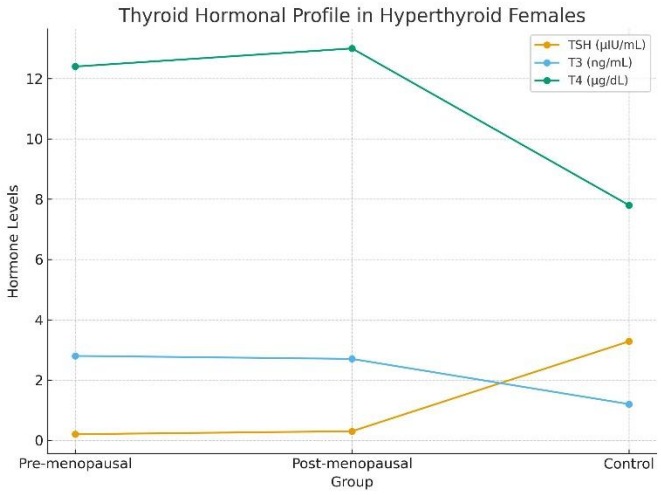

Figure 2 Thyroid Hormonal Profile in Hyperthyroid Females

DISCUSSION

Thyroid disorders represent the second most common endocrine abnormality after diabetes, with a disproportionately higher prevalence among women, particularly in the form of hypothyroidism and autoimmune thyroiditis (15). The findings of this study reaffirm the high occurrence of thyroid dysfunction in both pre- and postmenopausal women, approximating 26%, and highlight its increasing frequency with advancing age. These results are consistent with prior reports indicating that the risk of thyroid disorders escalates after menopause due to hormonal transitions that influence thyroid function and peripheral metabolism of thyroid hormones (16). The current study observed a significantly elevated mean thyroid-stimulating hormone (TSH) level and decreased free triiodothyronine (FT3) and free thyroxine (FT4) concentrations in hypothyroid females compared to hyperthyroid and euthyroid controls. This hormonal pattern reflects impaired thyroid hormone synthesis or reduced peripheral conversion, supporting earlier studies demonstrating diminished T4 to T3 conversion in hypothyroidism (17). Conversely, hyperthyroid women exhibited suppressed TSH levels and increased FT3 and FT4, indicative of enhanced glandular activity, likely associated with autoimmune thyroiditis or Graves' disease (18). Such findings reinforce the intricate feedback mechanism between pituitary and thyroid function, wherein excessive circulating thyroid hormones suppress TSH secretion, leading to persistent hormonal imbalance. The study also identified that premenopausal hypothyroid women experienced more pronounced menstrual irregularities (80%) compared to their hyperthyroid counterparts (65%). These outcomes are in line with previous research that described hypothyroidism as a major contributor to oligomenorrhea, polymenorrhea, or amenorrhea due to disrupted hypothalamic–pituitary–ovarian (HPO) axis function (19). Elevated TSH and altered prolactin levels in hypothyroidism may inhibit gonadotropin-releasing hormone secretion, thereby affecting ovulation and menstrual regularity. Hyperthyroidism, in contrast, tends to cause hypomenorrhea or menstrual shortening as a result of increased sex hormone–binding globulin (SHBG) and accelerated estrogen metabolism (20). These reproductive manifestations emphasize the direct and indirect influence of thyroid hormones on ovarian activity and sex hormone regulation. In postmenopausal women, the effect of thyroid dysfunction appeared more pronounced hormonally, although menstrual disturbances were absent. The decline in estrogen following menopause alters thyroid-binding globulin (TBG) and peripheral hormone metabolism, contributing to fluctuations in TSH levels across ethnic and demographic groups (21). The study found significant correlation between T4 and T3 levels in postmenopausal hypothyroid females, signifying synchronized downregulation of thyroid function, while the expected inverse correlation between TSH and T3 was not observed in hyperthyroid postmenopausal subjects, possibly due to adaptive feedback variations or small sample heterogeneity.

The clinical implication of these findings lies in the strong association between thyroid dysfunction and reproductive health. Thyroid hormones exert a direct effect on ovarian folliculogenesis and an indirect impact through SHBG modulation, thereby influencing fertility outcomes. These results support routine thyroid screening in women presenting with menstrual disorders or unexplained infertility to

facilitate early diagnosis and treatment (22,23). The strengths of this study include its comparative evaluation across both pre- and postmenopausal stages and the use of standardized biochemical assays, which enhanced the accuracy of hormonal estimation. However, the study was limited by its cross-sectional design, which precludes causal inference. The relatively small sample size within each subgroup and lack of adjustment for confounders such as body mass index, diet, and concurrent autoimmune conditions may have influenced the observed hormonal variations. Additionally, the study did not include assessment of thyroid antibody titers, which could have provided insight into the autoimmune etiology of thyroid dysfunction. Future studies should incorporate longitudinal follow-up and regression modeling to elucidate causal relationships between thyroid hormones, menopausal status, and reproductive outcomes. Expanding the sample size and including parameters such as thyroid peroxidase antibodies, estrogen levels, and SHBG concentrations could improve understanding of the endocrine interplay between thyroid and reproductive axes (24). In conclusion, this study demonstrates that thyroid dysfunction, whether hypothyroid or hyperthyroid, significantly affects hormonal equilibrium in women, with premenopausal subjects manifesting reproductive abnormalities and postmenopausal women showing heightened endocrine dysregulation. These findings highlight the importance of early hormonal screening and management of thyroid dysfunction as a crucial step in maintaining female reproductive and metabolic health.

CONCLUSION

The present study concludes that thyroid dysfunction profoundly influences hormonal equilibrium and reproductive well-being in both premenopausal and postmenopausal women. The findings confirm that disturbances in thyroid hormone secretion alter the balance of TSH, T3, and T4, leading to significant endocrine disruptions. Among premenopausal women, thyroid abnormalities were closely linked with menstrual irregularities, while in postmenopausal women, menopause appeared to intensify hormonal instability associated with thyroid disease. Overall, the research underscores that both hypothyroidism and hyperthyroidism have far-reaching implications for women's health, reinforcing the need for routine thyroid screening as part of reproductive and general health evaluations. Early identification and appropriate management of thyroid disorders can play a pivotal role in restoring hormonal harmony and safeguarding long-term well-being in women.

AUTHOR CONTRIBUTION

Author	Contribution
Fareen Bano Iftakhar	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Syeda Hijab Zainab	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Usama Ehsan	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Muhammad Umar*	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Aqsa Shaukat	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Taha Rehman	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published

Author	Contribution
Rimsha Ali	Contributed to study concept and Data collection Has given Final Approval of the version to be published
Wasi ur Rehman	Writing - Review & Editing, Assistance with Data Curation

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