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Original Research Paper

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A COMPARATIVE STUDY OF THYROID FUNCTION AND CLINICAL PROFILE OF NORMOTENSIVE AND HYPERTENSIVE EUTHYROID PATIENTS

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ABSTRACT

Background: Hypertension and subclinical thyroid variations both contribute significantly to cardiovascular disease risk. This study compares thyroid function parameters and cardiovascular risk profiles between normotensive and hypertensive euthyroid adults. **Methods:** This comparative observational study included 100 consecutive euthyroid adult patients (50 hypertensive, 50 normotensive) aged 25 to 70 years at a tertiary care hospital. Patients were evaluated for demographic profiles, thyroid function (TSH, FT3, FT4), C-reactive protein (CRP), lipid profiles, and the Reynolds Risk Score. **Results:** The mean age of hypertensive subjects was 55.32 years, significantly higher than normotensives at 49.48 years ($p < 0.01$). TSH levels were significantly elevated in the hypertensive group (1.95 mU/L) compared to normotensives (1.41 mU/L) ($p < 0.01$). The FT4×TSH product and CRP levels were also significantly higher in hypertensive patients ($p < 0.01$). Furthermore, the mean Reynolds Risk Score was significantly higher in hypertensives (11.86) versus normotensives (5.49). TSH showed a positive correlation with both systolic and diastolic blood pressure. **Conclusion:** Hypertensive euthyroid individuals carry a higher cardiovascular risk burden, marked by elevated TSH, FT4×TSH product, and CRP levels. Routine screening of thyroid profiles in hypertensive patients may offer early insights into peripheral thyroid resistance and overall cardiovascular risk.

KEYWORDS : Hypertension, Euthyroid, Thyroid Stimulating Hormone, Reynolds Risk Score, Cardiovascular Risk.

INTRODUCTION

Hypertension is a major public health problem globally and a leading cause of cardiovascular morbidity and mortality in both developed and developing countries. Uncontrolled hypertension can cause severe end-organ damage and is characterized by abnormalities in cardiac output, systemic vascular resistance, and arterial compliance. Thyroid hormones directly and indirectly influence blood pressure by affecting systemic vascular resistance, resting heart rate, endothelial function, and left ventricular contractility.

While overt hyperthyroidism and hypothyroidism are well-established secondary causes of hypertension, the relationship between subtle thyroid profile variations within the normal reference range and clinical outcomes in hypertensive individuals remains largely unexplored. This study evaluates the thyroid profile (TSH, FT3, FT4) alongside clinical features in normotensive and hypertensive euthyroid patients. Additionally, it assesses the association between systemic arterial blood pressure, peripheral thyroid sensitivity, and overall cardiovascular risk, measured by the Reynolds Risk Score, in these euthyroid subjects.

MATERIAL AND METHODS

This comparative observational study was conducted in the Department of General Medicine at a tertiary care hospital from September 2023 to December 2024. A total of 100 adult patients (aged 25 to 70 years) were enrolled, consisting of 50 established hypertensive patients on treatment for at least six months and 50 normotensive controls. All participants had confirmed euthyroid status based on thyroid function testing. Patients receiving thyroxine therapy or thyroid-altering medications, and those with a history of stroke, myocardial infarction, sepsis, or diabetes mellitus, were excluded from the study.

Following ethical approval and written informed consent, demographic data, clinical history, and physical examination metrics—including body mass index (BMI) and waist-to-hip ratio (WHR)—were recorded. Blood pressure was measured using a mercury sphygmomanometer after 5 minutes of rest, with the average of three readings recorded. Laboratory investigations included free triiodothyronine (FT3), free thyroxine (FT4), thyroid-stimulating hormone (TSH), C-reactive protein (CRP), and lipid profiles. The FT4×TSH product was calculated to evaluate altered peripheral sensitivity to thyroid hormones, and the Reynolds Risk Score was calculated to assess 10-year cardiovascular risk.

Data were entered into a pre-designed proforma and analyzed using SPSS Version 26.0. Quantitative data were analyzed using unpaired t-tests or Mann-Whitney tests, while qualitative data associations were assessed via Chi-Square tests. A p-value < 0.05 was considered statistically significant.

RESULTS

The study cohort consisted of 100 participants, evenly divided into hypertensive (n=50) and normotensive (n=50) groups. The mean age of hypertensive patients was 55.32 ± 10.17 years, which was significantly higher than the normotensive group (49.48 ± 8.17 years, $p < 0.01$). Gender distribution, BMI, and WHR showed no statistically significant differences between the two groups. However, a significant positive family history of hypertension was observed in 50% of the hypertensive group, compared to 16% in the normotensive group ($p < 0.01$).

Table 1. Baseline Demographic Characteristics of Study Groups

Variable	Category	Normotensive (n=50)	Hypertensive (n=50)	p-value
Age (years)	Mean ± SD	49.48 ± 8.17	55.32 ± 10.17	< 0.01
Gender	Male	18 (36.0%)	25 (50.0%)	0.09
	Female	32 (64.0%)	25 (50.0%)	
BMI (kg/m²)	Mean ± SD	25.42 ± 4.33	26.82 ± 4.33	0.27
WHR	Mean ± SD	0.87 ± 0.05	0.89 ± 0.07	0.51
Family Hx of HTN	Yes	8 (16.0%)	25 (50.0%)	< 0.01
	No	42 (84.0%)	25 (50.0%)	

Symptoms suggestive of subtle thyroid dysfunction and smoking status showed no significant variation between the two groups, confirming similarity in clinical presentation across the euthyroid participants.

Table 2. Distribution of Clinical Symptoms and Smoking Risk Factors

Variable	Category	Normotensive (n=50)	Hypertensive (n=50)	p-value
Smoking Status	Non-Smoker	21 (42.0%)	34 (68.0%)	0.15
	Ex-smoker	4 (8.0%)	2 (4.0%)	
	< 10 years	10 (20.0%)	6 (12.0%)	
	10-19 years	6 (12.0%)	2 (4.0%)	
	> 20 years	9 (18.0%)	6 (12.0%)	
Hypothyroid Symptoms	Yes	9 (18.0%)	12 (24.0%)	0.59
	No	41 (82.0%)	38 (76.0%)	
Hyperthyroid Symptoms	Yes	10 (20.0%)	9 (18.0%)	1.00
	No	40 (80.0%)	41 (82.0%)	

Biochemical analysis (Table 3) revealed that TSH levels were significantly elevated in the hypertensive group (1.95 ± 0.98 mU/L) compared to the normotensive group (1.41 ± 0.99 mU/L, $p < 0.01$). While FT3 and FT4 levels remained comparable, the FT4×TSH product was significantly higher among hypertensives (2.55 ± 0.94) than normotensives (2.10 ± 0.78, $p < 0.01$). Systemic inflammation, marked by CRP, was also markedly elevated in hypertensive individuals (17.77 ± 16.05 mg/L) versus controls (5.31 ± 9.87 mg/L, $p < 0.01$). Lipid profiles were statistically comparable.

Table 3. Biochemical and Thyroid Profile Parameters Among Study Groups

Variable	Normotensive (Mean ± SD)	Hypertensive (Mean ± SD)	p-value
TSH (mU/L)	1.41 ± 0.99	1.95 ± 0.98	< 0.01
FT3 (pg/mL)	3.18 ± 0.53	3.19 ± 0.69	0.942
FT4 (pmol/L)	1.49 ± 0.22	1.31 ± 0.35	0.223
FT4 × TSH	2.10 ± 0.78	2.55 ± 0.94	< 0.01
CRP (mg/L)	5.31 ± 9.87	17.77 ± 16.05	< 0.01
HDL (mg/dL)	45.88 ± 11.54	42.70 ± 28.05	0.46
TC (mg/dL)	186.84 ± 32.35	185.42 ± 42.15	0.85

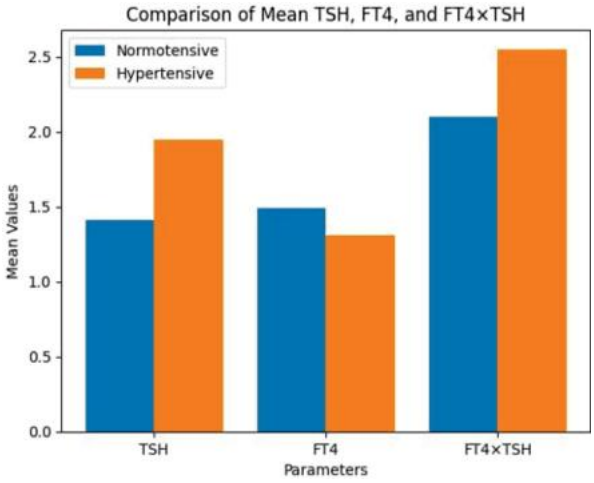

Figure 1: Comparison of Thyroid Profile Parameters Among Study Groups. TSH and the FT4×TSH Product are Significantly Elevated in the Hypertensive Group Despite Both Groups Remaining Clinically Euthyroid.

Cardiovascular risk assessment demonstrated that the mean Reynolds Risk Score was significantly higher in hypertensive subjects (11.86 ± 10.06) compared to normotensive subjects (5.49 ± 1.52, $p < 0.01$). High cardiovascular risk was identified in 20% of the hypertensive cohort, whereas no normotensive individuals fell into this category. Furthermore, TSH levels exhibited a statistically significant positive correlation with both systolic blood pressure ($r=0.34$, $p=0.02$) and diastolic blood pressure ($r=0.21$, $p=0.04$).

Table 4. Reynolds Risk Score Category Distribution

Reynolds Risk Category	Normotensive (n=50)	Hypertensive (n=50)	p-value
Not Applicable (NA)	11 (22.0%)	7 (14.0%)	< 0.01
Low	12 (24.0%)	12 (24.0%)	
Moderate	27 (54.0%)	21 (42.0%)	
High	0 (0.0%)	10 (20.0%)	

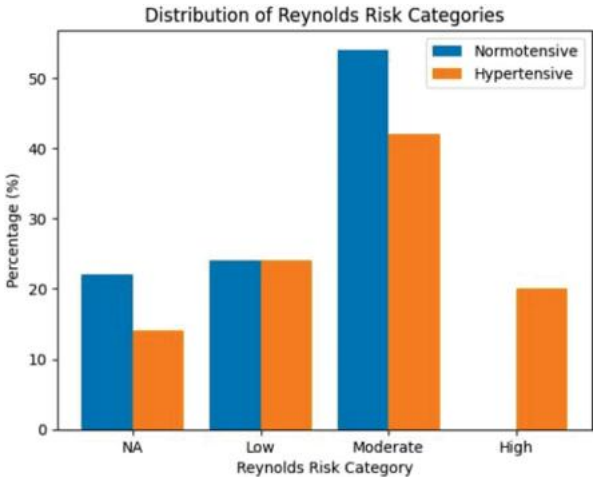

Figure 2: Distribution of Study Groups Per Reynolds Risk Score Category. A Significantly Higher Percentage of Hypertensive Patients Fall into the High-risk Category Compared to Normotensive Subjects.

DISCUSSION

This study demonstrates that subtle variations in TSH and peripheral thyroid indices occur in clinically euthyroid hypertensive patients and correlate with elevated cardiovascular risk. Our observation of significantly higher TSH levels in the hypertensive group supports the hypothesis that higher-normal TSH is associated with increased systolic and diastolic blood pressure. The positive correlation between TSH and arterial blood pressure further implicates TSH in influencing vascular resistance, even within the normal reference range.

The FT4×TSH product, an indicator of peripheral sensitivity to thyroid hormones, was significantly elevated in hypertensive patients. This suggests a possible phenomenon of peripheral thyroid hormone resistance, wherein reduced tissue-level action of thyroid hormones prompts a compensatory TSH elevation. Modest impairments in thyroid hormone activity can lead to altered vascular compliance, increased systemic vascular resistance, and increased arterial stiffness.

Additionally, the hypertensive cohort exhibited a significantly greater inflammatory burden, evidenced by elevated CRP levels. Systemic inflammation likely contributes to endothelial dysfunction, which acts synergistically with hormonal dysregulation to elevate cardiovascular risk. Consequently, the Reynolds Risk Score, which integrates CRP alongside traditional factors, was substantially higher in hypertensive subjects, emphasizing the multidimensional nature of their cardiovascular vulnerability. The absence of significant lipid profile differences between the groups highlights the limitations of relying exclusively on traditional lipid parameters for cardiovascular risk stratification.

Limitations of this study include its cross-sectional design, which precludes the establishment of temporal or causal relationships between elevated TSH, the FT4×TSH product, and the onset of systemic hypertension. Furthermore, being a single-center study without specific genetic profiling or thyroid antibody testing limits the broader generalizability of the mechanistic findings.

CONCLUSION

Hypertensive euthyroid patients exhibit raised TSH levels, higher FT4×TSH products, and elevated Reynolds Risk Scores compared to normotensive controls. The FT4×TSH product serves as a potentially valuable biomarker for identifying early peripheral thyroid hormone resistance, which may contribute to systemic hypertension and heightened cardiovascular risk. Routine screening of thyroid profiles and inflammatory markers in hypertensive patients enables more precise cardiovascular risk stratification and may provide new individualized starting points for early intervention.

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