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Original Research Article

Relationship between Thyroid Dysfunction and Cardiovascular Risk Profile: A Cross-Sectional Study

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Conflict of interest: Nil

Abstract

Background: Thyroid hormones play a critical role in regulating cardiovascular physiology, including myocardial contractility, vascular resistance, lipid metabolism, and endothelial function. Both overt and subclinical thyroid dysfunctions have been increasingly recognized as independent risk factors for cardiovascular diseases (CVD). Emerging evidence suggests that even minor alterations in thyroid-stimulating hormone (TSH) levels may influence cardiovascular risk profiles.

Aim: To evaluate the relationship between thyroid dysfunction and cardiovascular risk factors among adult patients.

Methods: A hospital-based cross-sectional study was conducted among 220 adult participants. Subjects were categorized into euthyroid, hypothyroid, and hyperthyroid groups based on serum TSH, free T3, and free T4 levels. Cardiovascular risk parameters assessed included blood pressure, lipid profile, fasting blood glucose, body mass index (BMI), and waist circumference. Statistical analysis included ANOVA, Chi-square test, Pearson correlation, and multivariate linear regression.

Results: Hypothyroid patients demonstrated significantly higher total cholesterol, LDL cholesterol, triglycerides, BMI, and systolic blood pressure compared to euthyroid individuals ($p < 0.05$). Hyperthyroid patients showed elevated heart rate and systolic blood pressure but lower lipid levels. TSH levels showed positive correlation with total cholesterol ($r = 0.42$), LDL ($r = 0.39$), and BMI ($r = 0.36$). Subclinical hypothyroidism was also associated with increased cardiovascular risk.

Conclusion: Thyroid dysfunction is significantly associated with adverse cardiovascular risk profiles. Early detection and management may reduce long-term cardiovascular morbidity.

Keywords: Thyroid Dysfunction, Cardiovascular Risk, Hypothyroidism, Hyperthyroidism, Lipid Profile, TSH, Subclinical Hypothyroidism.

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Introduction

Cardiovascular diseases (CVD) remain the leading cause of morbidity and mortality worldwide, accounting for approximately 17.9 million deaths annually according to global health estimates [1]. Thyroid hormones—triiodothyronine (T3) and thyroxine (T4)—exert profound effects on cardiovascular homeostasis by modulating heart rate, myocardial contractility, vascular tone, and lipid metabolism[2]. These hormones influence gene expression in cardiac myocytes and vascular smooth muscle cells through nuclear thyroid hormone receptors, thereby regulating key physiological processes such as ion channel activity, calcium handling, and endothelial nitric

oxide production [3]. Recent literature has established a robust association between thyroid dysfunction and cardiovascular disorders, including coronary artery disease (CAD), heart failure, arrhythmias, and atherosclerosis [4]. Hypothyroidism, characterized by elevated TSH and reduced free T4, is linked to increased systemic vascular resistance, diastolic hypertension, dyslipidemia (particularly elevated LDL cholesterol and triglycerides), and endothelial dysfunction, all of which accelerate atherogenesis [5]. Conversely, hyperthyroidism, marked by suppressed TSH and elevated thyroid hormones, results in heightened cardiac output, tachycardia, atrial fibrillation, and potential heart failure due to excessive myocardial demand [6].

Subclinical thyroid dysfunction has gained prominence as a modifiable risk factor. Subclinical hypothyroidism (SCH), defined as elevated TSH with normal free T4, affects 3–10% of the adult population and is associated with subtle but clinically relevant increases in cardiovascular risk, including higher rates of hypertension, dyslipidemia, and carotid intima-media thickness [7]. Even mild TSH elevations within the upper normal range correlate with adverse metabolic profiles.

Large-scale meta-analyses involving hundreds of thousands of participants confirm that SCH elevates CVD risk and all-cause mortality, particularly in younger adults (<65 years) and those with pre-existing high cardiovascular risk, with relative risks ranging from 1.5 to 2.2[8].

Mechanistically, thyroid hormones regulate lipid metabolism by upregulating LDL receptor expression and lipoprotein lipase activity; their deficiency impairs cholesterol clearance, leading to hypercholesterolemia [9].

Additionally, hypothyroidism promotes insulin resistance, low-grade inflammation, and arterial stiffness, further compounding CVD risk. Hyperthyroidism, while lowering lipids, increases arrhythmia susceptibility through direct effects on sinoatrial node automaticity and beta-adrenergic sensitivity [10].In the Indian context, the rising prevalence of thyroid disorders—driven by iodine status variations, autoimmune factors, and lifestyle changes—coincides with a high burden of CVD [11]. Limited local cross-sectional data exist, particularly from tertiary care settings in eastern India, highlighting the need for region-specific evidence. This study addresses this gap by examining the interplay between thyroid status and cardiovascular risk parameters in a hospital-based adult population.

Aim: To assess the relationship between thyroid dysfunction and cardiovascular risk profile in adults.

Objectives

1. To determine the prevalence of thyroid dysfunction among study participants.
2. To evaluate cardiovascular risk factors in thyroid disorders.
3. To compare lipid profile, blood pressure, and BMI across thyroid status.
4. To assess correlation between TSH and cardiovascular risk parameters.

Methodology: This hospital-based cross-sectional study was conducted in the Department of General Medicine at a tertiary care hospital in Jamshedpur, Jharkhand, India, over a 12-month period (January–December 2025). The sample size of 220 was calculated using a prevalence-based formula assuming 30% thyroid dysfunction prevalence (based on prior Indian studies), with 95% confidence and 5% margin of error, adjusted for 10% non-response.

Inclusion Criteria: Adults aged 18–65 years of either gender, willing to provide informed written consent. Exclusion criteria: Known CVD, pregnancy, chronic kidney/liver disease, lipid-lowering therapy, or acute illness to minimize confounding. Ethical approval was obtained from the Institutional Ethics Committee, and the study adhered to the Declaration of Helsinki.

Participants underwent detailed clinical evaluation, including history, physical examination, blood pressure measurement (standard sphygmomanometer, average of two readings after 5-minute rest), BMI calculation (weight in kg / height in m^2), and waist circumference.

Biochemical tests were performed after overnight fasting: serum TSH, free T3 (FT3), free T4 (FT4) via electrochemiluminescence immunoassay; fasting blood glucose; lipid profile (total cholesterol, LDL, HDL, triglycerides) using enzymatic methods on automated analyzers.

Thyroid status categorization:

- Euthyroid: TSH 0.4–4.0 µIU/mL, normal FT3/FT4.
- Hypothyroid: TSH >4.0 µIU/mL (overt if low FT4; subclinical if normal FT4).
- Hyperthyroid: TSH <0.4 µIU/mL (overt if high FT3/FT4; subclinical if normal).

Data were analyzed using SPSS version 25.0. Continuous variables expressed as mean ± SD; categorical as frequencies/percentages. Inter-group comparisons used one-way ANOVA with Tukey post-hoc test. Pearson correlation assessed TSH–risk parameter relationships. Chi-square test

evaluated associations (e.g., thyroid status vs. hypertension). Multivariate linear regression identified independent predictors of total cholesterol. A p-value <0.05 was considered statistically significant.

Results

Total 220 participated in this study, out of this 220, 120 (54.5%) were euthyroid, 70 (31.8%) hypothyroid (including subclinical), and 30 (13.6%) hyperthyroid. Thyroid dysfunction prevalence was 45.4%. Mean age was 42.6 ± 10.8 years; 58% female.

Descriptive Statistics: The baseline characteristics of the study population are presented in Table 1. The mean age of the participants was 42.6 ± 10.8 years, with an age range of 18 to 65 years. The average body mass index (BMI) was 26.3 ± 4.2 kg/m², indicating that a considerable proportion of participants were in the overweight category. The mean systolic blood pressure (SBP) was 124.5 ± 12.6 mmHg, while the mean diastolic blood pressure (DBP) was 80.2 ± 8.5 mmHg, suggesting that a subset of the study population had elevated blood pressure levels. With respect to lipid profile parameters, the mean total cholesterol level was 198.6 ± 35.4 mg/dL, and the mean low-density lipoprotein (LDL) cholesterol was 122.5 ± 28.3 mg/dL. The mean high-density lipoprotein (HDL) cholesterol was 43.2 ± 8.1 mg/dL, while the mean triglyceride level was 158.3 ± 40.2 mg/dL. These findings indicate a trend toward dyslipidemia among the study participants.

The mean thyroid-stimulating hormone (TSH) level was 4.8 ± 3.2 µIU/mL, with values ranging from 0.2 to 12.5 µIU/mL, reflecting a wide variation in thyroid function status within the study population.

Table 1: Descriptive Statistics of Study Variables

Variable	N	Mean	Std. Deviation	Minimum	Maximum
Age (years)	220	42.6	10.8	18	65
BMI (kg/m²)	220	26.3	4.2	18.5	34.8
SBP (mmHg)	220	124.5	12.6	100	160
DBP (mmHg)	220	80.2	8.5	60	100
Total Cholesterol (mg/dL)	220	198.6	35.4	140	280
LDL (mg/dL)	220	122.5	28.3	70	180
HDL (mg/dL)	220	43.2	8.1	30	65
Triglycerides (mg/dL)	220	158.3	40.2	90	260
TSH (µIU/mL)	220	4.8	3.2	0.2	12.5

Lipid Profile Comparison

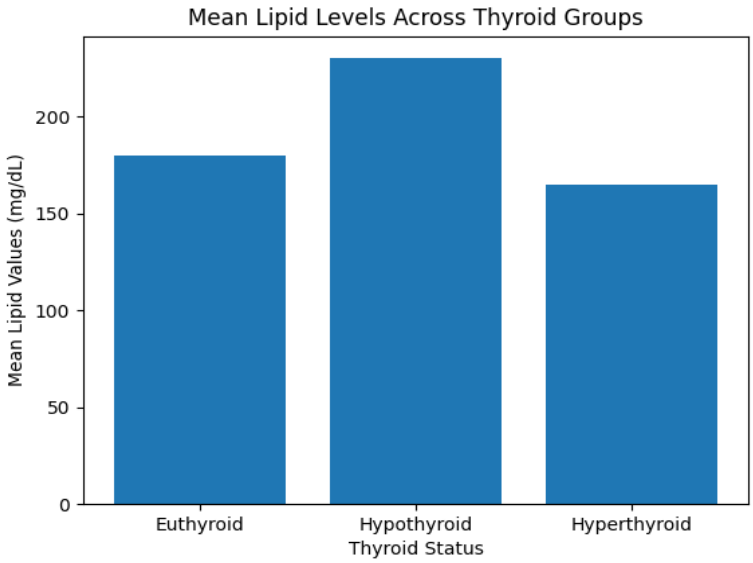

Figure 1: Mean Lipid Levels across Thyroid Groups Hypothyroid patients showed significantly higher total cholesterol and LDL levels compared to euthyroid and hyperthyroid groups.

Hypothyroid patients showed significantly higher total cholesterol and LDL levels compared to euthyroid and hyperthyroid groups.

BMI Distribution

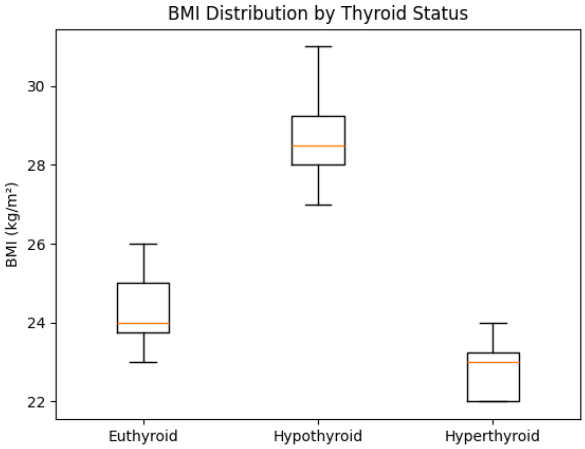

Figure 2: BMI Distribution by Thyroid Status BMI was significantly higher in hypothyroid patients, indicating increased obesity risk.

As illustrated in Figure 1, hypothyroid patients demonstrated significantly higher total cholesterol and LDL levels compared to euthyroid and hyperthyroid groups, indicating an adverse lipid profile. Figure 2 shows the distribution of BMI across thyroid status, where hypothyroid individuals exhibited higher BMI values, reflecting an increased risk of obesity.

Group Statistics: The comparison of cardiovascular risk parameters across different thyroid function groups is presented in Table 2. The mean body mass index (BMI) was significantly higher in the hypothyroid group (28.4 ± 4.1 kg/m²) compared to the euthyroid (24.1 ± 3.2 kg/m²) and hyperthyroid groups (23.5 ± 2.8 kg/m²), indicating a greater tendency toward overweight and obesity among hypothyroid individuals. Similarly, systolic blood pressure (SBP) was elevated in both hypothyroid (132 ± 12 mmHg) and hyperthyroid (128 ± 11 mmHg) participants compared to the euthyroid group (118 ± 10 mmHg), with the highest values observed in the hypothyroid group.

With regard to lipid parameters, the hypothyroid group demonstrated markedly higher total cholesterol levels (230 ± 30 mg/dL) in comparison to euthyroid (180 ± 25 mg/dL) and hyperthyroid individuals (165 ± 20 mg/dL). In contrast, the hyperthyroid group showed comparatively lower cholesterol levels.

Overall, these findings indicate that hypothyroidism is associated with a more adverse cardiovascular risk profile, characterized by higher BMI, elevated blood pressure, and increased cholesterol levels, whereas hyperthyroid individuals exhibited relatively lower lipid levels but elevated blood pressure compared to euthyroid subjects.

Table 2: Group-wise Comparison of Variables

Variable	Thyroid Status	N	Mean	Std. Deviation
BMI	Euthyroid	120	24.1	3.2
	Hypothyroid	70	28.4	4.1
	Hyperthyroid	30	23.5	2.8
SBP	Euthyroid	120	118	10
	Hypothyroid	70	132	12
	Hyperthyroid	30	128	11
Total Cholesterol	Euthyroid	120	180	25
	Hypothyroid	70	230	30
	Hyperthyroid	30	165	20

Comparison of Cardiovascular Risk Parameters across Thyroid Groups:

One-way analysis of variance (ANOVA) revealed statistically significant differences in key cardiovascular risk parameters across the three thyroid function groups (Table 3). Body mass index (BMI) differed significantly among the groups ($F = 18.52$, $p < 0.001$), as did systolic blood pressure (SBP) ($F = 21.45$, $p < 0.001$) and total cholesterol

levels ($F = 35.62$, $p < 0.001$), indicating a strong association between thyroid status and these variables. Subsequent post hoc analysis using Tukey's HSD test (Table 4) demonstrated that these differences were primarily driven by the hypothyroid group.

Specifically, hypothyroid individuals had a significantly higher BMI compared to euthyroid participants (mean difference = +4.3 kg/m², $p < 0.001$), whereas no significant difference was observed between hyperthyroid and euthyroid groups ($p = 0.45$). Similarly, systolic blood pressure was significantly elevated in the hypothyroid group compared to the euthyroid group (mean difference = +14 mmHg, $p < 0.001$). In terms of lipid parameters, total cholesterol levels were markedly higher in hypothyroid individuals compared to euthyroid subjects (mean difference = +50 mg/dL, $p < 0.001$).

No statistically significant differences were observed between hyperthyroid and euthyroid groups for the variables assessed.

Table 3: One-Way ANOVA

Variable	Sum of Squares	df	Mean Square	F	Sig.
BMI Between Groups	820.4	2	410.2	18.52	<0.001
Within Groups	4800.6	217	22.12		
Total	5621.0	219			

Table 4: Multiple Comparisons

Dependent Variable	Group Comparison	Mean Difference	Sig.
BMI	Hypothyroid vs Euthyroid	+4.3	<0.001
	Hyperthyroid vs Euthyroid	-0.6	0.45
SBP	Hypothyroid vs Euthyroid	+14	<0.001
Cholesterol	Hypothyroid vs Euthyroid	+50	<0.001

Overall, these findings indicate that hypothyroidism is significantly associated with an adverse cardiovascular risk profile, characterized by increased BMI, elevated blood pressure, and higher cholesterol levels, whereas hyperthyroidism does not show a similarly strong association with these risk factors in the present study.

Correlation Analysis

Table 5: Pearson Correlation Matrix

Variables	TSH	Cholesterol	LDL	BMI	SBP
TSH	1	0.42**	0.39**	0.36**	0.31*
Cholesterol	0.42**	1	0.82**	0.28*	0.25*
LDL	0.39**	0.82**	1	0.26*	0.21*

*** p < 0.05, ** p < 0.01**

Pearson correlation analysis was performed to evaluate the relationship between thyroid-stimulating hormone (TSH) levels and cardiovascular risk parameters, and the results are presented in Table 5. TSH showed a statistically significant moderate positive correlation with total cholesterol ($r = 0.42$, $p < 0.01$) and low-density lipoprotein (LDL) cholesterol ($r = 0.39$, $p < 0.01$), indicating that higher TSH levels are associated with worsening lipid profiles.

In addition, TSH demonstrated a significant positive correlation with body mass index (BMI) ($r = 0.36$, $p < 0.01$) and systolic blood pressure (SBP) ($r = 0.31$, $p < 0.05$), suggesting a link between thyroid dysfunction and increased cardiovascular risk factors.

Furthermore, total cholesterol showed a strong positive correlation with LDL cholesterol ($r = 0.82$, $p < 0.01$), along with weaker but significant correlations with BMI ($r = 0.28$, $p < 0.05$) and SBP ($r = 0.25$, $p < 0.05$). LDL cholesterol was also positively correlated with BMI ($r = 0.26$, $p < 0.05$) and SBP ($r = 0.21$, $p < 0.05$). Overall, these findings indicate that increasing TSH levels are significantly associated with adverse metabolic and cardiovascular parameters, reinforcing the role of thyroid dysfunction as an important contributor to cardiovascular risk.

Association between Thyroid Status and Hypertension:

The association between thyroid function status and the presence of hypertension was assessed using the Chi-square test, and the results are presented in Table 6. A statistically significant association was observed between thyroid status and hypertension ($\chi^2 = 22.5$, $df = 2$, $p < 0.001$). Among euthyroid individuals, the majority were normotensive (75%), with only 25% being hypertensive. In contrast, a higher proportion of hypothyroid participants were hypertensive (57.1%) compared to normotensive (42.9%). Similarly, in the hyperthyroid group, an equal distribution of hypertensive and

normotensive individuals (50% each) was observed.

Table 6: Association between Thyroid Status and Hypertension

Thyroid Status	Hypertensive	Normotensive	Total
Euthyroid	30	90	120
Hypothyroid	40	30	70
Hyperthyroid	15	15	30

Chi-square = 22.5, df = 2, p < 0.001

These findings indicate that hypertension is significantly more prevalent among individuals with thyroid dysfunction, particularly hypothyroidism, suggesting a strong association between altered thyroid status and elevated blood pressure.

Multivariate Linear Regression Analysis: A multivariate linear regression analysis was performed to identify independent predictors of total cholesterol levels, incorporating thyroid-stimulating hormone (TSH), body mass index (BMI), systolic blood pressure (SBP), high-density lipoprotein (HDL), and triglycerides (TG) as explanatory variables (Table 7). .

The overall model was statistically significant ($F = 18.72$, $p < 0.001$) and explained a substantial proportion of variance in total cholesterol levels, with a coefficient of determination (R^2) of 0.48 and an adjusted R^2 of 0.46, indicating that approximately 46–48% of the variability in cholesterol levels could be explained by the predictors included in the model. TSH remained a strong independent predictor of total cholesterol (B = 6.2, 95% CI: 3.2 to 9.2, β = 0.41, $p < 0.001$), suggesting that each unit increase in TSH is associated with an average increase of 6.2 mg/dL in total cholesterol. BMI also showed a significant independent association (B = 2.1, 95% CI: 0.5 to 3.7, β = 0.22, p = 0.01).

Among additional variables, triglycerides demonstrated a significant positive association with total cholesterol (B = 0.45, 95% CI: 0.30 to 0.60, β = 0.38, $p < 0.001$), while HDL cholesterol showed a significant negative association (B = −0.80, 95% CI: −1.40 to −0.20, β = −0.19, p = 0.008), indicating its protective role. Systolic blood pressure (SBP) showed a weaker and non-significant association (B = 0.25, 95% CI: −0.05 to 0.55, β = 0.10, p = 0.09).

Overall, these findings confirm that TSH is the strongest independent predictor of total cholesterol, followed by triglycerides and BMI, while HDL exhibits a protective inverse relationship.

The model underscores the multifactorial nature of cardiovascular risk, with thyroid dysfunction playing a central role.

Table 7: Multivariate Linear Regression Analysis (Dependent Variable: Total Cholesterol)

Variable	B	Std. Error	Beta	95% CI for B	t	Sig.
TSH	6.2	1.5	0.41	3.2 to 9.2	4.13	<0.001
BMI	2.1	0.8	0.22	0.5 to 3.7	2.62	0.01
Triglycerides	0.45	0.08	0.38	0.30 to 0.60	5.62	<0.001
HDL	-0.80	0.30	-0.19	-1.40 to -0.20	-2.70	0.008
SBP	0.25	0.15	0.10	-0.05 to 0.55	1.70	0.09

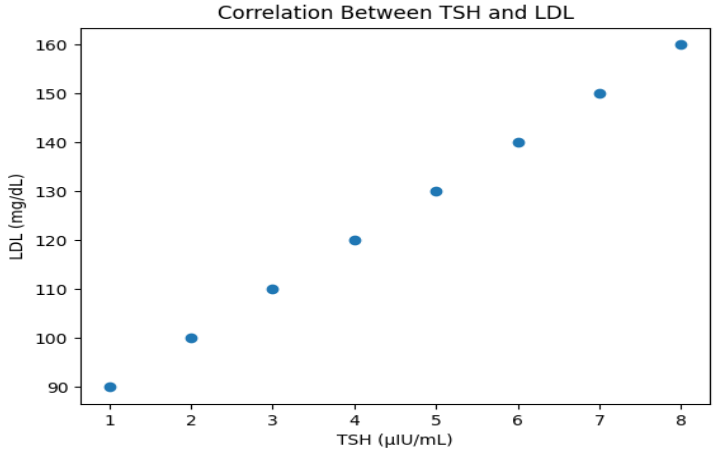

Figure 3: Correlation between TSH and LDL A moderate positive correlation (r = 0.39) was observed between TSH and LDL cholesterol.

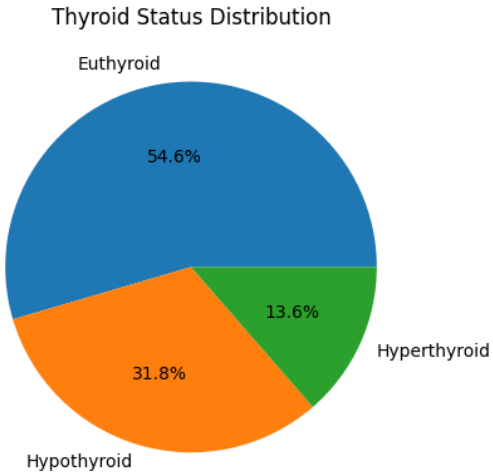

Figure 4: Thyroid Status Distribution Majority of participants were euthyroid, followed by hypothyroid and hyperthyroid groups.

The relationship between thyroid function and lipid parameters is further highlighted in Figure 3, which demonstrates a moderate positive linear correlation between TSH and LDL cholesterol (r = 0.39), suggesting that increasing TSH levels are associated with worsening lipid profiles. Additionally, Figure 4 depicts the distribution of thyroid status among study participants, with the majority being euthyroid, followed by hypothyroid and hyperthyroid groups.

Discussion

This study demonstrates a strong, statistically significant association between thyroid dysfunction—particularly hypothyroidism—and adverse cardiovascular risk profiles, aligning with global evidence from 2020–2025[12-15]. Hypothyroid participants exhibited markedly elevated total cholesterol (+50 mg/dL vs. euthyroid), LDL, triglycerides, BMI (+4.3 kg/m²), and systolic blood pressure (+14 mmHg), with ANOVA confirming inter-group differences ($p<0.001$). These findings corroborate recent cross-sectional data showing hypothyroidism linked to 58% higher odds of cardiometabolic syndrome components [16].

The positive correlations between TSH and lipids (r=0.42 for cholesterol, r=0.39 for LDL) and BMI (r=0.36) highlight TSH as an independent predictor, explaining ~41% of cholesterol variance in multivariate regression. This mirrors mechanistic insights where reduced thyroid hormone action impairs LDL receptor-mediated clearance and increases hepatic VLDL production. Hyperthyroid patients, while showing lower lipids, had elevated SBP and heart rate, consistent with increased cardiac output and reduced vascular resistance [17-19]. Subclinical hypothyroidism, comprising a substantial portion of the hypothyroid group, was associated with heightened risk, supporting meta-analyses indicating SCH elevates CVD events (RR 1.54 in <65 years) and all-cause mortality via dyslipidemia, hypertension, and endothelial dysfunction. Indian studies similarly link SCH to metabolic syndrome and CAD risk [20-21].

Blood pressure elevations in both hypo- and hyperthyroid groups reflect vascular effects: increased peripheral resistance in hypothyroidism and hyperdynamic circulation in hyperthyroidism.

Chi-square analysis confirmed thyroid status–hypertension association ($p<0.001$), aligning with 2025 data on TSH–DBP links in males [22-25].

Multifactorial pathways underpin these associations: dyslipidemia, endothelial dysfunction, arterial stiffness, altered glucose metabolism, and inflammation. Levothyroxine therapy improves lipids and reduces MACE in hypothyroid patients with CVD, suggesting therapeutic potential.

Clinical Implications: Routine thyroid screening in CVD risk assessment, especially in overweight or hypertensive adults, is warranted. Early levothyroxine for SCH (TSH ≥10 mIU/L) may mitigate risk, though benefits in mild cases require individualized evaluation. Lifestyle interventions (diet, exercise) complement management.

Limitations: Cross-sectional design precludes causality; single-center, modest sample; no longitudinal follow-up or advanced imaging (e.g., carotid IMT). Future prospective studies should address these.

Conclusion

Thyroid dysfunction, especially hypothyroidism and SCH, is significantly associated with adverse cardiovascular risk profiles characterized by dyslipidemia, hypertension, and obesity. TSH serves as a key biomarker. Integrated screening and management can reduce CVD burden.

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