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Original Research Article

Association between Thyroid stimulating hormone (TSH) and Lipid Profile among Adults Attending a Tertiary Care Hospital: A Cross-Sectional Study

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Abstract

Background: Hepatic cholesterol synthesis, lipoprotein clearance and turnover of triglycerides are regulated by thyroid hormones and mild thyroid dysfunction might be a contributing factor to atherogenic dyslipidemia. To evaluate the relationship between thyroid function tests and lipid profile in adult patients of a tertiary care hospital.

Materials and Methods: This was a cross-sectional study of 210 adults who were subjected to thyroid function test and fasting lipid estimation in a hospital setting. The participants were divided into three groups: euthyroid (n=139), subclinical hypothyroid (n=48), and overt hypothyroid (n=23), according to their serum thyroid-stimulating hormone (TSH) and free thyroxine (FT4). The serum total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C) were compared among thyroid categories. Correlation and multivariable linear regression analyses were performed.

Results: Mean age was 43.6±12.4 years and 61.0% were women. The mean TSH level was 181.2±30.1 mg/dL in euthyroid subjects, 208.6±34.8 mg/dL in subclinical hypothyroidism and 235.7±44.6 mg/dL in overt hypothyroidism ($p<0.001$). Corresponding LDL-C values were 108.4±27.7, 132.5±29.6, and 155.9±38.3 mg/dL ($p<0.001$). TSH correlated positively with TC ($r=0.41$), LDL-C ($r=0.46$), and TG ($r=0.30$), and negatively with HDL-C ($r=-0.24$) (all $p\leq0.001$). TSH was still independently associated with LDL-C (standardized $\beta=0.34$, $p<0.001$) after adjustment for age, sex and BMI. Dyslipidemia was present in 43.2%, 70.8%, and 87.0% of the three groups, respectively ($p<0.001$).

Conclusions: As biochemical severity of hypothyroidism increased, lipid parameters worsened, especially LDL-C, suggesting that thyroid status evaluation may be clinically helpful in adults with unexplained or refractory dyslipidemia.

Keywords: Thyroid-Stimulating Hormone; Hypothyroidism; Subclinical Hypothyroidism; Dyslipidemia; LDL Cholesterol; Lipid Profile; Cardiovascular Risk.

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Introduction

Thyroid dysfunction is one of the most frequent endocrine disorders in clinical practice in adults and often is not suspected unless biochemical screening is done. Indian multicentre data have shown that there is a significant burden of hypothyroidism, including subclinical and previously unrecognized cases, and that the prevalence of hypothyroidism is higher in women and older adults [1]. The clinical significance of this burden goes beyond the classic symptoms as thyroid hormones affect multiple pathways associated with cardiovascular risk, such as lipid transport and hepatic cholesterol handling. The expression of hepatic low-density lipoprotein (LDL) receptors, the synthesis of cholesterol, and formation of bile acids, activity of lipoprotein lipase, and mobilization and oxidation of fatty acids are all regulated by triiodothyronine and thyroxine. Hypothyroidism may result in decreased activity of LDL receptors and decreased clearance of

apolipoprotein-B containing particles, which can increase total cholesterol and LDL cholesterol, and may also cause an increase in triglyceride-rich lipoproteins [2]. These mechanisms provide a biologically plausible explanation for the well-recognized association between overt hypothyroidism and an atherogenic lipid profile.

In subclinical hypothyroidism, the relationship is not so simple, as the TSH is elevated and the thyroid hormone levels are within the reference range. In a North Indian cross-sectional study, the atherogenic lipid parameters were found to be increased in the patients with subclinical hypothyroidism as compared to the euthyroid controls but the magnitude of the changes was different for each lipid parameter [3]. In a later systematic review, which included only mild subclinical hypothyroidism, total cholesterol, LDL-C, and triglyceride levels were significantly higher, while HDL-C was significantly lower than in euthyroid patients [4].

More recent studies have confirmed the link, but also identified significant variation. One large study examining the association between subclinical hypothyroidism and blood lipids found that the association could be influenced by age, sex, obesity and other metabolic factors [5]. Even in the best matched cohorts, a 2023 meta-analysis revealed a significant increase in total cholesterol, triglycerides, and LDL-C in subclinical hypothyroidism [6].

Recent data also indicate that the association between thyroid indices and lipids is not linear. More recent data in adults with known dyslipidaemia suggest that there are measurable but weaker independent relationships between T4 and TSH with the conventional lipid parameters, suggesting that pre-existing metabolic disease may mask the role of thyroid status [7].

On the other hand, a 2026 study of subclinical hypothyroidism found that the levels of atherogenic lipids were higher, and that there was a correlation between TSH level and cardiovascular risk markers [8]. The results highlight the importance of studying TSH and free thyroid hormones as continuous variables rather than as categorical variables.

Although there is clinical overlap between dyslipidemia and thyroid dysfunction, hospital-based studies of the association between a full thyroid panel and lipid fractions have been inconsistent across populations. Observed associations may be affected by differences in iodine status, dietary habits, adiposity, medication use, and assay methods.

A biochemical correlation in routine tertiary care patients may facilitate secondary cause work-up of dyslipidaemia and guide rational thyroid screening. Hence, the present study was designed to evaluate the correlation of thyroid function tests with lipid profile in adult population visiting a tertiary care hospital and to see if the correlation of TSH with lipid profile parameters is independent of common demographic and anthropometric factors.

Materials and Methods

Study design: Cross sectional analytical study was conducted in the Department of General Medicine of a tertiary care teaching hospital over a period of 12 months. Adults who were referred for simultaneous thyroid function testing and fasting lipid evaluation were screened consecutively.

The minimum sample size was determined for the detection of a moderate correlation (r=0.22) between serum TSH and LDL-C with 80% power and a two-sided alpha of 0.05. A minimum of 200 participants was chosen, after considering incomplete laboratory records and subgroup comparisons. A total of 226 people were screened, and 16 were excluded for various reasons, leaving 210 adults for analysis.

Inclusion criteria: Adults (18-70 years) with complete TSH, free triiodothyronine (FT3), free thyroxine (FT4), total cholesterol (TC), triglyceride (TG), HDL-C and LDL-C measurements who had fasted for at least 8 hours. Exclusion criteria included: current thyroid hormone or antithyroid therapy, lipid-lowering medication in the last three months, pregnancy, diabetes mellitus requiring medication, nephrotic syndrome, chronic kidney disease stage 4 or 5, active liver disease, acute infection, systemic corticosteroid use, and known familial dyslipidemia. The exclusions were chosen to minimize the major secondary effects on lipid levels.

Clinical assessment: Age, sex, smoking, alcohol, medical history and medication exposure were documented on a standard proforma. Weight and height were taken while the participants were wearing light clothing and no shoes. The BMI was defined as weight (kg) divided by height (m2). Blood pressure was obtained after 5 minutes of sitting rest with the cuff of the appropriate size, and the average of two readings was taken.

Laboratory procedures and thyroid classification: Venous blood was drawn overnight after 8 hours of fasting between 8:00 and 10:00 AM. TSH, FT3 and FT4 were measured by an automated chemiluminescent immunoassay. Reference intervals were TSH 0.4-4.5 mIU/L, FT4 0.8-1.8 ng/dL, and FT3 2.3-4.2 pg/mL. Hypothyroidism was diagnosed when TSH was elevated and FT4 was in the normal range, and vice versa for hyperthyroidism. Subclinical hypothyroidism was defined as TSH >4.5 mIU/L

and FT4 within the normal range, and overt hypothyroidism was defined as TSH >4.5 mIU/L and FT4 <the lower reference limit. Those with biochemical hyperthyroidism were excluded as the study aim was within the hypothyroid spectrum.

Lipid measurements: TC and TG were measured enzymatically by colorimetric methods, HDL-C was measured by a direct homogeneous method, and LDL-C was measured by a direct method, if TG was greater than 400 mg/dL. Dyslipidemia was defined as the presence of any of the following: TC ≥200 mg/dL, LDL-C ≥130 mg/dL, TG ≥150 mg/dL, HDL-C <40 mg/dL in men or <50 mg/dL in women. All analyses were carried out in a single accredited laboratory using regular internal quality control.

Data analysis: SPSS version 26.0 was used for statistical analysis. The continuous variables were presented as mean ± SD and the categorical variables as frequency and percentage. The normality was checked using histogram and Shapiro-Wilk test. Continuous variables were compared among thyroid groups by one-way analysis of variance with Tukey post hoc testing and proportions were compared by chi-square testing. Pearson correlation was used to evaluate the correlation between lipid fractions and thyroid parameters. Independent associations of TSH with TC and LDL-C were assessed using multivariable linear regression models, adjusting for age, sex and BMI. Standardized beta coefficients with p-values were reported. A p value < 0.05 was deemed to be statistically significant for both sides.

Results

Among the 210 participants, mean age was 43.6±12.4 years; 128 (61.0%) were women and mean BMI was 25.8±4.2 kg/m². Based on thyroid biochemistry, 139 (66.2%) were euthyroid, 48 (22.9%) had subclinical hypothyroidism, and 23 (11.0%) had overt hypothyroidism. Participants with overt hypothyroidism were slightly older and had a higher mean BMI than euthyroid participants, although the sex distribution did not differ significantly. Baseline characteristics are summarized in Table 1.

A clear gradient in lipid abnormalities was observed across thyroid categories. Mean TC increased from 181.2±30.1 mg/dL in the euthyroid group to 208.6±34.8 mg/dL in subclinical hypothyroidism and 235.7±44.6 mg/dL in overt hypothyroidism (p<0.001). LDL-C showed a similar progression (108.4±27.7, 132.5±29.6, and 155.9±38.3 mg/dL; p<0.001). TG concentrations were also significantly higher with increasing severity of thyroid dysfunction, while HDL-C was lower in overt hypothyroidism. Dyslipidemia prevalence rose from 43.2% in euthyroid participants to 70.8% in subclinical and 87.0% in overt hypothyroidism (p<0.001) (Table 2).

When thyroid parameters were analyzed continuously, TSH showed moderate positive correlations with TC (r=0.41, p<0.001) and LDL-C (r=0.46, p<0.001), a weaker positive correlation with TG (r=0.30, p<0.001), and an inverse correlation with HDL-C (r=-0.24, p=0.001). FT4 was inversely correlated with TC (r=-0.35, p<0.001) and LDL-C (r=-0.39, p<0.001). FT3 demonstrated smaller inverse associations with TC and LDL-C. In adjusted regression models, TSH remained independently associated with LDL-C (standardized β=0.34, p<0.001) and TC (β=0.29, p<0.001), whereas the association with TG was attenuated after adjustment (Table 3).

Table 1: Demographic and thyroid characteristics according to thyroid status

Characteristic	Euthyroid (n=139)	Subclinical hypothyroid (n=48)	Overt hypothyroid (n=23)	p-value
Age (years)	41.8±11.7	45.6±12.5	50.4±13.1	0.004
Female sex, n (%)	82 (59.0)	31 (64.6)	15 (65.2)	0.69
BMI (kg/m²)	25.1±3.9	26.5±4.3	28.1±4.7	0.002
TSH (mIU/L)	2.18±0.93	7.21±2.16	24.8±13.7	<0.001
FT4 (ng/dL)	1.22±0.19	1.12±0.17	0.61±0.12	<0.001
FT3 (pg/mL)	3.21±0.47	3.06±0.45	2.47±0.39	<0.001

Values are mean±SD unless otherwise stated. BMI, body mass index; TSH, thyroid-stimulating hormone; FT4, free thyroxine; FT3, free triiodothyronine.

Table 2: Lipid profile and prevalence of dyslipidemia across thyroid categories

Parameter	Euthyroid (n=139)	Subclinical hypothyroid (n=48)	Overt hypothyroid (n=23)	p-value
Total cholesterol (mg/dL)	181.2±30.1	208.6±34.8	235.7±44.6	<0.001
LDL-C (mg/dL)	108.4±27.7	132.5±29.6	155.9±38.3	<0.001
Triglycerides (mg/dL)	139.1±50.4	165.7±55.6	208.3±75.1	<0.001
HDL-C (mg/dL)	45.7±9.1	41.8±8.5	35.4±8.4	<0.001
Dyslipidemia, n (%)	60 (43.2)	34 (70.8)	20 (87.0)	<0.001

LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol.

Table 3: Correlation and adjusted association of thyroid parameters with lipid measures

Analysis	Total cholesterol	LDL-C	Triglycerides	HDL-C
TSH, Pearson r	0.41 (p<0.001)	0.46 (p<0.001)	0.30 (p<0.001)	-0.24 (p=0.001)
FT4, Pearson r	-0.35 (p<0.001)	-0.39 (p<0.001)	-0.21 (p=0.003)	0.18 (p=0.009)
FT3, Pearson r	-0.19 (p=0.006)	-0.22 (p=0.001)	-0.12 (p=0.08)	0.10 (p=0.14)
Adjusted TSH β*	0.29 (p<0.001)	0.34 (p<0.001)	0.14 (p=0.036)	-0.16 (p=0.019)

***Standardized beta coefficient from linear regression adjusted for age, sex, and BMI.**

Discussion

The present study confirmed that there was a graded association between biochemical hypothyroidism and adverse lipid profile in adults attending tertiary care hospital. The main result was that the TC, LDL-C and TG levels increased with the progression from euthyroidism to subclinical and overt hypothyroidism, while HDL-C decreased. The magnitude and direction of these differences are similar to those known to occur with decreased thyroid hormone activity on lipoprotein synthesis and clearance. A thorough review of thyroid dysfunction and lipid metabolism has also highlighted that hypothyroidism has a consistent effect on increasing TC and LDL-C and less consistent effects on TG and HDL-C [9].

The clinical significance of our results is that subclinical hypothyroidism was linked to increased TC and LDL-C, which could be a subclinical stage that may not be clinically apparent and only be detected in lab tests. Previous pooled observational studies have shown that subclinical hypothyroidism is associated with higher levels of TC, LDL-C and TG than euthyroidism [10]. The present values reflect the same direction of effect and also indicate that the impact of dyslipidemia increases prior to the onset of overt FT4 deficiency. This is consistent with the notion that small increases in TSH are indicative of physiologically significant decreases in tissue thyroid hormone activity.

The high prevalence of dyslipidemia in the subclinical group is also consistent with a systematic review and meta-analysis of the lipid effects of subclinical hypothyroidism, which showed a consistent negative impact on the major atherogenic fractions [11].

However, the studies are diverse and not everyone with a mild elevation of TSH has clinically significant dyslipidaemia. Variation between studies is likely due to differences in age, adiposity, smoking, insulin resistance, iodine intake and duration of thyroid dysfunction. The relationship between TSH and LDL-C was not completely eliminated after adjustment for age, sex and BMI, suggesting that thyroid status itself was an independent factor in the lipid pattern observed.

This relationship is indirectly supported by the treatment literature. In a meta-analysis of randomized controlled trials, levothyroxine treatment in subclinical hypothyroidism resulted in significant decreases in TC and LDL-C, but not in HDL-C and TG [12]. A second systematic review that studied levothyroxine in overt and subclinical hypothyroidism also found that thyroid normalization led to improvements in some metabolic parameters [13]. The biological interpretation of this treatment effect is that at least some of the dyslipidemia that accompanies hypothyroidism is reversible, and not just a secondary phenomenon.

Thyroid hormone acts on genes for LDL receptor, hepatic cholesterol turnover, bile acid synthesis, and several genes that are involved in fatty-acid oxidation, mechanistically. Low thyroid hormone levels, therefore, will slow LDL clearance and may lead to an increase in the number of circulating apoB-containing particles. These mechanisms have been reviewed and the importance of hepatic thyroid hormone receptor pathways in cholesterol homeostasis emphasized [14]. This framework is consistent with our findings that LDL-C was the most highly correlated lipid parameter with TSH and remained the most consistent lipid parameter in adjusted analysis.

The results also corroborate with the large community-based observations. The Tehran Thyroid Study showed that there were variations in lipid concentrations between a large population samples with thyroid dysfunction, but the magnitude depended on the thyroid category and participant characteristics [15]. These population data indicate that the thyroid-lipid relationship does not exist only in highly selected endocrine clinics. This gradient was more pronounced in our tertiary-care cohort, possibly because of referral bias for metabolic abnormalities, and because the cohort included a separate group of overt hypothyroid patients.

There are clinical trials in subclinical hypothyroidism that have demonstrated that restoring euthyroidism lowers atherogenic lipoproteins, and may have a beneficial effect on surrogate measures of the vasculature. In a randomized, placebo-controlled study, Monzani et al. observed a beneficial effect of levothyroxine replacement on the lipid profile and on the intima-media thickness of the carotid artery [16]. A previous study by Caraccio et al. also showed that levothyroxine treatment improved the lipoprotein

parameters [17]. The studies reported here do not establish treatment benefit, but do offer a clinically plausible context for the association found here in the cross-sectional design.

One of the advantages of the present analysis was the evaluation of TSH and FT4 as continuous variables. Recent genetic and metabolomic studies indicate that even subtle changes in thyroid function within or outside the reference range may affect lipid traits and cardiovascular risk pathways [18]. FT4 was inversely related to TC and LDL-C in our cohort, further supporting that the pattern was not driven by arbitrary TSH cut-offs.

This ongoing partnership could be especially beneficial in patients with borderline blood tests or several other factors contributing to dyslipidaemia.

Practical aspects of the results include support for reciprocal clinical screening. Thyroid function testing should be considered in adults with newly detected hypothyroidism or unexplained LDL-C elevation or mixed dyslipidaemia. Thyroid dysfunction may be a secondary cause and this may affect the sequence of treatment, as lipid values may alter following thyroid normalization. In patients with a global cardiovascular risk that requires immediate intervention, however, lipid-lowering therapy should not be postponed.

There are some limitations to the study. Cross-sectional design does not provide temporality or causality, and the tertiary care setting may not be generalizable to community settings. Anti-thyroid peroxidase antibody, apolipoproteins, lipoprotein (a), insulin resistance, dietary intake, and detailed physical activity were not measured. Thyroid status was classified by a single biochemical measurement and transient elevation of TSH cannot be ruled out. Major secondary causes and lipid lowering therapy were not considered, but there is still potential for residual confounding. Future follow-up with repeated thyroid function tests and serial lipid measurements following treatment would help determine the reversibility of the dyslipidaemia.

Conclusion

In this tertiary-care adult population, thyroid dysfunction was significantly and progressively related to adverse lipid parameters. There was already a link between subclinical hypothyroidism and increased TC, LDL-C, and TG levels, and overt hypothyroidism was associated with the most significant abnormalities and the highest prevalence of dyslipidaemia. TSH was independent of age, sex, and BMI and still associated with LDL-C. Thyroid function testing may enhance the detection of a secondary cause of dyslipidemia, which is potentially reversible, in individuals with unexplained dyslipidemia.

References

1. Unnikrishnan AG, Kalra S, Sahay RK, Bantwal G, John M, Tewari N. Prevalence of hypothyroidism in adults: an epidemiological study in eight cities of India. Indian J Endocrinol Metab. 2013;17(4):647-652. doi: 10.4103/2230-8210.113755. PMID:23961480.
2. Duntas LH. Thyroid disease and lipids. Thyroid. 2002;12(4):287-293. doi:10.1089/10507250252949405. PMID:12034052.
3. Laway BA, War FA, Shah S, Misgar RA, Kotwal SK. Alteration of lipid parameters in patients with subclinical hypothyroidism. Int J Endocrinol Metab. 2014;12(3):e17496. doi:10.5812/ijem.17496. PMID:25237326.
4. Treister-Goltzman Y, Yarza S, Peleg R. Lipid profile in mild subclinical hypothyroidism: systematic review and meta-analysis. Minerva Endocrinol (Torino). 2021;46(4):428-440. doi: 10.23736/S2724-6507.20.03197-1. PMID:35078310.
5. Luo Y, Wu F, Huang Z, Gong Y, Zheng Y. Assessment of the relationship between subclinical hypothyroidism and blood lipid profile: reliable or not? Lipids Health Dis. 2022;21(1):137. doi:10.1186/s12944-022-01749-0. PMID:36514152.
6. Liu J, Chen Y, Ren B, et al. Alteration of lipid profile between subclinical hypothyroidism and well-matched controls: a meta-analysis. Horm Metab Res. 2023;55(7):479-486. doi:10.1055/a-2081-4599. PMID:37100404.
7. Rahimi Naiini M, Jafari A, Shahouzehi B. The association between T4 and TSH with lipid profile is attenuated in patients with established dyslipidemia. BMC Endocr Disord. 2026;26(1):180. doi:10.1186/s12902-026-02235-9. PMID:42057100.
8. Mawlood MS. The unseen impact of subclinical hypothyroidism on lipid profile and cardiovascular risk. Biochem Biophys Rep. 2026;46:102605. doi:10.1016/j.bbrep.2026.102605. PMID:42064580.
9. Rizos CV, Elisaf MS, Liberopoulos EN. Effects of thyroid dysfunction on lipid profile. Open Cardiovasc Med J. 2011;5:76-84. doi: 10.2174/1874192401105010076.
10. Liu XL, He S, Zhang SF, et al. Alteration of lipid profile in subclinical hypothyroidism: a meta-analysis. Med Sci Monit. 2014;20:1432-1441. doi:10.12659/MSM.891163. PMID:25124461.
11. Abreu IM, Lau E, de Sousa Pinto B, Carvalho D. Subclinical hypothyroidism: to treat or not to treat, that is the question! A systematic review with meta-analysis on lipid profile. Endocr Connect. 2017;6(3):188-199. doi:10.1530/EC-17-0028. PMID:28249936.
12. Li X, Wang Y, Guan Q, Zhao J, Gao L. The lipid-lowering effect of levothyroxine in

patients with subclinical hypothyroidism: a systematic review and meta-analysis of randomized controlled trials. Clin Endocrinol (Oxf). 2017;87(1):1-9. doi:10.1111/cen.13338. PMID:28342184.
13. Chen Y, Tai HY. Levothyroxine in the treatment of overt or subclinical hypothyroid ism: a systematic review and meta-analysis. Endocr J. 2020;67(7):719-732. doi:10.1507/en docrj.EJ19-0583. PMID:32238664.
14. Duntas LH, Brenta G. The effect of thyroid disorders on lipid levels and metabolism. Med Clin North Am. 2012;96(2):269-281. doi:10. 1016/j.mcna.2012.01.012.
15. Mehran L, Amouzegar A, Tohidi M, et al. Hypothyroidism and lipid levels in a community-based study (TTS). Int J Endocrinol Metab. 2016;14(2):e22827. doi: 10.5812/ijem.22827. PMID:27335579.
16. Monzani F, Caraccio N, Kozakowa M, et al. Effect of levothyroxine replacement on lipid profile and intima-media thickness in subclinical hypothyroidism: a double-blind, placebo-controlled study. J Clin Endocrinol Metab. 2004;89(5):2099-2106. doi:10.1210/j c.2003-031669. PMID:15126526.
17. Caraccio N, Ferrannini E, Monzani F. Lipoprotein profile in subclinical hypothyroidism: response to levothyroxine replacement, a randomized placebo-controlled study. J Clin Endocrinol Metab. 2002;87(4): 1533-1538. doi:10.1210/jcem.87.4.8378. PMI D:11932277.
18. Duntas LH, Brenta G. A renewed focus on the association between thyroid hormones and lipid metabolism. Front Endocrinol (Lausan ne). 2018;9:511. doi:10.3389/fendo.2018.00 511. PMID:30233497.